

Causal Inference Methods in Data Science

Lecture 5: Methods for dealing with unmeasured confounding

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IV

One motivating example

- Labor economists have long been interested in determining the causal effect of education on wage
- However, no randomized trials can be conducted to randomly assign people to or not to get higher education
- The only hope is to rely on observational studies
- Consider the following causal DAG:



- Obviously, if the data does not contain measurements of **ability** (almost impossible to measure it anyway), association between education and wage is not causation

One motivating example

- Labor economists have long been interested in determining the causal effect of education on wage
- However, no randomized trials can be conducted to randomly assign people to or not to get higher education
- The only hope is to rely on observational studies
- Instead, Card (1995) consider the following causal DAG:



- Can we identify $\tau_{E \rightarrow W}$, since $\tau_{B \rightarrow W}$ is composed of $\tau_{B \rightarrow E}$ and $\tau_{E \rightarrow W}$, and both causations, $\tau_{B \rightarrow W}$ and $\tau_{B \rightarrow E}$, are associations?

A real data analysis

let's analyze Card's data

Example 1

```
1 library(ivreg)
2 data("SchoolingReturns", package = "ivreg")
3
4 ## simple linear regression
5 edu_wage_ols <- lm(log(wage) ~ education + poly(experience,
6           2, raw = TRUE) + ethnicity + smsa + south, data =
7           SchoolingReturns)
8 summary(edu_wage_ols)
9
10 ## IV regression
11 edu_wage_iv <- ivreg(log(wage) ~ education + poly(experience
12           , 2, raw = TRUE) + ethnicity + smsa + south |
13           nearcollege + poly(age, 2, raw = TRUE) + ethnicity +
14           smsa + south, data = SchoolingReturns)
```

Another motivating example: the role of lipoprotein subfractions on heart diseases

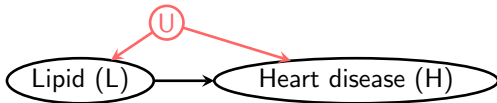
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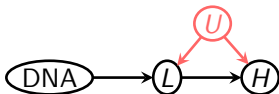
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- The causal DAG:



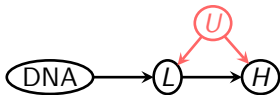
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- The IV “revolution” in genetics, led by **George Davey Smith** from University of Bristol

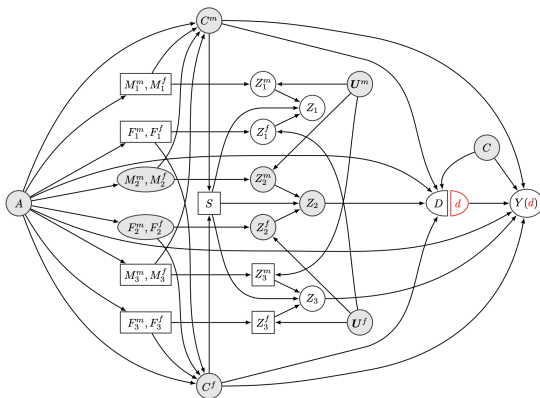


the random mating process roughly renders our DNA as a random variable, not influenced by other factors (not exactly though); maybe, based on biological knowledge, the particular mutation does not biologically affect our heart

ideal:

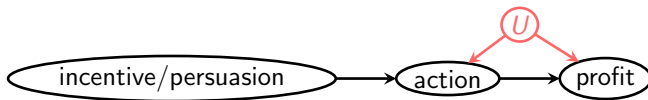


reality (REF: Almost exact Mendelian randomization)



Another motivating example: incentive or persuasion mechanism in behavior economics

- A central agent (e.g. Uber) may want to “manipulate” other agents (e.g. drivers and passengers) to increase utility
- However, the central agent cannot directly dictate what other agents do – the only thing the central agent can do is to provide incentive (e.g. money prize) or persuasion (e.g. revealing certain information of the states of the world)
- The incentive/persuasion itself may have no direct effect on the final utility
- The causal DAG



Another motivating example: effects of price on quantity

- IV was actually invented by Philip Wright (Sewell Wright's father) in 1928
- Wright wanted to study the effect of price on demand: e.g. to cut the smoking population by half, what the price of cigarette should have been?

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- Wright wanted to study the effect of price on demand: e.g. to cut the smoking population by half, what the price of cigarette should have been?
- The supply-demand model (from theoretical economics, possibly quite ideal):

$$\log Q = \beta_0 + \beta_1 \log P + U$$

U is not independent of $\log P$ (from economic theory, both determined by supply and demand curve), creating the problem of “endogeneity”

- Wright concluded to learn β_1 , one needs to find some extra information to solve this “endogeneity” problem

Some other real examples

- military lottery, actually military service/war experience, psychological health (famous Vietnam war study)
- randomly giving gifts, taking covid vaccine, risk of dying from covid
- randomly giving money to students doing less well in school, actually attending school with more enthusiasm, academic achievement (famous field experiments conducted by super-star economist Roland Fryer)
- etc.

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This story tells us:

- (1) Z causes A
- (2) No unmeasured confounding between Z and $\{A, Y\}$
- (3) Z causes Y only through A

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Z satisfying the above three assumptions is called an “Instrumental Variable” (IV); IV is simply an IMPERFECT INTERVENTION!

One analysis strategy: intention-to-treat (ITT) analysis

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- But do you think ITT analysis really answer our scientific question of interest?

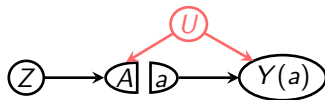
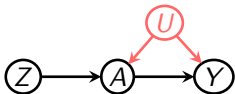
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- But do you think ITT analysis really answer our scientific question of interest?
- After this course, **DO NOT CONFUSE ITT ANALYSIS AS IF IT IS CAUSAL!**

The Instrumental Variable DAG/SWIG



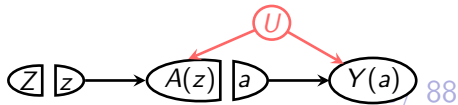
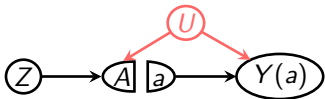
- From SWIG, one reads $Y(a) \perp\!\!\!\perp Z$ so $\mathbb{E}[Y(a)] = \mathbb{E}[Y(a)|Z]$ for all a

Core assumptions of IV

For simplicity we silence conditioning on the baseline confounders X

Z is an IV if

- relevance: $Z \not\perp\!\!\!\perp A$



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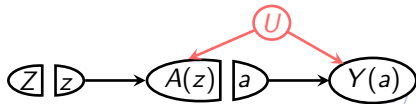
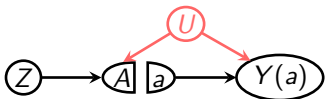
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- exogeneity (no unmeasured confounders between Z and A and between Z and Y):

$$Z \perp (A(z), Y(z, a)) \quad \forall a, z$$

[or can be relaxed to $Z \perp Y(z, a)$]



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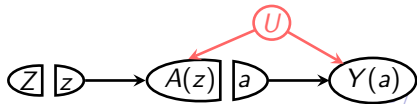
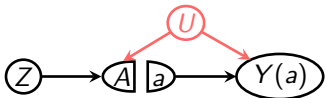
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- exclusion restriction (no direct effect from Z to Y):

$$Y(z, a) \equiv Y(a) \quad \forall a, z$$



IV point identification: Linear SEM illustration

Let's consider the following linear SEM related to the IV DAG/SWIG:
assuming U has $\mathbb{E}[U] = 0$

$$Y = \tau A + \eta U + \varepsilon_Y$$

$$A = \pi Z + \beta U + \varepsilon_A$$

$$Z = \varepsilon_Z,$$

$$\varepsilon_Y \perp\!\!\!\perp \varepsilon_A \perp\!\!\!\perp \varepsilon_Z \perp\!\!\!\perp U$$

Then

$$\mathbb{E}[A|Z] = \pi Z + \beta \mathbb{E}[U|Z] = \pi Z + \beta \mathbb{E}[U] = \pi Z$$

$$\mathbb{E}[Y|Z] = \tau \mathbb{E}[A|Z] + \eta \mathbb{E}[U|Z] = \tau \pi Z + \eta \mathbb{E}[U] = \tau \pi Z = \gamma Z$$

(called “reduced-form” regression in econometrics)

so

$$\tau = \frac{\gamma}{\pi}, \text{ assuming } \pi \neq 0$$

This is the so-called 2SLS estimator of ATE under linear IV setting

IV point identification: nonparametric result

Based on the three core IV assumptions, in particular $Y(a) \perp\!\!\!\perp Z$, can we identify $\mathbb{E}[Y(a)]$ or the ACE $\mathbb{E}[Y(1)] - \mathbb{E}[Y(0)]$?

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Consider binary instrument $Z \in \{0, 1\}$

Compliance table

subgroups	$A(z = 1)$	$A(z = 0)$
always taker	1	1
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Without non-compliance, by $Y(a) \perp\!\!\!\perp Z$,

$$\begin{aligned}\mathbb{E}[Y(a)] &= \mathbb{E}[Y(a)|Z = a] = \mathbb{E}[Y(a)|Z = a, A(z) = a] \\ &= \mathbb{E}[Y|Z = a, A = a]\end{aligned}$$

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With non-compliance, unidentified in general

$$\begin{aligned}\mathbb{E}[Y(a)] &= \mathbb{E}[Y(a)|Z = z] \\ \Rightarrow \mathbb{E}[Y(a)] &= \mathbb{E}[Y|Z = z, A = a] \\ &\quad + \underbrace{P(A(z) = 1 - a)}_{P(A=1-a|Z=z)} \underbrace{\{\mathbb{E}[Y(a)|Z = z, A = 1 - a] - \mathbb{E}[Y|Z = z, A = a]\}}_{\text{unidentified}}\end{aligned}$$

IV point identification: nonparametric result

Derivation:

$$\begin{aligned}\mathbb{E}[Y(a)] &= \mathbb{E}[Y(a)|Z = z] \\&= \mathbb{E}[Y(a)|Z = z, A(z) = a]P(A(z) = a|Z = z) \\&\quad + \mathbb{E}[Y(a)|Z = z, A(z) = 1 - a]P(A(z) = 1 - a|Z = z) \\&= \mathbb{E}[Y|Z = z, A = a]P(A = a|Z = z) \\&\quad + \mathbb{E}[Y(a)|Z = z, A(z) = 1 - a]P(A = 1 - a|Z = z) \\&= \mathbb{E}[Y|Z = z, A = a] - \mathbb{E}[Y|Z = z, A = a]P(A = 1 - a|Z = z) \\&\quad + \mathbb{E}[Y(a)|Z = z, A(z) = 1 - a]P(A = 1 - a|Z = z) \\&= \mathbb{E}[Y|Z = z, A = a] \\&\quad + P(A = 1 - a|Z = z)\{\mathbb{E}[Y(a)|Z = z, A = 1 - a] - \mathbb{E}[Y|Z = z, A = a]\}\end{aligned}$$

by far, we have used every IV conditions but we still have a non-identifiable counterfactual quantity $\mathbb{E}[Y(a)|Z = z, A(z) = 1 - a]$

A more essential way of understanding non-identifiability

The following strategy is always helpful: counting free parameters by taking everything to be $\{0, 1\}$ -valued

Since $A, Z \in \{0, 1\}^2$, we have only four possible values that can be calculated from the observed data $\mathbb{E}[Y|Z = 0, A = 0]$, $\mathbb{E}[Y|Z = 0, A = 1]$, $\mathbb{E}[Y|Z = 1, A = 0]$, $\mathbb{E}[Y|Z = 1, A = 1]$

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Then

$$\mathbb{E}[Y(a)] = \mathbb{E}[Y(a)|Z = z]$$

$$\begin{aligned} \Rightarrow \mathbb{E}[Y(a)] &= \mathbb{E}[Y|Z = z, A = a] \\ &\quad + \underbrace{P(A(z) = 1 - a)}_{P(A=1-a|Z=z)} \{ \mathbb{E}[Y(a)|Z = z, A = 1 - a] - \mathbb{E}[Y|Z = z, A = a] \} \end{aligned}$$

$$\begin{aligned} \Rightarrow \mathbb{E}[Y(1)] &= \mathbb{E}[Y|Z = z, A = 1] \\ &\quad + P(A = 0|Z = z) \{ \mathbb{E}[Y|Z = z, A = 0] + \tau - \mathbb{E}[Y|Z = z, A = 1] \} \end{aligned}$$

$$\begin{aligned} \mathbb{E}[Y(0)] &= \mathbb{E}[Y|Z = z, A = 0] \\ &\quad + P(A = 1|Z = z) \{ \mathbb{E}[Y|Z = z, A = 1] - \tau - \mathbb{E}[Y|Z = z, A = 0] \} \end{aligned}$$

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$\forall z \in \{0, 1\}$:

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$$\begin{aligned}\tau &= \mathbb{E}[Y(1)] - \mathbb{E}[Y(0)] \\ &= \underbrace{P(A = 0|Z = z)}_{1 - \mathbb{E}[A|Z=z]} \tau + \underbrace{P(A = 1|Z = 1 - z)}_{\mathbb{E}[A|Z=1-z]} \tau \\ &\quad + \underbrace{P(A = 0|Z = z)\mathbb{E}[Y|Z = z, A = 0] + P(A = 1|Z = z)\mathbb{E}[Y|Z = z, A = 1]}_{\mathbb{E}[Y|Z=z]} \\ &\quad - \mathbb{E}[Y|Z = 1 - z]\end{aligned}$$

So: ATE τ can be computed as 2SLS (two-stage least square)

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So: ATE τ can be computed as 2SLS (two-stage least square)

$$\tau = \frac{\mathbb{E}[Y|Z = z] - \mathbb{E}[Y|Z = 1 - z]}{\mathbb{E}[A|Z = z] - \mathbb{E}[A|Z = 1 - z]} = \frac{\text{second stage LS coefficient}}{\text{first stage LS coefficient}}$$

IV point identification: 1st attempt alternative derivation

(1) Causal structural assumption gives us $Y(a) \perp\!\!\!\perp Z$, implying

$$\mathbb{E}[(Y(0) - \mathbb{E}[Y(0)])h(Z)] = 0 \quad \forall h$$

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$$Y(0) = Y - \tau A$$

$$\Rightarrow \mathbb{E}[Y(0)] = \mathbb{E}[Y] - \tau \mathbb{E}[A]$$

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(3) Combining (1) + (2):

$$\begin{aligned} \mathbb{E}[(Y - \tau A - \mathbb{E}[Y] + \tau \mathbb{E}[A])h(Z)] &= 0 \\ \Rightarrow \tau &= \frac{\mathbb{E}[Yh(Z)] - \mathbb{E}[Y]\mathbb{E}[h(Z)]}{\mathbb{E}[Ah(Z)] - \mathbb{E}[A]\mathbb{E}[h(Z)]} = \frac{\text{Cov}(Y, h(Z))}{\text{Cov}(A, h(Z))} \end{aligned}$$

Choose $h(Z) = \mathbb{E}[A|Z]$ (first stage regression), we have

$$\begin{aligned} \tau &= \frac{\text{Cov}(Y, \mathbb{E}[A|Z])}{\text{Cov}(A, \mathbb{E}[A|Z])} \equiv \frac{\mathbb{E}[Y|Z=1] - \mathbb{E}[Y|Z=0]}{\mathbb{E}[A|Z=1] - \mathbb{E}[A|Z=0]} \\ &\quad \text{(two-stage least square (2SLS))} \end{aligned}$$

IV point identification: 2nd attempt, monotonicity assumption

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Ruling out defiers: what can we identify?

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$$\begin{aligned} P(A(1) > A(0)) &= P(A(1) = 1, A(0) = 0) \\ &= P(A(1) = 1) - P(A(1) = 1, A(0) = 1) \\ &= P(A(1) = 1) - P(A(0) = 1) \underbrace{P(A(1) = 1|A(0) = 1)}_{\equiv 1} \\ &= P(A(1) = 1) - P(A(0) = 1) \\ &= \mathbb{E}[A|Z = 1] - \mathbb{E}[A|Z = 0] \end{aligned}$$

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This important conceptual leap together with extremely impactful applications in labor economics wins the Nobel prize

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- Wang and Tchetgen Tchetgen 2018: “no unmeasured confounder-treatment interactions”

$$\mathbb{E}[Y(1) - Y(0)|U] = \mathbb{E}[Y(1) - Y(0)]$$

- In both cases, 2SLS helps identify ATE

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derivation under “no instrument-treatment interaction”: define mimicking counterfactual

$$\tilde{Y}(\gamma) := Y - \gamma \cdot A$$

by SNMM, we have $\mathbb{E}[\tilde{Y}(\gamma^*)|Z, A] = \mathbb{E}[Y(0)|Z, A]$

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$$\begin{aligned}\mathbb{E}[\tilde{Y}(\gamma^*)|Z] &= \mathbb{E}[\tilde{Y}(\gamma^*)] \\ \Rightarrow \mathbb{E}[(\tilde{Y}(\gamma^*) - \mathbb{E}[\tilde{Y}(\gamma^*)])h(Z)] &= 0, \forall h \\ \Rightarrow \mathbb{E}[(Y - \gamma^* \cdot A - \mathbb{E}[Y] + \gamma^* \mathbb{E}[A])Z] &= 0 \\ \Rightarrow \gamma^* &= \frac{\mathbb{E}[(Y - \mathbb{E}[Y])Z]}{\mathbb{E}[(A - \mathbb{E}[A])Z]}\end{aligned}$$

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- Economists strongly recommend to report the first-stage F-statistic whenever using 2SLS (simply output by every regression model in R)
- Convention: “if F-statistic is bigger than 10, one can safely use 2SLS”

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- What if we have multiple, say K endogenous exposures?
- In general, one needs to get at least one IV per endogenous exposure – in economics, this is called the “just-identified” case
- If you have less IVs than needed, it is called the “under-identified” case
- If you have more IVs than needed, it is called the “over-identified” case

What to do with many IVs?

- To illustrate the main idea, let's again consider the linear SEM:

$$Y = \tau A + \eta U + \varepsilon_Y$$

$$A = \pi^\top Z + \beta U + \varepsilon_A$$

with Z now a k -dimensional vector

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- Let's write down the n -sample version of the above linear SEM

$$\mathbf{Y}_{n \times 1} = \mathbf{A}_{n \times 1} \tau + \mathbf{U} \eta + \varepsilon_Y = \mathbf{A} \tau + \xi$$

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- Denote $P_Z = \mathbf{Z}(\mathbf{Z}^\top \mathbf{Z})^{-1} \mathbf{Z}^\top$, the following estimator is referred to as the 2SLS with the presence of many IVs

$$\hat{\tau}_{2SLS} = \frac{\mathbf{A}^\top P_Z \mathbf{Y}}{\mathbf{A}^\top P_Z \mathbf{A}} = \frac{\mathbf{A}^\top P_Z (\mathbf{A} \tau + \xi)}{\mathbf{A}^\top P_Z \mathbf{A}} = \tau + \underbrace{\frac{\mathbf{A}^\top P_Z \xi}{\mathbf{A}^\top P_Z \mathbf{A}}}_{\text{mean zero}}$$

Alternative popular estimator: Limited Information Maximum Likelihood (LIML)

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- It can be “roughly” viewed as a linear combination between OLS and 2SLS
- How to set λ ? LIML particularly chooses the following strategy: λ is the smallest root of the following equation

$$\det [(\mathbf{A} \ \mathbf{Y})_{2 \times n}^\top \{I - \lambda P_Z^\perp\} (\mathbf{A} \ \mathbf{Y})_{n \times 2}] = 0$$

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- When IVs are weak, it does not help to have many of them...
- Because otherwise, one could have generated so many random noises to serve as IVs to completely solve the endogeneity problem
- What happens when many IVs are weak? for simplicity, let's say $\mathbf{Z} \perp \mathbf{A}$ so we also have $\mathbf{Z} \perp \boldsymbol{\xi}$; we also have $\mathbb{E}[P_Z] \approx I$

$$\begin{aligned}\hat{\tau}_{2SLS} &= \tau + \frac{\mathbf{A}^\top P_Z \boldsymbol{\xi}}{\mathbf{A}^\top P_Z \mathbf{A}} \\ &\approx \tau + \frac{\mathbb{E}[\mathbf{A}^\top P_Z \boldsymbol{\xi}]}{\mathbb{E}[\mathbf{A}^\top P_Z \mathbf{A}]} \\ &= \tau + \frac{\mathbb{E}[\mathbf{A}^\top \mathbb{E}[P_Z] \boldsymbol{\xi}]}{\mathbb{E}[\mathbf{A}^\top \mathbb{E}[P_Z] \mathbf{A}]} = \tau + \frac{\mathbb{E}[\mathbf{A}^\top \boldsymbol{\xi}]}{\mathbb{E}[\mathbf{A}^\top \mathbf{A}]} \\ &\approx \tau + \frac{\mathbf{A}^\top \boldsymbol{\xi}}{\mathbf{A}^\top \mathbf{A}} = \hat{\tau}_{OLS}\end{aligned}$$

- People tend to view LIML as a more robust version of 2SLS under many weak IVs

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- A joke among economists: it takes an economist's life-time to find a good IV
- In practice, it is difficult to find IVs for a particular social science or economic problem
- But in clinical medicine and biology, IVs seem to be much easier to find, such as non-compliance in clinical trials
- And more recently, Mendelian randomization (MR) that makes biologists both happy and sad ...

Mendelian Randomization (MR): A natural IV

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- Probably not due to GWAS study we kind of know Z is associated with A : weak IV

General strategies for dealing with weak IVs in MR

- In general, assume linear treatment effect (not necessarily completely linear model)
- Weak IV ($A - Z$ weak dependence):
 - Filter out weak IVs by hypothesis testing using F -statistic:
Andrews, Stock, Sun Annual Reviews of Econometrics 2019

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 - Alternative modeling strategy by random effect model: Zhao, Chen, Wang, Small IJE 2019

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 - GENIUS: Sun, Tchetgen Tchetgen, Walter Stat. Sci. 2020

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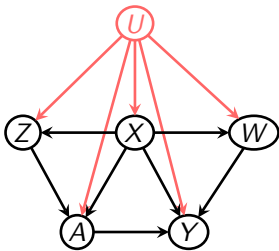


- Difficult to handle invalid IVs
- See [Li and Ye, 2022](#) for some recent progress on testing if the effects are zero

Proximal causal inference or negative controls

Proximal causal learning (motivated from negative control in experimental biology)

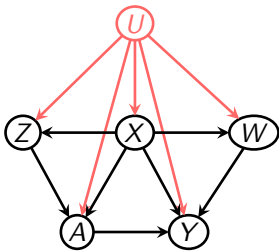
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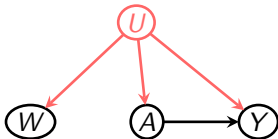
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In the above DAG, $\tau = \mathbb{E}[Y(1) - Y(0)]$ is point identifiable without modeling assumptions, but under some extra conditions

Application of proximal causal learning

- Genomics: CRISPR-Cas9 gene-perturbation experiments – often we do not know exactly
- Environmental health:
- Proxies can also be viewed as the measurements of the true underlying biological mechanisms

Proximal causal learning comes from negative control

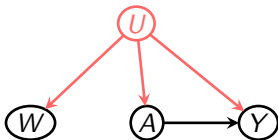


W : negative control outcome (NCO), not causally affected by A

Inspired from experimental biology: always compare to something that is known not to be affected by the chemical treatment

e.g. Y : death due to lung cancer, A : smoking, W : non-smoking related death (e.g. diabetes)

Proximal causal learning comes from negative control



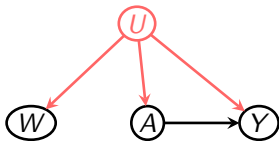
W : negative control outcome (NCO), not causally affected by A

Inspired from experimental biology: always compare to something that is known not to be affected by the chemical treatment

e.g. Y : death due to lung cancer, A : smoking, W : non-smoking related death (e.g. diabetes)

Intuition: any difference of W between $A = 1$ and $A = 0$ is due to U

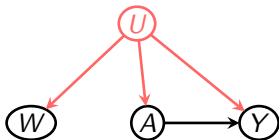
Illustration via linear models



$$\mathbb{E}[Y|A, U] = \beta_{AY}A + \beta_{UY}U$$

$$\mathbb{E}[W|A, U] = \beta_{UW}U$$

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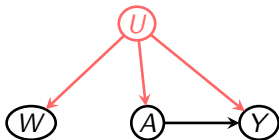
The above equations imply the following linear models over observables:

$$\mathbb{E}[Y|A] = \beta_{AY}A + \beta_{UY}\mathbb{E}[U|A]$$

$$\mathbb{E}[W|A] = \beta_{UW}\mathbb{E}[U|A]$$

$$\Rightarrow \mathbb{E}[Y|A] = \beta_{AY}A + \frac{\beta_{UY}}{\beta_{UW}}\mathbb{E}[W|A]$$

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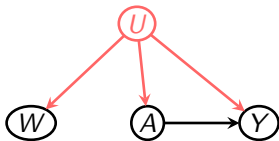
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When assuming $\frac{\beta_{UY}}{\beta_{UW}}$ is known, we can recover β_{AY}

Illustration via linear models



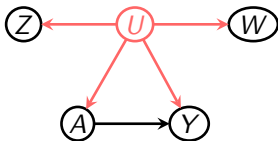
$$\mathbb{E}[Y|A, U] = \beta_{AY}A + \beta_{UY}U$$

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So NCO is quite like IV: helpful but not enough for point identification

50% of proximal causal learning: double negative control

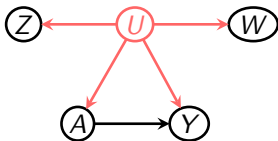
What if in addition we have a negative control treatment (NCT) Z ?



Q: Is Z a valid IV?

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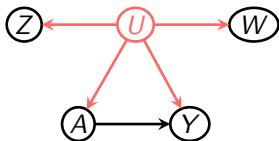
What if in addition we have a negative control treatment (NCT) Z ?



Q: Is Z a valid IV?

Obviously not

50% of proximal causal learning: double negative control



Implication on observables:

$$\mathbb{E}[Y|A, Z, U] = \beta_{AY}A + \beta_{UY}U$$

$$\mathbb{E}[W|A, Z, U] = \beta_{UW}U$$

$$\mathbb{E}[U|A, Z] = \beta_{AU}A + \beta_{ZU}Z$$

$$\mathbb{E}[Y|A, Z] = \beta_{AY}A + \beta_{UY}\mathbb{E}[U|A, Z]$$

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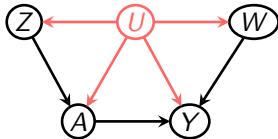
$$\Rightarrow \mathbb{E}[Y|A, Z] = \beta_{AY}A + \frac{\beta_{UY}}{\beta_{UW}}\mathbb{E}[W|A, Z]$$

Non-rigorously argue yourself why we do not need to know the value of $\frac{\beta_{UY}}{\beta_{UW}}$ when $\mathbb{E}[W|A, Z]$ does depend on Z .

So it is quite important that $\mathbb{E}[U|A, Z]$ varies with Z

100% of proximal causal learning except for X

In fact, we can further relax the setting

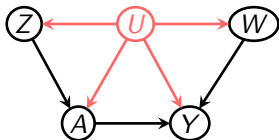


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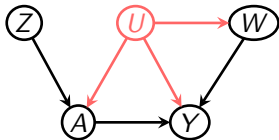
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$$\mathbb{E}[Y|A, Z, U] = \beta_{AY}A + \beta_{UY}U$$

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But what if



$$\mathbb{E}[Y|A, Z, U] = \beta_{AY}A + \beta_{UY}U$$

$$\mathbb{E}[W|A, Z, U] = \beta_{UW}U$$

Can you still argue $\mathbb{E}[U|A, Z]$ varies with Z ?

Nonparametric identification: confounding bridge & completeness

1. Confounding bridge: there exists a function $h(a, w)$ such that

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NOTE: Try to draw connections between these two assumptions and what we have done with linear model!

Proximal identification: Step 1

(1) “confounding bridge equation $\mathbb{E}[Y|Z, A] = \mathbb{E}[h(A, W)|Z, A]$ ” +
“exclusion restriction: $Y \perp\!\!\!\perp Z|U, A$ ”:

$$\begin{aligned}\mathbb{E}[\mathbb{E}[Y|U, A]|Z, A] &= \mathbb{E}[\mathbb{E}[Y|U, Z, A]|Z, A] = \mathbb{E}[Y|Z, A] \\ \Rightarrow \mathbb{E}[\mathbb{E}[Y|U, A]|Z, A] &= \mathbb{E}[h(A, W)|Z, A]\end{aligned}$$

Proximal identification: Step 2

(1) “confounding bridge equation $\mathbb{E}[Y|Z, A] = \mathbb{E}[h(A, W)|Z, A]$ ” +
“exclusion restriction: $Y \perp\!\!\!\perp Z|U, A$ ”:

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(2) “completeness: $\mathbb{E}[v(U)|Z, A] = 0 \Rightarrow v(U) = 0$ ” + “ $W \perp\!\!\!\perp Z, A|U$ ”

$$\begin{aligned}\mathbb{E}[\mathbb{E}[Y|U, A]|Z, A] &= \mathbb{E}[h(A, W)|Z, A] = \mathbb{E}[\mathbb{E}[h(A, W)|U, Z, A]|Z, A] \\ \Rightarrow \mathbb{E}[Y|U, A] &= \mathbb{E}[h(A, W)|U, Z, A] = \int h(A, w) \underbrace{f(w|U, Z, A)}_{\equiv f(w|U)} dw\end{aligned}$$

Proximal identification: Step 3

- (1) “confounding bridge equation $\mathbb{E}[Y|Z, A] = \mathbb{E}[h(A, W)|Z, A]$ ” +
“exclusion restriction: $Y \perp\!\!\!\perp Z|U, A$ ”:

$$\mathbb{E}[\mathbb{E}[Y|U, A]|Z, A] = \mathbb{E}[h(A, W)|Z, A]$$

- (2) “completeness: $\mathbb{E}[v(U)|Z, A] = 0 \Rightarrow v(U) = 0$ ” + “ $W \perp\!\!\!\perp Z, A|U$ ”

$$\mathbb{E}[Y|U, A] = \int h(A, w)f(w|U)dw$$

(3)

$$\begin{aligned}\mathbb{E}[Y(a)] &= \mathbb{E}[\mathbb{E}[Y(a)|U]] \\ &= \mathbb{E}[\mathbb{E}[Y|U, A = a]] \\ &= \int_u \mathbb{E}[Y|U = u, A = a]f(u)du \\ &\stackrel{(2)}{=} \int_u \int_w h(a, w)f(w|u)dwf(u)du \\ &= \int_u \int_w h(a, w)f(w, u)dwdu \\ &= \int_w h(a, w) \left\{ \int_u f(w, u)du \right\} dw \\ &= \int_w h(a, w)f(w)dw\end{aligned}$$

Proximal identification: Complete

- (1) “outcome bridge equation $\mathbb{E}[Y|Z, A] = \mathbb{E}[h(A, W)|Z, A]$ ” +
“exclusion restriction: $Y \perp\!\!\!\perp Z|U, A$ ”:

$$\mathbb{E}[\mathbb{E}[Y|U, A]|Z, A] = \mathbb{E}[h(A, W)|Z, A]$$

- (2) “completeness: $\mathbb{E}[v(U)|Z, A] = 0 \Rightarrow v(U) = 0$ ” + “ $W \perp\!\!\!\perp Z, A|U$ ”

$$\mathbb{E}[Y|U, A] = \int h(A, w)f(w|U)dw$$

- (3)

$$\mathbb{E}[Y(1)] = \mathbb{E}[\mathbb{E}[Y|U, A = 1]] = \int_w h(1, w)f(w)dw$$

Proximal identification: IPW form

- (1) “treatment bridge equation $\frac{1}{\mathbb{P}(A=1|W)} = \mathbb{E}[q(Z, A)|A = 1, W]$ ” +
“exclusion restriction: $W \perp\!\!\!\perp Z, A|U$ ”:

$$\mathbb{E} \left[\frac{1}{\mathbb{P}(A = 1|U)} | A = 1, W \right] = \mathbb{E}[q(Z, A)|A = 1, W]$$

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$$\frac{1}{\mathbb{P}(A = 1|U)} = \int q(z, A) f(z|U, A = 1) dz$$

- (3)

$$\mathbb{E}[Y(1)] = \mathbb{E} \left[\frac{AY}{\mathbb{P}(A = 1|U)} \right] = \mathbb{E} [Aq(Z, A)Y]$$

Naturally, two forms give us “doubly robust” proximal ATE identification

$$\mathbb{E}[Y(1)] = \mathbb{E} [Aq(Z, A)(Y - h(A, W)) + h(1, W)]$$

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- The origin of proximal causal learning is from the measurement error literature, in particular the work [Kuroki and Pearl, 2014](#)

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- It is possible to use techniques from causal graphical models to design algorithms to select valid proxies from data
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Other related frameworks

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- Examples: Difference-in-Difference, Synthetic Control, Regression Discontinuity, Multiple Treatments, Bespoke IV, Data Combination ... (study on your own if interested)

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- Athey & Imbens praised SC as “the most important innovation in the policy evaluation literature in the last 15 years”
- SC is designed to answer causal questions when we have the so-called “panel data” (longitudinal data in biostatistics)

Motivation of SC

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- Data: 1960 – 2003 GDP information for Germany and 16 other countries without such a reunification
- $Y_{1,t}, t = 1, \dots, T$: the GDPs for Germany
- $Y_{i,t}, i = 2, \dots, N; t = 1, \dots, T$: the GDPs for 16 other countries (untreated)
- T_0 : the year of reunification, so $Y_{1,t}$ is untreated when $t \leq T_0$, but treated when $t > T_0$

The data

- Downloadable from <https://doi.org/10.7910/DVN/24714>
- including information on: country, year, gdp, and other time-varying covariates

SC: linear model case

- Suppose the following linear SEM for Germany:

$$Y_{1,t} = \begin{cases} \tau_t + \alpha_1^\top U_t + \varepsilon_{1,t} & t > T_0 \\ \alpha_1^\top U_t + \varepsilon_{1,t} & t \leq T_0 \end{cases}$$

where U_t is a stochastic process that changes with t

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- Potential outcome & consistency assumption:

$$Y_{1,t} = \begin{cases} Y_{1,t}(0) = \alpha_1^\top U_t + \varepsilon_{1,t} & t \leq T_0 \\ Y_{1,t}(1) = Y_{1,t}(0) + \tau_t & t > T_0 \end{cases}$$

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- From the single time series alone, $\tau_t, t > T_0$ is not identified

Synthetic control by other countries

- Abadie then realized that we also have data from other untreated countries – can we do something similar to matching to create a hypothetical “Germany” that was never re-unified from the data

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- Under (1), we achieve **identification**: $t > T_0$

$$\begin{aligned} \tau_t &= \mathbb{E}[Y_{1,t}(1) - Y_{1,t}(0)] = \mathbb{E}[Y_{1,t}] - \alpha_1^\top \mathbb{E}[U_t] \\ &= \mathbb{E}[Y_{1,t}] - \sum_{i=2}^N w_i \alpha_i^\top \mathbb{E}[U_t] = \mathbb{E}[Y_{1,t}] - \sum_{i=2}^N w_i \mathbb{E}[Y_{i,t}] \end{aligned}$$

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- This observation leads to the following constrained least-square estimator of the weights:

$$\hat{\mathbf{w}} = \arg \min_{0 \leq \mathbf{w} \leq \mathbf{1}, \mathbf{1}^\top \mathbf{w} = 1} \frac{1}{T_0} \sum_{t=1}^{T_0} \left(Y_{1,t} - \sum_{i=2}^N w_i Y_{i,t} \right)^2$$

Germany reunification example

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- See “exercises.R”

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- The theoretical justification of the constrained least square methods is tricky – the noise term is correlated with the “regressors” in the model (because $Y_{i,t}$ is determined by $\varepsilon_{i,t}$)
- SC is also connected with matrix completion (the statistical problem that arises from the Netflix challenge)

$$\mathbf{Y} = \begin{pmatrix} \checkmark & \checkmark & \cdots & \checkmark & \checkmark & \checkmark \\ \checkmark & \checkmark & \cdots & \checkmark & \checkmark & \checkmark \\ \text{NA} & \checkmark & \cdots & \checkmark & \checkmark & \checkmark \\ \vdots & \vdots & \ddots & \vdots & \vdots & \vdots \\ \text{NA} & \checkmark & \cdots & \checkmark & \checkmark & \checkmark \end{pmatrix}$$

for more connections, see [Athey et al. '21](#) and [Amjad, Shah, Shen '19](#)

partial identification, nontrivial inequality
constraints and a first encounter of quantum
mechanics in causal inference

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- We will not cover the quantum mechanics part (read the materials if interested)

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- Trivia:

$$\begin{aligned}\tau &= \Pr(Y(1) = 1, A = 1) + \Pr(Y(1) = 1, A = 0) - \Pr(Y(0) = 1, A = 1) - \Pr(Y(0) = 1, A = 0) \\ &= \Pr(Y = 1, A = 1) - \Pr(Y = 1, A = 0) + \underbrace{\Pr(Y(1) = 1, A = 0)}_a - \underbrace{\Pr(Y(0) = 1, A = 1)}_b\end{aligned}$$

$$\Rightarrow \tau \begin{cases} \geq \Pr(Y = 1, A = 1) - \Pr(Y = 1, A = 0) - \Pr(A = 1) & a = 0, b \leq \Pr(A = 1) \\ \leq \Pr(Y = 1, A = 1) - \Pr(Y = 1, A = 0) + \Pr(A = 0) & a \leq \Pr(A = 0), b = 0 \end{cases}$$

$$\Leftrightarrow \tau \begin{cases} \geq -\Pr(Y = 0, A = 1) - \Pr(Y = 1, A = 0) \\ \leq \Pr(Y = 1, A = 1) + \Pr(Y = 0, A = 0) \end{cases}$$

Conclusion:

$$-\Pr(Y = 0, A = 1) - \Pr(Y = 1, A = 0) \leq \tau \leq \Pr(Y = 1, A = 1) + \Pr(Y = 0, A = 0)$$

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- Trivia:

$$\begin{aligned}\tau &= \Pr(Y(1) = 1, A = 1) + \Pr(Y(1) = 1, A = 0) - \Pr(Y(0) = 1, A = 1) - \Pr(Y(0) = 1, A = 0) \\ &= \Pr(Y = 1, A = 1) - \Pr(Y = 1, A = 0) + \underbrace{\Pr(Y(1) = 1, A = 0)}_a - \underbrace{\Pr(Y(0) = 1, A = 1)}_b\end{aligned}$$

$$\Rightarrow \tau \begin{cases} \geq \Pr(Y = 1, A = 1) - \Pr(Y = 1, A = 0) - \Pr(A = 1) & a = 0, b \leq \Pr(A = 1) \\ \leq \Pr(Y = 1, A = 1) - \Pr(Y = 1, A = 0) + \Pr(A = 0) & a \leq \Pr(A = 0), b = 0 \end{cases}$$

$$\Leftrightarrow \tau \begin{cases} \geq -\Pr(Y = 0, A = 1) - \Pr(Y = 1, A = 0) \\ \leq \Pr(Y = 1, A = 1) + \Pr(Y = 0, A = 0) \end{cases}$$

Conclusion:

$$-\Pr(Y = 0, A = 1) - \Pr(Y = 1, A = 0) \leq \tau \leq \Pr(Y = 1, A = 1) + \Pr(Y = 0, A = 0)$$

Width of this trivial bound is 1, so almost always covers 0

Can we do better than trivial bounds?

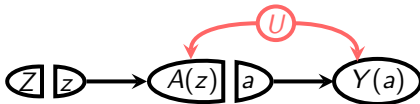
- Yes, using IV
Hernan, Robins. Instruments for Causal Inference: An Epidemiologist's Dream?

Can we do better than trivial bounds?

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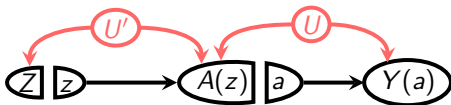
Hernan, Robins. Instruments for Causal Inference: An Epidemiologist's Dream?

- IV SWIG (intervening both Z and A simultaneously)



Can we do better than trivial bounds?

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Hernan, Robins. Instruments for Causal Inference: An Epidemiologist's Dream?
- In fact, for partial identification purpose, we can consider a more relaxed IV SWIG

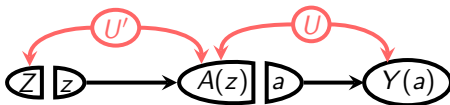


Can we do better than trivial bounds?

- Yes, using IV

Hernan, Robins. Instruments for Causal Inference: An Epidemiologist's Dream?

- In fact, for partial identification purpose, we can consider a more relaxed IV SWIG



- Even under the relaxed IV SWIG, we have
latent-variable exclusion restriction & exogeneity

$$\Pr(Y(z=1, a) = 1|U) = \Pr(Y(z=0, a) = 1|U), a \in \{0, 1\};$$
$$Z \perp\!\!\!\perp U; Y(z, a) \perp\!\!\!\perp Z, A(z)|U, a, z \in \{0, 1\}^2$$

Narrow it down using IV: Robins-Manski bounds

Marginalizing U , “relaxed” IV core becomes **marginal IV assumptions**

$$Y(z, a) \perp\!\!\!\perp Z, P(Y(1, a) = 1) = P(Y(0, a) = 1), a, z \in \{0, 1\}^2$$

Robins (1989) & Manski (1990) showed

1. When conditioning on the same z

$$\tau = \mathbb{E}[Y(1)] - \mathbb{E}[Y(0)] = \mathbb{E}[Y(1)|Z = z] - \mathbb{E}[Y(1)|Z = z] \Rightarrow$$

$$\tau \in \left[\begin{array}{c} \max_{z=0,1} \{ -\Pr(Y=0, A=1|Z=z) - \Pr(Y=1, A=0|Z=z) \}, \\ \min_{z=0,1} \{ \Pr(Y=1, A=1|Z=z) + \Pr(Y=0, A=0|Z=z) \} \end{array} \right]$$

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$$\begin{aligned} \tau &= \Pr(Y(1) = 1, A = 1|Z = z) + \Pr(Y(1) = 1, A = 0|Z = z) \\ &\quad - \Pr(Y(0) = 1, A = 1|Z = z') - \Pr(Y(0) = 1, A = 0|Z = z') \\ &= \Pr(Y = 1, A = 1|Z = z) - \Pr(Y = 1, A = 0|Z = z') \\ &\quad + \Pr(Y(1) = 1, A = 0|Z = z) - \Pr(Y(0) = 1, A = 1|Z = z') \\ &= \Pr(A = 1|Z = z) - \Pr(Y = 0, A = 1|Z = z) - \Pr(Y = 1, A = 0|Z = z') \\ &\quad + \Pr(Y(1) = 1, A = 0|Z = z) - \Pr(Y(0) = 1, A = 1|Z = z') \\ &= -\Pr(Y = 0, A = 1|Z = z) - \Pr(Y = 1, A = 0|Z = z') \\ &\quad + \Pr(A = 1|Z = z) + \Pr(Y(1) = 1, A = 0|Z = z) - \Pr(Y(0) = 1, A = 1|Z = z') \end{aligned}$$

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Narrow it down using IV: Robins-Manski bounds

Robins (1989) & Manski (1990)

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Robins-Manski bounds

$$\tau \in \left[\begin{array}{l} \max \left\{ \begin{array}{l} - \Pr(Y = 0, A = 1|Z = 1) - \Pr(Y = 1, A = 0|Z = 1), \\ - \Pr(Y = 0, A = 1|Z = 0) - \Pr(Y = 1, A = 0|Z = 0), \\ - \Pr(Y = 0, A = 1|Z = 1) - \Pr(Y = 1, A = 0|Z = 0) \\ \quad + \Pr(A = 1|Z = 1) - \Pr(A = 1|Z = 0), \\ - \Pr(Y = 0, A = 1|Z = 0) - \Pr(Y = 1, A = 0|Z = 1) \\ \quad + \Pr(A = 1|Z = 0) - \Pr(A = 1|Z = 1) \end{array} \right\}, \\ \min \left\{ \begin{array}{l} \Pr(Y = 1, A = 1|Z = 1) + \Pr(Y = 0, A = 0|Z = 1), \\ \Pr(Y = 1, A = 1|Z = 0) + \Pr(Y = 0, A = 0|Z = 0), \\ \Pr(Y = 1, A = 1|Z = 1) + \Pr(Y = 0, A = 0|Z = 0) \\ \quad + \underbrace{\Pr(A = 0|Z = 1) - \Pr(A = 0|Z = 0)}_{\Pr(A=1|Z=0) - \Pr(A=1|Z=1)}, \\ \Pr(Y = 1, A = 1|Z = 0) + \Pr(Y = 0, A = 0|Z = 1) \\ \quad + \underbrace{\Pr(A = 0|Z = 0) - \Pr(A = 0|Z = 1)}_{\Pr(A=1|Z=1) - \Pr(A=1|Z=0)} \end{array} \right\} \end{array} \right],$$

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Width of the above bounds?

$$\text{Width} \leq \underbrace{\Pr(A = 0|Z = 1) + \Pr(A = 1|Z = 0)}_{\text{sum of the probabilities of observed non-compliance}}$$

if $\Pr(A = 0|Z = 1) + \Pr(A = 1|Z = 0) \leq \min\{1, \Pr(A = 0|Z = 0) + \Pr(A = 1|Z = 1)\}$

Can we strengthen Robins-Manski bounds?

Assume the following instead

$$Y(z = 0, a = 0) = Y(z = 1, a = 0) = Y(0)$$

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Compare with Robins-Manski's assumption

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What are the differences?

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What are the differences? The new IV assumptions are cross-world and hence much stronger than the old assumptions!

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In fact, we have

cross-world IV assumptions \Rightarrow latent-variable IV assumptions \Rightarrow marginal IV assumptions

Balke-Pearl bounds: tightening Robins-Manski bounds with cross-world assumption

- Recall the derivation of Robins-Manski bounds:

$$\begin{aligned}\tau &= \Pr(Y(1) = 1, A = 1|Z = z) + \Pr(Y(1) = 1, A = 0|Z = z) \\ &\quad - \Pr(Y(0) = 1, A = 1|Z = z') - \Pr(Y(0) = 1, A = 0|Z = z') \\ &= \Pr(Y = 1, A = 1|Z = z) - \Pr(Y = 1, A = 0|Z = z') \\ &\quad + \Pr(Y(1) = 1, A = 0|Z = z) - \Pr(Y(0) = 1, A = 1|Z = z') \\ &= \Pr(A = 1|Z = z) - \Pr(Y = 0, A = 1|Z = z) - \Pr(Y = 1, A = 0|Z = z') \\ &\quad + \Pr(Y(1) = 1, A = 0|Z = z) - \Pr(Y(0) = 1, A = 1|Z = z') \\ &= -\Pr(Y = 0, A = 1|Z = z) - \Pr(Y = 1, A = 0|Z = z') \\ &\quad + \Pr(A = 1|Z = z) + \Pr(Y(1) = 1, A = 0|Z = z) - \Pr(Y(0) = 1, A = 1|Z = z') \\ &\geq -\Pr(Y = 0, A = 1|Z = z) - \Pr(Y = 1, A = 0|Z = z') \\ &\quad + \Pr(A = 1|Z = z) - \Pr(A = 1|Z = z')\end{aligned}$$

Seemingly quite hopeless to improve!

Balke-Pearl bounds: tightening Robins-Manski bounds with cross-world assumption

- But let's do a coupling argument! Choose $z, z', z'' = 0, 1, 0$ or $1, 0, 1$

$$\begin{aligned}\tau &= \Pr(Y(1) = 1) - \Pr(Y(0) = 1) \\&= \Pr(Y(1) = 1, Y(0) = 1|Z = z) + \Pr(Y(1) = 1, Y(0) = 0|Z = z) \\&\quad - \Pr(Y(0) = 1, Y(1) = 1|Z = z') - \Pr(Y(0) = 1, Y(1) = 0|Z = z'') \\&= \Pr(Y = 1, Y(0) = 1, A = 1|Z = z) + \Pr(Y(1) = 1, Y = 1, A = 0|Z = z) \\&\quad + \Pr(Y = 1, Y(0) = 0, A = 1|Z = z) + \Pr(Y(1) = 1, Y = 0, A = 0|Z = z) \\&\quad - \Pr(Y(0) = 1, Y = 1, A = 1|Z = z') - \Pr(Y = 1, Y(1) = 1, A = 0|Z = z') \\&\quad - \Pr(Y(0) = 1, Y = 0, A = 1|Z = z'') - \Pr(Y = 1, Y(1) = 0, A = 0|Z = z'') \\&\geq \Pr(Y = 1, A = 1|Z = z) + \underbrace{\Pr(Y(1) = 1, A = 0|Z = z)}_{\geq 0} \\&\quad - \Pr(Y = 1, A = 1|Z = z') - \Pr(Y = 1, A = 0|Z = z') \\&\quad - \Pr(Y = 0, A = 1|Z = z'') - \Pr(Y = 1, A = 0|Z = z'') \\&\geq \Pr(Y = 1, A = 1|Z = z) - \Pr(Y = 1|Z = z') \\&\quad - \Pr(Y = 0, A = 1|Z = z'') - \Pr(Y = 1, A = 0|Z = z'')\end{aligned}$$

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- By finessing the calculations for the blue and green terms, we also get

$$\begin{aligned}\tau \geq & \Pr(Y = 0, A = 0|Z = z) - \Pr(Y = 0|Z = z') \\ & - \Pr(Y = 0, A = 1|Z = z'') - \Pr(Y = 1, A = 0|Z = z'')\end{aligned}$$

- Upper bounds similar technique; omitted

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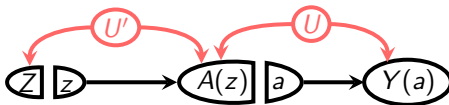
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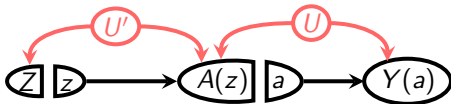


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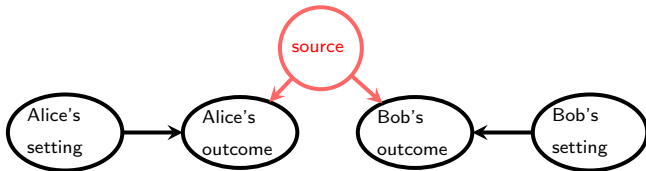
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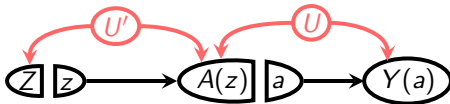


- Let's compare it with the DAG describing Bell-CHSH experiment:

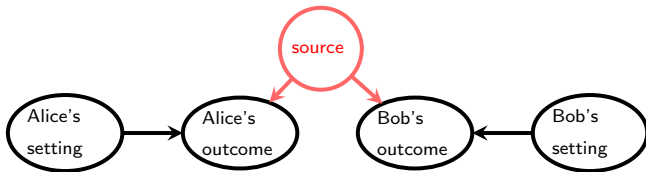


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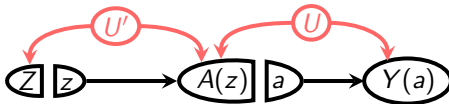


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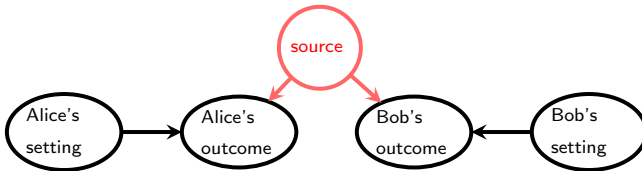


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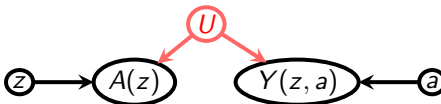
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- Mapping the notation a bit:



Bell-CHSH experiment

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Think about what statisticians usually do in practice: We always assume data are random draws from some stochastic process, e.g. regression model $Y = \beta X + \mathcal{N}(0, 1)$; but have you ever doubted why we cannot just develop data analysis methods for deterministic models? Are we statisticians fundamentally quantum? Not really

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Bell-CHSH experiment can be described as follows: Two particles are prepared. One particle X travels to Alice and the other Y travels to Bob, who are light years apart. Alice and Bob measure the particle spin along directions $z \in \{0, 1\}$ and $a \in \{0, 1\}$ and observe $A(z) \in \{0, 1\}$ and $Y(a) \in \{0, 1\}$

Bell-CHSH experiment

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Bell-CHSH experiment can be described as follows: Two particles are prepared. One particle A travels to Alice and the other Y travels to Bob, who are light years apart. Alice and Bob measure the particle spin along directions $z \in \{0, 1\}$ and $a \in \{0, 1\}$ and observe $A(z) \in \{0, 1\}$ and $Y(a) \in \{0, 1\}$

If “local realism” (i.e. existence of U) were true, then the correlation between Alice's outcome $A(z)$ and Bob's outcome $Y(a)$ must satisfy certain constraints, discovered by John Clauser, Michael Horne, Abner Shimony, and Richard Holt

CHSH-like inequality

Theorem 1 (CHSH-like inequality)

Z, A, Y are all $\{0, 1\}$ -valued. Under **latent-variable IV assumptions**

$$\begin{aligned} \Pr(Y(z = 1, a) = 1 | U) &= \Pr(Y(z = 0, a) = 1 | U), a \in \{0, 1\}; \\ Z &\perp U; Y(z, a) \perp Z, A(z) | U, a, z \in \{0, 1\}^2 \end{aligned}$$

we have

$$\begin{aligned} 0 &\leq \Pr(Y(z, a) = 1, A = 1 | Z = z) + \Pr(Y(z, 1 - a) = 0, A = 0 | Z = z) \\ &\quad + \Pr(Y(1 - z, a) = 0, A = 0 | Z = 1 - z) \\ &\quad - \Pr(Y(1 - z, 1 - a) = 0, A = 0 | Z = 1 - z) \leq 1 \end{aligned}$$

Bell experiment showed CHSH inequality can be violated; hence Bohr were right and Einstein were wrong – reality is non-local, God does play dice, and our world is intrinsically stochastic

What does CHSH-like inequality have to do with Balke-Pearl bounds?

Theorem 2 (Theorem 5.1 of F. Richard Guo's PhD thesis)

CHSH inequality closes the gap between Balke-Pearl and Robins-Manski bounds.

Proof.

Computer assisted proof. Balke-Pearl bounds can be derived symbolically using polytope optimization algorithms. In fact, one can set up and solve the following mathematical program:

$$\max_{\dots} \text{ or } \min_{\dots} \Pr(Y(z=0, a=1) = 1) - \Pr(Y(z=0, a=0) = 1)$$

s.t. trivial inequalities for prob., consistency, **marginal IV**, **CHSH inequality**

where \dots stands for parametrized variables, including

$\Pr(Y = y, A = a | Z = z)$ and $\Pr(A = a, Y(0,0) = y_{00}, Y(0,1) = y_{01}, Y(1,0) = y_{10}, Y(1,1) = y_{11} | Z = z)$. The solution to this program is in fact Balke-Pearl bounds



Summary

cross-world IV assumptions \Rightarrow latent-variable IV assumptions \Rightarrow marginal IV assumptions

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CHSH inequality

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\Downarrow

CHSH inequality + marginal IV assumptions

\Downarrow

Balke-Pearl

More references on partial identification using IVs

- REF: Balke, Pearl. Bounds on Treatment Effects from Studies with Imperfect Compliance. JASA 1997.
- REF: Swanson et al. Partial Identification of the Average Treatment Effect Using Instrumental Variables. JASA 2018.
- REF: Richardson, Robins. Analysis of the Binary Instrumental Variable Model. 2014.

Software

- R package [causaloptim](#)
- Learn how to use this package from <https://sachsmc.github.io/causaloptim/articles/example-code.html>
- Symbolic computation and directly giving you the formula of the bounds
- Including multiple IV bounds and outcome measurement error with proxies
- For some contrived applications in legal contexts, see [Tian and Pearl, 2000 UAI](#)

Next chapter

- Causal discovery and structure learning; some more causal graphs