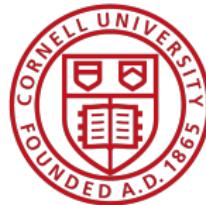


Causal Mediation in Natural Experiments

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Labour Work in Progress Seminar
6 March 2025

Introduction

Have you ever read an epidemiology/psychology/medicine paper's abstract, and seen claims of causal effects **mediated** through some mechanism?

[Family communication patterns, family environment, and the impact of parental alcoholism on offspring self-esteem \[PDF\]](#) sagepub

S Rangarajan, L Kelly - *Journal of Social and Personal ...*, 2006 - journals.sagepub.com

This study examined the role of perceptions of family environment and family communication as mediators of the effects of parental alcoholism on the self-esteem of adult children of alcoholics. Participants (N= 227) completed self-reports of parental alcoholism, family environment, family communication patterns (FCP), and self-esteem. Results indicated a negative relationship between the seriousness of both maternal and paternal alcoholism and self-esteem. Paternal and maternal alcoholism were related to the two dimensions of ...

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Introduction

Have you ever read an epidemiology/psychology/medicine paper's abstract, and seen claims of causal effects **mediated** through some mechanism?

[HTML] Persistent depressive symptomatology and inflammation: to what extent do health behaviours and weight control mediate this relationship?

[HTML] sciel

M Hamer, GJ Molloy, C de Oliveira... - Brain, Behavior, and ..., 2009 - Elsevier

We examined if persistent depressive symptoms are associated with markers of inflammation (C-Reactive Protein-CRP) and coagulation (fibrinogen), and if this association can be partly explained by weight control and behavioural risk factors (smoking, alcohol, physical activity).

The study sample included 3609 men and women (aged 60.5 ± 9.2 years) from The English Longitudinal Study of Ageing, a prospective study of community dwelling older adults.

Depressive symptoms (using the 8-item CES-D scale), health behaviours ...

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Introduction

- 1980s: Psychometrics defined mediation (distinct from moderation).
- 1920s: Application of early econometric path analysis (Wright 1928).
- 2020s: Popular in epidemiology, medicine, psychology.

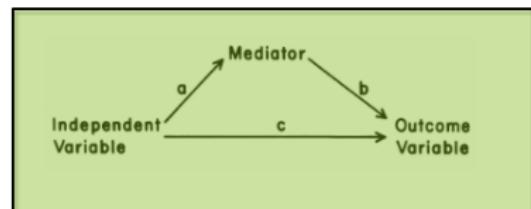
Figure: Baron Kelly (1986), p. 1176.

1176

REUBEN M. BARON AND DAVID A. KENNY

gression equation, as described by Cohen and Cohen (1983) and Cleary and Kessler (1982). So if the independent variable is denoted as X , the moderator as Z , and the dependent variable as Y , Y is regressed on X , Z , and XZ . Moderator effects are indicated by the significant effect of XZ while X and Z are controlled. The simple effects of the independent variable for different levels of the moderator can be measured and tested by procedures described by Aiken and West (1986). (Measurement error in the moderator requires the same remedies as measurement error in the independent variable under Case 2.)

The quadratic moderation effect can be tested by dichotomizing the moderator at the point at which the function is presumed to accelerate. If the function is quadratic, as in Figure 2, the effect of the independent variable should be greatest for those who are high on the moderator. Alternatively, quadratic moderation can be tested by hierarchical regression procedures described by Cohen and Cohen (1983). Using the same notation as in the previous paragraph, Y is regressed on X , Z , XZ , Z^2 , and XZ^2 . The test of quadratic moderation is given by the test



model, which recognizes that an active organism intervenes between stimulus and response, is perhaps the most generic formulation of a mediation hypothesis. The central idea in this model is that the effects of stimuli on behavior are mediated by various transformation processes internal to the organism. Theorists as diverse as Hull, Tolman, and Lewin shared a belief in the importance of postulating entities or processes that intervene between input and output. (Skinner's blackbox approach represents the notable exception.)

Introduction

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- 2020s: Popular in epidemiology, medicine, psychology.

Figure: Imai Keele Tingley Yamamoto (2010–).

A general approach to causal **mediation** analysis.

[K Imai, L Keele, D Tingley - Psychological methods, 2010 - psycnet.apa.org](#)

... Following prior work (eg, **Imai**, Keele, & Yamamoto, 2010; Pearl, 2001; Robins & Greenland...

mediation effects using the potential outcomes notation. We then review the key result of **Imai**...

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Identification, inference and sensitivity analysis for causal mediation effects

[K Imai, L Keele, T Yamamoto - 2010 - projecteuclid.org](#)

Causal mediation analysis is routinely conducted by applied researchers in a variety of disciplines. The goal of such an analysis is to investigate alternative causal mechanisms by ...

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Mediation: R package for causal mediation analysis

[D Tingley, T Yamamoto, K Hirose, L Keele... - Journal of statistical ..., 2014 - jstatsoft.org](#)

In this paper, we describe the R package mediation for conducting causal mediation analysis in applied empirical research. In many scientific disciplines, the goal of researchers ...

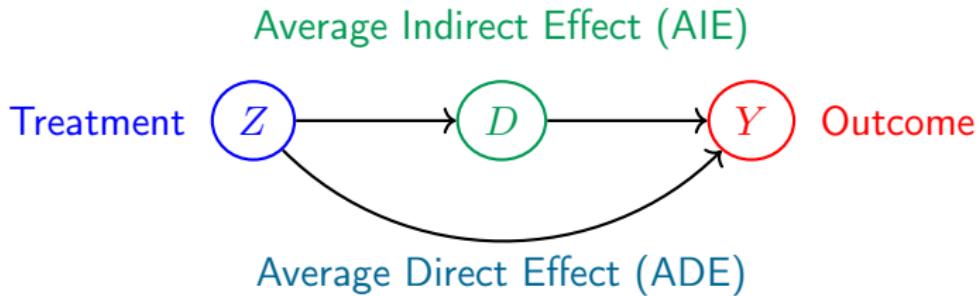
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Introduction:

1. [familiar] Causal design to estimate a treatment effect.



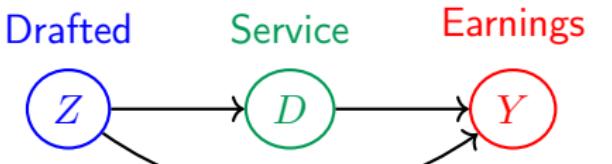
2. [unfamiliar] CM decomposes ATE along a mechanism pathway.



3. ATE \implies Average causal effect $Z \rightarrow Y$
- AIE \implies How much $Z \rightarrow Y$ effect through mediator D ?
- ADE \implies How much $Z \rightarrow Y$ effect is left over?

Introduction— CM Examples:

1. Lottery military draft 1969 (Angrist 1990).

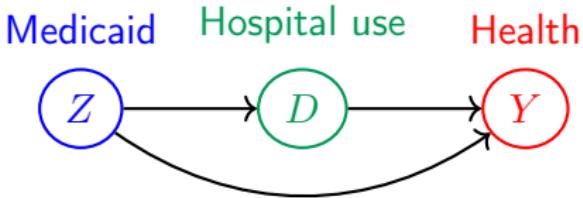
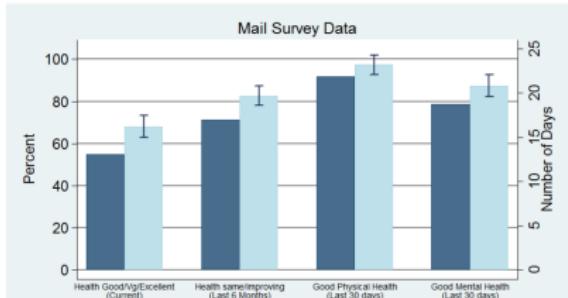


Draft avoidance (education deferment)

Note: IV further assumes direct = 0 (exclusion restriction).

2. Oregon health insurance experiment (Finkelstein+ 2009).

Medicaid Improves Self-Reported Health



All else (e.g., less worry)

Introduction

This project examines CM methods from an economic perspective:

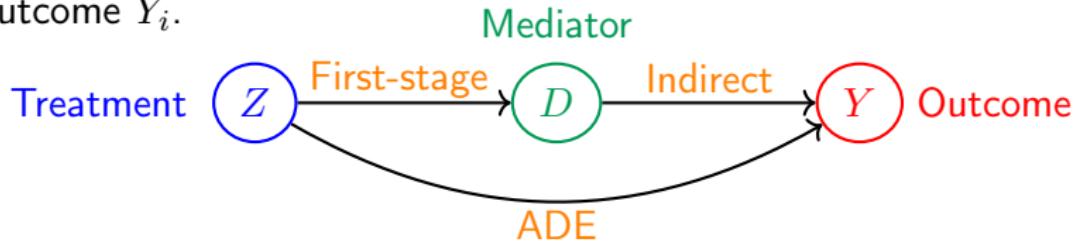
1. Problems with conventional, selection-on-observables, approach to CM in social science settings — including natural experiments.
[Negative result]
2. Recovering valid CM effects under selection-into-mediator, using a selection model.
[Positive result]

Brings together ideas from two different literatures:

- ▶ **Causal mediation.**
Baron Kelly (1986), Imai Keele Yamamoto (2010), Flores Flores-Lagunes (2009), Frölich Huber (2017), Huber (2020), Kwon Roth (2024).
- ▶ **Selection-into-treatment, selection models/MTEs.**
Roy (1951), Heckman (1979), Heckman Honore (1990), Florens Heckman Meghir Vytlacil (2008).

Direct & Indirect Effects — Model

Consider binary treatment $Z_i = 0, 1$, binary mediator $D_i = 0, 1$, and continuous outcome Y_i .



D_i is a function of Z_i :

$$D_i = Z_i D_i(1) + (1 - Z_i) D_i(0).$$

Y_i is a function of both Z_i, D_i :

$$Y_i = Z_i Y_i(1, D_i(1)) + (1 - Z_i) Y_i(0, D_i(0)).$$

Assume Z_i is ignorable, conditional on \mathbf{X}_i .

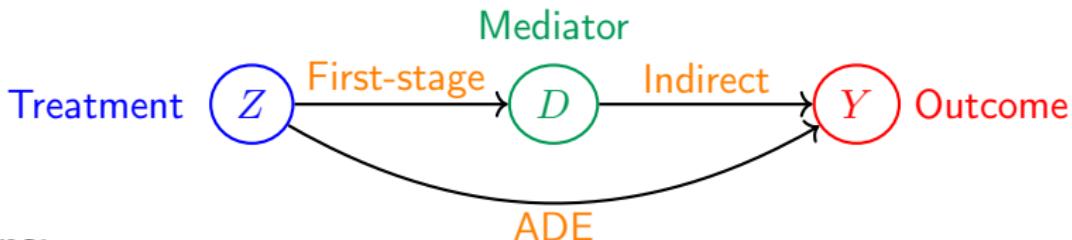
$$Z_i \perp\!\!\!\perp D_i(z), Y_i(z', d) \mid \mathbf{X}_i \text{ for } z, z', d = 0, 1.$$

Only two causal effects are identified so far.

ATE: $\mathbb{E}[Y_i(1, D_i(1)) - Y_i(0, D_i(0))] = \mathbb{E}[Y_i \mid Z_i = 1] - \mathbb{E}[Y_i \mid Z_i = 0]$

Average first-stage: $\mathbb{E}[D_i(1) - D_i(0)] = \mathbb{E}[D_i \mid Z_i = 1] - \mathbb{E}[D_i \mid Z_i = 0]$

Direct & Indirect Effects — Definitions



Definitions:

Average Direct Effect (ADE) : $\mathbb{E} [Y_i(1, D_i(Z_i)) - Y_i(0, D_i(Z_i))] ,$

Average Indirect Effect (AIE): $\mathbb{E} [Y_i(Z_i, D_i(1)) - Y_i(Z_i, D_i(0))] .$

- ▶ ADE is average effect $Z \rightarrow Y$, blocking the D path.
- ▶ AIE is causal effect of $D \rightarrow Y$, times number of $D(Z)$ compliers.¹

$$\text{AIE} = \mathbb{E} [D_i(1) - D_i(0)] \mathbb{E} [Y_i(Z_i, 1) - Y_i(Z_i, 0) | D_i(1) = 1, D_i(0) = 0] .$$

¹Assume mediator monotonicity to simplify notation.

Direct & Indirect Effects — Identification

Sequential ignorability (SI, Imai Keele Yamamoto 2010):

Assume mediator D_i is *also* ignorable, conditional on \mathbf{X}_i and Z_i realisation

$$D_i \perp\!\!\!\perp Y_i(z', d) \mid \mathbf{X}_i, Z_i = z', \text{ for } z', d = 0, 1.$$

If **SI** holds then ADE and AIE are identified by two-stage regression:

$$\text{ADE} = \mathbb{E} \left[\underbrace{\mathbb{E} [Y_i \mid Z_i = 1, D_i = d', \mathbf{X}_i] - \mathbb{E} [Y_i \mid Z_i = 0, D_i = d', \mathbf{X}_i]}_{\text{Second-stage regression, } Y_i \text{ on } Z_i \text{ holding } D_i, \mathbf{X}_i \text{ constant}} \right]$$

$$\begin{aligned} \text{AIE} = \mathbb{E} & \left[\underbrace{\left(\mathbb{E} [D_i \mid Z_i = 1, \mathbf{X}_i] - \mathbb{E} [D_i \mid Z_i = 0, \mathbf{X}_i] \right)}_{\text{First-stage regression, } D_i \text{ on } Z_i} \right. \\ & \times \left. \underbrace{\left(\mathbb{E} [Y_i \mid Z_i = z', D_i = 1, \mathbf{X}_i] - \mathbb{E} [Y_i \mid Z_i = z', D_i = 0, \mathbf{X}_i] \right)}_{\text{Second-stage regression, } Y_i \text{ on } D_i \text{ holding } Z_i, \mathbf{X}_i \text{ constant}} \right] \end{aligned}$$

Direct & Indirect Effects — Identification

Sequential ignorability (SI, Imai Keele Yamamoto 2010):

Assume mediator D_i is *also* ignorable, conditional on \mathbf{X}_i and Z_i realisation

$$D_i \perp\!\!\!\perp Y_i(z', d) \mid \mathbf{X}_i, Z_i = z', \text{ for } z', d = 0, 1.$$

E.g., OLS simultaneous regression (Imai Keele Yamamoto, 2010):

$$\begin{array}{ll} Z_i \leftarrow \text{Treatment} & \text{First-stage: } D_i = \phi + \pi Z_i + \psi'_1 \mathbf{X}_i + \eta_i \\ D_i \leftarrow \text{Mediator} & \text{Second-stage: } Y_i = \alpha + \beta D_i + \gamma Z_i + \delta Z_i D_i + \psi'_2 \mathbf{X}_i + \varepsilon_i \\ Y_i \leftarrow \text{Outcome} & \implies \text{ADE} = \gamma + \delta \mathbb{E}[D_i] \\ & \qquad \qquad \qquad \text{AIE} = \pi(\beta + \delta \mathbb{E}[Z_i]) \end{array}$$

i.e., a regression decomposition.

Other estimation methods do the same decomposition, avoiding linearity assumptions (see Huber 2020 for an overview).

Direct & Indirect Effects — Selection

⇒ Great, we can use the Imai Keele Yamamoto (2010) approach to CM all our respective applied projects.

⇒ Learn the mechanism pathways in causal research → big gain!

Before we join epidemiologists/psychologists/medical researchers in this conclusion, let us interrogate the **SI** assumption.

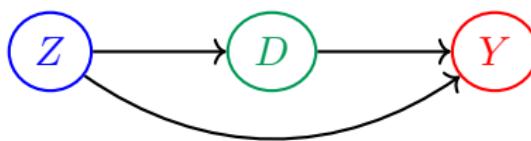
$$D_i \perp\!\!\!\perp Y_i(z', d) \mid X_i, Z_i = z', \text{ for } z', d = 0, 1.$$

Would this assumption hold true in settings that social scientists consider?

Return to the Oregon health insurance experiment (Finkelstein+ 2009).



Medicaid Hospital use Health

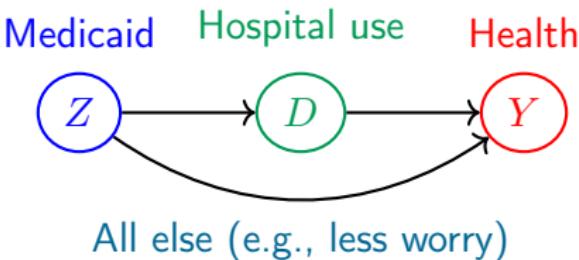


All else (e.g., less worry)

Direct & Indirect Effects — Selection

SI: $D_i \perp\!\!\!\perp Y_i(z', d) \mid X_i, Z_i = z', \text{ for } z', d = 0, 1.$

Oregon health insurance experiment (Finkelstein+ 2009).



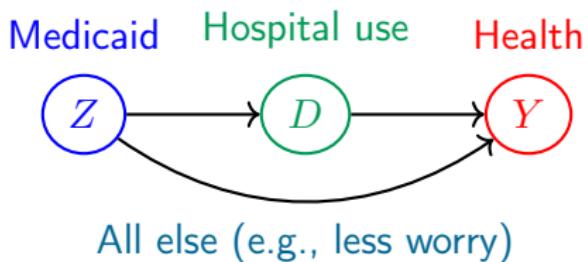
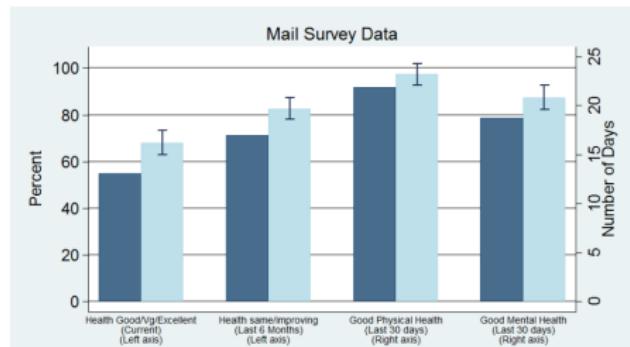
SI in this setting:

1. Medicaid assigned randomly (ensured by studying the 2008 Oregon waitlist lottery).
2. Hospital usage is quasi-random, conditional on Medicaid assignment Z_i and demographics X_i .

Direct & Indirect Effects — Selection

SI: Hospital usage is quasi-random, conditional on Medicaid assignment
 Z_i and demographics X_i .

Medicaid Improves Self-Reported Health



Consider the case individuals go to the hospital to maximise health.

$$D_i(z') = \mathbb{1} \left\{ \underbrace{Y_i(z', 1) - Y_i(z', 0)}_{\text{Benefits}} \geq \underbrace{C_i}_{\text{Costs}} \right\}, \quad \text{for } z' = 0, 1.$$

i.e., Roy (1951) selection into D_i .

Direct & Indirect Effects — Selection

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Theorem: If selection is Roy-style, and benefits are not 100% explained by Z_i, X_i , then **SI** does not hold.

Proof sketch: suppose D_i is ignorable \implies selection-into- D_i is explained 100% by $\{C_i, Z_i, X_i\}$, while unobserved gains explain 0%.

Direct & Indirect Effects — Selection

SI: Hospital usage is quasi-random, conditional on Medicaid assignment Z_i and demographics X_i .

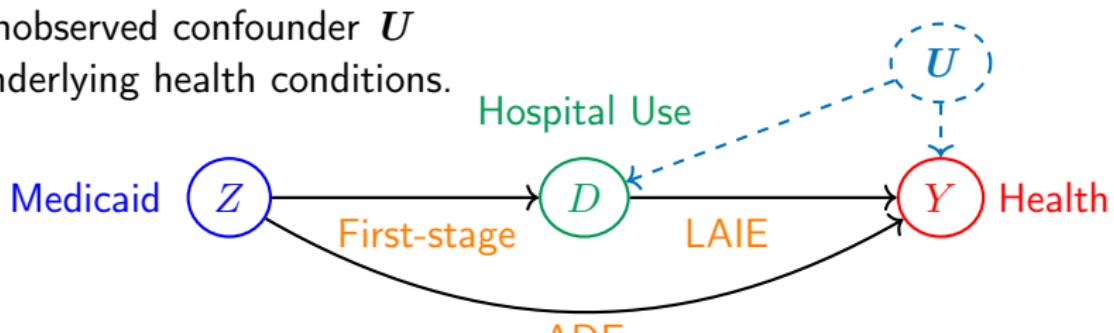
Consider the case **individuals go to the hospital** to maximise health.

$$D_i(z') = \mathbb{1} \left\{ \underbrace{Y_i(z', 1) - Y_i(z', 0)}_{\text{Benefits}} \geq \underbrace{C_i}_{\text{Costs}} \right\}, \quad \text{for } z' = 0, 1.$$

i.e., Roy (1951) selection into D_i .

⇒ unobserved confounder U

e.g., underlying health conditions.



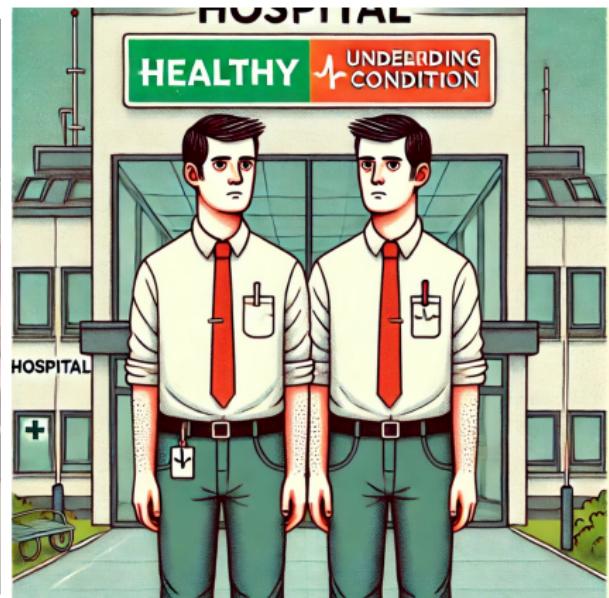
Direct & Indirect Effects — Selection

In practice, the only way to believe the **SI** assumption (selection-on-observables) is to study a case with another natural experiment for D_i — in addition to the one that guaranteed Z_i is ignorable.

(a) Cells in a lab → **SI** believable.



(b) People choosing healthcare → **SI** not.



Direct & Indirect Effects — Selection Bias

- ▶ What happens if you go ahead and estimate CM anyway?
 - ▶ Would this be problematic?
 - ▶ Estimating causal effects with an unobserved confounder is usually quite bad. . .
-

Definition: Selection bias (Heckman Ichimura Smith Todd, 1998).

Estimating $D \rightarrow Y$, if D not ignorable:

$$\begin{aligned} & \mathbb{E}[Y_i | D_i = 1] - \mathbb{E}[Y_i | D_i = 0] \\ &= \text{ATT} \\ &+ \underbrace{\left(\mathbb{E}[Y_i(., 0) | D_i = 1] - \mathbb{E}[Y_i(., 0) | D_i = 0] \right)}_{\text{Selection Bias}}. \end{aligned}$$

Direct & Indirect Effects — Selection Bias

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$$\begin{aligned} & \mathbb{E}[Y_i | D_i = 1] - \mathbb{E}[Y_i | D_i = 0] \\ &= \text{ATE} \\ &+ \underbrace{\left(\mathbb{E}[Y_i(., 0) | D_i = 1] - \mathbb{E}[Y_i(., 0) | D_i = 0] \right)}_{\text{Selection Bias}} \\ &+ \underbrace{\Pr(D_i = 0) (\text{ATT} - \text{ATU})}_{\text{Group-differences Bias}}. \end{aligned}$$

Direct & Indirect Effects — Selection Bias

⇒ CM Effects have this same flavour, causal effects contaminated by (less interpretable) bias terms.

$$\text{CM Estimand} = \text{ADE} + (\text{Selection Bias} + \text{Group difference bias})$$

$$\underbrace{\mathbb{E}_{D_i} [\mathbb{E}[Y_i | Z_i = 1, D_i] - \mathbb{E}[Y_i | Z_i = 0, D_i]]}_{\text{Estimand, Direct Effect}}$$

$$= \underbrace{\mathbb{E}[Y_i(1, D_i(Z_i)) - Y_i(0, D_i(Z_i))]}_{\text{Average Direct Effect}}$$

$$+ \underbrace{\mathbb{E}_{D_i} [\mathbb{E}[Y_i(0, D_i(Z_i)) | D_i(1) = d] - \mathbb{E}[Y_i(0, D_i(Z_i)) | D_i(0) = d]]}_{\text{Selection Bias}}$$

$$+ \underbrace{\mathbb{E}_{D_i} \left[\begin{aligned} & \left(1 - \Pr(D_i(1) = d) \right) \\ & \times \left(\mathbb{E}[Y_i(1, D_i(Z_i)) - Y_i(0, D_i(Z_i)) | D_i(1) = 1 - d] \right. \\ & \quad \left. - \mathbb{E}[Y_i(1, D_i(Z_i)) - Y_i(0, D_i(Z_i)) | D_i(0) = d] \right) \end{aligned} \right]}_{\text{Group difference bias}}$$

Direct & Indirect Effects — Selection Bias

⇒ CM Effects have this same flavour, causal effects contaminated by (less interpretable) bias terms. Put $\pi = \Pr(D_i(1) = 1, D_i(0) = 0)$.

$$\text{CM Estimand} = \text{AIE} + (\text{Selection Bias} + \text{Group difference bias})$$

$$\underbrace{\mathbb{E}_{Z_i} \left[\left(\mathbb{E}[D_i | Z_i = 1] - \mathbb{E}[D_i | Z_i = 0] \right) \times \left(\mathbb{E}[Y_i | Z_i, D_i = 1] - \mathbb{E}[Y_i | Z_i, D_i = 0] \right) \right]}_{\text{Estimand, Indirect Effect}}$$

$$= \underbrace{\mathbb{E}[Y_i(Z_i, D_i(1)) - Y_i(Z_i, D_i(0))]}_{\text{Average Indirect Effect}}$$

$$+ \underbrace{\pi \left(\mathbb{E}[Y_i(Z_i, 0) | D_i = 1] - \mathbb{E}[Y_i(Z_i, 0) | D_i = 0] \right)}_{\text{Selection Bias}}$$

$$+ \pi \left[\left(1 - \Pr(D_i = 1) \right) \begin{pmatrix} \mathbb{E}[Y_i(Z_i, 1) - Y_i(Z_i, 0) | D_i = 1] \\ - \mathbb{E}[Y_i(Z_i, 1) - Y_i(Z_i, 0) | D_i = 0] \end{pmatrix} \right.$$

$$\left. + \left(\frac{1 - \Pr(D_i(1) = 1, D_i(0) = 0)}{\Pr(D_i(1) = 1, D_i(0) = 0)} \right) \begin{pmatrix} \mathbb{E}[Y_i(Z_i, 1) - Y_i(Z_i, 0) | D_i(1) = 0 \text{ or } D_i(0) = 1] \\ - \mathbb{E}[Y_i(Z_i, 1) - Y_i(Z_i, 0)] \end{pmatrix} \right]$$

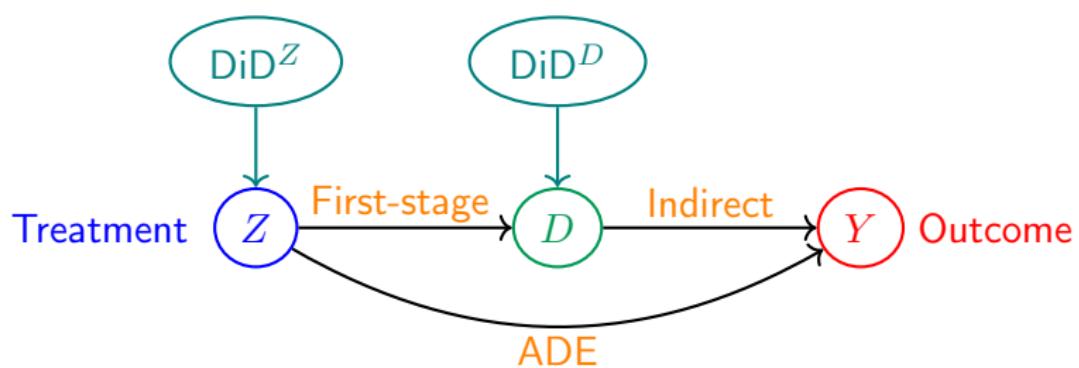
$$\text{Groups difference Bias}$$

Identification Under Selection

That was a long way of giving negative results. Is there any hope?

If you can use a two-way research design, then please do!

Figure: Two-way Diff-in-Diff (see Deuchert Huber Schelker, 2019).



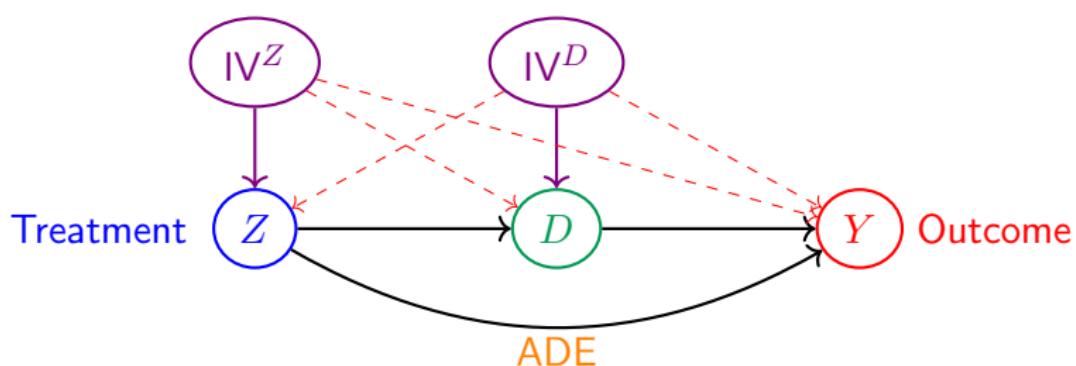
Note: assumes common trends across complier groups, identifies ADE + AIE local to complier groups.

Identification Under Selection

That was a long way of giving negative results. Is there any hope?

If you can use a two-way research design, then please do!

Figure: Two-way IV (see Frölich Huber, 2017).



Note: two-way exclusion restriction, identifies ADE + AIE local to overlapping complier groups. Also avoid 2SLS (see Kim 2025)!

Identification Under Selection

That was a long way of giving negative results. Is there any hope?

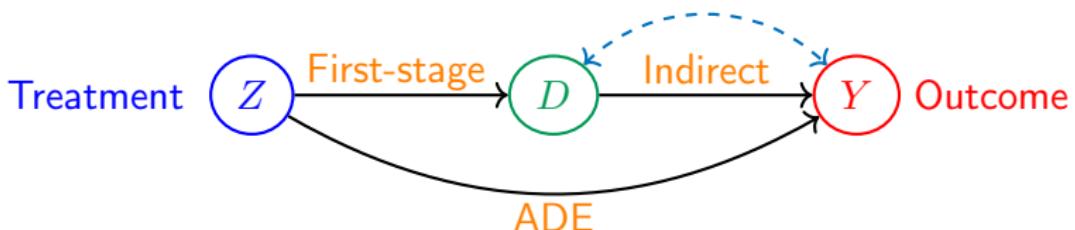
What about the mainstream case, with research design for only Z ?
How do economists do causal effects in these systems?

1. Estimate the ATE, and call it a day.
2. (optional) Present suggestive evidence of mechanisms. . . . ▶ suggestive

Overview of a selection model solution to identification.

Identification with a Selection Model

Suppose Z is ignorable, D is not, so we have the following causal model.



Put $\mu_{d'}(z'; \mathbf{X}_i) = \mathbb{E}[Y_i(z', d') | \mathbf{X}_i]$, and $U_{d'}$ the corresponding errors.

$$Y_i(Z_i, 0) = \mu_0(Z_i; \mathbf{X}_i) + U_{0,i}, \quad Y_i(Z_i, 1) = \mu_1(Z_i; \mathbf{X}_i) + U_{1,i}.$$

Then this system has the following regression equations:

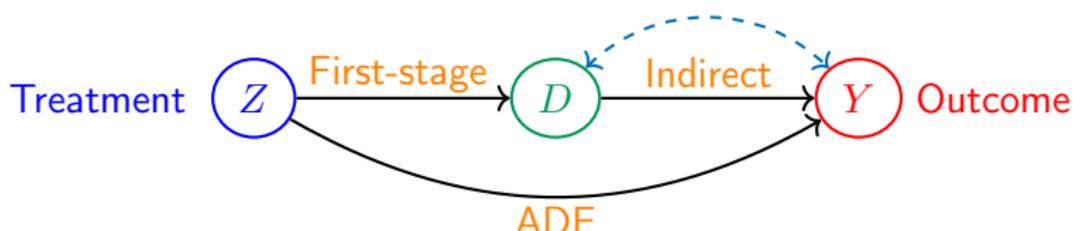
$$D_i = \phi + \pi Z_i + \varphi(\mathbf{X}_i) + \eta_i$$

$$Y_i = \alpha + \beta D_i + \gamma Z_i + \delta Z_i D_i + \zeta(\mathbf{X}_i) + \underbrace{(1 - D_i) U_{0,i} + D_i U_{1,i}}_{\text{Correlated error term.}}$$

Where β, γ, δ comprise the ADE and AIE.

Identification with a Selection Model

Suppose Z is ignorable, D is not, so we have the following causal model.



Then this system has the following regression equations:

$$D_i = \phi + \pi Z_i + \varphi(\mathbf{X}_i) + \eta_i$$

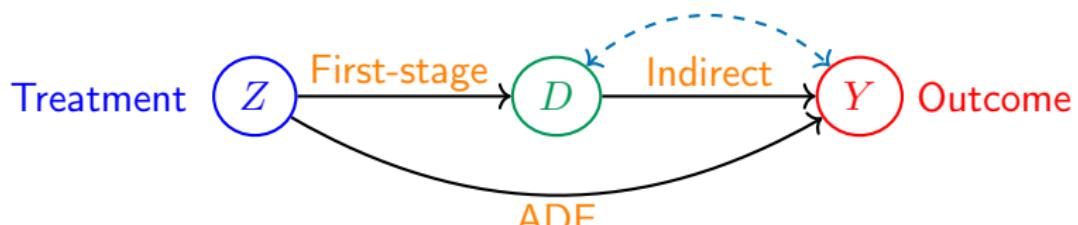
$$Y_i = \alpha + \beta D_i + \gamma Z_i + \delta Z_i D_i + \zeta(\mathbf{X}_i) + \underbrace{(1 - D_i) U_{0,i} + D_i U_{1,i}}_{\text{Correlated error term.}}$$

Where β, γ, δ comprise the ADE and AIE.

⇒ Identify second-stage (despite correlated error term).

Identification with a Selection Model

Suppose Z is ignorable, D is not, so we have the following causal model.



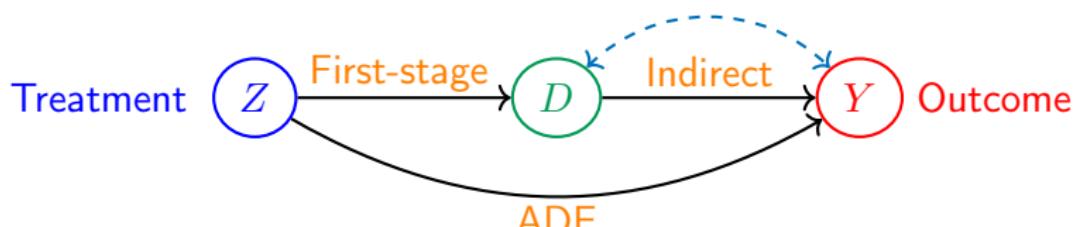
Note that Roy selection has first-stage errors correlated with second-stage errors.

$$D_i = \mathbb{1} \left\{ Z_i(\delta + \beta) + (1 - Z_i)\beta - C_i \geq -\left(U_{1,i} - U_{0,i} \right) \right\}$$

$$Y_i = \alpha + \beta D_i + \gamma Z_i + \delta Z_i D_i + \zeta(\mathbf{X}_i) + \underbrace{(1 - D_i) U_{0,i} + D_i U_{1,i}}_{\text{Correlated error term.}}$$

Identification with a Selection Model

Suppose Z is ignorable, D is not, so we have the following causal model.

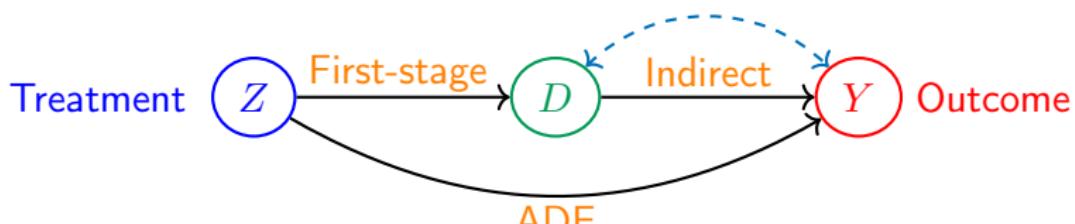


Heckman (1979) Selection model, assumptions:

1. Mediator monotonicity $\Pr(D_i(1) \geq D_i(0) | \mathbf{X}_i) = 1$.
 2. First-stage errors inform second-stage heterogeneity,
 $\text{Corr}[\eta_i, (1 - D_i) U_{0,i} + D_i U_{1,i}] > 0$.
 3. Error-term distribution $\eta_i, U_{0,i}, U_{1,i} \sim \text{Normal}(\mathbf{M}, \boldsymbol{\Sigma})$.
- ⇒ identifies second-stage, and thus ADE + AIE.

Identification with a Selection Model

Suppose Z is ignorable, D is not, so we have the following causal model.



Semi-parametric control function (Newey Imbens 2009), assumptions:

1. Mediator monotonicity $\Pr(D_i(1) \geq D_i(0) | \mathbf{X}_i) = 1$.
2. First-stage errors inform second-stage heterogeneity,
 $\text{Corr}[\eta_i, (1 - D_i) U_{0,i} + D_i U_{1,i}] > 0$.
3. Valid instrument for D_i .

⇒ identifies second-stage, and thus ADE + AIE (without unrealistic error distribution assumption).

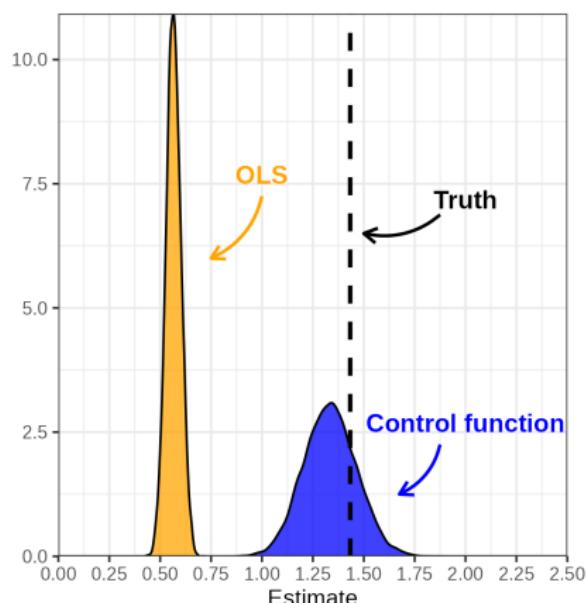
► MTEs

Simulation evidence

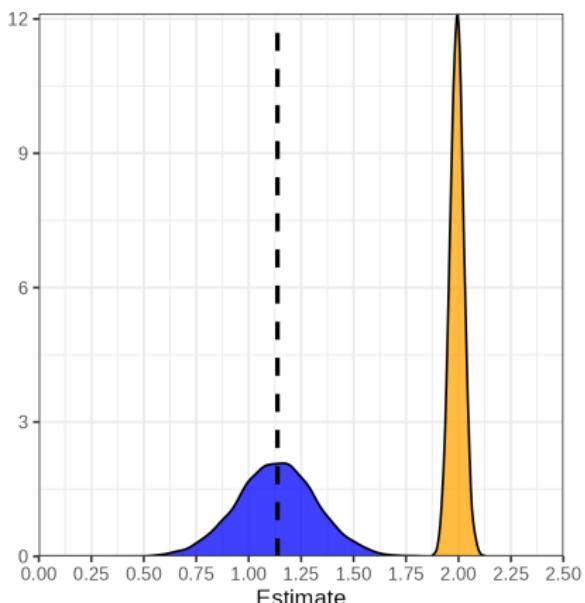
Simulation with Roy selection, trivariate normal errors, unobserved costs.

Figure: Simulated Distribution of CM Effect Estimates.

(a) ADE.



(b) AIE.

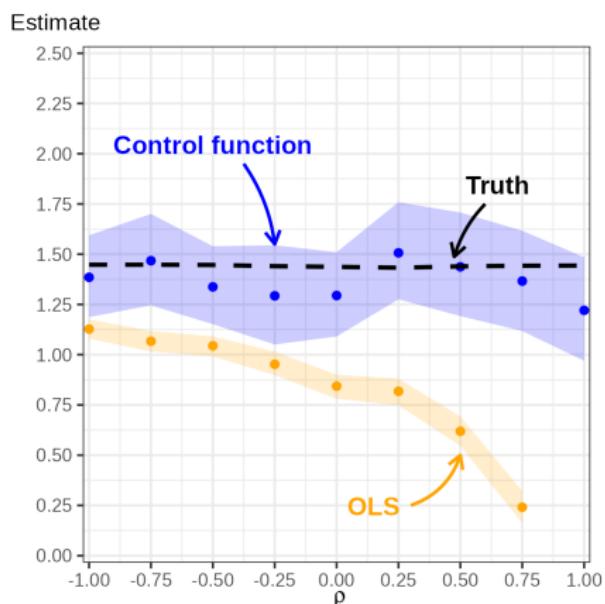


Simulation evidence

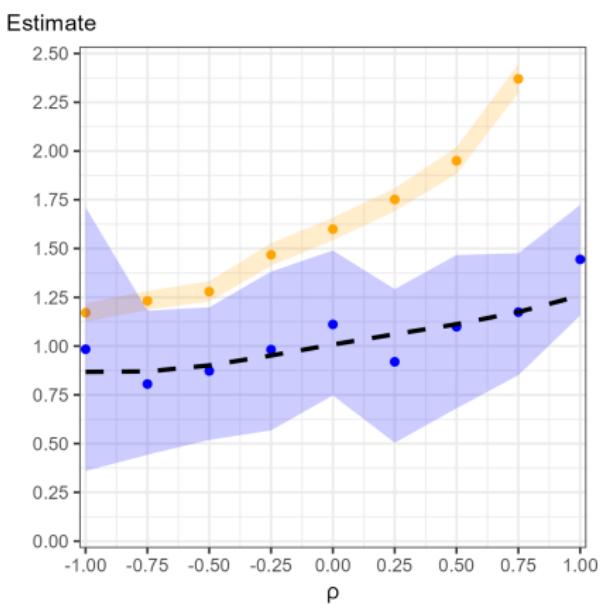
Simulation with Roy selection, trivariate normal errors, unobserved costs.

Figure: Point Estimates of CM Effects, OLS versus Control Function, varying ρ values with $\sigma_0 = 1, \sigma_1 = 2$ fixed.

(a) ADE.



(b) AIE.



Conclusion

Overarching goals:

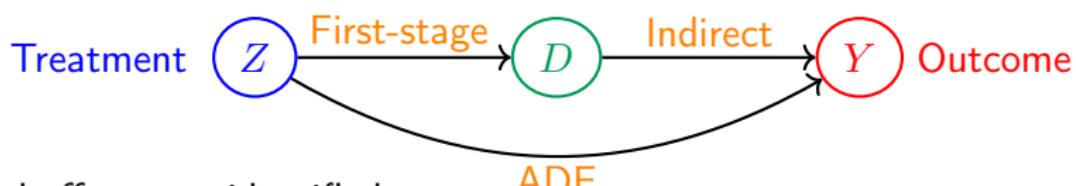
1. Ward economists away from using CM methods unabashedly.
→ Noted problems in the most popular methods for CM effects, pertinent for economic applications.
2. Bring CM methods into the credibility revolution (Angrist Card Imbens 2022), and labour selection-into-treatment (Heckman 2000) agendas.
→ Traced out identification by selection models.

Work-in-progress part of LWIPS:

- ▶ Connect the control function approach to MTE methods
- ▶ Large sample properties + analytical SEs
- ▶ Use this approach to estimate direct and indirect effects of genetics and education (JMP companion paper)
- ▶ (eventually) Package for selection-adjusted CM effects, by Heckman model and IV-assisted CF/MTE.

Appendix: Suggestive Evidence of Mechanisms

How empirical economists currently give evidence for mechanisms/mediators in causal effects.



Two causal effects are identified:

$$\text{ATE: } \mathbb{E}[Y_i(1, D_i(1)) - Y_i(0, D_i(0))] = \mathbb{E}[Y_i | Z_i = 1] - \mathbb{E}[Y_i | Z_i = 0]$$

$$\text{Average first-stage: } \mathbb{E}[D_i(1) - D_i(0)] = \mathbb{E}[D_i | Z_i = 1] - \mathbb{E}[D_i | Z_i = 0]$$

⇒ Show results of these two effects and assume indirect effect is positive, constant → suggestive evidence of mechanisms!

See Blackwell Matthew Ruofan Opacic (2024) for this in full, and a partial identification approach to avoid its unrealistic assumptions.

Appendix: Connection to MTES

The ADE is fine to estimate with a selection model/CF, but AIE refers to mediator gains only among mediator compliers.

$$\text{AIE} = \mathbb{E}[D_i(1) - D_i(0)] \mathbb{E}[Y_i(Z_i, 1) - Y_i(Z_i, 0) | D_i(1) = 1, D_i(0) = 0].$$

Outline of MTE approach to identifying AIE:

1. Mediator monotonicity has a selection model for D_i (Vycatil 2002).

$$D_i(z') = \mathbb{1}\{\mu(z'; \mathbf{X}_i) \geq U_i\}$$

2. Identify Marginal Indirect Effect (MIE), with instrument by LIV.

$$\mathbb{E}[Y_i(Z_i, 1) - Y_i(Z_i, 0) | U_i = u']$$

3. Mediator compliers' AIE is an integral of the MIE (Mogstad Santos Torgovitsky, 2017).

$$\int_W \mathbb{E}[Y_i(Z_i, 1) - Y_i(Z_i, 0) | U_i = u'] ,$$

$$\text{for } W = \{u' \mid D_i(1) = 1, D_i(0) = 0\} .$$