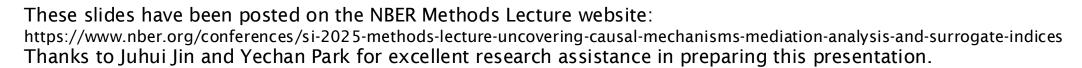
Uncovering Causal Mechanisms Mediation Analysis and Surrogate Indices

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NBER Methods Lecture

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A Recent Motivating Inquiry

An inquiry regarding a new workforce training program in NYC that builds connections between people to obtain jobs, motivated by research on social capital and upward mobility:

We would love to gather any ideas to improve predictions we can make [about the program's impacts]. As you know, in government, we **don't have the luxury of waiting 10 years** to report solid outcomes. We need to be able to tell the program's story of impact on an ongoing basis. We are continuously **innovating on how to be more efficient**.

- Diana Franco, NYC Economic Development Corporation July 16, 2025

Two Questions

- 1. Can we measure impacts of interventions more quickly rather than waiting to observe long-term outcomes (e.g., changes in career trajectories)?
 - More generally, when we can't measure primary outcomes of interest directly, can we use other variables as proxies?
- 2. What are the mechanisms through which an intervention matters (e.g., job referrals from connections vs. information about career paths)?
 - Understanding mechanisms critical for "innovating to improve efficiency"

- Both questions involve the analysis of intermediate outcomes either as predictors of long-term outcomes or as mediators
- Goal of this lecture: give an applied introduction to methods to answer these questions

Outline

- 1. Motivating Applications
- 2. Mediation Analysis
- 3. Surrogate Indices
- 4. Application and Recommendations for Empirical Practice

Motivating Example 1: Peer Effects on College Attendance

 Cattan, Salvanes, and Tominey (2025) analyze the effects of having high-school peers whose parents attended elite colleges on elite college attendance rates in Norway

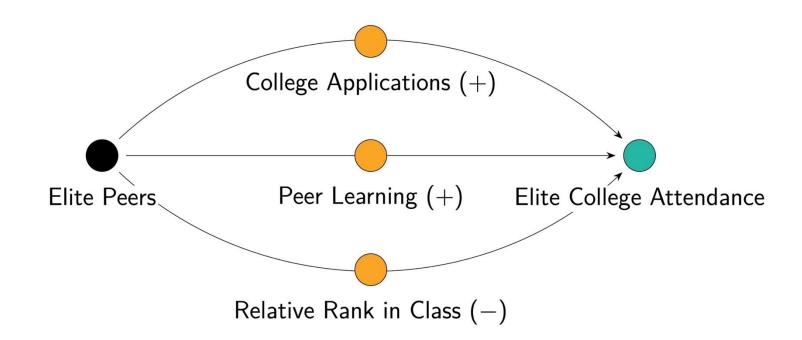
 Research Design: random variation in peer composition arising from finite-sample fluctuations across cohorts within schools

Cattan, Salvanes, and Tominey (2025): Reduced-Form Treatment Effect



1 SD increase in share of peers with elite-educated parents in school cohort →
2.6 pp increase in probability of enrolling in elite college (relative to mean of 10 pp)

Cattan, Salvanes, and Tominey (2025): Causal Mechanisms

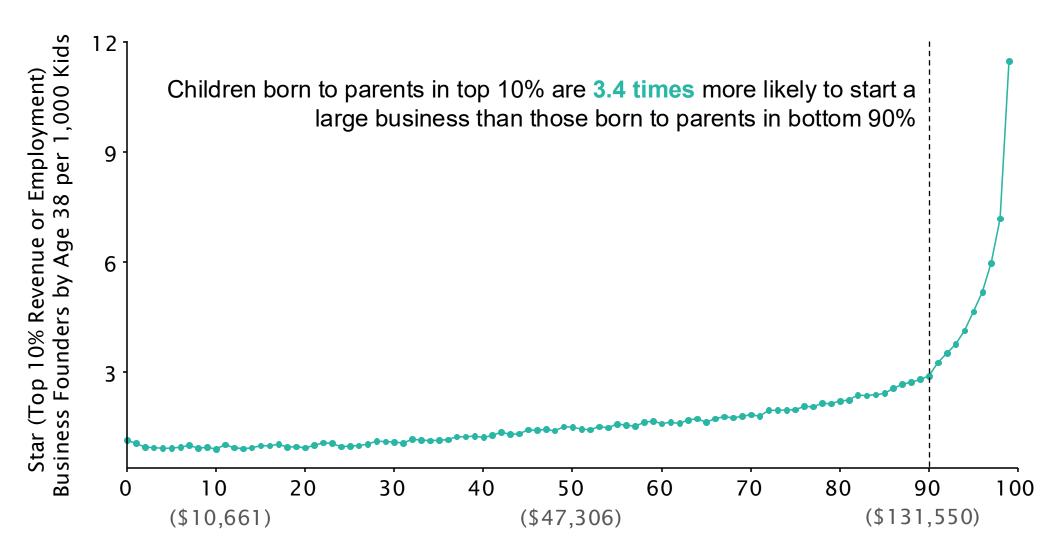


Direct Acyclic Graph (DAG) Representation of Causal Mechanisms [Pearl 2000]

Motivating Example 2: Determinants of Entrepreneurship

 Chetty, Dossi, Smith, van Reenen, Zidar, Zwick (2025) analyze determinants of and returns to entrepreneurship in the U.S. using data from anonymized tax records

Entrepreneurship Rates by Parental Income



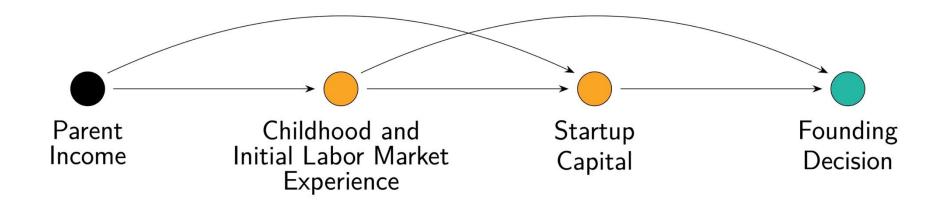
Parent Household Income Percentile

The Entrepreneurial Pipeline: Causal Mechanisms





The Entrepreneurial Pipeline: Causal Mechanisms



A Common Approach to Mediation Analysis: Controlling for Mediators

How much of the difference in entrepreneurship rates between children from low vs. high income families is explained by difference in access to capital at startup?

 Intuitive, commonly used approach: control for wealth at startup and measure how much of the raw gap in founding by parental income is "explained" by wealth

A Common Approach to Mediation Analysis: Controlling for Mediators

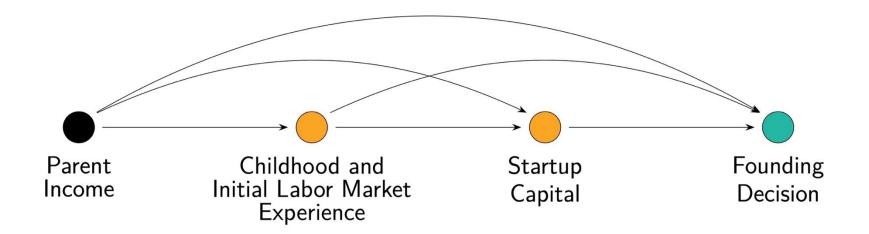
Regress indicator for business startup on having high-income (top decile) parents:

$$Y_i = \alpha_i + \beta \cdot (\text{Parent in Top } 10\%)_i + \gamma \cdot (\text{Wealth at Startup})_i + \epsilon_i$$

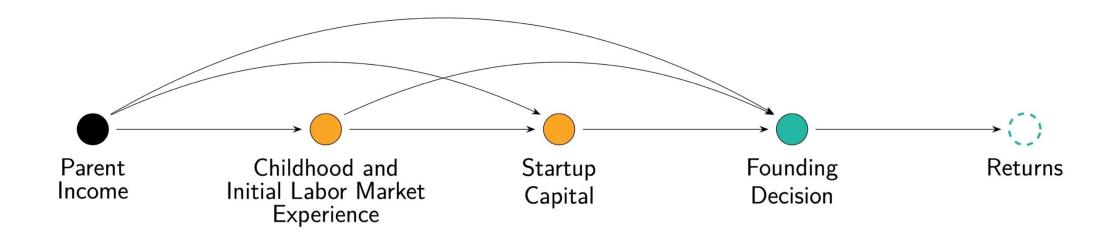
Dep. Variable: Founded a Large Firm (per 1,000 kids)	
Parents in Top Decile	7.69
	(0.04)
Wealth (\$1 mil) at Startup	

- \rightarrow Wealth accounts for 1-0.18/0.769 = 77% of effect of parent income on entrepreneurship
- Part 1 of this lecture (mediation analysis): under what assumptions is this approach valid? How can we identify the role of mediators when those assumptions fail?

The Entrepreneurial Pipeline

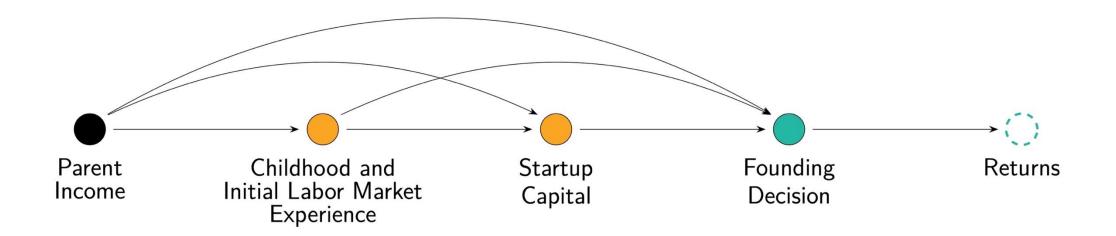


The Entrepreneurial Pipeline: Predicting Returns



- What are the marginal returns to inducing new business startups?
- Challenge: takes many years to measure returns → difficult to answer directly given censored data (need data from childhood to a decade after business startup)

The Entrepreneurial Pipeline: Predicting Returns



- Common heuristic practice: predict long-term returns using proxies measured at earlier stages (e.g., employment, revenues)
- Part 2 of this lecture (surrogate indices): under what assumptions is this approach valid? How can we predict long-term outcomes when those assumptions fail?

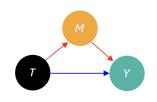
Part 1: Mediation Analysis

Identifying Mechanisms Underlying Treatment Effects on Primary Outcomes

Part I. Introduction to Mediation

Causal Mechanism as Direct and Indirect Effects

- Directed Acyclic Graph (DAG; Pearl, 2000)
 - $T \in \mathcal{T} = \{0, 1\}$: treatment
 - $M \in \mathcal{M}$: mediator (mechanism variable)
 - $Y \in \mathcal{Y}$: observed outcome



- Direct effect: Effect of T on Y while holding M constant
- Indirect effect: Effect of T on Y through M
- DAG = Nonparametric Structural Equation Model (NPSEM)

$$Y = f_Y(M, T, \epsilon)$$
$$M = f_M(T, \eta)$$

where ϵ and η are i.i.d. and are usually omitted from DAG

Controlled Direct Effect (CDE)

- $Y(t, m) \in \mathcal{Y}$: potential outcome when T = t and M = m
- Definition

Individual:
$$CDE_i(m) := Y_i(1, m) - Y_i(0, m)$$

Average: $\overline{CDE}(m) := \mathbb{E}[Y(1, m) - Y(0, m)]$

for a given mediator value $m \in \mathcal{M}$

- Interpretation
 - direct effect of treatment while holding the mediator constant at m
 - effect of joint intervention on T and M
- If M fully captures treatment effect, CDEs will be zero for all m
- Potential interaction effects:

$$CDE_i(m) \neq CDE_i(m')$$
 for some i and $m \neq m'$

Natural Indirect Effect (NIE)

Definition (Robins and Greenland, 1992; Pearl, 2001)

```
Individual: NIE_i(t) := Y_i(t, M_i(1)) - Y_i(t, M_i(0))

Average: \overline{NIE}(t) := \mathbb{E}[Y(t, M(1)) - Y(t, M(0))]
```

- Interpretation
 - effect of change in M on Y induced by T
 - change M from M(0) to M(1) while holding T at t=0 or t=1
 - zero treatment effect on M implies zero NIE

- Represents the causal effect of T on Y through M
- Complete mediation \rightsquigarrow NIE_i = TE_i := $Y_i(1, M_i(1)) Y_i(0, M_i(0))$

Treatment Effect Decomposition

Natural direct effect (NDE):

Indiviual:
$$NDE_i(t) := Y_i(1, M_i(t)) - Y_i(0, M_i(t))$$

Average: $\overline{NDE}(t) := \mathbb{E}[Y(1, M(t)) - Y(0, M(t))]$

- change T from 0 to 1 while holding M constant at M(t)
- causal effect of T on Y, holding M constant at its potential value that would be realized when T=t
- Represents all mechanisms other than through M
 - Complete mediation $\rightsquigarrow \mathsf{NDE}_i(t) = 0$
 - No mediation \rightsquigarrow NDE_i = TE_i
- Effect decomposition:

$$\underbrace{Y_i(1,M_i(1)) - Y_i(0,M_i(0))}_{= \text{total effect (TE}_i)} = \text{NIE}_i(t) + \text{NDE}_i(1-t)$$

$$= \frac{1}{2} \sum_{t=0}^{1} \{ \text{NIE}_i(t) + \text{NDE}_i(t) \}$$

Gender Bias and Educational Attainment (Chen et al. 2019)

- Data on Taiwanese families
 - Y: educational attainment of the oldest child who is female
 - T: gender of the second oldest child
 - M: number of siblings
- Gender bias
 - Direct effect: having a brother takes away resources from a female child
 - Indirect effect: having a brother leads to a smaller number of siblings and hence more resources
 - Direct and indirect effects may have opposite signs
- Causal effects of interest
 - CDE: effect of having a brother while keeping sibling size constant at a fixed value, e.g., 2
 - NDE: effect of having a brother while keeping sibling size constant at a value that would result, e.g., if the second child were male
 - NIE: effect of having a brother through sibling size

Take-aways I

- Causal mechanism
 - how and why (not just whether) treatment affects outcome
 - understanding of causal structure (DAG = NPSEM)

- Causal quantities of interest
 - Controlled direct effect (CDE)
 - Natural direct and indirect effects (NDE, NIE)
 - Effect decomposition: TE = NDE + NIE
 - No similar decomposition for CDE
 - Complete mediation: CDE = NDE = 0 and NIE = TE
 - No mediation: NIE = 0 and NDE = TE

Part II. Mediation Analysis Under Pretreatment Confounding

Linear Structural Equation Model (LSEM)

- Let's build some intuition with LSEM
- Homogeneous effects without interaction:

$$Y_i = \alpha_Y + \beta_Y T_i + \gamma_Y M_i + \epsilon_i$$

$$M_i = \alpha_M + \beta_M T_i + \eta_i$$

- $\overline{\mathsf{CDE}}(m) = \overline{\mathsf{NDE}}(t) = \beta_Y$ for any m and t
- $\overline{\text{NIE}}(t) = \beta_M \times \gamma_Y$ for any t
- CDE and NDE are identical
- Homogeneous effects with interaction:

$$Y_i = \alpha_Y + \beta_Y T_i + \gamma_Y M_i + \delta_Y T_i M_i + \epsilon_i$$

- $\overline{\mathsf{CDE}}(m) = \beta_{\mathsf{Y}} + m\delta_{\mathsf{Y}}$
- $\overline{\text{NDE}}(t) = \beta_Y + \delta_Y(\alpha_M + t\beta_M)$
- $\overline{\text{NIE}}(t) = \beta_M \times \gamma_Y + t\beta_M \times \delta_Y$
- CDE is different from NDE

LSEM with Heterogeneous Effects and Interaction

Model

$$Y_i = \alpha_Y + \beta_Y^{(i)} T_i + \gamma_Y^{(i)} M_i + \delta_Y^{(i)} T_i M_i + \epsilon_i$$
$$M_i = \alpha_M + \beta_M^{(i)} T_i + \eta_i$$

- $\overline{\mathsf{CDE}}(m) = \bar{\beta}_Y + m\bar{\delta}_Y$ where $\bar{\beta}_Y = \mathbb{E}[\beta_Y^{(i)}]$ and $\bar{\delta}_Y = \mathbb{E}[\delta_Y^{(i)}]$
- $\overline{\mathsf{NDE}}(t) = \bar{\beta}_Y + \alpha_M \times \bar{\delta}_Y + \mathbb{E}[\delta_Y^{(i)}(t\beta_M^{(i)} + \eta_i)]$
- $\overline{\mathsf{NIE}}(t) = \mathbb{E}[\beta_M^{(i)} \times (\gamma_Y^{(i)} + t\delta_Y^{(i)})]$
- Heterogeneous effects may be correlated with one another
 - For example, $\mathbb{E}[\beta_M^{(i)} \times \gamma_Y^{(i)}] \neq \bar{\beta}_M \times \bar{\gamma}_Y$
 - Possible to have $\bar{\beta}_M, \bar{\gamma}_Y > 0$ but $\mathbb{E}[\beta_M^{(i)} \times \gamma_Y^{(i)}] < 0$ or vice versa
- $\bar{\beta}_M$, $\bar{\gamma}_Y$, $\bar{\delta}_Y$, etc. are identifiable under exogeneity
- But, $\mathbb{E}[\beta_M^{(i)} \times \gamma_Y^{(i)}]$, $\mathbb{E}[\beta_M^{(i)} \times \delta_Y^{(i)}]$, etc. are unidentifiable
- This is essentially a problem of unobserved pre-treatment confounding

Identification of CDE with Pre-treatment Confounding

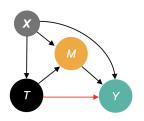
- Assumptions:
 - Unconfoundedness

$$\begin{aligned} &\{Y_i(t,m), M_i(t)\}_{t,m} \perp \!\!\! \perp T_i \mid \boldsymbol{X}_i = \boldsymbol{x} \\ &\{Y_i(t,m)\}_m \perp \!\!\! \perp M_i \mid T_i = t, \boldsymbol{X}_i = \boldsymbol{x} \end{aligned}$$

Overlap

$$P(T_i = t \mid X_i = x) > 0$$

 $P(M_i = m \mid T_i = t, X_i = x) > 0$



• Identification:

$$\overline{CDE}(m)$$

$$= \sum_{\mathbf{X}} (\mathbb{E}[Y \mid T = 1, M = m, \mathbf{X}] - \mathbb{E}[Y \mid T = 0, M = m, \mathbf{X}]) P(\mathbf{X})$$

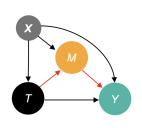
Identification of NDE/NIE with Pretreatment Confounding

• Replace the following assumption

$$\{Y_i(t,m)\}_m \perp \!\!\! \perp \underbrace{M_i}_{=M_i(t)} \mid T_i = t, X_i = x$$

with the cross-world independence

$$\{Y_i(\mathbf{t'},m)\}_{\mathbf{t'},m} \perp M_i(\mathbf{t}) \mid T_i = t, \mathbf{X}_i = \mathbf{x}$$



- Additional conditional independence between $Y_i(t', m)$ and $M_i(t)$
- Identification (Imai et al. 2010)

$$\overline{\mathsf{NDE}}(t) = \sum_{M, \mathbf{X}} (\mathbb{E}[Y \mid M, T = 1, \mathbf{X}] - \mathbb{E}[Y \mid M, T = 0, \mathbf{X}]) \times P(M \mid T = t, \mathbf{X}) P(\mathbf{X})$$

$$\overline{\mathsf{NIE}}(t) = \sum_{M,\mathbf{Y}} \mathbb{E}[Y \mid M, T = t, \mathbf{X}]$$

$$\times \{ P(M \mid T = 1, \mathbf{X}) - P(M \mid T = 0, \mathbf{X}) \} P(\mathbf{X})$$

Experimental Identification (Imai et al. 2013)

- Parallel design
 - ullet Randomize T and observe M and Y
 - \bigcirc Randomize T and M and observe Y
- We can identify P(M(t)), P(Y(t, M(t))), and P(Y(t, m))
- CDE is identified
- NDE/NIE is still not identifiable:
 - randomization cannot break correlation between Y(t', m) and M(t)
 - partial identification: sharp bounds contain zero
- Crossover design
 - lacktriangledown Randomize T and observe M and Y
 - ② On the same sample, change T to the opposite condition while holding M at the same value and observe Y
- Y(t, M(t)), M(t), and Y(1 t, M(t)) are observable
- Additional assumption: no carryover effects
- NDE/NIE is identifiable

No Interaction Assumption

No individual-level interaction

$$Y_i(1, m) - Y_i(0, m) = Y_i(1, m') - Y_i(0, m')$$

- $NDE_i(t) = CDE_i(m) = CDE_i$
- $\overline{\mathsf{NDE}}(t) = \overline{\mathsf{CDE}}(m) = \overline{\mathsf{CDE}}$
- $\overline{\mathsf{NIE}}(t) = \mathsf{ATE} \overline{\mathsf{NDE}}$
- Testable implication:

$$\mathbb{E}[Y_i(1,m) - Y_i(0,m) \mid \mathbf{X}_i = \mathbf{x}] = \mathbb{E}[Y_i(1,m') - Y_i(0,m') \mid \mathbf{X}_i = \mathbf{x}]$$

for all x

- NDE/NIE is identifiable so long as CDE can be identified
- Experimental identification, and identification with pretreatment and posttreatment confounding are all possible

Estimation of Natural Direct and Indirect Effects

Recall the identification formula (NIE)

$$\overline{\mathsf{NIE}}(t) = \sum_{M, \mathbf{X}} \mathbb{E}[Y \mid M, T = t, \mathbf{X}]$$

$$\times \{P(M \mid T = 1, \mathbf{X}) - P(M \mid T = 0, \mathbf{X})\} P(\mathbf{X})$$

- predict M given each treatment value: $\{M_i(1), M_i(0)\}$
- ② predict Y by first setting $T_i = t$ and $M_i = M_i(0)$, and then $T_i = t$ and $M_i = M_i(1)$: $\{Y_i(t, M_i(0)), Y_i(t, M_i(1))\}$
- 3 compute the average difference between two predicted outcomes
- Estimation of NDE is similar

$$\overline{\mathsf{NDE}}(t) = \sum_{M, \boldsymbol{X}} (\mathbb{E}[Y \mid M, T = 1, \boldsymbol{X}] - \mathbb{E}[Y \mid M, T = 0, \boldsymbol{X}])$$

$$\times P(M \mid T = t, \boldsymbol{X})P(\boldsymbol{X})$$

• One can also do: $\overline{\mathsf{NDE}}(t) = \mathsf{ATE} - \overline{\mathsf{NIE}}(1-t)$

Weighting Methods for NDE and NIE

• Three weighting formulae:

$$\mathbb{E}[Y(t, M(t'))] = \mathbb{E}\left[\underbrace{\frac{1\{T = t'\}}{\Pr(T = t' \mid \boldsymbol{X})}}_{\text{weighting to get } P(M(t') \mid \boldsymbol{X})} \times \mathbb{E}[Y \mid M, T = t, \boldsymbol{X}]\right]$$

$$= \mathbb{E}\left[\underbrace{\frac{1\{T = t\}}{\Pr(T = t \mid \boldsymbol{X}_i)}}_{\text{treatment weighting}} \times \underbrace{\frac{P(M \mid T = t', \boldsymbol{X})}{P(M \mid T_i = t, \boldsymbol{X}_i)}}_{\text{mediator weighting}} \times Y\right]$$

$$= \mathbb{E}\left[\frac{1\{T = t\}}{\Pr(T = t \mid M, \boldsymbol{X})} \times \frac{\Pr(T = t' \mid M, \boldsymbol{X})}{\Pr(T = t' \mid \boldsymbol{X})} \times Y\right]$$

- The third expression follows from Bayes rule
- Useful when the mediator is high-dimensional
- Multiply-robust semiparametric estimator (Tchetgen Tchetgen and Shpitser, 2012); Double machine learning (Farbmacher et al. 2022)

Sensitivity Analysis

- Examine the robustness of empirical findings to the violation of untestable assumptions
- How large a departure from the key identification assumption must occur for the conclusions to no longer hold?
- ullet Potential existence of unobserved pretreatment confounding (T is assumed to be unconfounded)

$$\{Y_i(t',m)\}_{t',m} \not\perp \!\!\!\!\perp M_i(t) \mid T_i = t, \boldsymbol{X}_i = \boldsymbol{x}$$

Recall LSEM (or more generally, additive semiparametric model)

$$Y_{i} = \alpha_{Y} + \beta_{Y} T_{i} + \gamma_{Y} M_{i} + \underbrace{\frac{\lambda_{\epsilon} U_{i} + \tilde{\epsilon}_{i}}{=\epsilon_{i}}}_{=\epsilon_{i}}$$

$$M_{i} = \alpha_{M} + \beta_{M} T_{i} + \underbrace{\frac{\lambda_{\eta} U_{i} + \tilde{\eta}_{i}}{=\epsilon_{i}}}_{=\epsilon_{i}}$$

• How much does U_i have to matter for the results to go away?

Sensitivity Parameters

- R² parameterization
 - **1** Proportion of previously unexplained variance explained by U_i

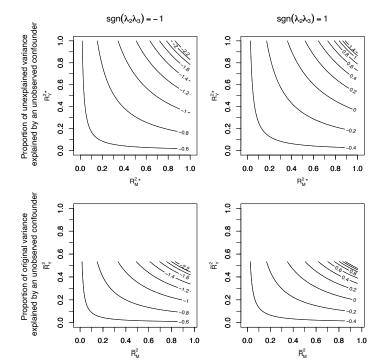
$$R_M^{2*} \equiv \frac{\mathbb{V}(\lambda_{\eta} U_i)}{\mathbb{V}(\eta_i)}$$
 and $R_Y^{2*} \equiv \frac{\mathbb{V}(\lambda_{\epsilon} U_i)}{\mathbb{V}(\epsilon_i)}$

2 Proportion of original variance explained by U_i

$$\widetilde{R}_{M}^{2} \equiv \frac{\mathbb{V}(\lambda_{\eta}U_{i})}{\mathbb{V}(M_{i})} \text{ and } \widetilde{R}_{Y}^{2} \equiv \frac{\mathbb{V}(\lambda_{\epsilon}U_{i})}{\mathbb{V}(Y_{i})}$$

• We also need to specify the direction of effects:

$$\operatorname{sgn}(\lambda_{\eta}\lambda_{\epsilon}) \ = \ egin{cases} 1 & ext{if same direction} \ -1 & ext{if opposite directions} \end{cases}$$



Gender Bias Application: Standard Mediation Analysis

The original analysis fits LSEM with interaction

$$Y_i = \alpha_Y + \beta_Y T_i + \gamma_Y M_i + \delta_Y T_i M_i + \boldsymbol{\xi}_Y^\top \boldsymbol{X}_i + \epsilon_i$$
$$M_i = \alpha_M + \beta_M T_i + \boldsymbol{\xi}_M^\top \boldsymbol{X}_i + \eta_i$$

- Y_i: university admission
- T_i: the second child is male
- M_i : sibling size is greater than two
- Estimates:

ÂTE	0.0020 (0.0013)
$\widehat{CDE}(\widehat{M})$	-0.0010 (0.0014)
$\widehat{NDE}(1)$	-0.0001 (0.0014)
$\widehat{NIE}(0)$	0.0022 (0.0005)

- Also, fits a random coefficient model to address heterogeneity
- Sensitivity analysis based on a semiparametric random coefficient model (Imai and Yamamoto, 2013)

Take-aways II

- Linear structural equation model
 - two key assumptions beyond exogeneity:
 - homogeneous effects
 - 2 no interaction
 - CDE = NDE under those assumptions
 - Relaxing these assumptions lead to different interpretations and identification issues
- Nonparametric identification analysis under pretreatment confounding
 - CDE is identifiable under standard exogeneity
 - NDE/NIE requires cross-world independence
 - \bullet alternatively, $\mathsf{CDE} = \mathsf{NDE}$ if we assume no individual-level interaction
- Difficulty of identification
 - even when M is randomized, NIE/NDE are unidentifiable
 - sensitivity analysis plays an important role for assessing robustness

Part III. Coping with Identification Difficulties

Instrumenting the Mediator

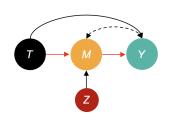
- Instrument: Z_i
- Mediator: $M_i(t,z)$
- Exclusion restriction

$$Y_i(t, m, z) = Y_i(t, m)$$

NPSEM:

$$Y = f_Y(M, T, \epsilon)$$

 $M = f_M(T, Z, \eta)$ where $\epsilon \not\perp \!\!\! \perp \eta$



- If M and Z are continuous, we can use the control function approach (Imbens and Newey, 2009)
 - **1** Independence: $Z \perp \!\!\! \perp (\epsilon, \eta)$
 - ② Monotonicity: η is a continuous scalar variable with its CDF and $f_M(\cdot,\cdot,\eta)$ being strictly monotonic in η
- Then, $(M, T) \perp \!\!\! \perp \!\!\! \perp \in C$ where $C = F_{M|T,Z}(T,Z) = F_{\eta}(\eta)$
 - Recall the control function approach to 2SLS
 - Regress Y on M,T and the first stage residual $\hat{\eta}$
- Extension: an additional instrument for T (Florich and Huber, 2017)

Gender Bias Application: IV Analysis

• Instrument Z: twinning at the second birth

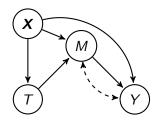
$$M_i = \alpha_M + \beta_M T_i + \zeta_M Z_i + \lambda_M T_i Z_i + \boldsymbol{\xi}_M^\top \boldsymbol{X}_i + \eta_i$$

- Assumptions:
 - exogenous instrument: twinning is random conditional on X
 - ullet exclusion restriction: twinning affects Y only through M
- Findings:

	Standard analysis	IV analysis
ÂTE	0.0020 (0.0013)	0.0021 (0.0013)
$\widehat{CDE}(\widehat{M})$	$-0.0010 \ (0.0014)$	$-0.0092 \ (0.0061)$
$\widehat{NDE}(1)$	-0.0001 (0.0014)	-0.0203 (0.0106)
$\overline{NIE}(0)$	0.0022 (0.0005)	0.0224 (0.0105)

Complete Mediation Analysis (Kwon and Roth 2024)

- Complete mediation: $Y_i(t, m) = Y_i(m)$
- Assumption: No unobserved confounding between T and M and between T and Y
- Possible unobserved confounding between M and Y



• Under monotonicity $M_i(1) \ge M_i(0)$ (in the binary mediator case), we can use the following test of instrumental validity

$$P(Y, M = 0 \mid T = 0, X) \ge P(Y, M = 0 \mid T = 1, X)$$

 $P(Y, M = 1 \mid T = 1, X) \ge P(Y, M = 1 \mid T = 0, X)$

- Randomized experiment: test of complete mediation
- ullet Observational study: unobserved confounding between T and Y can also lead to the rejection of the null hypothesis

^{*} This slide has been revised to more accurately represent the method proposed by Kwon and Roth (2024).

Implicit Mediation

- What if we want to avoid the untestable assumptions at all costs?
- What can we infer from ATE_M and ATE_Y that are identifiable without such assumptions?

Table 1. Possible Implicit-Mediation Findings

Result	Inference	Rationale
X affects M and Y	M may be a mediator.	X appears to influence M, and this effect seems to coincide with a change in Y, as would be expected if M were a mediator.
X affects M but not Y	M appears not to be a mediator.	Although <i>X</i> affects <i>M</i> , this effect seems not to have any consequences for <i>Y</i> .
X affects Y but not M	Some variable other than M may be a mediator.	<i>X</i> appears to have no effect on <i>M</i> , which means that <i>X</i> 's apparent effect on <i>Y</i> is not due to changes in <i>M</i> .
X affects neither M nor Y	There seem to be no indirect pathways from <i>X</i> to <i>Y</i> through <i>M</i> or other mediators.	X seems not to set in motion any causal effects.

Identification Analysis of Implicit Mediation

- Questions:
 - ① Does ATE_M = 0 imply $\overline{\text{NIE}} = 0$ and/or $\overline{\text{NDE}} \neq 0$?
 - ② Does $ATE_M > 0$ and $ATE_Y > 0$ imply $\overline{NIE} > 0$?
- No! Recall even the no-assumption bounds from the parallel experiment design always contain zero
- The decomposition under a binary mediator:

$$\overline{\mathsf{NIE}}(t) = \underbrace{\mathbb{E}[Y_i(t,1) - Y_i(t,0) \mid M(1) = 1, M(0) = 0]}_{\mathsf{ATE} \text{ of } M \text{ on } Y \text{ for compliers}} \cdot p_{10}$$

$$-\underbrace{\mathbb{E}[Y_i(t,1) - Y_i(t,0) \mid M(1) = 0, M(0) = 1]}_{\mathsf{ATE} \text{ of } M \text{ on } Y \text{ for defiers}} \cdot p_{01}$$

where
$$p_{m_1m_0} = \Pr(M(1) = m_1, M(0) = m_0)$$

Cross-world assumption or homogeneity assumption leads to the usual product estimator

$$\overline{\mathsf{NIE}}(t) = \underbrace{\mathbb{E}[Y_i(t,1) - Y_i(t,0)]}_{\mathsf{=ATE of }M \text{ on }Y} \times \underbrace{(p_{10} - p_{01})}_{\mathsf{=ATE}_M}$$

Identification under Monotonicity

(Blackwell et al. 2024; Kwon and Roth 2024)

Monotonicity assumption (no defier) yields:

$$\overline{\mathsf{NIE}}(t) = \mathbb{E}[Y_i(t,1) - Y_i(t,0) \mid M(1) = 1, M(0) = 0] \cdot p_{10}$$

Sharp bounds

$$\max\{-\mathsf{ATE}_M, -q_{1-t,t|t}\} \leq \overline{\mathsf{NIE}}(t) \leq \min\{\mathsf{ATE}_M, q_{tt|t}\}$$
 where $q_{ym|t} = \mathsf{Pr}(Y=y, M=m \mid T=t)$

- Two fundamental difficulties remain:
 - effect heterogeneity
 - endogeneity of mediator
- Even under an additional assumption of $\mathbb{E}[Y(t,1) Y(t,0)] > 0$, the sharp bounds still contain zero

Take-aways III

- Instrumental variable approach
 - addressing the endogeneity problem
 - the instrument must be exogeneous
 - exclusion restriction needs to be satisfied
 - nonparametric estimation is possible
- Complete mediation
 - hypothesis testing approach
 - no need to assume the exogeneity of mediator
 - ullet no unobserved confounding between T and Y (satisfied in RCT)
- Implicit mediation
 - an attempt to sidestep assumptions
 - not informative even about the signs of NIE/NDE
 - monotonicity is not sufficient

Part IV. Mediation Analysis under Posttreatment Confounding

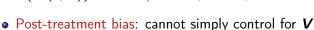
Identification of CDE with Posttreatment Confounding

• Replace the following assumption

$$\{Y_i(t,m)\}_m \perp \!\!\! \perp M_i \mid T_i = t, \boldsymbol{X}_i = \boldsymbol{x},$$

with

$$\{Y_i(t,m)\}_m \perp M_i \mid \mathbf{V}_i = \mathbf{v}, T_i = t, \mathbf{X}_i = \mathbf{x}$$



$$\overline{\mathsf{CDE}}(m) \neq \sum_{X,Y} (\mathbb{E}[Y \mid T=1, M=m, X, Y])$$

$$-\mathbb{E}[Y \mid T=0, M=m, \boldsymbol{X}, \boldsymbol{V}])P(\boldsymbol{X}, \boldsymbol{V})$$

ullet Identification: model $oldsymbol{V}$ given $\mathcal T$ and $oldsymbol{X}$

$$\overline{\mathsf{CDE}}(m) \ = \ \sum \left\{ \mathbb{E}[Y \mid T=1, M=m, \boldsymbol{X}, \boldsymbol{V}] P(\boldsymbol{V} \mid T=1, \boldsymbol{X}) \right\}$$

$$-\mathbb{E}[Y \mid T=0, M=m, \mathbf{X}, \mathbf{V}]P(\mathbf{V} \mid T=0, \mathbf{X})\}P(\mathbf{X})$$

Estimation of Controlled Direct Effects

Directly use the identification formula

$$\bar{\xi}(m) = \sum_{\boldsymbol{X},\boldsymbol{V}} \{ \mathbb{E}[Y \mid T = 1, M = m, \boldsymbol{X}, \boldsymbol{V}] P(\boldsymbol{V} \mid T = 1, \boldsymbol{X})$$
$$-\mathbb{E}(Y \mid T = 0, M = m, \boldsymbol{X}, \boldsymbol{V}) P(\boldsymbol{V} \mid T = 0, \boldsymbol{X}) \} P(\boldsymbol{X})$$

- regression of Y on T, M, X, V
- model \boldsymbol{V} given T and $\boldsymbol{X} \leadsto$ difficult if \boldsymbol{V} is high-dimensional
- Marginal structural models (Robins et al. 2000)

$$\mathbb{E}[Y(t,m)] = \mathbb{E}\left[\underbrace{\frac{1\{T=t,M=m\}}{\Pr(T=t\mid \boldsymbol{X})}}_{\text{IPW for treatment}} \cdot \underbrace{\frac{1}{\Pr(M=m\mid T=t,\boldsymbol{X},\boldsymbol{V})}}_{\text{IPW for mediator given treatment}} \times Y\right]$$

- ullet no need to model $oldsymbol{V}$
- covariate balancing methods are also available (Imai and Ratkovic, 2015)

Identification of NDE/NIE with Posttreatment Confounding

- Identification is impossible with observed posttreatment confounding
- Consider the following NPSEM

$$Y = f_{Y}(M, \mathbf{V}, T, \epsilon)$$

 $M = f_{M}(\mathbf{V}, T, \eta)$
 $\mathbf{V} = f_{\mathbf{V}}(T, \xi)$

Cross-world independence cannot hold

$$\underbrace{\boldsymbol{V}(1)}_{=f_{\boldsymbol{V}}(1,\xi)} \not\perp \underbrace{\boldsymbol{V}(0)}_{=f_{\boldsymbol{V}}(0,\xi)} \implies Y(t',m,\boldsymbol{V}(t'),\epsilon) \not\perp \!\!\!\!\perp M(t,\boldsymbol{V}(t),\eta)$$

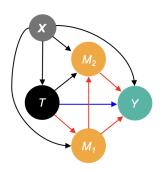
ullet Conditioning on T and $oldsymbol{V}$ does not solve this problem

Multiple Causally Related Mediators

- Same as the posttreatment confounding setting
- Path specific effects
- Combined effect:

$$T \to M_1 \rightsquigarrow Y$$

$$= (T \to M_1 \to Y) + (T \to M_1 \to M_2 \to Y)$$



- Generalized cross-world independence assumptions:

 - $\{M_{2i}(t', m_1), Y_i(t', m_1, m_2)\}_{t', m_1, m_2} \bot M_{1i}(t) \mid T_i = t, X_i = x$
 - $\{Y_i(t', m_1, m_2)\}_{t', m_2} \perp \perp M_{2i}(t, m_1) \mid M_{1i} = m_1, T_i = t, X_i = x$
- Identifiable decomposition:

$$\mathsf{ATE} \ = \ (\ \mathsf{T} \to \mathsf{Y}) + (\ \mathsf{T} \to \mathsf{M}_2 \to \mathsf{Y}) + (\ \mathsf{T} \to \mathsf{M}_1 \leadsto \mathsf{Y})$$

Interventional Direct and Indirect Effects (IDE and IIE)

- $\mathcal{P}_{M(t)}$: interventional distribution that independently generates M(t)
- Definition (Geneletti, 2007; Lok, 2016)

Individual:
$$\begin{cases} \mathsf{IIE}_i(t) &= Y_i(t, \mathcal{P}_{M(1)}) - Y_i(t, \mathcal{P}_{M(0)}) \\ \mathsf{IDE}_i(t) &= Y_i(1, \mathcal{P}_{M(t)}) - Y_i(0, \mathcal{P}_{M(t)}) \end{cases}$$
 Average:
$$\begin{cases} \overline{\mathsf{IIE}}(t) &= \mathbb{E}[Y(t, \mathcal{P}_{M(1)}) - Y(t, \mathcal{P}_{M(0)})] \\ \overline{\mathsf{IDE}}(t) &= \mathbb{E}[Y(t, \mathcal{P}_{M(1)}) - Y(t, \mathcal{P}_{M(0)})] \end{cases}$$

- Interpretation
 - similar to NIE and NDE
 - IDE is a function of CDE:

$$\mathsf{IDE}_i(t) = \sum_{m} \mathsf{CDE}_i(m) \times P(M(t) = m)$$

- no mediation: zero treatment effect on M implies zero IIE
- Effect decomposition

$$\underbrace{Y_i(1,\mathcal{P}_{M(1)}) - Y_i(0,\mathcal{P}_{M(0)})}_{\text{Interventional Total Effect (ITE)} \neq \text{TE}} = \text{IIE}_i(t) + \text{IDE}_i(1-t)$$

Identification of IDE and IIE

Once CDE is identified, we can identify IDE:

$$\overline{\mathsf{IDE}}(t) = \sum_{m} \overline{\mathsf{CDE}}(m) P(M(t) = m)$$

IIE is also identifiable:

$$\overline{\mathsf{IIE}}(t) = \sum_{m} \mathbb{E}[Y(t,m)] \left\{ P(M(1) = m) - P(M(0) = m) \right\}$$

Effect decomposition

$$\underbrace{\mathbb{E}[Y(1,\mathcal{P}_{M(1)}) - Y(0,\mathcal{P}_{M(0)})]}_{\neq \mathbb{E}[Y(1,M(1)) - Y(0,M(0))]} = \overline{\mathsf{IDE}}(t) + \overline{\mathsf{IIE}}(1-t)$$

- Complete mediation: $\overline{\mathsf{IDE}} = 0$
- Identification is possible with observed pretreatment and posttreatment confounding
- Experimental identification via parallel design is also possible

Take-aways IV

- Posttreatment confounding
 - CDE can be identified under exogeneity
 - estimation of CDE requires marginalizing posttreatment confounders
 - NIE/NDE are not identifiable under exogeneity
 - Different decomposition is identifiable under cross-world independence

- Alternative estimands
 - interventional direct and indirect effects (IDE/IIE)
 - interventional distribution on M
 - enables decomposition of alternative total effect
 - identification of CDE implies that of IDE/IIE

Conclusion, Resources, and References

Concluding Remarks on Causal Mechanisms

- Study of causal mechanisms is essential but challenging
- Triangulation of evidence is necessary
 - causal quantities
 - CDE
 - NDE/NIE, path specific effects
 - IDE/IIE
 - causal identification strategies
 - selection on observables
 - instrumental variables
 - experimental designs
 - partial identification
 - statistical methodologies
 - weighting and regression
 - sensitivity analysis
 - nonparametric modeling and machine learning

Resources

- Statistical software:
 - mediation (R and Stata)
 - Valeri and VanderWeele macros (SPSS, SAS, Stata)
- Review article by an economist:

Huber, Martin (2020). "Mediation Analysis".

Handbook of Labor, Human Resources and Population Economics.

Ed. by Klaus F. Zimmermann. Cham: Springer.

Monographs:

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 <u>Statistical Science</u> 25.1, pp. 51–71.

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 Proc. of the 17th Conference on Uncertainty in Artificial Intelligence.
 San Francisco, CA: Morgan Kaufmann, pp. 411–420.
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Part 2: Surrogate Indices

Identifying Treatment Effects on Primary Outcomes Using Mediators

Identifying Long-Term Treatment Effects Using Proxies

- In this part of the lecture, focus on three methods that formalize when and how we can
 use proxies to predict treatment effects on a primary outcome of interest
 - 1. Surrogates: predict long-term outcomes using intermediate outcomes [e.g., Prentice 1989, VanderWeele 2013, Athey, Chetty, Imbens, Kang 2025]
 - 2. Remotely Sensed Variables: predict outcomes using post-treatment proxies [Rambachan, Singh, Viviano 2025]
 - 3. Experimental Selection Correction: control for selection using intermediate outcomes [Athey, Chetty, Imbens 2025]

Surrogates

Predicting Treatment Effects Using Intermediate Outcomes

Estimating Long-Term Impacts of Interventions



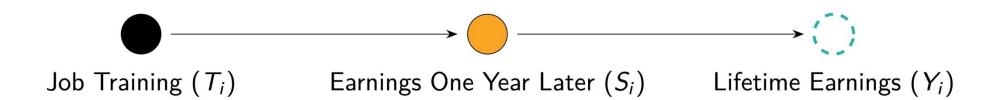
How do job training programs affect career trajectories?

Would take decades to measure full impact on lifetime earnings

- Intuitive, widely used heuristic: use short-term proxies to predict long-term outcomes
 - If job training improves earnings one year later, and earnings are highly serially correlated over time, can we extrapolate to predict long-term earnings gains?

Surrogate Model: Setup

Experimental Data (e.g., Job Training Experiment) $G_i = E$



Observational Data (e.g., Administrative Tax Records) $G_i = O$



Identification Using Surrogates: Key Assumptions

Assumption 1. Unconfounded Experiment (or Quasi-Experiment)

$$T_i \perp \!\!\! \perp (Y_i(0), Y_i(1), S_i(0), S_i(1)) \mid G_i = E$$

Assumption 2. Comparability of Experimental and Observational Samples

$$Y_i \mid S_i, G_i = E \sim Y_i \mid S_i, G_i = O$$

Comparability Assumption in Linear Models

$$Y_i \mid S_i, G_i = E \sim Y_i \mid S_i, G_i = O$$

• Suppose the conditional expectation of $Y_i \mid S_i$ is linear in both samples:

Experimental Data: $E[Y_i \mid S_i, G_i = E] = \alpha_E + \beta^E \cdot S_i$

Observational Data: $E[Y_i \mid S_i, G_i = O] = \alpha_O + \beta^O \cdot S_i$

In this case, comparability requires that the slopes are the same in both samples:

$$\beta^E = \beta^O$$

Identification Using Surrogates: Key Assumptions

Assumption 1. Unconfounded Experiment (or Quasi-Experiment)

$$T_i \perp \!\!\! \perp (Y_i(0), Y_i(1), S_i(0), S_i(1)) \mid G_i = E$$

Assumption 2. Comparability of Experimental and Observational Samples

$$Y_i \mid S_i, G_i = E \sim Y_i \mid S_i, G_i = O$$

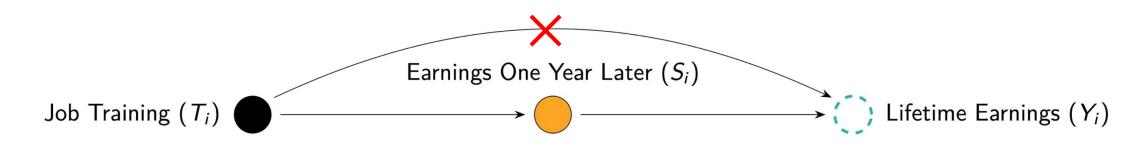
Assumption 3. Surrogacy: T_i has no effect on long-term outcome Y_i once we control for S_i

$$T_i \perp \!\!\!\perp Y_i \mid S_i, G_i = E$$

Violations of Surrogacy Assumption

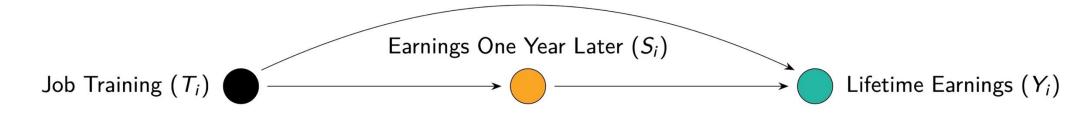
$$T_i \perp \!\!\!\perp Y_i \mid S_i, G_i = E$$

Experimental Data

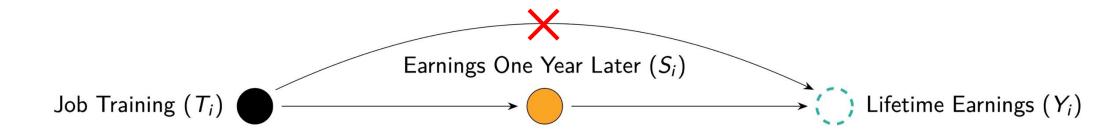


Surrogacy vs. Mediation

Mediation: Allow treatment to have direct effect on Y_i and estimate direct vs. indirect effects using data on Y_i



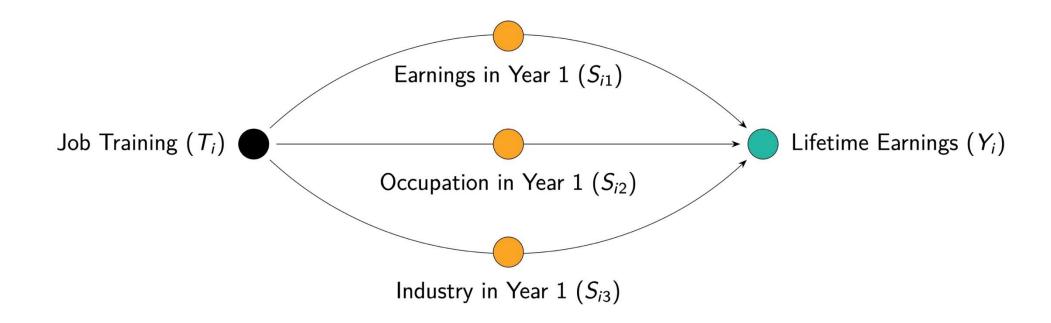
Surrogacy: Assume no direct effect of treatment on $Y_i \rightarrow$ indirect effect = total effect; do not observe Y_i in experimental sample, so use observational data to predict it



Surrogacy with Multiple Mediators

In many social science applications, unlikely to have a single mediator that captures causal pathway from treatment to outcome (unlike specific biomarkers in biology)

More plausible that causal pathways are spanned by multiple mediators that together satisfy surrogacy assumption



Surrogate Index: Combining Multiple Mediators

- Combine multiple mediators by constructing a surrogate index
- Surrogate index is conditional expectation of long-term outcome (e.g., total lifetime earnings) given the surrogates (e.g., early-career earnings) in the observational data

$$\mu(s, O) = E[Y_i \mid S_i = s, G_i = O]$$

In a linear model, surrogate index is the predicted value from a regression of long-term outcome on surrogates S_{in} :

$$Y_i = eta_0 + \sum_{n=1}^N \ eta_n S_{in} + \epsilon_i$$
 $\mu(S_i, O) = eta_0 + \sum_{n=1}^N \ \hat{eta}_n S_{in}$

Identification Using Surrogate Index

Under Assumptions 1-3 (and regularity conditions), treatment effect on the surrogate index in the experimental sample is the average treatment effect on long-term outcome:

$$ATE_{Y} = E[\mu(S_{i}, O) \mid T_{i} = 1, G_{i} = E] - E[\mu(S_{i}, O) \mid T_{i} = 0, G_{i} = E]$$

Two steps to estimate treatment effect on long-term outcome:

- Regress long-term outcome on surrogates to construct surrogate index in observational data reg lifetime_earnings earnings_yr1 earnings_yr2 earnings_yr3 if obs == 1 predict lifetime_earnings_pred
- 2. Regress surrogate index on treatment in experimental data reg lifetime_earnings_pred treatment if exp == 1

Note: standard errors must be adjusted to account for noise in estimating surrogate index; can be estimated using bootstrap

Application: California GAIN Training Program

California Greater Avenues to Independence (GAIN) program: job training program implemented in late 1980s to help welfare recipients find work

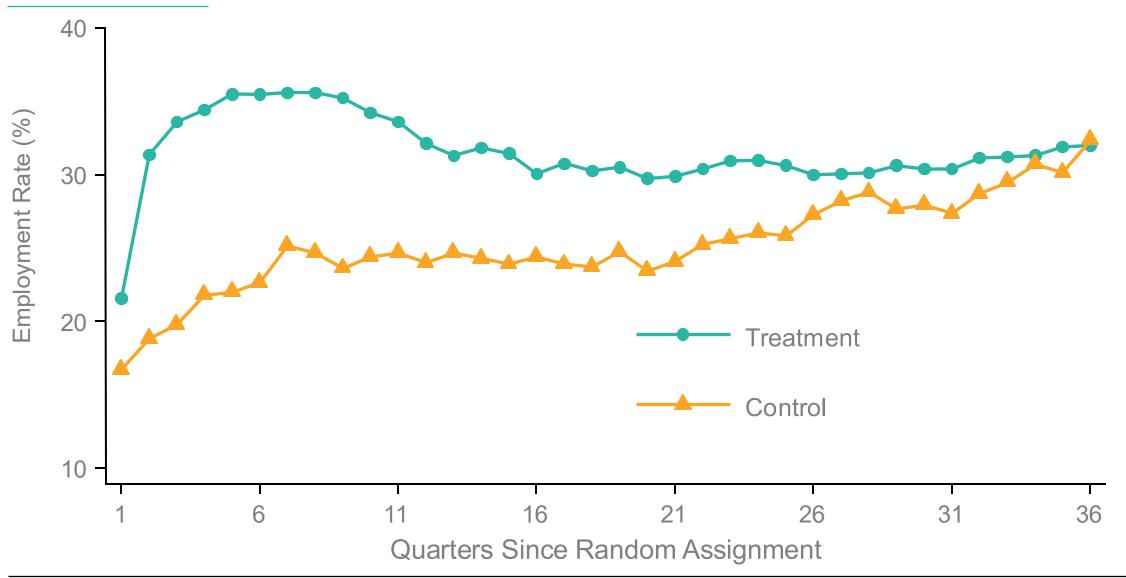
Evaluated using randomized trials in Oakland, Los Angeles, Riverside, and San Diego

 Focus first on Riverside program, which had the largest impacts, perhaps because of its "jobs first" (rather than training-based) approach

Then return to the other sites, which we use for out-of-sample validation

Employment Rates in Treatment vs. Control Group

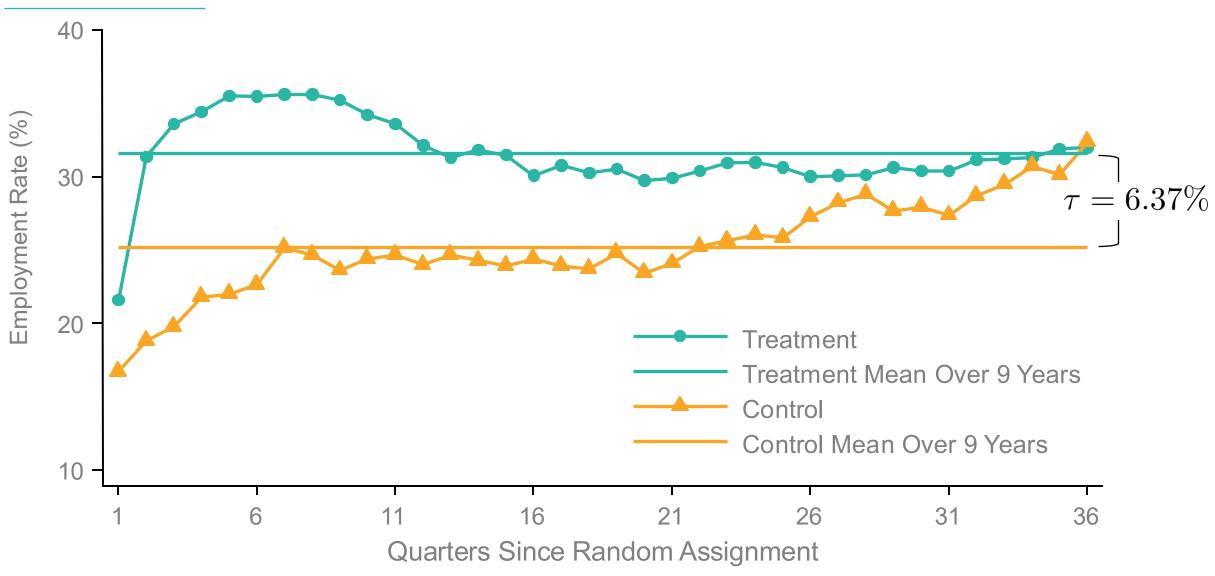
By Quarter in Riverside



Source: Hotz, Imbens, Klerman (2006); Athey, Chetty, Imbens, Kang (2025)

Employment Rates in Treatment vs. Control Group

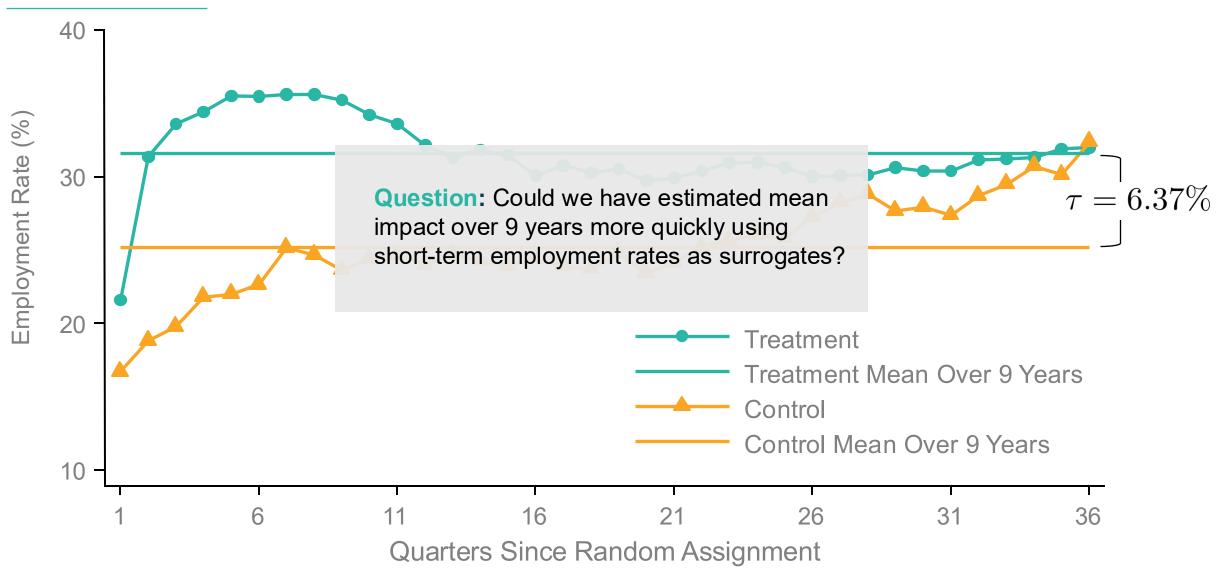
By Quarter in Riverside



Source: Hotz, Imbens, Klerman (2006); Athey, Chetty, Imbens, Kang (2025)

Employment Rates in Treatment vs. Control Group

By Quarter in Riverside



Source: Hotz, Imbens, Klerman (2006); Athey, Chetty, Imbens, Kang (2025)

Construction of the Surrogate Index

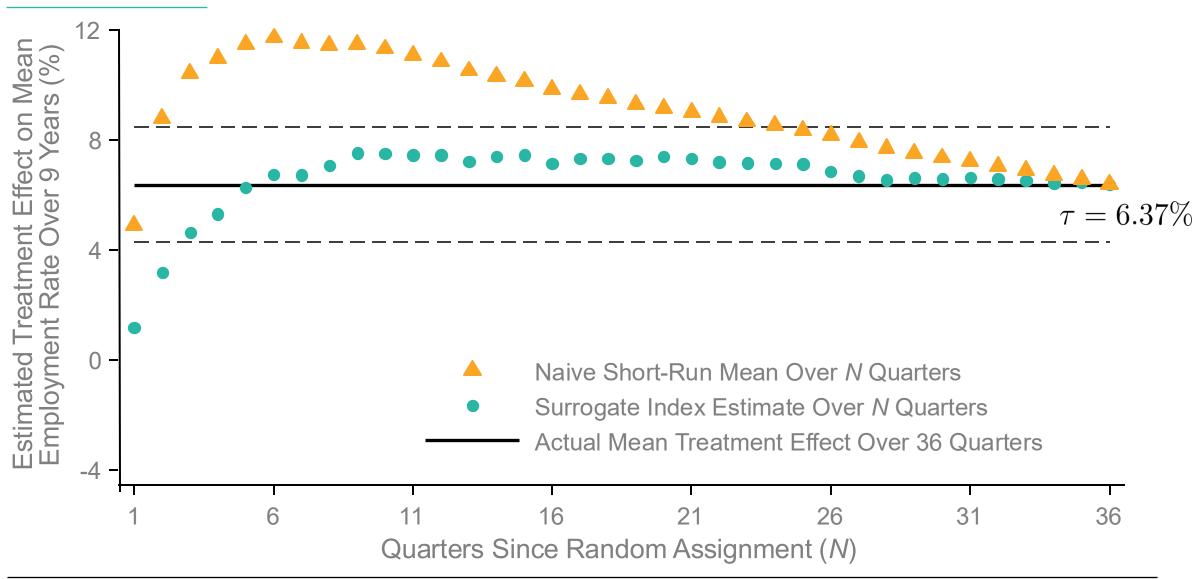
- Construct surrogate index using data on quarterly employment rates immediately after job training program
- Regress mean employment rate over 36 quarters on employment indicators from quarter 1 to quarter N:

$$\overline{Y}_{iT} = \beta_0 + \sum_{n=1}^{N} \beta_n Y_{in} + \epsilon_i$$

- Then estimate treatment effect on surrogate index based on employment rates up to quarter N
- Evaluate how quickly (at what value of N) we can estimate nine-year mean impact accurately

Estimate of Treatment Effect on Mean Employment Rate Over Nine Years

Varying Quarters of Data used to Construct Estimate



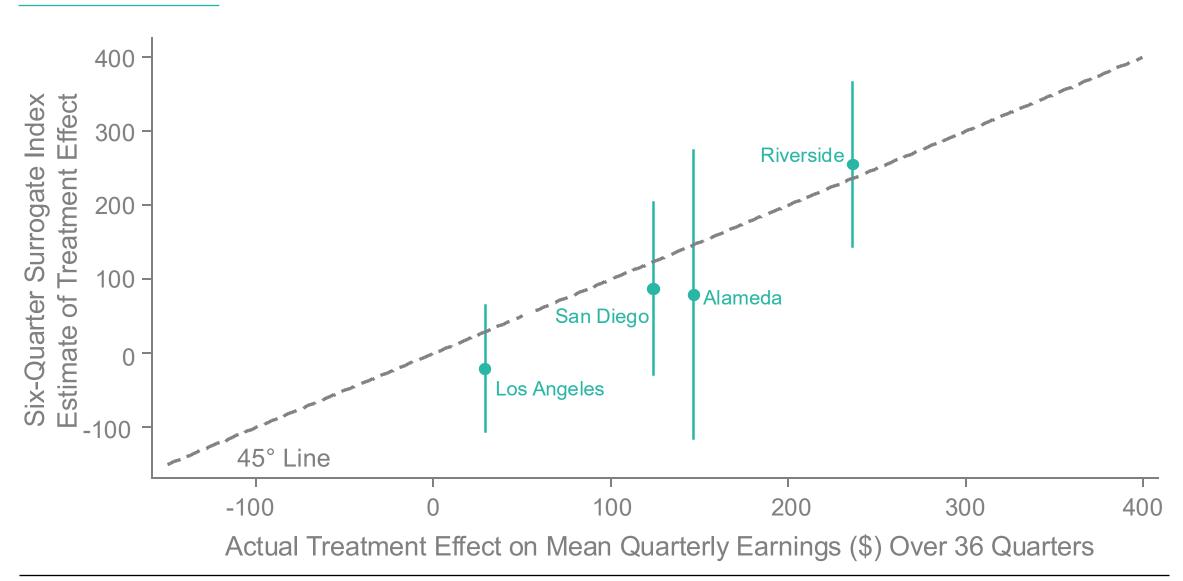
Source: Athey, Chetty, Imbens, Kang (2025)

Out of Sample Validation: Predicting Cross-Site Heterogeneity

- Now use six-quarter surrogate index estimated in Riverside and ask how well it performs in predicting heterogeneity in treatment effects across other sites
 - Out-of-sample validation that jointly tests surrogacy and comparability assumptions

Surrogate Index Estimates vs. Actual Experimental Estimates, by Site

Mean Quarterly Earnings (\$) Over Nine Years

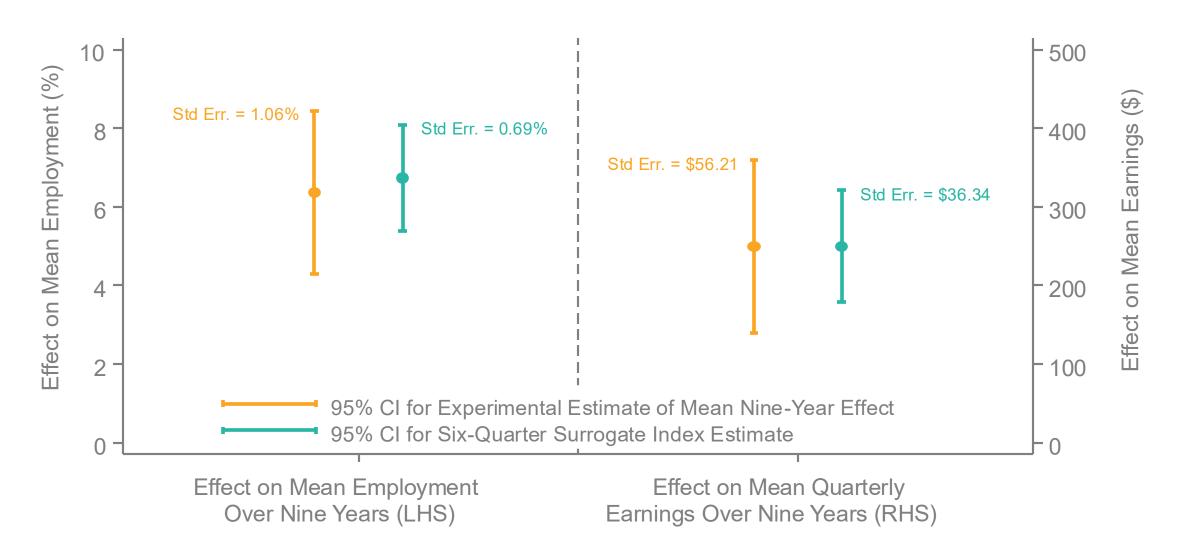


Source: Athey, Chetty, Imbens, Kang (2025)

Secondary Benefit of Surrogate Indices: Gains in Precision

- Even when evaluating impacts with nine-years of data, may be preferable to use surrogate index based on first six quarters to gain precision
 - Intuition: under surrogacy assumption, any residual variation in long-term outcome conditional on surrogate index is noise orthogonal to treatment
 - Reduce standard errors by estimating treatment effect on surrogate index instead of long-term outcome itself

Gains in Precision from Using Surrogate Index



Source: Athey, Chetty, Imbens, Kang (2025)

Further Reading on Surrogate Indices

Identification with Alternative Data Configurations

- Battocchi et al. 2021 [dynamically adjusted treatments]
- Bibaut et al. 2023 [combining many weak experiments]
- Rambachan, Singh, Viviano 2025 [post-treatment outcomes]

Estimation

- Chen and Ritzwoller 2022 [efficient influence function]
- Kallus and Mao 2024 [role of surrogacy assumption in efficiency]

Applications

- Chetty, Deming, Friedman 2023
- Carlana, Miglino, Tincani 2024
- Athey, Castillo, Chandar 2025



Remotely Sensed Variables

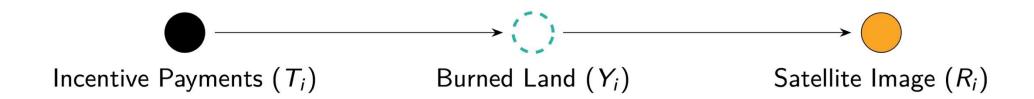
Predicting Treatment Effects Using Post-Treatment Outcomes

Post-Outcome Remotely Sensed Variables

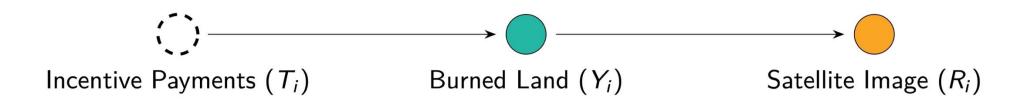
- In some settings, variables used as proxies for primary outcomes are not intermediate outcomes but instead measured after the primary outcome
- Example: Jack, Jayachandran, Kala, and Pande (2025) randomize incentive payments (T_i) to farmers in India to stop burning crop residue from land (Y_i)
 - Since objective measures of land burning are costly to obtain, use satellite images (remotely sensed variable R_i) to predict burning

Post-Outcome Remotely Sensed Variables: Setup

Experimental Data



Observational Data

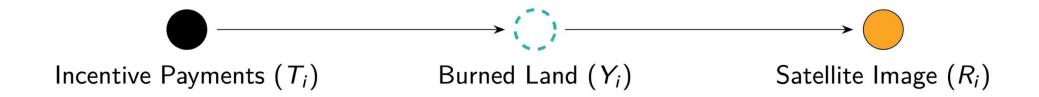


Post-Outcome Remotely Sensed Variables

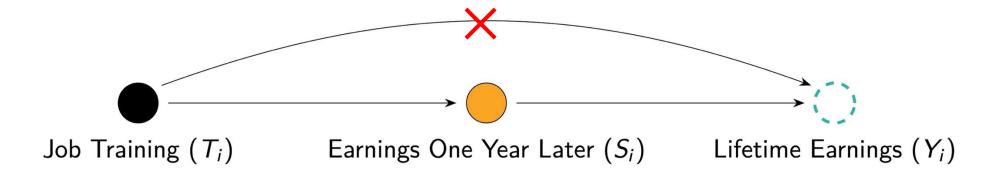
- Common, intuitive practice is to use surrogate approach in such settings: predict primary outcome Y_i using R_i and then estimate treatment effect on predicted outcome
- Rambachan, Singh, Viviano (2025) show that this generally yields biased estimates, because surrogacy assumption is violated: T_i directly affects Y_i independent of R_i .

Remote Sensing vs. Surrogate Model

Experimental Data in Remote Sensing Model



Experimental Data in Surrogate Model



Post-Outcome Remotely Sensed Variables

- Common, intuitive practice in such settings is to use surrogate approach: predict primary outcome Y_i using R_i and then estimate treatment effect on predicted outcome
- Rambachan, Singh, Viviano (2025) show that this generally yields biased estimates, because surrogacy assumption is violated: T_i directly affects Y_i independent of R_i
- They show how one can identify treatment effect on Y_i in general setting without parametric restrictions
- Focus here on a linear example that captures key difference relative to surrogate model

Identification Using Remotely Sensed Variables: Key Assumptions

Assumption 1. Unconfounded Treatment in Experimental Sample

$$T_i \perp \!\!\! \perp (Y_i(0), Y_i(1)) \mid G_i = E$$

Assumption 2. Stability Across Experimental and Observational Samples

$$R_i \mid Y_i, T_i, G_i = E \sim R_i \mid Y_i, T_i, G_i = O$$

Assumption 3. No Direct Effect of T_i on post-treatment outcome R_i conditional on Y_i

$$T_i \perp \!\!\!\perp R_i \mid Y_i$$

Source: Rambachan, Singh, Viviano (2025)

Stability Assumption in Linear Case

• Suppose the conditional expectation of $R_i \mid Y_i$ is linear in both samples:

Experimental Data:
$$E[R_i \mid Y_i, G_i = E] = \alpha_E + \beta^E \cdot Y_i$$

Observational Data:
$$E[R_i \mid Y_i, G_i = O] = \alpha_O + \beta^O \cdot Y_i$$

In this case, stability requires that the slopes are the same in both samples:

$$\beta^{\mathsf{E}} = \beta^{\mathsf{O}} = \beta$$

Identification Using Remotely Sensed Variables

Under Assumptions 1-3 (and regularity conditions), treatment effect on Y_i is:

$$ATE_{Y} = \{E[R_{i} \mid T_{i} = 1, G_{i} = E] - E[R_{i} \mid T_{i} = 0, G_{i} = E]\} \cdot \frac{1}{\beta}$$
 $ATE_{Y} = ATE_{R}/\beta$

Three steps to estimate treatment effect on Y_i :

- Regress the RSV on treatment in experimental data to identify ATE_R
 reg satellite_prediction T if exp == 1
- 2. Regress the RSV on outcomes in observational data to estimate β reg satellite_prediction burned_land if obs == 1
- 3. Divide ATE_R from (1) by β from (2)

Application of Remote Sensing Estimator

$$ATE_{Y} = ATE_{R}/\beta$$

	(1)	(2)	(3)
Estimand	$\mathrm{ATE}_{\mathrm{R}}$	$d\mathbf{R}$ / $d\mathbf{Y}$ (β)	$\mathrm{ATE_Y} = \mathrm{ATE_R} \; / \; \beta$
Estimate	0.079^{*}	0.530***	0.148*
	(0.041)	(0.072)	(0.084)

Note: R_i is a prediction that a farmer has not burned their field, based on satellite imagery in Jack et al. (2025).

Intuition: satellite prediction captures 53% of actual non-burned plots > inflate treatment effect on satellite prediction by 1/.53 to get effect on Y

Source: Rambachan, Singh, Viviano (2025)

Experimental Selection Correction

Controlling for Selection Using Intermediate Outcomes

Failures of Surrogacy Assumption

Many applications where surrogacy assumptions may not hold

 Test scores often used as a surrogate for long-term outcomes (e.g., high school graduation or earnings)

 But early interventions affect long-term outcomes through many channels besides test scores [e.g., Heckman et al. 2006, Deming 2009, Chetty et al. 2011]

How Can We Make Progress When Surrogacy Assumption Fails?

How can we make progress when we have data on intermediate outcomes such as test scores but they do not satisfy surrogacy assumptions?

- One approach: experimental selection correction [Athey, Chetty, Imbens 2025]
 - Key requirement: must observe treatment (T_i) along with intermediate and long-term outcomes (S_i, Y_i) in observational data
 - With this additional information, we can strictly weaken surrogacy assumptions

- Goal: causal inference when we have observational data on long-term outcomes for interventions implemented in the past (e.g., class size reduction)
 - Not applicable to new interventions for which we do not yet have long-term outcome data (unlike surrogate estimator)

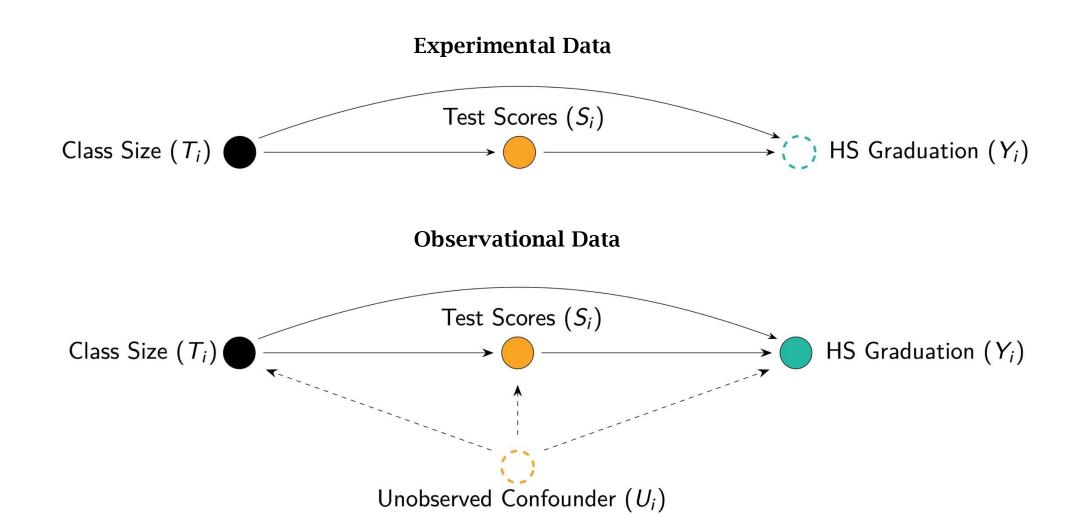
Treatment Effects of Class Size: Experimental vs. Observational Data

	$G_i = E$ Experimental (Tennessee STAR)	$G_i = O$ Observational (NYC Admin Data)
Treatment Effect of Assignment to Small 3 rd Grade Class on:		
3^{rd} Grade Test Scores (S_i)	0.19 SD (0.04)	-0.12 SD (0.01)
HS Graduation Rate (Y _i)	?	-1.76 pp (0.29)

Note: All estimates based on OLS regressions, controlling for school x cohort fixed effects

Source: Athey, Chetty, Imbens (2025)

Experimental Selection Correction Model: Setup



Experimental Selection Correction: Key Identification Assumptions

Assumption 1. Unconfounded Experiment

$$T_i \perp \!\!\! \perp (Y_i(0), Y_i(1), S_i(0), S_i(1)) \mid G_i = E$$

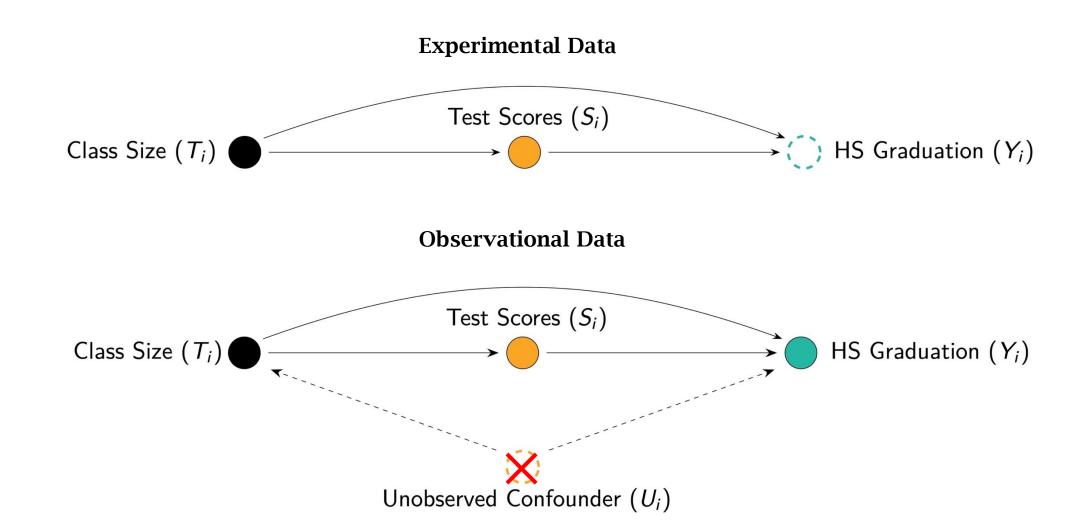
Assumption 2. External Validity of Experimental Sample for Observational Sample

$$Y_{i}(t), S_{i}(t) \mid G_{i} = E \sim Y_{i}(t), S_{i}(t) \mid G_{i} = O$$

Assumption 3. Latent Unconfoundedness: Confounds that affect S_i and Y_i are the same

$$T_i \perp \!\!\!\perp Y_i(t) \mid S_i(t), G_i = O \quad \forall t$$

Violations of Latent Unconfoundedness Assumption



Linear Case

Linear models for intermediate and long-term outcomes

$$S_i = \tau^{\mathsf{S}} \ T_i + \alpha_i^{\mathsf{S}}$$
 $Y_i = \tau^{\mathsf{Y}} \ T_i + \alpha_i^{\mathsf{Y}}$ score class size parental input HS graduation class size residual variation in Y

- $(\alpha_i^{\mathsf{Y}}, \alpha_i^{\mathsf{S}}) \perp \!\!\! \perp T_i$ in experimental sample, but not in observational sample
- Here, latent unconfoundedness assumption requires:

$$\alpha_i^{\mathsf{Y}} = \delta \alpha_i^{\mathsf{S}} + \epsilon_i \quad \text{with} \quad T_i \perp \!\!\! \perp \epsilon_i \mid \alpha_i^{\mathsf{S}}, G_i = O$$

• Once we control for parental input, residual variation in graduation rates (ϵ_i) must be independent of class size (T_i) in observational sample

Identification in Linear Case

• If we observed α_i^S (parent input), we could identify treatment effect by controlling for α_i^S :

$$Y_i = \tau^{\mathsf{Y}} T_i + \gamma \ \alpha_i^{\mathsf{S}} + \epsilon_i$$

• We do not observe α_i^S , but can infer it by comparing scores in observational sample to **predicted** scores based on the experimental estimate:

$$\alpha_i^{\mathsf{S}} = S_i - \hat{\tau}^{\mathsf{S}} T_i$$

where $\hat{\tau}^S$ is estimated in the experimental sample.

In application, we can infer that there is negative selection into smaller classrooms in NYC schools because children in smaller classes have lower test scores

Identification with Experimental Selection Correction

Under Assumptions 1-3 (and regularity conditions), treatment effect on Y_i is:

$$ATE_{Y} = E[Y_{i} \mid T_{i} = 1, \alpha_{i}^{S}, G_{i} = O] - E[Y_{i} \mid T_{i} = 0, \alpha_{i}^{S}, G_{i} = O]$$

Three steps to estimate treatment effect on Y_i :

1. Regress S_i on T_i in experimental sample to estimate τ^S

2. Construct residuals of S_i in the observational sample based on predicted values

```
predict score_pred
gen selection = score - score_pred if obs == 1
```

3. Regress Y_i on T_i in observational sample, controlling for residuals from Step 2

```
reg graduation treatment selection if obs == 1
```

Experimental Selection Correction: Connections to Existing Estimators

- Analogous to control function methods with instrumental variables
 - In IV models, can identify by controlling for first-stage residuals (which captures endogenous variation due to selection) in second-stage estimating equation
 - Here, control for difference between observed short-term outcome and prediction based on experimental estimate (which again captures variation due to selection)

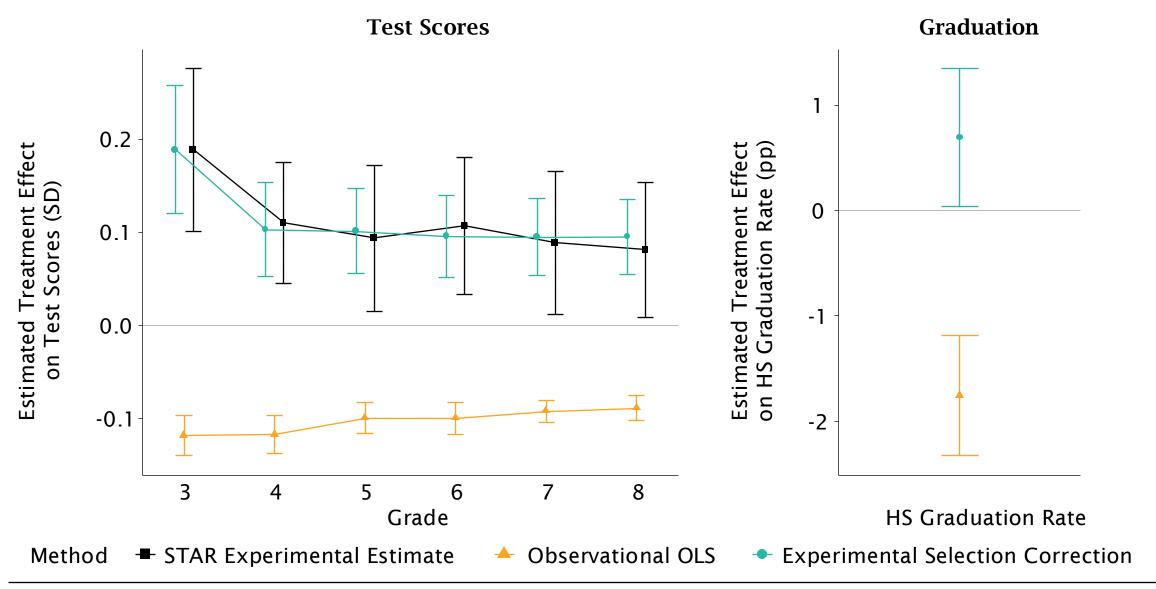
- Also connected to traditional selection correction based on parametric assumptions and exclusion restrictions [e.g., Heckman 1979]
 - Here, selection correction is based on an experimental estimate of treatment effect on intermediate outcome

Application: Causal Effects of Class Size on High School Graduation

Observational data from New York City public schools with information on 3rd grade class size, subsequent test scores, and high school graduation [Chetty, Friedman, Rockoff 2014, Mariano et al. 2024]

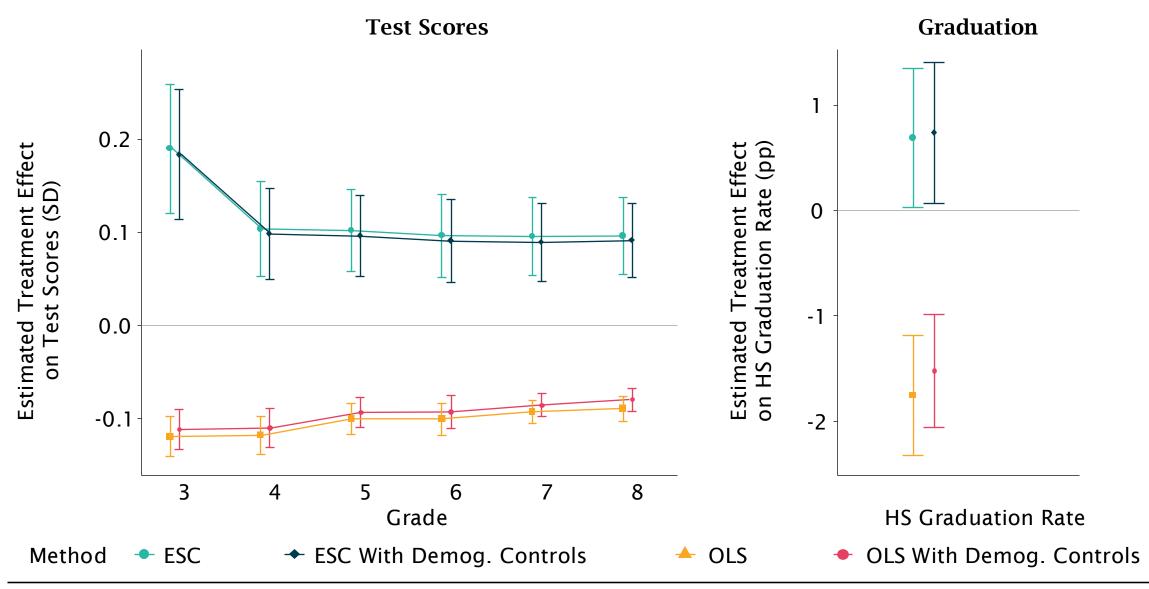
- Experimental data from Tennessee STAR, which randomly assigned 12,000 children to small vs. large classrooms [see e.g., Krueger 1999]
 - Many papers on impacts of STAR on short-term outcomes, but much less evidence on longterm impacts; no estimates of impacts on graduation rates in particular

Treatment Effects of Assignment to Small Class in 3rd Grade



Source: Athey, Chetty, Imbens (2025)

Effect of Controlling for Observables on Treatment Effect Estimates



Source: Athey, Chetty, Imbens (2025)

Further Reading on Experimental Selection Correction

Identification with Alternative Assumptions

- Heckman and Pinto 2015, Garcia et al. 2020 [assumptions based on economic theory]
- Ghassami et al. 2022 [parallel trends]
- Park and Sasaki 2024a,b [partial identification]

Identification with Alternative Data Configurations

- Obradovic 2024 [partial identification with additional instruments]
- Imbens et al. 2025 [identification with sequential intermediate outcomes]

Estimation

Meza and Singh 2024 [nested nonparametric IV]

Applications

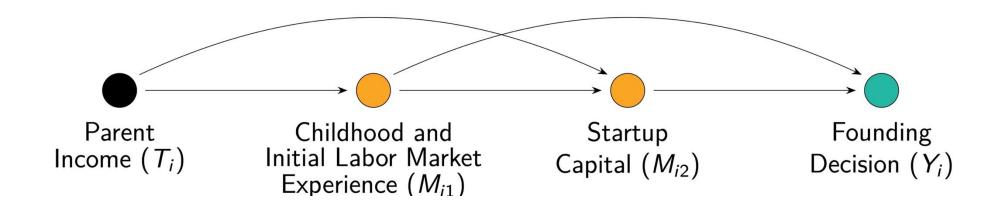
Aizer, Early, Eli, Imbens, Lee, Lleras-Muney, Strand 2024



Application: Determinants of and Returns to Entrepreneurship

Now apply these methods to return to motivating example of determinants and returns to entrepreneurship [Chetty, Dossi, Smith, van Reenen, Zidar, Zwick 2025]

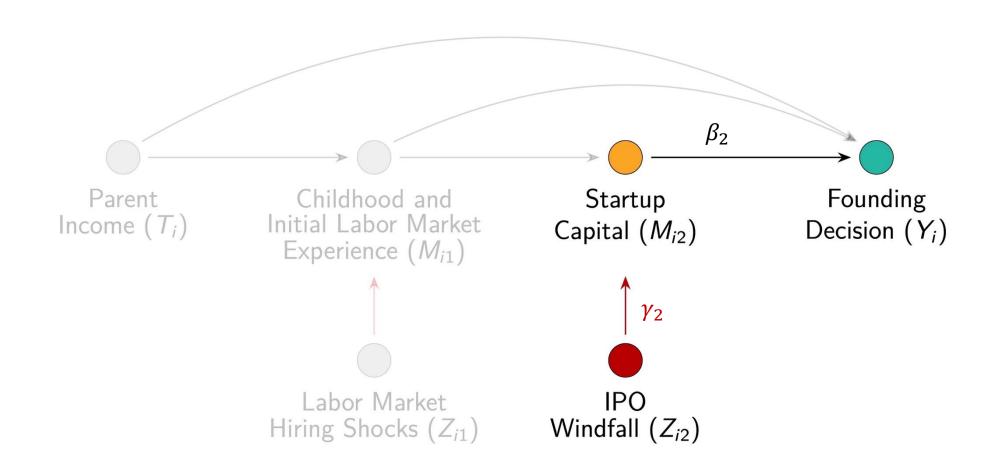
The Entrepreneurial Pipeline: Causal Mechanisms



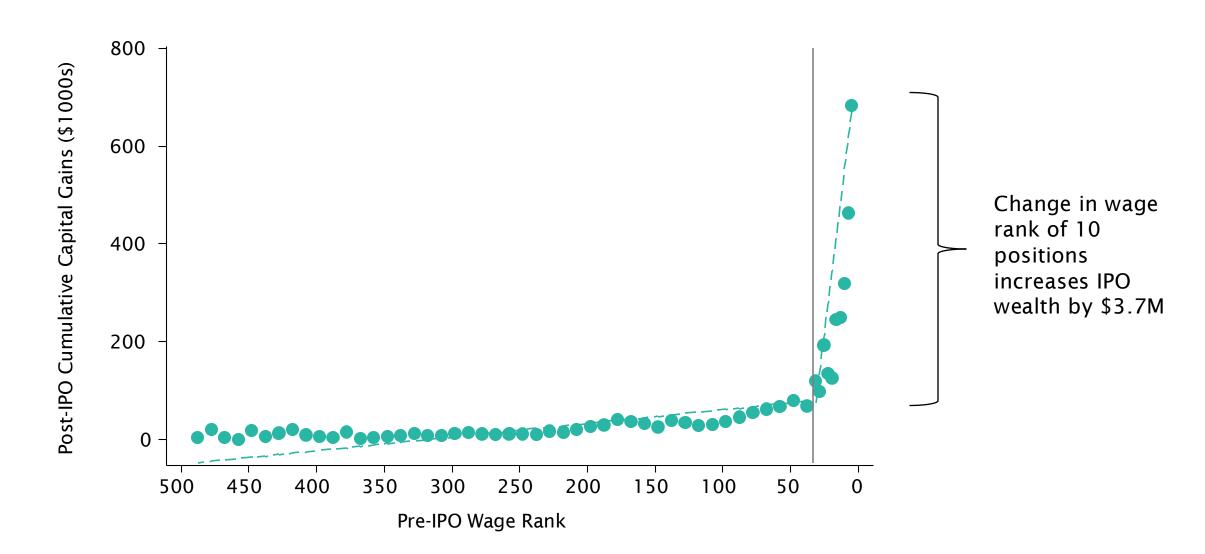
Identifying Mediating Mechanisms

- Use multiple instruments to obtain exogenous variation in mediators and quantify role of each mechanism
- Start by assuming homogeneous treatment effects and additivity of treatment effects
- Then revisit validity of these assumptions

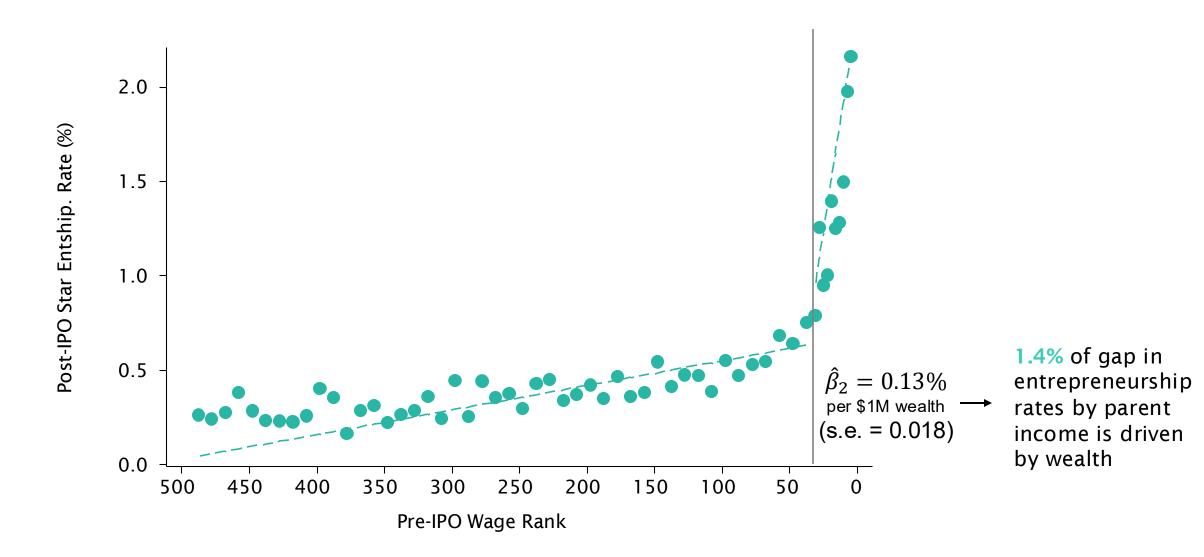
The Entrepreneurial Pipeline: Causal Mechanisms



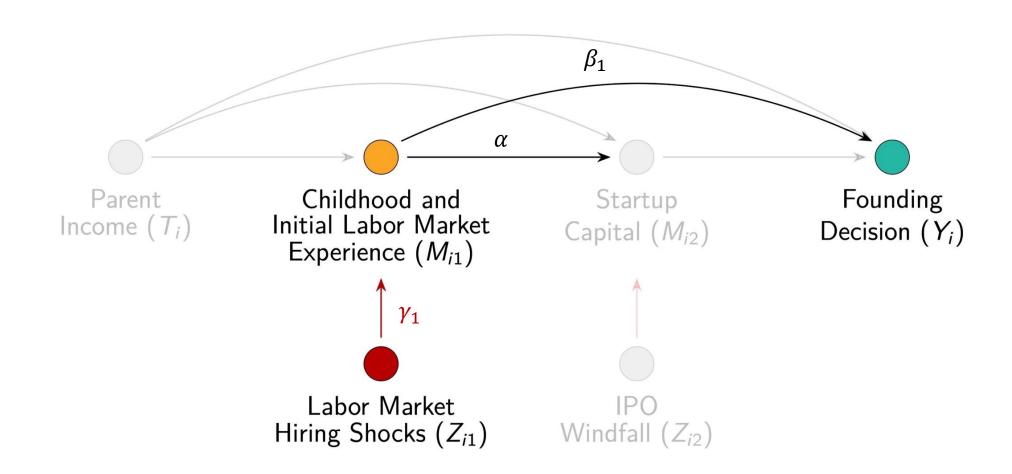
Wealth Windfall by Wage Rank in Initial Public Offerings



Impact of IPO Cash Windfalls on Subsequent Entrepreneurship Rates



The Entrepreneurial Pipeline: Causal Mechanisms



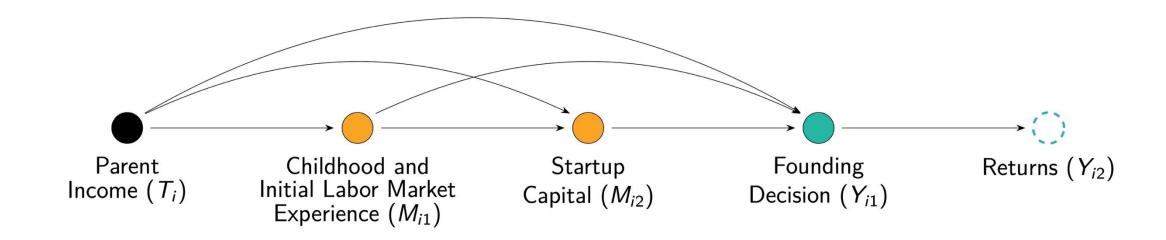
Addressing Potentially Heterogeneous Treatment Effects

- Key implication of mediation analysis: Wealth at startup relatively unimportant in explaining gaps compared to upstream pipeline (e.g., sector of first job)
- Conclusion hinges on assuming homogeneous treatment effects. Two concerns:
 - Samples used to estimate treatment effects in each design differ on observables (e.g. age, cohort)
 - Solution: condition on observables by reweighting on cohort and age
 - Yields wealth effect that is similar to baseline estimate

Addressing Potentially Heterogeneous Treatment Effects

- Key implication of mediation analysis: Wealth at startup relatively unimportant in explaining gaps compared to upstream pipeline (e.g., sector of first job)
- Conclusion hinges on assuming homogeneous treatment effects. Two concerns:
 - Samples used to estimate treatment effects in each design differ on observables (e.g. age, cohort)
 - 2. LATE estimated by instruments may differ due to unobserved heterogeneity: those involved in IPOs may have good access to capital markets already
 - Solution: use instruments that generate different LATEs
 - Similarly small effects on business startup estimated using lottery winnings [Golosov et al. 2024]

The Entrepreneurial Pipeline: Predicting Returns



What are the marginal returns to new businesses startups induced by a shock to labor market experience? Are they as successful as existing businesses?

Predicting Returns Using a Surrogate Index

- Long-term returns (e.g., 8-10 years after startup) observed for few firms due to censoring
- Use surrogate index to predict outcomes using historical data where we observe both short-run (2 year after founding) and long-run (8-year) outcomes
 - Regress eight-year returns on revenues, wages, and number of employees two years after founding
- Compare predicted returns of marginal entrepreneurs induced to enter by labor market experience shock to returns of average entrepreneur
 - Key finding: returns of marginal entrepreneur are very similar to those of average entrepreneur

Takeaways from Empirical Application

- 1. IV-based mediation analysis shows that low-income children are less likely to start firms because of a lack of exposure to entrepreneurial industries, not lack of wealth
 - Opposite of conclusion obtained from controlling for wealth
 - Sequential ignorability assumption fails here because wealth is correlated with many other unobservable factors that directly affect entrepreneurship
- Surrogate indices reveal that increasing labor market experience induces startups whose returns are similar to current mean

→ Early-career incubator or apprenticeship programs targeted to certain subgroups could increase business startups and growth significantly

Start by writing down causal graphs (DAGs) underlying empirical analysis

- Mediation analysis:
 - 1. Avoid common practice of controlling for endogenous regressors unless sequential ignorability (conditionally exogenous mediators) can be justified
 - Generally need to find instruments or experiments that vary mediators, as we do for treatment itself in design-based paradigm
 - 3. Start by assuming additivity + no heterogeneity, then evaluate robustness by reweighting, estimating different LATEs, and estimating interaction effects

- Predicting long-term/primary outcomes using intermediates or proxies:
 - 1. When intermediate outcomes are likely to span set of causal pathways to later outcomes, use surrogate index to maximize efficiency
 - Often works well when using early labor market or firm outcomes as surrogates for later outcomes, perhaps because there are certain paths to later success
 - When long-term outcome is observed, surrogate index may still be helpful for analyzing heterogeneity or additional tests that demand more power

- Predicting long-term/primary outcomes using intermediates or proxies:
 - 1. When intermediate outcomes are likely to span set of causal pathways to later outcomes, use surrogate index to maximize efficiency
 - 2. If surrogacy assumptions are debated and treatment is observed alongside long-term outcome in observational data, use experimental selection correction
 - Ex: when forecasting long-term impacts of early childhood interventions, where there are many causal pathways beyond those that run through test scores

- Predicting long-term/primary outcomes using intermediates or proxies:
 - 1. When intermediate outcomes are likely to span set of causal pathways to later outcomes, use surrogate index to maximize efficiency
 - 2. If surrogacy assumptions are debated and treatment is observed alongside long-term outcome in observational data, use experimental selection correction
 - 3. If proxies are observed post-treatment, use remote sensing estimator

- In general: validate any surrogate/proxy method using direct experimental or quasiexperimental estimates on holdout outcomes
 - Both in a given paper and more generally as a field → will yield better understanding of best proxies and causal pathways

Potential Directions for Further Methodological Research

- Alternative assumptions and data configurations
 - Ex: Identification when surrogacy fails and treatment unobserved in observational data
- 2. Incorporating common issues in applications
 - Ex: Measurement error in surrogates or short-term outcomes; noncompliance in experiments; dynamic selection; spillover effects
- 3. Target parameters beyond average treatment effect
 - Ex: quantile treatment effects; marginal treatment effects; heterogeneity
- 4. Connections to traditional econometric methods
 - Ex: data combination; panel data [e.g., Ridder and Moffitt 2007, Arkhangelsky and Imbens 2024]

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