Notes for long version

1. Translate the wording for everyone (mechanism, quasi-random), and be clearer about suggestive. Use words like necessary but not sufficient.
2. Needs a clearer introduction, which accurately overviews and previews the approach findings
3. Novelty needs to be loud, so put it first, Write ATE $=$ ADE $+$ AIE for Levon, and enumerates folks theorems for why CM did not take off in econ but did in medicine epi psych)
4. Evan Riehl recommends a slide with quotes from top 5s that investigates mechanisms (note the approach is necessary but not sufficient for mechanism analysis)
5. Mention Kwon Roth result on my data, reject null then move on....
6. Longer presentation needs clear reasoning on the IV.

Notes for long version: empirical IV

1. Longer explanation of the IV in Oregon for applied audience
2. Options for the included IV, mainly to consider as illustrative (and do not want people to expect a super clean IV, but then get an illustrative one).
3. Talk through the quasi-experimental concerns (why is D_i endogenous?)
4. Show the IV set-up (clean pre-Z_i first-stage, but exclusion restriction maybe lacking). Can it be made binary to simplify the interpretation (and linearise the estimation?).
5. Develop at least one slide that talks through the controlling for *already diagnosed* illnesses
6. See what the CM estimates look like without controlling for them already.

Causal Mediation in Natural Experiments

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Cornell, Labor Economics Seminar
17 November 2025

Presentation Plan

Introduction

First ten minutes: introduction, road-map, and preview of findings.

- Mechanism are important (show top 5 quotes), and we do not know much about them and given (at best) suggestive evidence.
- CM is a framework from elsewhere which gives sufficient evidence, though has not taken off in economics
- I give explicit reasoning for why conventional CM methods are unlikely to work in applied econ settings, then develop a structural approach to get back to what you want.
- Apply these methods to Oregon, showing how these methods work in practice (suggestive, conventional CM, then my MTE approach).

For this part, no model and no maths notation, just vibes.

Presentation Plan

1. Oregon

Second ten-minutes, the model and suggestive evidence in the OHIE.

- Introduce the OHIE, and say why the mechanisms are important and unknown
- Show suggestive evidence
- Question whether suggestive evidence is enough, when $D_i \to Y_i$ correlation is zero.

Presentation Plan

2. Causal Mediation

15-minutes, the CM framework.

- Model: Define the ADE and AIE, identifying assumption, and thus sufficient evidence on this mechanism.
- Explain why this does not hold (e.g., Roy model), and get selection bias (figure of simulation)
- Show the figure of conventional CM (i.e., assume the mechanism) estimate for OHIE. Explain why MI would not hold for Oregon

Presentation Plan

3. **CM with Selection** 15-minutes, My approach to CM.

- Write the two-stage equation, giving intuition on where non-identification comes from (second-stage).
- Use my 3 assumption approach to identify the mediator mechanism MTE, then ADE + AIE (write equations, and short guide, but be careful)
- Explain estimation with my simulation figures.

Presentation Plan

4. **Return to Oregon** 10-minutes, my application of CM.

- Re-introduce the setting (good lottery first-stage, but hospital visits are free choice).
- Walk through my MTE model, with monotonicity + relevance, then walk through the IV (if illustrative)
- Show my estimates of CM effects, then give discussion of findings. Include an adjusted slide of not controlling for pre-diagnosed illnesses, alluding to Oster (2019) reasoning on controlling for more ailments to get a sharper answer.

Finding notes: very different CM effects from conventional, noting direct (psychological) effects of socialised healthcare access. Agreed with suggestive evidence, giving clear uncertainty on the proportion of the mechanism. Note $\rightarrow$ apply the Kitagawa (2015) Kwon Roth (2024) test to these data rejects null (there are direct effects).

Presentation Plan

Conclusion Bullet-point overview of everything that has happened..

Causal Mediation in Natural Experiments

Natural experiments gives credible estimates of causal effects

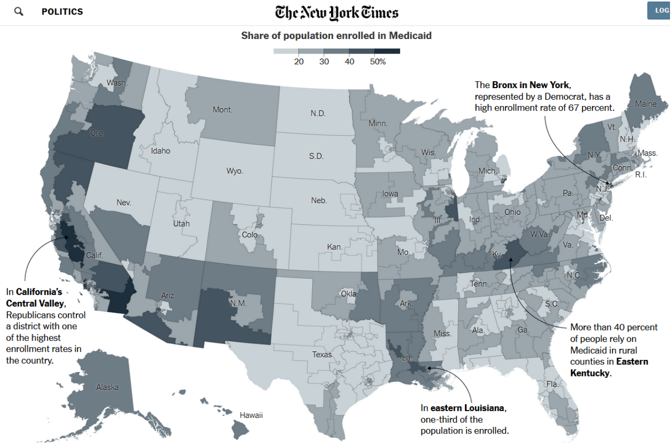
- Little information on the mechanisms through which they operate
- Limits understanding of the decisions and underlying economic system
-

School meals $\rightarrow$ adult income, because increased education (Lundborg Rooth Alex-Petersen 2022).

Senan note: unsure how to start the presentation with 5 minutes that grab attention. Just start section 1, introducing the data succinctly.

Oregon Health Insurance Experiment

In the USA, healthcare is only provided by the government in special cases $\rightarrow$ Medicaid is the government programme which provides health insurance for those close to the poverty line (> 70 million people in 2025).



Oregon Health Insurance Experiment

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2004: Oregon Medicaid enrolment frozen

↓

2008: Enrolment reopens...
90,000 sign up (far exceeding funding allocated)

↓

Wait-list Lottery:

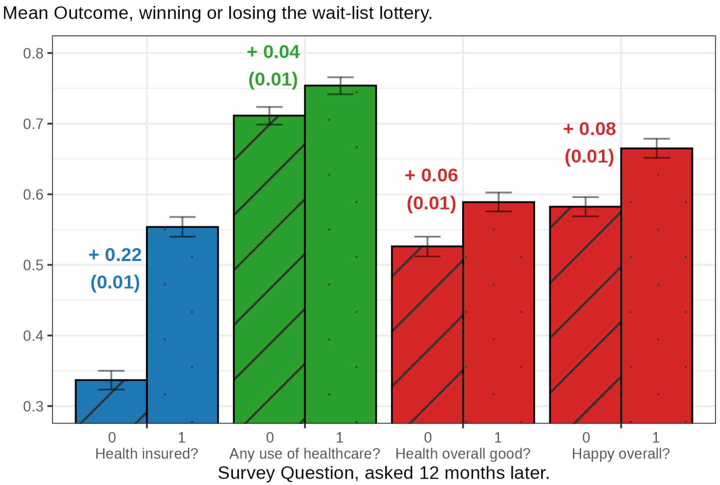
↙ ↘

35,000 Won (Treatment)

55,000 Lost (Control)

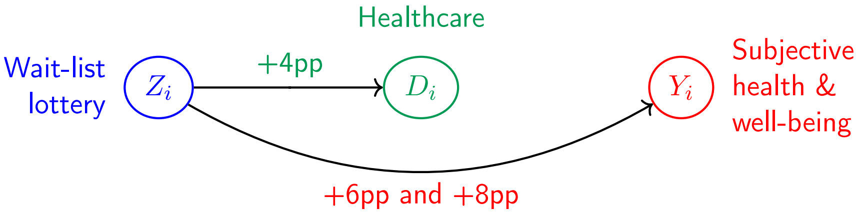
Oregon Health Insurance Experiment

Winning this wait-list lottery significantly increased healthcare usage, plus subjective health and well-being (Finkelstein et al, 2012).



Oregon — Suggestive Evidence

Winning this wait-list lottery significantly increased healthcare usage, plus subjective health and well-being (Finkelstein et al, 2012).



Suggestive evidence:

- If first-stage $\neq 0$, then healthcare may be a mediating mechanism
- This gives **suggestive evidence for healthcare as mechanism**.

Oregon — Suggestive Evidence

Suggestive evidence is primarily how economics investigates mechanisms.

Abstract — Lundborg Rooth Alex-Petersen (2022, ReStud).

"…Exposure to the [free school meals] programme also had substantial effects on educational attainment and health, which can explain a large part of the effect of the programme on lifetime income."

Abstract — Bloom Mahajan McKenzie Roberts (2013, QJE).

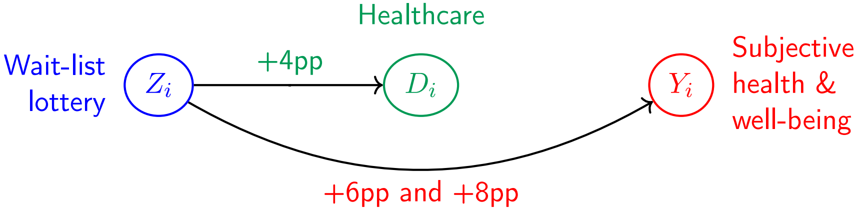
"…We find that adopting these management practices had three main effects. First, it raised average productivity by 11% through improved quality and efficiency and reduced inventory […]."

Abstract — Carvhalo (2025, JPE Micro).

"… Evidence suggests fluid intelligence and self-control partly mediate the relationship between the [education polygenic index] and education."

Oregon — Suggestive Evidence

Winning this wait-list lottery significantly increased healthcare usage, plus subjective health and well-being (Finkelstein et al, 2012).

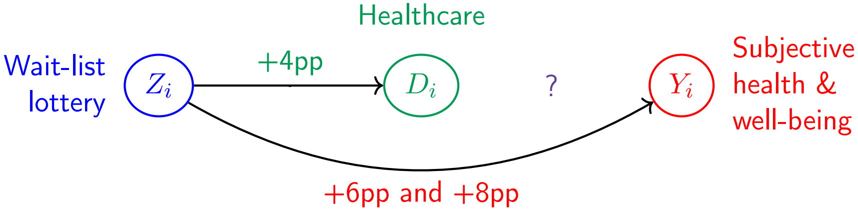


What about direct effects?

- Winning access to Medicaid means you can file for free health insurance (income effect)
- Less stress from no longer having to be uninsured (psychological gains).

Oregon — Suggestive Evidence

Winning this wait-list lottery significantly increased healthcare usage, plus subjective health and well-being (Finkelstein et al, 2012).



There is one missing piece to make a **definitive conclusion**:

Size of causal effect $D_i \to Y_i \ldots$

- If large, then healthcare explains all the lottery effect
- If small/zero then, then all direct (e.g., psychological) gains.

Causal Mediation (CM)

CM is an alternative framework to studying mechanisms, giving sufficient evidence on the mediating mechanism.

$$\text{Wait-list lottery} \quad Z_i \xrightarrow{\text{Treatment Effect (ATE)}} Y_i \quad \text{Subjective health \& well-being}$$

Define

- Treatment $Z_i = 0, 1$, wait-list lottery
- Mediator mechanism $D_i = 0, 1$, healthcare usage
- Outcome Y_i, subjective health and well-being.

CM aims to decompose the ATE in two channels, direct and indirect effects

$$\text{ATE} = \text{ADE} + \text{AIE}.$$

Causal Mediation (CM)

CM is an alternative framework to studying mechanisms, giving sufficient evidence on the mediating mechanism.

Write $D_i(z')$ and $Y_i(z', d')$ for the potential outcomes.

Two average causal effects are identified, with Z_i randomly assigned:

1. Average first-stage

$$\mathbb{E}\left[D_i(1) - D_i(0)\right] = \mathbb{E}\left[D_i \mid Z_i = 1\right] - \mathbb{E}\left[D_i \mid Z_i = 0\right]$$

2. Average Treatment Effect (ATE)

$$\mathbb{E}\left[Y_i(1, D_i(1)) - Y_i(0, D_i(0))\right] = \mathbb{E}\left[Y_i \mid Z_i = 1\right] - \mathbb{E}\left[Y_i \mid Z_i = 0\right].$$

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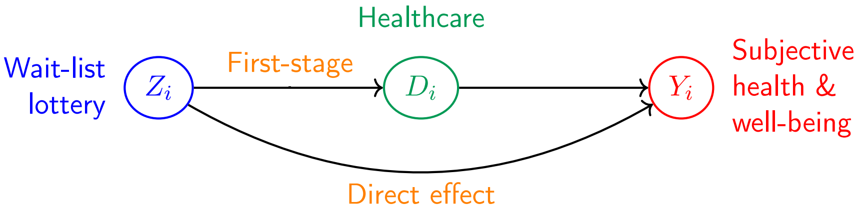


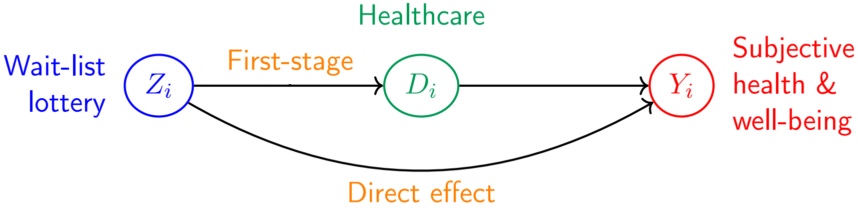
CM decomposes the ATE into components

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

AIE represents the average effect going through healthcare.

Causal Mediation (CM)

CM is an alternative framework to studying mechanisms, giving sufficient evidence on the mediating mechanism.



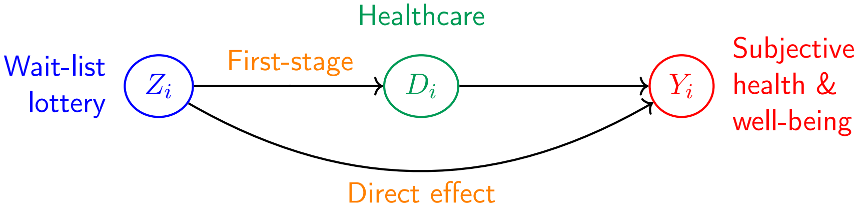
CM decomposes the ATE into components

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

ADE represents the average effect going absent healthcare.

Causal Mediation (CM)

CM effects (ADE + AIE) are not identified without further assumptions.

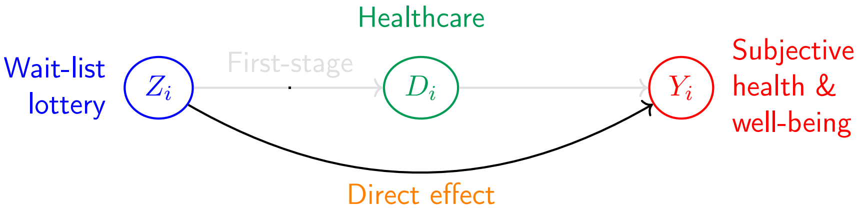


Conventional CM relies on two identifying assumptions for ADE + AIE,

1. Treatment Z_i is (quasi-)randomly assigned
2. Mediator D_i is (quasi-)randomly assigned, conditional on Z_i realisation (and covariates $\boldsymbol{X}_i$).

Causal Mediation (CM)

Under assumptions (1) + (2), the ADE + AIE are separately identified by two-stage regression (Imai Keele Yamamoto 2010).



ADE is the effect of Z_i after controlling for D_i

$$\begin{aligned}\text{ADE} &= \mathbb{E}\left[Y_i\left(\boxed{1}, D_i(Z_i)\right) - Y_i\left(\boxed{0}, D_i(Z_i)\right)\right] \\ &= \mathbb{E}\left[Y_i \,\middle|\, \boxed{Z_i = 1}, D_i\right] - \mathbb{E}\left[Y_i \,\middle|\, \boxed{Z_i = 0}, D_i\right].\end{aligned}$$

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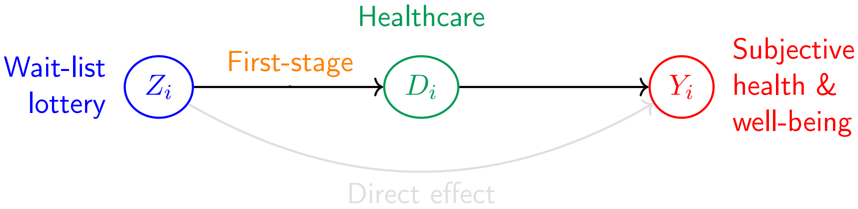


AIE is the effect of D_i after controlling for Z_i, times average first-stage.

$$\text{AIE} = \mathbb{E}\left[Y_i\left(Z_i, \boxed{D_i(1)}\right) - Y_i\left(Z_i, \boxed{D_i(0)}\right)\right]$$
$$= \left(\mathbb{E}\left[D_i \mid Z_i = 1\right] - \mathbb{E}\left[D_i \mid Z_i = 0\right]\right)$$
$$\times \left(\mathbb{E}\left[Y_i \,\middle|\, \boxed{D_i = 1}, Z_i\right] - \mathbb{E}\left[Y_i \,\middle|\, \boxed{D_i = 0}, Z_i\right]\right).$$

Causal Mediation (CM)

This approach (conventional CM) is used heavily in epidemiology and medicine to give evidence for the channels of a treatment effect, but there is a reason why this is not prominent in economics.

Identifying assumptions:

1. Treatment Z_i is (quasi-)randomly assigned
2. Mediator D_i is (quasi-)randomly assigned, conditional on Z_i realisation (and covariates $\boldsymbol{X}_i$).

Translation: Healthcare is a random choice, conditional on wait-list lottery realisation and demographic controls.

Would this be plausible in settings economists study?

Causal Mediation (CM) — Roy Model

Consider the case that people, after the lottery, choose to visit the doctor in the next 12 months based on subjective costs and benefits,

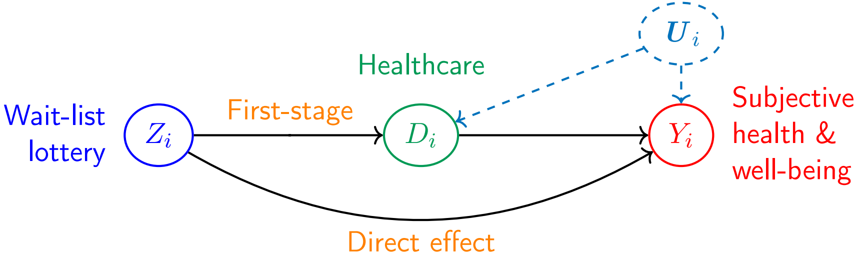
$$D_i\left(z'\right) = \mathbb{1}\left\{ \underbrace{C_i}_{\text{Costs}} \leq \underbrace{Y_i\left(z', \boxed{1}\right) - Y_i\left(z', \boxed{0}\right)}_{\text{Benefits}} \right\}.$$

The wait-list lottery has no strategic selection, but visiting healthcare after is an unconstrained choice.

Theorem: If choice to attend healthcare is unconstrained, based on costs and benefits (Roy model) and demographics do not explain all benefits $\implies$ mediator mechanism is not random, there is unobserved confounding.

Causal Mediation (CM) — Selection Bias

Individual unobserved benefits are an unobserved confounder $\boldsymbol{U}_i$ here,



In economic settings, Conventional CM analyses have bias similar to classical selection bias (Heckman Ichimura Smith Todd 1998).

- Direct: $\quad$ CM Estimand $=$ ADE $+$ (Selection Bias $+$ Group difference bias)
- Indirect: $\quad$ CM Estimand $=$ AIE $+$ (Selection Bias $+$ Group difference bias)

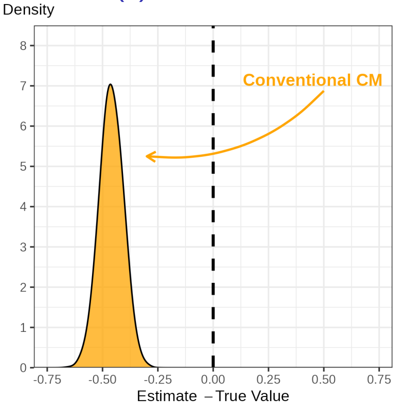
ADE biases AIE biases

Causal Mediation (CM) — Selection Bias

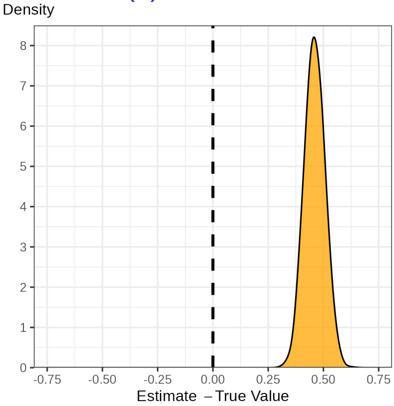
With strategic selection, the bias terms can be large and mislead inference on how much goes through the mediating channel.

Figure: Simulated Distribution of CM Effect Estimates from 10,000 DGPs.

(a) $\widehat{\text{ADE}} - \text{ADE}$.



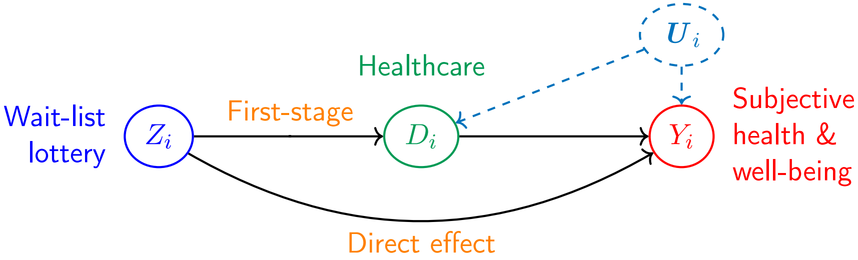
(b) $\widehat{\text{AIE}} - \text{AIE}$.



CM with Selection

Conventional CM methods do not identify ADE + AIE in settings, so I build a structural model for natural experiment settings.

Take as given that Z_i is quasi-randomly assigned, but D_i is not:



1. Average first-stage, $Z_i \to D_i$, is identified
2. Average second-stage, $Z_i, D_i \to Y_i$, is not — represented by $\boldsymbol{U}_i$.

Intuition: model $\boldsymbol{U}_i$ via mediator MTE to identify ADE + AIE.

CM with Selection

Conventional CM methods do not identify ADE + AIE in settings, so I build a structural model for natural experiment settings.

Write potential outcomes as mean + unobserved, in choosing to visit healthcare or not, $D_i = 0, 1$:

$$Y_i(z', 0) = \mathbb{E}\left[Y_i(z', 0) \,\middle|\, \boldsymbol{X}_i\right] + U_{0,i}, \quad Y_i(z', 1) = \mathbb{E}\left[Y_i(z', 1) \,\middle|\, \boldsymbol{X}_i\right] + U_{1,i}.$$

CM has two-stage regression equations:

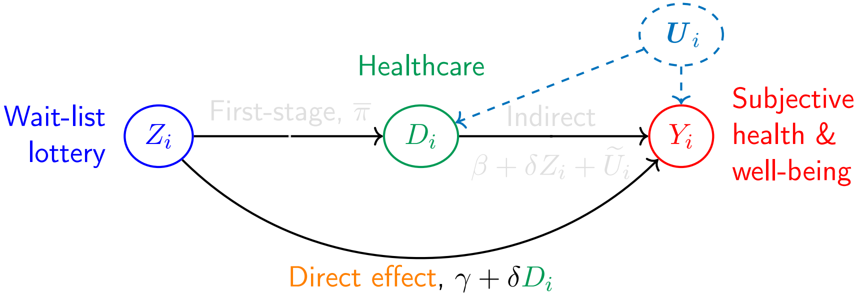
$$D_i = \phi + \overline{\pi} Z_i + \varphi(\boldsymbol{X}_i) + V_i$$

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

1. $\overline{\pi}$ is average first-stage, effect $Z_i \to D_i$
2. β, γ, δ are separated effects of Z_i, D_i.

CM with Selection

Conventional CM methods do not identify ADE + AIE in settings, so I build a structural model for natural experiment settings.



ADE composes effects of Z_i, holding D_i constant:

$$\text{ADE} = \mathbb{E}\left[\gamma + \delta D_i\right].$$

CM with Selection

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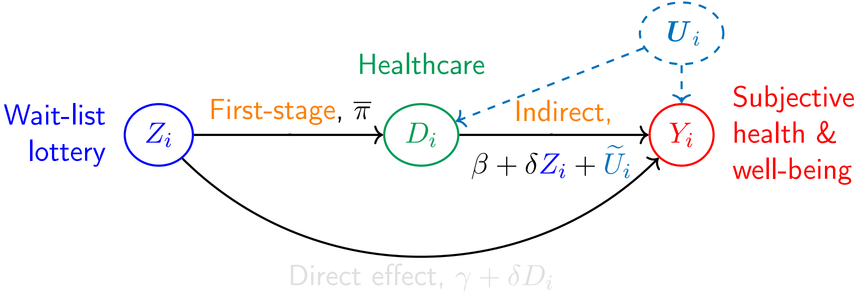


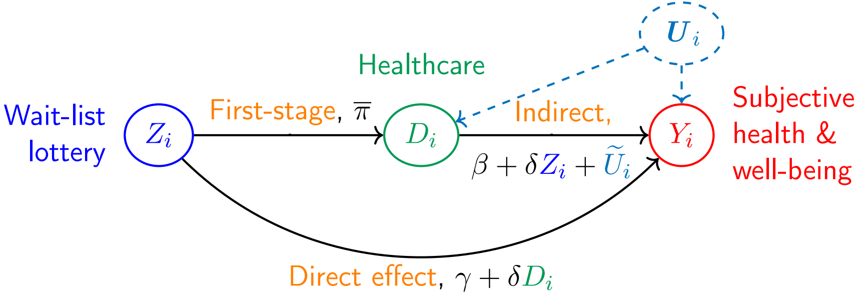
AIE composes effects of D_i, holding Z_i constant:

$$\text{AIE} = \mathbb{E}\left[\overline{\pi}\left(\beta + \delta Z_i + \widetilde{U}_i\right)\right],$$

where $\widetilde{U}_i = \mathbb{E}\left[U_{1,i} - U_{0,i} \mid \boldsymbol{X}_i, D_i(0) \neq D_i(1)\right]$ unobserved complier gains.

CM with Selection

Conventional CM methods do not identify ADE + AIE in settings, so I build a structural model for natural experiment settings.

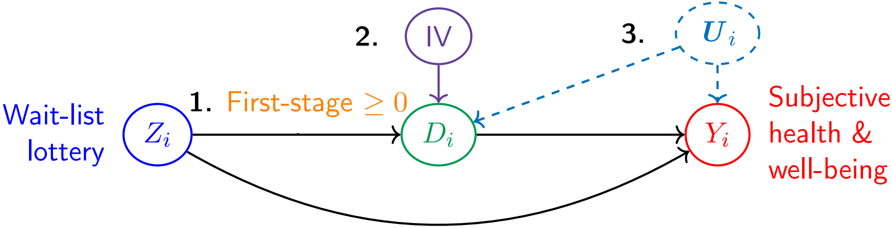


Structural model must solve the following issues:

1. β, γ, δ are not identified (see: selection bias)
2. $\widetilde{U}_i$ is also not known (unobserved complier healthcare gains).

MTE Model

The structural model is based on 3 assumptions.



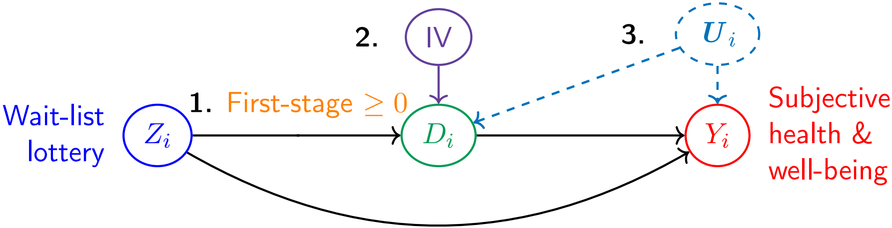
1. First-stage monotonicity,

$$\Pr\left(D_i(0) \leq D_i(1)\right) = 1.$$

Intuition: No defiers — no one visits healthcare less if winning wait-list lottery, relative to losing.

MTE Model

The structural model is based on 3 assumptions.



2. Mediator take-up cost IV

Requires an IV, which affects Y_i only via D_i.

Key example: Cost-shifting IV — random variation in healthcare take-up (not gains), e.g. type of healthcare providers charge different prices.

MTE Model

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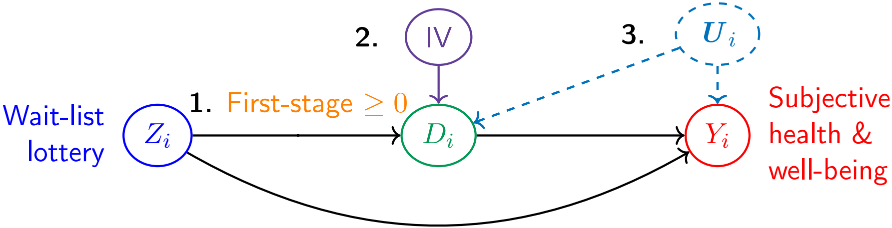

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Key example: Cost-shifting IV — random variation in healthcare take-up (not gains), e.g. type of healthcare providers charge different prices.

Conclusion

Explain in the applied example — build in the motivation of the health location cost IV.

Note why this fits so conceptually well for CM, connecting selection in the first-stage to causal effects in the second-stage.