

Frontier Topics in Empirical Economics: Chapter 1

Outline of Causal Inference

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- “Frontier Topics in Empirical Economics” by Zibin Huang
- Assembled from previous slides and notes
- We will cover one chapter in each week
 - Main content (must read)
 - Application paper (read original paper + ref report)
 - Homework (mandatory + selective)
 - Practice problem (selective)

Plan of This Course

- Basic causal inference and statistical tools (Chapter 1-4)
Potential outcome framework, RCT, matching vs regression, non-parametric method, machine learning, DAG framework
- IV (Chapter 5-7)
IV, LATE, GMM, MTE, Bartik IV
- Panel data and RDD (Chapter 8-9)
DID and event study, pre-trend testing, synthetic control, DDD, staggered DID, RDD, RKD
- Other topics (Chapter 10-14)
Cluster standard error, intro to discrete choice model, peer effect and spillover
- Student Presentation (TBD)

Plan of This Course

- The goal of this course is to get all students to stop being reg monkeys
- What is regression monkey? $\Rightarrow$ Run regs without creativity
 - Running regressions without knowing why
 - Only know very basic statistical off-the-shelf methods
 - Have no economic sense, do not know any economic theory
- This is no economist, this is a BAD statistician!
- This course aims to teach you
 - The logic behind regression and causal inference
 - Statistical tools beyond regression in causal inference
 - How to regularize data with your economic theory and intuition

Motivating Example: Female Labor Participation

- Consider a female labor participation problem
- You would run a regression:

$$y_i = \beta_0 + \beta_1 w_i + u_i \quad (1)$$

- y_i is labor participation dummy, w_i is wage
- This is what we call "reduced-form" method
- But where does it come from? Why is it called "reduced-form"?
- Let's start from the very beginning: a behavioral model

Motivating Example: Female Labor Participation

This is an example from Professor Chao Fu.

- Utility maximization of female i :

$$\begin{aligned} \max_{l_i} \quad & U_i(c_i, 1 - l_i) + \epsilon_{il} \\ \text{s.t.} \quad & c_i = w_i l_i \end{aligned} \tag{2}$$

c_i : consumption; l_i : labor supply; ϵ_{il} : unobserved taste shock; w_i : wage

Motivating Example: Female Labor Participation

- Assume that l_i is binary (work, not work)
- $l_i = 1$ if $U(l = 1) \geq U(l = 0)$:

$$U_i(w_i, 0) + \epsilon_{i1} \geq U_i(0, 1) + \epsilon_{i0} \quad (3)$$

- Then given w_i , we have a threshold value of $\epsilon_{i0} - \epsilon_{i1}$ for i to choose to work:

$$\begin{aligned} l_i &= 1 \quad \text{if} \quad \epsilon_{i0} - \epsilon_{i1} \leq \epsilon^* \\ \epsilon^* &= U_i(w_i, 0) - U_i(0, 1) \end{aligned} \quad (4)$$

Motivating Example: Female Labor Participation

- Assume that shock $\epsilon_{i0} - \epsilon_{i1}$ has a CDF F_ϵ , taken to be independent of w_i
- We have the following working probability for i :

$$\begin{aligned} G(w) &= \Pr(I = 1|w) = \int_{-\infty}^{\epsilon^*} dF_\epsilon \\ &= F_\epsilon(\epsilon^*(w)) \end{aligned} \tag{5}$$

- Two empirical research approaches for this question

Motivating Example: Female Labor Participation

1. We can directly estimate probability function G with linearity assumption

- Assume that G is a linear function

$$G(w) \approx \beta_0 + \beta_1 w_i \quad (6)$$

- Linear Probability Model $\Rightarrow$ We can use OLS to estimate β
- This is called "Reduced-form" approach
- It is reduced from the underlying behavioral model
- We usually identify it by some research "design" (IV, RDD, DID)
- Thus, it is also called "Design-based" approach

Motivating Example: Female Labor Participation

2. We can estimate ϵ 's CDF F , and utility function U

- We have the likelihood function as:

$$L(\Theta^U, \Theta^F; data) = \prod_{i=1}^N F_{\epsilon}(\epsilon^*)^{l_i} [1 - F_{\epsilon}(\epsilon^*)]^{1-l_i} \quad (7)$$

Θ^U is the parameter set of utility function; Θ^F is the parameter set of shock's CDF

- We use MLE to estimate Θ^U and $\Theta^F \Rightarrow$ Recover choice structure directly
- This is called "Structural" / "Model-based" approach

Motivating Example: Female Labor Participation

For example,

- Assume a linear utility function $U_i(w_i l_i, 1 - l_i) = \alpha w_i l_i + \phi(1 - l_i)$
- And $\epsilon_{i0}, \epsilon_{i1}$ are i.i.d. T1EV, so their difference $\epsilon_{i0} - \epsilon_{i1}$ follows a logistic distribution
- We have the likelihood function as:

$$\begin{aligned} L(\Theta^U, \Theta^F; data) &= \prod_{i=1}^N F_{\epsilon}(\epsilon^*)^{l_i} [1 - F_{\epsilon}(\epsilon^*)]^{1-l_i} \\ &= \prod_{i=1}^N \left(\frac{\exp(\alpha w_i)}{\exp(\alpha w_i) + \exp(\phi)} \right)^{l_i} \times \left(\frac{\exp(\phi)}{\exp(\alpha w_i) + \exp(\phi)} \right)^{1-l_i} \end{aligned} \quad (8)$$

- We will show more details in Chapter 12

Two Approaches: Internal vs External Validity

- Now we compare these two approaches
- First, we need to clarify two important concepts
 - Internal validity
 - External validity
- Internal means the validity within the current specific context or environment
- External means the validity outside the current context or environment
- External refers to our attempt to extrapolate our analysis

Two Approaches: Internal vs External Validity

- There are three layers of policy evaluation (Heckman and Vytlacil, 2007)
- Take One Child Policy (OCP) as an example
 - Evaluating the impact of a **historical intervention**
What was the impact of the OCP on fertility rate?
 - Forecasting the impact of an intervention previously happened in environment A to happen **in another environment B**
What would be the impact if we restart the OCP in 2023?
 - Forecasting the impact of an intervention **never happened in history** in any environment
What would be the impact if we force all women to give birth to at least one child?
- The first one is "internal"
- The second and the third are "external"

Two Approaches: Structural/Model-based Approach

- Target: Primitive parameters $\Rightarrow$ Choice structure
Agent's utility function, firm's production function, market structure...
- Advantages
 - Deeper economic thinking: we can understand the original decision-making process
 - Great external validity $\Rightarrow$ Solid under Lucas' critique (Lucas, 1976)
 - More reliable counterfactual analysis
- Disadvantages
 - Need more (untestable) assumptions on functional form, distribution of unobservable...
 - Low internal validity

Two Approaches: Reduced-form/Design-based Approach

- Target: Some marginal effect of conditional expectation function
What is the impact of A on B?
- Do not care about the mechanism $\Rightarrow$ A black box of causal effect
- Advantages
 - Very reliable if you have a good exogenous shock
 - Great internal validity, not so many assumptions
- Disadvantages
 - No mechanism is revealed $\Rightarrow$ More of a statistician than an economist
 - Usually effects are very local $\Rightarrow$ Low external validity
The causal effect is estimated for group A. Can it be applied to group B?
 - Hard to have external counterfactual interpretation
Lucas' critique, General Equilibrium effect...

Two Approaches: Deduction vs Induction

- The two approaches reflect two fundamental modes of scientific inquiry
- Structural approach $\Rightarrow$ Deduction
 - Incorporate economic theory and prior knowledge into a model
 - Derive implications from general principles to specific cases
- Reduced-form approach $\Rightarrow$ Induction
 - Rely primarily on observed data, with minimal theoretical structure
 - "Letting the data speak"
- Neither is superior; they complement each other
- Recent trend: Combining the two
 - Construct a structural model, then estimate its key parameters using design-based methods (RCT, DID...)

Two Approaches: Reduced-form/Design-based Approach

- This course will mainly focus on the Reduced-form/Design-based Approach
- Specifically, I will carefully go through traditional regression tools used in Applied Economics
- And introduce tools beyond simple regression
- I will also introduce a little Structural/Model-based Approach (DCM)
- In general, let's try not to be Reg Monkeys!

Potential Outcome Framework and RCT

- Example : Health status and hospitalization

Group	Sample Size	Mean Health Status
Hospital	7,774	3.21
No hospital	90,049	3.93

- Going to hospital makes you more sick?
- No! People go to hospital because they are sick.
- Correlation is NOT causality!!!

Potential Outcome Framework and RCT



Potential Outcome Framework and RCT

Potential Outcome Framework/Rubin Causal Model (Rubin, 1974)

- Binary treatment D_i for individual i , some outcome Y_i
- Y_{0i} : The "potential outcome" of i if he/she is *not treated*, regardless of the treatment status in reality
- Y_{1i} : The "potential outcome" of i if he/she is *treated*, regardless of the treatment status in reality
- Thus, we have:

$$\begin{aligned} Y_i &= \begin{cases} Y_{1i} & \text{if } D_i = 1 \\ Y_{0i} & \text{if } D_i = 0 \end{cases} \\ &= Y_{0i} + (Y_{1i} - Y_{0i})D_i \end{aligned} \tag{9}$$

Potential Outcome Framework and RCT

Two average treatment effects are of primary interest

- Average Treatment Effect (ATE):

$$ATE = E[Y_{1i} - Y_{0i}]$$

Expected treatment effect for a randomly drawn individual from the population

- Average Treatment Effect on the Treated (ATT):

$$ATT = E[Y_{1i} - Y_{0i} | D_i = 1]$$

Expected treatment effect for those who actually receive the treatment

- ATE and ATT generally differ when treatment is not random

Potential Outcome Framework and RCT

- Individual treatment effect: $Y_{1i} - Y_{0i}$
- Not available: There is only one world! Given i , you see either Y_{0i} or Y_{1i}
- But we can consider averages: By differencing mean outcomes from the two groups

$$\begin{aligned} & E[Y_i | D_i = 1] - E[Y_i | D_i = 0] \\ &= \underbrace{E[Y_{1i} | D_i = 1] - E[Y_{0i} | D_i = 1]}_{\text{Average Treatment on the Treated (ATT)}} + \underbrace{E[Y_{0i} | D_i = 1] - E[Y_{0i} | D_i = 0]}_{\text{Selection bias}} \end{aligned} \tag{10}$$

- ATT: Causal effect on the treated group
- Selection bias: Original difference between treated and untreated group
- Give me an example of the selection bias

Potential Outcome Framework and RCT

Randomization can solve the selection problem

- Assume that we randomly assign the treatment to the population:

$$D_i \perp\!\!\!\perp Y_{0i}, Y_{1i} \quad (11)$$

- Then we have selection bias to be zero:

$$E[Y_{0i}|D_i = 1] - E[Y_{0i}|D_i = 0] = 0$$

- Thus, simple difference between the mean of treated and untreated group is ATT (and overall ATE)

$$E[Y_i|D_i = 1] - E[Y_i|D_i = 0] = E[Y_{1i}|D_i = 1] - E[Y_{0i}|D_i = 1] = ATT = ATE$$

Regression, CEF and Causal Inference

- Regression is the most useful tool in applied econometrics
- When can we interpret regression coefficient as causal effect?
- What are the relations among regression, conditional expectation function (CEF) and treatment effect?

Regression, CEF and Causal Inference

Conditional Expectation Function (CEF)

- CEF is the conditional expectation of an outcome Y_i , given some predictor vector X_i

$$E[Y_i | X_i = x] = \int t f_y(t | X_i = x) dt \quad (12)$$

where f_y is pdf

- This is a **population** concept ($n \rightarrow \infty$)
- It describes a prediction of X on Y , but **NOT necessarily causal**
- We can always decompose Y_i as predicted part (CEF) + error part

$$Y_i = E[Y_i | X_i] + \epsilon_i \quad (13)$$

where $E[\epsilon_i | X_i] = 0$ (conditional mean zero)

Regression, CEF and Causal Inference

- CEF is the best predictor of Y_i given X_i
- It minimizes the mean squared prediction errors

Theorem 1.5.1: CEF Minimizes MSE

Let $m(X_i)$ be any function of X_i . The CEF solves

$$E[Y_i|X_i] = \arg \min_{m(X_i)} E[(Y_i - m(X_i))^2]$$

so it is the MMSE predictor of Y_i given X_i .

Regression, CEF and Causal Inference

Linear Regression

- Regression is a **linear prediction** that minimizes the mean squared error

$$Y_i = X_i' \beta + \epsilon_i$$

$$\beta = \arg \min_b E[(Y_i - X_i' b)^2]$$

- We have the first order condition (moment condition) as:

$$E[X_i \underbrace{(Y_i - X_i' \beta)}_{\epsilon_i}] = 0$$

- The solution can be written as:

$$\beta = E[X_i X_i']^{-1} E[X_i Y_i]$$

Regression, CEF and Causal Inference

Tips: Difference between β and $\hat{\beta}_{OLS}$

- Definition

$$\beta = E[X_i X_i']^{-1} E[X_i Y_i]$$

$$\hat{\beta}_{OLS} = (X'X)^{-1} X'Y$$

- $\hat{\beta}_{OLS}$ is an estimator of β (there can be alternative estimators, e.g. MLE)
- Population vs Sample, Identification vs Estimation
- X_i is an $1 \times k$ vector, Y_i is a scalar. They are random variables
- X is an $n \times k$ matrix, Y is an $n \times 1$ vector. They are realizations of random variables (real data)

Regression, CEF and Causal Inference

CEF and linear regression

- $E[\epsilon_i | X_i] = 0$ vs $E[X_i \epsilon_i] = 0$
- Minimizing MMSE: Best predictor (CEF) vs Best linear predictor (linear regression)
- CEF is stronger than linear regression
- If CEF is linear, then linear regression is identical to CEF
- Even if CEF is not linear, regression is the best linear approximation to CEF (Minimize MSE)

Regression, CEF and Causal Inference

- For any data, you can always run a regression (as long as the rank condition is satisfied)
- But the coefficient β is not necessarily a causal effect
- When does a regression coefficient have a causal meaning?

Regression, CEF and Causal Inference

Case 1: We assume randomization (no need for controls) and constant TE

- When we have a random experiment with $D_i \perp\!\!\!\perp Y_{0i}, Y_{1i}$ and regression

$$Y_i = \alpha + \rho D_i + \epsilon_i$$

- Under this randomization, with binary D_i , the CEF is automatically linear in D_i , then:

$$\rho = E[Y_i|D_i = 1] - E[Y_i|D_i = 0] = E[Y_{1i} - Y_{0i}|D_i = 1]$$

- Regression coefficient ρ is the ATT/ATE

Regression, CEF and Causal Inference

Case 2: We assume randomization after controls

- Key to go from correlation/prediction to causality: **Conditional Independent Assumption (CIA)/Selection on Observables**

$$D_i \perp\!\!\!\perp Y_{0i}, Y_{1i} | X_i$$

- Treatment is random, after controlling for covariates X_i

Regression, CEF and Causal Inference

Case 2: We assume randomization after controls

- Homogeneous (constant) treatment effect case is simple
- For each $X_i = x$, we have the following regression:

$$Y_i = \alpha + \rho_r D_i + X_i' \gamma + \nu_i \quad (14)$$

- With linear CEF, regression coefficient ρ_r is the treatment effect

$$\rho_r = E[Y_i | X_i, D_i = 1] - E[Y_i | X_i, D_i = 0] = E[Y_{1i} - Y_{0i}]$$

Regression, CEF and Causal Inference

Case 2: We assume randomization after controls

- Heterogeneous treatment effect case is more complicated
- Let δ_x be the within group ATE/ATT:

$$\delta_x = E[Y_i|X_i, D_i = 1] - E[Y_i|X_i, D_i = 0] = E[Y_{1i}|X_i, D_i = 1] - E[Y_{0i}|X_i, D_i = 1]$$

- It can be shown that ρ_r is the treatment-variance weighted average of δ_x :

$$\rho_r = \frac{E[\sigma_D^2(X_i)\delta_x]}{E[\sigma_D^2(X_i)]} \quad (15)$$

$$\sigma_D^2(X_i) \equiv E[(D_i - E[D_i|X_i])^2|X_i]$$

- Proof: see Chapter 3.3.1 in Angrist and Pischke (2009)

Regression, CEF and Causal Inference

- Important! How to understand/interpret equation (15)?
- More weights are assigned to cells with largest treatment variance
- Zero weight if a cell is full of treated/untreated individuals

Regression, CEF and Causal Inference

- Homework: What is the implication of expression (15) when unconditional independence holds (Like in an RCT)? That is, when $D \perp\!\!\!\perp Y_{1i}, Y_{0i}$?

Regression, CEF and Causal Inference

Let's compare assumptions of Regression, CEF and Causal Model

- $y = f(D) + e$
- Linear Regression: $f(D) = \beta D, E(De) = 0$ Uncorrelated
- CEF: $E(e|D) = 0$ Mean Independence
- Causal Model: $e \perp\!\!\!\perp D \quad (D_i \perp\!\!\!\perp y_{0i}, y_{1i})$ Independence
- Tips: A regression of Y on a single dummy D recovers the CEF exactly

Regression, CEF and Causal Inference

Main takeaways from this part

- Strength of assumptions regarding unobservable e
Causal model (CIA) > CEF (Mean Independence) > Linear regression (Uncorrelated)
- CEF is the best predictor of Y given X
- Linear regression is the best "linear" predictor of Y given X
- Linear regression is the best linear approximation of CEF
- Under CIA and homogeneous TE, regression coefficient is the TE
- Under CIA and heterogeneous TE, regression coefficient is the treatment-variance weighted average of group ATE

Simpson Paradox, Omitted Variables and Bad Controls

- Consider two treatments A and B for COVID
- We examine the effect of the treatments by patients' conditions (mild/severe)
- We have the death rate by treatments and conditions as:

	Mild	Severe	Total
A	15% (210/1400)	30% (30/100)	16% (240/1500)
B	10% (5/50)	20% (100/500)	19% (105/550)

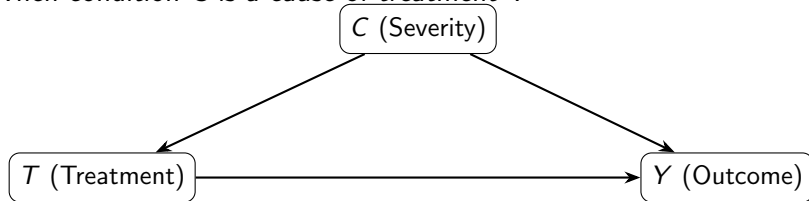
- Total death rate: $A < B$
- Death rate within condition group: $A > B$

Simpson Paradox, Omitted Variables and Bad Controls

- Which one is better? A or B? $\Leftrightarrow$ Should we control for condition (C)?
- It depends on the causal structure!
- Condition is the cause or the consequence of the treatment?

Simpson Paradox, Omitted Variables and Bad Controls

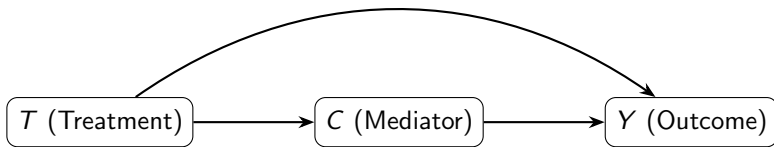
Case 1: When condition C is a cause of treatment T



- C causes T and Y; T causes Y
- C is a pre-determined variable to T
- We should control for C $\Rightarrow$ B is better
- If we do not control for C $\Rightarrow$ Omitted Variable Bias

Simpson Paradox, Omitted Variables and Bad Controls

Case 2: When treatment T is a cause of condition C



- T causes C and Y ; C causes Y
- C is a post-determined variable $\Rightarrow C$ is the consequence of T
- We should not control for $C \Rightarrow A$ is better
- If we do control for $C \Rightarrow$ Bad Control Problem
- **Never control a channel!!!**

Simpson Paradox, Omitted Variables and Bad Controls

- Rule of thumb: Control pre-determined variables, not post-determined ones
- But sometimes controlling for pre-determined variables can also be wrong
- Let's discuss this "bad control" issue in more details in Week 4
- DAG will offer you a clear and powerful tool to determine which variables to control, given your proposed causal structure

Simpson Paradox, Omitted Variables and Bad Controls

- Quiz: Should we control for X?
 - $Y=\text{wage}$, $D=\text{education}$, $X=\text{natural ability}$
 - $Y=\text{wage}$, $D=\text{education}$, $X=\text{labor participation decision}$
 - $Y=\text{GDP at } t+1$, $D=\text{R\&D expenditure at } t$, $X=\text{trade volume at } t+1$

Matching

- Regression is only one of the tools we use to tackle causal effect
- Matching is another common tool
- It is simple and non-parametric
- Basic idea
 - (1) Compare treated and control units with same covariates;
 - (2) Put together to produce a single overall weighted average treatment effect
- Regression is a particular sort of weighted matching estimator

Matching

- Assume that for treatment D_i , we have CIA: $Y_{0i}, Y_{1i} \perp\!\!\!\perp D_i | X_i$
- We can express ATT as:

$$\begin{aligned}\delta_{ATT} &= E[Y_{1i} - Y_{0i} | D_i = 1] = E[E[Y_{1i} - Y_{0i} | X_i, D_i = 1] | D_i = 1] \\ &= E[E[Y_{1i} | X_i, D_i = 1] - E[Y_{0i} | X_i, D_i = 1] | D_i = 1] \\ &= E[E[Y_i | X_i, D_i = 1] - E[Y_i | X_i, D_i = 0] | D_i = 1] \\ &= E[\delta_x | D_i = 1]\end{aligned}$$

- The corresponding matching estimator (sample analog) is:

$$\hat{\delta}_{ATT} = \sum_x \hat{\delta}_x \hat{P}(X_i = x | D_i = 1)$$

- Similarly, we can derive a matching estimator for ATE:

$$\hat{\delta}_{ATE} = \sum_x \hat{\delta}_x \hat{P}(X_i = x)$$

Matching vs Regression

Regression is one of the matching estimators!

- Matching estimator of ATT: $\hat{\delta}_{ATT} = \sum_x \hat{\delta}_x P(X_i = x | D_i = 1)$
Weighted by probability mass for treated group
- Matching estimator of ATE: $\hat{\delta}_{ATE} = \sum_x \hat{\delta}_x P(X_i = x)$
Weighted by probability mass for all units
- Regression estimator: $\frac{\sum_x \hat{\sigma}_D^2(x) \hat{\delta}_x \hat{P}(X_i = x)}{\sum_x \hat{\sigma}_D^2(x) \hat{P}(X_i = x)}$
Weighted by treatment variances

Matching vs Regression

- Explain the meaning of the weights in these three estimators. To which observation/cell are they going to give the largest weights?

Propensity Score Matching

- Assume that we want to estimate college premium on wages
- To have CIA, we need a lot of controls:
Gender, race, nationality, birth weight, IQ, parents' education, parents' income...
- Curse of dimensionality: There are too many dimensions in X_i
- We will not have enough observations for each value of X_i to estimate $\hat{\delta}_x$
- Maybe you have 10,000 observations
- But only 2 of them are Han boys with IQ 150, family income 100,000 RMB/year, parents are high-school educated
- Very hard to implement the matching estimator (but regression is still feasible)

Propensity Score Matching

- Propensity Score Matching (PSM) is a simple method to reduce the dimensionality
- **Assumption 1 (CIA):** $Y_{1i}, Y_{0i} \perp\!\!\!\perp D_i | X_i$
- Assumption 2 (Overlap): $0 < P(D_i = 1 | X_i) < 1$
- PSM Theorem (Rosenbaum and Rubin, 1983): If Assumptions 1 and 2 hold, we have $Y_{1i}, Y_{0i} \perp\!\!\!\perp D_i | P(X_i)$, where $P(X_i) = P(D_i = 1 | X_i)$
- We are fine, as long as we control for the propensity score $P(X)$

Propensity Score Matching

- Go back to the college premium example
- Instead of matching across all controls (gender, family income...)
- We can match for the predicted probability $P(X)$ for each person to go to college
- We just replace all X_i with $P(X_i)$ in the matching estimator, and get the PSM estimator.

Regression vs PSM

- Regression usually does not suffer from the curse of dimensionality
- Since we are regularizing controls by linear function (next class)
- We can also combine regression and PSM by running a regression, controlling for propensity score (but not each variable)

Regression vs PSM

- In general, Angrist prefers regression (Angrist and Pischke, 2009)
- Because some parts of the process to implement PSM are not standardized
- e.g. how to estimate the propensity score $P(X)$? (Logit? LPM? Probit?)
- PSM CANNOT solve the endogeneity issue!!!!!!
- PSM CANNOT solve the endogeneity issue!!!!!!
- PSM CANNOT solve the endogeneity issue!!!!!!

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