

Rubin's Causal Model

Endogeneity and Exogeneity

Tadao Hoshino (星野匡郎)

ver. 2018 Fall Semester

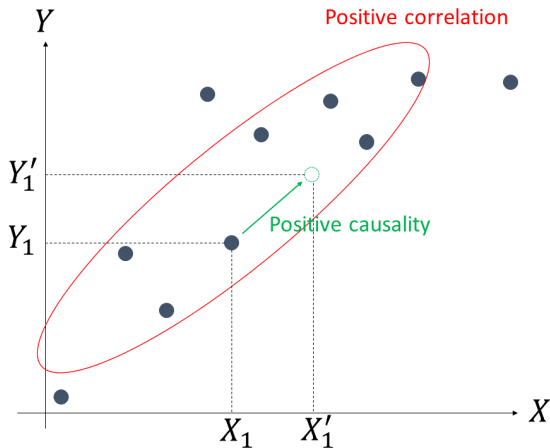
Introduction: Correlation and Causality

- For variables X and Y , we say they are **correlated** when
the two variables vary together
(Y tends to change as X changes)

Correlation = overall tendency of the observed data

- On the other hand, when
a change in X causes a change in Y
(one can change X in order to change Y)
we say there is a **causal relationship** from X to Y .

Introduction: Correlation and Causality



- For example, consider a case where X : weight and Y : height.
- Clearly, X and Y are correlated; but there is no causality from X to Y .

Introduction: Correlation and Causality

- In general, causality implies correlation, however correlation does not imply causality.
- We are often interested in quantifying the causality between variables, rather than a mere correlation.

Does raising minimum wage increase/decrease unemployment rate?

Even if we can observe a statistical correlation

“the unemp rate tends to be smaller in regions with lower mini wages”

this does *not* imply the causality

“the unemp rate can be reduced by lowering the mini wage”

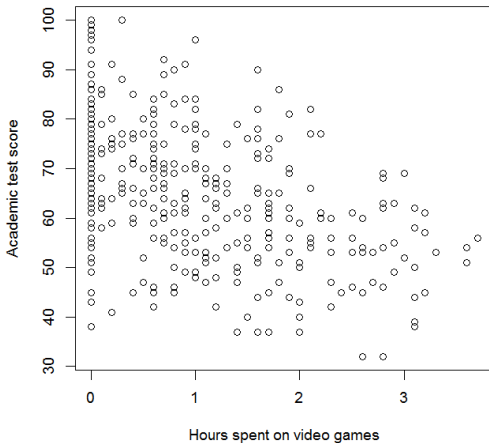
- The above example highlights the importance of distinguishing between correlation and causality for policy analysis.

=> **Statistical Causal Inference**

Correlation \neq Causality

Spurious Correlation

Playing video games and academic performance¹



¹This is simulated data created by myself.

Spurious Correlation

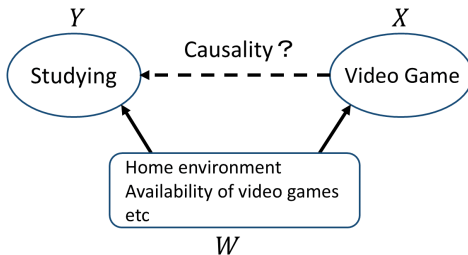
- We observe an overall negative correlation between video game and academic performance (correlation coef. : -0.49).
- From this result, we *cannot* conclude that “playing video games has a negative influence on student’s academic performance”.
- Regardless of whether playing video games negatively or even positively affects the academic performance, a negative correlation can exist between them. Why?

Spurious Correlation

A **spurious correlation** occurs when two variables (X and Y) that actually have no logical connection can be correlated due to the presence of an unobserved variable (W) that affects both X and Y .

Spurious Correlation

- Unobserved variables (W) that can affect both playing video games (X) and studying (Y):
 - home environment (e.g., both parents work late)
 - availability of video games in the child's own bedroom
 - etc
- In order to improve Y , improving the W 's is much more important than merely decreasing X .

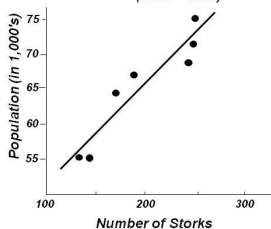


Spurious Correlation

Examples of spurious correlation:

- *Storks bring babies.*²

Population of Oldenburg, Germany, at Year's End
vs. Number of Storks Observed Each Year
(1930 - 1936)



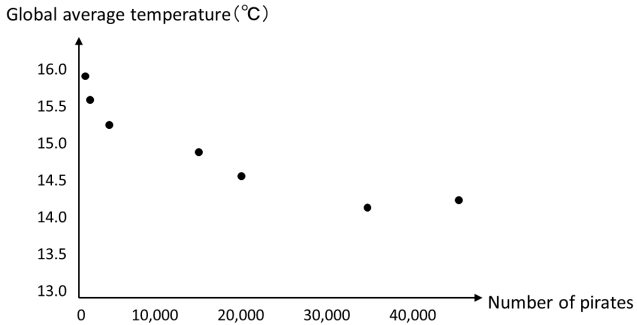
Source: Statistics for Experimenters,
by Box, Hunter & Hunter

- Consumption of beers and the number of water accidents.
- etc

²storks = コウノトリ

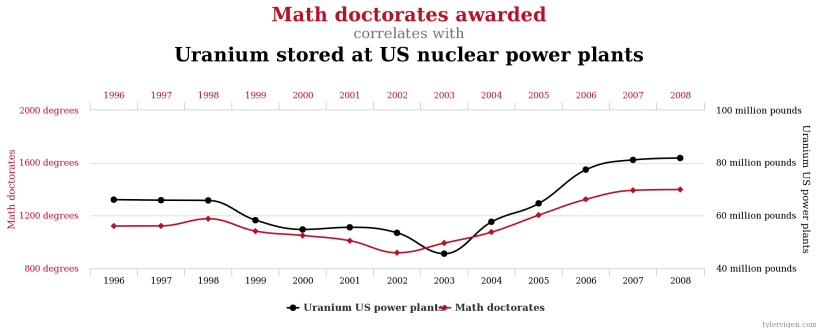
Correlation just by chance

Number of pirates and global warming



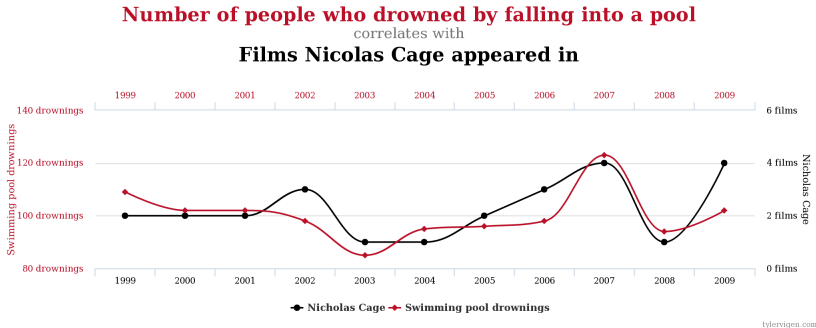
(ref.) <http://sparrowism.soc.srcf.net/home/pirates.html>

Correlation just by chance



(ref.) <http://www.tylervigen.com/spurious-correlations>

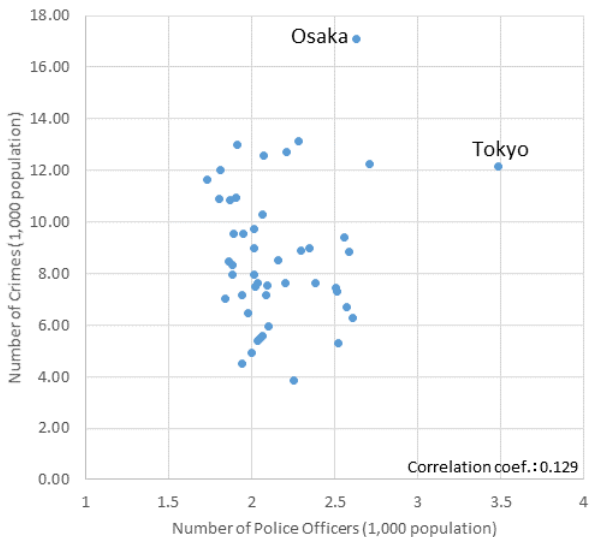
Correlation just by chance



(ref.) <http://www.tylervigen.com/spurious-correlations>

Simultaneity

Number of police officers and crimes per 1,000 population.



Simultaneity

- It is normally believed that increasing police reduces crimes.
=> We expect a negative causal relationship between police and crime.
- However, the observed correlation has the opposite sign to the expected causal effect. This phenomenon can be explained by the **simultaneity** of police levels and crime.

Simultaneity

For variables X and Y , **simultaneity** means that X is a cause of Y and at the same time Y is a cause of X .

- If the arrest rates are about the same across regions, this would generate a tendency “police \uparrow - crime \uparrow ” and “police \downarrow - crime \downarrow ”, which results in positive correlation between police and crime.

Linear Regression = Correlation Analysis \nRightarrow Causality

Correlation Coefficient

Correlation Coefficient

Let $\{(X_1, Y_1), \dots, (X_n, Y_n)\}$ be the sample data of (X, Y) of size n . The (sample) **correlation coefficient** r_{XY} is defined as

$$r_{XY} = \frac{\sum_{i=1}^n (X_i - \bar{X}_n)(Y_i - \bar{Y}_n)}{\sqrt{\sum_{i=1}^n (X_i - \bar{X}_n)^2} \sqrt{\sum_{i=1}^n (Y_i - \bar{Y}_n)^2}}$$

where \bar{X}_n and \bar{Y}_n are the sample averages of X and Y , respectively.

The correlation coefficient takes a value ranging from -1 to 1 :

- $r_{XY} > 0$ positive correlation ($r_{XY} = 1$: perfect positive correlation)
- $r_{XY} = 0$ no correlation
- $r_{XY} < 0$ negative correlation ($r_{XY} = -1$: perfect negative correlation)

Linear Regression = Correlation Analysis

- Consider a simple linear regression model:

$$Y_i = \beta_0 + X_i\beta_1 + \varepsilon_i, \quad i = 1, \dots, n$$

- Recall that the OLS estimator of β_1 is given by

$$\hat{\beta}_{n1} = \frac{\sum_{i=1}^n (X_i - \bar{X}_n)(Y_i - \bar{Y}_n)}{\sum_{i=1}^n (X_i - \bar{X}_n)^2}$$

Linear Regression = Correlation Analysis

- Observe that

$$r_{XY} = \frac{\sum_{i=1}^n (X_i - \bar{X}_n)(Y_i - \bar{Y}_n)}{\sqrt{\sum_{i=1}^n (X_i - \bar{X}_n)^2} \sqrt{\sum_{i=1}^n (Y_i - \bar{Y}_n)^2}}$$

multiplying both the num and denom by $\sqrt{\sum_{i=1}^n (X_i - \bar{X}_n)^2}$

$$= \frac{\sum_{i=1}^n (X_i - \bar{X}_n)(Y_i - \bar{Y}_n)}{\sum_{i=1}^n (X_i - \bar{X}_n)^2} \cdot \frac{\sqrt{\sum_{i=1}^n (X_i - \bar{X}_n)^2}}{\sqrt{\sum_{i=1}^n (Y_i - \bar{Y}_n)^2}}$$

$$= \hat{\beta}_{n1} \cdot \underbrace{\frac{\sqrt{\sum_{i=1}^n (X_i - \bar{X}_n)^2}}{\sqrt{\sum_{i=1}^n (Y_i - \bar{Y}_n)^2}}}_{\text{positive constant}}$$

- Therefore, the regression coefficient $\hat{\beta}_{n1}$ and the correlation coefficient r_{XY} are proportionally related, i.e., $\hat{\beta}_{n1} = 0 \iff r_{XY} = 0$.

Linear Regression = Correlation Analysis

- As stated above, correlation does not imply causation. Therefore, for a regression model

$$Y_i = \beta_0 + X_i\beta_1 + \varepsilon_i, \quad i = 1, \dots, n$$

when β_1 is estimated to be significantly positive, although we can say that

“ Y tends to increase as X increases” – correlation,

we cannot say that

“an increase in X causes an increase in Y ” – causality

in general.³

³As described later, if X has a property called *exogeneity*, the estimated β_1 can be interpreted as the causal effect of X on Y .

Rubin's Causal Model

In order to statistically deal with “causality”, first of all, there is a question:

How can we mathematically define the causal relationship btwn variables?

In the literature, there are several different approaches:

- Rubin's Causal Model (D. Rubin)
- Structural Causal Models (J. Pearl)
- etc.

- Among them, the Rubin's Causal Model (RCM) is the most commonly used framework in econometrics.
- We consider causal relationship between two variables (Y, T) .
 - Y is an outcome variable of interest, and T is a variable that potentially affects the value of Y .
 - T is called the **treatment** variable.
- In the following, we focus on the case where the treatment T is a dummy variable, i.e., $T \in \{0, 1\}$.

Rubin's Causal Model

Examples of (Y, T) :

Y	T	Causal effect of interest
Wage	Completion of college degree yes: $T = 1$, no: $T = 0$	Wage returns to a college degree
Academic grade	Participation in remedial courses yes: $T = 1$, no: $T = 0$	Effect of remedial education on academic performance
Emission of pollutants	Introduction of emission ctrl tech yes: $T = 1$, no: $T = 0$	Effect of the control technology on pollutants

Rubin's Causal Model

- For each individual i , suppose that we observe (Y_i, T_i) .
- Each individual has two **potential outcomes**, Y_1 and Y_0 , one for each value of the treatment T :
 - Y_{1i} = the value of Y_i when $T_i = 1$ is true,
 - Y_{0i} = the value of Y_i when $T_i = 0$ is true.
- Note that it is *impossible* to observe an individual under two different treatment statuses (T_i must be either one or zero).
- That is, when $T_i = 1$ is realized, we can observe an outcome $Y_i = Y_{1i}$, but Y_{0i} is an unobservable potential outcome (the so-called **counterfactual outcome**.)

Treatment status	Observed outcome	Counterfactual outcome
$T_i = 1$	$Y_i = Y_{1i}$	Y_{0i}
$T_i = 0$	$Y_i = Y_{0i}$	Y_{1i}

Rubin's Causal Model

- Thus, the observed outcome Y_i can be expressed as

$$Y_i = T_i \cdot Y_{1i} + (1 - T_i) \cdot Y_{0i}$$

- In this framework, the causal effect of T on Y for individual i is defined as

$$Y_{1i} - Y_{0i}.$$

This quantity is referred to as the (individual) **treatment effect** for i .

- Namely, the causal effect (i.e., treatment effect) is defined as the difference between

the observed outcome when $T_i = 1$ (0) is actually selected
and
the counterfactual outcome when $T_i = 0$ (1) is hypothetically selected.

Rubin's Causal Model

- The treatment effect may be heterogeneous among individuals.
 - It is possible that the treatment is beneficial for some people but not for others.
- Unless there is someone completely identical to i , we cannot observe both potential outcomes Y_{0i} and Y_{1i} at the same time, and thus the individual treatment effect cannot be directly measured.
- However, it is still possible to estimate the treatment effect averaged over some subpopulation.

Average Treatment Effect (ATE)

Average Treatment Effect (ATE)

- It is impossible to directly estimate the individual treatment effects from data.
- Let us focus on, not individual specific treatment effect, but the treatment effect averaged over the whole population, the so-called **average treatment effect (ATE)**:

$$E[Y_{1i}] - E[Y_{0i}].$$

- When a sample of n observations $\{(Y_i, T_i) : i = 1, \dots, n\}$ is available, how can we estimate the ATE?

Average Treatment Effect (ATE)

- **Treatment group** $\{i : T_i = 1\}$: individuals who actually receive the treatment.
- **Control group** $\{i : T_i = 0\}$: individuals who do not receive the treatment.
 - The sample size of the treatment group and that of the control group are $\sum_{i=1}^n T_i$ and $\sum_{i=1}^n (1 - T_i)$, respectively.
- A naive estimator of ATE: $E[Y_{1i}] - E[Y_{0i}]$ is just the difference between the average outcome of the treatment group and that of the control group:

$$\underbrace{\frac{\sum_{i=1}^n T_i Y_i}{\sum_{i=1}^n T_i}}_{\text{average } Y \text{ in TG}} - \underbrace{\frac{\sum_{i=1}^n (1 - T_i) Y_i}{\sum_{i=1}^n (1 - T_i)}}_{\text{average } Y \text{ in CG}} \quad (1)$$

Average Treatment Effect (ATE)

- In fact, (1) is numerically equivalent to the OLS slope estimator $\hat{\beta}_{n1}$ of the following simple linear regression model:

$$Y_i = \beta_0 + T_i\beta_1 + \varepsilon_i, \quad i = 1, \dots, n$$

that is,

$$\frac{\sum_{i=1}^n T_i Y_i}{\sum_{i=1}^n T_i} - \frac{\sum_{i=1}^n (1 - T_i) Y_i}{\sum_{i=1}^n (1 - T_i)} = \frac{\sum_{i=1}^n (T_i - \bar{T}_n)(Y_i - \bar{Y}_n)}{\sum_{i=1}^n (T_i - \bar{T}_n)^2}$$

holds. (This equality can be confirmed by direct calculation.)

- Thus, the simple difference estimator (1) only shows the correlation of T and Y , and therefore, it may not necessarily reflect the true causal effect of T on Y .

Summary of the Discussion So Far

- Correlation does not imply causality due to the presence of, e.g., spurious correlation and simultaneity.
- The linear least squares estimator of the regression coefficients is proportional to the correlation coefficient.
=> Linear regression is a correlational analysis.
- In the RCM framework, the causal effect of T on Y averaged over the population is called the average treatment effect (ATE):

$$\text{ATE: } E[Y_{1i} - Y_{0i}].$$

Summary of the Discussion So Far (cont')

- At first glance, it seems that the ATE can be estimated by the difference between the average outcome of the treatment group and that of the control group.
- However, since such simple difference estimator is equivalent to the OLS estimator of β_1 in a regression model

$$Y_i = \beta_0 + T_i\beta_1 + \varepsilon_i,$$

the resulting estimate only reflects the correlation between T and Y .

Q. Under what conditions does the simple difference estimator correctly recover the ATE?

A. A sufficient condition is that the treatment variable T has a property called “exogeneity” with respect to the outcome variable Y .

Endogeneity and Exogeneity

Endogeneity and Exogeneity

- Consider a simple regression model:

$$Y_i = \beta_0 + X_i\beta_1 + \varepsilon_i, \quad i = 1, \dots, n,$$

where $E[\varepsilon_i] = 0$. The causal impact of a unit increase in X on Y is β_1 .

- The variable X is said to be **exogenous** in the model if it is uncorrelated with the error term ε .
- That is, when

$$\underbrace{Cov(X_i, \varepsilon_i)}_{=E[X_i\varepsilon_i]} = 0, \quad i = 1, \dots, n$$

holds, X is an exogenous variable.

- The uncorrelation with the error term is referred to as **exogeneity**.

Endogeneity and Exogeneity

- Taking the expectation of both sides of the simple regression model yields $\beta_0 = E[Y_i] - E[X_i]\beta_1$.
- Thus, the model can be re-written as

$$Y_i - E[Y_i] = (X_i - E[X_i])\beta_1 + \varepsilon_i, \quad i = 1, \dots, n. \quad (2)$$

- If X is an exogenous variable, multiplying X_i on both sides of (2) and taking the expectation of them lead to

$$\begin{aligned} Cov(X_i, Y_i) &= V(X_i)\beta_1 + Cov(X_i, \varepsilon_i) \\ &= V(X_i)\beta_1. \end{aligned}$$

Hence, the slope parameter β_1 can be characterized by

$$\beta_1 = \frac{Cov(X_i, Y_i)}{V(X_i)}.$$

Recall: The OLS estimator of β_1 is obtained by replacing Cov and V with the sample covariance and variance, respectively.

Endogeneity and Exogeneity

- When X is not exogenous, i.e.,

$$\underbrace{\text{Cov}(X_i, \varepsilon_i)}_{=E[X_i \varepsilon_i]} \neq 0$$

X is said to be an **endogenous variable**.

- The correlation of X with the error term ε is referred to as **endogeneity** of X .
- When X is an endogenous variable, we have

$$\frac{\text{Cov}(X_i, Y_i)}{V(X_i)} = \beta_1 + \underbrace{\frac{\text{Cov}(X_i, \varepsilon_i)}{V(X_i)}}_{\text{endogeneity bias}}$$
$$\neq \beta_1$$

Endogeneity and Exogeneity

- Therefore, in this case, if one tries to estimate β_1 based on the formula

$$\text{Cov}(X_i, Y_i) / V(X_i),$$

this entails an estimation bias (so-called **endogeneity bias**) that amounts to

$$\text{Cov}(X_i, \varepsilon_i) / V(X_i).$$

- Unfortunately, the magnitude and even the sign of the endogeneity bias are uncertain (because ε is unobservable).
- In the presence of endogeneity, the OLS estimate is uninformative about the causal impact of X on Y (it only tells us the correlation between X and Y), i.e., the OLS estimator is inconsistent.

Endogeneity and Exogeneity

Recall that when the following assumptions are met, the OLS estimator $\hat{\beta}_{n1}$ becomes consistent.

Assumption 1. The conditional expectation of Y given X is given by a linear function $\beta_0 + X\beta_1$. That is,

$$Y = \beta_0 + X\beta_1 + \varepsilon$$
$$E(Y|X) = \beta_0 + X\beta_1 \quad (\iff E(\varepsilon|X) = 0)$$

Assumption 2. $E(X^2)$ is finite.

Assumption 3. The observations $\{(Y_1, X_1), \dots, (Y_n, X_n)\}$ are independent and sampled from the same population.

Assumption 4. The error term ε is independent of X , and its variance is given by $E(\varepsilon^2) = \sigma^2$.

Endogeneity and Exogeneity

- Under these conditions,

$$\text{OLS } \hat{\beta}_{n1} = \frac{\text{sample Cov}(X, Y)}{\text{sample } V(X)} \xrightarrow{p} \frac{\text{pop Cov}(X, Y)}{\text{pop } V(X)} = \beta_1.$$

- This implies that X is exogenous under these conditions.
- Indeed, by Assumption 1 and the law of iterated expectations, we have

$$E(X\varepsilon) = E[X \underbrace{E(\varepsilon|X)}_0] = 0.$$

- Endogeneity of X indicates the violation of Assumption 1.

ATE can be estimated by OLS under exogeneity.

- For simplicity, we assume that

$$Y_{1i} - Y_{0i} = \beta, \quad i = 1, \dots, n$$

i.e., the treatment effect is homogeneous among individuals:⁴

- In this case, the ATE is simply

$$\text{ATE: } E[Y_{1i} - Y_{0i}] = \beta.$$

⁴This assumption is not necessary but is made for simplicity only.

Endogeneity and Exogeneity

ATE can be estimated by OLS under exogeneity (cont').

- Since $Y_i = T_i \cdot Y_{1i} + (1 - T_i) \cdot Y_{0i}$, we can write

$$\begin{aligned} Y_i &= Y_{0i} + T_i \beta \\ &= \underbrace{E(Y_{0i})}_{=\alpha} + T_i \beta + \underbrace{Y_{0i} - E(Y_{0i})}_{=\varepsilon_i} \\ &= \alpha + T_i \beta + \varepsilon_i \text{ such that } E(\varepsilon_i) = 0. \end{aligned}$$

- Thus, if T is exogenous such that $Cov(T_i, \varepsilon_i) = 0$, the treatment effect (ATE) β can be estimated by OLS.
- However, unfortunately the exogeneity of T cannot be tested empirically since ε is unobservable. We have to evaluate the plausibility of the exogeneity assumption on a case by case basis.

Sources of Endogeneity

Sources of Endogeneity: Omitted Variables

- Consider the following multiple regression model with two regressors X_1 and X_2 :

$$Y_i = \beta_0 + X_{1i}\beta_1 + X_{2i}\beta_2 + \varepsilon_i, \quad i = 1, \dots, n$$

- Here, we assume that both X_1 and X_2 are exogenous:

$$E[X_{1i}\varepsilon_i] = 0, \quad E[X_{2i}\varepsilon_i] = 0$$

- Then, when both X_1 and X_2 are observable variables, the slope parameters β_1 and β_2 can be correctly (consistently) estimated by multiple OLS regression.

Sources of Endogeneity: Omitted Variables

- Now consider a case in which X_2 is unobservable for some reason.
- Excluding unobservable X_2 from the model, we regress Y only on X_1 based on the following simple regression model:

$$Y_i = \alpha + X_{1i}\beta_1 + \eta_i, \quad i = 1, \dots, n$$

where $\alpha = \beta_0 + E[X_{2i}]\beta_2$, and η_i is a new error term defined by

$$\eta_i = (X_{2i} - E[X_{2i}])\beta_2 + \varepsilon_i \text{ such that } E(\eta_i) = 0.$$

- In this simple regression model, the explanatory variable X_2 , which needs to be included in the model if $\beta_2 \neq 0$, is omitted from the analysis: **omitted variables**.

Sources of Endogeneity: Omitted Variables

- In order to correctly estimate β_1 in this model, X_1 needs to be an exogenous variable, in the sense that

$$E[X_{1i}\eta_i] = 0.$$

- However, by the definition of η ,

$$\begin{aligned} E[X_{1i}\eta_i] &= E \{ X_{1i} [(X_{2i} - E[X_{2i}]) \beta_2 + \varepsilon_i] \} \\ &= Cov(X_{1i}, X_{2i}) \beta_2. \end{aligned}$$

Hence, unless either $Cov(X_{1i}, X_{2i}) = 0$ or $\beta_2 = 0$ (or both), X_1 is an endogenous variable: $E[X_{1i}\eta_i] \neq 0$.

Sources of Endogeneity: Omitted Variables

Interpretation as the spurious correlation problem:

- Let Y and X_1 denote the academic test score and the hours spent on video games, respectively.
- Consider estimating the causal effect of X_1 on Y using a simple regression model.
- In addition, as a factor affecting both X_1 and Y , there is a variable X_2 , e.g., student's home environment.

Home env X_{2i} affects video games $X_{1i} \iff Cov(X_{1i}, X_{2i}) \neq 0$

Home env X_{2i} affects academic test score $Y_i \iff \beta_2 \neq 0$

Sources of Endogeneity: Omitted Variables

- Therefore, when the variable X_2 is omitted from the regression model, the video game variable X_1 is necessarily an endogenous variable, and thus the causal effect cannot be estimated by OLS.
- The amount of the estimation bias is

$$\frac{Cov(X_{1i}, \eta_i)}{V(X_{1i})} = \frac{Cov(X_{1i}, X_{2i})\beta_2}{V(X_{1i})}$$

- If X_2 is an index of home educational environment, it is expected that $Cov(X_{1i}, X_{2i}) < 0$ and $\beta_2 > 0$, i.e., the estimation bias is negative.
- Even when the causal impact of video game on studying is actually zero (i.e., $\beta_1 = 0$), the estimate of β_1 can be significantly negative.

The non-identifiability of causality due to spurious correlation and the endogeneity bias due to omitted variables are mathematically equivalent problems.

Sources of Endogeneity: Simultaneity

- As an example, we consider the estimation of the causal effect of police on crime.
- Let X_i be the number of police officers in a district i , and Y_i be the number of crime incidents in this district.
- Simultaneity: If crime rate increases, a larger police force is needed. At the same time, increasing police force can reduce crime.
- This relationship can be expressed as:

$$Y_i = \beta_0 + X_i\beta_1 + \varepsilon_i \quad (\beta_1: \# \text{ of police officers} \rightarrow \# \text{ of crimes})$$

$$X_i = \gamma_0 + Y_i\gamma_1 + u_i \quad (\gamma_1: \# \text{ of crimes} \rightarrow \# \text{ of police officers})$$

For simplicity we do not consider other factors that can influence Y and X .

Sources of Endogeneity: Simultaneity

- If X_i is an endogenous variable such that $E[X_i\varepsilon_i] \neq 0$, we cannot correctly estimate β_1 .
- However, according to the second model,

$$\begin{aligned}E[X_i\varepsilon_i] &= E[(\gamma_0 + Y_i\gamma_1 + u_i)\varepsilon_i] \\&= E[Y_i\varepsilon_i]\gamma_1 + E[u_i\varepsilon_i].\end{aligned}$$

Further,

$$\begin{aligned}E[Y_i\varepsilon_i] &= E[(\beta_0 + X_i\beta_1 + \varepsilon_i)\varepsilon_i] \\&= E[X_i\varepsilon_i]\beta_1 + \underbrace{E[\varepsilon_i^2]}_{>0}.\end{aligned}$$

- Thus, $E[X_i\varepsilon_i] = 0$ does not hold.

Econometric methods to circumvent endogeneity

Randomized experiment

- Experimental study designs in which individuals are randomly assigned to a treatment group or a control group.
- The randomized experiment is the "gold-standard" to identify and test causal relationships. However, conducting a randomized experiment is often costly not only financially, but also ethically and politically.
- More details follow in the next lecture.

Matching method

- Matching method is a method to evaluate causal effects of a treatment by comparing individuals with similar characteristics (socio-economic background, ethnicity, gender, etc) but receiving different treatment assignment.
- Matching method can be used when the source of endogeneity for T is spurious correlation (omitted variables), and the third variable(s) W can be observed by researchers.
- This is the last topic of this course.

Quasi-experimental approaches

Instrumental variables

- Consider a simple regression model $Y = \beta_0 + X\beta_1 + \varepsilon$.
- Whatever the source of endogeneity, if we can find a variable Z that satisfies

(A) Z is not a direct determinant of Y such that $E(Z\varepsilon) = 0$

(B) Z is a direct determinant of X such that $Cov(Z, X) \neq 0$,

multiplying Z on both sides of (2) and taking the expectation of them lead to

$$Cov(Z_i, Y_i) = Cov(Z_i, X_i)\beta_1 + \underbrace{Cov(Z_i, \varepsilon_i)}_{E(Z_i\varepsilon_i)=0 \text{ by (A)}}$$
$$\iff \beta_1 = \frac{Cov(Z_i, Y_i)}{\underbrace{Cov(Z_i, X_i)}_{\neq 0 \text{ by (B)}}}.$$

Instrumental variables (cont')

- The last equality implies that we can estimate β_1 by replacing the population covariances with their sample counterparts.
- A random variable Z satisfying the conditions (A) and (B) is called the **instrumental variable** for X .
- Example: X : video games, Y : academic performance.
 - Z : proximity to an internet cafe.
- Technical details of instrumental variable method are beyond the scope of this course.

There are many other quasi-experimental approaches often used in empirical studies in economics: regression discontinuity design (RDD), difference in differences (DID), synthetic control method, etc.

- average treatment effect (ATE), 28
- causality, causal relationship, 2
- control group, 29
- correlation, 2
- correlation coefficient, 16
- counterfactual outcome, 24
- endogeneity, 36
- endogeneity bias, 37
- endogenous variable, 36
- exogeneity, 34
- exogenous variable, 34
- instrumental variables, 54
- omitted variables, 44
- potential outcome, 24
- simultaneity, 14

spurious correlation, 7

treatment, 22

treatment group, 29