

Statistical Models for Causal Analysis

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Introduction to Causal Inference
Spring 2016

Three Modes of Statistical Inference

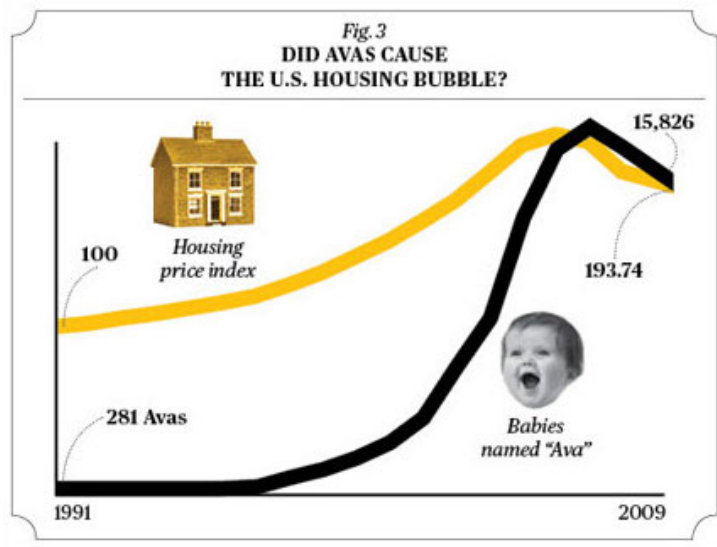
1. Descriptive Inference: summarizing and exploring data
 - Inferring ideal points from roll-call votes
 - Inferring topics from texts and speeches
 - Inferring social networks from surveys
2. Predictive Inference: forecasting out-of-sample data points
 - Inferring future state failures from past failures
 - Inferring population average turnout from a sample of voters
 - Inferring individual level behavior from aggregate data
3. Causal Inference: predicting *counterfactuals*
 - Inferring the effects of ethnic minority rule on civil war onset
 - Inferring why incumbency status affects election outcomes
 - Inferring whether the lack of war among democracies can be attributed to regime types

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Causal inference is the most difficult of the three.

Correlation \neq Causation



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- Incumbency advantage:
What *would* have been the election outcome if the candidate had not been an incumbent?
- Democratic peace:
Would the two countries have fought each other if they had been both autocratic?
- Policy intervention:
How many more disadvantaged youths *would* get employed under the new job training program?

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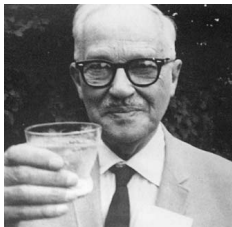
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We need a statistical model that can explicitly distinguish factials and counterfactuals.

Neyman-Rubin Potential Outcomes Model

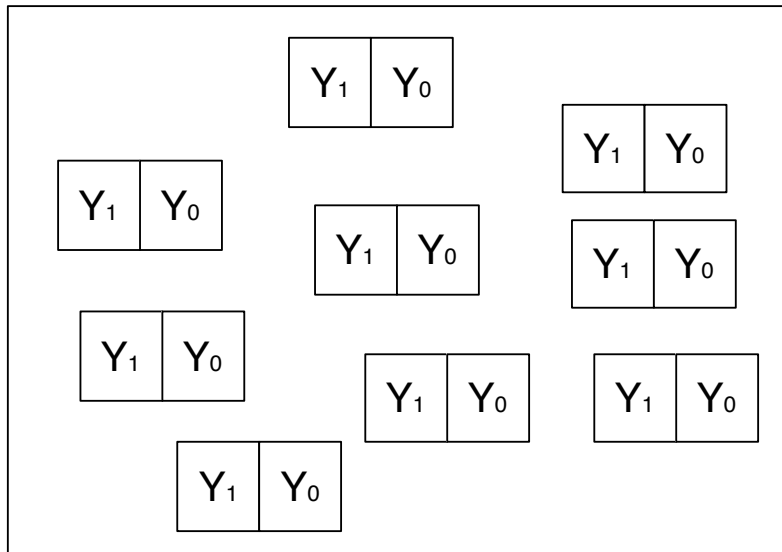


Jerzy Neyman (1894–1981)

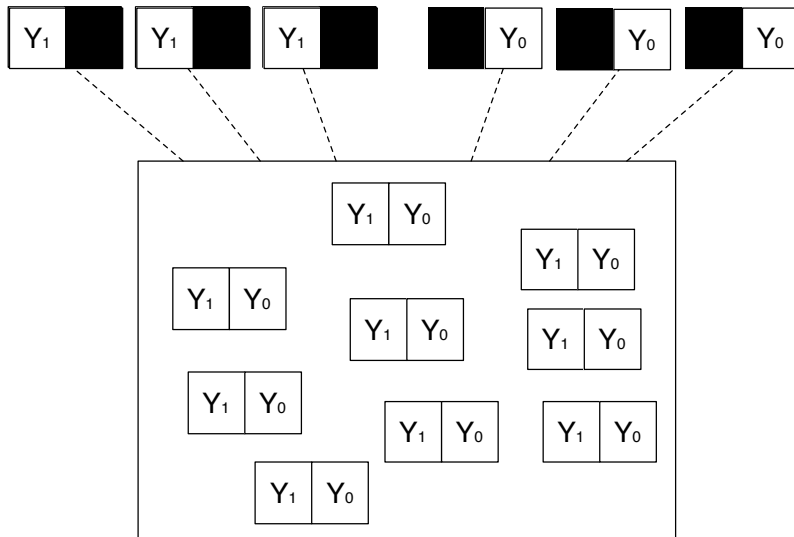


Donald Rubin (1943–)

Neyman Urn Model



Neyman Urn Model



Causality with Potential Outcomes

Definition (Treatment)

D_i : Indicator of treatment intake for unit i , where $i = 1, \dots, N$

$$D_i = \begin{cases} 1 & \text{if unit } i \text{ received the treatment} \\ 0 & \text{otherwise} \end{cases}$$

Definition (Observed Outcome)

Y_i : Variable of interest whose value may be affected by the treatment

Definition (Potential Outcomes)

Y_{di} : Value of the outcome that *would* be realized if unit i received the treatment d , where $d = 0$ or 1

$$Y_{di} = \begin{cases} Y_{1i} & \text{Potential outcome for unit } i \text{ with treatment} \\ Y_{0i} & \text{Potential outcome for unit } i \text{ without treatment} \end{cases}$$

Alternative notation: $Y_i(d)$, Y_i^d , etc.

Causality with Potential Outcomes

Definition (Causal Effect, or Unit Treatment Effect)

Causal effect of the treatment on the outcome for unit i is the difference between its two potential outcomes:

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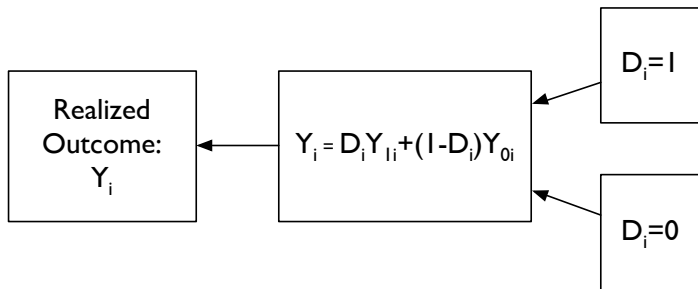
Fundamental Problem of Causal Inference (Holland 1986):

We can never observe both Y_{1i} and Y_{0i} for the same i

This makes τ_i **unidentifiable** without further assumptions.

Causal Inference as a Missing Data Problem

Problem: Causal Inference is difficult because it involves missing data.
How can we calculate $\tau_i = Y_{1i} - Y_{0i}$?



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- One “solution”: Assume **unit homogeneity**

$$\tau_i = \tau \quad \text{for all } i$$

- If Y_{1i} and Y_{0i} are constant across individual units, then cross-sectional comparisons will recover $\tau = \tau_i$
- If Y_{1i} and Y_{0i} are constant across time, then before-and-after comparisons will recover $\tau = \tau_i$

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- This may be sometimes plausible in physical sciences
- Unfortunately, rarely true in social sciences

Stable Unit Treatment Value Assumption (SUTVA)

- Recall: $Y_i = Y_{D_i i}$, or equivalently $Y_i = D_i Y_{1i} + (1 - D_i) Y_{0i}$
- This notation implicitly makes the following assumption:

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Assumption (SUTVA)

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Violation examples: Vaccination, fertilizer on plot yield, communication

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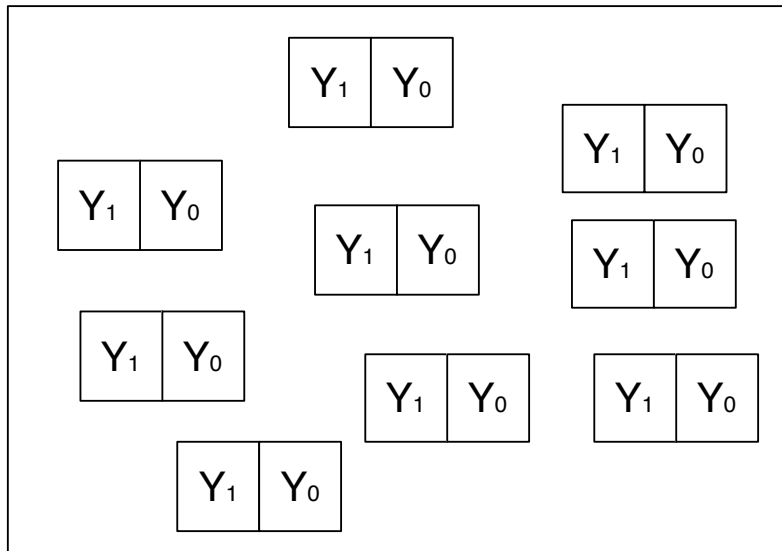
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Without SUTVA, causal inference becomes exponentially more difficult as N increases.

Back to the Neyman Urn Model



Causal Quantities of Interest, or Estimands

- Unit-level causal effects are fundamentally unobservable
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- For now, assume we observe all units in the population of interest, i.e. N = size of population

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Definition (Average treatment effect, ATE)

$$\tau_{ATE} = \frac{1}{N} \sum_{i=1}^N \{Y_{1i} - Y_{0i}\}$$

or equivalently

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

- Note that τ_{ATE} is still unidentified
- In the rest of this course, we will consider various assumptions under which τ_{ATE} can be identified from observed information

Other Causal Estimands

Definition (Average treatment effect on the treated, ATT)

$$\tau_{ATT} = \frac{1}{N_1} \sum_{i=1}^N D_i \{Y_{1i} - Y_{0i}\} \quad \text{where} \quad N_1 = \sum_{i=1}^N D_i$$

or equivalently $\tau_{ATT} = \mathbb{E}[Y_{1i} - Y_{0i} | D_i = 1]$

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where X_i is a **pre-treatment covariate** for unit i

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- In words, $\tau_{CATE}(x)$ is a **subgroup effect**, treatment effect on units who have particular characteristics x .

Illustration: Average Treatment Effect

Suppose we observe a population of 4 units:

i	D_i	Y_i
1	1	3
2	1	1
3	0	0
4	0	1

What is $\tau_{ATE} = \mathbb{E}[Y_{1i} - Y_{0i}]$?

Illustration: Average Treatment Effect

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3	0	0
4	0	1
$\mathbb{E}[Y_i \mid D_i = 1]$		2
$\mathbb{E}[Y_i \mid D_i = 0]$		0.5
$\mathbb{E}[Y_i \mid D_i = 1] - \mathbb{E}[Y_i \mid D_i = 0]$		1.5

What is $\tau_{ATE} = \mathbb{E}[Y_{1i} - Y_{0i}]$?

Naïve estimator:

$$\begin{aligned}\tilde{\tau} &= \mathbb{E}[Y_i \mid D_i = 1] - \mathbb{E}[Y_i \mid D_i = 0] \quad (\text{observed difference in means}) \\ &= \frac{3 + 1}{2} - \frac{0 + 1}{2} = 1.5 \quad \text{Could this be wrong?}\end{aligned}$$

Illustration: Average Treatment Effect

Suppose we observe a population of 4 units:

i	D_i	Y_i	Y_{1i}	Y_{0i}	τ_i
1	1	3	3	?	?
2	1	1	1	?	?
3	0	0	?	0	?
4	0	1	?	1	?

What is $\tau_{ATE} = \mathbb{E}[Y_{1i} - Y_{0i}]$? We need potential outcomes that we do not observe!

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Suppose hypothetically: $Y_{01} = 0, Y_{02} = Y_{13} = Y_{14} = 1$.

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Why $\tau_{ATE} \neq \tilde{\tau}$? When would they be equal?

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Selection Bias

- Comparisons of observed outcomes for the treated and the untreated do not usually give the right answer:

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Example: Church attendance and turnout

- churchgoers differ from individuals who do not attend church in many ways (e.g. civic duty)
- turnout for churchgoers would be higher than for non-churchgoers even if churchgoers never attended church ($E[Y_{0i}|D_i = 1] - E[Y_{0i}|D_i = 0] > 0$)

Selection Bias

- Comparisons of observed outcomes for the treated and the untreated do not usually give the right answer:

$$\begin{aligned}\tilde{\tau} &= \mathbb{E}[Y_i|D_i = 1] - \mathbb{E}[Y_i|D_i = 0] \\ &= \mathbb{E}[Y_{1i}|D_i = 1] - \mathbb{E}[Y_{0i}|D_i = 0] \\ &= \underbrace{\mathbb{E}[Y_{1i} - Y_{0i}|D_i = 1]}_{\tau_{ATT}} + \underbrace{\mathbb{E}[Y_{0i}|D_i = 1] - \mathbb{E}[Y_{0i}|D_i = 0]}_{\text{Bias}}\end{aligned}$$

- Bias term $\neq 0$ if **selection into treatment** is associated with potential outcomes

Example: Job training program for the disadvantaged

- participants are self-selected from a subpopulation of individuals in difficult labor situations
- post-training period earnings for participants would be lower than those for nonparticipants in the absence of the program ($E[Y_{0i}|D_i = 1] - E[Y_{0i}|D_i = 0] < 0$)

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An Alternative Causal Model: Causal Graphs

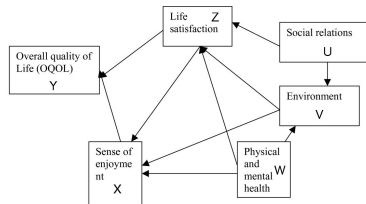
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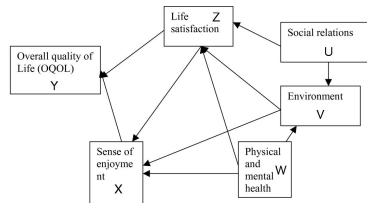
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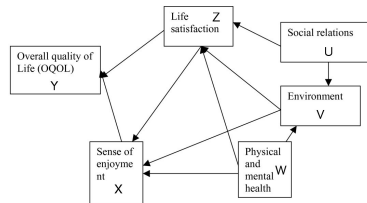
$$Y = \alpha_0 + \alpha_1 X + \alpha_2 Z + \varepsilon_\alpha$$

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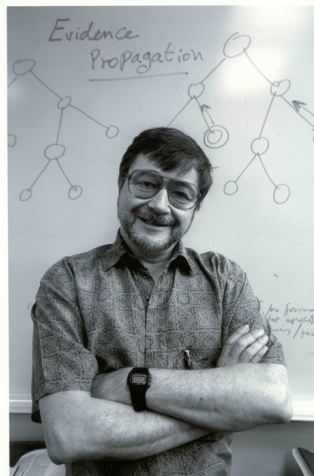
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- Went out of fashion:
 - Strong distributional/functional form assumptions
 - No language to distinguish causation from association

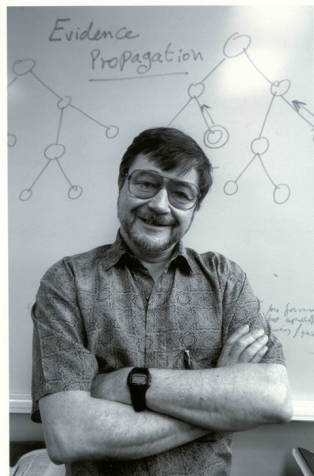
Pearl's Attack



Judea Pearl (1936–) proposed a new causal inference framework based on **nonparametric structural equation modeling (NPSEM)**

- Originally a computer scientist
- Previous important work on artificial intelligence
- *Causality* (2000, Cambridge UP)
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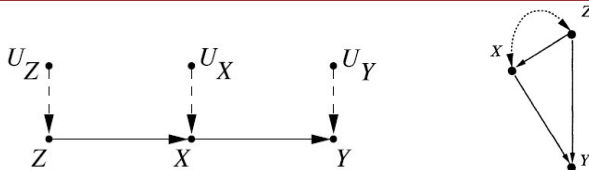


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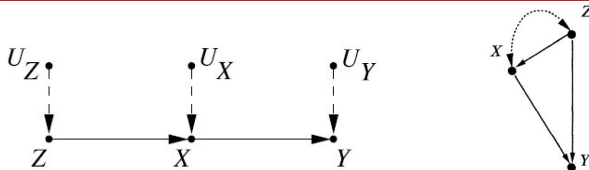
Pearl's framework builds on SEMs and revives it as a formal language of causality.

Causal Diagram



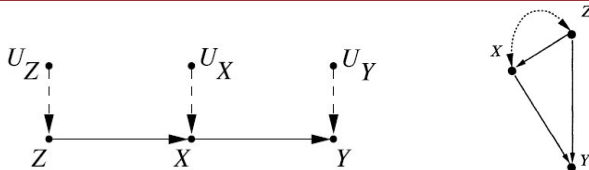
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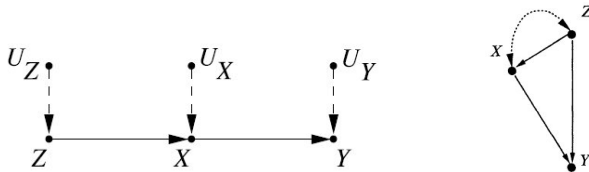
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- Note that it is *missing* edges that encode causal assumptions
 - Missing arrows encode exclusion restrictions
 - Missing dashed arcs encode independencies between error terms

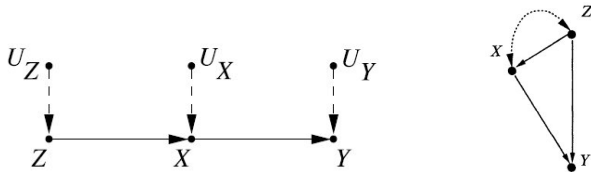
NPSEM and Treatments



- A causal DAG has a one-to-one relationship with an NPSEM:

$$z = f_Z(u_Z), \quad x = f_X(z, u_X), \quad y = f_Y(x, u_Y)$$

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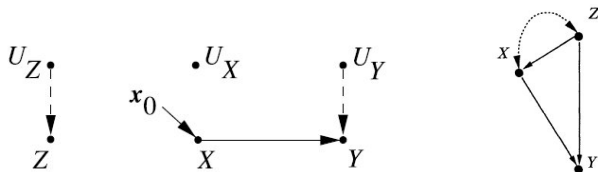


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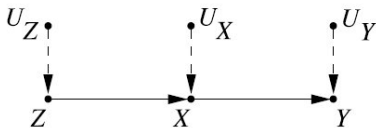
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- Treatments (interventions) are represented by the *do()* operator
- For example, $do(x_0)$ holds X at x_0 exogenously and thus replaces the structural equation for X with this value:

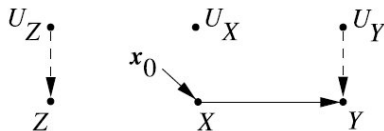
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Causal Effects and Identification



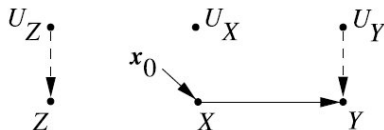
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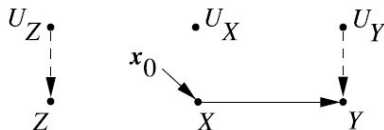
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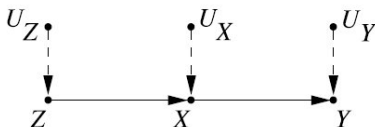


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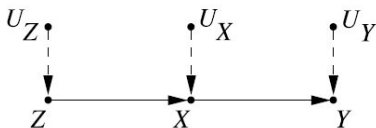
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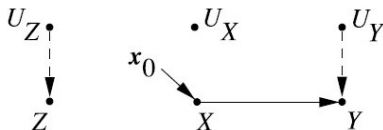
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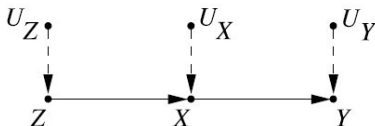
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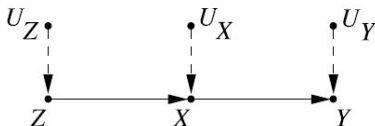
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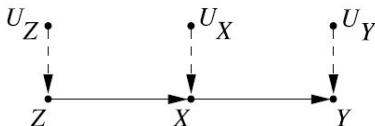
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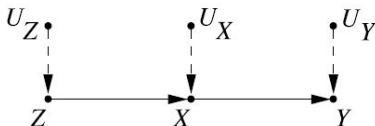
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- Because of this fundamental equivalence, we will mostly work with potential outcomes, currently the standard framework in social sciences.
- Graphs are useful for expressing and visualizing a causal model in empirical research.

Aside: Modern Schools of Statistical Causal Inference

- ① Potential Outcomes (Rubin, Rosenbaum, Imbens)
 - Most widely used in applied sciences, especially economics and political science
 - Causal inference as missing data problem
- ② NPSEM (Pearl)
 - Uses mathematical theory of graphs
 - Borrows concepts from Bayes net and neural networks
- ③ Sufficient Component Causes (Rothman, VanderWeele)
 - Originates in epidemiology; “causal pies”
 - Resembles the qualitative comparative analysis (QCA)
- ④ Decision-theoretic causality (Dawid)
 - Does not assume existence of counterfactuals
- Causal inference as an independent field of science: *Journal of Causal Inference*

Summing Up

- Potential outcomes framework (Neyman-Rubin model) as a dominant framework for causal inference
- Causal quantities are defined by potential outcomes (counterfactuals), not by realized (observed) outcomes
- No assumption of unit homogeneity; causal effects allowed to vary unit by unit
- Observed association is neither necessary nor sufficient for causality
- Estimation of causal effects often starts with studying the treatment assignment mechanism