A (Brief) Introduction to Causal Inference

Evan A. Jones
University of Maryland – College Park

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The “Why?” and “What If?” Questions

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- Human children explore the world as scientists do (2, 4):
  - Asking questions
  - Forming hypotheses
  - Testing hypotheses via interventions (5)

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- Human children explore the world as scientists do (2, 4):
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- By adulthood, we have fairly solid causal intuition about the physical world

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- When can we interpret β as a causal effect?

What is causality?

Outline

- Logic of Causal Inference
- Experiments vs the World
- Potential Outcomes vs Structural Causal Models

Logic of Causal Inference

Beyond Description

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- Break down phenomena into constituent parts and define how parts interact to produce emergent behavior (*data-generating process*) (12)
- Once uncovered, causal mechanisms are powerful

Beyond Description

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- (Incumbency effect) What *would have* been the election outcome if the candidate were an incumbent?

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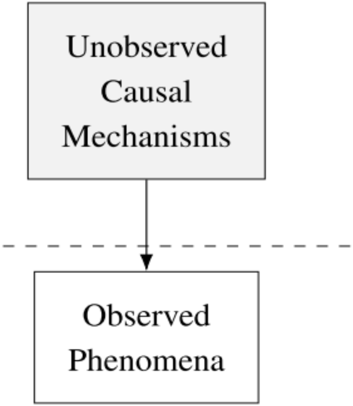
- (Incumbency effect) What *would have* been the election outcome if the candidate were an incumbent?
- (Resource curse) What *would have* been the GDP growth rate without oil?
- (Democratic peace) *Would* the two countries have escalated conflict similarly if they were both autocratic?

Causal mechanisms allow us to make unbiased predictions about the effect of *interventions* (10) and *counterfactual situations*

The Ladder of Causation

Answering causal queries requires more than observing data. Why?



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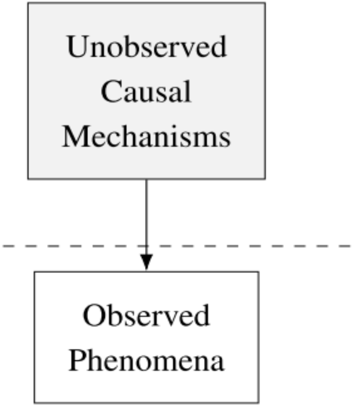

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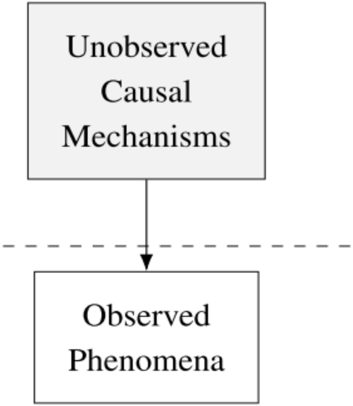

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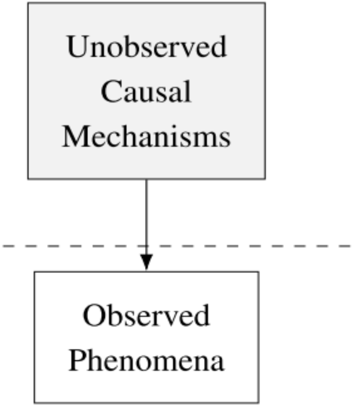

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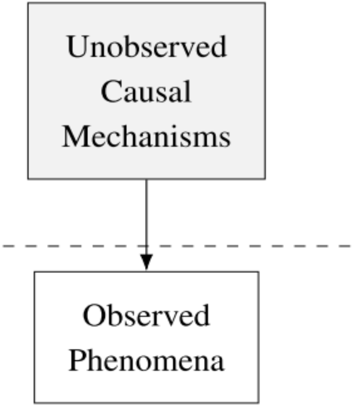
4. Data suggest paradoxical effects



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Simpson's Paradox

Believe the Election Was Stolen

		Total
Misinfo	Yes	47% ($\frac{582}{1240}$)
	No	60% ($\frac{456}{760}$)
		$\mathbb{E}[Y \mid T]$

- Social media data on user behavior
- Consumers of misinformation are *less* likely to believe the election was stolen
- Hmm... what is happening?

Simpson's Paradox

Believe the Election Was Stolen

		D	R	Total
Misinfo	Yes	30% ($\frac{240}{800}$)	78% ($\frac{342}{440}$)	47% ($\frac{582}{1240}$)
	No	11% ($\frac{16}{150}$)	72% ($\frac{440}{610}$)	60% ($\frac{456}{760}$)
		$\mathbb{E}[Y \mid T, C{=}D]$	$\mathbb{E}[Y \mid T, C{=}R]$	$\mathbb{E}[Y \mid T]$

- When we group by party, the effect of misinformation flips

$$\underbrace{\frac{800}{1240}(0.3)}_{\text{upweight}} + \frac{440}{1240}(0.78) = 0.47$$

$$\frac{150}{760}(0.11) + \underbrace{\frac{610}{760}(0.73)}_{\text{upweight}} = 0.60$$

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$$\mathbb{E}[Y\mid T=1]-\mathbb{E}[Y\mid T=0]=-0.13$$

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What is the effect of misinformation?

- When we group by party, the effect of misinformation flips

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Layer (Symbolic)	Typical Activity	Typical Question	Example	Statistics

Based on table from (1, p. 8)

The Ladder of Causation

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$\mathcal{L}_1$	Associational $P(y\|x)$	Seeing	What is? How would seeing X change my belief in Y?	What does a speech tell us about a politician's ideology?	Regression / Model fitting / MLE
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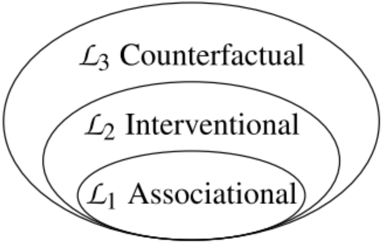
$\mathcal{L}_3$	Counterfactual $P(y_x\|x', y')$	Imagining	Why? What if I had acted differently?	Was it the Russians that caused Trump to win?	—

Based on table from (1, p. 8)

The Problem of Causal Inference

- The layers of the causal hierarchy are nested



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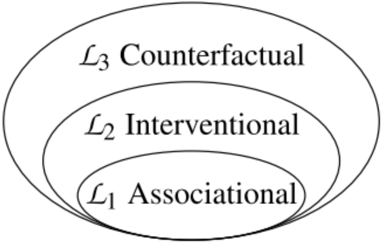
- *Generally impossible* to draw higher-layer inferences with only lower-layer information (1)



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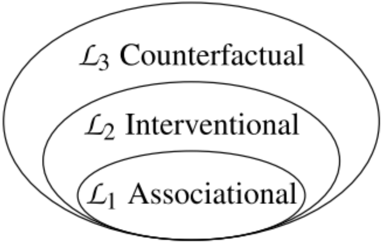

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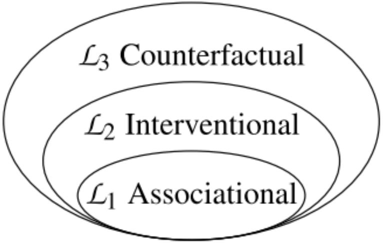
- A: *Causal assumptions*



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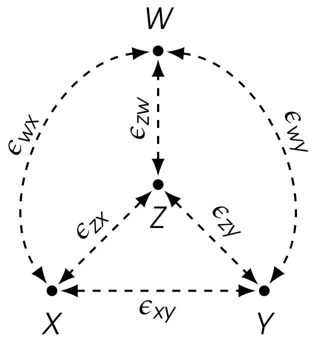
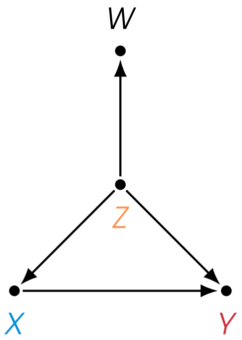


Observed data ($\mathcal{L}_1$)

- At $\mathcal{L}_1$ we have a variable "salad"
- In terms of probability, all we know is $P(X, Y, Z, W)$
- Everything *could be* related to everything else
- Best we can do is estimate associations (correlations)

The Problem of Causal Inference



observed data ($\mathcal{L}_1$) +
causal assumptions

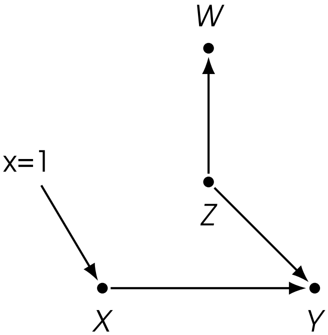
- With knowledge + additional evidence, we *assume* away some paths

- Arrows imply conditional dependencies:

$$\implies \quad P(Y|Z,X)P(X,W|Z)P(Z)$$

- Still *no intervention*, observed effect of X on Y depends on Z

The Problem of Causal Inference



Intervention data ($\mathcal{L}_2$)

- We set the value of X:

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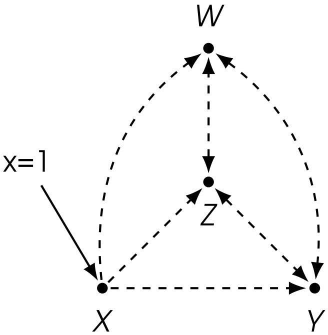

Intervention data ($\mathcal{L}_2$)

- We set the value of X:
  $do(x = 1)$
- Causal assumptions rendered moot
- If X influences Y, then a change in X will appear as a change in Y

  $$\mathbb{E}[Y|X = x_1] - \mathbb{E}[Y|X = x_0] \neq 0$$

Simpson's Paradox Revisited

- What is the effect of misinformation on the belief that the 2020 election was fraudulent?

$$\mathbb{E}[Y|T=1] - \mathbb{E}[Y|T=0] = -0.13$$

or

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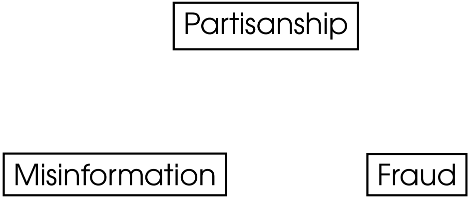

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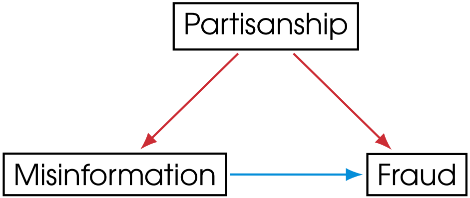

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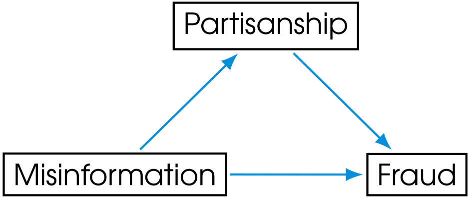

The Problem of Causal Inference

"The central question in the analysis of causal effects is the question of *identification*: can the controlled (post-intervention) distribution, $P(Y = y|do(x))$, be estimated from data governed by the pre-intervention distribution $P(X, Y, Z, W)$?"

- Pearl (2009, p. 108)

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- Thus, the key to causal inference is achieving identification
- In experiments, identification is built-in since *we* control the treatment
- In observational data, identification is tougher and, sometimes, *unachievable*
- So why not only do experiments?

Experiments vs the World

Experiments: Pros and Cons

Pros

- Identification guaranteed

Cons

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- Causal mechanism still an assumption

Observational Studies: Pros and Cons

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$\implies$ external validity

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Observational Studies: Pros and Cons

Pros

- Can study phenomena of interest
- ...in the population of interest

$\Longrightarrow$ external validity

Cons

- Identification challenged by
  - selection bias
  - non-random treatment
  - data limitations
- Identification may be impossible without more data or experiment

Potential Outcomes
vs
Structural Causal Models

Potential Outcomes

- Associated with Neyman (7) and Rubin (11)
- Widely adopted in social sciences and medicine
- Randomized experiment serve as its ruling paradigm

Potential Outcomes

- Object of analysis is a unit-based response variable
  - patients
  - survey respondents
  - cities

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- "The value outcome Y would obtain in experimental unit i had treatment T_i been t"

Potential Outcomes

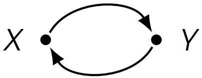
- Units: $i = 1, \ldots, N$
- "Treatment":
  - $T_i = 1$ if treated
  - $T_i = 0$ otherwise
- *Observed* outcome: Y_i
- Pre-treatment covariates: X_i
- Potential outcomes: $Y_i(1)$ and $Y_i(0)$

Voters	**Contact**	**Turnout**		**Age**	**Party ID**
i	T_i	$Y_i(1)$	$Y_i(0)$	X_i	X_i
1	1	1	?	19	D
2	0	?	0	45	D
3	0	?	1	36	R
$\vdots$	$\vdots$	$\vdots$	$\vdots$	$\vdots$	$\vdots$
N	1	0	?	71	R

Potential Outcomes Assumptions

Core Assumptions

1. No simultaneity

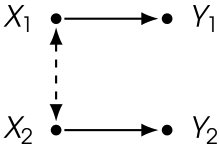


Potential Outcomes Assumptions

Core Assumptions

1. No simultaneity

2. No interference between units



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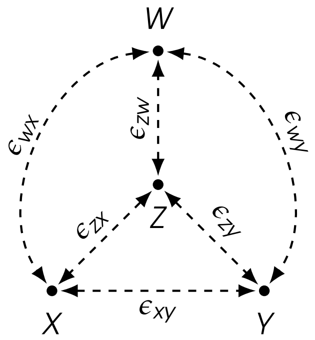
- Stable Unit Treatment Value Assumption (SUTVA)
- Potential violations:
  - feedback effects
  - spill-over effects
  - different treatment administration
- Observed outcome is random because treatment is random
- Multi-valued treatment: more potential outcomes for each unit

Potential Outcomes Assumptions

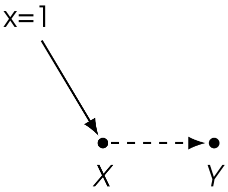


Observed data ($\mathcal{L}_1$)

Crux of PO is randomized treatment

- Causal mechanism too complex to rule out no omitted variable with certainty

Potential Outcomes Assumptions



Intervention data ($\mathcal{L}_2$)

Crux of PO is randomized treatment

- Causal mechanism too complex to rule out no omitted variable with certainty
- Looks for "as-if" random treatments or proxy treatments
- Allows you to ignore possible confounders

Potential Outcomes Research Designs

- Preferred research designs based on exogeneity assumption:
  - Instrumental Variables (IV)
  - Regression Discontinuity Design (RDD)
  - Difference-in-Difference (DiD)
- When we cannot find intervention data: matching
- Criticisms:
  - exogeneity assumption almost always untestable
  - finding guaranteed random treatments in the wild is extremely rare
  - OR the randomized "treatment" doesn't quite align with the theory we want to test

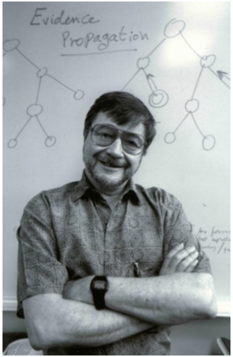
Structural Causal Models

- Associated with Pearl (8) but many predecessors and successors

- Emerged from computer science field, but builds on:
    - structural equation models (SEM) (3)
    - potential outcomes
    - probabilistic graphical models (6, 13)

- The causal graph serves as ruling paradigm

- sometimes referred to as a "DAG" (directed acyclic graph)



Structural Causal Models

- Based on a directed graph that displays casual relationships between variables

- Models sometimes defined as ordered triples $\langle U, V, E \rangle$:
  - Exogenous variables U
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  - Set of equations E that defining relationships between V

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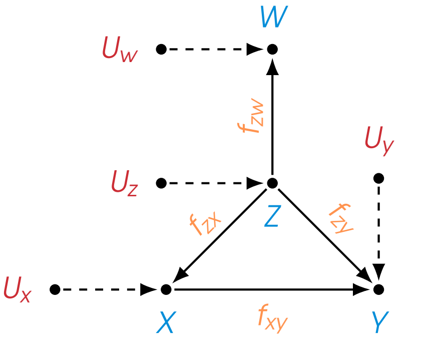
- The models are probabilistic and represent a unique factorization of a joint probability distribution into conditional probabilities
- Use *do*-calculus to achieve identification on observed data

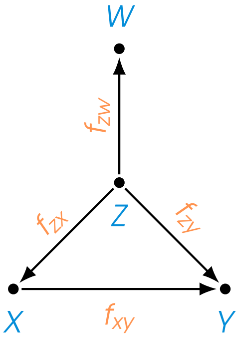
SCM Assumptions



observed data ($\mathcal{L}_1$) +
causal assumptions

- The notation seems scary, but we saw this before

SCM Assumptions



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  - U are independent of what happens within the system
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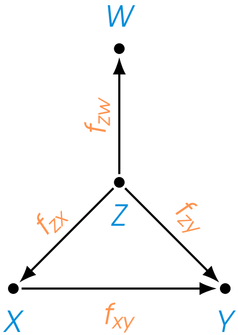

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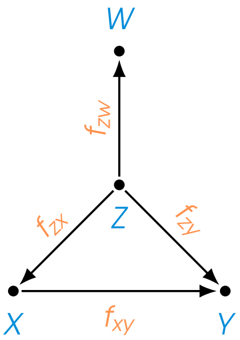

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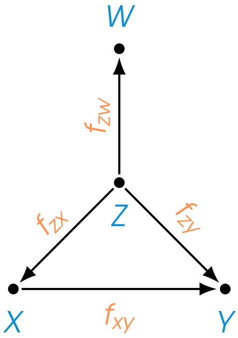

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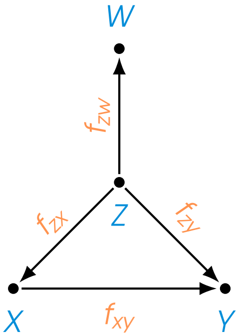
SCM Assumptions



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  - E represents functional relationships
- All assumptions are encoded into the graph itself
- Since the graph represents conditional probabilities, we can determine what variables to adjust for from it
- Theory $\Longrightarrow$ assumptions

Model Elements

All DAGs are built from three fundamental relationships

Model Elements

Chain

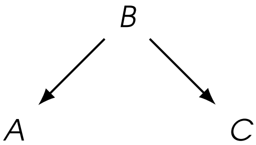
- Straight line connections with arrows pointing from cause to effect
- B mediates effect of A on C

$$A \longrightarrow B \longrightarrow C$$

Model Elements

Fork

- One cause has multiple effects
- There exists spurious correlation between A and C due to B
- Eliminate by adjusting for B



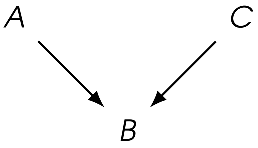
Model Elements

Collider

- Multiple causes affect one outcome

- Conditioning on B often induces a non-causal negative relationship between A and C

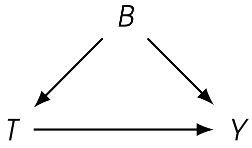
- Collider bias, wherein B explains away correlation between A and C



Identification with DAGS

Identification is achieved via *do*-calculus

- Set of rules for determining a minimally-sufficient set of adjustment variables
- Examine all paths between treatment and outcome, control for confounders
- Not too complicated, but beyond scope of presentation



B confounds effect of T on Y

SCM as a Language

- SCMs represents a *language* of causality

- All other approaches to causal inference can be encoded in a DAG (i.e. PO is subsumed by SCM)

- Can also be used to determine when and how to escape from selection bias

- Criticisms:
    - Encoding our theory into a DAG can be *hard*
    - Complex theory $\implies$ complex DAG
    - $\hookrightarrow$ DAGs can become overwhelming, fast
    - do-calculus only guarantees identification if theory is correct

- Dagitty: tool that performs do-calculus for you, has `R` package too

Conclusion

Conclusion

- Randomized experiments are a gold standard for causal inference
- But they are black boxes
- The key to causal inference on observational data is:
  - make stronger assumptions about the relationships between variables
  - Search for interventional $\mathcal{L}_2$ setups that match theory
- In SCM, we determine if causal query is identify; if not, identify minimal adjustment set from DAG
- In PO, identifiability is guaranteed so long as we believe intervention is truly random
- Once identified, we can interpret β as a causal effect

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