### A (Brief) Introduction to Causal Inference

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Prepared for GVPT's GSA Method Workshop, Spring 2021.

Understanding the world around us is an inherently human endeavor

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



- Understanding the world around us is an inherently human endeavor
- Human children explore the world as scientists do (2, 4):
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  - Testing hypotheses via interventions (5)
- By adulthood, we have fairly solid causal intuition about the physical world



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- Take the following, for instance :

$$Y = \alpha + \beta X + \theta Z + \epsilon$$

- When can we interpret  $\beta$  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

- Causal explanations are "more than mere descriptions ... of the observed data" (1, p. 3)

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- Break down phenomena into constituent parts and define how parts interact to produce emergent behavior (*data-generating process*) (11)
- Once uncovered, causal mechanisms are powerful

We can answer counterfactual questions:

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

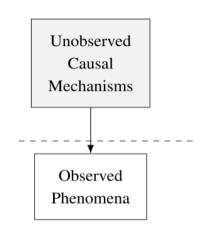
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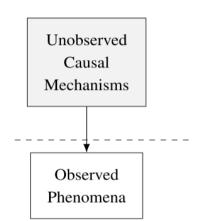
- (Incumbency effect) What *would have* been the election outcome if the candidate were an incumbent?
- (Resource curse) What would have been the GDP growth rate without oil?
- (Democratic peace) Would the two countries have escalated conflict similarly if they were both autocratic?

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

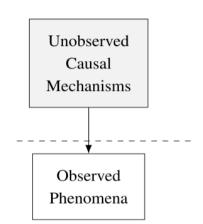


Answering causal queries requires more than observing data. Why?

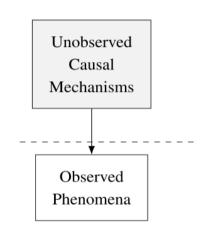
1. Causal mechanisms are generally *unobservable* (8)



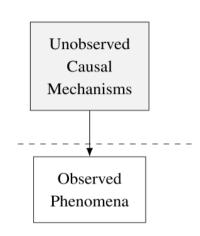
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- Causal mechanisms are generally unobservable (8)
- 2. Data represent single realized outcome of an intervention (traces of mechanism)
- 3. Sample does not match the population/group we want to study
- 4. Data suggest paradoxical effects



# Simpson's Paradox

#### Believe the Election Was Stolen

		Total	
Misinfo	Yes	47% ( <u>582</u> )	
	No	$60\% \left(\frac{456}{760}\right)$	
		$\mathbb{E}[Y T]$	

- 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

# Misinfo

	D	R	Total
		78% ( <del>342</del> )	
No	11% ( <del>16</del> )	$72\% \left(\frac{440}{610}\right)$	$60\% \left(\frac{456}{760}\right)$
	$\mathbb{E}[Y T,C=D]$	$\mathbb{E}[Y T,C=R]$	$\mathbb{E}[Y T]$

 When we group by party, the effect of misinformation flips

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

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

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Layer (Symbolic)	Typical Activity	Typical Question	Example	Statistics
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Based on table from (1, p. 8)

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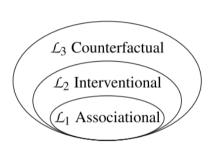
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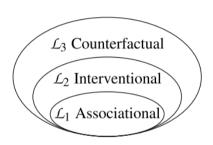
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$\mathcal{L}_3$	Counterfactual $P(y_x x',y')$	Imagining	Why? What if I had acted differently?	Was it the Russians that caused Trump to win?	_
	Based on table from (1, p. 8)				

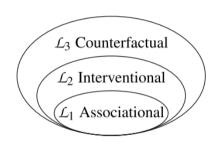
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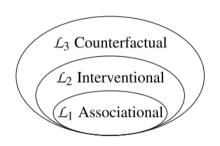
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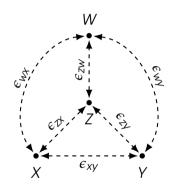


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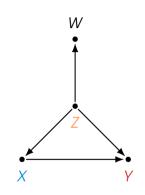
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- Q: So what "information" allows us to move up the ladder?
- A: Causal assumptions





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)

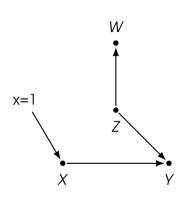


observed data ( $\mathcal{L}_1$ ) + causal assumptions

- With knowledge + additional evidence, we assume away some paths
- Arrows imply conditional dependencies:

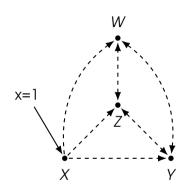
$$\implies P(Y|Z,X)P(X,W|Z)P(Z)$$

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



- We set the value of X: do(x = 1)

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



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

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

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

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

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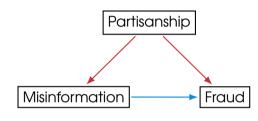
Misinformation

Fraud

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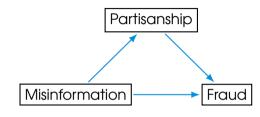
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It depends on your causal assumptions



"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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- 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

# Pros

Cons

- Identification guaranteed

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 $\Longrightarrow$  Internal validity

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- Causal mechanism still an assumption

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- Identification challenged by
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  - data limitations
- Identification may be impossible without more data or experiment

# Potential Outcomes VS Structural Causal Models

- Associated with Neyman (7) and Rubin (10)
- Widely adopted in social sciences and medicine
- Randomized experiment serve as its ruling paradigm





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  - survey respondents
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- "The value outcome Y would obtain in experimental unit i had treatment  $\mathcal{T}_i$  been t"

### Potential Outcomes

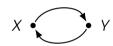
- Units: 
$$i = 1, ..., N$$

- "Treatment":
  - $T_i = 1$  if treated
  - $T_i = 0$  otherwise
- Observed outcome: Yi
- 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
;	;	:	:	:	:
Ň	i	0	?	71	R

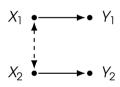
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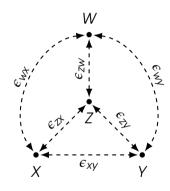
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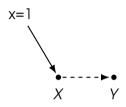
- Stable Unit Treatment Value Assumption (SUTVA)
- Potential violations:
  - feedback effects
  - spill-over effects
  - different treatment administration
- Observed outcome is random because treatment is random
- Multi-valued treatment: more potential outcomes for each unit



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

#### Crux of PO is randomized treatment

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



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

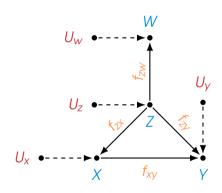
- Associated with Pearl (8) but many predecessors and successors
- Emerged from computer science field, but builds on:
  - structural equation models (SEM) (3)
  - potential outcomes
  - probabilistic graphical models (6, 12)
- The causal graph serves as ruling paradigm
- sometimes referred to as a "DAG" (directed acyclic graph)



- 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*
  - Endogenous variables V
  - Set of equations  $\it E$  that defining relationships between  $\it V$

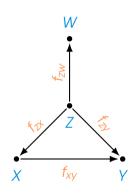
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- Use do-calculus to achieve identification on observed data



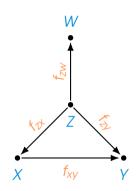
observed data ( $\mathcal{L}_1$ ) + causal assumptions

- The notation seems scary, but we saw this before



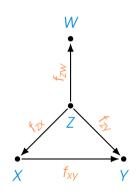
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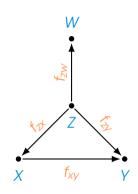
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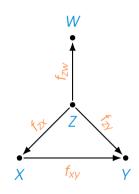
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- Theory  $\Longrightarrow$  assumptions

All DAGs are built from three fundamental relationships

#### Chain

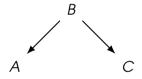
Straight line connections with arrows pointing from cause to effect

$$A \longrightarrow B \longrightarrow C$$

- B mediates effect of A on C

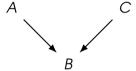
#### Fork

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



#### Collider

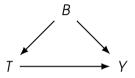
- Multiple causes affect one outcome
- Conditioning on B often induces a non-causal negative relationship between A and C
- Collider bias, wherein B explains away correlation between A and C



### 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 ⇒ complex DAG
  - → 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  $\beta$  as a causal effect

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