#### 12 Maggio 2025

### INTRODUCTION TO

### CAUSAL MODELLING AND REASONING

Martina Cinquini & Isacco Beretta

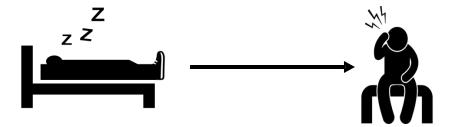


### REICHENBACH COMMON CAUSE PRINCIPLE

Let X and Y be two variables such that X and Y are **statistically dependent** then it holds:

- X is indirectly causing Y
- Y is indirectly causing X
- There is a possibly unobserved common cause Z that indirectly causes both
   X and Y

Sleeping with shoes on is strongly correlated with waking up with a headache



Sleeping with shoes on is strongly correlated with waking up with a headache

Common cause: drinking the night before

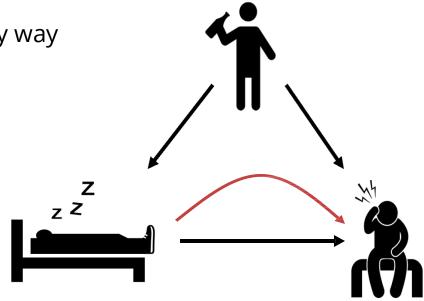
1. Shoe-sleepers differ from non-shoe-sleepers in a key way

Sleeping with shoes on is strongly correlated with waking up with a headache

Common cause: drinking the night before

1. Shoe-sleepers differ from non-shoe-sleepers in a key way

2. Confounding



Sleeping with shoes on is strongly correlated with waking up with a headache

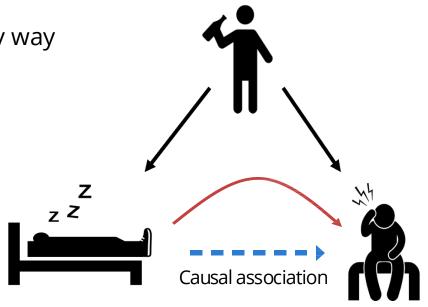
Common cause: drinking the night before

1. Shoe-sleepers differ from non-shoe-sleepers in a key way

2. Confounding

Total association (e.g., correlation):

Mixture of causal and confounding association



### INGREDIENTS OF A STATISTICAL THEORY OF CAUSALITY

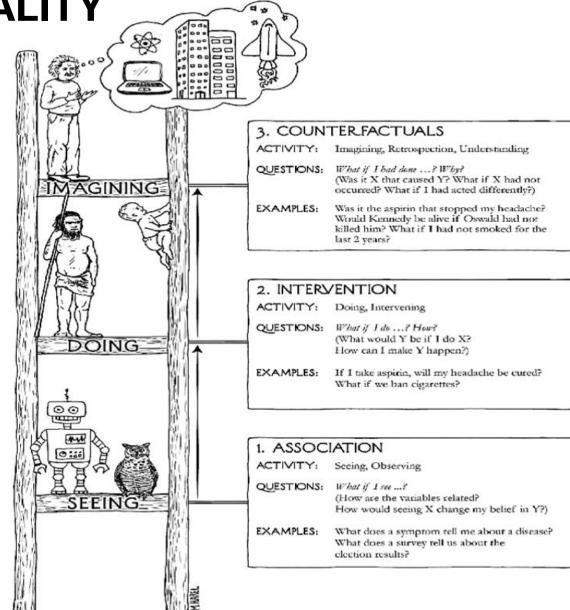
- Working definition of causation
- Method for creating causal models
- Method for linking causal models with features of data
- Method for reasoning over model and data

THE LATTER OF CAUSALITY

"Actual" Causality

"Causality-in-mean"

**Statistics** 



### **RANDOMIZED EXPERIMENTS**

Which kind of post works better?

Interventional data

Post 1

Post 2

Post ...

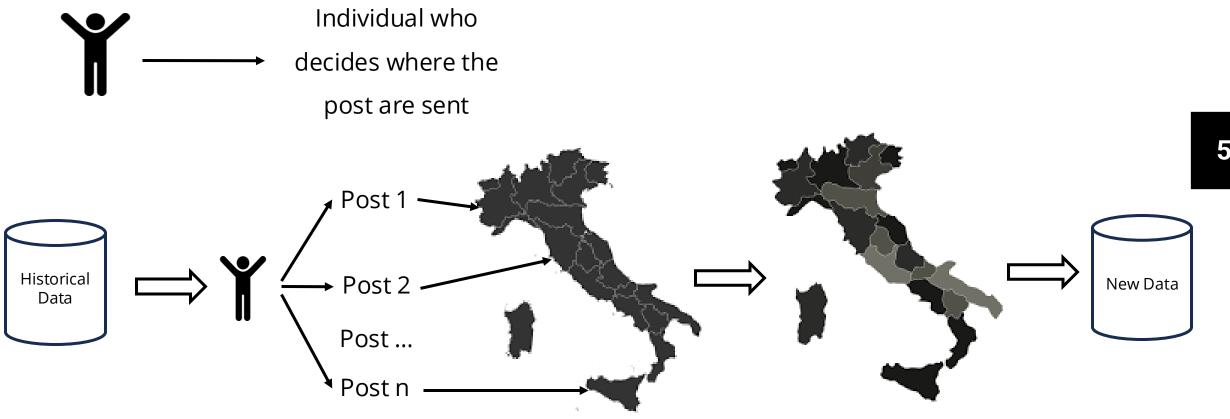
#### Limitations

- Can not use historical data
- It **cannot** be applied to **certain situations** (e.g., long-term effect, selected demographics, content virality)

Post n

### **BEYOND RANDOMIZED EXPERIMENTS**

Associational data



### **CAUSAL MODEL FRAMEWORKS**

Potential Outcomes (PO)

Structural Causal Model (SCM)

Antecedents in the earlier econometric literature

Demand and Supply Models (Haavelmo, 1944)

Path analysis (Wright, 1934)

These frameworks are complementary, with different strengths that make them appropriate to address different problems in specific situations.

### **CAUSAL MODEL FRAMEWORKS**

Potential Outcomes (PO)

Structural Causal Model (SCM)

Antecedents in the earlier econometric literature

Demand and Supply Models (Haavelmo, 1944) Path analysis (Wright, 1934)

Specifically, to deal with:

Estimating individual-level causal effects

Complex models with a large number of variables

# POTENTIAL OUTCOME: INTUITION

Inferring the effect of treatment on some outcome



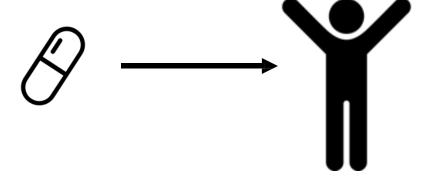
## POTENTIAL OUTCOME: INTUITION

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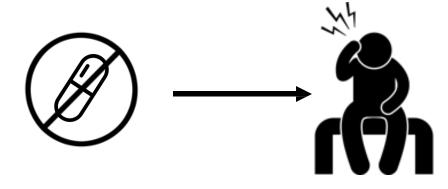


Causal Effect?

Take a pill



Don't take a pill



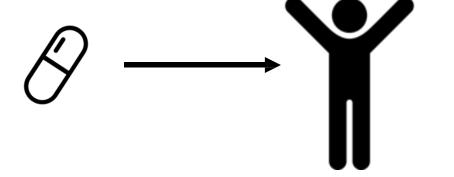
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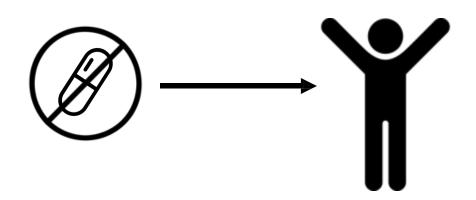


No Causal Effect

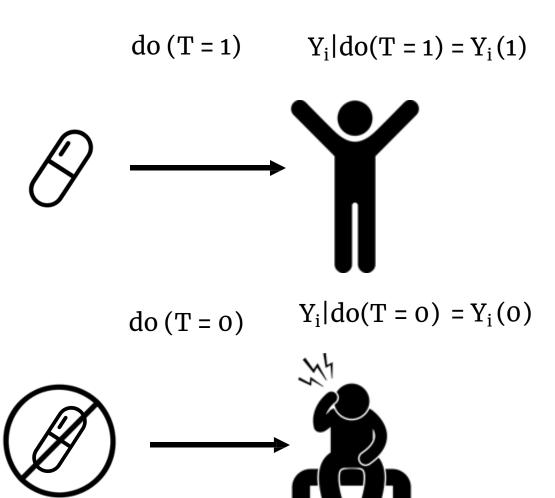
Take a pill



Don't take a pill



### POTENTIAL OUTCOME: NOTATION



T: Observed Treatment

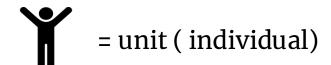
Y: Observed Outcome

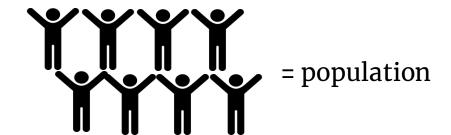
i: used in subscript to denote a specific individual

 $Y_i(1)$ : PO under treatment

 $Y_i(0)$ : PO under no treatment

### **OTHER DEFINITIONS**

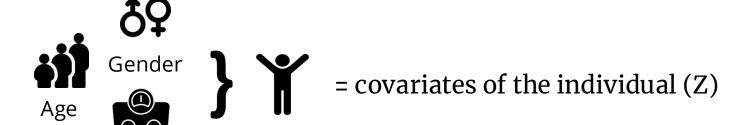




#### INDIVIDUAL TREATMENT EFFECT (ITE)

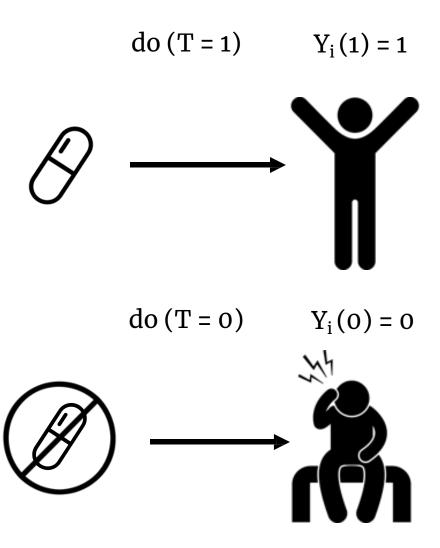
The ITE for the  $i^{th}$  unit is defined as follows:

$$Y_{i}(1) - Y_{i}(0)$$



Weight

### POTENTIAL OUTCOME: NOTATION



T: Observed Treatment

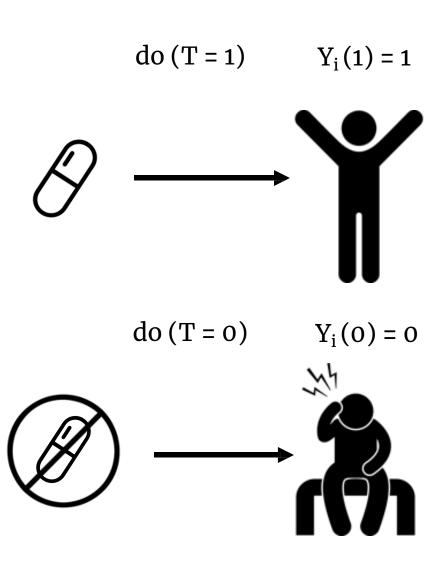
Y: Observed Outcome

i: used in subscript to denote a specific individual

 $Y_i(1)$ : PO under treatment

 $Y_i(0)$ : PO under no treatment

Causal Effect:  $Y_i(1) - Y_i(0) = 1$ 



#### **Fundamental Problem.**

We cannot observe both  $Y_i(1)$  and  $Y_i(0)$ , therefore we cannot observe the

Causal Effect:  $Y_i(1) - Y_i(0)$ 

The PO that you do not (and cannot) observe are known as **COUNTERFACTUALS** because they are counter to fact (reality).

Due to the fundamental problem, we know that we can't access to ITE

#### **AVERAGE TREATMENT EFFECT (ATE)**

The ATE is obtained by taking an average over the ITEs:

$$E[Y_i(1) - Y_i(0)] = E[Y(1) - Y(0)]$$

where we recall that the average is over the individuals i if  $Y_i(x)$  is deterministic.

How would we actually compute the ATE?

i	T	Y	Y(1)	Y(o)	Y(1)—Y(0)
1	0	0	?	0	?
2	1	1	1	?	?
3	1	0	0	?	?
4	0	0	?	0	?
5	0	1	?	1	?
6	1	1	1	?	?

The fundamental problem of CI can be seen as a **MISSING DATA** 

#### **PROBLEM**

The question mark means that we do not observe the value

 $E[Y_i(1) - Y_i(0)] = ?$ 

i	T	Y	Y(1)	Y(o)	Y(1)—Y(0)
1	0	0	?	0	?
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3	1	0	0	?	?
4	0	0	?	0	?
5	0	1	?	1	?
6	1	1	1	?	?

$$E[Y_i(1) - Y_i(0)] = E[Y(1)] - E[Y(0)]$$

i	T	Y	Y(1)	Y(0)	Y(1)—Y(0)
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3	1	0	0	?	?
4	0	0	?	0	?
5	0	1	?	1	?
6	1	1	1	?	?

$$E[Y_i(1) - Y_i(0)] = E[Y(1)] - E[Y(0)] = E[Y | T = 1] - E[Y | T = 0]$$

i	T	Y	Y(1)	Y(o)	Y(1)—Y(0)
1	0	0	?	0	?
2	1	1	1	?	?
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4	0	0	?	0	?
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The fundamental problem of CI can be seen as a **MISSING DATA PROBLEM** 

2/3 1/3

$$E[Y_i(1) - Y_i(0)] = E[Y(1)] - E[Y(0)] = E[Y | T = 1] - E[Y | T = 0]$$

i	T	Y	Y(1)	Y(o)	Y(1)—Y(0)
1	0	0		0	?
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The fundamental problem of CI can be seen as a **MISSING DATA PROBLEM** 

2/3 - 1/3 = 1/3

$$E[Y_i(1) - Y_i(0)] = E[Y(1)] - E[Y(0)] \ge E[Y \mid T = 1] - E[Y \mid T = 0]$$

i	T	Y	Y(1)	Y(0)	Y(1)—Y(0)
1	0	0		0	?
2	1	1	1		?
3	1	0	0		?
4	0	0		0	?
5	0	1		1	?
6	1	1	1		?

2/3 - 1/3 = 1/3

What does it mean?

causation is not simply

association

In general, they are not equal due to **CONFOUNDING** 

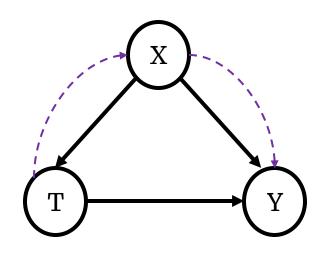
What **ASSUMPTIONS** would make the ATE equal to the associational difference?

## IGNORABILITY - $(Y(1), Y(0)) \perp T$

$$E[Y_i(1)] - E[Y_i(0)] = E[Y(1) | T = 1] - E[Y(0) | T = 0]$$
  
=  $E[Y | T = 1] - E[Y | T = 0]$ 

- We can ignore how individual ended up in the treatment/control group, and treat their PO as <a href="mailto:exchangeable">exchangeable</a>. However, it is **unrealistic** in observational data.
- ( ) Unconfoundeness

$$(Y(1), Y(0)) \perp T \mid X$$

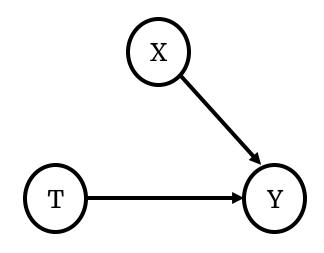


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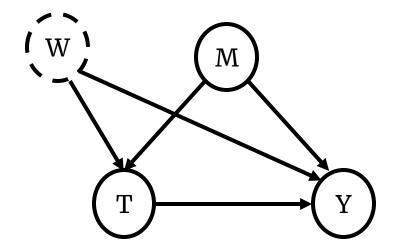
$$(Y(1), Y(0)) \perp T \mid X$$



When conditioning on **X**, **non-causal** association between *T* and **Y no longer exists**.

### **UNCONFOUNDENESS**

- While is not a problem in randomized experiments, it is an **untestable assumption** in observational data
- There may be some **unobserved confounders** that are not part of  $X = \{M\}$ , meaning unconfoundedness is <u>violated</u>.

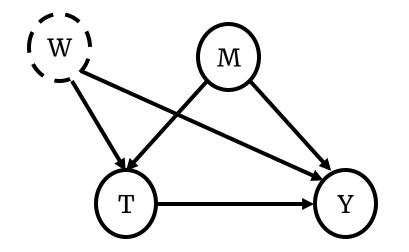


Ignorability

$$(Y(1),Y(0))\perp T\mid X$$

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Ignorability
(Y(1), Y(0)) T | X

### **POSITIVITY**

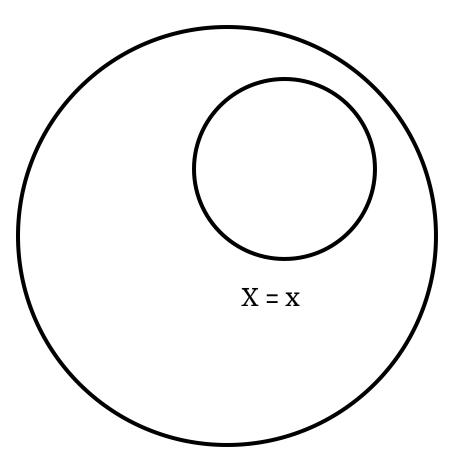
For all values x of covariates x present in the population of interest (i.e., z such that P(X = x > 0))

$$0 < P(T = 1 | X = x) < 1$$

Positivity is the condition that **all subgroups of the data** with different value x for covariates X have some probability of receiving any value of treatment T

# **POSITIVITY: INTUITION**





No one treated

$$T = 0$$

$$T = 0$$

$$T = 0$$

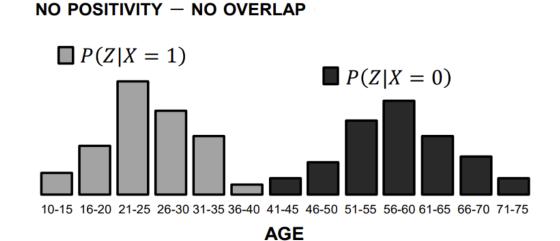
Everyone treated

$$T = 1$$

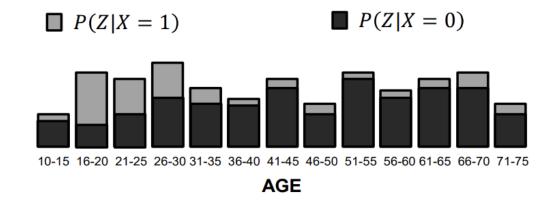
$$T = 1$$

$$T = 1$$

## **POSITIVITY: OVERLAP**



POSITIVITY — OVERLAP



No overlap means severe positivity violation

Complete overlap means no positivity violation

adjusting (conditioning) on more covariates **Z** 

could lead to

13

higher chance of satisfying unconfoundedness



could lead to

higher chance of violating positivity



demanding too much from models and getting very bad behavior in return



fit a model to  $\mathbb{E}[Y|X, \mathbf{Z}]$  using the available data  $(x, y, \mathbf{z})$ 

increase the "dimension" of the covariates **Z** 



makes the subgroups for any level **z** of the covariates **Z** smaller



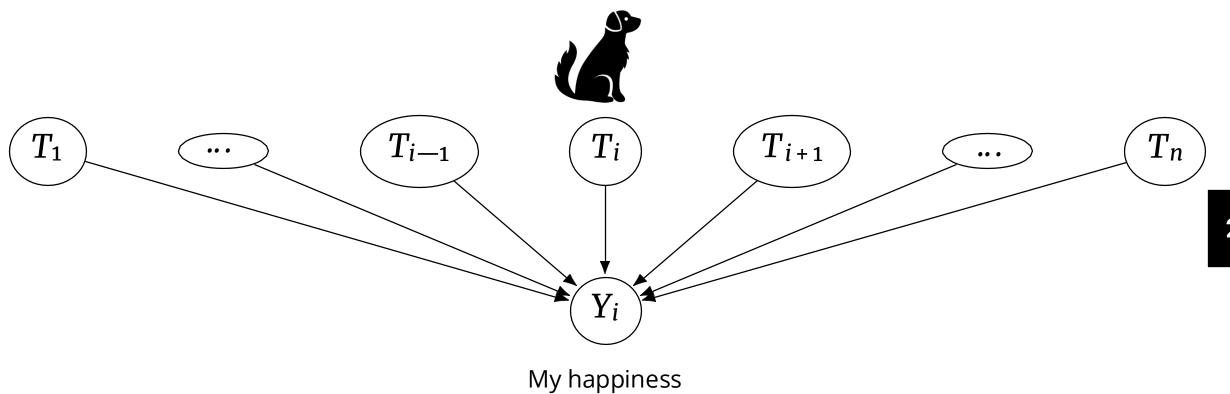
**CURSE OF DIMENSIONALITY** 

### **NO INTERFERENCE**

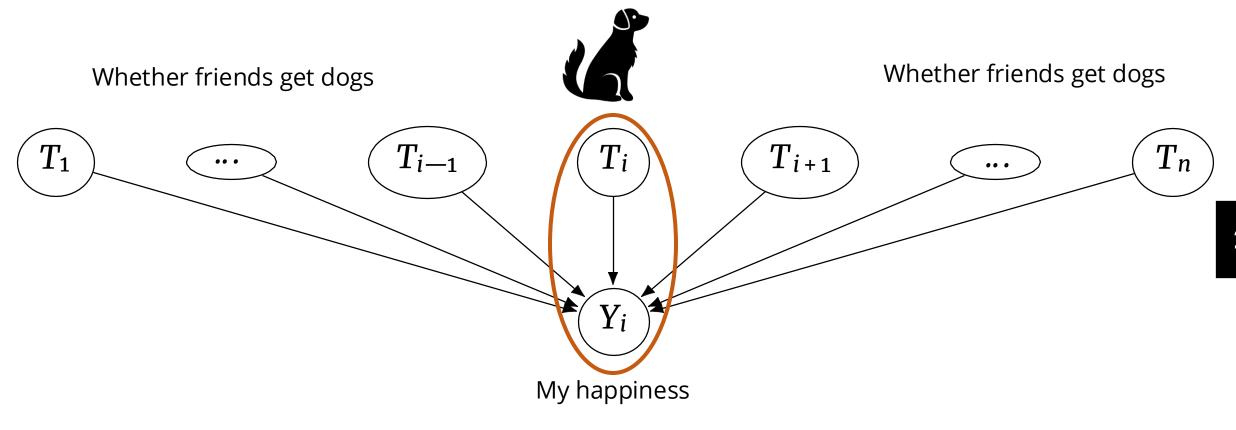
The outcome  $Y_i$  of each unit  $\boldsymbol{i}$  is unaffected by anyone else's treatment  $T_j$   $j\neq i$ 

$$Y_i(t_1, t_2, ...., t_{i-1}, t_{i+1}, ..., t_{n-1}, t_n) = Y_i(t_i)$$

## **NO INTERFERENCE**



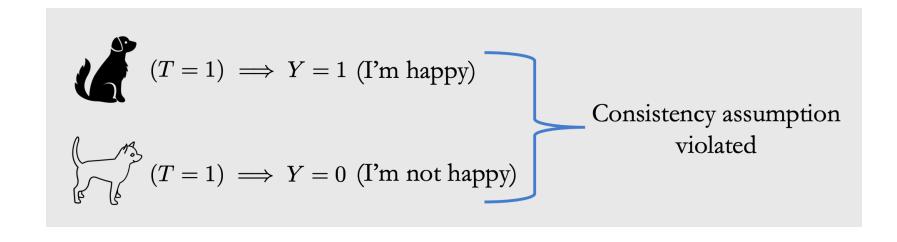
## **NO INTERFERENCE**



#### **CONSISTENCY**

If the treatment is T, then the observed outcome Y is the potential outcome under treatment X.

Formally, 
$$T = t \implies Y = Y(t)$$



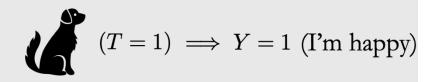
### **SUTVA**

A combination of consistency and no interference. Specifically, the PO of a unit **do not** 

**depend** on the treatments assigned to others.

But in real world ...



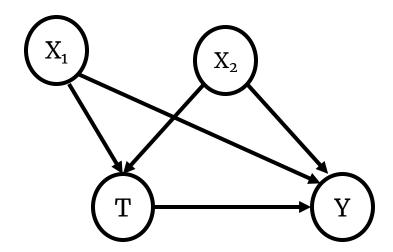




#### **HOW TO USE THE PO: AN EXAMPLE**

#### **PROPENSITY SCORE MATCHING (PSM)**

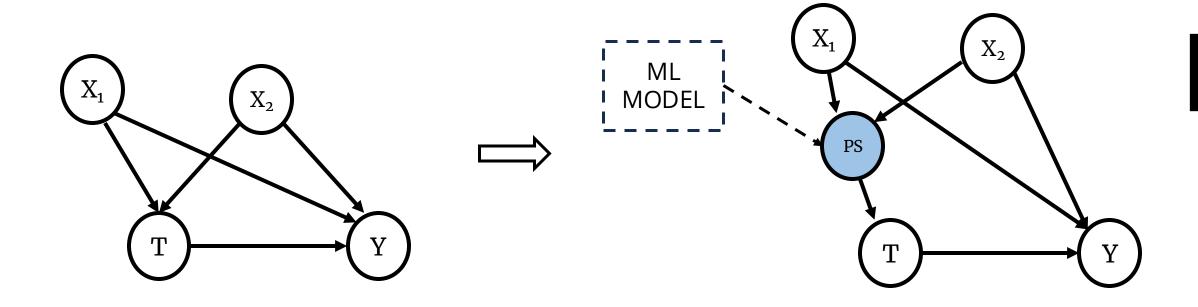
It match T=0 and T=1 observations on the estimated probability of being treated.



### **HOW TO USE THE PO: AN EXAMPLE**

#### **PROPENSITY SCORE MATCHING (PSM)**

It match T=0 and T=1 observations on the estimated probability of being treated.



#### **PO RECAP**

- Mainly used for estimating average effects of binary treatments
- Convincing empirical applications

#### LIMITATIONS:

- An expert of the field should verify whether all the previous assumptions are valid.

  It is challenging and you need some people working on it.
- No use of causal diagrams

### **CAUSAL MODEL FRAMEWORKS**

Potential Outcomes (PO)

Structural Causal Model (SCM)

Antecedents in the earlier econometric literature

Demand and Supply Models (Haavelmo, 1944)

Path analysis (Wright, 1934)

Specifically, to deal with:

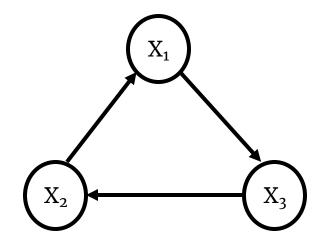
Estimating individual-level causal effects

Complex models with a large number of variables

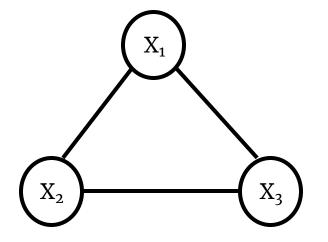
# STRUCTURAL CAUSAL MODEL

Mathematically, a Structural Causal Model (SCM) consists of **a set of Endogenous (V)** and a set of **Exogenous (U)** variables connected by **a set of functions (F)** that determine the values of the the variables in V based on the values of the variables in U.

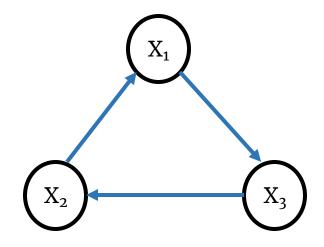
Each SCM is associated with a **graphical model** where **each node** is a **variable in V** and each edge is a **function f**.



**Directed Graph** 

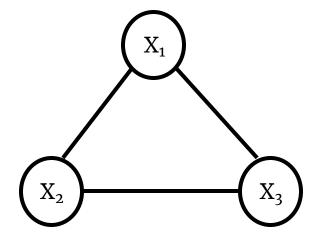


**Undirected Graph** 

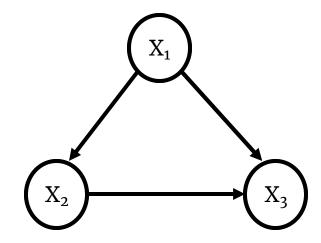


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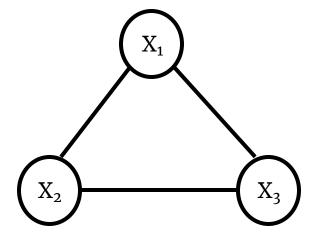
This graph contains a cycle



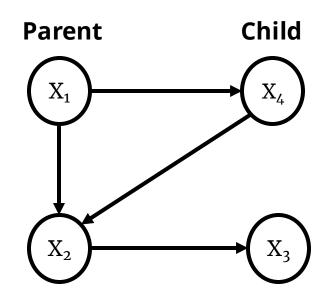
**Undirected Graph** 

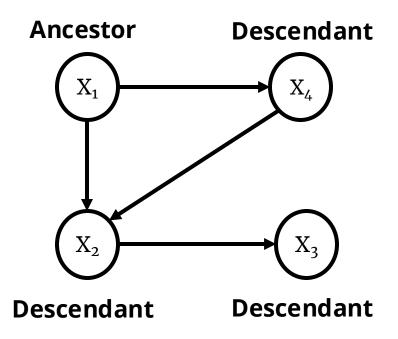


**Directed Acyclic Graph** 

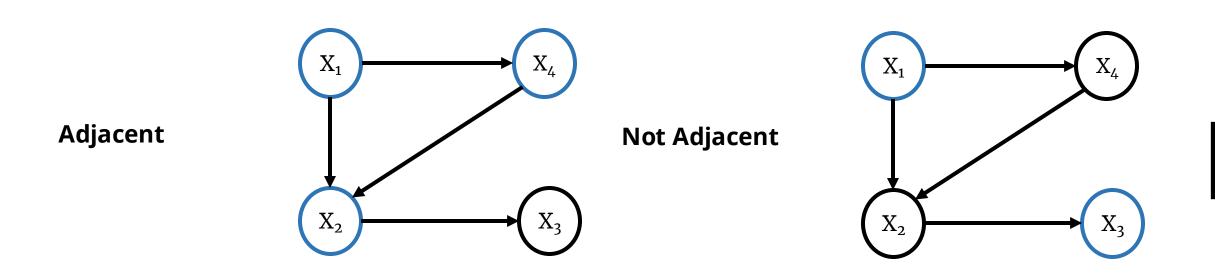


**Undirected Graph** 

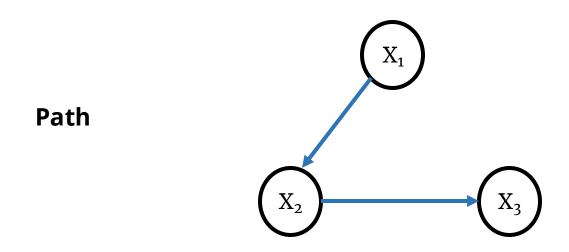




Descendant is a **broader** term than child because it includes **not only the immediate children** but also **their children and so forth** 



Ajdacent is a node that is directly connected to another node within a graph



A path is a sequence of nodes where each node is connected to the next node by an edge

## STRUCTURAL CAUSAL MODEL: EXAMPLE

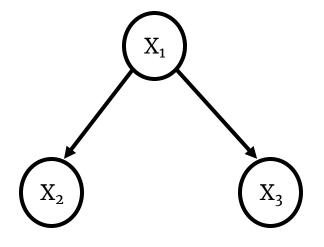
$$X = \{X_1, X_2, X_3\}$$

 $X_1 := Uniform(0, 1)$ 

 $X_2 := \sin(X_1) + \text{Normal}(0, 1)$ 

 $X_3 := 2 * X_1 + Normal(0, 1)$ 

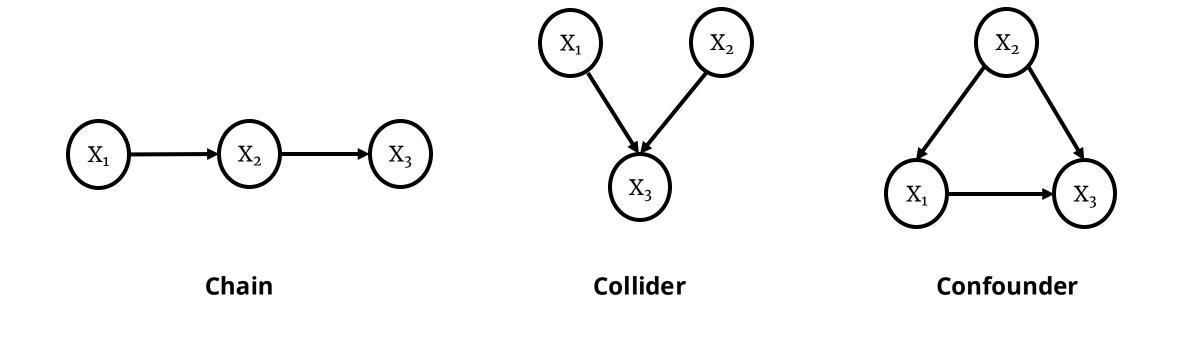
Structural Equation (SE)



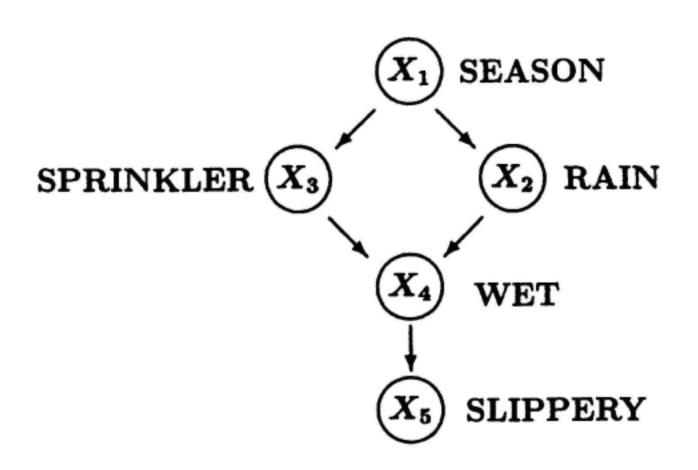
Directed Acyclic Graph (DAG)

#### **37**

# CAUSAL STRUCTURES

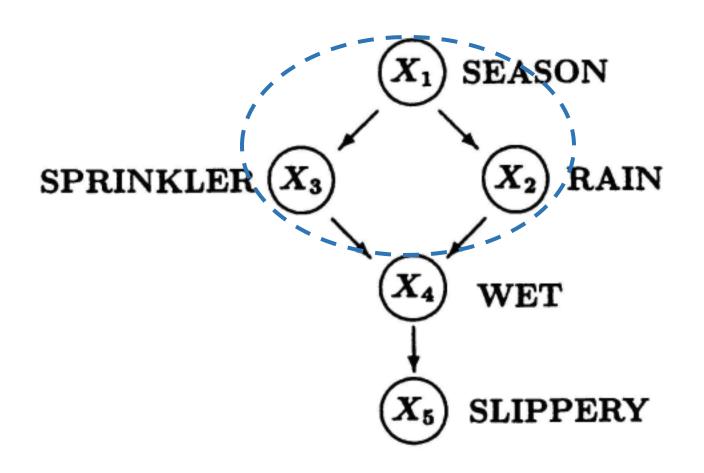


## CAUSAL STRUCTURES: EXAMPLE



#### 38

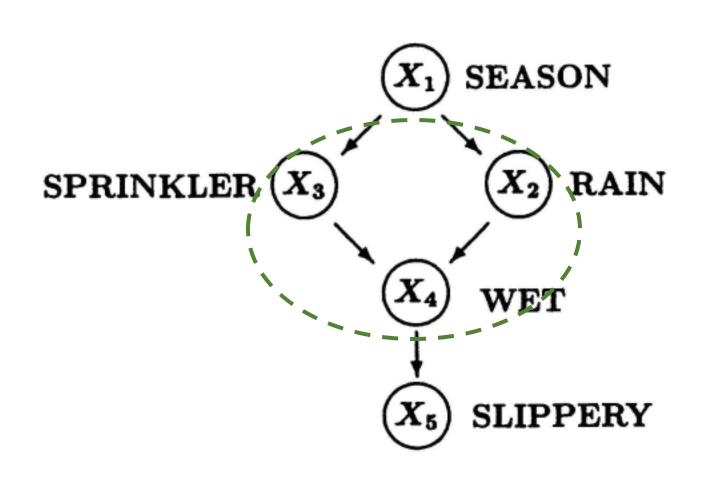
## **CAUSAL STRUCTURES: EXAMPLE**



Confounder

#### 38

## **CAUSAL STRUCTURES: EXAMPLE**



Collider

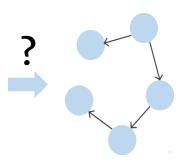
### **LEVELS OF INVESTIGATION**

Causal Discovery (CD)

Given a set of variables, is it possible to **determine the**causal relationship

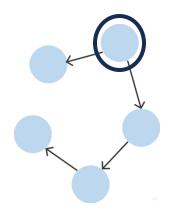
between them?

A	В	С	D	E
3.2	2.2	1.6	7.5	2.4
2.9	3.1	1.3	8.2	5.1

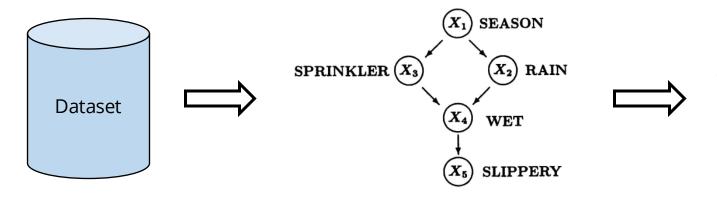


Causal Inference (CI)

If we manipulate
the value of one variable,
how much would
the others change?



### **CAUSAL PIPELINE**



**Causal Discovery** 

What are the consequences of turning on the sprinkler?

(The floor gets wet)

Causal Inference

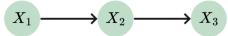
#### 40

## **CAUSAL DISCOVERY: METHODS**

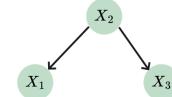
Constraintbased Scorebased

Markov Equivalence Class

$$X_1 \!\perp\!\!\!\perp X_3 \!\mid\! X_2$$
 and  $X_1 \!\not\!\perp\!\!\!\perp X_3$ 



$$X_1 \longleftarrow X_2 \longleftarrow X_3$$



#### 40

# **CAUSAL DISCOVERY: METHODS**

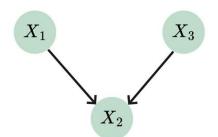
Constraintbased



Markov Equivalence Class

$$X_1 \perp \!\!\! \perp X_3 \mid X_2 \text{ and } X_1 \perp \!\!\! \perp X_3$$
 $X_1 \longrightarrow X_2 \longrightarrow X_3$ 
 $X_1 \longleftarrow X_2 \longleftarrow X_3$ 
 $X_1 \longleftarrow X_2 \longleftarrow X_3$ 

V-structure



# **CAUSAL DISCOVERY: METHODS**

Constraintbased

Scorebased Markov Equivalence Class

$$X_1 \perp \!\!\! \perp X_3 \mid X_2 \text{ and } X_1 \not \perp \!\!\! \perp X_3$$
 $X_1 \longrightarrow X_2 \longrightarrow X_3$ 

$$X_1 \longleftarrow X_2 \longleftarrow X_3$$

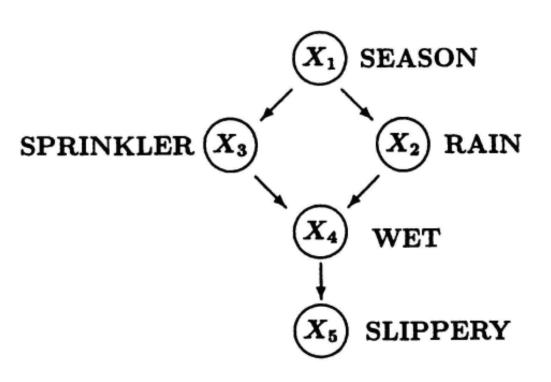


Functional Causal Models



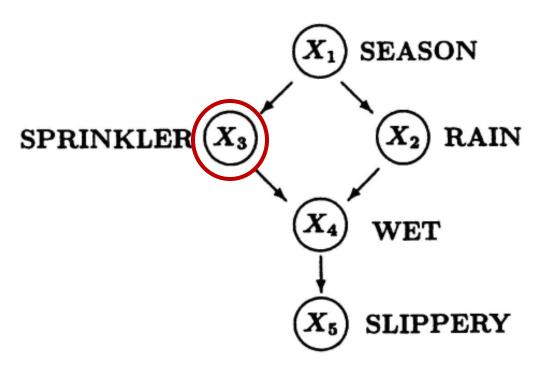
- Strong assumptions but they can uniquely identify the true DAG
- Linear and non-Gaussian, Additive noise, Post-nonlinear

#### **INTERVENTION**



Interpreting edges as cause-effect relationships enable reasoning about the outcome of interventions using the do-operator

### **INTERVENTION**

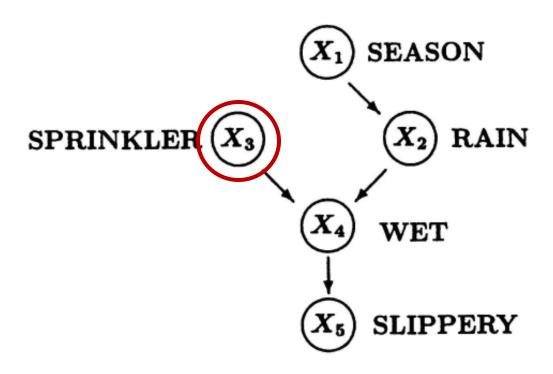


The notation do(Sprinkler := ON) denotes an intervention by which variable Sprinkler is set to value ON.

Externally forcing the variable to assume a particular value makes it **independent of its** causes and breaks their causal influence on it.

#### **INTERVENTION**

Interventional Data



Graphically, the effect of an intervention can be captured by **removing all incoming edges to the intervened variable**.

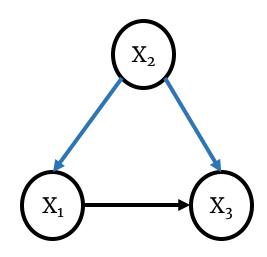
#### **BACK-DOOR CRITERION**

The best-known technique to find causal estimands given a graph.

A set of variables  $\mathbf{Z}$  satisfies the **back-door criterion** relative to an ordered pair of variables  $(X_i, X_j)$  in a DAG  $\mathbf{G}$  if:

- $\bigcirc$  no node in **Z** is a descendant of  $X_i$
- $\bigodot$  **Z** blocks every path between  $X_i$  and  $X_j$  that contains an arrow into  $X_i$ .

#### **BACK-DOOR CRITERION: EXAMPLE**



Backdoor path

$$X_1 < -X_2 -> X_3$$

This path is **not causal**.

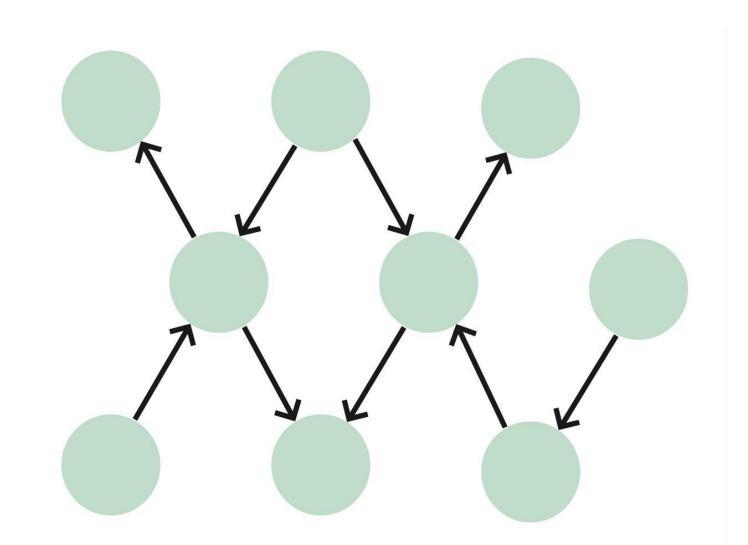
It is a process that creates **spurious correlations** between  $X_1$  and  $X_3$  that are driven solely by fluctuations in the  $X_2$  random variable.

If we can **close all of the open backdoor paths**, then we can isolate the causal effect of  $X_1$  and  $X_3$  using an identification strategy.

$$P(X_3 \mid do(X_1) = \sum_{X_2} P(X_3 \mid X_1, X_2) P(X_2)$$

# **EXERCISE**

Find the discovered graph



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# THANK FOR YOUR ATTENTION