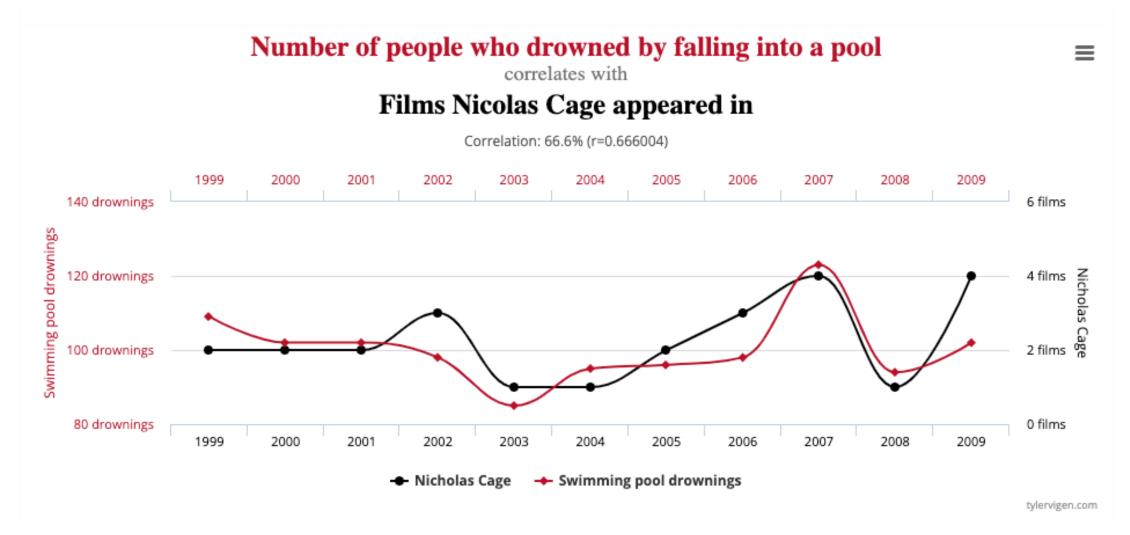
07 Maggio 2024

INTRODUCTION TO

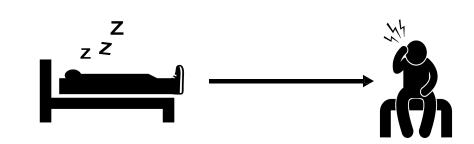
CAUSAL MODELLING AND REASONING

Martina Cinquini & Isacco Beretta





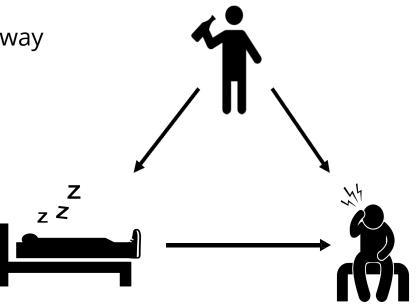
Sleeping with shoes on is strongly correlated with waking up with a headache



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

Common cause: drinking the night before

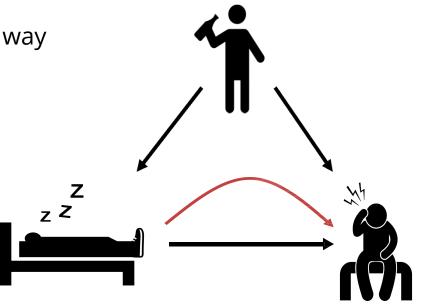
1. Shoe-sleepers differ from non-shoe-sleepers in a key way



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

Common cause: drinking the night before

Shoe-sleepers differ from non-shoe-sleepers in a key way
Confounding



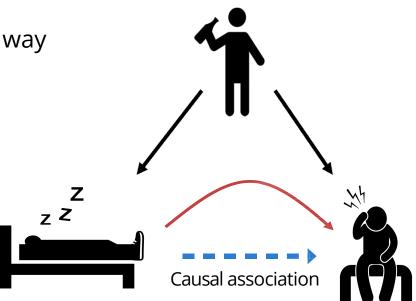
Sleeping with shoes on is strongly correlated with waking up with a headache

Common cause: drinking the night before

Shoe-sleepers differ from non-shoe-sleepers in a key way
Confounding

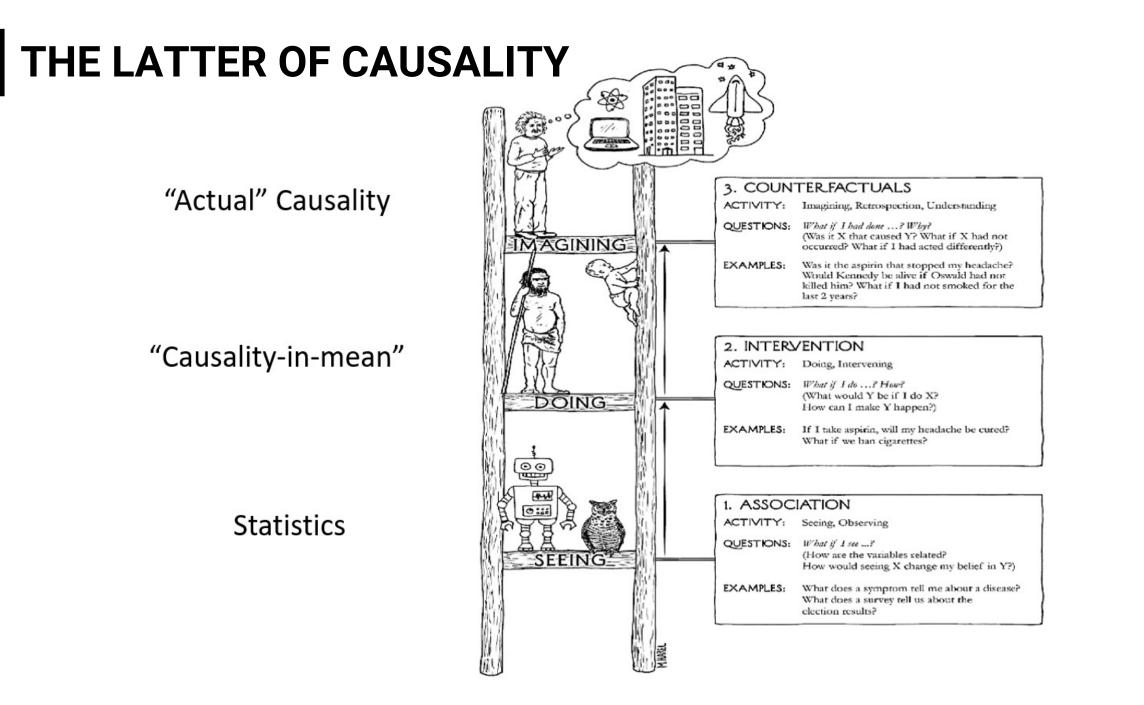
Total association (e.g., correlation):

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
- Θ
- Method for reasoning over model and data



RANDOMIZED EXPERIMENTS



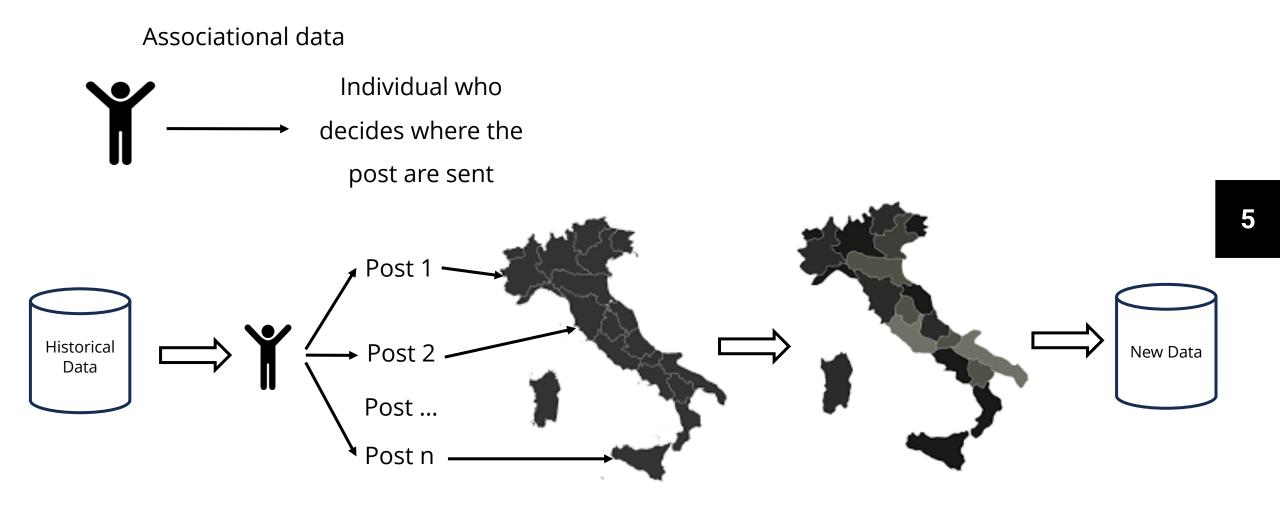
Limitations



Can not use **historical** data

It cannot be applied to certain situations (e.g., long-term effect, selected demographics, content virality)

BEYOND RANDOMIZED EXPERIMENTS



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

Inferring the effect of treatment on some outcome

Take a pill





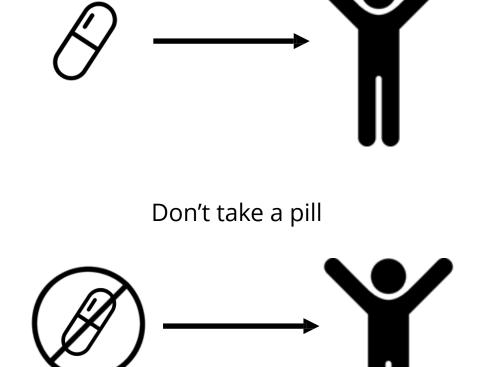
Causal Effect?

POTENTIAL OUTCOME: INTUITION

Inferring the effect of treatment on some outcome

Take a pill

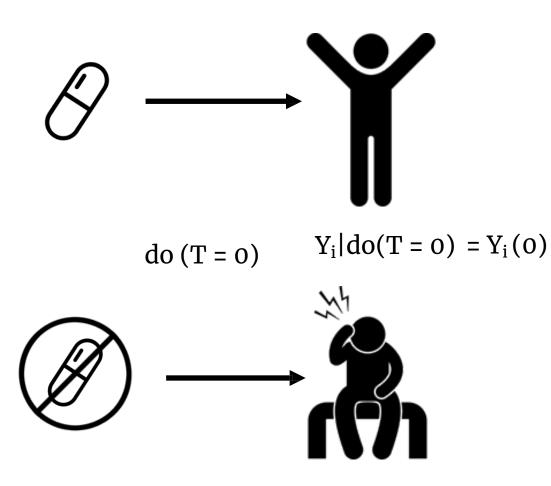


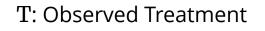


No Causal Effect

POTENTIAL OUTCOME: NOTATION

do (T = 1) $Y_i | do(T = 1) = Y_i(1)$





Y: Observed Outcome

i: used in subscript to denote a specific individual

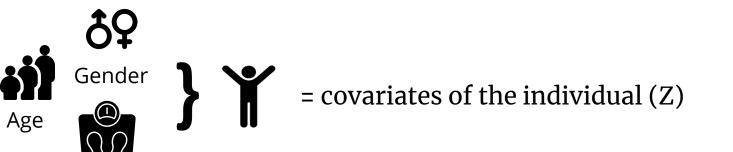
 $Y_i(1)$: PO under treatment

 $Y_i(0)$: PO under no treatment

OTHER DEFINITIONS

= unit (individual)

= population



INDIVIDUAL TREATMENT EFFECT (ITE)

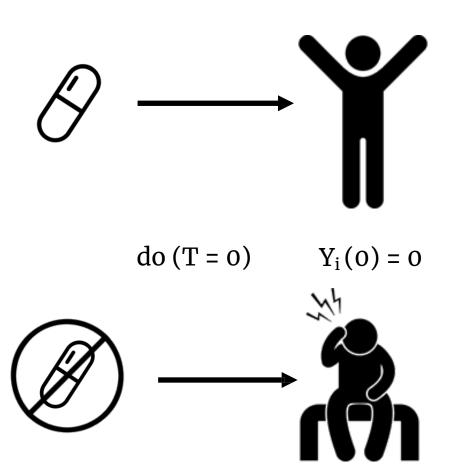
The ITE for the i^{th} unit is defined as follows:

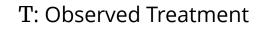
 $Y_i(1) - Y_i(0)$

Weight

POTENTIAL OUTCOME: NOTATION

do (T = 1) $Y_i(1) = 1$





Y: Observed Outcome

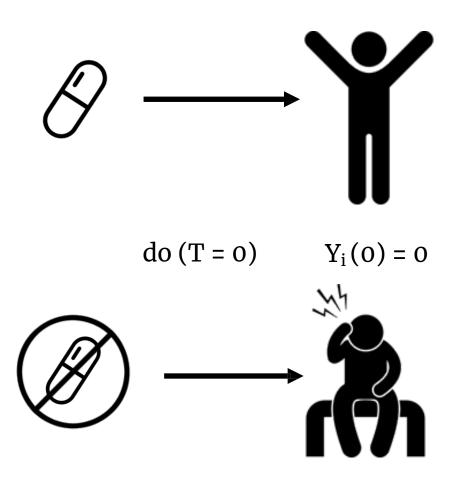
i: used in subscript to denote a specific individual

 $Y_i(1)$: PO under treatment

 $Y_i(0)$: PO under no treatment

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

do (T = 1)
$$Y_i(1) = 1$$



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

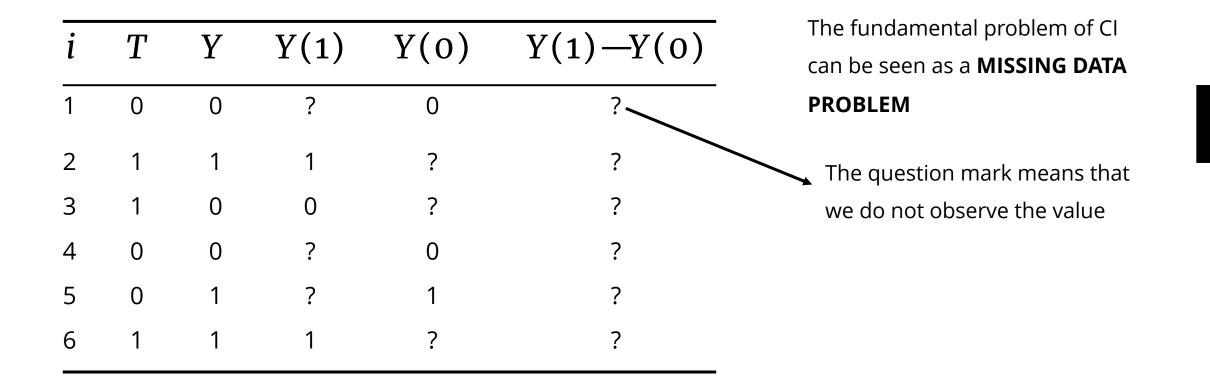
AVERAGE TREATMENT EFFECT (ATE)

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?



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

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	?	?

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

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	?	?

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

i	T	Y	Y(1)	Y(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	?	?

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

i	Т	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		?

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

i	T	Y	Y(1)	Y(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		?

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(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					?

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

2/3 - 1/3 = 1/3

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

i	Т	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 simply association

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

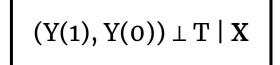
What **ASSUMPTIONS** would make the ATE equal to the associational difference?

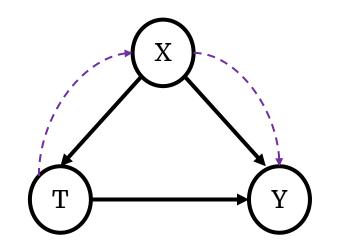
IGNORABILITY - (Y(1), Y(0)) \perp **T**

$E[Y_i(1)] - E[Y_i(0)] = E[Y(1) | T = 1] - E[Y(0) | T = 0]$ = 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 <u>exchangeable</u>. However, it is **unrealistic** in observational data.

Unconfoundeness





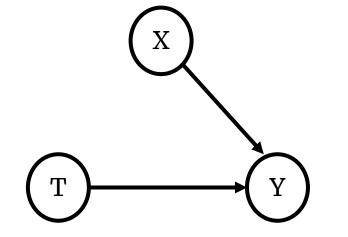
IGNORABILITY - (Y(1), Y(0)) \perp **T**

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We can ignore how individual ended up in the treatment/control group, and treat their PO as <u>exchangeable</u>. However, it is **unrealistic** in observational data.

Unconfoundeness

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

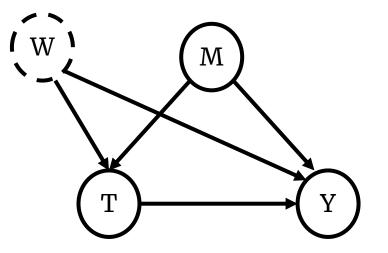


When conditioning on **X**, **noncausal** association between *T* and **Y no longer exists**.

UNCONFOUNDENESS

O While is not a problem in randomized experiments, it is an **untestable assumption** in observational data

 \bigcirc There may be some **unobserved confounders** that are not part of X = {M}, meaning unconfoundedness is <u>violated</u>.

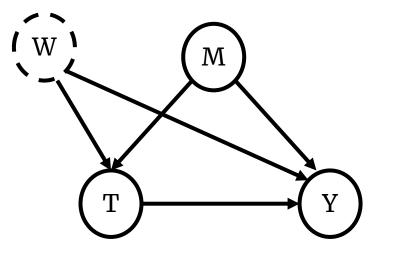


Ignorability $(Y(1), Y(0)) \perp T \mid X$

UNCONFOUNDENESS

O While is not a problem in randomized experiments, it is an **untestable assumption** in observational data

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

ASIDE: IDENTIFIABILITY

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

$$= E[Y | T = 1] - E[Y|T = 0]$$

Causal quantities

Statistical quantities

A causal quantity (e.g. E[Y(t)]) is **identifiable** if we can compute it from a purely statistical quantity (e.g. E[Y | t))

POSITIVITY

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

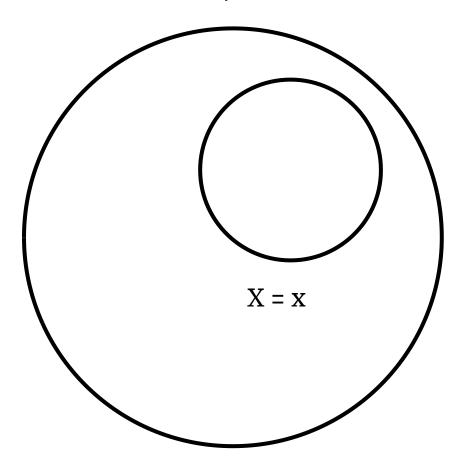
0 < P(T = 1 | X = x) < 1

 $\overline{\mathbf{\Theta}}$

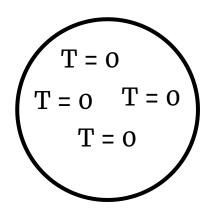
Positivity is the condition that **all subgroups of the data** with different value x for covariates X have some probability of receiving any value of treatment T

POSITIVITY: INTUITION

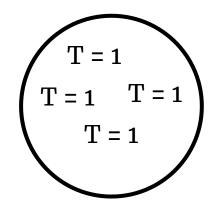
Total Population



No one treated

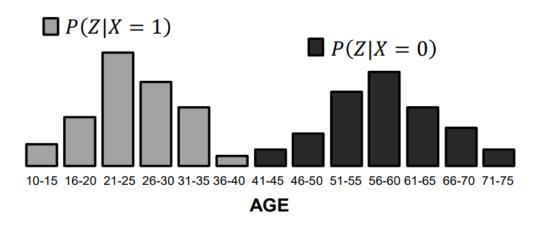


Everyone treated

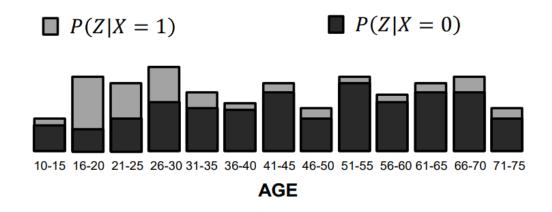


POSITIVITY: OVERLAP

NO POSITIVITY - NO OVERLAP

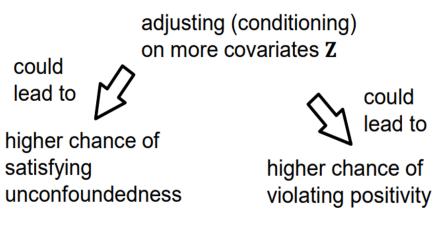


POSITIVITY - OVERLAP



No overlap means severe positivity violation

Complete overlap means no positivity violation



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**

 Λ

makes the subgroups for any level z of the covariates \mathbf{Z} smaller

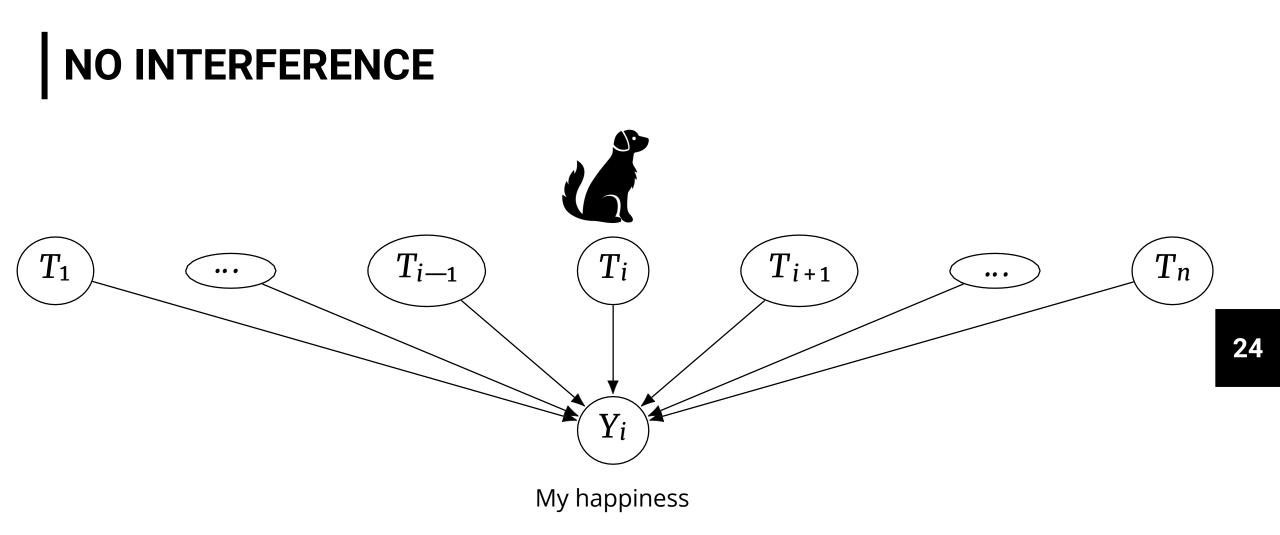
 \mathcal{P}

CURSE OF DIMENSIONALITY

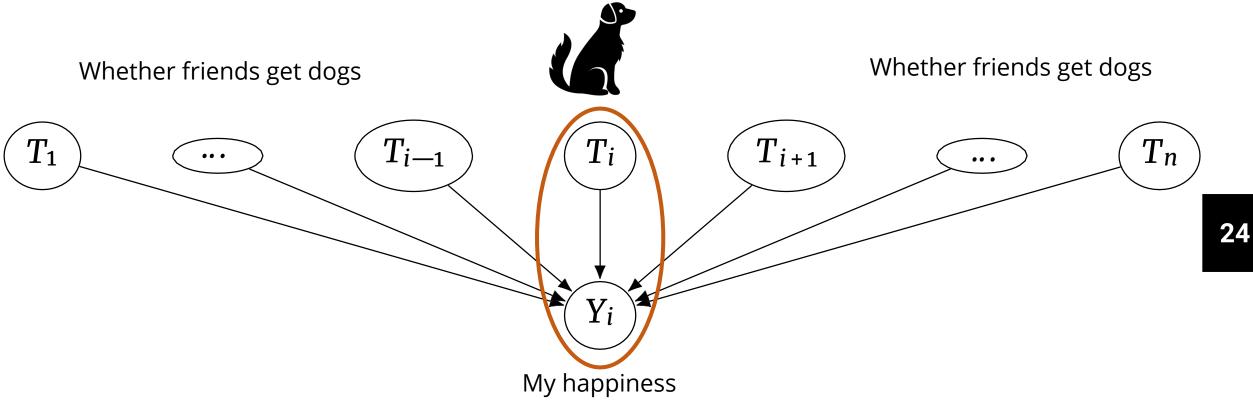
NO INTERFERENCE

The outcome Y_i of each unit 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





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

Formally, T = t $\Box > Y = Y(t)$

$$(T = 1) \implies Y = 1 \text{ (I'm happy)}$$

Consistency assumption
violated

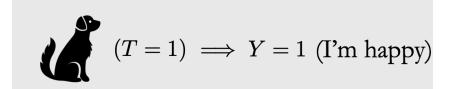


A combination of consistency and no interference. Specifically, the PO of a unit **do not**

depend on the treatments assigned to others.

But in real world ...



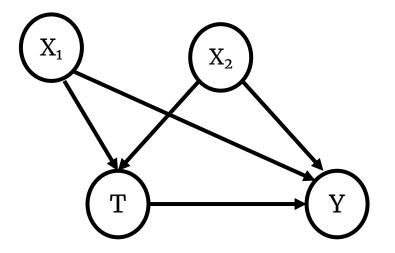


<image>

HOW TO USE THE PO: AN EXAMPLE

PROPENSITY SCORE MATCHING (PSM)

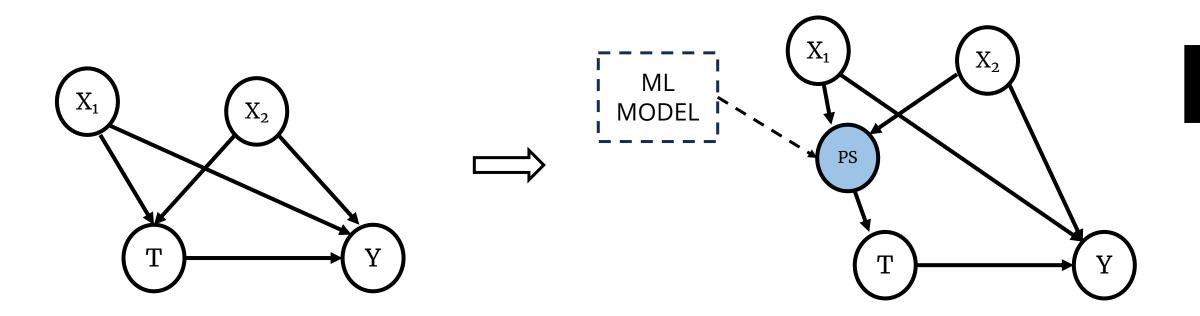
It match T=0 and T=1 observations on the estimated probability of being treated.



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.





€

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)

29

Specifically, to deal with:

Estimating individual-level causal effects

Complex models with a large number of variables

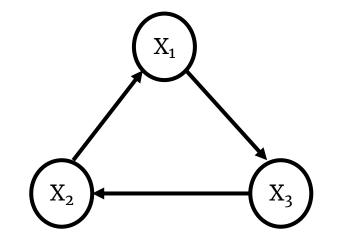
STRUCTURAL CAUSAL MODEL

Mathematically, a Structural Causal Model (SCM) consists of a set of Endogenous (V) and

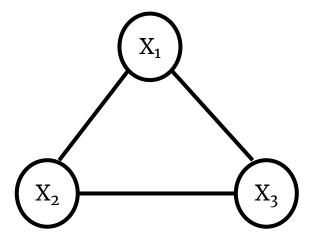
a set of **Exogenous (U)** variables connected by **a set of functions (F)** that determine the

values of the the variables in V based on the values of the variables in U.

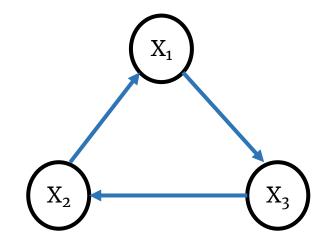
Each SCM is associated with a **graphical model** where **each node** is a **variable in V** and each edge is a **function f**.



Directed Graph

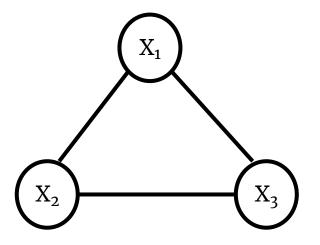


Undirected Graph

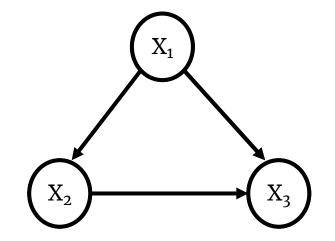




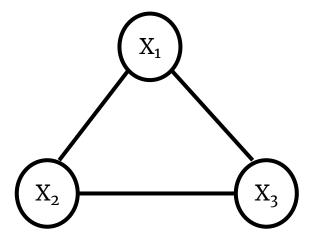
This graph contains a cycle



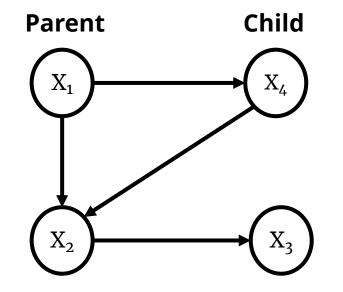
Undirected Graph

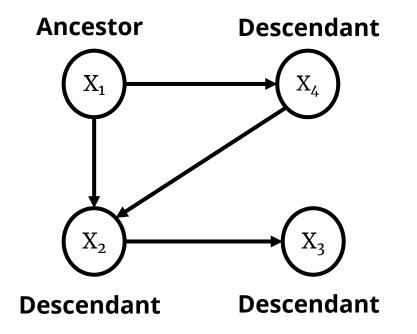


Directed Acyclic Graph

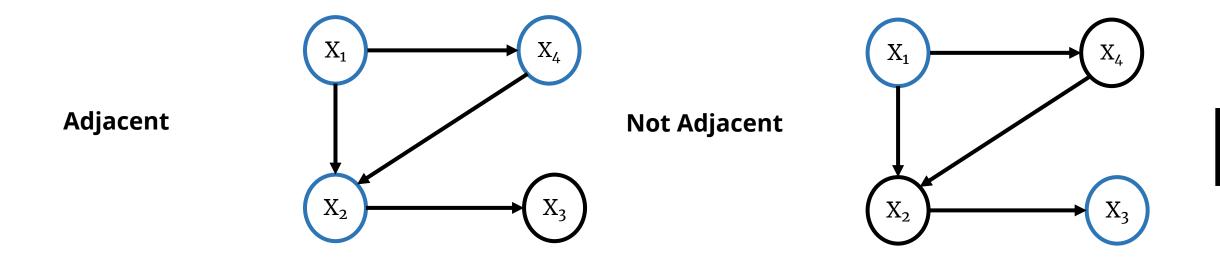


Undirected Graph

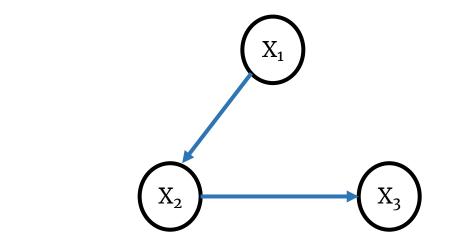




Descendant is a **broader** term than child because it includes **not only the immediate children** but also **their children and so forth**



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



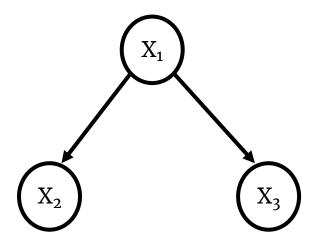
Path

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

STRUCTURAL CAUSAL MODEL: EXAMPLE

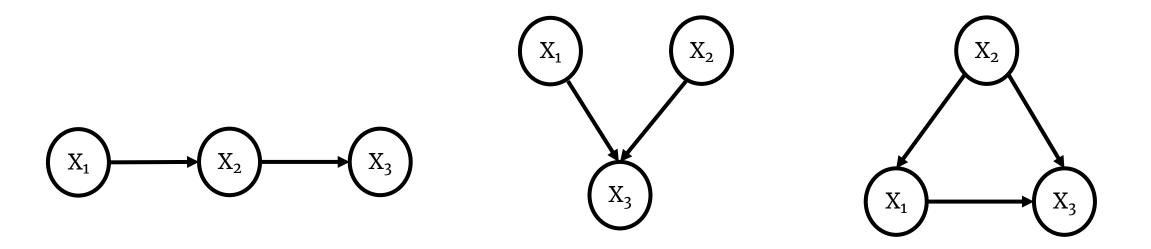
 $X = \{X_1, X_2, X_3\}$

- $X_1 := \text{Uniform}(0, 1)$
- $X_2 := sin(X_1) + Normal(0, 1)$
- $X_3 := 2 * X_1 + Normal(0, 1)$
 - Structural Equation (SE)



Directed Acyclic Graph (DAG)

CAUSAL STRUCTURES

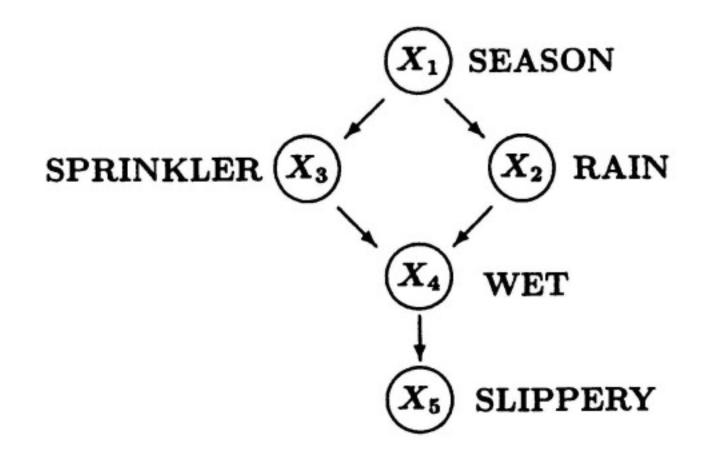


Chain

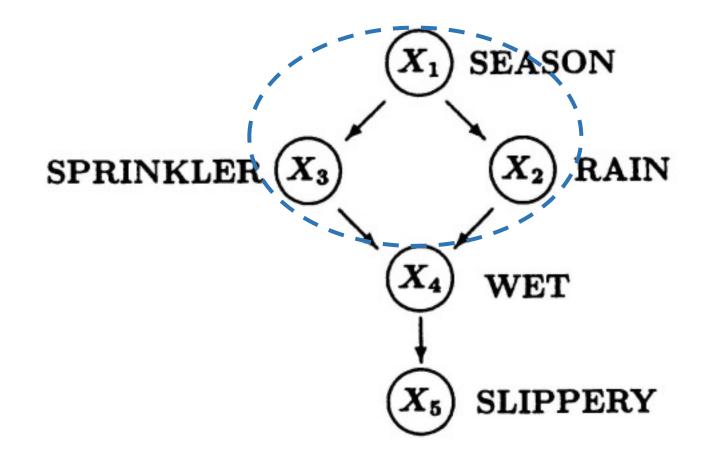
Collider

Confounder

CAUSAL STRUCTURES: EXAMPLE

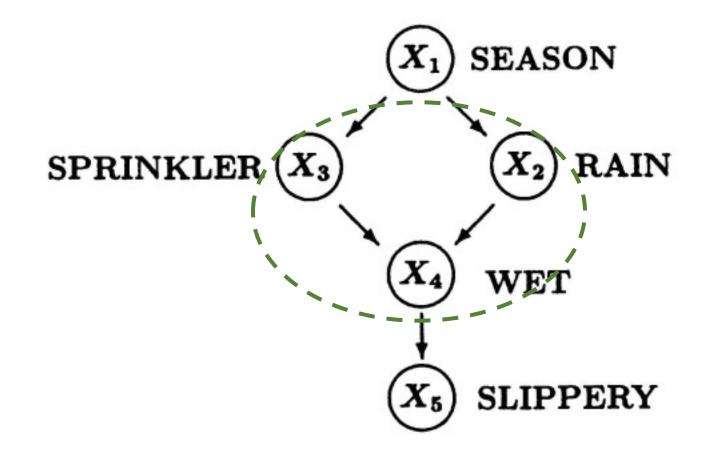


CAUSAL STRUCTURES: EXAMPLE



Confounder

CAUSAL STRUCTURES: EXAMPLE





LEVELS OF INVESTIGATION

Causal Discovery (CD)

Given a set of variables,

is it possible to **determine the**

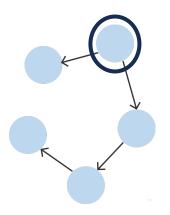
causal relationship

between them?

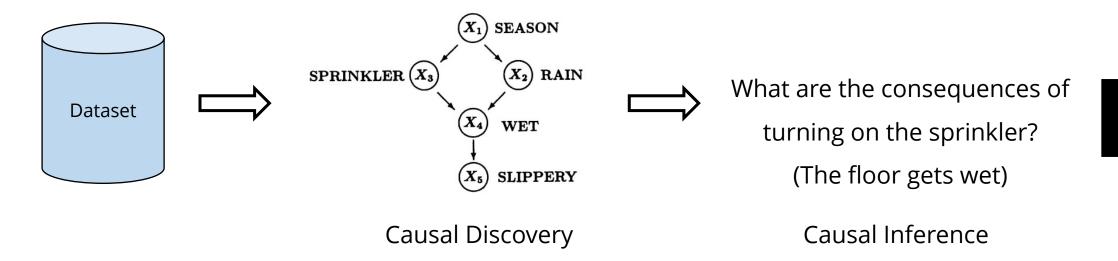
А	В	С	D	Е	2
3.2	2.2	1.6	7.5	2.4	
2.9	3.1	1.3	8.2	5.1	
		-			

Causal Inference (CI)

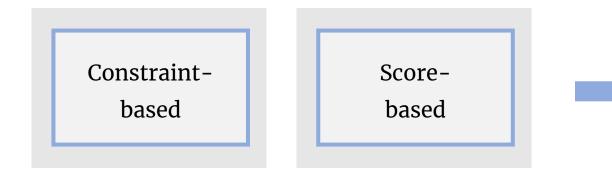
If we manipulate the value of one variable, **how much would the others change**?



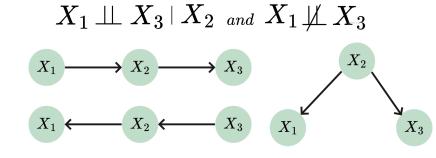
CAUSAL PIPELINE



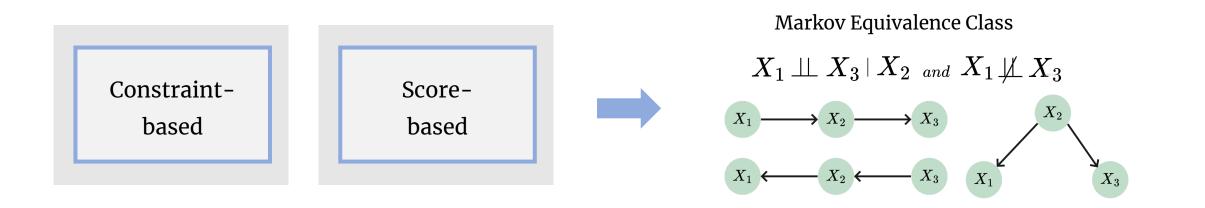
CAUSAL DISCOVERY: METHODS

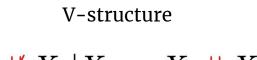


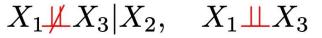
Markov Equivalence Class

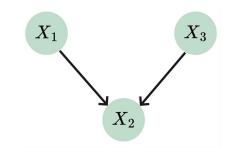


CAUSAL DISCOVERY: METHODS

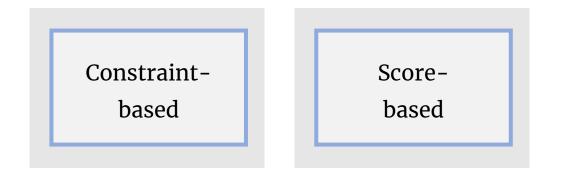




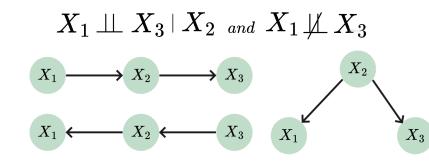




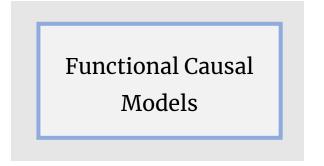
CAUSAL DISCOVERY: METHODS



Markov Equivalence Class



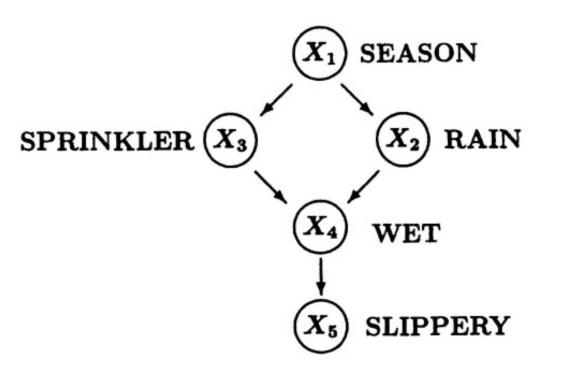






- Strong assumptions but they can uniquely identify the true DAG
- Linear and non-Gaussian, Additive noise, Post-nonlinear

INTERVENTION

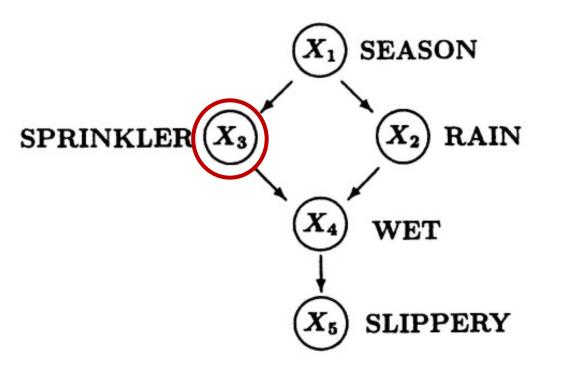


Interpreting edges as cause-effect relationships

enable reasoning about the outcome of

interventions using the do-operator

INTERVENTION

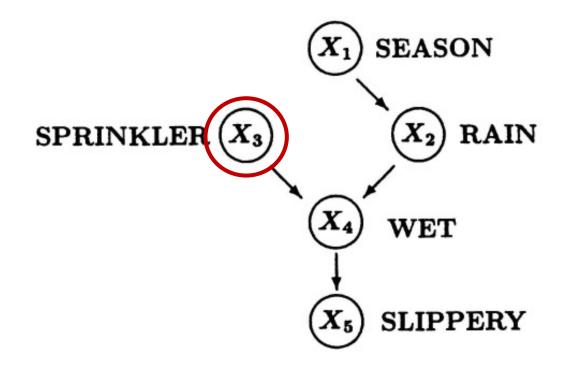


The notation do(Sprinkler := ON) denotes an intervention by which variable Sprinkler is set to value ON.

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

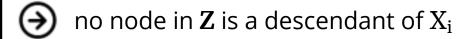


Graphically, the effect of an intervention can be captured by **removing all incoming edges to the intervened variable**.

BACK-DOOR CRITERION

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

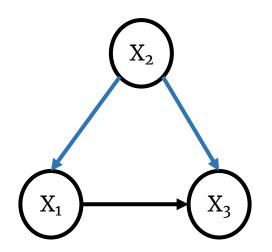
A set of variables **Z** satisfies the **back-door criterion** relative to an ordered pair of variables (X_i, X_j) in a DAG G if:





 \mathbf{Z} blocks every path between X_i and X_j that contains an arrow into X_i .

BACK-DOOR CRITERION: EXAMPLE



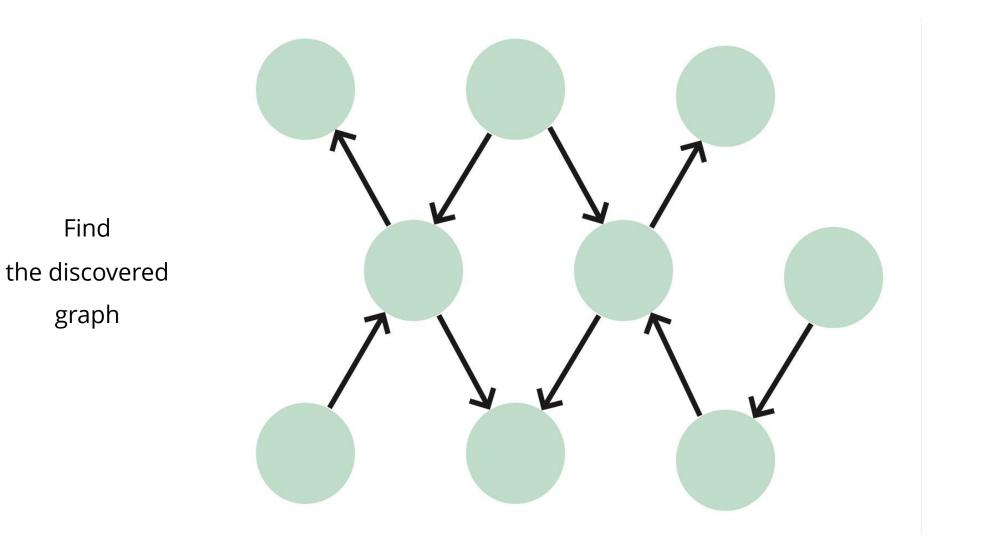
Backdoor path $X_1 < -X_2 \rightarrow X_3$

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.

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

EXERCISE





Pearl, Judea, and Dana Mackenzie. The book of why: the new science of cause and effect. Basic books, 2018.

Imbens, Guido W. "Potential outcome and directed acyclic graph approaches to causality: Relevance for empirical practice in economics." *Journal of Economic Literature* 58.4 (2020): 1129-1179.

Nogueira, Ana Rita, et al. "Methods and tools for causal discovery and causal inference." *Wiley interdisciplinary reviews: data mining and knowledge discovery* 12.2 (2022): e1449.

Pearl, Judea, Madelyn Glymour, and Nicholas P. Jewell. *Causal inference in statistics: A primer*. John Wiley & Sons, 2016.

https://www.bradyneal.com/causal-inference-course

THANK FOR YOUR ATTENTION