

Machine Learning for econometrics

Reminders of potential outcomes and Directed Acyclic Graphs

Matthieu Doutreligne

January 10, 2025

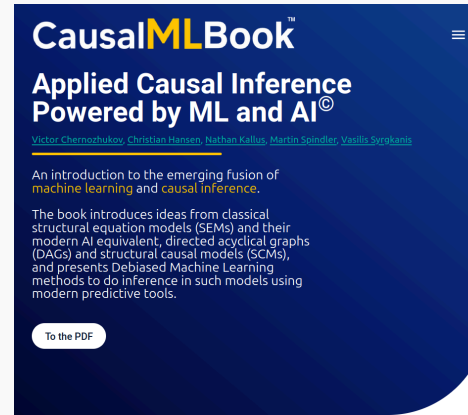
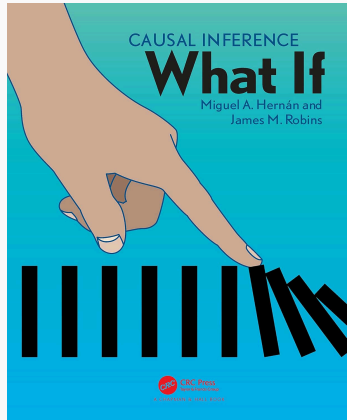
Thanks to Judith Abecassis for the slides on DAGs

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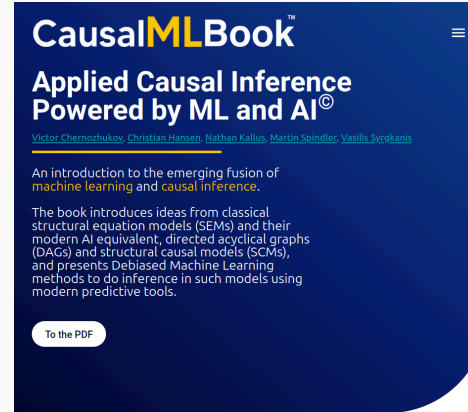
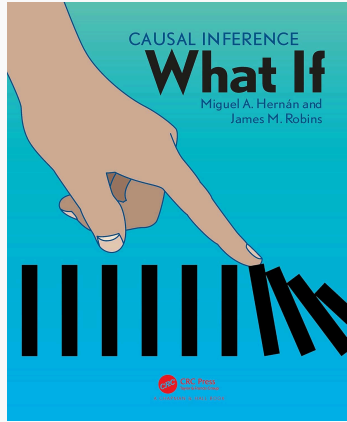
Introduction

Causal inference: subfield of statistics dealing with "why questions"



At the center of epidemiology (Hernan & Robins, 2020), econometrics (Chernozhukov et al., 2024), social sciences,

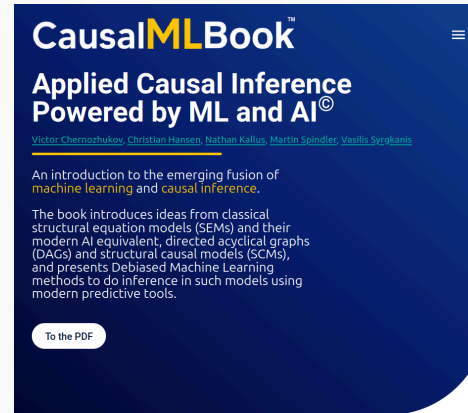
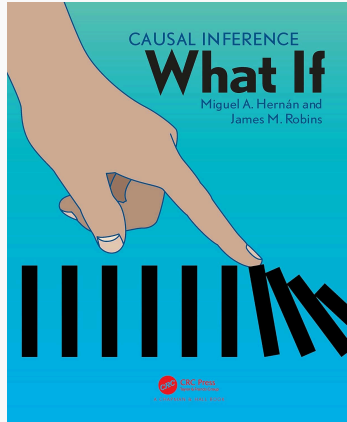
Causal inference: subfield of statistics dealing with "why questions"



At the center of epidemiology (Hernan & Robins, 2020), econometrics (Chernozhukov et al., 2024), social sciences, machine learning...

Now, bridging with machine learning (Kaddour et al., 2022) : Fairness, reinforcement learning, causal discovery, causal inference for LLM, causal representations...

Causal inference: subfield of statistics dealing with "why questions"



At the center of epidemiology (Hernan & Robins, 2020), econometrics (Chernozhukov et al., 2024), social sciences,

This session:

- Reminder on causal inference
- Importance of Directed Acyclic Graphs and causal status of covariates

What is a "why question"?

- Economics: How does supply and demand (causally) depend on price?
- Policy: Are job training programmes actually effective?
- Epidemiology: How does this treatment affect the patient's health?
- Public health : Is this prevention campaign effective?
- Psychology: What is the effect of family structure on children's outcome?
- Sociology: What is the effect of social media on political opinions?

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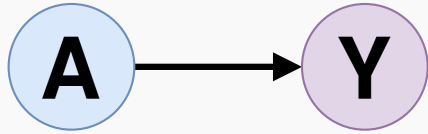
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This is different from predictive questions

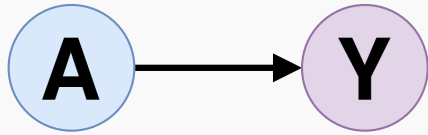
Prediction (ML): What usually happens in a given situation?



Prediction models (X, Y)

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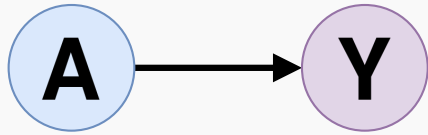


Prediction models (X, Y)

- What will be the weather tomorrow?
- What will be the outcome of the next election?
- How many people will get infected by flue next season?
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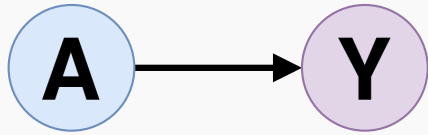


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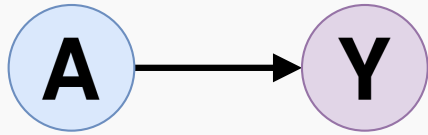


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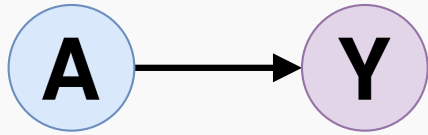


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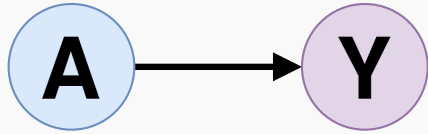


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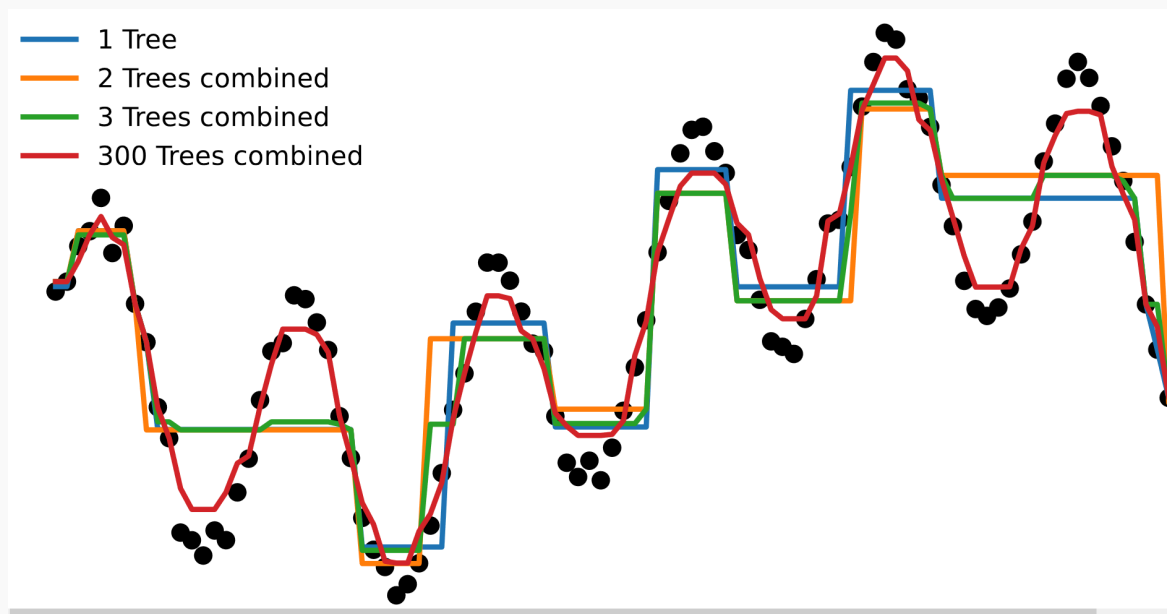
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Assumption Train and test data are drawn from the same distribution.

Machine learning is pattern matching

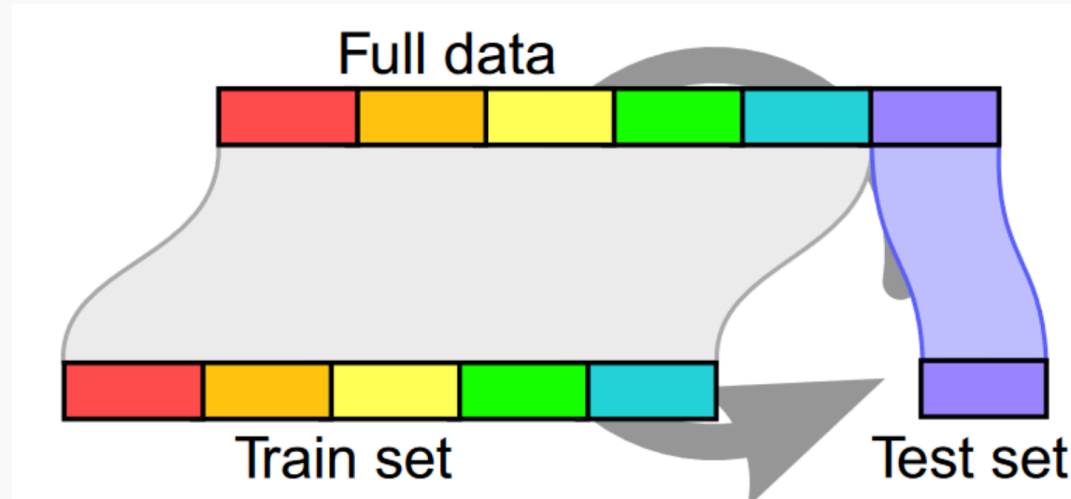
Find an estimator $f : x \rightarrow y$ that approximates the true value of y so that $f(x) \approx y$



Boosted trees : iterative ensemble of decision trees

Machine learning is pattern matching that generalizes to new data

Select models based on their ability to generalize to new data : (train, test) splits and cross validation (Stone, 1974).



“Cross validation” (Varoquaux et al., 2017)

Machine learning is great for prediction on complex data

- Images: Image classification with deep convolutional neural networks (Krizhevsky et al., 2012)



ImageNet 1K: 1.5 million images, 1000 classes

Machine learning is great for prediction on complex data

- Images: Image classification with deep convolutional neural networks (Krizhevsky et al., 2012)
- Speech-to-text: Towards end-to-end speech recognition with recurrent neural networks (Graves & Jaitly, 2014)
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- Images: Image classification with deep convolutional neural networks (Krizhevsky et al., 2012)
- Speech-to-text: Towards end-to-end speech recognition with recurrent neural networks (Graves & Jaitly, 2014)
- Text: Attention is all you need (Vaswani, 2017)

Motif :

Le patient est admis le 29 août date pour des difficultés respiratoires custom .

Antécédents familiaux :

Le père du patient n'est pas asthmatique custom .

HISTOIRE DE LA MALADIE

Le patient dit avoir de la toux cim10 R05 depuis trois jours date . Elle a empiré jusqu'à nécessiter un passage aux urgences.

Named entity recognition

Machine learning might be less successful for what if questions

Machine learning is not driven by causal mechanisms

- For example people that go to the hospital die more than people who do not¹:
- Naive data analysis might conclude that hospitals are bad for health.
- The fallacy is that we are comparing different populations: people who go to the hospital typically have a worse baseline health than people who do not.

This is a confounding factor: a variable that influences both the treatment and the outcome.

¹Example from https://inria.github.io/scikit-learn-mooc/concluding_remarks.html?highlight=causality

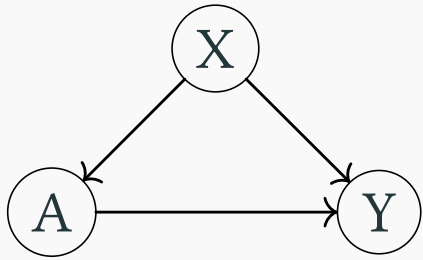
Why is prediction different from causation? (2/2)

Causal inference (most part of economists) : What would happen if we changed the system ie. under an intervention?

Why is prediction different from causation? (2/2)

Causal inference (most part of economists) : What would happen if we changed the system ie. under an intervention?

Assumption: No unmeasured variables influencing both treatment and outcome \rightarrow confounders.



Causal inference models

$(X, A, Y(A = 1), Y(A = 0))$

the covariate shift between treated and control units.

Illustration of the fundamental problem of causal inference

Example from epidemiology

- Population: patients experiencing a stroke
- Intervention $A = 1$: patients had access to a MRI scan **in less than 3 hours** after the first symptoms
- Comparator $A = 0$: patients had access to a MRI scan **in more than 3 hours** after the first symptoms
- $Y = \mathbb{P}[\text{Mortality}]$: the mortality at 7 days
- $X = \mathbb{P}[\text{Charlson score}]$: a comorbidity index summarizing the overall health state of the patient. Higher is bad for the patient.

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Illustration: observational data

Draw a population sample without treatment status

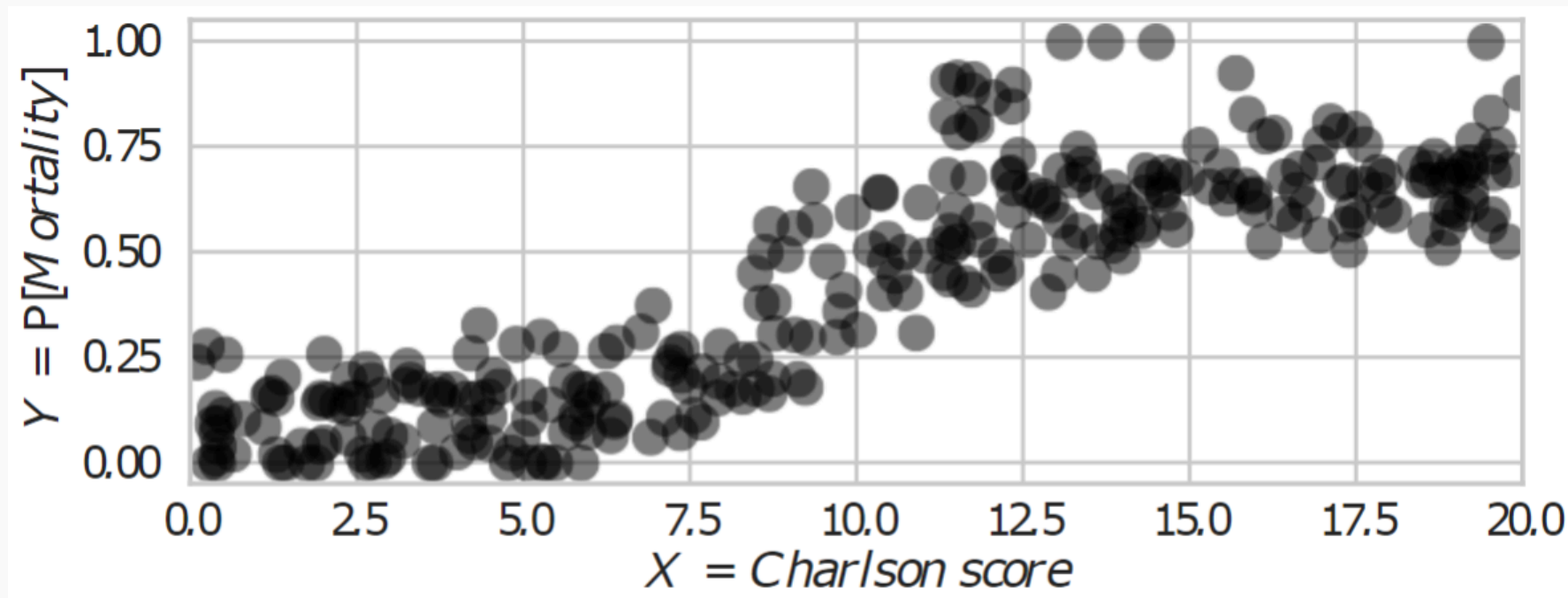


Illustration: observational data

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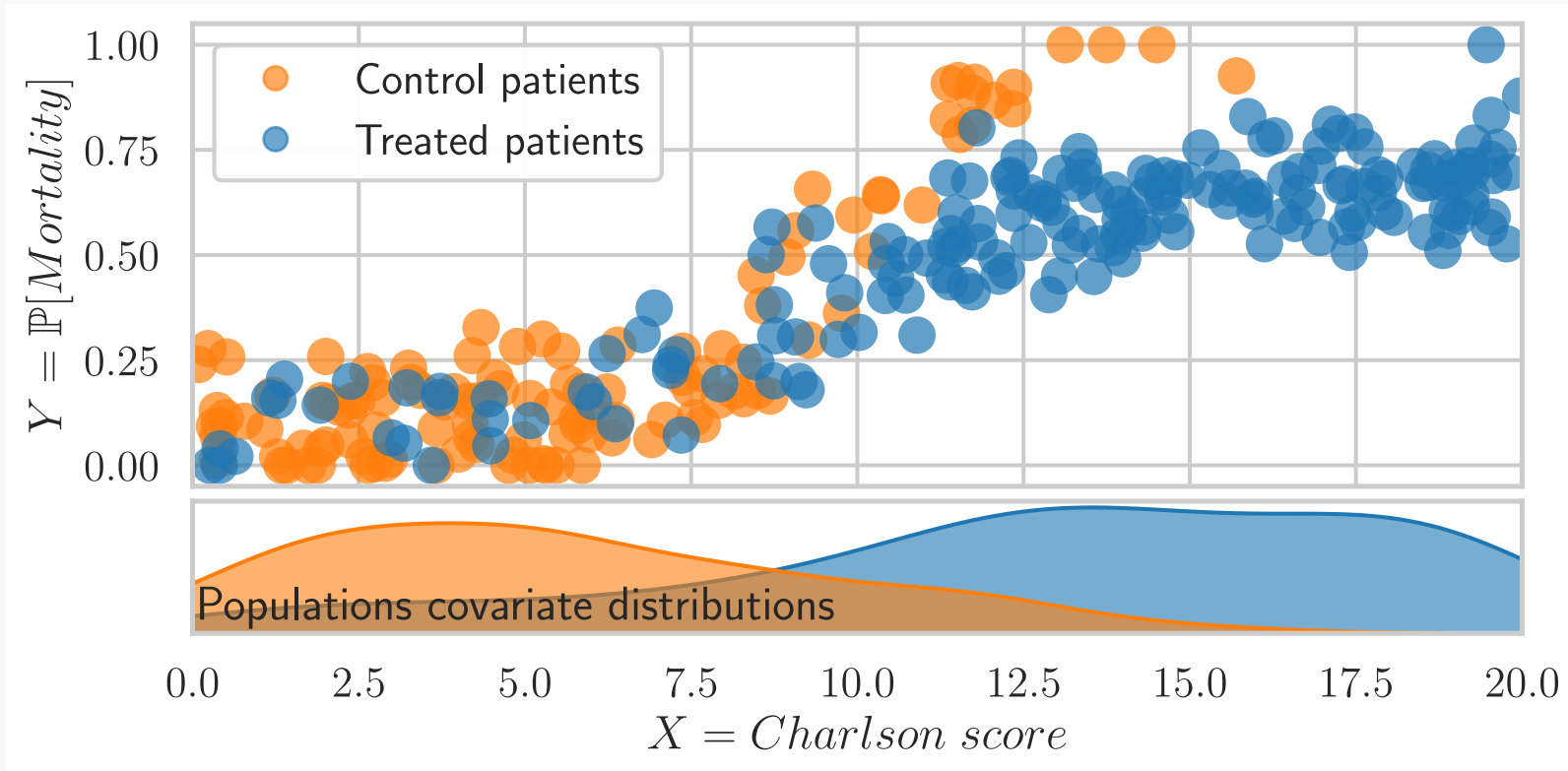
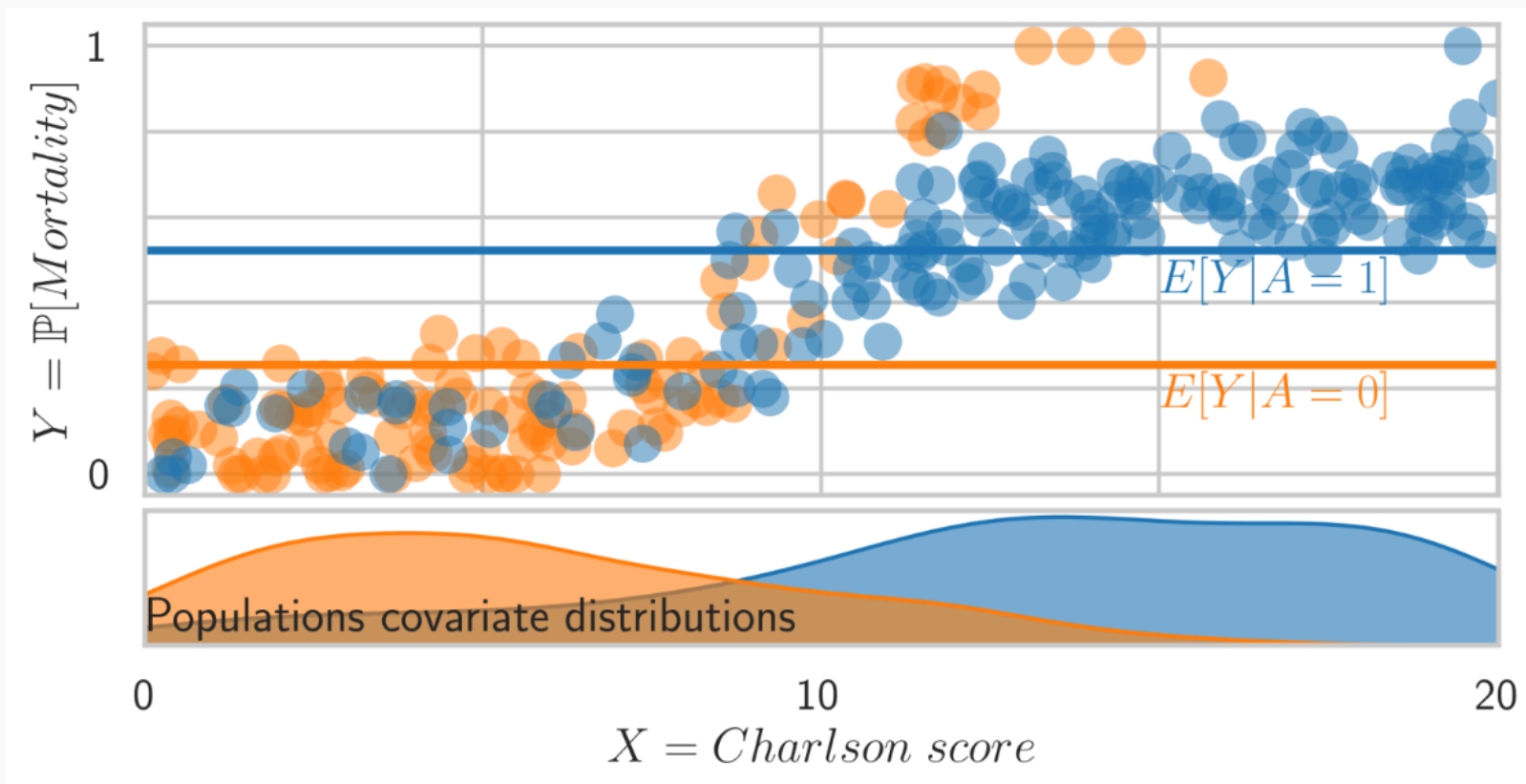


Illustration: observational data, a naive solution

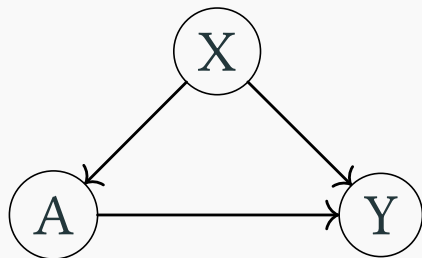
Compute the difference in mean (DM): $\tau_{\text{DM}} = \mathbb{E}[Y(1)] - \mathbb{E}[Y(0)]$

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Observational data

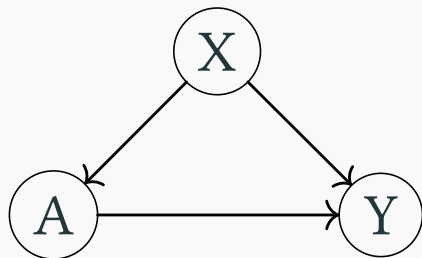


$$Y(1), Y(0) \not\perp\!\!\!\perp A$$

Intervention is not random
(with respect to the confounders)

RCT case: No problem of confounding

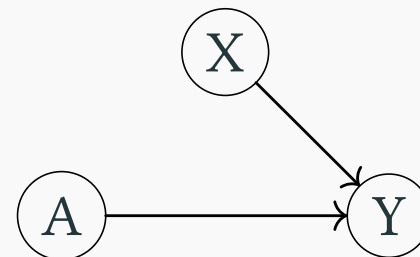
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$$Y(1), Y(0) \not\perp\!\!\!\perp A$$

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RCT data



$$Y(1), Y(0) \perp\!\!\!\perp A$$

**Force random assignment of the
intervention**

Illustration: RCT data

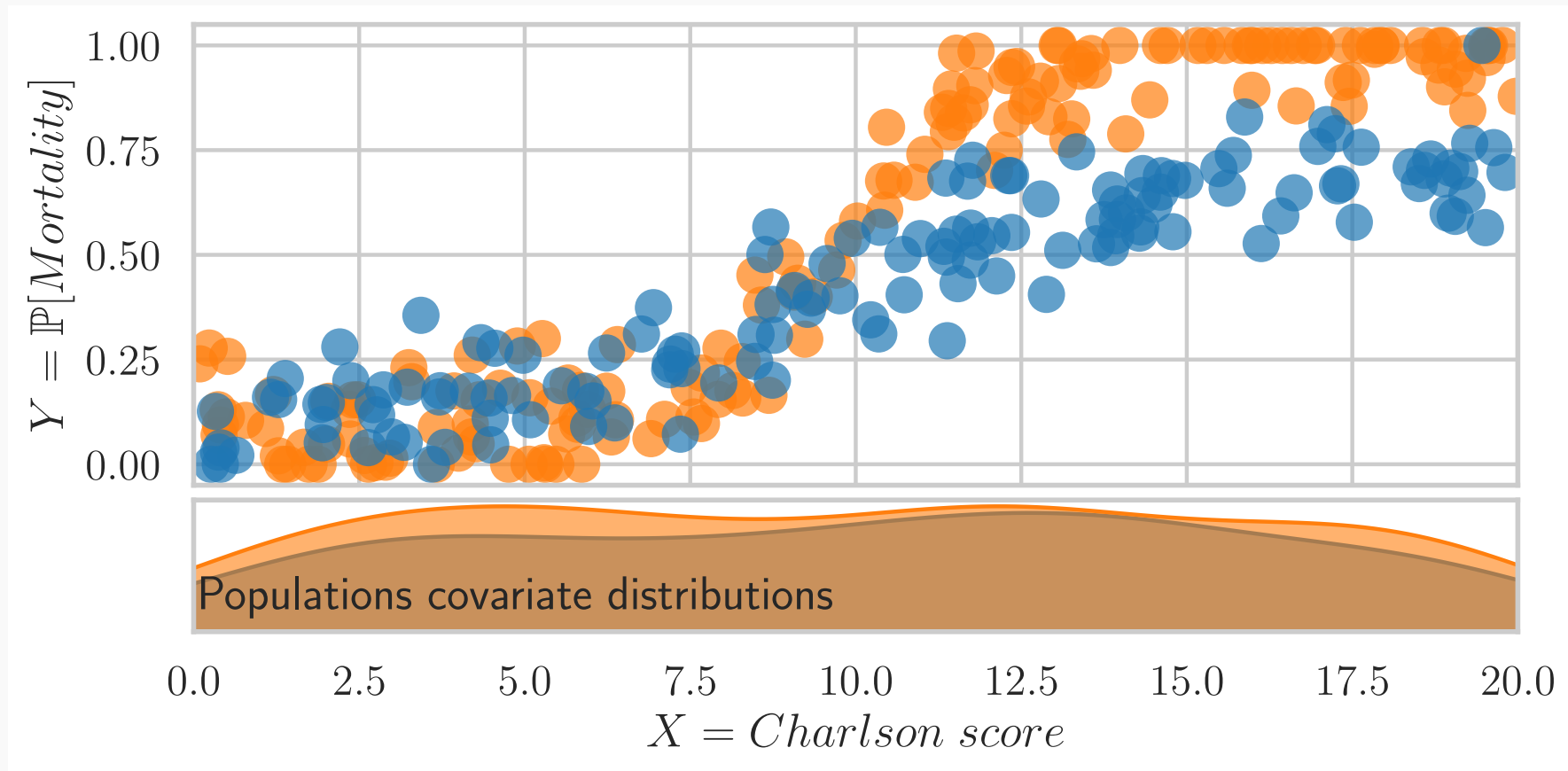
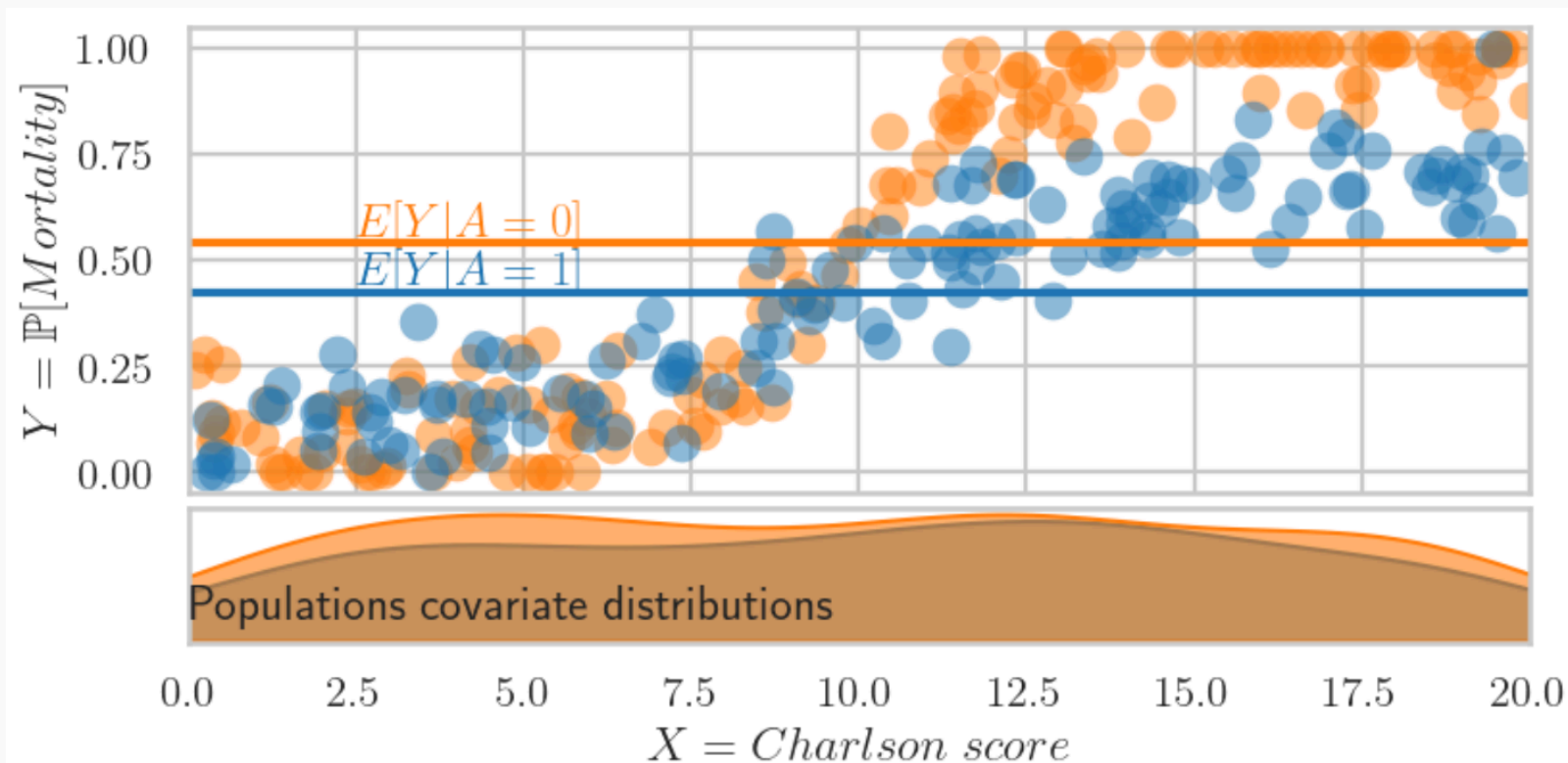


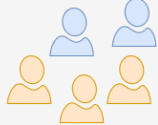
Illustration: RCT data, a naive solution

Compute the difference in mean (DM): $\tau_{\text{DM}} = \mathbb{E}[Y(1)] - \mathbb{E}[Y(0)]$




Four steps of causal inference


1 - Framing



Population
Type 2 diabetes

 **Intervention**
 $A = 1$, second line antidiabetics

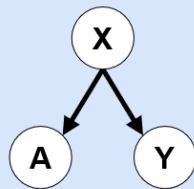
 **Comparator**
 $A = 0$, metformin

 **Outcome**
 $Y = \text{HbA1c}$

 **Time**

2 - Identification

Confounders

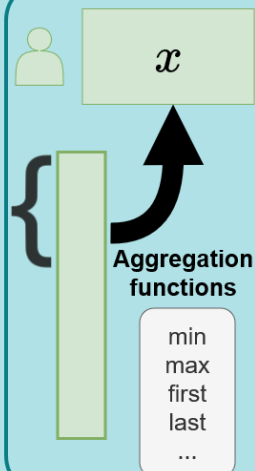


Estimand
 $\mathbb{E}[Y(1)]$
 $-\mathbb{E}[Y(0)]$

Look for other sources of bias

3 - Estimation

Feature extraction



Causal estimator

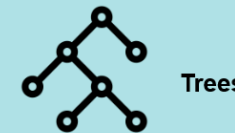
G-formula
 $\mu(a; x) = \mathbb{E}[Y|a; x]$

IPW
 $e(x) = \mathbb{P}[A = 1|x]$

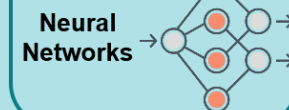
Double-robust
 $(e(x), \mu(a; x))$

Nuisance estimator

Linear Regression



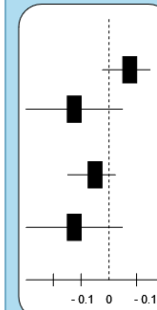
Trees



Neural Networks

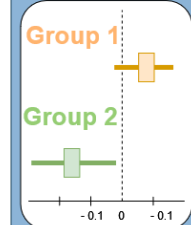
4 - Vibration Analysis

Question analyses choices

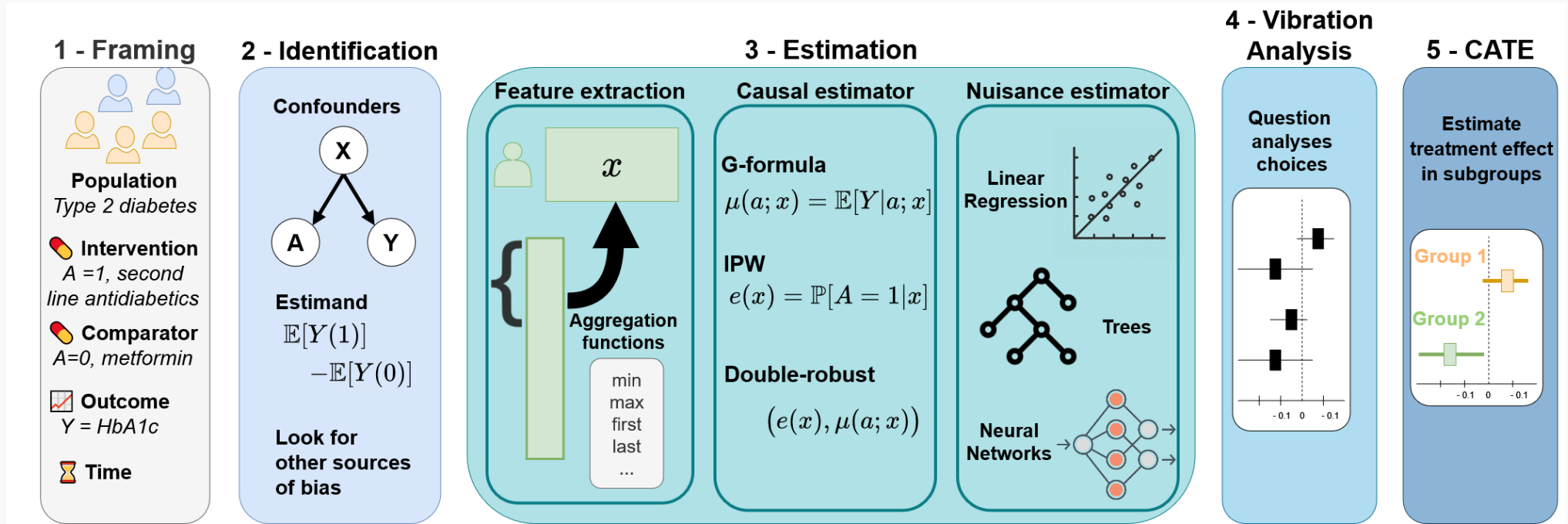


5 - CATE

Estimate treatment effect in subgroups



Four steps of causal inference



Today: Framing and identification with DAGs

Framing: How to ask a sound causal question

PICO framework (Richardson et al., 1995)

Originally designed for clinical research. It is a structured approach to formulate a research question. Critical for health technology assessment (eg. Haute Autorité de santé).

PICO stands for

- Population : Who are we interested in?
- Intervention : What treatment/intervention do we study?
- Comparison : What are we comparing it to?
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Example with the job dataset (LaLonde, 1986)

Built to evaluate the impact of the National Supported Work (NSW) program. The NSW is a transitional, subsidized work experience program targeted towards people with long-standing employment problems.

The PICO framework

Component	Description	Example
Population	What is the target population of interest?	People with longstanding employment problems
Intervention	What is the intervention?	On-the-job training lasting between nine months and a year
Control	What is the relevant comparator?	No training
Outcome	What are the outcomes?	Earnings in 1978
Time	Is the start of follow-up aligned with intervention assignment?	The period of follow-up for the earning is the year after the intervention

PICO: other examples in econometrics

The Oregon Health Insurance Experiment (Finkelstein et al., 2012) : A randomized experiment by lottery assessing the impact of Medicaid on low-income adults in Oregon.

- P: Low-income adults in Oregon
- I: Medicaid
- C: No insurance
- O: Healthcare uses and expenditures, health outcomes

PICO: other examples in econometrics

The economic impact of climate change on US agricultural land. (Deschênes & Greenstone, 2007): difference-in-differences design assessing the impact of climate change on agricultural profits.

- P: US agricultural land
- I: Climate change
- C: No climate change
- O: Agricultural profits

PICO: other examples in econometrics

The impact of class size on test scores. (Angrist & Lavy, 1999): regression discontinuity design.

- P: Fourth and fifth grades school in Israel
- I: Class size increases by one unit
- C: No class size increase
- O: Test scores (math and reading)

Identification: List necessary information to answer the causal question

Identification: Build the causal model

A causal effect is said to be identified if it is possible, with ideal data (infinite sample size and no measurement error), to purge an observed association of all noncausal components such that only the causal effect of interest remains.

— (Elwert & Winship, 2014)

Steps

- Potential outcome framework : mathematical tool to reason about causality
- Directed acyclic graphs (DAG) : graphical tool to reason about causality
- Causal estimand : what is the targeted quantity?

Potential outcomes, (Neyman, 1923; Rubin, 1974)

The Neyman-Rubin model, let:

- Y be the outcome,
- A the (binary) treatment

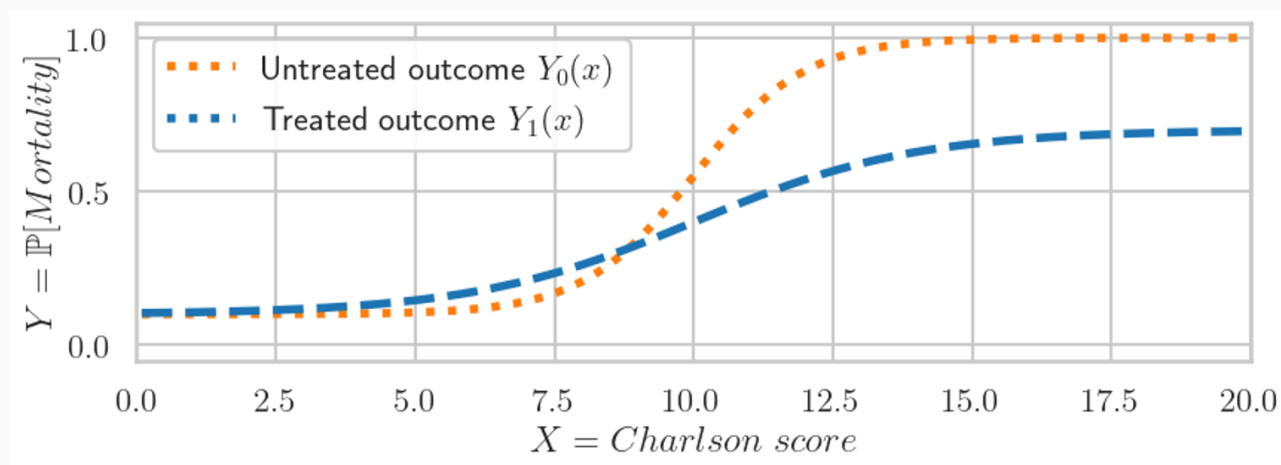
For each individual, we have two potential outcomes: $Y(1)$ and $Y(0)$. But only one is observed, depending on the treatment assignment: $Y(A)$.

Potential outcomes, (Neyman, 1923; Rubin, 1974)

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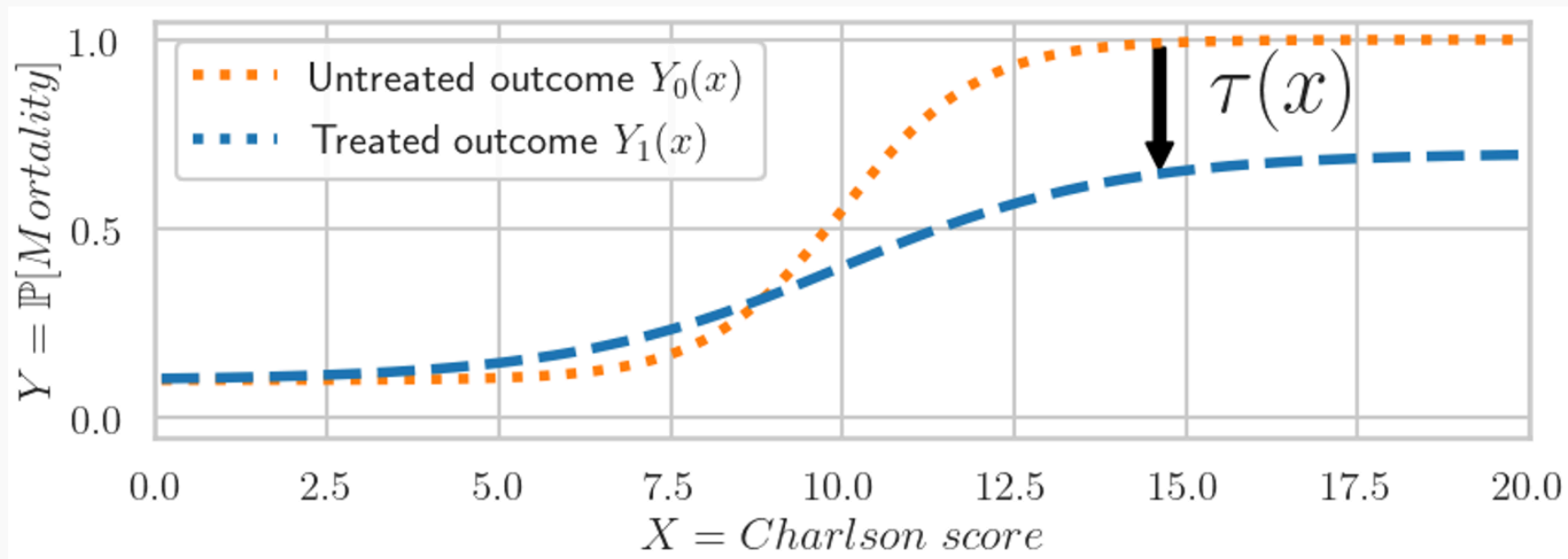


Causal estimand: What is the targeted quantity (with potential outcomes)?

- Average treatment effect (ATE): $\mathbb{E}[Y(1) - Y(0)]$
- Conditional average treatment effect (CATE): $\mathbb{E}[Y(1) - Y(0) \mid X]$

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Causal estimand: What is the targeted quantity (with potential outcomes)?

Other estimands

- Average treatment effect on the treated (ATT): $\mathbb{E}[Y(1) - Y(0) \mid A = 1]$
- Conditional average treatment effect on the treated (CATT):

$$\mathbb{E}[Y(1) - Y(0) \mid A = 1, X]$$

Causal estimand: What is the targeted quantity (with potential outcomes)?

Other estimands

- Average treatment effect on the treated (ATT): $\mathbb{E}[Y(1) - Y(0) \mid A = 1]$
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Other estimands more used in epidemiology

- Risk ratio (RR): $\frac{\mathbb{E}[Y(1)]}{\mathbb{E}[Y(0)]}$
- Odd ratio (OR) for binary outcome: $\left(\frac{\mathbb{P}[Y(1)=1]}{\mathbb{P}[Y(1)=0]} \right) / \left(\frac{\mathbb{P}[Y(0)=1]}{\mathbb{P}[Y(0)=0]} \right)$

See (Colnet et al., 2023) for a review of the different estimands and the impact on generalization.

PICO framework, link to the potential outcomes

Component	Description	Notation	Example
Population	What is the target population of interest?	$X \sim P(X)$	People with longstanding employment problems
Intervention	What is the intervention?	$A \sim P(A = 1) = p_A$	On-the-job training lasting between nine months and a year
Control	What is the relevant comparator?	$1 - A \sim 1 - p_A$	No training
Outcome	What are the outcomes?	$Y(1), Y(0) \sim P(Y(1), Y(0))$	Earnings in 1978
Time	Is the start of follow-up aligned with intervention assignment?	N/A	The period of follow-up for the earning is the year after the intervention

What can we learn from the data?

- Four assumptions, referred as strong ignorability
- Required to assure identifiability of the causal estimands with observational data (Rubin, 2005)

Assumption 1: Unconfoundedness, also called ignorability

Treatment assignment is as good as random given the covariates X

$$\{Y(1), Y(0)\} \perp\!\!\!\perp A \mid X$$

- Equivalent to conditional independence on the propensity score:

$e(X) \stackrel{\text{def}}{=} \mathbb{P}(A = 1|X)$ (Rosenbaum & Rubin, 1983):

$$\{Y(1), Y(0)\} \perp\!\!\!\perp A \mid e(X)$$

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$$\{Y(1), Y(0)\} \perp\!\!\!\perp A \mid X$$

- Equivalent to conditional independence on the propensity score:

$e(X) \stackrel{\text{def}}{=} \mathbb{P}(A = 1|X)$ (Rosenbaum & Rubin, 1983):

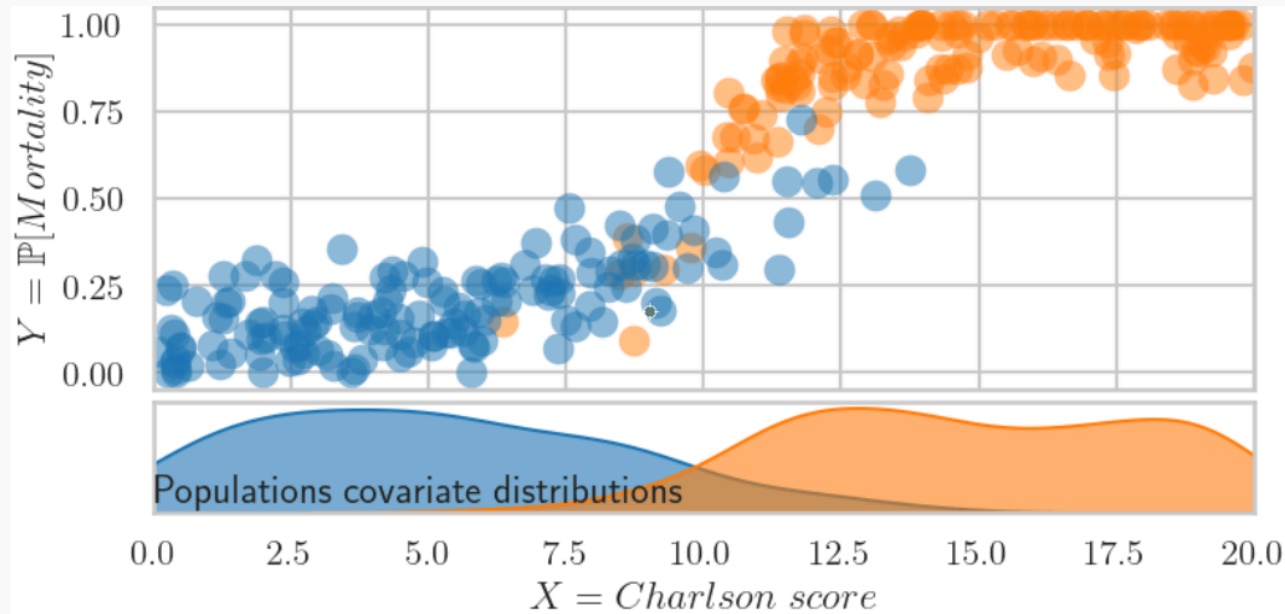
$$\{Y(1), Y(0)\} \perp\!\!\!\perp A \mid e(X)$$

- Knowledge based ie. cannot be validated with data
 - Because of possibly unmeasured confounders
 - In practice : ask yourself if you have measured all the relevant variables that could influence both the treatment and the outcome.

Assumption 2: Overlap, also known as positivity

The treatment is not deterministic given X

$$\eta < e(x) < 1 - \eta \text{ with } e(X) \stackrel{\text{def}}{=} \mathbb{P}(A = 1|X)$$



- Choice of covariates X : trade-off between ignorability and overlap (D'Amour et al., 2021)

Assumption 3 and 4: Consistency and generalization

Consistency, also called Stable Unit Treatment Values (SUTVA)

The observed outcome is the potential outcome of the assigned treatment for each unit i .

$$Y_i = A_i Y_i(1) + (1 - A_i) Y_i(0)$$

- The intervention A is well defined (Hernan & Robins, 2020)
- There is no interference ie. network effect

Assumption 3 and 4: Consistency and generalization

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Generalization, also called no-covariate shift

Training and test data are drawn from the same distribution

Directed acyclic graphs (DAGs)

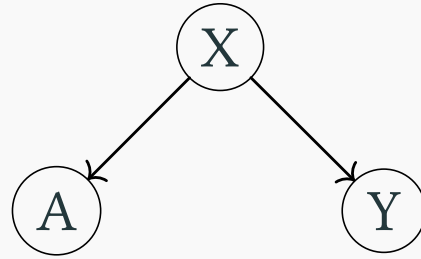
DAGs encode the causal structure of the data generating process

Introduced by (Pearl, 1995), (Pearl & others, 2000). Good practical overview in (Vander-Weele, 2019).

Motivation

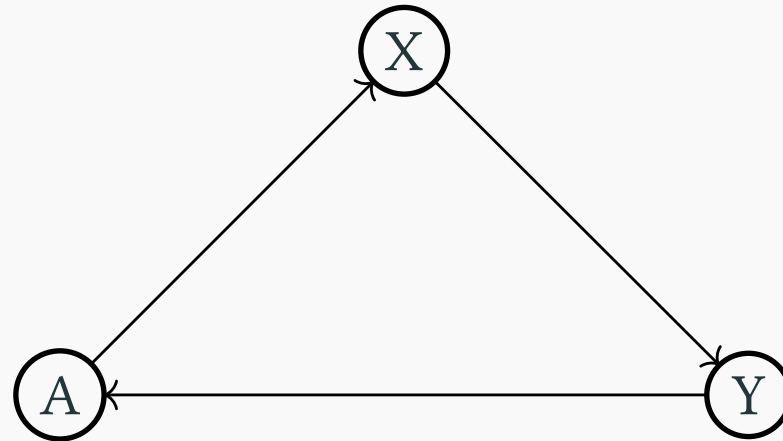
- Reason about the relation between variables.
- Help identify for which (minimal) set of variables, the ATE is identifiable.

Directed acyclic graphs (DAG), definitions

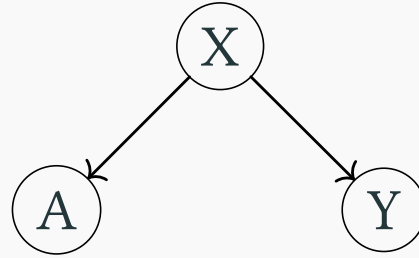


- **Graph:** A set of relations between nodes described by edges between those nodes
- **Directed:** Edges between nodes have direction. The direction of the arrow represents a cause-effect relationship.
- **Acyclic:** : There are no cycles or loops in the causal structure. A variable can't be a cause of itself.

This is not a DAG



DAGs: nodes

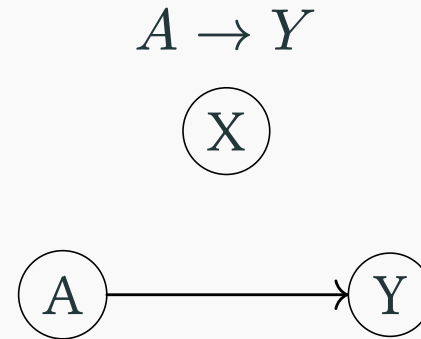
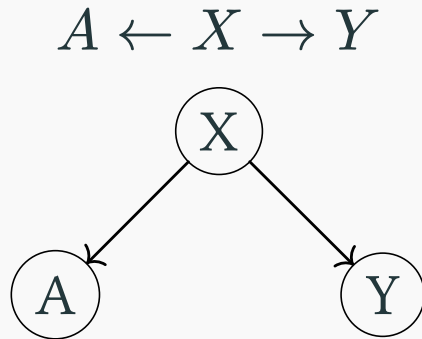


- **Nodes** represent random variables.
- **Edges** between nodes denote the presence of causal effects (i.e. difference in potential outcomes). Here, $Y_i(a) \neq Y_i(a')$ for two different levels of A_i because there is an arrow from A to Y.
- **Lack of edges** between nodes denotes the absence of a causal relationships.

Not drawing an arrow makes a stronger assumption about the relationship between those two variables than drawing an arrow.

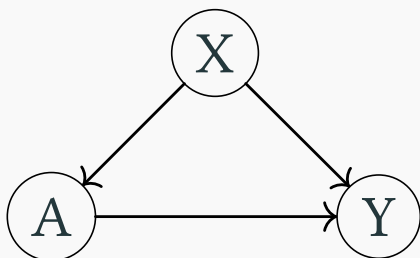
DAGs: paths

- A path between two nodes is a route that connects the two nodes following non-intersecting edges.
- A path exists even if the arrows are not pointing in the good direction.
- Two examples of paths between A and Y:



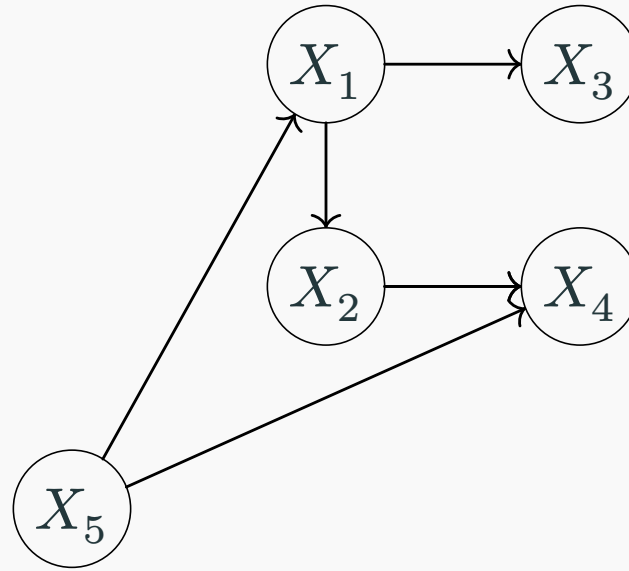
DAGs: causal paths

- Paths encode dependencies between random variables (not necessarily causal dependencies; it can be mere associations).
- We distinguish:
 - **Causal** paths: arrows are all in the same direction.
 - From **Non-causal** paths: arrows pointing in different directions
- When there is a causal path between two variables A and B, we say that B is a **descendant** of A (it is causally impacted by A)



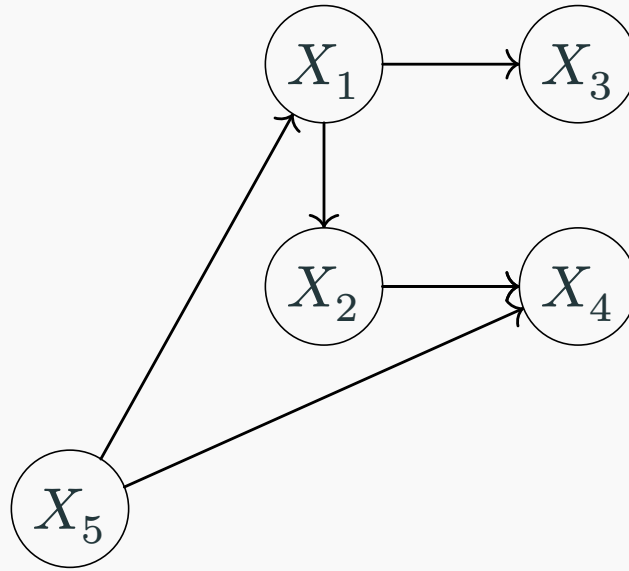
$A \rightarrow Y$ is **causal**
 $Y \rightarrow A$ is **non-causal**

Your turn



Paths between X_1 and X_4 ? Which of them are causal?

Your turn



Paths between X_1 and X_4 ? Which of them are causal?

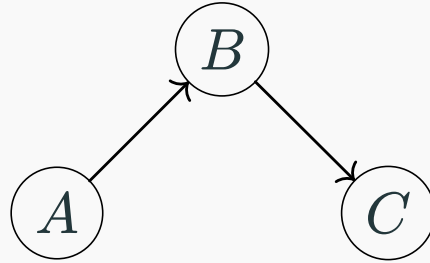
- $X_5 \rightarrow X_1 \rightarrow X_2 \rightarrow X_4$
- $X_5 \rightarrow X_4$
- $X_1 \leftarrow X_5 \rightarrow X_4$: non causal

- $X_3 \leftarrow X_1 \rightarrow X_2 \rightarrow X_4$: non causal

Three types of directed edges: path

There are three kinds of “triples” or paths with three nodes: These constitute the most basic building blocks for causal DAGs.

First, a **causal path (or chain):** $A \rightarrow B \rightarrow C$

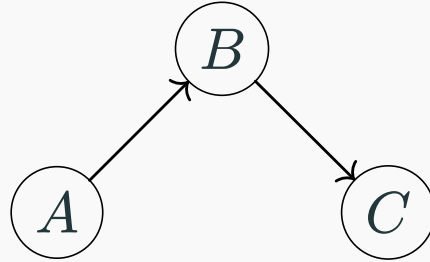


- The effect of A “flows” through B. A and C are not independent and the relationship is causal.

Three types of directed edges: path

There are three kinds of “triples” or paths with three nodes: These constitute the most basic building blocks for causal DAGs.

First, a **causal** path (or chain): $A \rightarrow B \rightarrow C$

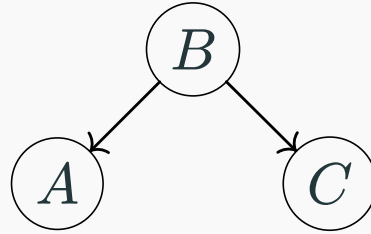


Example

An individual receiving a message (A) encouraging them to vote causes that individual to actually vote (C) only if the individual actually reads (B) the message.

Three types of directed edges: mutual dependence

Second, **mutual dependence** or fork or confounder: $A \leftarrow B \rightarrow C$

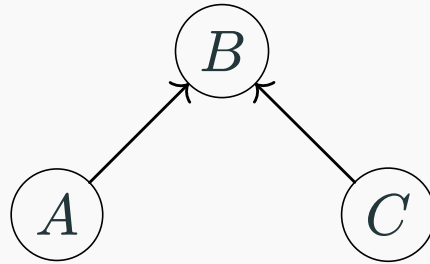


- A and C are not causally related but B is a common cause of both.
- A and C are not independent, but are independent conditional on B.
- **Not conditioning on X** introduces bias.

- Rise in temperature (B) causes both the thermometer (A) to change, and ice to melt (C), but the thermometer changing does not cause ice to melt.
- A is prostate cancer; B is age; and C is Alzheimer's disease.

Three types of directed edges: collider

Third, **collider**: $A \rightarrow B \leftarrow C$

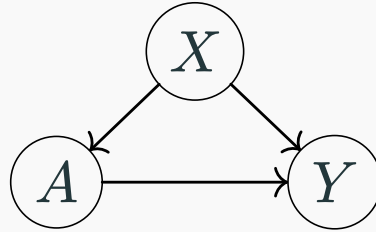


- A and C are both common causes of B : they collide into B.
- A and C are independent, but conditionnaly dependent given B.
- **Conditionning on B** introduces a spurious correlation between A and C.

- A is result from dice 1, C is results from dice 2, B is sum of dice 1 and dice 2.
- A is height, C is speed, B is whether an athlete plays in the NBA.

Open and blocked paths

We can open or block paths between variables by conditioning on them:



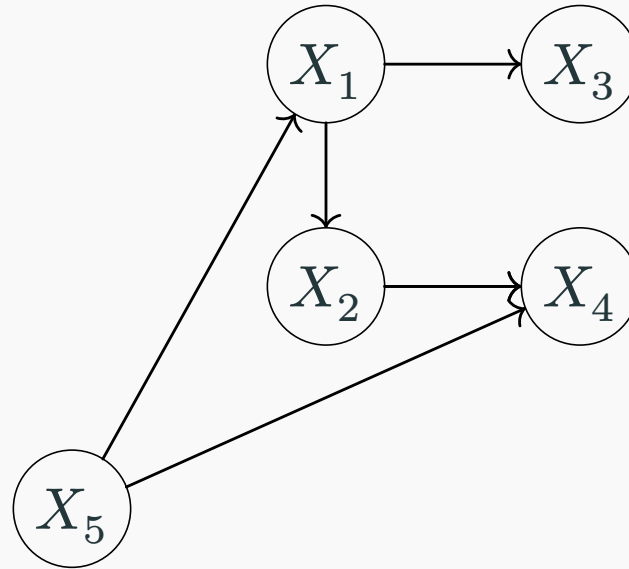
A path is **blocked** (or d-separated) if:

- the path contains a non-collider that has been conditioned on.
- or the path contains a collider that has not been conditioned on (and has no descendants that have been conditioned on).

Conditioning on a variable:

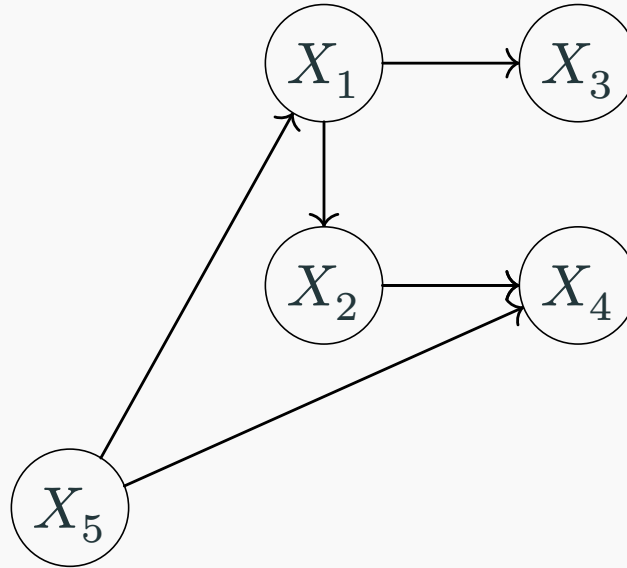
- **Blocks** a path if that variable is **not a collider** on that path.
- **Opens** a path if that variable is a **collider** on that path.

Your turn



Paths from X_5 to X_2 ? Which of them are opened/blocked?

Your turn



Paths from X_5 to X_2 ? Which of them are opened/blocked?

$X_5 \rightarrow X_1 \rightarrow X_2$ (blocked by conditioning on X_1)

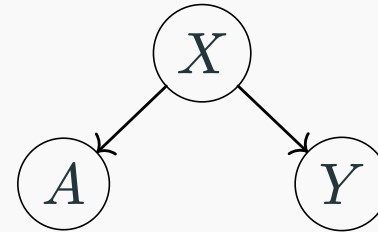
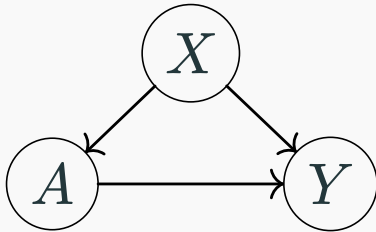
$X_5 \rightarrow X_1 \rightarrow X_2 \rightarrow X_4$ (opened by conditioning X_4)

Backdoor paths: a special type of paths

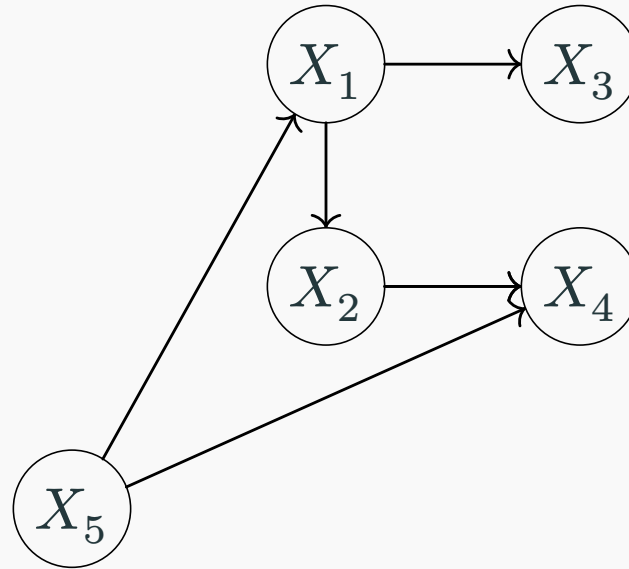
Backdoor path: A Backdoor path from a variable A to another Y, is any non-causal path between A and Y that does not include descendants of A

Identifying backdoor paths: Backdoor paths from A to Y are all those paths that remain between A and Y after removing all arrows coming out of A.

These paths are responsible for confounding bias: they imply association not causation.

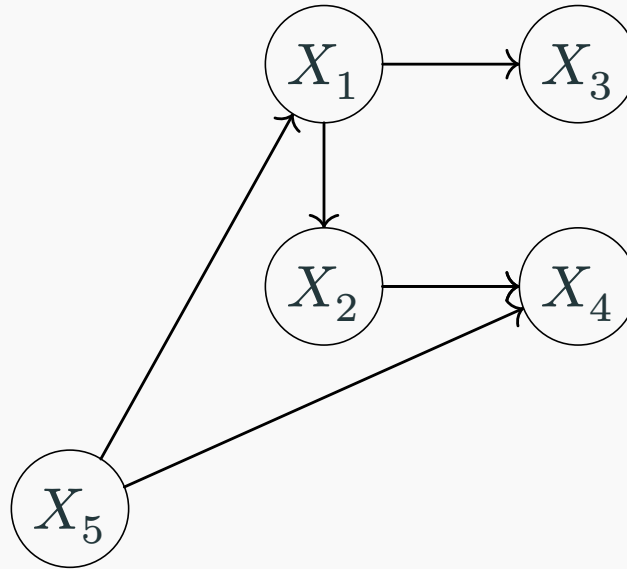


Your turn



What are the backdoor paths from X_1 to X_4 ? from X_2 to X_4 ?

Your turn



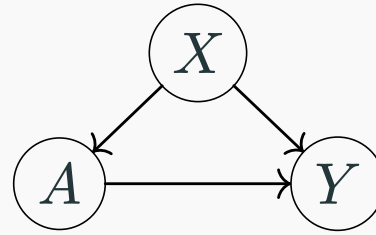
What are the backdoor paths from X_1 to X_4 ? from X_2 to X_4 ?

$$X_1 \leftarrow X_5 \rightarrow X_4$$

$$X_2 \leftarrow \leftarrow X_5 \rightarrow X_4$$

Graphical identification

DAGs help us know whether observed covariates are enough to identify a treatment effect.



In other words, how can we make it so that there are no non-causal dependencies between treatment and outcome?

Graphical identification (Pearl & others, 2000)

The effect of A on Y is identified if all backdoor paths from A to B are blocked, and no descendant of A is conditioned on.

On which variables should we condition? General rules

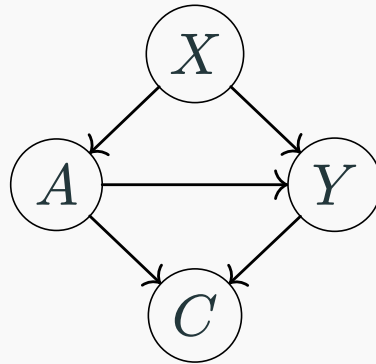
- Do not condition for variables on causal paths from treatment to outcome
- Condition on variables that block non-causal backdoor paths
- Don't condition on colliders! Eg. don't condition on post-treatment variables.

On which variables should we condition? General rules

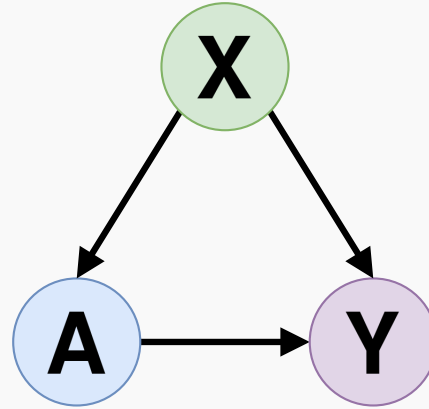
- Do not condition for variables on causal paths from treatment to outcome
- Condition on variables that block non-causal backdoor paths
- Don't condition on colliders! Eg. don't condition on post-treatment variables.

In the following example, to estimate the effect of T on Y, we should:

- Condition on X
- NOT condition on M because it is a descendant of T



Famous examples of confounders

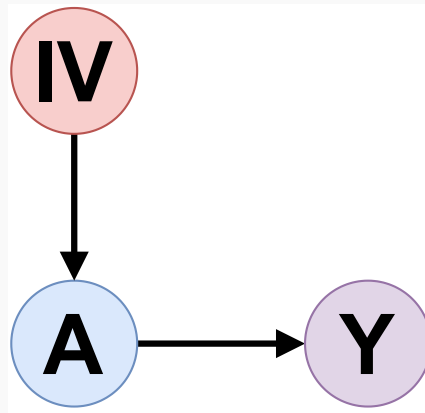


Effect of education on earnings

The family background can act as a confounder: Wealthier families may provide better education opportunities AND influence earnings independently of the education itself.

Famous examples of instrumental variables

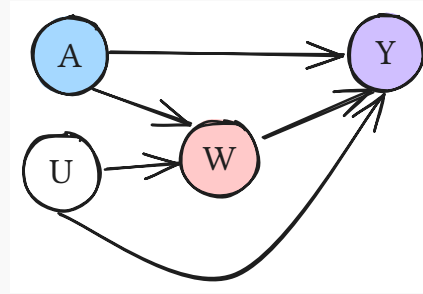
Instrumental variable (IV): influences only the treatment.



Effect of education on earnings (Angrist & Krueger, 1991)

Quarter of birth are randomly assigned but influence the lengths of schooling due to school entry laws.

Famous examples of colliders

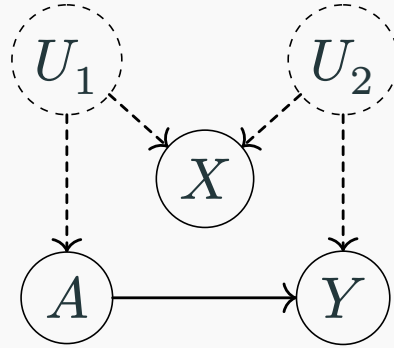


Special case of collider: consequence of both the treatment and the outcome.

Effect of smoking on mortality (Hernández-Díaz et al., 2006)

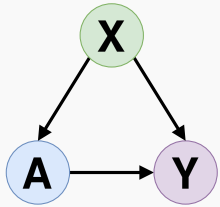
Birth weight is influenced by smoking and other factors. Conditioning on birth weight (a collider) creates a spurious negative correlation between smoking and other risk factors, leading to the paradoxical conclusion that smoking reduces infant mortality, even though it harms overall health.

More colliders: M-bias

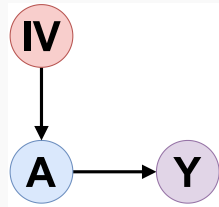


- Do not condition on any pre-exposure variable that you have at disposal!
- Should we condition on X in trying to estimate the effect of A on Y ?
- There is a backdoor path through two unobserved variables (U_1, U_2). But it is blocked because X is a collider along that path.
- Conditioning on X opens up that path, inducing a non-causal association between T and Y .

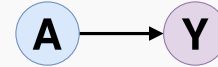
Which variable to include into your analysis?



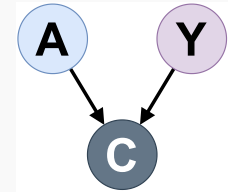
Confounder ✓



Instrumental variable ✗
(generally)



Outcome parent ✓
(generally)

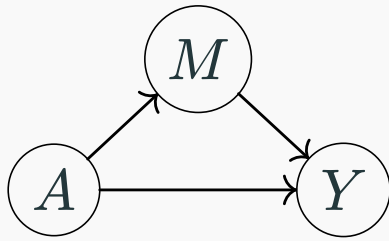


Collider ✗

- High-level strategy: Control solely for pre-treatment variables that influences both the outcomes, the treatment or both.

Special types of variables: mediators

A **mediator** block the path from the treatment to the outcome.



Here, two causal paths from A:

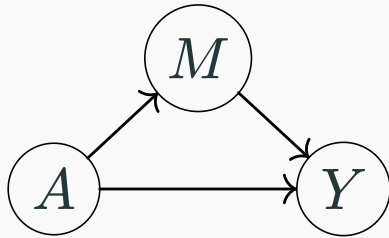
- $A \rightarrow Y$ - a “direct effect”
- $A \rightarrow M \rightarrow Y$ - an “indirect effect” through M

Effect of children poverty on economic outcomes (Bellani & Bia, 2019)

- Y is economic outcomes in adulthood, A is child poverty, M is education.
- What part of the effect of poverty on outcome is mediated by education?

Special types of variables: mediators

A **mediator** block the path from the treatment to the outcome.

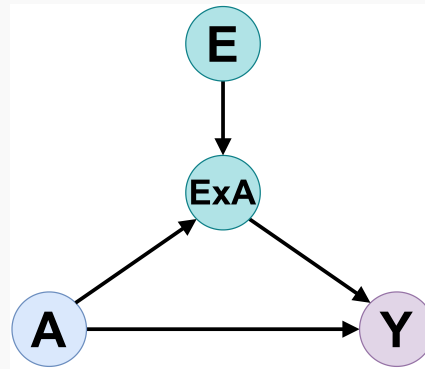


Here, two causal paths from A :

- $A \rightarrow Y$ - a “direct effect”
- $A \rightarrow M \rightarrow Y$ - an “indirect effect” through M

- All causal paths from a treatment capture its overall treatment effect.
- The average treatment effect of T combines both the “direct effect” and the “indirect effect”.

Effect modifier: influences the treatment effect on the outcome.



Take away on DAGs

- DAGs are a powerful tool to reason about causality
- Useful to identify the variables to condition on / to include into the analysis
- NB: Drawing the true DAG is often hard / not feasible

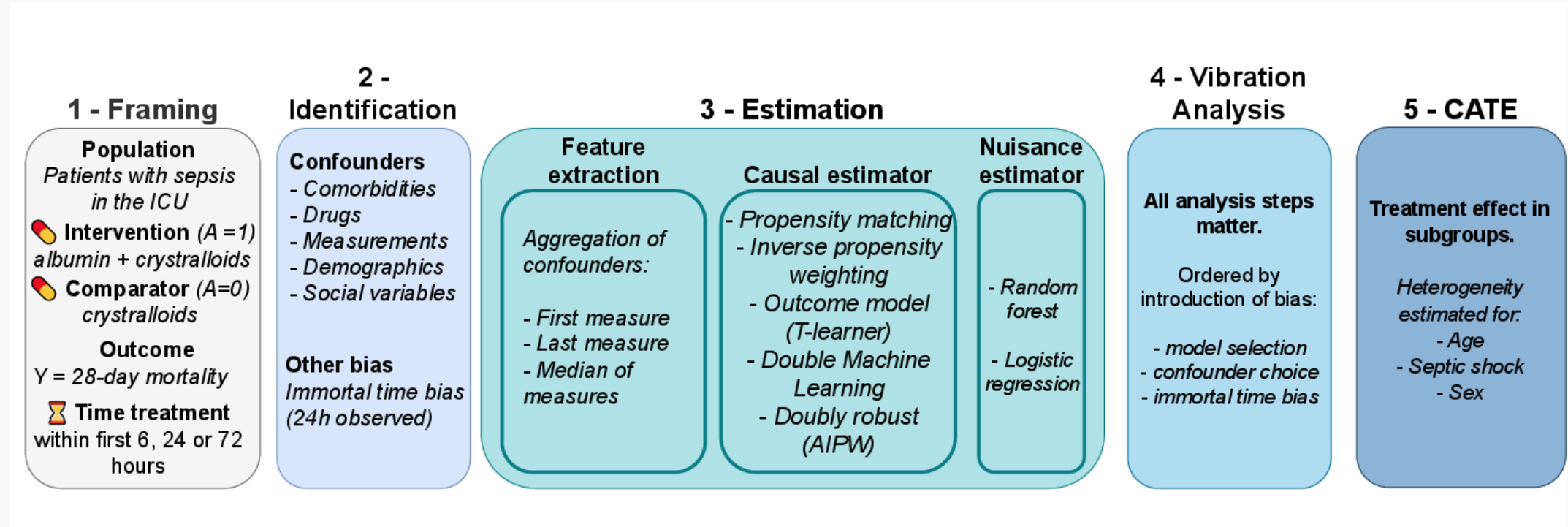
Take away on covariate selection

What is the most important part to insure validity?

- The covariate included (an appropriate DAG)?
- The design of the study?
- The causal estimator (IPW, G-formula, AIPW...)
- The statistical estimator (Linear regression, Logistic regression...)

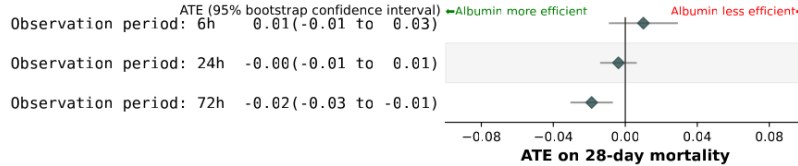
Comparing different choices for the study steps (Doutreligne et al., 2024)

Question of interest

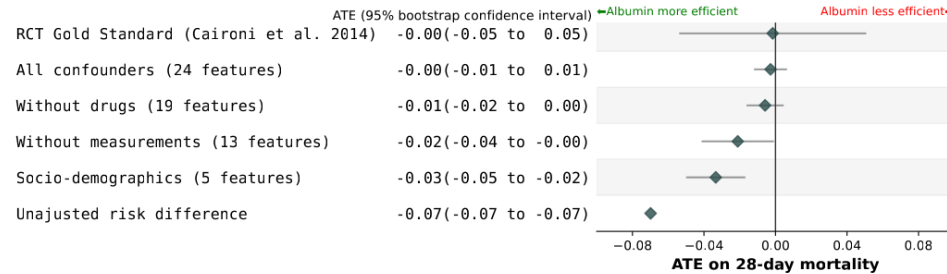


Results of the sensitivity analysis

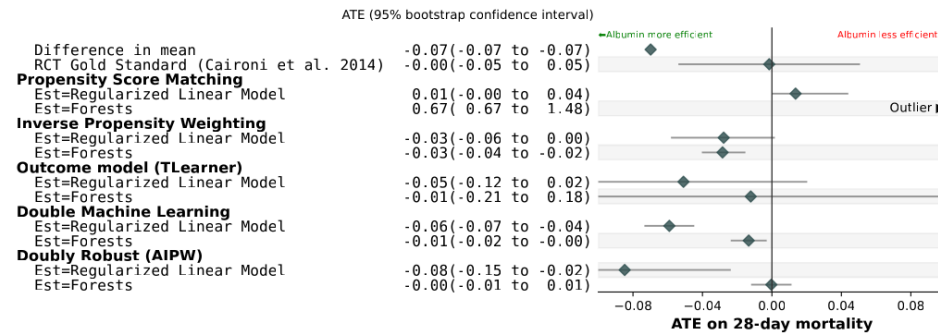
(a) Framing – Immortal Time Bias



(b) Identification – confounders choice



(c) Model selection



Practical session

To your notebooks! 

- url: https://straymat.github.io/causal-ml-course/practical_sessions.html

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Supplementary material

A word on structural equation models

TODO