

Introduction to Causal Inference

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Machine Learning in High Energy Physics Summer School
June 6, 2019

Why bother?

Predictive models are great, why do we need causal inference?

- ▶ in real life today's train could differ from tomorrow's test
- ▶ especially if we want to act on the results of the predictions!
- ▶ causal mechanisms are more stable than correlations

What is causality?

Lewis D. (1973) *Causation*. The journal of philosophy: 556-567: causation is “something that makes a difference, and the difference it makes must be a difference from what would have happened without it”.

The “interventionis” definition: T causes Y iff changing T leads to a change in Y , *keeping everything else constant*.

The causal effect is the magnitude by which Y is changed by a unit change in T .

Keeping everything else constant: parallel, counterfactual reality.

Causal questions are weird!

The Three Layer Causal Hierarchy

Level	Typical Activity	Typical Question	Examples
1. Association $\mathbf{P}(y x)$	Seeing	What is?	What does a symptom tell me about a disease? What does a survey tell us about the election results?
2. Intervention $\mathbf{P}(y do(x), z)$	Doing, Intervening	What if? What if I do X?	What if I take aspirin, will my headache be cured? What if we ban cigarettes?
3. Counterfactual $\mathbf{P}(y_x x', y')$	Imagining, Retrospection	Why? Was it X that caused Y? What if I had acted differently?	Was it the aspirin that stopped my headache? What I had not been smoking the past 2 years?

Pearl J. *Theoretical Impediments to Machine Learning with Seven Sparks from the Causal Revolution*. arXiv:1801.04016v1, 2018

Potential outcomes framework

Y_{1i} — the outcome for unit i that would be observed in condition $T = 1$ (“treatment”),

Y_{0i} — the outcome that would be observed, all else held constant, in condition $T = 0$ (“control”).

Causal effect of treatment on Y :

$$\tau_i = Y_{1i} - Y_{0i}$$

Fundamental problem of causal inference: only one outcome is observed for each unit
⇒ causal effect cannot be measured.

Solution — estimate something else, e.g. average causal effect:

$$\text{ATE} = \mathbb{E}(\tau_i) = \mathbb{E}(Y_{1i} - Y_{0i}) = \mathbb{E}(Y_{1i}) - \mathbb{E}(Y_{0i})$$

(population) average treatment effect.

Randomized experiment

- ▶ A large population of experimental units
- ▶ Treatment T with support $\{0, 1\}$
- ▶ Each unit in $i \in U$ has potential outcomes Y_{0i}, Y_{1i}
- ▶ Population average treatment effect:

$$\text{ATE} = \mathbb{E}(Y_1 - Y_0)$$

- ▶ Random sample of size N from the population
- ▶ Sample average treatment effect — an estimate of ATE:

$$\text{SATE} = \frac{1}{N} \sum_{i=1}^N (Y_{1i} - Y_{0i})$$

- ▶ Randomly assign N_1 units to treatment ($T_i = 1$) and $N_0 = N - N_1$ to control ($T_i = 0$)
- ▶ Observe $Y_i = T_i Y_{1i} + (1 - T_i) Y_{0i}$
- ▶ Because treatment assignment is random,

$$\widehat{\text{SATE}} = \frac{1}{N_1} \sum_{i: T_i=1} Y_i - \frac{1}{N_0} \sum_{j: T_j=1} Y_j = \bar{Y}_1 - \bar{Y}_0$$

is an unbiased estimate of SATE (and ATE)

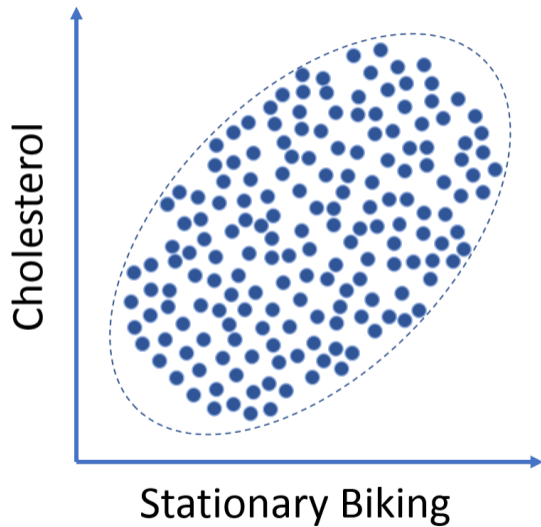
Randomized experiment

Experiment is not always feasible:

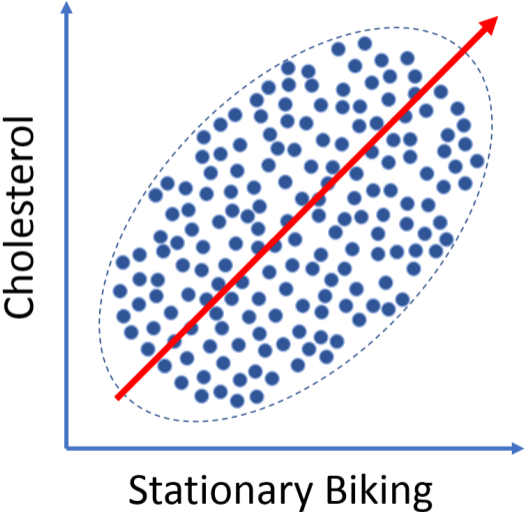
- ▶ thunderstorms → forest fires — we cannot manipulate the treatment
- ▶ TV violence → cruelty — treatment is difficult to fix, response is difficult to measure in a lab
- ▶ alcohol consumption → performance in school — unethical

In such cases we have to resort to observational data.

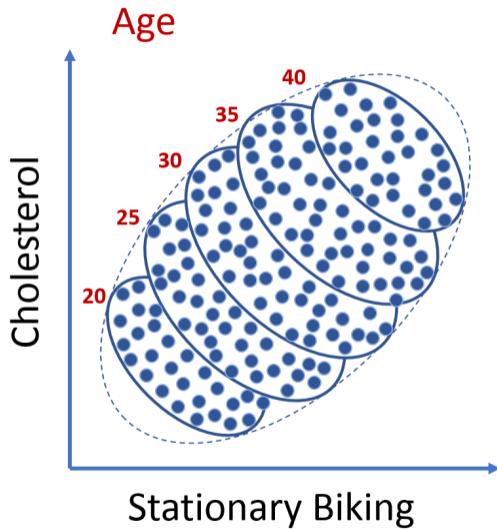
Cholesterol and exercise



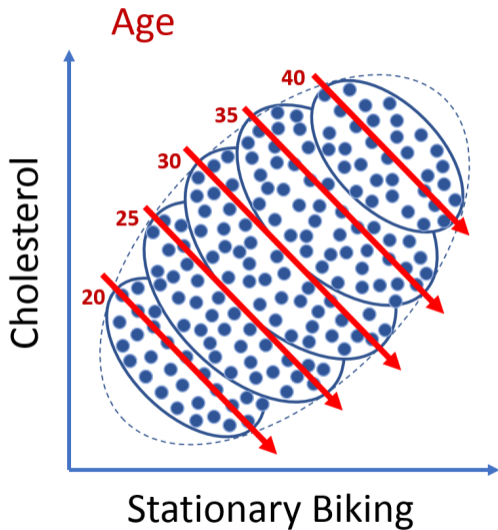
Cholesterol and exercise



Cholesterol and exercise



Cholesterol and exercise



Simpson's paradox

Example 1:

Σ	Recovered	Not recovered	Recovery rate
Drug	273	77	78%
Placebo	289	61	83%

Placebo is 5% more effective

Men	Recovered	Not recovered	Recovery rate
Drug	81	6	93%
Placebo	234	36	87%

Drug is 5% more effective

Women	Recovered	Not recovered	Recovery rate
Drug	192	71	73%
Placebo	55	25	69%

Drug is 4% more effective

Simpson's paradox

Does the drug increase chance to recover compared to placebo?

Conclusion 1: drug is 5% worse than placebo.

$$\widehat{ATE} = \mathbf{P}(recovery | drug) - \mathbf{P}(recovery | placebo)$$

Conclusion 2: drug is 4.51% better than placebo (assuming patients are 49% women).

$$\widehat{ATE} = \sum_{sex_i} (\mathbf{P}(recovery | drug, sex_i) - \mathbf{P}(recovery | placebo, sex_i)) \mathbf{P}(sex_i)$$

Which one is correct?

What would happen if we intervene?

Simpson's paradox

Example 2:

Σ	Recovered	Not recovered	Recovery rate
Drug	273	77	78%
Placebo	289	61	83%

Placebo is 5% more effective

Low pressure by the end of treatment	Recovered	Not recovered	Recovery rate
Drug	81	6	93%
Placebo	234	36	87%

Drug is 5% more effective

High pressure by the end of treatment	Recovered	Not recovered	Recovery rate
Drug	192	71	73%
Placebo	55	25	69%

Drug is 4% more effective

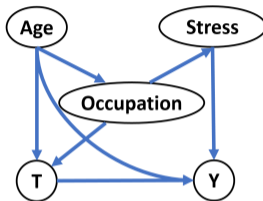
Simpson's paradox

In example 1, conclusion 2 is correct, in example 2 — conclusion 1.

Everything depends on the directions of causal relationships between a feature determining subgroups and the rest of features.

Causal graphs

Causal relationships could be represented on graphs where variables are vertices and directed edges are causal relationships.



Edges — direct causes, directed paths — indirect causes.

Graph encodes all causal assumptions:

- ▶ occupation does affect outcome Y
- ▶ age does not affect stress
- ▶ stress does not affect occupation
- ▶ treatment does not affect stress
- ▶ ...

Elements of causal graph

$A \rightarrow B \rightarrow C$ — **chain**

B — mediator

Example:

- ▶ A — school budget
- ▶ B — average score of graduates
- ▶ C — proportion of students admitted to college

Properties:

1. A and B , B and C are dependent:
 $\exists a, b : \mathbf{P}(B = b | A = a) \neq \mathbf{P}(B = b)$
 $\exists b, c : \mathbf{P}(C = c | B = b) \neq \mathbf{P}(C = c)$
2. C and A are likely dependent
3. $C \perp A | B$ conditionally independent: $\forall a, b, c$

$$\mathbf{P}(C = c | A = a, B = b) = \mathbf{P}(C = c | B = b)$$

(if B is fixed, then A and C are independent)

Elements of causal graph

$$B \leftarrow A \rightarrow C \text{ — fork}$$

A — confounder

Example:

- ▶ A — ice cream sales
- ▶ B — average daily temperature
- ▶ C — number of violent crimes per day

Properties:

1. A and B , A and C are dependent
2. B and C are likely dependent
3. $B \perp C | A$ are conditionally independent

Elements of causal graph

$$B \rightarrow A \leftarrow C \text{ — collider}$$

A — also collider

Example (Monty Hall problem):

- ▶ A — choice of the game host
- ▶ B — choice of the player
- ▶ C — position of the prize

Properties:

1. B and A , C and A are dependent
2. B and C are independent
3. $B \not\perp C|A$ conditionally dependent

Intervention

We need to use observational data to estimate the effect of **intervention**: what would happen with Y if we set the value of T equal to t ?

Notation: $do(T = t)$.

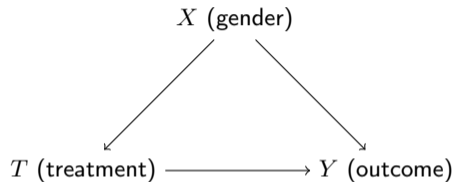
Potential outcomes are outcomes under intervention:

$$Y_{1i} = Y_i | do(T = 1) , Y_{0i} = Y_i | do(T = 0)$$

Hence, causal effect could be represented through intervention:

$$ATE = \mathbb{E}(Y_{1i}) - \mathbb{E}(Y_{0i}) = \mathbb{E}(Y_i | do(T = 1)) - \mathbb{E}(Y_i | do(T = 0))$$

Intervention



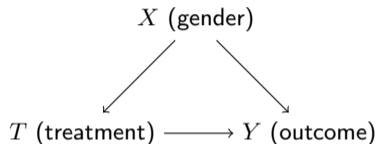
Drug effect in terms of interventions:

$$\text{ATE} = \mathbf{P}(Y = \text{recovery} | do(T = \text{drug})) - \\ - \mathbf{P}(Y = \text{recovery} | do(T = \text{placebo})) .$$

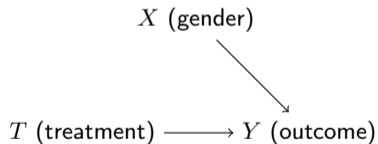
Graph surgery

Graph surgery — removal of all edges directed into treatment variable X .

Example 1, original graph G :



Modified graph G_m :



$$\mathbf{P}(Y = y | do(X = x)) = \mathbf{P}_m(Y = y | X = x)$$

Graph surgery

In the modified graph:

$$\mathbf{P}_m(X = x) = \mathbf{P}(X = x),$$

$$\mathbf{P}_m(Y = y|T = t, X = x) = \mathbf{P}(Y = y|T = t, X = x),$$

because the edges pointing to T and Y did not change \Rightarrow

$$\begin{aligned}\mathbf{P}(Y = y|do(T = t)) &= \mathbf{P}_m(Y = y|T = t) = \\ &= \sum_z \mathbf{P}_m(Y = y|T = t, X = x) \mathbf{P}_m(X = x) = \\ &= \sum_z \mathbf{P}(Y = y|T = t, X = x) \mathbf{P}(X = x).\end{aligned}$$

Graph surgery

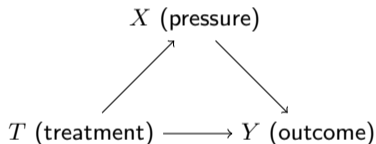
In example 1:

$$\mathbf{P}(Y = \text{recovery} | do(T = \text{drug})) = 0.832,$$

$$\mathbf{P}(Y = \text{recovery} | do(T = \text{placebo})) = 0.7818$$

$$\Rightarrow \text{ATE} = 0.05.$$

In example 2 $G = G_m$:



Therefore,

$$\mathbf{P}(Y = y | do(T = t)) = \mathbf{P}_m(Y = y | T = t) = \mathbf{P}(Y = y | T = t)$$

$$\mathbf{P}(Y = \text{recovery} | do(T = \text{drug})) = 0.78,$$

$$\mathbf{P}(Y = \text{recovery} | do(T = \text{placebo})) = 0.83$$

$$\Rightarrow \text{ATE} = -0.05.$$

Adjustment formula

Adjustment formula allows to calculate the effect of an intervention by conditioning on the vertices of X :

$$\mathbf{P}(Y = y | do(T = t)) = \sum_x \mathbf{P}(Y = y | T = t, X = x) \mathbf{P}(X = x).$$

What is X ?

Causal effect formula:

$$\mathbf{P}(Y = y | do(T = t)) = \sum_x \mathbf{P}(Y = y | T = t, PA = x) \mathbf{P}(PA = x),$$

where PA — parents of T .

Assumptions of conditioning on X

Ignorability (no unmeasured confounders)

Under random experiments, $T \perp X$ for both observed and unobserved covariates.

But conditioning and related techniques can only construct $T \perp X$ for observed covariates.

So assume that after conditioning on observed covariates, any unmeasured covariates are irrelevant:

$$\mathbf{P}(Y_T | X) = \mathbf{P}(Y_T | X, T)$$

Stable Unit Treatment Value (SUTVA) (no spillover)

The effect of treatment on an individual is independent of whether or not others are treated:

$$\mathbf{P}(Y_i | do(T_i, T_j)) = \mathbf{P}(Y_i | do(T_i))$$

Overlap (common support)

There should be overlap on observed covariates between treated and untreated individuals:

$$0 < \mathbf{P}(T = 1 | X = x) < 1$$

Unknown parents

S (socioeconomical status) \rightarrow W (weight)



T (treatment) \longrightarrow Y (outcome)

Socioeconomical status — unobservable variable; how can we estimate the effect of intervention on T ?

More definitions

Path — a sequence of vertices where each vertex is connected to the next one with an edge.

Directed path — a path where all edges have the same direction.

Backdoor path from A to B starts with $A \leftarrow$ and ends with $\rightarrow B$.

A path P is **blocked** by variable X , if:

1. P contains $A \rightarrow B \rightarrow C$, $A \leftarrow B \rightarrow C$, $B \in X$
2. P contains $A \rightarrow B \leftarrow C$, $B \notin X$ and all the descendants of $B \notin X$

Backdoor criterion

For an ordered pair of vertices (A, B) in acyclic graph G a set of vertices X satisfies **backdoor criterion**, if it:

- ▶ X does not contain the descendants of A
- ▶ X blocks all backdoor paths from A to B

If X satisfies backdoor criterion for (T, Y) , then

$$\mathbf{P}(Y = y | do(T = t)) = \sum_x \mathbf{P}(Y = y | T = t, X = x) \mathbf{P}(X = x)$$

(backdoor formula).

Backdoor criterion

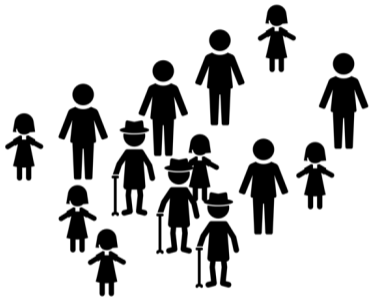
To calculate less conditional probabilities, backdoor formula could be simplified:

$$\begin{aligned}\mathbf{P}(Y = y | do(T = t)) &= \sum_x \mathbf{P}(Y = y | T = t, X = x) \mathbf{P}(X = x) = \\ &= \sum_x \frac{\mathbf{P}(Y = y, T = t, X = x)}{\mathbf{P}(T = t | X = x)}\end{aligned}$$

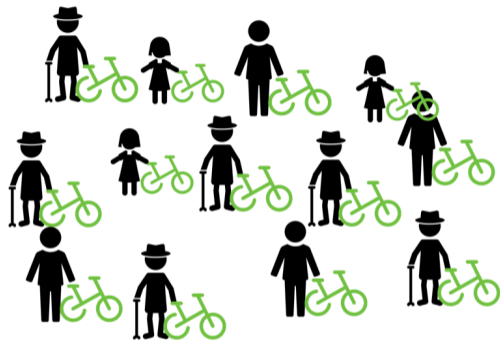
This way

- ▶ the method is called **inverse probability weighting**
- ▶ denominator $e_i = \mathbf{P}(T = t | X = x)$ — propensity score.

Biking vs Cholesterol



Avg Cholesterol = 200



Avg Cholesterol = 206

Regression

Model Y as a function of T and X :

$$Y = \beta_0 + \beta_1 X_1 + \dots + \beta_k X_k + \alpha T + \varepsilon,$$

i.e., $Cholesterol = \beta_0 + \beta_1 \cdot Age + \alpha \cdot Exercise + \varepsilon$.

$\hat{\alpha}$ — an estimate of the average effect of changing T from 0 to 1, **if** among X_1, \dots, X_k there are:

- ▶ all the parents of T , or a set of variables that satisfies backdoor criterion for (T, Y)
- ▶ no colliders of T and Y

Also, the model must be true.

Matching



Matching

- ▶ Paired individuals provide the counterfactual estimate for each other
- ▶ Reduces sample size
- ▶ Could be approximate:
 - ▶ on distances in X space
 - ▶ on propensity scores $e_i = \mathbf{P}(T = 1 | X = x)$

Stratification

180



180



200



190



240



230



Stratification

- ▶ Many:many matching
- ▶ Stratum sizes — bias-variance tradeoff
- ▶ You can stratify on binned propensity scores! But they must be well-calibrated.

Weighting

Propensity scores could be used as weights:

$$\widehat{\text{ATE}} = \frac{1}{N_1} \sum_{i: T_i=1} w_i Y_i - \frac{1}{N_0} \sum_{j: T_j=1} w_j Y_j,$$
$$w_i = \frac{T}{e_i} + \frac{1 - T}{1 - e_i}$$

Inverse Probability of Treatment Weighting (IPTW).

- ▶ High variance when e_i close to 0 or 1 (could be stabilized heuristically)
- ▶ Assumes propensity score model is correctly specified

Doubly robust

Combines models $\hat{Y}_{T=t}$ and propensity scores \hat{e} :

$$DR_1 = \begin{cases} \frac{Y}{\hat{e}} - \frac{\hat{Y}_{T=1}(1-\hat{e})}{\hat{e}}, & T = 1, \\ \hat{Y}_{T=1}, & T = 0; \end{cases}$$
$$DR_0 = \begin{cases} \hat{Y}_{T=0}, & T = 1, \\ \frac{Y}{1-\hat{e}} - \frac{\hat{Y}_{T=1}\hat{e}}{1-\hat{e}}, & T = 0 \end{cases}$$

Causal effect on T — difference between mean DR_1 and DR_0 .

- ▶ Works if at least one of two is correctly specified
- ▶ But if both propensity score or regression are slightly incorrect, may become very biased

Causal analysis simple checks

- ▶ Adding random covariates should not change the analysis
- ▶ AA-test: randomizing the treatment should turn causal effect into 0
- ▶ Subsampling should not change the conclusions

References

- ▶ theory:
 - ▶ Pearl J., Glymour M., Jewell N.P. *Causal Inference in Statistics: A Primer*, 2016
 - ▶ Pearl J., Mackenzie D. *The Book of Why: The New Science of Cause and Effect*, 2018
 - ▶ Morgan S.L., Winship C. *Counterfactuals and Causal Inference* (2015, 2nd ed)
- ▶ good introduction: <https://causalinference.gitlab.io/kdd-tutorial/>
- ▶ implementations:
 - ▶ <http://www.bnlearn.com/> (R)
 - ▶ <https://github.com/microsoft/dowhy> (Python)