Methods for Causal Inference
Lecture 1

Ava Khamseh
School of Informatics

2021-2022

Logistics

These lectures are being recorded.

- Lectures: **Mondays** and **Thursdays** at **10:00-11:00am**
- Tutorials: Every other week **Wednesdays 12:00-1:00pm** 40GS_LG.07 Teaching Studio, **first session: 26/01/2022**
- Slides and recordings will appear on Learn
- Office Hours: Wednesdays 15:00-17:00
- Email me any questions, happy to discuss!

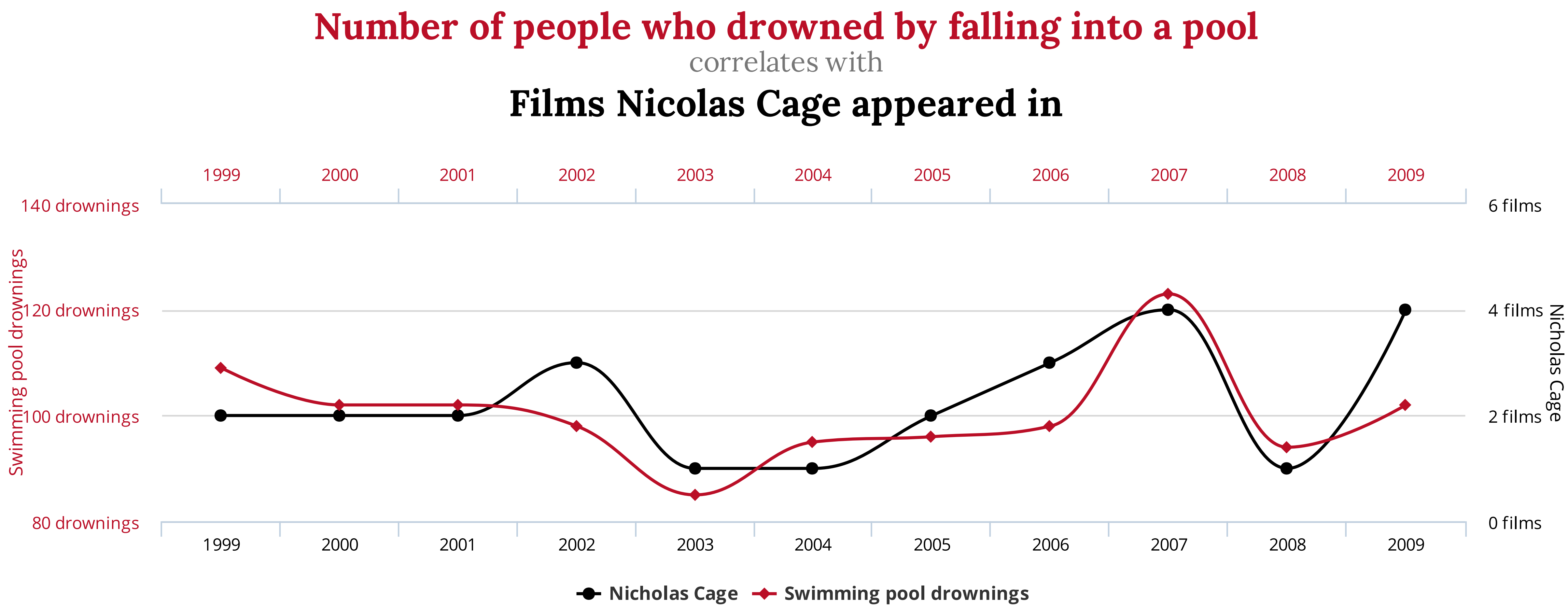
References

- Causal Inference in Statistics: A Primer (Pearl, Glymour, Jewell, 2016)
- More advanced: Causality (Pearl, 2009)
- Elements of Causal Inference: Foundations and Learning Algorithms (Peters, Janzing and Schölkopfk)
- Many other papers from the literature … (will be referenced)

“Correlation does not imply causation”

“Correlation does not imply causation”

Spurious correlation (random coincidence)

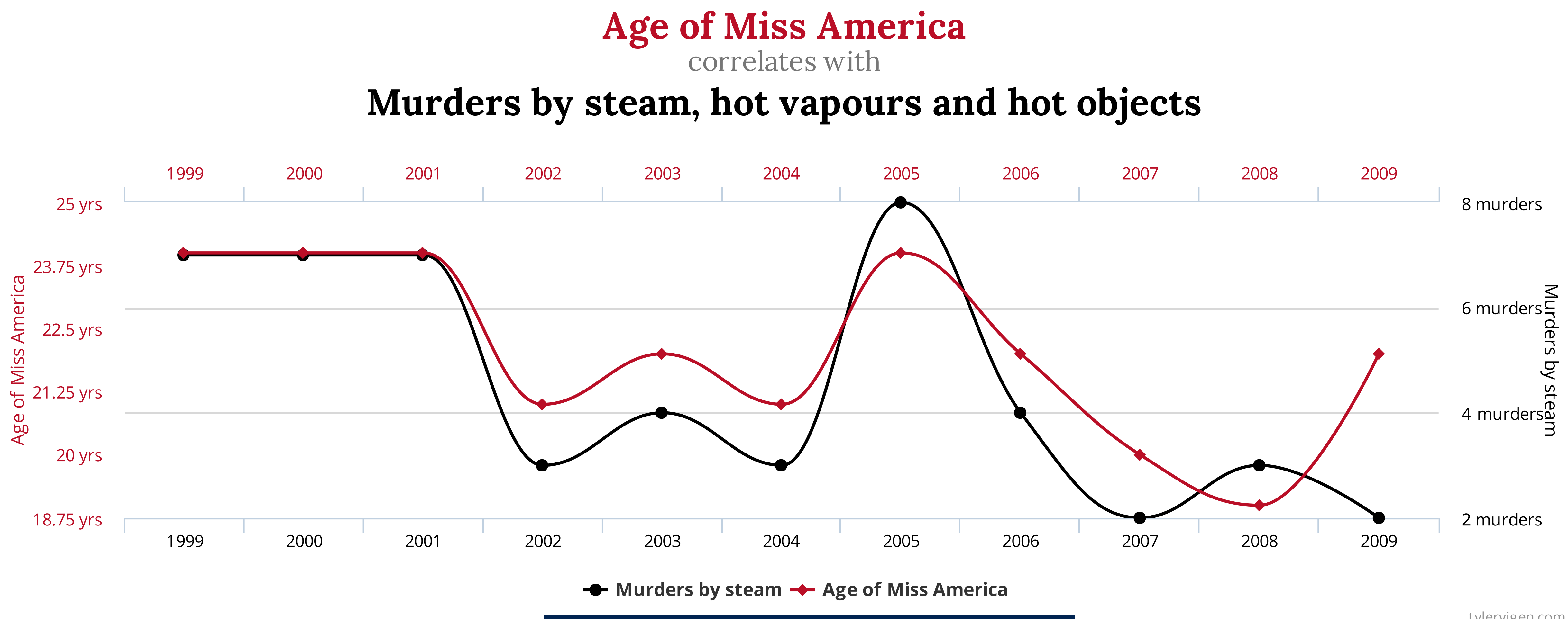


Correlation: 66.6% (r=0.666)

tylervigen.com/spurious-correlations

> “Correlation does not imply causation”

Spurious correlation (random coincidence)

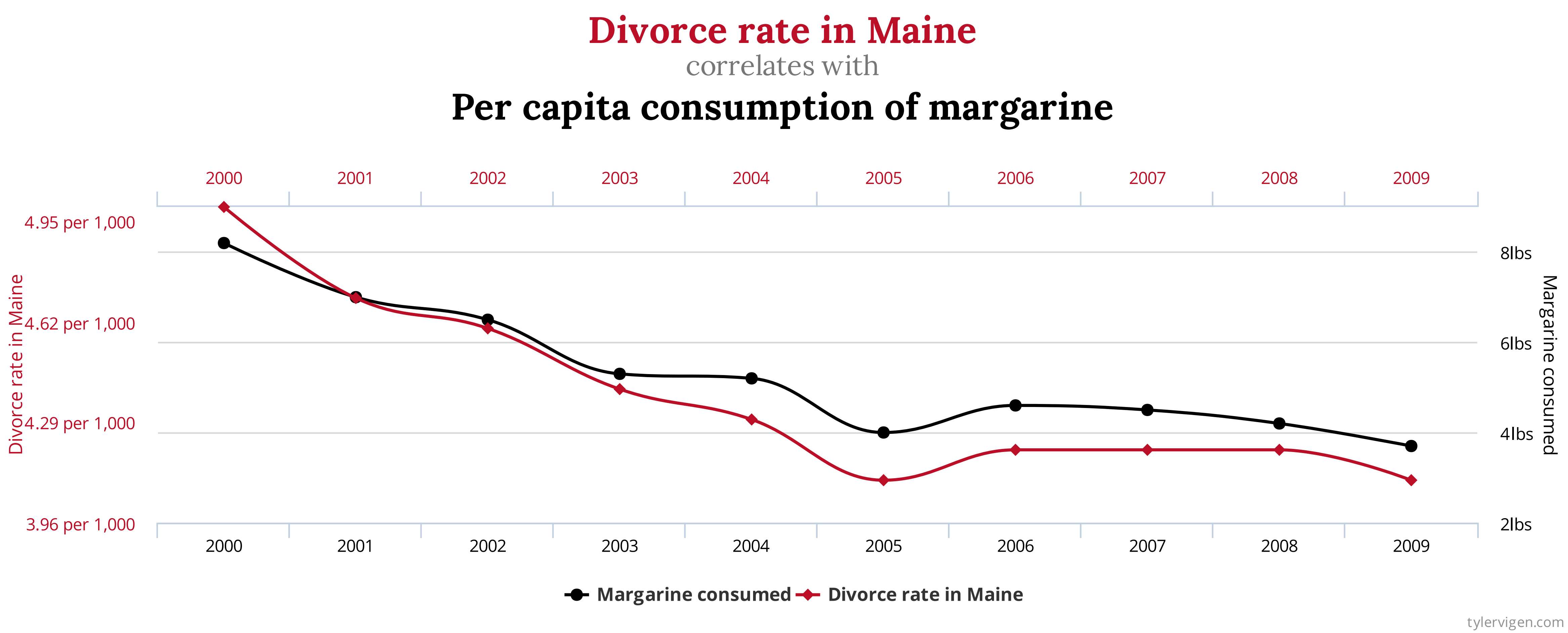


Correlation: 87.01% (r=0.8701)

tylervigen.com/spurious-correlations

“Correlation does not imply causation”

Spurious correlation (random coincidence)



Correlation: 99.26% (r=0.9926)

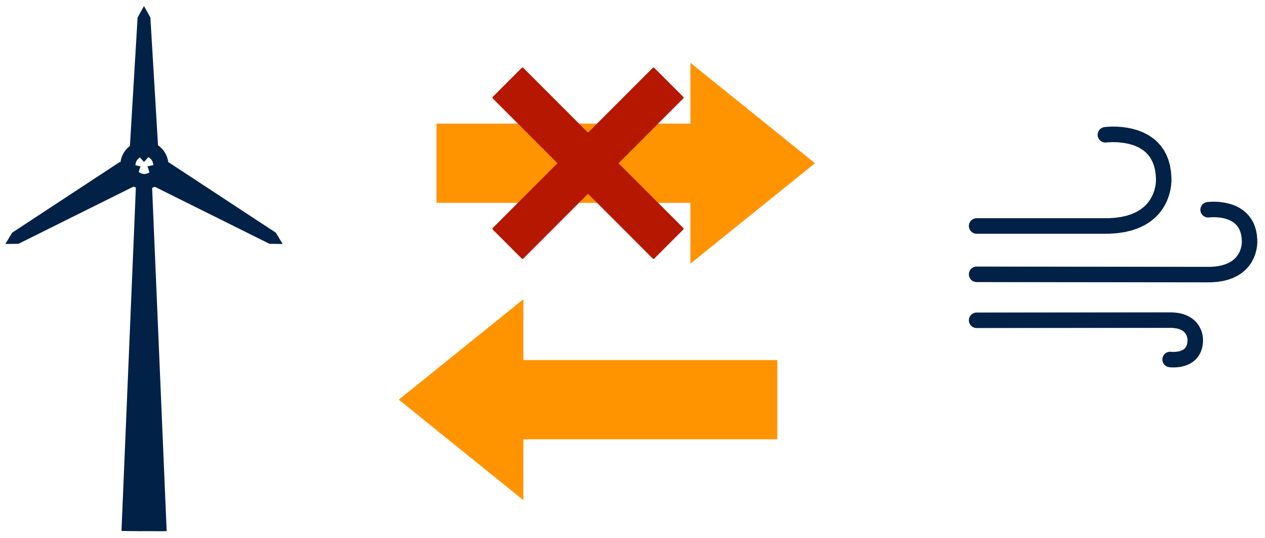
tylervigen.com/spurious-correlations

“Correlation does not imply causation”

Reverse causation:

The faster the wind-turbine rotates, the more wind is observed.

Therefore, rotation of turbines is the cause for winds!



“Correlation does not imply causation”

you can argue both ways

Circular/bidirectional cause and consequence:

Hours spent on Netflix and weight gain

! If we aren't given enough data it can be hard to guess which way it goes => what influences what !

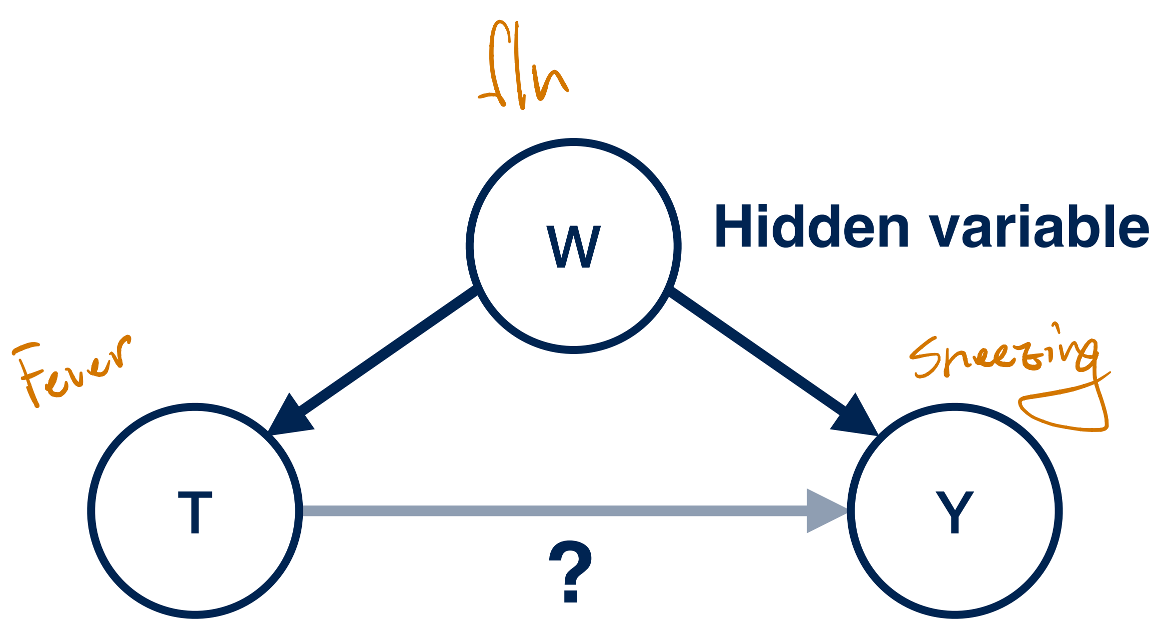
Hours spent on Netflix -> Less activity -> increase in weight

Weight gain -> exercising becomes harder -> more time online as hobby

“Correlation does not imply causation”

Confounding factor:

- Fever is not a cause of sneezing, they are both symptoms of flu (No arrow)
- Treatment outcome relationship confounded by age



Why should we care about **causation**?

- To guide **actions** and **policies**:
- To understand *how* and *why* **interventions** affect outcomes
- **Predict** what would have happened under a different intervention:
  "What if I were to act differently?"

Why should we care about **causation**?

- To guide **actions** and **policies**:
- To understand *how* and *why* **interventions** affect outcomes
- **Predict** what would have happened under a different intervention: "What if I were to act differently?"

- Controversial examples:

due to FALSE claims regarding CAUSATION

- **Biomedical**: "Vaccines lead to autism"
- **Political/Economical**: "increases minimum wage, increases unemployment (people become lazy)"

Why should we care about **causation**?

- To guide **actions** and **policies**:
- To understand *how* and *why* **interventions** affect outcomes
- **Predict** what would have happened under a different intervention: "What if I were to act differently?"

- Examples:
- **Biomedical**: What drug, what dose, when, how often, … (see later)
- **Political**: How social media posts from famous individuals (e.g. celebrities, ex-political figures, etc.) influence elections
- **Environmental**: Is the constant energy consumption in region X due to the regions's energy efficiency standards or due to its mild climate, demographics
- **Education**: People with feature X are more likely to obtain an internship in tech

Personalised Medicine

EXAMPLE

- Patient diagnosed with a particular **disease**

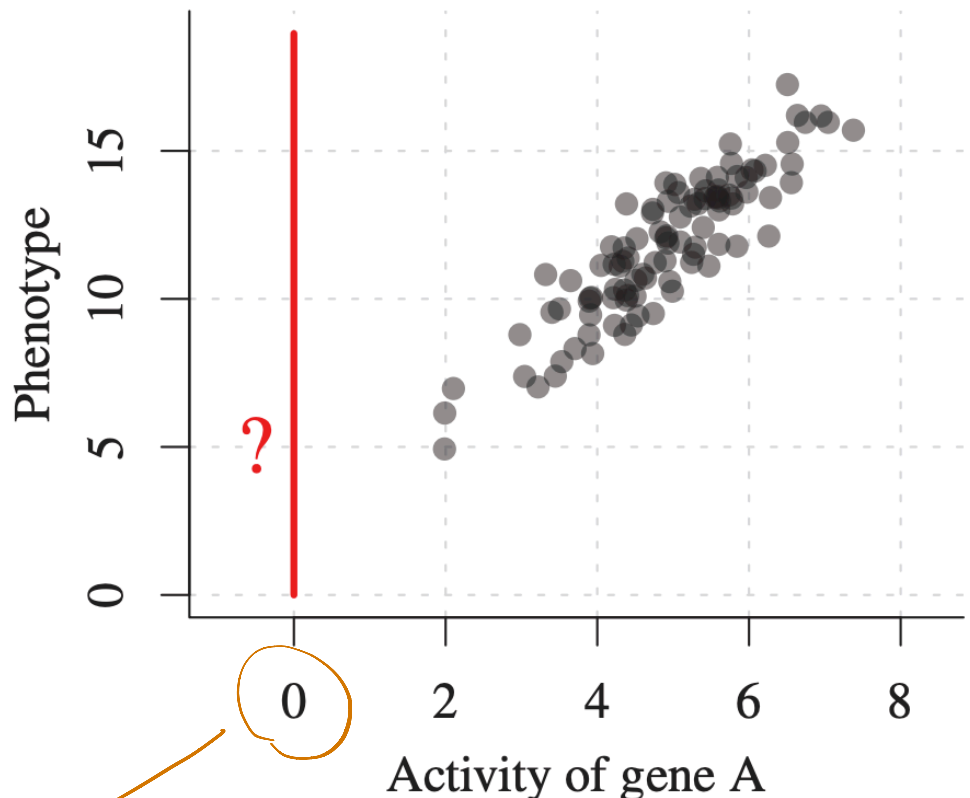
- Certain **baseline covariates** are known, e.g. age, weight, BMI, blood sugar, …

  → our response/decision can be different based on these features

- **Question:** Should **treatment A** or **treatment B** be given
  - What is the causal effect of A vs B
  - Design a **policy**: Features —> {A,B}
  - i.e. best treatment for **a given individual**

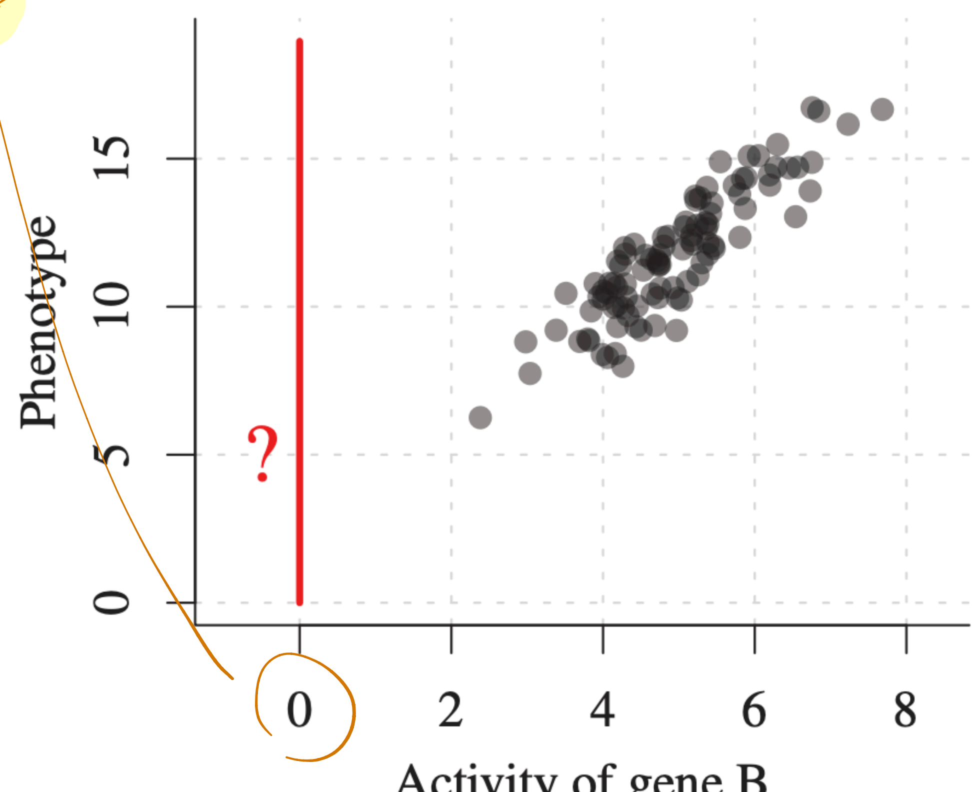
- Source: **Electronic Health Records**

Gene Perturbation



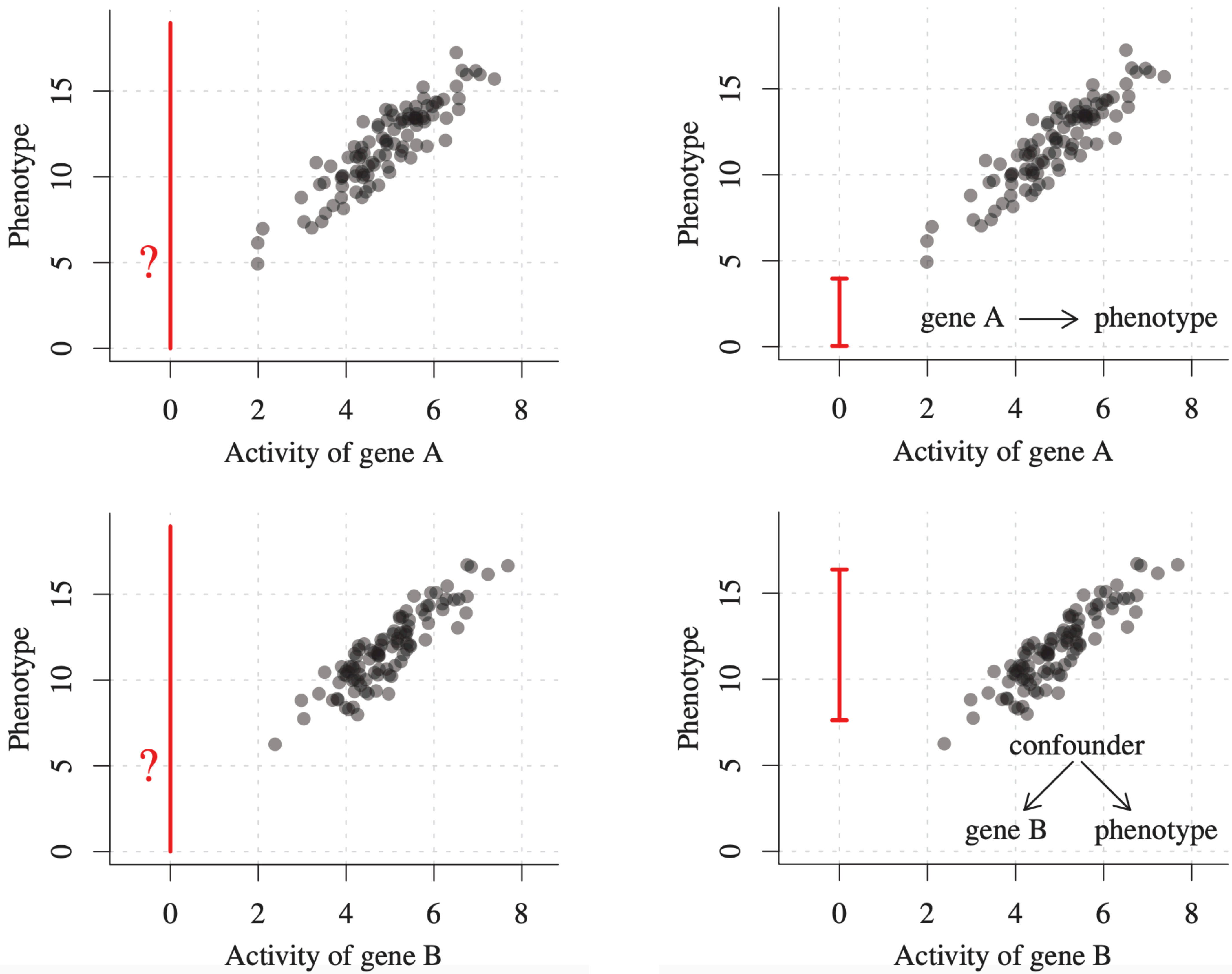
Is one of the genes (A|B) causal? Or is it just correlational?

0 = no activity



Elements of Causal Inference, Peters et al.

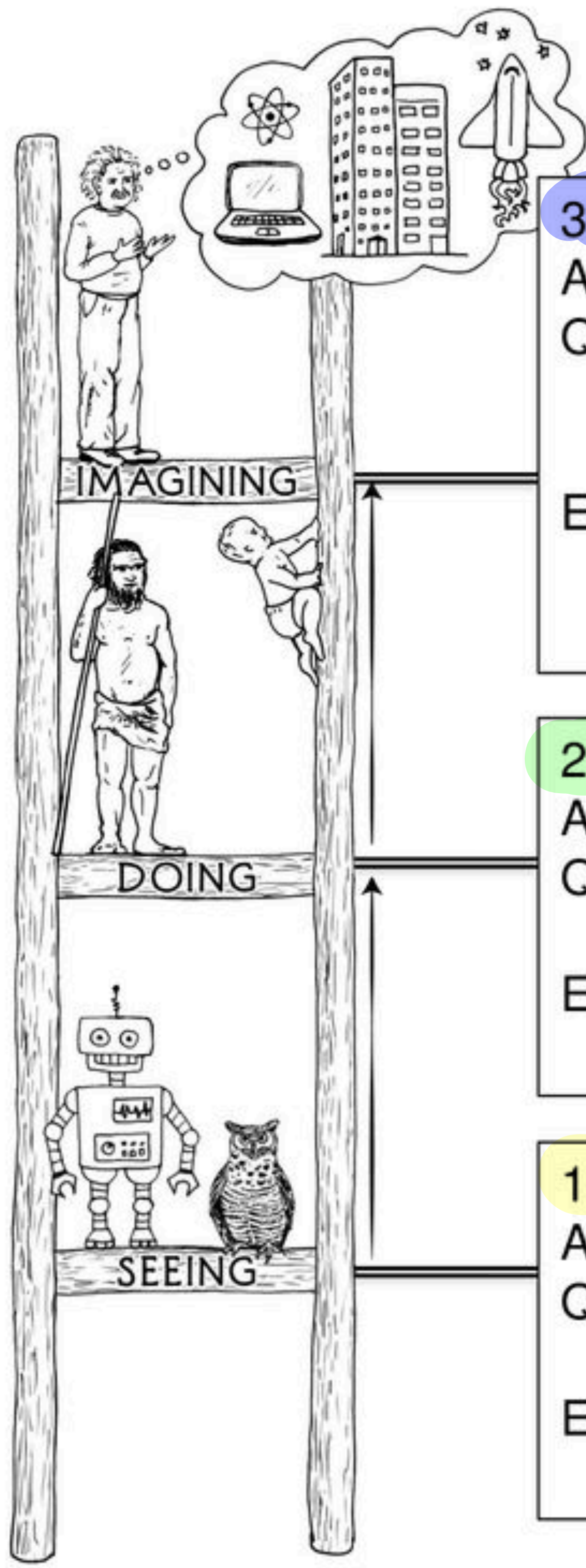
Gene Perturbation



Elements of Causal Inference, Peters et al.

Pearl's ladder of causation

making a change



3-LEVEL HIERARCHY

3. COUNTERFACTUALS
ACTIVITY: Imagining, Retrospection, Understanding
QUESTIONS: *What if I had done . . . ? Why?*
(Was it X that caused Y? What if X had not occurred? What if I had acted differently?)
EXAMPLES: Was it the aspirin that stopped my headache? Would Kennedy be alive if Oswald had not killed him? What if I had not smoked the last 2 years?

2. INTERVENTION
ACTIVITY: Doing, Intervening
QUESTIONS: *What if I do . . . ? How?*
(What would Y be if I do X?)
EXAMPLES: If I take aspirin, will my headache be cured? What if we ban cigarettes?

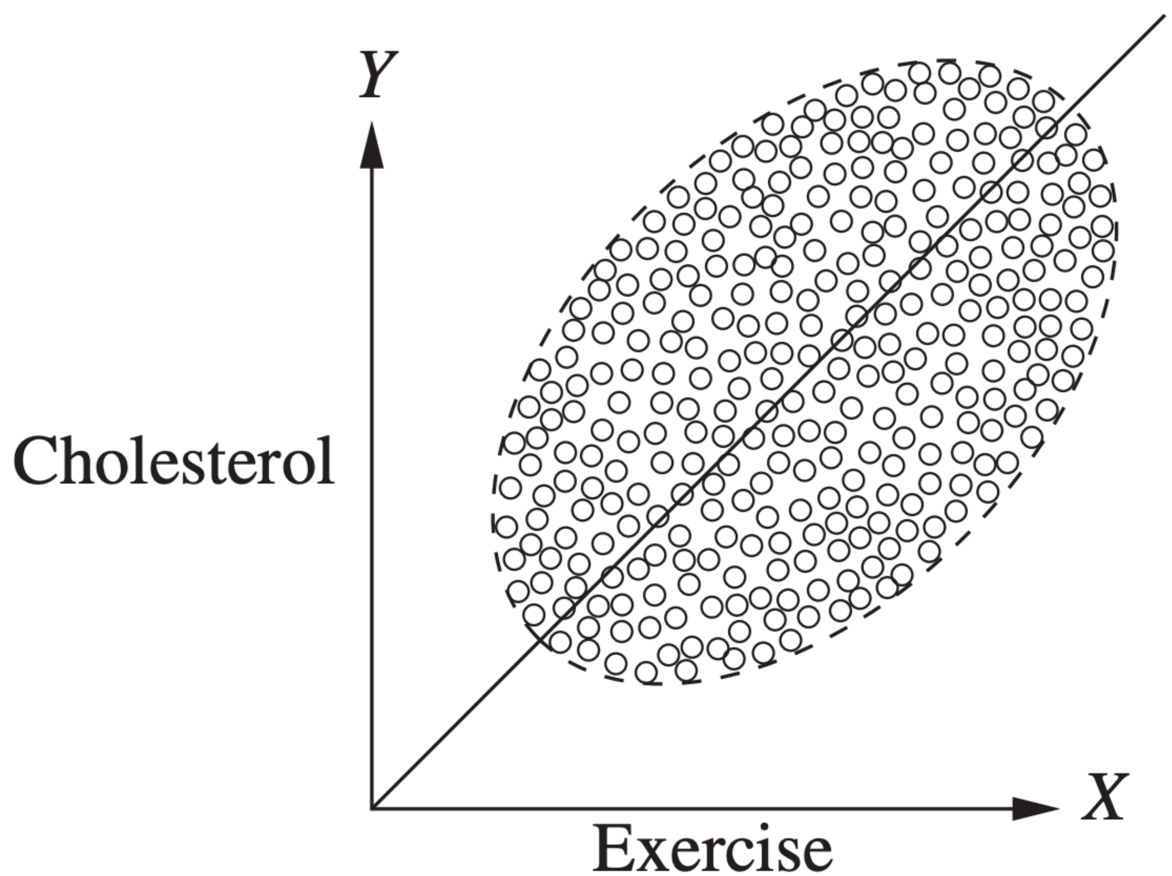
1. ASSOCIATION
ACTIVITY: Seeing, Observing
QUESTIONS: *What if I see . . . ?*
(How would seeing X change my belief in Y?)
EXAMPLES: What does a symptom tell me about a disease? What does a survey tell us about the election results?

Simpson's Paradox

- Why concluding causality from purely associational measures, i.e. correlation, can be **very wrong** (not just neutral): "It would have better not to make any statements!"

Simpson's Paradox – a phenomenon in probability and statistics in which a trend appears in several groups of data but disappears or reverses when groups are joined together

can be resolved by identifying CONFOUNDING VARIABLES and CAUSAL RELATIONS ARE APPROPRIATELY ADDRESSED.



A well-known example of that:

UC Berkely Gender Bias

UC Berkeley gender bias [edit]

One of the best-known examples of Simpson's paradox comes from a study of gender bias among graduate school admissions to University of California, Berkeley. The admission figures for the fall of 1973 showed that men applying were more likely than women to be admitted, and the difference was so large that it was unlikely to be due to chance.[13][14]

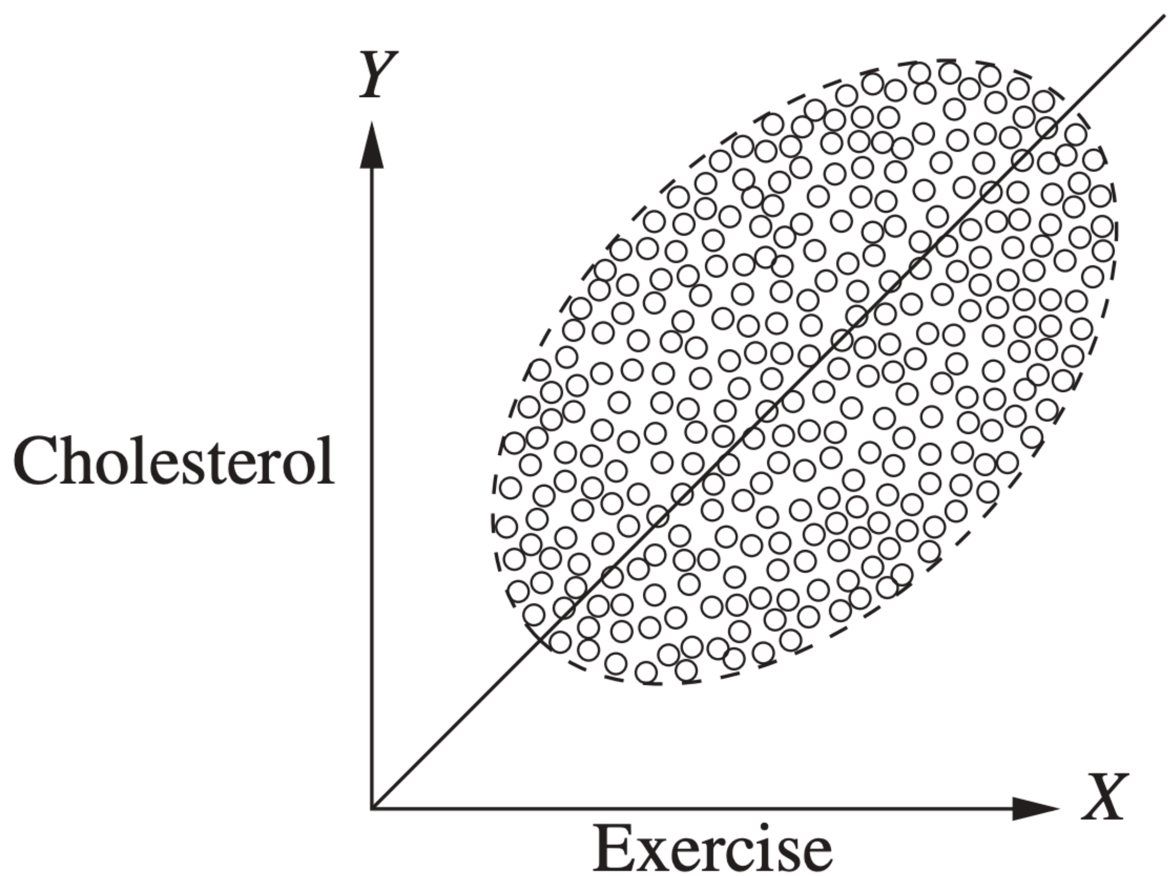
	All		Men		Women	
	Applicants	Admitted	Applicants	Admitted	Applicants	Admitted
Total	12,763	41%	8,442	**44%**	4,321	35%

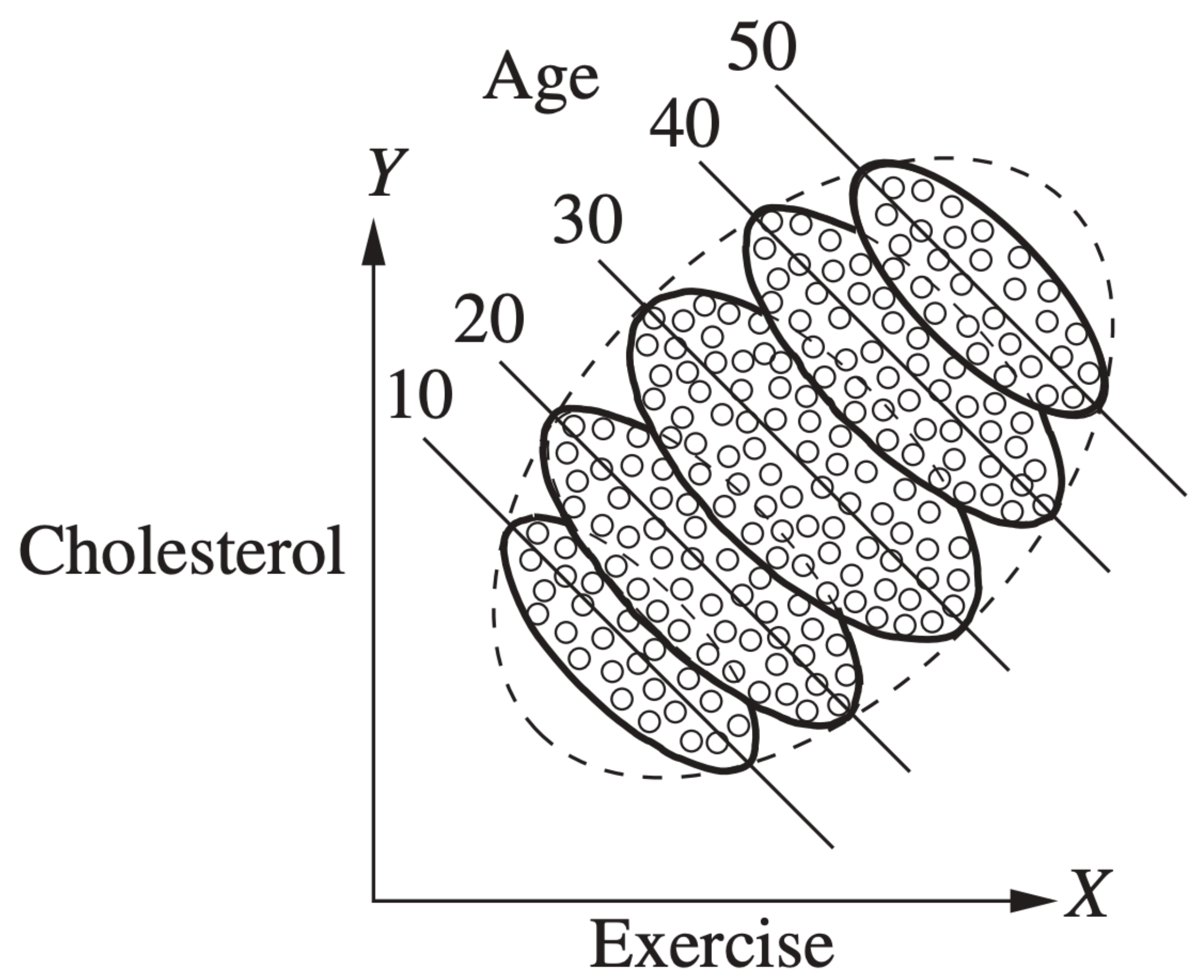
However, when examining the individual departments, it appeared that 6 out of 85 departments were significantly biased against men, while 4 were significantly biased against women. In total, the pooled and corrected data showed a "small but statistically significant bias in favor of women".[14] The data from the six largest departments are listed below, the top two departments by number of applicants for each gender italicised.

Department	All		Men		Women	
	Applicants	Admitted	Applicants	Admitted	Applicants	Admitted
A	933	64%	***825***	62%	108	**82%**
B	585	63%	***560***	63%	25	**68%**
C	918	35%	325	**37%**	***593***	34%
D	792	34%	417	33%	375	**35%**
E	584	25%	191	**28%**	***393***	24%
F	714	6%	373	6%	341	**7%**
Total	4526	39%	2691	45%	1835	30%

Simpson's Paradox

- Why concluding causality from purely associational measures, i.e. correlation, can be **very wrong** (not just neutral): "It would have better not to make any statements!"





Causal Inference in Statistics, Pearl (2016)

Example with numbers

We will come back to this example after having built the causal calculus how the causal effect can be detected accurately (without us suffering …)

Language of causality and the roles of variables

"What intervention", "how much", "when", "how often", "Control", "effect of", "why did", "what if", …

Causality language

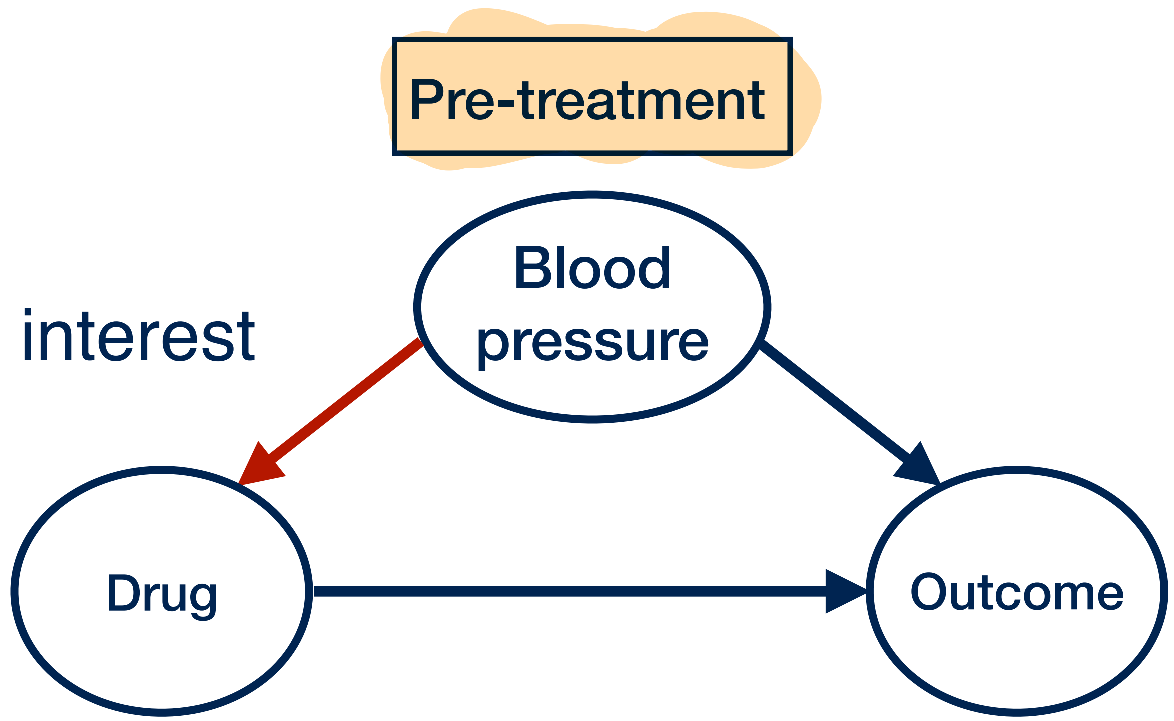
Consider all variables affecting the system of interest and **the role each plays.**

Patient: Info on DNA variants and biomarkers, traits/disease, confounders
Clinician: What drug, what dose, when, how often, …

a variable that influences both the dependent and independent variable ⇒ causing a suspicious association

Consider all variables affecting the system of interest and **the role each play** (as far as possible)

Blood pressure is a **confounder** here:



Language of causality and the roles of variables

“What intervention”, “how much”, “when”, “how often”, “Control”, “effect of”, “why did”, “what if”, …

Causality language

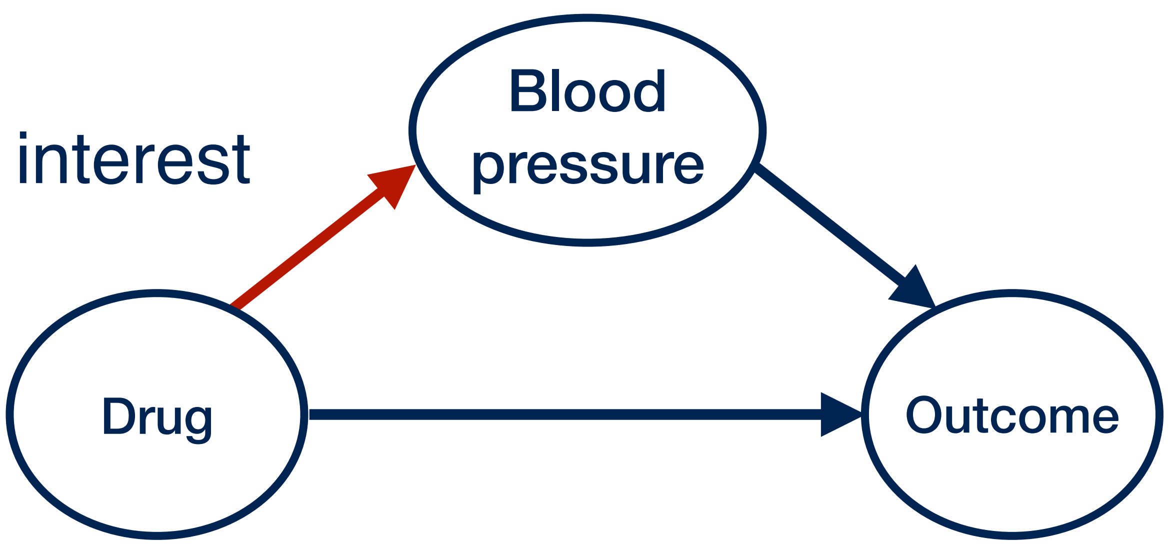
Consider all variables affecting the system of interest and **the role each plays.**

Patient: Info on DNA variants and biomarkers, traits/disease, confounders
Clinician: What drug, what dose, when, how often, …

Post-treatment

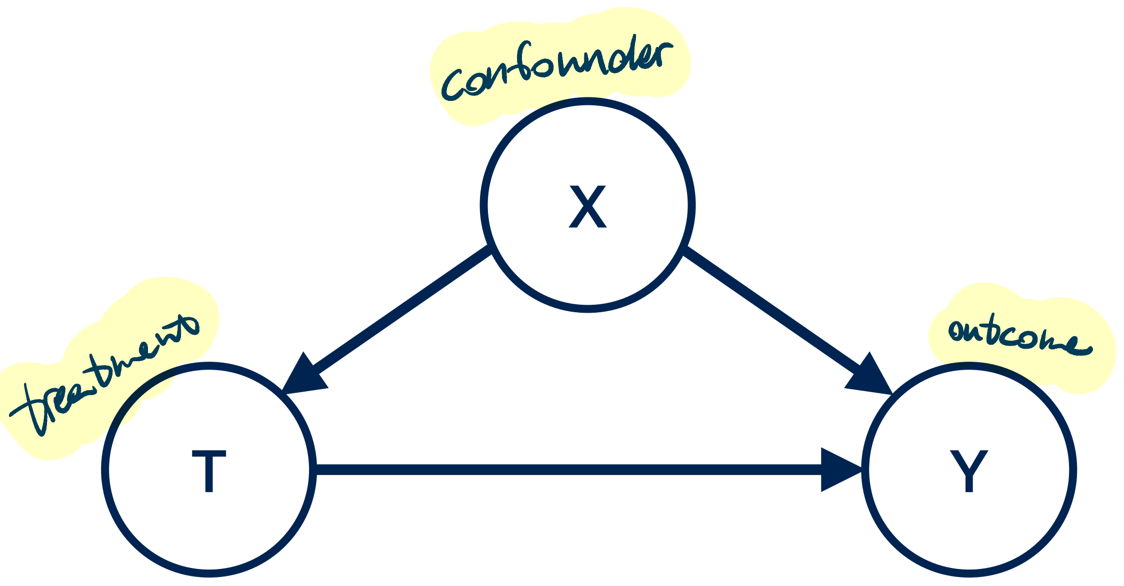
Consider all variables affecting the system of interest and **the role each play** (as far as possible)

Blood pressure is a **mediator** here:



Conventions

- Variable to be manipulated: **treatment (T)**, e.g. drug
- Variable we observe as response: **outcome (Y)**, e.g. success/failure of drug
- Other observable variables that can affect treatment and outcome causally and we wish to correct for: **confounders (X)**, e.g. age, sex, socio-economic status, …
- Unobservable confounder **(U)**



Causal Estimation of Effects

- Have a prior causal knowledge (may be incomplete) and know the treatment/outcome pair, c.e., weight gain, hours online

- Interested in estimating the **effect size**:

extended version:

Expected Value

treatment applied

treatment isn't applied

$$\mathbb{E}\left[y_{t=1}(x) - y_{t=0}(x)\right] = \int \big(y_1(x) - y_0(x)\big)p(x)dx$$

X – features of confounders

p(x) – distribution of patients in the population

Note: The features/confounders x for both treatment and control groups are drawn from the **same** distribution p(x)

- Goal: Find an **unbiased estimator**, e.g. signal/noise ratio

Randomised experiments: Already in causal framework

- In a **randomised experiment**, p(x) is designed to be the same for both treatment groups (t=0 or t=1), typically uniform
- Paired '**clones**' in treatment and outcome groups
- Simply take the difference of the averages:

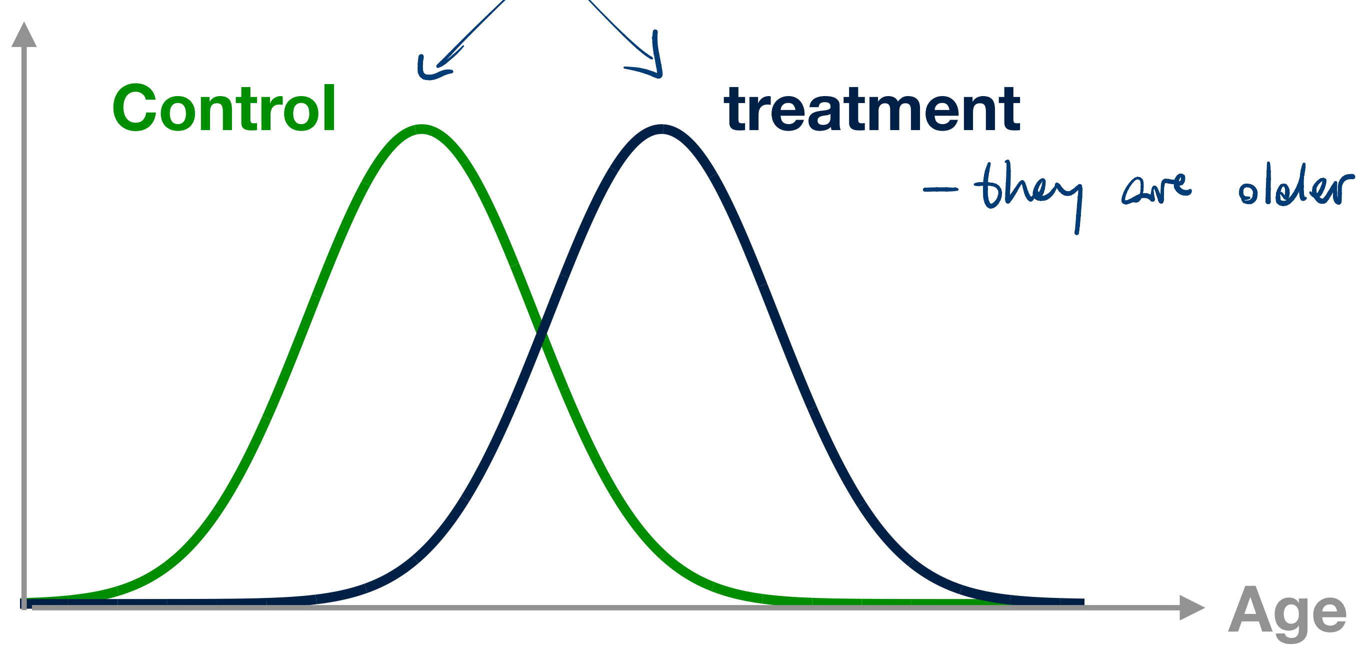
$$\Delta\hat{\mu} = \hat{\mathbb{E}}\big[y_{t=1}(x) - y_{t=0}(x)\big] = \frac{1}{N}\sum_{i=1}^{N}\big(y_1^{(i)}(x) - y_0^{(i)}(x)\big)$$

- Statistical test: e.g. T-test and p-values …

$$\frac{\Delta\hat{\mu}}{\sqrt{\frac{(\hat{\sigma}_{\Delta\mu})^2}{N}}} > t^*$$

Observational data: What goes wrong?

$$p(x|t=1) \neq p(x|t=0)$$



– they are older! #noBueno

$$\left(\int y_1(x)p(x|t=1)dx \; - \int y_0(x)p(x|t=0)dx\right) \neq \int \big(y_1(x) - y_0(x)\big)p(x)dx$$

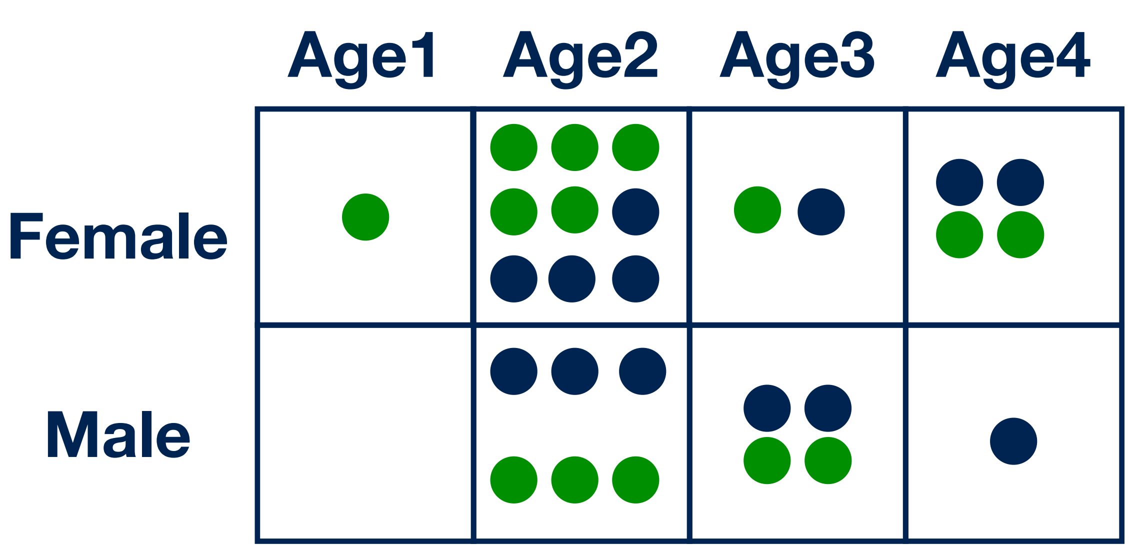
Observational data: Stratification

- Measure outcome (success/failure), **within each** of the young/old groups **separately**
- Take weighted average by the probability of being young/old

$$\mathbb{E}(\text{Healed}|t=1) = \mathbb{E}(\text{Healed}|t=1, \text{young})p(\text{young}) + \mathbb{E}(\text{Healed}|t=1, \text{old})p(\text{old})$$

weighted average!

- Disadvantages:
    - All possible confounders need to be observed
    - Assumes overlap between the two distributions (if there is no overlap, sample is not representative, e.g. performing the experiment only for old people)
    - Bad estimates as confounder dimensionality increases



$\Rightarrow$

Need specific causal effect estimation techniques

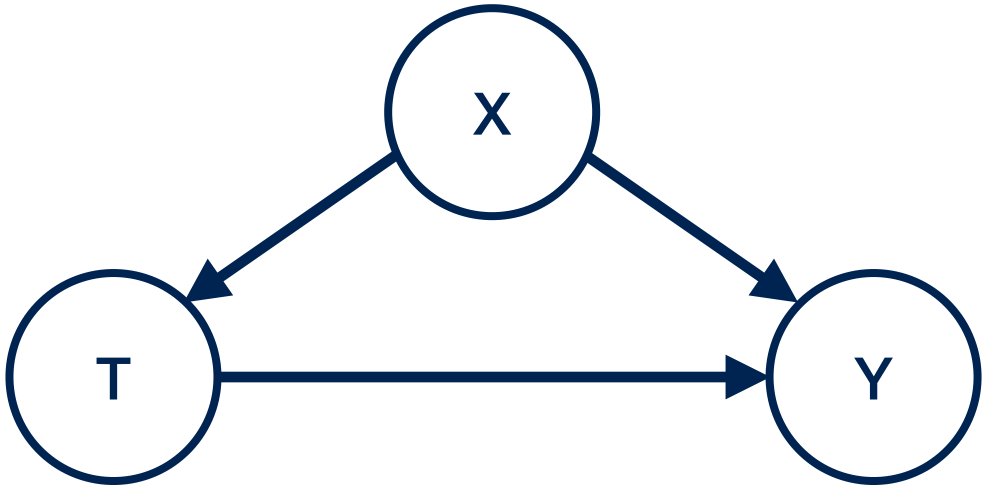
Two main Frameworks for causal estimation/discovery

- **Potential outcomes (Rubin):**

    - Requires a given treatment-outcome pair (known directionality)
    - Mainly applies to causal estimation (learning effects)
    - More familiar to biologists

- **Structural causal models (Pearl):**

    - Causal graph
    - Structural equations
    - Algorithmic: Causal Discovery



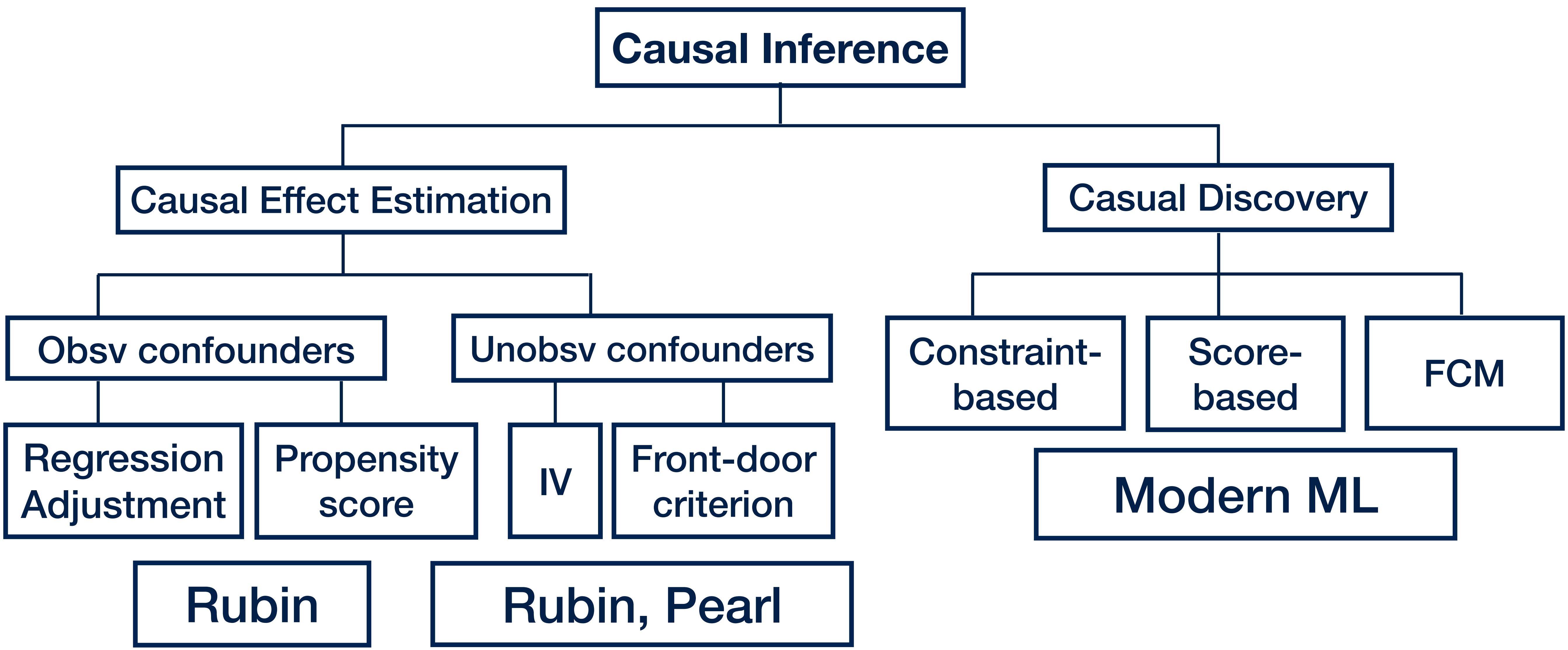
$$x = f_x(\epsilon_x),\ t = f_t(x, \epsilon_t),\ y = f_y(x, t, \epsilon_y)$$

Extend the language of probability theory:
do-calculus

Assumption: Independent noise terms: $\epsilon_x \perp\!\!\!\perp \epsilon_t \perp\!\!\!\perp \epsilon_y$

Overview of the course

- **Lecture 1**: Introduction & motivation, why do we care about causality?
- **Lecture 2**: Recap of probability theory, e.g., variables, events, conditional probabilities, independence, law of total probability, Bayes' rule
- **Lecture 3**: Recap of regression, multiple regression, graphs, SCM
- **Lectures 4-20**:



Causal Estimation of Effects vs Causal Discovery

- **How much would some variables (features or labels) change if we manipulate the value of another variable?**
    - Have a prior causal knowledge (may be incomplete)
    - Wish to estimate degrees of causal dependencies

- **By modifying the value of which variables could we change the value of another variable?**
    - Wish to discover the causal graph
    - Apply causal inference