

Causality

An introduction for ML practitioners

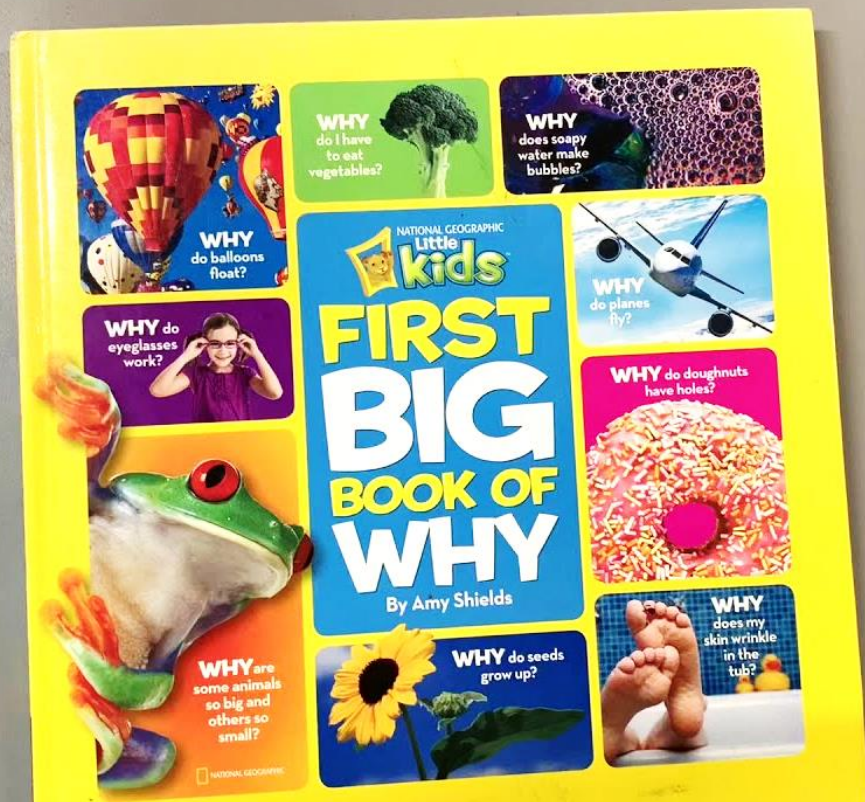
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Why?



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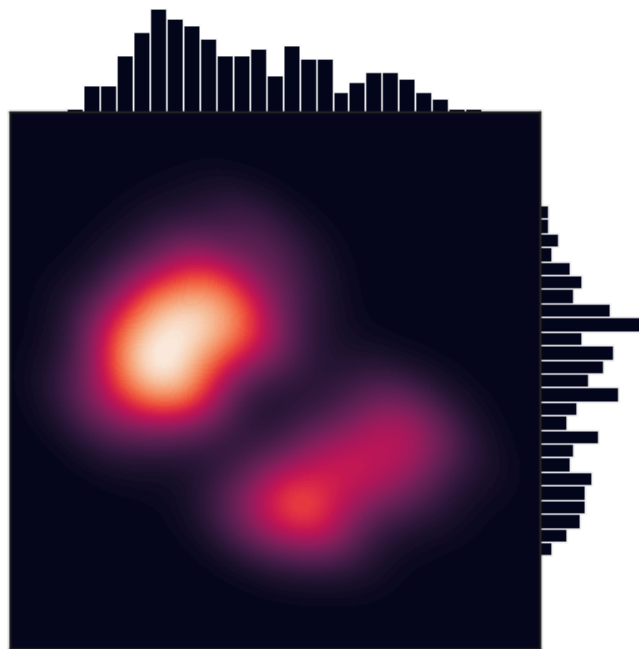


Cause and effect are integral to decision-making:

- What is the cause of these symptoms?
→ Decide between different treatments.
- Why is the cost of living so high?
→ Determine which economic intervention is more effective.
- Why did my computer break down?
→ Decide to update software or replace a part.

...

Why?

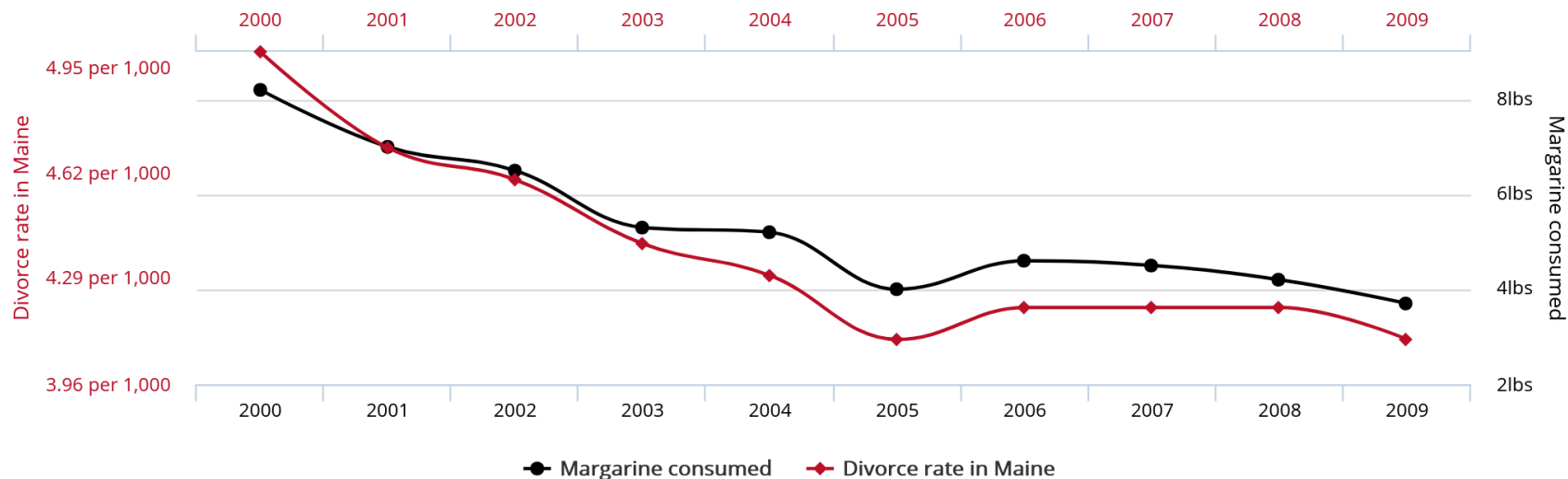


- Statistics and probability is usually concerned with the *description* of data, i.e. inference:
 - ↳ Finding a parsimonious description of the **joint probability distribution** of data.
- Inference and hypothesis testing **cannot** answer the questions discussed in the previous slide – “**why**” and “**what if**” – used in decision-making.
- However, making **informed decisions** is often why we reach for statistics in the first place!

Correlation \neq causation

Divorce rate in Maine correlates with Per capita consumption of margarine

Correlation: 99.26% ($r=0.992558$)



Data sources: National Vital Statistics Reports and U.S. Department of Agriculture

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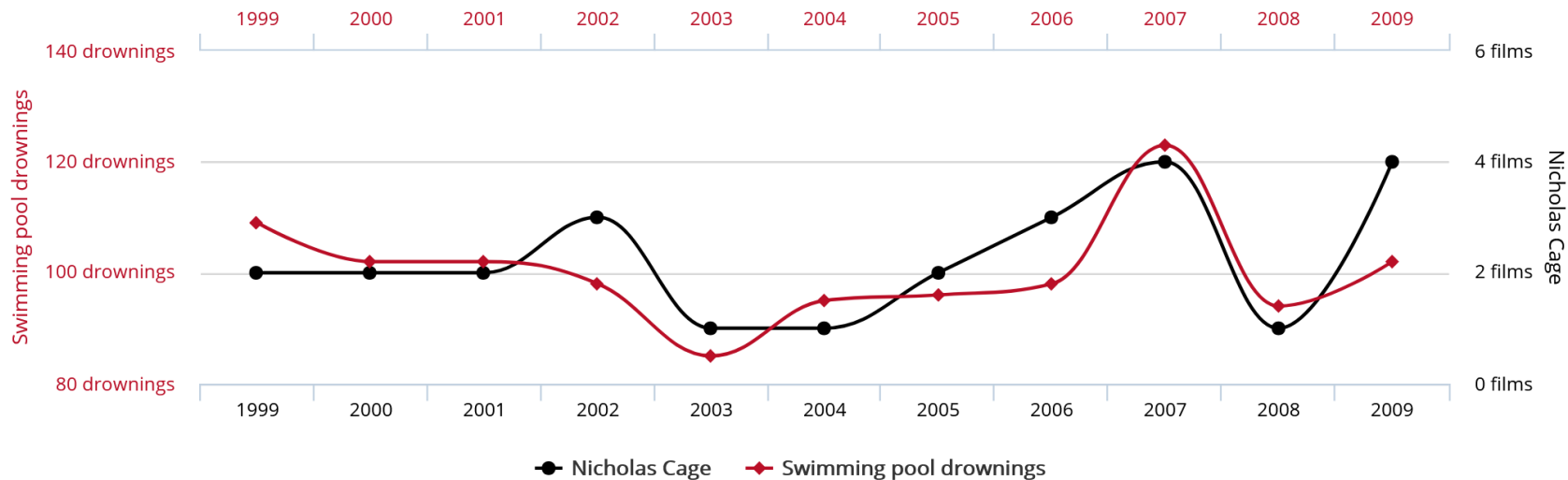
Correlation \neq causation

Number of people who drowned by falling into a pool

correlates with

Films Nicolas Cage appeared in

Correlation: 66.6% ($r=0.666004$)



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Data sources: Centers for Disease Control & Prevention and Internet Movie Database

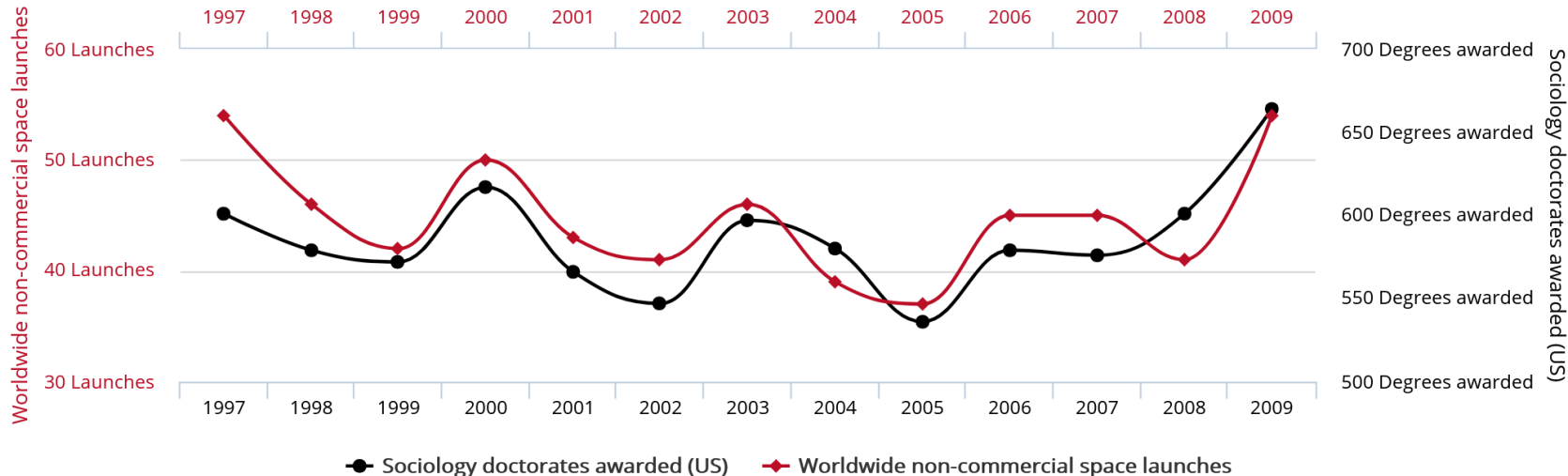
Correlation \neq causation

Worldwide non-commercial space launches

correlates with

Sociology doctorates awarded (US)

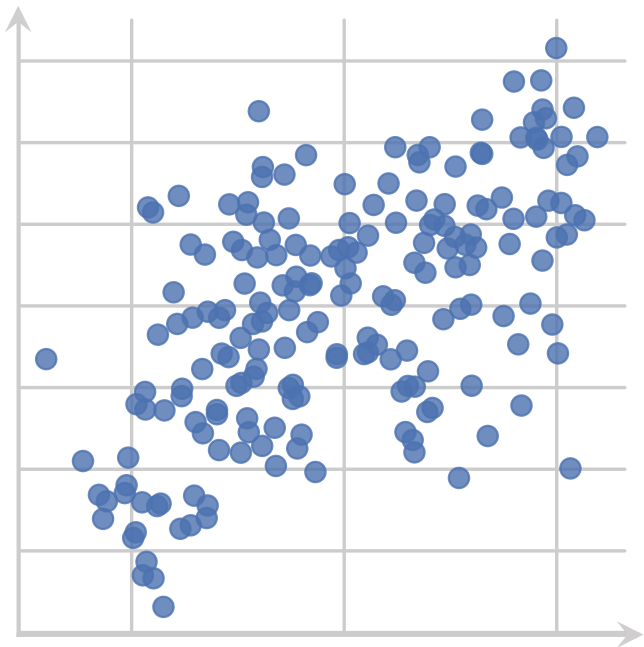
Correlation: 78.92% ($r=0.78915$)



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Data sources: Federal Aviation Administration and National Science Foundation

Simpson's paradox

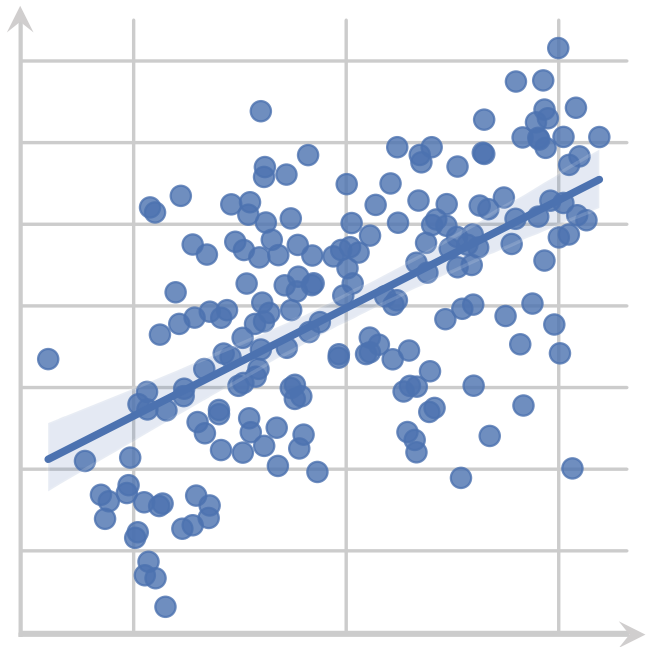


- A dataset of 2 variables, plotted as (X, Y) .
- Collected to answer an empirical question (e.g. hypothesis in biology, performance of ML methods vs. some factors...).

Simpson's paradox

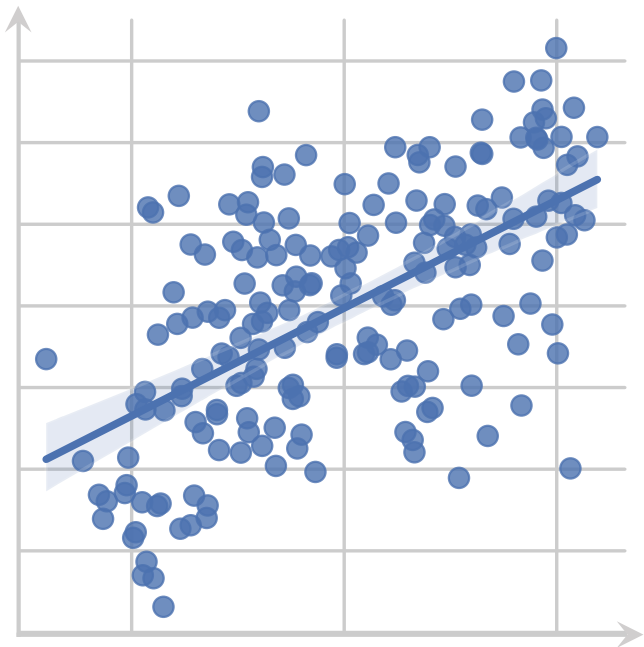


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- Both variables seem to be very correlated, with an “ok” linear fit.
- A statistical study could conclude that one variable *may* cause the other (with the usual caveat that correlation \neq causation).
- Trust the data! Are we done?

Simpson's paradox



- What if we know more about the domain (*not encoded in the statistics*) that leads us to doubt this conclusion?

Example:

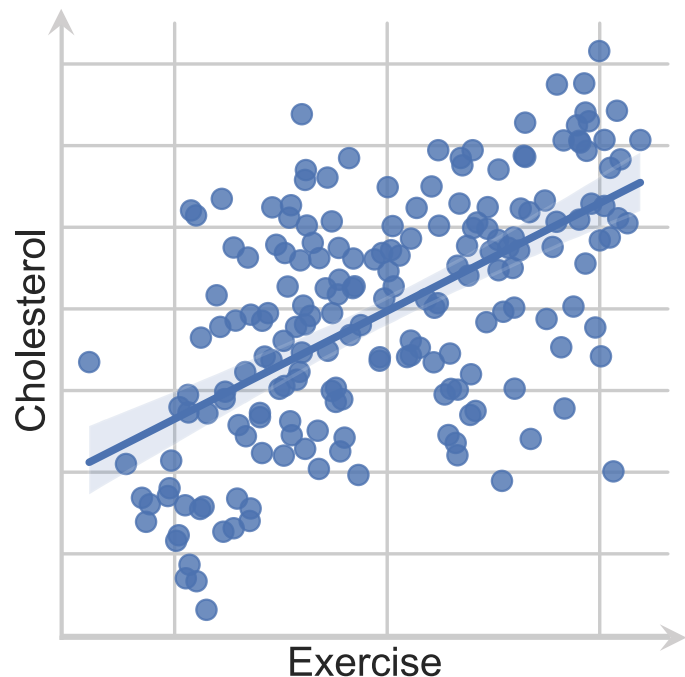
- X axis: hours of weekly exercise.
- Y axis: amount of cholesterol in the blood.

Implies that **more exercise leads to higher cholesterol** (or vice-versa)?

Simpson's paradox

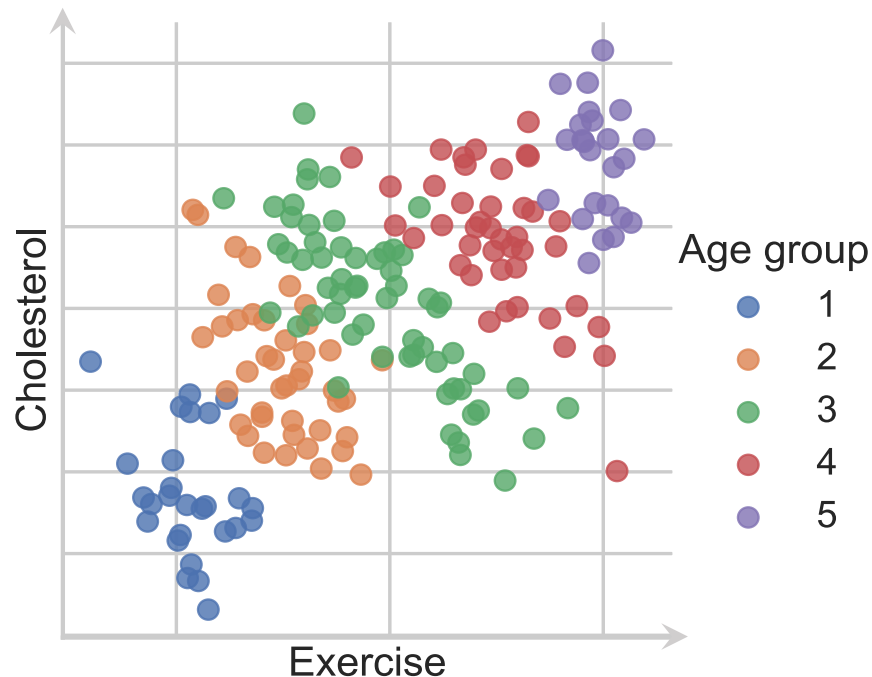


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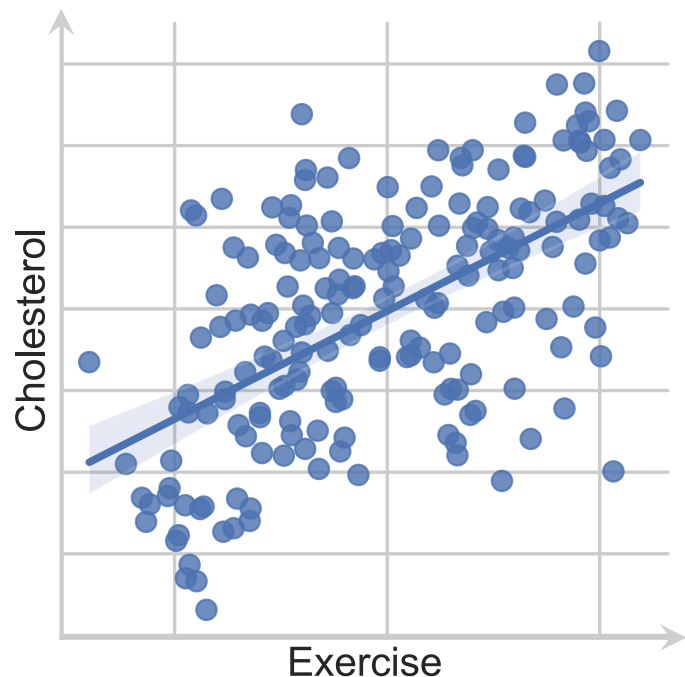
- Don't cancel your gym membership yet.
- What if we group (segregate) the *same* data according to a person's age?
- Also referred to as *conditioning* on that variable.

Simpson's paradox



- Don't cancel your gym membership yet.
- What if we group (segregate) the *same* data according to a person's age?
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Simpson's paradox



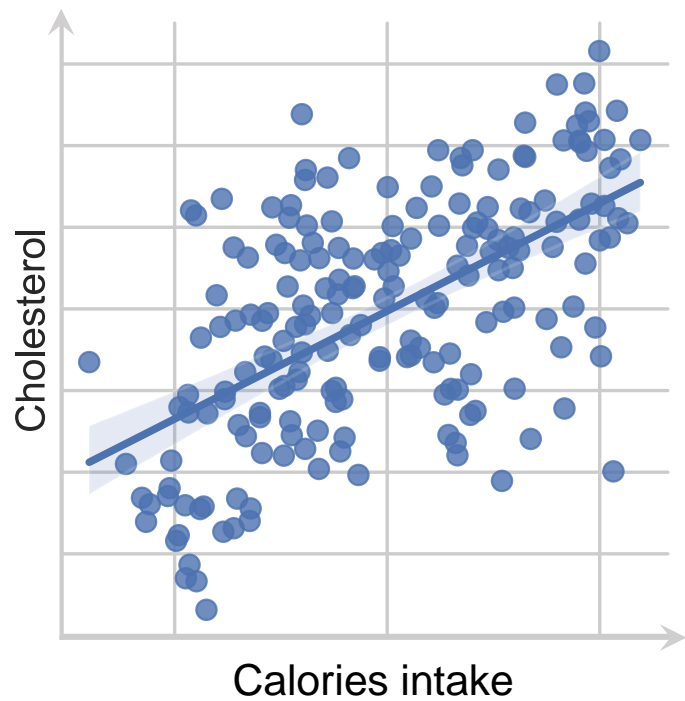
- Suddenly there is a *negative* correlation in each age group.

Explanation:

- As people get older, they tend to exercise more **and** have higher cholesterol (regardless of exercise).
- However, comparing people of the same age, more exercise does lead to lower cholesterol – as expected.

Paradox: Ignoring age, the correlation is the *opposite* of what we expect (exercise leads to high cholesterol).

Simpson's paradox



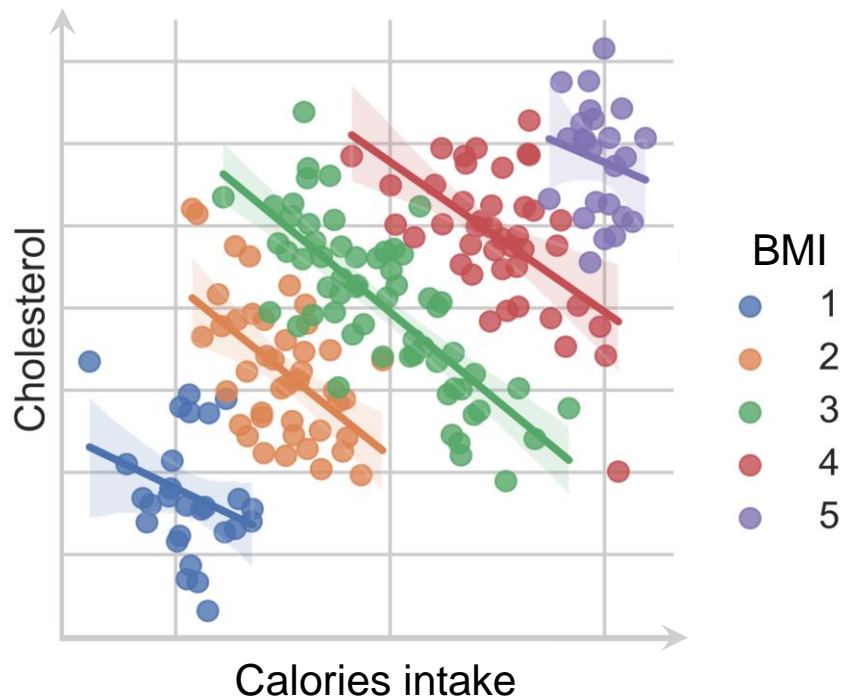
- Does this mean that segregated data always gives us the correct answer?

No. Let's imagine that the axes are relabeled:

- X axis: calories per day.
- Y axis: blood cholesterol (as before).

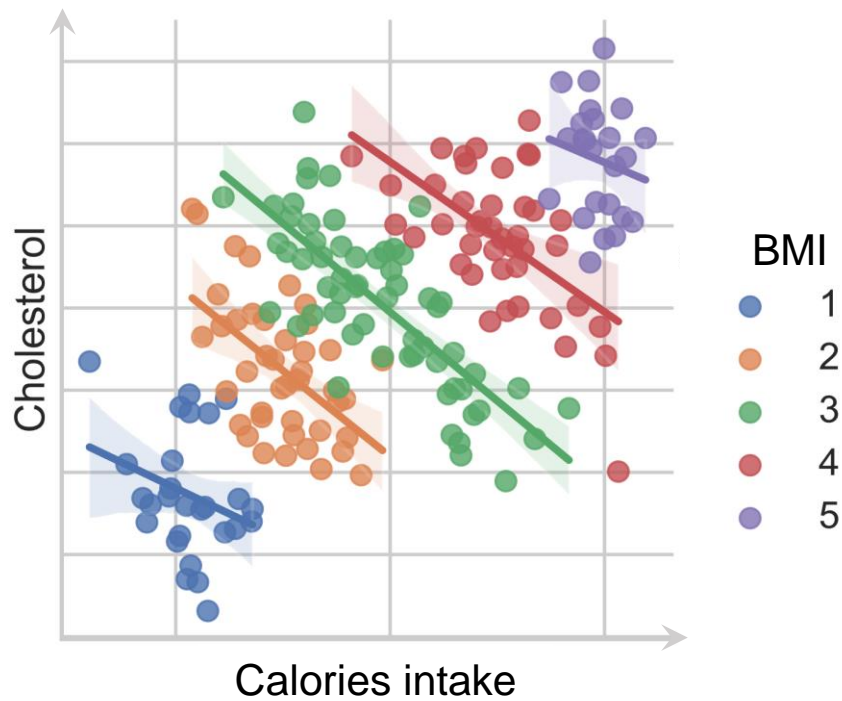
Now, common sense says that there *should* be a positive correlation (eating more food in general leads to higher cholesterol).

Simpson's paradox



- Now group the data by body mass index (BMI, an indicator of weight relative to height).
- There is now a *negative* correlation in each group!
- Should the conclusion be that more calories leads to lower cholesterol? (Would be true if grouping/conditioning is always the answer, as before.)

Simpson's paradox

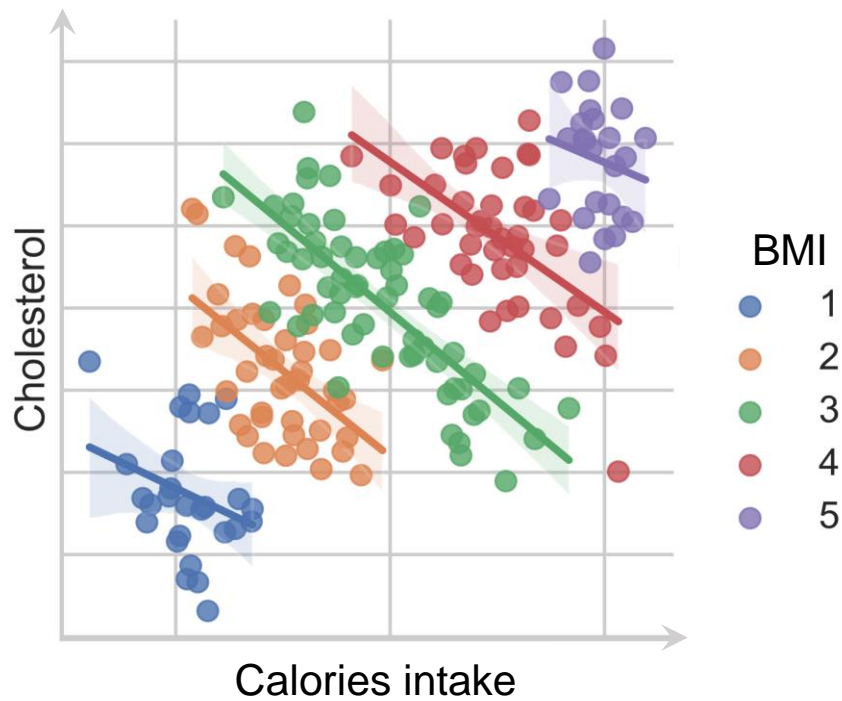


Plausible explanation: (**I'm not a physician*)

- Increasing calories increases BMI, which in turn is associated with higher cholesterol.
- For a *fixed BMI group*, if the calories intake increases, there must be a corresponding energy expenditure (exercise), otherwise we would move to a higher BMI group. And this exercise lowers cholesterol.

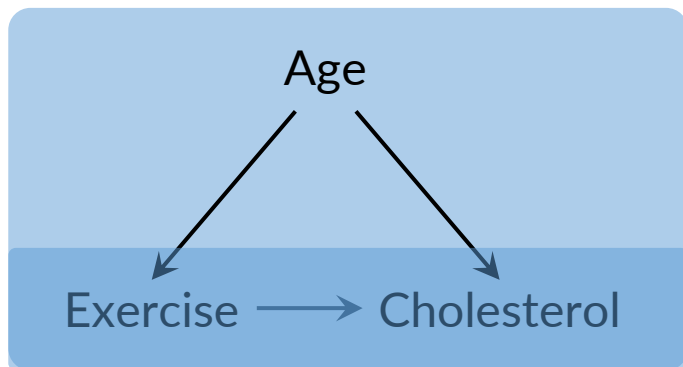
In other words, since BMI is **part of the mechanism** that affects cholesterol, it does **not** make sense to segregate data by it.

Simpson's paradox



- **Simpson's paradox** describes situations where grouping reverses correlations *arbitrarily* – and standard statistics cannot tell us which one is correct!
- We needed *domain knowledge* to navigate these examples correctly.
- How can we hope to make correct decisions in much more complex situations (many variables, complex dependencies)?
- We need a *language* to describe this domain knowledge and be systematic.

Graphs of dependencies – intuition

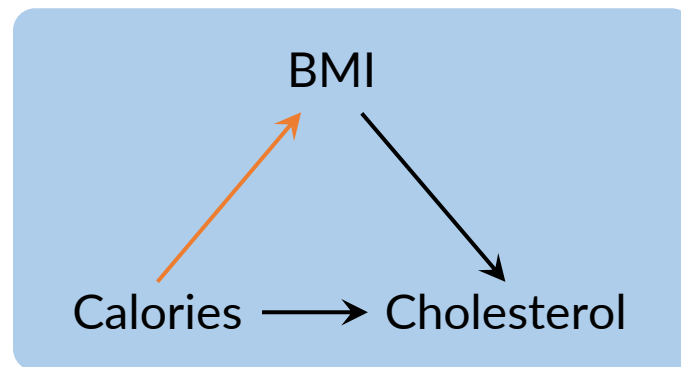
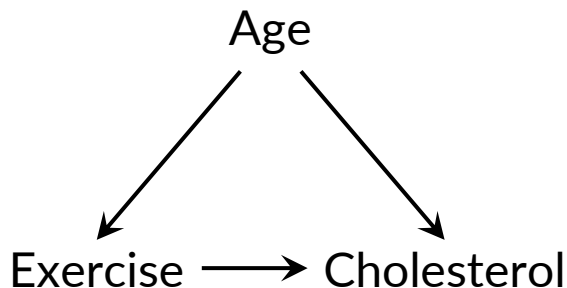


- “Age” influences both other variables.
- In other words, it is a **confounder** when studying exercise vs. cholesterol.
- **Ignoring** age may induce a spurious correlation between them.
- **Conditioning** on age (splitting the data by age group) fixes it.

Graphs of dependencies – intuition



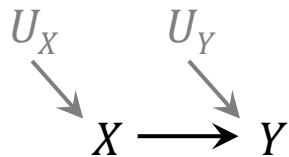
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- In the other example, BMI influences cholesterol but is influenced by calories.
- **Conditioning** on BMI would not have the same effect as for age, as it's not a confounder (we will elaborate on this later).

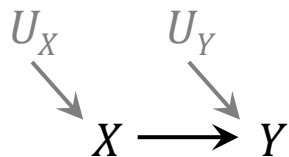
Let us be a bit more formal:

- Define a Structural Causal Model (**SCM**) as a graph.
 - ↪ *Think of it like a “blueprint” or “story” of how the data is generated.*
- This graph has a node for each variable of interest (e.g. X , Y).
- It has a directed edge (arrow) $X \rightarrow Y$ if **knowledge** of X is **necessary** to calculate Y .
 - ↪ In other words, for some function f we have $Y = f(X, \dots)$.



Structural Causal Model (SCM)

- This is a Directed Acyclic Graph (DAG) – it has no cycles.
- If $X \rightarrow Y$, we say that X is a **direct cause** of Y .
- If X is an ancestor of Y , it is an (indirect) **cause** of Y .
- Conversely, if two variables are not ancestors of each other, they are **independent** (in the statistical sense).

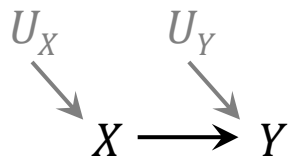


Example: U_X and U_Y .

Structural Causal Model (SCM)

We split the nodes into 2 sets:

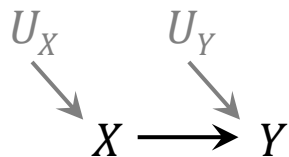
- Exogenous variables $U = \{U_X, U_Y\}$.
 - ↪ They are **external** to our model.
 - ↪ Represent **unknown** factors: their values are modelled as **probability distributions**.
 - ↪ They are also **independent** of each other.
- Endogenous variables $V = \{X, Y\}$.
 - ↪ They are **internal** to our model.
 - ↪ They are **deterministic**: knowing the values of all exogenous variables, we can set their values with perfect certainty.



Structural Causal Model (SCM)

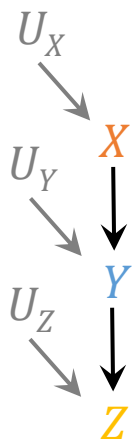
Why are SCMs useful?

- We can predict patterns of **independence** solely from the graph.
 \hookrightarrow Says a lot about the model, even **before** deciding which functions to use or estimating any parameters.
- Predict effect of **interventions** (“what if”) with just an SCM and data.
 \hookrightarrow Makes statistics “actionable”: correctly supports decision-making.
- Use **independence** to help estimate parameters more efficiently:



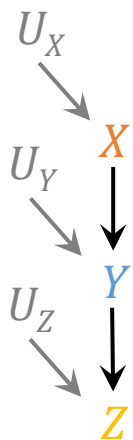
$$\underbrace{P(A, B)}_{\substack{\text{\#parameters} \\ \text{\#samples}}} = \underbrace{P(A)}_{\substack{\text{\#parameters} \\ \text{\#samples}}} \underbrace{P(B)}_{\substack{\text{\#parameters} \\ \text{\#samples}}}$$

Independence patterns – chains



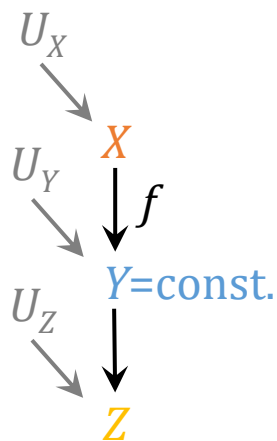
- *Example SCM:*
 - School funding X , exam scores Y , college acceptance Z .
 - Exogenous (noise) variables U (all independent).
- All pairs of variables are likely **dependent**:
 $X \rightarrow Y$, $Y \rightarrow Z$, $X \rightarrow Z$
- E.g. a change in X in general results in a change in Y (and indirectly in Z).

Independence patterns – chains



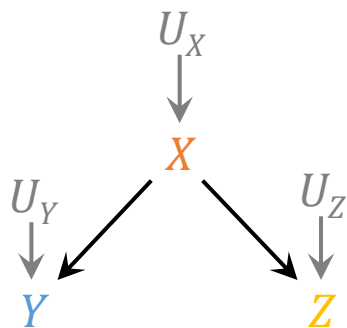
- X and Z are **independent, conditional** on Y .
- What does it mean to condition on Y ?
- It means we filter the data into groups, for each distinct value of Y (e.g. $Y = a$, $Y = b$, ...).
- In practice, this happens when we “know” that for a particular case $Y = a$, (e.g. an exam score was a) and we want to model the remaining probabilities.

Independence patterns – chains



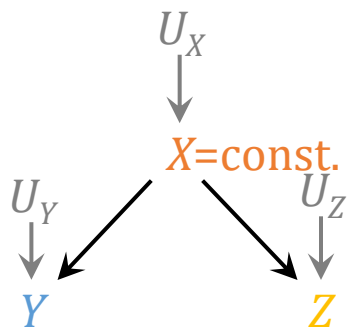
- For a **fixed** value $Y = a$ (grouping the data), let us analyse the dependence between X and Z .
- As we change X to take on different values, then U_Y will **have** to change to keep the **fixed** value $Y = a$.
- *Example:* If $Y = f(X) + U_Y$, then in the group $Y = a$ we'd always observe noise set **exactly** to $U_Y = a - f(X)$.
- But Z does not depend on U_Y , only on U_Z and Y , so it is **independent** of X .
- Reiterating: X and Z are **independent**, conditional on Y .
- Applies to **any configuration** of variables that **contains a chain**.

Independence patterns – forks



- *Example SCM:*
 - Temperature X , ice cream sales Y , crime Z .
 - Exogenous (noise) variables U (all independent).
- All pairs of variables are likely **dependent**:
 $X \rightarrow Y$, $X \rightarrow Z$, Y and Z
- Y and Z are dependent because, **some** of the time, **both** will change due to X .
- *Example:* Based on this SCM, we would expect correlations between ice cream sales and crime, though there is no direct causal connection between them.

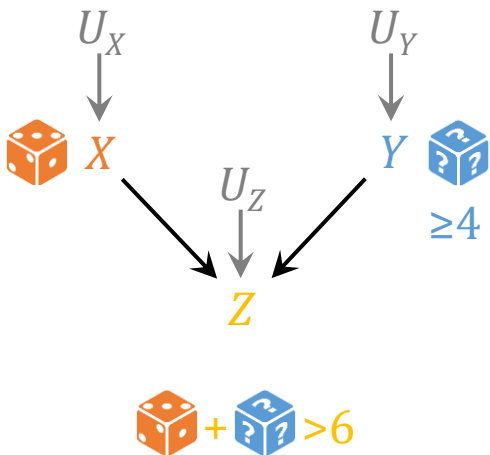
Independence patterns – forks



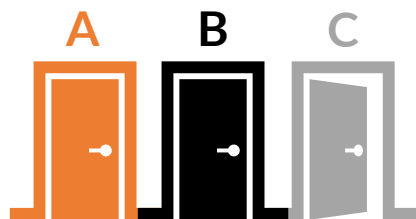
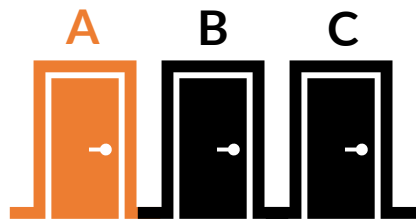
- Y and Z are **independent, conditional** on X .
- To understand this conditioning, again we filter the data into groups ($X = a$, $X = b$, ...).
- Now we're comparing only cases where X is constant.
- Since X is constant, Y and Z only depend on their respective noise variables U , which are independent.
- So Y and Z are **independent** too.
- This conditional independence applies to **any** configuration of variables with a “common cause” like X (called a “fork”).

Independence patterns – colliders

- *Example SCM:*
 - 1st dice X , 2nd dice Y , sum of dice results > 6 (Boolean) Z .
 - Exogenous (noise) variables U (all independent).



- Some pairs of variables are likely **dependent**:
 $X \rightarrow Z$, $Y \rightarrow Z$
- X and Y are **independent, unconditionally**.
- X and Y are **dependent, conditionally** on Z .
- *Example:* If we know that the first dice result (X) is 3, and that the sum is larger than 6 (Z), then the second dice result (Y) must be 4 or more – the dice results **become dependent**.

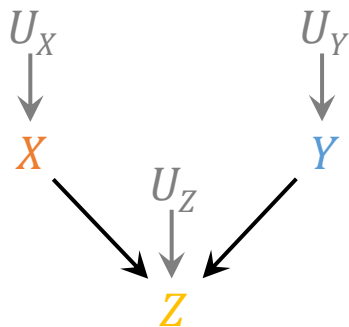
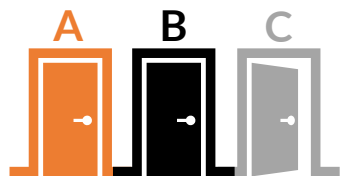
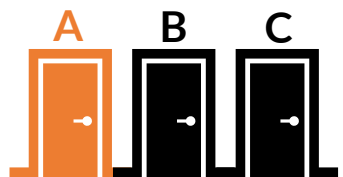


Colliders are very common and create very **unintuitive** results in conditional probabilities.

One such example is the “Monty Hall problem”:

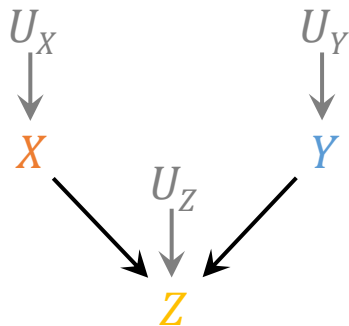
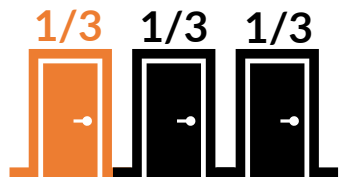
- There are 3 doors, 1 has a prize behind it (equal probabilities).
- You choose **door A**.
- Another door, **door C**, is shown to have no prize behind it.
- Is it better to switch to **door B**, or keep the choice of **door A**?
 - *Naïve answer*: Both are the same ($1/2$ chance of winning).
 - *Real answer*: There is a $2/3$ chance of winning by switching to **door B**, and $1/3$ by staying with **door A** – so you have a better chance of winning if you switch to **door B**!

Trouble with colliders



- Why? Let us write it as a SCM:
 - X is your initial choice of door.
 - Y is the door with the prize.
 - Z is the door shown to you with no prize.
 - ↪ Z is causally dependent on X , and on Y . (The door shown must not have the prize, and not be the one you chose.)
- Since this SCM is a **collider**, conditioning on Z (i.e. **knowing** which other door has no prize) makes X and Y **dependent**.
 - ↪ This challenges our expectation: Your choice of door (X) and the door with the prize (Y) were picked **independently**, so you may expect them to remain independent!
- Realizing that this is a collider helps us avoid this mistake.

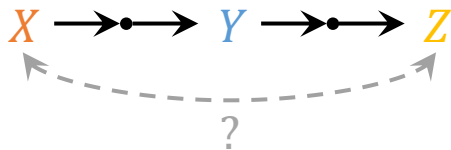
Trouble with colliders



Just to get a quantitative answer to the Monty Hall problem:

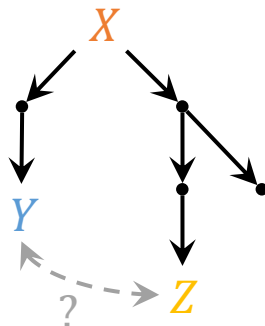
- The **door you chose** has $1/3$ probability of having the prize.
- So when another door is opened showing no prize, **your door** still has $1/3$ probability of having the prize.
- Since the opened door has 0 probability of having the prize, and the total probability must sum to 1 , the probability of the **remaining door** must be $1 - 1/3 = 2/3$, making it the best option.

Generalizing to larger graphs



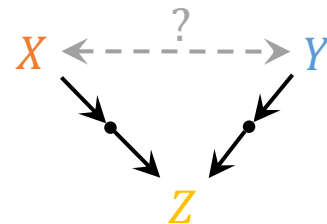
Chain with longer paths between the nodes:

- X and Z are **dependent**, unconditionally.
- X and Z are **independent**, conditional on Y .



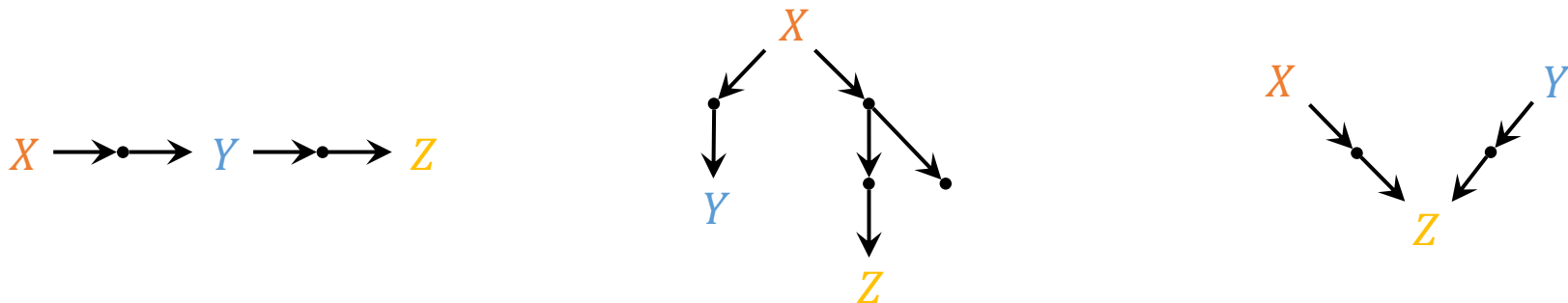
Fork where Z and Y are **descendants** of X :

- Y and Z are **dependent**, unconditionally.
- Y and Z are **independent**, conditional on X .



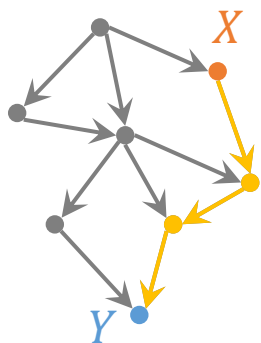
Collider with a **single path** between X , Z and Y :

- X and Y are **independent**, unconditionally.
- X and Y are **dependent**, conditional on Z .

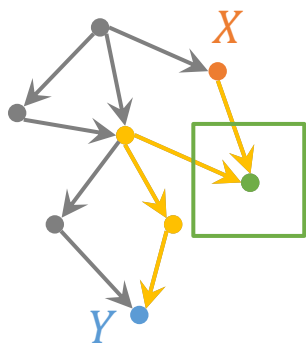


- Can we find dependence/independence patterns more systematically?
↳ Yes: We can check for **d-separation**.
- “d” stands for *directional*: two nodes are “separated” in a graph, taking the edges’ directions into account.
- You can think about it as **d-separated** = **independent**.

d -separation – unconditional

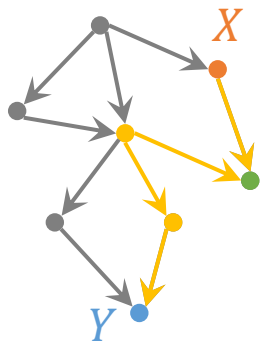


- Consider an arbitrary graph with two nodes X and Y .
- To know whether the two nodes are d -separated, consider **all paths** between them (ignoring edge direction).
- If there is any **unblocked path** between them, they are **d -connected** (i.e. not d -separated).
 - ↳ Then X and Y are likely to be **dependent**.



- Let us delete one edge to see an example of a **blocked path**.
- The **highlighted path** connects X and Y .
- But it is **blocked** because it goes through a **collider node** (i.e. that node has two incoming edges in the path).
- All paths between X and Y are blocked, so they are **d -separated**.
 $\hookrightarrow X$ and Y are **independent**.

Analogy:

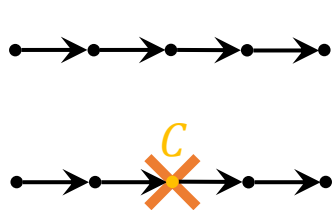


- Think about dependence between two nodes like water flowing.
- A **single blockage** in one path blocks flow through it.
- If **all paths are blocked**, there is no flow – they are d -separated.
- But a **single unblocked** path allows some flow to go through.

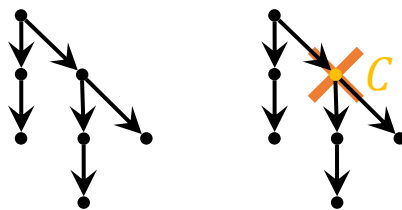
When not conditioning, **only colliders** block dependence.

When conditioning on some variables, there are more ways to block.

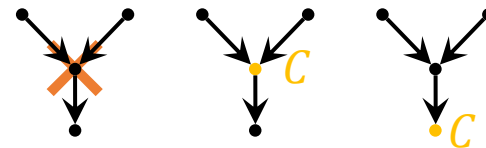
Conditioning on a node C :



- **Chains** that contain C block dependence.

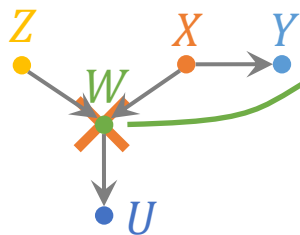
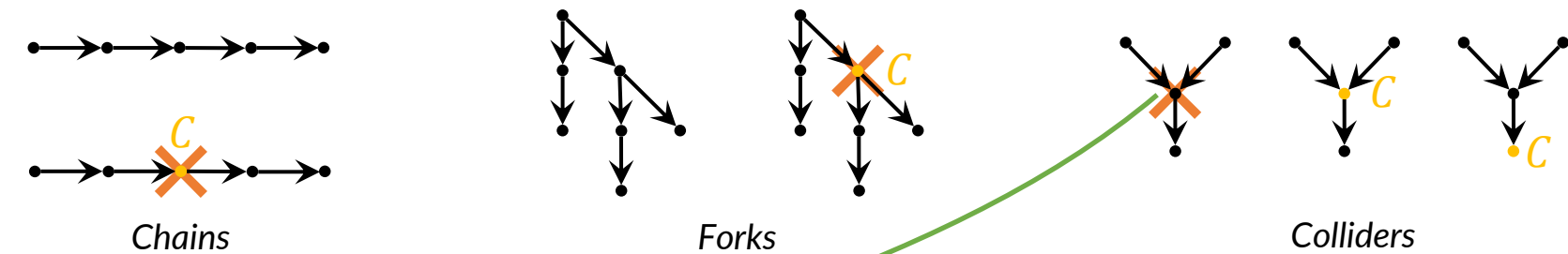


- **Forks** with C as the middle node block dependence.



- **Colliders** that **do not** have C as the middle node or as a descendant block dependence.

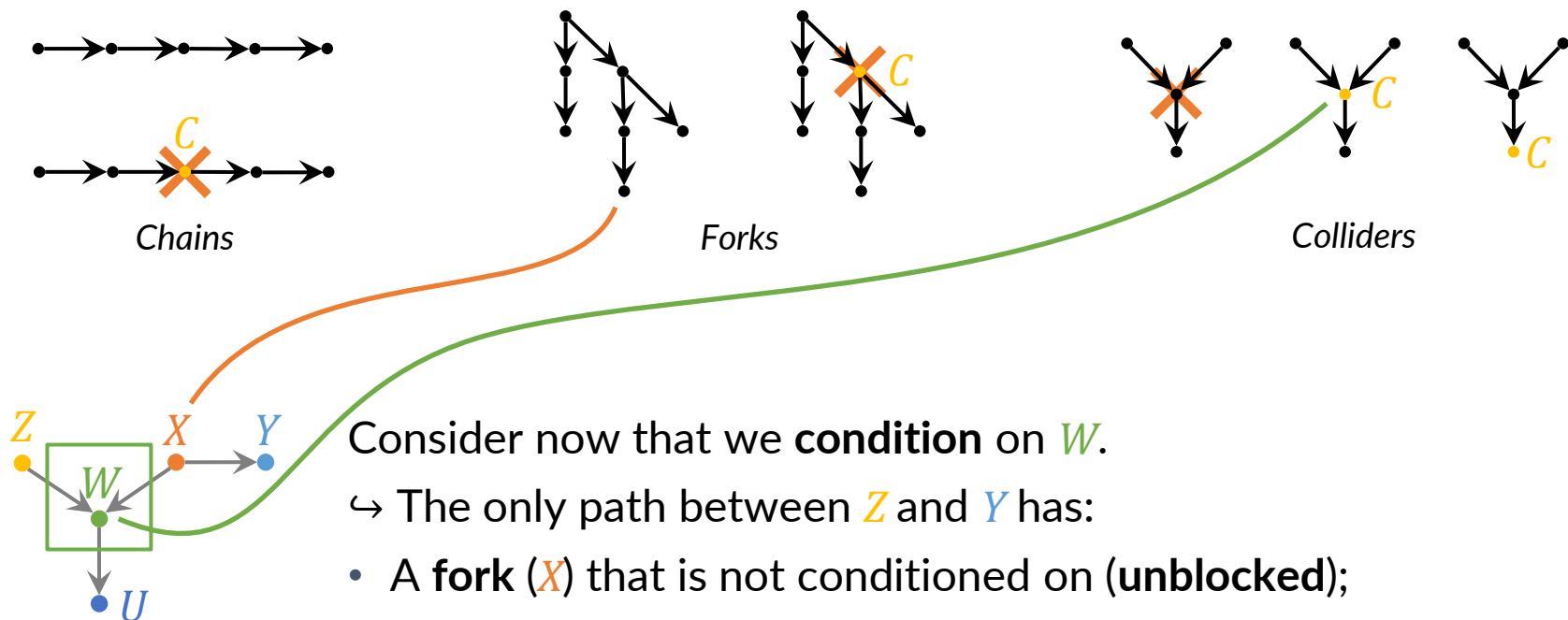
d -separation – example



Consider the dependence between **Z** and **Y** in this SCM.

↪ **Unconditionally**, they're d -separated (**independent**), since the path between them is **blocked** by the collider **W**.

d-separation – example



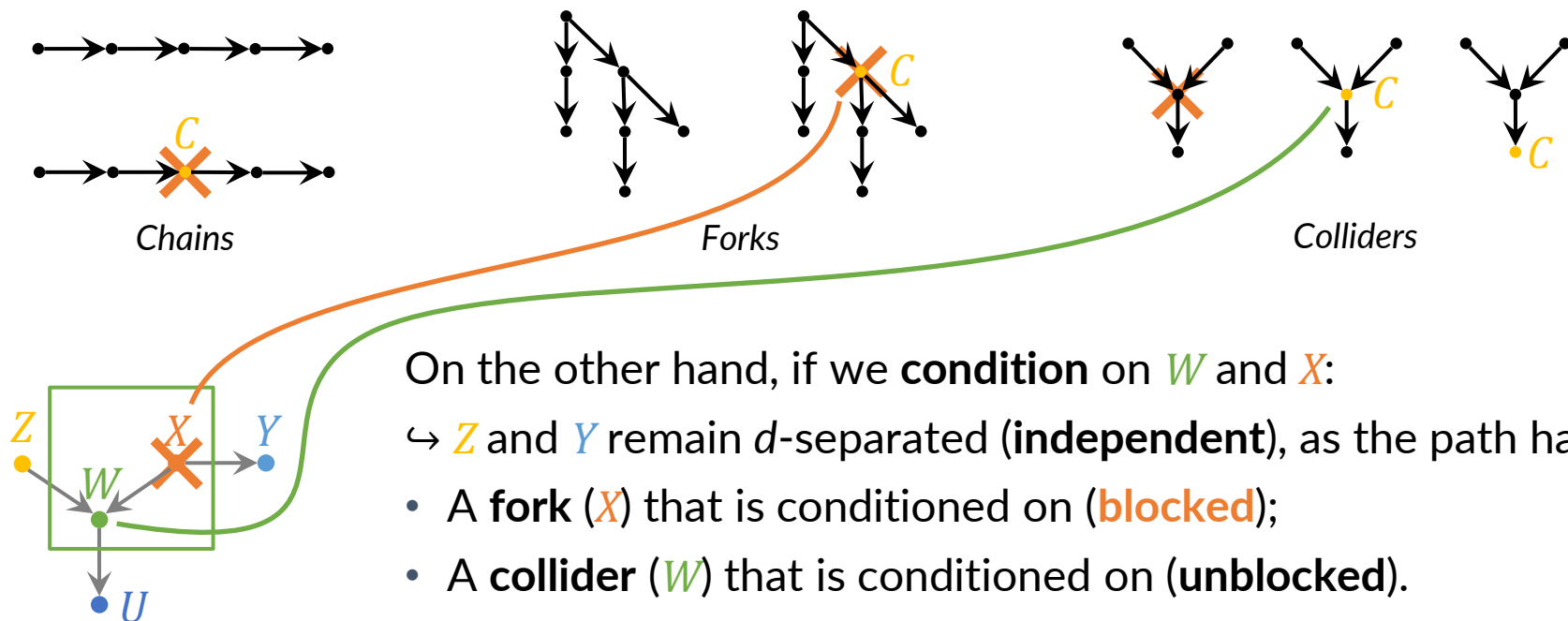
Consider now that we **condition** on W .

↪ The only path between Z and Y has:

- A **fork** (X) that is not conditioned on (**unblocked**);
- A **collider** (W) that is conditioned on (**unblocked**).

With an unblocked path, Z and Y are now d -connected (**dependent**).

d-separation – example



On the other hand, if we **condition** on **W** and **X**:

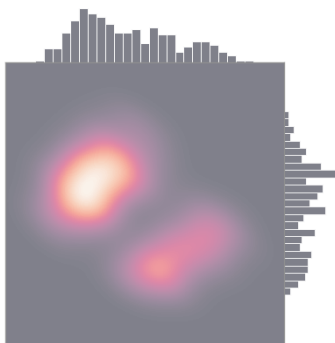
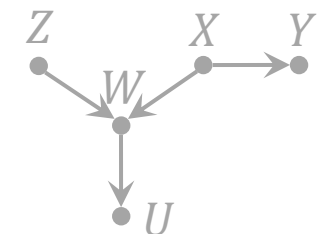
↪ **Z** and **Y** remain d-separated (**independent**), as the path has:

- A **fork** (**X**) that is conditioned on (**blocked**);
- A **collider** (**W**) that is conditioned on (**unblocked**).

But a single blocked node in each path is enough to block dependence completely.

But why? What is the usefulness of this in ML practice?

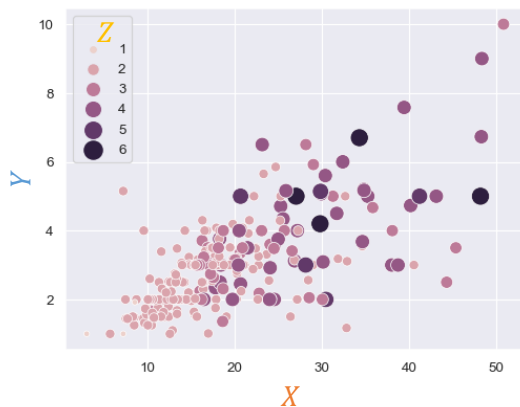
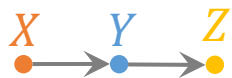
- Causal models have **testable implications** for generated data.
- So if a structural causal model (SCM) is correct, it will **predict** patterns of conditional (in)dependence, which **the data must have**.
↪ Otherwise the model is incorrect!



So, given a dataset and a SCM that you think must explain it:

- Use *d*-separation criteria on the SCM to **list** which variables are independent, conditional on other variables.
- Use a statistical independence test to **check** if those variables are independent in the dataset, conditionally (e.g. by grouping).

If a test fails, the SCM does not fit the data, and must be changed.

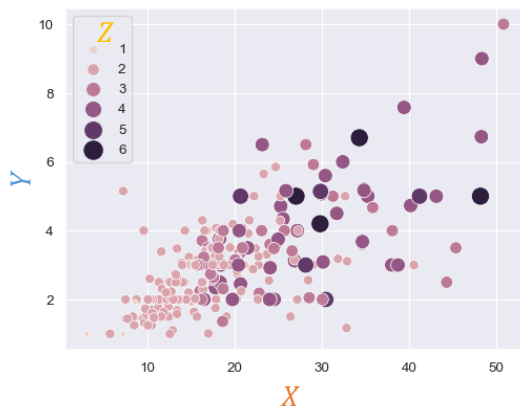
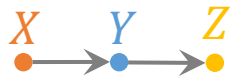


Example – the graph on the left, with associated dataset:

- We know that X and Z are dependent through Y , but **independent when conditioned** on Y .
- So we regress Z from X and Y , for example by fitting the data with a linear model:

$$Z = aX + bY + c$$

- We expect a to be 0. Otherwise, there is a (linear) dependence between X and Z , and the SCM is **wrong**. (*Conditional correlation implies conditional dependence.*)
- We also know how to **repair** it: we must add a path between X and Z , that is not d -separated by Y .

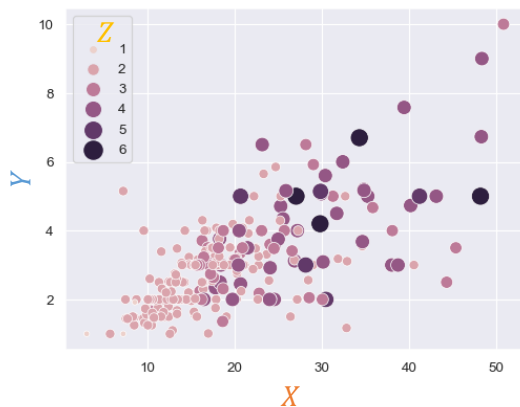
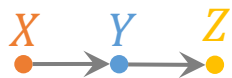


Example:

- Alternatively, we could **group the data** based on Y (condition on Y).
- Then we expect X and Z to be **independent** in each group.
- To verify this, we use a statistical test of independence.

Use independence tests from standard Python packages:

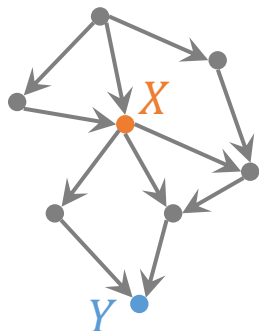
- χ^2 test (discrete) – `scipy.stats.chi2_contingency`
- Mutual Information (continuous) – `sklearn.feature_selection.mutual_info_regression`



Advantages over fitting a model to data directly:

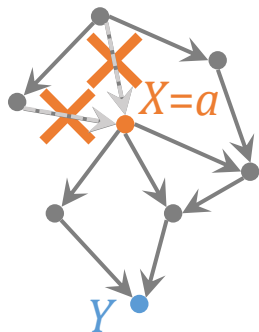
- d -separation is **nonparametric**: it only uses dependency patterns, and results are the same **regardless** of the distributions or functions that relate variables.
- It is **local**: it tells us **where** the model doesn't fit the data, so we can repair it.
 - ↪ If instead we fit a model to data directly, and it's a poor fit, there would be no clue as to what went wrong.
- Since it's local, we can get **partial information** even when other parts of the model are unknown, or have parameters that are impossible to estimate from data.

A brief appetizer – interventions



- We can also use SCMs to analyse the effect of **interventions**:
 - What is the outcome if a patient takes a given drug?
 - Would a given policy result in fewer wildfires?
 - Does a given change in a system yield better performance?

A brief appetizer – interventions



- **Intervening \neq conditioning.**
- An intervention **changes** the graph:
 - The intervened variable is set **deterministically** to a value (e.g. $X = a$).
 - All incoming edges are **cut** (removed).
- The SCM obtained after the intervention **shares** the remaining structure (functional relations, exogenous distributions) but will generally result in **different** overall probabilities (e.g. $P(Y)$).
- This answers “what if” (decision-making) questions, while avoiding many pitfalls (confounding, Simpson’s paradox, etc).

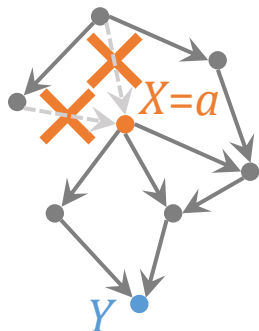
A brief appetizer – interventions

“Do”-notation

- To write the probabilities in a modified graph (obtained by intervening), insert a “do” operator in the conditioned variables:

$$P(Y \mid \text{do}(X = a))$$

- Meaning: Set $X = a$ (deterministically), cutting off incoming edges; then evaluate $P(Y)$ normally in this new graph.



Average Causal Effect

- Binary intervention (e.g. take drug $X = 1$ vs. no drug $X = 0$),

$$\text{ACE} = P(Y \mid \text{do}(X = 1)) - P(Y \mid \text{do}(X = 0))$$

- Interpretation:* **difference** between the **fraction** of the population that recovers (Y) when all take drug vs. when none take the drug.

Parting thoughts

- We still don't have the whole “book of why”.
- But we can be aware of the kinds of structures that hide behind the data we observe, and how they can trick our intuition.



For a more complete (but still relatively short) picture, check: *Judea Pearl et al., “Causal Inference in Statistics: A Primer”* ...from which I borrowed many of the examples in these slides.

Thank you!