Mediators Confounders and Colliders, oh my!

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Goals for understanding

Use causal diagrams to understand:

What is a **mediator**?

What is a **confounder**?

What is a **collider**?

Use causal diagrams and domain expert knowledge to answer:

Which variables should we condition on in a regression model?

Pearl 2018

Building blocks of causal diagrams

$$X \longrightarrow Y$$

"When we draw an arrow from X to Y, we are implicitly saying some probability rule or function specifies how Y would change if X were to change." Pearl 2018

Building blocks of causal diagrams

Definition:

A back-door path is any path from **X** to **Y** that starts with an arrow pointing into **X**.

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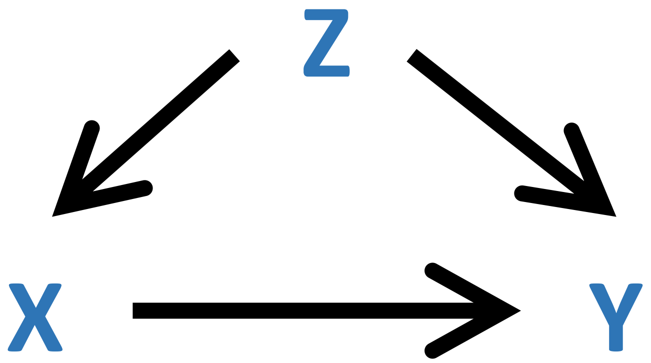

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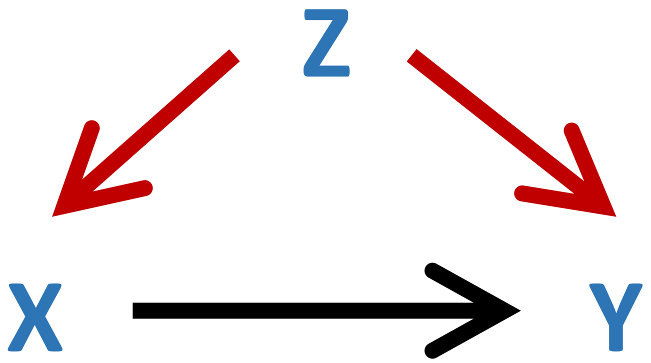

Backdoor path from
X ← Z → Y

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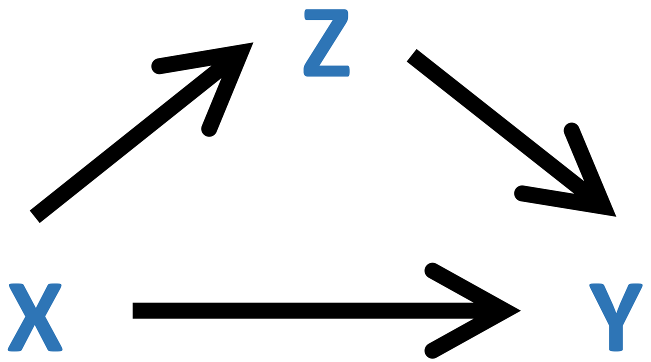

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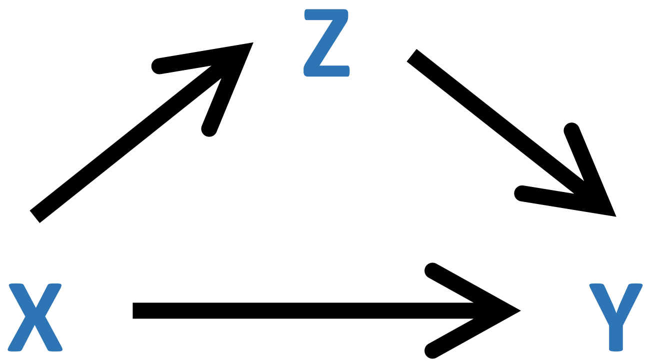

No backdoor path

Building blocks of causal diagrams: motivating example

Data shows increased ice cream sales are associated with more crime…

Ice cream sales cause crime?

Ice cream sales 🍦 ⟶ Crime 🔫

Building blocks of causal diagrams: motivating example

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Ice cream sales cause crime? That’s what the data say!

But our brains 🧠 say that doesn’t make any sense!

Ice cream sales 🍦 $\xrightarrow{?}$ Crime 🔫

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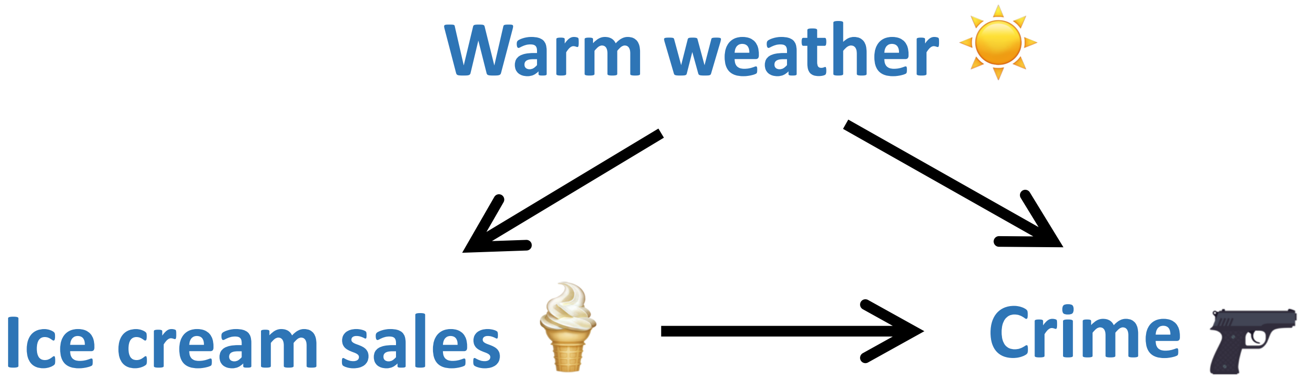

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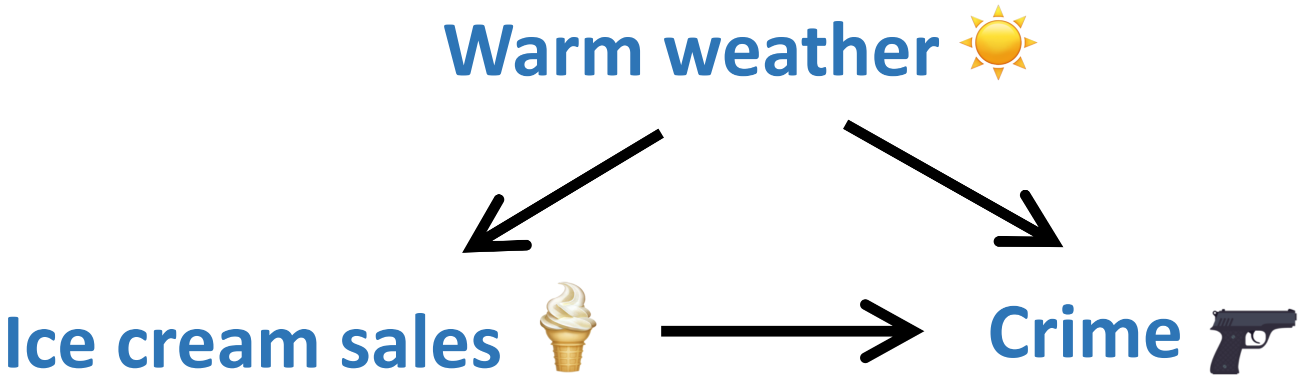

Solution: Condition on "background factors" (aka confounders) K.

VERY IMPORTANT QUESTION:

***Which* variables should we condition on?**

Building blocks of causal diagrams

3 kinds of *junctions*

1. Chain: A → B → C
2. Fork: A ← B → C
3. Collider: A → B ← C

1. Chain: A → B → C

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B is the *mechanism* or <u>mediator</u> that transmits the effect of A to C.

Fire 🔥 → Smoke → Alarm

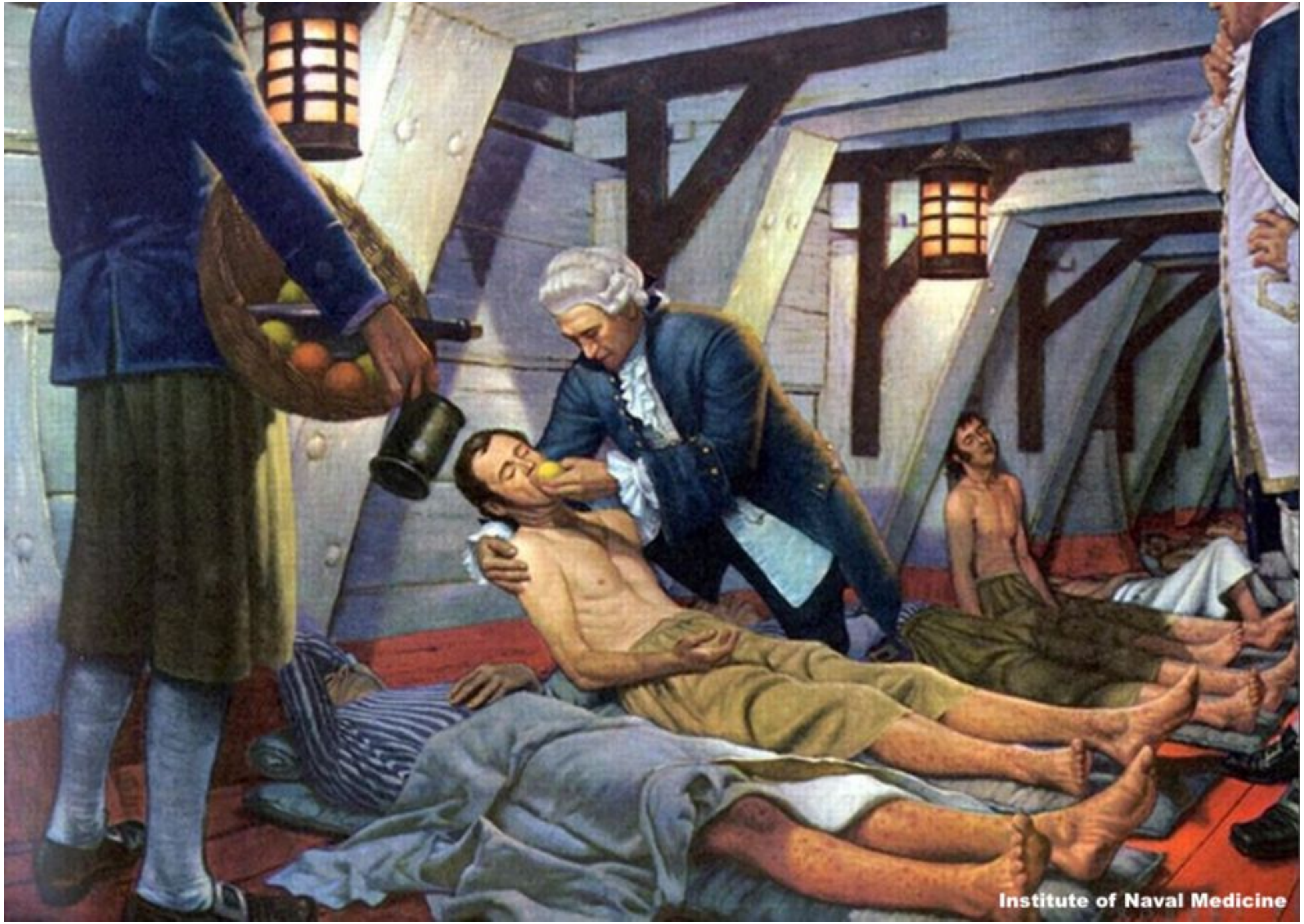
Fire only causes the alarm to go off via producing smoke.
There is no direct arrow from Fire → Alarm.

Controlling for B (mediator) prevents information about A (exposure) from getting to C (outcome), and vice versa.

Why do a mediation analysis?

To understand causal mechanisms and
to answer questions like “How?” and “Why?”

Scurvy



Dr. James Lind credited with the first clinical trial in 1747

Administered 6 different treatments to 12 afflicted sailors:

- 2 spoons of vinegar
- 1 quart of cider
- 1 cup of seawater
- 2 oranges, 1 lemon
- 25 drops elixir of vitriol
- Paste of garlic, mustard seed, horseradish

Sailors who were given the citrus fruits got better.

Citrus $\longrightarrow$ Scurvy

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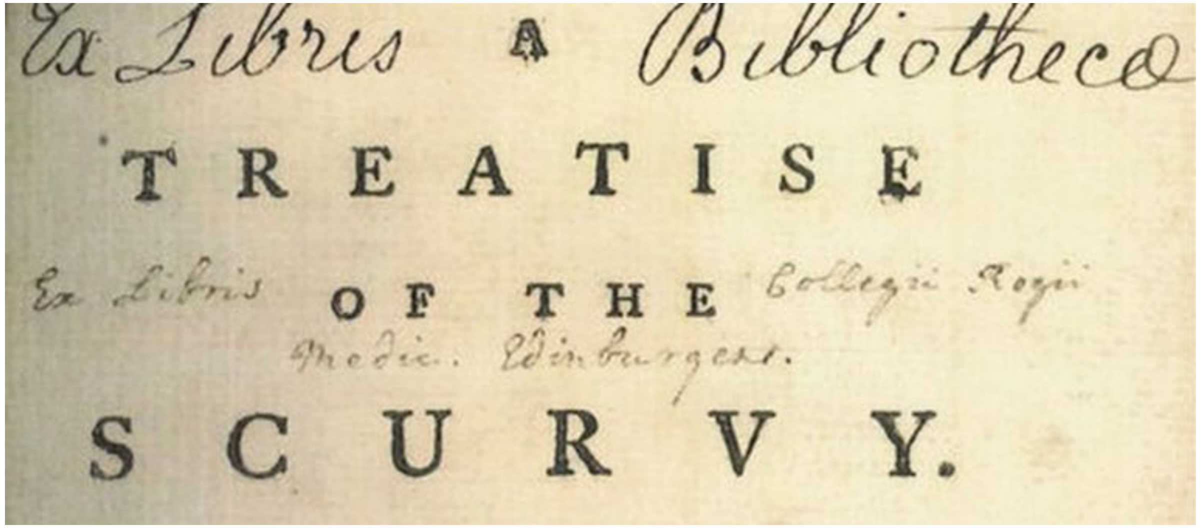
Sailors who were given the citrus fruits got better. ***BUT WHY?***

$$\text{Citrus} \xrightarrow{\quad ? \quad} \text{Scurvy}$$

Lind suggested lemons worked due to their acidity

Published *Treatise of the Scurvy* in 1753

Citrus → Acidity → Scurvy



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TREATISE

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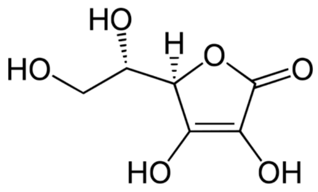
SCURVY.

Vitamin C discovered by Albert Szent-Gyorgyi in 1933

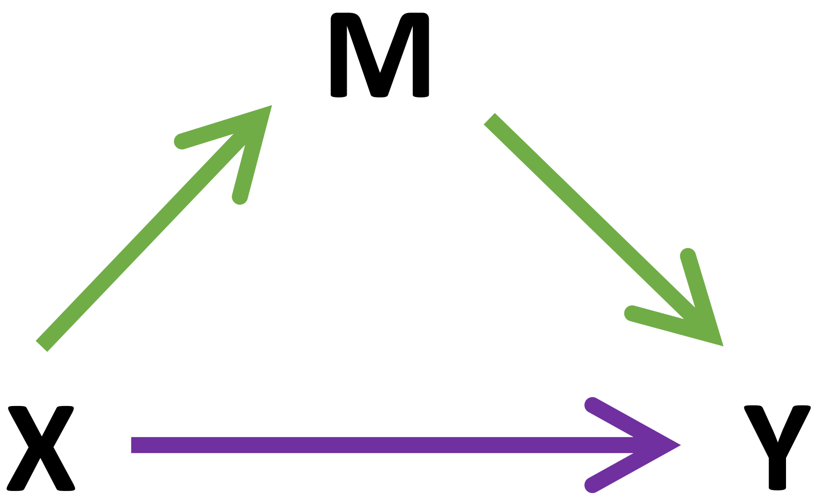
Feeding the sailors citrus fruits increased their vitamin C levels, which in turn remedied their scurvy



Citrus $\longrightarrow$ Vitamin C $\longrightarrow$ Scurvy

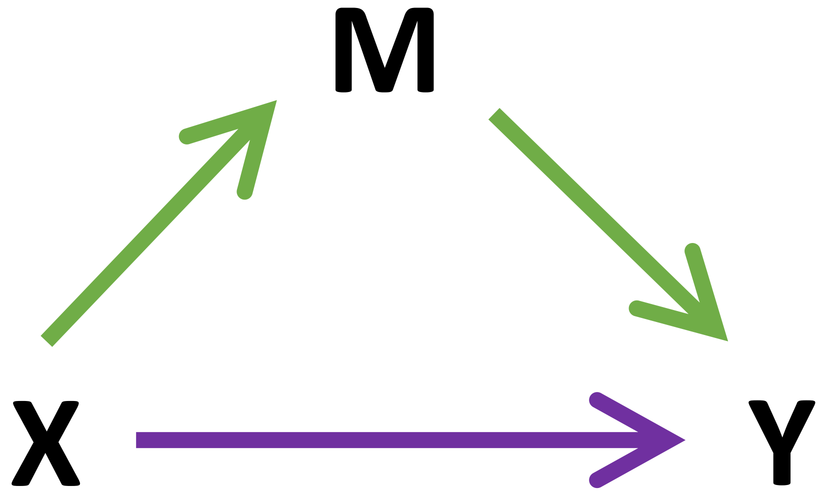
Vitamin C is a mediator of the effect of citrus on scurvy

Mediation Analysis decomposes the total effect of exposure X on outcome Y into the direct effect and the mediated effect transmitted through M.



To estimate the total effect of X on Y, DO NOT adjust for the mediator M.

To estimate the the direct effect of X on Y, DO adjust for the mediator M.



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Confounder makes A and C statistically correlated even though there is no direct causal link between A and C.

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Confounder makes A and C statistically correlated even though there is no direct causal link between A and C.

Shoe size 👞 ← Age of child → Reading ability 📚

Children with larger shoes tend to read at a higher level. But giving a child larger shoes won’t make him read better…

Controlling for B (confounder) prevents information about A (exposure) from getting to C (outcome), and vice versa.

Confounder Example:

1996 observational study data comparing endoscopic vs open surgery showed:

- Higher probability of successful removal of **small kidney stones** using **open surgery**
- Higher probability of successful removal of **large kidney stones** using **open surgery**
- Higher probability of successful removal of **kidney stones** using **endoscopic surgery**

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- Higher probability of successful removal of **kidney stones** using **endoscopic surgery**

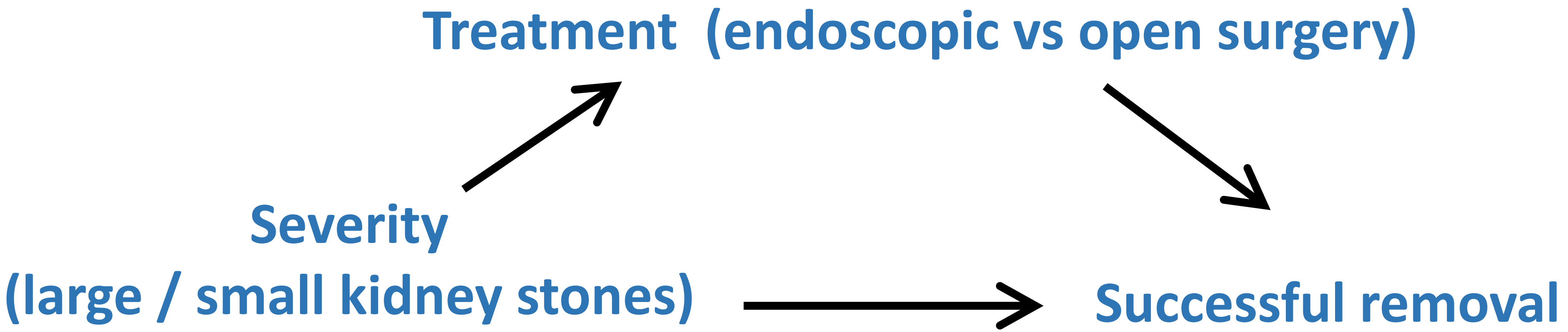
Open surgery is better for small and large kidney stones, but worse for all kidney stones?

HUH? 🤔

Confounder Example:

Kidney Stone Severity is a confounder of Treatment and Successful Removal.

To obtain unbiased estimates of Treatment’s effect on Success, we need to adjust for kidney stone severity. **In this case, one should report the adjusted estimates.**



3. Collider: A → B ← C

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B is a *common effect* or <u>collider</u> of A and C.

Conditioning on collider B *creates a dependence* between A and C even though they are unrelated in the general population.
(collider bias is also known as <u>selection bias</u>)

We should *not* condition on colliders.

Collider Example: Smoking birthweight paradox

Prior studies have shown evidence of
Smoking mother 🚬 → Low birthweight 👶 → Mortality

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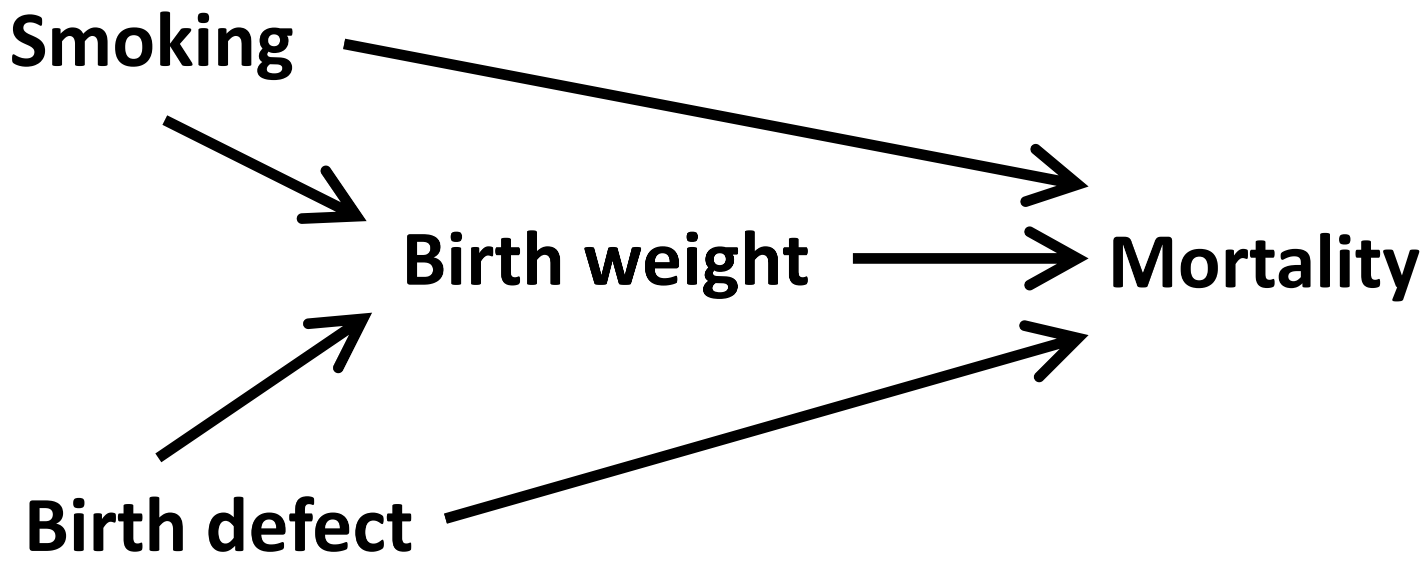
Smoking mother 🚬 → Low birthweight 👶 → Mortality

Mid 1960s study by Yerushalmy on 15,000 children found that low birthweight babies of smoking mothers had *better survival* than low birthweight babies of nonsmoking mothers.

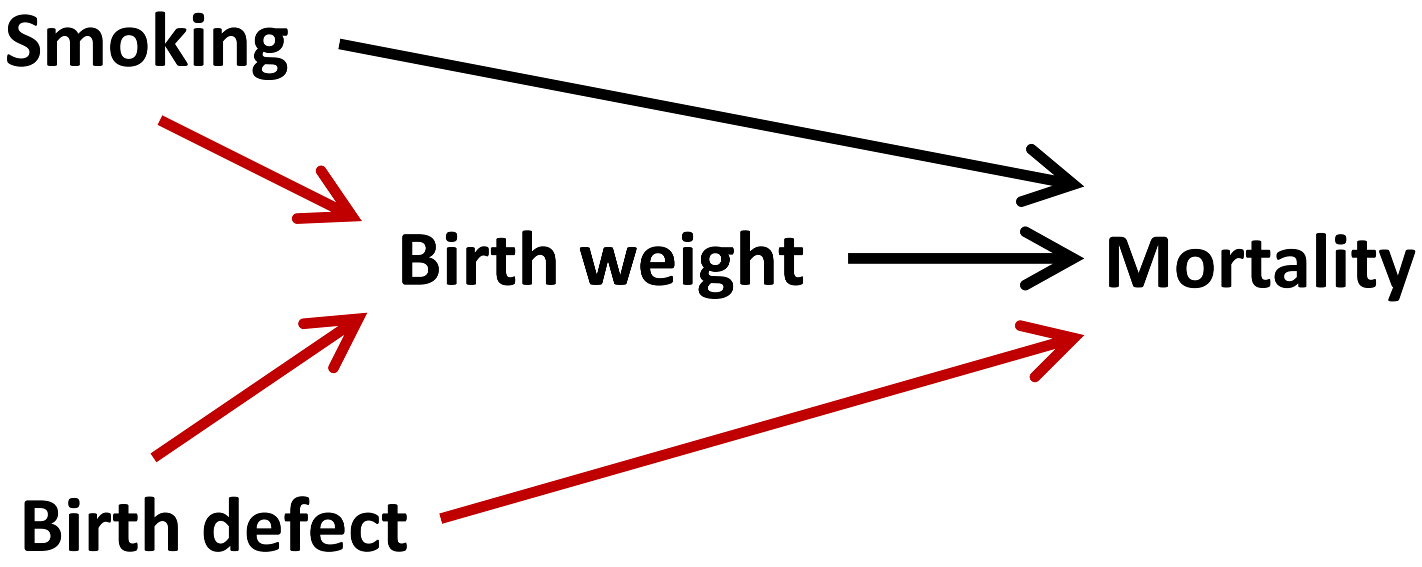
Mothers' smoking is protective? That doesn't make sense!

Collider Example: Smoking birthweight paradox



Collider Example: Smoking birthweight paradox

Birth weight is a collider!



When we look only at low birthweight babies (conditioning on a collider), we open a backdoor path from Smoking → Birth weight ← Birth defect → Mortality.

Collider Example: Smoking birthweight paradox

Stumped epidemiologists for decades!

“In this case, **the collider bias was detected because the apparent phenomenon was too implausible**, but just imagine how many cases of collider bias go undetected because the bias does not conflict with theory.”

Pearl 2018

Returning to our very important question: *Which* variables should we condition on?

To estimate the total effect of exposure X on outcome Y:

DO adjust for confounders (common causes of X and Y)

DO NOT adjust for colliders (common effects of X and Y)

DO NOT adjust for mediators (variables on the causal pathway)*

Returning to our very important question: *Which* variables should we condition on?

To estimate the total effect of exposure X on outcome Y:

DO adjust for confounders (common causes of X and Y)

DO NOT adjust for colliders (common effects of X and Y)

DO NOT adjust for mediators (variables on the causal pathway)*

* In a mediation analysis, you DO adjust for the mediator to estimate the *direct effect* of X on Y adjusted for M.

Games from "Toward *a Clearer Definition of Confounding*" by Clarice Weinburg (a deputy chief at NIH)

American Journal of Epidemiology
Published by Oxford University Press on behalf of the Johns Hopkins Bloomberg School of Public Health 2012.

Vol. 176, No. 6
DOI: 10.1093/aje/kws127
Advance Access publication:
August 17, 2012

Practice of Epidemiology

"Toward a Clearer Definition of Confounding" Revisited With Directed Acyclic Graphs

Penelope P. Howards*, Enrique F. Schisterman, Charles Poole, Jay S. Kaufman, and Clarice R. Weinberg

Game 1:

$$
\begin{tikzcd}
X \arrow[r] & Z \arrow[r] \arrow[d] & Y \\
 & B &
\end{tikzcd}
$$

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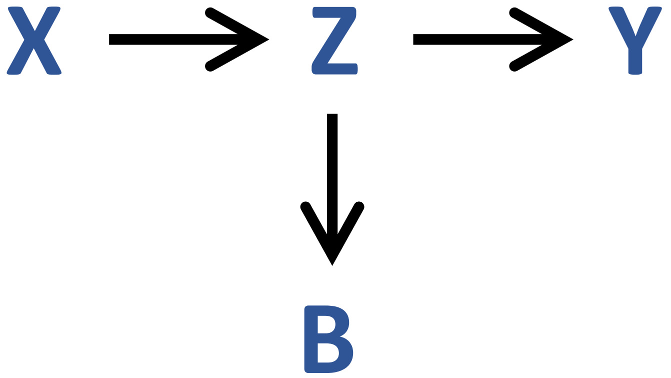
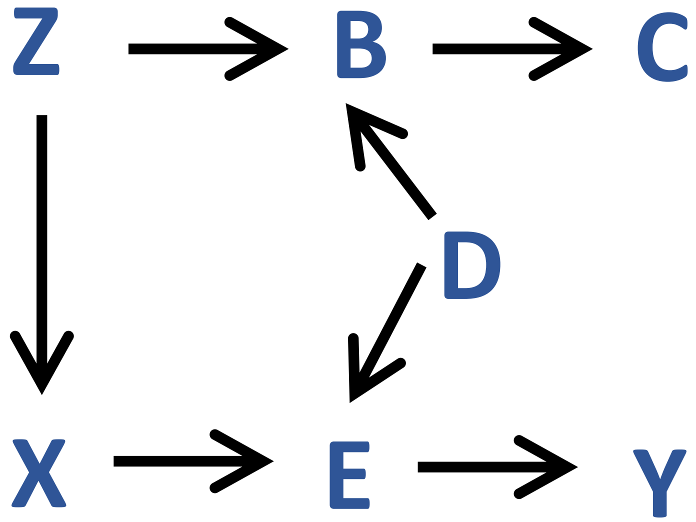


There are no back-door paths from X to Y (no arrows coming into X) ,
so we don't need to control for anything.

Game 2:



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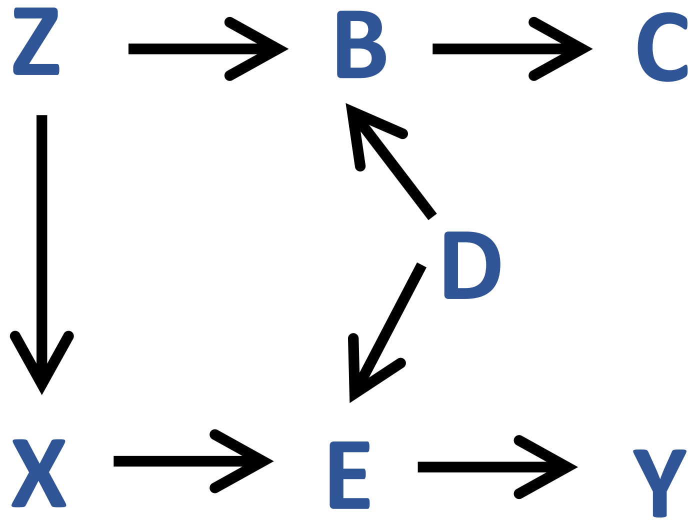
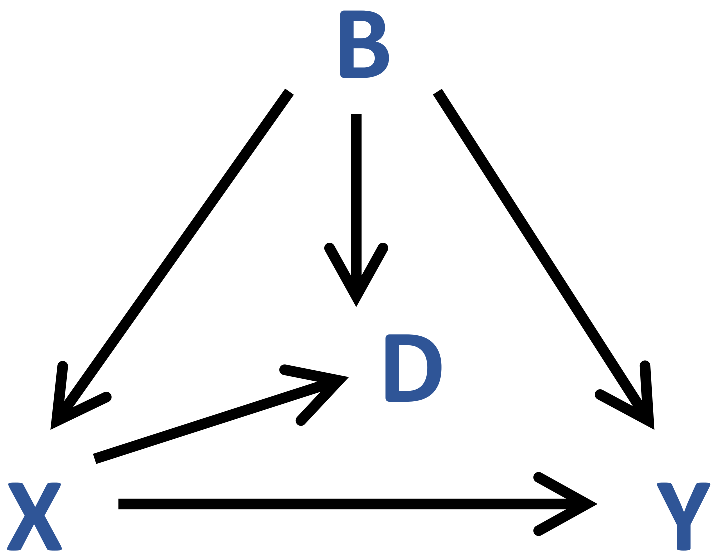


Backdoor path X ← Z → B → D → E → Y is already blocked by the collider B.
If we did adjust for B, we could “re-close” the path by conditioning on Z and/or D.
There is more than 1 way to de-confound the X to Y relationship!

Game 3:



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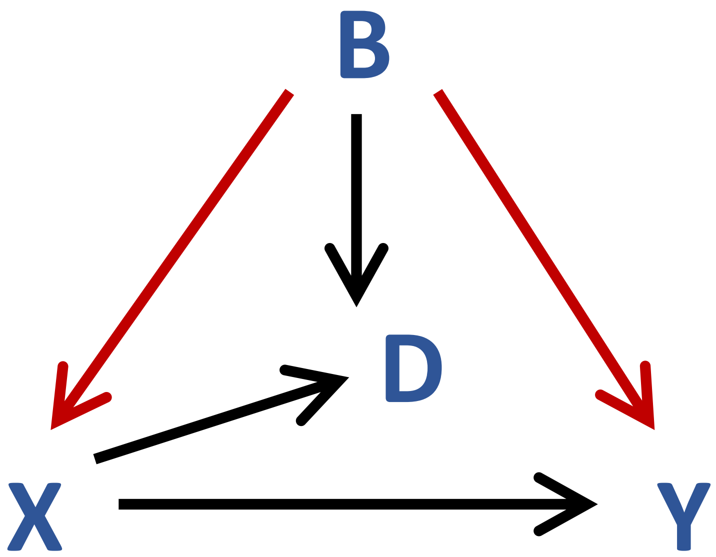

There is a backdoor path from X ← B → Y.
Need to adjust for confounder B.

Simpson’s Paradox (1951):

Same (fictional) data
2 stories
2 different conclusions

Story 1: "The Bad / Bad / Good Drug"

	Control Group (no drug)		Treatment Group (took drug)		
	Heart Attack	No HA	Heart Attack	No HA	
Female	1	19	3	37	60
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Total	13	47	11	49	

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→Drug is bad for women

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30% (12/40) men in **control** group had heart attack
40% (8/20) men in **treatment** group had heart attack

→Drug is bad for men

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40% (8/20) men in **treatment** group had heart attack

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22% (13/60) **controls** had heart attack

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30% (12/40) men in **control** group had heart attack
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22% (13/60) **controls** had heart attack
18% (11/60) **treatment** group had heart attack

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22% (13/60) **controls** had heart attack
18% (11/60) **treatment** group had heart attack

→Drug is good for people

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→Drug is bad for men

→Drug is good for people

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→Drug is bad for women
→Drug is bad for men
→Drug is good for people

We know it is impossible for a "Bad/Bad/Good drug" to exist!
So what's going on here?

“In vain will you seek guidance from [the data]. To answer the question, we must look **beyond the data** to the **data generating process**.”

Pearl 2018

Data generating process

- Gender is associated with Heart attack (men at greater risk).

  Gender → Heart Attack
- Gender is associated with Drug (women had preference for taking Drug).

  Gender → Drug
- Presumably, Drug is related to Heart Attack

  Drug → Heart Attack

Data generating process

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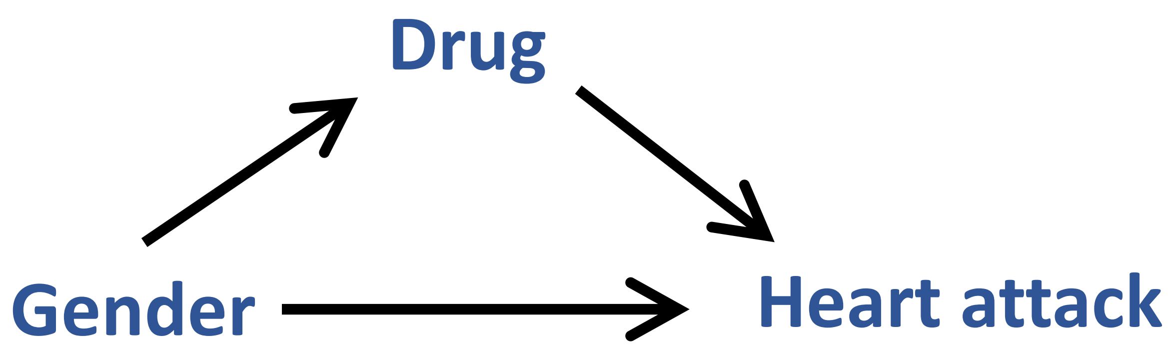

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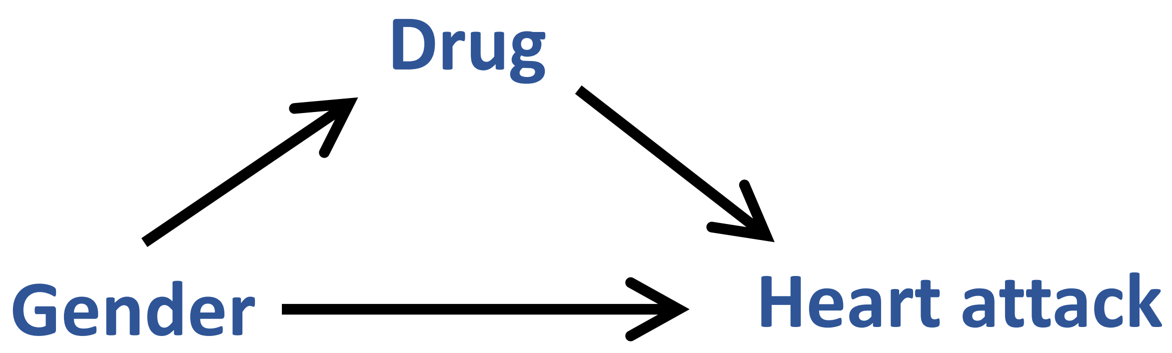

Gender is a **confounder** of Drug and Heart attack, so **use gender-adjusted estimates** of Drug's effect on Heart Attack and **conclude drug is bad**.

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To get average Drug Effect adjusting for Gender (a confounder):

Take simple average (because sample is split 50% men and 50% women),

Pr(Heart Attack | Drug) = (7.5% + 40%)/2 = **23.75 %**

Similarly, **Pr(Heart Attack | No Drug)**

= (5% + 30%)/2 = **17.5 %**

Therefore, drug is Bad! No more paradox.

Story 2: “The Bad / Bad / Good Drug”

	Control Group (no drug)		Treatment Group (took drug)		
	Heart Attack	No HA	Heart Attack	No HA	
Low BP	1	19	3	37	60
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→Drug is bad for low BP

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	Control Group (no drug)		Treatment Group (took drug)		
	Heart Attack	No HA	Heart Attack	No HA	
Low BP	1	19	3	37	60
High BP	12	28	8	12	60
Total	13	47	11	49	

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7.5 % (3/40) low BP in **treatment** group had heart attack

→Drug is bad for low BP

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- Blood Pressure → Heart Attack
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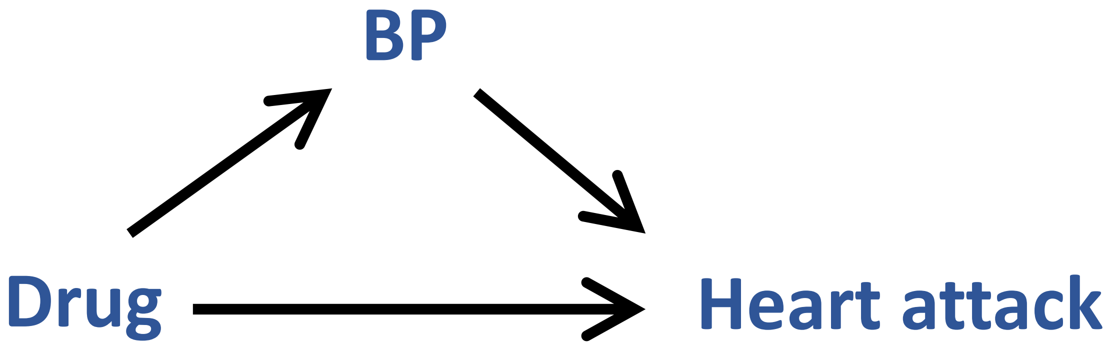

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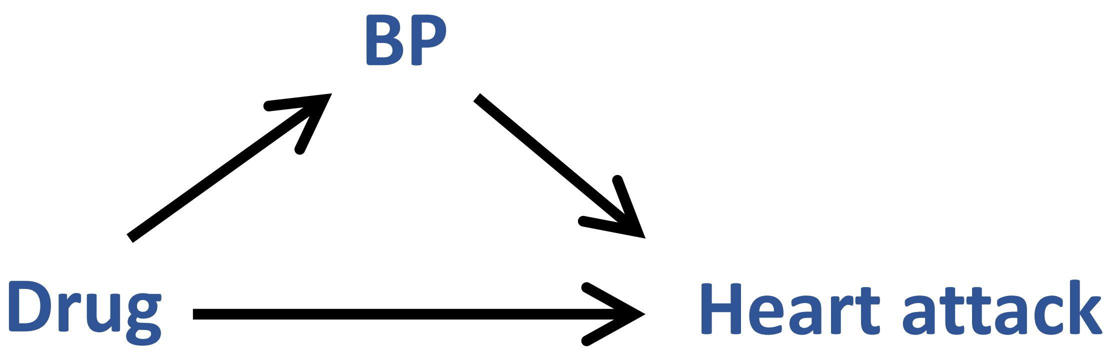

BP is a **mediator** of Drug and Heart attack, so to obtain an unbiased estimate of Drug's effect on Heart Attack **we should not adjust for BP**.

Conditioning on BP would disable one of the causal paths (maybe the main one) by which the drug works.

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We should not adjust for Blood Pressure, so we use the unadjusted estimates:

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Same data
2 stories
2 different conclusions.

Q: So do we aggregate data or partition data?

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A: <u>It depends</u> on the process that generated the data.

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If we see X, the probability of Y increases. This could be due to:

i. Y causes X $Y \longrightarrow X$

ii. Some other variable Z is the cause of both X and Y

$$\begin{tikzcd} & Z \arrow[dl] \arrow[dr] & \\ X \arrow[rr] & & Y \end{tikzcd}$$

iii. X causes Y $X \longrightarrow Y$

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You can control for too much. Sometimes you end up controlling away the effect you're trying to measure by conditioning on a mediator, or you induce bias by conditioning on a common effect (aka collider).

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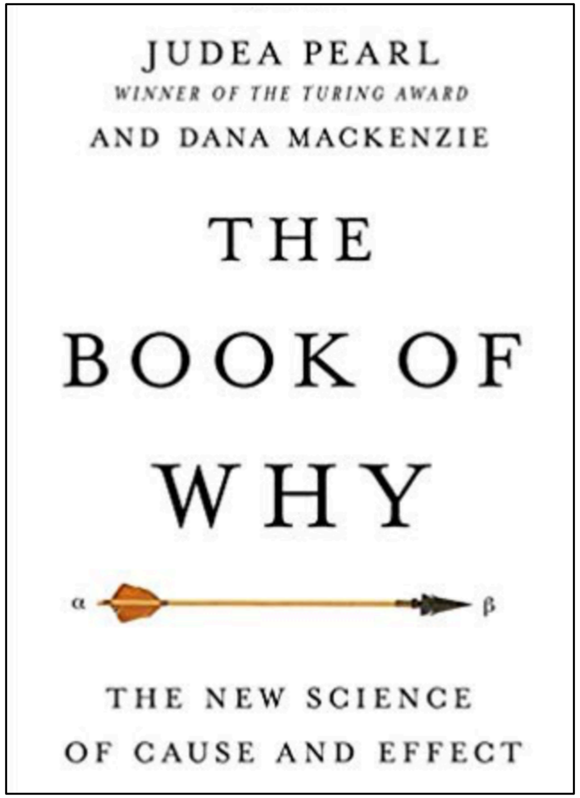
NO! Z could be a mediator or a collider.

To learn more:



MULTIVARIATE BEHAVIORAL RESEARCH
https://doi.org/10.1080/00273171.2018.1552109

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Christina T. Saunders and Jeffrey D. Blume

Department of Biostatistics, Vanderbilt University

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