

Week 5: A Causal Inference Perspective on Methodological Issues

Causal Inference & Structural Equation Modeling

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based on slides by Oisín Ryan and Ellen Hamaker

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Causal graphs and conceptual clarity

causal models have very specific info. on statistical result you might expect, but if you have certain statistical results, that does not contain all the info. we need on our causal operation... which means that we lose some info. if we only look at the statistics, e.g., means & cov. matrices... compared to if we knew the entire causal structure.

Causal models imply statistical models - causal models contain information not contained in statistical models

- ▶ Many questions which are confusing, difficult or impossible to answer in purely statistical terms become clear when we take a causal inference perspective.
- ▶ I.e., Draw the relevant causal model!

Motto of Miguel Hernan: Draw your assumptions before your conclusions!



Overview

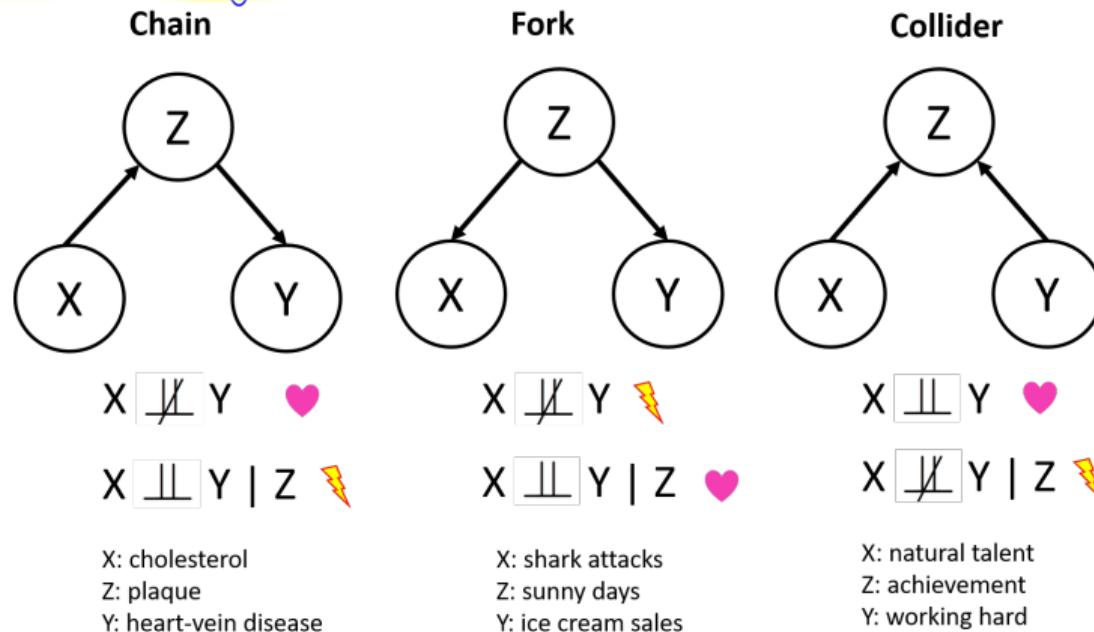
Interventions are more explicit in potential outcome framework.



- ▶ DAGs & Interventions & the RCT
- ▶ Selection Bias, Berksons Paradox, Simpsons Paradox ~ various paradoxes related to selection bias.
- ▶ Repeated Measures: Change Score vs Controlling for Pre-measure

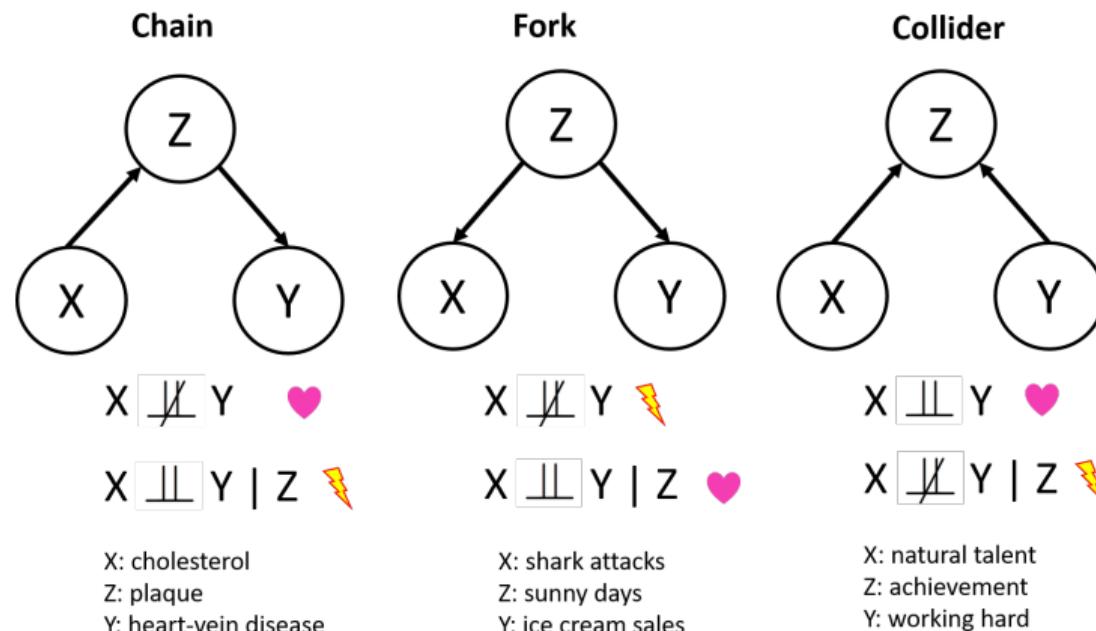
DAGs, SCMs, and interventions

Lecture 1: On which variables should we (not) condition to obtain the causal effect for observational data? ~w/o any interventions,, what variable should we control for, based on a causal DAG?



DAGs, SCMs, and interventions

Lecture 1: On which variables should we (not) condition to obtain the causal effect for **observational** data?



Then how can we represent an intervention in a DAG?

- ▶ What if we **intervened** on the system represented by the DAG?

DAGs, SCMs, and interventions



Remember: Conditioning in observational data, and intervening are not the same actions.

another way of saying = statistical relationships are not the same as causal relationship !!

DAGs, SCMs, and interventions

Remember: Conditioning in observational data, and intervening are not the same actions.

- ▶ Conditioning on X , observational data: Let's look at people in our dataset that have $X=1$, and then at the people who have $X=0$.

DAGs, SCMs, and interventions

Remember: Conditioning in observational data, and intervening are not the same actions.

Here we don't know how they got that X , there might have been some confounding involved...

- ▶ Conditioning on X , observational data: Let's look at people in our dataset that have $X=1$, and then at the people who have $X=0$.
- ▶ Intervening on X : Let's set these people's X to 1 (experimental group), and set these people's X to 0 (control group).

We have full control of on which ppl have 1 & on which ppl have 0 \rightarrow very impo difference!

that's why RCT is helpful becaz it allows us to take control over values of a particular variable.

★Keep in mind they're diff! ★

DAGs, SCMs, and interventions: The "do-operator"

To represent interventions in SCMs, we use the "do-operator":

do-operator: *Set X to a specific value*

The do-operator $do(X = x)$ represents a "surgical intervention" to set the value of the variable X to a constant value x

DAGs, SCMs, and interventions: The "do-operator"

To represent interventions in SCMs, we use the "do-operator":

do-operator:

The do-operator $do(X = x)$ represents a “surgical intervention” to set the value of the variable X to a constant value x



“Surgical” interventions: Modularity assumption: Remainer of the DAG (causal structure) remains completely intact!

Assume that it is possible to intervene on a variable without fundamentally changing how it relates to other variables, e.g.:

- ▶ We can intervene on X without changing $p(Z | X)$
- ▶ We can intervene on one cause-effect mechanism without changing the others!

Read more about such assumptions by searching the jargon 'Modularity', 'Localized Interventions' and 'Fat Hand Interventions'. \rightarrow affecting other variables by accident

Average Causal Effect - DAG edition

We can use the DAGs, SCM and the do-operator to define and estimate any causal effect based on an intervention we want:

Often we are interested in the effect of an intervention on the mean of our outcome variable - the effect of the intervention on average across different people.

Average causal effect of X (0 vs 1) on Y:

$$ACE = E[Y \mid do(X = x_1)] - E[Y \mid do(X = x_0)]$$

(expected value of Y given that we set $X = x_1$) - (exp. val. of Y given that we set $X = x_0$)

Average Causal Effect - DAG edition

We can use the DAGs, SCM and the do-operator to define and estimate any causal effect based on an intervention we want:

Often we are interested in the effect of an intervention on the *mean* of our outcome variable - the effect of the intervention on average across different people.

Average causal effect of X (0 vs 1) on Y:

$$ACE = E[Y \mid do(X = x_1)] - E[Y \mid do(X = x_0)]$$

Here it's more implicit in the do-operator... potential outcome framework, there it was very explicit what all of those assumptions are.

Note: $ACE = E[Y \mid do(X = x_1)] - E[Y \mid do(X = x_0)] = E[Y_i^1] - E[Y_i^0]$

The idea is that the expression w/ "do-operator" is the same as

the expression w/ the potential outcomes we had last week \rightarrow ofc, that means that you have to adhere to

"Essentially your intervention is like ← all of those assumptions we specified last week a successful RCT, that's the idea behind do-operator."

Interventions with DAGs and SCMs: Partial Mediation Example

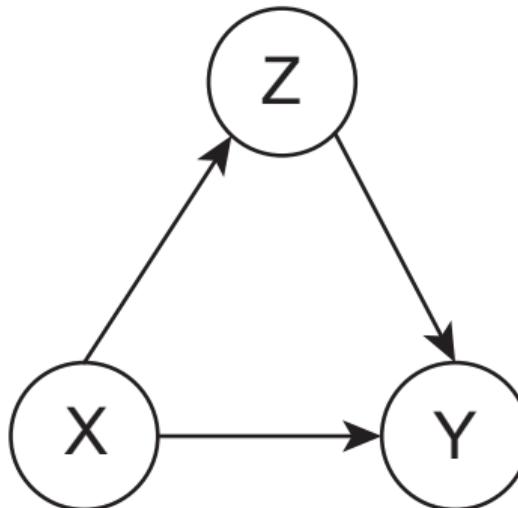
The observational DAG and SCM:

$$\begin{aligned} X &: \text{bernoulli}(0.5) \\ Z &:= 2X + \epsilon_Z \\ Y &:= 1X + 2Z + \epsilon_Y \end{aligned} \quad \left. \begin{array}{l} \text{How they're generated} \end{array} \right\}$$

In observational data set, this case we didn't wanna control for
Z, cuz we're interested in a total effect of X on Y.

where

- ▶ X is bernoulli distributed (0 or 1) with probability 0.5, ϵ_Z, ϵ_Y are iid, $\sim \mathcal{N}(0, 1)$



Interventions with DAGs and SCMs: Partial Mediation Example

The observational DAG and SCM:

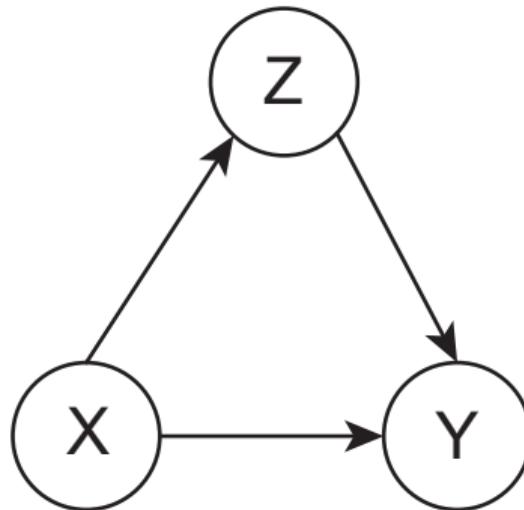
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"Naive Causal Estimate,"

Prima Facie Effect: $E[Y | X = 1] - E[Y | X = 0] =$

In this case, you already know P.F effect is the same as causal effect becuz there's no confounders or anything... 8 / 71

Interventions with DAGs and SCMs: Partial Mediation Example

The observational DAG and SCM:

$$X : \text{bernoulli}(0.5)$$

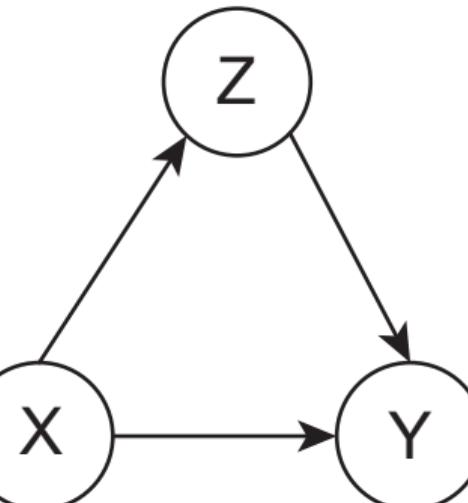
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where

- ▶ X is bernoulli distributed (0 or 1) with probability 0.5, ϵ_Z, ϵ_Y are iid, $\sim \mathcal{N}(0, 1)$

For ppl w/ $X=1$, what values do they have for Y ?
expected values



Then again look at ppl w/ $X=0$, and see the expected value of Y

Prima Facie Effect: $E[Y | X = 1] - E[Y | X = 0] =$

$5 - 0 = 5$ and this is combination of indirect path & direct path

Interventions with DAGs and SCMs: Partial Mediation Example

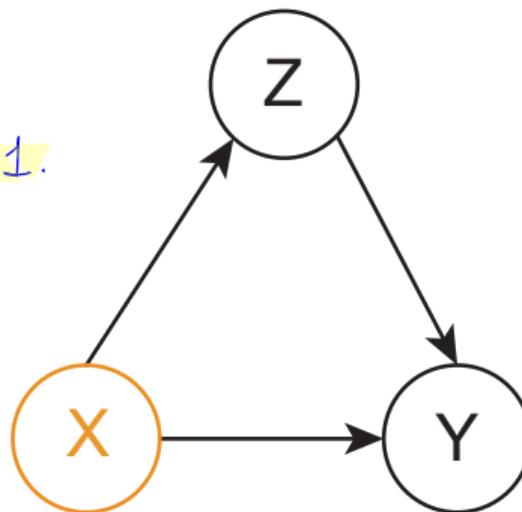
SCMs model with intervention " $do(X = 1)$ " ~ meaning the SCM for X changes

$X := 1$ ↙ No more probabilities involved any longer.
 we just force it to be 1.

$$Z := 2X + \epsilon_Z$$
$$Y := 1X + 2Z + \epsilon_Y$$

where

- ϵ_Z, ϵ_Y are iid, $\sim \mathcal{N}(0, 1)$



Interventions with DAGs and SCMs: Partial Mediation Example

SCMs model with intervention $do(X = 1)$.

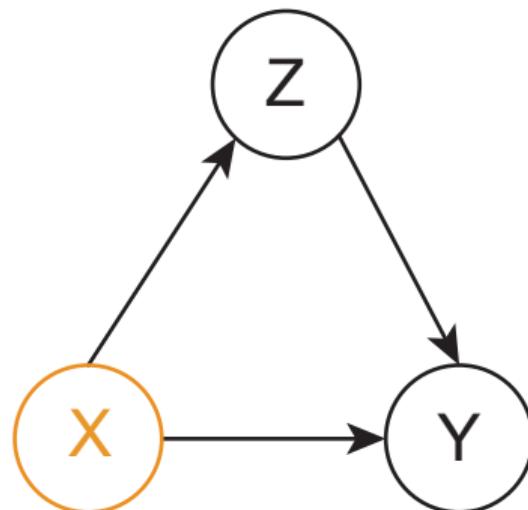
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Average Causal Effect: $E[Y | do(X = 1)] - E[Y | do(X = 0)] =$

Interventions with DAGs and SCMs: Partial Mediation Example

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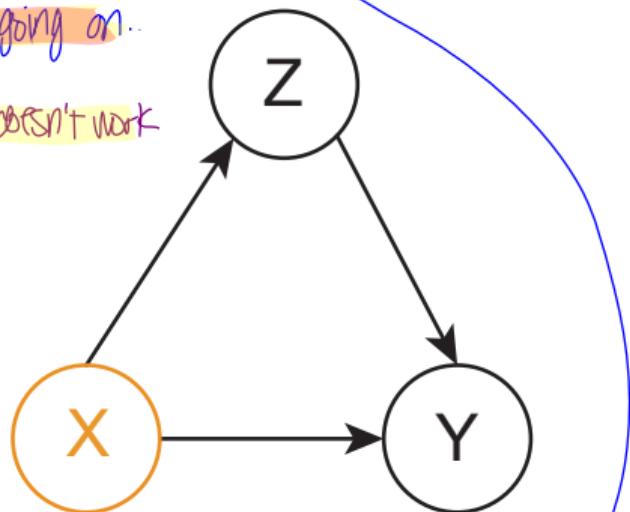
In this case that happens, becuz Prima Facie effect is a good estimator of true causal effect, becuz we don't have confounding going on.

$$X := 1$$

$$Z := 2X + \epsilon_Z$$

$$Y := 1X + 2Z + \epsilon_Y$$

It works here, but it doesn't work in general...



where

- ϵ_Z, ϵ_Y are iid, $\sim \mathcal{N}(0, 1)$

Average Causal Effect: $E[Y | do(X = 1)] - E[Y | do(X = 0)] =$

$$5 - 0 = 5$$

Same thing as before.

Interventions with DAGs and SCMs: "Partial Confounding" Example

Another observational DAG and SCM:

logistic reg.

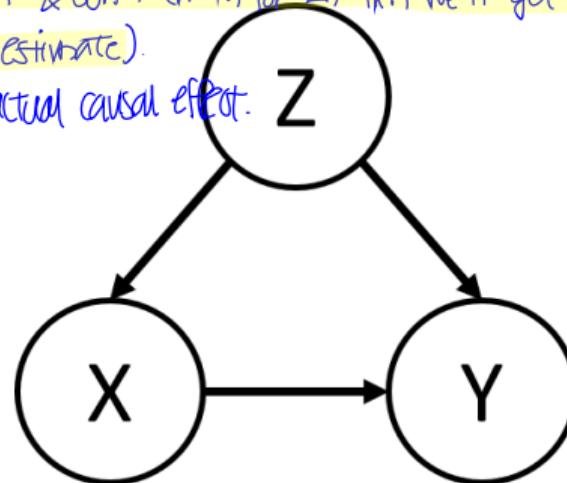
$$\text{logodds}(X) := 2Z$$

$$Z := \epsilon_Z$$

$$Y := 1X + 2Z + \epsilon_Y$$

Now we have a situation w/ confounding! \rightarrow If we just look at the effect of X on Y & don't control for Z , then we'll get a wrong estimate. (biased estimate).

You won't get an actual causal effect.



where

- ▶ X can be 0 or 1 and follows a logistic regression model, ϵ_Z, ϵ_Y are iid, $\sim \mathcal{N}(0, 1)$

Interventions with DAGs and SCMs: Partial Confounding Example

Another observational DAG and SCM:

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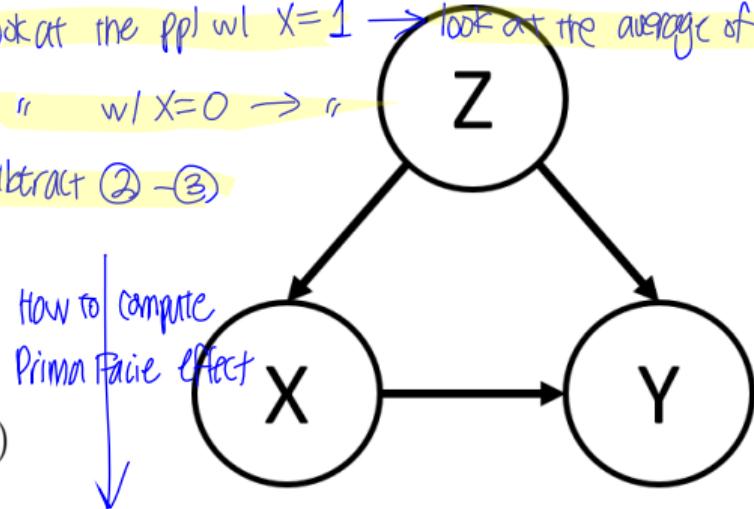
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where

- ▶ X can be 0 or 1 and follows a logistic regression model, ϵ_Z, ϵ_Y are iid, $\sim \mathcal{N}(0, 1)$

- ① Simulate the data according to SCM
- ② look at the ppl w/ $X=1 \rightarrow$ look at the average of Y in that group
- ③ " " w/ $X=0 \rightarrow$ "
- ④ subtract ② - ③



Prima Facie Effect: $E[Y | X = 1] - E[Y | X = 0] \sim \text{appx. } 4$

d

Interventions with DAGs and SCMs: Partial Confounding Example

Another observational DAG and SCM:

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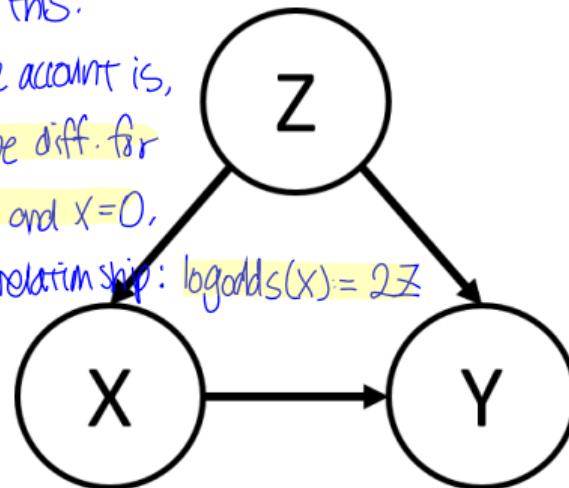
you can also prove this:

what you need to take account is,

the Z value would be diff. for

pp) that have $X=1$ and $X=0$,

becuz of the first relationship: $\text{logodds}(X) := 2Z$



where

- ▶ X can be 0 or 1 and follows a logistic regression model, ϵ_Z, ϵ_Y are iid, $\sim \mathcal{N}(0, 1)$

Prima Facie Effect: $E[Y | X = 1] - E[Y | X = 0] \sim$

$$\sim 2.5 - (-1.5) = 4$$

biased effect would be 4

If someone proves this exactly I'll treat the class to boterkoek in our next meeting
(for the approximation via simulation see rcode on bb).

Interventions with DAGs and SCMs: Example 2

SCMs model the intervention $do(X = 1)$. Since

Note: X is now no longer affected by Z . We fully control the value of X , so there's no way Z affects X anymore.

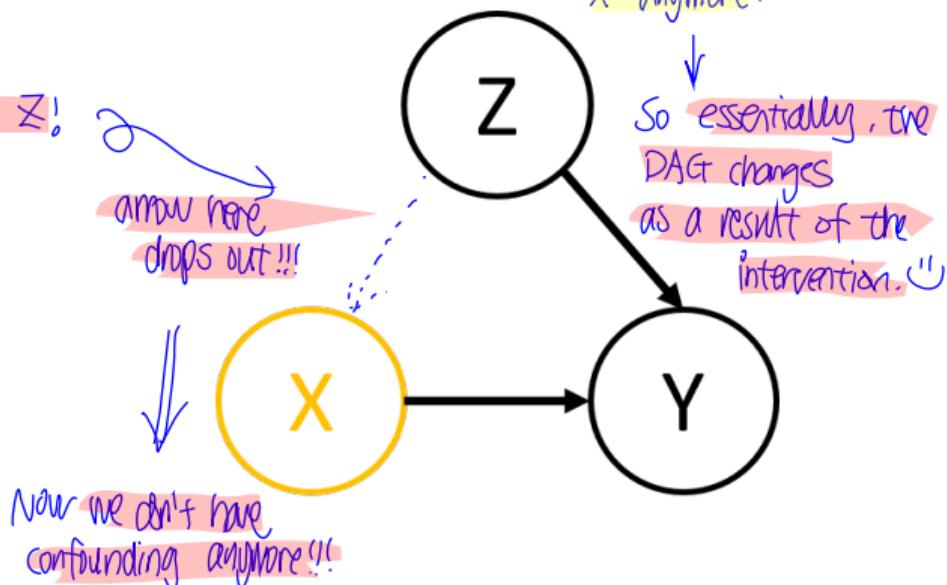
$X := 1 \rightarrow X$ no longer depends on Z !

$Z := \epsilon_Z$

$Y := 1X + 2Z + \epsilon_Y$

where

- ϵ_Z, ϵ_Y are iid, $\sim \mathcal{N}(0, 1)$



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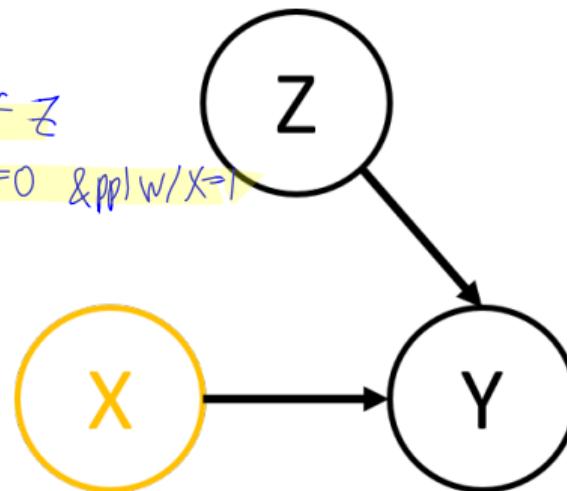
$X := 1$ ↗ In this situation, the expected value of Z

$Z := \epsilon_Z$ ↗ does not differ between $p(p|w/X=0)$ & $p(p|w/X=1)$

$$Y := 1X + 2Z + \epsilon_Y$$

where

- ϵ_Z, ϵ_Y are iid, $\sim \mathcal{N}(0, 1)$



$$1 + E(2Z) - E(2Z) = 4 \text{ actual causal effect } = 1$$

$$\text{Average Causal Effect: } E[Y | do(X = 1)] - E[Y | do(X = 0)] = \left\{ \begin{array}{l} \\ \end{array} \right.$$

in *prima facie*, it was 4.

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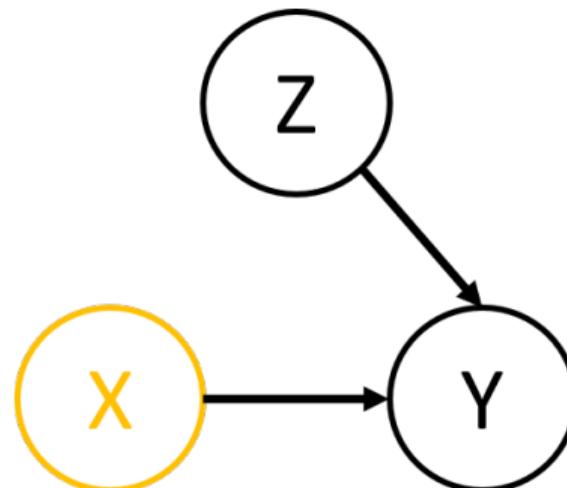
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Average Causal Effect: $E[Y | do(X = 1)] - E[Y | do(X = 0)] =$

$1 - 0 = 1$, while in the observational case, we saw 4.

Interventions vs Conditioning

The point is again, observing is not the same as intervening!

Observing/Seeing \neq Intervening/Doing:

$E[Y | A = a]$ is not necessarily the same as $E[Y | \text{do}(A = a)]$

If there's difference between the two, then there's

When statistical relationship \neq causal effect, we say the former is confounded. confounding going on.

Example 1 - partial mediation:

- ▶ $E[Y | \text{do}(X = 1)] - E[Y | \text{do}(X = 0)] = 5 - 0 = 5$
- ▶ $E[Y | X = 1] - E[Y | X = 0] = 2.5 - 0 = 5$

This is just another way of saying what confounding is!

Interventions vs Conditioning

Observing/Seeing \neq Intervening/Doing:

$E[Y | A = a]$ is *not* necessarily the same as $E[Y | do(A = a)]$

When statistical relationship \neq causal effect, we say the former is *confounded*.

Example 1 - partial mediation:

- ▶ $E[Y | do(X = 1)] - E[Y | do(X = 0)] = 5 - 0 = 5$) There was no diff.
- ▶ $E[Y | X = 1] - E[Y | X = 0] = 2.5 - 0 = 5$

Example 2 - partial confounding:

- ▶ $E[Y | do(X = 1)] - E[Y | do(X = 0)] = 1 - 0 = 1$) diff → there's confounding!
- ▶ $E[Y | X = 1] - E[Y | X = 0] \sim 2.5 - (-1.5) = 4$

Interventions vs Conditioning

Observing/Seeing \neq Intervening/Doing:

$E[Y | A = a]$ is *not* necessarily the same as $E[Y | do(A = a)]$

When statistical relationship \neq causal effect, we say the former is *confounded*.

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Example 2 - partial confounding:

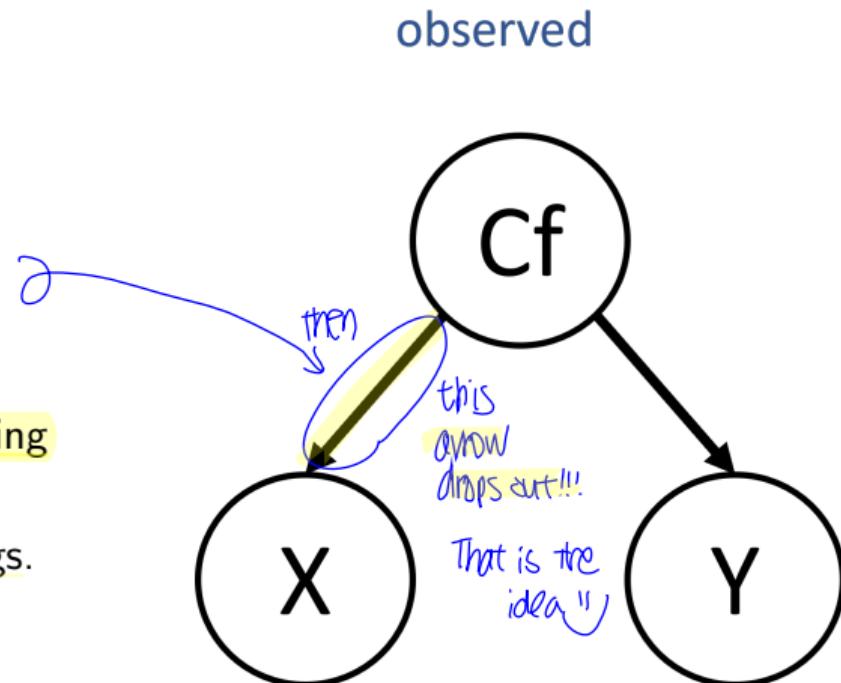
- ▶ $E[Y | do(X = 1)] - E[Y | do(X = 0)] = 1 - 0 = 1$
- ▶ $E[Y | X = 1] - E[Y | X = 0] \sim 2.5 - (-1.5) = 4$

Observationally, people with X=1 will have higher expected values for Z than people with X=0 (~ .8 vs -.8), so the mean of y will also be higher for the former!

Randomized Control Trials - Why They Work

RCTs are extremely powerful because randomization ensures no confounding.

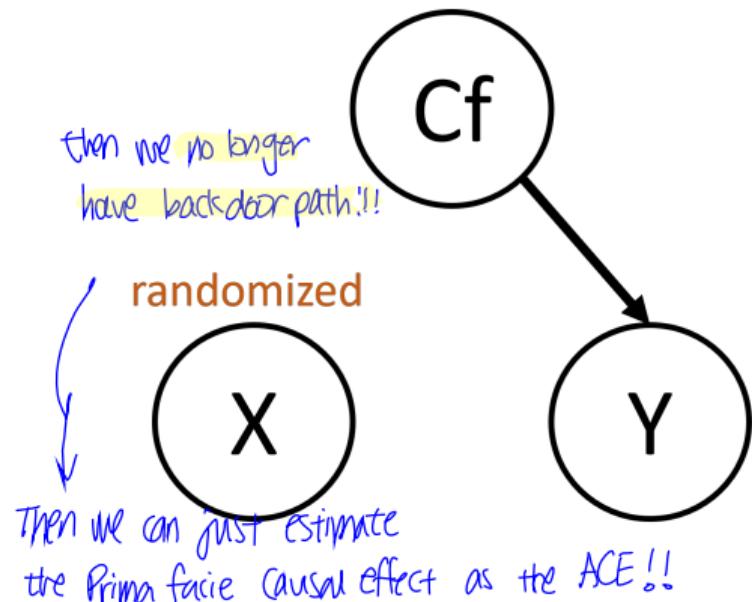
- ▶ Randomization (if successful) means having full control over the treatment variable.
- ▶ There can't be any backdoor paths if everyone has an equal probability of being treated or not
- ▶ But, RCTs not possible in many settings.



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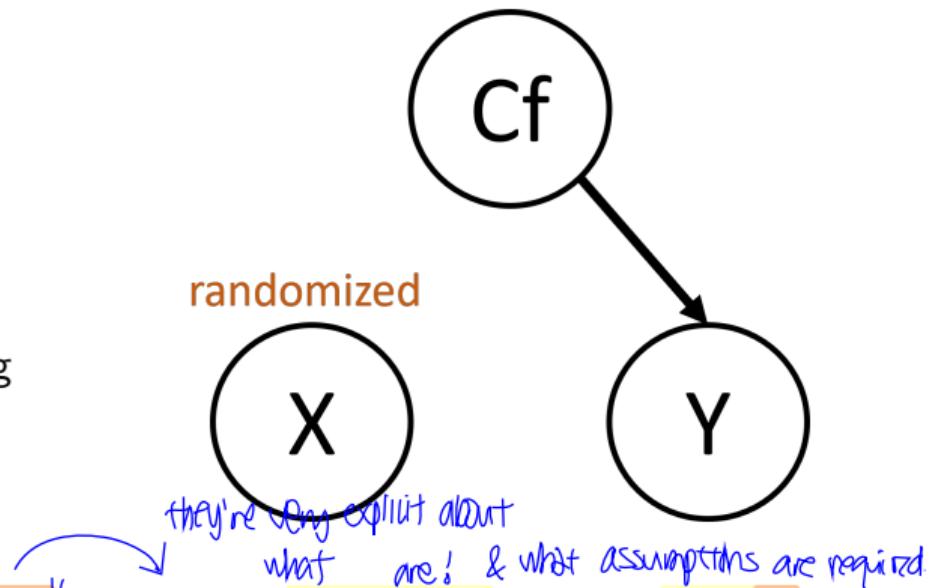
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- ▶ But, RCTs not possible in many settings.



Note: From the Potential Outcomes perspective, (successful) RCTs work because they by design adhere to all the assumptions (e.g., exchangeability, positivity, etc).

Simpsons Paradox

Simpsons Paradox

Statistical phenomena where a relationship which is present when aggregating over the population may be reversed or absent when looking at sub-populations



Changes the sign or close to zero...

when we look at the specific part of population,
the association changes in some ways..

≈ "marginal relationships are not the same as conditional relationships!!"
And it also help thinking about what the underlying DAG is..

Simpsons Paradox

Simpsons Paradox

Statistical phenomena where a relationship which is present when aggregating over the population may be reversed or absent when looking at sub-populations

Example (Pearl, Glymour & Jewell, 2016):

no randomization

- ▶ 700 sick patients are given the choice to take a new drug: 350 "choose to" take it.
- ▶ We are interested in effects of a drug (D) on recovery (R). We also record the gender (G)
- ▶ Should we prescribe the drug?

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Example (Pearl, Glymour & Jewell, 2016):

- ▶ 700 sick patients are given the choice to take a new drug: 350 choose to take it.
- ▶ We are interested in effects of a drug (D) on recovery (R). We also record the gender (G)
- ▶ Should we prescribe the drug? *<statistical result ↴>*

Table 1.1 Results of a study into a new drug, with gender being taken into account

	Drug	No drug	
Men	81 out of 87 recovered (93%)	>	234 out of 270 recovered (87%)
Women	192 out of 263 recovered (73%)	>	55 out of 80 recovered (69%)
Combined data	273 out of 350 recovered (78%)	<	289 out of 350 recovered (83%)

↳ combined data shows the "opposite" result !!

Taking drug is

a good idea!

Taking drug

is NOT good!?

Overview

- ▶ DAGs & Interventions & the RCT
- ▶ **Selection Bias, Berksons Paradox, Simpsons Paradox**
- ▶ Repeated Measures: Change Scores vs Controlling for Pre-measure

Simpsons Paradox

Counter-intuitive, but not really a paradox ~it's completely in line w/ all the probability rules and whatnot.

- ▶ A marginal dependency ($P(R | D)$) is not necessarily the same as a conditional dependency ($P(R | D, G = 0)$)
 - ▶ But which piece of information should we use to make treatment decisions?
 - ▶ (Who) should we treat?! But it still does not really help us to decide what should we do then...
Which of these dependencies are relevant..
- ↳ marginal dependency (where we don't control for Gender) is \neq conditional dependency (where we do control for Gender)

Simpsons Paradox

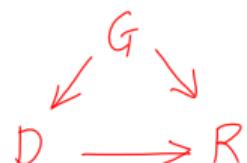
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- ▶ A marginal dependency ($P(R | D)$) is not necessarily the same as a conditional dependency ($P(R | D, G = 0)$)
- ▶ But which piece of information should we use to make treatment decisions?
- ▶ (Who) should we treat?!



Draw your DAG! based on 3 variables ↴

Variables: Gender ('G'), Drug ('D') and Recovery ('R')



Simpsons Paradox

- ▶ Estrogen levels negatively affect recovery
- ▶ Women are more likely to take the drug than men

Yes, We should condition on Gender - it blocks a backdoor path!

And then, the conclusion is: we do give drugs, becuz it helps recovering, Becuz it's a confounder in this case

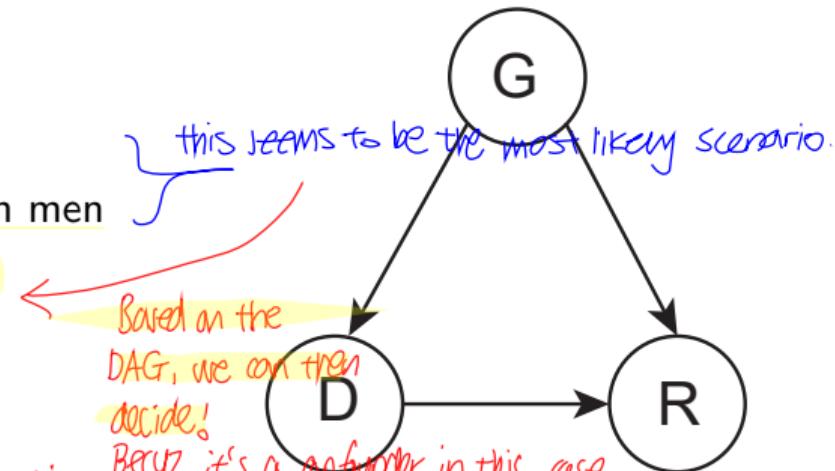


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Simpsons Paradox

Suppose that we measured **post-treatment blood pressure (B)** instead of gender, next to Drug taking (D) and Recovery (R).

Draw your DAG!

Table 1.2 Results of a study into a new drug, with posttreatment blood pressure taken into account

	Drug		No drug
Low BP	81 out of 87 recovered (93%)	>	234 out of 270 recovered (87%)
High BP	192 out of 263 recovered (73%)	>	55 out of 80 recovered (69%)
Combined data	273 out of 350 recovered (78%)	<	289 out of 350 recovered (83%)

Simpsons Paradox

Suppose that we measure post-treatment blood pressure (B) instead

- ▶ Statistical information is exactly the same!!
- ▶ B cannot cause drug taking
- ▶ The drug works in part by decreasing blood pressure
- ▶ We should not condition on blood pressure and in this case, that means we do NOT give the drugs!

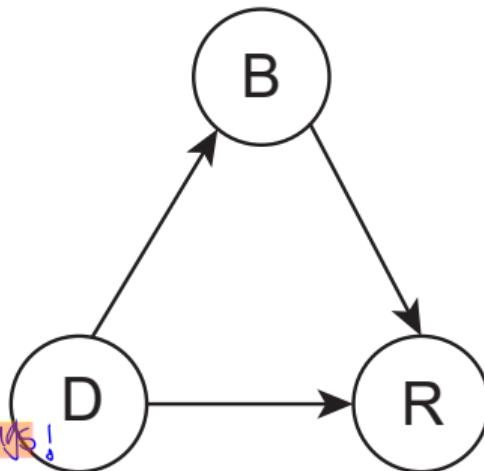
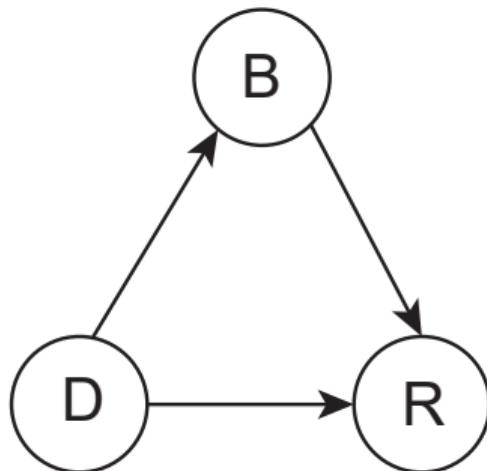
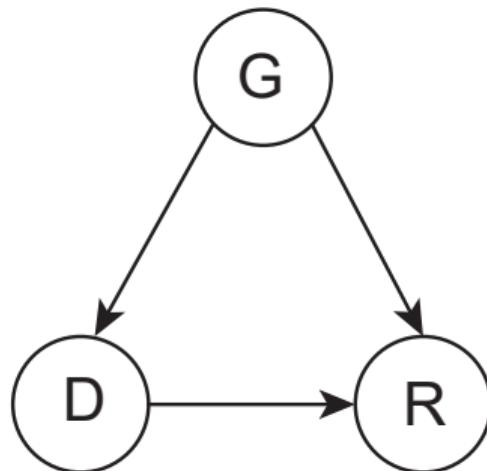


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Simpsons Paradox

- ▶ Statistical information alone cannot provide the answer on when to treat (!)
- ▶ Two different DAGs can produce the exact same statistical dependencies in the observational setting
 - ¹¹Observationally equivalent¹⁰
- ▶ These DAGs imply different causal effects, and hence different models to estimate those effects from observational data.



Selection Bias: when we condition on a specific sub-group, for ex. by doing some kinds of selection, for ex. we only look at student population... Usually it's a confounding situation becuz of how you sampled. Sampling only patients, students...etc → then we see the diff. between what we observe in the general population and what we had in a specific sub-population...

Berkson's Paradox

Two phenomena which are statistically *independent* in the general population are statistically *dependent* in a sub-population that was selected.

Also know as: Selection Bias, Endogenous Selection Bias, Berkson's bias

Classic example: We are interested in the relationship between *Lung Cancer* (L) and *Diabetes* (D)

- ▶ General population, these two variables are independent.
- ▶ In a sample of *hospital patients*, there is a negative dependency - patients who don't have diabetes are *more likely* to have lung cancer.

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Berkson's Paradox

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Again, to figure out what's going on here?
...Draw your DAG!

ppi often don't think about
Selection Bias : we get the wrong result becaz collecting patient samples, we condition on hospitalization.?

So, In Simpson's paradox, it's very explicit. we just include a certain variable in the analysis or we don't.

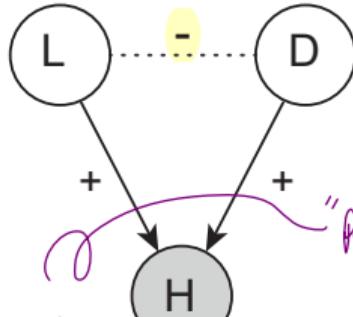
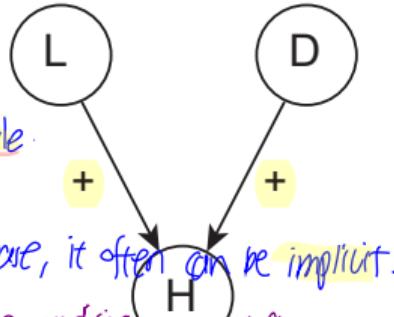
But In Berksons paradox,

we might have accidentally

condition on some kind of variable.

& you might not be aware of

this problem... In Berksons case, it often can be implicit.



"potential Berksons paradox,"

seems the most likely scenario

EX) very clear example where we condition on specific samples all the time
= student population!!! we do so many experiments exclusively in students.

- ▶ Lung cancer L and diabetes D cause hospitalization H
- ▶ By selecting participants from a hospital we condition on hospitalization ($H = 1$)
- ▶ If you are hospitalised, and you don't have diabetes, probably you do have lung cancer (Otherwise - why would you be in hospital?).
- ▶ $P(D|L = 1, H = 1) \neq P(D|L = 1) \neq P(D|do(L) = 1)$
- ▶ We have conditioned on a collider! \Rightarrow This would explain why we get a diff. result in the full population

conditional dependencies do not have to be equal to marginal dependencies

Simpsons or Berksons or?

these & dependencies do not equal to causal dependencies

Simpsons Paradox

Statistical phenomena where a relationship which is present when aggregating over the population may be reversed or absent when looking at sub-populations

Berksons Paradox ~typically mentioned in context of (pre-) selection idea

Two phenomena which are statistically *independent* in the general population are statistically *dependent* in a sub-population that was selected.

What's the difference?

- In Simpsons, we find different relations when we control vs not control for a variable
- In Berksons, we find different relations as a result of 'accidental' selection via our sampling procedure.
- Either can be the result of collider bias or confounder bias or overcontrol bias (controlling for a mediator)

Berksons

Simpsons

POINT is* \Rightarrow conditioning on a variable either by including in your analysis somehow, or by accidentally selecting a particular sample, might be problematic. It can be becuz of collider bias/confounder bias/overcontrol bias... And to clear nif what's going on \rightarrow Draw A -> G \star Think about a causal model. (It's not so impo. whether

Simpsons or Berksons or?

you call it a Simpsons / Berksons paradox)

Simpsons Paradox

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Note 1. When we have a **partial mediation** where (for example) the **direct effect is positive** and the **indirect effect is negative** (or other way around), so the **total effect is near zero** - This is called a '**supression effect**'.

↳ keep in mind : time order of variables , & what the actual possible DAG might be.

cuz remember, "mediation & Confounding" \rightarrow we cannot tell them apart !!

Simpsons or Berksons or?

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Note 1. When we have a **partial mediation** where (for example) the **direct effect is positive** and the **indirect effect is negative** (or other way around), so the **total effect is near zero** - This is called a '**supression effect**'.

Note 2. Some people relate **Simpsons** expressedly to **confounding bias** and **Berksons** to **collider bias**.

In any case...draw the causal model! ~Always a good idea!! Be very explicit about the mechanism that your summarizing

Conditioning on a variable either by including in your analysis in some ways, or by accidentally selecting a particular sample, it might be problematic. It can be becauz collider bias, confounder bias, or overcontrol bias.

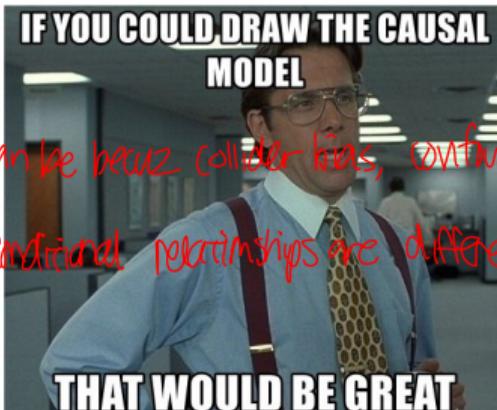
If you have this pattern, where your marginal and conditional relationships are different, Draw a DAG!

Think about the causal model! ☺



THE CAUSAL MODEL
WHAT IF I TOLD YOU

YOU COULD DRAW THE CAUSAL
MODEL



memegenerator.net

ONE DOES NOT SIMPLY

DO INFERENCE WELL WITHOUT
DRAWING THE CAUSAL MODEL

memegenerator.net



memegenerator.net

either w/ equations or w/
drawing ...

Recap: Where are we?

We are interested in **the effect of treatment X on outcome Y**:

- ▶ What is the effect of dieting on psychological well-being (Schafer & Kang, 2008)?
- ▶ What is the effect of out-of-home-placement on children's well-being (Berger et al., 2009)?
- ▶ What is the effect of physical punishment of children's behavioral problems (Larzelere et al., 2010)?
- ▶ What is the effect of extra schooling on social economic status?

If we have only **observational data** for this, we should:

- ▶ be concerned about **confounding**, collider bias, overcontrol bias
- ▶ **including the right covariates** to account for this
- ▶ we can use DAGs to see what we should control for (i.e., condition on)

But a **DAG** is of course only as good as our theory is...

What covariates should be included?

Steiner, Cook, Shadish and Clark (2010) used a creative design:

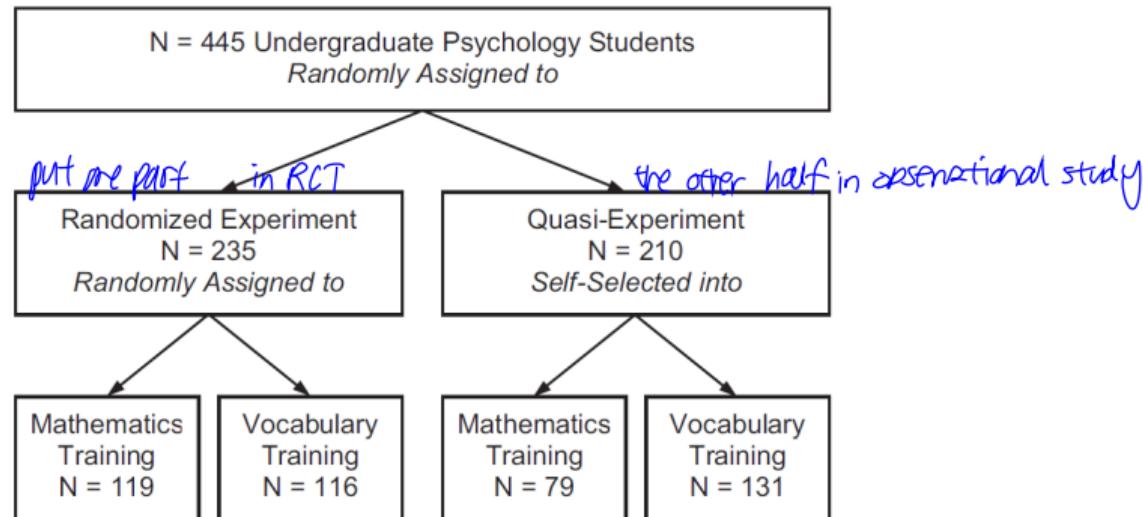
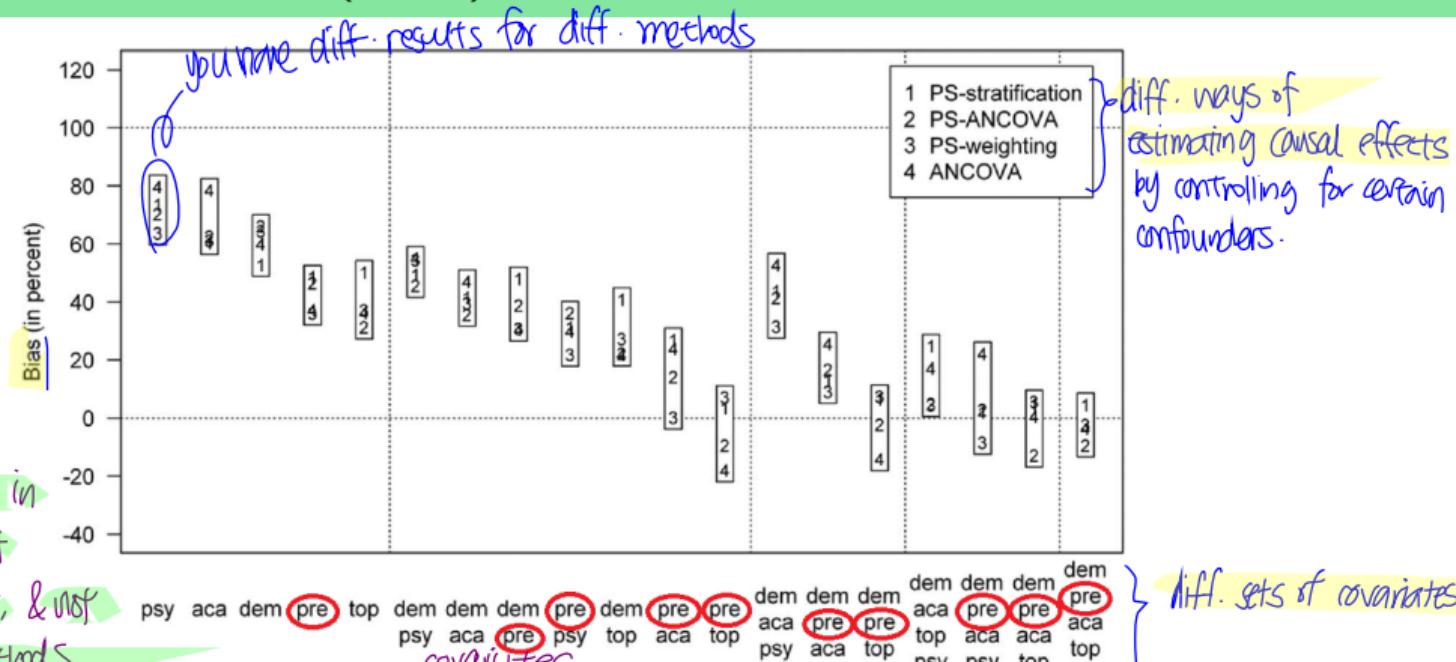


Figure 1. Overall design of the within-study comparison of the randomized experiment and the quasi-experiment.

This allows for a comparison of RCT results (true ACE) and observational results.

↳ How well the obs.-study is recovering the true causal effect, assuming RCT was done properly

Results from Steiner et al. (2010)



• What they also found:

Overall, pre-test measure seems a valuable covariate to include.

very often including "pre-treatment" as a confounder decreases bias

Overview

p
ofc, you can already make a DAG
in your mind & see when this might
or might not apply!
That's what we're gonna do
now... ☺

- ▶ DAGs & Interventions & the RCT
- ▶ Selection Bias, Berksons Paradox, Simpsons Paradox
- ▶ **Repeated Measures: Change Scores vs Controlling for Pre-measure**

Pre-Post Designs

Pre-post test designs: when the outcome is measured twice

- ▶ Lord's paradox
- ▶ ANCOVA vs. change score analysis
- ▶ Five scenarios
- ▶ How DAGs can help (Pearl, 2016)
- ▶ Unmeasured confounders (Kim & Steiner, 2019)

Lord's paradox

Comparing non-randomly assigned groups

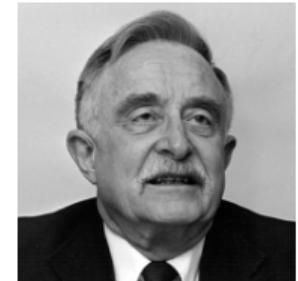
Psychological Bulletin
1967, Vol. 68, No. 5, 304-305

A PARADOX IN THE INTERPRETATION OF GROUP COMPARISONS

FREDERIC M. LORD

Educational Testing Service

Attention is called to a basic source of confusion in the interpretation of certain types of group comparison data.



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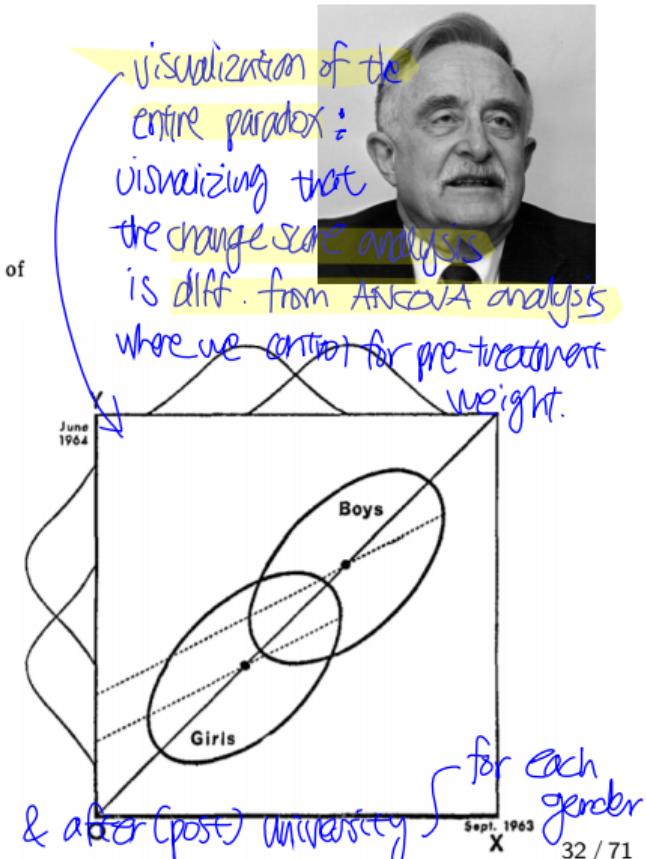
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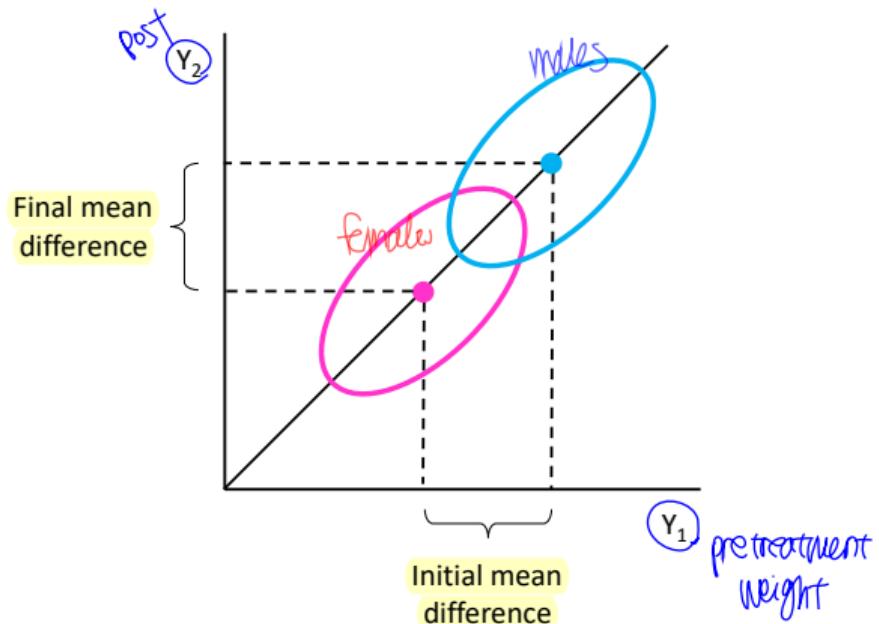
"A large university is interested in investigating the effects on the students of the diet provided in the university dining halls and any sex difference in these effects." (p.304)

treatment var. = diet

measure the weight before (pre) & after (post) university

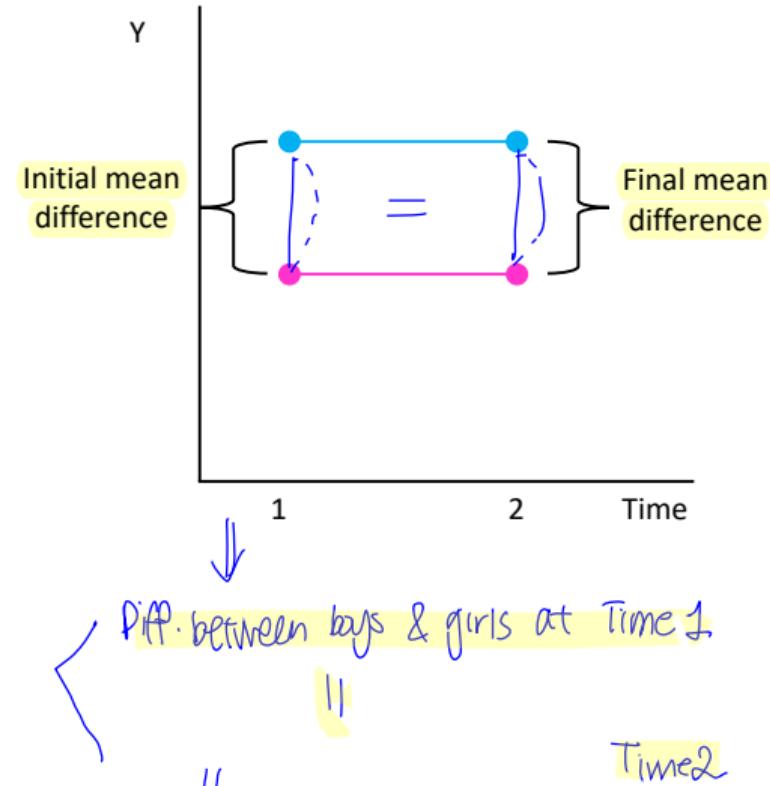
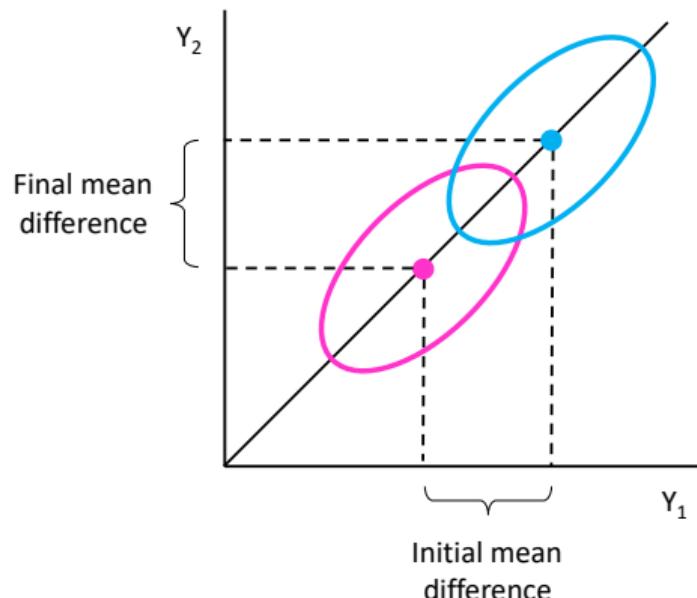


Statistician 1: Looks at the difference in the differences

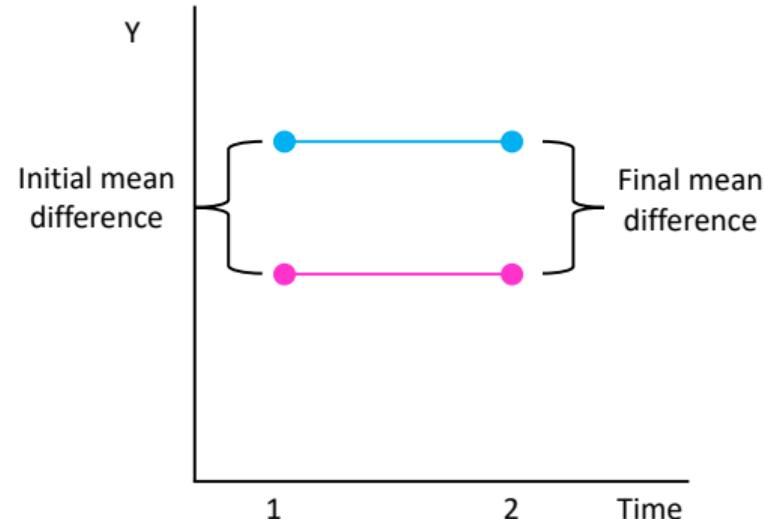
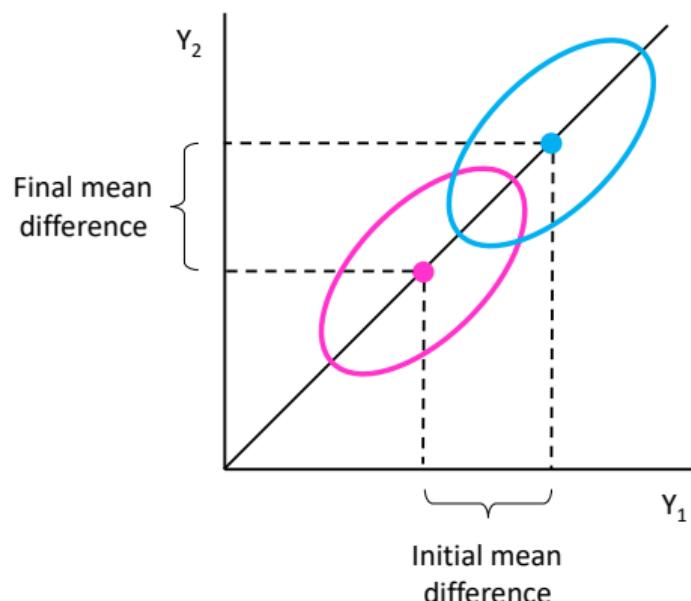


⇒ Diff. in pre-weight between males & females is the same as in post-weight difference!

Statistician 1: Looks at the difference in the differences



Statistician 1: Looks at the difference in the differences

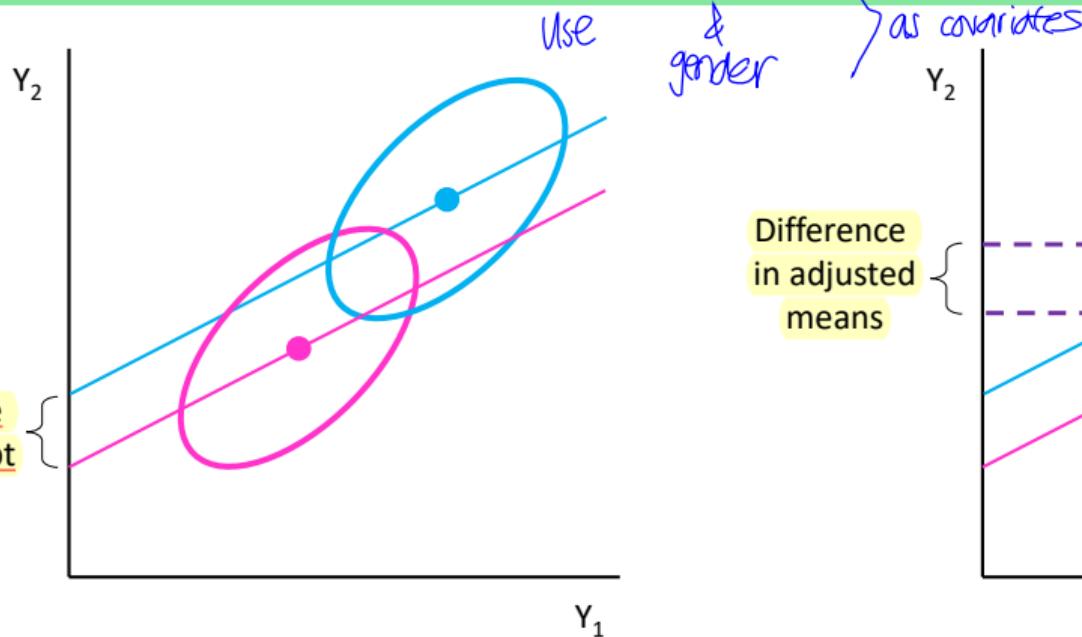


Conclusion: No change per group, so no difference in their change either.

University diet doesn't really matter...

Or more generally: There is no difference over time in the differences between the groups.

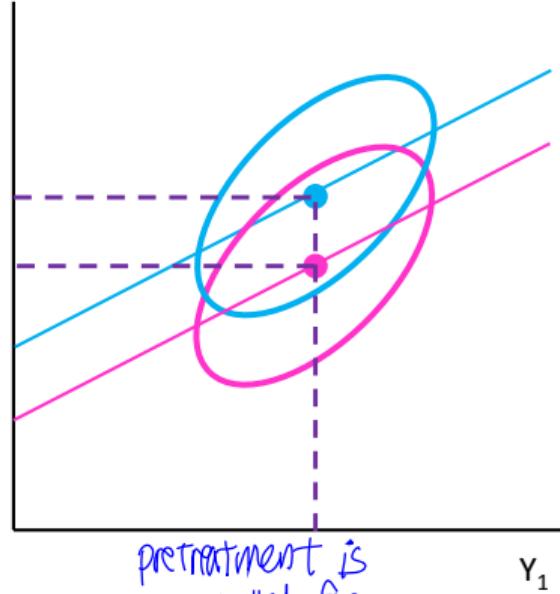
Statistician 2: Uses ANCOVA - Pre-weight as control variable



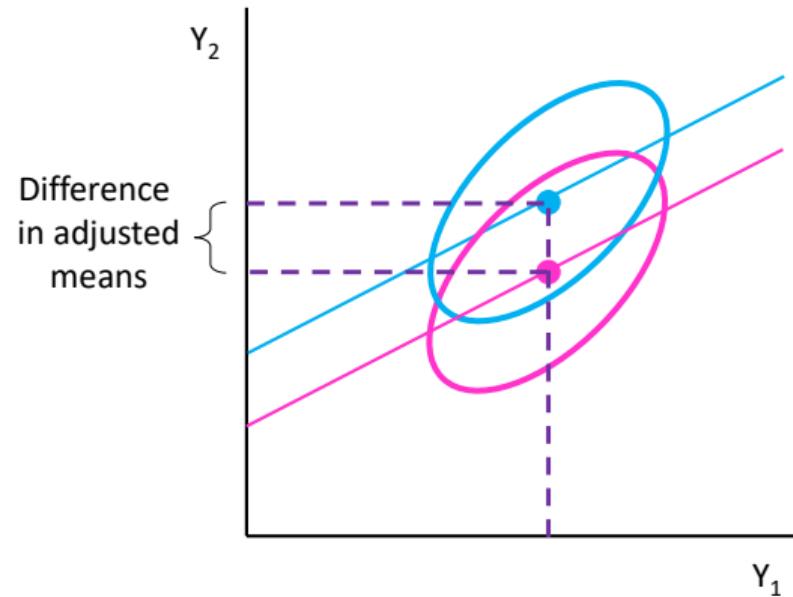
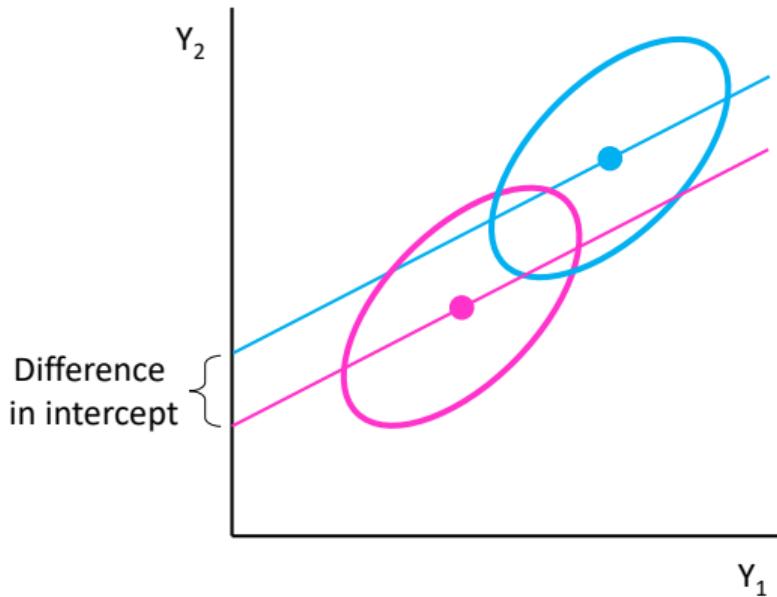
now we DO see
differences between them!

If we were to compare boys & girls that have the same pre-weights, then the adj mean in post-weights differ!!

Difference
in adjusted
means

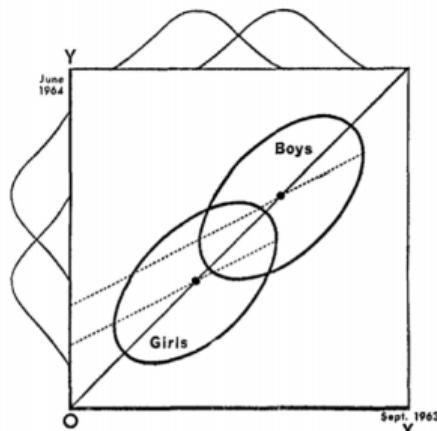


Statistician 2: Uses ANCOVA - Pre-weight as control variable



Conclusion: When comparing a boy and girl of **equal weight to begin with**, the **boy tends to weigh more afterwards** than the girl; hence, **there is a difference, after correcting for initial differences!**

Sex differences in effect of dining hall diet



Statistician 1: No difference Statistician 2: Boys gain more than
girls

FIG. 1. Hypothetical scatterplots showing initial and final weight for boys and for girls.

→ So in essence, he also is saying that statistics is not sufficient to decide what the correct approach is.

Lord's conclusion (p.305, 1967):

"The researcher wants to know how the groups would have compared if there had been no preexisting uncontrolled differences. The usual research study of this type is attempting to answer a question that simply cannot be answered in any rigorous way on the basis of available data."

Lord's paradox in empirical research

Larzelere et al. (2010) studied the effect of **corrective actions** on **problem behaviors** of 1,464 children aged 4 and 5.

Corrective action	ANCOVA result	Change score result
Antisocial behavior		
Professional interventions		
Psychotherapy visits	.07**	.00
Ritalin	.07**	.04
Parental disciplinary actions		
Non-physical punishment	.03	-.08**
Physical punishment	.07**	-.05
Scolding/yelling	.06*	-.08**
“Hostile/ineffective” scale	.09**	-.15**
Hyperactivity		
Professional interventions		
Psychotherapy visits	.03	-.02
Ritalin	.05*	-.00
Parental disciplinary actions		
Non-physical punishment	.07**	.01
Physical punishment	.03	.01
Scolding/yelling	.04*	-.05
“Hostile/ineffective” scale	.09**	-.08**

You see there arise very diff patterns.

In ANCOVA context, "disciplining action" seems to increase antisocial behavior.

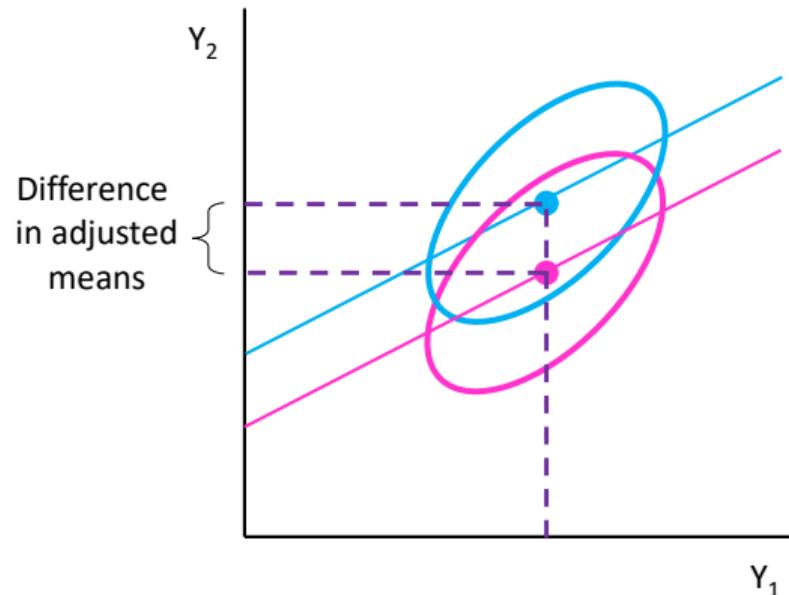
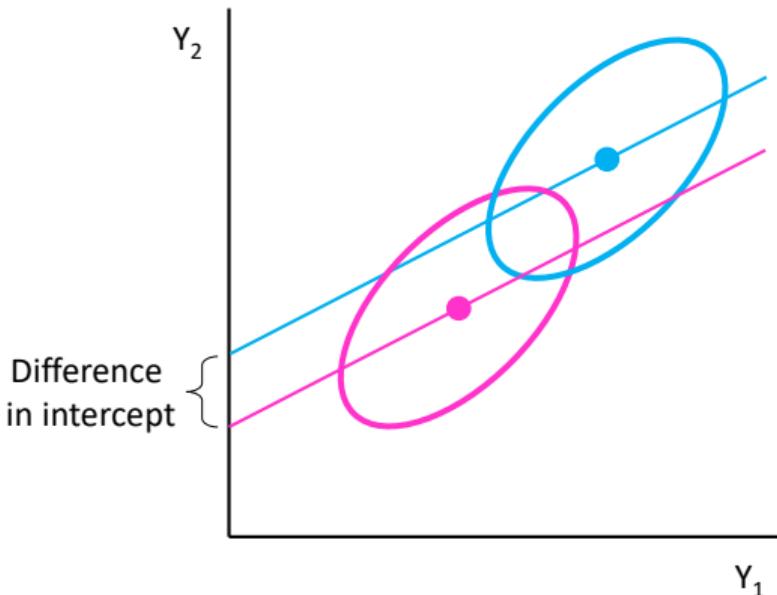
In Change score, it decreases antisocial behavior. similar w/ hyperactivity.

⇒ Not figuring out what result is correct, it'll imply very different practical advice to parents!!!

ANCOVA vs. Change score model

ANCOVA (popular in psychology)

Analysis of covariance (ANCOVA) in a pre-posttest design is based on including the pretest as covariate.



ACE based on ANCOVA model

The causal effect β_1 in the ANCOVA model is the difference between treated and untreated persons with identical values on covariate (i.e. the pretest). We can write this as the expected difference in potential outcomes:

$$ACE_{ANCOVA} = E[Y_2^1] - E[Y_2^0]$$

$$= E[Y_2^1 | X = 1, Y_1] - E[Y_2^0 | X = 0, Y_1]$$

No unobserved confounding

$$= \underline{E[Y_2 | X = 1, Y_1]} - \underline{E[Y_2 | X = 0, Y_1]}$$

Consistency

$$= (\beta_0 + \beta_1 + \beta_2 Y_1) - (\beta_0 + \beta_2 Y_1)$$

$$\text{here } X=1 \quad \text{here } X=0 \rightarrow \beta_1 \cdot 0$$

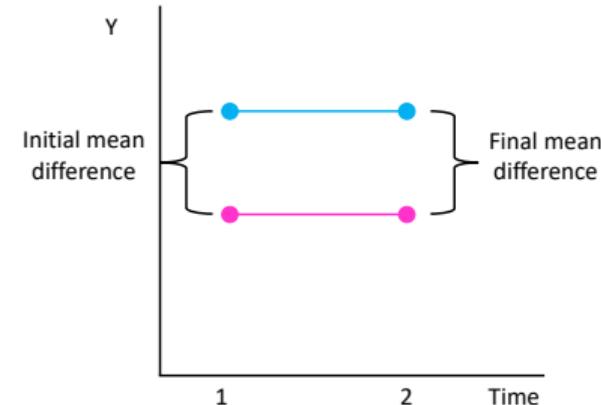
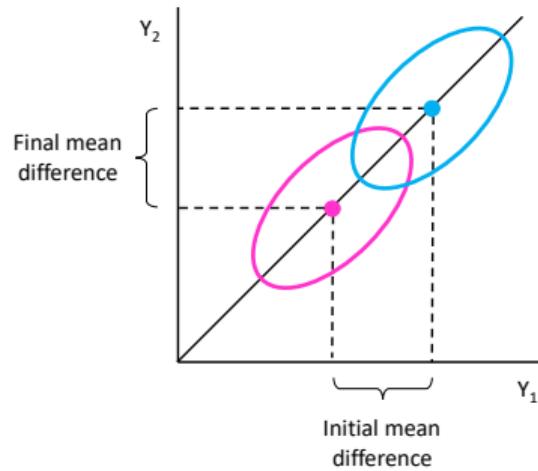
Correct model

$$= \beta_1$$

our estimate of effect of treatment X : just the regression coefficient of our treatment variable is our estimate of causal effect.

Change score model (popular in econometrics)

The change score model (or gain score model) is based on investigating difference-in-differences.



Change score model:

$$Y_{2i} - Y_{1i} = \gamma_0 + \gamma_1 X_i + \epsilon_{2i}$$

~ regress only on the treatment variable

only control for X : the treatment of interest

where γ_1 is interpreted as the causal effect of X .

ACE based on Change score model

Let $G_i = Y_{2i} - Y_{1i}$ represent a person's **gain score** (aka change or difference score).

Then the **average causal effect** can be expressed as the expected difference in potential outcomes of G_i :

$$\begin{aligned} ACE_{CS} &= E[G^1] - E[G^0] \\ &= E[G^1|X = 1] - E[G^0|X = 0] && \text{No unobserved confounding} \\ &= E[G|X = 1] - E[G|X = 0] && \text{Consistency} \\ &= E[\gamma_0 + \gamma_1] - E[\gamma_0] && \text{Correct model} \\ &= \underline{\gamma_1} \end{aligned}$$

Alternative expression of ACE_{CS}

Instead of expressing the **ACE** of the changes score model in terms of the regression parameters, we can also express it in terms of the **pre- and post-test means**, that is:

$$\begin{aligned} ACE_{CS} &= E[G^1] - E[G^0] \\ &= E[G^1|X = 1] - E[G^0|X = 0] && \text{No unobserved confounding} \\ &= E[G|X = 1] - E[G|X = 0] && \text{Consistency} \\ &= E[\{Y_2 - Y_1\}|X = 1] - E[\{Y_2 - Y_1\}|X = 0] \\ &= (E[Y_2|X = 1] - E[Y_1|X = 1]) - (E[Y_2|X = 0] - E[Y_1|X = 0]) \end{aligned}$$

ACE_{CS} as difference-in-differences

Thus we have

$$ACE_{CS} = (E[Y_2|X=1] - E[Y_1|X=1]) - (E[Y_2|X=0] - E[Y_1|X=0])$$

diff. over time in group 1 diff. over time in group 0

that is, the **ACE is equal to the difference between groups in their gain scores.**

Alternatively, we can write

$$ACE_{CS} = (E[Y_2|X=1] - (E[Y_2|X=0])) - (E[Y_1|X=1] - E[Y_1|X=0])$$

group diff. at time 2 group diff. at time 1

that is, the **ACE is equal to the difference over time in the difference between the groups (difference-in-differences).**

This
is
usually
seen
in
change score
model

Conclusion so far

With the **ANCOVA model** we answer the question: If the groups had been equal on the pre-test, would we observe a difference between them on the post-test?

$Y_{2i} = \beta_0 + \beta_1 X_i + \beta_2 Y_{1i} + e_{2i}$ If so (i.e., $\beta_1 \neq 0$), we conclude **treatment has an effect**.

With the **CS model**, we answer the question: Is the change over time different for the two groups?

$Y_{2i} - Y_{1i} = \gamma_0 + \gamma_1 X_i + \epsilon_i$ If so (i.e., $\gamma_1 \neq 0$), we conclude **treatment has an effect**.

It's impo. to realize that these are in essence diff. questions, which in result might have diff. answers.



Conclusion so far

With the **ANCOVA model** we answer the question: If the groups had been equal on the pre-test, would we observe a difference between them on the post-test?

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Yet, these questions can lead to **different answers**!

Question: Would it help if we could decide whether we are interested in Y_{2i} or $G_i = Y_{2i} - Y_{1i}$ as the outcome? → They're diff. effects.. okay.. But still then we want to know what should we do then?

We have **two models**:

- ↳ ANCOVA: $Y_{2i} = \beta_0 + \beta_1 X_i + \beta_2 Y_{1i} + e_{2i}$
- ↳ CSM: $Y_{2i} - Y_{1i} = \gamma_0 + \gamma_1 X_i + \epsilon_i$

- 1) **Rewrite** the ANCOVA model as a changes score model (i.e., with G_i as the outcome); what does this tell you?
- 2) **Rewrite** CSM as ANCOVA model (i.e., with Y_{2i} as the outcome); what does this tell you?

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- It shows that doing an ANCOVA controlling for pre-test with either Y_{2i} or $G_i = Y_{2i} - Y_{1i}$ as the outcome leads to the same effect of X_i . *this will stay intact.*
 - reg. coef. for the pre-treatment variable is $\beta_2 - 1$

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- 2) CSM as ANCOVA: $Y_{2i} = \gamma_0 + \gamma_1 X_i + Y_{1i} + \epsilon_i$ This shows that the CSM can be considered a special case of ANCOVA (with $\beta_2 = 1$). In original ANCOVA situation, β_2 can take diff. values.
 ⇒ So, in a very specific situation, two models may give the same results!! for ex, when $\beta_2 = 1$, then no

DIY: When are the ACEs the same?

also other scenarios ...

When will the ANCOVA model and the change score model give the same ACE?

$$ACE_{CS} = (E[Y_2|X=1] - E[Y_2|X=0]) - (E[Y_1|X=1] - E[Y_1|X=0])$$

If we express the change score
ACE in terms of β_1 & β_2 , we
get this

$$ACE_{ANCOVA} = \beta_1$$

$$ACE_{CS} = \beta_1 + (\beta_2 - 1)(E[Y_1|X=1] - E[Y_1|X=0])$$

difference between pre-treatment between the groups

ANSWER: These are **identical when** $(\beta_2 - 1)(E[Y_1|X=1] - E[Y_1|X=0]) = 0$

there're 2 scenarios !!

meaning that

~ there's no diff. in pre-treatment between two groups

We'll also get the same result !! as w/ ANCOVA

This is the case when either:

1) $\beta_2 = 1$: the **effect of the pretest on posttest within groups is 1** (as you had already found when rewriting a CSM as an ANCOVA model); or

2) $(E[Y_1|X=1] - E[Y_1|X=0])$: there are **no initial group differences** (as in an RCT!)
 $M_{Y1} = M_{Y0}$

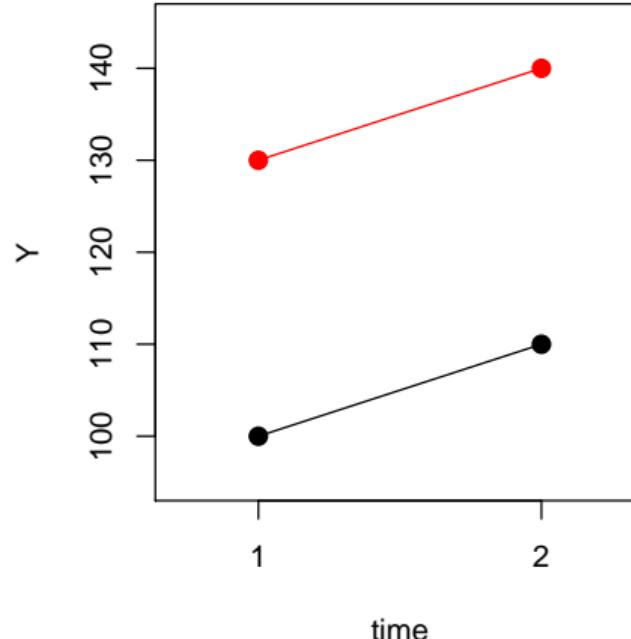
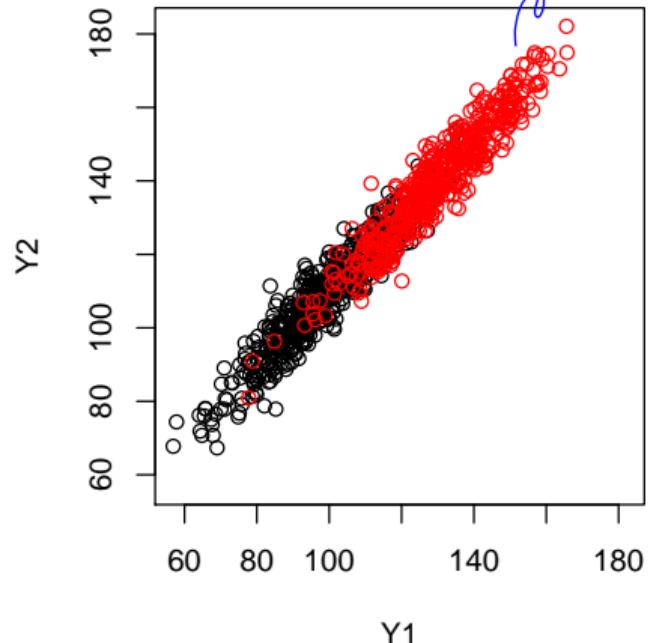
where there's no pre-treatment diff

Five scenarios

Scenario 1: No causal effect

shape here, bcz

$\beta_2 = 1$: relationship between Y_1 & Y_2 is 1.

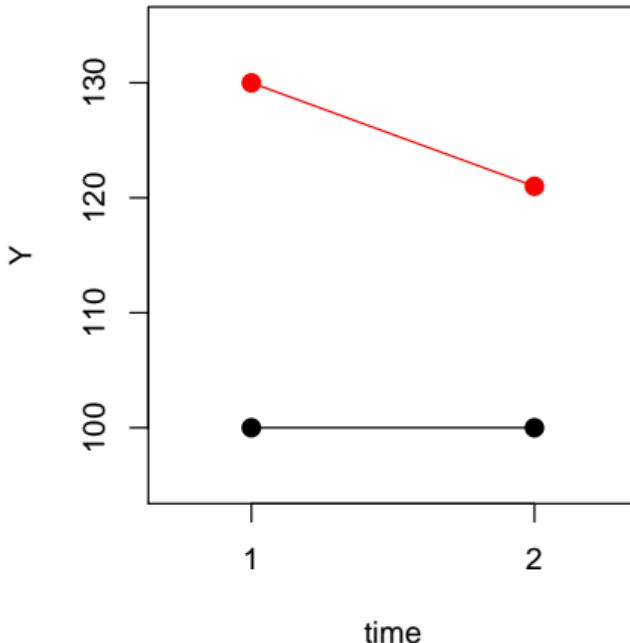
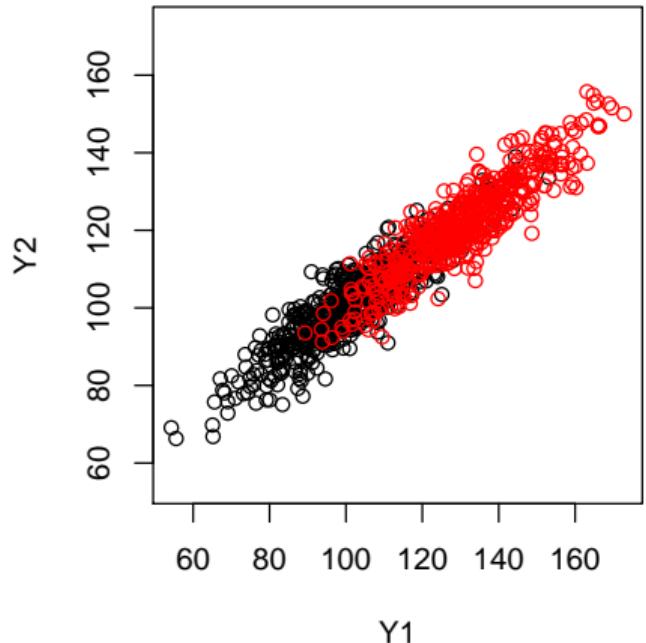


ANCOVA: $\beta_1 = 0$ so no causal effect

CSM: $\gamma_1 = 0$ so no causal effect

Scenario 2: CS model negative causal effect

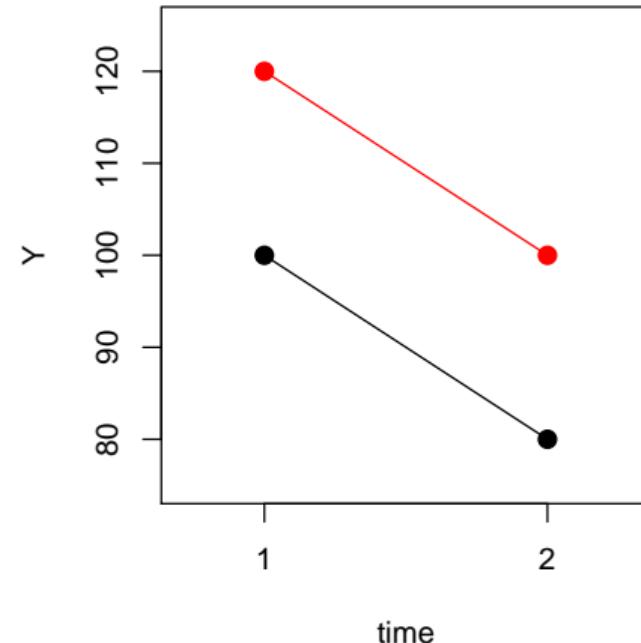
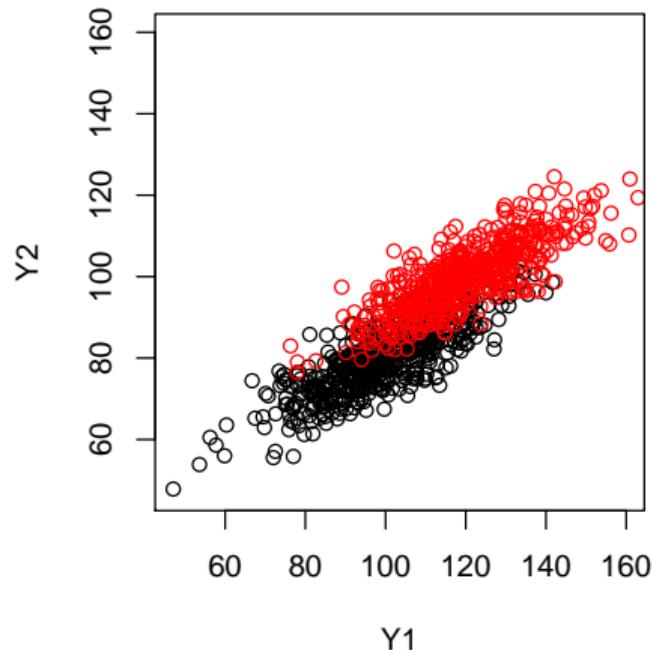
~They have diff. results.
↳ Beacuz there're group differences in pre-treatment.



ANCOVA: $\beta_1 = 0$ so no causal effect

CSM: $\gamma_1 < 0$ so a (negative) causal effect

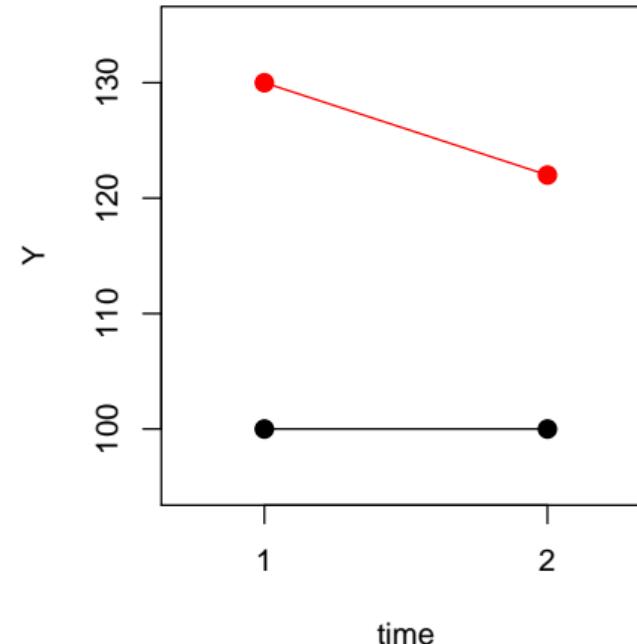
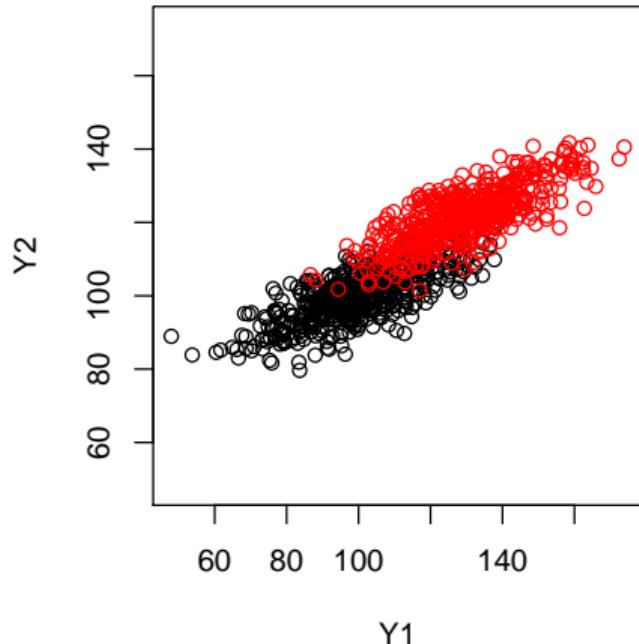
Scenario 3: ANCOVA model positive causal effect



ANCOVA: $\beta_1 > 0$ so a (positive) causal effect

CSM: $\gamma_1 = 0$ so no causal effect

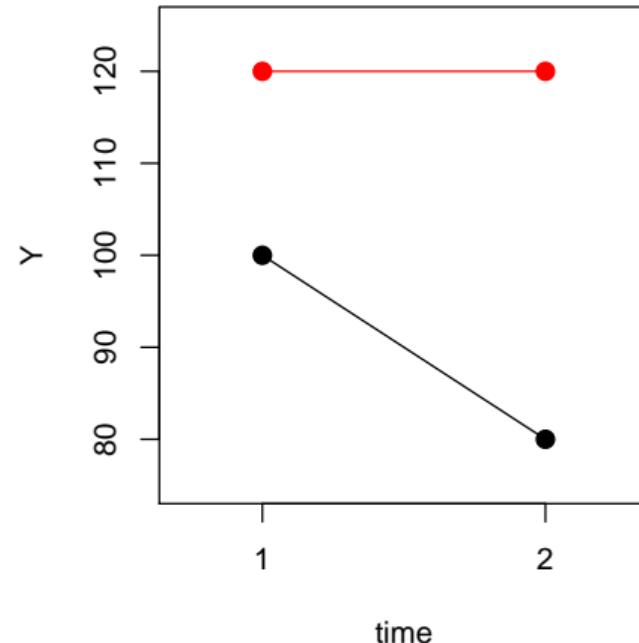
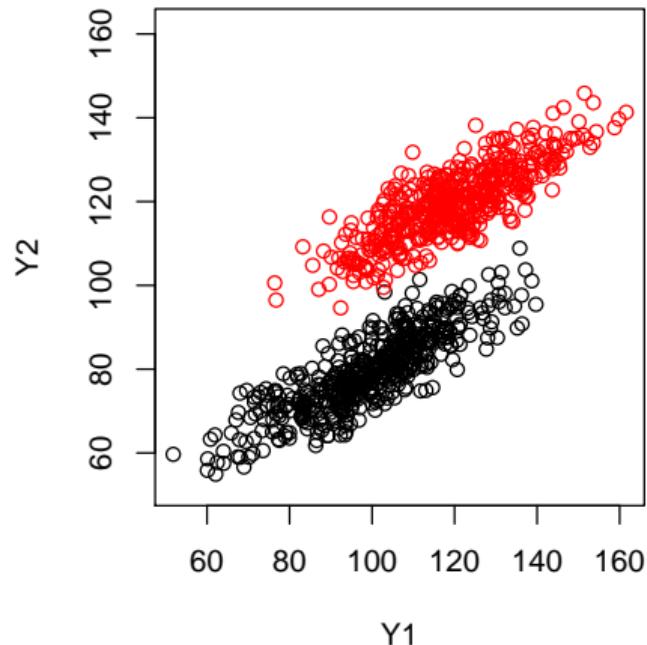
Scenario 4: Opposite conclusions regarding direction!



ANCOVA: $\beta_1 > 0$ so a (positive) causal effect

CSM: $\gamma_1 < 0$ so a (negative) causal effect

Scenario 5: Some agreement



ANCOVA: $\beta_1 > 0$ so a (positive) causal effect

CSM: $\gamma_1 > 0$ so a (positive) causal effect

So what now?

* It can be shown that only when 1) $\beta_2 = 1$ and/or 2) $\mu_{1|1} = \mu_{1|0}$, are β_1 (ACE_{ANCOVA}) and γ_1 (ACE_{CSM}) identical.

Allison (p.109, 1990):

"It is unrealistic to expect either model to be best in all situations; [...] the choice will rarely be obvious, and there will almost always be some residual uncertainty. One should also consider the possibility that neither of these models is appropriate [...]."

Allison (p.100, 1990):

"A problem with much of the work comparing change score and regressor variable methods is that the conclusions are rarely based on an explicit model for generation of the data."

Draw your DAG! → to figure out what the right thing is.

How DAGs can help

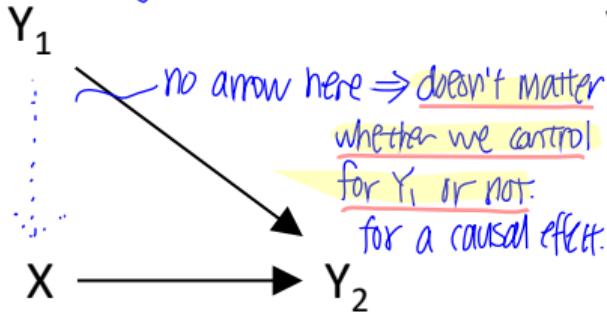
One thing we saw in the previous result is that,
so as we include the pre-test as a control variable,
also in the change-score model, then it becomes equivalent to ANCOVA.
So \Rightarrow one impo. consideration is: (Do we need to control for pre-treatment
or not?)

Should we control for pretest? - Y₂ as outcome

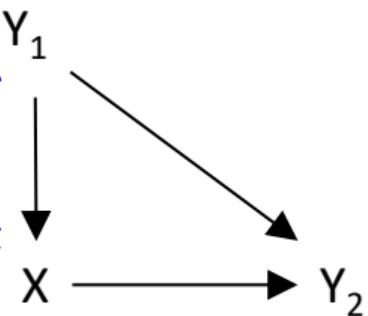
Ofc, it depends on the actual DAG!

The key issue is whether **assignment** is:

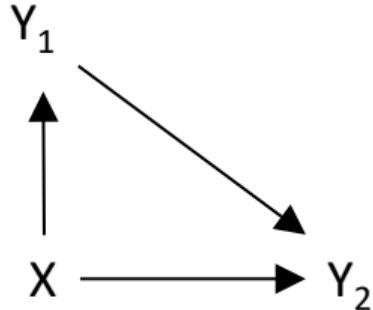
Random assignment



Based on pretest



Treatment status determines the pre-treatment
Existing groups: mediating situation



ANCOVA is preferred,
(statistical)

it has **more power**

But in terms of getting the right causal effect,
it doesn't matter if we include
it or not.

ANCOVA is correct;

pretest is a **confounder**

↓
And in that case, we
need to control for it to remove
confounding

We do not want to control unless you're only
interested in
ANCOVA gives **direct effect**

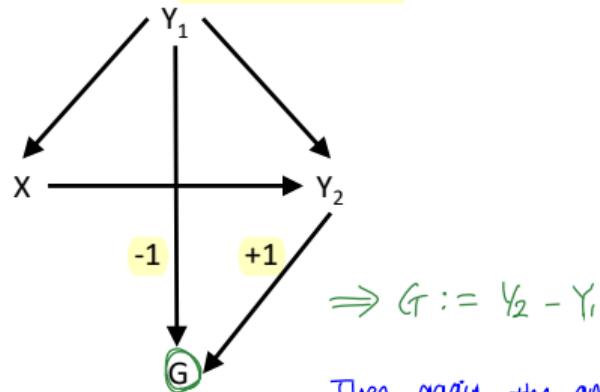
pretest is a **mediator**

↓
we do not want to control for
pre-treatment if we're interested in
Total effect.

Controlling for Pre-Treatment - change score

Change/Gain score: $G_i = Y_{2i} - Y_{1i}$

Pretest as confounder



Need to block backdoor paths:

$$X \leftarrow Y_1 \rightarrow G$$

$$X \leftarrow Y_1 \rightarrow Y_2 \rightarrow G$$

Control for pre-treatment (use ANCOVA) to get

$$X \rightarrow Y_2 \rightarrow G$$

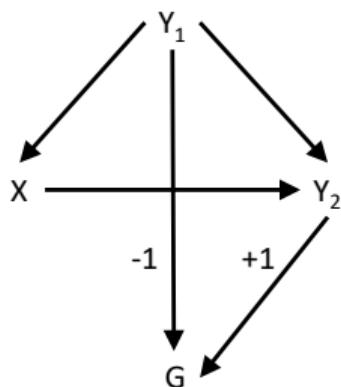
Then, again, the answer is the same as before:

we should actually include pre-treatment in our change-score analysis to get the correct causal effect, the change-score.

Controlling for Pre-Treatment - change score

Change/Gain score: $G_i = Y_{2i} - Y_{1i}$

Pretest as confounder



Need to block backdoor paths:

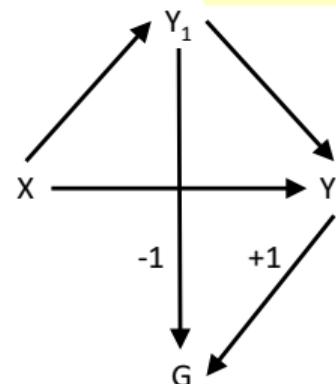
$$X \leftarrow Y_1 \rightarrow G$$

$$X \leftarrow Y_1 \rightarrow Y_2 \rightarrow G$$

Control for pre-treatment (use ANCOVA) to get

$$X \rightarrow Y_2 \rightarrow G$$

Pretest as mediator



Total effect consists of:

$$X \rightarrow Y_2 \rightarrow G$$

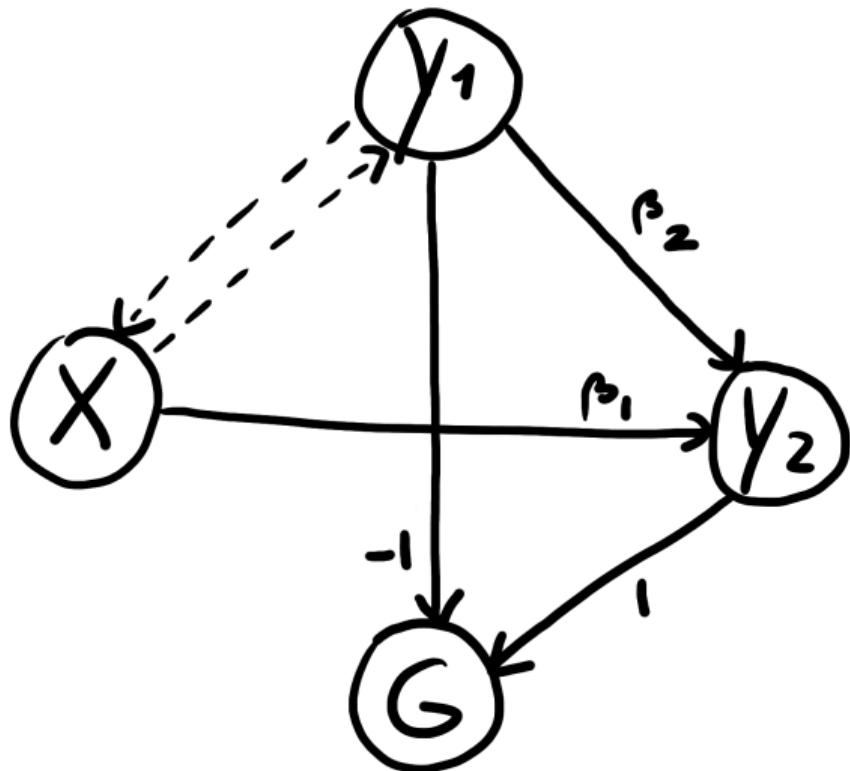
$$X \rightarrow Y_1 \rightarrow G$$

$$X \rightarrow Y_1 \rightarrow Y_2 \rightarrow G$$

And then again, we do not want to control for pre-treatment.

Do not control for pre-treatment (classical change score model) to get total effect; control for pre-treatment (ANCOVA) to get direct effect.

Revisit: When classical Change Score and ANCOVA model have the same results

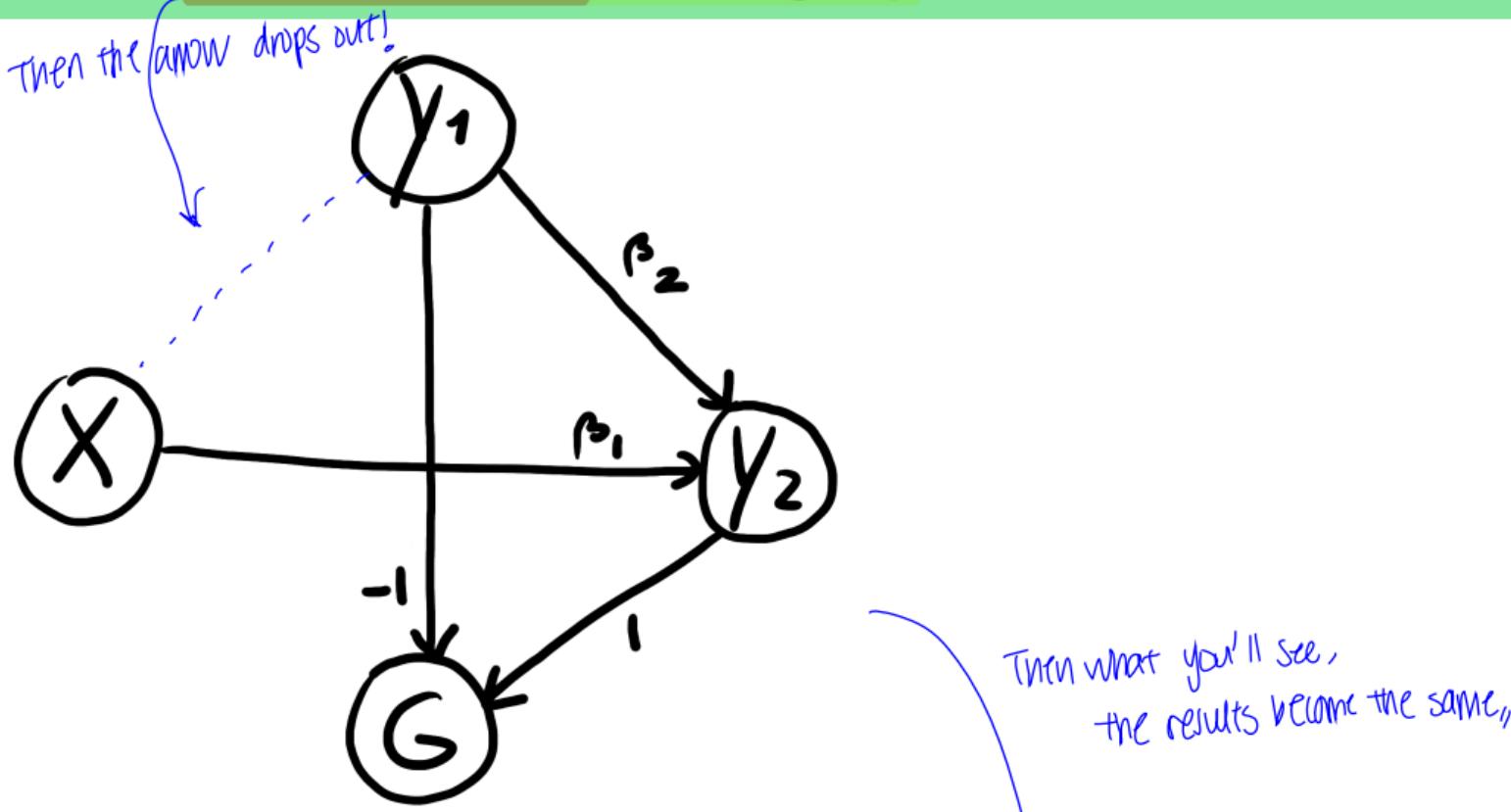


When the expected values for Y_1 are the same in each group (no group differences in Y_1) $\mu_{11} = \mu_{10}$

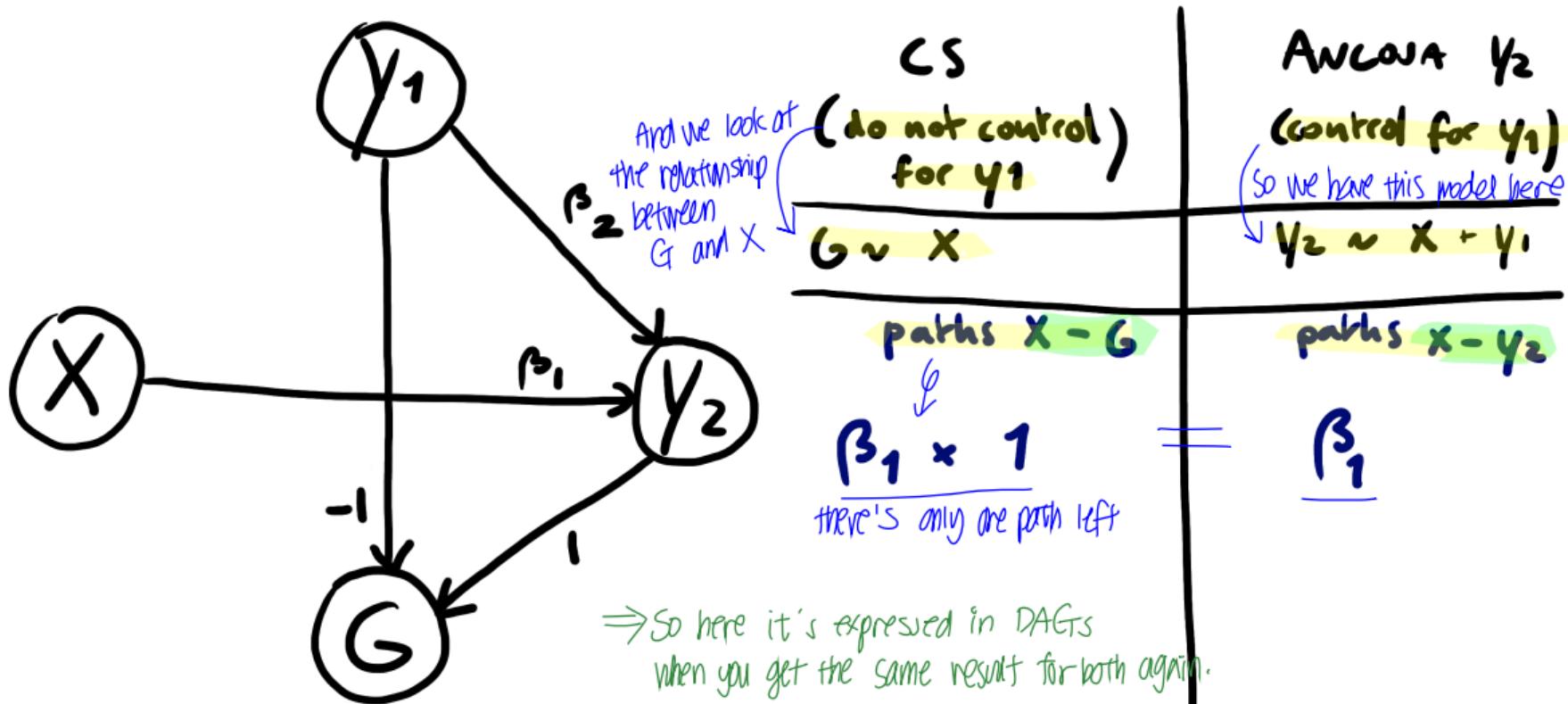
$\beta_2 = 1$

When the effect of Y_1 on Y_2 is equal to 1

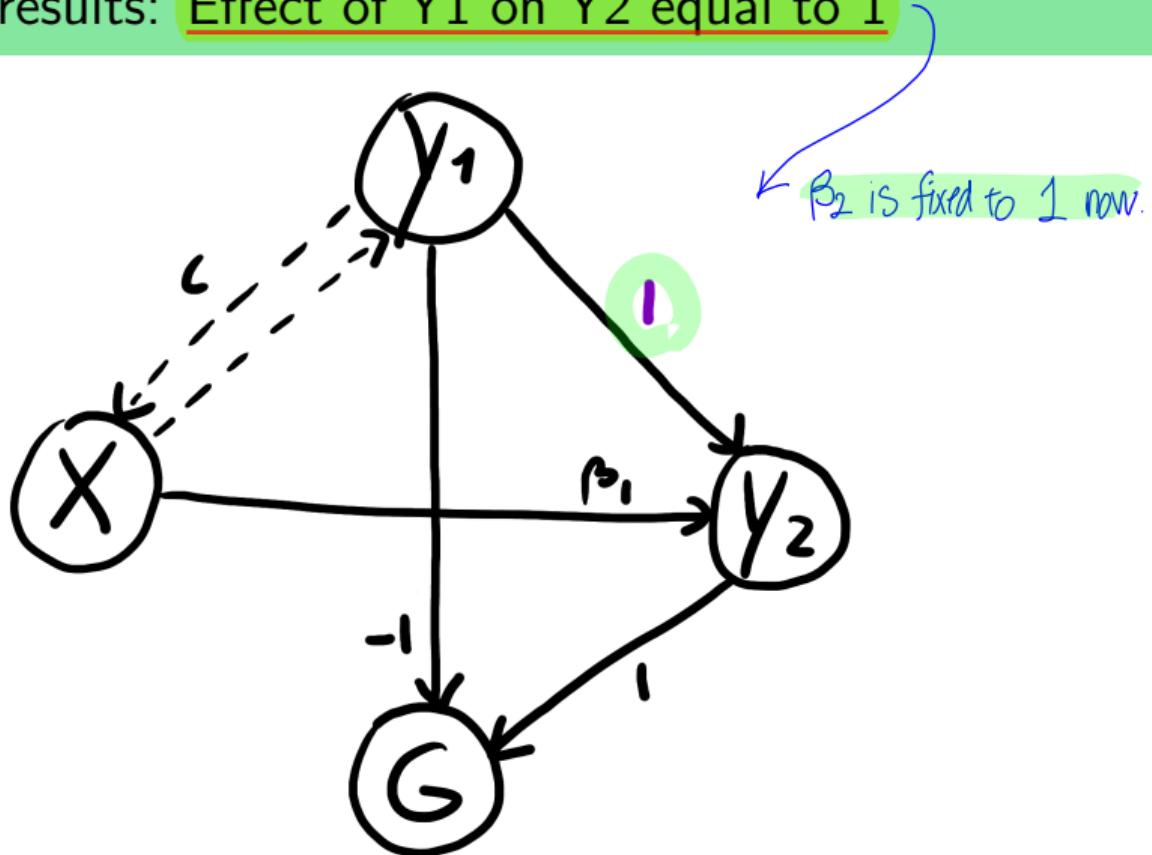
Revisit: When classical Change Score and ANCOVA model have the same results: EVs Y_1 the same in each group



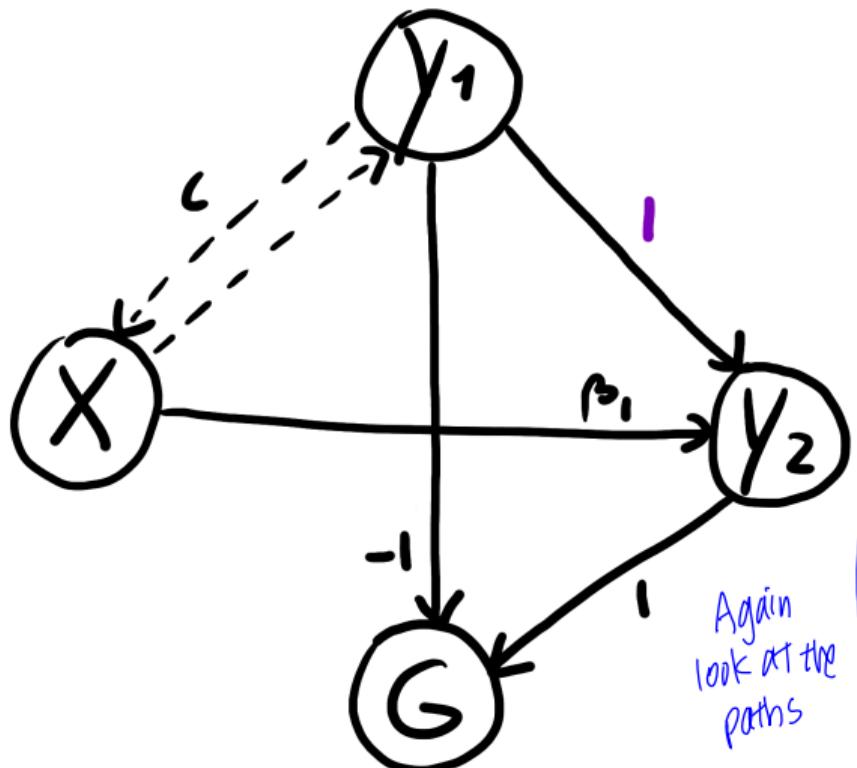
Revisit: When classical Change Score and ANCOVA model have the same results: EVs Y_1 the same in each group



Revisit: When classical Change Score and ANCOVA model have the same results: Effect of Y_1 on Y_2 equal to 1



Revisit: When classical Change Score and ANCOVA model have the same results: Effect of Y_1 on Y_2 equal to 1



CS
(do not control)
for Y_1

$$G \sim X$$

paths $X - G$

$$X \xrightarrow{1} Y_1 \xrightarrow{1} Y_2 \xrightarrow{1} G$$

$$X \xrightarrow{1} Y_1 \xrightarrow{1} G$$

$$X \xrightarrow{\beta_1} Y_2 \xrightarrow{1} G$$

$$1 + (-1) + \beta_1$$

ANCOVA Y_2
(control for Y_1)

$$Y_2 \sim X + Y_1$$

paths $X - Y_2$

$$X \xrightarrow{1} Y_1 \xrightarrow{1} Y_2$$

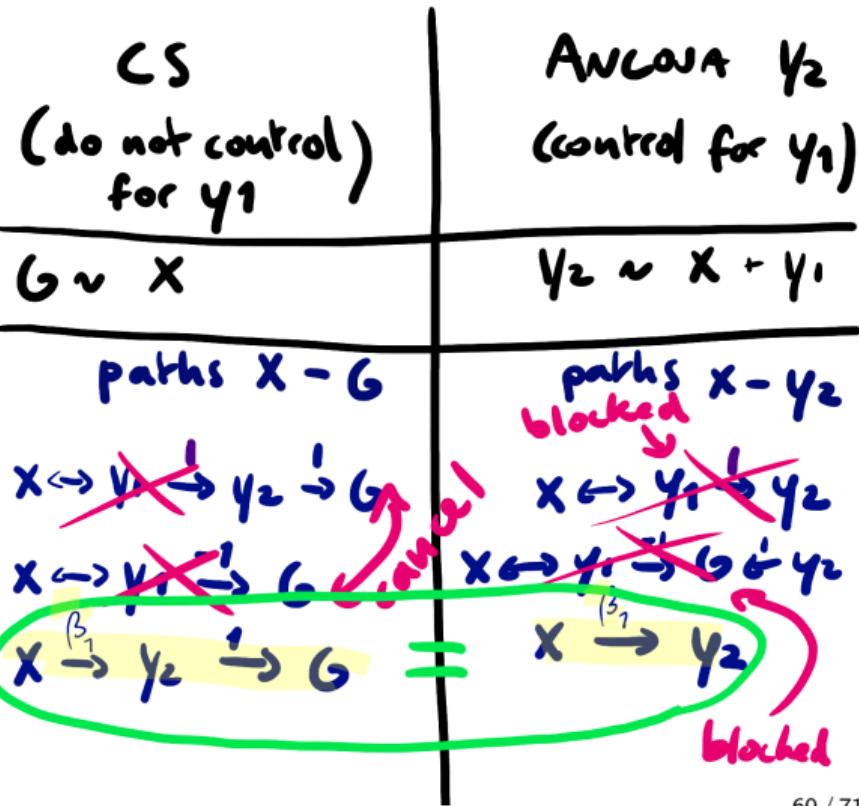
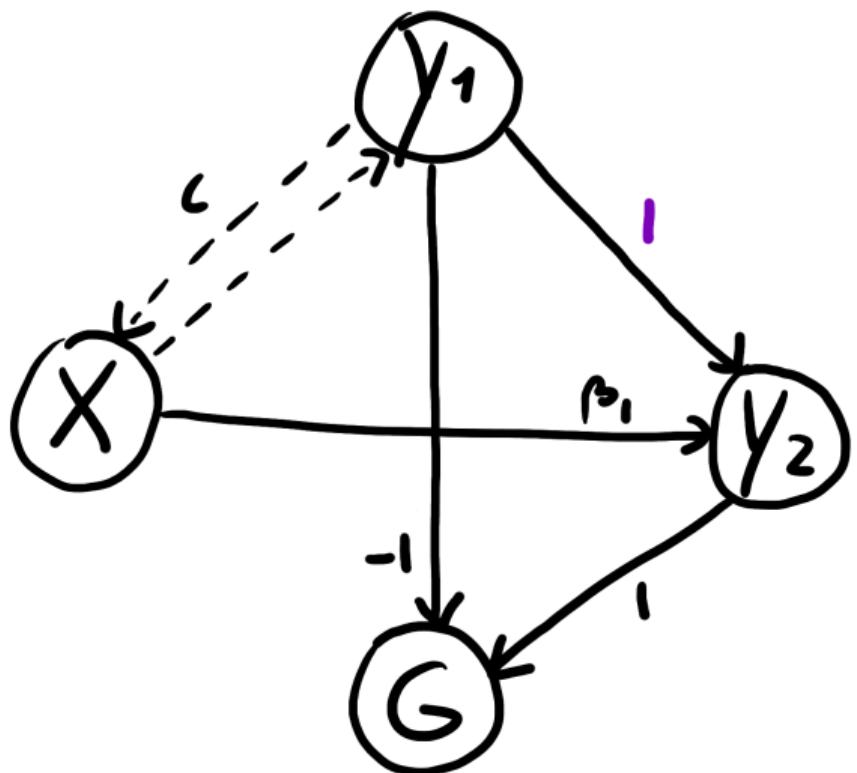
$$X \xrightarrow{1} Y_1 \xrightarrow{1} G \& Y_2$$

$$X \xrightarrow{\beta_1} Y_2$$

blocked :: we're controlling for X

blocked :: G is a collider

Revisit: When classical Change Score and ANCOVA model have the same results: Effect of Y_1 on Y_2 equal to 1



★ Timing is critical

We need to think about the relationship between the pre-treatment variable & the post
↳ Do we need to control for it, YES/NO to get the correct causal effect. This stays the same
whether you use change-score or ANCOVA...

The DAGs show that the **causal relation** between treatment X and pre-test Y1 is critical; it is about whether X or Y1 came first (i.e., their temporal order).

In Rubin's causal framework (Week 2), the timing of treatment, outcome and covariates is also considered critical.

Holland (1986):

- ▶ exposure to a cause (i.e., treatment) occurs at a **specific time point or time interval**
- ▶ variables are thus divided into **pre-exposure** and **post-exposure**
- ▶ "The role of a response variable Y is to measure the effect of the cause, and thus **response variables** must fall into the **post-exposure class**."
(p.946, Holland, 1986)
- ▶ **covariates** should come from the **pre-exposure phase**; then they cannot be affected by the treatment.

If you know the timing of treatment & outcome variable, then you can make decision more easily, whether it is collider / mediator / confounder & accordingly whether you should control for it or not...

Critique of using change scores in DAGs

~ ppl do have opinions on whether you should use change score in the context of causal models...

Journal of Evaluation in Clinical Practice

International Journal of Public Health Policy and Health Services Research



Causal diagrams and change variables

Eyal Shahar MD MPH¹ and Doron J. Shahar²

↓
Should you look at change score at all?
or should you really focus on Y_2 ?

Abstract

Background The true change in the value of a variable between two time points is often assumed to be a cause or an effect of interest. To our knowledge, this assumption is based on intuition, rather than on any formal theoretical justification.

Methods We used causal directed acyclic graphs to explore the causal properties of a change variable, and critically examined competing structures.

Results Based on the proposed causal structure, a change variable (true change) is no more than a derived variable. It does not cause anything and is not of causal interest.

Conclusions A true change is not a variable in the physical world. Therefore, modelling the change between two time points is justified only in a few situations.

so ↓ The idea is that, there cannot really be arrows pointing outwards of G.

Would it be possible for G to ever be a causal variable in that sense,

or can it only always be an outcome variable somewhere at the end...

& they're saying G is not so relevant, & not really interesting

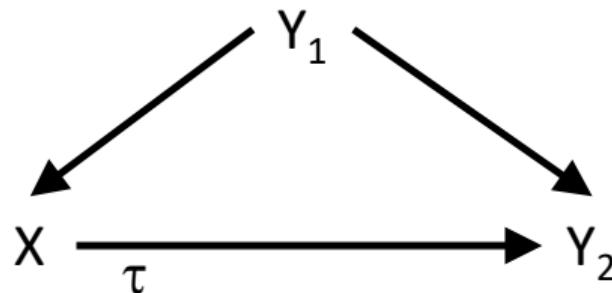
Unmeasured confounding

in the pre-post test design

When there's a specific kind of unmeasured confounding,
looking at Change score can be very useful!! 😊

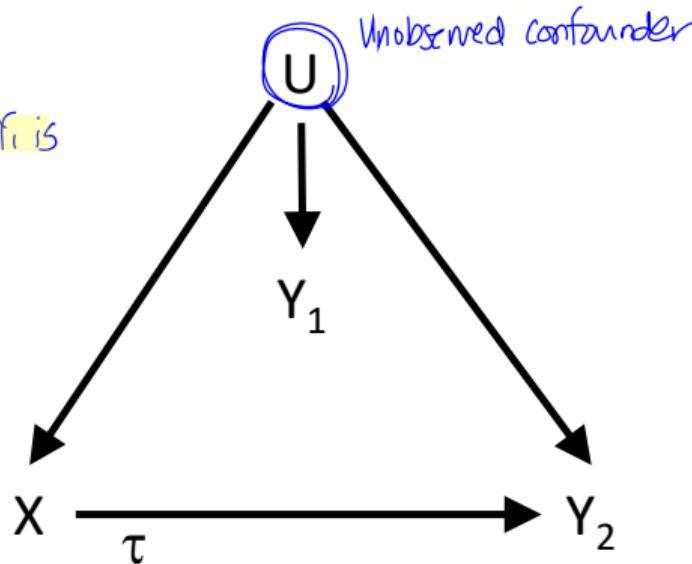
Pretest as a proxy for confounder

When Y_1 was **measured prior to treatment**, it could be a **confounder**; you need to control for it then (e.g., use ANCOVA model).

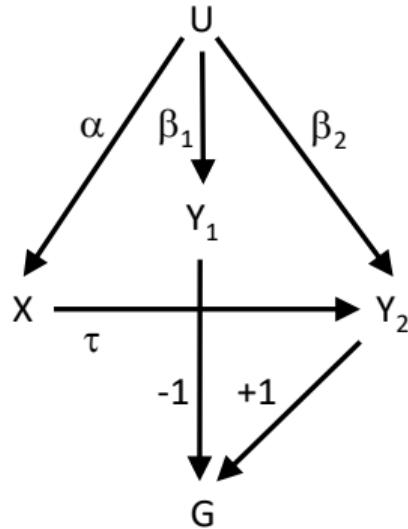


But Y_1 may also be a **proxy of an unobserved confounder**; controlling for Y_1 will **only partly remove bias** due to $X \leftarrow U \rightarrow Y_2$.

depending on
how strongly Y_1 is
related to U .



How can classical change score analysis help?



The interest is in $X \rightarrow G$; this is equal to $X \rightarrow Y_2$

Other paths between X and G:

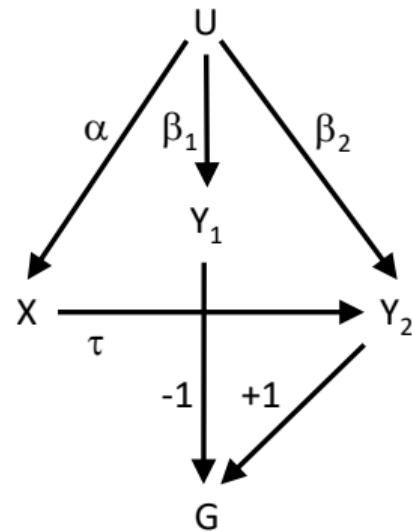
- ▶ $X \leftarrow U \rightarrow Y_1 \rightarrow G$
- ▶ $X \leftarrow U \rightarrow Y_2 \rightarrow G$
- ▶ $X \rightarrow Y_2 \leftarrow U \rightarrow Y_1 \rightarrow G$



When we have **linear relations** (and the variance of U is equal to 1), we get:

- ▶ First path: $-\alpha\beta_1$
- ▶ Second path: $\alpha\beta_2$
- ▶ Third path: 0 (contains the **collider** Y_2)

How can classical change score analysis help?



The interest is the causal path $X \rightarrow Y_2 \rightarrow G$. This is the causal effect from $X \rightarrow G$ ($\tau \times 1$) but also the causal effect of X on Y_2 (just τ)

The interest is in $X \rightarrow G$; this is equal to $X \rightarrow Y_2$

Other paths between X and G :

- ▶ $X \leftarrow U \rightarrow Y_1 \rightarrow G$
- ▶ $X \leftarrow U \rightarrow Y_2 \rightarrow G$
- ▶ $X \rightarrow Y_2 \leftarrow U \rightarrow Y_1 \rightarrow G$

When we have **linear relations** (and the variance of U is equal to 1), we get:

- ▶ First path: $-\alpha\beta_1$
- ▶ Second path: $\alpha\beta_2$
- ▶ Third path: 0 (contains the **collider** Y_2)

So in that case, we'd do a better job than just controlling for Y_1 , as we could completely remove the confounding effect of U by using change-score approach.

If $\beta_1 = \beta_2$, the first and second path **cancel each other out** (cf. Kim & Steiner, 2019)!

→ So in this case, using Change Score analysis might actually be a good idea.

Important to realize (i.e., conclusion so far)

We have written the change score model as a special case of the ANCOVA model.

This may suggest we should just test which model fits better.

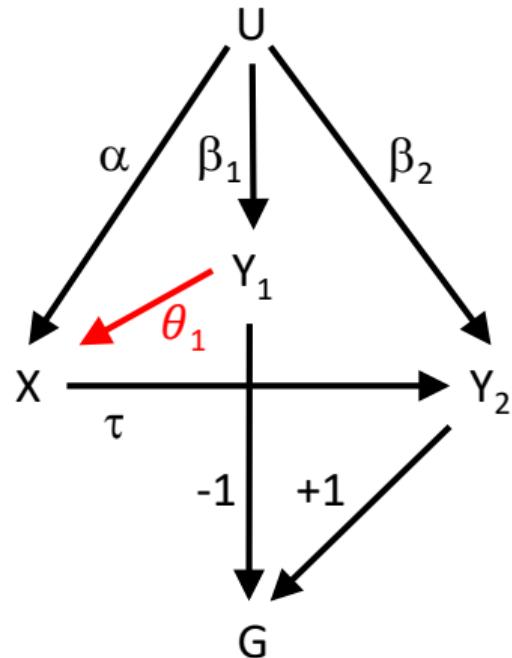
However, the point is **NOT** that we want to determine which of these two models generated the data!

The goal is to "estimate the treatment effect without bias".
true causal effect

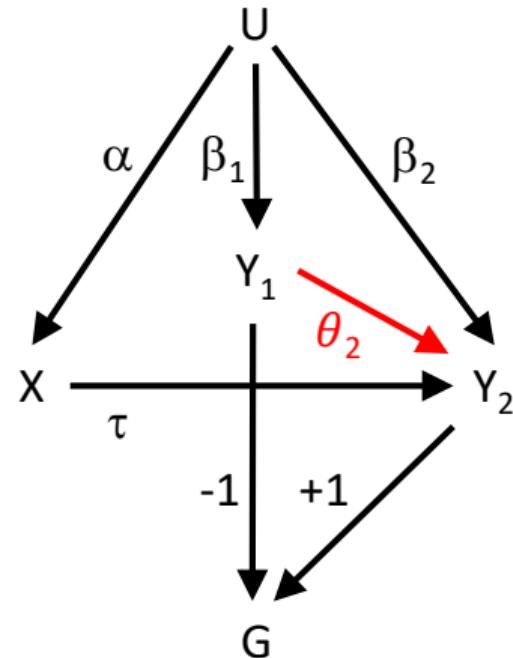
The **change score model** is:

- typically NOT considered a reasonable model as a data generating mechanism
- but a very useful model for estimating the causal effect (under specific circumstances) $\sim \text{ex } \beta_1 = \beta_2$

What if... we have slightly diff. situations, then it no longer works!!

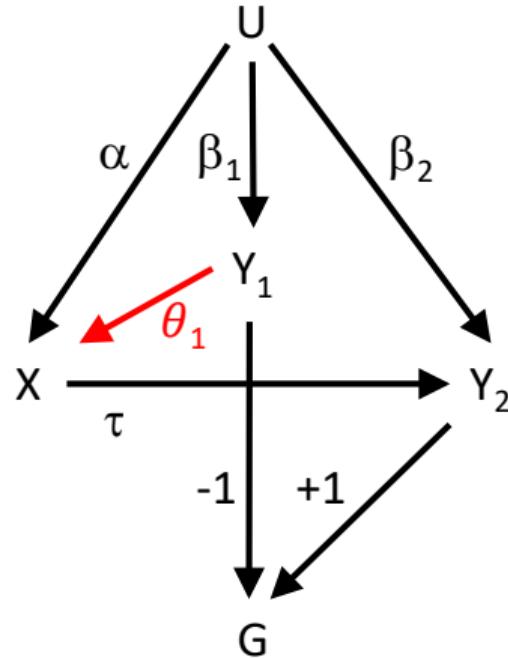


Pretest affects treatment

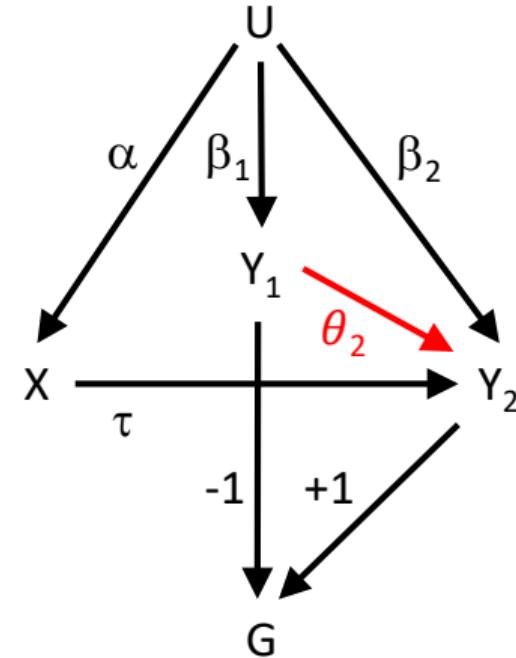


Pretest affects outcome

What if...



Pretest affects treatment

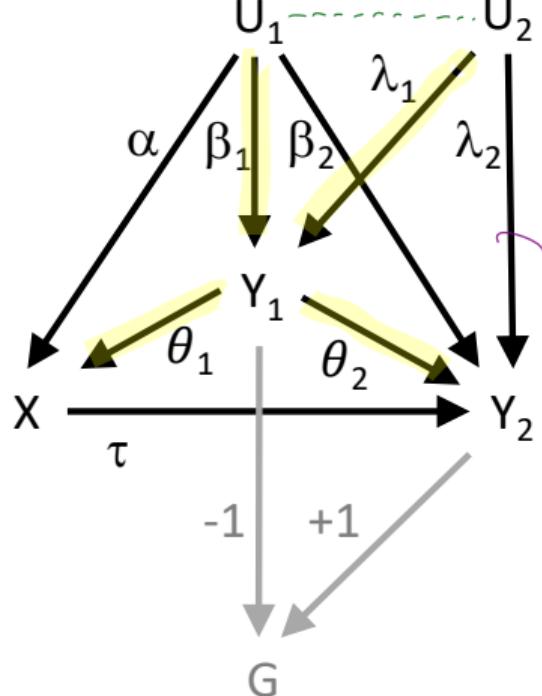


Pretest affects outcome

In these scenarios neither the Change score model nor the ANCOVA model give an unbiased estimate of the causal effect of X on Y_2 .

Also important to realize (wrt timing)

When pretest is from the **pretreatment phase**, it does **NOT** mean it can **only** be a confounder. Becuz there might be all kinds of unobserved variables that we didn't take into account.



↓
We can have a variable that can be **both confounder & collider**, or **both mediator & confounder**... specially when you have multiple variables that are affecting each other over time. Then, you have a problem...

The **pretest Y_1** is:

- ▶ a **confounder**: $X \leftarrow Y_1 \rightarrow Y_2$
- ▶ a **collider**: $X \leftarrow U_1 \rightarrow Y_1 \leftarrow U_2 \rightarrow Y_2$

Var. can be **both confounder & collider**...!!

So if we control for Y_1 , we open a backdoor path by introducing a **spurious relationship** between U_1 & U_2 and introduce bias!

Summary

Use

> **ANCOVA** (regress Y_2 or $G=Y_2-Y_1$ on X and Y_1):

- ▶ when Y_1 is confounder of X and Y_2
- ▶ or to get direct effect when Y_1 is mediator

Use

> **Marginal model** (regress Y_2 on X):

- ▶ when Y_1 is mediator and interest is in total effect of X on Y_2

Use

> **Change score model** (regress $G=Y_2-Y_1$ on X):

- ▶ when there is time-invariant unobserved confounding with stable effect : when we have this very specific kind of unobserved confounding
- ▶ (or when Y_1 is mediator and the interest is in total effect of X on Y_2-Y_1)

Summary

ANCOVA (regress Y_2 or $G=Y_2-Y_1$ on X and Y_1):

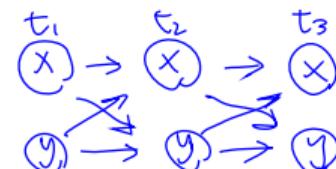
- ▶ when Y_1 is confounder of X and Y_2
- ▶ or to get direct effect when Y_1 is mediator

Marginal model (regress Y_2 on X):

- ▶ when Y_1 is mediator and interest is in total effect of X on Y_2

Change score model (regress $G=Y_2-Y_1$ on X):

- ▶ when there is time-invariant unobserved confounding with stable effect
- ▶ (or when Y_1 is mediator and the interest is in total effect of X on Y_2-Y_1)



There is a lot more to say and study about causality and time

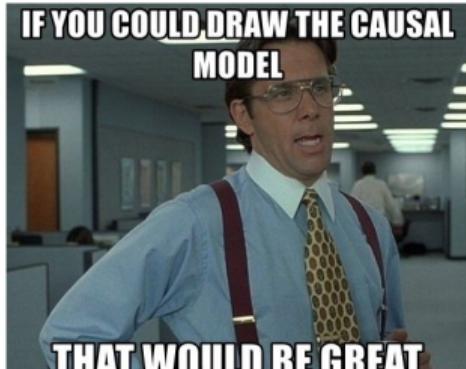
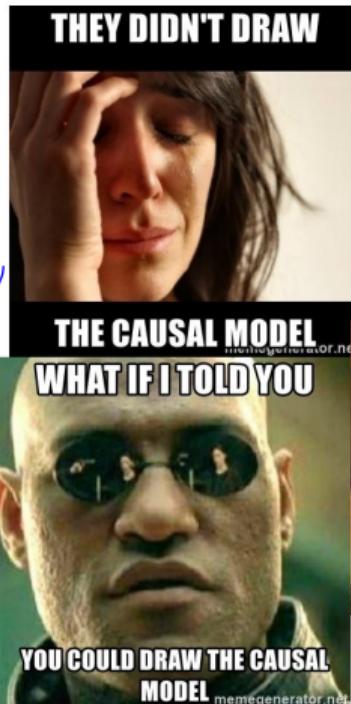
- ▶ Consider time-varying treatments, outcomes, and covariates
- ▶ Variables affecting themselves and each other continuously through time, effects may change over time, ...
- ▶ In (these) more complicated scenarios we may have variables that are simultaneously confounders/mediators/colliders.

Ex)

In any case...

Do causal inference in a principled way!

- ▶ Be explicit and clear about your causal interests/questions
- ▶ Specify your ideas (causal theory) in some causal graph *draw the DAG! 😊* and/or in equations (and SCM)..
- ▶ make assumptions explicit
- ▶ Choose a causal analysis best tailored to your particular problem.
- ▶ Replicate, triangulate, critique, etc!



In any case...

Finally, remember these science key three...

- ▶ Measurement
- ▶ Theory formation
- ▶ Causal Inference

