Introduction to causal inference

Session 11

MATH 80667A: Experimental Design and Statistical Methods
HEC Montréal

Outline

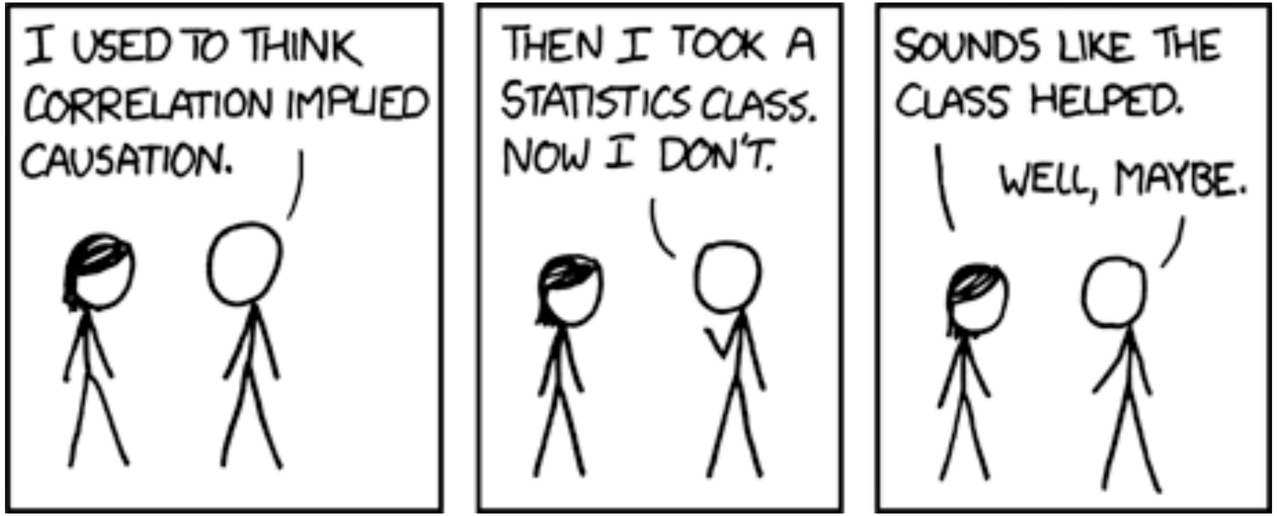
Basics of causal inference

Directed acyclic graphs

Causal mediation

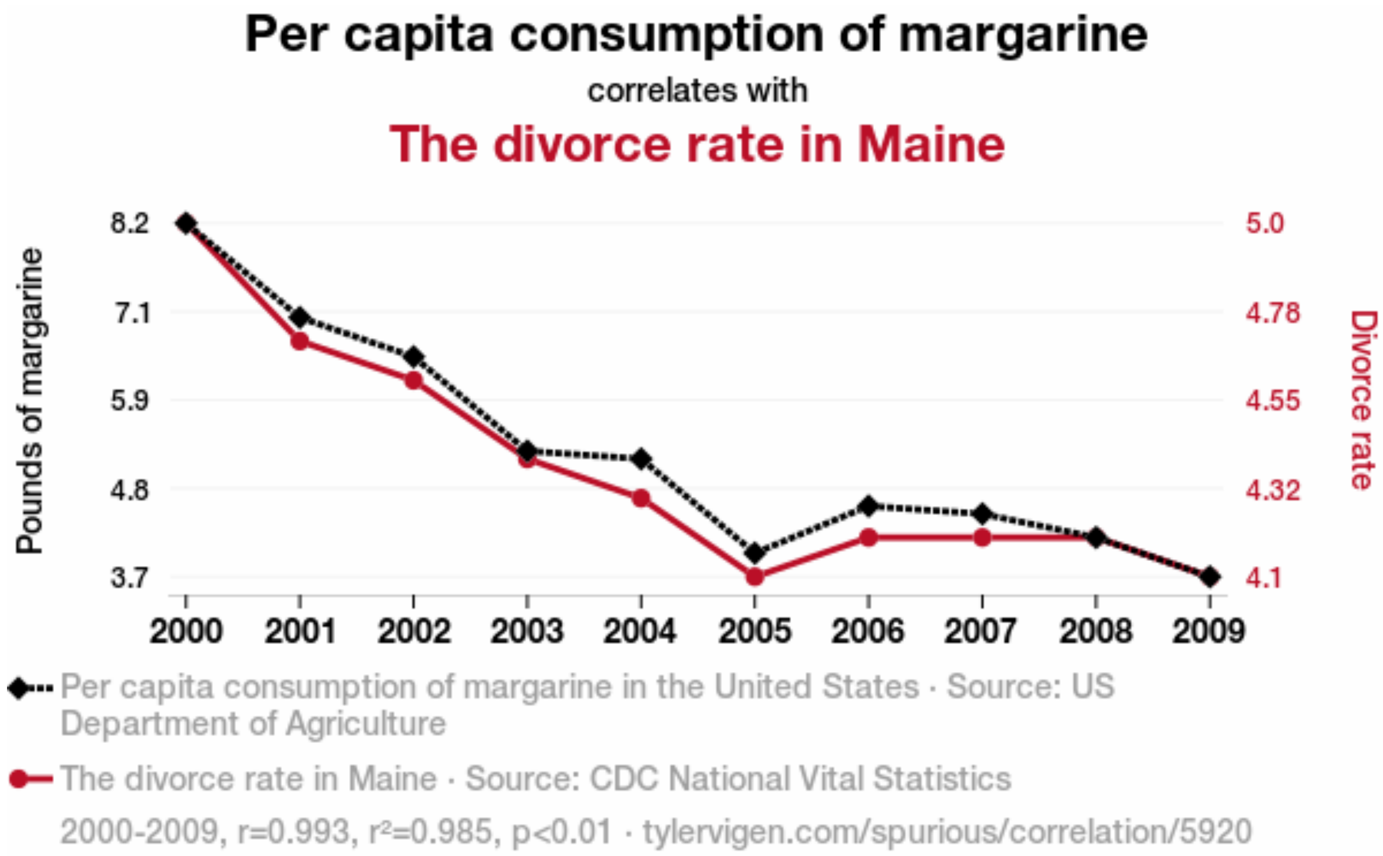
Causal inference

Correlation is not causation



xkcd comic 552 by Randall Munroe, CC BY-NC 2.5 license. Alt text: Correlation doesn't imply causation, but it does waggle its eyebrows suggestively and gesture furtively while mouthing 'look over there'.

Spurious correlation



Correlation vs causation

$$\overbrace{\underbrace{\mathbb{E}(y \mid \mathrm{do}(x))}_{\text{Causation}}}^{\text{The average population-level change in } y \text{ when } \textit{experimentally doing } x} \neq \overbrace{\underbrace{\mathbb{E}(y \mid x)}_{\text{Correlation}}}^{\text{The average population-level change in } y \text{ when accounting for } \textit{observed } x}$$

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Potential outcomes

For individual i, we postulate the existence of a potential outcomes

- $Y_i(1)$ (response for treatment $X = 1$) and
- $Y_i(0)$ (response for control $X = 0$).

Both are possible, but only one will be realized.

Observe outcome for a single treatment

- Result $Y(X)$ of your test given that you either party $(X = 1)$ or study $(X = 0)$ the night before your exam.

Fundamental problem of causal inference

With binary treatment X_i, I observe either $Y_i \mid \mathrm{do}(X_i = 1)$ or $Y_i \mid \mathrm{do}(X_i = 0)$.

i	X_i	$Y_i(0)$	$Y_i(1)$	$Y_i(1) - Y_i(0)$
1	1	?	4	?
2	0	3	?	?
3	1	?	6	?
4	0	1	?	?
5	0	5	?	?
6	1	?	7	?

Causal assumptions?

Since we can't estimate individual treatment, we consider the **average** treatment effect (average over population) $\mathsf{E}\{Y(1) - Y(0)\}$.

The latter can be estimated as

$$\mathsf{ATE} = \underbrace{\mathsf{E}(Y \mid X = 1)}_{\substack{\text{expected response among} \\ \text{treatment group}}} - \underbrace{\mathsf{E}(Y \mid X = 0)}_{\substack{\text{expected response among} \\ \text{control group}}}$$

When is this a valid causal effect?

(Untestable) assumptions

For the ATE to be equivalent to $\mathsf{E}\{Y(1) - Y(0)\}$, the following are sufficient:

1. *ignorability*, which states that potential outcomes are independent of assignment to treatment
2. lack of interference: the outcome of any participant is unaffected by the treatment assignment of other participants.
3. consistency: given a treatment X taking level j, the observed value for the response $Y \mid X = j$ is equal to the corresponding potential outcome $Y(j)$.

Directed acyclic graphs

Types of data

Experimental

You have control over which units get treatment

Observational

You don't have control over which units get treatment

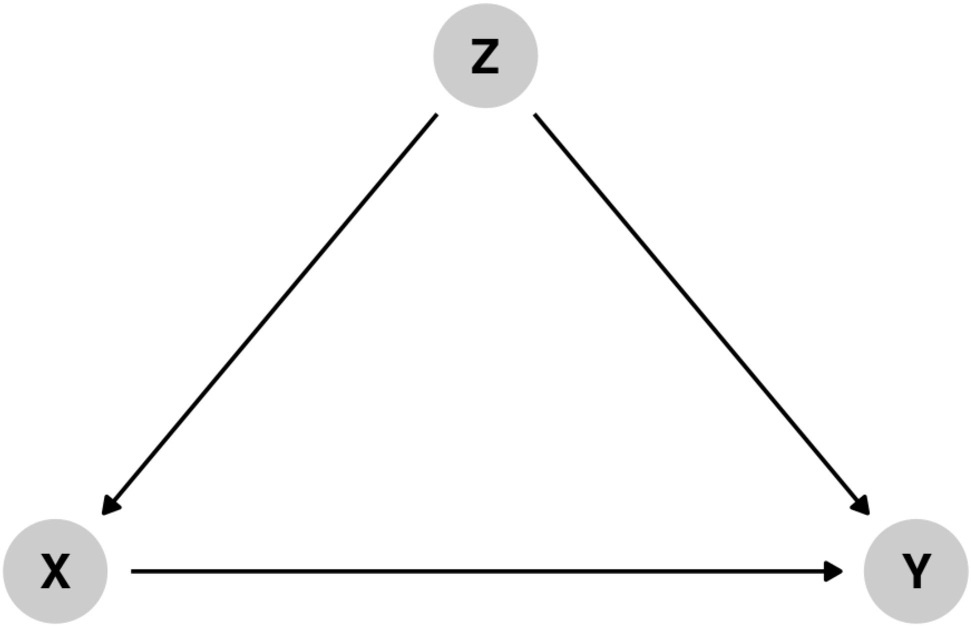
Causal diagrams

Directed acyclic graphs (DAGs)

Directed: Each node has an arrow that points to another node

Acyclic: You can't cycle back to a node (and arrows only have one direction)

Graph: A set of nodes (variables) and vertices (arrows indicating interdependence)

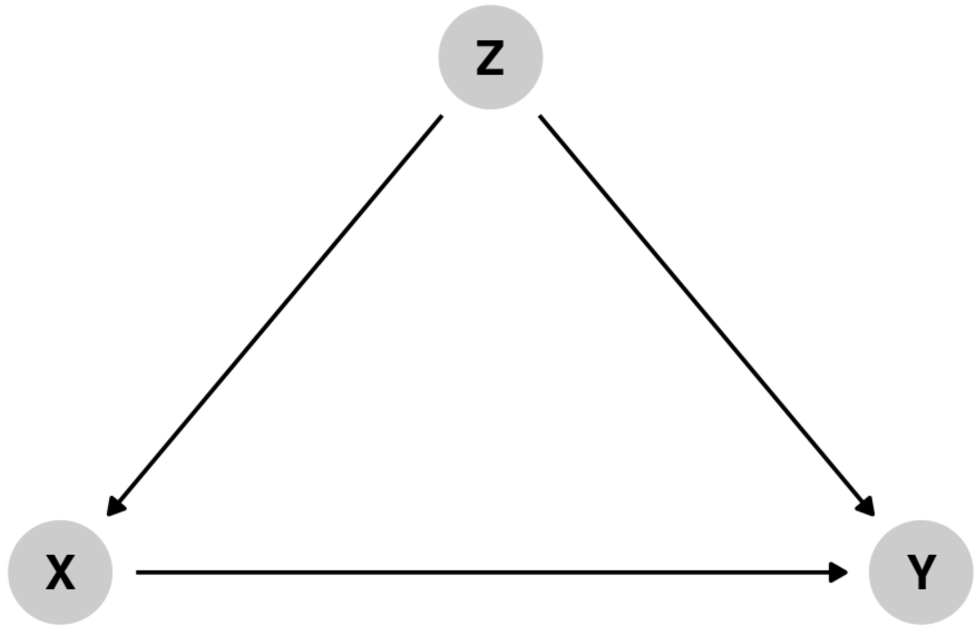


Causal diagrams

Directed acyclic graphs (DAGs)

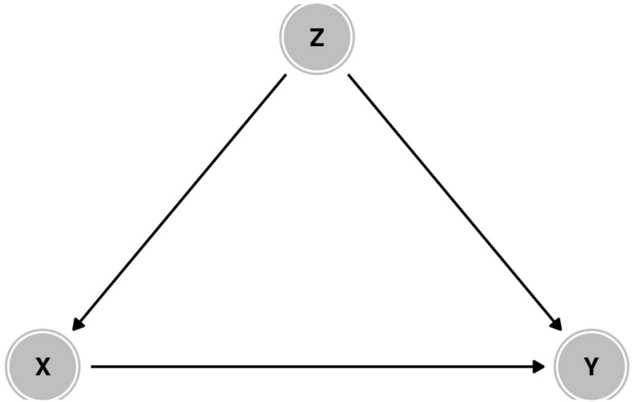
Graphical model of the process that generates the data

Maps your logical model



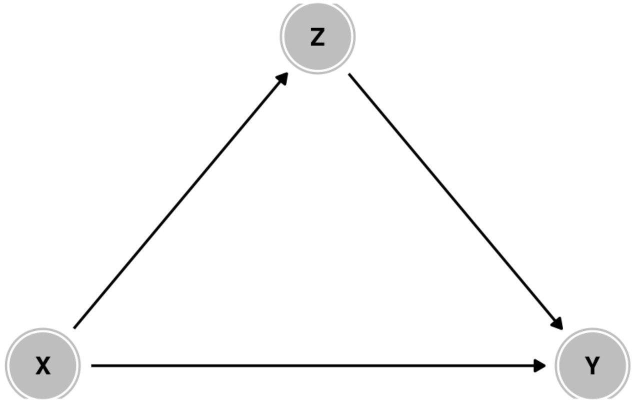
Three types of associations

Confounding



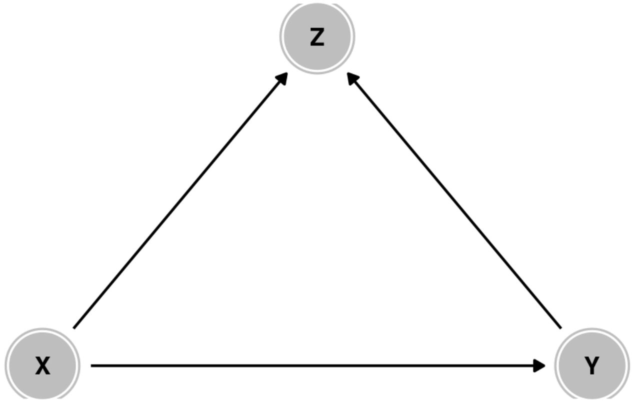
Common cause

Causation



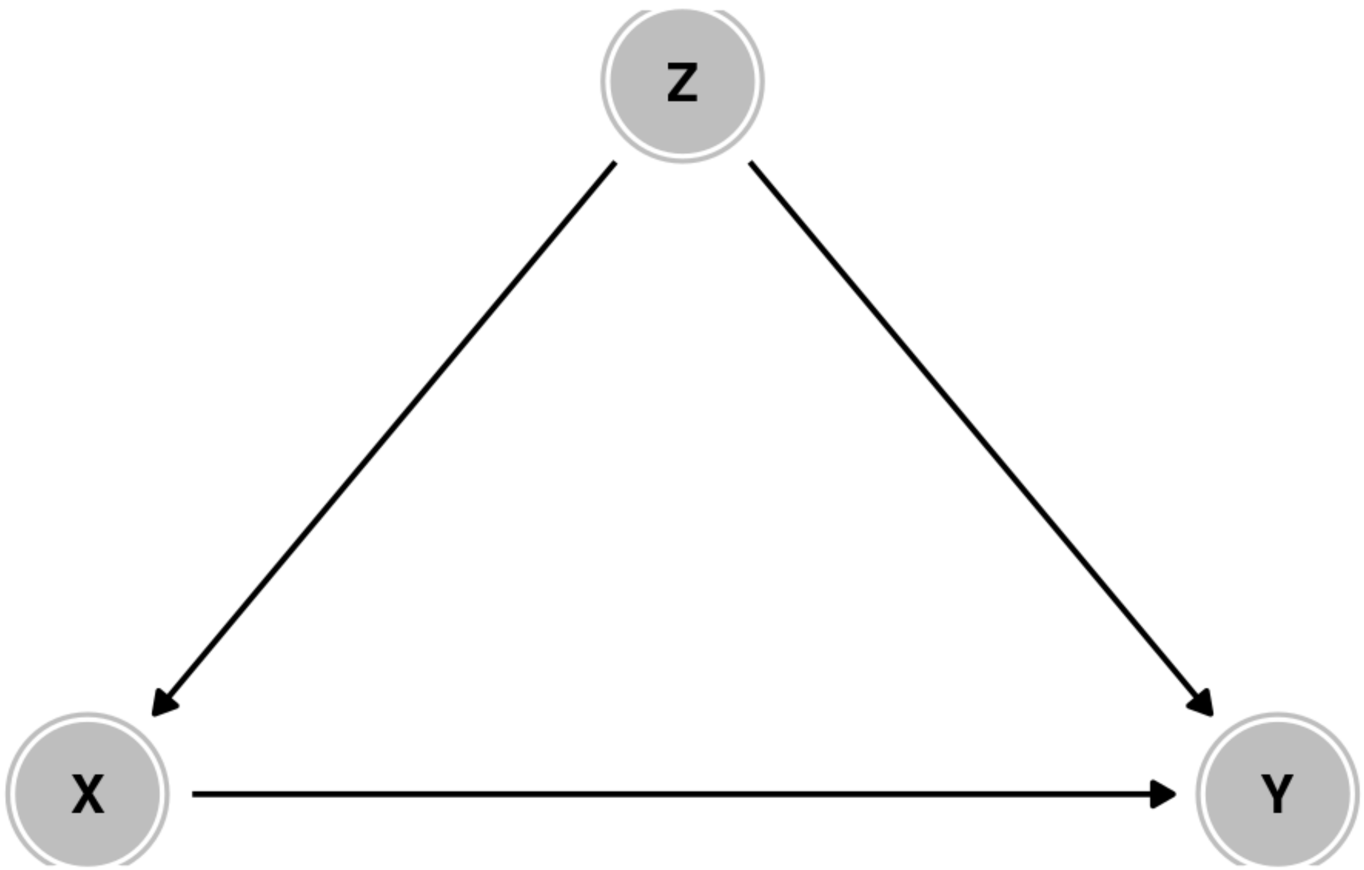
Mediation

Collision



Selection / endogeneity

Confounding



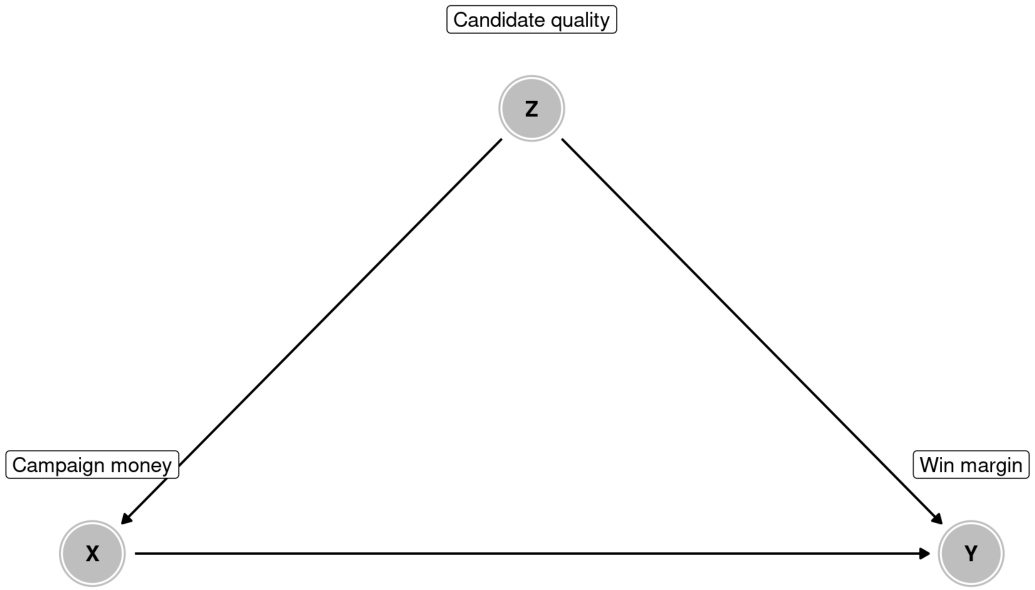
X causes Y

But Z causes both X and Y

Z *confounds* the X → Y association

Confounder: effect of money on elections

What are the paths between money and win margin?



Money → Margin

Money ← Quality → Margin

Quality is a *confounder*

Experimental data

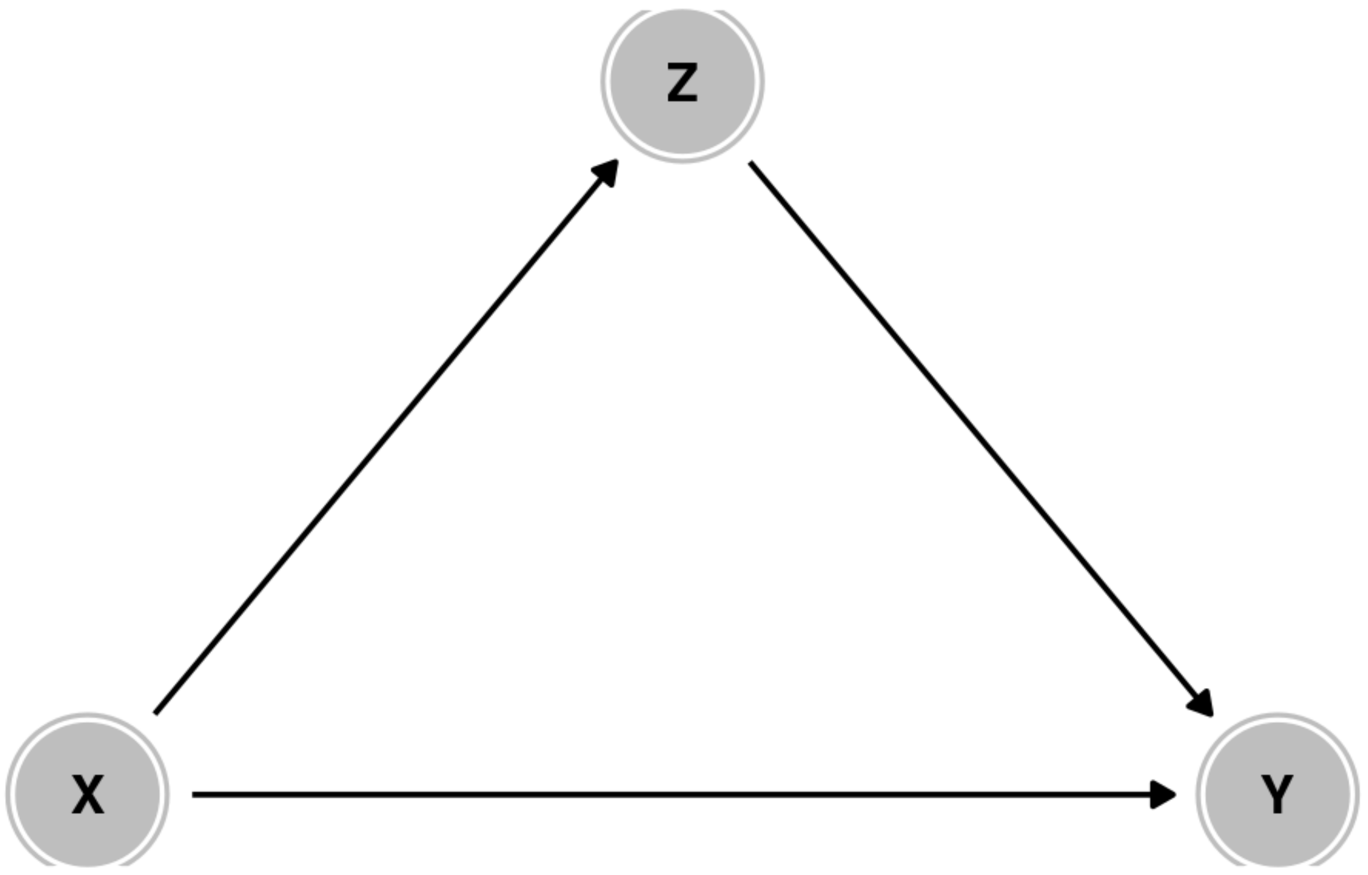
Since we randomize assignment to treatment X, all arrows **incoming** in X are removed.

With observational data, we need to explicitly model the relationship and strip out the effect of X on Y.

How to adjust with observational data

- Include covariate in regression
- Matching: pair observations that are more alike in each group, and compute difference between these
- Stratification: estimate effects separately for subpopulation (e.g., young and old, if age is a confounder)
- Inverse probability weighting: estimate probability of self-selection in treatment group, and reweight outcome.

Causation

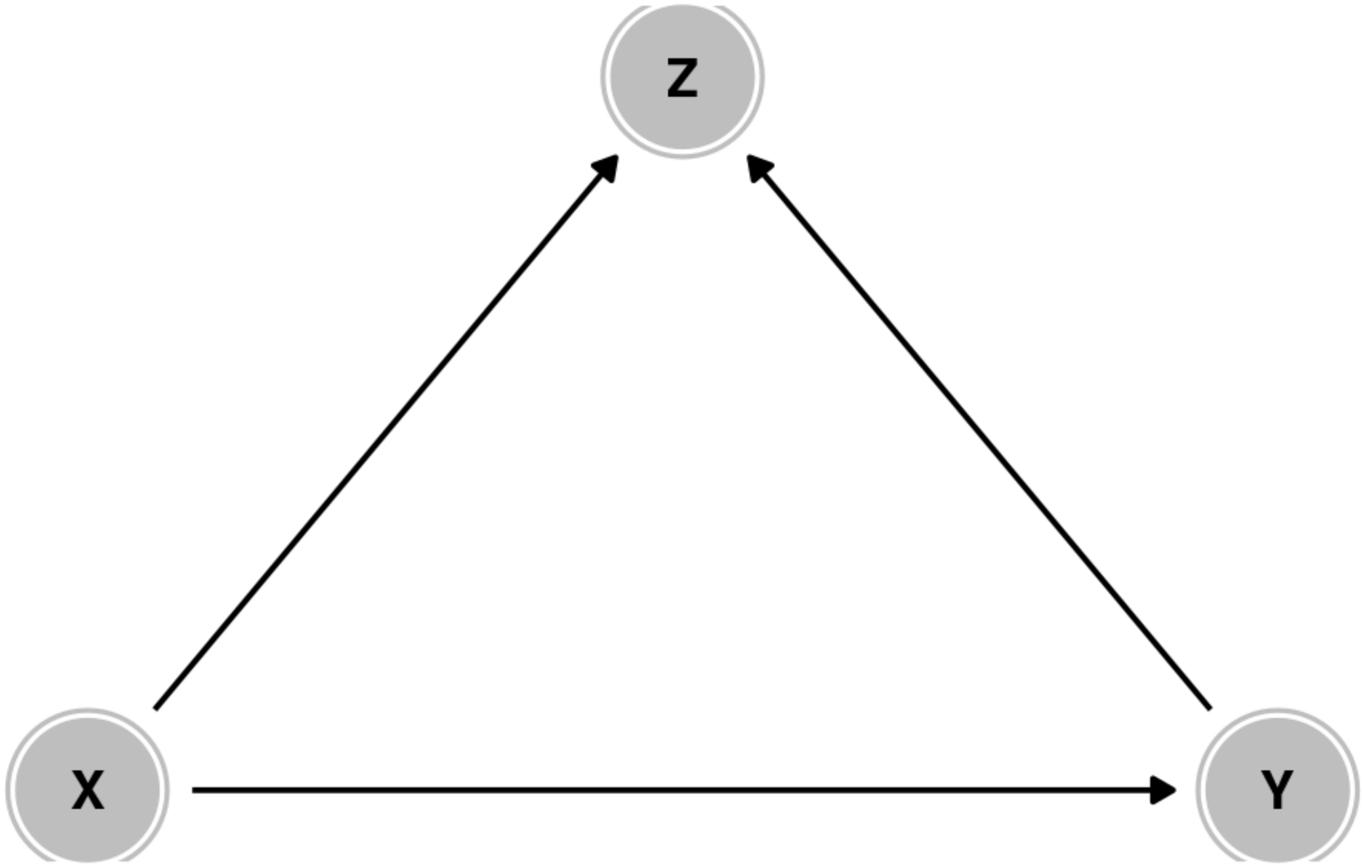


X causes Y

X causes
Z which causes Y

Z is a mediator

Colliders



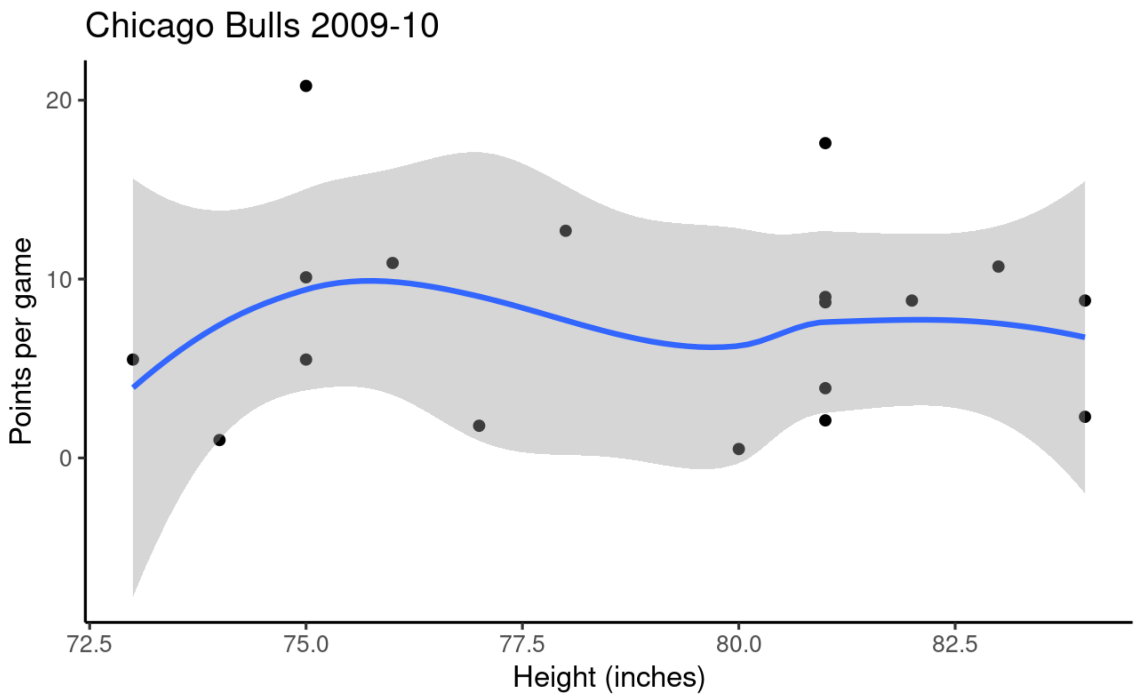
X causes Z

Y causes Z

Should you control for Z?

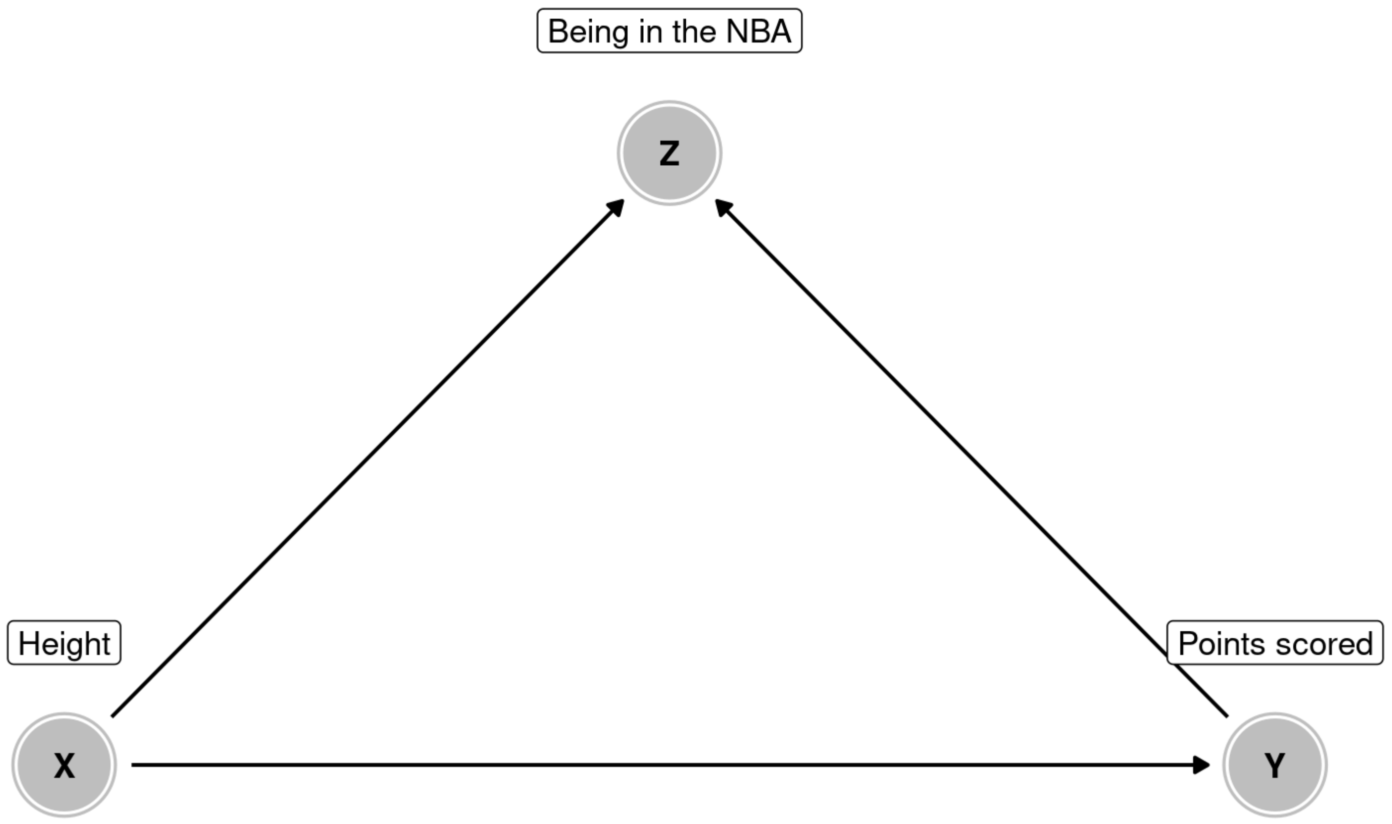
Colliders can create fake causal effects

Colliders can hide real causal effects



Height is unrelated to basketball skill… among NBA players

Colliders and selection bias

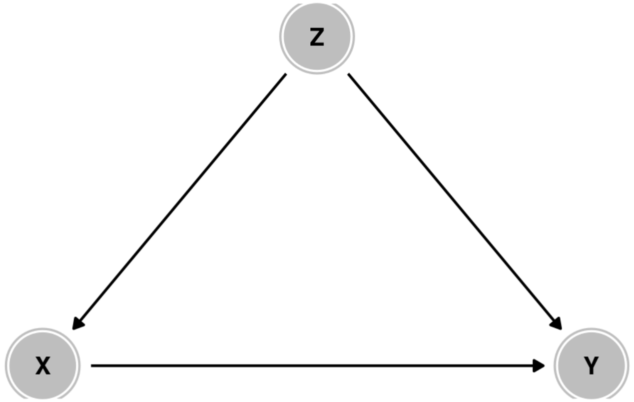


Conditioning on colliders

- Omnipresent in the literature
- Example: When and how does the number of children affect marital satisfaction? An international survey
- Example: The Predictive Validity of the GRE Across Graduate Outcomes

Three types of associations

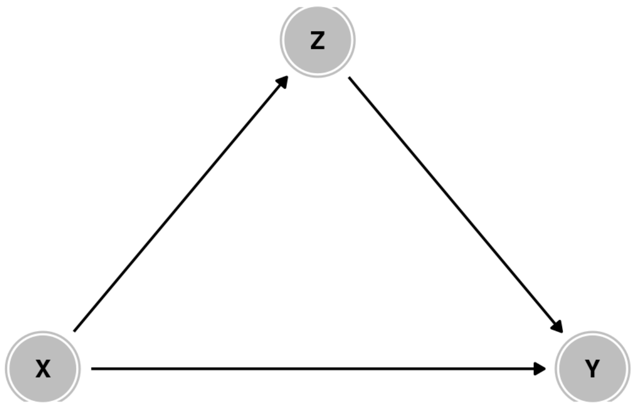
Confounding



Common cause

Causal forks **X** ← **Z** → **Y**

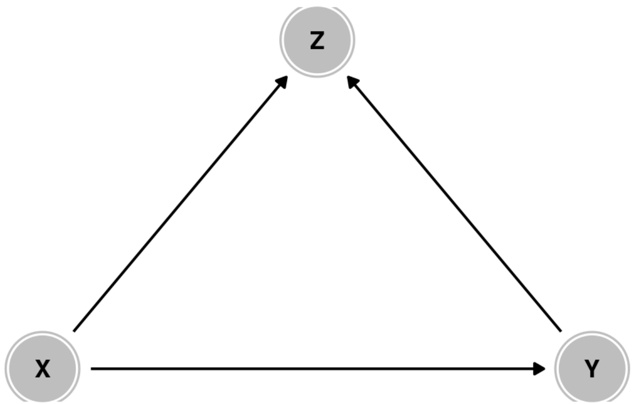
Causation



Mediation

Causal chain **X** → **Z** → **Y**

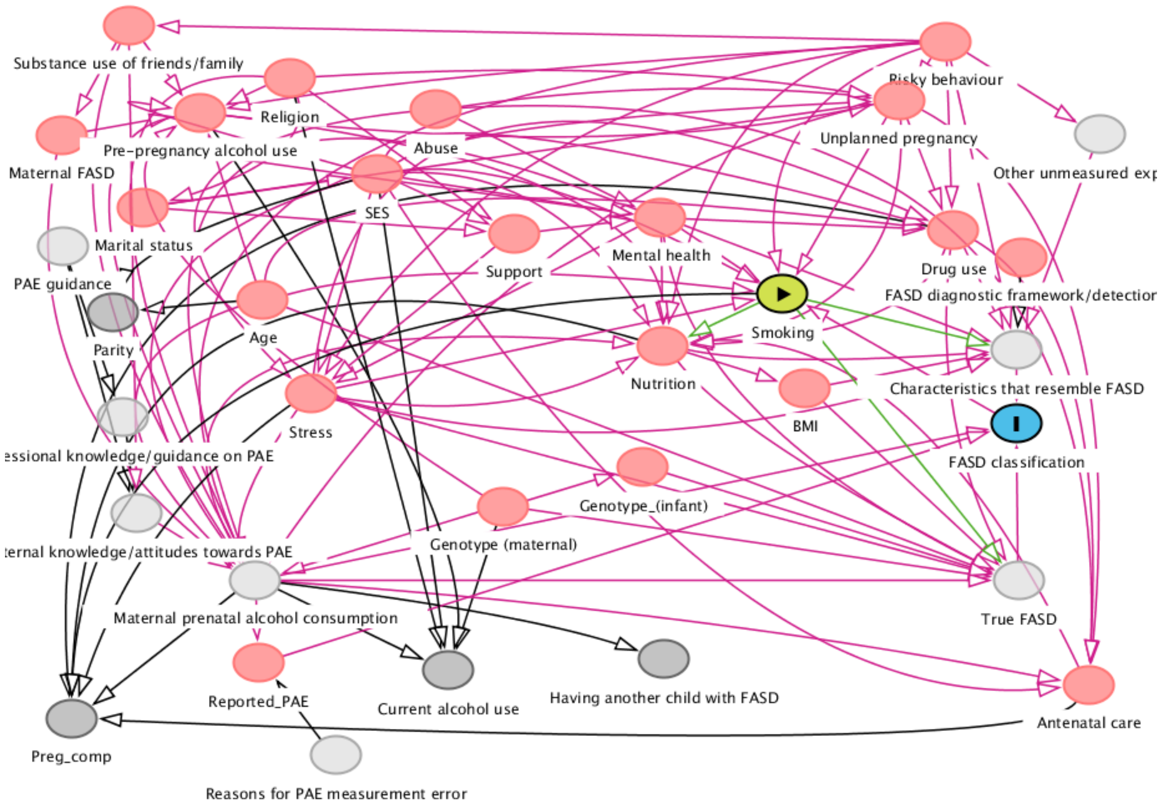
Collision



Selection / endogeneity

inverted fork **X** → **Z** ← **Y**

Life is inherently complex



Postulated DAG for the effect of smoking on fetal alcohol spectrum disorders (FASD)