Week 9: Frameworks for Causal Inference

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Princeton

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[1]These slides are heavily influenced by Matt Blackwell, Justin Grimmer, Jens Hainmueller, Erin Hartman and Kosuke Imai

Where We've Been and Where We're Going...

- Last Week
  - regression diagnostics
- This Week
  - core ideas in causal inference
  - potential outcomes
  - nonparametric structural equation models and DAGs
  - experiments
- Next Week
  - selection on observables
- Long Run
  - probability $\rightarrow$ inference $\rightarrow$ regression $\rightarrow$ causal inference

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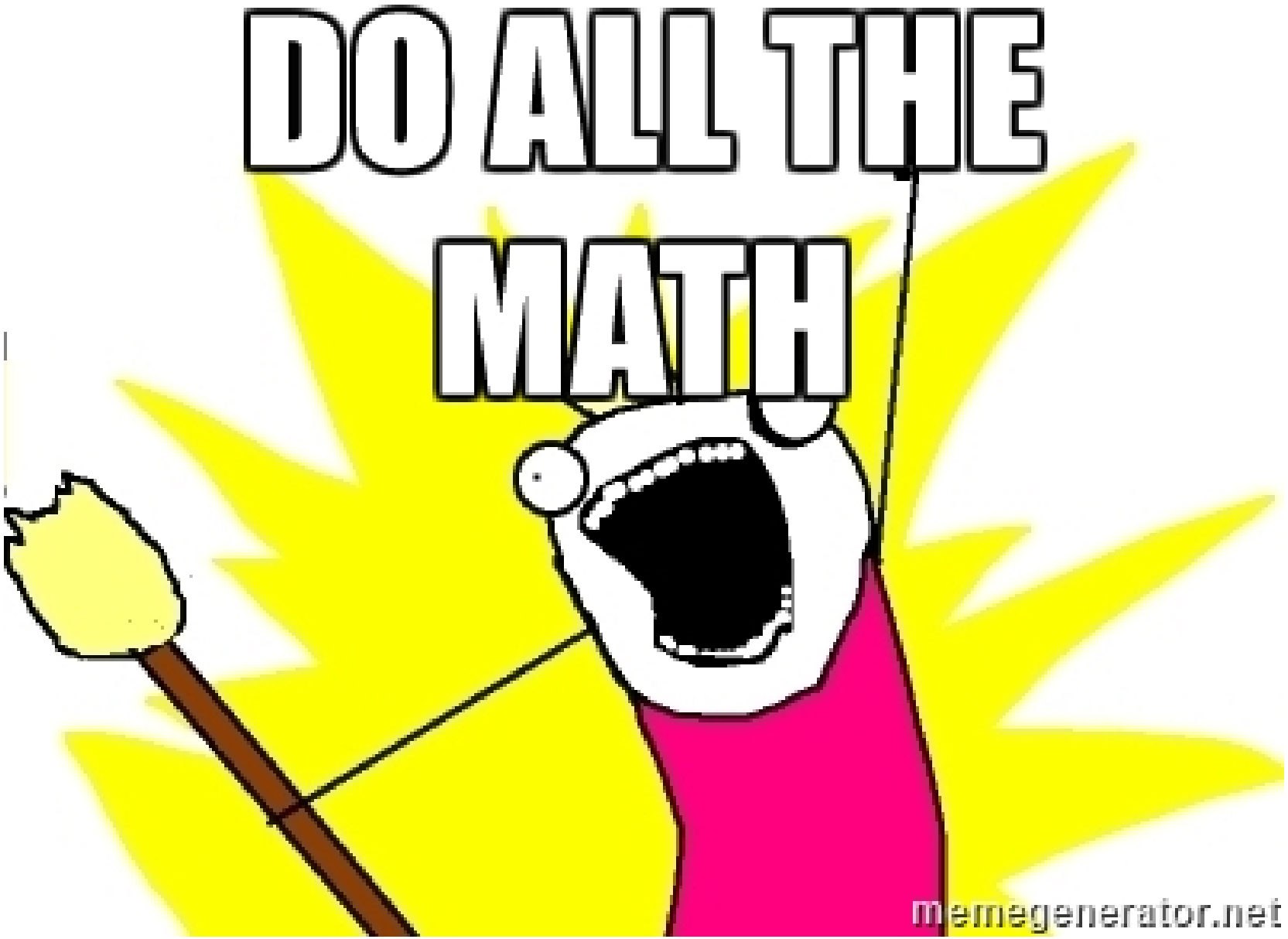
- Reason 2: When you understand how the methods work you can make choices based on substantive knowledge rather than what software spits out at you.


DO ALL THE
MATH
memegenerator.net

Regression as a Tool: A Review

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- This is because they are about different groups of units not about the same unit under intervention.

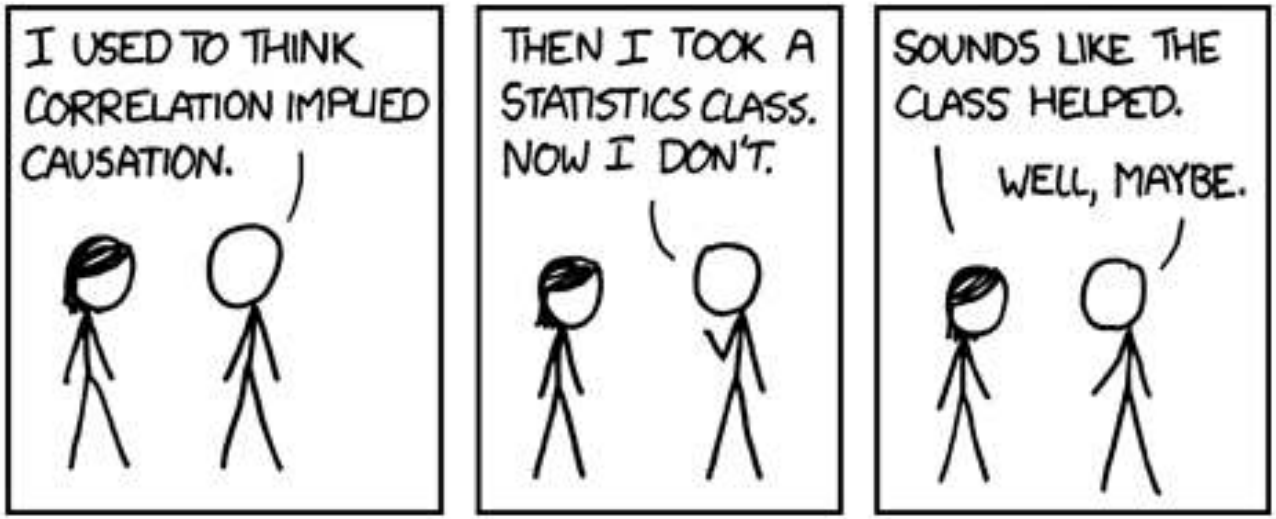
1. Core Ideas in Causal Inference

2. Potential Outcomes
   - Framework
   - Estimands
   - Three Big Assumptions
   - What Gets to Be a Cause

3. Nonparametric Structural Equation Models and Causal Directed Acyclic Graphs

4. The Experimental Ideal

Causation


I USED TO THINK CORRELATION IMPLIED CAUSATION.
THEN I TOOK A STATISTICS CLASS. NOW I DON'T.
SOUNDS LIKE THE CLASS HELPED.
WELL, MAYBE.

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- By convention we often call the counterfactual levels we care about treated and control and we start with binary treatment variables (continuous things get much more complicated!).

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We will read Lundberg, Johnson, and Stewart 2021 where theoretical and empirical estimand terminology comes from.

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- Identification depends on assumptions not statistical models.
- As we will see this is not a conversation about estimation: in other words, if someone answers "regression" they have made a category error

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The role of assumptions:

- Often identification requires (hopefully minimal) assumptions
- Even when identification is possible, estimation may impose additional assumptions (i.e. that the linear approximation to the CEF is good enough)
- Law of Decreasing Credibility (Manski): The credibility of inference decreases with the strength of the assumptions maintained

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   - Framework
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The Potential Outcomes Framework

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- It is a way of thinking about counterfactuals and the assumptions required to make statements about them.
- We will first step through the framework, then discuss estimands, three big assumptions and finally what counts as a cause.

Potential Outcomes

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T_i: Dichotomous Treatment assignment for unit i (multi-valued treatments are possible too–just more potential outcomes for each unit)

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τ_i: The treatment effect

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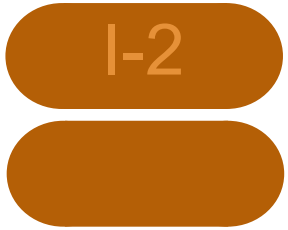

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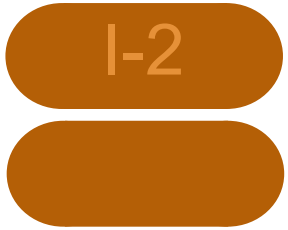

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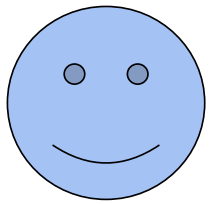

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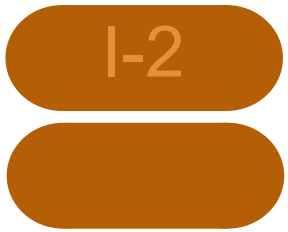

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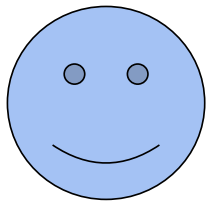

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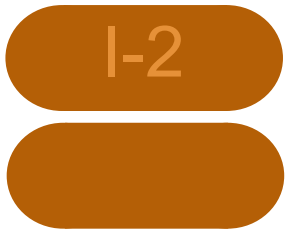

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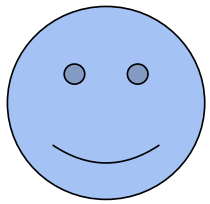

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Illustrated potential outcomes here and later courtesy of Erin Hartman

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Treatment assignment is the source of randomness

Causal Inference is a Missing Data Problem

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No methodology allows us to simultaneously observe both potential outcomes, $Y_i(1)$ and $Y_i(0)$, making τ_i unobservable–and unidentifiable without additional assumptions (Fundamental Problem of Causal Inference Holland (1986))

Causal Inference is a Missing Data Problem

Example: Asprin's Impact on Headaches

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Step 1: (Randomly) sample units

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Step 2: Randomly assign treatment

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Step 3: Measure revealed potential outcome

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- Treatment effect heterogeneity: Zero ATE doesn't mean zero effect for everyone

1. Core Ideas in Causal Inference

2. Potential Outcomes
   - Framework
   - Estimands
   - Three Big Assumptions
   - What Gets to Be a Cause

3. Nonparametric Structural Equation Models and Causal Directed Acyclic Graphs

4. The Experimental Ideal

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  - Ex: in an experiment with 3 units, if the potential outcomes for unit i depend on the treatment assignment of units j and k, the potential outcomes for unit i are defined by $Y(i, j, k)$:

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- Same version of the treatment

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We also need to assume Positivity $0 < P(T_i = 1) < 1 \; \forall \; i$ with probability 1.

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- Another way of thinking of it: The distributions of the potential outcomes $(Y_i(1),\ Y_i(0))$ are the same for the treatment and control group.
- Yet another way of thinking of it: The treatment and control group are exchangeable, or balanced (on observables and unobservables) on average

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- Assume a completely randomized trial (fixed number of units assigned to treatment)
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Design-based Inference for SATE

Variance of $\hat{\tau}$:

$$V[\hat{\tau}] = \frac{1}{n}\left(\frac{n_c}{n_t}S_1^2 + \frac{n_t}{n_c}S_0^2 + 2S_{0,1}\right)$$

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- This is conditional ignorability.

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- Selection bias: how different the treated and control groups are in terms of their potential outcome under control.

Selection Makes Us Care About Assignment Mechanisms

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"The process that determines which units receive which treatments, hence which potential outcomes are realized and thus can be observed, and, conversely, which potential outcomes are missing."
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- Unconfounded assignment: Disallows dependence of the assignment mechanism on the potential outcomes

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Most statistical models of causal inference attain identification of treatment effects by restricting the assignment mechanism in some way.

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- Identification involves using the do-calculus to write the causal quantity (which involves do()) in terms of an empirical quantity (which involves only observed data).

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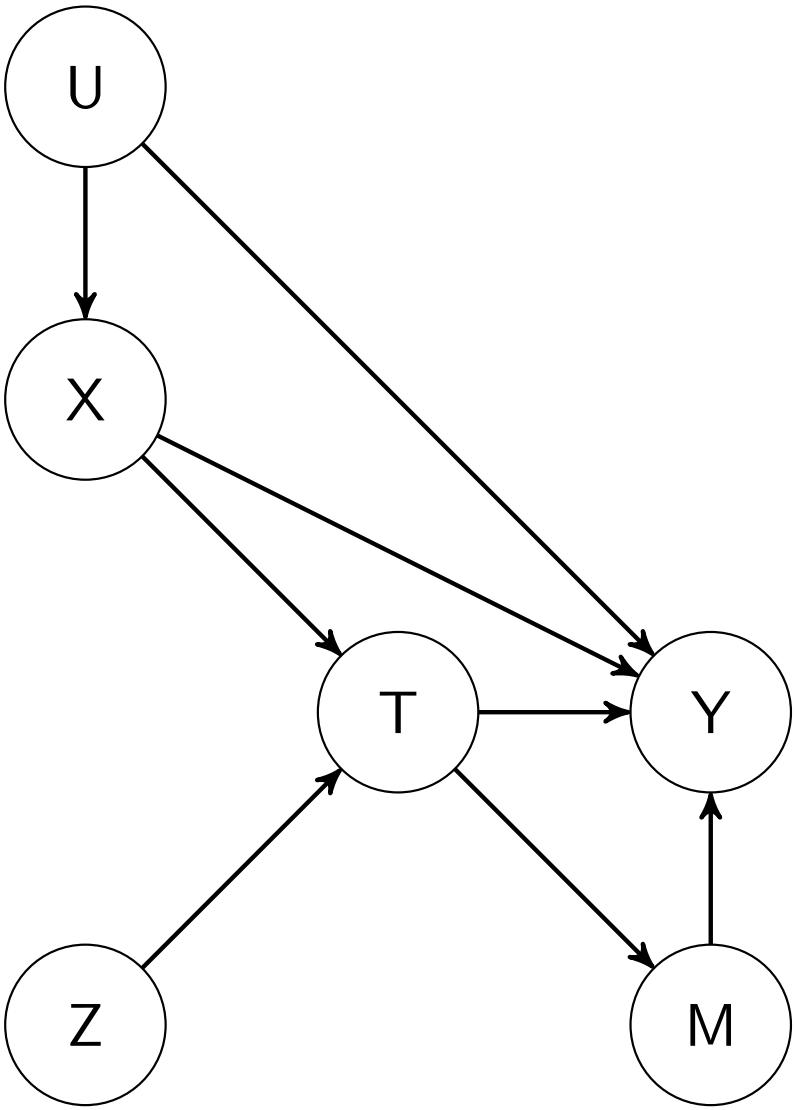
Code Idea: causal reasoning before statistical reasoning

Components of a DAG



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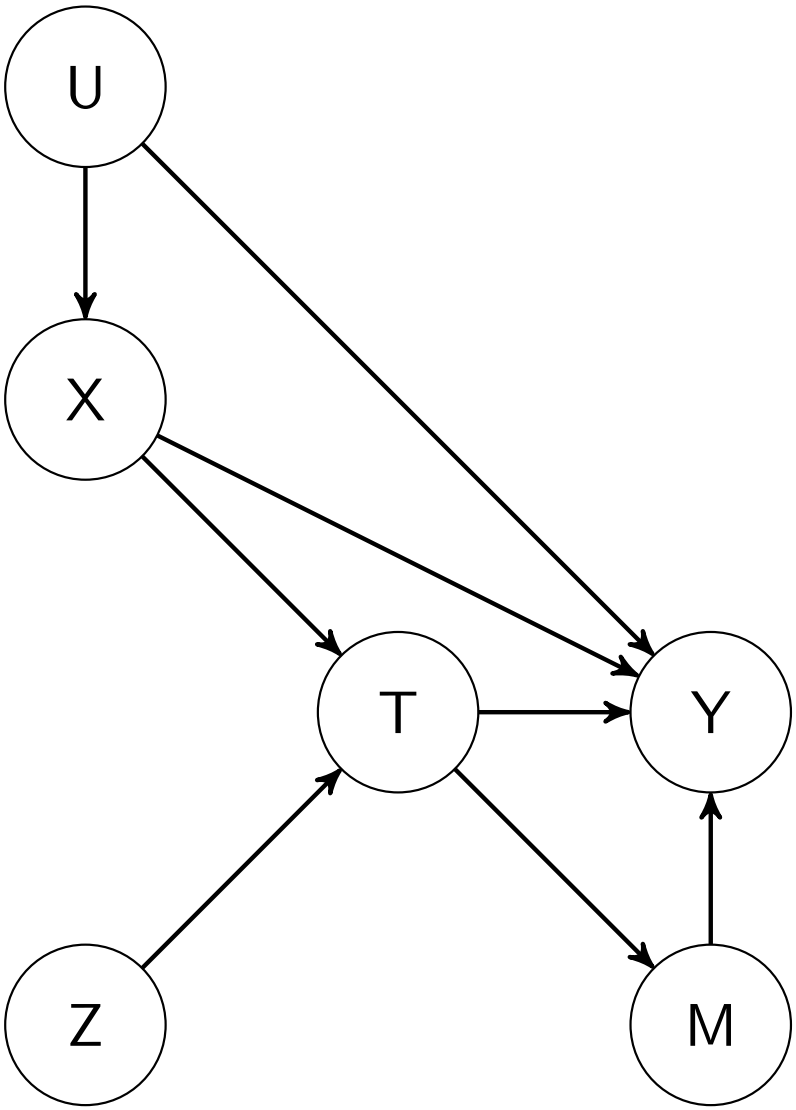

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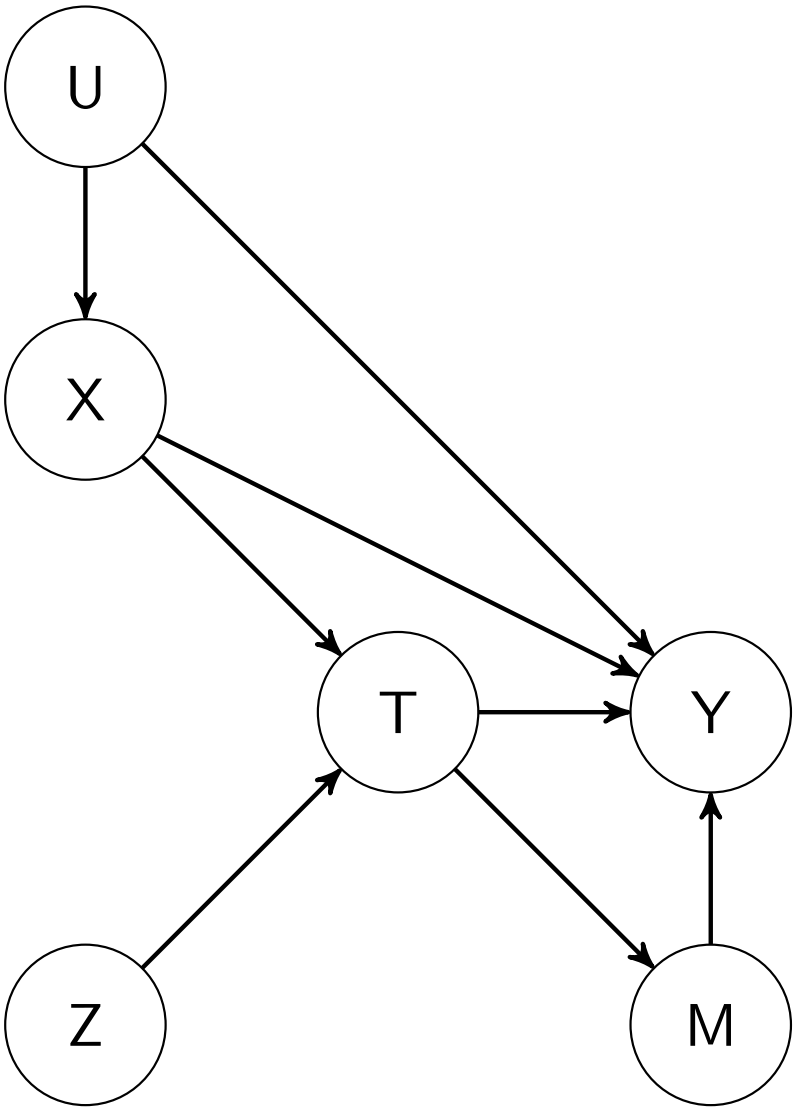

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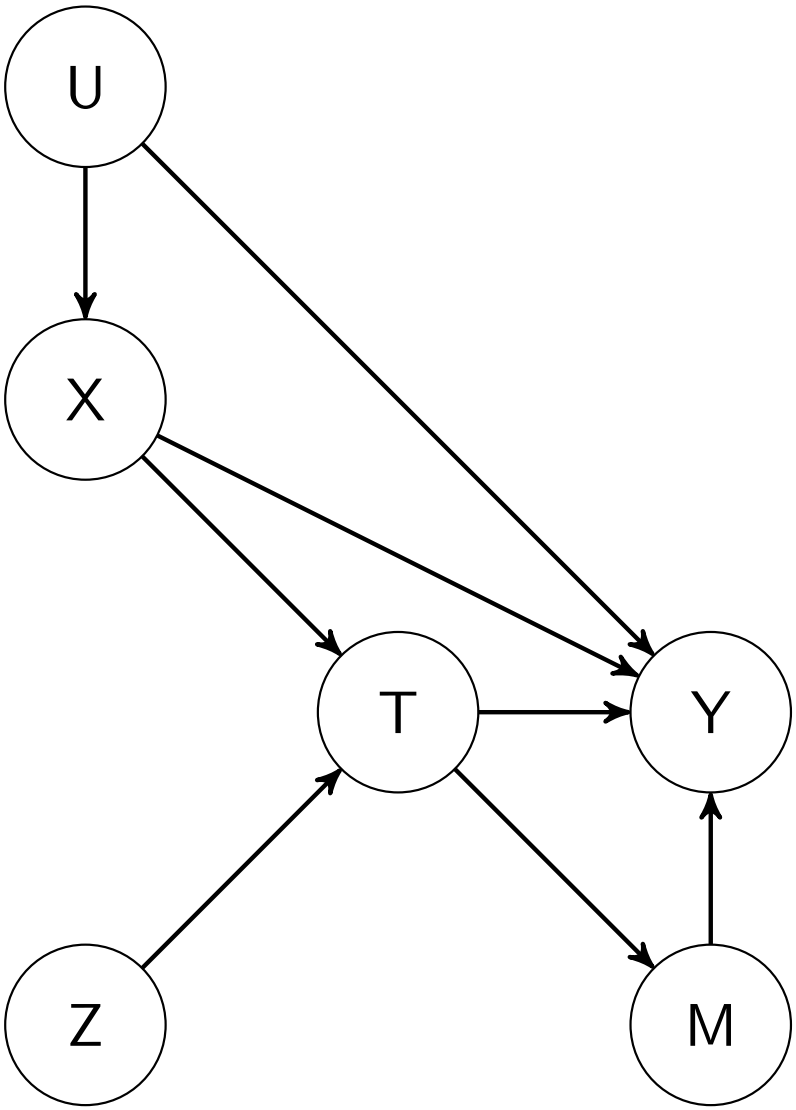

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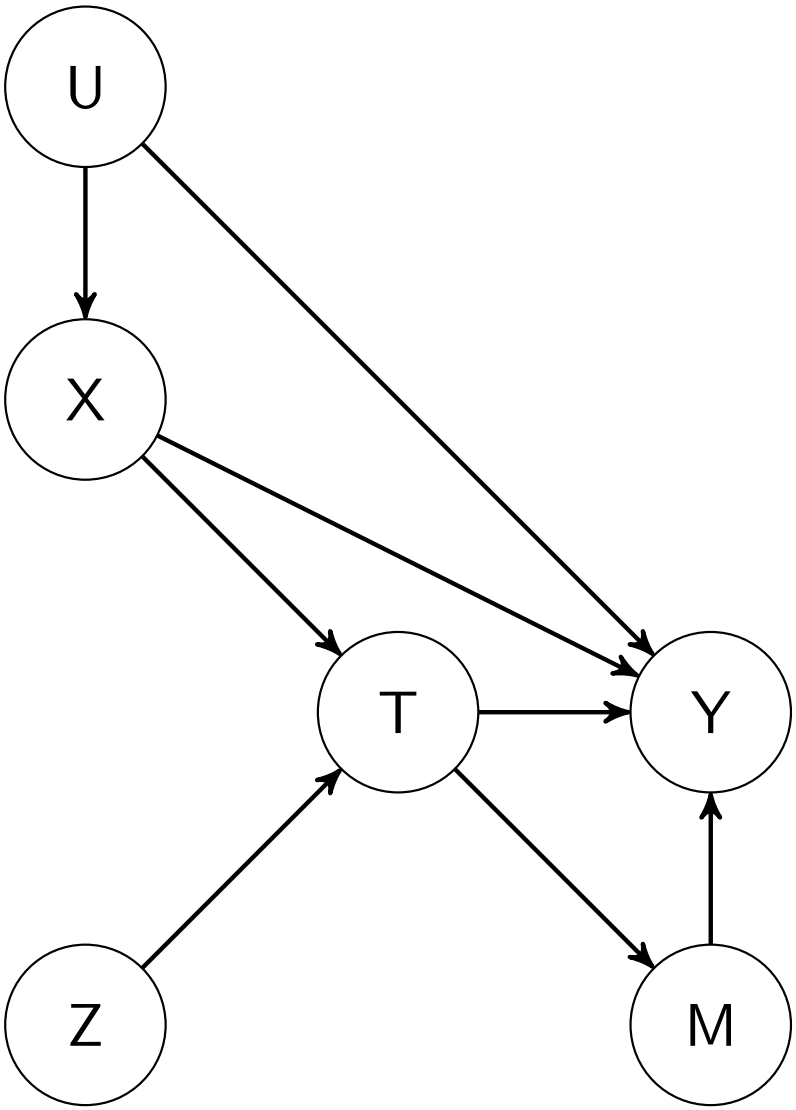

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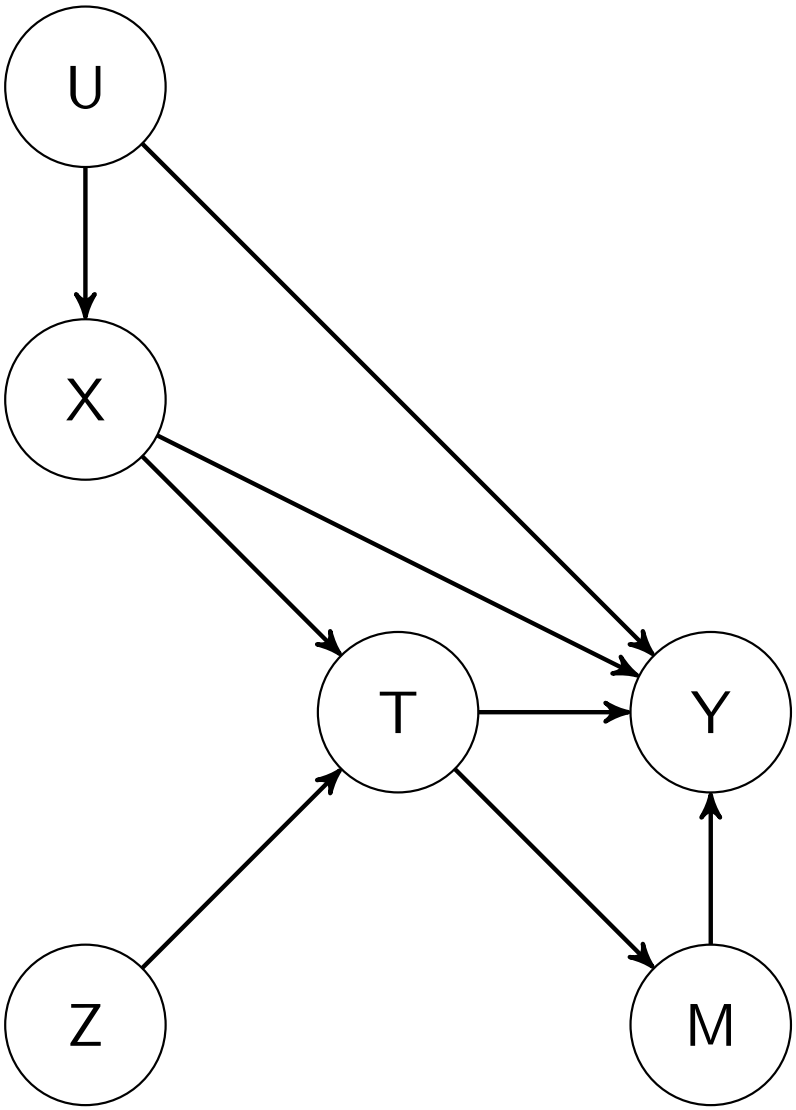

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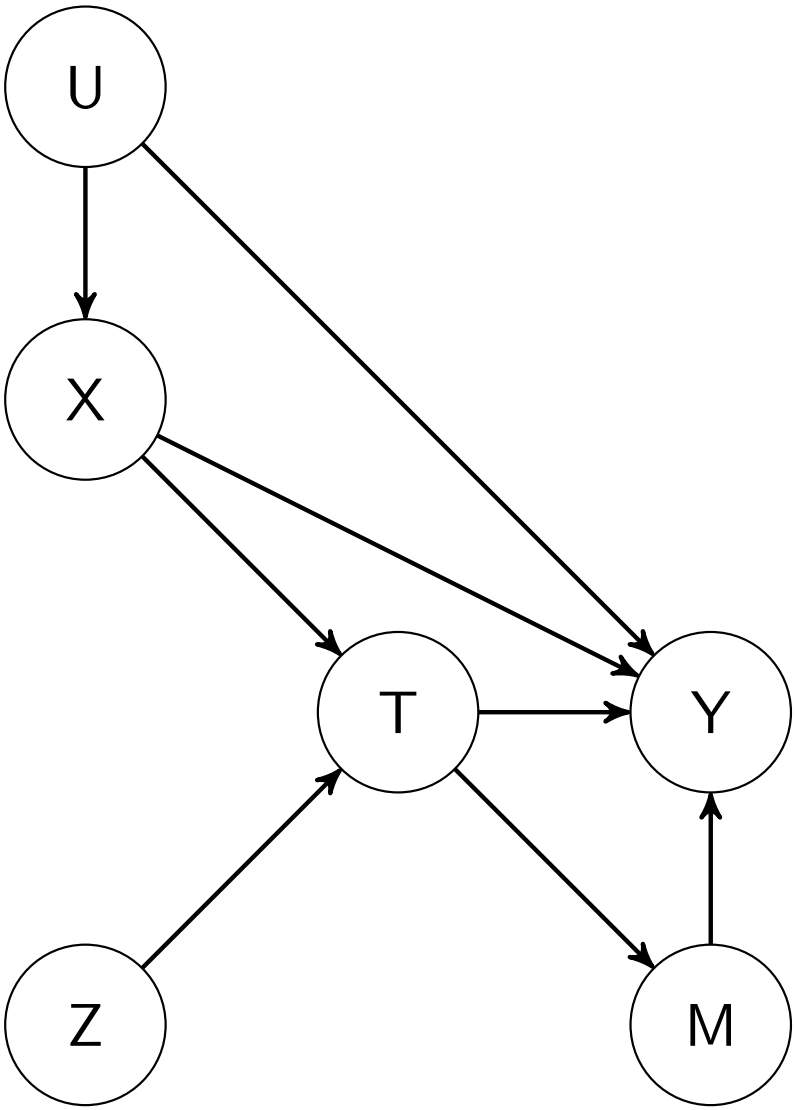

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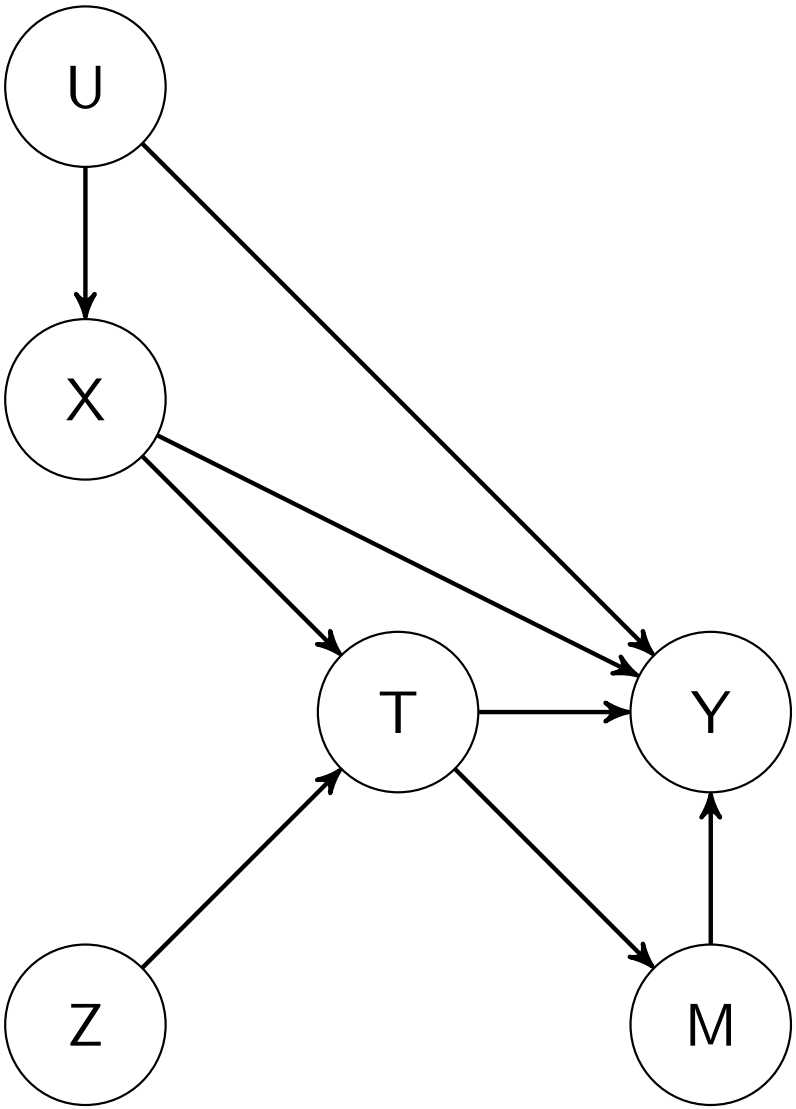

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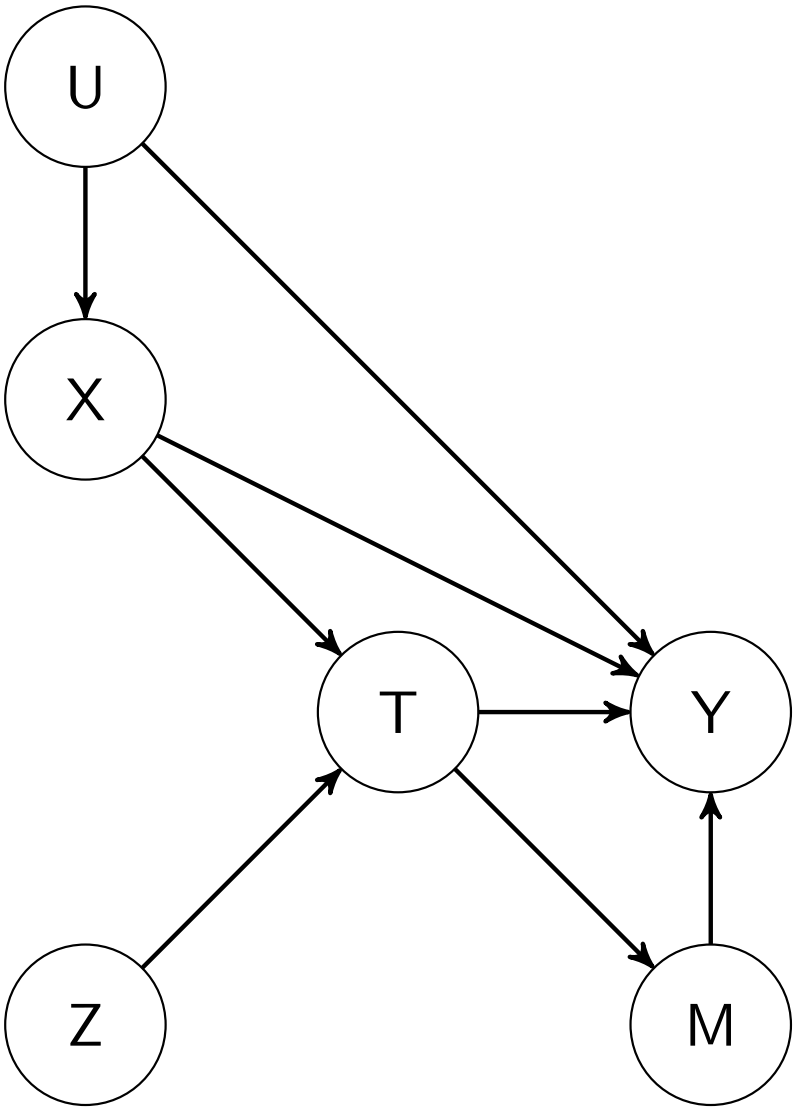

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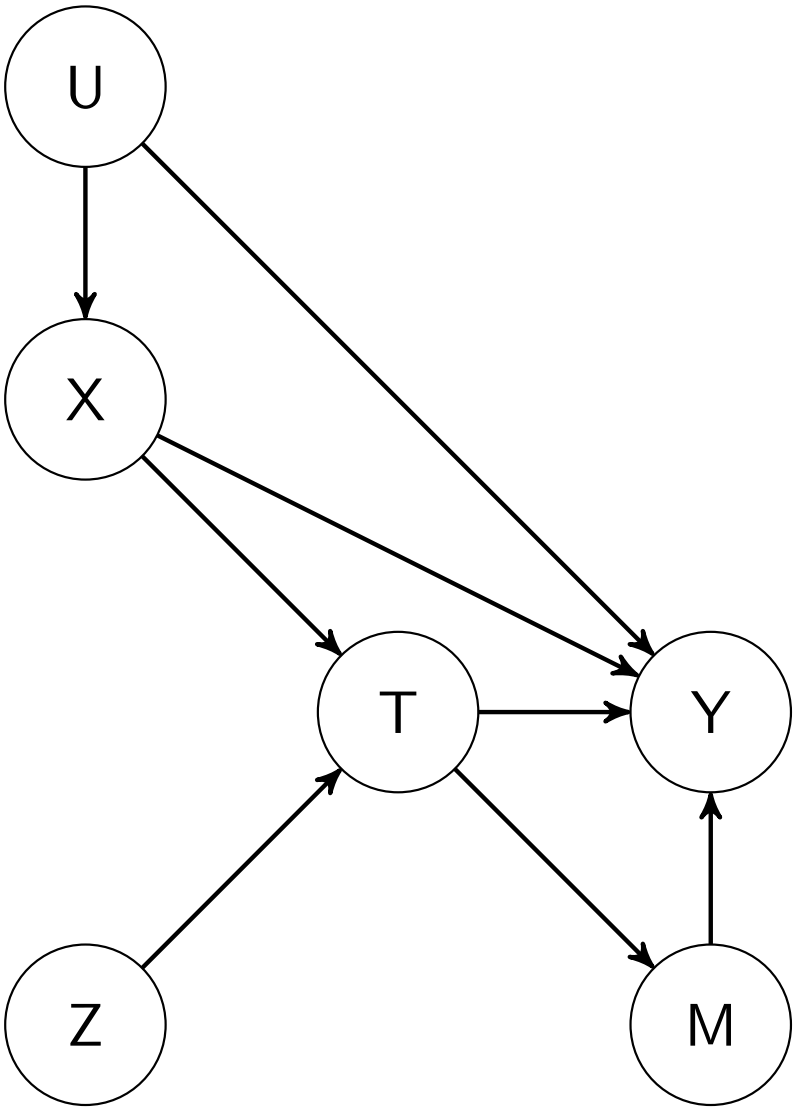

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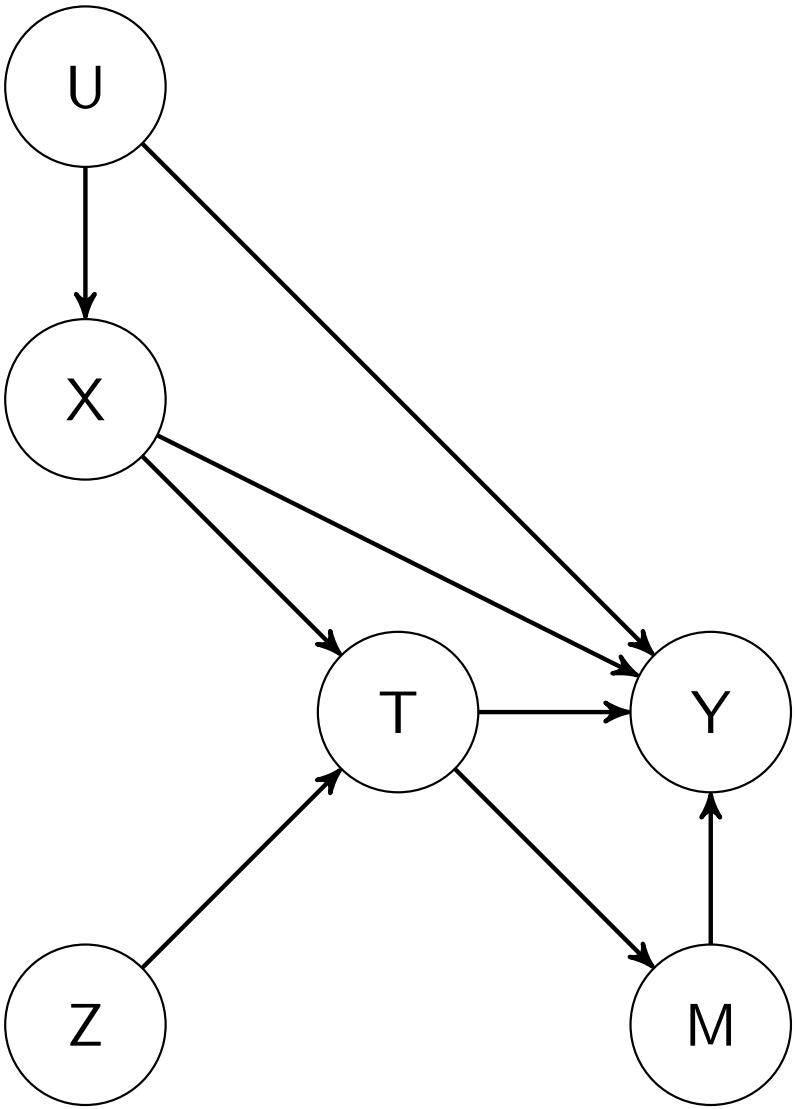

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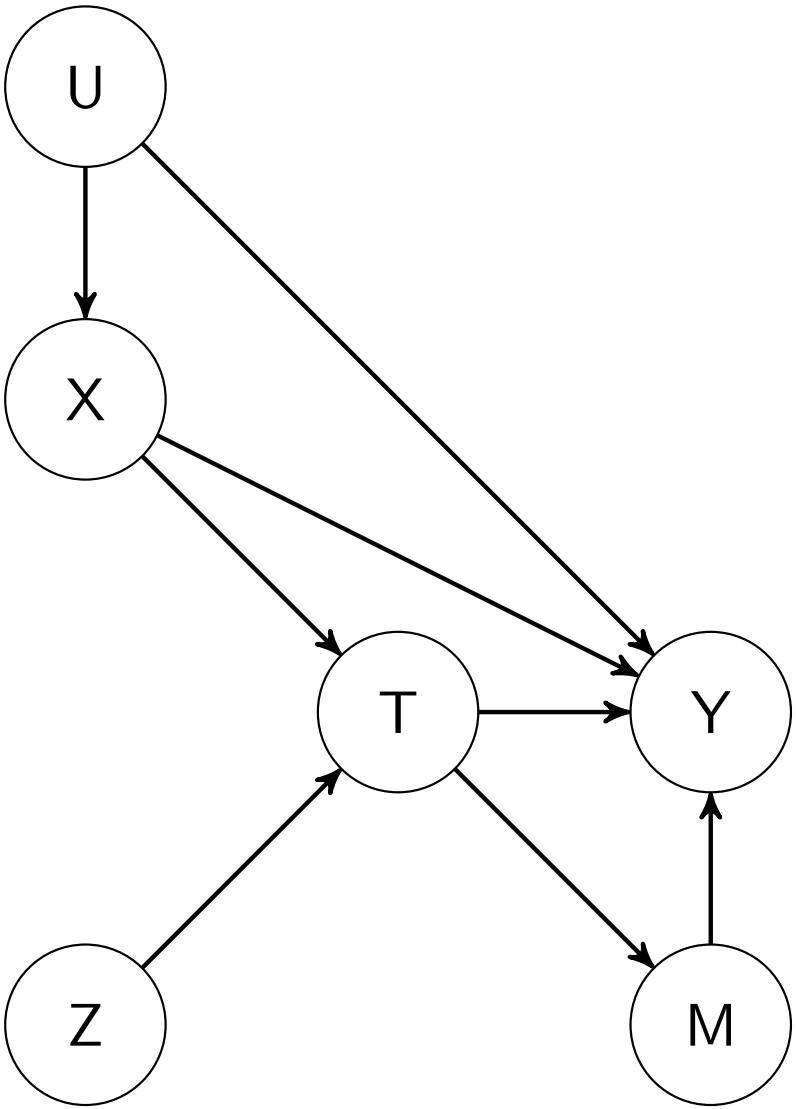

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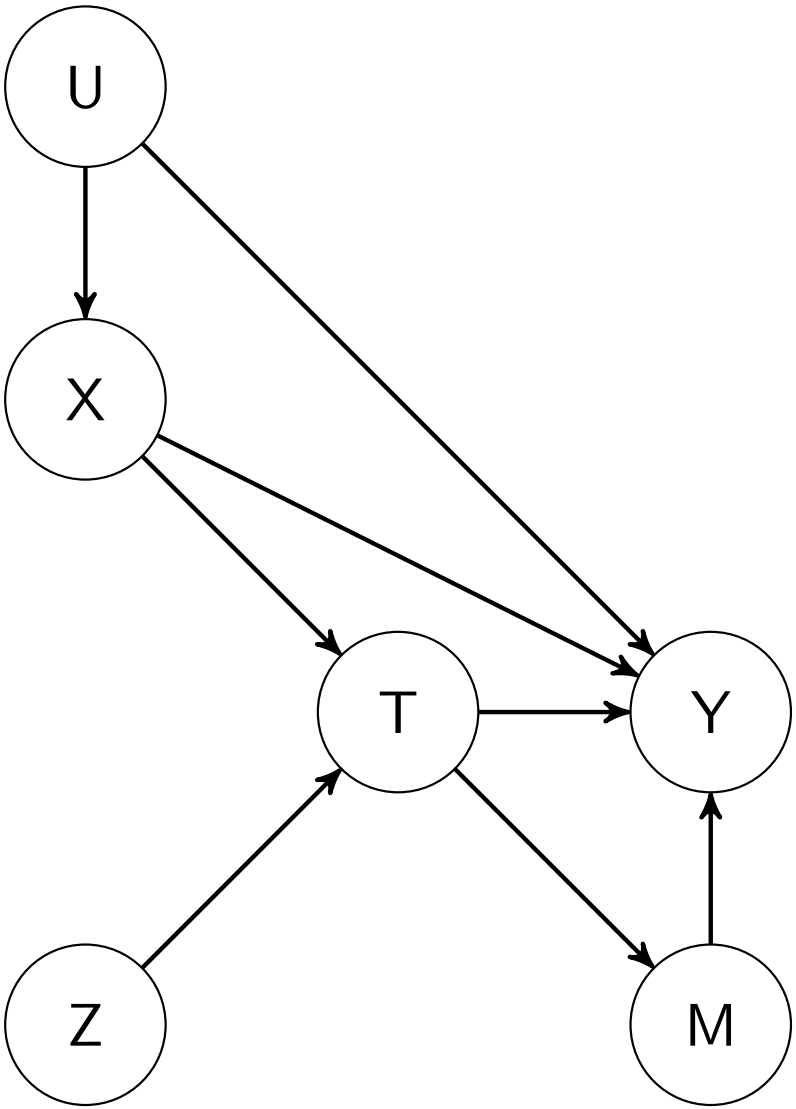

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- We will talk in depth about two types of relationships: confounders and colliders

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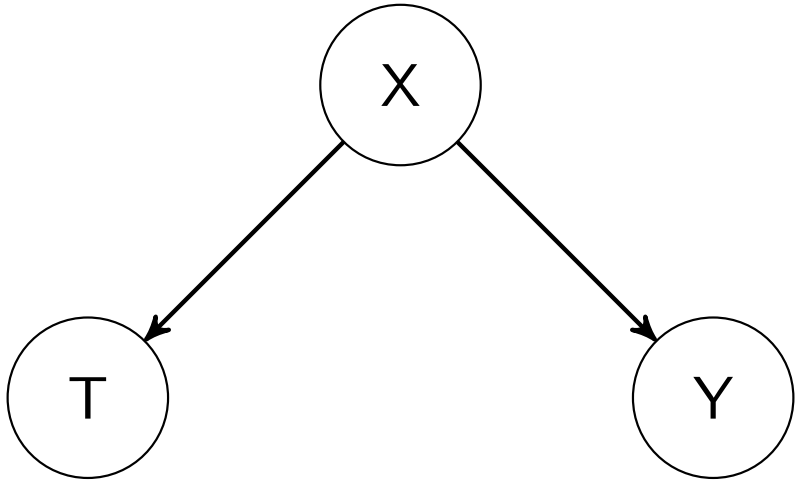

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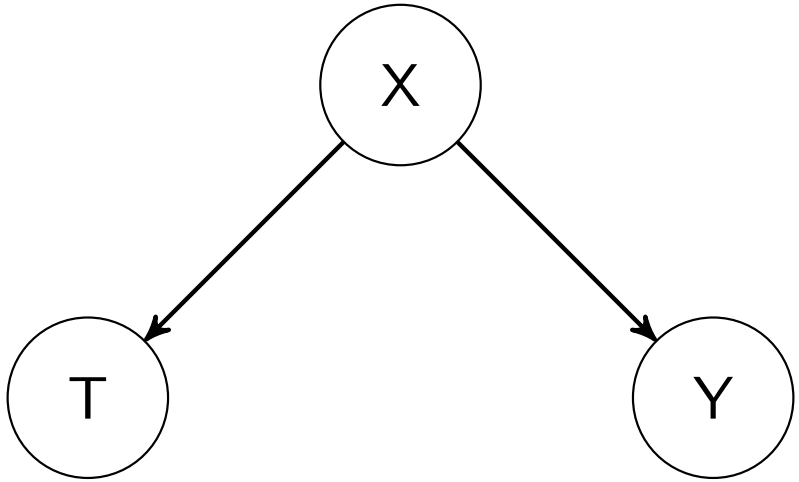

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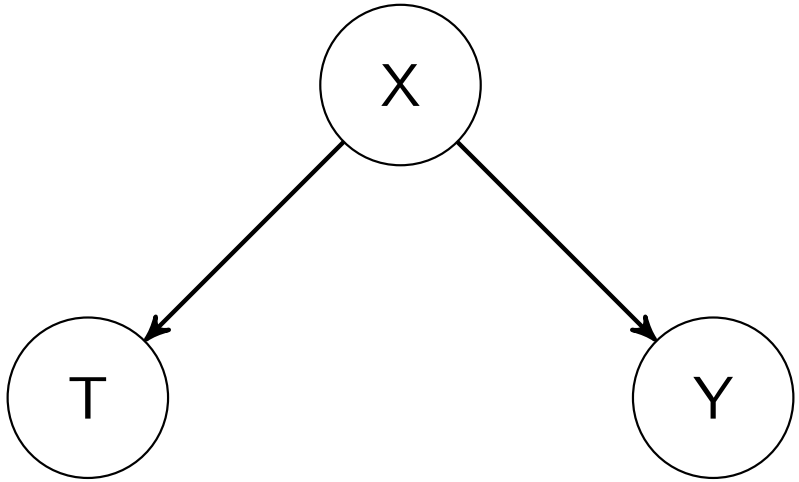

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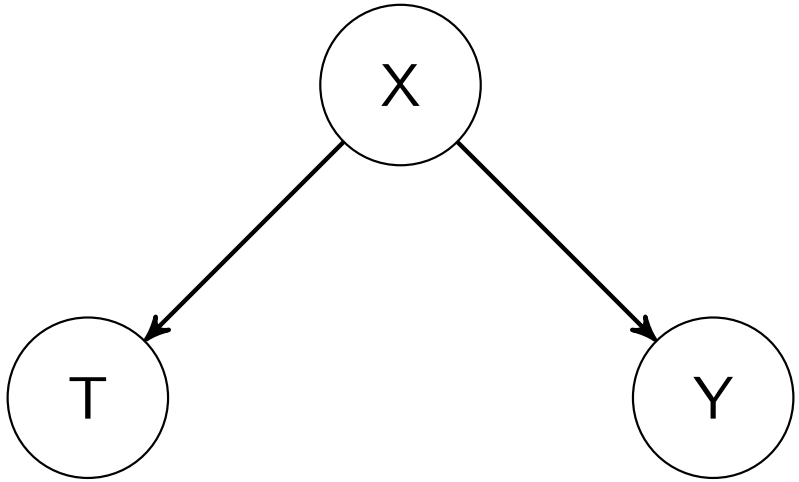

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- We can think of conditioning on a confounder as blocking the flow of association.

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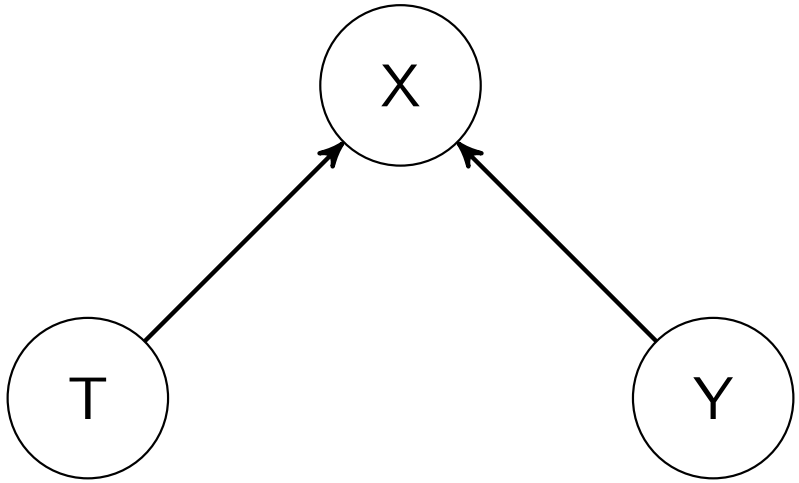

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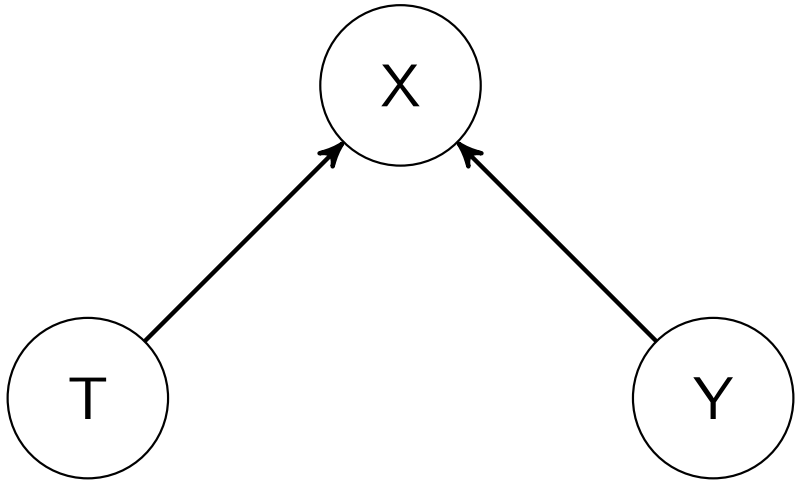

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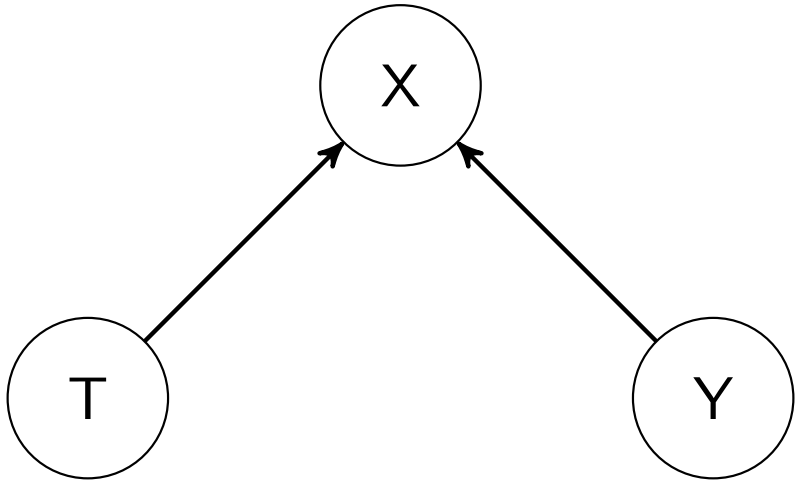

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- In this scenario T and Y are not marginally associated
- If we control for X they become associated and create a connection between T and Y

Colliders are scary because you can induce dependence

Endogenous Selection Bias: The Problem of Conditioning on a Collider Variable

Felix Elwert[1] and Christopher Winship[2]

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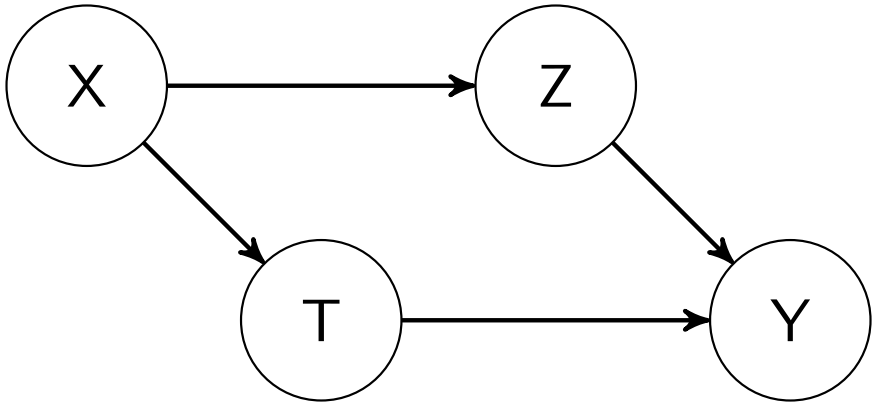
Keywords

causality, directed acyclic graphs, identification, confounding, selection

Abstract

Endogenous selection bias is a central problem for causal inference. Recognizing the problem, however, can be difficult in practice. This article introduces a purely graphical way of characterizing endogenous selection bias and of understanding its consequences (Hernán et al. 2004). We use causal graphs (directed acyclic graphs, or DAGs) to highlight that endogenous selection bias stems from conditioning (e.g., controlling, stratifying, or selecting) on a so-called collider variable, i.e., a variable that is itself caused by two other variables, one that is (or is associated with) the treatment and another that is (or is associated with) the outcome. Endogenous selection bias can result from direct conditioning on the outcome variable, a post-outcome variable, a post-treatment variable, and even a pre-treatment variable. We highlight the difference between endogenous selection bias, common-cause confounding, and overcontrol bias and discuss numerous examples from social stratification, cultural sociology, social network analysis, political sociology, social demography, and the sociology of education.

From Confounders to Back-Door Paths



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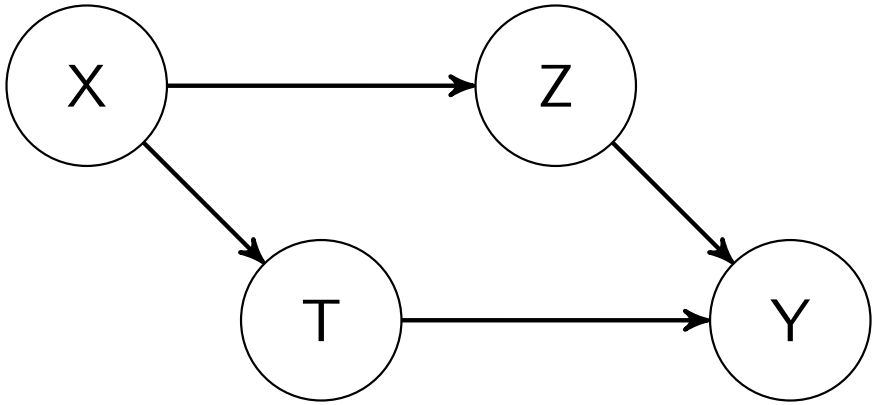

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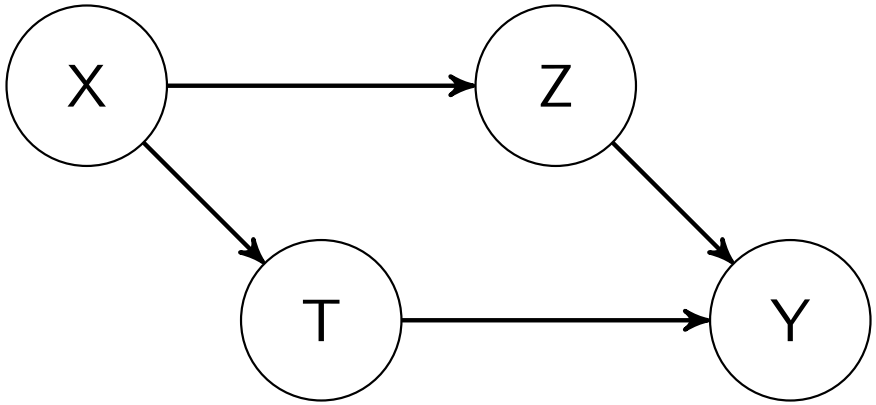

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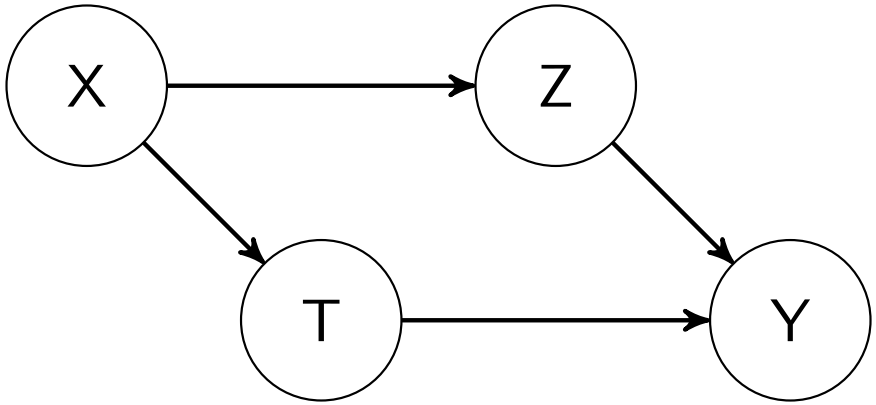

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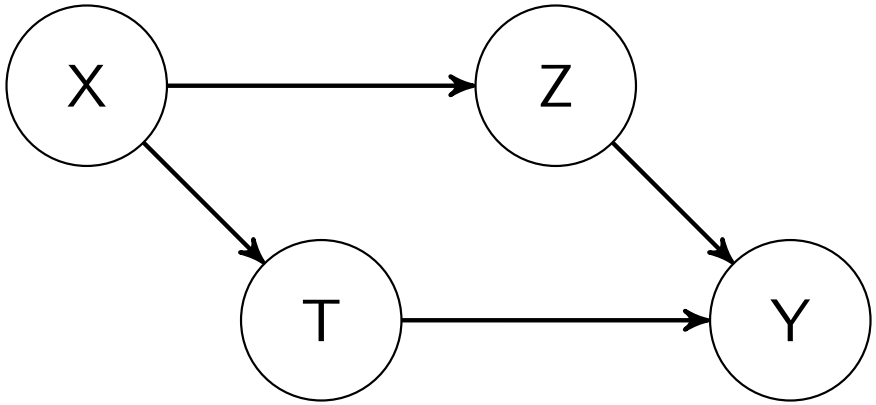

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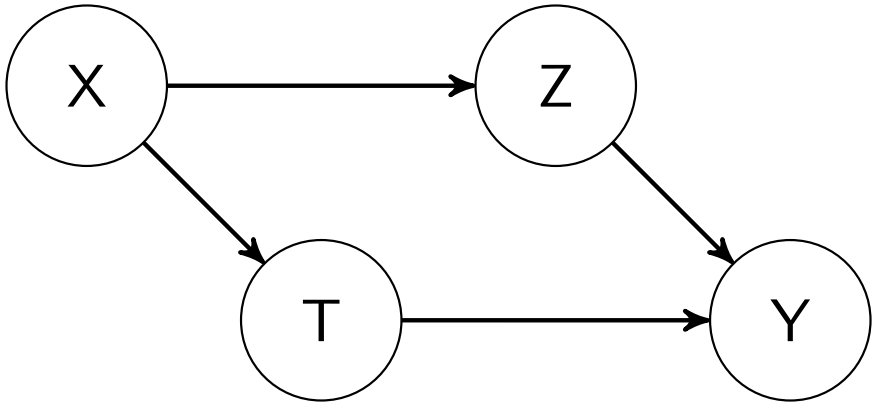

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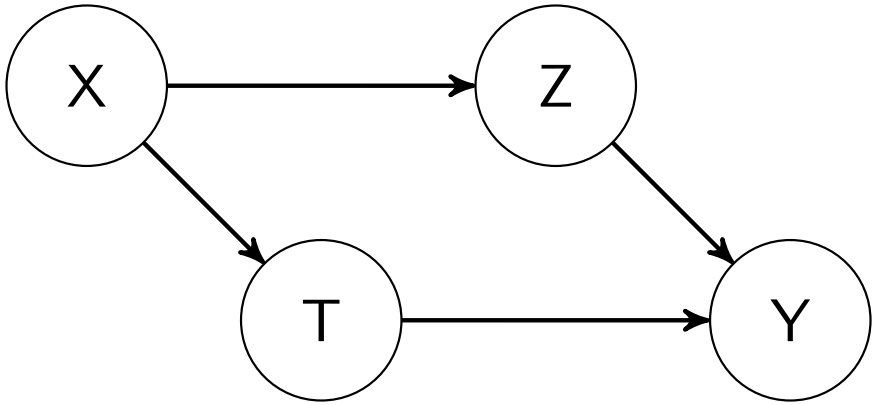
From Confounders to Back-Door Paths



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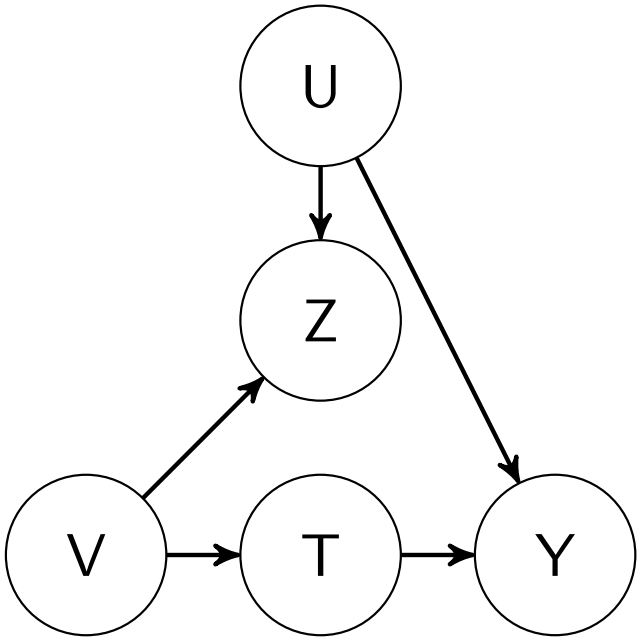
Colliders and Back-Door Paths



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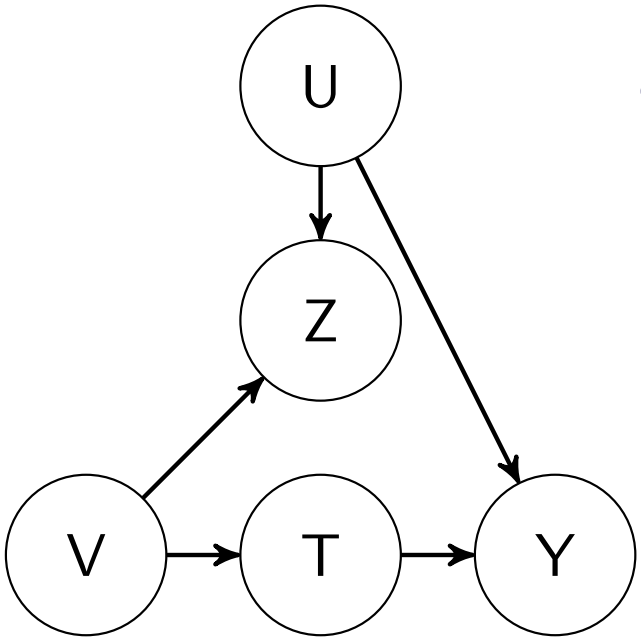

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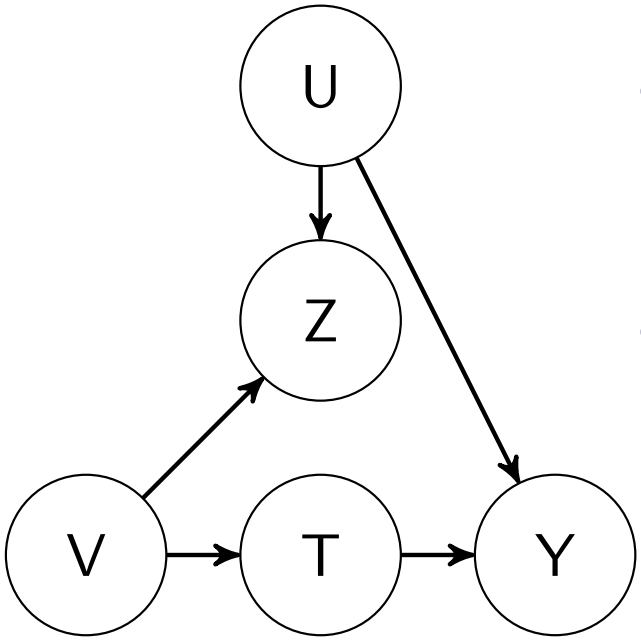

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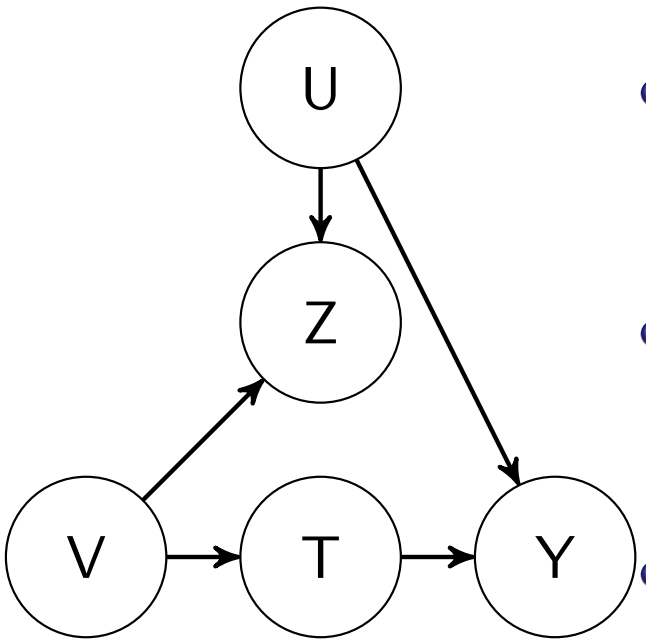

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- So how do we know which back-door paths to block?

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- If A is not D-separated from B by C we say that A is D-connected to B by C

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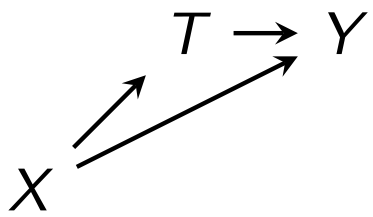
- We will see some other approaches late in the semester.

Blocking backdoor paths: College and earnings

What do we need to include to block all backdoor paths between college and earnings?



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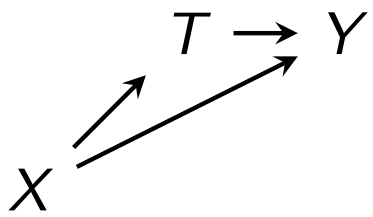

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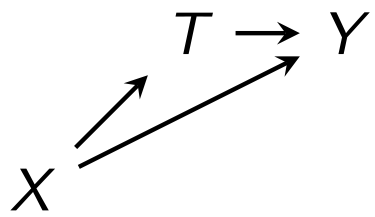

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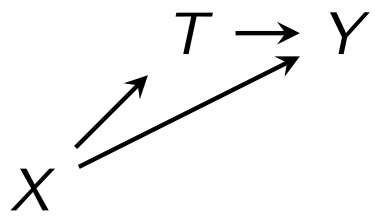
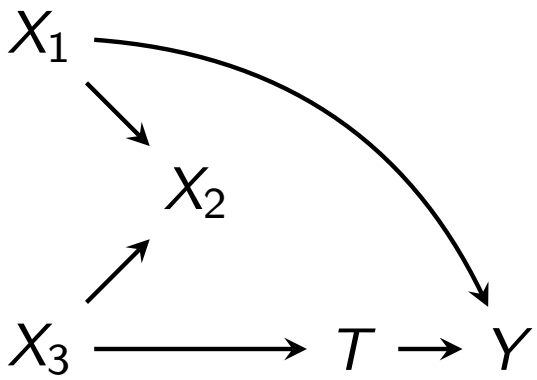


Ability, parents' income, parents' education, extended family who pay for college and help you find a job, neighborhood characteristics that affect high school quality and also the availability of local jobs, ... lots of things!

Non-causal paths: Part 2

Now consider this graph. Is there an unblocked backdoor path from T to Y?



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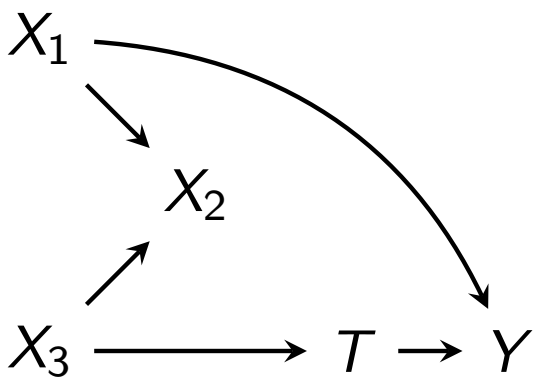
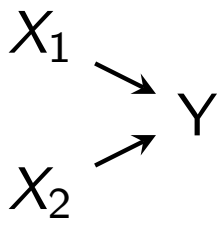


No need to condition! X_2 already blocks this path. it is a collider.

Colliders: Be careful!



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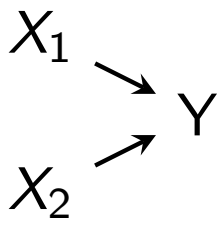

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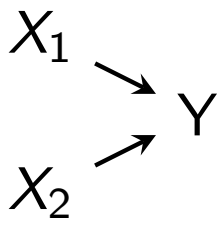

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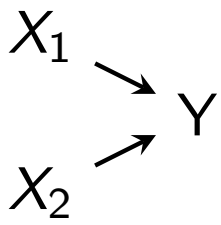

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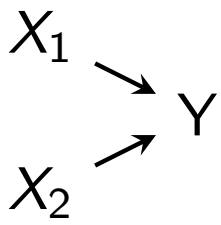

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Should we worry about this design? It depends on our theory about how these variables are related. We can argue about identification with a DAG.

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Suppose working in Hollywood is a function of two factors: acting ability and beauty. In the general population, these two are uncorrelated. However, among those who work in Hollywood, those who are bad at acting must be beautiful.

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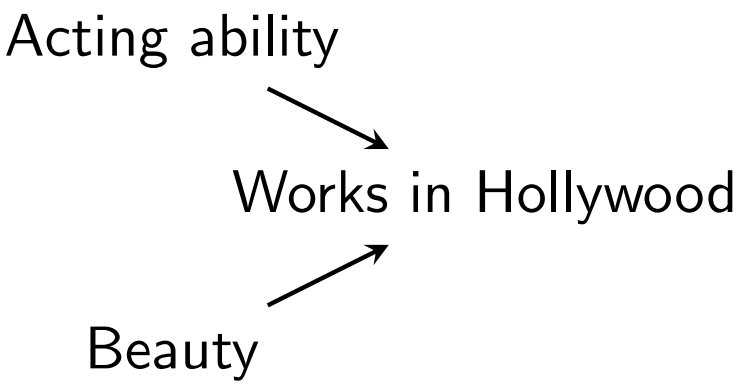

Conditional on Hollywood

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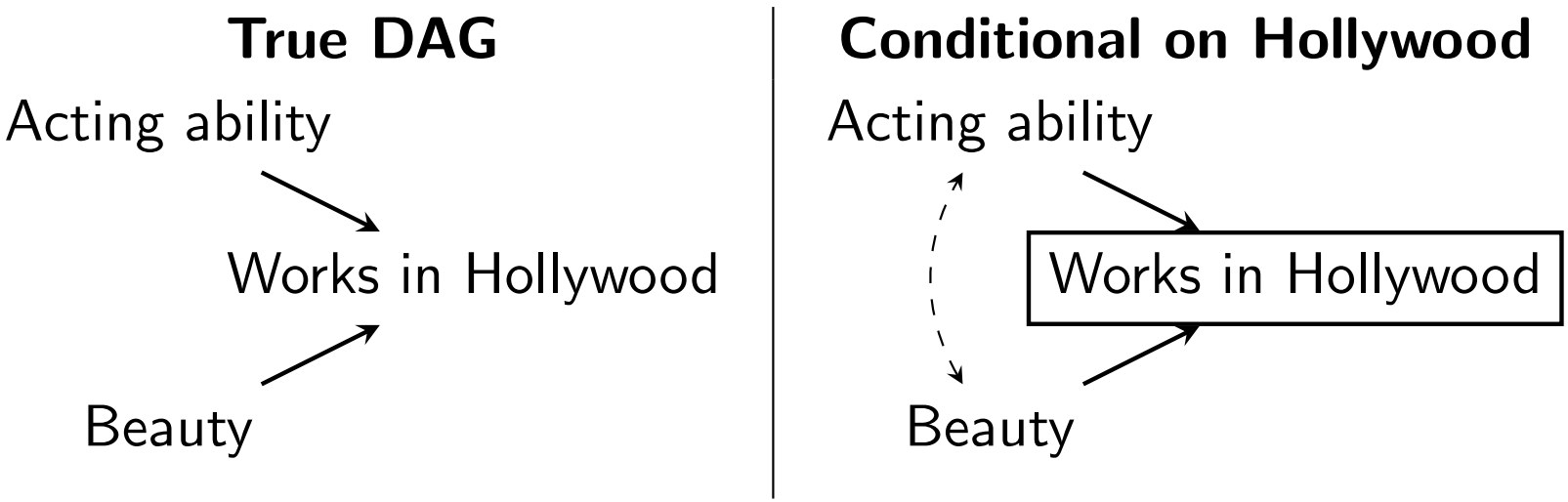

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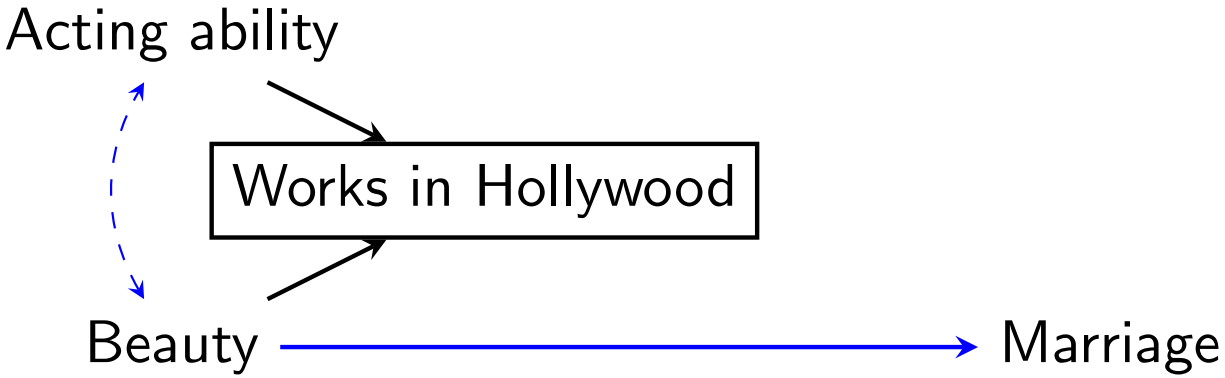
This is an example of conditioning on a collider! We induce a negative association between acting ability and beauty.



Under the assumptions above, our results are driven by collider conditioning!

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   - Three Big Assumptions
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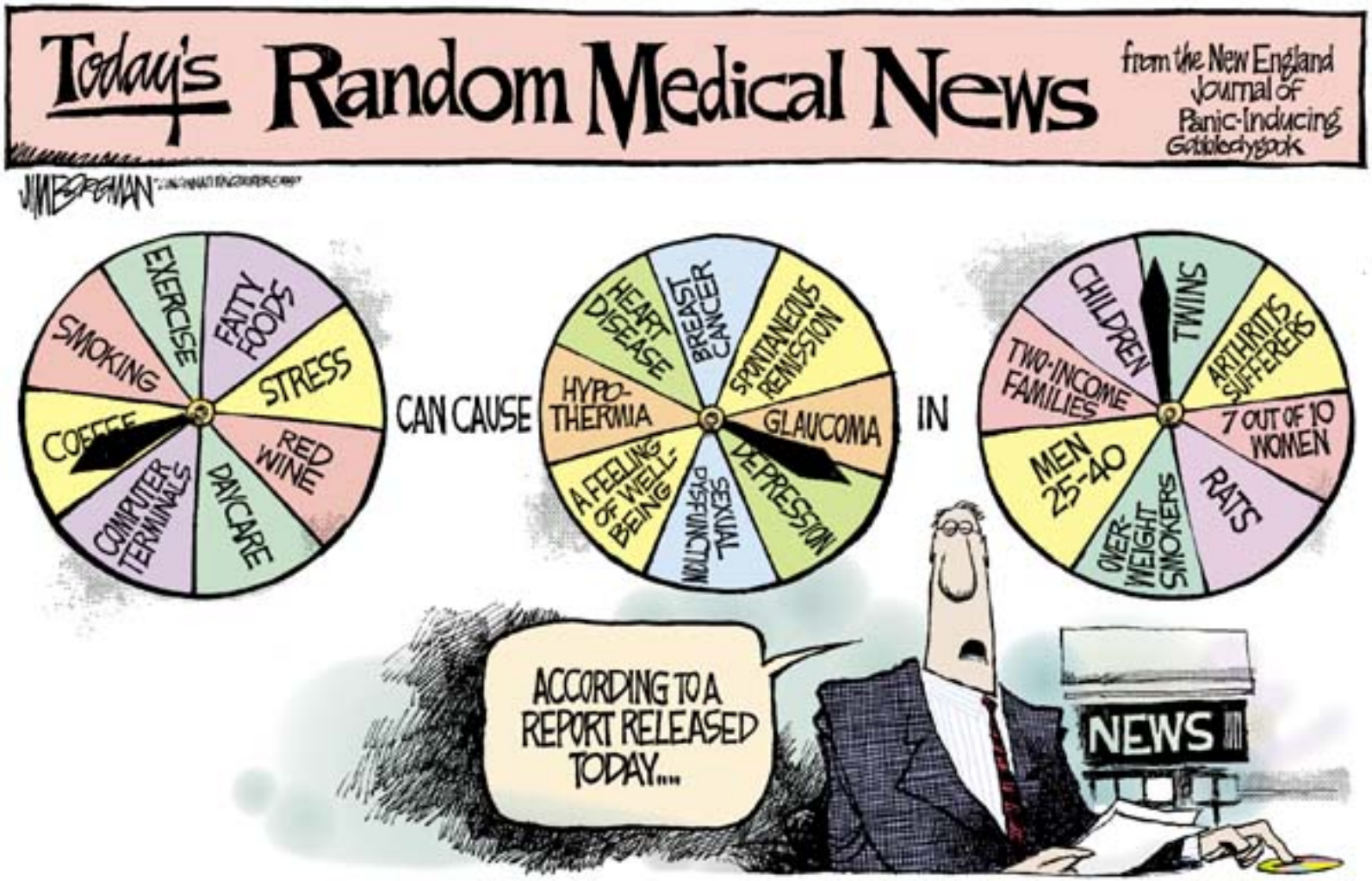
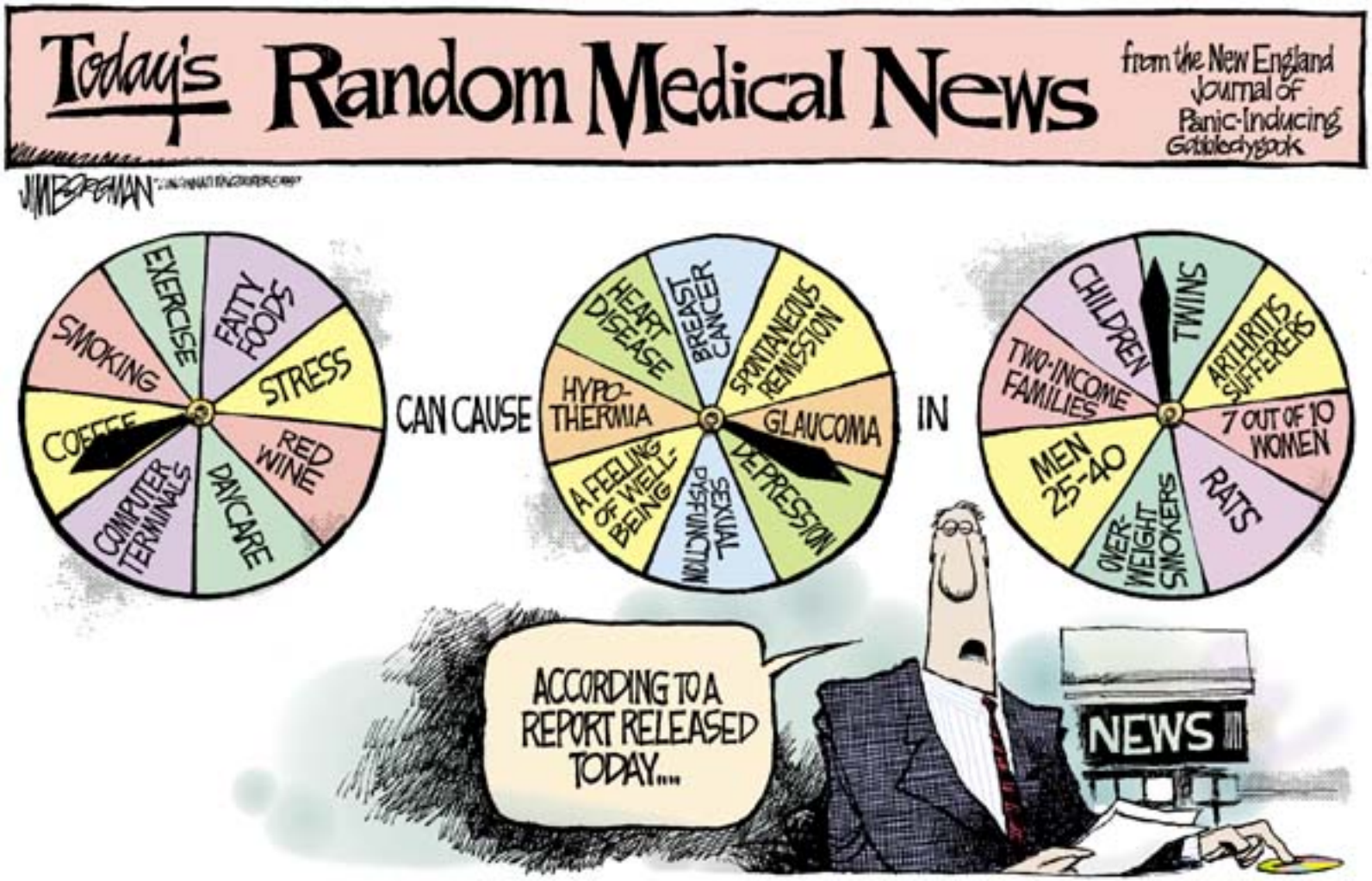
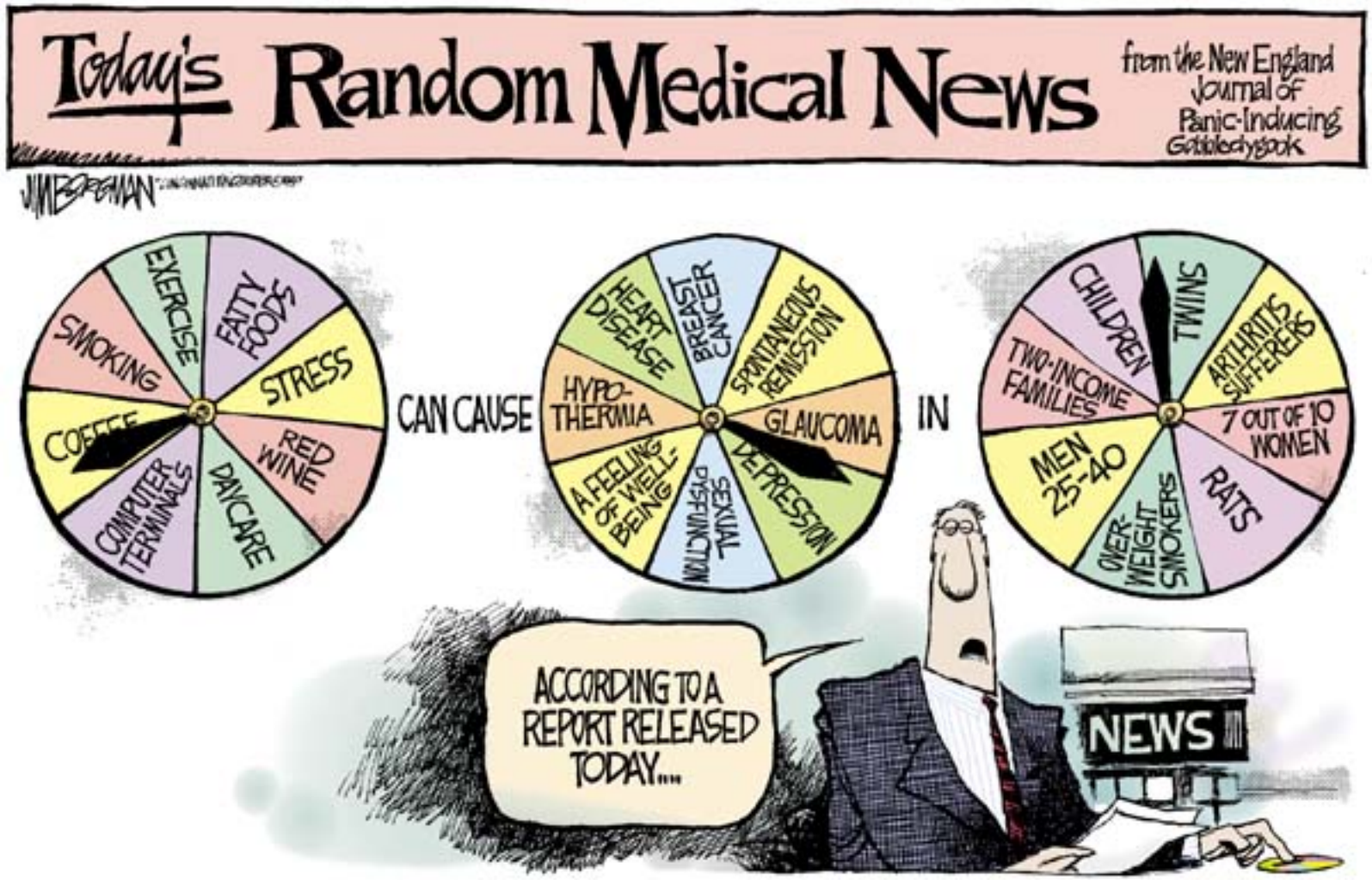


Lancet 2001: negative correlation between coronary heart disease mortality and level of vitamin C in bloodstream (controlling for age, gender, blood pressure, diabetes, and smoking)



Lancet 2002: no effect of vitamin C on mortality in controlled placebo trial (controlling for nothing)



Lancet 2003: comparing among individuals with the same age, gender, blood pressure, diabetes, and smoking, those with higher vitamin C levels have lower levels of obesity, lower levels of alcohol consumption, are less likely to grow up in working class, etc.

Why So Much Variation?

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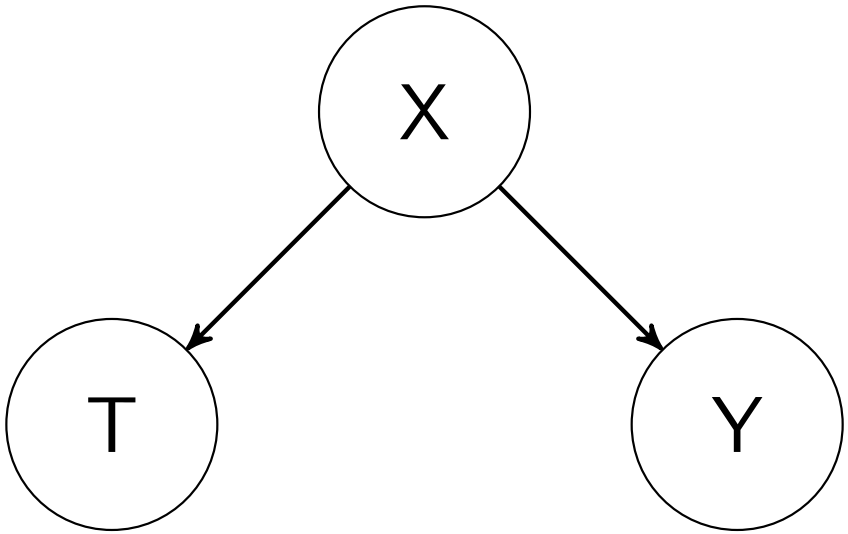
Confounders



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  - "The planner of an observational study should always ask himself [sic]: How would the study be conducted if it were possible to do it by controlled experimentation" (Cochran 1965)

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  1. Positivity: assignment is probabilistic: $0 < P(T_i = 1) < 1$
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     - ★ Treatment assignment does not depend on any potential outcomes.
     - ★ Sometimes written as $T_i \perp\!\!\!\perp (\mathbf{Y}(1), \mathbf{Y}(0))$

Why do Experiments Help?

Remember selection bias?

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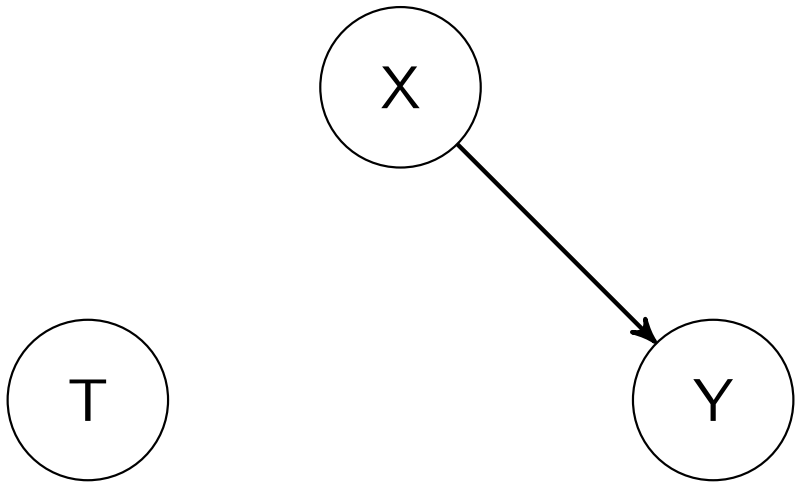
When all goes well, an experiment eliminates selection bias.

Randomization Removes the Arrows into the Treatment



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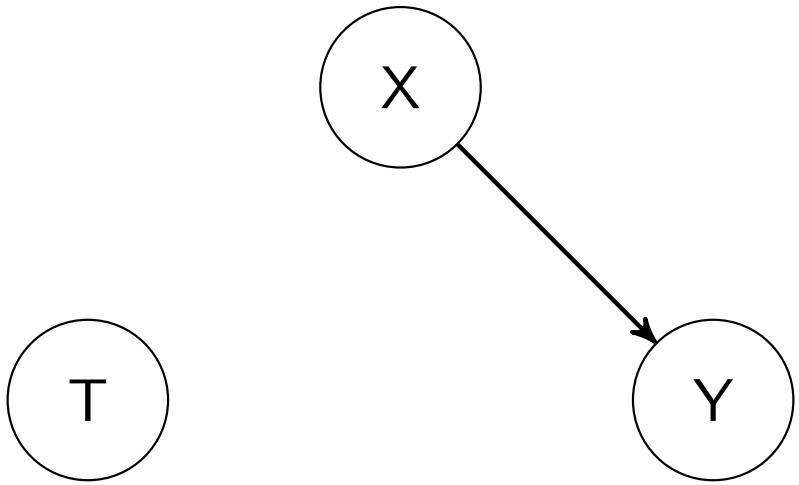

This ensures any observed or unobserved pretreatment covariates have the same distribution in the treatment and the control group. That is, they are balanced.

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- Generally, stratified designs mean that the probability of treatment depends on a covariate, X_i: $P(T_i = 1 | X_i = x)$.
- Conditional randomization assumptions:
  - Positivity: $0 < P(T_i = 1 | X_i = x) < 1$ for all i and x.
  - Unconfoundedness: $P(T_i = 1 | \mathbf{X}, \mathbf{Y}(1), \mathbf{Y}(0)) = P(T_i = 1 | X_i)$

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\end{aligned}$$

- ATE is just the weighted average of the within-strata differences in means.
- Identified because the last line is a function of observables.
- The averaging is over the distribution of the strata $\rightsquigarrow$ size of the blocks.

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- We cover a bit because experiments are a dominant metaphor. Much of the work on observational studies is trying to find a circumstance where we can pretend we have a stratified experiment.
- Of course, in real experiments sometimes people don't always do what we say (compliance problems).

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- estimation of causal effects does not require identical treatment and control groups
- you need a clear counterfactual to have a well-defined causal effect. For example of 'the recession was caused by Wall Street' may make intuitive sense but is it well-defined?

http://egap.org/methods-guides/10-things-you-need-know-about-causal-inference

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Next Time: Causality with Measured Confounding