

## Reminders:

- **Tim's office hours 11:20-11:50am PT today**
- **Final project presentations in person G01/G10 on Monday, June 3, 10:30am-12:30pm** (check Ed for links, schedule, details)
  - Great opportunity to learn about each other's projects. Lots of Q&A!
  - Attendance is mandatory
  - Active participation rewarded with extra credit
- **Upload your deliverables on Gradescope by Sunday 23:59pm PT – no late periods so that we can prepare the session and give you feedback and grades quickly**
  - Project Report
  - Presentation Video (and slides PDF)
    - 6 minutes (no credit if longer – we need to be fair across groups)
  - Metadata (primarily dataset info)

# Causal Inference I

## Introduction to Counterfactual Reasoning

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CSE547 Machine Learning for Big Data

Tim Althoff



# Plan for today:

## Introduction to Counterfactual Reasoning

- When is prediction / big data not enough?
- What is causality?
- Potential Outcomes Framework (Rubin)
  - How can we define and compute causal effects?
- Unobserved Confounds & Simpson's Paradox
  - Why we should always worry about confounding in decision making?
- Structural Causal Model Framework (Pearl)
  - How can we make our assumptions explicit?
  - Given our assumptions, is causal inference feasible? Can we *identify* a causal effect?

# Plan for Thursday

- We will learn today that more efficient estimators are needed.
- Effective estimation of causal effects
  - Conditioning on Key Variables
  - Matching
  - Stratification
  - Weighting
  - Regression
  - Sensitivity Analyses

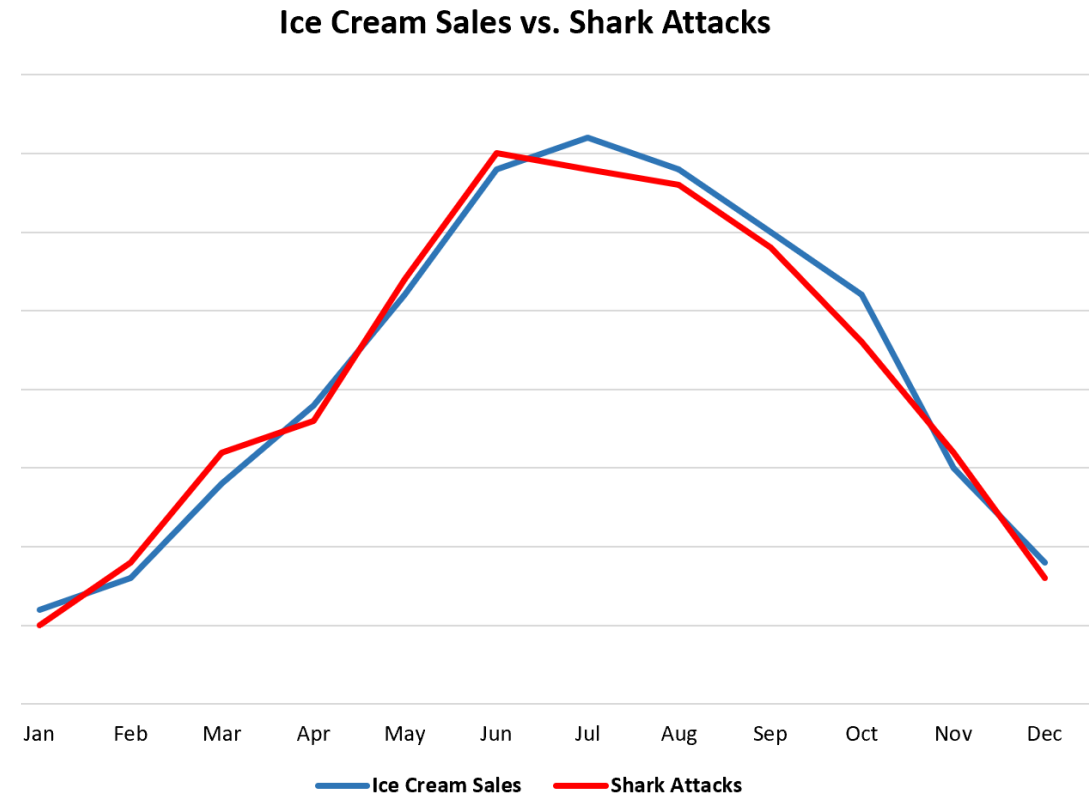
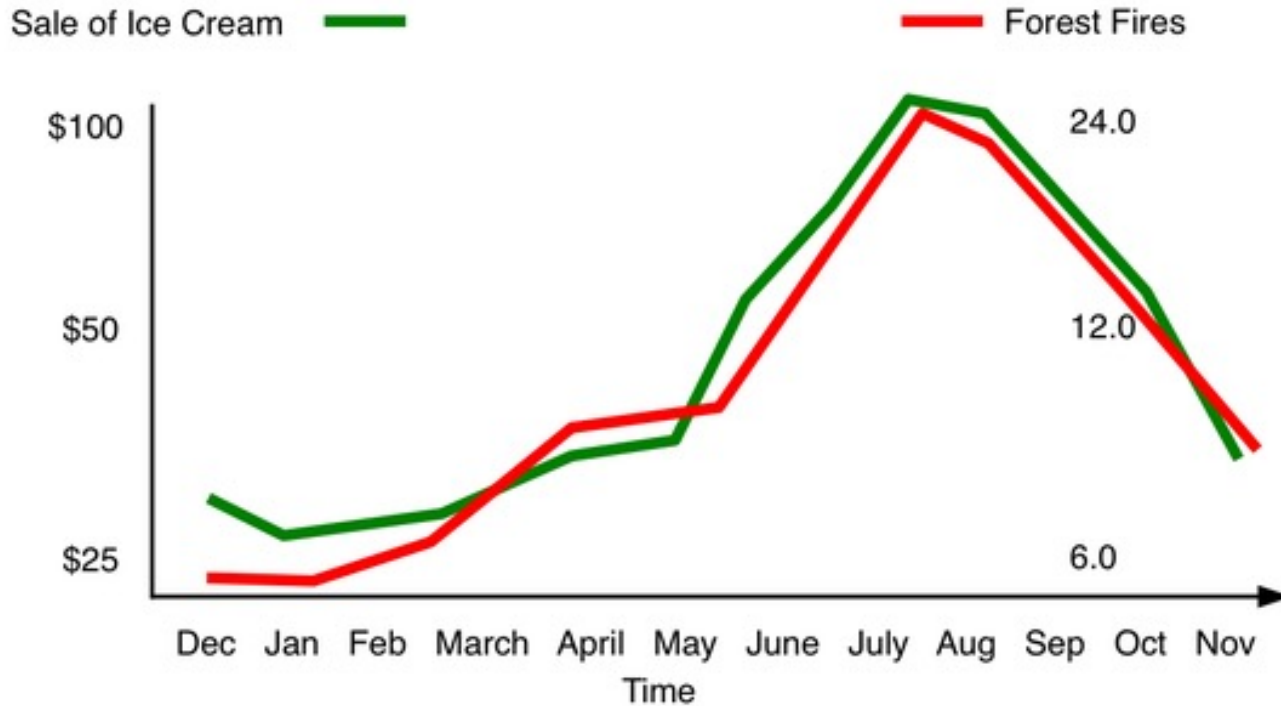
**When is prediction / big data not enough?**

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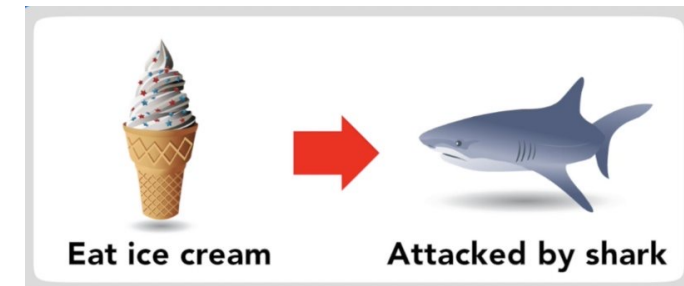
# Prediction is everywhere!

- Recommender Systems: Predicting future rating/consumption
  - Social Networks: Link prediction
  - Course projects ...
- 
- We have increasing amounts of data and increasingly accurate predictions! Why do we need causal inference? When is more data not enough?

# Do prediction models guide decision-making?



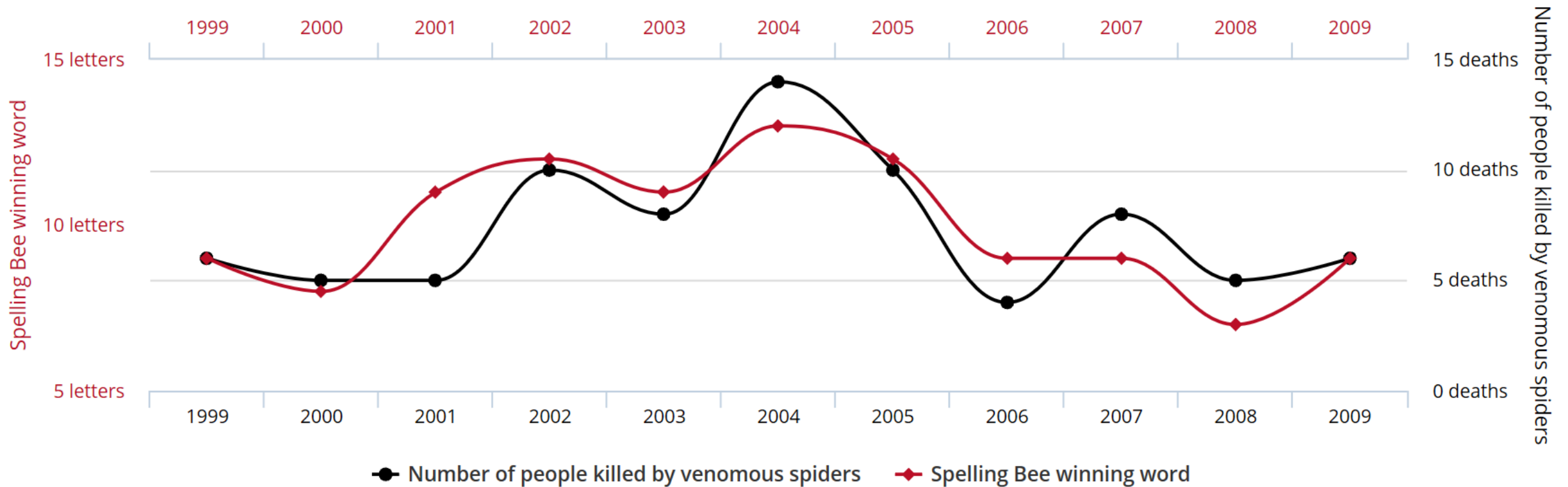
Do we need to regulate ice cream sales?



# Letters in Winning Word of Scripps National Spelling Bee correlates with

## Number of people killed by venomous spiders

Correlation: 80.57% (r=0.8057)



tylervigen.com

Data sources: National Spelling Bee and Centers for Disease Control & Prevention

Source: <http://www.tylervigen.com/spurious-correlations>

# Takeaways

- If it is a spurious correlation without underlying causal connection, we likely won't be able to predict the future well!
- But wait! Wouldn't we be able to predict shark attacks well next summer based on our ice cream sales model?
- Changes in the environment & interventions may cause your predictive model to fail
  - What if we move to southern hemisphere? July is winter now.
- Typically assume that train/test sets are drawn from same distribution in supervised learning - No such guarantee in real life!
- Problematic: Acting on a prediction changes distribution!
  - Echo chamber: Recommend political news – if we start recommending only certain articles, we will see less clicks on other articles in the future, even if someone might have read them if recommended.
  - Incl. critical domains: healthcare or adversarial scenarios



# What if I have a ton of data?



# Big data to the rescue?

- “Look at how much data I had...”
- “How could I be wrong? I used 3 billion data points!”
- “This is just noise. All the problems will cancel out...”
  
- Beware! You do need to worry about bias **and** variance!
- **More data does not help you reduce bias!**
  
- **This week: Sources of bias, how to model it, and what to do about it**

# The Reasonable Uneffectiveness of Big Data

- Play on “The Unreasonable Effectiveness of Data”
  - By Alon Halevy, Peter Norvig, and Fernando Pereira at Google
  - Simple models + Lots of data work very well
- Now consider context of **causal inference**
  - Common threats to causal inference are **independent of sample size** (more details later)
  - When we **cannot observe counterfactuals**, observing more data will not help us! (formal definition coming later)

# Big Data does not address...

...common threats to causal inference, including:

1. **Construct validity**

- E.g. measurement error

2. **Internal Validity**

- E.g. confounding

3. **External Validity**

- E.g. selection effects

# Challenge 1: Construct Validity

- Def: Are you measuring what you think you are measuring?
  - Especially important operationalization of theoretical construct / new “sensor”  
(e.g. social media, linguistic construct such as empathy)
  - Measurement error (e.g. drift in accelerometer sensor)
- How to demonstrate?
  - Convergent validity: Simultaneous measures of same construct correlate
  - Discriminant validity: Doesn't measure what it shouldn't

Big Data typically means little control over how anything was measured

# Challenge 2: Internal Validity

- Def: Soundness of research design
  - Are you able to appropriately answer your research question with the right level of evidence? (e.g., correlational, causal)
- **What potential selection effects / confounding are there?**
  - Is data missing non-randomly?
  - Could measurement be biased across key groups? (e.g. phone steps count for women vs. pockets)
  - Does population change across multiple analyses (complicating comparisons)?

# Internal Validity (cont.)

- How robust are findings across different analytic choices along the way?
  - How robust are results with respect to inclusion/exclusion of outliers?
- How many hypotheses are being tested?
  - May need to control false discovery rate (cf. Bonferroni and Benjamini-Hochberg correction)
- Are distributional / parametric assumptions valid?
  - Consider non-parametric models incl. bootstrapping

Big Data typically means observational data, convenience samples, and no pre-registration

# Challenge 3: External Validity

- Def: Can findings be generalized to other situations and to other people?
- How biased is the study population?
  - Ex: “Internet Explorer users”
  - Ex: “Chrome latest beta users”
  - Ex: “Smartphone owner + health app installed”
  - Convenience samples can be WEIRD, especially motivated, lack key groups of interest, ...

Big Data typically means more data,  
but more of the same!



# Recap: Prediction is insufficient for choosing interventions, more data may not help!

Do they lead us to the right decision? Not necessarily

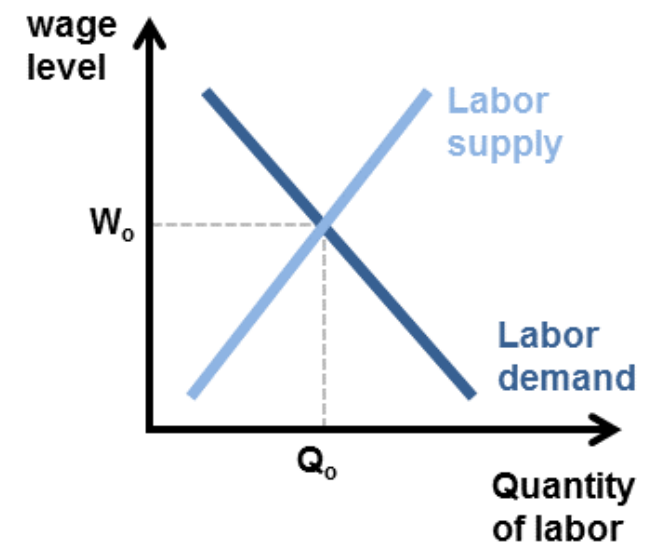
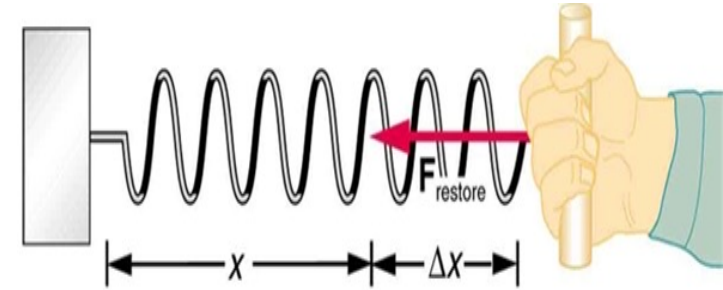
- Unclear, predictive algorithms provide no insight on effects of decisions

**What is causality?**

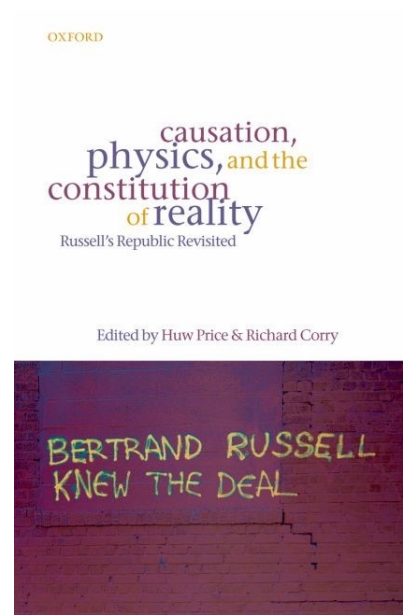
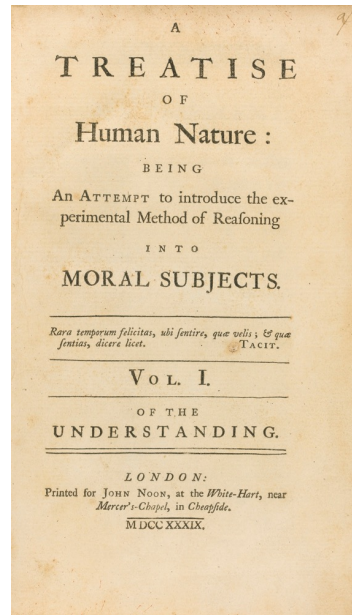
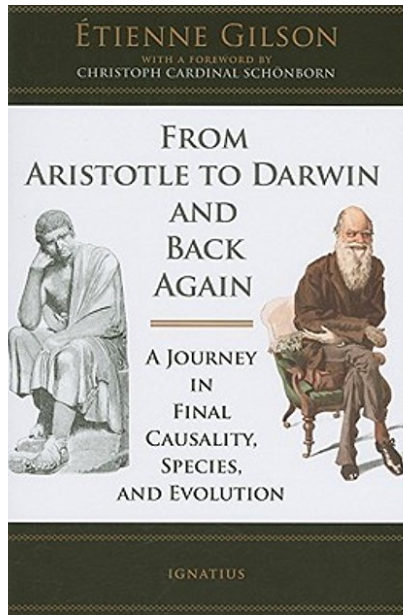
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# Cause and Effect

- Questions of cause and effect common in biomedical and social sciences
- Such questions form the basis of almost all scientific inquiry
  - Medicine: drug trials, effect of a drug
  - Social sciences: effect of a certain policy
  - Genetics: effect of genes on disease
- So what is causality?
- What does it mean to *cause* something?



# A big scholarly debate, from Aristotle to Russell



# What is causality?

- A fundamental question
- Surprisingly, until very recently---maybe the last 30+ years--- we have not had a mathematical language of causation. We have not had an arithmetic for representing causal relationships.

*"More has been learned about causal inference in the last few decades than the sum total of everything that had been learned about it in all prior recorded history."*

--Gary King, Harvard University

# The Three Layer Causal Hierarchy

Pearl, Theoretical Impediments to Machine Learning with Seven Sparks from the Causal Revolution, arXiv:1801.04016v1. 11 Jan 2018

Level	Typical Activity	Typical Question	Examples
1. Association $P(y   x)$	Seeing	What is? How would seeing $X$ change my belief in $Y$ ?	What does a symptom tell me about a disease? What does a survey tell us about the election results?

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2. Intervention $P(y   do(x), z)$	Doing, Intervening	What if? What if I do $X$ ?	What if I take aspirin, will my headache be cured? What if we ban cigarettes?
3. Counterfactuals $P(y_x   x', y')$	Imagining, Retrospection	Why? Was it $X$ that caused $Y$ ? What if I had acted differently?	Was it the aspirin that stopped my headache? Would Kennedy be alive had Oswald not shot him? What if I had not been smoking the past 2 years?



# A practical definition

**Definition:** T causes Y iff  
changing T leads to a change in Y,  
*keeping everything else constant.*

The **causal effect** is the magnitude by which Y is changed by a unit change in T.

T is often binary, but can be categorical, ordinal, continuous.

Called the “interventionist” definition of causality.

\**Interventionist* definition [<http://plato.stanford.edu/entries/causation-mani/>]

# Keeping everything else constant: Imagine a *counterfactual* world

“What-if” questions

Reason about a world that does not exist.

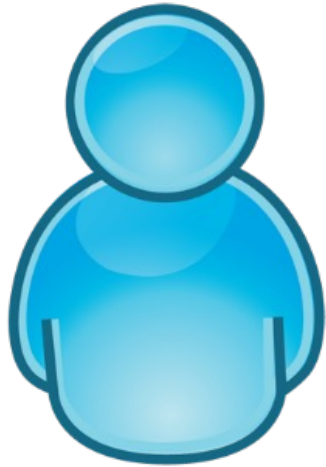


- What if a system intervention was not done?
- What if an algorithm had been changed?
- What if we give a drug to a patient?

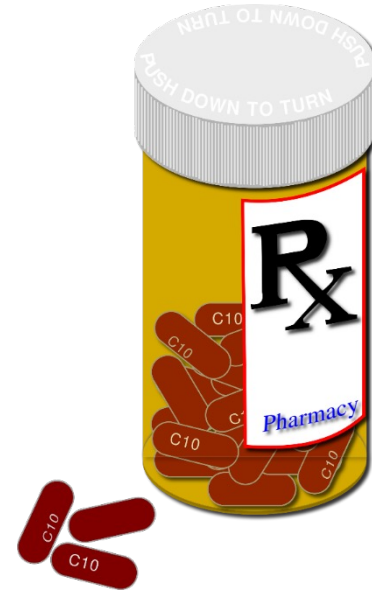
# Potential Outcomes Framework

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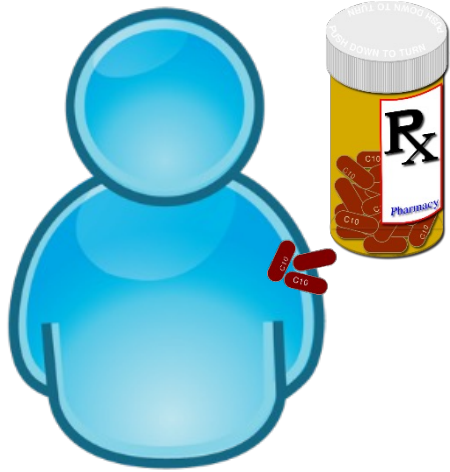


Alice



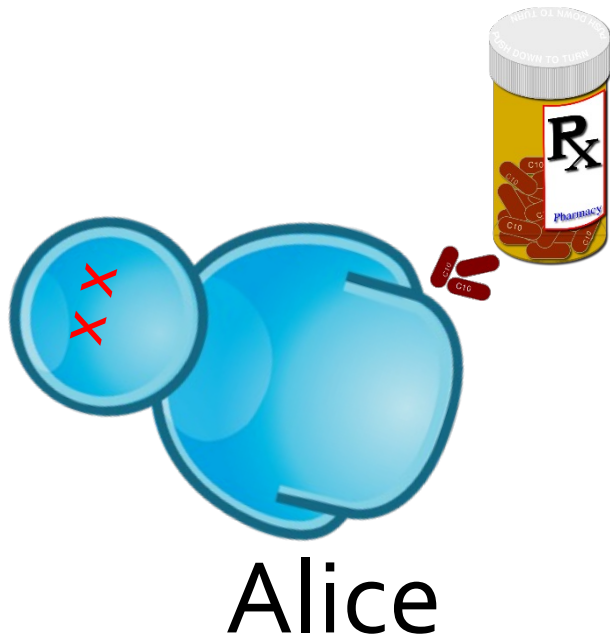
Treatment

# Potential Outcomes framework

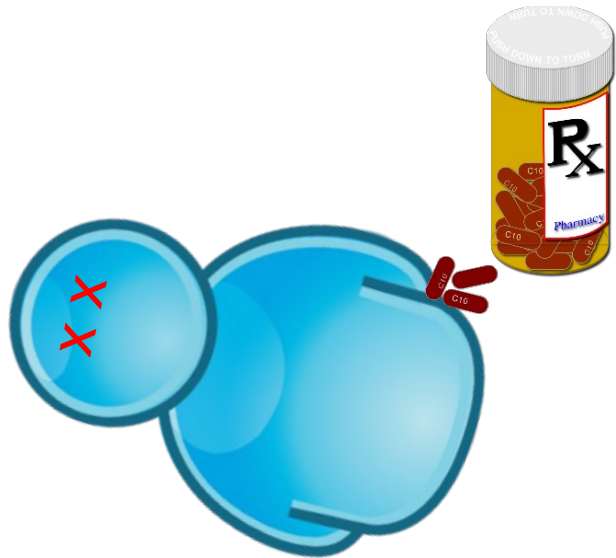


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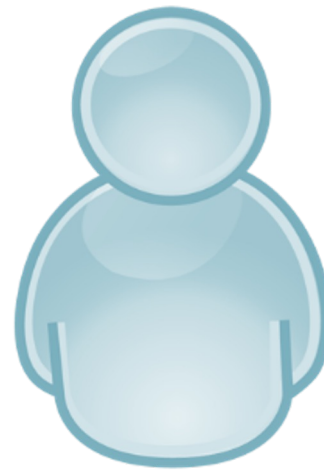
# Potential Outcomes framework



# Potential Outcomes framework: Introduce a counterfactual quantity



$Y_{T=1}$



$Y_{T=0}$



Causal effect of  
treatment =  
 $E[Y_{T=1} - Y_{T=0}]$

Average Treatment Effect (ATE)

# Causal inference is the problem of estimating the counterfactual $Y_{t=\sim t}$

Person	T	$Y_{T=1}$	$Y_{T=0}$
P1	1	0.4	0.3
P2	0	0.8	0.6
P3	1	0.3	0.2
P4	0	0.3	0.1
P5	1	0.5	0.5
P6	0	0.6	0.5
P7	0	0.3	0.1

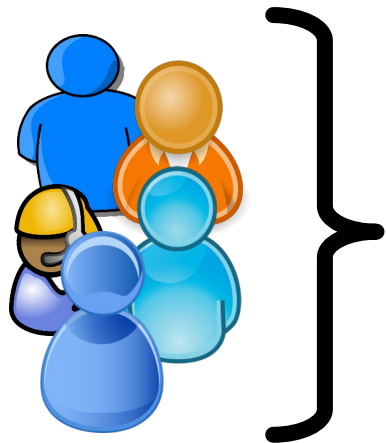
Causal effect:  $E[Y_{t=1} - Y_{t=0}]$


**Fundamental problem of causal inference:** For any person, observe only one: either  $Y_{t=1}$  or  $Y_{t=0}$

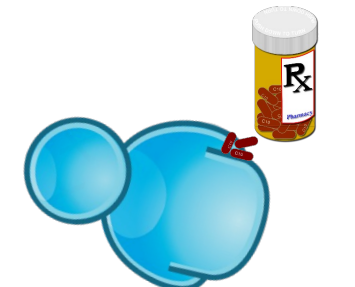


# Fundamental problem: Counterfactual outcome is not observed

- Causal inference is really a “missing data” problem
- Estimate missing data values using various methods
- $Y_{T=0}$  now becomes an estimated quantity, based on outcomes of other people who did not receive treatment

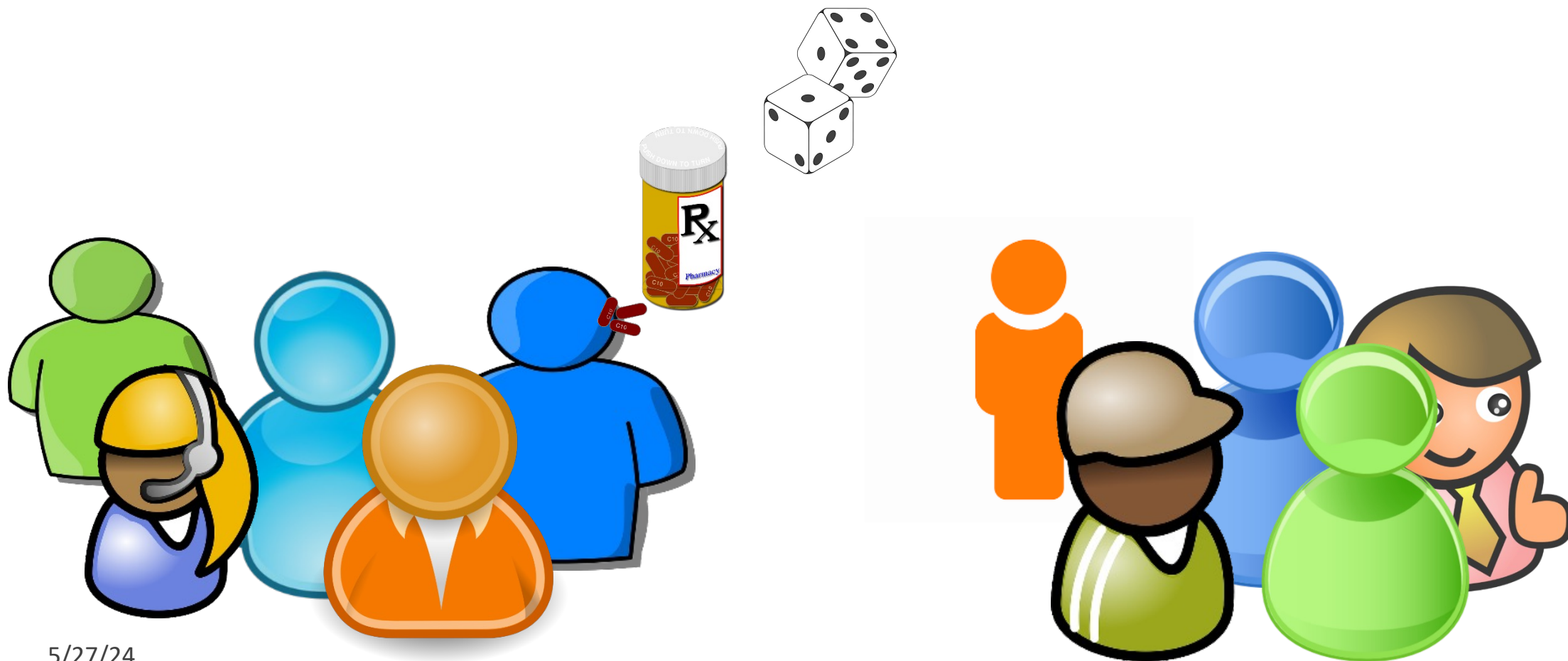



$$\hat{Y}^{T=0}$$


$$Y^{T=1}$$

# Randomized Experiments are the “gold standard”

One way to estimate counterfactual.



5/27/24

Simple: sample mean difference gives you unbiased ATE estimate

# Experiments are not always possible!

In many cases, we cannot randomize / intervene / A-B test (cf. offline evaluation).

- **Practicality:** Exposure to treatment may be hard to manipulate
  - Ex: Environmental effects (air pollution)
- **Ethical concerns:** Known negative effects
  - Ex: Smoking with known negative effect (today known; more on Thu)
  - Extreme Ex: Is suicide contagious?
- **Efficiency:** Experimental science is expensive and takes time
  - Ex: Studying impact on mortality 10 years later
- ...

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In many cases, we cannot randomize / intervene / A-B test (cf. offline evaluation).

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  - Extreme Experiments (e.g., drug trials; more on Thu)
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  - Ex: Studying impact on mortality 10 years later
- ...

What can we do when an experiment is not possible?  
More on Thursday!

# What causal effects might you want to estimate?

- So far: ATE – Average Treatment Effect
  - $E[Y_{T=1} - Y_{T=0}]$ : This is average causal effect across entire population
- ATE could be different on treated vs untreated group
  - Often we care about particular populations!
  - Ex: Hip Surgery -> Walking Ability
    - Doctors are not interested in effect of hip surgery on healthy population. What does it change for someone who has difficulty walking?
- ATT – Average Treatment Effect **on the Treated**
  - $E[Y_{T=1} - Y_{T=0} \mid \mathbf{T}=1]$

# Recap: Potential Outcomes Framework

- **Potential outcomes** reasons about causal effects by comparing outcome of treatment to outcome of no-treatment
- **The Fundamental Problem of Causal Inference:**  
For any individual, we cannot observe both treatment and no-treatment.
- **Randomized experiments** are one elegant solution, but not always possible
  - We'll discuss other solutions on Thursday that rely only on observational data

# Unobserved Confounds / Simpson's Paradox

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# Unobserved Confounds

- Which treatment should a doctor recommend for kidney stones?
- **Simpson's paradox:** After accounting for the confounder (stone size) the best choice reverses.
- Critical for decision making

Treatment A	Treatment B
78% (273/350)	83% (289/350)

Charig et al., BMJ 1986



# Questions for you 😊

- What explains this “paradox”? Concretely, why did treatment B look so effective?
- What could researchers have done to fix this at the time?

	Treatment A	Treatment B
Small stones	<b>93%</b> (81/87)	87% (234/270)
Large stones	<b>73%</b> (192/263)	69% (55/80)
Both	78% (273/350)	<b>83%</b> (289/350)

Charig et al., BMJ 1986

# Recap: Unobserved Confounds

- Unobserved confounds are a threat to causal reasoning and to decision making

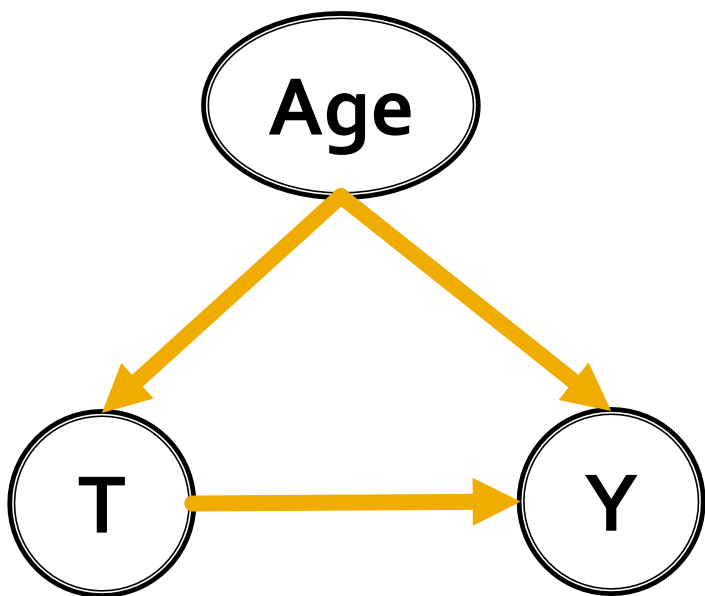
# Structural Causal Model Framework

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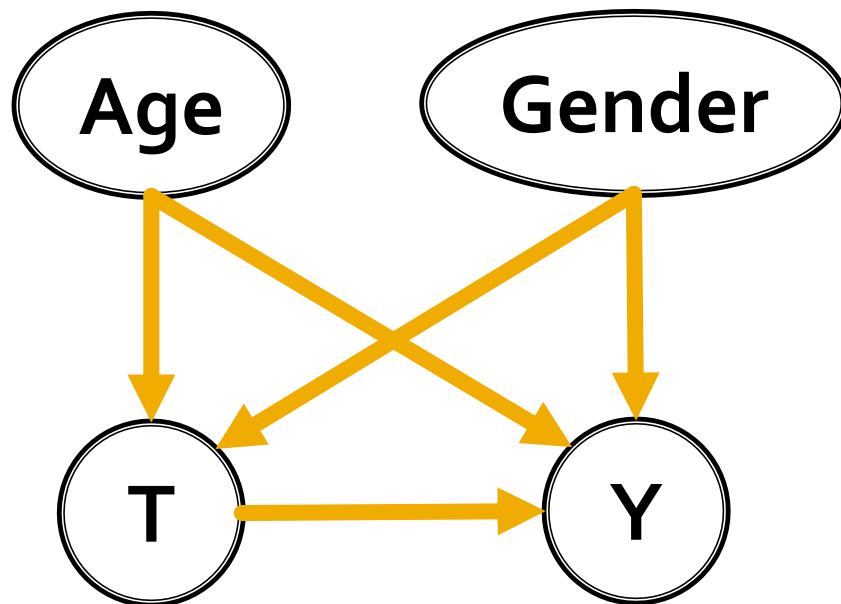
# The real world is complicated

- We observed that accounting for kidney stone size was critical
- Many other factors might influence the observed outcome
  - How do they affect treatment and outcome?
  - Which ones to include?
- How to we formalize all of our **assumptions**?
  - Causal inference cannot be done with data alone. It requires making assumptions about the world.
- How to **identify** the causal effect in such cases?
  - The task of causal **identification** is to determine an expression, the causal estimand, that expresses our target value as a function of the observable correlational relationships in our system.
  - We can use graphical model framework to answer this

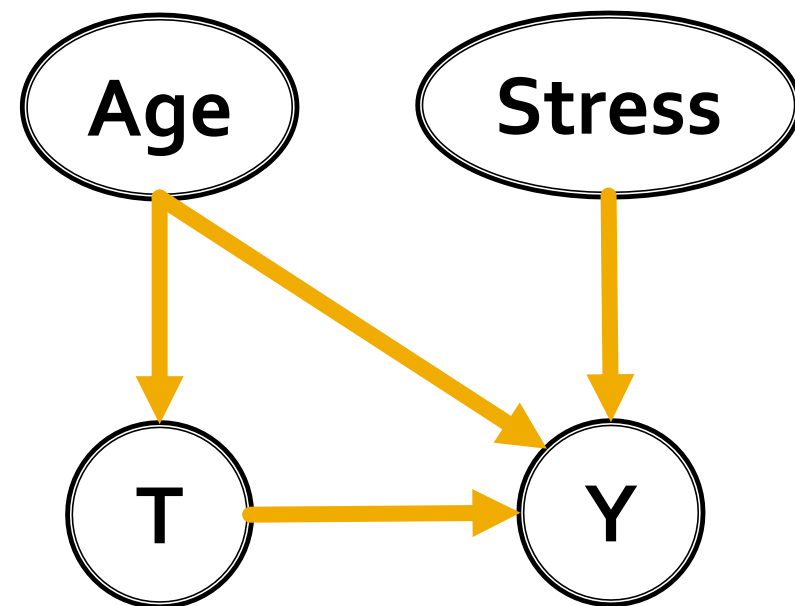
# Which variables to condition on?



$$X = \{Age\}$$

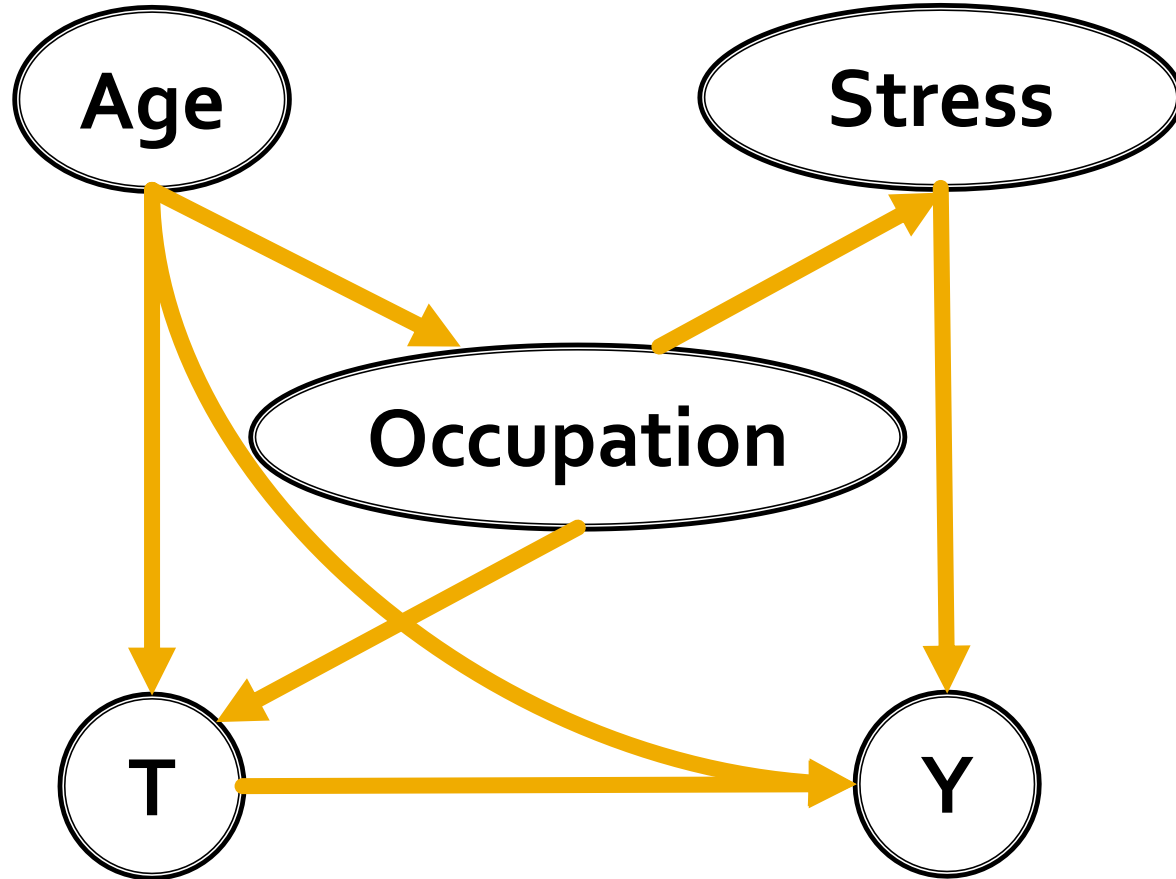


$$X = \{Age, Gender\}$$

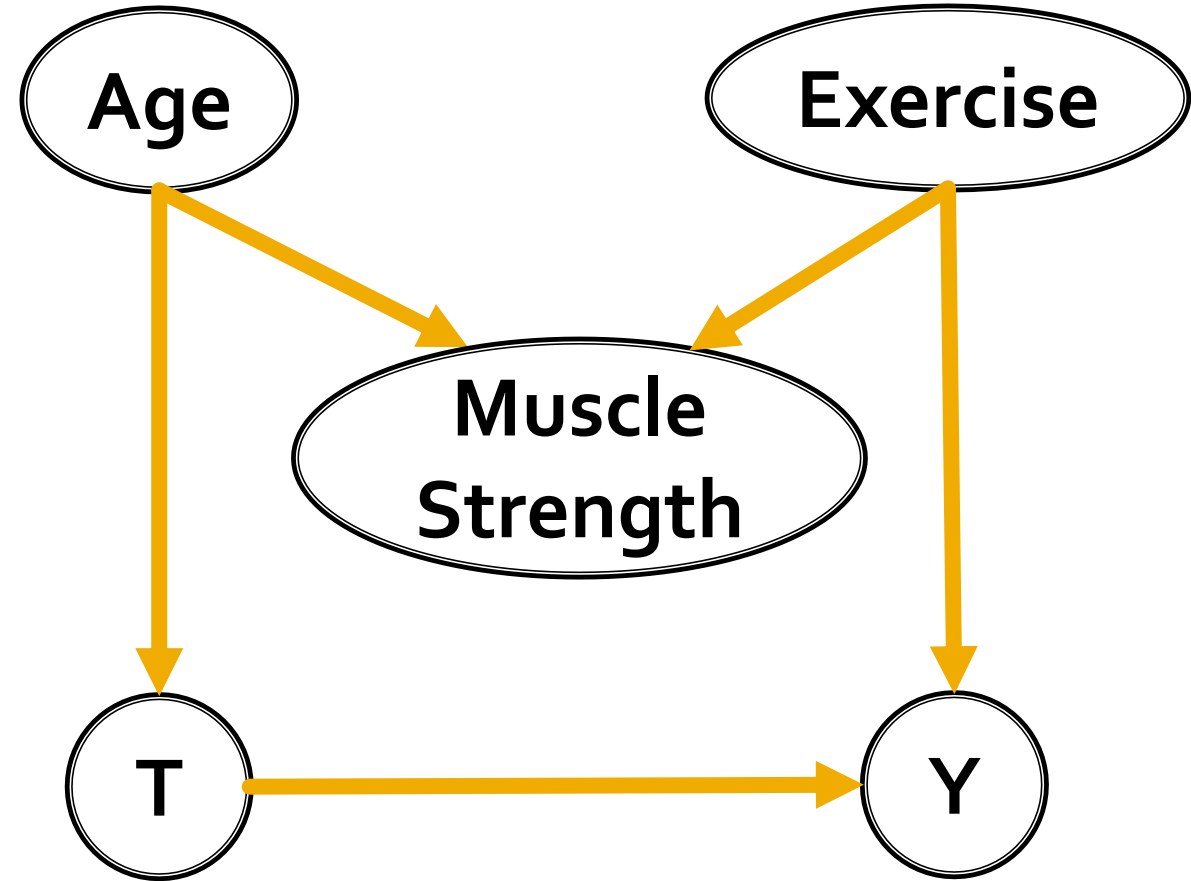


$$X = \{Age\}$$

# What about these?

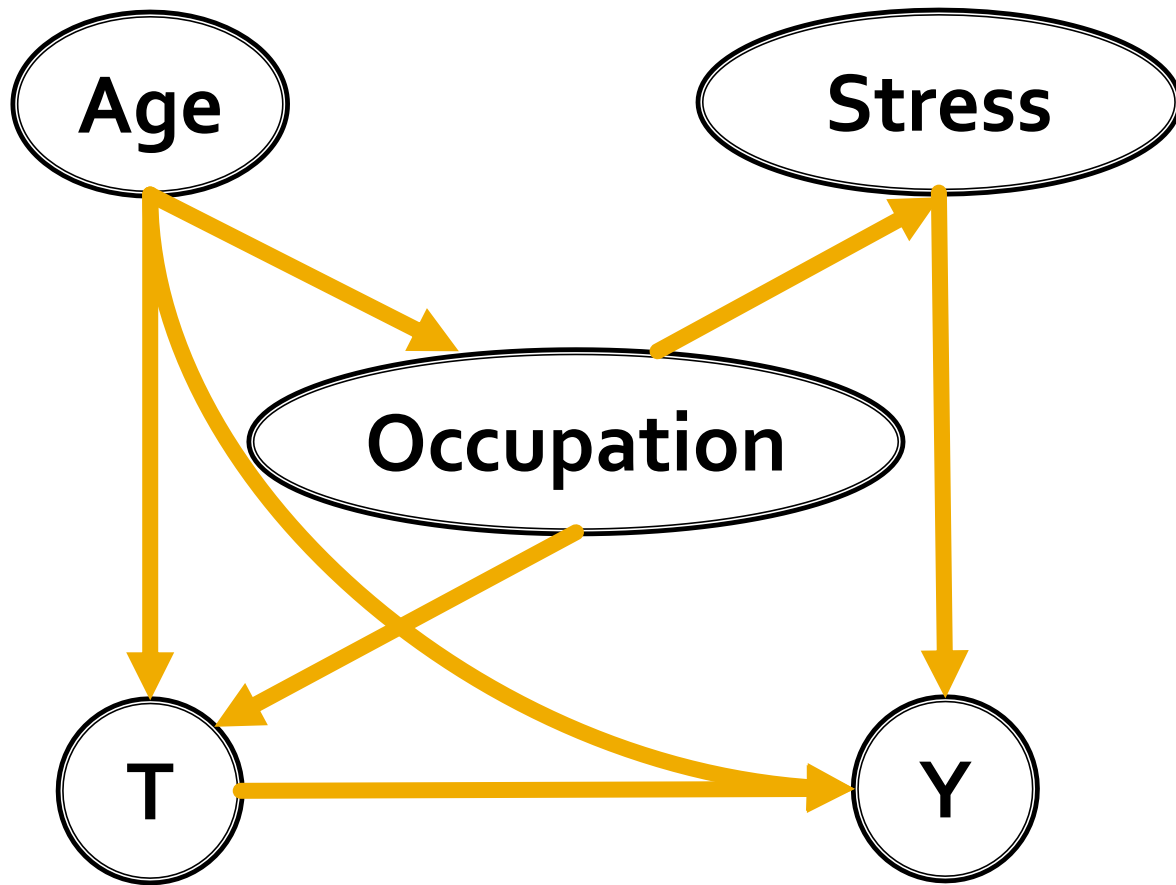


$X = ?$



$X = ?$

# Structural Causal Model: A framework for expressing complex causal relationships



Structural Equation Models with Random Errors  $u$ 's are "error variables" or "exogenous variables"

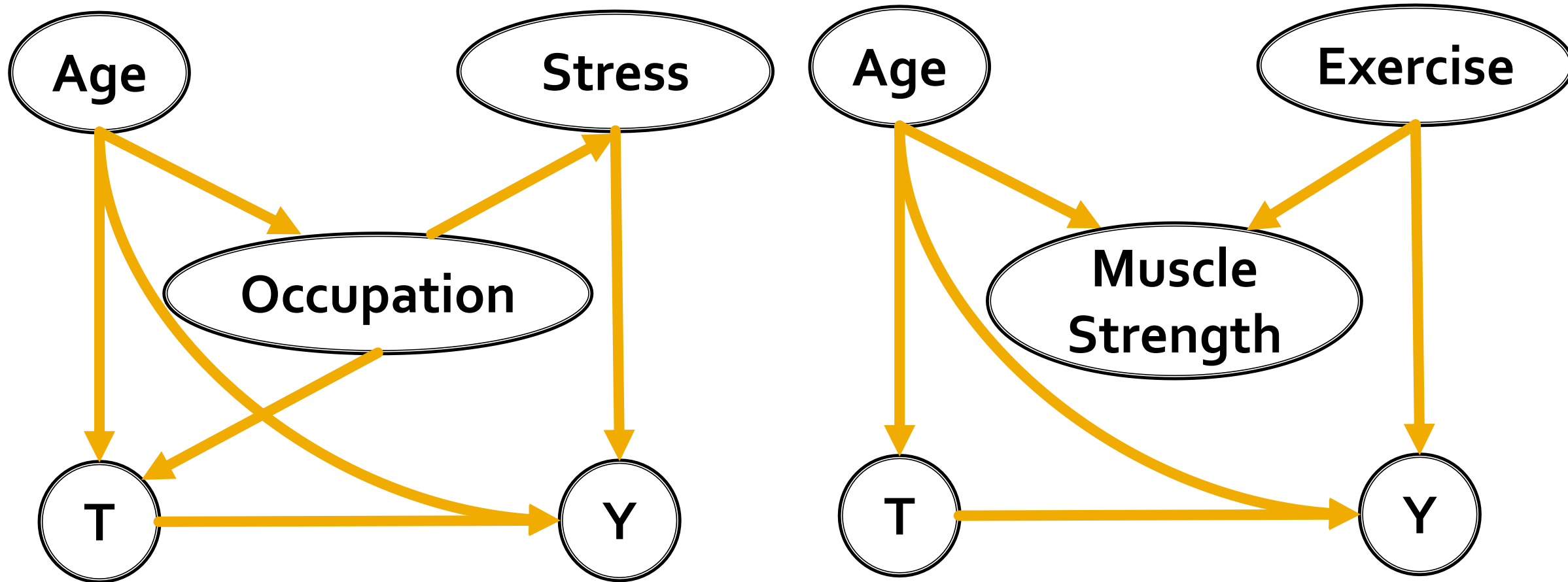
$$\begin{aligned} \textit{Occupation} &= h(\textit{Age}, u_o) \\ \textit{Stress} &= k(\textit{Occupation}, u_s) \end{aligned}$$

$$\begin{aligned} T &= g(\textit{Age}, \textit{Occupation}, u_t) \\ Y &= f(T, \textit{Age}, \textit{Stress}, u_y) \end{aligned}$$

Edges represent *direct* causes.

Directed paths represent *indirect* causes.

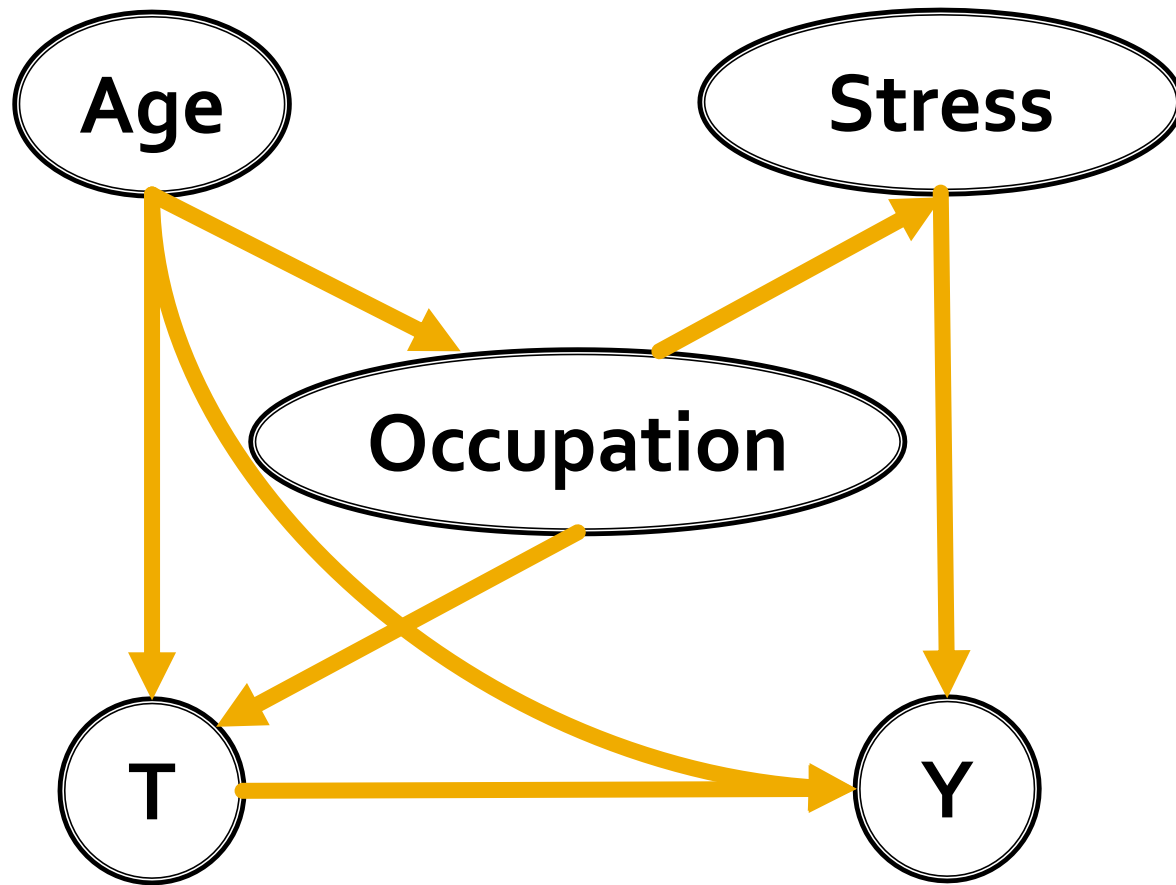
# Structural Causal Model makes assumptions explicit



**The graph encodes all causal assumptions.**



# Important: Assumptions are the edges that are *missing*



**Assumption 1:** Occupation does not directly affect outcome Y.

**Assumption 2:** Age does not directly affect stress.

**Assumption 3:** Stress does not directly affect Occupation.

**Assumption 4:** Treatment does not directly affect stress.

*..and so on.*

**Condition for validity:** The graph reflects all relevant causal processes.

# Key Benefit (1) of SCM: Provides a language for expressing counterfactuals

*If a person was given treatment, what is the probability that he would be cured if he was not given treatment?*

$$P(Y = 1|T = 1, T = 0)$$

**Non-sensical.**

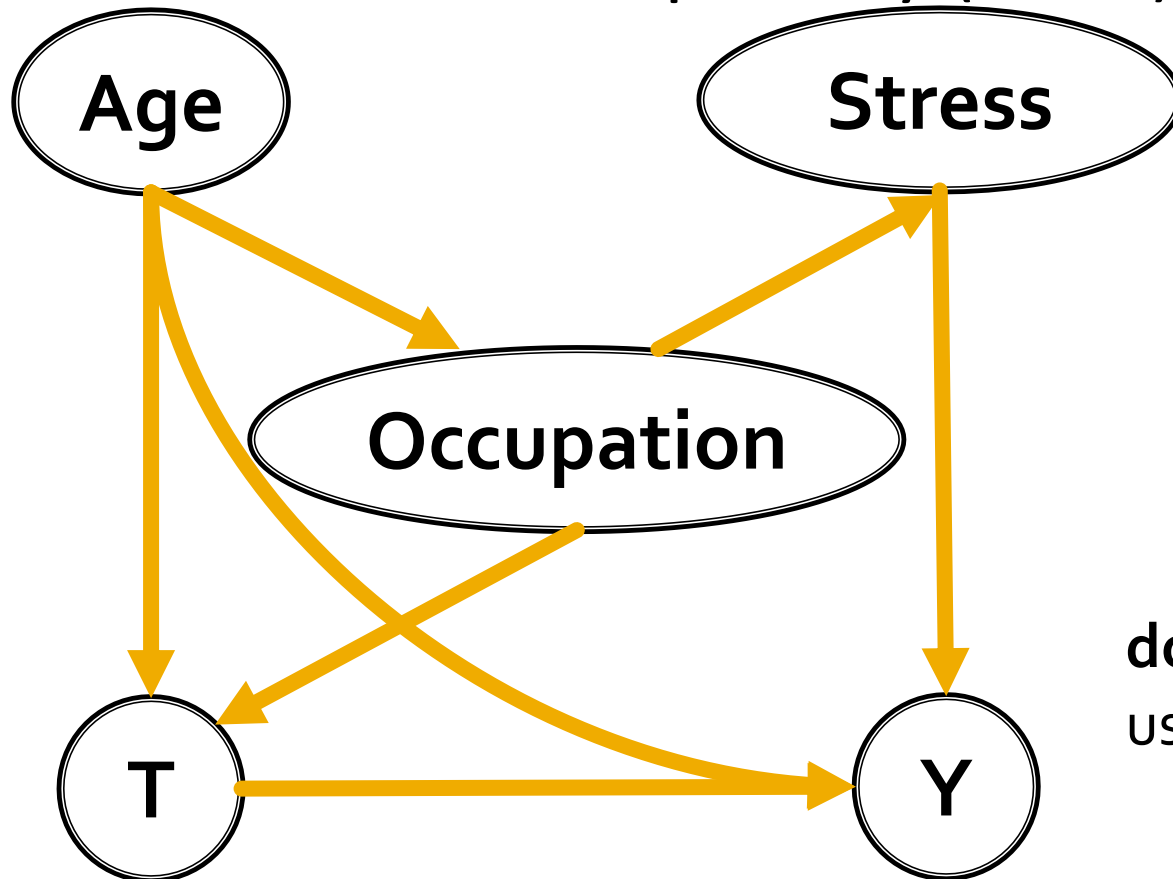
*Can write it as:*

$$P(Y_{T=0} = 1|T = 1), \text{ or} \\ P(Y = 1|T = 1, do(T = 0))$$

$P(Y|do(T))$  avoids confusion with  $P(Y|T)$

# Key Benefit 2 of SCM: Provides a mechanistic way of identifying causal effect

**do-calculus:** A rule-based calculus that can help identify any counterfactual quantity (Pearl)

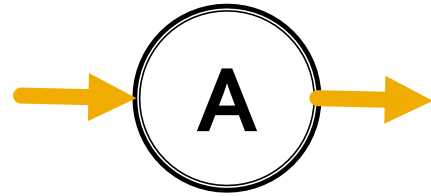


E.g.,  
 $P(Y|do(T))$   
 $= \dots do\text{-calculus rules} \dots$   
 $= \sum_x P(Y|T, X = x) P(X = x)$

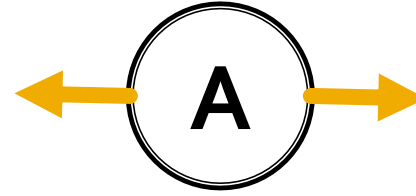
**do-calculus is complete:** If we cannot identify using do-calculus, causal effect is unidentifiable.

# Advanced Topic: Back-door criterion

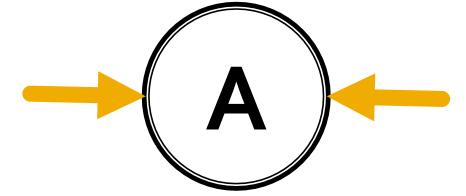
Three kinds of node-edges  
Path is “blocked”



If conditioned on A



If conditioned on A



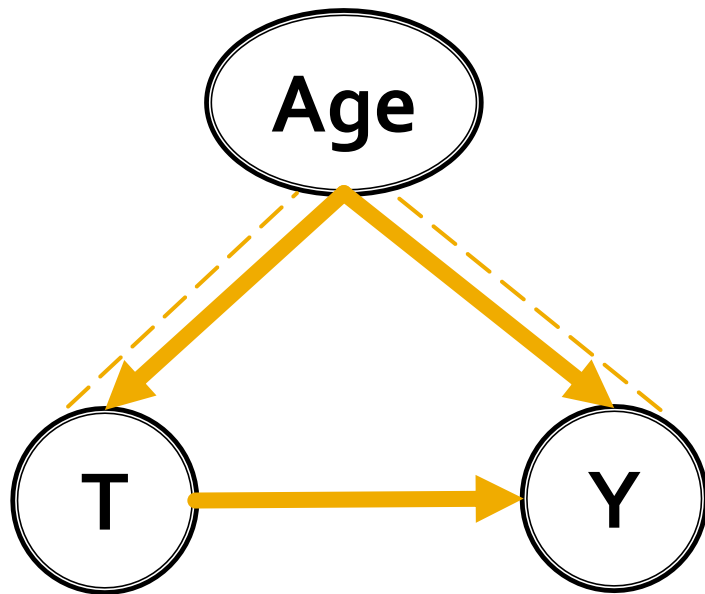
If **not** conditioned on A

“Back-door” path: Any undirected path that starts with  and ends with 

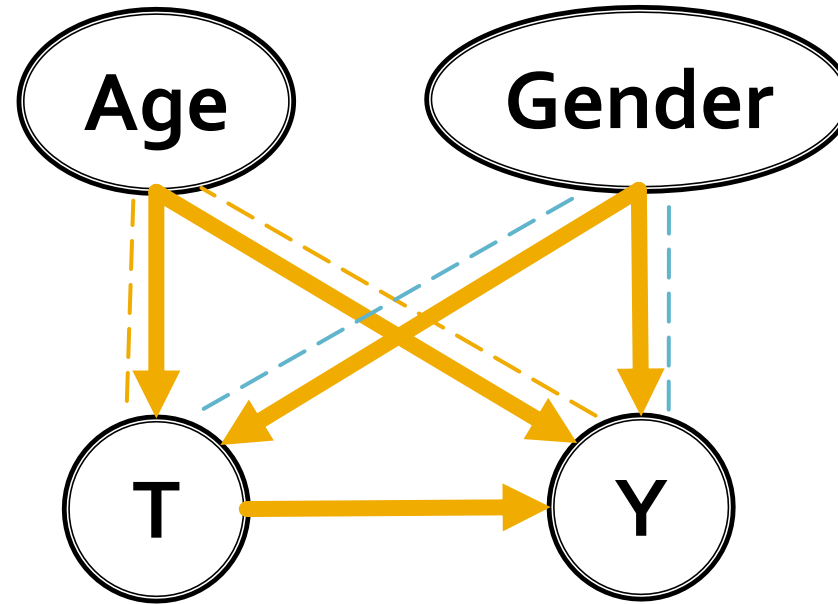
**Back-door criterion:** If conditioning on  $X$  blocks all back-door paths between treatment  $T$  and outcome  $Y$ , and  $X$  does not include any descendants of  $T$ , then

$$P(Y|\mathit{do}(T)) = \sum_x P(Y|T, X = x)P(X = x)$$

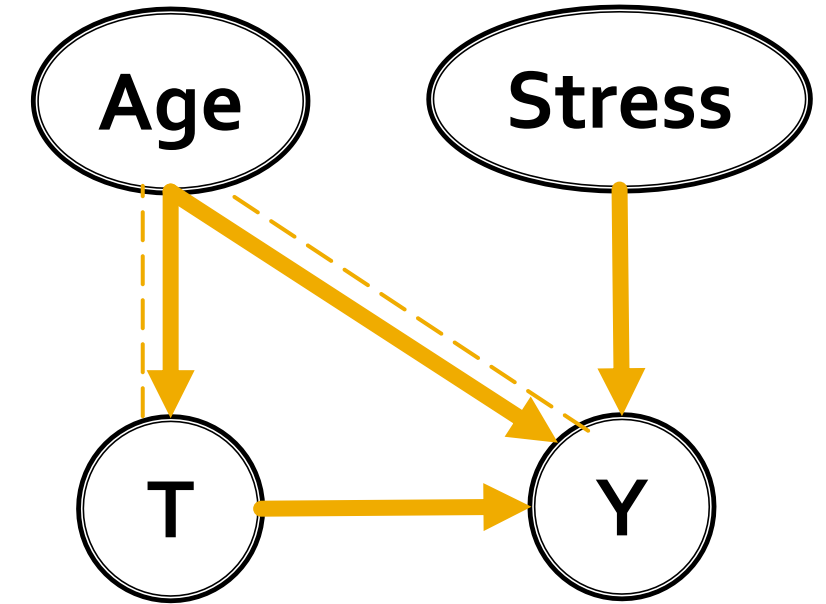
# Back-door criterion provides a precise way to find the set of variables to condition on



$$X = \{Age\}$$

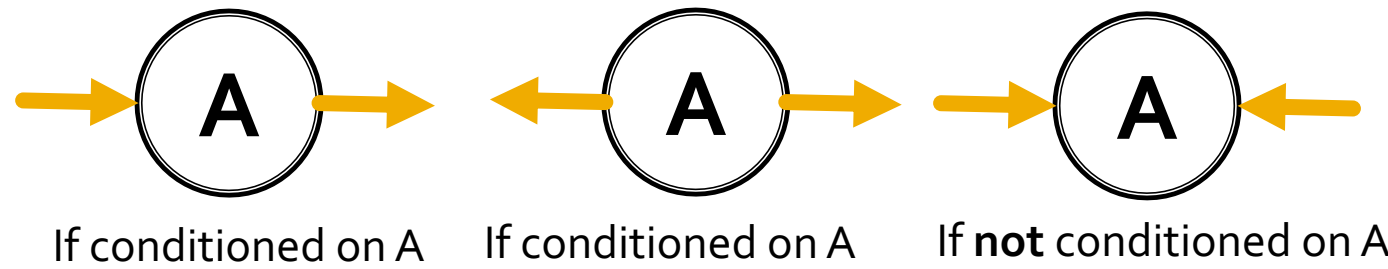


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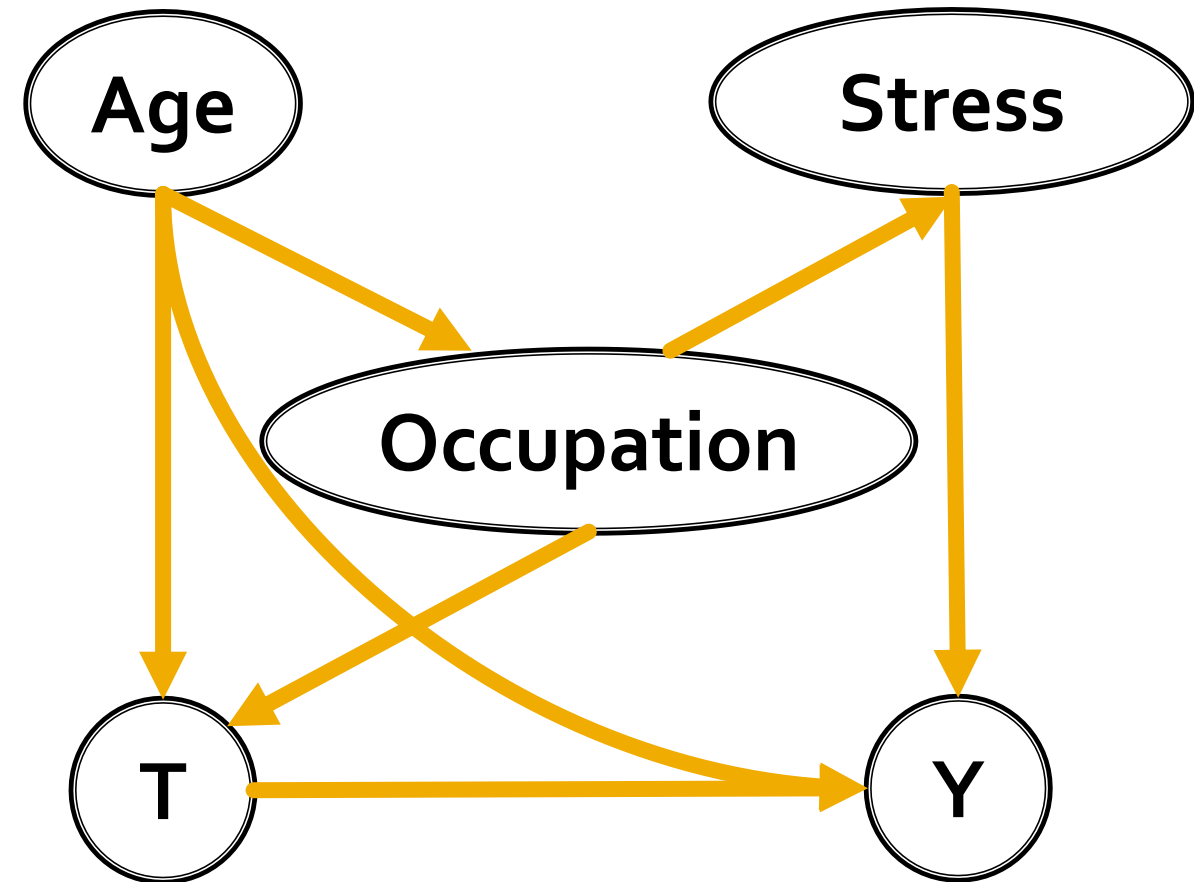


$$X = \{Age\}$$

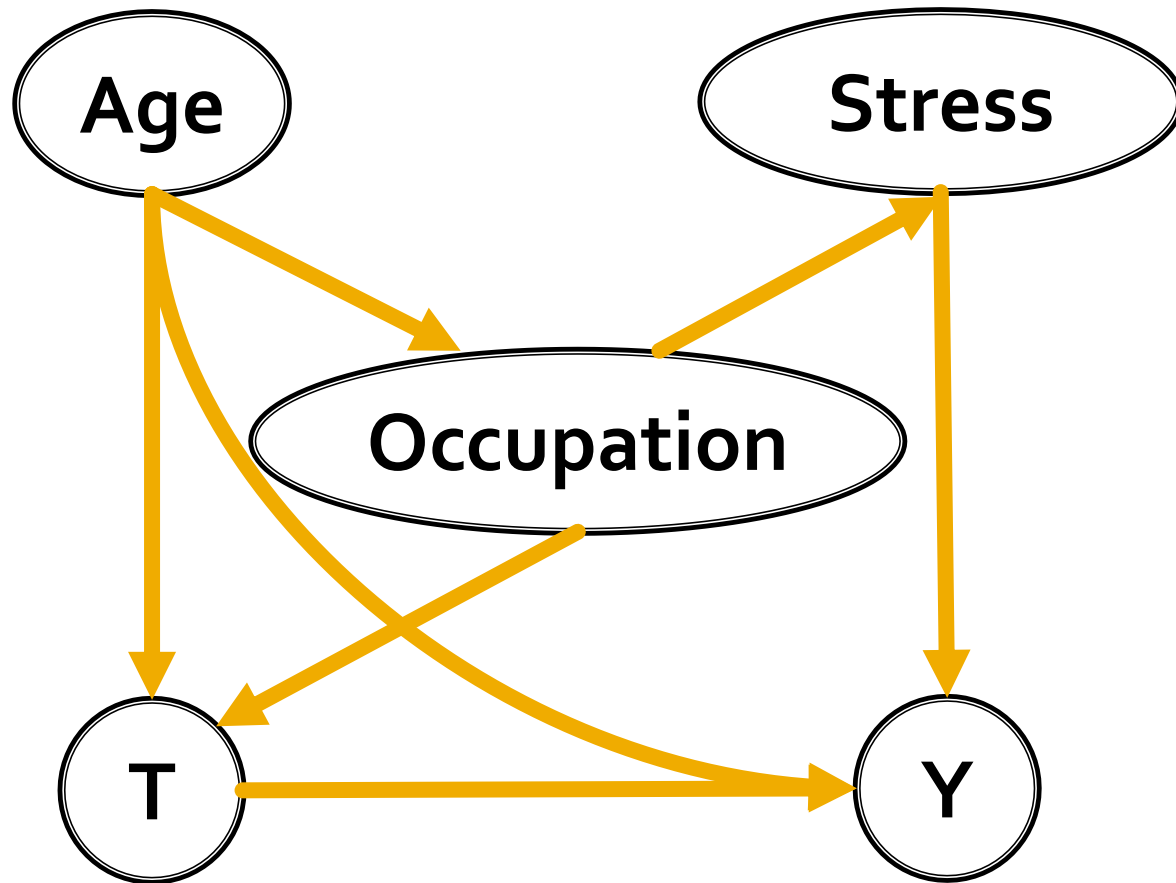
# Now it's your turn!



- Find a partner close to you.
  - If watching recording: I invite you to pause now and try it out.
- **Q:** What variables do we need to condition on to block all backdoor paths?
- **Too easy?** Find all such sets



# Correct Answer

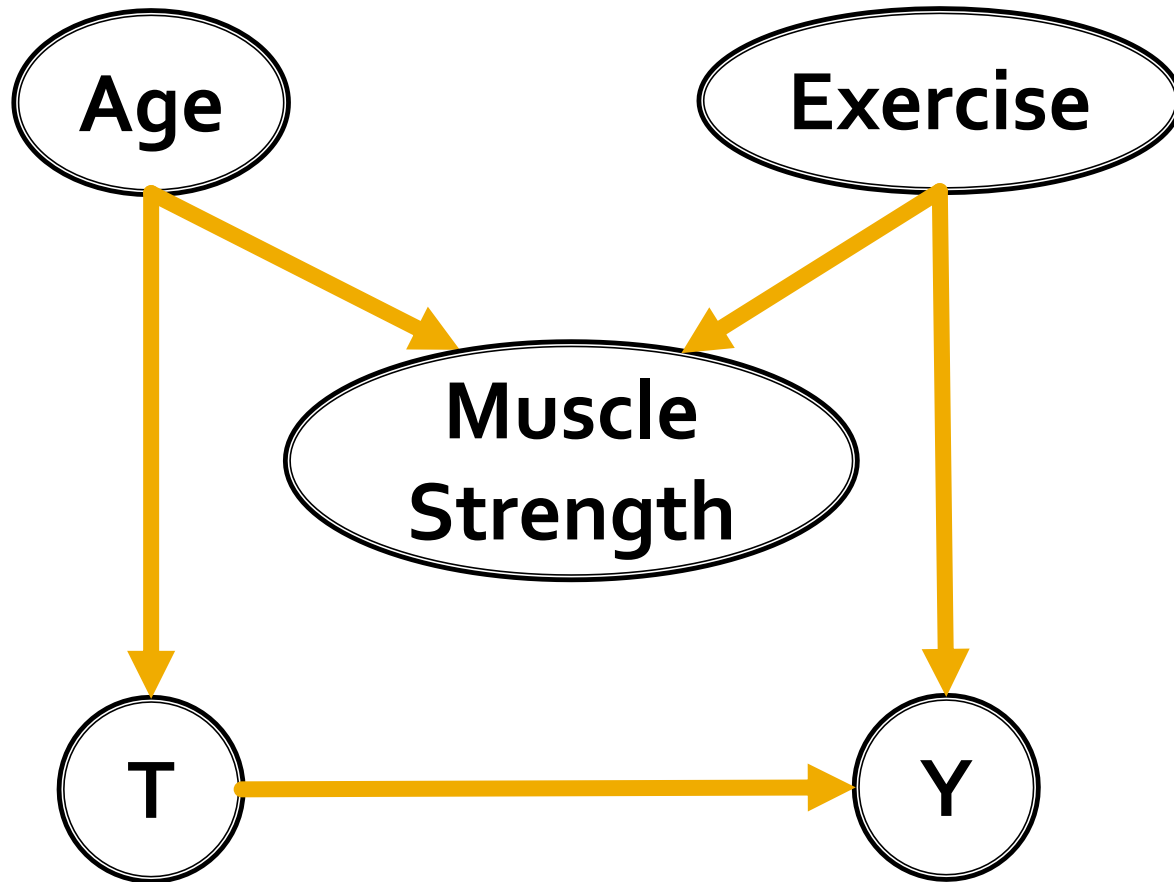


$X = \{\text{Age}, \text{Stress}\}$

$X = \{\text{Age}, \text{Occupation}\}$

$X = \{\text{Age}, \text{Stress}, \text{Occupation}\}$

# Next example:



$X = \{\}$  – Muscle Strength is a collider!

$X = \{\text{MuscleStrength, Exercise}\}$

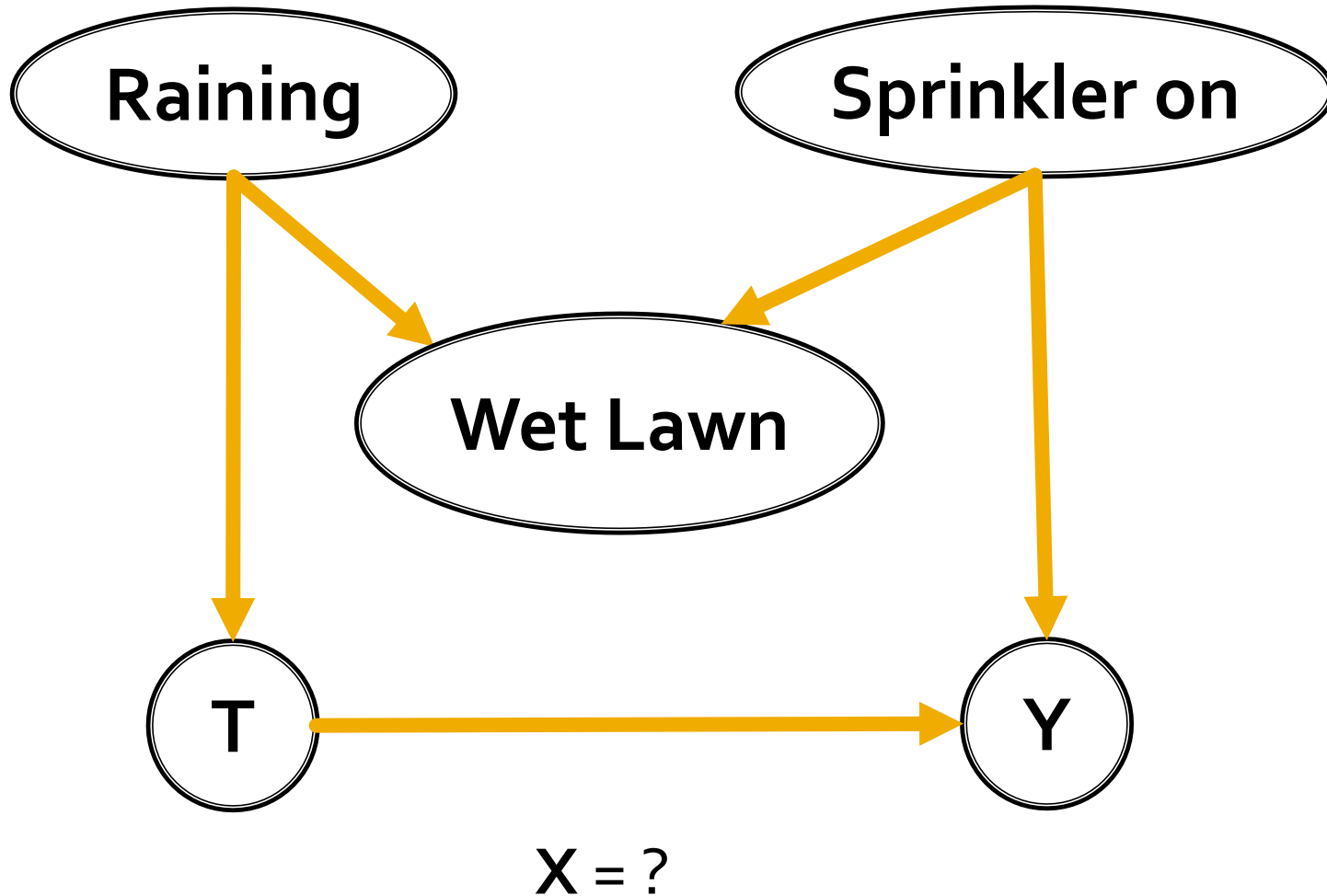
$X = \{\text{MuscleStrength, Age}\}$

$X = \{\text{MuscleStrength, Age, Exercise}\}$

It is important to know about colliders! **Controlling for more variables can even hurt!**



# Special Case: Colliders



Q: Why are colliders special and we should not condition on them to block the backdoor path?

A: If you know that the lawn is wet, but sprinkler is not on. Now you know it must be raining.

**Conditioning on collider induces correlation between parent nodes.**

# Why is computing conditional probability not enough?

- **Back-door criterion:** If conditioning on  $X$  blocks all back-door paths between treatment  $T$  and outcome  $Y$ , and  $X$  does not include any descendants of  $T$ , then

$$P(Y|do(T)) = \sum_x P(Y|T, X = x)P(X = x)$$

- **This is a correct, unbiased estimator. But it can be so inefficient, essentially requiring “infinite” data, that it loses all practical utility.**
  - This happens when  $X$  is high-dimensional and/or incl. continuous variables
  - Example: Say  $X$  is lots of sentences in the English Language and we want to identify the causal effect of my advising on how well my students are doing?
  - Example:  $X$  is your entire genome. How often will you observe this?
  - Example:  $X$  is continuous dosage amount of a drug
- More on Thu about effective estimation techniques
  - We will carefully make **bias variance trade-offs** that allow for more efficient estimation

# Let's do the course evaluation right now

- <https://uw.iasystem.org/survey/290650>
  - Also available from Ed link.
- We take your feedback very seriously. It makes a difference!
- Consider sharing what you liked about the course. What is working well? What should we keep?
- Thank you for participating!

# Recap: Structural Causal Models

- Allow us to make causal assumptions explicit
  - Assumptions are the *missing* edges!
- Provide language for expressing counterfactuals
- Well-defined mechanisms for reasoning about causal relationships
  - E.g., Backdoor criterion

# Both PO & SCM frameworks have merits

Use **structural causal model** and **do-calculus** for

modeling the problem

making **assumptions** explicit

**identifying** the causal effect

Use **potential outcomes-based** methods for

**estimating** the causal effect

# Recap of today:

- **Causality** is important for decision-making and study of effects
- **Big Data** does not necessarily address threats to causal inference
- **Potential Outcomes Framework** gives practical method for estimating causal effects. Translates causal inference into counterfactual estimation
- **Unobserved confounds** are a critical challenge – what might we miss?
- **Structural Causal Model Framework** gives language for expressing and reasoning about causal relationships: Causal **Identification**
- **On Thursday:** Methods for efficient **Estimation** of causal treatment effects in observational data

# Additional Resources

- Slides based in part on KDD 2018 Tutorial by Emre Kiciman and Amit Sharma: <http://causalinference.gitlab.io/kdd-tutorial/>
- Courses
  - UW Econ 488: Causal Inference
  - [UW Stat 566: Causal Modeling](#)
- Books
  - Pearl. Book of Why
  - Rosenbaum. Design of Observational Studies
  - Kiciman & Sharma. <https://causalinference.gitlab.io/> (free, in-progress)

Please give us feedback 😊  
<https://bit.ly/CSE547feedback2024>