



#### NOW...Scientific Evidence on Effects of Smoking!





#### Hey kids... don't smoke

#### R. A. Fisher on smoking and lung cancer (in 1957)

... the B.B.C. gave me the opportunity of putting forward examples of the two classes of alternative theories which **any statistical association**, **observed without the predictions of a definite experiment**, allows--namely, (1) that the supposed effect **is really the cause**, or in this case that incipient cancer, or a pre-cancerous condition with chronic inflammation, is a factor in inducing the smoking of cigarettes, or (2) that cigarette smoking and lung cancer, though not mutually causative, are **both influenced by a common cause**, in this case the individual genotype

### **Graphical notation for causality**



Variables: vertices (or nodes)

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Relationships: directed edges (arrows)

Shaded node / dashed edges: unobserved variable



### Fisher: correlation is not causation

He did not use graphical notation like this

But the graphs can be very useful

# Explaining an observed correlation

We find a statistically significant correlation between X and Y

What does it mean?

- 1. False positive (spurious correlation)
- 2. X causes Y
- 3. Y causes X
- 4. Both have common cause U [possibly unobserved]

Statistically indistinguishable cases (without "experimental" data)

Importantly different consequences!

# A simple mathematical model of causality

Think about interventions that change some target variable  ${\cal T}$ 

- Forget about the arrows pointing into T (intervention makes them irrelevant)
- Change T, e.g. setting it to some arbitrary new value T=t
- This change propagates along directed paths out of T to all descendant variables of T in the graph, causing their values to change

All of these changes could be deterministic, but most likely in our usage they are probabilistic

**Exercise**: in each of these cases, if we intervene on X which other variable(s) are changed as a result?



Y

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#### **Computing counterfactuals**

X

If we know/estimate *functions* represented by edges, we can simulate/compute the consequences of an intervention



#### Causal inference: much more difficult

Predictive machine learning is about

 $p_{Y|X}(y|x)$ 

and regression--conditional expectation, conditional quantile, etc. If we passively observe some value of x, what would we observe about y?

*Causal inference* is about (various notations)

$$p(y| ext{do}[X=x]), \quad ext{i.e.} \quad p(y|X\leftarrow x)$$

i.e. what happens to  $\boldsymbol{Y}$  when we actually intervene on  $\boldsymbol{X}$ 

If we actively change x, what would we observe about y?

#### Experiments

Actually do interventions while collecting data

#### **Observational studies**

Try to infer causal relationships without interventions, by using dark arts more/specialized assumptions/methods that require careful interpretation

Scientific progress: be wrong in more interesting/specific ways

#### Potential outcomes: another causal framework

Relative strengths/weaknesses compared to DAGs

- Narrow focus: goal is to estimate one edge in a graph
- Difficult to express more complex relationships

#### This is not a course on causal inference

Covering a few basics for interesting connections to ML!

So when is ML (and e.g. regression) useful for causal inference?

# Idea: adjusting for confounders

**Confounders**: other variables that obscure the (causal) relationship from X to Y, e.g.

- *Y*: health outcome
- *X*: treatment dose
- *Z*: disease severity

Without considering Z, it might seem like larger doses of X correlate with worse health outcomes

#### Solution: add more variables to the model

Make model complex enough to capture important factors

Similarly, might need to model *non-linear causal relationships* 

#### Strategy: two staged regression

Two-stage least squares (2SLS)

Suppose we want to learn the causal relationship of D on Y, but

 $Y = D heta + Xeta + arepsilon_Y$  $D = Xlpha + arepsilon_D$ 

In words: X is confounding the relationship

- First stage: regress out X
- Second stage: using residuals from first stage,

regress  $Y - X \hat{eta}$  on  $D - X \hat{lpha}$ 

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# Powerful, intuitive idea Orthogonal projection We'll come back to this

(Think about fitting the relationships using ML instead of regression)

## Propensity

General theme: applying ML strengths to causality

Special case: if the "treatment" (causal) variable is categorical

Many causal methods for this case involve predicting the treatment itself

i.e. prediction with categorical outcome, **classification** 

We'll also come back to this (propensity methods) after covering classification

# Summary: a key ingredient in many causal inference methods involves classification, can leverage ML tools

# Guiding ideas / warnings

• More complex models (ML) do not guarantee better causal inference

Might even make things worse (just like with prediction)

• Models with better predictions may be worse for causal inference

Even if prediction accuracy is measured on test data!

• Inference = causal inference...?

Or, at least, causal interpretations can be special case of tradeoff between prediction and inference/interpretation

# **Causal inference**

## An exciting interdisciplinary field

#### Practically important, connections to ML

"Data scientists have hitherto only predicted the world in various ways; the point is to change it" -Joshua Loftus

Mixtape... remix? 🤔

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