Questions and Answers

Macartan Humphreys, Honors Seminar

Design overview

Outline

- We will talk about what a research design *is* and what its main components are
- We will recap (or introduce) key ideas on:
  - causal inference and hypothesis development and testing
  - qualitative inference
- This lecture will take place over 2 days (Day 1 likely up to beginning of "Answer strategies")

MIDA

The four elements of a design:

- **M** Your model. How you think the world works
- **I** Your inquiry: The question you want to ask of the world
- **D** Your data strategy: how you plan to gather data from the world: this includes interventions and measurement
- **A** Your answer strategy: the conclusions you will draw once you have data

If you can define a MIDA design then you have provided enough information to assess the quality of your design.

MIDA

We are going to talk about:

- Models
- Inquiries
- Quantitative data and answer strategies
- Qualitative data and answer strategies

Models: Nodes and graphs

Types of claims

$$X \to Y$$

Types of claims

$$X \rightarrow Y$$

- Analytic claims: e.g. $X = 1$ *implies* $Y = 1$
- Descriptive claims: e.g. $Y = 1$ when $X = 1$
- Causal claims: e.g. $X = 1$ causes $Y = 1$

We mostly focus on causal claims. Even claims we think of as descriptive are often causal claims.

What are X and Y?

$$X \to Y$$

- **Names for** X: independent variable, explanatory variable, input, exogeneous variable, cause, driver, right hand side variable
- **Names for** Y: dependent variable, output, outcome, endogenous variable, left hand side variable
- Both implicitly have a location and a timestamp "$Y = 1 \Leftrightarrow$ `The US was a democracy in 2000`"

What's the question?

- $X \rightarrow Y$
- $? \rightarrow Y$
- $X \rightarrow ?$
- $? \rightarrow ?$

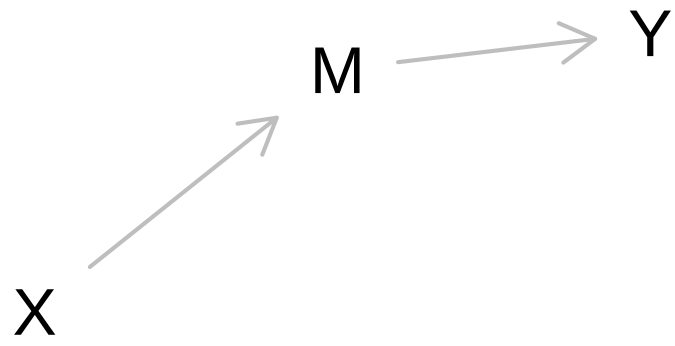
Population and case level claims:

- Does X affect Y in general? (effects of causes)
- Did $X = 1$ cause $Y = 1$ in this case? (causes of effects)

Other types of variables

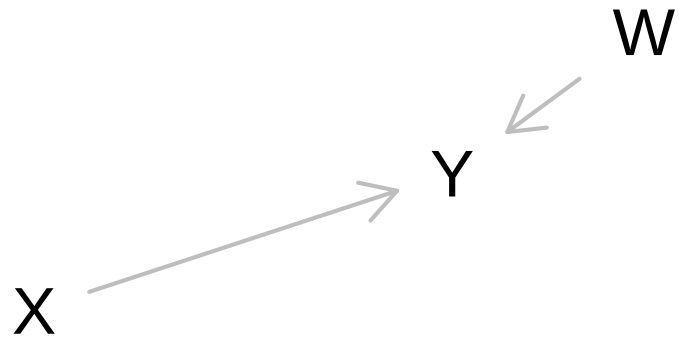
1. mediating variables
2. conditioning or moderating variables
3. confounding variables
4. instrumental variables

Mediating variables



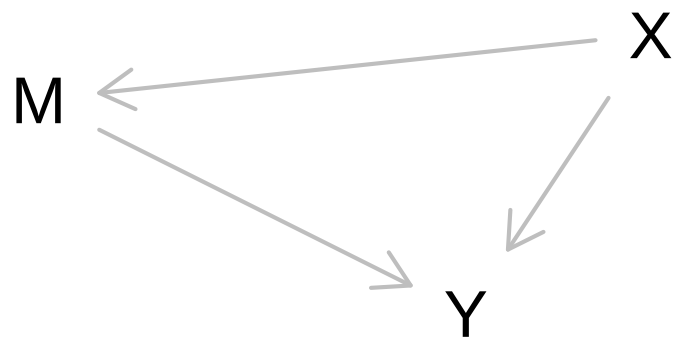
- "Oil wealth produces grievances which cause conflict"
- We often say: "M is a mechanism through which X causes Y.

Conditioning or moderating variables



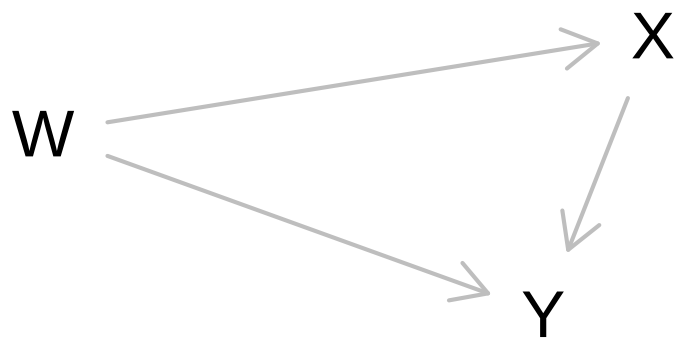
- e.g. “The effect of oil wealth on conflict is weaker when institutions are strong”

Variables might mediate *and* moderate



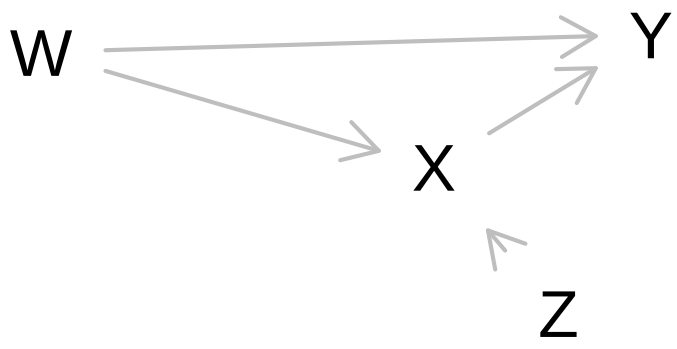
- "Oil wealth produces grievances which cause conflict"
- "There are many other channels through which oil wealth affects conflict and which exacerbate the effects of grievances"

Confounding variables



- "The effect of education on voting behavior is hard to assess because wealth affects both education and voting behavior"
- So: a correlation between education and voting might *not* imply a causal relation between the two.
- How might one address this?

Instrumental variables



"It's hard to assess the effect of military service on future earnings because of individual characteristics that might explain both. But date of birth affects the chances of serving and so can be used to recover estimates of service on earnings."

Models: The arguments

An argument:

Here is a complete, albeit barebones (and possibly incorrect), argument:

- Good institutions (***I***) cause economic growth (***G***), except in countries that have large stocks of natural resources (***N***).
- The reason is that institutions encourage people to invest (***V***) which spurs growth (this effect does not kick in in natural resource rich countries however since people live off rent rather than off production).
- Growth also makes it easier to maintain good institutions, which creates a virtuous cycle.
- Being an ally (***A***) of the United States also helps economic growth, but it sometimes corrupts domestic institutions.
- Historically, places with climates (***C***) suitable for colonizers to settle in ended up with better institutions. Except for their effect on institutions, these climatic conditions are irrelevant for understanding contemporary economic growth.

Some counterarguments:

- Places with climates suitable for colonizers benefited from better access to international markets which led to growth.
- Good soil is also important for growth!
- Good institutions also make sure that investments yield greater returns and that's what causes growth

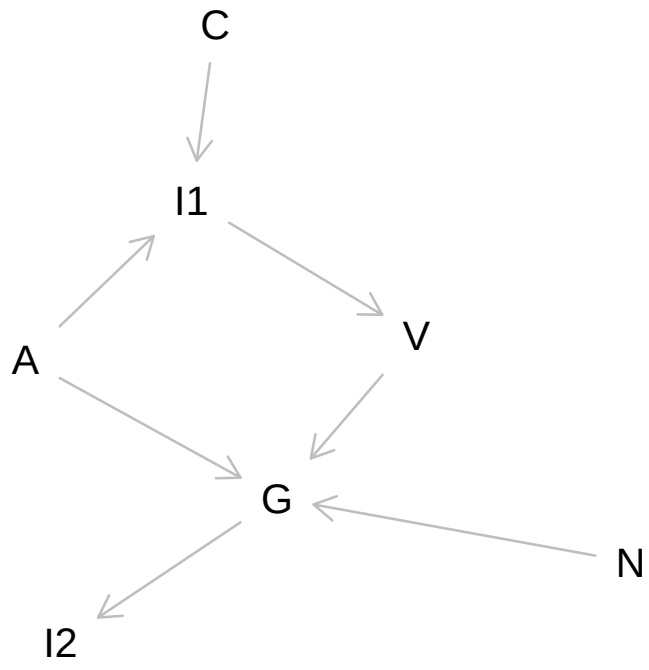
Questions on Nodes

- What are the dependent variables?
- What are the independent variables?
- What are the mediating variables?
- What are the conditioning variables?
- What are the confounding variables?
- What are the instrumental variables?
- Graph the relations between the variables.

Questions on Inference

- Say I and G are positively correlated. Does this mean that I causes G?
- Say I and G are negatively correlated. Does this mean that I does not cause G?
- How might you estimate the effect of I on G?
- How does C help establish the link between I and G?
- Where is the theory? Is it in equivalent to the graph or does it generate the graph?
- How might you check if the proposed theory is correct?
- Which of the counterarguments are strong and why?

A graph



Exercises: Dissect these arguments

Four arguments. For each one you should identify the:

- type of argument (effect of X, cause of Y, effect of X on Y)
- unit of analysis
- dependent variable(s)
- independent variable(s)
- mediator(s)
- possible conditioning variable(s)
- possible confounder(s)
- possible identification strategy
- relevant key agent(s) (actor(s))
- measurement strategy

A. Natural resources and conflict

In developing countries that discover natural resources, such as oil, the ruling elite can extract wealth without needing to tax citizens and develop the state apparatus. Because the state does not rely on taxation for government revenue, it does not need to set up accountability structures or extend its reach and citizens do not feel that they have ownership over the state. The state therefore becomes both less democratic and weaker than if it had not discovered the resources.

B. Democracy and growth

Rich countries are more likely to be democratic for the simple reason that when people become wealthier they refuse to be dictated to by others and they demand a role in government. The marginal effects of income increases are greater for poorer countries because the impacts on eduction are greatest at these levels. You can test this proposition by exploiting natural variation in commodity prices which provide shocks to national income, especially for countries dependent on primary commodity exports.

C. Factor Endowments and Coalitions

When countries increase trade (imports and exports), the returns to economic factors (such as labor, land and capital) are affected differently. Specifically, the returns to factors that are the most *abundant* are positive, while the returns to factors that are the most *scarce* are negative. Therefore, the relative factor endowments of a country will predict what sort of political coalitions will form (eg Land versus Labor + Capital) and which groups will favor free trade policies.

D. Democratic peace

In democratic states, leaders are accountable for any losses incurred as a result of the wars that they enter into. Two states with democratic leaders are also more likely to share a common set of norms, and to engage in trade with one another. Therefore, two democracies are far less likely to enter into war with one another than a democracy and a non-democracy, or two non-democracies.

Inquiries: Causal claims

Motivation

The *intervention* based motivation for understanding causal effects:

- We want to know if a particular intervention (like aid) caused a particular outcome (like reduced corruption).
- We need to know:
  1. What happened?
  2. What would the outcome have been if there were no intervention?
- The problem
  1. . . . this is hard
  2. . . . this is impossible

  The problem in 2 is that you need to know what would have happened if things were different. You need information on a **counterfactual**

Potential Outcomes: Simple case

- For each unit we assume that there are two **post-treatment** outcomes: $Y_i(1)$ and $Y_i(0)$.
  - $Y(0)$ is the outcome that **would** obtain *if* the unit did not receive the treatment
  - $Y(1)$ is the outcome that **would** obtain *if* the unit received the treatment
- The **causal effect** of Treatment (relative to Control) is:

$$\tau_i = Y_i(1) - Y_i(0)$$

- Note:
  - the causal effect is defined at the *individual level*.
  - there is no "data generating process" or functional form
  - the causal effect is defined relative to something else (did Germany cause the second world war?)

Causal claims: What is seen?

- We have talked about what's potential, now what do we *observe*?
- Say X_i indicates whether the unit i is assigned to treatment ($X_i = 1$) or not ($X_i = 0$). Then what we observe is:

$$Y_i = X_i Y_i(1) + (1 - X_i) Y_i(0)$$

- Say X is a random variable, then this is a sort of data generating process. BUT the key things to note is
  - Y_i is random but the randomness comes from X_i — the potential outcomes, $Y_i(1)$, $Y_i(0)$ are fixed

Implications

Five implications of the counterfactual definition:

1. You *infer* causation you don't observe of measure causes
2. Causal relations are not transitive
3. Causal paths are not spatially connected
4. Causes are not rival
5. No causation without manipulation

1. Causal statements are partly about what did not happen

Inference: We define causes in terms of things that did not happen. This puts **inference** front and center.

- Assessing effects is not a measurement problem, it is an inference problem. This is true whether we use quantitative or qualitative methods.

2. Causal relations are not transitive

- If for a given unit A causes B and B causes C, does that mean that A causes C?

  A boulder is flying down a mountain. You duck. This saves your life.

  So the boulder caused the ducking and the ducking caused you to survive. So: *did the boulder cause you to survive?*

What implications of non-transitivity?

3: Causal paths are not spatially connected

Say A causes B — does that mean that there is a spatiotemporally continuous sequence of causal intermediates?

- Person A is planning some action Y
- Person B sets out to stop them
- Person X intervenes and prevents B from stopping person A

In this case Person A may complete their action, producing Y, without any knowledge that B and X even exist; in particular X need not be anywhere close to the action.

But still, X caused Y

What implications of non-connectedness?

4: Causes are not rival

The counterfactual model focuses on contribution. It can be used to assess attribution in a very conditional sense (i.e. given $X = Y = 1$ did X cause Y) but not in an absolute sense ("the" cause).

- Focus is on **non-rival** contributions. Not: what caused Y but **what is the effect of** X?
- At most it provides a conditional account of what caused Y—whether a particular thing caused Y, given other things.

Consider at outcome Y that might depend on two causes X_1 and X_2:

$$Y(0,0) = 0$$

$$Y(1,0) = 0$$

$$Y(0,1) = 0$$

$$Y(1,1) = 1$$

What caused Y? Which cause was most important?

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The counterfactual model focuses on contribution. It can be used to assess attribution in a very conditional sense (i.e. given $X = Y = 1$ did X cause Y) but not in an absolute sense ("the" cause).

- This is problem for research programs that define "explanation" in terms of figuring out *the* things that cause Y
- Real difficulties conceptualizing what it means to say one cause is more important than another cause. What does that mean?

5: No causation without manipulation

- Some seemingly causal claims not admissible.
- To get the definition off the ground, **manipulation must be imaginable** (whether practical or not)
- This renders thinking about effects of race and gender difficult
- What does it mean to say that Aunt Pat voted for Brexit because she is old?

5: No causation without manipulation

- Some seemingly causal claims not admissible.
- To get the definition off the ground, **manipulation must be imaginable** (whether practical or not)
- This renders thinking about effects of race and gender difficult
- **Compare**: What does it mean to say that Southern counties voted for Brexit because they have many old people?

Exercise your potential outcomes 1

Consider the following potential outcomes table:

Unit	Y(0)	Y(1)	τ_i
1	4	3	
2	2	3	
3	1	3	
4	1	3	
5	2	3	

Questions for us: What are the unit level treatment effects? What is the average treatment effect?

Exercise your potential outcomes 2

Consider the following potential outcomes table:

In treatment?	Y(0)	Y(1)
Yes		2
No	3	
No	1	
Yes		3
Yes		3
No	2	

Questions for us: Fill in the blanks.

- Assuming a constant treatment effect of $+1$
- Assuming a constant treatment effect of -1
- Assuming an *average* treatment effect of 0

What is the actual treatment effect?

Takeaway on Causal Claims

- Takeaway: A causal claim is always partly a claim about what **did not happen**

Inquiries: Estimands

Estimands

- The estimand is the thing you want to estimate
- If you are estimating something you should be able to say what your estimand is
- You are responsible for your estimand. Your estimator will not tell you what your estimand is
- Just because you can calculate something does not mean that you have an estimand (You can test a hypothesis without having an estimand)

Examples of estimands

- The average effect in the population
- The average effect in the sample
- The average effect for those treated
- The average (counterfactual) effect for those not treated
- The effect of assigning treatment
- The effect of receiving treatment
- The effect for men, for women
- The effect of one effect on another effect
- Whether $X = 1$ was the cause of $Y = 1$
- More difficult: the share of the effect of X on Y that passes through mediator M

Examples of estimands

Consider the factorial design:

		X2	
		0	1
X1	0	Y(0,0)	Y(0,1)
	1	Y(1,0)	Y(1,1)

Multiple targets of inference in a factorial design

- **Interactive** effect is

$$(Y(1,1) - Y(0,1)) - (Y(1,0) - Y(0,0))$$

- **Interpretation**: How much bigger is the effect of $X1$ when $X2 = 1$ than it is when $X2 = 0$

Examples of estimands

Consider the factorial design:

		X2	
		0	1
X1	0	Y(0,0)	Y(0,1)
	1	Y(1,0)	Y(1,1)

Question: What is the estimand "The overall effect of X1"?

Takeaway on Estimands

- Takeaway: Estimands (the target of inference) should be defined clearly.
- It is good practice to define them in terms of potential outcomes.

Inquiries: Hypotheses

Characteristics of good hypotheses

- They are possibly TRUE or FALSE
- They are **falsifiable**
- They are statements about **the world**, not your analysis
- They are simple (not double barreled)
- They involve clear concepts
- They are **few**, and they are motivated
- They are **contested**: You will learn something whether the data supports them or rejects them. Most importantly: you are not sure if they are true or false
- They are numbered, and maybe even named

Some hypotheses

Consider these:

- Education is very important
- Education increases your income
- Education either increases, decreases, or has no effect on your income
- Education is good for you because it strengthens your character in very fundamental ways that you could never measure

Now:

- Just one of these is not a hypothesis. Which one?
- Just one of these is a good hypothesis. Which one?

Nulls: A point of confusion

Because of an unusual convention, social scientists often describe hypotheses in terms of what they **expect** but then *test* the null hypothesis of no effect

eg:

- H1: Competition reduces prices
- H-null: Competition has no effect on prices

Take home ideas

- You don't need them, but stating expectations in terms of hypotheses provides *discipline* to a research project.
- Hypotheses are statements about the world that you seek to *reject*
- A good hypothesis is simple and falsifiable
- A p value is the probability of data like what you see under some particular hypothesis

Answers: Differences in Means with Experimental Data

The problem with the case level estimand

The simplest estimand is the case level causal effect.

- The causal effect of Treatment (relative to Control) is:

$$\tau_i = Y_i(1) - Y_i(0)$$

- This is what we want to estimate

The problem with the case level estimand

The simplest estimand is the case level causal effect.

- The causal effect of Treatment (relative to Control) is:

$$\tau_i = Y_i(1) - Y_i(0)$$

- This is what we want to estimate
- BUT: We never can observe both $Y_i(1)$ and $Y_i(0)$!
- This is called the **fundamental problem of causal inference** (Holland)

The possibility of estimating *average* effects

- Now for some magic. We really want to estimate:

$$\tau_i = Y_i(1) - Y_i(0)$$

  but we cannot

- Say we lower our sights and try to estimate an **average** treatment effect:

$$\tau = E(Y(1) - Y(0))$$

- Now make use of the fact that

$$E(Y(1) - Y(0)) = E(Y(1)) - E(Y(0))$$

- In words: *The average of differences is equal to the difference of averages*
- The good news is that *while we can't hope to measure the differences; we are good at measuring averages*.

The possibility of estimating *average* effects

- So we want to estimate $E(Y(1))$ and $E(Y(0))$.
- We know that we can estimate averages of a quantity by taking the average value from a **random sample** of units
- So one approach is to to select a random sample of the $Y(1)$ values and a random sample of the $Y(0)$ values, in other words, we **randomly assign** subjects to conditions.
- When we do that we can in fact estimate:

$$E(Y_i(1)|Z_i = 1) - E(Y_i(0)|Z_i = 0)$$

which in expectation equals:

$$E(Y_i(1)|Z_i = 1 \text{ or } Z_i = 0) - E(Y_i(0)|Z_i = 1 \text{ or } Z_i = 0)$$

- This highlights a deep connection between **random assignment** and **random sampling**

How randomization helps

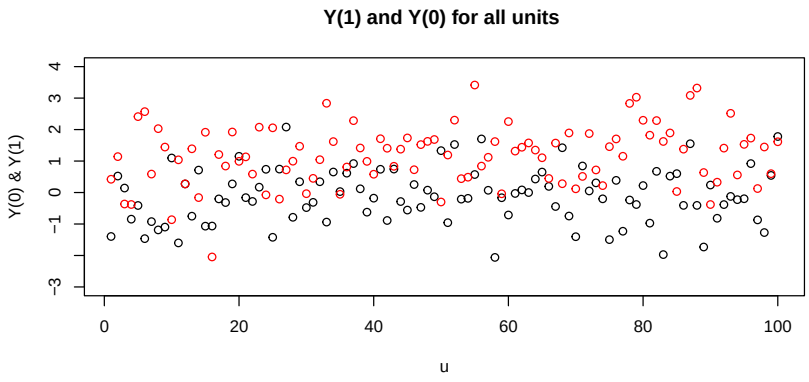
This provides a **positive argument** for causal inference from randomization, rather than simply saying with randomization "everything else is controlled for''

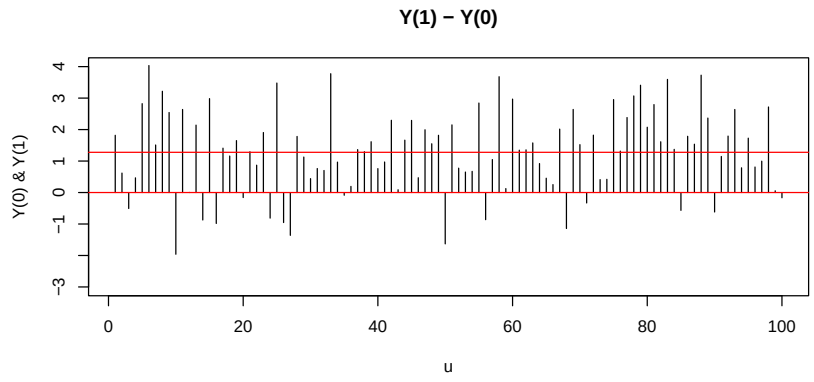
Let's discuss:

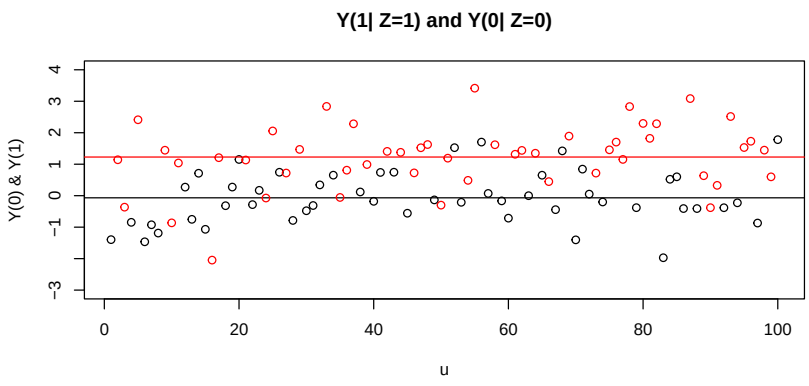
- Does the fact that an estimate is unbiased mean that it is right?
- Can a randomization "fail"?
- Where are the covariates?

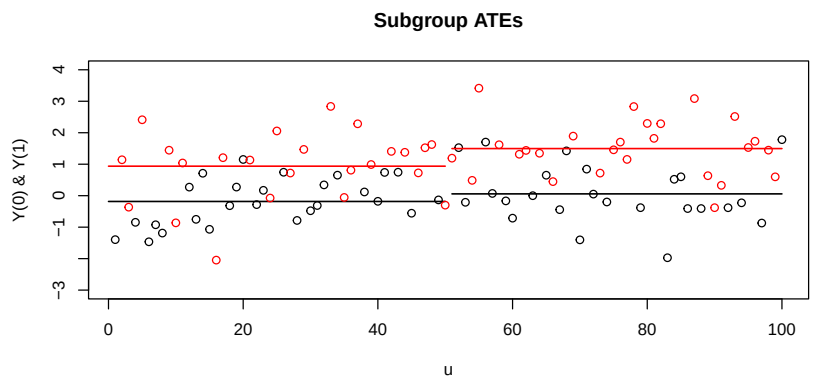
Potential outcomes: why randomization works

The average of the differences $\approx$ difference of averages (and exactly equal "in expectation").









For discussion:

How does the approach here relate to Mill's "method of difference"?

> *If an instance in which the phenomenon under investigation occurs, and an instance in which it does not occur, have every circumstance save one in common, that one occurring only in the former; the circumstance in which alone the two instances differ, is the effect, or cause, or an indispensable part of the cause, of the phenomenon.*

John Stuart Mill (1843) *A System of Logic, Vol. 1.*

Take away on randomization and differences in means

- Takeaway: Random **assignment** to treatment is random **sampling** from alternative universes. Differences in means estimates the differences between two universes.
- In practice we don't always have situations where treatment is randomly assigned. A *big challenge* in research design is to figure ways to make the case that assignment can be treated "as if" it is random.

Answers: Tests

Two approaches

There are two broad approaches for drawing conclusions about hypotheses from data.

- Frequentist (classical) approach
  - Key ideas: p value, confidence interval
- Bayesian approach
  - Key ideas: Posterior distribution

Tests

In the classical approach to testing a hypothesis we ask:

How likely are we to see data like this if the hypothesis is true?

- If the answer is "not very likely" then we treat the hypothesis as suspect.
- If the answer is *not* "not very likely" then the hypothesis is maintained (some say "accepted" but this is tricky as you may want to "maintain" multiple incompatible hypotheses)

How unlikely is "not very likely"

Weighing Evidence

When we test a hypothesis we decide first on what sort of evidence we need to see in order to decide that the hypothesis is not reliable.

- **Othello** has a hypothesis that Desdemona is innocent.
- **Iago** confronts him with evidence:
- See how she looks at him: would she look a him like that if she were innocent?
- See how she defends him: would she defend him like that if she were innocent?
- See he carries her handkerchief: would he have her handkerchief if she were innocent?
- Othello, the chances of all of these things arising if she were innocent is surely less than 5%

Hypotheses are often rejected, sometimes maintained, but rarely accepted

Note that Othello is focused on the probability of the events if she were innocent but not the probability of the events if Iago were trying to trick him.

He is not directly assessing his belief in whether she is faithful, but rather how likely the data would be if she were faithful.

That is, he assesses:

$$\Pr(\text{Data}|\text{Hypothesis is TRUE})$$

In contrast a "Bayesian" would try to assess:

$$\Pr(\text{Hypothesis is TRUE}|\text{Data})$$

Not Bayes

Note: $Pr(\text{Data}|\text{Hypothesis is TRUE})$ and $Pr(\text{Hypothesis is TRUE}|\text{Data})$ are connected but in a slightly complex way (Bayes Rule):

$$\Pr(\text{H}|\text{D}) = \frac{\Pr(\text{D}|\text{H})\Pr(\text{H})}{\Pr(\text{D}|\text{H})\Pr(\text{H}) + \Pr(\text{D}|\text{NOT H})\Pr(\text{NOT H})}$$

So your belief about the hypothesis should depend not just on the likelihood of seeing the data given the hypothesis but also on your prior belief about how plausible the hypothesis is. But this second part is ignored in classical tests.

Bayes and Frequentist Inference: Example

Imagine a great test:

- If you have a disease then there is a 99% probability that the test will say you have the disease.
- If you do not have a disease then there is a 99% probability that the test will say you do not have the disease.
- 1% of people like you have this disease

The test says you have the disease:

- Will the frequentist reject the null that you have the diseaese?
- Does a Bayesian think you have the disease?

Bayes and Frequentist Inference: Example

Answer with a figure:

Bayes and Frequentist Inference: Example

Bayes answer with a formula:

$$\Pr(S|P) = \frac{\Pr(P|S)\Pr(S)}{\Pr(P|S)\Pr(S) = \Pr(P|H)\Pr(H)} = \frac{.99 \times .01}{.99 \times .01 + .01 \times .99} = \frac{1}{2}$$

Frequentist answer:

The p value is: $\Pr(P|H) = 0.01$. This is very small so reject hypothesis that you are healthy

Calculate a p value in your head

- Illustrating p values via "randomization inference"...
- Say you randomized assignment to treatment and your data looked like this.

Unit	1	2	3	4	5	6	7	8	9	10
Treatment	0	0	0	0	0	0	0	1	0	0
Healthy?	3	2	4	6	7	2	4	9	8	2

- Does the treatment improve your health?
- $p =?$

Calculate a p value in your head

- Illustrating p values via "randomization inference"...
- Say you randomized assignment to treatment and your data looked like this.

Unit	1	2	3	4	5	6	7	8	9	10
Treatment	0	0	0	0	0	0	0	0	1	0
Healthy?	3	2	4	6	7	2	4	9	8	2

- Does the treatment improve your health?
- $p =?$

Data and Answers: Other Quantitative Strategies

Recap: Differences in means worked when we had randomization

- We want to estimate:

$$\frac{1}{n}\sum_i((Y_i(1)) - Y_i(0))$$

- We estimate using:

$$\frac{1}{n_t}\sum_{i\in T} Y_i - \frac{1}{n_c}\sum_{i\in C} Y_i$$

- This works because, with randomization $\frac{1}{n_t}\sum_{i\in T} Y_i = \frac{1}{n}\sum_i((Y_i(1))$ *in expectation* – that is, on average the sample average is the population average. Similarly $\frac{1}{n_c}\sum_{i\in C} Y_i = \frac{1}{n}\sum_i((Y_i(0))$ *in expectation*.

But in the absence of randomization there are inferential risks

Difficulties once assignment is related to potential outcomes.

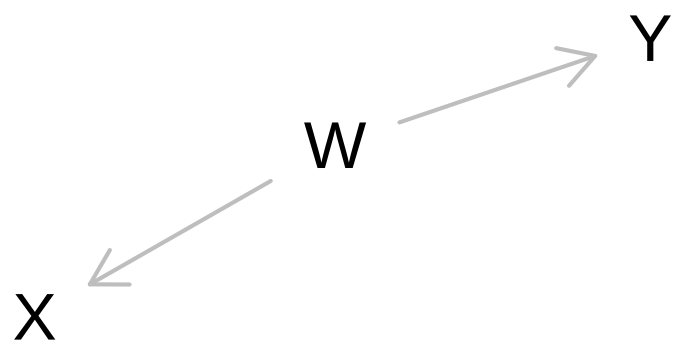


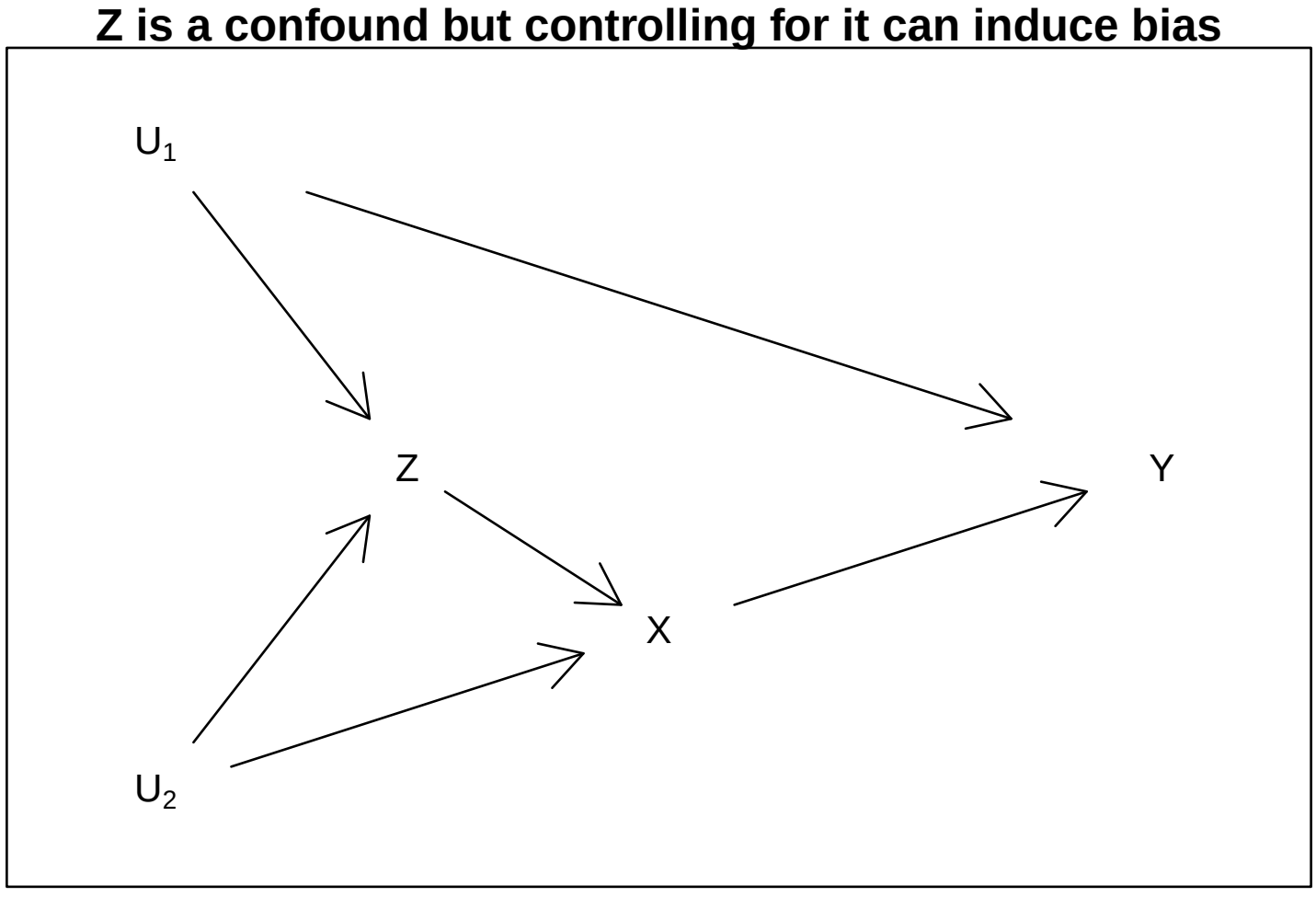
Figure 1: Here X might be related to Y even though it does not cause Y

In fact in the absence of randomization a model is *required*

- Randomization is **not required** for causal inference.
- But without it you need some alternative argument for why your estimates from the treatment group capture *what would occur in the control group* if they were treated (and vice versa)
- What's more you will need a **model**:
  - Some variables might *have* to be taken into account in order to ensure no confounding
  - Some variables might *have* to *not* be taken into account in order to ensure no confounding
  - Sometimes these might be the same variables!

In the absence of randomization a model is *required*

This is shown by Pearl (1995):



Alternatives

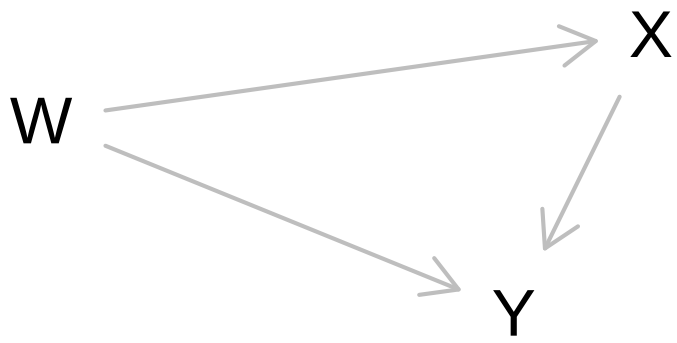
Randomization provides a very useful benchmark. Other strategies seek to approximate the magic of randomization:

- **Experimental Control** seeks to ensure that *ceteris* is *paribus* (induced unit homogeneity)
- **Adjustments**. Controlling: Regression, Matching and Weighting
- **Instrumental variables** or **Natural experiments**—seeks a shock that approximates randomization.
- **Difference in differences**—assumes that once you account for common time trends cases are as-if randomized
- **Regression discontinuity**—assumes that cases are as-if randomized *around a threshold* (there are also motivations for RDD that do not assume as-if randomization)
- **Synthetic Matching** and other model based approaches

Adjustment methods Intuition

Key idea is to figure out effects *conditional* on the values others nodes my take.

Our problem:



Our solution:

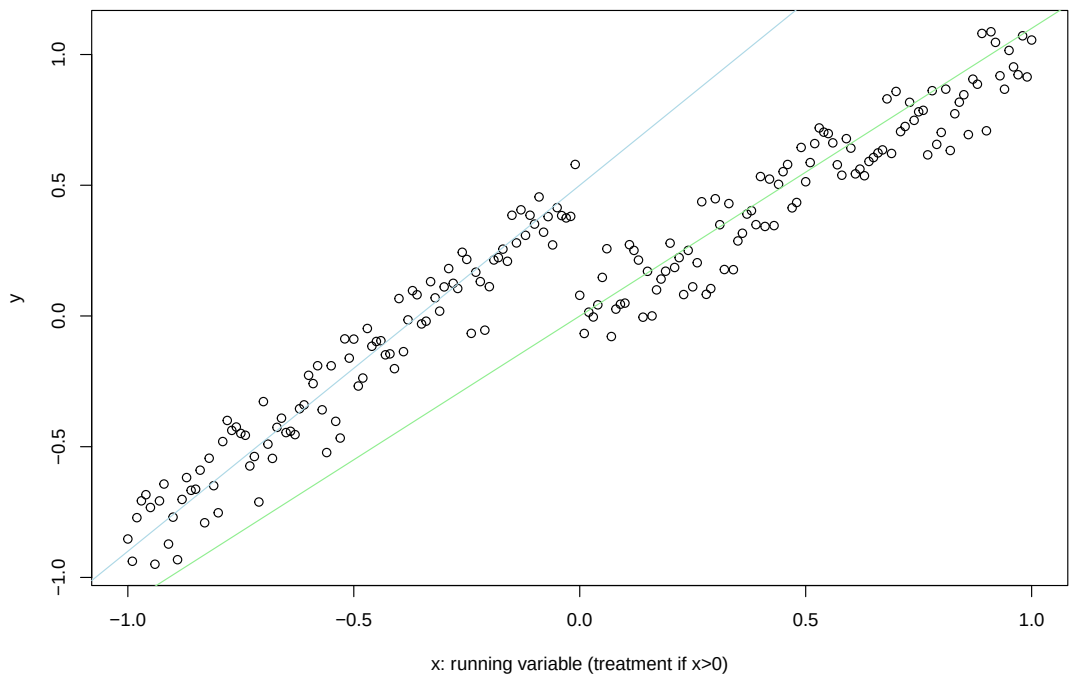
$$X \rightarrow Y \quad (\text{given } W = 0)$$

$$X \rightarrow Y \quad (\text{given } W = 1)$$

We can estimate effects *within* similar sets and then average the results (weighting by the size of the sets)

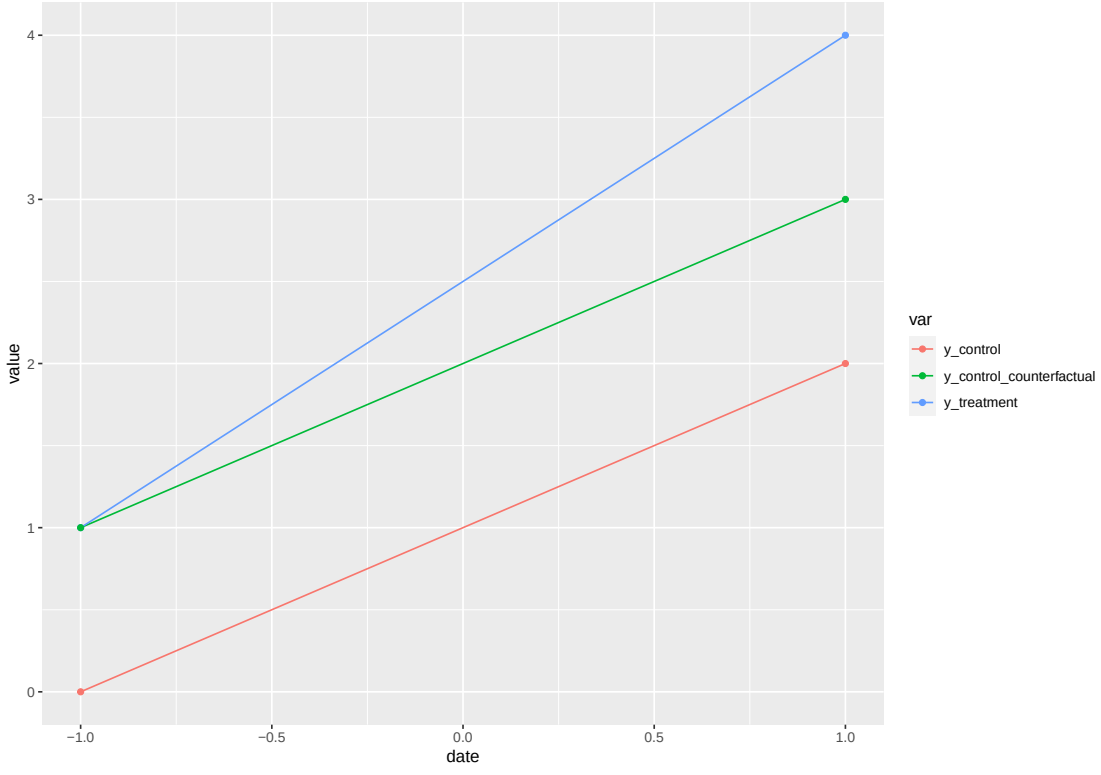
RDD Intuition:

e.g. compare units in which the margin of victory was 1 vote for democrats against those for which it was -1. We expect that these are approximately identical, on average, is *all* regards and get an estimate of the effect of victory on some outcome *at the threshold*



Diff-in-diff Intuition:

- examine the difference between treated and control in the before-after difference
- OK under the assumption that the change in the control group is the same as what the change would have been in the treatment group absent treatment



Data and Answers: Other Qualitative Strategies

Qualitative estimands

- A common qualitative estimand is the "cause of an effect": given $Y = 1$ and $X = 1$ is $Y = 1$ because $X = 1$?
- Randomization (alone) does not justify answers to "causes of effects" questions
- We might be able to say that we think that the effect of a treatment on a given outcome is $1/3$
- But that does not justify claiming that the probability that the outcome is due to the treatment is $1/3$

Qualitative estimands

Here is the logic:

- Say I know that (binary) X increases Y from $\frac{1}{3}$ to $\frac{2}{3}$ on average.
- Say I observe $Y = 1$. What are the chances it is due to X?
  - One possibility is that X has a positive effect for $2/3$ of cases and a negative effect for $1/3$ of cases. In that case whenever $X = Y = 1$ this is due to X.
  - Another is that X never has a negative effect and it has a positive effect for $1/3$ of cases and no effect on the rest. Then there's a 50% chance that $Y = 1$ is due to $X = 1$ (why 50%?)

Learning from clues

- Classically qualitative strategies use *auxiliary* information to understand the relationship between X and Y.
- For instance: You want to know if the swamp caused malaria
  - Quantitative approach: Compare malaria incidence in places with and without swamps
  - Qualitative approach: Look to see whether there mosquitoes are breeding at the swamp
- This approach typically requires a theory: conditional on theory T, observing K increases my confidence that $X = 1$ caused $Y = 1$ in this case.

Process tracing

- In process tracing such clues are called "CPO"–causal process observations
- How informative a clue is is sometimes called its "probative value"
  - sometimes you learn a lot when you *do* see a clue ($K = 1$)
  - sometimes you learn a lot when you do *not* see a clue despite looking for it ($K = 0$)
- Classic tests:
  - A "smoking gun" clue gives a lot of confidence when you find it
  - A "hoop" clue gives a lot of confidence when you do not find it

Bayesian updating

See for instance Fairfield and Charman

We observe clue K and want to update about hypothesis A (given hypotheses A or B under consideration):

$$\Pr(A|K) = \frac{\Pr(K|A)\Pr(A)}{\Pr(K|A)\Pr(A) + \Pr(K|B)\Pr(B)}$$

The action is in the difference between $\Pr(K|A)$ and $\Pr(K|B)$

Qualitative tests: b or d?

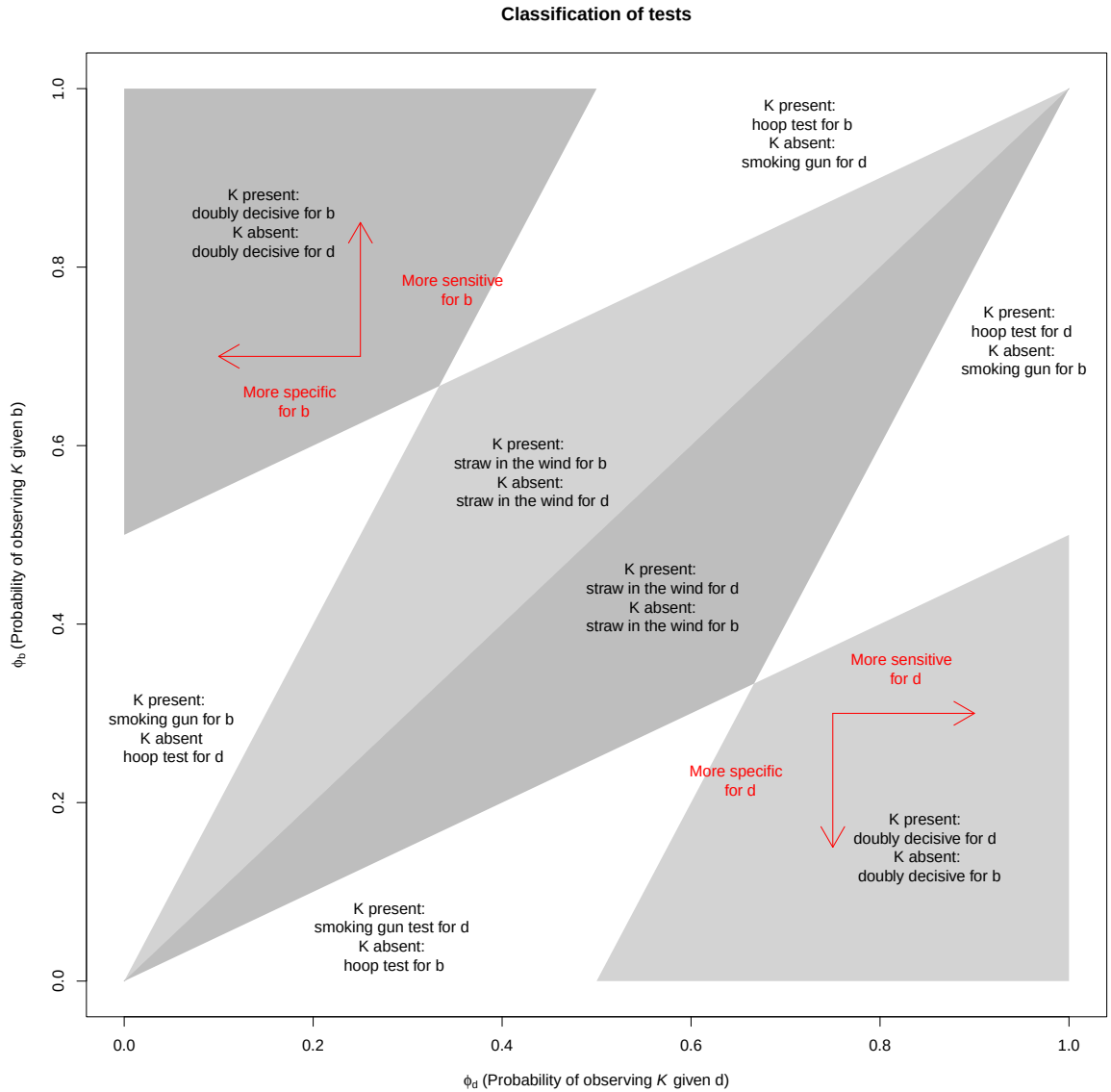


Figure 2: Mapping from the probability of observing a clue if b (ϕ_b) or d (ϕ_d) to a generalization of the Van-Evera tests.

How might beliefs about probative value be supported?

- Most common answer: "theory"
- Better answer: background knowledge + specific assumptions

For instance:

- Say we know in a given population that:
  - $X \rightarrow Y \leftarrow K$
  - $\Pr(Y = 1|X = 0, K = 0) = 1$
  - $\Pr(Y = 1|X = 1, K = 0) = .5$
  - $\Pr(Y = 1|X = 0, K = 1) = 0$
  - $\Pr(Y = 1|X = 1, K = 1) = .5$
- The unit we care about is "exchangeable" with other units in this population

Then for an $X = Y = 1$ case:

- seeing $K = 1 \rightarrow X$ caused Y (why?)
- seeing $K = 0 \rightarrow X$ did not cause Y (why?)

How might beliefs about probative value be supported?

- Thus, doubly decisive clues are possible
- But likely rare: most often best you can do is put bounds on causal effects

Case selection: What do you do with a case?

- Establish that X is indeed X and Y is indeed Y
- Assess whether scope conditions of theory are indeed present
- Assess whether the argument has "face validity"
- Ideally look for pre-specified clues that support or weaken the claim

Case selection

- Case selection depends on the estimand. Are you interested in a case level estimand or a population level estimand?

Common strategies:

1. On the regression line
2. Off the regression line
3. Most likely cases, least likely cases
4. Proportionate to distribution (safe rule of thumb)

less common but good:

5. Follow the probative value:
   - Perhaps K is informative in one case not another
   - For instance in the example above there is no point selecting a $X = Y = 0$ case since you already know that in that case $K = 1$: learning about K will not be informative

Case selection: n

How many cases?

- No good answer
- More always better except insofar as they reduce quality of analysis
- If you are doing causal inference with case comparison methods only then you want as many as possible and *at least* as many as you have explanations that you want to distinguish from each other

Mixed methods [advanced]

Insight:

- If observation of X and Y lets you update your beliefs about a causal effect
- And if observation of K also lets you update your beliefs about a causal effect
- Then you can update jointly from X, Y, K

$$Pr(H|X, Y, K) = \frac{Pr(X, Y, K|H)Pr(H)}{Pr(X, Y, K)}$$

Back to MIDA: Design Evaluation

Design declaration of a simple RCT

```
simple_design <-

  # M: model
  declare_population(N = 100, u = rnorm(N)) +
  declare_potential_outcomes(Y ~ 0.25*Z + u) +

  # I: inquiry: the average effect
  declare_inquiry(PATE = mean(Y_Z_1 - Y_Z_0)) +

  # D: data strategy
  # sampling
  declare_sampling(S = complete_rs(N = N, n = 50)) +
  # assignment
  declare_assignment(Z = simple_ra(N, prob = 0.5)) +
  declare_reveal(Y, Z) +

  # A: answer strategy
  declare_estimator(Y ~ Z, inquiry = "PATE")
```

Design diagnosis

If you then "run" this design many times you can see the distribution of the "true" answers, the distribution of the estimates, and how these relate to each other.

Estimand Label	Bias	RMSE	Power	Coverage
PATE	0.00 (0.01)	0.28 (0.01)	0.14 (0.01)	0.95 (0.01)

Design library

You can explore some designs here:

- https://declaredesign.org/library/
- https://eos.wzb.eu/ipi/DDWizard/
- https://declaredesign.org/blog/2020-01-08-dding-on-the-internets.html

To dos

To dos

- Graph your problem
- Provide a paragraph encapsulating the theory
- Try to define the primary estimand
- Clarify top two or three hypotheses
- Re-consider estimation strategy in light of estimand: does the estimator shoot at this estimand?