

# Introducing Counterfactual Causal Inference

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## What might cause mean?: Definitions and Conceptualization

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## Some of my recent causal questions

Did a new Hausa television station in northern Nigeria change attitudes about violence, the role of women in society, or the role of youth in society?

Will adding education counselors to public housing in the USA increase the numbers of low income youth enrolled in post-secondary education (like university) and receiving financial aid for their education?

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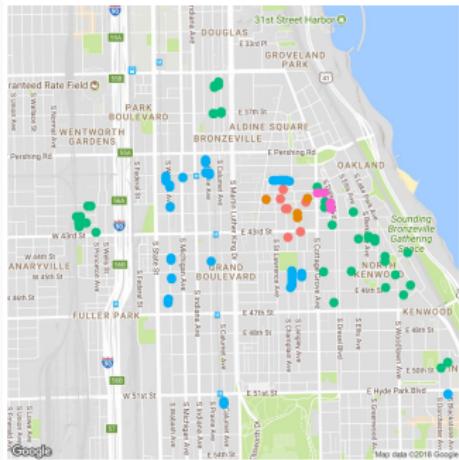
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Figure 1: Example of overlapping AMPs in Chicago



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## What are we doing when we talk about causation?

I think that social scientists try to work collectively to **build evidence** for explanations. So: we make individual contributions, we try to persuade ourselves that we have learned something, we correct our past misunderstandings, etc.

Strong evidence persuades — it is harder to argue against than weak evidence.

Randomized experiments, we'll show, are especially persuasive about explanations involving **cause** in very focused ways.

*“[The experimenter’s] aim is to draw valid conclusions of determinate precision and generality from the evidence...”* (Joan Fisher Box quoted in Pearl and Mackenzie (2018))

## Why not just talk about correlation and association?

Why the growing interest in *causal* inference rather than *population* inference or *measurement* inference?

My answer: Humanity needs a kind of engineering turn within part of the social sciences because of the growth in “How” questions: “How can we make government work better? How can we deliver development aid better?”

Moving from “Why” to “How” involves the need to know about the effects of causes.

For examples of this move: EGAP, J-PAL, Behavioral Insights teams, the Evidence-Based Policy Movement, McKinsey, Deloitte and see (Bowers and Testa, 2019).

## What does “cause” mean?

When someone says “ $X$  causes  $Y$ ” they might mean:

**Persistent association** “We always/mostly see  $Y = 1$  when  $X = 1$  and  $Y = 0$  when  $X = 0$ .”

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**Difference after operation of a mechanism** “Once upon a time A changed  $X$ , and then one day X changed  $B$ , and because of that  $B$  changed  $C$ , and finally  $C$  changed  $Y$ .”

other...

## What does “cause” mean?

Often, experiments aim to manipulate (by randomization) parts of expected/theoretical mechanisms to reveal counter-factuals rather than aim to document persistent and wide-spread association.

This week we will be focusing on the counterfactual approach because we focusing on experiments. It is not that we think it is wrong to conceptualize “cause” in any other way. But that it has been productive to use the counterfactual approach.

{ Extra: If you want to dig into this see Brady (2008).

<http://egap.org/resources/guides/causality/> }

## How to interpret “X causes Y”?

- “X causes Y” need not imply that W and V do not cause Y: X is a part of the story, not the whole story. (The whole story is not necessary in order to learn about whether X causes Y).
- Counterfactual causation does not require “spatiotemporally continuous sequence of causal intermediates” ex: Person A plans event Y. Person B’s action would stop Y (say, a random bump from a stranger). Person C doesn’t know about Person A or action Y but stops B (maybe thinks B is going to trip). So, Person A does action Y. And Person C causes action Y (without Person C’s action, Y would not have occurred). (Holland, 1986)
- “X causes Y” requires a **context**: matches cause flame but require oxygen; small classrooms improve test scores but require experienced teachers and funding (Cartwright and Hardie, 2012).

## How to interpret “X causes Y”?

- We can establish that X causes Y without knowing mechanism. The mechanism can be complex, it can involve probability: X causes Y sometimes because of A and sometimes because of B.
- “X causes Y” can mean “With X, probability of Y is higher than would be without X.” or “Without X there is no Y.” Either is compatible with the counterfactual idea.
- Correlation is not causation: Favorite examples?
- “X causes Y” is a statement about what didn’t happen: “If X had not operated, occurred, then Y would not have occurred.” (More about the fundamental problem of counterfactual causation later)

Randomization for Interpretable  
Comparisons and Clarity about  
Uncertainty

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## Exercise: Observational studies vs. Randomized studies

**Discuss in small groups:** Help me design the next project to answer one of these questions (or one of your own causal questions). Just sketch the key features of two designs — one observational and the other randomized.

### Possible research questions:

- Can edutainment (like the Hausa TV Station or radio programs currently being used in Niger) change attitudes about violence and extremism? (Goal: Reduce violence and extremism.)
- Does information about words spoken to infants/toddlers improve early language acquisition in this group? (Goal: reduce inequality in early verbal skills and eventually reducing inequality in school readiness at age 5)

## Exercise: Observational studies vs. Randomized studies

### Tasks:

1. Sketch an ideal observational study design? (no randomization, no researcher control but infinite resources for data collection) What questions would critical readers ask when you claim that your results reflect a causal relationship?
2. Sketch an ideal experimental study design? (including randomization and control) What questions would critical readers ask when you claim that your results reflect a causal relationship?

## Why randomize?

1. Randomization produces **fair** comparisons (ex. impersonal, no systematic differences between groups).
2. Randomization helps us reason about information/uncertainty.

*“Fisher realized that an uncertain answer to the right question is much better than a highly certain answer to the wrong question...If you ask the right question, getting an answer that is occasionally wrong is much less of a problem [than answers to the wrong question]. You can still estimate the amount of uncertainty in your answer, because the uncertainty comes from the randomization procedure (which is known) rather than the characteristics of the soil (which are unknown).” (Pearl and Mackenzie, 2018)*

## Using randomization to reason about causal Inference

How can we use what we **see** to learn about what we want to **know** ?

City	Pair	Treat	Turnout			Newspaper	$y_1$	$y_0$
			Baseline	Outcome				
Saginaw	1	0	17	16			?	16
Sioux City	1	1	21	22		Sioux City Journal	22	?
Battle Creek	2	0	13	14			?	14
Midland	2	1	12	7		Midland Daily News	7	?
Oxford	3	0	26	23			?	23
Lowell	3	1	25	27		Lowell Sun	27	?
Yakima	4	0	48	58			?	58
Richland	4	1	41	61		Tri-City Herald	61	?

**Table 1:** Design and outcomes in the Newspapers Experiment. The Treatment column shows treatment randomized within pair with the newspaper ads as 1 and lack of treatment as 0. The potential outcomes are  $y_1$  for treatment and  $y_0$  for control. Panagopoulos (2006) provides more detail on the design of the experiment.

## Using randomization to reason about causal Inference

How can we use what we see to learn about potential outcomes  
(causal effect $_i = f(y_{i,1}, y_{i,0})$ )?

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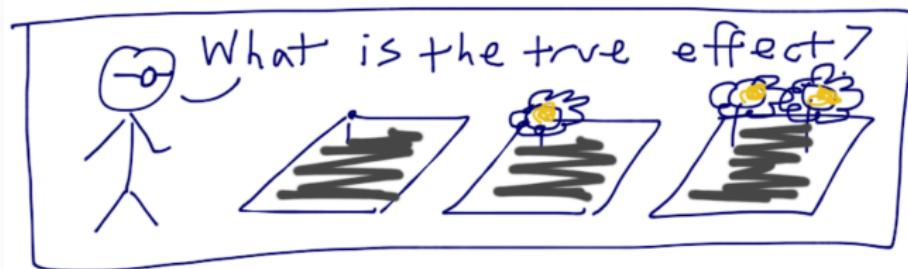
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What is the true effect of the treatment assignment?}



We don't know.



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I don't know the truth, but I can provide a good guess of the average causal effect.

i	$Z_i$	$Y_i$	$y_{i1}$	$y_{i0}$
A	0	16	?	16
B	1	22	22	?
C	0	7	?	7
D	1	14	14	?

$$\begin{aligned}\widehat{ATE} &= \bar{Y}_i | Z_i = 1 - \bar{Y}_i | Z_i = 0 \\ &= \frac{22+14}{2} - \frac{16+7}{2} = 6.5\end{aligned}$$

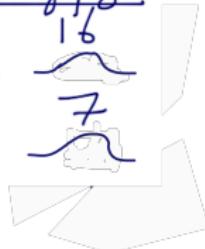
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I dew nut knew thee truth,  
but, given pryers, I cane  
predikte itf  
probabeeleetee.



i	$Z_i$	$y_i$	$y_{i+1}$	$y_{i+2}$
A	0	16	wave	16
B	1	22	22	wave
C	0	7	wave	7
D	1	14	14	wave

$$P(\text{[wave icon]}, f(y_1 - y_0)) = \text{[wave icon]}$$



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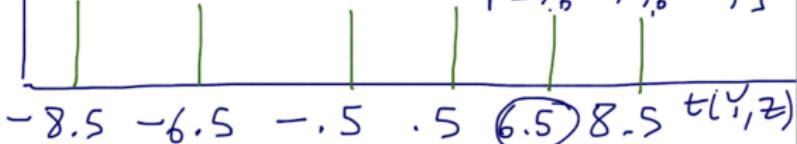
I don't know the truth,  
but I can assess specific  
claims about the truth.


$$H_0: y_{i1} = y_{i0}$$

i	$z_i$	$y_i$	$y_{i1}$	$y_{i0}$
A	0	16	?	16
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$$P(t(y, z))$$

$$\frac{1}{6}$$



What is the true effect of the treatment assignment?}

See also Pearl (2000) and also Richardson and Robins (2013). For more on the potential outcomes approach see Imbens and Rubin (2015).

## Estimating an Average Treatment Effect}

```
options(width=132)

Z <- c(0,1,0,1)
Y <- c(16,22,7,14)
estate <- mean(Y[Z==1]) - mean(Y[Z==0]) ## same as coef(lm(Y~Z))
estate

## [1] 6.5
```

## Testing the Sharp Null of No Effects

```
options(width=132)

Om <- matrix(0, ncol=choose(4,2), nrow=length(z)) ## All possible
whotrted <- combn(1:4,2)
for(i in 1:choose(4,2)){ Om[cbind(whotrted[,i],i)]<-1 }
meandifftz <- function(y,z){ mean(y[z==1]) - mean(y[z==0]) }
thedist<-apply(Om,2, function(z){ meandifftz(Y,z) })
rbind(Om, thedist)
```

```
##      [,1] [,2] [,3] [,4] [,5] [,6]
##      1.0  1.0  1.0  0.0  0.0  0.0
##      1.0  0.0  0.0  1.0  1.0  0.0
##      0.0  1.0  0.0  1.0  0.0  1.0
##      0.0  0.0  1.0  0.0  1.0  1.0
## thedist 8.5 -6.5  0.5 -0.5  6.5 -8.5
```

What do we need to interpret our calculations as teaching about causal quantities?

For the sharp null test: Randomization occurred as reported.

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For the sharp null test: Randomization occurred as reported.

For the average treatment effect: Randomization occurred as reported plus no interference between units.

## Weaknesses of RCTs (To Discuss)

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## References i

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