

Causal Inference in Environmental & Social Science

Nils Droste

2022 ClimBEco course



Structure of the Course

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model
Structural Causal Models
Mechanism

Controlled Trial

Design

Empirics Conservation

Conservatio

Randomistas



	time	Day 1: May 30, 2022	Day 2: May 31, 2022	Day 3: June 01, 2022	Day 4: June 02, 2022	Day 5: June 03, 2022
Lectures	10-12h	Greetings, Introduction to Causal inference, and randomized controlled trials	(Semi) Natural Experiments: Panel data regressions, two-way fixed effects, and recent cor- rections for staggered treatment	Simulated Counterfactu- als: matching methods, syn- thetic controls, and Bayesian Structural time series	Instruments & Interruptions: instrumental variables, regression discontinuity design	Cutting edges: Structural equation modelling for causal inference (and machine learning techniques?)
Seminars	13-15h	Replication: Jayachandran et al. (2017) Science	Replication: Card	Krueger (1994) <i>JAERE</i>	Replication: LaLonde (1986) PNAS	Replication: Abou-Chadi
Krause (2020) <i>RPP</i>	Student present- ations		'	'	'	'
Consultations	15-16h					

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My offer

- I will provide you with different "entry points" (words, graphs, math) to sharpen your intuition and conceptual understanding of quantitative causal inference
- We will collaboratively replicate exemplary works / causal inference strategies

My ask price

I want feedback what goes nice and what does not?

Your task

You apply one of the methods to a problem of your choice, write a short report and provide replication code

Motivation – My answer

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If you think about policies as if

- they were instruments / mechanisms / interventions
- with a potential to fix societal problems

Would you not want to know which ones actually work?



Example I: Epidemiology

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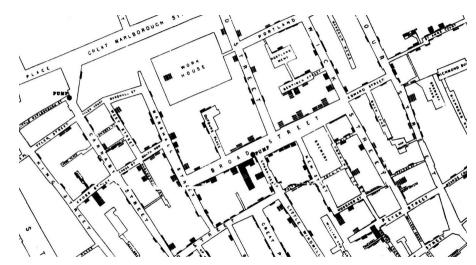
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John Snow's original dot map of the 1854 Broad street cholera outbreak. Image sources & info: wikipedia

Motivation – Greater minds' answers

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Development of Western science is based on **two great achievements**: the invention of the formal logical system (in Euclidean geometry) by the Greek philosophers, and the discovery of the possibility to find out **causal relationships by systematic experiment** (during the Renaissance)."

Albert Einstein (1953), as cited in Pearl (Pearl2009), my emphasis



Motivation – Greater minds' answers

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My interpretation:

 \rightarrow If we want to check our theories about how the world works, we can use systematic obervations (i.e. data) to test our assumptions.



Motivation – Greater minds' answers

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My interpretation:

- \rightarrow If we want to check our theories about how the world works, we can use systematic obervations (i.e. data) to test our assumptions.
- \rightarrow That does not *necessarily* entail quantitative analysis, but large number of observations have benefits for robustness (see next slide).

A short detour into probability

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Is the coin fair?



A short detour into probability

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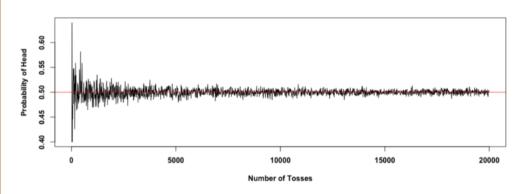
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Is the coin fair?



→ The law of large numbers allows to aproximate "true" values.

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BLIND MASTER PO: Close your eyes. What do you hear?

YOUNG KWAI CHANG CAINE: I hear the water, I hear the birds.

MASTER PO: Do you hear your own heartbeat?

KWAI CHANG CAINE: No.

MASTER PO: Do you hear the grasshopper that is at your feet?

KWAI CHANG CAINE: Old man, how is it that you hear these things?

MASTER PO: Young man, how is it that you do not?

Kung Fu, Pilot. Cited from Angrist2015, own emphasis



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→ We assume a measurable reality (positivism, empiricism).



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To answer questions of causality we need an epistemological framework to

- formulate testable hypothesis
- find a suitable method to test hypothesis



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To answer questions of causality we need an *epistemological framework* to

- formulate testable hypothesis
- find a suitable method to test hypothesis

Statistical causal inference is one such approach, suitable for

- both inductive and deductive reasoning
- generalizable, reproducible, falsifiable research



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We have a population of units; for each unit i we observe a variable D and a variable Y.

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We have a population of units; for each unit i we observe a variable D and a variable Y.

We observe that *D* and *Y* are correlated. Does *correlation* imply *causation*?



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We observe that *D* and *Y* are correlated. Does *correlation* imply *causation*?

In general no, because of

- confounding factors;
- reverse causality



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Randomista:

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We observe that *D* and *Y* are correlated. Does *correlation* imply *causation*?

In general no, because of

- confounding factors;
- reverse causality

We would like to understand in which circumstances one can conclude from the evidence that D causes Y.

source: lecture notes Sascha Becker 2014



Example II: Storcks & Babies

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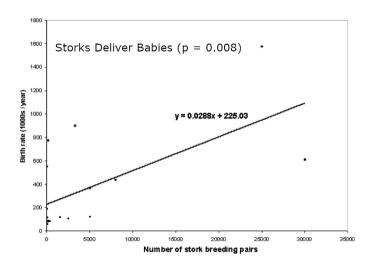
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Do storcks deliver babies? Image source: Matthews (Matthews2000)

Example II: Storcks & Babies

What happened, why did we get it so wrong?

Country	Area (km²)	Storks (pairs)	Humans (10 ⁶)	Birth rate (10 ³ /yr)
Albania	28,750	100	3.2	83
Austria	83,860	300	7.6	87
Belgium	30,520	1	9.9	118
Bulgaria	111,000	5000	9.0	117
Denmark	43,100	9	5.1	59
France	544,000	140	56	774
Germany	357,000	3300	78	901

Subset of original data. Source: Matthews (Matthews2000)

Besides outcome variable and variable of interest, we forgot confounding variables.

$$\mathbf{Y}_{i} = \alpha + \beta_{1} \mathbf{D}_{i} + \beta_{2} \mathbf{C}_{i} + \varepsilon_{i} \tag{1}$$

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Problem I: Confounding variables

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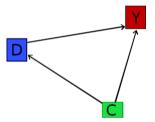
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Directed acyclic graph where variable C affects both textcolorblueD and Y. Image source: Modified from Huntington-Klein 2018

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Recall, we let Y denote our outcome variable, and D our treatment or intervention which we are interested in.

Letter *i* is an index of the individuals within our population.



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Recall, we let Y denote our outcome variable, and D our treatment or intervention which we are interested in.

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For **D** we have two possible realizations:



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Recall, we let Y denote our outcome variable, and D our treatment or intervention which we are interested in.

Letter *i* is an index of the individuals within our population.

For **D** we have two possible realizations:

- $\mathbf{D} = \mathbf{1}$ if *i* has received treatment;
- D = 0 if i has not received treatment.



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Recall, we let Y denote our outcome variable, and D our treatment or intervention which we are interested in.

Letter *i* is an index of the individuals within our population.

For **D** we have two possible realizations:

- $\mathbf{D} = \mathbf{1}$ if *i* has received treatment;
- $\mathbf{D} = \mathbf{0}$ if *i* has *not* received treatment.

Thus, $Y_i(D_i)$ indicates the *potential outcome* according to treatment:

- \bigvee_{i} (1) is the outcome in case of treatment;
- $V_i(0)$ is the outcome in case of *no* treatment.



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The hypothetical outcome for each unit can be written as

$$\Delta \mathbf{Y}_i = \mathbf{Y}_i(1) - \mathbf{Y}_i(0) \tag{2}$$

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The hypothetical outcome for each unit can be written as

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■ This approach requires to think in terms of "counterfactuals".



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The hypothetical outcome for each unit can be written as

$$\Delta \mathbf{Y}_i = \mathbf{Y}_i(1) - \mathbf{Y}_i(0) \tag{2}$$

- This approach requires to think in terms of "counterfactuals".
- While theoretically ideal, the identification and the measurement of a pure counterfactual is logically impossible:



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- This approach requires to think in terms of "counterfactuals".
- While theoretically ideal, the identification and the measurement of a pure counterfactual is logically impossible:
- We can only observe one state of the world, i.e. we cannot *directly* measure what would have happened in the counterfactual case (cf. **Holland1986**).



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The best we can do to infer an average treatment effect (ATE) by comparing sufficiently large subsamples from the overall population I: i.e. $I = \{A, B...\}$.



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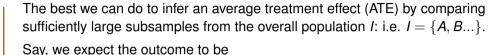
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$$E\{\Delta Y_i\} = E\{Y_i(1) - Y_i(0)\} = E\{Y_i(1)\} - E\{Y_i(0)\}.$$
(3)



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The best we can do to infer an average treatment effect (ATE) by comparing sufficiently large subsamples from the overall population I: i.e. $I = \{A, B...\}$. Say, we expect the outcome to be

$$E\{\Delta Y_i\} = E\{Y_i(1) - Y_i(0)\} = E\{Y_i(1)\} - E\{Y_i(0)\}.$$
 (3)

We can approximate this theoretical effect by treating individuals a from A, and compare their average to the one of untreated individuals $b \in B$:

$$E\{\Delta Y_i\} \approx E\{Y_a(1)\} - E\{Y_b(0)\} \tag{4}$$



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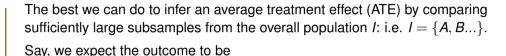
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In this case we exploit *random chance* within sufficiently large samples that makes these groups comparable. Such a setting can be generated by randomized controlled experiments.



Structural Causal Models I

by directed acyclic graphs (DAG). For example:

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Directed acyclic graph where D affects Y. Image source: modified from Huntington-Klein 2018



Another approach is to specify the assumed causal relation within a system

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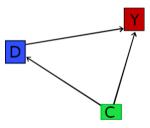
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Another approach is to specify the assumed causal relation within a system by directed acyclic graphs (DAG). For example:



Directed acyclic graph where D affects Y. Image source: modified from Huntington-Klein 2018



Directed acyclic graph where variable C affects both D and Y. Image source: Huntington-Klein 2018

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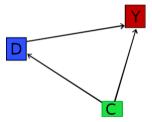
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Directed acyclic graph where D affects Y. Image source: modified from Huntington-Klein 2018

Directed acyclic graph where variable C affects both D and Y. Image source: Huntington-Klein 2018

In the second case we need to close the <u>back-door path</u> by controlling for *C*.

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Judea Pearl et al. (**Pearl2016**) developed the do-calculus to express the effect of an intervention you *do*:



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Judea Pearl et al. (**Pearl2016**) developed the do-calculus to express the effect of an intervention you *do*:

P(Y|D) is the *conditional probability* of Y given D.



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Judea Pearl et al. (**Pearl2016**) developed the do-calculus to express the effect of an intervention you *do*:

P(Y|D) is the conditional probability of Y given D.

If we have a confounding variable C and we want an unbiased estimate of intervention D's effects on Y, we shall control for C and assess the probability of Y given both D and C:



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P(Y|D) is the conditional probability of Y given D.

If we have a confounding variable C and we want an unbiased estimate of intervention D's effects on Y, we shall control for C and assess the probability of Y given both D and C:

$$P(\mathbf{Y}|do(D)) = \sum_{C} P(\mathbf{Y}|D,C)P(C)$$
 (5)



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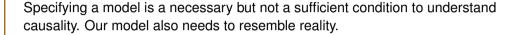
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Randomista:

Specifying a model is a necessary but not a sufficient condition to understand causality. Our model also needs to resemble reality.

We therefore need an understanding of the underlying *mechanism*.



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Specifying a model is a necessary but not a sufficient condition to understand causality. Our model also needs to resemble reality.

We therefore need an understanding of the underlying *mechanism*.

"Causal processes, causal interactions, and causal laws provide the mechanisms by which the world works; to understand why certain things happen, we need to see how they are produced by these mechanisms."

Salmon1984 as cited in Samantha Kleinberg Causal Inference, lecture 9



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Randomista:

Specifying a model is a necessary but not a sufficient condition to understand causality. Our model also needs to resemble reality.

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"Causal processes, causal interactions, and causal laws provide the mechanisms by which the world works; to understand why certain things happen, we need to see how they are produced by these mechanisms."

Salmon1984 as cited in Samantha Kleinberg Causal Inference, lecture 9

My take: \rightarrow *We need theory!* Theory can be developed (and tested) through many (inductive & deductive) methods.



Ontology - Epistemology - Theory

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Plato's allegory of the cave. Image source: Studio Binder 2020

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Do you have developed an intuition for the following?

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Do you have developed an intuition for the following?

■ How large numbers of obervations allow more robust inference?



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Do you have developed an intuition for the following?

- How large numbers of obervations allow more robust inference?
- That correlation does not imply causation?



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Do you have developed an intuition for the following?

- How large numbers of obervations allow more robust inference?
- That correlation does not imply causation?
- That causal analysis require some form of framework to ...
 - formulate hypothesis
 - test hypothesis ?



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- How large numbers of obervations allow more robust inference?
- That correlation does not imply causation?
- That causal analysis require some form of framework to ...
 - formulate hypothesis
 - test hypothesis ?
- That quantitative causal inference needs theory / an understanding of the causal mechanism to work?



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■ 1747 James Lind conducted a clinical trial on the treatment of scurvy

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■ 19th century: experimental psychology (Wilhelm Wundt)



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■ 1747 James Lind conducted a clinical trial on the treatment of scurvy

- 19th century: experimental psychology (Wilhelm Wundt)
- up to early 20th century: experimental sociology (Comte vs. Hegel vs. Marx)



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- Ronald Fisher's 1935 *Design of Experiments* (agricultural field experiments)
- since 1960's standard for approval of medicine (double blind clinical trials)



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- since 1960's standard for approval of medicine (double blind clinical trials)
- 1970's RAND Health Insurance Experiment (cf. Angrist2015)



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- 2019 Nobel Prize in Economics to *randomistas* (Banerjee2011)



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- 1970's RAND Health Insurance Experiment (cf. Angrist2015)
- 2019 Nobel Prize in Economics to randomistas (Banerjee2011)

References and further reading

- RCTs: Pearce2014; DeSouzaLeao2019; Jamison2019
- Experiments in a broader sense, cf. Wikipedia, Britannica

In order to assess the effect of a "treatment" (of sorts), we can

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In order to assess the effect of a "treatment" (of sorts), we can

■ take two random samples from a population

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In order to assess the effect of a "treatment" (of sorts), we can

- take two random samples from a population
- treat one, and compare it to the other (as if "counterfactual")

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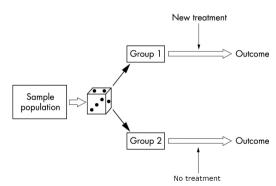
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Conservation



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- take two random samples from a population
- treat one, and compare it to the other (as if "counterfactual")



Schematic outline of a randomized controlled trial. Image source: Adapted from **Kendall2003**

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Experiments – statistical approach

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Recall that we approach the average treatment effect (ATE) by comparing sufficiently large subsamples from the overall population: i.e. $I = \{A, B...\}$.



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Recall that we approach the average treatment effect (ATE) by comparing sufficiently large subsamples from the overall population: i.e. $I = \{A, B...\}$.

To approximate this treatment effect we can treat individuals $a \in A$, and compare their average to the one of untreated individuals $b \in B$. This is called a *difference-in-means* estimator:

$$E\{\Delta Y_i\} \approx E\{Y_a(1)\} - E\{Y_b(0)\}$$
 (6)



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Random chance in sufficiently large samples makes these groups comparable (remember the law of large numbers).



Experiments – graphical approach

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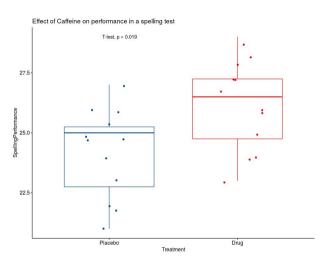
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A hypothetical experiment. Image source: Adapted from personality-project

Experiments – methodological note of caution

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When designing an experiment we need a big enough sample size.

What is big enough can be calculated based on

- false positive probability (e.g. no more than 5%)
- minimum detectable effect (MDE)
- the power required at MDE (e.g. 80%)

Reference: cf. Coleman2018 or Ramesh Johari MS&E 226 lecture 18



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RESEARCH ARTICLE

ECONOMICS

Cash for carbon: A randomized trial of payments for ecosystem services to reduce deforestation

Seema Jayachandran, 18 Joost de Laat, 2 Eric F. Lambin, 3,4 Charlotte Y. Stanton, 5 Robin Audy, 6 Nancy E. Thomas 7

We evaluated a program of payments for ecosystem services in Uganda that offered forestowning households annual payments of 70,000 Ugandan shillings per hectare if they conserved their forest. The program was implemented as a randomized controlled trial in 121 villages, 60 of which received the program for 2 years. The primary outcome was the change in land area covered by trees, measured by classifying high-resolution satellite imagery. We found that tree cover declined by 4.2% during the study period in treatment villages, compared to 9.1% in control villages. We found no evidence that enrollees shifted their deforestation to nearby land. We valued the delayed carbon dioxide emissions and found that this program benefit is 2.4 times as large as the program costs.

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Deforestation contributes to climate change.

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Deforestation contributes to climate change.

■ As much land is private, paying land users for conservation (a public good) is a common approach.



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Deforestation contributes to climate change.

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- Payments for ecosystem services (PES) schemes became popular (in the developing world).



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Deforestation contributes to climate change.

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- Jayachandran et al. (Jayachandran2017) run an experiment in Uganda (third highest deforestation rate in the world 2005-2010)



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- They
 - pay 563 private forest owners (PFO) in 60 treated villages (there are 535 PFO in 61 control villages), and
 - monitor deforestation rates by satelite imagery.

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 - monitor deforestation rates by satelite imagery.
- Jayachandran et al. (**Jayachandran2017**) analyse the effect on tree cover.

Results

Primary outcomes

Village boundaries

	TimeBe neumanites		
	∆Tree cover (ha)	∆Tree cover (ha)	∆Log of tree cover
	(1)	(2)	(3)
Treatment group	5.549*	5.478**	0.0521**
	[2.888]	[2.652]	[0.021]
Control group	-13.371	-13.371	-0.095
Control variables	No	Yes	Yes
Observations	121	121	121

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Results

Secondary outcomes

Increased patrolling Has any fence around IHS of food IHS of nonfood Allow others Cut any trees in to gather firewood of the forest in land with expend. in past expend. in past the past year from own forest last 2 years natural forest 30 days 30 days (1) (2) (3) (4)(5) (6) -0.140*** -0.170*** 0.109*** 0.036 0.156** Treatment group 0.065 [0.034] [0.039] [0.074] [0.066] Lee bound (lower) -0.161*** -0.185*** 0.094** 0.007 -0.0290.053 [0.034] [0.039] [0.064] Lee bound (upper) -0.104*** -0 148*** 0.132*** 0.055 0.144* 0.215*** FO 0341 FO.0391 FO.0751 TO.0641 Control group mean 0.453 0.427 0.378 0.667 2.524 4.363 Control group SD F0.4981 TO.4951 TO.485 TO.4721 T1.3541 Observations 1018 9767 984 1020 1020 Observations (Lee bounds) 994 957 965 998 998 998

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Paying forest owners for conservation reduces deforestation rates

 \rightarrow No afforestation is observable at that payment level.



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Results

Paying forest owners for conservation reduces deforestation rates

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"This study also adds to the literature on PES [by]"

"satellite images with very high resolution, which enables us to detect selective tree-cutting in addition to clear-cutting"



Javachandran2017

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Results

Paying forest owners for conservation reduces deforestation rates

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"This study also adds to the literature on PES [by]"

- "satellite images with very high resolution, which enables us to detect selective tree-cutting in addition to clear-cutting"
- "cost-benefit analysis allows policy-makers to assess the cost-effectiveness of the PES program in comparison to other options for reducing global carbon emissions"

Javachandran2017

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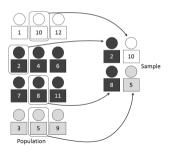
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A note on methods

The authors used a *stratification* strategy to ensure balanced randomization:

number of PFOs



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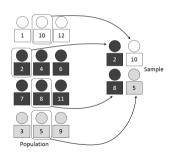
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- av. household earnings / capita



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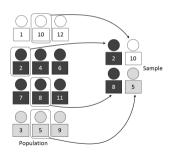
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- av. household earnings / capita
- distance to a road, and



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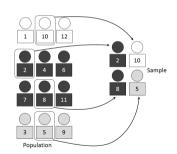
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- average landsize



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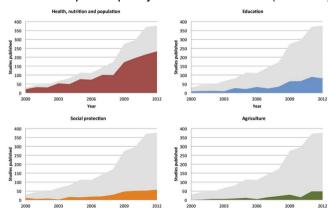
Discussion questions

- Do you think it is a good idea to pay private land owners for the provision of public goods ecosystem services?
- Do you think it is a market-based approach if the government pays it? If so why?
- What could be alternative approaches to ensure provision of public (environmental) goods?



Randomistas on a roll

Increased use of development policy evaluation studies (or RCTs)



Share of quasi-experimental studies in color. Image source: Cameron et al. **Cameron2016**, cf. **Tollefson2015**

Year



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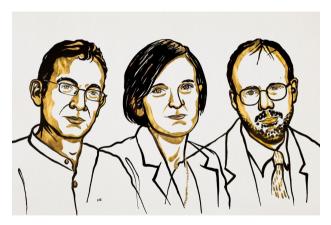
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Banerjee, Duflo & Kremer win the 2019 Nobel Prize in Economics. Image source: Sverige Riksbank

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■ Some treatments cause ethical concern.

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■ RCTs target individuals not structural causes.



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- RCTs target individuals not structural causes.
- A solid design is needed for internal and external validity.



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- RCTs target individuals not structural causes.
- A solid design is needed for internal and external validity.
- There is / was a replication crisis and p-hacking.



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- RCTs target individuals not structural causes.
- A solid design is needed for internal and external validity.
- There is / was a replication crisis and p-hacking.
- There can be secondary, unintended outcomes.



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- RCTs target individuals not structural causes.
- A solid design is needed for internal and external validity.
- There is / was a replication crisis and p-hacking.
- There can be secondary, unintended outcomes.
- Experiments can be costly.



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Experiments do not deliver all answers (cf. Howe2004)

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A fuller picture may be provided by mixed method research (cf. Imai2011; Latour2012; Blok2014)



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- A fuller picture may be provided by mixed method research (cf. Imai2011; Latour2012; Blok2014)
- Observational data and identification strategies provide alternative quantitative approaches for causal inference (cf. Gelman2014)



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- Experiments do not deliver all answers (cf. Howe2004)
- A fuller picture may be provided by mixed method research (cf. Imai2011; Latour2012; Blok2014)
- Observational data and identification strategies provide alternative quantitative approaches for causal inference (cf. Gelman2014)
- ightarrow My own take: I believe more systematical experiments in the implementation of policies can increase effectiveness (compared to trial-and-error)

Econometricians

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MASTER JOSHWAY: In a nutshell, please, Grasshopper.

GRASSHOPPER: Causal inference compares potential outcomes, descriptions of the world when alternative roads are taken.

MASTER JOSHWAY: Do we compare those who took one road with those who took another?

GRASSHOPPER: Such comparisons are often contaminated by selection bias, that is, differences between treated and control subjects that exist even in the absence of a treatment effect.

MASTER JOSHWAY: Can selection bias be eliminated?

GRASSHOPPER: Random assignment to treatment and control conditions eliminates selection bias. Yet even in randomized trials, we check for balance.

MASTER JOSHWAY: Is there a **single causal truth**, which all randomized investigations are sure to reveal?

GRASSHOPPER: I see now that there can be **many truths**, Master, some compatible, some in contradiction. We therefore take special note when findings from two or more experiments are similar.

Angrist2015, own emphasis

Experiment lessons

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- (internal and external) validity
- ethical implications
- Interpretation of the results is a big part of the story / political recommendation.



Further readings

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- Behaghel, L., Macours, K., & Subervie, J. (2019). How can randomised controlled trials help improve the design of the common agricultural policy? *European Review of Agricultural Economics*, 46(3), 473-493. doi: 10.1093/erae/jbz021
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