

Causal Inference in Environmental & Social Science

Nils Droste

2021 ClimBEco course



Structure of the Course

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model Structural Causal Models Mechanism

Controlled Trials

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Empirics Conservation

Randomistas

References



	time	Day 1: May 10, 2021	Day 2: May 11, 2021	Day 3: May 12, 2021	Day 4: May 13, 2021	Day 5: May 14, 2021
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: Marcus & Sant'Anna (2020) JAERE	Replication: Bayer & Aklin (2020) PNAS	Replication: Kim & Urpelainen (2017) RPP	Student presenta- tions
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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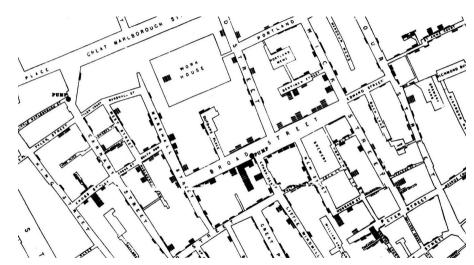
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John Snow's original dot map of the 1854 Broad street cholera outbreak. Image sources & info: wikipedia

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 (2009), 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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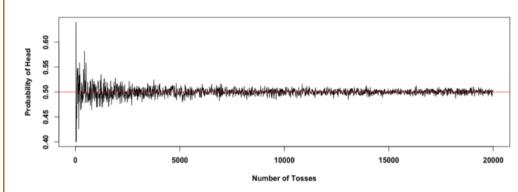
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Is the coin fair?



 \rightarrow 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 Angrist and Pischke 2015, (p. xi), 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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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*?

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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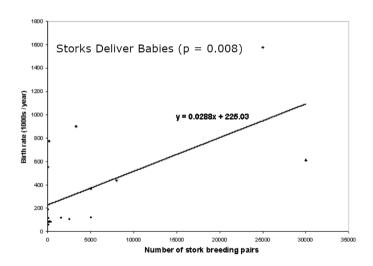
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Do storcks deliver babies? Image source: Matthews (2000)

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 (2000)

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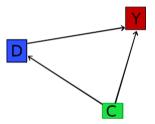
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Directed acyclic graph where variable C affects both textcolorblueD and Y. Image source: Modified from Huntington-Klein 2018

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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.

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

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

$$\Delta Y_i = Y_i(1) - Y_i(0) \tag{2}$$

■ 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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$$\Delta Y_i = Y_i(1) - Y_i(0) \tag{2}$$

- This approach requires to think in terms of "counterfactuals".
- While theoretically ideal, the identification and the measurement of a pure counterfactual is logically impossible:
- We can only observe one state of the world, i.e. we cannot *directly* measure what would have happened in the counterfactual case (cf. Holland 1986).

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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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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)

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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 $b \in B$:

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

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

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

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References



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

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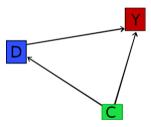
References



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



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

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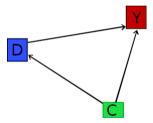
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Another approach is to specify the assumed causal relation within a system by directed acyclic graphs (DAG). For example:





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

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. (2016) developed the do-calculus to express the effect of an intervention you *do*:

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Judea Pearl et al. (2016) 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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References



Judea Pearl et al. (2016) 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:

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

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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.

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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*.

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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."

Salmon 1984 as cited in Samantha Kleinberg Causal Inference, lecture 9

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Salmon 1984 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?

■ How large numbers of obervations allow more robust inference?

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

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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?

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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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- 1747 James Lind conducted a clinical trial on the treatment of scurvy
- 19th century: experimental psychology (Wilhelm Wundt)

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

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- 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. Angrist and Pischke 2015)

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- 2019 Nobel Prize in Economics to randomistas (Banerjee and Duflo 2011)

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- 2019 Nobel Prize in Economics to *randomistas* (Banerjee and Duflo 2011)

References and further reading

- RCTs: **DeSouzaLeao2019**; **Jamison2019**; Pearce and Raman 2014
- 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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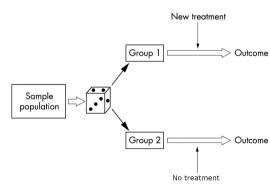
Deference



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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")



Schematic outline of a randomized controlled trial. Image source: Adapted from Kendall 2003

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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)

Experiments – statistical approach

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

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

Experiments – graphical approach

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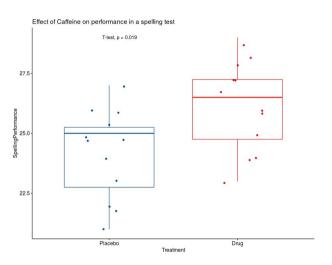
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A hypothetical experiment. Image source: Adapted from personality-project

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. Coleman 2018 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, 1: 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.

- As much land is private, paying land users for conservation (a public good) is a common approach.
- Payments for ecosystem services (PES) schemes became popular (in the developing world).

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- As much land is private, paying land users for conservation (a public good) is a common approach.
- Payments for ecosystem services (PES) schemes became popular (in the developing world).
- Jayachandran et al. (2017) run an experiment in Uganda (third highest deforestation rate in the world 2005-2010)

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Deforestation contributes to climate change.

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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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- 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.
- Jayachandran et al. (2017) analyse the effect on tree cover.

Results

Primary outcomes

Treatment group

Control group
Control variables
Observations

Village boundaries

 Δ Tree Δ Tree Δ Log of cover (ha) cover (ha) tree cover

(1)	(2)	(3)
5.549*	5.478**	0.0521**
2.888]	[2.652]	[0.021]
3 371	-13 371	-0.095

Yes Yes 121 121

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Results

Secondary outcomes

	Cut any trees in the past year	Allow others to gather firewood from own forest	Increased patrolling of the forest in last 2 years	Has any fence around land with natural forest	IHS of food expend. in past 30 days	IHS of nonfood expend. in past 30 days
	(1)	(2)	(3)	(4)	(5)	(6)
Treatment group	-0.140***	-0.170***	0.109***	0.036	0.065	0.156**
	[0.034]	[0.033]	[0.039]	[0.033]	[0.074]	[0.066]
Lee bound (lower)	-0.161***	-0.185***	0.094**	0.007	-0.029	0.053
	[0.034]	[0.033]	[0.039]	[0.033]	[0.070]	[0.064]
Lee bound (upper)	-0.104***	-0.148***	0.132***	0.055	0.144*	0.215***
	[0.033]	[0.032]	[0.039]	[0.034]	[0.075]	[0.064]
Control group mean	0.453	0.427	0.378	0.667	2.524	4.363
Control group SD	[0.498]	[0.495]	[0.485]	[0.472]	[1.177]	[1.354]
Observations	1018	9767	984	1020	1020	1020
Observations (Lee bounds)	994	957	965	998	998	998



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Results

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

 \rightarrow No afforestation is observable at that payment level.

"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"

Jayachandran et al. 2017, p. 6

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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"

Jayachandran et al. 2017, p. 6

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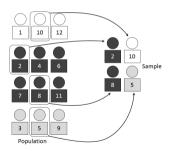
Conservation



A note on methods

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

number of PFOs



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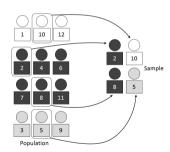
References



A note on methods

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

- number of PFOs
- av. household earnings / capita



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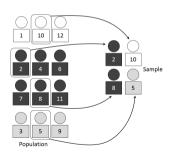
Conservation



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

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



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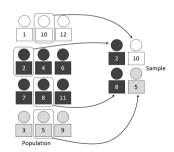
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A note on methods

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- distance to a road, and
- average landsize



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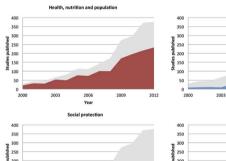


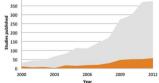
Discussion questions

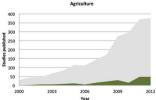
- Do you think it is a good idea to pay private land owners for the provision of public goods ecosystem services?
- Do you think it is a market-based approach if the government pays it? If so why?
- What could be alternative approaches to ensure provision of public (environmental) goods?

Randomistas on a roll

Increased use of development policy evaluation studies (or RCTs)







2006

Year

2009

2012

Education

Share of quasi-experimental studies in color. Image source: Cameron et al. 2016, cf. Tollefson 2015



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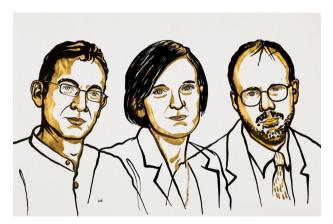
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Banerjee, Duflo & Kremer win the 2019 Nobel Prize in Economics.
Image source: Sverige Riksbank

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■ Some treatments cause ethical concern.

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- Some treatments cause ethical concern.
- RCTs target individuals not structural causes.

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- Some treatments cause ethical concern.
- RCTs target individuals not structural causes.
- A solid design is needed for internal and external validity.

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- Some treatments cause ethical concern.
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- There is / was a replication crisis and p-hacking.

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- Some treatments cause ethical concern.
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- There can be secondary, unintended outcomes.

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- Some treatments cause ethical concern.
- 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. Howe 2004)

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- Experiments do not deliver all answers (cf. Howe 2004)
- A fuller picture may be provided by mixed method research (cf. Imai et al. 2011; Latour et al. 2012; Blok and Pedersen 2014)

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- Experiments do not deliver all answers (cf. Howe 2004)
- A fuller picture may be provided by mixed method research (cf. Imai et al. 2011; Latour et al. 2012; Blok and Pedersen 2014)
- Observational data and identification strategies provide alternative quantitative approaches for causal inference (cf. Gelman 2014)

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- Experiments do not deliver all answers (cf. Howe 2004)
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- Observational data and identification strategies provide alternative quantitative approaches for causal inference (cf. Gelman 2014)
- 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.

Angrist and Pischke 2015, (p. 30), own emphasis

Experiment lessons

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- The design of the experiments matter for it's
 - (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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