



LUND
UNIVERSITY

Causal Inference in Environmental & Social Science

Nils Droste

2022 ClimBEco course



Structure of the Course

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Neyman-Rubin Model

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	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, <i>Introduction to Causal inference</i> , and randomized controlled trials	<i>(Semi) Natural Experiments</i> : Panel data regressions, two-way fixed effects, and recent corrections for staggered treatment	<i>Simulated Counterfactuals</i> : matching methods, synthetic controls, and Bayesian Structural time series	<i>Instruments & Interruptions</i> : instrumental variables, regression discontinuity design	<i>Cutting edges</i> : Structural equation modelling for causal inference (and machine learning techniques?)
Seminars	13-15h	<i>Replication</i> : Jayachandran et al. (2017) <i>Science</i>	<i>Replication</i> : Card & Krueger (1994) <i>JAERE</i>	<i>Replication</i> : LaLonde (1986) <i>PNAS</i>	<i>Replication</i> : Abou-Chadi & Krause (2020) <i>RPP</i>	Student presentations
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?



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



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Albert Einstein (1953), as cited in Pearl (2009), my emphasis

My interpretation:

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



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Albert Einstein (1953), as cited in Pearl (2009), my emphasis

My interpretation:

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

→ 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?



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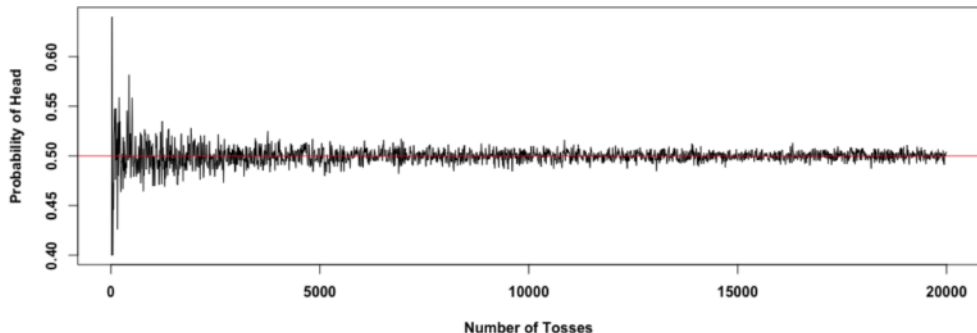
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Is the coin fair?



→ The law of large numbers allows to approximate "*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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MASTER PO: *Young man, **how is it that you do not?***

Kung Fu, Pilot. Cited from Angrist and Pischke 2015, (p. xi), own emphasis

→ 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



Causation

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

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References

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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References

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



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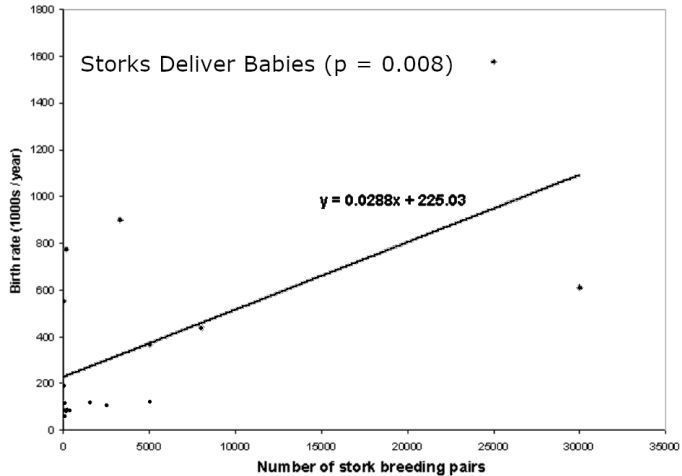
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Do storcks deliver babies? Image source: Matthews (2000)

Example II: Storcks & Babies

What happened, why did we get it so wrong?

Country	Area (km ²)	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**.

$$Y_i = \alpha + \beta_1 D_i + \beta_2 C_i + \varepsilon_i \quad (1)$$



Problem I: Confounding variables

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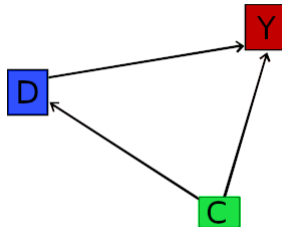
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Directed acyclic graph where variable **C** affects both **D** and **Y**. Image source: Modified from Huntington-Klein 2018

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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:

- $D = 1$ 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:

- $D = 1$ if i has received treatment;
- $D = 0$ if i has *not* received treatment.

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

- $Y_i(1)$ is the outcome in case of treatment;
- $Y_i(0)$ is the outcome in case of *no* treatment.

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The hypothetical outcome for each unit can be written as

$$\Delta Y_i = Y_i(1) - Y_i(0) \quad (2)$$



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The hypothetical outcome for each unit can be written as

$$\Delta Y_i = Y_i(1) - Y_i(0) \quad (2)$$

- This approach requires to think in terms of “*counterfactuals*”.



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The hypothetical outcome for each unit can be written as

$$\Delta Y_i = Y_i(1) - Y_i(0) \quad (2)$$

- 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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The hypothetical outcome for each unit can be written as

$$\Delta Y_i = Y_i(1) - Y_i(0) \quad (2)$$

- 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).

Neyman-Rubin Model III

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

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



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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. $I = \{A, B, \dots\}$.

Say, we expect the outcome to be

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

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

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

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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. $I = \{A, B, \dots\}$.

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

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

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.

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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](#)



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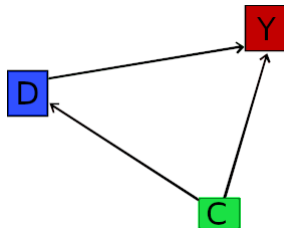
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Another approach is to specify the assumed causal relation within a system by directed acyclic graphs (DAG). For example:



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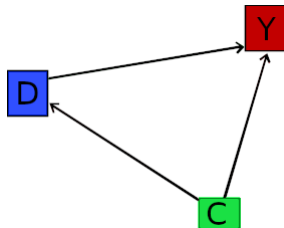
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Another approach is to specify the assumed causal relation within a system by directed acyclic graphs (DAG). For example:



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 back-door path 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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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 .



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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 .

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 :

$$P(Y|do(D)) = \sum_C P(Y|D, C)P(C) \quad (5)$$



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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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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](#)

My take: → ***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 observations allow more robust inference?



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Do you have developed an intuition for the following?

- How large numbers of observations 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 observations 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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Do you have developed an intuition for the following?

- How large numbers of observations 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?

The history of randomized controlled trials (RCT)

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



The history of randomized controlled trials (RCT)

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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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- 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)



The history of randomized controlled trials (RCT)

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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)
- Ronald Fisher's 1935 *Design of Experiments* (agricultural field experiments)



The history of randomized controlled trials (RCT)

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- since 1960's standard for approval of medicine (double blind clinical trials)



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- 1970's RAND Health Insurance Experiment (cf. Angrist and Pischke 2015)



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- 1970's RAND Health Insurance Experiment (cf. Angrist and Pischke 2015)
- 2019 Nobel Prize in Economics to *randomistas* (Banerjee and Duflo 2011)

References and further reading

- RCTs: Pearce and Raman 2014; de Souza Leão and Eyal 2019; Jamison 2019
- Experiments in a broader sense, cf. [Wikipedia](#), [Britannica](#)

Experiments – the ”gold” standard

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

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Experiments – the "gold" standard

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

- take two random samples from a population

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Experiments – the "gold" standard

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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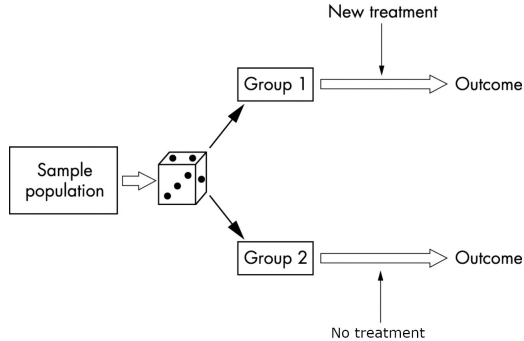
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Experiments – the "gold" standard

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. $I = \{A, B\dots\}$.



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

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:

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

Experiments – statistical approach

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

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

Experiments – graphical approach

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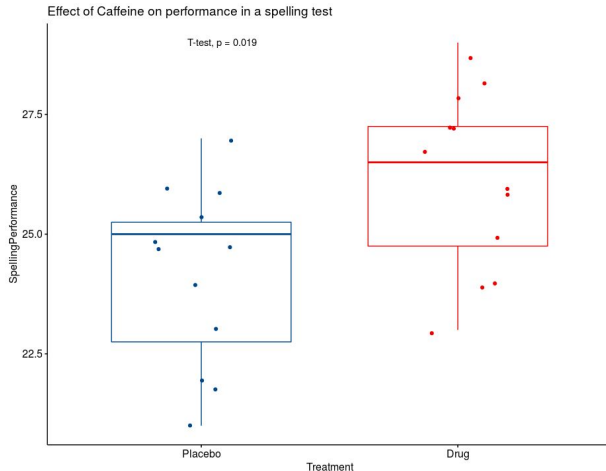
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A hypothetical experiment. Image source: Adapted from [personality-project](#)

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. Thomas⁷

We evaluated a program of payments for ecosystem services in Uganda that offered forest-owning 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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Paying households for conservation

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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- They
 - pay 563 private forest owners (PFO) in 60 treated villages (there are 535 PFO in 61 control villages), and
 - monitor deforestation rates by satellite 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 satellite imagery.
- Jayachandran et al. (2017) analyse the effect on tree cover.

Paying households for conservation

Results

Primary outcomes

Village boundaries

	Δ Tree cover (ha)	Δ Tree cover (ha)	Δ 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



Paying households for conservation

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*** [0.034]	-0.170*** [0.033]	0.109*** [0.039]	0.036 [0.033]	0.065 [0.074]	0.156** [0.066]
Lee bound (lower)	-0.161*** [0.034]	-0.185*** [0.033]	0.094** [0.039]	0.007 [0.033]	-0.029 [0.070]	0.053 [0.064]
Lee bound (upper)	-0.104*** [0.033]	-0.148*** [0.032]	0.132*** [0.039]	0.055 [0.034]	0.144* [0.075]	0.215*** [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



Paying households for conservation

Results

Paying forest owners for conservation reduces deforestation rates
→ 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”

Jayachandran et al. 2017, p. 6



Paying households for conservation

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



Paying households for conservation

A note on methods

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

■ number of PFOs

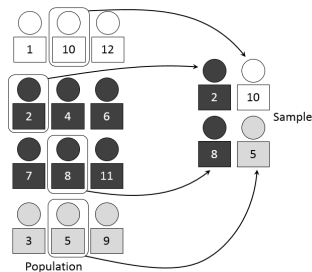


Image source: [wikipedia](https://en.wikipedia.org/wiki/File:Stratification_diagram.png)



Paying households for conservation

A note on methods

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- number of PFOs
- av. household earnings / capita

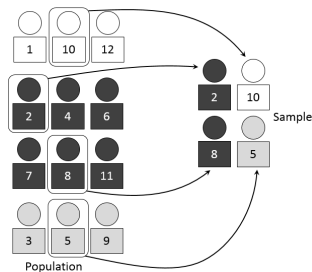


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Paying households for conservation

A note on methods

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

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

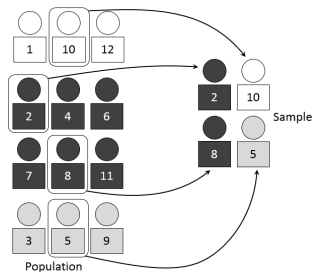


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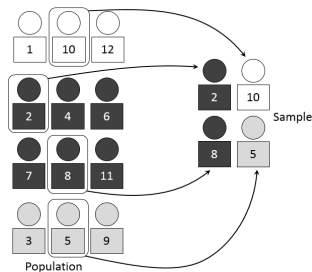


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Discussion questions

- 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)

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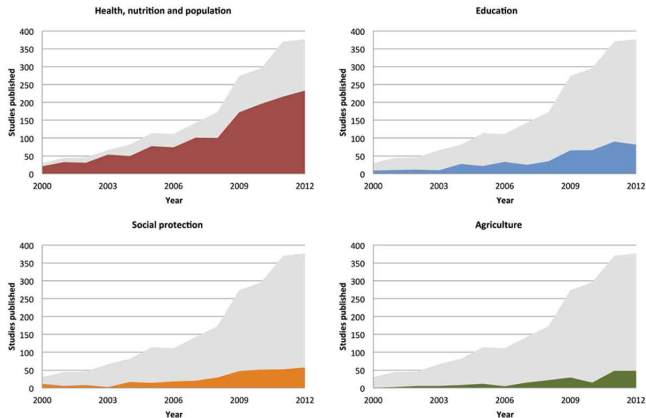
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Share of quasi-experimental studies in color. Image source: Cameron et al. 2016, cf. Tollefson 2015



Randomistas on a roll

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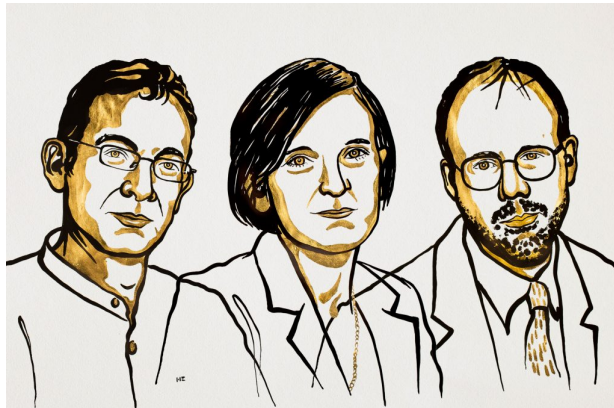
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Banerjee, Duflo & Kremer win the 2019 Nobel Prize in Economics.

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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- 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.



Not all that glitters is gold

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- Some treatments cause ethical concern.
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- 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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References

- 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.



A rejoinder

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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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- Observational data and identification strategies provide alternative quantitative approaches for causal inference (cf. Gelman 2014)



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- Observational data and identification strategies provide alternative quantitative approaches for causal inference (cf. Gelman 2014)

→ *My own take*: I believe more systematical experiments in the implementation of policies can increase effectiveness (compared to trial-and-error)

Econometricians

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



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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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References III

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

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