



LUND
UNIVERSITY

Causal Inference in Environmental & Social Science

Nils Droste

2023 ClimBEco course



Structure of the Course

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model
Structural Causal Models
Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References

	time	Day 1: April 24, 2023	Day 2: April 25, 2023	Day 3: April 26, 2023	Day 4: April 27, 2023	Day 5: April 28, 2023
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					



Learning Contract

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References



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

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References



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

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model
Structural Causal Models
Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References

*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

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model
Structural Causal Models
Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References

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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.



Motivation – Greater minds' answers

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model
Structural Causal Models
Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References



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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

Motivation

Epistemes

Causation

Theory

- Neyman-Rubin Model
- Structural Causal Models
- Mechanism

Controlled Trials

Design

Empirics

- Conservation

Randomistas

References



Is the coin fair?



A short detour into probability

Motivation

Epistemes

Causation

Theory

- Neyman-Rubin Model
- Structural Causal Models
- Mechanism

Controlled Trials

Design

Empirics

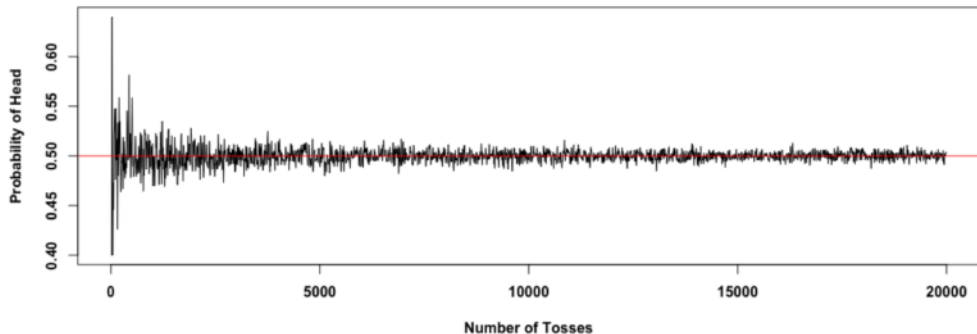
- Conservation

Randomistas

References



Is the coin fair?



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

Epistemological & Ontological Foundations

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References

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



Epistemological & Ontological Foundations

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References

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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).



Epistemological & Ontological Foundations

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model
Structural Causal Models
Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References

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

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



Epistemological & Ontological Foundations

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References

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 .

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References



Causation

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*?

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References



Causation

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

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References



Causation

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model
Structural Causal Models
Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

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

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

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model
Structural Causal Models
Mechanism

Controlled Trials

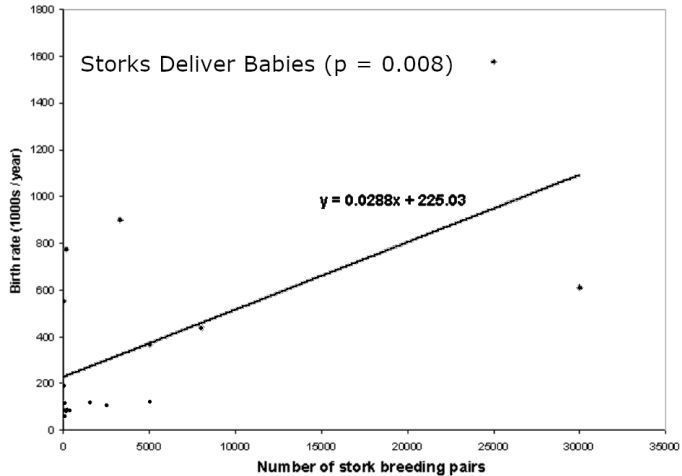
Design

Empirics

Conservation

Randomistas

References



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

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model
Structural Causal Models
Mechanism

Controlled Trials

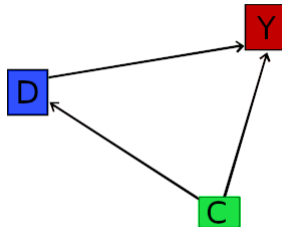
Design

Empirics

Conservation

Randomistas

References



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

Neyman-Rubin Model I

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References

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.



Neyman-Rubin Model I

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References

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:



Neyman-Rubin Model I

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References

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.



Neyman-Rubin Model I

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References



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.

Neyman-Rubin Model II

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References

The hypothetical outcome for each unit can be written as

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



Neyman-Rubin Model II

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References

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*”.



Neyman-Rubin Model II

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References



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:

Neyman-Rubin Model II

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References



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

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References



Neyman-Rubin Model III

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References

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

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



Neyman-Rubin Model III

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References



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 $b \in B$:

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

Neyman-Rubin Model III

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References



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.

Structural Causal Models I

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

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](#)



Structural Causal Models I

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

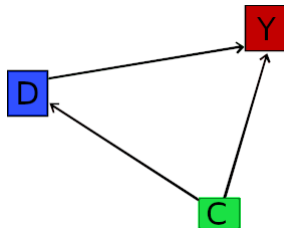
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Another approach is to specify the assumed causal relation within a system by directed acyclic graphs (DAG). For example:



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](#)

Structural Causal Models I

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

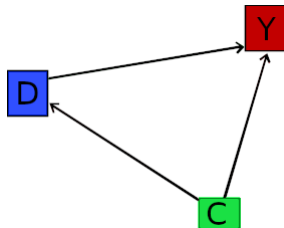
References



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](#)



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**.

Structural Causal Models II

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References

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



Structural Causal Models II

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

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 .



Structural Causal Models II

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

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 :



Structural Causal Models II

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References

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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 :

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



Mechanism

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References

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



Mechanism

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References

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***.



Mechanism

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References



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](#)

Mechanism

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References



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

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model
Structural Causal Models
Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References



Plato's allegory of the cave. Image source: [Studio Binder 2020](#)

Intermediate lessons

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References

Do you have developed an intuition for the following?



Intermediate lessons

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References

Do you have developed an intuition for the following?

- How large numbers of observations allow more robust inference?



Intermediate lessons

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References

Do you have developed an intuition for the following?

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



Intermediate lessons

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References



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 ?

Intermediate lessons

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References



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)

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References



The history of randomized controlled trials (RCT)

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References

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



The history of randomized controlled trials (RCT)

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References

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



The history of randomized controlled trials (RCT)

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References

- 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)

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References

- 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)

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model
Structural Causal Models
Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References

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



The history of randomized controlled trials (RCT)

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model
Structural Causal Models
Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References

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



The history of randomized controlled trials (RCT)

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model
Structural Causal Models
Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References

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The history of randomized controlled trials (RCT)

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model
Structural Causal Models
Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References

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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

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References



Experiments – the "gold" standard

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

- take two random samples from a population

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References



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")

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

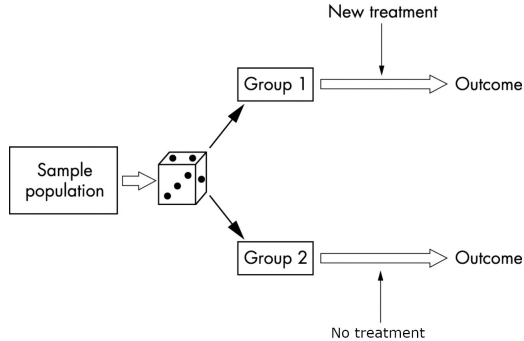
References



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



Experiments – statistical approach

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References

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



Experiments – statistical approach

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model
Structural Causal Models
Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References



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:

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

Experiments – statistical approach

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model
Structural Causal Models
Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References



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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This is called a *difference-in-means* estimator:

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

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

Experiments – graphical approach

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model
Structural Causal Models
Mechanism

Controlled Trials

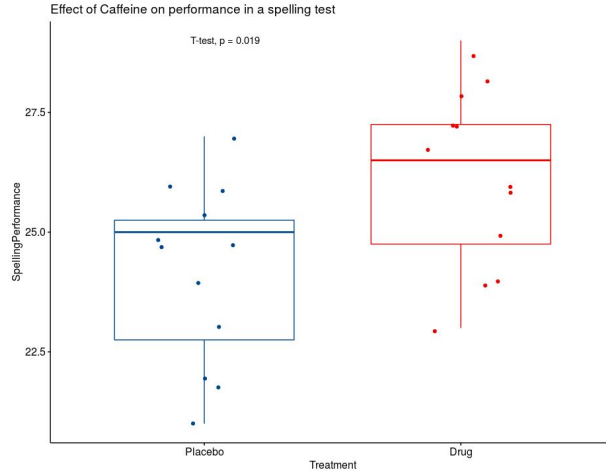
Design

Empirics

Conservation

Randomistas

References



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

Experiments – methodological note of caution

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References

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](#)



Paying households for conservation

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References

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.



Paying households for conservation

Deforestation contributes to climate change.

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References



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.

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References



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).

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model
Structural Causal Models
Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References



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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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- Jayachandran et al. (2017) run an experiment in Uganda (third highest deforestation rate in the world 2005-2010)

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model
Structural Causal Models
Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References



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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.

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model
Structural Causal Models
Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References



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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.

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model
Structural Causal Models
Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References



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.

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References



Paying households for conservation

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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
→ 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”
- ”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

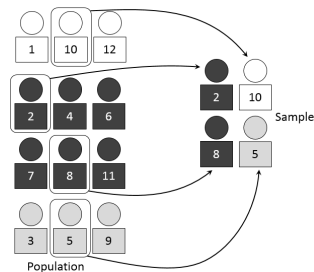


Image source: [wikipedia](https://en.wikipedia.org/wiki/Stratified_sampling)



Paying households for conservation

A note on methods

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- number of PFOs
- av. household earnings / capita

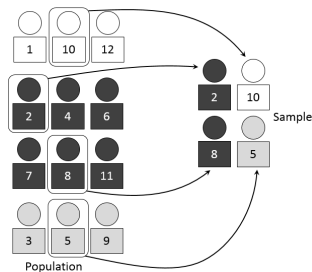


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Paying households for conservation

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

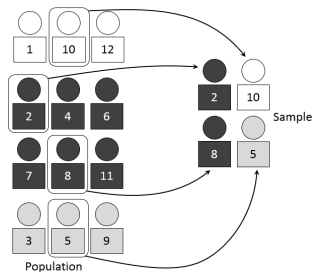


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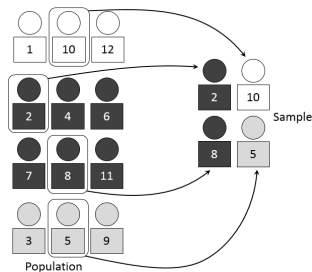


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Paying households for conservation

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model
Structural Causal Models
Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References



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)

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

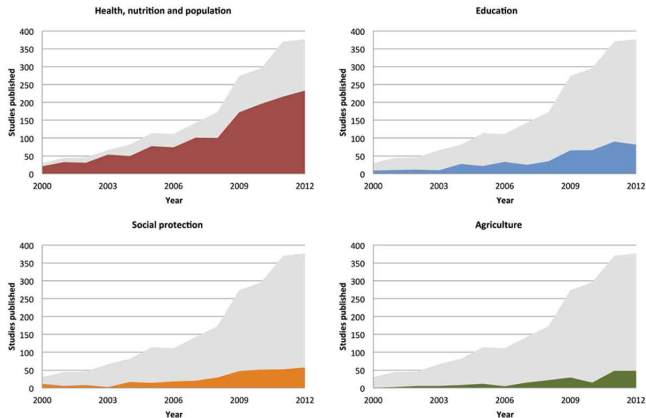
Design

Empirics

Conservation

Randomistas

References



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



Randomistas on a roll

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

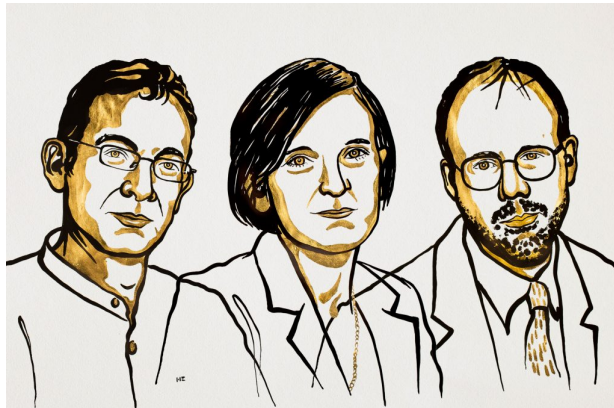
Design

Empirics

Conservation

Randomistas

References



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

Image source: [Sverige Riksbank](#)



Not all that glitters is gold

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model
Structural Causal Models
Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References

- Some treatments cause ethical concern.



Not all that glitters is gold

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model
Structural Causal Models
Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References

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



Not all that glitters is gold

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References

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



Not all that glitters is gold

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model
Structural Causal Models
Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References

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- There is / was a replication crisis and p-hacking.



Not all that glitters is gold

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model
Structural Causal Models
Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References

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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.



Not all that glitters is gold

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

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

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References

- Experiments do not deliver all answers (cf. Howe 2004)



A rejoinder

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model
Structural Causal Models
Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References

- 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)



A rejoinder

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References

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



A rejoinder

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References



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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



Experiment lessons

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References

- 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

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References

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References I

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model
Structural Causal Models
Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

References

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References II

Motivation

Epistemes

Causation

Theory

Neyman-Rubin Model

Structural Causal Models

Mechanism

Controlled Trials

Design

Empirics

Conservation

Randomistas

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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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