PS207 Quantitative Causal Inference, Spring 2018

Introduction + Potential Outcomes Model

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UC Santa Barbara

Special thanks to Chad Hazlett (UCLA) for select slides, used with permission

Three Modes of Statistical Inference

1. Descriptive Inference: summarizing and exploring data
   - Inferring “ideal points” from roll-call votes
   - Inferring “topics” from texts and speeches
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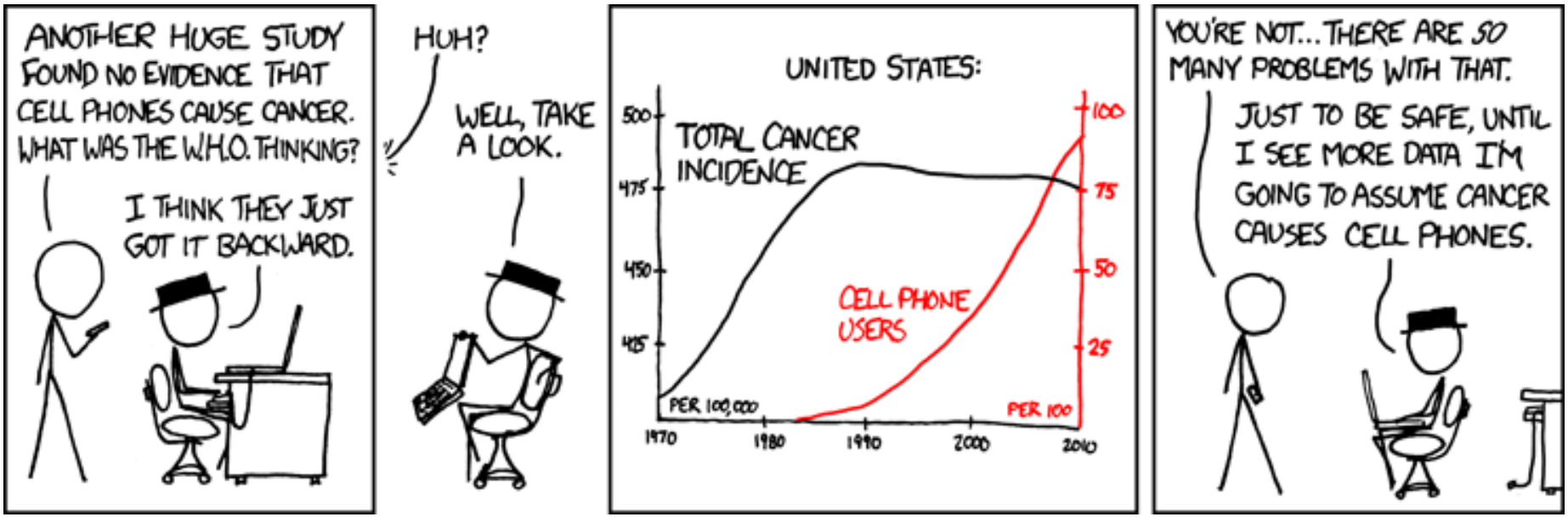
Causal inference is the most difficult!

(because of the assumptions it requires, not the math)

Correlation $\neq$ Causation



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- Among them, the Nurses Health Study involved tens of thousands of participants, showed a 30% lower risk of heart attack, and was basis for wide scale prescription of estrogen replacement
- Grodstein and Stampfer (1998):

  *"Consistent evidence from over **40** epidemiologic studies demonstrates that postmenopausal women who use estrogen therapy after the menopause have significantly lower rates of heart disease than women who do not take estrogen. . . the evidence clearly supports a clinically important protection against heart disease for postmenopausal women who use estrogen."*

Case Study: Post-Menopausal Estrogen

April 22, 2003

Hormone Studies: What Went Wrong?

By GINA KOLATA

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- Why do you think the results were so different?

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- Experiment showed 40% *increase* in risk of heart attack; also increased risk of breast cancer, and dementia.
- Had to halt experiment, issue new advice to physicians
- Why do you think the results were so different?
- Later randomized trials:
  - reaffirmed the WHI result, showed it holds for estrogen-only replacement
  - showed the risk depends on age and other factors.

A Traditional Approach

John Stuart Mill's **Method of Difference**:

> *If an instance in which the phenomenon under investigation occurs, and an instance in which it does not occur, have every circumstance in common save one, that one occurring only in the former; the circumstance in which alone the two instances differ is the effect, or the cause, or an indispensable part of the cause, of the phenomenon.*

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This could work, but we rarely have such comparisons in the social sciences...and worse, data cannot in general tell us when this holds.

We need a way to know when the data we have will allow us to estimate a causal effect: **identification**.

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Something is **identifiable** if you can get it from the joint distribution of observable random variables.

Causal Identification (Manski 1995)

> *...it is useful to separate the inferential problem into statistical and identification components. Studies of **identification** seek to characterize the conclusions that could be drawn if one could...obtain an unlimited number of observations. Identification problems cannot be solved by gathering more [data].*

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Manski continues,

> *"The study of identification logically comes first. Negative identification findings imply that statistical inference is fruitless... positive identification findings imply that one should go on to study the feasibility of statistical inference."*

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We are interested in causal identification: the combination of assumptions and data needed to make a causal conclusion.

$$\text{Assumptions} + \text{Data} \rightarrow \text{Causal Conclusions}$$

Design-based inference

Many assumptions are best made reasonable by *design*.

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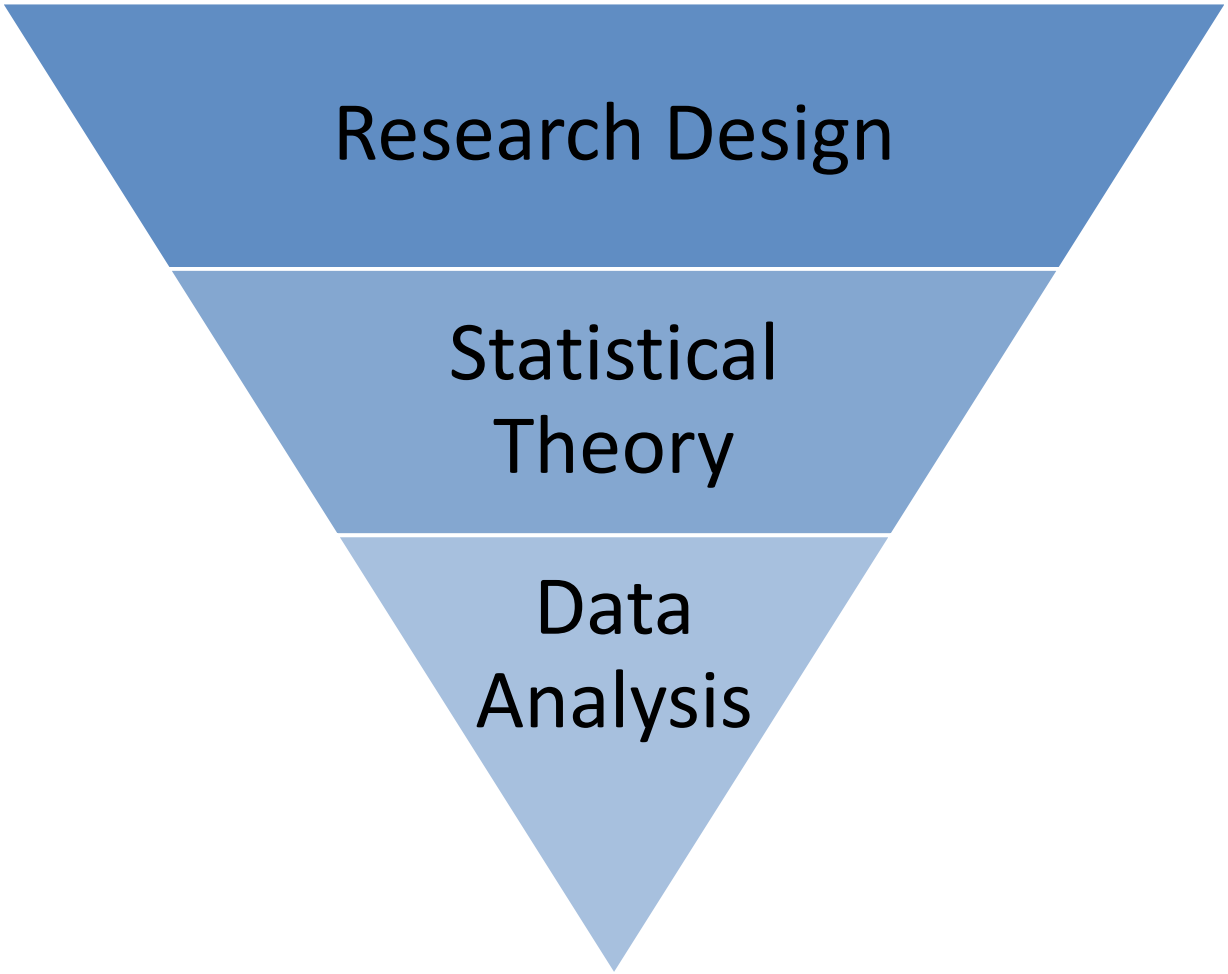
Hence, “design-based inference”:



Questions Answered in This Course

- Why do we accept results from randomized experiments as causal? What makes experiments so special?
- In social sciences we often cannot run randomized experiments. Can we still make a causal inference?
- Can observational studies ever produce causal conclusions? If so, when?
- Some have said statistics is only useful for analyzing association, not causation. Is that not true?
- *How can I publish a paper in a top social science journal?*

WBER editors Alain de Janvry and Elisabeth Sadoulet:

Question: Any pet peeves with submissions or with referees that it would be good for people to avoid?

> *Unfortunately yes. Our main two criteria in selecting papers for publication are rigorous identification and policy relevance. The two go together as we cannot have credible policy recommendations without strong causal inference. Too many of the submitted papers offer simple "determinants" that are partial correlates with no causal value, and yet are the basis for bold policy recommendations... This includes a large number of cross-country panel regressions with only mechanical, and hence not credible, identification, and yet eventually huge claims of policy implications.*

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  - design your research to maximize the credibility of causal claims
  - know where a study falls on the credibility spectrum
  - know tools for testing, maximizing the credibility of causal claims.
  - goal is NOT to tell you you cannot study certain topics, rather to let you answer questions of interest in the best way possible.

Roadmap for the Course

- Introduction and review
- Potential outcome model
- Randomized experiments, natural experiments
- Causal inference under "selection on observables"
  - matching
  - weighting
  - propensity scores
  - regression
  - extensions: effect heterogeneity, mediation, mechanism
- Sensitivity analyses
- Parallel-trend related approaches
  - difference in difference
  - panel methods
  - synthetic controls
- Instrumental variables
- Discontinuity designs
- Qualitative causal inference (e.g. process-tracing)

Readings

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- Angrist, Joshua D. and Jörn-Steffen Pischke. 2009. *Mostly Harmless Econometrics: An Empiricist's Companion*. Princeton University Press.

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Other assigned articles and chapters: will be posted on course website as necessary

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- A short research paper, typically applying a method learned in this course to an empirical problem of substantive interest.
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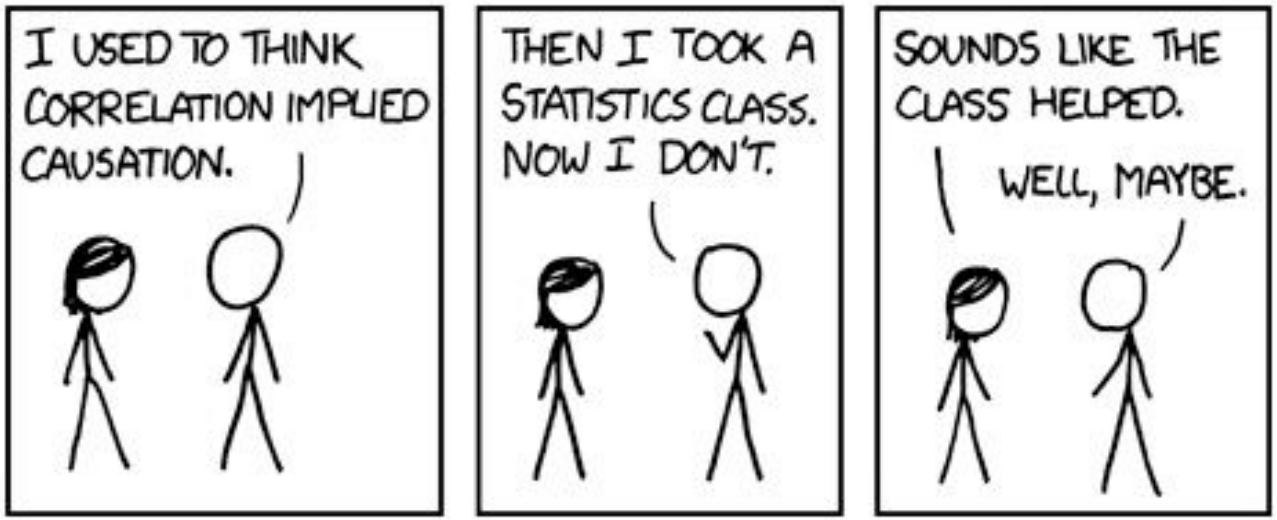
- Key attribute: *follow the guidelines of the course in terms of maximizing and characterizing the credibility of causal claims*
- Coauthoring strongly encouraged!
- We will likely have class presentations, but depends on size

Paper Milestones

- Week 3: submit teams and (if possible) a notional topic or title for your project
- Week 5: submit a brief description of your project
- Week 7: submit a two-page progress report on your project
- Finals Week, submit final project by email to mildenberger@polsci.ucsb.edu

Housekeeping

- Course website (Gauchospace): Slides and code, psets, data sets
- Interrupt any time if you have a question! If something is unclear to you it is probably unclear to others.
- Problem set submissions: give hard copy of write-up to me. Problem sets will be completed in RMarkdown, so you will simply need to print out your executed code.
- Office hours: open-door and by appointment


I USED TO THINK CORRELATION IMPLIED CAUSATION.
THEN I TOOK A STATISTICS CLASS. NOW I DON'T.
SOUNDS LIKE THE CLASS HELPED.
WELL, MAYBE.

Questions?

Purpose, Scope, and Examples

Goal in causal inference is to assess the causal effect some potential cause (e.g. an institution, intervention, policy, or event) on some outcome.

Examples of such research questions include: What is the effect of

- political institutions on corruption?
- voting technology on voting fraud?
- incumbency status on vote shares?
- peace-keeping missions on peace?
- mass media on voter preferences?
- church attendance on turnout?

The Neyman-Rubin Potential Outcome Model

Much of the progress on causal inference in recent years made possible by the Neyman-Rubin causal model, aka the Potential Outcomes Model (POM).

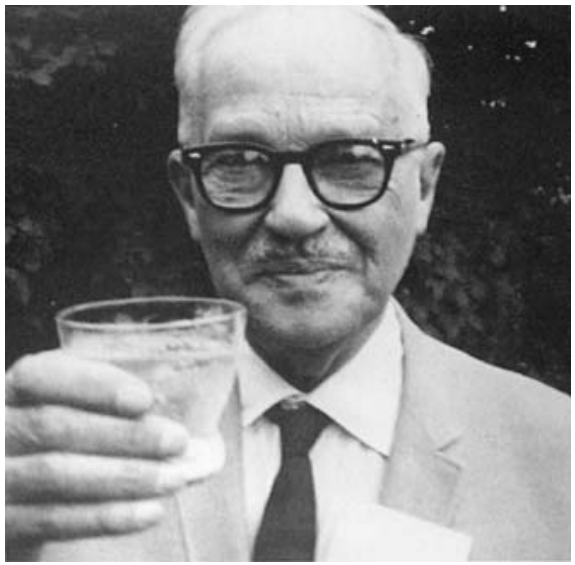


Figure: Neyman

Figure: Rubin

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- The trouble is you do not get to see both Y_{1i} and Y_{0i} for unit i. This is the fundamental problem of causal inference.
- Everything we do will be about filling in the "missing" potential outcome for each unit through various assumptions and statistical tricks. That is it!

Fundamental Quantities (so far)

- Y_{1i} and Y_{0i}, the potential outcomes
- D_i, the treatment for unit i
- Observed outcome, $Y_i = D_i Y_{1i} + (1 - D_i) Y_{0i}$
- $\tau_i = Y_{1i} - Y_{0i}$
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Warning: regression goggles

- tempting to think of the (observed) Y_i as "responding" to observed D_i.
- but here we see the observed Y_i as just a revelation of either Y_{1i} or Y_{0i}.
- Put differently, the potential outcomes $\{Y_{1i}, Y_{0i}\}$
  - are not *changed* by D_i.
  - they may even be independent of D_i (which is great)
  - D_i just switches which one you can see.

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- So can we estimate $\mathbb{E}[Y_{1i}]$? And $\mathbb{E}[Y_{0i}]$?
- What does a sample estimator of $\mathbb{E}[Y_i|D_i = 1] - \mathbb{E}[Y_i|D_i = 0]$ tell us?

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- If you estimate $\mathbb{E}[Y_i|D_i = 1] - \mathbb{E}[Y_i|D_i = 0]$, what will we get? Does this tell us anything about $\mathbb{E}[Y_{1i} - Y_{0i}]$?

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Scenario 3: Suppose you don't know anything about how D_i is assigned w.r.t. Y_{1i} and Y_{0i}.

- Can we say anything about how $\mathbb{E}[Y_i|D_i = 1] - \mathbb{E}[Y_i|D_i = 0]$ compares to $\mathbb{E}[Y_{1i} - Y_{0i}]$?

Quick Intuitions II

Scenario 2: Suppose $Y_{1i} = Y_{0i}$ for all i. But now, someone sees Y_{1i}, and chooses $D_i = 1$ more often when Y_{1i} is higher.

- How would the Y_{1i} you see compare to Y_{1i} you don't see?
- So does $\mathbb{E}[Y_{1i}|D_i = 1] = \mathbb{E}[Y_{1i}|D_i = 0] = \mathbb{E}[Y_{1i}]$?
- How would the Y_{0i} you see compare to the ones you don't?
- If you estimate $\mathbb{E}[Y_i|D_i = 1] - \mathbb{E}[Y_i|D_i = 0]$, what will we get? Does this tell us anything about $\mathbb{E}[Y_{1i} - Y_{0i}]$?

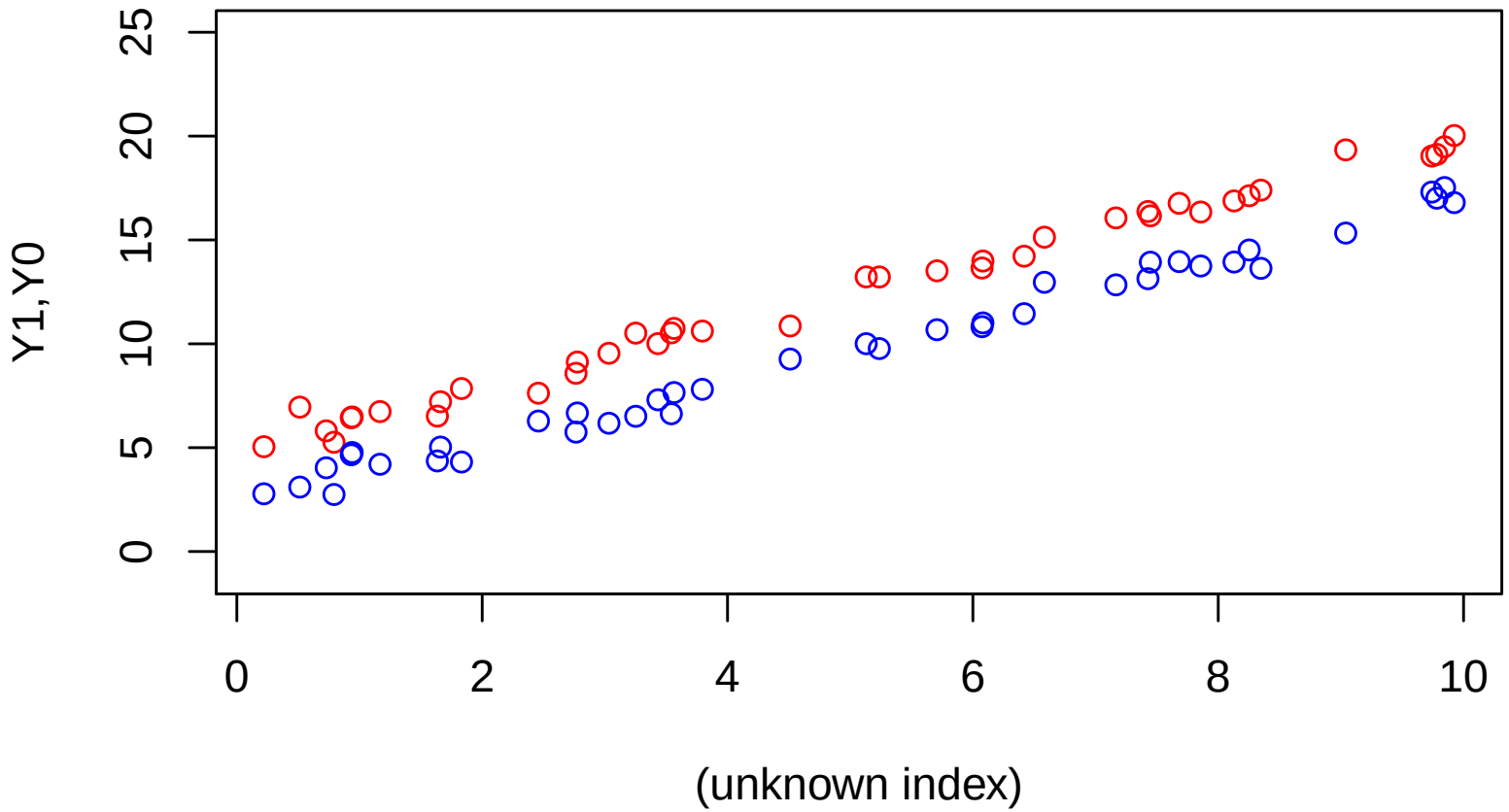
Scenario 3: Suppose you don't know anything about how D_i is assigned w.r.t. Y_{1i} and Y_{0i}.

- Can we say anything about how $\mathbb{E}[Y_i|D_i = 1] - \mathbb{E}[Y_i|D_i = 0]$ compares to $\mathbb{E}[Y_{1i} - Y_{0i}]$?

Which of these scenarios are we usually in with observational data?

Visual Practice

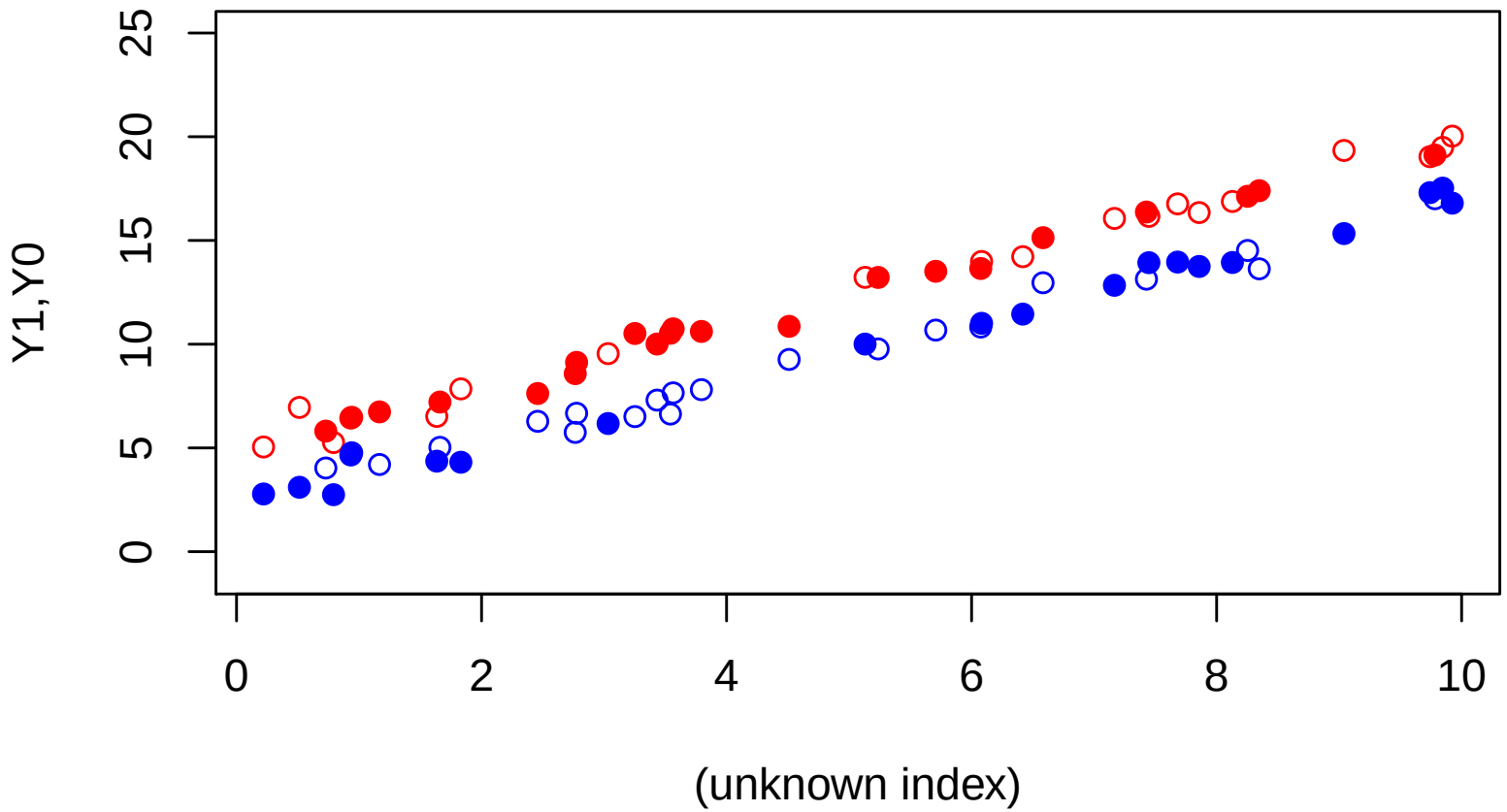
Let's looks at Y_{1i} and Y_{0i} alone first.



True $\mathbb{E}[Y_{1i} - Y_{0i}] = ATE = 3$

Random Assignment of Treatment

Now suppose $D_i = 1$ is randomly assigned. We see only the filled dots:



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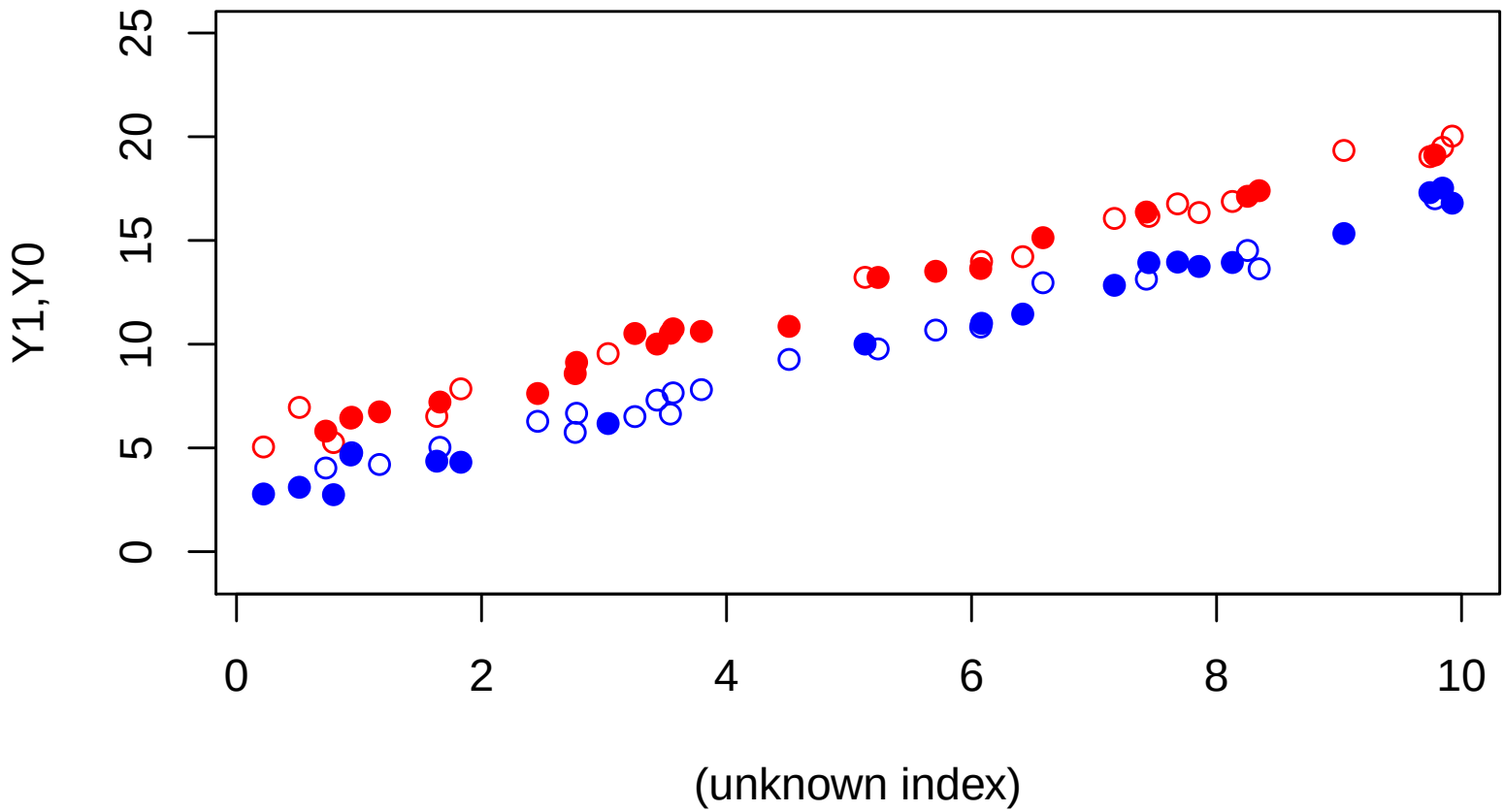
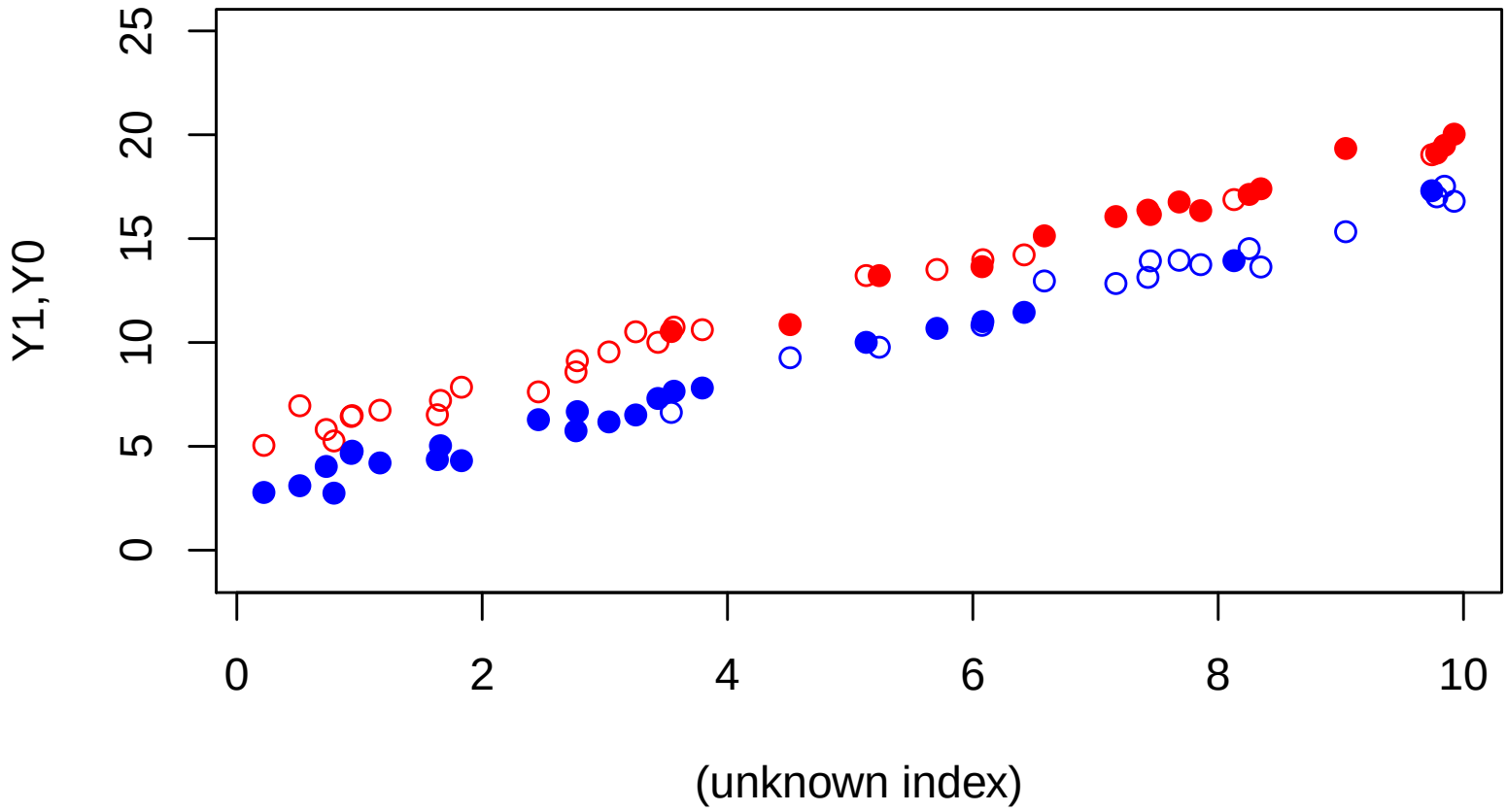


We get

$$\hat{\mathbb{E}}[Y_i|D_i = 1] - \hat{\mathbb{E}}[Y_i|D_i = 0] = \widehat{ATE} = 3.01$$

Non-Random Assignment of Treatment 1

Suppose $Pr(D_i = 1)$ increases to the right. Now we observe (filled dots):



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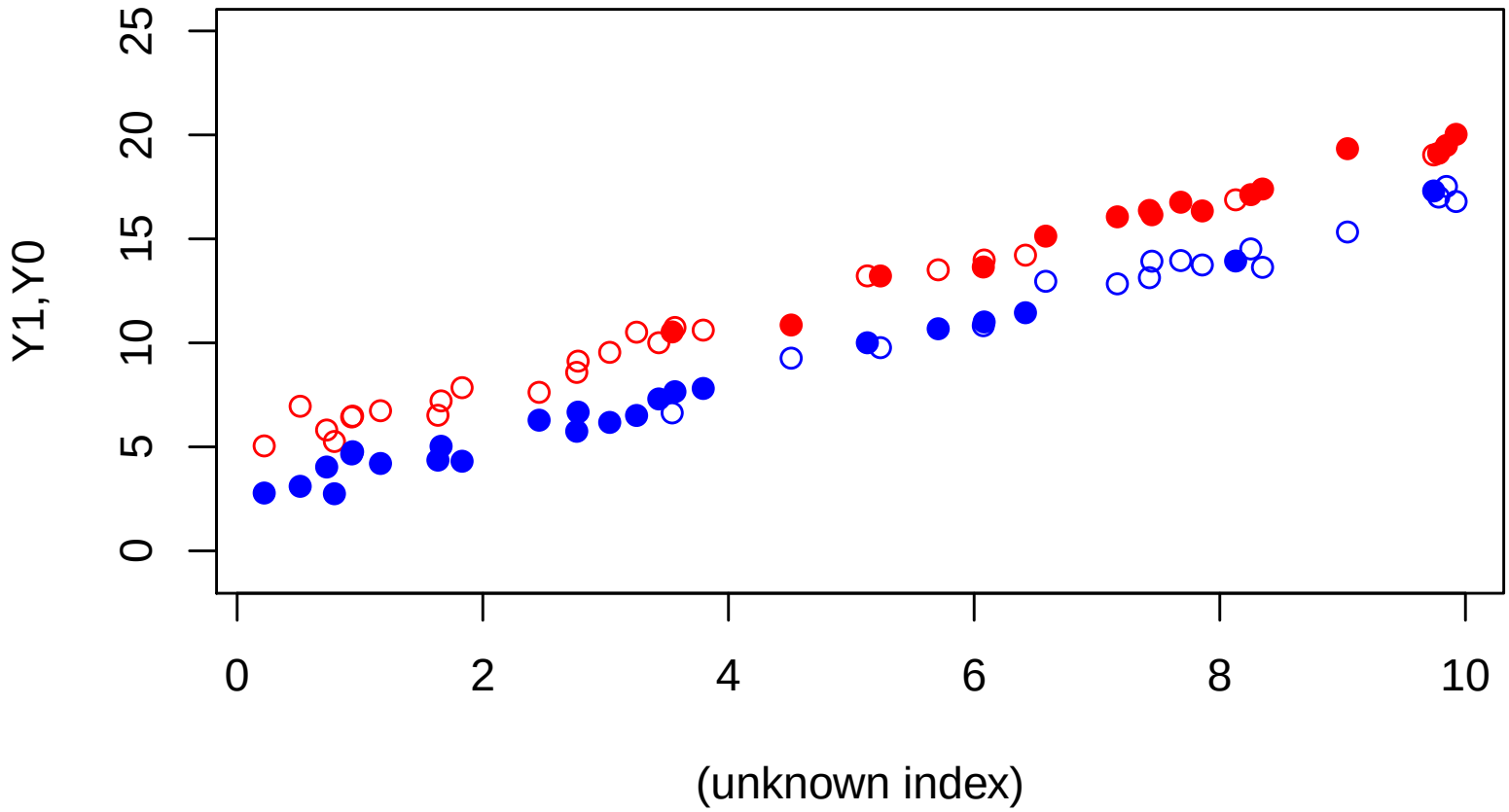

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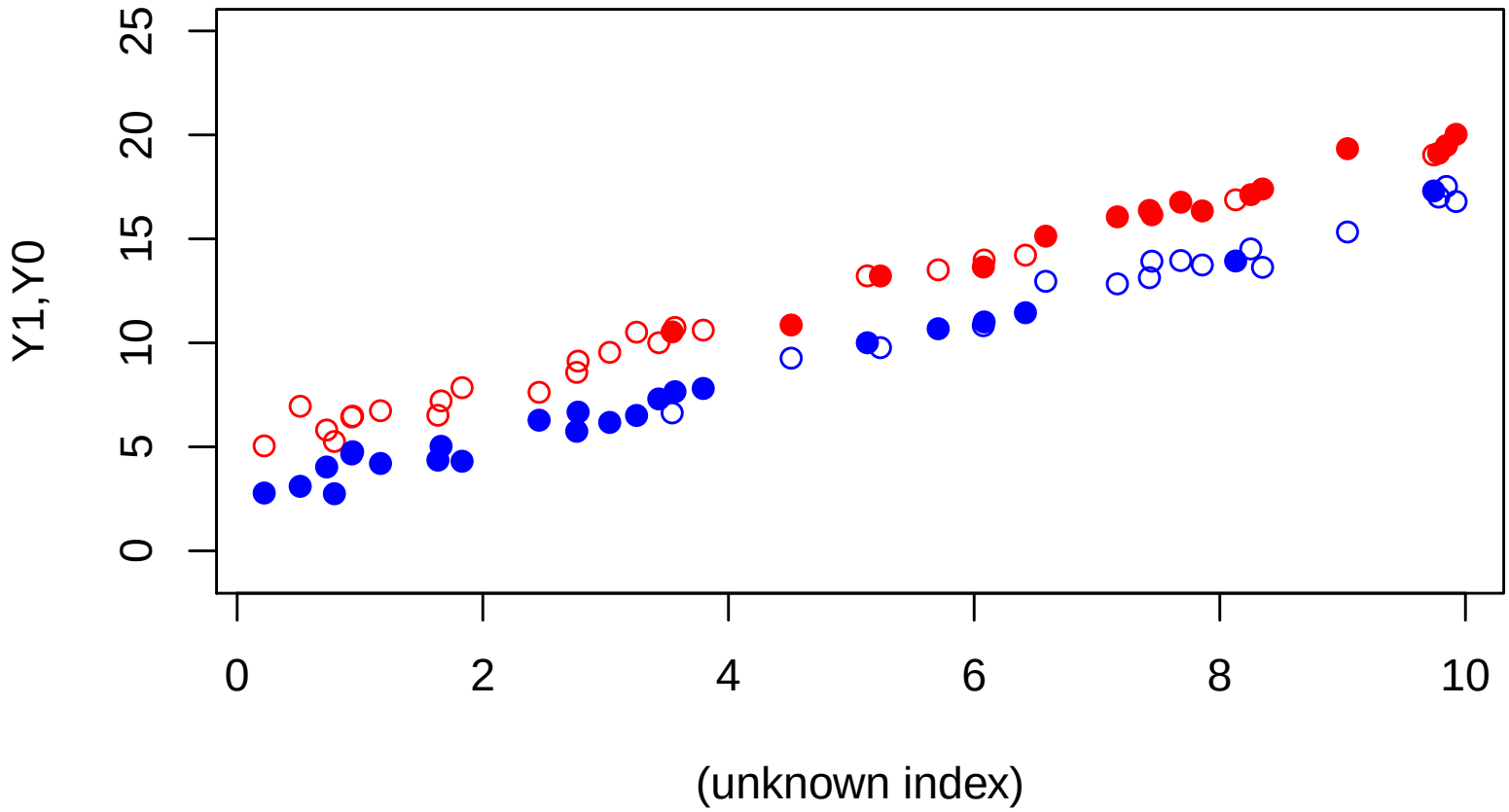

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$$\hat{\mathbb{E}}[Y_i|D_i = 1] - \hat{\mathbb{E}}[Y_i|D_i = 0] = \widehat{ATE} = 9.97$$

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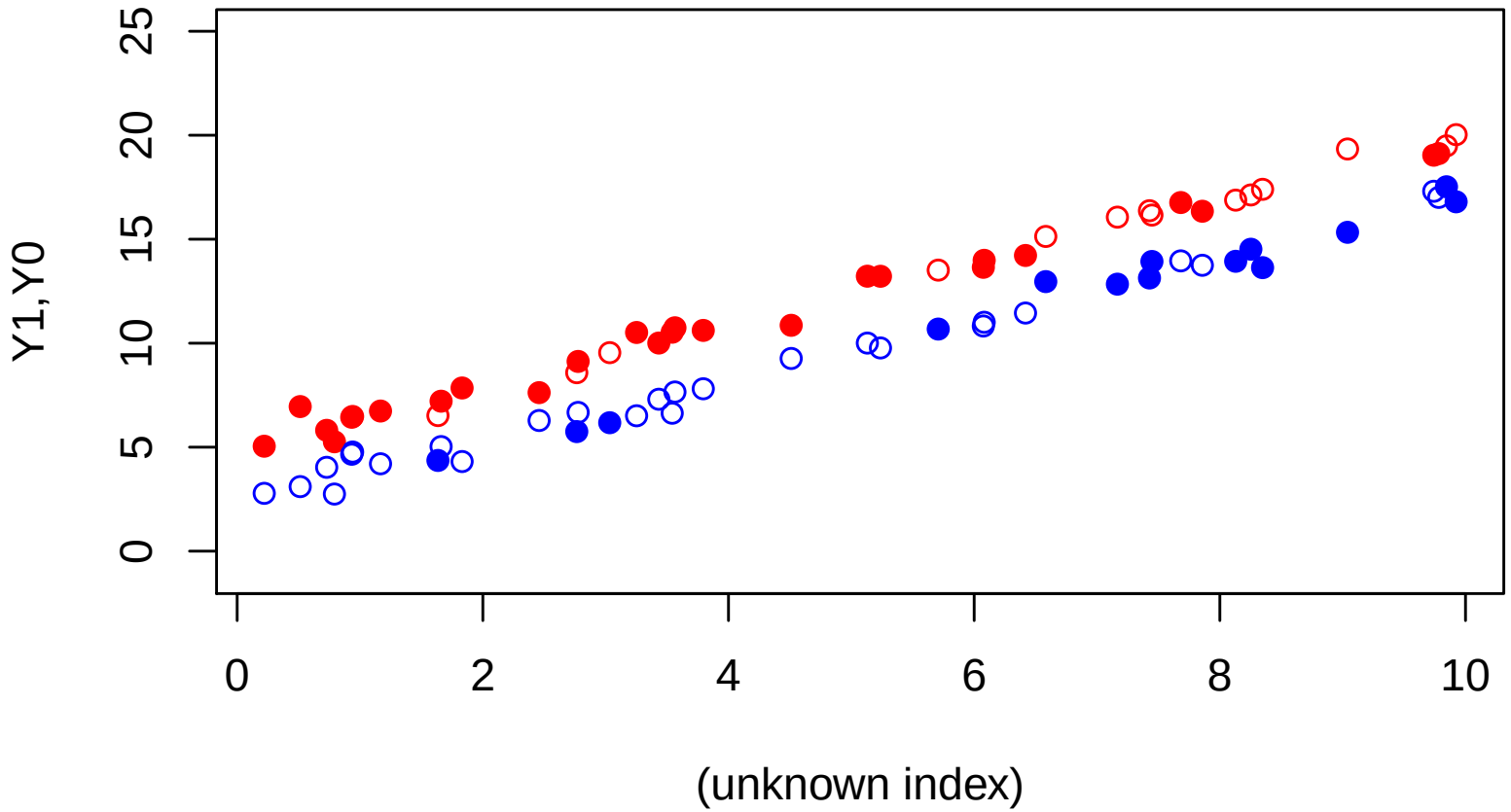

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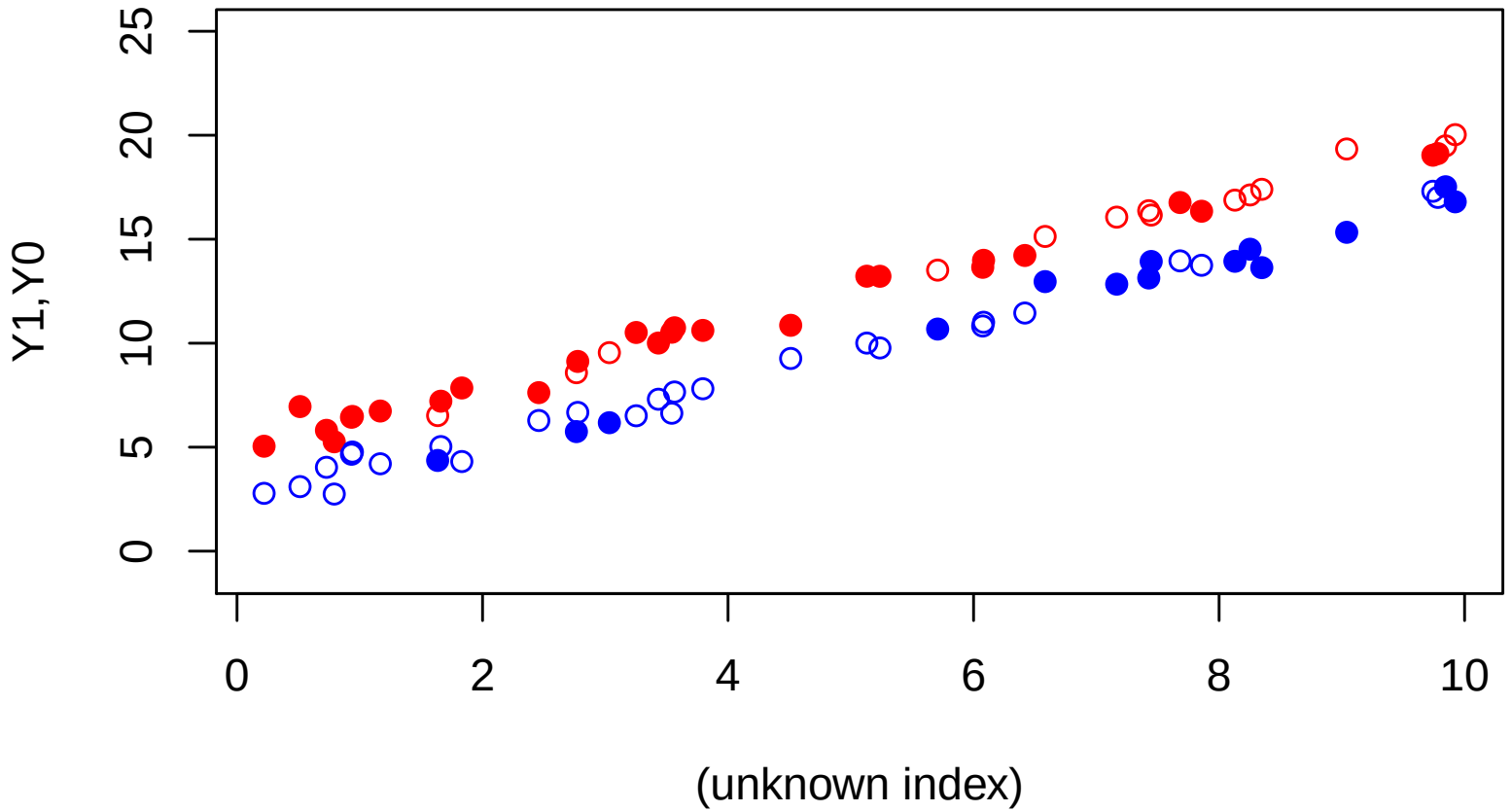

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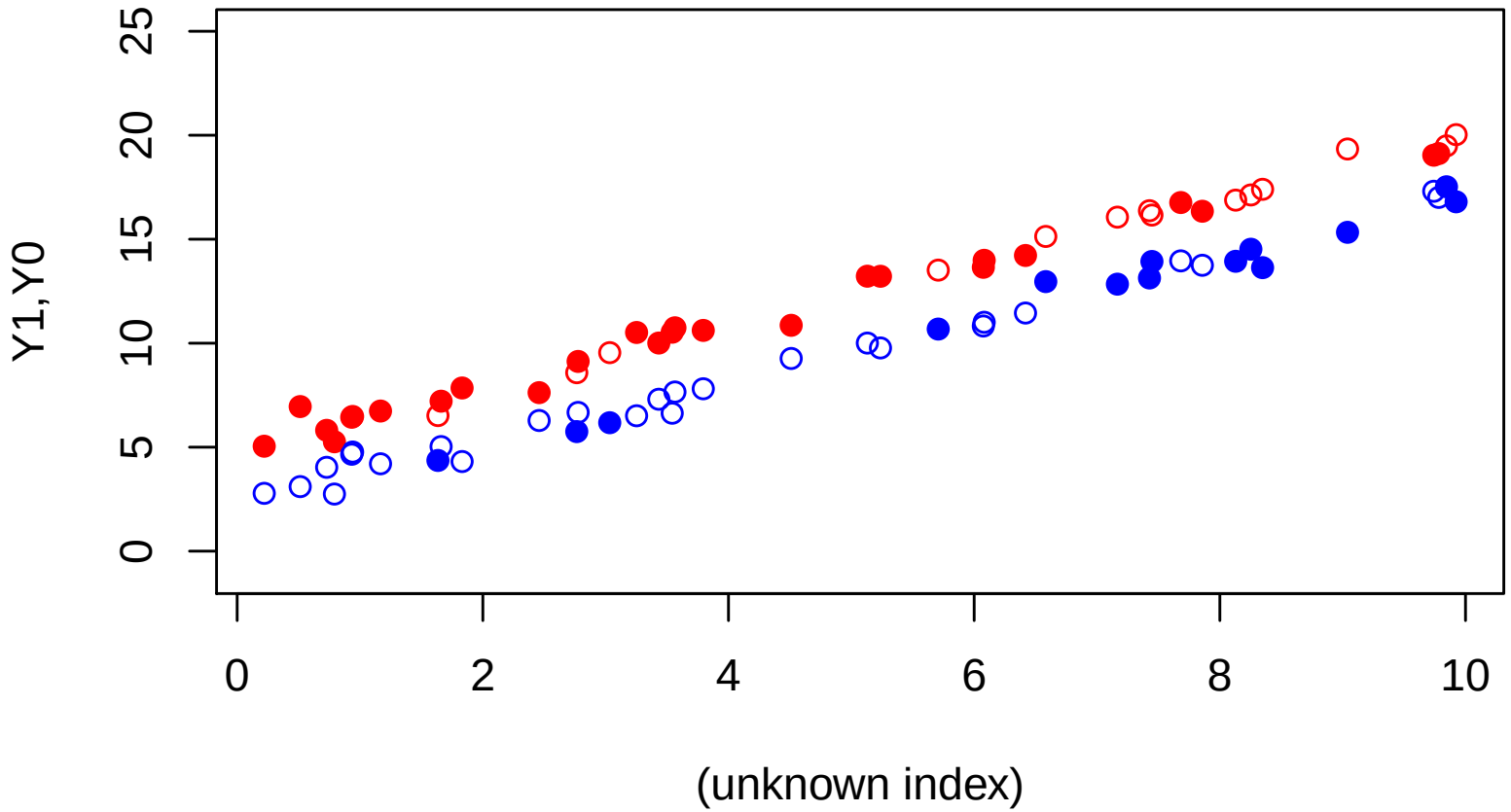

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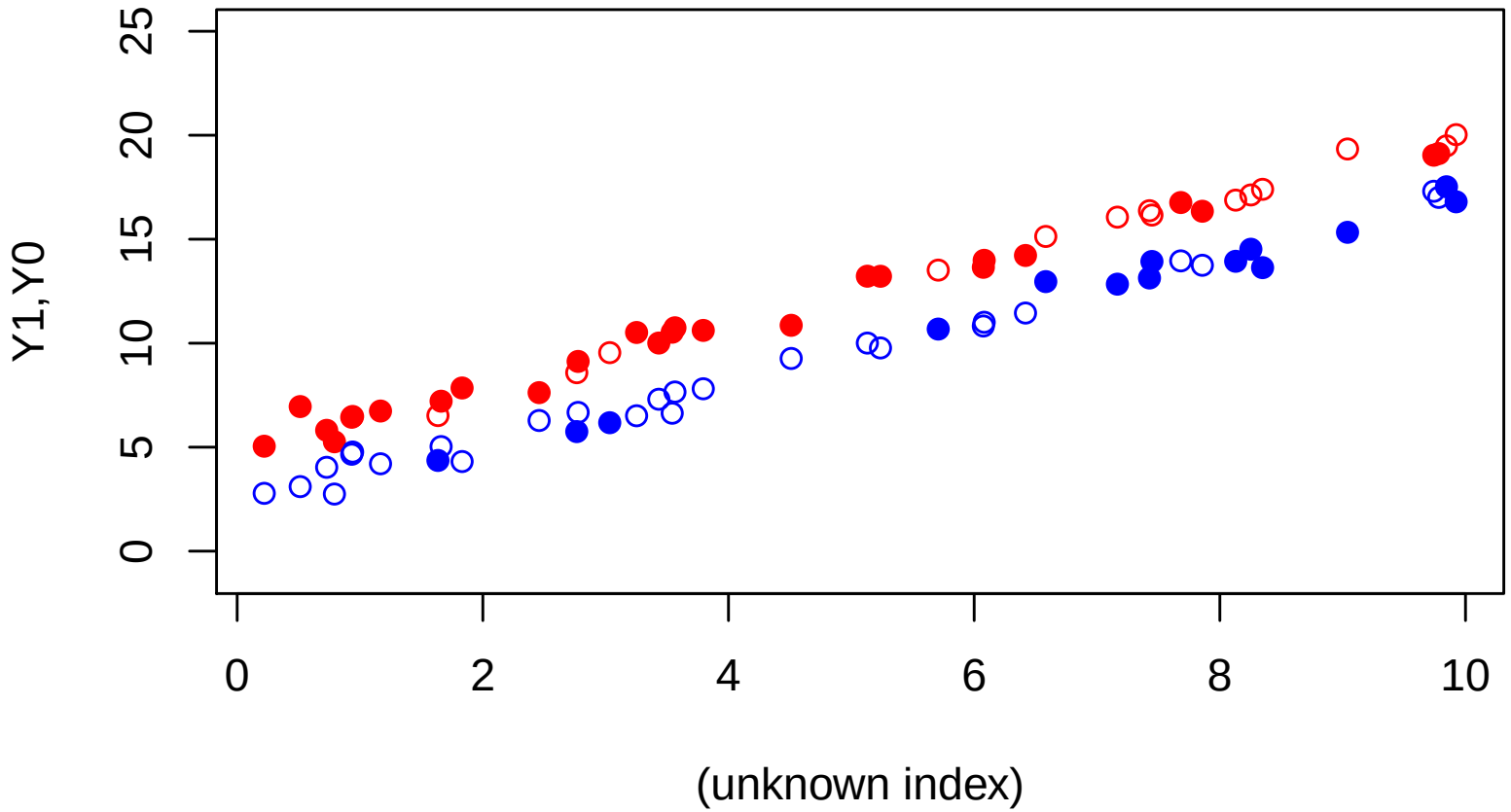

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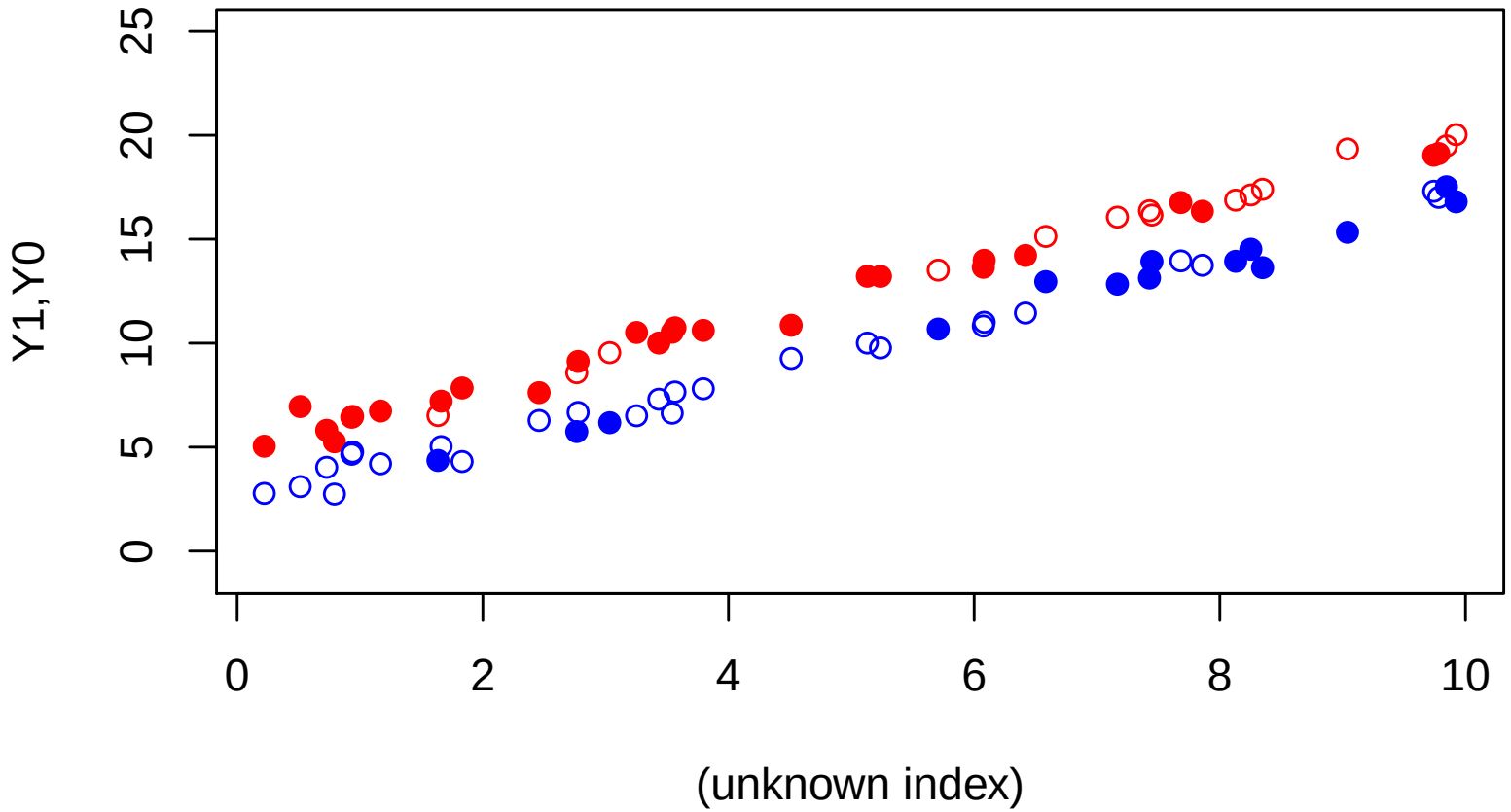

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What can we do about this? Nothing, yet

Preview of Things to Come

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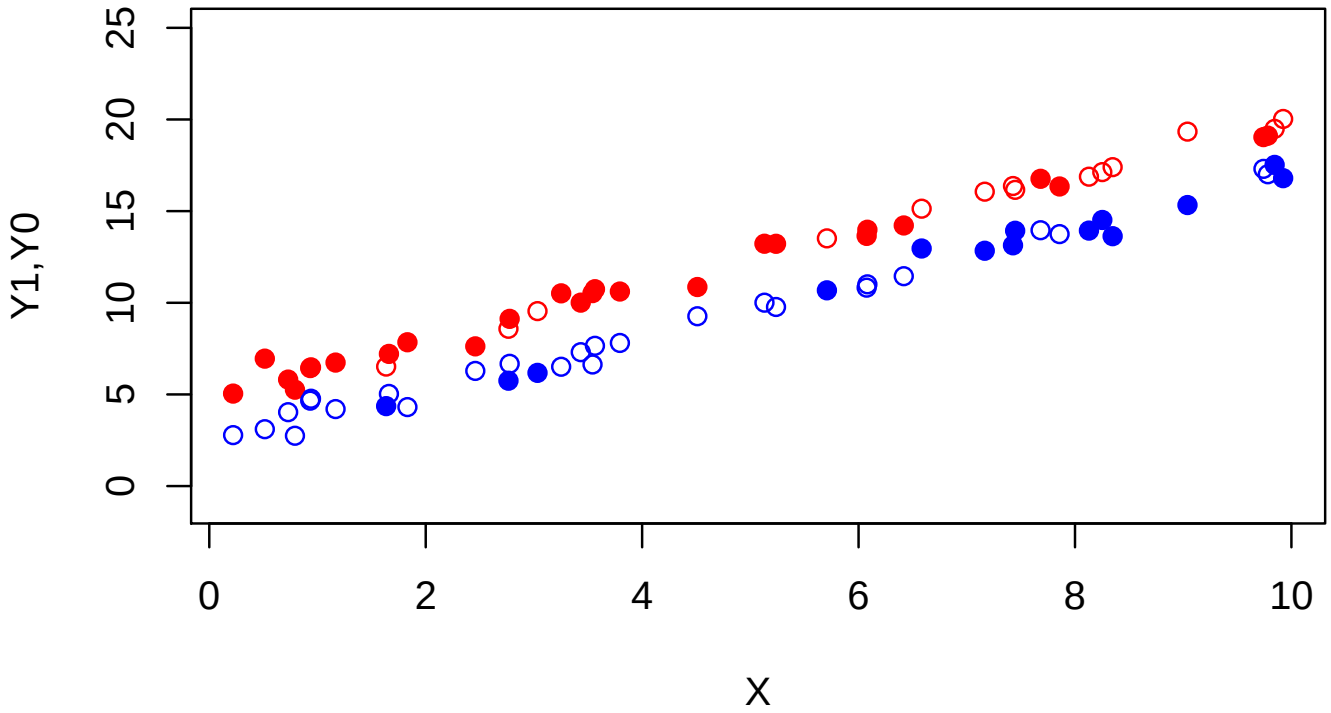

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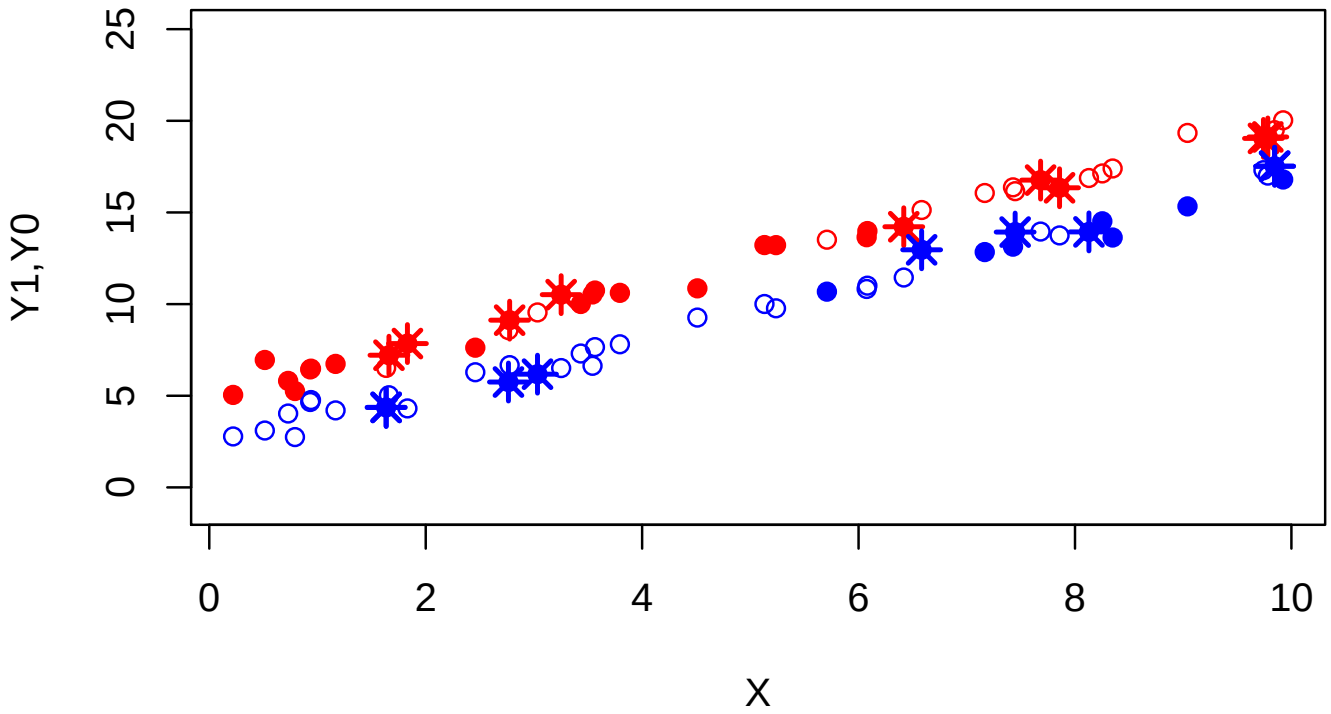

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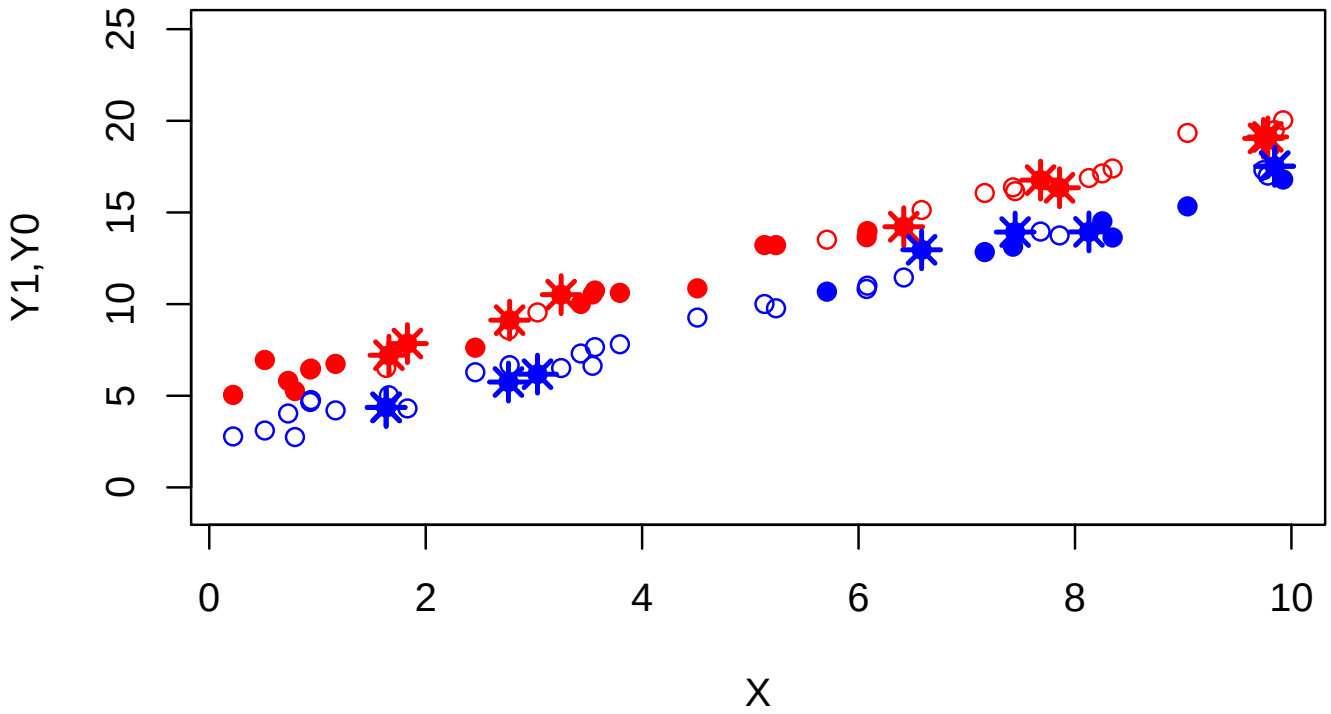

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$$[\overline{Y}_i|D = 1] - [\overline{Y}_i|D = 0] = \widehat{ATE} = 2.63$$

Notation and Definitions

Unit-level effects (τ_i) are fundamentally unidentifiable. On occasion we will be able to identify various average effects. Some estimands include:

- Treatment effect, $\tau_i = Y_{1i} - Y_{0i}$
- Average Treatment Effect (ATE):

$$ATE = \mathbb{E}[Y_{1i} - Y_{0i}] = \mathbb{E}[\tau_i] = \int \tau p(\tau) d\tau$$

- Average treatment effect on the treated (ATT):

$$ATT = \mathbb{E}[Y_{1i} - Y_{0i} | D_i = 1] = \int \tau p(\tau | D = 1) d\tau$$

- Average treatment effect on the controls (ATC):

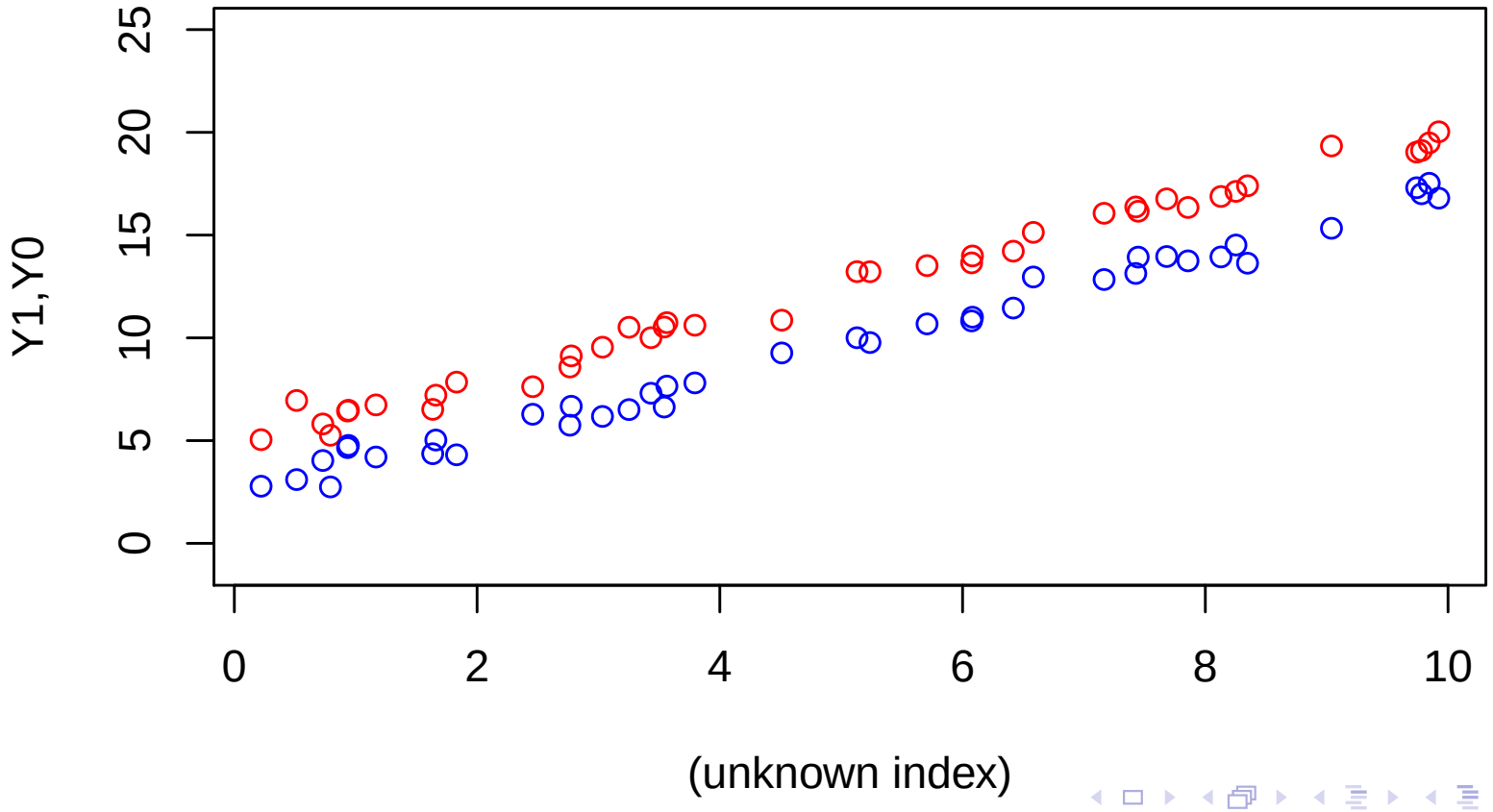
$$ATC = \mathbb{E}[Y_{1i} - Y_{0i} | D_i = 0] = \int \tau p(\tau | D = 0) d\tau$$

- Average treatment effect for sub-groups ($ATE(X)$):

$$ATE(X) = \mathbb{E}[Y_{1i} - Y_{0i} | X = x] = \int \tau p(\tau | X = x) d\tau$$

Practice

Looking at this plot, describe the meaning of the *ATE*, *ATT*, *ATC*, & *ATE*(X) graphically



A Common Assumption: SUTVA

The use of notation $\{Y_{1i}, Y_{0i}\}$ implicitly means we only care about treatment status of i and not of other units, j

- We also defined observed to depend only on unit i's treatment: $Y_i = Y_{1i}D_i + Y_{0i}(1 - D_i)$
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Causal inference in the presence of interference is an area of active research. e.g. sometimes "spillover" or "saturation" is deliberately varied

Some practice

Imagine a study population with 4 units:

i	D_i	Y_{1i}	Y_{0i}	τ_i
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What are the terms in blue & green?

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Example: $D =$ Church Attendance, $Y =$ Political Participation

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Example: Human rights treaties

- Countries willing to sign a human rights treaty will often be those with better human rights already.
- Human rights better in signatory countries even if they had not signed ($\mathbb{E}[Y_0|D = 1] - \mathbb{E}[Y_0|D = 0] > 0$)

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- When we don't know anything, we're in trouble.
- We previewed how knowledge that D_i is random conditional on X can be used.

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In our graphical example, we saw that identification relied on being able to make assumptions about D_i's assignment:

- When we know it was random, we knew unobserved POs would look just like the observed ones.
- When we don't know anything, we're in trouble.
- We previewed how knowledge that D_i is random conditional on X can be used.

Those represent important assignment mechanisms: random assignment, selection on observables, and selection on unobservables.

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- Multiple definitions of "causal effect", need to be precise about estimand.

From here: random assignment, then random-conditional-on-observables.