Week 12: Repeated Observations and Panel Data

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Princeton

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[1] These slides are heavily influenced by Matt Blackwell, Adam Glynn, Jens Hainmueller and Erin Hartman.

Where We've Been and Where We're Going...

- Last Week
  - causal inference with unmeasured confounding
- This Week
  - Monday:
    - panel data
    - diff-in-diff
    - fixed effects
  - Wednesday:
    - spillover of material
    - Q&A
    - wrap-up
- The Following Week
  - break!
- Long Run
  - probability $\rightarrow$ inference $\rightarrow$ regression $\rightarrow$ causality

Questions?

Motivation

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    - possess some cultural trait correlated with better health outcomes
- If we have data on countries over time, can we make any progress in spite of these problems?

Ross Data

```
##       cty_name year democracy infmort_unicef
## 1 Afghanistan 1965         0            230
## 2 Afghanistan 1966         0             NA
## 3 Afghanistan 1967         0             NA
## 4 Afghanistan 1968         0             NA
## 5 Afghanistan 1969         0             NA
## 6 Afghanistan 1970         0            215
```

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  - The main difference is what level of analysis we care about (individual, city, county, state, country, etc).
- Time is a typical application, but applies to other groupings:
  - counties within states
  - states within countries
  - people within professions

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- Panel data: large n, relatively short T
- Time series, cross-sectional (TSCS) data: smaller n, large T
- We are primarily going to focus on similarities today but there are some differences.

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$$y_{it} = \mathbf{x}_{it}'\beta + a_i + u_{it}$$

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We will start by considering performance of estimators assuming this model is true.

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- Has two problems:
  1. Heteroskedasticity (see clustering from diagnostics week)
  2. Possible violation of zero conditional mean errors
- Both problems arise out of ignoring the unmeasured heterogeneity inherent in a_i

Pooled OLS with Ross data

```
pooled.mod <- lm(log(kidmort_unicef) ~ democracy + log(GDPcur),
                 data = ross)
summary(pooled.mod)
```

```
##
## Coefficients:
##              Estimate Std. Error t value Pr(>|t|)
## (Intercept)  9.76405    0.34491   28.31   <2e-16 ***
## democracy   -0.95525    0.06978  -13.69   <2e-16 ***
## log(GDPcur) -0.22828    0.01548  -14.75   <2e-16 ***
## ---
## Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1
##
## Residual standard error: 0.7948 on 646 degrees of freedom
##   (5773 observations deleted due to missingness)
## Multiple R-squared:  0.5044, Adjusted R-squared:  0.5029
## F-statistic: 328.7 on 2 and 646 DF,  p-value: < 2.2e-16
```

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- Example: democratic institutions correlated with time-invariant unmeasured aspects of health outcomes, like quality of health system or a lack of ethnic conflict.
- Ignore the heterogeneity $\rightsquigarrow$ correlation between the combined error and the independent variables:
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- Pooled OLS will be biased and inconsistent because zero conditional mean error fails for the combined error.

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- Due to 'no perfect collinearity': $\mathbf{x}_{it}$ has to change over time for some units. High variance if its slow moving.
- Differencing will reduce the variation in the independent variables and thus increase standard errors.

First Differences in R (via plm package)

```
library(plm)

fd.mod <- plm(log(kidmort_unicef) ~ democracy + log(GDPcur), data = ross,
                      index = c("id", "year"), model = "fd")
summary(fd.mod)

## Oneway (individual) effect First-Difference Model
##
## Call:
## plm(formula = log(kidmort_unicef) ~ democracy + log(GDPcur),
##     data = ross, model = "fd", index = c("id", "year"))
##
## Unbalanced Panel: n=166, T=1-7, N=649
##
## Residuals :
##    Min. 1st Qu.  Median 3rd Qu.    Max.
## -0.9060 -0.0956  0.0468  0.1410  0.3950
##
## Coefficients :
##              Estimate Std. Error  t-value Pr(>|t|)
## (intercept) -0.149469   0.011275 -13.2567  < 2e-16 ***
## democracy   -0.044887   0.024206  -1.8544  0.06429 .
## log(GDPcur) -0.171796   0.013756 -12.4886  < 2e-16 ***
## ---
## Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1
##
## Total Sum of Squares:    23.545
## Residual Sum of Squares: 17.762
## R-Squared      :  0.24561
##       Adj. R-Squared :  0.24408
## F-statistic: 78.1367 on 2 and 480 DF, p-value: < 2.22e-16
```

Motivation: Studying the Minimum Wage

Minimum Wages and Employment:
A Case Study of the Fast-Food Industry
in New Jersey and Pennsylvania

By DAVID CARD AND ALAN B. KRUEGER*

On April 1, 1992, New Jersey's minimum wage rose from $4.25 to $5.05 per hour. To evaluate the impact of the law we surveyed 410 fast-food restaurants in New Jersey and eastern Pennsylvania before and after the rise. Comparisons of employment growth at stores in New Jersey and Pennsylvania (where the minimum wage was constant) provide simple estimates of the effect of the higher minimum wage. We also compare employment changes at stores in New Jersey that were initially paying high wages (above $5) to the changes at lower-wage stores. We find no indication that the rise in the minimum wage reduced employment. (*JEL* J30, J23)

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- Economics conventional wisdom: higher minimum wages decrease low-wage jobs.
- Card and Krueger (1994) study a 1992 New Jersey minimum wage increase (\$4.25 to \$5.05).
- Idea: compare employment rates in 410 fast-food restauarants in New Jersey and eastern Pennsylvania (where there wasn't a wage increase) both before and after the change.

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- Idea: compare employment rates in 410 fast-food restauarants in New Jersey and eastern Pennsylvania (where there wasn't a wage increase) both before and after the change.
- Based on survey data:
  - Wave 1: March 1992, one month before the minimum wage increased
  - Wave 2: December 1992, eight months after increase

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- d_t is a dummy variable for the second time period
  - $d_2 = 1$ and $d_1 = 0$
- β_1 is the quantity of interest: it's the effect of being treated

Diff-in-Diff Mechanics

- Let's take differences:

$$(y_{i2} - y_{i1}) \;\; = \;\; \delta_0(1-0) + \beta_1(x_{i2} - x_{i1}) + (a_i - a_i) + (u_{i2} - u_{i1})$$

- This represents
  - δ_0: the difference in the average outcome from period 1 to period 2 in the untreated group
  - $(x_{i2} - x_{i1}) = 1$ for the treated group and 0 for the control group
  - β_1 represents the additional change in y over time (on top of δ_0) associated with being in the treatment group.

Diff-in-Diff Mechanics

- Let's take differences:

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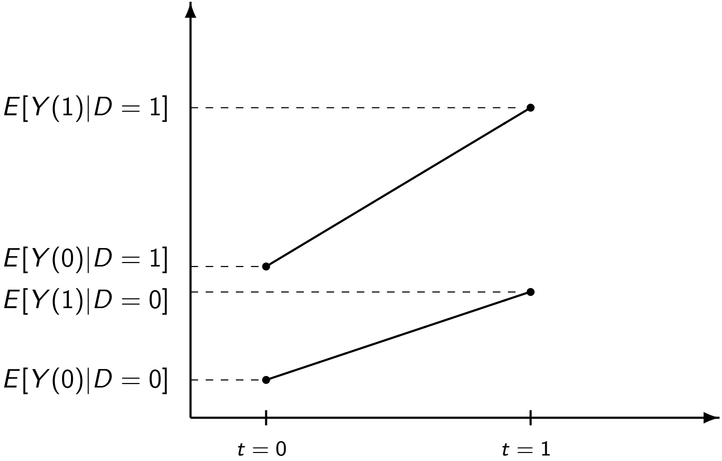
Graphical Representation: Difference-in-Differences

Define $D = 1$ when $x_{i2} - x_{i1} = 1$ and 0 otherwise



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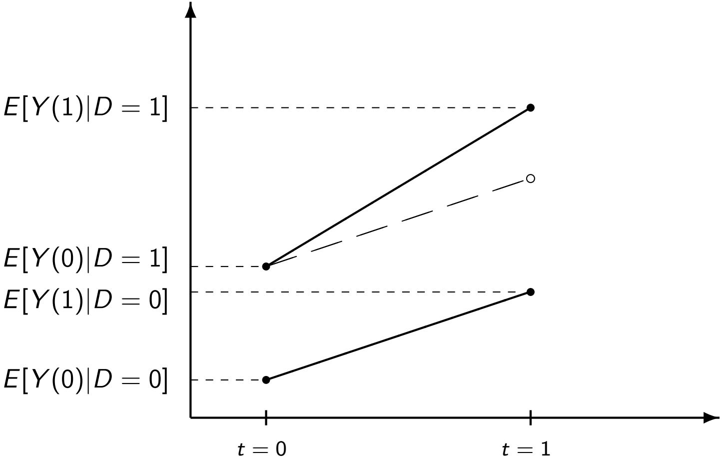

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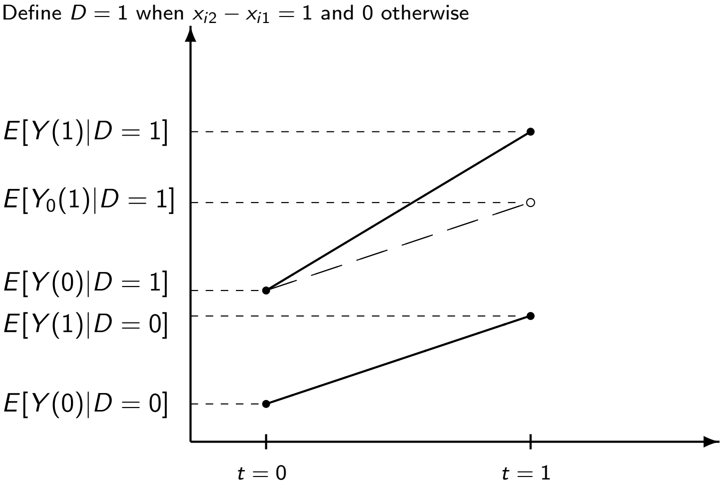

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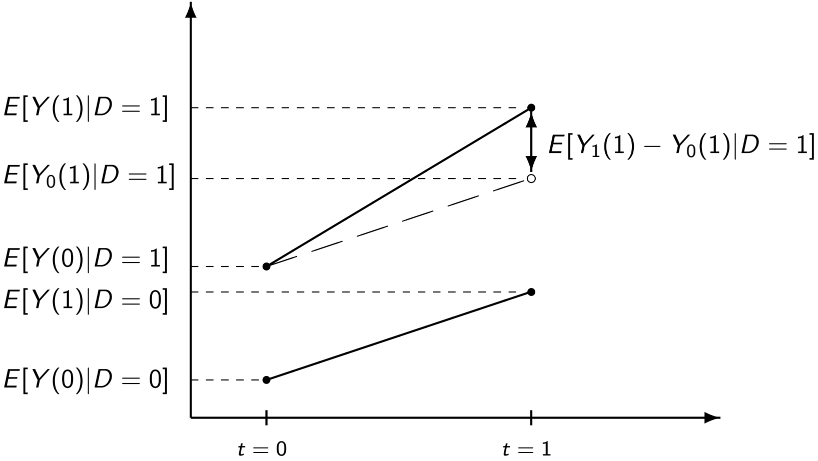

Identification with Difference-in-Differences

Identification Assumption (parallel trends)

$E[Y_0(1) - Y_0(0)|D = 1] = E[Y_0(1) - Y_0(0)|D = 0]$

Identification Result

Given parallel trends the ATT is identified as:

$$\begin{aligned} E[Y_1(1) - Y_0(1)|D = 1] &= \Big\{E[Y(1)|D = 1] - E[Y(1)|D = 0]\Big\} \\ &- \Big\{E[Y(0)|D = 1] - E[Y(0)|D = 0]\Big\} \end{aligned}$$

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Proof.

Note that the identification assumption implies
$E[Y_0(1)|D = 0] = E[Y_0(1)|D = 1] - E[Y_0(0)|D = 1] + E[Y_0(0)|D = 0]$
plugging in we get

$$
\begin{aligned}
& \{E[Y(1)|D = 1] - E[Y(1)|D = 0]\} - \{E[Y(0)|D = 1] - E[Y(0)|D = 0]\} \\
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- & \{E[Y_0(0)|D = 1] - E[Y_0(0)|D = 0]\} \\
= & E[Y_1(1) - Y_0(1)|D = 1] + \{E[Y_0(0)|D = 1] - E[Y_0(0)|D = 0]\} \\
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= & E[Y_1(1) - Y_0(1)|D = 1]
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Example: Lyall (2009)

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Does Indiscriminate Violence Incite Insurgent Attacks?

Evidence from Chechnya

Jason Lyall
Department of Politics and the Woodrow Wilson School
Princeton University, New Jersey

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- Counterintuitive findings: shelled villages experience a 24% reduction in insurgent attacks relative to controls.

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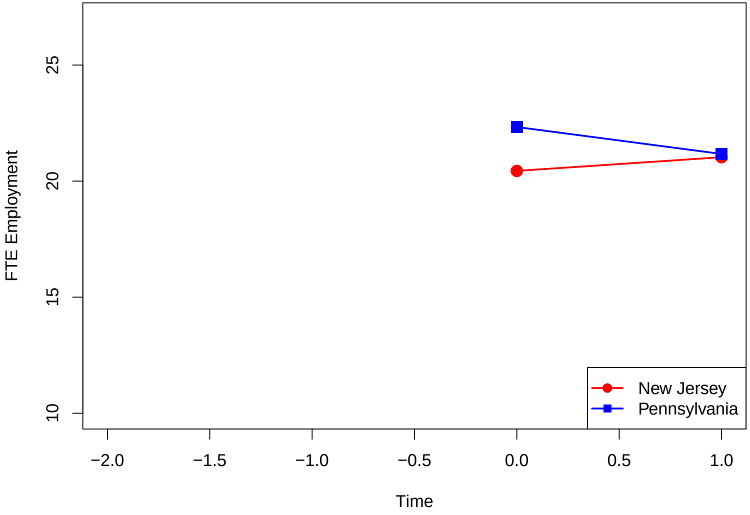
- NJ_i indicates which stores received the treatment of a higher minimum wage at time period $t = 2$

Parallel Trends?



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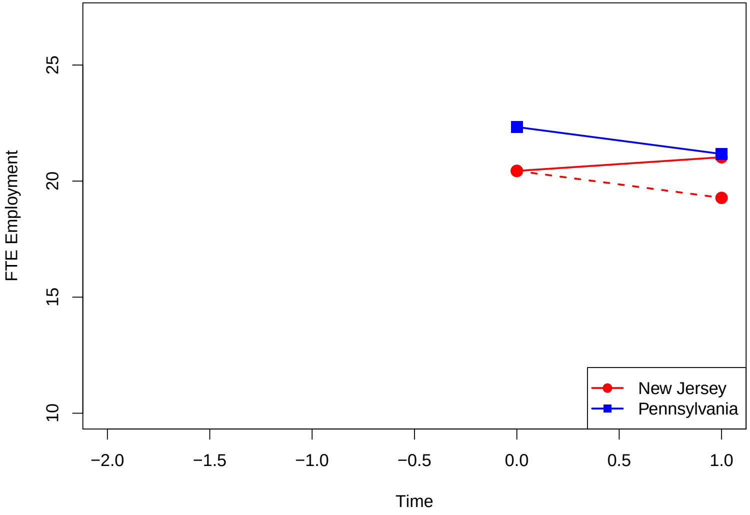

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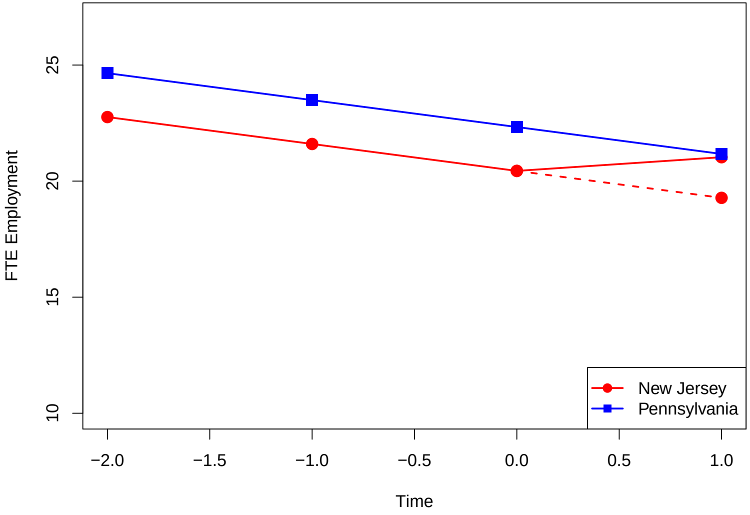

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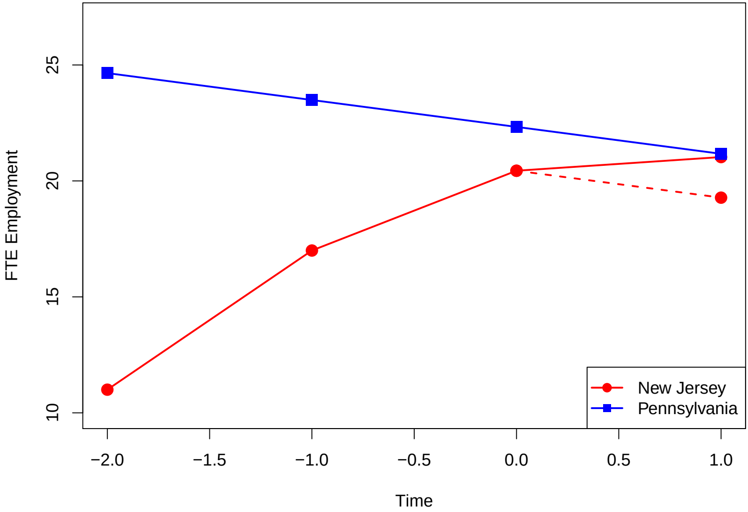
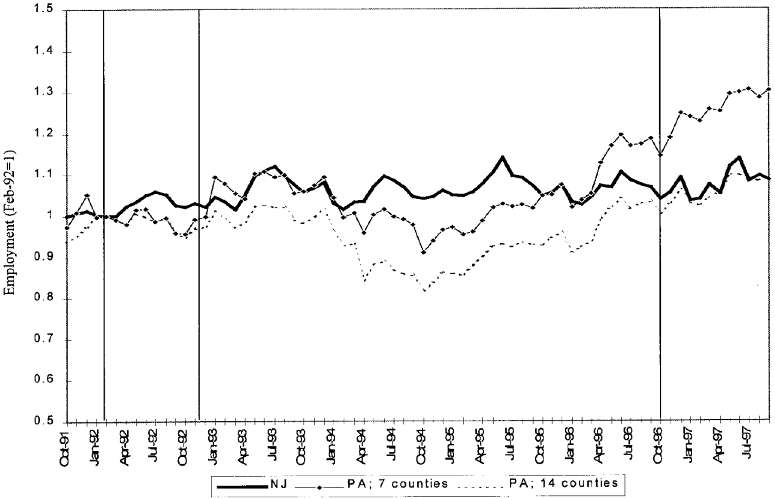


Longer Trends in Employment (Card and Krueger 2000)



First two vertical lines indicate the dates of the Card-Krueger survey. October 1996 line is the federal minimum wage hike which was binding in PA but not NJ

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4) Long-term vs. Short-term Effects
parallel trends are less credible over a long time horizon, but many policies need time to take effect.

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6) Endogenous Control Variables
can add (time-varying) covariates to help with some of above concerns $\rightsquigarrow$ "regression diff-in-diff"

$$y_{i2} - y_{i1} = \delta_0 + \mathbf{z}_i'\tau + \beta(x_{i2} - x_{i1}) + (u_{i2} - u_{i1})$$

but need to be careful that they aren't affected by the treatment.

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- Two questions to ask:
  1. 'what is the counterfactual?' or
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- Personal Gripe: 'Two-way Fixed Effects' models often called a DiD or Generalized-DiD design but the parallel trend assumptions are different in important ways.

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- We discussed a differencing approach to this model
- The Fixed effects model is an alternative way to remove time-invariant unmeasured confounding
- We will start by assuming the model and discussing properties and in the next section, we will consider non-parametric identification.

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- Key fact: because it is time-constant the mean of a_i is just a_i

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&= \left(\frac{1}{T}\sum_{t=1}^{T}\mathbf{x}_{it}'\right)\boldsymbol{\beta} + \frac{1}{T}\sum_{t=1}^{T} a_i + \frac{1}{T}\sum_{t=1}^{T} u_{it} \\
&= \overline{\mathbf{x}}_i'\boldsymbol{\beta} + a_i + \overline{u}_i
\end{aligned}
$$

- Key fact: because it is time-constant the mean of a_i is just a_i
- This regression is sometimes called the "between regression"

Within Transformation

Within Transformation

- The "fixed effects," "within," or "time-demeaning" transformation is when we subtract off the over-time means from the original data:

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- NB: you must demean the X variables not just the Y variables.

Fixed Effects with Ross data

```
fe.mod <- plm(log(kidmort_unicef) ~ democracy + log(GDPcur), data = ross, index = c("id", "year"),
 model = "within")
summary(fe.mod)
```

```
## Oneway (individual) effect Within Model
##
## Call:
## plm(formula = log(kidmort_unicef) ~ democracy + log(GDPcur),
##     data = ross, model = "within", index = c("id", "year"))
##
## Unbalanced Panel: n=166, T=1-7, N=649
##
## Residuals :
##     Min.   1st Qu.    Median   3rd Qu.     Max.
## -0.70500 -0.11700  0.00628  0.12200  0.75700
##
## Coefficients :
##              Estimate Std. Error  t-value  Pr(>|t|)
## democracy   -0.143233   0.033500  -4.2756 2.299e-05 ***
## log(GDPcur) -0.375203   0.011328 -33.1226 < 2.2e-16 ***
## ---
## Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1
##
## Total Sum of Squares:    81.711
## Residual Sum of Squares: 23.012
## R-Squared      :  0.71838
##       Adj. R-Squared :  0.53242
## F-statistic: 613.481 on 2 and 481 DF, p-value: < 2.22e-16
```

Strict Exogeneity

- FE models are valid if $\mathbb{E}[\mathbf{u}|\mathbf{X}] = 0$: all errors are uncorrelated with covariates in every period:

$$\mathbb{E}[\ddot{u}_{it}|\ddot{\mathbf{X}}] = \mathbb{E}[u_{it}|\ddot{\mathbf{X}}] - \mathbb{E}[\overline{u}_i|\ddot{\mathbf{X}}] = 0 - 0 = 0$$

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- This is because the composite errors, $\ddot{u}_{it}$ are function of the errors in every time period through the average, $\overline{u}_i$
- This rules out, for instance, lagged dependent variables, since $y_{i,t-1}$ has to be correlated with $u_{i,t-1}$. Thus it can't be a covariate for y_{it}.

Fixed Effects and Time-Invariant Covariates

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- Basic message: any time-constant variable gets "absorbed" by the fixed effect. It has nothing to contribute because the comparison is within the units.
- Can include interactions between time-constant and time-varying variables, but lower order term of the time-constant variables get absorbed by fixed effects too

Time-constant variables

- Pooled model with a time-constant variable, proportion Islamic:

```
library(lmtest)
p.mod <- plm(log(kidmort_unicef) ~ democracy + log(GDPcur) + islam,
             data = ross, index = c("id", "year"), model = "pooling")
coeftest(p.mod)
```

```
##
## t test of coefficients:
##
##                Estimate  Std. Error  t value  Pr(>|t|)
## (Intercept) 10.30607817  0.35951939  28.6663 < 2.2e-16 ***
## democracy   -0.80233845  0.07766814 -10.3303 < 2.2e-16 ***
## log(GDPcur) -0.25497406  0.01607061 -15.8659 < 2.2e-16 ***
## islam        0.00343325  0.00091045   3.7709 0.0001794 ***
## ---
## Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1
```

Time-constant variables

- FE model, where the islam variable drops out, along with the intercept:

```
fe.mod2 <- plm(log(kidmort_unicef) ~ democracy + log(GDPcur) + islam,
               data = ross, index = c("id", "year"), model = "within")
coeftest(fe.mod2)
```

```
##
## t test of coefficients:
##
##              Estimate Std. Error  t value  Pr(>|t|)
## democracy   -0.129693   0.035865  -3.6162 0.0003332 ***
## log(GDPcur) -0.379997   0.011849 -32.0707 < 2.2e-16 ***
## ---
## Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1
```

Alternate Computation: Least Squares Dummy Variable

- As an alternative to the within transformation, we can also include a series of $n-1$ dummy variables for each unit:

$$y_{it} = \mathbf{x}'_{it}\boldsymbol{\beta} + d_i^{(1)}\alpha_1 + d_i^{(2)}\alpha_2 + \cdots + d_i^{(n)}\alpha_n + u_{it}$$

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- Why are these equivalent? (remember partialing out strategy and Frisch-Waugh-Lovell theorem)

Example with Ross data

```
library(lmtest)
lsdv.mod <- lm(log(kidmort_unicef) ~ democracy + log(GDPcur) +
               as.factor(id), data = ross)
coeftest(lsdv.mod)[1:6,]
coeftest(fe.mod)[1:2,]
```

```
##                     Estimate Std. Error    t value      Pr(>|t|)
## (Intercept)       13.7644887 0.26597312  51.751427 1.008329e-198
## democracy         -0.1432331 0.03349977  -4.275644  2.299393e-05
## log(GDPcur)       -0.3752030 0.01132772 -33.122568 3.494887e-126
## as.factor(id)AGO   0.2997206 0.16767730   1.787485  7.448861e-02
## as.factor(id)ALB  -1.9309618 0.19013955 -10.155498  4.392512e-22
## as.factor(id)ARE  -1.8762909 0.17020738 -11.023558  2.386557e-25
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- Note that when the second dimension isn't time, fixed effects will be relevant more often.

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- We've seen the trouble with constant effects before, it goes back to Lecture 10 and results on regression with heterogenous treatment effects more generally.

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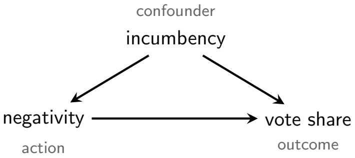
Examples of static and dynamic causal inference problems:



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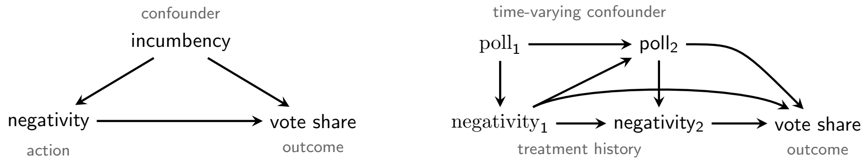

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A Framework for Dynamic Causal Inference in Political Science

Matthew Blackwell University of Rochester

Dynamic strategies are an essential part of politics. In the context of campaigns, for example, candidates continuously recalibrate their campaign strategy in response to polls and opponent actions. Traditional causal inference methods, however, assume that these dynamic decisions are made all at once, an assumption that forces a choice between omitted variable bias and posttreatment bias. Thus, these kinds of "single-shot" causal inference methods are inappropriate for dynamic processes like campaigns. I resolve this dilemma by adapting methods from biostatistics, thereby presenting a holistic framework for dynamic causal inference. I then use this method to estimate the effectiveness of an inherently dynamic process: a candidate's decision to "go negative." Drawing on U.S. statewide elections (2000–2006), I find, in contrast to the previous literature and alternative methods, that negative advertising is an effective strategy for nonincumbents. I also describe a set of diagnostic tools and an approach to sensitivity analysis.

What candidate would plan all of their rallies, write all of their speeches, and film all of their advertisements at the beginning of a campaign, then sit back and watch them unfold until Election Day? Clearly this is absurd, and yet it is the only setup that the usual ways of making causal inferences allows us to study. While political science has seen enormous growth in attention to causal inference over the past decade, these advances have heavily focused on snapshots where the dynamic nature of politics is crammed into a single point in time. As political science finds itself with a growing number of motion pictures—panel data, time-series cross-sectional data—a tension has emerged between substance and method. Indeed, applied to dynamic data, the best practices of *single-shot* causal inference methods provide conflicting advice and fail to alleviate omitted variable or posttreatment bias.

This article focuses on a specific dynamic process: negative advertising in 176 U.S. Senate and gubernatorial elections from 2000 until 2006. Candidates in these races change their tone over the course of the campaign, react-

are more likely to go negative than those that are safe. Attempting to correct for this dynamic selection by controlling for polls leads to posttreatment bias since earlier campaign tone influences polling. The inappropriate application of single-shot causal inference therefore leaves scholars between a rock and a hard place, steeped in bias with either approach. This dilemma is not limited to negative advertising or campaigns—every field of political science has a variable of interest that evolves over time.

This article solves this dilemma by presenting a framework for dynamic causal inference and a set of tools, developed in biostatistics and epidemiology (Robins, Hernán, and Brumback 2000), to estimate dynamic causal effects. These tools directly model dynamic selection and overcome the above problems of single-shot causal inference. Actions (such as campaign tone) are allowed to vary over time along with any confounding covariates (such as polling). Thus, we can study the effects of the *action history* (candidate's tone across the entire campaign) as opposed to a single action (simply "going negative").

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This article solves this dilemma by presenting a framework for dynamic causal inference and a set of tools, developed in biostatistics and epidemiology (Robins, Hernán, and Brumback 2000), to estimate dynamic causal effects. These tools directly model dynamic selection and overcome the above problems of single-shot causal inference. Actions (such as campaign tone) are allowed to vary over time along with any confounding covariates (such as polling). Thus, we can study the effects of the *action history* (candidate's tone across the entire campaign) as opposed to a single action (simply "going negative").

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How to Make Causal Inferences with Time-Series Cross-Sectional Data under Selection on Observables

MATTHEW BLACKWELL *Harvard University*
ADAM N. GLYNN *Emory University*

Repeated measurements of the same countries, people, or groups over time are vital to many fields of political science. These measurements, sometimes called time-series cross-sectional (TSCS) data, allow researchers to estimate a broad set of causal quantities, including contemporaneous effects and direct effects of lagged treatments. Unfortunately, popular methods for TSCS data can only produce valid inferences for lagged effects under some strong assumptions. In this paper, we use potential outcomes to define causal quantities of interest in these settings and clarify how standard models like the autoregressive distributed lag model can produce biased estimates of these quantities due to post-treatment conditioning. We then describe two estimation strategies that avoid these post-treatment biases—inverse probability weighting and structural nested mean models—and show via simulations that they can outperform standard approaches in small sample settings. We illustrate these methods in a study of how welfare spending affects terrorism.

INTRODUCTION

Many inquiries in political science involve the study of repeated measurements of the same countries, people, or groups at several points in time. This type of data, sometimes called time-series cross-sectional (TSCS) data, allows researchers to draw on a larger pool of information when estimating causal effects. TSCS data also give researchers the power to ask a richer set of questions than data with a single measurement for each unit (for example, see Beck and Katz 2011). Using this data, researchers can move past the narrowest contemporaneous questions—what are the effects of a single event?—and instead ask how the *history* of a process affects the political world. Unfortunately, the most common approaches to modeling TSCS data require strict assumptions to estimate the effect of treatment histories without bias and make it difficult to understand the nature of the counterfactual comparisons.

This paper makes three contributions to the study of TSCS data. Our first contribution is to define some counterfactual causal effects and discuss the assumptions needed to identify them nonparametrically. We also relate these quantities of interest to common quantities in the TSCS literature, like impulse responses, and show how to derive them from the parameters of a common TSCS model, the autoregressive distributed lag (ADL) model. These treatment effects can be nonparametrically identified under a key selection-on-observables assumption called sequential ignorability; unfortunately, however, many common TSCS approaches rely on more stringent assumptions, including a lack of causal feedback between the treatment and time-varying covariates. This feedback, for example, might involve a country's level of welfare spending affecting the vote share of left wing parties, which in turn might affect future levels of spending. We argue that this type of feedback is common in TSCS settings. While we focus on a selection-on-observables assumption in this paper, we discuss the tradeoffs with this choice compared to standard fixed-effects methods, noting that the latter may also rule out this type of dynamic feedback.

Our second contribution is to provide an introduction to two methods from biostatistics that can estimate the effect of treatment histories without bias and under weaker assumptions than common TSCS models. We focus on two methods: (1) *structural nested mean models* or SNMMs (Robins 1997) and (2) *marginal structural models* (MSMs) *with inverse probability of treatment weighting* (IPTWs) (Robins, Hernán, and

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Core Conundrum

There is a (possibly irresolvable) tension: modeling causal dynamics between treatment and outcomes OR addressing unobserved time-invariant confounders. Three great recent papers:

A Framework for Dynamic Causal Inference in Political Science

Matthew Blackwell University of Rochester

Dynamic strategies are an essential part of politics. In the context of campaigns, for example, candidates continuously recalibrate their campaign strategy in response to polls and opponent actions. Traditional causal inference methods, however, assume that these dynamic decisions are made all at once, an assumption that forces a choice between omitted variable bias and posttreatment bias. Thus, these kinds of "single-shot" causal inference methods are inappropriate for dynamic processes like campaigns. I resolve this dilemma by adapting methods from biostatistics, thereby presenting a holistic framework for dynamic causal inference. I then use this method to estimate the effectiveness of an inherently dynamic process: a candidate's decision to "go negative." Drawing on U.S. statewide elections (2000–2006), I find, in contrast to the previous literature and alternative methods, that negative advertising is an effective strategy for nonincumbents. I also describe a set of diagnostic tools and an approach to sensitivity analysis.

What candidate would plan all of their rallies, write all of their speeches, and film all of their advertisements at the beginning of a campaign, then sit back and watch them unfold until Election Day? Clearly this is absurd, and yet it is the only setup that the usual ways of making causal inferences allows us to study. While political science has seen enormous growth in attention to causal inference over the past decade, these advances have heavily focused on snapshots where the dynamic nature of politics is crammed into a single point in time. As political science finds itself with a growing number of motion pictures—panel data, time-series cross-sectional data—a tension has emerged between substance and method. Indeed, applied to dynamic data, the best practices of *single-shot* causal inference methods provide conflicting advice and fail to alleviate omitted variable or posttreatment bias.

This article focuses on a specific dynamic process: negative advertising in 176 U.S. Senate and gubernatorial elections from 2000 until 2006. Candidates in these races change their tone over the course of the campaign, react-

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When Should We Use Unit Fixed Effects Regression Models for Causal Inference with Longitudinal Data?*

Kosuke Imai† In Song Kim‡

Forthcoming in *American Journal of Political Science*

Abstract

Many researchers use unit fixed effects regression models as their default methods for causal inference with longitudinal data. We show that the ability of these models to adjust for unobserved time-invariant confounders comes at the expense of dynamic causal relationships, which are permitted under an alternative selection-on-observables approach. Using the nonparametric directed acyclic graph, we highlight two key causal identification assumptions of unit fixed effects models: past treatments do not directly influence current outcome, and past outcomes do not affect current treatment. Furthermore, we introduce a new nonparametric matching framework that elucidates how various unit fixed effects models implicitly compare treated and control observations to draw causal inference. By establishing the equivalence between matching and weighted unit fixed effects estimators, this framework enables a diverse set of identification strategies to adjust for unobservables in the absence of dynamic causal relationships between treatment and outcome variables. We illustrate the proposed methodology through its application to the estimation of GATT membership effects on dyadic trade volume.

Key Words: before-and-after design, directed acyclic graph, matching, panel data, time series cross sectional data, weighted least squares

Replication Materials: The data, code, and any additional materials required to replicate all analyses in this article are available on the American Journal of Political Science Dataverse within the Harvard Dataverse Network, at: `https://dx.doi.org/10.7910/DVN/YUM3K8`

Core Conundrum

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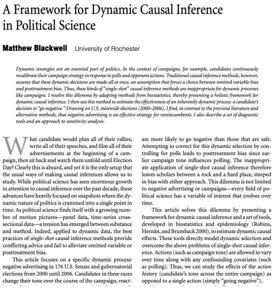
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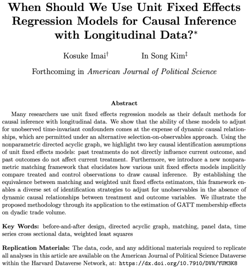
When Should We Use Unit Fixed Effects Regression Models for Causal Inference with Longitudinal Data?*

Kosuke Imai† In Song Kim‡

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We are going to focus on addressing unobserved time-invariant confounders using the last paper.

Next several slides are based on slides graciously provided by In Song Kim and Kosuke Imai.

Directed Acyclic Graph (DAG)

Non-parametric identification assumptions for fixed effects:

$$Y_{it} \quad = \quad g(X_{it}, \mathbf{U}_i, \epsilon_{it}) \quad \text{and} \quad \epsilon_{it} \perp\!\!\!\perp \{\mathbf{X}_i, \mathbf{U}_i\}$$

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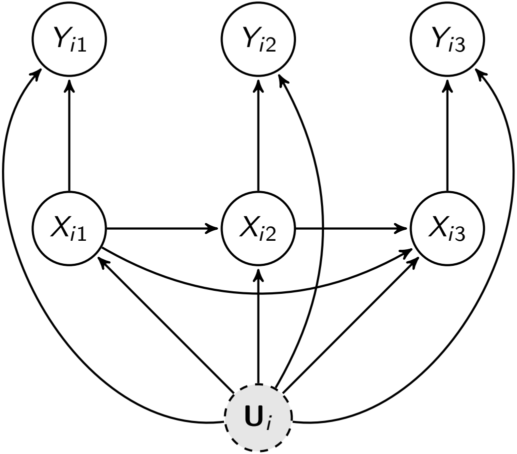

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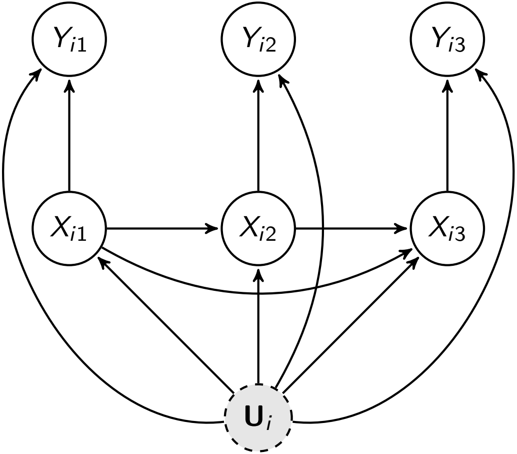

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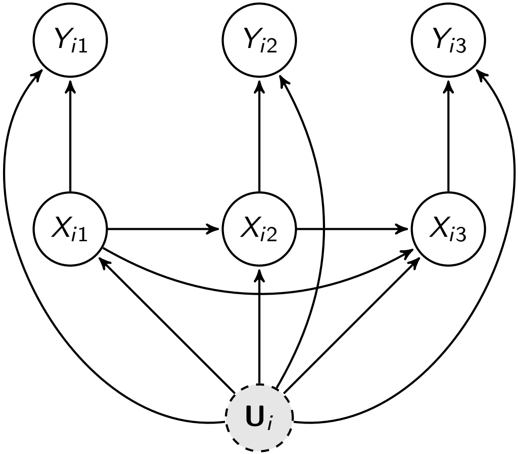

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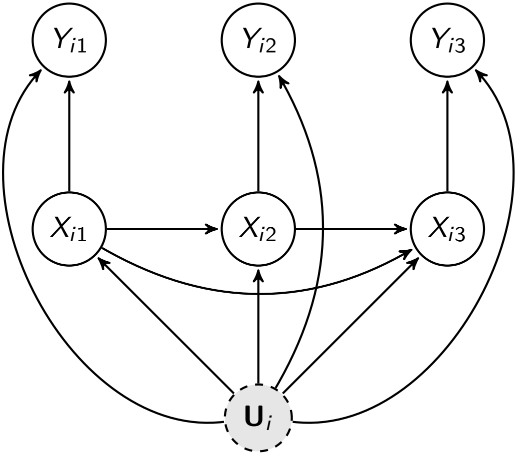

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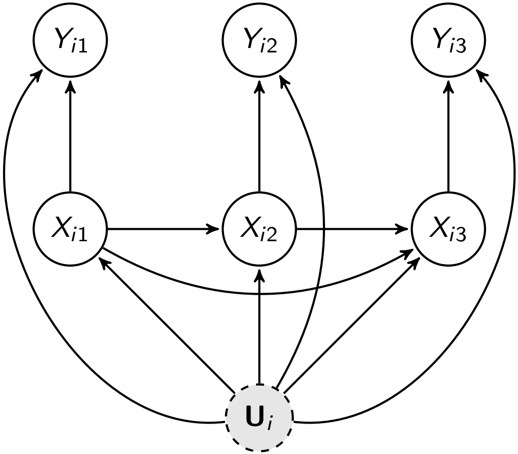

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4. Past treatments do not directly affect current outcome

the result implies that the counterfactual outcome for a treated observation in a given time period is estimated using the observed outcomes of different time periods of the same unit. Since such a comparison is valid only when no causal dynamics exist, this finding underscores the important limitation of linear regression models with unit fixed effects.

- Imai and Kim (Forthcoming)

What Ideal Experiment Corresponds to the Fixed Effects Model?

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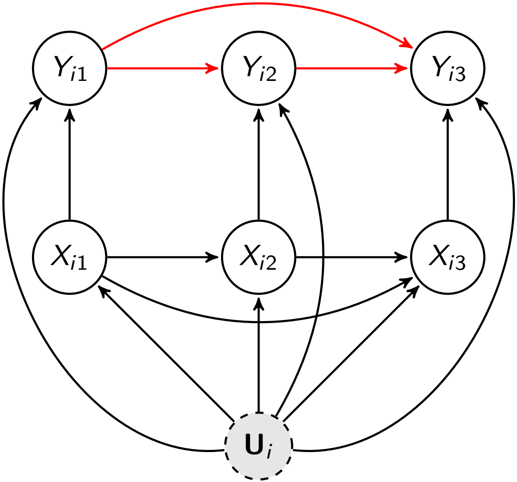
  4. and so on
- Now let's consider each assumption in turn.

Past Outcomes Don't Directly Affect Current Outcome



- Strict exogeneity still holds.

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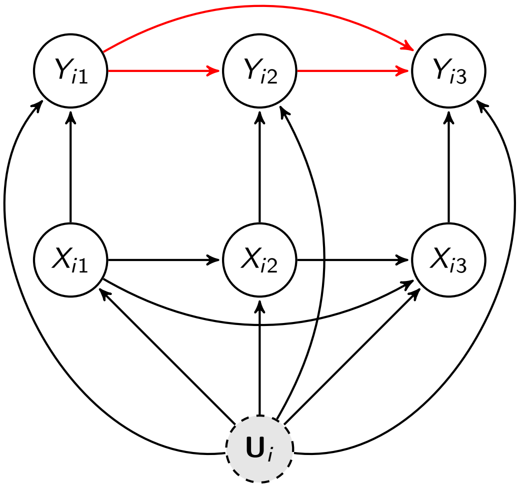

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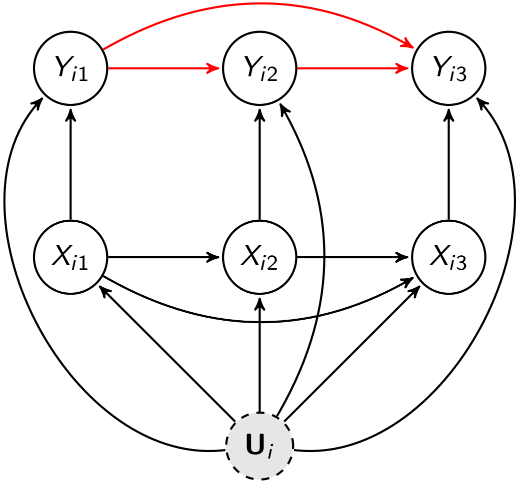

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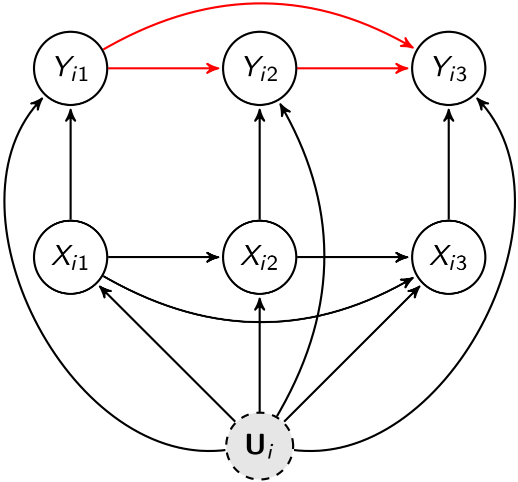

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- No need to adjust for past outcomes.
- Should use cluster robust standard errors for inference.

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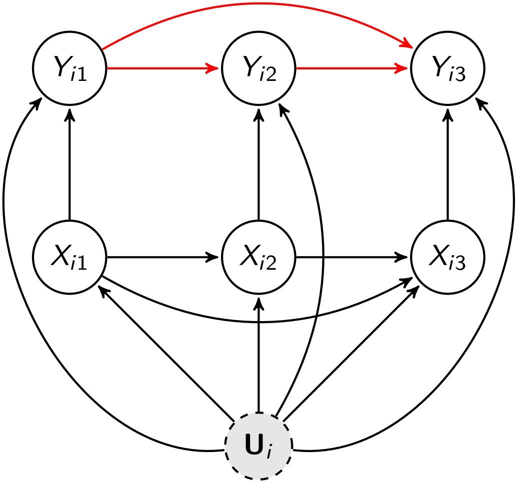
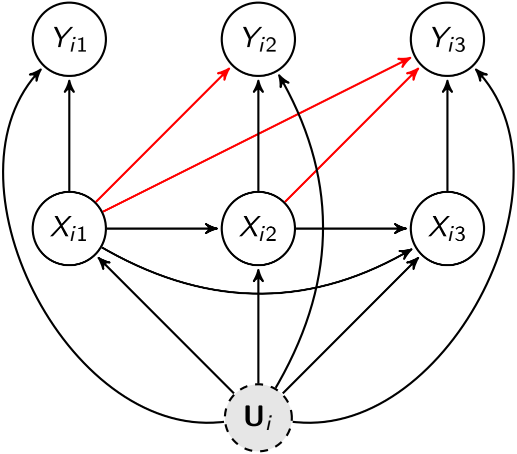


- Strict exogeneity still holds.
- Past outcomes do not confound $X_{it} \longrightarrow Y_{it}$ given $\mathbf{U}_i$.
- No need to adjust for past outcomes.
- Should use cluster robust standard errors for inference.
- Conclusion: The assumption can be relaxed

Past Treatments Don't Directly Affect Current Outcome



- Need to adjust for past treatments

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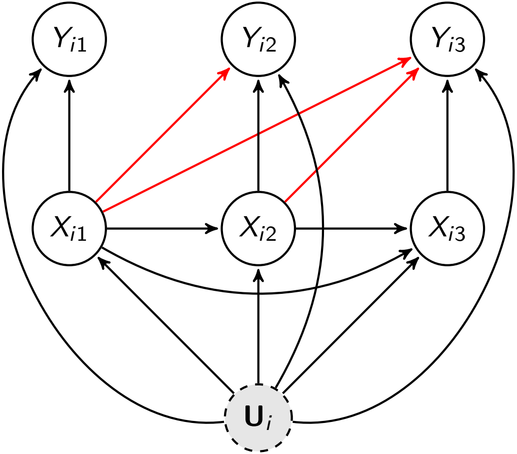

- Need to adjust for past treatments
- Strict exogeneity holds given past treatments and $\mathbf{U}_i$

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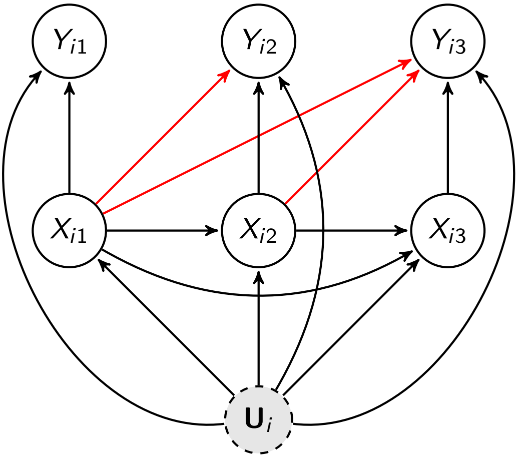

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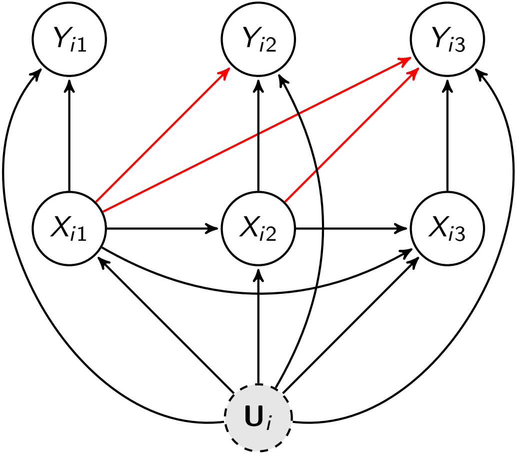

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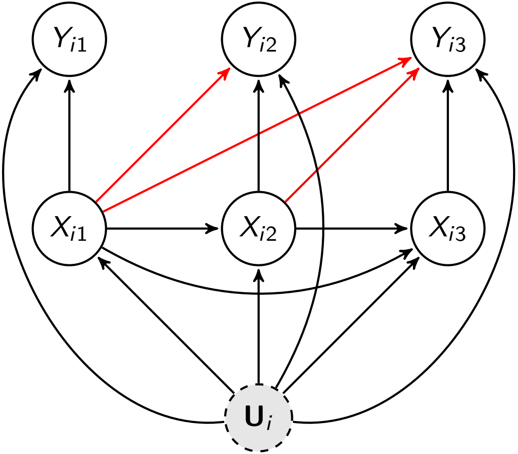

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- Conclusion: The assumption can be partially relaxed

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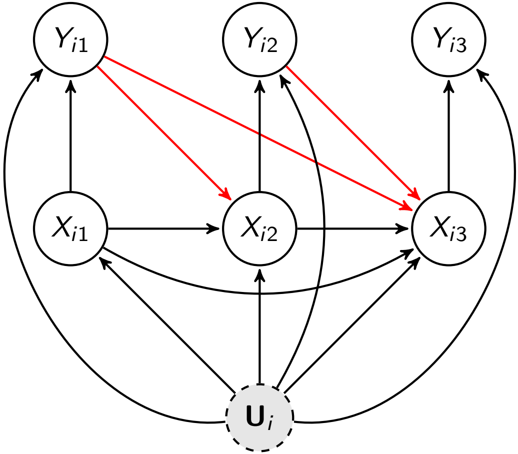

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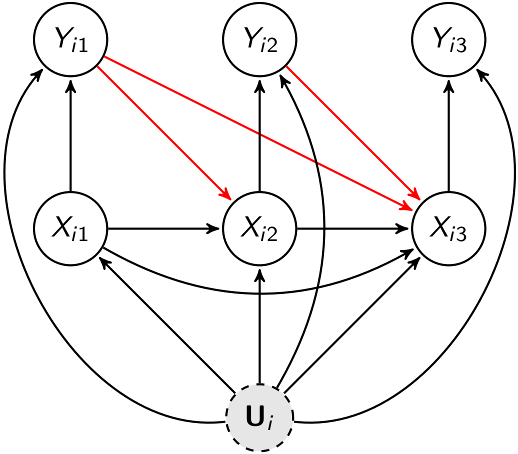

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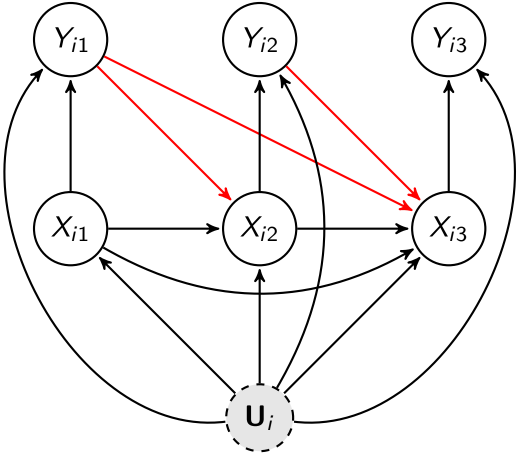

- Correlation between error term and future treatments
- Violation of strict exogeneity

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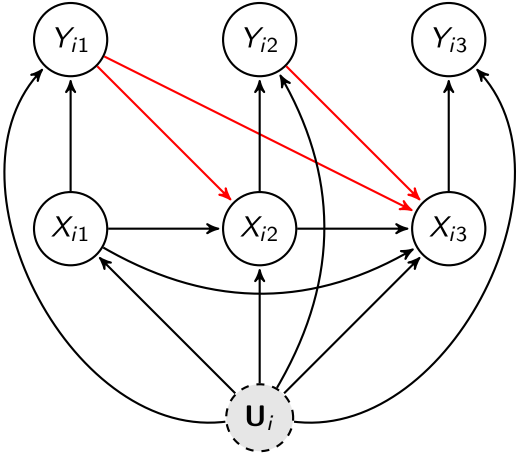

- Correlation between error term and future treatments
- Violation of strict exogeneity
- No adjustment is sufficient

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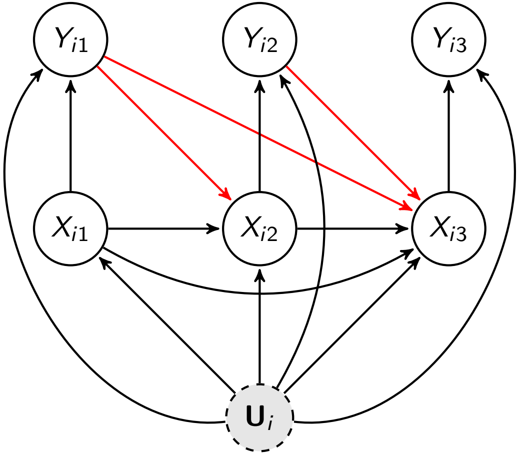

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- Implication: No dynamic causal relationships between treatment and outcome variables

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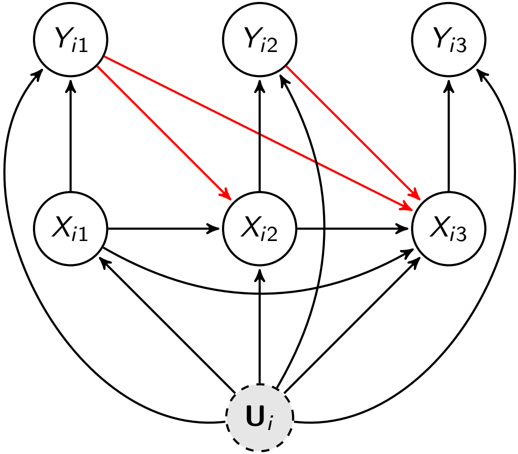

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- Conclusion: The assumption cannot be relaxed

Can't We Just Adjust for Time-Varying Confounders?

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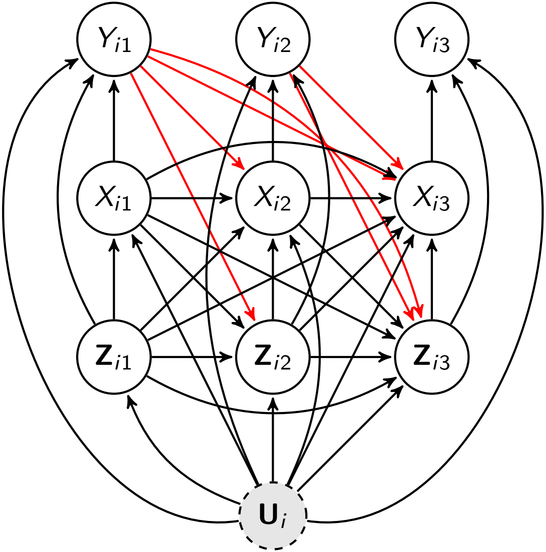

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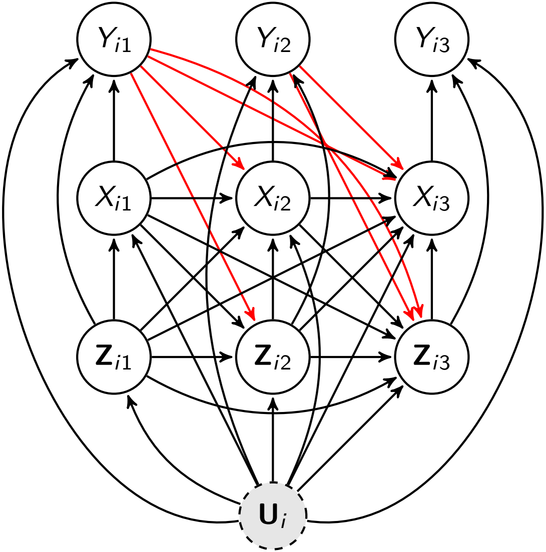

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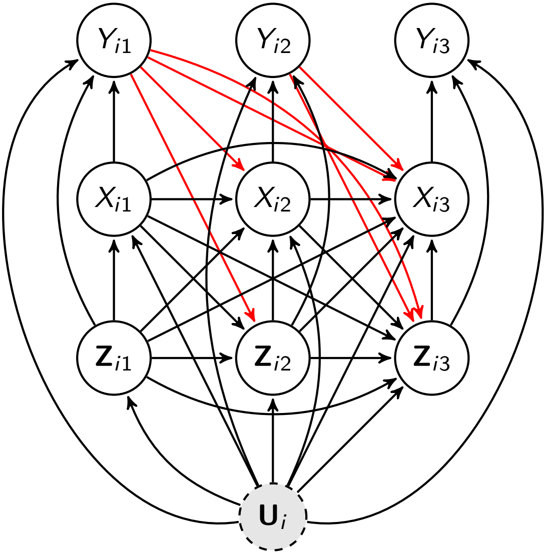

- $Y_{it} = \alpha_i + \beta X_{it} + \gamma^\top \mathbf{Z}_{it} + \epsilon_{it}$
- past outcomes cannot directly affect current treatment
- past outcomes cannot *indirectly* affect current treatment through $\mathbf{Z}_{it}$

But What If I Have Causal Dynamics?

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Alternative: Marginal Structural Models (Robins, Hernán and Brumback, 2000)

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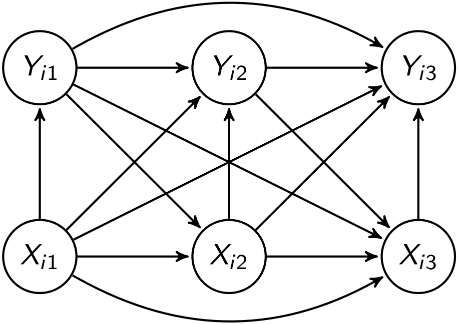

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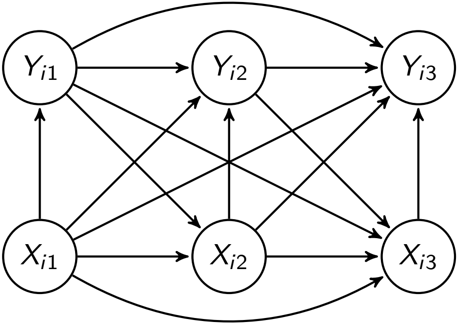

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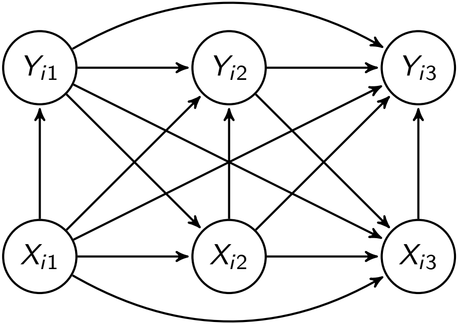

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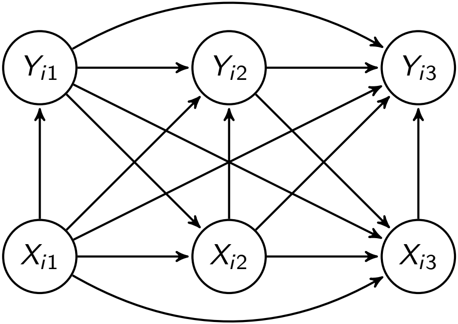

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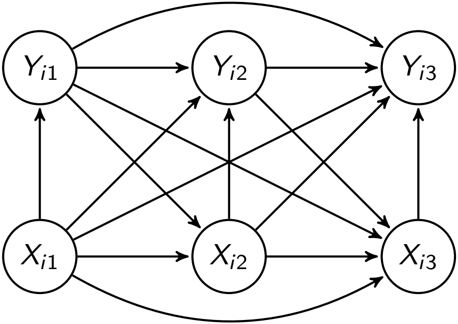

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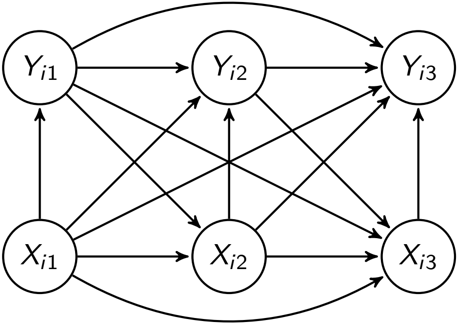

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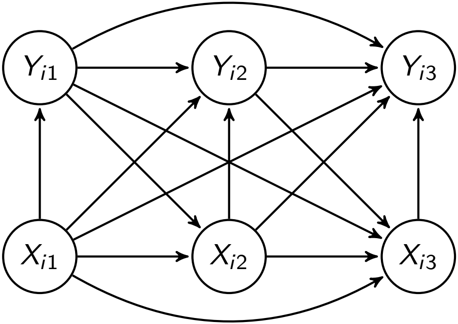

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  2) causal dynamics between treatment and outcome $\rightsquigarrow$ selection-on-observables

Q: What conditions do we need to infer causality?

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A: A clear estimand, an identification strategy and an estimation strategy.

Identification Strategies in This Class

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Essentially everything assumes: consistency/SUTVA (no interference between units, variation in the treatment is irrelevant) and positivity (there is some chance of all getting treatment)

Some Estimation Strategies

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- Stratification

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- Regression (and relatives)

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- Stratification
- Regression (and relatives)
- Matching (not covered)
- Weighting (not covered)

Q: Can you review how to read DAGs?

[2] Courtesy of Erin Hartman's slides for this.

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A: Sure[2]

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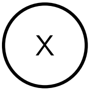
Notation

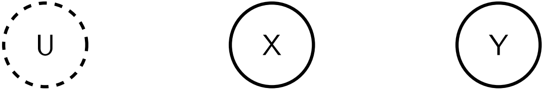


Node – A random Variable. Sometimes drawn as a solid circle $\overset{X}{\bullet}$.

Notation

Dashed line means its unobserved. Sometimes drawn as a hollow circle $\overset{U}{\circ}$.

Notation



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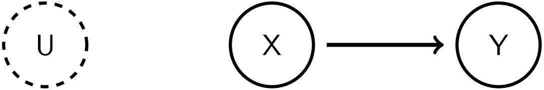
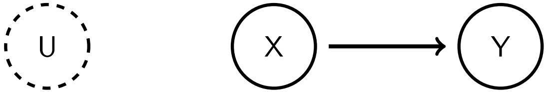


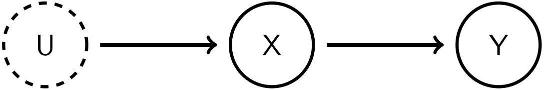
Arrow means "X causes Y".

Notation



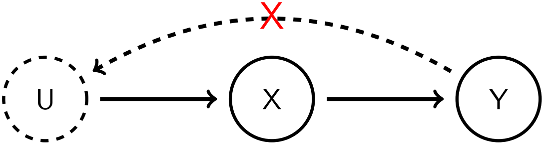
A parent is a direct cause of a child, a child is directly caused by a parent.

Notation



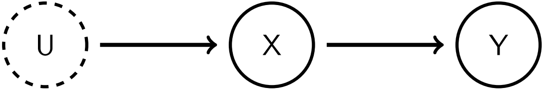
An ancestor is a direct or indirect cause, a descendant is caused, directly or indirectly, by an ancestor.

Notation



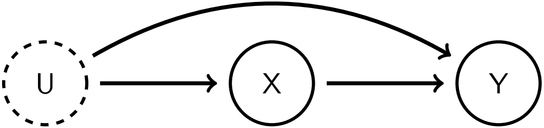
Acyclic implies there are no paths from a variable back to itself.

Notation



A lack of arrows implies no causal relationship.

Notation



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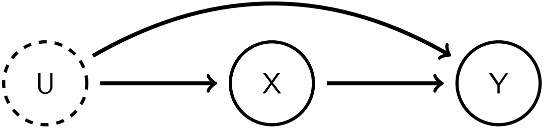
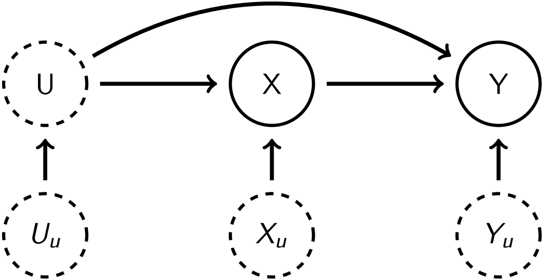


A lack of variables indicates a lack of common causes in the DGP.

Notation



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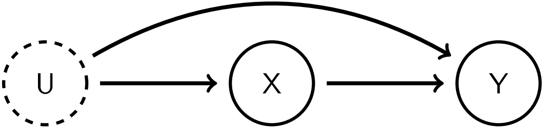
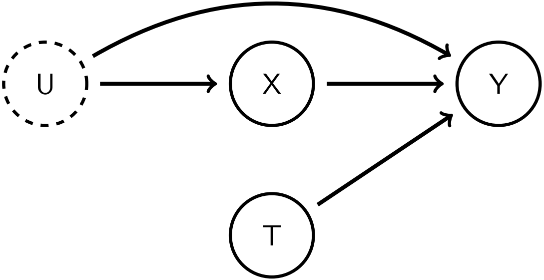


DAGs encode non-parametric structural models.

$$X = f_X(U)$$

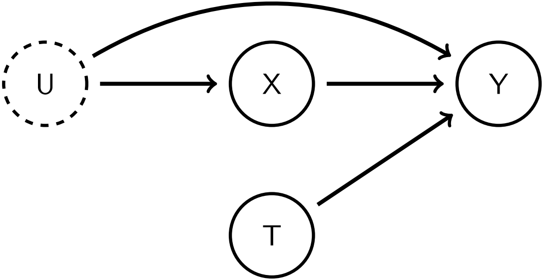
$$Y = f_Y(X, U)$$

Notation



A collider is when a node receives edges from two, or more, other nodes.

Notation

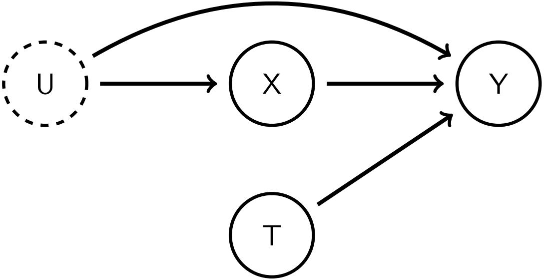


A causal effect can be defined using the *do* operator.

$$P(Y = y \mid do(X = x)) = \sum_{z} P(Y = y \mid X = x, PA = z)P(PA = z)$$

where PA are parents of X, and z ranges of all the combinations of values that the variables in PA can take.

Notation



Then, if T is binary,

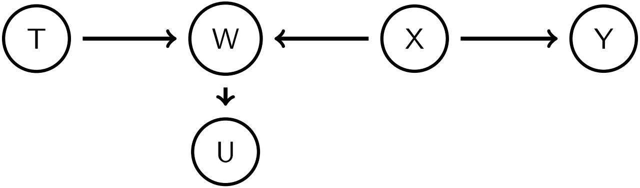
$$ACE = P(Y = 1 \mid do(T = 1)) - P(Y = 1 \mid do(T = 0))$$

and if T is randomized, then:

$$ACE = P(Y = 1 \mid T = 1) - P(Y = 1 \mid T = 0)$$

because there are no parents of T.

d-separation



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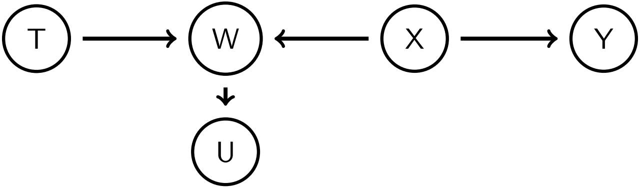


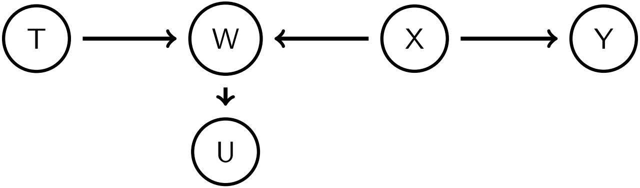
A path p is blocked by a set of nodes Z if and only if:

(1) p contains a chain of nodes $A \rightarrow B \rightarrow C$ or a fork $A \leftarrow B \rightarrow C$ such that the middle node B is in Z or

(2) p contains a collider $A \rightarrow B \leftarrow C$ such that the collision node B is not in Z and no descendant of B is in Z

If Z blocks every path between two nodes X and Y, then X and Y are *d*-separated, conditional on Z, and thus are conditionally independent given Z.

d-separation



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T and Y are d-separated conditional on $\{\}$, because they are blocked by the collider W, meets (2)

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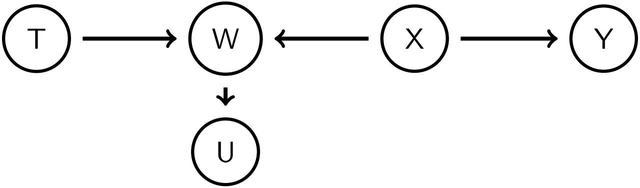

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T and Y are d-connected conditional on $\{W\}$, violates (2).

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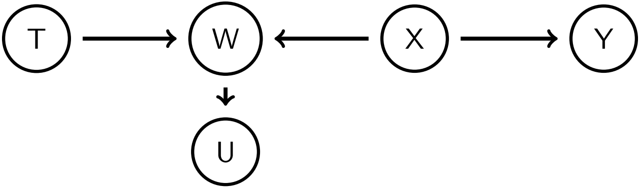

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T and Y are d-separated conditional on $\{W, X\}$, because X blocks the path by criterion (1).

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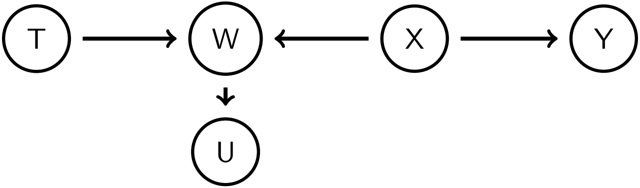

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We can use *d*-separation to do calculate causal effects via the "back-door" criterion, so long as Z does not contain descendants of our treatment of interest.

Q: Can you review how instrumental variables deal with issues of confounding?

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A: We use only the units whose treatment status was effectively randomized by the instrument (because they are compliers).

Q: What are degrees of freedom and how do they play into standard errors?

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A: Let's consider the anatomy of a standard error.

Anatomy of the Standard Error

Imagine we have a regression of Y on a variable of interest X and a vector of other variables $\mathbf{Z}$.

$$\widehat{\text{Var}}(\widehat{\beta}_X) = \frac{\frac{1}{(n-k-1)}\sum_{i=1}^{n} \hat{u}_i^2}{(1 - R^2_{X\sim\mathbf{Z}})\sum_{i=1}^{n}(X_i - \overline{X})^2}$$

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$$\widehat{\text{SE}}(\widehat{\beta_X}) = \sqrt{\widehat{\text{Var}}(\widehat{\beta}_X)}$$

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A: Regression adjustment of experiments can be helpful for improving precision. We don't need it for confounding, but it can improve our standard errors to adjust for pre-treatment covariates that are highly predictive of the output. If done correctly and in moderate-to-large samples, this can dramatically improve your standard errors. Even better though is blocking which is adjustment by design.

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Further Reading:

- Lin, W., 2013. 'Agnostic notes on regression adjustments to experimental data: Reexamining Freedmans critique.' *The Annals of Applied Statistics*
- Athey, S. and Imbens, G.W., 2017. 'The Econometrics of Randomized Experiments.' In *Handbook of Economic Field Experiments* (Vol. 1, pp. 73-140).
- Egap Methods Guide: 10 things you need to know about covariate adjustment. `https://egap.org/methods-guides/10-things-know-about-covariate-adjustment`

Q: Can you discuss the difference between having an instrument and having a mediator?

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A: If we think of the treatment as the mediator of the instrument, it is by the exclusion restriction a total mediator (the direct effect is 0).

Q: How do propensity scores and matching fit into all of this?

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A: They are different ways of conditioning on variables in a selection on observables strategy. Importantly: they are tools for estimation not tools for identification.

Propensity Score as a Low-Dimensional Summary

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- Curse of dimensionality: there will be very few, if any, units in a given stratum of X_i.

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- $\leadsto$ stratifying on e_i is the same in expectation as stratifying on the full X_i.
- The true propensity score is actually a balancing score, which means that $D_i \perp\!\!\!\perp X_i \mid e(X_i)$

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  - NB: propensity scores only achieve balance in expectation

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(Ho, Imai, King, Stuart, 2007: fig.1, Political Analysis)

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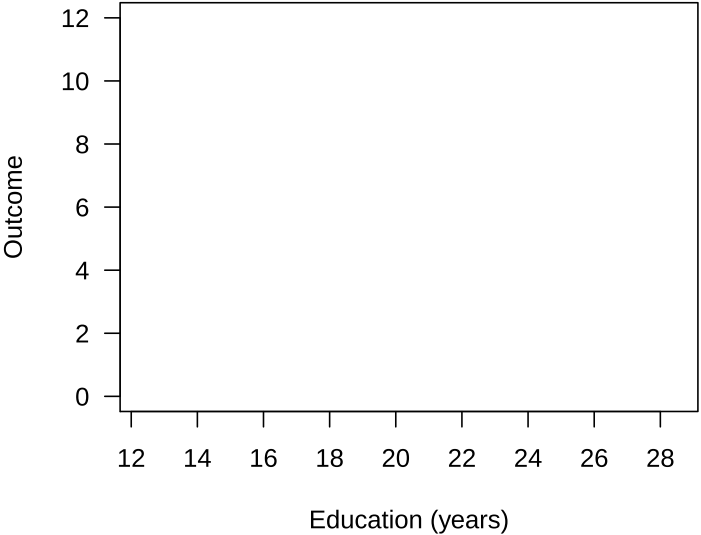

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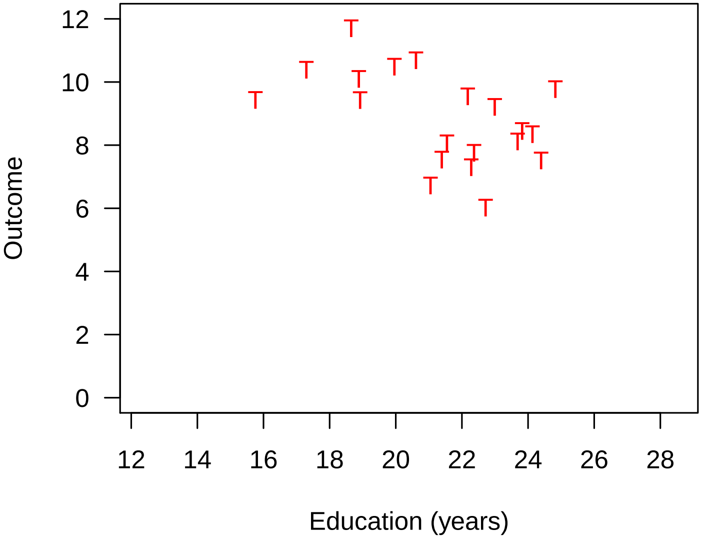

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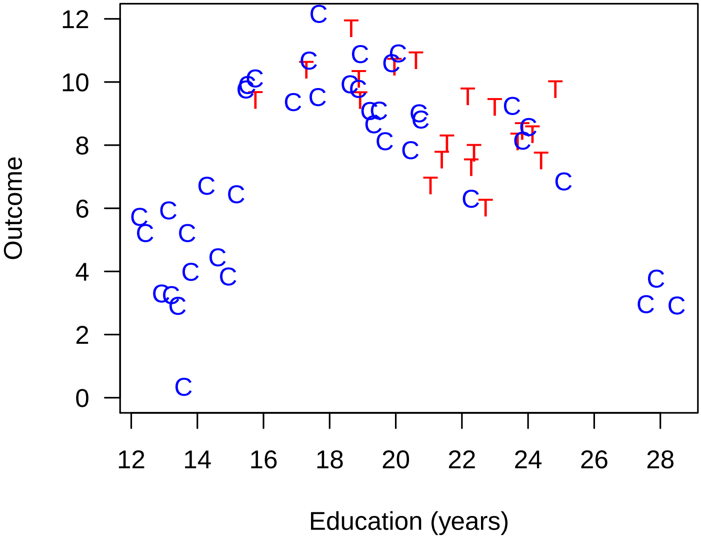

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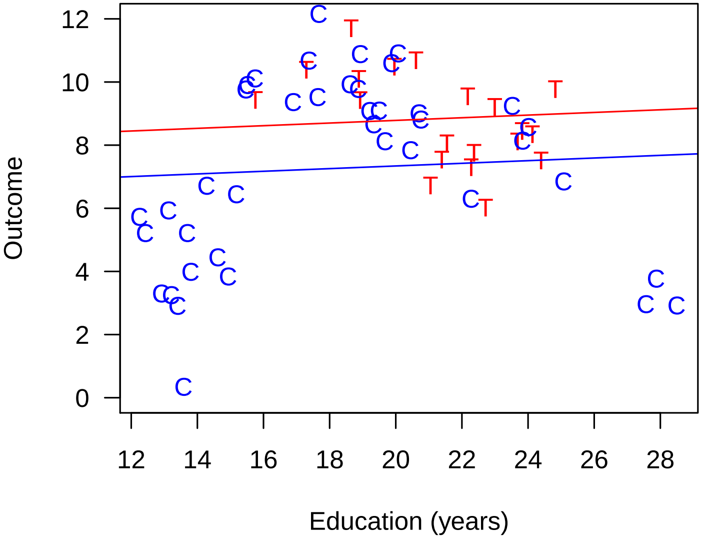

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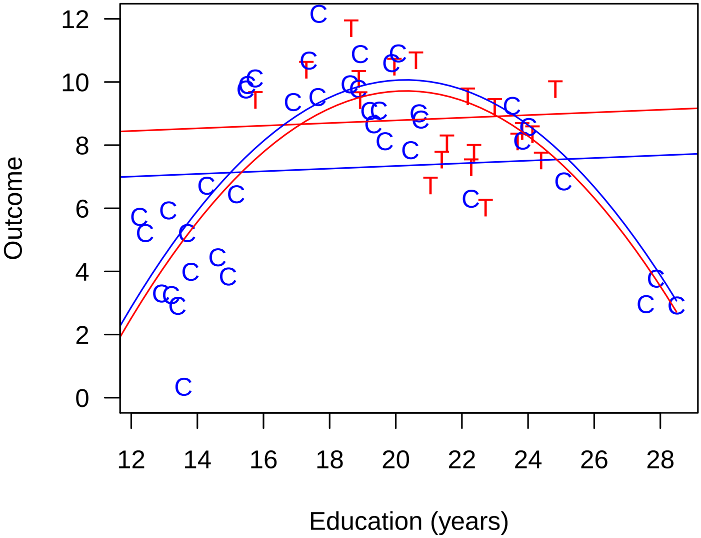

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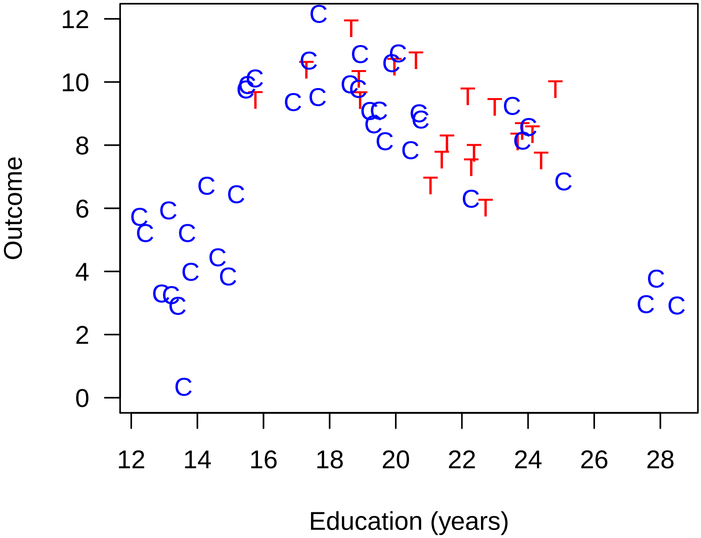

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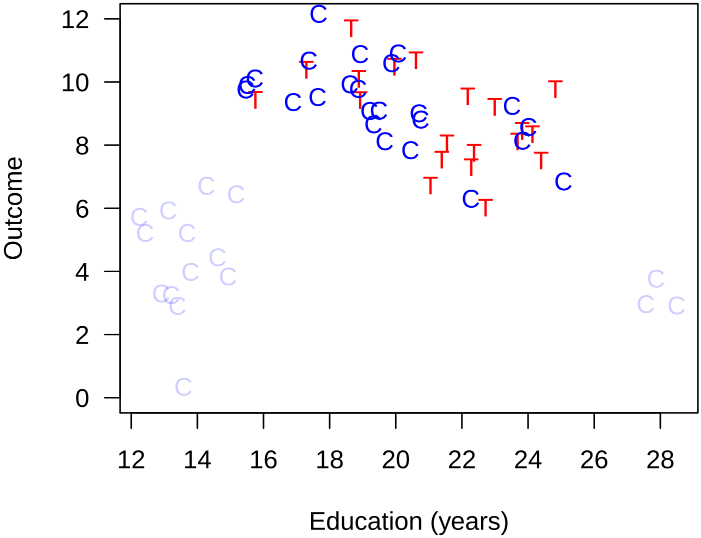

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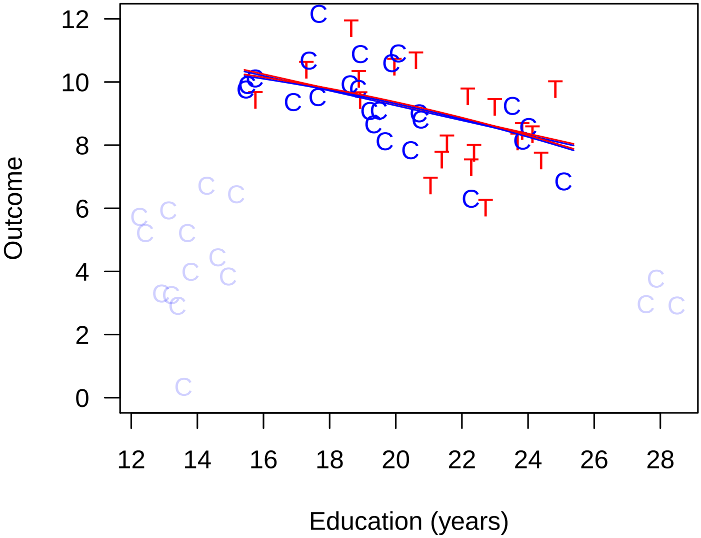

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- Which is the best method? The one that produces the best balance!

Next few slides based on slides by Gary King and Rich Nielsen

Method 1: Mahalanobis Distance Matching

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(Approximates Fully Blocked Experiment)

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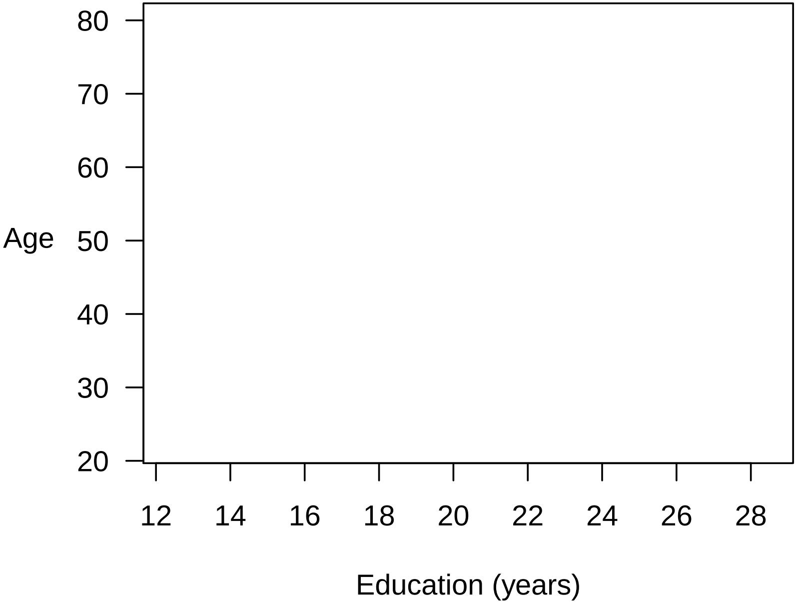

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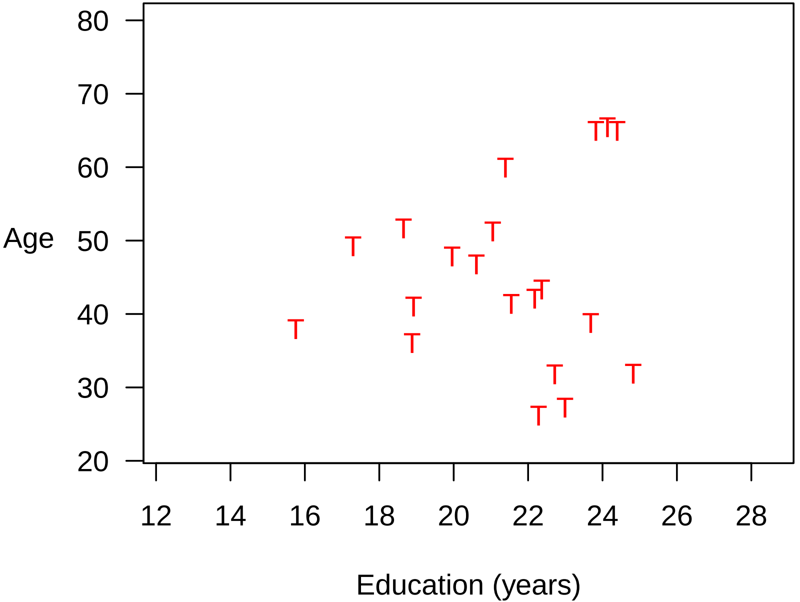

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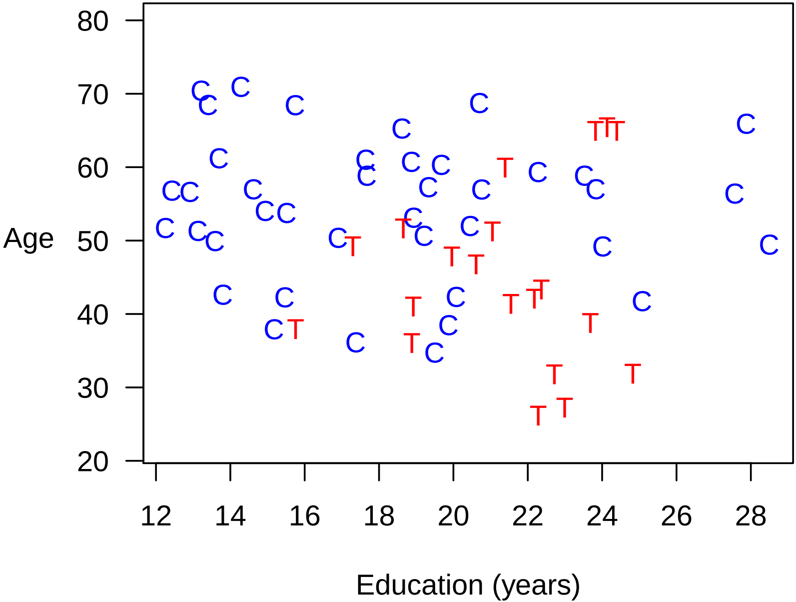

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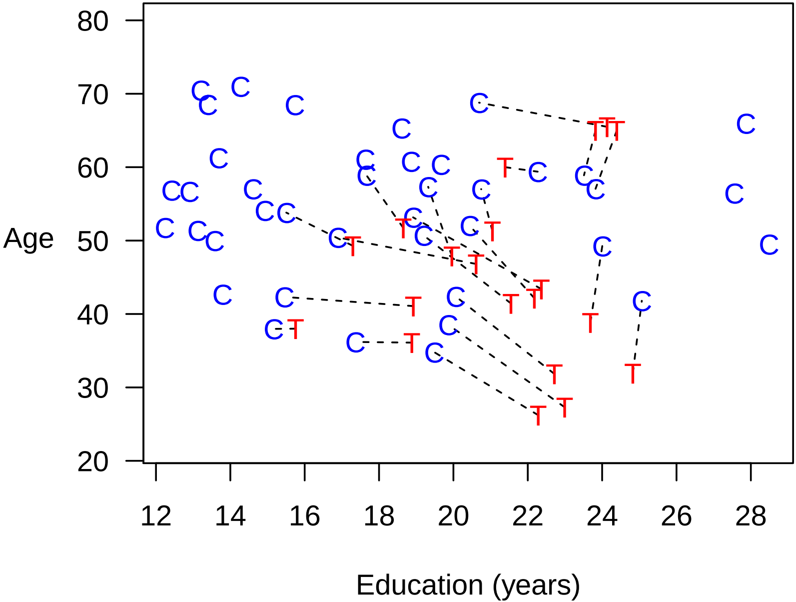

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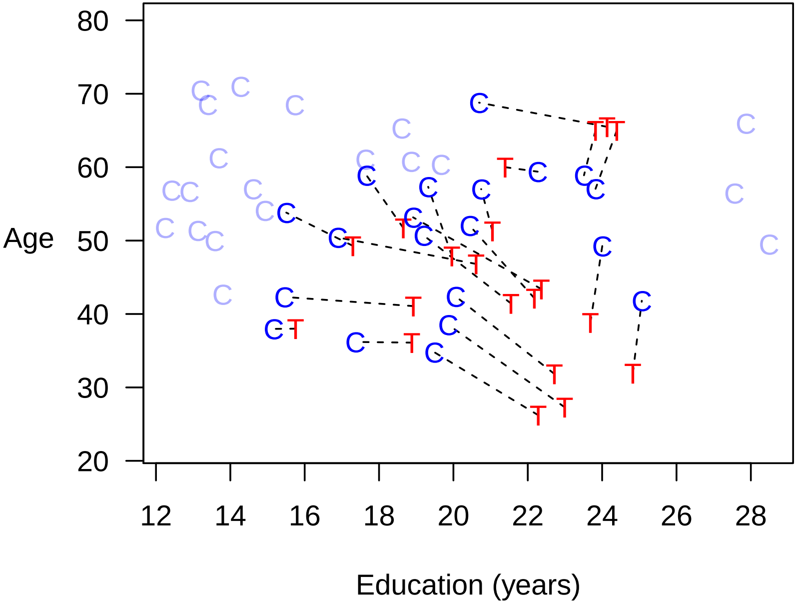

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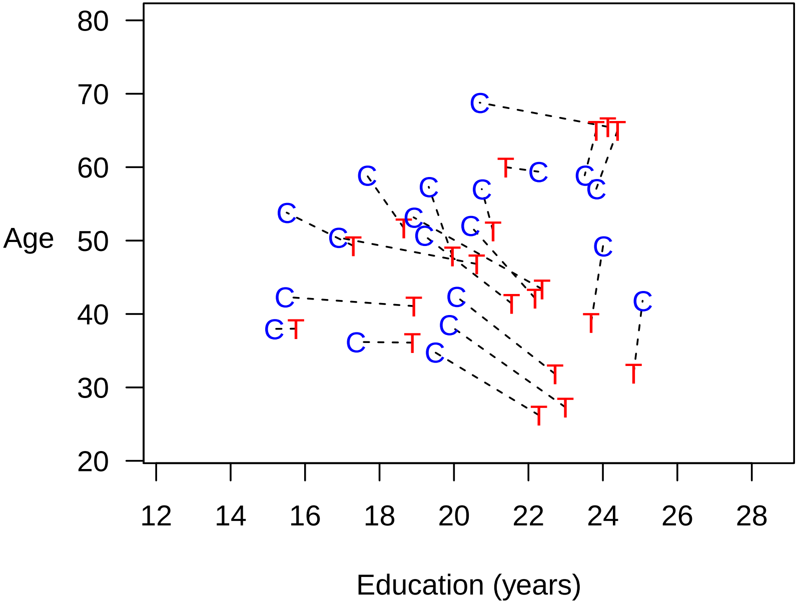

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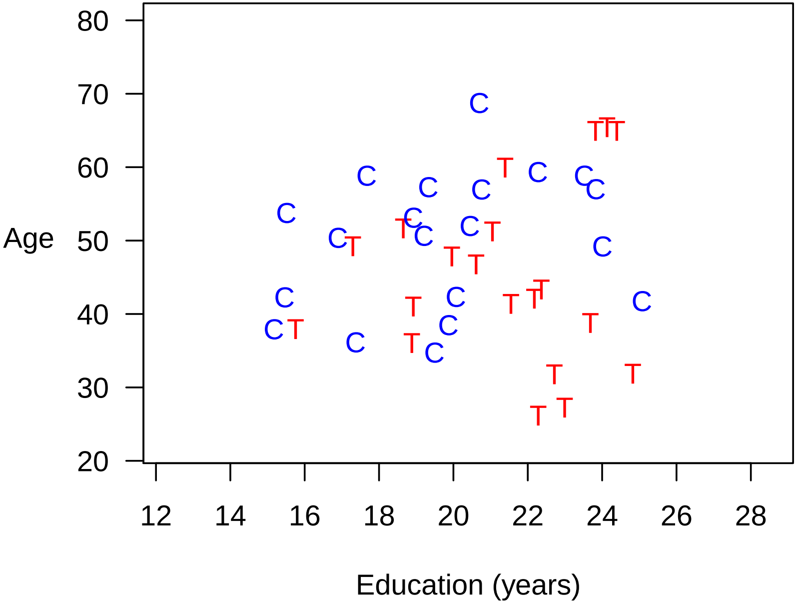

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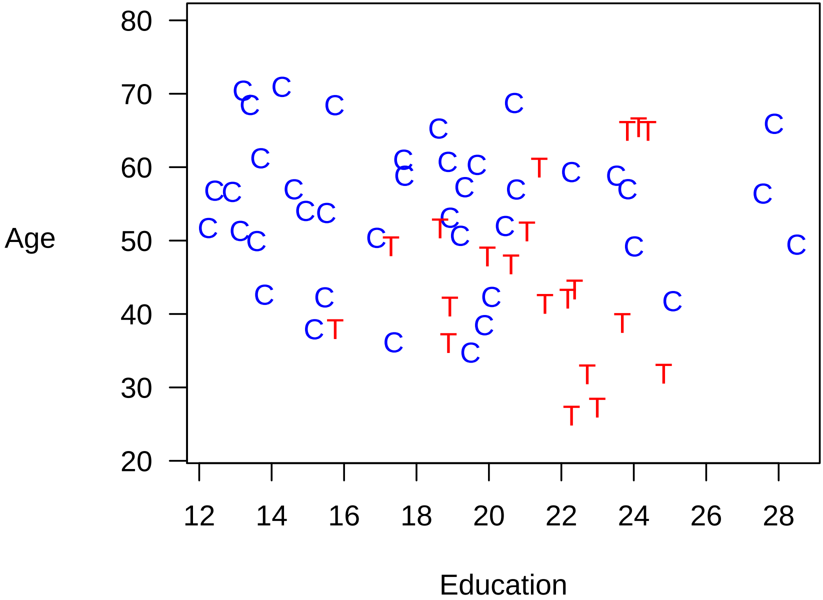

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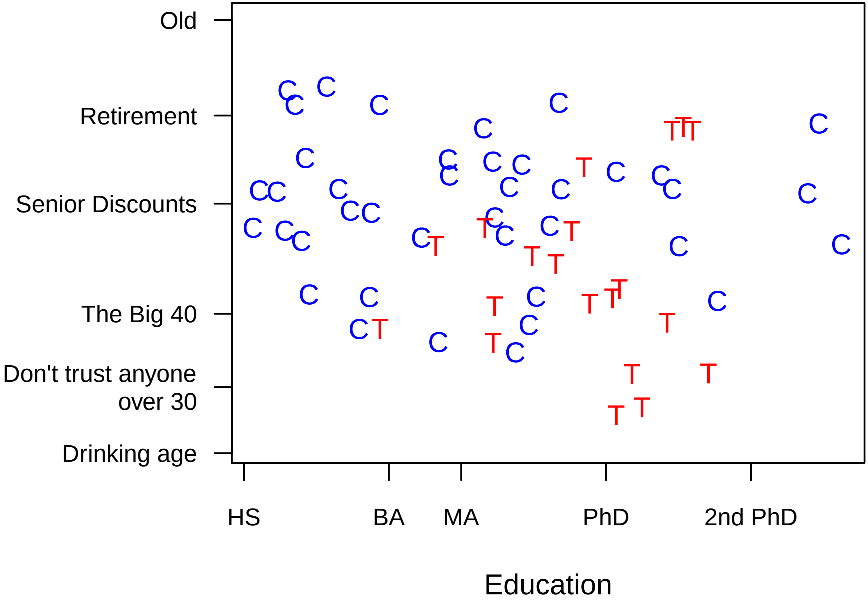

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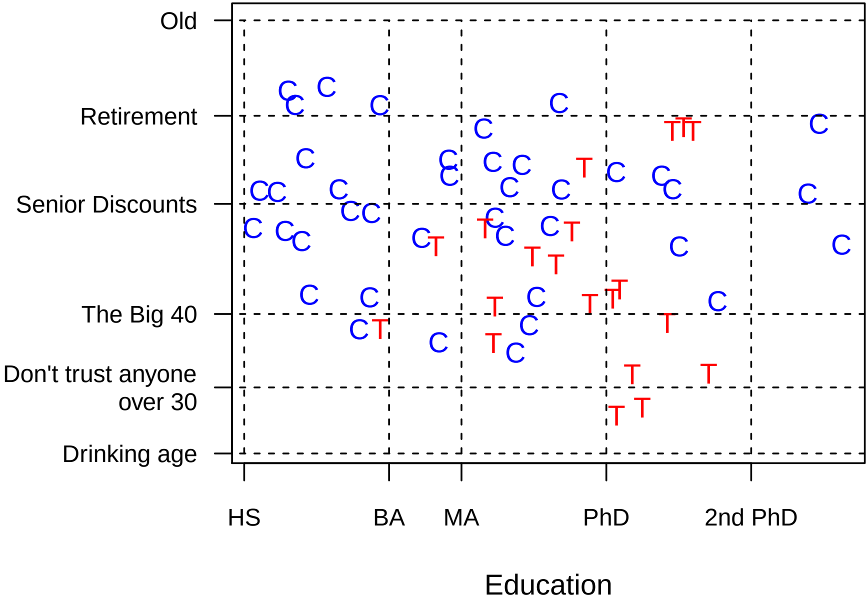

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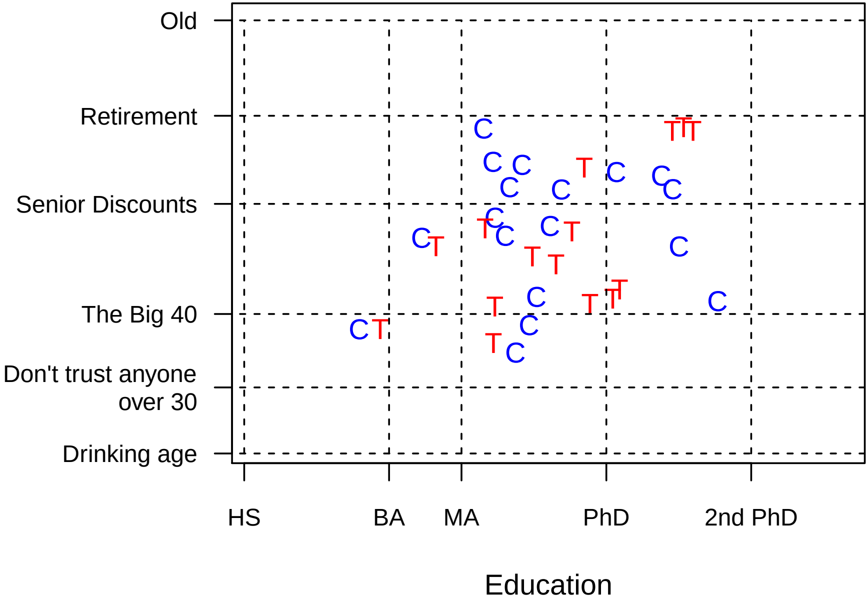

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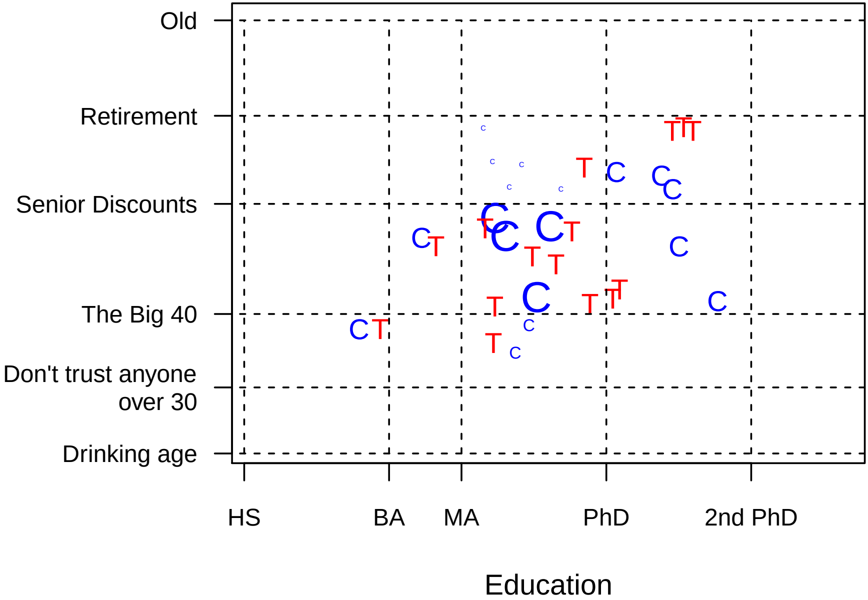

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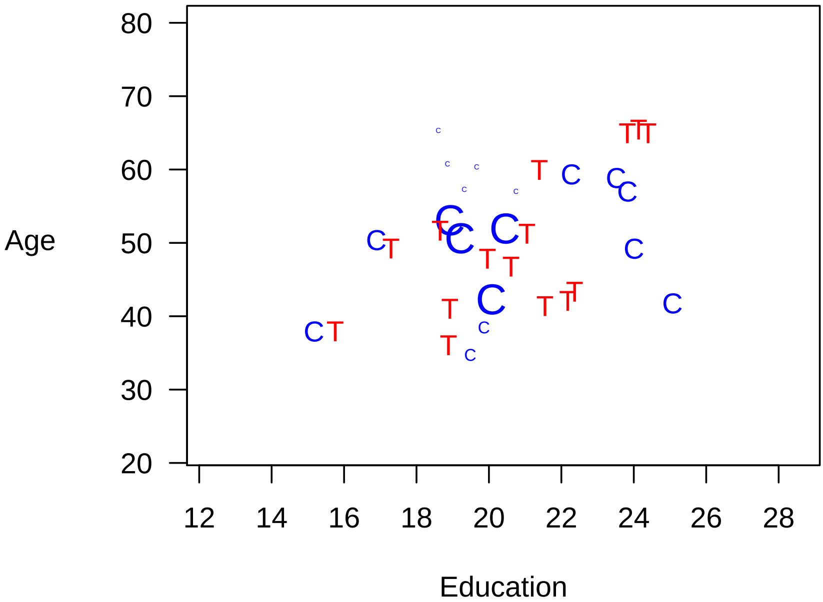

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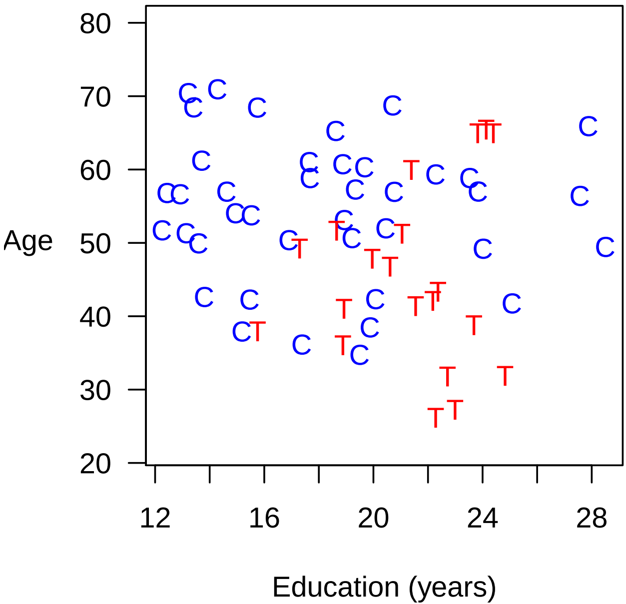

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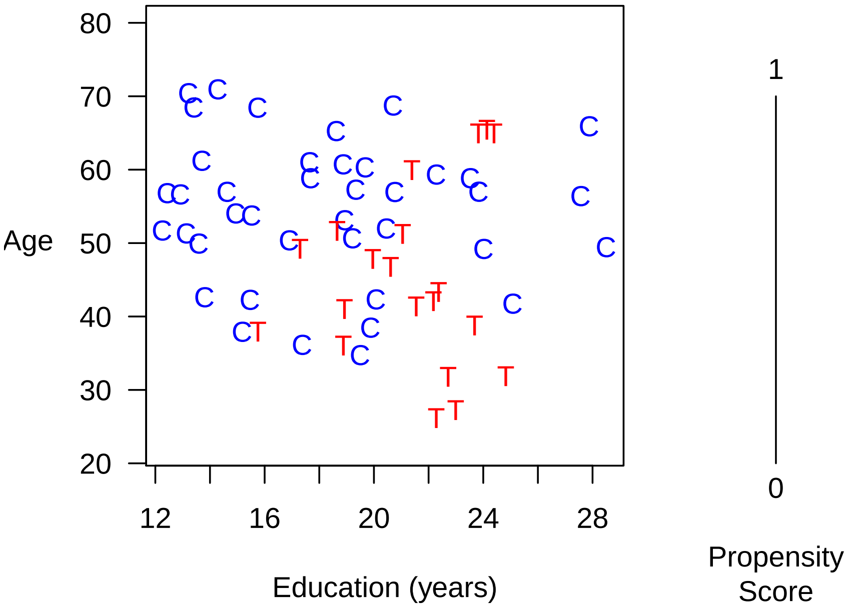

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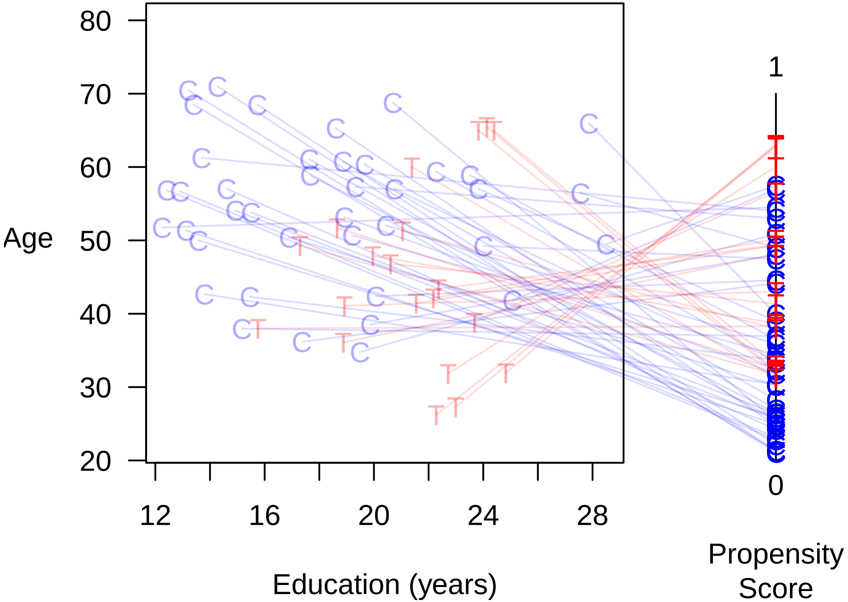

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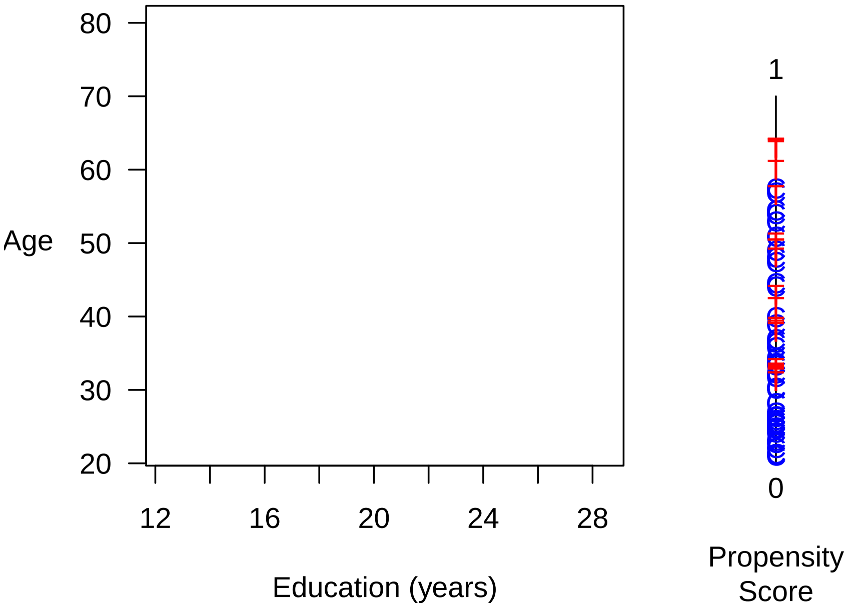

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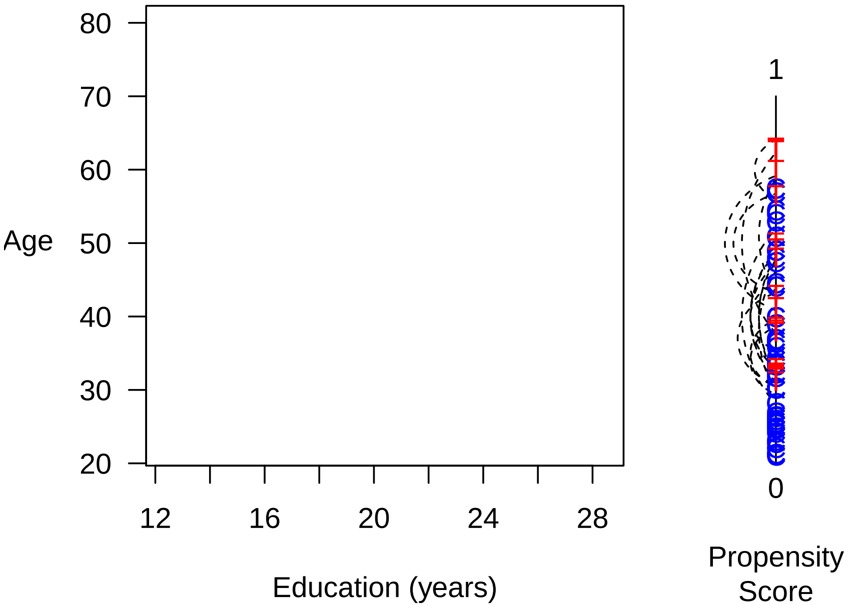

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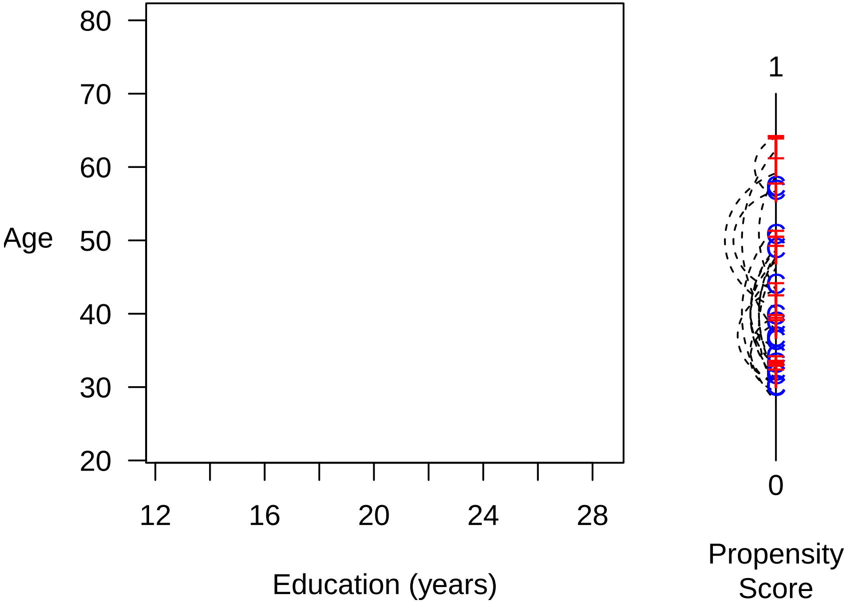

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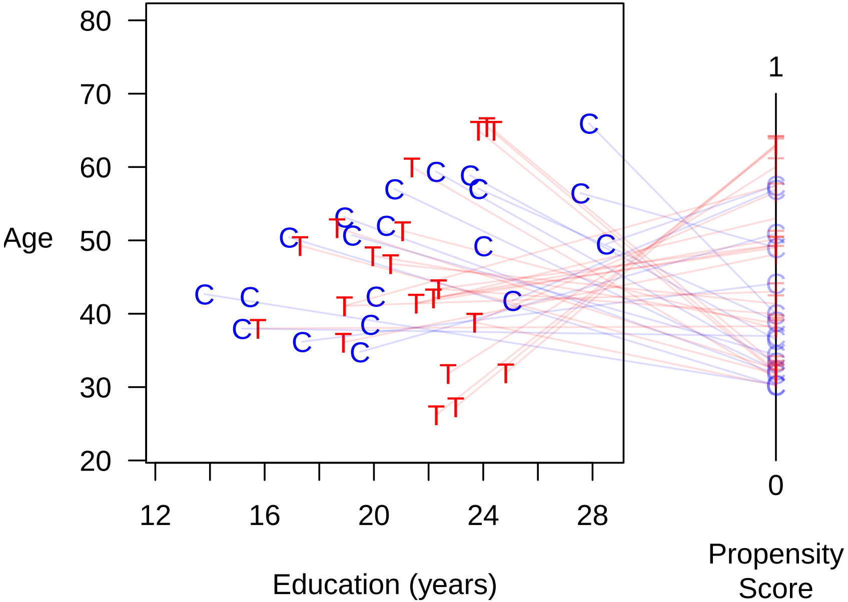

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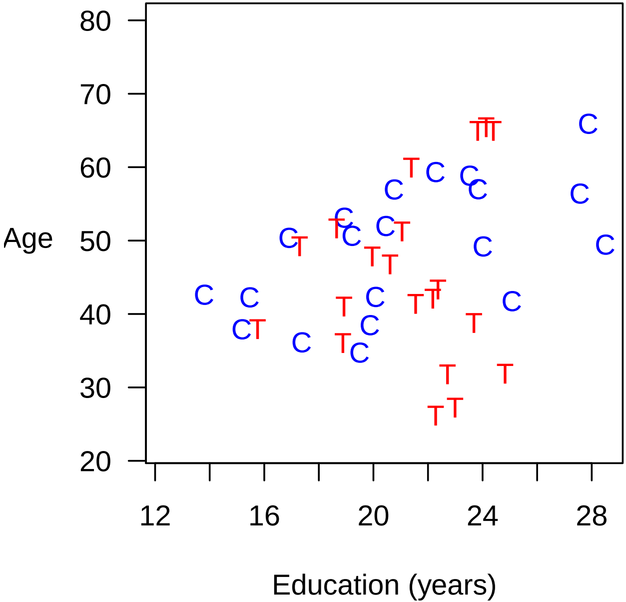

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A: Sure. Generally speaking, they are a way of borrowing information.

Eight Schools Data

School	Est. Effect	SE
A	28	15
B	8	10
C	-3	16
D	7	11
E	-1	9
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Policy Question: What is the effect size in School A?

Eight Schools Background

- ETS analyzes special coaching program on test scores
- 8 separate parallel experiments in different high schools
- Outcome was the score on a special administration of SAT-V with scores varying between 200 and 800 ($\mu = 500, \sigma = 100$)
- SAT is designed to be resistant to short-term efforts intended to boost performance, but each school thought it was a success.
- No prior reason to believe that one program would be more effective than the others
- Treatment effects estimated controlling for PSAT-M and PSAT-V scores
- A bit over the 30 students in each school
- For the sake of scale: an 8-point increase in the score indicates about 1 more test item was answered correctly.
- (Analysis is from Rubin 1981, treatment via Gelman et al 2015)

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- It is the "same course" in every school, but they are different schools.

Options for Analysis

There are two clear options:

1. an unpooled analysis in which we use separate estimates for every school- in this case directly from the table

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$$\bar{y}_{\cdot} = \frac{\sum_{j=1}^{8} \frac{1}{\sigma_j^2} \bar{y}_j}{\sum_{j=1}^{8} \frac{1}{\sigma_j^2}}$$

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   - the pooled estimate is 7.7 with standard error of 4.1. Thus the confidence interval is $[-.5, 15.9]$

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  2. assume that the observed effect in each school is sampled from a normal distribution with a mean equal to its true effect and standard deviation given in the table
- This model contains both the separate and pooled estimates as limiting special cases. If we force the standard deviation of the true effects to be zero, then all school get the same estimate, if we let it go to infinity we get the separate estimates

The Model

$$\bar{y}_j|\theta_j \sim \text{Normal}(\theta_j, \sigma_j^2)$$
$$\theta_j|\mu, \tau \sim \text{Normal}(\mu, \tau^2)$$
$$p(\mu, \tau) = p(\mu|\tau)p(\tau) \propto p(\tau)$$

Known: $\bar{y}_j, \sigma_j^2$
Unknown: τ, μ, θ

Some Mechanics

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\hat{\theta}_j &= \frac{\frac{1}{\sigma_j^2}\bar{y}_j + \frac{1}{\tau^2}\mu}{\frac{1}{\sigma_j^2} + \frac{1}{\tau^2}} \\
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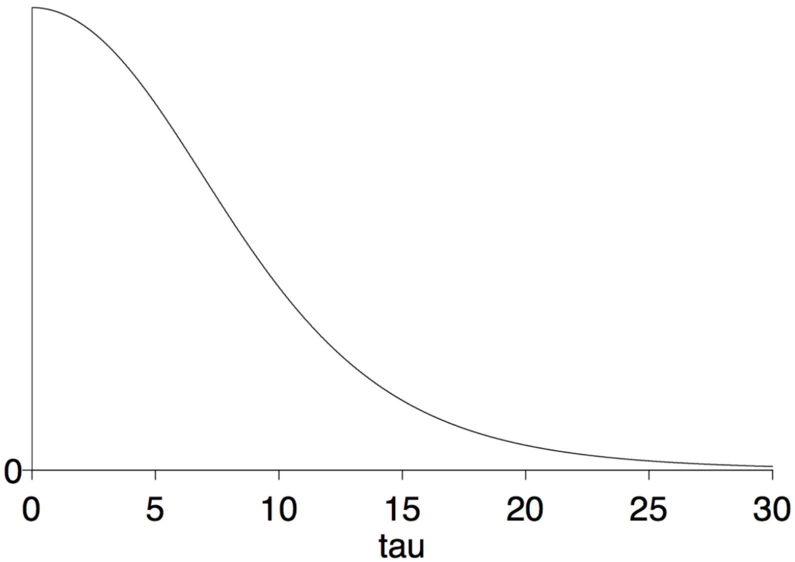
- This captures our intuition that while School A might have a larger effect, it is perhaps an overestimate
- The form show us that the amount of shrinkage is relative to our certainty about the estimate and how much we believe the individual effects matter
- Our final guess is that the median effect for school A is about 10 points with 50% probability between 7 and 16

Results



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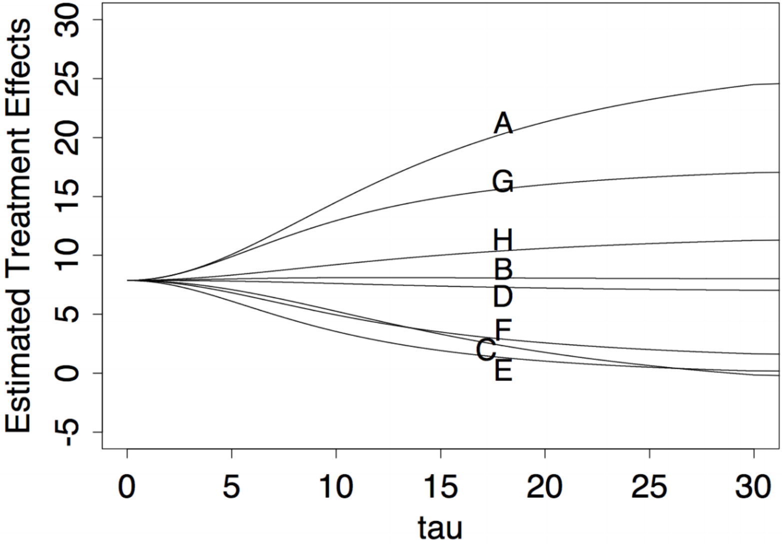

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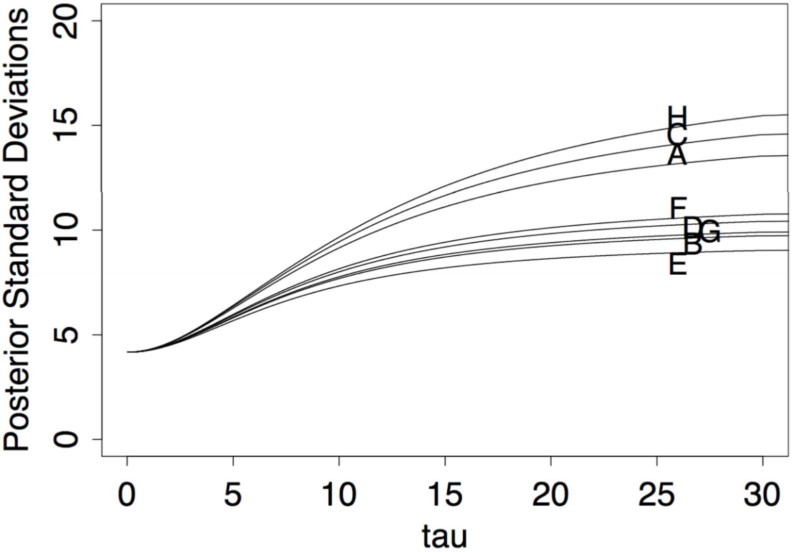
Posterior Standard Deviations
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5
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15
20
25
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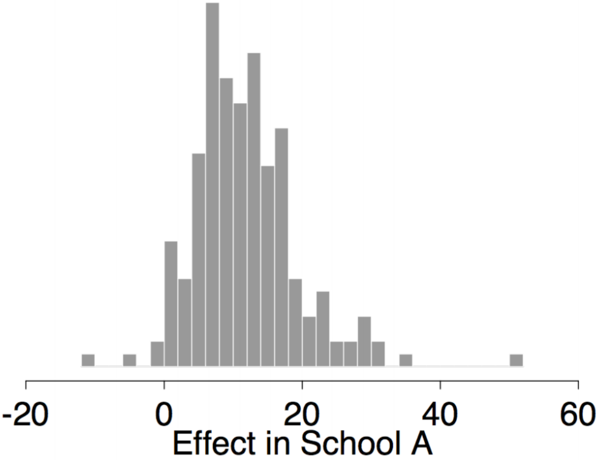

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- Allows us to capture variability in our treatment effects, variances etc.
- Allows us to model dependence in our error terms

Q: How do we determine power?

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A: A combination of the effect size, the variance and the sample size. Unfortunately, only one of which we know. See the `DeclareDesign` suite of packages for this and so much more!

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- lack of clarity about the counterfactual and common support

Q: When should you pick your statistical strategy? How do you balance pre-planning research / literature reviews with potential problems with data/causal assumptions? How much data exploration should you do up front compared to exploration throughout the question? If you have a causal question or idea but arent sure of data, how should you go about searching for potential data and making sure assumptions are reasonable?

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A: Let's chat.

Q: What do you believe will be the biggest applications for social statistics in the future?

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4. Try to explain it to someone else

Where are you?

Where are you?

You've been given a powerful set of tools

Your New Weapons

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  - Expected value, variances, independence
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- Univariate Inference
  - Interval estimation (normal and non-normal Population)
  - Confidence intervals, hypothesis tests, p-values
  - Practical versus statistical significance

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- Multiple Regression
  - OLS estimator for multiple regression
  - Regression assumptions
  - Properties: Bias, Efficiency, Consistency
  - Standard errors, testing, p-values, and confidence intervals
  - Polynomials, Interactions, Dummy Variables
  - F-tests
  - Matrix notation

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- And you learned how to use `R`: you're not afraid of trying something new!

Using these Tools

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So, Admiral Ackbar, now that you've learned how to run these regressions we can just use them blindly, right?


IT'S A TRAP!

Beyond Linear Regressions

You need more training

Beyond Linear Regressions

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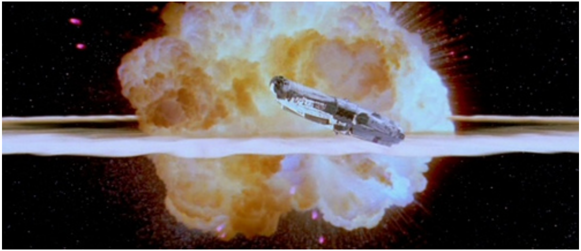
There is so much more to learn! Take classes, read books!

Thanks!

Thanks so much for an amazing semester.



Fill out your evaluations!

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$$\mathbb{E}\left[\frac{1}{N}\sum_{i=1}^{N} \frac{Z_i Y_i}{\pi_i}\right] = \frac{1}{N}\sum_{i=1}^{N} \frac{\mathbb{E}[Z_i] Y_i}{\pi_i} = \frac{1}{N}\sum_{i=1}^{N} \frac{\pi_i Y_i}{\pi_i} = \bar{Y}_N$$

- Reweights the sample to be representative of the population.

Back to causal effects

- With a completely randomized experiment, we can just use the simple differences in means:

$$\mathbb{E}[Y_i|D_i = 1] - \mathbb{E}[Y_i|D_i = 0] = \mathbb{E}[Y_i(1)] - \mathbb{E}[Y_i(0)]$$

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- With subclassification, we binned X_i, calclulated within-bin differences and then averaged across the bins, just like this.

Searching for the weights

$$\mathbb{E}[Y_i(d)] = \sum_{x \in \mathcal{X}} \mathbb{E}[Y_i | D_i = d, X_i = x] \mathbb{P}(X_i = x)$$

- Compare this to the the within treatment group average:

$$\mathbb{E}[Y_i | D_i = d] = \sum_{x \in \mathcal{X}} \mathbb{E}[Y_i | D_i = d, X_i = x] \mathbb{P}(X_i = x | D_i = d)$$

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- How should we reweight the data from an observational study?

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- How should we reweight the data from an observational study?
- If we were to reweight the data by $W_i = 1/\mathbb{P}(D_i = d | X_i)$, then we would break the relationship between D_i and X_i.

Weights

- Single binary covariate. Define the weight function:

$$w(d,x) = \frac{1}{e(x)^d(1-e(x))^{1-d}}$$

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- If $(D_i, X_i) = (0, 0)$:

$$W_i = \frac{1}{1 - e(0)} = \frac{1}{\mathbb{P}(D_i = 0|X_i = 0)}$$

Example

	$X_i = 0$	$X_i = 1$
$D_i = 0$	4	3
$D_i = 1$	4	9

- $\mathbb{P}(D_i = 1 | X_i = 0) = 0.5$

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	$X_i = 0$	$X_i = 1$
$D_i = 0$	$1/0.5$	$1/0.25$
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- $\mathbb{P}_W(D_i = 1 | X_i = x) = 0.5$ for all x

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- Important point: $\mathbb{P}_W(D_i = 1 | X_i = 1) = \mathbb{P}_W(D_i = 1 | X_i = 0) = \frac{1}{\omega^*}$
- $\rightsquigarrow D_i$ independent of X_i in the reweighted data.

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- We want to see what the conditional weighted mean identifies:

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$$\hat{\tau} = \frac{1}{N}\sum_{i=1}^{N}\left(\frac{D_i Y_i}{e(X_i)} - \frac{(1-D_i)Y_i}{1-e(X_i)}\right)$$

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- The above two results give us that this esimator is unbiased.
- This is sometimes called the Horvitz-Thompson estimator due to the close connection to the survey sampling estimator.