GSERM - St. Gallen 2022

Analyzing Panel Data

June 9, 2022

Causation

The goal: **Making causal inferences from observational data.**

- Establish and measure the *causal* relationship between variables in a non-experimental setting.
- The *fundamental problem of causal inference*:

  It is impossible to observe the causal effect of a treatment or a predictor on a single unit.

- Specific challenges:
  - *Confounding*
  - *Selection bias*
  - *Heterogenous treatment effects*

Causation and Counterfactuals

Causal statements imply <u>counterfactual</u> reasoning.

- "If the cause(s) had been different, the outcome(s) would be different, too."
- Conditioning, probabilistic and causal:

Probabilistic conditioning	Causal conditioning
$\Pr(Y\|X = x)$	$\Pr[Y\|do(X = x)]$
Factual	Counterfactual
Select a sub-population	Generate a new population
Predicts passive observation	Predicts active manipulation
Calculate from full DAG*	Calculate from surgically-altered DAG*
Always identifiable when X and Y are observable	Not always identifiable even when X and Y are observable

*See below. Source: Swiped from Shalizi, "Advanced Data Analysis from an Elementary Point of View", Table 23.1.

- Causality (typically) implies / requires:
  - *Temporal ordering*
  - *Mechanism*
  - *Correlation*

Notation

- N observations indexed by i, $i \in \{1, 2, ...N\}$
- Outcome variable Y
- Interest: the effect on Y of a treatment variable W:
  - $W_i = 1 \leftrightarrow$ observation i is "treated"
  - $W_i = 0 \leftrightarrow$ observation i is "control"

Potential Outcomes

- Y_{0i} = the value of Y_i if $W_i = 0$
- Y_{1i} = the value of Y_i if $W_i = 1$
- $\delta_i = (Y_{1i} - Y_{0i})$ = the treatment effect of W

Treatment Effects

The average treatment effect (ATE) is just:

$$\begin{aligned} \text{ATE} \equiv \bar{\delta} &= \mathrm{E}(Y_{1i} - Y_{0i}) \\ &= \frac{1}{N}\sum_{i=1}^{N} Y_{1i} - Y_{0i}. \end{aligned}$$

BUT we observe only Y_i:

$$Y_i = \begin{cases} Y_{0i} \text{ if } W_i = 0, \\ Y_{1i} \text{ if } W_i = 1. \end{cases}$$

or (equivalently)

$$Y_i = W_i Y_{1i} + (1 - W_i) Y_{0i}.$$

Estimating Treatment Effects

Key to estimating treatment effects: **Assignment mechanism for** W.

Neyman/Rubin/Holland: Treat inability to observed Y_{0i} / Y_{1i} as a missing data problem.

[press "pause"]

Notation:

$$\underset{N\times K}{\mathbf{X}} \cup \{\mathbf{W}, \mathbf{Z}\}$$

W have some missing values,
Z are "complete"

Consider a matrix **R** with:

$$R_{ik} = \begin{cases} 1 & \text{if } X_{ik} \text{ is missing,} \\ 0 & \text{otherwise.} \end{cases}$$

$$\pi_{ik} = \Pr(R_{ik} = 1)$$

Missing Data (continued)

Rubin's flavors of missingness:

- Missing completely at random ("MCAR") (= "ignorable"):

$$\mathbf{R} \perp \{\mathbf{Z}, \mathbf{W}\}$$

- Missing at random ("MAR") (conditionally "ignorable"):

$$\mathbf{R} \perp \mathbf{W} | \mathbf{Z}$$

- Anything else is "informatively" (or "non-ignorably") missing.

[press "play"]

Estimating Treatment Effects

Key to estimating treatment effects: **Assignment mechanism for** W.

Neyman/Rubin/Holland: Treat inability to observed Y_{0i} / Y_{1i} as a missing data problem.

- If the "missingness" due to the value of W_i is orthogonal to the values of Y, then it is ignorable. Formally:

$$\Pr(W_i|\mathbf{X}_i, Y_{0i}, Y_{1i}) = \Pr(W_i|\mathbf{X}_i)$$

- If that "missingness" is non-orthogonal, then it is not ignorable, and can lead to bias in estimation
- Non-ignorable assignment of W requires understanding the mechanism by which that assignment occurs

SUTVA

One more thing: the stable unit-treatment value assumption ("SUTVA")

- Requires that there be two and only two possible values of Y for each observation i...
- "the observation (of Y_i) on one unit should be unaffected by the particular assignment of treatments to the other units."
- $\equiv$ the "assumption of no interference between units," meaning:
  - Values of Y for any two i, j ($i \neq j$) observations do not depend on each other
  - Treatment effects are homogenous within categories defined by W

Treatment Effects Under Randomization of W

If W_i is assigned randomly, then:

$$\Pr(W_i) \perp Y_{0i}, Y_{1i}$$

and so:

$$\Pr(W_i | Y_{0i}, Y_{1i}) = \Pr(W_i) \forall\, Y_{0i}, Y_{1i}.$$

This means that the "missing" data on Y_0/Y_1 are ignorable (here, in the special case where the $\mathbf{X}_i$ on which W_i depends is null). This in turn means that:

$$f(Y_{0i}|W_i = 0) = f(Y_{0i}|W_i = 1) = f(Y_i|W_i = 0) = f(Y_i|W_i = 1)$$

and

$$f(Y_{1i}|W_i = 0) = f(Y_{1i}|W_i = 1) = f(Y_i|W_i = 0) = f(Y_i|W_i = 1)$$

Randomized W (continued)

Implication: Y_{0i} and Y_{1i} are (not identical but) *exchangeable*...

This in turn means that:

$$E(Y_{0i}|W_i) = E(Y_{1i}|W_i)$$

and so

$$\begin{aligned} \widehat{\text{ATE}} &= \text{E}(Y_i|W_i = 1) - \text{E}(Y_i|W_i = 0) \\ &= \bar{Y}_{W=1} - \bar{Y}_{W=0}. \end{aligned}$$

will be an unbiased estimate of the ATE.

Observational Data: W Depends on $\mathbf{X}$

Formally,

$$Y_{0i}, Y_{1i} \perp W_i | \mathbf{X}_i.$$

Here,

- $\mathbf{X}$ are *known confounders* that (stochastically) determine the value of W_i,
- Conditioning on $\mathbf{X}$ is necessary to achieve exchangeability.

So long as W is entirely due to $\mathbf{X}$, we can condition:

$$f(Y_{1i}|\mathbf{X}_i, W_i = 1) = f(Y_{1i}|\mathbf{X}_i, W_i = 0) = f(Y_i|\mathbf{X}_i, W_i)$$

and similarly for Y_{0i}.

W Depends on $\mathbf{X}$ (continued)

Estimands:

- the *average treatment effect for the treated* (ATT):

$$\text{ATT} = \text{E}(Y_{1i}|W_i = 1) - \text{E}(Y_{0i}|W_i = 1).$$

- the *average treatment effect for the controls* (ATC):

$$\text{ATC} = \text{E}(Y_{1i}|W_i = 0) - \text{E}(Y_{0i}|W_i = 0).$$

Corresponding estimates:

$$\widehat{\text{ATT}} = \text{E}\{[\text{E}(Y_i|\mathbf{X}_i, W_i = 1) - \text{E}(Y_i|\mathbf{X}_i, W_i = 0)]|W_i = 1\}.$$

and

$$\widehat{\text{ATC}} = \text{E}\{[\text{E}(Y_i|\mathbf{X}_i, W_i = 1) - \text{E}(Y_i|\mathbf{X}_i, W_i = 0)]|W_i = 0\}.$$

Note that in both cases **the expectation of the whole term is conditioned on** W_i.

Confounding

Confounding occurs when one or more observed or unobserved factors $\mathbf{X}$ affect the causal relationship between W and Y.

Formally, confounding requires that:

- $\text{Cov}(\mathbf{X}, W) \neq 0$ (the confounder is associated with the "treatment")
- $\text{Cov}(\mathbf{X}, Y) \neq 0$ (the confounder is associated with the outcome)
- $\mathbf{X}$ does not "lie on the path" between W and Z (that is, $\mathbf{X}$ is not affected by either W or Y).

Digression: DAGs

Directed acyclic graphs (DAGs) are a tool for visualizing and interpreting structural/causal phenomena.

- DAGs comprise:
  - Nodes (typically, variables / phenomena) and
  - Edges (or lines; typically, relationships/causal paths).
- Directed means each edge is *unidirectional*.
- Acyclical means exactly what it suggests: If a graph has a "feedback loop," it is not a DAG.
- Read more at the Wikipedia page, or at this useful page.

Know your DAG

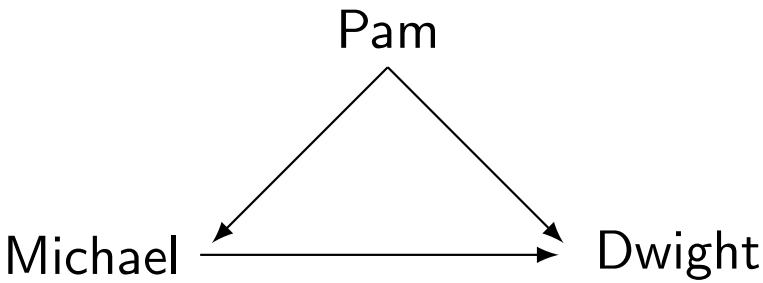


Figure: A DAG

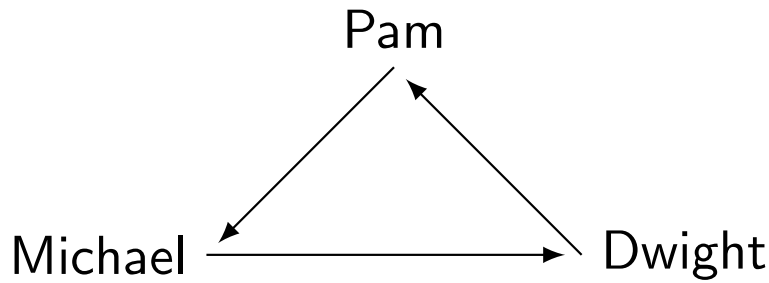
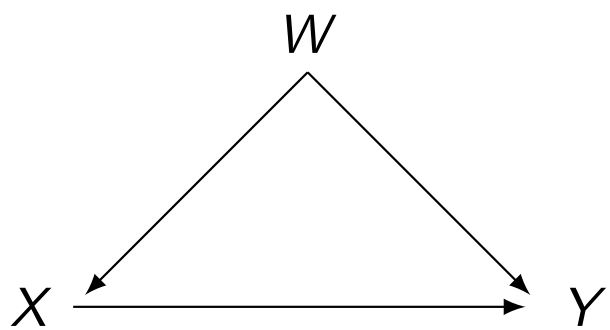


Figure: Not a DAG

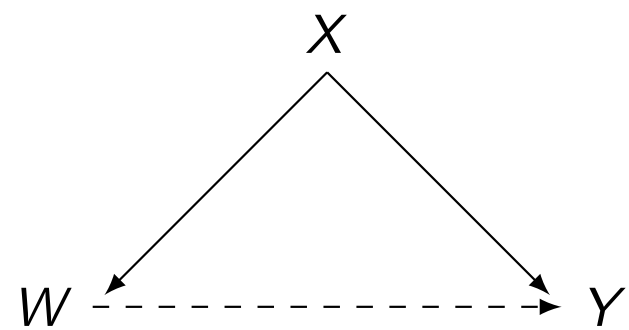
DAGs and Confounding

No Confounding

A "Collider"



Confounding



What We're On About

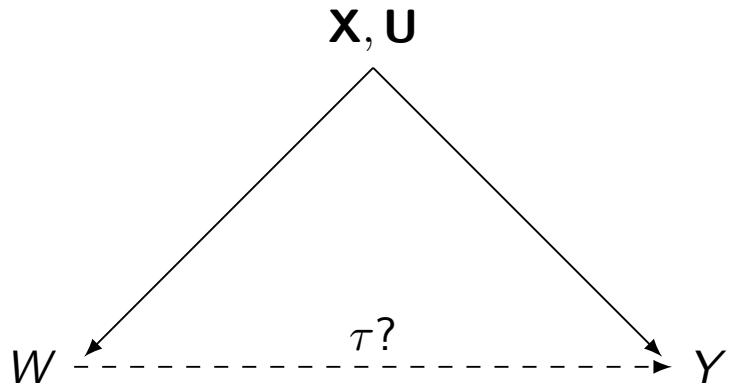


Figure: Potential Confounding

Here:

- Y is the outcome of interest,
- W is the primary predictor / covariate ("treatment") of interest,
- T_i is the "treatment indicator" for observation i,
- We're interested in estimating τ, the "treatment effect" of W on Y,
- $\mathbf{X}$ are observed confounders,
- $\mathbf{U}$ are unobserved confounders.

Things We Can Do

- **Randomize**

  (or...)

- Instrumental Variables Approaches
- Selection on Observables:
  - Regression / Weighting
  - Matching (propensity scores, multivariate/minimum-distance, genetic, etc.)
- Regression Discontinuity Designs ("RDD")
- Differences-In-Differences ("DiD")
- Synthetic Controls
- Others...

Under Randomization

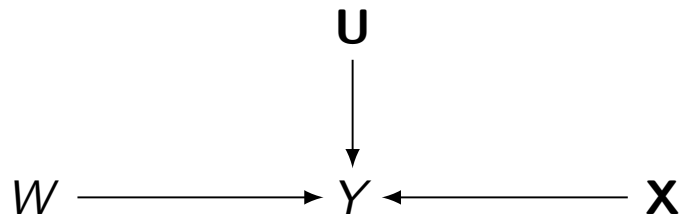


Figure: = no confounding!

Note:

- Randomized assignment of W "balances" covariate values – both observed and unobserved – *on average*...
- That is, under randomization of W:

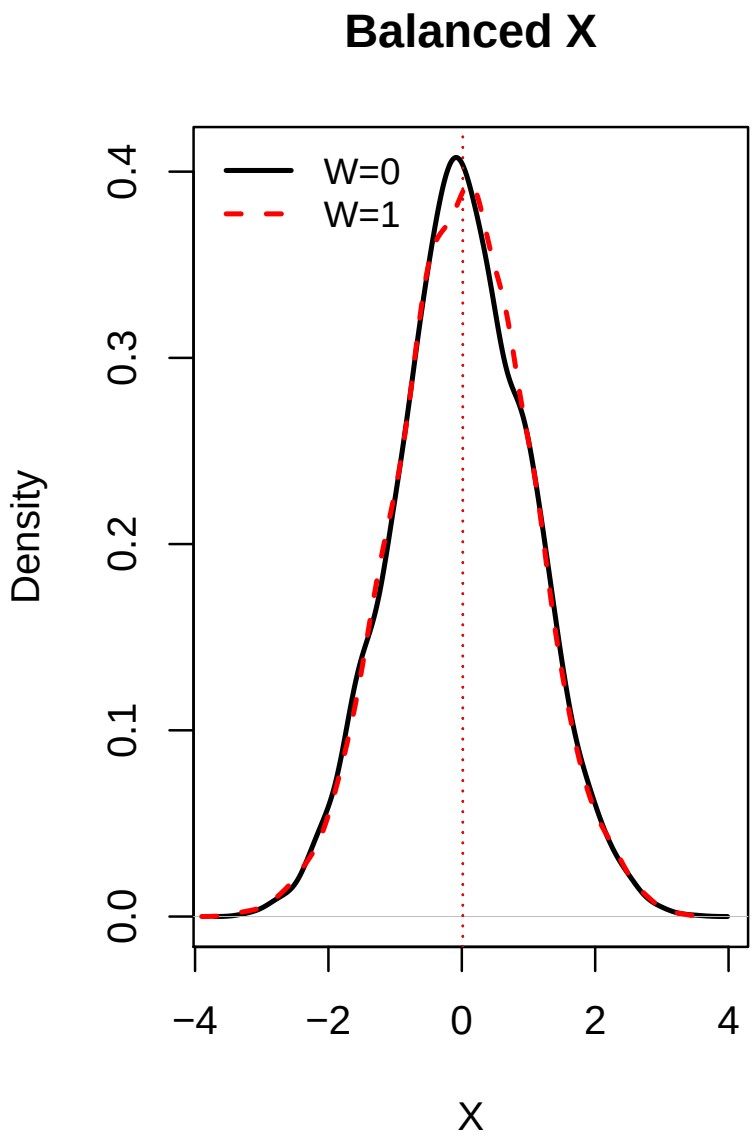
$$\mathrm{E}(\mathbf{X}_i, \mathbf{U}_i \mid W_i = 0) = \mathrm{E}(\mathbf{X}_i, \mathbf{U}_i \mid W_i = 1)$$

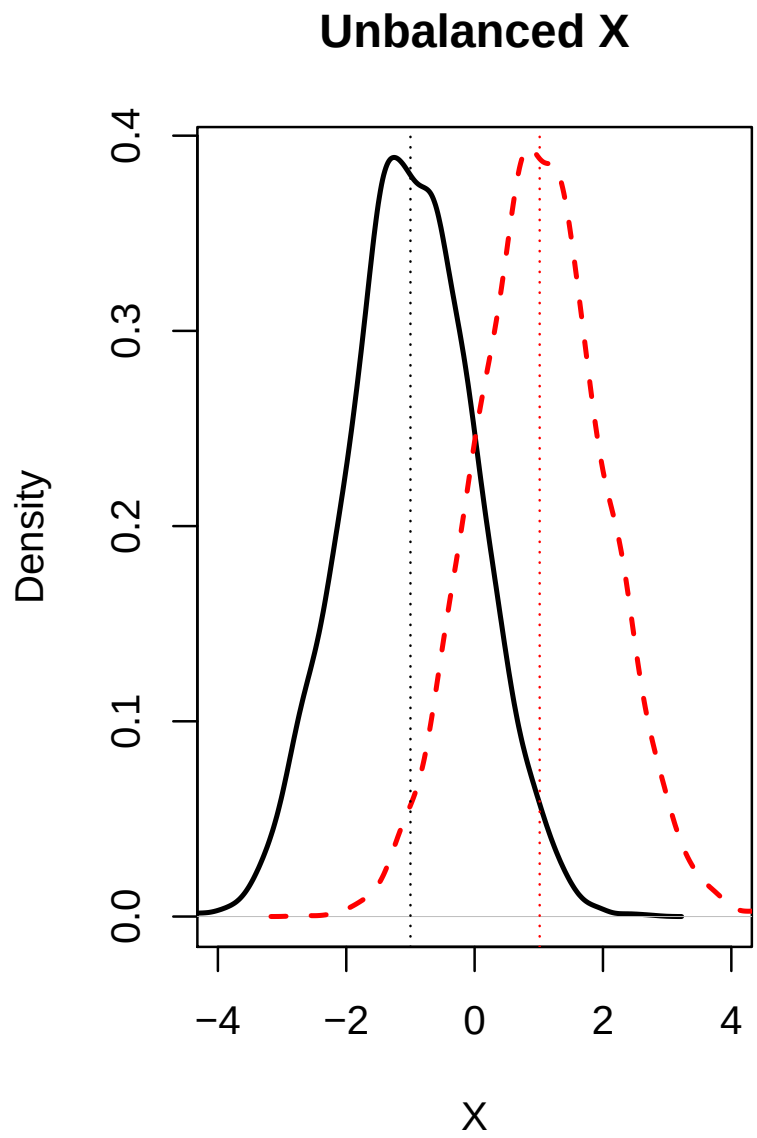
  or, more demandingly,

$$\mathrm{E}[f(\mathbf{X}, \mathbf{U}) \mid W_i = 0] = \mathrm{E}[f(\mathbf{X}, \mathbf{U}) \mid W_i = 1]$$

- Can yield imbalance by random chance...

Covariate Balance / Imbalance





Nonrandom Assignment of W_i

Valid causal inference requires $Y_{0i}, Y_{1i} \perp W_i | \mathbf{X}_i, \mathbf{U}_i$

- That is, treatment assignment W_i is *conditionally ignorable*

"What if I have unmeasured confounders?"

- In general, that's a bad thing.
- One approach: obtain *bounds* on possible values of τ
  - Assume you have one or more unmeasured confounders
  - Undertake one of the methods described below to get $\hat{\tau}$
  - Calculate the range of values for $\hat{\tau}$ that could occur, depending on the degree and direction of confounding bias
  - Or ask: How strong would the effect of the **U**s have to be to make $\hat{\tau} \rightarrow 0$?
- Some useful cites:
  - Rosenbaum and Rubin (1983)
  - Rosenbaum (2002)
  - DiPrete and Gangl (2004)
  - Liu et al. (2013)
  - Ding and VanderWeele (2016)

Digression: Instrumental Variables

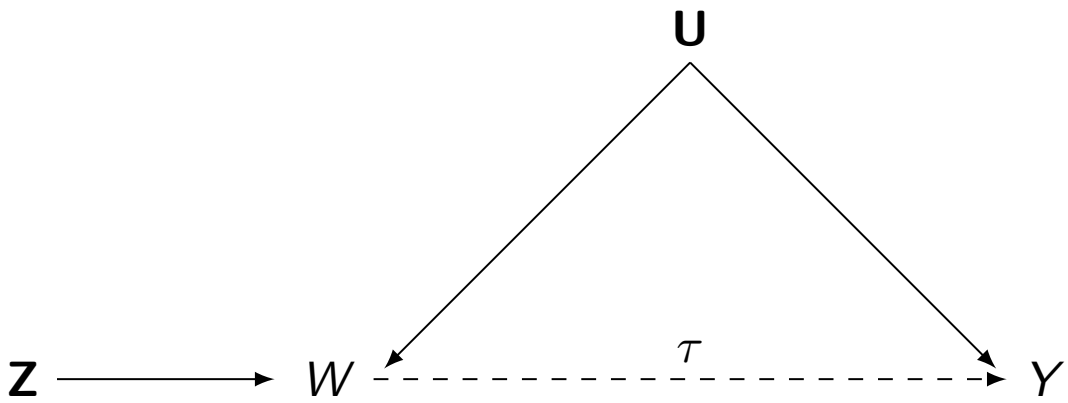


Figure: Instrumental Variables

As in the more general regression case where we have $\text{Cov}(\mathbf{X}, \mathbf{u}) \neq 0$, instrumental variables can be used to address confounding in causal analyses.

Instrumental Variables (continued)

Considerations:

- Requires:
  1. $\text{Cov}(\mathbf{Z}, W) \neq 0$)
  2. $\mathbf{Z}$ has no independent effect on Y, except through W
  3. $\mathbf{Z}$ is exogenous [i.e., $\text{Cov}(\mathbf{Z}, \mathbf{u}) = 0$]
- Arguably most useful when treatment compliance is uncertain / driven by unmeasured factors ("intent to treat" analyses)
- Mostly, they're not that useful at all...
  - Bound et al. (1995): Weak instruments are worse than endogeneity bias
  - Young (2020): Inferences in published IV work (in economics) are wrong and terrible
  - Shalizi (2020, chapters 20-21): Gathers all the issues together, sometimes hilariously
- Other useful references:
  - Imbens et al. (1996) (the overly-cited one)
  - Hernan and Robins (2006) (making sense of things)
  - Lousdal (2018) (a good intuitive introduction)

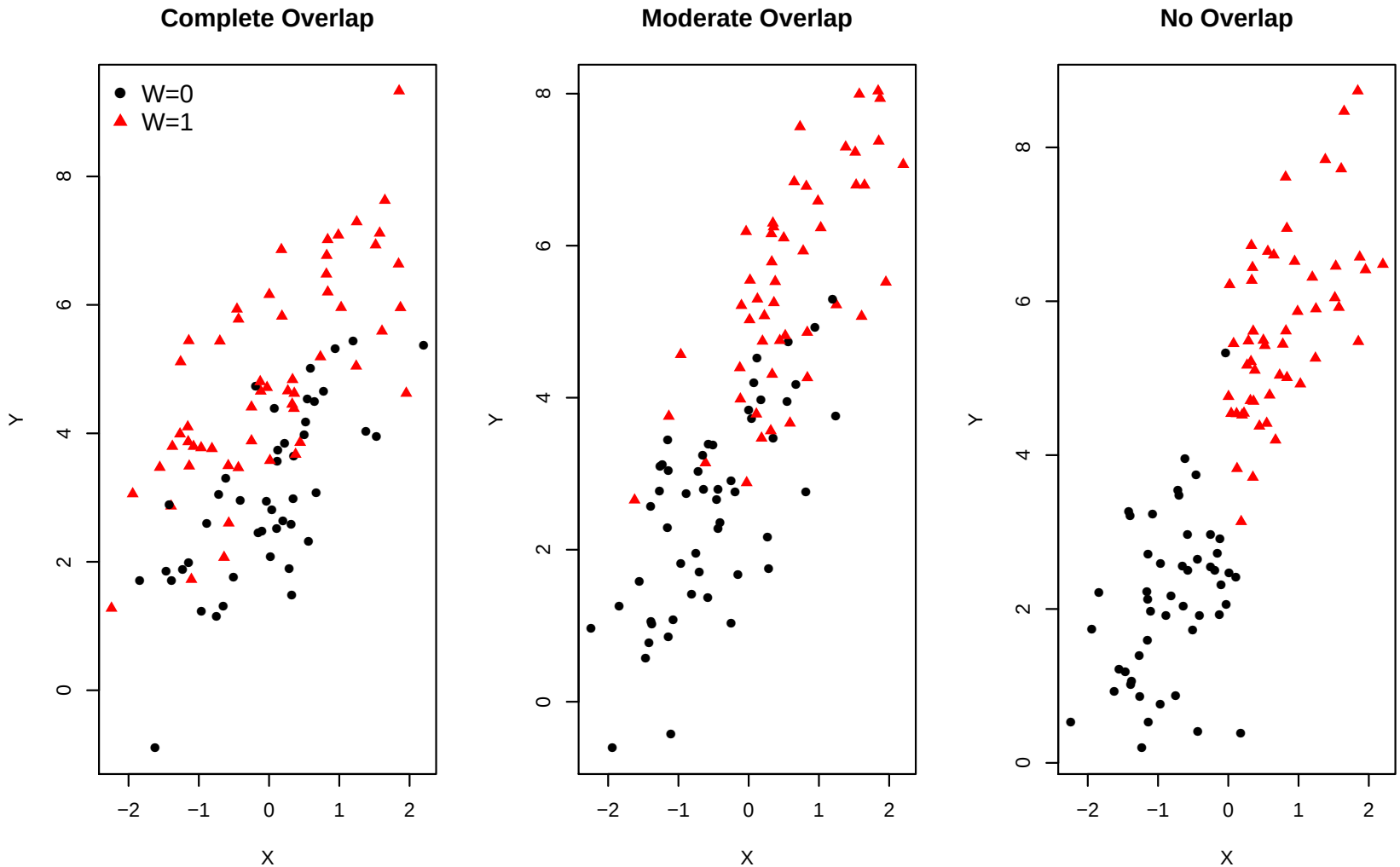
Nonrandom Assignment of W_i (continued)

So...

- Causal inference with observational data typically requires that $\mathbf{U} = \varnothing$...
- This typically requires a strong theoretical motivation in order to assume that the specification conditioning on the observed $\mathbf{X}$ exhausts the list of possible confounders.

- **Even if** this assumption is reasonable, there are two (related) important concerns:
  - Lack of *covariate balance* (as above)
  - Lack of *overlap* among observations with $W_i = 0$ vs. $W_i = 1$
  - The latter is related to *positivity*, the requirement that each observation's probability of receiving (or not receiving) the treatment is greater than zero

Overlap



Overlap and Balance

In general:

- Ensuring overlap allows us to make counterfactual statements from observational data
  - Requires that we have comparable $W_i = 0$ and $W_i = 1$ units
  - It's *necessary* – no overlap means any counterfactual statements are based on assumption
  - Think of this as an aspect of *model identification* (Crump et al. 2009)
  - Most often handled via matching

- Ensuring covariate balance corrects potential bias in $\hat{\tau}$ due to (observed) confounding
  - This can be done a number of different ways: stratification, weighting, regression...
  - Key: Adjusting for (observable) differences across groups defined by values of W

- In general, we usually address overlap first, then balance...

Matching

Matching is a way of dealing with one of both of covariate overlap and (im)balance.

The process, generally:

1. Choose the $\mathbf{X}$ on which the observations will be matched, and the matching procedure;
2. Match the observations with $W_i = 0$ and $W_i = 1$;
3. Check for balance in $\mathbf{X}_i$; and
4. Estimate $\hat{\tau}$ using the matched pairs.

Variants / considerations:

- 1:1 vs. 1:k matching
- "Greedy" vs. "Optimal" matching (see Gu and Rosenbaum 1993)
- Distances, calipers, and "common support"
- Post-matching: Balance checking...

Flavors of Matching

- Simplest: Exact Matching
  - For each of the n observations i with $W = 1$, find a corresponding observation j with $W = 0$ that has identical values of $\mathbf{X}$
  - Calculate $\hat{\tau} = \frac{1}{n} \sum (Y_i - Y_j)$
  - Generally not practical, especially for high-dimensional $\mathbf{X}$
  - Variants: "coarsened" exact matching (e.g., Iacus et al. 2011)

- Multivariate Matching
  - Match each observation i which has $W = 1$ with a corresponding observation j with $W = 0$, and whose values on $\mathbf{X}_j$ are the most similar to $\mathbf{X}_i$
  - One example: Mahalanobis distance matching, based on the distance:

$$d_M(\mathbf{X}_i, \mathbf{X}_j) = \sqrt{(\mathbf{X}_i - \mathbf{X}_j)' \mathbf{S}^{-1} (\mathbf{X}_i - \mathbf{X}_j)}.$$

Flavors of Matching (continued)

- Propensity Score Matching
  - Match observation i which has $W = 1$ with observation j having $W = 0$ based on the closeness of their *propensity score*
  - The propensity score is, $\Pr(W_i = 1|\mathbf{X}_i)$, typically calculated as the predicted value of T_i (the treatment indicator) from a logistic (or other) regression of T on $\mathbf{X}$.
  - The assumptions about matching [that Y is orthogonal to $W|\mathbf{X}$ and that $\Pr(W_i = 1|\mathbf{X}_i) \in (0, 1)$] mean that $Y \perp W|\Pr(T|\mathbf{X})$.
  - In practice: read this...

- Other variants: Genetic matching (Diamond and Sekhon 2013), etc.[1]

[1] Shalizi (2016) notes that "(A)pproximate matching is implicitly doing nonparametric regression by a nearest-neighbor method," and that "(M)aybe it is easier to get doctors and economists to swallow "matching" than "nonparametric nearest neighbor regression"; this is not much of a reason to present the subject as though nonparametric smoothing did not exist, or had nothing to teach us about causal inference."

Matching Software

Interestingly, quite a few of the good matching programs written for R have been written by political scientists...

- the `Match` package (does propensity score, M-distance, and genetic matching, plus balance checking and other diagnostics)
- the `MatchIt` package (for pre-analysis matching; also has nice options for checking balance)
- the `optmatch` package (suite for 1:1 and 1:k matching via propensity scores, M-distance, and optimum balancing)
- `matching` (in the `arm` package)

Regression Discontinuity Designs

"RDD":

- Treatment changes abruptly [usually at some threshold(s)] according to the value(s) of some measured, continuous, pre-treatment variable(s)
  - This is known as the "assignment" or "forcing variable(s)," sometimes denoted **A**
  - Formally:

$$W_i = \begin{cases} 0 \text{ if } A_i \leq c \\ 1 \text{ if } A_i > c \end{cases}$$

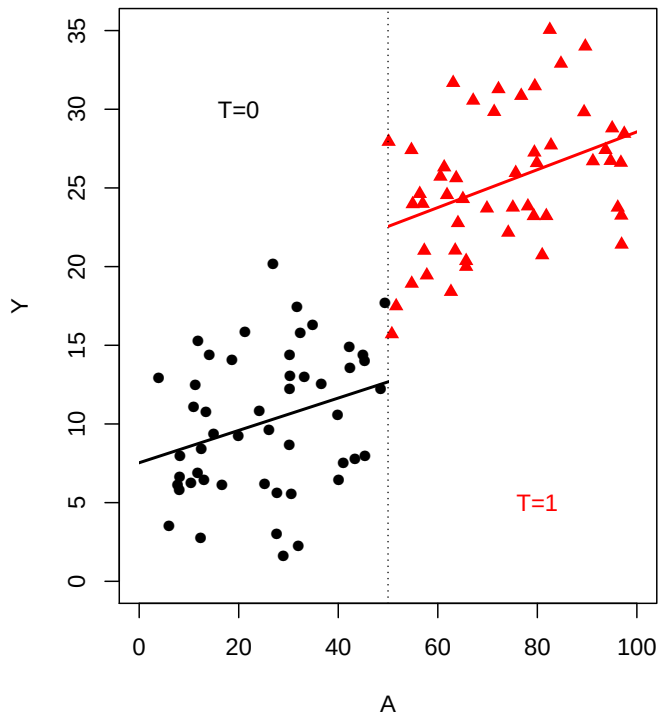
- Intuition: Observations near but on either side of the threshold(s) are highly comparable, and can be used to (locally) identify τ
- This is because variation in W_i near the threshold is effectively random (a "local randomized experiment")
- E.g. Carpenter and Dobkin (2011) (on the relationship between the legal drinking age and public health outcomes like accidental deaths)

RDD (continued)

- Pluses:
  - Can be estimated straightforwardly, as:

$$Y_i = \beta_0 + \beta_1 A_i + \tau W_i + \gamma A_i W_i + \epsilon_i$$

  - Generally requires fewer assumptions than IV or DiD (and those assumptions are easier to observe and test)
- Minuses:
  - Provides only an estimate of a local treatment effect
  - Fails if (say) subjects can manipulate A in the vicinity of c
- Lee and Lemieux (2010) is an excellent (if fanboi-ish) review
- R packages: `rddtools`, `rdd`, `rdrobust`, `rdpower`, `rdmulti`



Panel Data Approaches: Differences-In-Differences

"DiD":

- Leverages two-group, two-period data ($T = 2$):

	Pre-Treatment ($T = 0$)	Post-Treatment ($T = 1$)
Treated ($W = 1$)	A	B
Unreated ($W = 0$)	C	D

- Process (simple version):
  - Calculate the pre- vs. post-treatment difference for the treated group ($B - A$)
  - Calculate the pre- vs. post-treatment difference for the untreated group ($D - C$)
  - Calculate the differences between the differences [$DiD = (B - A) - (D - C)$]
  - This is the same as fitting the regression:

$$Y_{it} = \beta_0 + \beta_1 W_{it} + \beta_2 T_{it} + \beta_3 W_{it} T_{it} + u_{it}$$

- Validity depends on (a) all the usual assumptions required by OLS, plus (b) the equal trends assumption – that there are no time-varying differences between the two groups as we go from $T = 0$ to $T = 1$.
- Resources:
  - Our old friend Wikipedia
  - Pischke's slides on DiD
  - R: package `did`
  - Stata: `ieddtab` in the `ietoolkit`

Panel Data Approaches: Synthetic Controls

The "synthetic control method" (SCM):

- Addresses situations in which we have a single treated case (or small number of them)...
- Requires at least one (and ideally more) repeated measurements over time on the outcome of interest, and
- Also requires multiple (but not *too* many) non-treated cases
- Assumptions:
  - Possible control units are similar
  - Lack of spillover between treated and potential control units
  - Lack of exogenous shocks to potential control units
- Intuition:
  - Create a counterfactual "control" unit that is as similar to the (pre-treatment) treated case as possible
  - Do so by weighting the observed predictors across "control" cases to minimize the difference (in a MSE sense)
  - Also: compare the pre-treatment trend in the synthetic control to that in the treated case
  - The weights are then used to create a post-treatment trend for the synthetic control
  - Inference is via placebo methods (varying the timing of the intervention)
- Advantages:
  - Works with (very) small N
  - Doesn't require parallel trends (a la DiD)
  - Abadie et al. claim that SCM controls for both observed and unobserved time-varying confounders
- A few references:
  - A nontechnical introduction in the *BMJ*
  - Method of the Month Blog
  - The Development Impact blog post on SCM

Software Matters

- R
  - Packages for matching are listed above (`Matching`, `MatchIt`, etc.)
  - Similarly for RDD (`rddtools`, `rdd`, etc.) and DiD (`did`)
  - IV regression: `ivreg` (in `AER`), `tsls` (in `sem`), others
  - Synthetic controls are in `Synth` and `MicroSynth`
  - See generally the *Econometrics* and *SocialSciences* CRAN Task Views
- Stata also has a large suite of routines for attempting causal inference with observational data...
- And there's a pretty good NumPy/SciPy-dependent package for Python, called (creatively) Causalinference

Causal Inference: One-Way (FE) Models

Imai and Kim (2019):

- The punch line first: "(t)he ability of unit fixed effects regression models to adjust for unobserved time-invariant confounders comes at the expense of dynamic causal relationships between treatment and outcome variables."
- Also dependent on functional form assumptions (specifically, linearity)

Intuition: For the model:

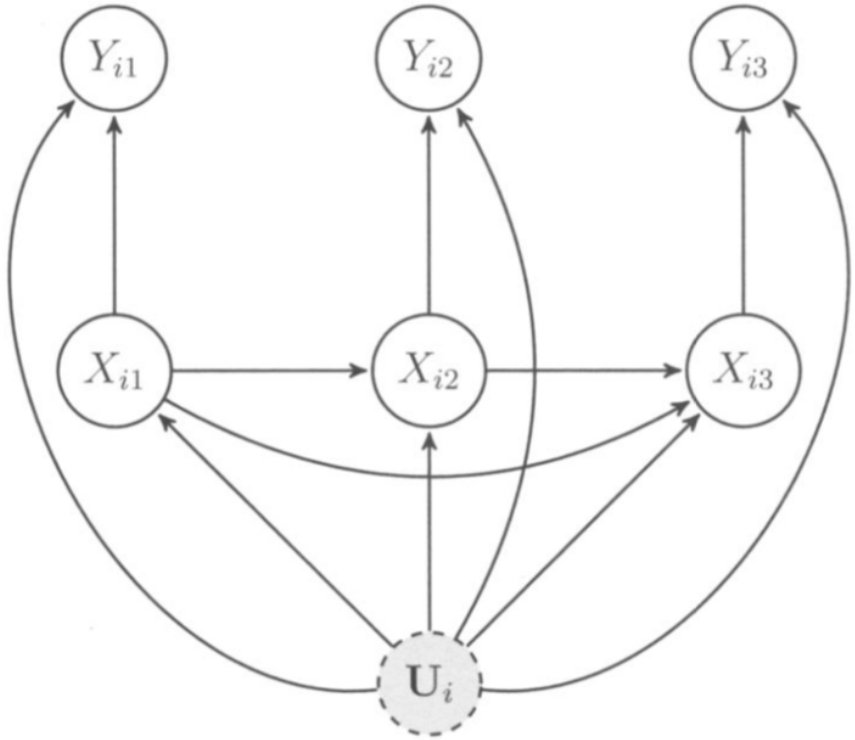
$$Y_{it} = \mathbf{X}_{it}\boldsymbol{\beta} + \alpha_i + u_{it}$$

where (for simplicity) X is a binary treatment for which we want to know a causal effect on Y:

- Identification is via $\text{Cov}[(\mathbf{X}_{it}, \alpha_i), u_{it}] = 0$
- In this framework, $\boldsymbol{\beta} = \tau$, the typical causal estimand (that is, the expected difference between $Y_{it}(0)$ and $Y_{it}(1)$)

A more flexible approach is to think of a FE model as a DAG...

Fixed-Effects DAG



Source: Imai and Kim (2019).

Key FE Takeaways

Summarizing Imai and Kim (2019):

- Three key identifying assumptions for FE models:
  - No unobserved time-varying confounders
  - Past treatments / values of $\mathbf{X}$ do not affect current values of Y[2]
  - Past outcomes Y do not affect current values of $\mathbf{X}$.
- Alternatively, one can select on observables (a la Blackwell and Glynn 2018) and model dynamics (albeit at the cost of failing to control for unobserved time-constant confounders).

"...researchers must choose either to adjust for unobserved time-invariant confounders through unit fixed effects models or to model dynamic causal relationships between treatment and outcome under a selection-on-observables approach. No existing method can achieve both objectives without additional assumptions" (Imai and Kim 2019, 484).

[2] Can be relaxed via IV, but that requires independence of past and present values of Y.

Two-Way Models

Imai and Kim redux (2020):

- In the simple $T = 2$ case, DiD is equivalent to a two-way FE model:

$$Y_{it} = \mathbf{X}_{it}\beta + \alpha_i + \eta_t + u_{it}$$

- I & K: The same is not true for $T > 2$...
- More important: two-way FEs' ability to control for unmeasured confounders depends on the (linearity of the) functional form...
- Upshot: two-way FEs aren't a (nonparametric) cure-all...
- Related: When we control for both α_i and η_t, what – exactly – is the counterfactual?

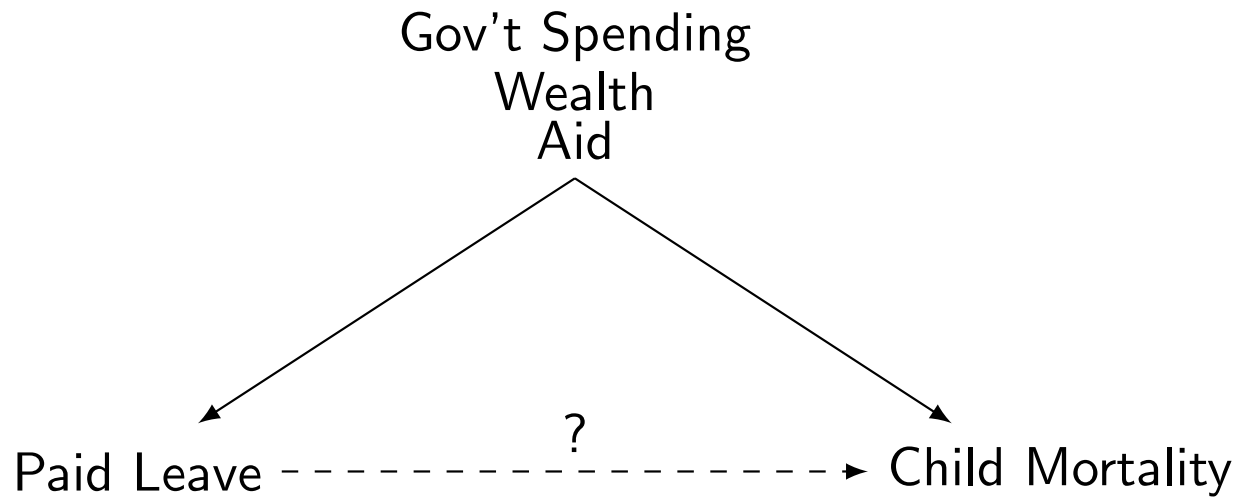
Back To The WDI

```
> describe(WDI,fast=TRUE,ranges=FALSE,check=TRUE)
                          vars     n             mean        sd             se
ISO3                         1 13330              NaN        NA             NA
Year                         2 13330              NaN        NA             NA
Region                       3 13330              NaN        NA             NA
country                      4 13330              NaN        NA             NA
RuralPopulation              5 13045            48.61 2.574e+01           0.23
UrbanPopulation              6 13045            51.39 2.574e+01           0.23
BirthRatePer1K               7 12112            28.32 1.310e+01           0.12
FertilityRate                8 11847             3.97 2.010e+00           0.02
PrimarySchoolAge             9 10696             6.14 6.200e-01           0.01
LifeExpectancy              10 11829            64.37 1.146e+01           0.11
AgeDepRatioOld              11 11731            10.34 6.360e+00           0.06
ChildMortality              12 10761            75.75 7.773e+01           0.75
GDP                         13  9585 242308268086.15 1.102e+12 11252014966.83
GDPPerCapita                14  9582        11685.74 1.868e+04         190.78
GDPPerCapGrowth             15  9598             1.89 6.210e+00           0.06
TotalTrade                  16  8363            78.18 5.414e+01           0.59
FDIIn                       17  8195             5.57 4.542e+01           0.50
NetAidReceived              18  8633    453209476.19 8.678e+08     9339331.98
MobileCellSubscriptions     19  9849            33.70 5.029e+01           0.51
NaturalResourceRents        20  8745             6.61 1.087e+01           0.12
GovtExpenditures            21  8012            16.20 8.190e+00           0.09
PaidParentalLeave           22  9776             0.10 3.000e-01           0.00
ColdWar                     23 13330             0.48 5.000e-01           0.00
YearNumeric                 24 13330          1990.50 1.790e+01           0.16
```

A New Question

Do paid parental leave policies decrease child mortality?

- Y = `ChildMortality` (N of deaths of children under 5 per 1000 live births) **(logged)**
- T = `PaidParentalLeave` (1 if provided, 0 if not)
- **X**s:
  - `GDPPerCapita` (Wealth; in constant $US) (logged)
  - `NetAidReceived` (Net official development aid received; in constant $US) (logged)
  - `GovtExpenditures` (Government Expenditures, as a percent of GDP)



Preliminary Regressions

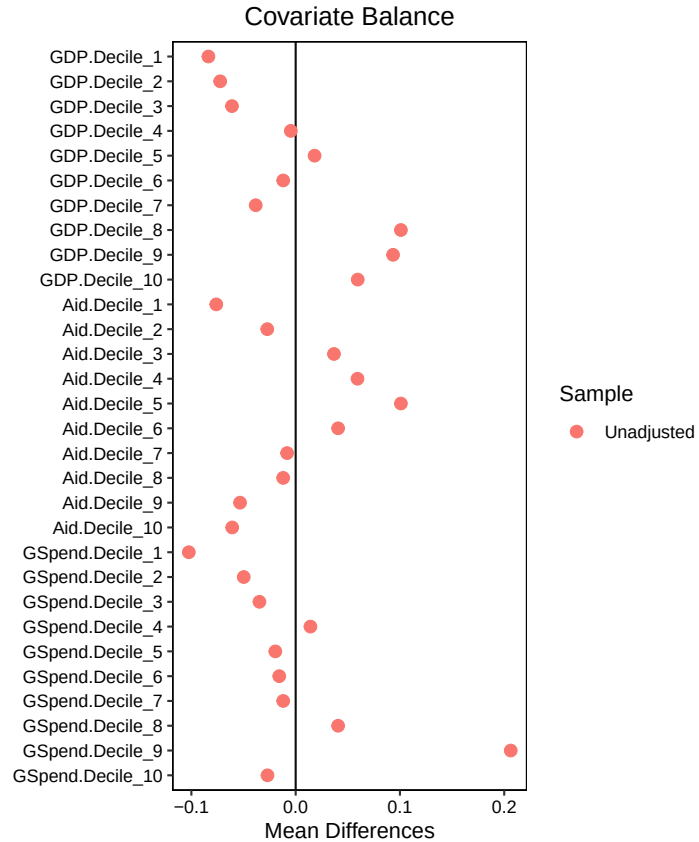
Table: Models of log(Child Mortality)

	BIV	OLS	FE.1way	FE.2way	FE.LDV
Paid Parental Leave	−1.812	−0.909***	−0.080*	−0.128***	−0.205***
	(0.036)	(0.039)	(0.043)	(0.025)	(0.026)
ln(GDP Per Capita)		−0.676***	−1.086***	−0.291***	−0.549***
		(0.010)	(0.017)	(0.013)	(0.012)
ln(Net Aid Received)		−0.073***	−0.088***	0.008**	0.003
		(0.007)	(0.007)	(0.004)	(0.004)
Government Expenditures		−0.002*	0.002**	0.002***	0.002**
		(0.001)	(0.001)	(0.001)	(0.001)
Lagged Child Mortality					0.008***
					(0.0001)
Constant	3.793*	10.670***			
	(0.012)	(0.182)			
Observations	9,167	4,946	4,946	4,946	4,942
R^2	0.215	0.585	0.477	0.113	0.805
Adjusted R^2	0.215	0.585	0.461	0.076	0.799

*p<0.1; **p<0.05; ***p<0.01

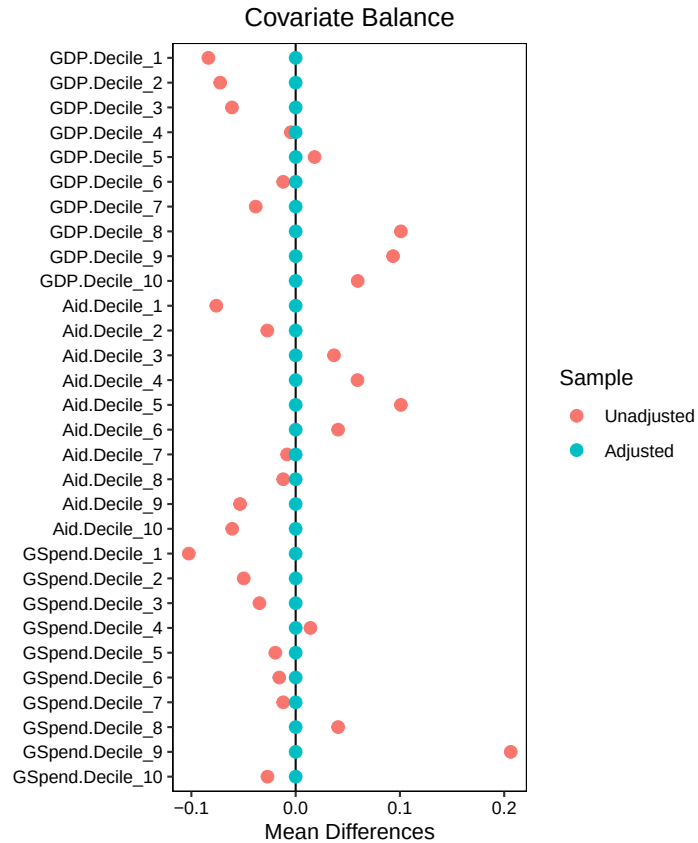
Checking Covariate Balance (Pre-Matching)

```
> # Subset data a little bit:

> vars<-c("ISO3","Year","Region","country","UrbanPopulation",
          "FertilityRate","PrimarySchoolAge","ChildMortality",
          "GDPPerCapita","NetAidReceived","NaturalResourceRents",
          "GovtExpenditures","PaidParentalLeave","ColdWar",
          "lnCM")
> wdi<-WDI[vars]
> wdi<-na.omit(wdi)


> # Create discrete-valued variables (i.e., coarsen) for
> # matching on continuous predictors:

> wdi$GDP.Decile<-as.factor(ntile(wdi$GDPPerCapita,10))
> wdi$Aid.Decile<-as.factor(ntile(wdi$NetAidReceived,10))
> wdi$GSpend.Decile<-as.factor(ntile(wdi$GovtExpenditures,10))


> # Pre-match balance statistics...
>
> BeforeBal<-bal.tab(PaidParentalLeave~GDP.Decile+
                     Aid.Decile+GSpend.Decile,data=wdi,
                     stats=c("mean.diffs","ks.statistics"))
```



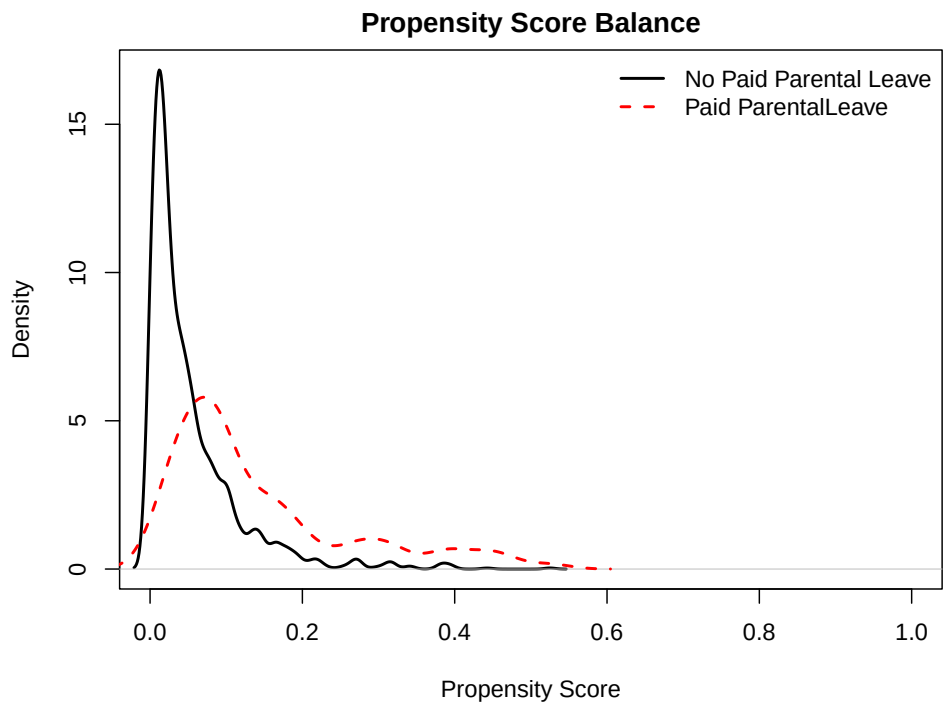
Exact Matching

```
> M.exact <- matchit(PaidParentalLeave~GDP.Decile+Aid.Decile+
+                    GSpend.Decile,data=wdi,method="exact")
> summary(M.exact)

Call:
matchit(formula = PaidParentalLeave ~ GDP.Decile + Aid.Decile +
    GSpend.Decile, data = wdi, method = "exact")

Summary of Balance for All Data:
.
.
.
Sample Sizes:
              Control Treated
All            4622.      282
Matched (ESS)   322.2     268
Matched         831.      268
Unmatched      3791.       14
Discarded         0.        0


> # Create matched data:
>
> wdi.exact <- match.data(M.exact,group="all")
> dim(wdi.exact)
[1] 1099   20
```



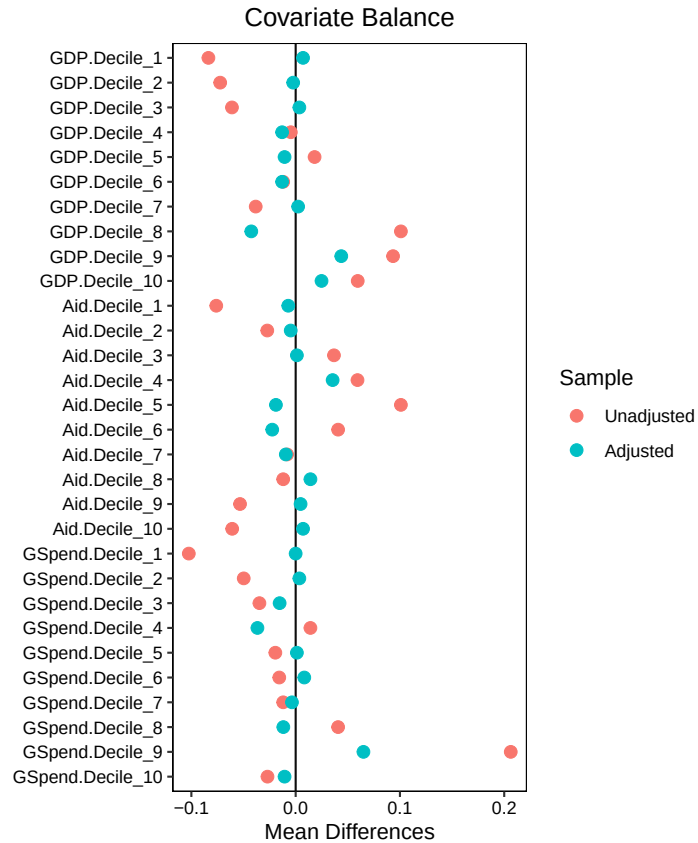
Propensity Scores

```
> # Model for propensity scores:
>
> PS.fit<-glm(PaidParentalLeave~GDP.Decile+Aid.Decile+
                GSpend.Decile,data=wdi,
                family=binomial(link="logit"))

> # Generate scores & check common support:
>
> PS.df<-data.frame(PS = predict(PS.fit,type="response"),
                    PaidParentalLeave=PS.fit$model$PaidParentalLeave)
```



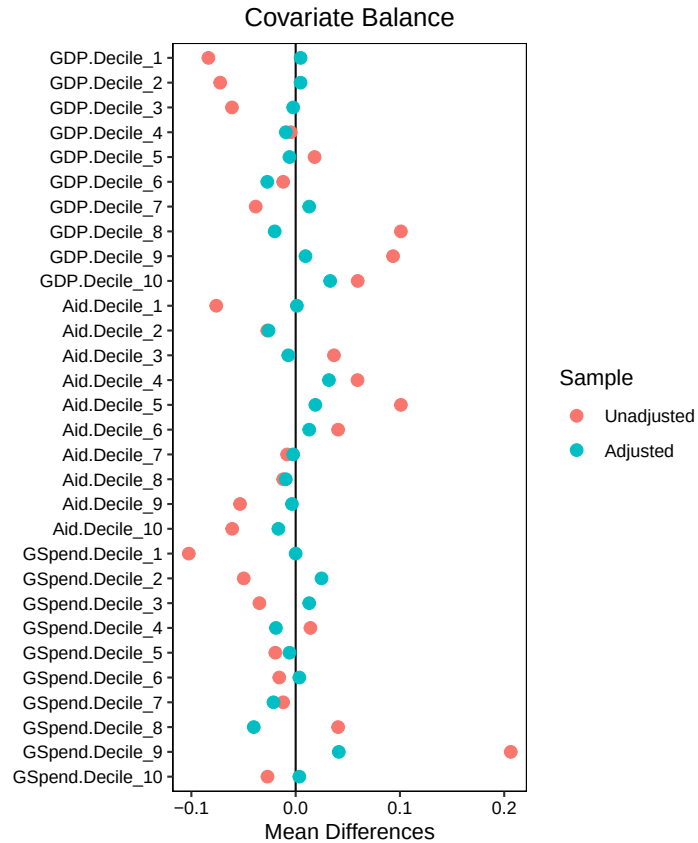
Propensity Score Matching

```
> M.prop <- matchit(PaidParentalLeave~GDP.Decile+Aid.Decile+
+                   GSpend.Decile,data=wdi,method="nearest",
+                   ratio=3)
> summary(M.prop)

Call:
matchit(formula = PaidParentalLeave ~ GDP.Decile + Aid.Decile +
    GSpend.Decile, data = wdi, method = "nearest", ratio = 3)

Summary of Balance for All Data:
.
.
.

Sample Sizes:
          Control Treated
All          4622     282
Matched       846     282
Unmatched    3776       0
Discarded       0       0

> # Matched data:
>
> wdi.ps <- match.data(M.prop,group="all")
> dim(wdi.ps)
[1] 1128   21
```



"Optimal" Matching

```
> M.opt <- matchit(PaidParentalLeave~GDP.Decile+Aid.Decile+
+                    GSpend.Decile,data=wdi,method="optimal",
+                  ratio=3)
> summary(M.opt)

Call:
matchit(formula = PaidParentalLeave ~ GDP.Decile + Aid.Decile +
    GSpend.Decile, data = wdi, method = "optimal", ratio = 3)
.
.
.

Sample Sizes:
          Control Treated
All          4622     282
Matched       846     282
Unmatched    3776       0
Discarded       0       0


> # Matched data:
>
> wdi.opt <- match.data(M.opt,group="all")
> dim(wdi.opt)
[1] 1128   21
```



Post-Matching Regressions

Table: Models of log(Child Mortality)

	PreMatch.FE	Exact.FE	PS.FE	Optimal.FE
Paid Parental Leave	-0.083^{*}	-0.163^{***}	-0.117^{**}	-0.164^{***}
	(0.044)	(0.053)	(0.053)	(0.051)
ln(GDP Per Capita)	-1.086^{***}	-1.004^{***}	-1.084^{***}	-1.263^{***}
	(0.017)	(0.035)	(0.035)	(0.033)
ln(Net Aid Received)	-0.087^{***}	-0.044^{**}	-0.079^{***}	-0.037^{**}
	(0.007)	(0.017)	(0.018)	(0.015)
Government Expenditures	0.002^{**}	0.004^{*}	0.004	0.005^{**}
	(0.001)	(0.003)	(0.003)	(0.002)
Observations	4,820	1,077	1,115	1,119
R^2	0.476	0.505	0.537	0.613
Adjusted R^2	0.459	0.443	0.482	0.571

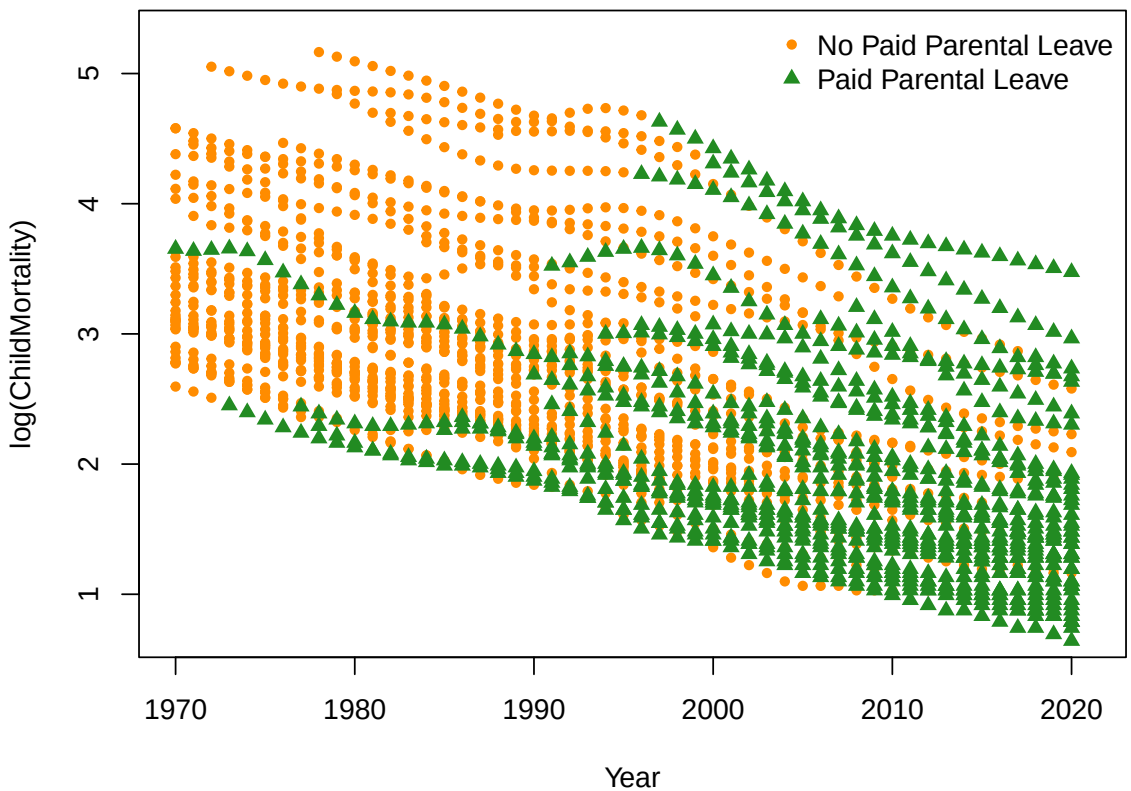
*p<0.1; **p<0.05; ***p<0.01

Another Approach: DiD

Intuition: Compare the child mortality "trajectories" of countries before and after they implement paid parental leave policies.

The model is:

$$\begin{aligned} \text{Child Mortality}_{it} &= \beta_0 + \beta_1(\text{Paid Parental Leave}_{it}) + \beta_2(\text{Time}_t) + \\ &= \beta_3(\text{Paid Parental Leave}_{it} \times \text{Time}_t) + (\text{confounders}) + u_{it} \end{aligned}$$



DiD Regressions

Table: DiD Models of log(Child Mortality)

	OLS		One-Way FE		Two-Way FE	
Paid Parental Leave	−22.780***	4.562	−10.720***	−6.400	−2.942	−3.461
	(3.937)	(8.099)	(2.028)	(6.073)	(2.572)	(9.244)
Time (1950=0)	−0.713***	−0.491***	−0.672***	−0.754***		
	(0.047)	(0.086)	(0.023)	(0.102)		
Paid Parental Leave x Time	0.421***	−0.140	0.179***	0.078	0.007	0.0003
	(0.077)	(0.153)	(0.038)	(0.111)	(0.050)	(0.174)
ln(GDP Per Capita)		−19.530***		−13.630***		−11.660***
		(0.697)		(2.256)		(2.648)
ln(Net Aid Received)		−2.990***		−0.480		−1.570***
		(0.452)		(0.440)		(0.494)
Government Expenditures		−0.754***		0.564***		0.491***
		(0.129)		(0.140)		(0.146)
Constant	49.800***	284.500***				
	(1.893)	(11.770)				
Observations	2,073	490	2,073	490	2,073	490
R^2	0.176	0.659	0.525	0.685	0.008	0.124
Adjusted R^2	0.175	0.655	0.514	0.665	-0.039	-0.042

*p<0.1; **p<0.05; ***p<0.01

Resources

- Good references:
  - Freedman (2012)*
  - Shalizi (someday)*
  - Morgan and Winship (2014)
  - Pearl et al. (2016)
  - Peters et al. (2017)
- Courses / syllabi (a sampling):
  - Eggers (2019)
  - Frey (2019)
  - Imai (2019)
  - Sekhon (2015)
  - Simpson (2019)
  - Xu (2018)
  - Yamamoto (2018)
- Other useful things:
  - The Causal Inference Book
  - Some useful notes

* I really like this one.