Matching EC 607, Set 8

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Prologue

Schedule

Last times

- DAGs
- The conditional independence assumption: $(\mathrm{Y}_{0i},\,\mathrm{Y}_{1i})\perp\!\!\!\!\perp\mathrm{D}_i|\mathrm{X}_i$
- Omitted variable bias
- Good vs. bad controls

Today

- First problem set!
- Matching estimators (MHE 3.2 and Cameron and Trivedi 25.4).

The gist

Remember the **conditional independence assumption**⁺ in a setting—*i.e.*, treatment is as-good-as random conditional on a known set of covariates?

Matching estimators take us at our word.

If we really believe $(\mathbf{Y}_{1i}, \mathbf{Y}_{0i}) \perp \mathbf{D}_i | \mathbf{X}_i$, then we can just calculate a bunch of treatment effects conditional on \mathbf{X}_i , *i.e.*,

$$au(x) = E[\mathrm{Y}_{1i} - \mathrm{Y}_{0i} \mid \mathrm{X}_i = x]$$

The idea: Estimate a treatment effect only using observations with (nearly?) identical values of X_i . The CIA buys us causality within these groups.

Goals

Let's return to **the fundamental problem of causal inference** for a moment.

- 1. We want/need to know $au_i = \mathrm{Y}_{1i} \mathrm{Y}_{0i}$.
- 2. We cannot simultaneously observe both Y_{1i} and Y_{0i} .

Most empirical strategies boil to strategies to estimate Y_{0i} for treated individuals—the unobservable counterfactual for the treatment group.

Matching is no different.

We match untreated observations to treated observations using X_i , *i.e.*, calculate a $\widehat{Y_{0i}}$ for each Y_{1i} , based upon "matched" untreated individuals.

More formally

We want to construct a counterfactual for each individual with $D_i = 1$.

The counterfactual for i should only use individuals that match X_i .

Let there be N_T treated individuals and N_C control individuals. We want

- N_T sets of weights
- with N_C weights in each set: $w_i(j)$ $(i=1,\,\ldots,\,N_T;\,j=1,\,\ldots,\,N_C)$

Assume $\sum_{j} w_{i}(j) = 1$. Our estimate for the counterfactual of treated *i* is

$$\widehat{\mathrm{Y}_{0i}} = \sum_{j \in (D=0)} w_i(j) \mathrm{Y}_j$$
 .

More formally

If our estimated counterfactual for treated individual i is

$$\widehat{\mathrm{Y}_{0i}} = \sum_j w_i(j) \mathrm{Y}_j$$

then our estimated treatment effect (for individual i) is

$${\hat au}_i = \mathrm{Y}_{1i} - \widehat{\mathrm{Y}_{0i}} = \mathrm{Y}_{1i} - \sum_j w_i(j) \mathrm{Y}_j$$

 \therefore a generic matching estimator for the treatment effect on the treated is

$$\hat{{ au}}_M = rac{1}{N_T}\sum_{i\in(\mathrm{D}=1)}\left(\mathrm{Y}_{1i}-\widehat{\mathrm{Y}_{0i}}
ight) = rac{1}{N_T}\sum_{i\in(\mathrm{D}=1)}\left(\mathrm{Y}_{1i}-\sum_{j\in(D=0)}w_i(j)\mathrm{Y}_j
ight)$$

Weight for it[†]

So all we need is those weights and we're done.^{††}

Q Where does one find these handy weights?

A You've got options, but you need to choose carefully/responsibly.

E.g., if $w_i(j) = \frac{1}{N_C}$ for all (i, j), then we're back to a difference in means. This weighting doesn't abide by our conditional independence assumption.

The plan Choose weights $w_i(j)$ that indicate **how close** X_j is to X_i .

Proximity

Our weights $w_i(j)$ should be a measure of **how close** X_j is to X_i .

If X is **discrete**, then we can consider equality, *i.e.*, $w_i(j) = \mathbb{I}(X_i = X_j)$, scaling as necessary to get $\sum_j w_i(j) = 1$.

Proximity

Our weights $w_i(j)$ should be a measure of **how close** X_j is to X_i .

If X is **continuous**, then we need *proximity* rather than *equality*.

Nearest-neighbor matching chooses the single closest control observation using the Euclidean distance between X_i and X_j , *i.e.*,

$$\mathrm{d}_{i,j} = \left(\mathrm{X}_i - \mathrm{X}_j
ight)' \left(\mathrm{X}_i - \mathrm{X}_j
ight)$$

• $\hat{ au}_i = Y_{1i} - Y^i_{0j}$, where Y^i_{0j} is *i*'s nearest neighbor in the control group.

- Estimator: $\hat{\tau}_M = \frac{1}{N_T} \sum_i \hat{\tau}_i$
- Produces causal estimates if CIA is valid *and* we have sufficient overlap.
- Suffers from arbitrary choices of units.

Proximity

Our weights $w_i(j)$ should be a measure of **how close** X_j is to X_i .

If X is **continuous**, then we need *proximity* rather than *equality*.

Nearest-neighbor matching with Mahalanobis distance chooses the single closest control using Mahalanobis distance between X_i and X_j , *i.e.*,

$$\mathrm{d}_{i,j} = \left(\mathrm{X}_i - \mathrm{X}_j
ight)' \Sigma_X^{-1} \left(\mathrm{X}_i - \mathrm{X}_j
ight)$$

where Σ_X^{-1} is the covariance matrix of **X**.

- Estimator: $\hat{ au}_M = rac{1}{N_T}\sum_i \hat{ au}_i$ where $\left(\hat{ au}_i = \mathrm{Y}_{1i} \mathrm{Y}^i_{0j}\right)$
- Produces causal estimates if CIA is valid *and* we have sufficient overlap.
- Does not suffer from arbitrary choices of units.

More neighbors?

Why limit ourselves to a **single** "best" match?

If we're going to let a function/algorithm choose the *nearest* match, can't we also let the function/algorithm choose *how many* matches?

Furthermore, if $N_C \gg N_T$, it we're throwing away *a lot* of information.

We could instead use this information and be more efficient.

More neighbors!

Kernel matching gives positive weight to all control observations within some **bandwidth** h, with higher weight for closer matches determined by some **kernel function** $K(\cdot)$,

$$w_i(j) = rac{K\!\!\left(rac{\mathrm{X}_j - \mathrm{X}_i}{h}
ight)}{\sum_{j \in (D=0)} K\!\!\left(rac{\mathrm{X}_j - \mathrm{X}_i}{h}
ight)}$$

Example The Epanechnikov kernel is defined as

$$K(z)=rac{3}{4}ig(1-z^2ig) imes \mathbb{I}(|z|<1)$$

The Epanechnikov kernel $K(z) = rac{3}{4} ig(1-z^2ig) imes \mathbb{I}(|z|<1)$







The Epanechnikov kernel $K(z)=rac{3}{4}ig(1-z^2ig) imes \mathbb{I}(|z|<1)$



The Triangle kernel $K(z) = (1 - |z|) imes \mathbb{I}(|z| < 1)$



The Uniform kernel $K(z) = rac{1}{2} imes \mathbb{I}(|z| < 1)$



The Gaussian kernel $K(z) = \left(2\pi
ight)^{-1/2} \expigl(-z^2/2igr)$



Kernels

Aside

Kernel functions are good for more than just matching.

You will most commonly see/use them smoothing out densities—providing a smooth, moving-window average.

E.g., R's (ggplot2's) smooth, density-plotting function geom_density().

geom_density() defaults to kernel = "gaussian", but you can specify many
other kernel functions (including "epanechnikov").

You can also change the bandwidth argument. The default is a bandwidthchoosing function called bw.nrd0().

Adding neighbors

As we add more neighbors—either moving from 1 to n > 1 or increasing our bandwidth—we potentially increase the efficiency of our estimator.

We need to **be careful not to add** *too many* **controls** for each treated *i*.

CIA requires that we're actually conditioning on the observables—it does not allow us to take a simple average across all control observations.

The curse of dimensionality[†]

It turns out kernel- and bandwidth-selection are not our biggest enemies.

As the dimension of **X** expands (matching on more variables), it becomes **harder and harder to find a nice, close control** for each treated unit.

We need a way to shrink the dimensionality of **X**.

Setup

Let's begin with two assumptions—one old and one new.

- 1. Conditional independence: $(Y_{0i}, Y_{1i}) \perp D_i | X_i$
- 2. **Overlap:** $0 < \Pr(D_i = 1 \mid X_i) < 1$

We can estimate an average treatment effect by conditioning on X_i .

However, overlap may fail if the dimensions of X are large and N is finite.

Propensity scores provide a solution to this mess.

The magic

It turns out that if $(\mathbf{Y}_{0i}, \mathbf{Y}_{1i}) \perp \mathbf{D}_i | \mathbf{X}_i$, then we actually only need to match/condition on $p(\mathbf{X}_i) = E[\mathbf{D}_i | \mathbf{X}_i]$.

 $p(X_i)$ is the **propensity score**, the probability of treatment given X_i .

Propensity-score theorem If $(Y_{0i}, Y_{1i}) \perp D_i | X_i$, then $(Y_{0i}, Y_{1i}) \perp D_i | p(X_i)$.

This theorem extends our CIA to a one-dimensional score, avoiding the curse of dimensionality.

Theorem If $(Y_{0i}, Y_{1i}) \perp D_i | X_i$, then $(Y_{0i}, Y_{1i}) \perp D_i | p(X_i)$.

Proof

$$egin{aligned} &\operatorname{Pr}\!\left[\operatorname{D}_{i}=1\Big|\operatorname{Y}_{0i},\,\operatorname{Y}_{1i},\,p(\operatorname{X}_{i})
ight] \ &=E\!\left[\operatorname{D}_{i}\Big|\operatorname{Y}_{0i},\,\operatorname{Y}_{1i},\,p(\operatorname{X}_{i})
ight] \ &=E\!\left[E\!\left(\operatorname{D}_{i}\Big|\operatorname{Y}_{0i},\,\operatorname{Y}_{1i},\,p(\operatorname{X}_{i}),\,\operatorname{X}_{i}
ight)\Big|\operatorname{Y}_{0i},\,\operatorname{Y}_{1i},\,p(\operatorname{X}_{i})
ight] \ &=E\!\left[E\!\left(\operatorname{D}_{i}\Big|\operatorname{Y}_{0i},\,\operatorname{Y}_{1i},\,\operatorname{X}_{i}
ight)\Big|\operatorname{Y}_{0i},\,\operatorname{Y}_{1i},\,p(\operatorname{X}_{i})
ight] \end{aligned}$$

Theorem If $(Y_{0i}, Y_{1i}) \perp D_i | X_i$, then $(Y_{0i}, Y_{1i}) \perp D_i | p(X_i)$.

Proof

$$egin{aligned} &\operatorname{Pr}iggl[\mathrm{D}_i = 1 \Big| \mathrm{Y}_{0i}, \, \mathrm{Y}_{1i}, \, p(\mathrm{X}_i) iggr] = \cdots = Eiggl[Eiggl(\mathrm{D}_i \Big| \mathrm{Y}_{0i}, \, \mathrm{Y}_{1i}, \, X_i iggr) \Big| \mathrm{Y}_{0i}, \, \mathrm{Y}_{1i}, \, p(\mathrm{X}_i) iggr] \ &= Eiggl[Eiggl(\mathrm{D}_i \Big| \mathrm{X}_i iggr) \Big| \mathrm{Y}_{0i}, \, \mathrm{Y}_{1i}, \, p(\mathrm{X}_i) iggr] \ &= Eiggl[p(\mathrm{X}_i) \Big| \mathrm{Y}_{0i}, \, \mathrm{Y}_{1i}, \, p(\mathrm{X}_i) iggr] \ &= p(\mathrm{X}_i) \end{aligned}$$

 $\therefore (\mathrm{Y}_{0i}, \, \mathrm{Y}_{1i}) \perp\!\!\!\perp \mathrm{D}_i | \mathrm{X}_i \implies (\mathrm{Y}_{0i}, \, \mathrm{Y}_{1i}) \perp\!\!\!\perp \mathrm{D}_i | p(\mathrm{X}_i) \quad \checkmark$

Intuition

Q What's going on here?

 X_i carries way more information than $p(X_i)$, so how can we still get conditional independence of treatment by only conditioning on $p(X_i)$?

A₁ Conditional independence of treatment isn't about extracting all of the information possible from X_i . We actually only care about creating a situation in which D_i |something is independent of (Y_{0i}, Y_{1i}) .

 A_2 Back to our main concern: **selection bias**. People select into treatment. If X says two people were equally likely to be treated, and if X_i explains all of selection (CIA), then there cannot be selection between these two people.

Estimation

So where do propensity scores come from?

We estimate them—and there are a lot of ways to do that.

- 1. Flexible (*i.e.*, interactions) logit specification
- 2. Kernel regression (remember kernel functions?)
- 3. Many others—machine learning, series-logit estimator, etc.

Q Can we just use plain OLS (linear probability model)?

A Sort of. Think about FWL. This route is going to be the same as a regression conditioning on X_i .

Estimation

From *MHE* (p. 83)

Question

A big question here is how to best model and estimate $p(\mathbf{X}_i)$...

Answer

The answer to this is inherently application-specific. A growing empirical literature suggests that a logit model for the propensity score with a few polynomial terms in continuous covariates works well in practice...

Application

So you have some estimated propensity scores $\hat{p}(\mathbf{X}_i)$. What next?

Option 1 Conditioning via regression

Option 1a Use a **regression to condition** on $p(X_i)$, *i.e.*,

$$\mathbf{Y}_i = \alpha + \delta \mathbf{D}_i + \beta p(\mathbf{X}_i) + u_i$$
 (1a)

Option 1b If we think treatment effects are heterogeneous and may covary with X, then we might want to also **interact** treatment with $p(X_i)$, *i.e.*,

$$\mathbf{Y}_i = \alpha + \delta_1 \mathbf{D}_i + \delta_2 \mathbf{D}_i p(\mathbf{X}_i) + \beta p(\mathbf{X}_i) + u_i$$
 (1b)

Heterogeneity with regression

Let's think a bit more about heterogeneous treatment effects in this setting.

$$egin{aligned} \mathrm{Y}_{0i} &= lpha + eta \mathrm{X}_i + u_i \ \mathrm{Y}_{1i} &= \mathrm{Y}_{0i} + \delta_1 + \delta_2 \mathrm{X}_i \end{aligned}$$

i.e., the treatment effect depends upon X_i .

$$\mathrm{Y}_i = \mathrm{D}_i \mathrm{Y}_{1i} + (1-\mathrm{D}_i) \, \mathrm{Y}_{0i}$$

$$=\mathrm{D}_{i}igg(\mathrm{Y}_{0i}+\delta_{1}+\delta_{2}\mathrm{X}_{i}igg)+\left(1-\mathrm{D}_{i}
ight)\mathrm{Y}_{0i}$$

$$=\mathrm{Y}_{0i}+\delta_{1}\mathrm{D}_{i}+\delta_{2}\mathrm{D}_{i}\mathrm{X}_{i}$$

$$= lpha + \delta_1 \mathrm{D}_i + \delta_2 \mathrm{D}_i \mathrm{X}_i + eta \mathrm{X}_i + u_i$$

Heterogeneity

This final equation

 $\mathbf{Y}_i = \alpha + \delta_1 \mathbf{D}_i + \delta_2 \mathbf{D}_i \mathbf{X}_i + \beta \mathbf{X}_i + u_i$

suggests that we want $p(X_i)$ and $D_i p(X_i)$, i.e.,

$$\mathbf{Y}_i = lpha + \delta_1 \mathbf{D}_i + \delta_2 \mathbf{D}_i p(\mathbf{X}_i) + \beta p(\mathbf{X}_i) + u_i$$
 (1b)

which yields

- 1. a group-specific treatment effect $\delta_1 + \delta_2 p(\mathbf{X}_i)$ for each \mathbf{X}_i
- 2. an average treatment effect $\delta_1 + \delta_2 \overline{p}(\mathbf{X}_i)$

More flexibility

We motivated propensity scores with a desire to reduce dimensionality and estimate/choose/assume fewer parameters.

Adding $p(X_i)$ and $D_i p(X_i)$ as covariates in a linear regression doesn't quite exhaust our potential for flexible/nonparametric estimation.

Blocking

Option 2 Block (stratify) on propensity scores.

- 1. Divide the range of $\hat{p}(\mathbf{X}_i)$ into K blocks (*e.g.*, 0.05-wide blocks).
- 2. Place each observation into a block via its $\hat{p}(\mathbf{X}_i)$.
- 3. Calculate $\hat{\tau}_k$ for each block via difference in means.
- 4. Average the $\hat{\tau}_k$ using their shares of the sample, *i.e.*,

$${\hat au}_{ ext{Block}} = \sum_{k=1}^K {\hat au}_k rac{N_{1k} + N_{0k}}{N}$$

Note Blocking is similar to NN/kernel matching using $p(X_i)$ as distance.

Choosing blocks

Blocking on propensity scores requires defining defining blocks.

One common route involves some iteration.

- 1. Choose blocks.
- 2. Check the **balance of the covariates** within each block.⁺
 - If covariates are not balanced, then split your blocks and repeat.
 - If covariates are balanced, then stop.

† Keep multiple-hypothesis testing in mind. With many covariates and many blocks, you are bound to find statistically significant relationships—even if you are balanced in truth.

Overlap

Blocking emphasizes our overlap assumption, *i.e.*, $0 < \Pr(D_i | X_i) < 1$.

If a block contains zero treated/control units, we cannot calculate $\hat{\tau}_k$.

Caution Logit can hide violations—it forces $0 < \hat{p}(X_i) < 1$.

Common practice Empirically enforce overlap:

- Drop control units with $\hat{p}(X_i)$ below the minimum propensity score in the treatment group.
- Drop treated units with $\hat{p}(\mathbf{X}_i)$ above the maximum propensity score in the control group.

Weighting

Option 3 Weight observations by the inverse propensity score.

Q How does weighting by $1/\hat{p}(\mathbf{X}_i)$ make sense?

A Consider our old (likely biased) friend the difference in means, *i.e.*,

$$\hat{{ au}}_{ ext{Diff}} = \overline{ ext{Y}}_{ ext{T}} - \overline{ ext{Y}}_{ ext{C}} = rac{\sum_i ext{D}_i ext{Y}_i}{\sum_i ext{D}_i} - rac{\sum_i (1 - ext{D}_i) ext{Y}_i}{\sum_i (1 - ext{D}_i)}$$

which we've discussed is biased due to selection into treatment, i.e.,

$$E[\mathrm{Y}_{0i}|\mathrm{D}_i=1]
eq E[\mathrm{Y}_{0i}]$$

Weighting, justified

Suppose we know $p(\mathrm{X}_i)$ and we weight each **treated** individual by $1/p(\mathrm{X}_i)$

$$\begin{split} E\bigg[\frac{\mathrm{D}_{i}\mathrm{Y}_{i}}{p(\mathrm{X}_{i})}\bigg] &= E\bigg[\frac{\mathrm{D}_{i}\left(\mathrm{D}_{i}\mathrm{Y}_{1i} + (1 - \mathrm{D}_{i})\mathrm{Y}_{0i}\right)}{p(\mathrm{X}_{i})}\bigg] = E\bigg[\frac{\mathrm{D}_{i}\mathrm{Y}_{1i}}{p(\mathrm{X}_{i})}\bigg] \\ &= E\bigg(E\bigg[\frac{\mathrm{D}_{i}\mathrm{Y}_{1i}}{p(\mathrm{X}_{i})}\bigg|\,\mathrm{X}_{i}\bigg]\bigg) = E\bigg(\frac{E[\mathrm{D}_{i}\mid\mathrm{X}_{i}]\,E[\mathrm{Y}_{1i}\mid\mathrm{X}_{i}]}{p(\mathrm{X}_{i})}\bigg) \\ &= E\bigg(\frac{p(\mathrm{X}_{i})\,E[\mathrm{Y}_{1i}\mid\mathrm{X}_{i}]}{p(\mathrm{X}_{i})}\bigg) = E\bigg(E[\mathrm{Y}_{1i}\mid\mathrm{X}_{i}]\bigg) = E[\mathrm{Y}_{1i}]$$

Similarly, weighting **control** individuals by $1/(1-p(\mathrm{X}_i))$ yields

$$Eigg[rac{(1-\mathrm{D}_i)\mathrm{Y}_i}{1-p(\mathrm{X}_i)}igg]=E[\mathrm{Y}_{0i}]$$

Weighting: The estimator

Thus, we can estimate an unbiased treatment effect via

$${\hat au}_{p ext{Weight}} = rac{1}{N}\sum_{i=1}^{N}\left[rac{ ext{D}_{i} ext{Y}_{i}}{p(ext{X}_{i})} - rac{(1- ext{D}_{i}) ext{Y}_{i}}{1-p(ext{X}_{i})}
ight]$$

Intuition We're trying to overcome selection bias, *i.e.*, treated individuals were more likely to be treated as a function of X_i —producing higher $p(X_i)$.

We want to get back to as-good-as random variation in treatment.

So we upweight (1) **treated** individuals with low $p(X_i)$ and (2) **control** observations with high $p(X_i)$.

Weighting: The example

Suppose for some individual *i*, $p(X_i) = 0.80$.

This propensity score says someone with this set of X_i was four-times more likely to be **treated** than **control**.

Our weights fix this imbalance for each X_i .

- If i is **treated**, then her weight is $1/p(\mathrm{X}_i) = 1/0.80 = 1.25$
- If i is **control**, then her weight is $1/(1-p(\mathrm{X}_i)) = 1/(1-0.80) = 5$

And guess what 5/1.25 is... 4! This weighting scheme gets us back to equal representation for each set of X_i .

Weighting: Last issue

Practical issue Nothing guarantees $\sum_{i} \hat{p}(\mathbf{X}_{i}) = 1$.

Solution Normalize weights by their total sum.

Applying the normalized (and estimated) propensity scores

$$\hat{ au}_{p ext{Weight}} = \sum_{i=1}^{N} rac{rac{\mathrm{D}_{i}\mathrm{Y}_{i}}{\hat{p}(\mathrm{X}_{i})}}{\sum_{i}rac{\mathrm{D}_{i}}{\hat{p}(\mathrm{X}_{i})}} - \sum_{i=1}^{N} rac{rac{(1-\mathrm{D}_{i})\mathrm{Y}_{i}}{1-\hat{p}(\mathrm{X}_{i})}}{\sum_{i}rac{(1-\mathrm{D}_{i})}{1-\hat{p}(\mathrm{X}_{i})}}$$

Hirano, Imbens, and Ridder (2003) suggests this estimator is efficient.

Why choose one?

There's nothing special about weighted averages—regression can weight.

Thus, a regression-based estimate

$$\mathrm{Y}_i = lpha + \mathrm{X}_ieta + au\mathrm{D}_i + u_i$$

with **weights**

$$w_i = \sqrt{rac{\mathrm{D}_i}{\hat{p}(\mathrm{X}_i)} + rac{(1-\mathrm{D}_i)}{1-\hat{p}(\mathrm{X}_i)}}$$

offers a *doubly robust* property—you have two chances to be right: $p(X_i)$ or the regression specification.

Why choose one? Part two

An alternative, doubly robust method combines propensity-score blocking with regression.

Step 1 For each block k, we run the regression

$$\mathbf{Y}_i = \alpha_k + \mathbf{X}_i \beta_k + \tau_k \mathbf{D}_i + u_i$$

Step 2 Aggregate block-level treatment-effect estimates

$$\hat{ au} = \sum_{k=1}^K \hat{ au}_k rac{N_{1k}+N_{0k}}{N}$$

Major requirements

Don't get (too) caught up in the bells and whistles.

We still have two **major** requirements for any of these methods to work.

1. Is the **conditional-independence assumption** true?

2. Do we have **overlap** between treatment and control units.

We can look for evidence of (2) in the data—particularly if we're using propensity-score methods.[†]

How? Plot the distributions of $p(\mathbf{X}_i)$ for **T** and **C**.

 $^{+}$ Checking for overlap in X-space, can be tough as the dimensions of X expand.

Missing overlap in $p(\mathbf{X}_i)$



Authentic (enforced) overlap in $p(\mathbf{X}_i)$





Logit-based $\hat{p}(\mathrm{X}_i)$ hiding some of the missing overlap in $p(\mathrm{X}_i)$

Overlap in one dimension does not guarantee in two dimensions.

Note Shading denotes **share of treatment:** white =0% and **pink**=100%.

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