

2. Matching and weighting estimators

LPO 8852: Regression II

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Selection bias

Lecture 1 showed why the simple difference in means between treated and untreated cases does not identify the ATT (or ATE):

$$\begin{aligned} E(Y|D=1) - E(Y|D=0) &= \\ E[Y(1)|D=1] - E[Y(0)|D=0] &= ATT + \underbrace{E[Y(0)|D=1] - E[Y(0)|D=0]}_{\text{selection bias}} \end{aligned}$$

Selection bias reflects differences in $Y(0)$ between the $D=1$ and $D=0$.

- Randomization of D eliminates selection bias!
- Regression can help under very strong conditions about potential outcomes.

Matching and weighting

Matching and weighting estimators construct comparison groups that are *balanced* on a set of observable variables. There are lots of ways to do this:

- Selecting specific matches
- Constructing a matched weighted sample
- Subclassification / stratification

Key assumption for causal interpretation: once we have conditioned on observables—by selecting matches, constructing weights, or stratifying—treatment assignment and potential outcomes are independent. This is the conditional independence assumption (CIA).

A note on weighted averages

What is a weighted average? Given a weight w_i for each observation i , the weighted average for Y is:

$$\frac{\sum_{i=1}^n w_i Y_i}{\sum_{i=1}^n w_i}$$

Weights are used for lots of reasons (Solon, Haider, & Wooldridge, 2015). In matching we may choose weights based on the values of confounders to eliminate differences in X between treated and untreated groups.

Example 1: private vs. public colleges, revisited

This is a stylized version of the private college example in Lecture 1:

Private				Public			
Student	Ivy	Leafy	Smart	All State	Tall State	Altered State	Earnings
A	1	Reject	Admit		Admit		110000
	2	Reject	Admit		Admit		100000
	3	Reject	Admit		Admit		110000
B	4	Admit		Admit		Admit	60000
	5	Admit		Admit		Admit	30000
C	6		Admit				115000
	7		Admit				75000
D	8	Reject		Admit	Admit		90000
	9	Reject		Admit	Admit		60000

Source: *Mastering Metrics* (2015). Shaded cell represents the student's chosen college, from those they were admitted to. Based on Dale & Krueger (2002).

Example 1

In the above table:

$$E[Y(1)|D = 1] - E[Y(0)|D = 0] = 92,000 - 72,500 = 19,500$$

$$= ATT + \underbrace{E[Y(0)|D = 1] - E[Y(0)|D = 0]}_{\text{selection bias}}$$

It is likely the treated group has a higher $Y(0)$ than the untreated group. This is suggested above by the higher mean earnings for students who applied and were admitted to private colleges (esp. groups A and C).

Example 1

What if we could create equivalent groups by conditioning on some X ?
For example, what if:

$$\underbrace{E[Y(0)|D=1, X]}_{\text{unobserved}} = \underbrace{E[Y(0)|D=0, X]}_{\text{observed!}}$$

In other words, there is no difference in potential outcomes $Y(0)$ between $D=0$ and $D=1$, once we condition on X . Then we could contrast the mean Y for each set of X and then average them.

In the private vs. public college example, assume there is no difference in $Y(0)$ conditional on application/admitted group A-D:

Example 1

	Student	Ivy	Leafy	Smart	All State	Tall State	Altered State	Earnings
A	1		R	A		A		110000
	2		R	A		A		100000
	3		R	A		A		110000
B	4	A			A		A	60000
	5	A			A		A	30000
C	6		A					115000
	7		A					75000
D	8	R			A	A		90000
	9	R			A	A		60000

$Avg(Y|D=1, \text{Group}=A)=105,000$

$Avg(Y|D=0, \text{Group}=A)=110,000$. Difference = $105,000 - 110,000 = -5,000$

$Avg(Y|D=1, \text{Group}=B)=60,000$

$Avg(Y|D=0, \text{Group}=B)=30,000$. Difference = $60,000 - 30,000 = 30,000$

Example 1

The simple average of the within-group differences (groups A and B) is:

$$(-5,000 + 30,000)/2 = \$12,500$$

A *weighted* average by group size gives more weight to the group with more students:

$$(-5,000) * (3/5) + (30,000) * (2/5) = \$9,000$$

Another weighted average assigns weights according to the number of *treated* students in each group:

$$(-5,000) * (2/3) + (30,000) * (1/3) = \$6,666$$

Example 1

Weighted averages use the data more efficiently, and also generalize appropriately to the groups included in the calculation. Note groups C and D are either all treated (private college) or all untreated (public college). There is no **common support** here. This term will come up again.

Example 1

Note in this example that neither the weighted nor unweighted average of groups A and B estimate the ATE or ATT for this population. This is due to the lack of common support.

- Without a counterfactual for the treated in group C, we can't estimate ATT (or ATE)
- Without a counterfactual for the untreated in group D, we can't estimate ATU (or ATE)

An illustration of the importance of being attentive to the population to which you are able to generalize with your data. Note: generalizing to groups A and B may still be interesting! It also may be the best you can do.

Example 1

Mastering Metrics explains how regression estimates are weighted averages of multiple matched comparisons. E.g., consider the regression:

$$Y_i = \alpha + \beta P_i + \gamma A_i + e_i$$

where $P_i = 1$ if the student attended a private college and $A_i = 1$ if the student was in group A (versus B). Groups C and D are excluded.

Using the Example 1 data, $\hat{\beta} = 10,000$. This is comparable to the averages found earlier, but is not identical to any of them. Regression effectively applies different weights. It estimates a **variance-weighted treatment effect**. See also Sloczynski (2022).

Example 1

The weighted averages in Example 1 can be characterized as:

- A **matching** approach: each treated case (private college attendee) is matched to one or more untreated case (public college attendee) with the same observable characteristic (application/admission group). Then the outcomes of the matched observations are compared.
- A **subclassification / stratification** approach: cases are stratified according to some observable characteristic (application/admission group), mean differences are calculated within each group, and then averaged across groups.

Identifying assumption: conditional on application/admissions group, potential outcomes are balanced across treated and untreated cases. Treatment assignment is “as good as random.”

Example 2: Catholic schools

Murnane & Willett (ch. 12) stratify the NELS sample by family income to estimate the effect of Catholic high school attendance on 12th grade math achievement:

Table 12.1 Descriptive statistics on annual family income, by stratum, overall and by type of high school attended, and average twelfth-grade mathematics achievement by income stratum and by high-school type ($n = 5,671$)

Stratum	Average Base-Year Annual Family Income (1988 dollars, 15-point ordinal scale)	Cell Frequencies	Average Mathematics Achievement (12th grade)	
			Public Catholic	Diff.
Label	Income Range	Sample Variance	Sample Mean	
			Public Catholic	
			(% of stratum total)	
<i>Ht_Inc</i>	\$35,000 to \$74,999	0.24	11.38 11.42 1,969 344 (14.87%)	53.60 55.72 2.12***†
<i>Med_Inc</i>	\$20,000 to \$34,999	0.22	9.65 9.73 1,745 177 (9.21%)	50.34 53.86 3.52***†
<i>Lo_Inc</i>	≤\$19,999	3.06	6.33 6.77 1,365 71 (4.94%)	46.77 50.54 3.76***†
				Weighted Average ATE 3.01
				Weighted Average ATT 2.74

† $p < 0.10$; * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$
†One-sided test.

Example 2

Calculate the difference within each strata and then the weighted average of these differences across strata.

The estimate of ATE (3.01) uses *total* cell sizes as weights; the estimate of ATT (2.74) uses counts of *treated* cases in each cell as weights. These estimates are smaller than the unconditional mean differences in math scores (3.895), suggesting an upward bias.

Note income is a continuous variable. M&W created three income strata with the aim of (1) creating balance in family income within each strata; (2) maintaining common support.

Identifying assumption: conditional on income (strata), enrollment in Catholic school is “as good as random” (!).

Example 2

One could stratify on multiple covariates, as M&W do here with income and a measure of prior achievement (12 total cells):

Table 12.2 Sample frequencies and average twelfth-grade mathematics achievement, by high-school type, within 12 strata defined by the crossing of stratified versions of base-year annual family income and mathematics achievement ($n = 5,671$)

Stratum		Cell Frequencies		Average Mathematics Achievement (12th Grade)		
Base-Year Family Income	Base-Year Mathematics Achievement	Public	Catholic	Public	Catholic	Diff.
<i>Hi_Inc</i>	<i>Hi_Ach</i>	1,159	227	58.93	59.66	0.72
	<i>MHi_Ach</i>	432	73	49.18	50.71	1.53 ^{*,†}
	<i>MLo_Ach</i>	321	38	42.75	44.23	1.48
	<i>Lo_Ach</i>	57	6	39.79	40.40	0.62
<i>Med_Inc</i>	<i>Hi_Ach</i>	790	93	57.42	59.42	2.00 ^{***,†}
	<i>MHi_Ach</i>	469	49	47.95	50.14	2.19 ^{***,†}
	<i>MLo_Ach</i>	390	33	41.92	44.56	2.64 ^{***,†}
	<i>Lo_Ach</i>	96	2	37.94	39.77	1.83
<i>Lo_Inc</i>	<i>Hi_Ach</i>	405	36	56.12	56.59	0.47
	<i>MHi_Ach</i>	385	13	47.12	48.65	1.53
	<i>MLo_Ach</i>	433	21	40.99	41.70	0.71
	<i>Lo_Ach</i>	142	1	36.81	42.57	5.76
				Weighted Average ATE		1.50
				Weighted Average ATT		1.31

* $p < 0.10$; ** $p < 0.05$; *** $p < 0.01$; **** $p < 0.001$

[†]One-sided test.

Curse of dimensionality

Finer strata may provide a stronger argument for the conditional independence assumption that treatment group membership is unrelated to potential outcomes (within strata), but they make it more and more difficult to achieve common support—the **curse of dimensionality**.

Matching

There are many approaches to constructing matched comparison groups:

- Exact matching
- Coarsened exact matching
- Nearest neighbor/distance matching
- Propensity score matching

Exact matching

As the name suggests, **exact matching** entails pairing each treated observation with one or more untreated observations with the same X (one or more matching variables). Estimate the ATT with:

$$\widehat{ATT} = \frac{1}{N_T} \sum_{D_i=1} (Y_i - Y_{j(i)})$$

where $Y_{j(i)}$ represents the Y for the matched case(s) for treated observation i . If multiple exact matches are used, $Y_{j(i)}$ stands in for the average of these.

Note: this could also be done for untreated observations to estimate ATU.

Nearest neighbor matching

Nearest neighbor, approximate, or distance matching relaxes the need for an exact match and identifies “nearest neighbors” based on one or more matching variables X . Some distance measures:

- Euclidean distance
- *Normalized* Euclidean distance
- Mahalanobis distance

\widehat{ATT} is the same as the previous slide, but the matched case(s) used for $Y_{j(i)}$ are based on distance (e.g., nearest neighbor) criteria.

Euclidean and normalized Euclidean distance

Suppose you have a vector X of k variables for two units i and j . The Euclidean distance between X_i and X_j is:

$$\|X_i - X_j\| = \sqrt{\sum_{m=1}^k (X_{mi} - X_{mj})^2}$$

These X_m variables are likely on different scales. The *normalized* Euclidean distance scales each variable by its variance:

$$\sqrt{\sum_{m=1}^k \frac{(X_{mi} - X_{mj})^2}{\sigma_m^2}}$$

Mahalanobis distance

Suppose you have a vector X of k variables for two units i and j . The Mahalanobis distance between X_i and X_j is:

$$d(X_i, X_j) = \sqrt{(X_i - X_j)' C^{-1} (X_i - X_j)}$$

Loosely, this is the sum of squared distances between values in X_i and X_j normalized by the covariance. (C is the covariance matrix for the matching variables in X). If there is no covariance between the X , this reduces to the normalized Euclidean distance.

Why “take out” the covariance? Suppose there is some latent characteristic that shows up in multiple matching variables. If those multiple variables are used to calculate distance, we may be “double-counting” by using distance on all of those variables.

Stata's teffects commands

Stata's `teffects` commands implement a wide array of treatment effect estimators using matching, weighting, regression adjustment, etc.

- `teffects nnmatch`: exact and/or nearest neighbor matching
- `teffects psmatch`: propensity score matching
- `teffects ipw`: inverse probability weighting
- ...and others

The `teffects` manual from Stata is actually worth reading! See also my handout: *Stata commands for matching*.

Stata's teffects nnmatch

`teffects nnmatch` implements exact or nearest neighbor matching—or a combination of these.

`teffects nnmatch (y x) (t), options`

y is the outcome, x are the matching variables, and t is the treatment indicator. In the options can use `ematch(vars)` to specify a list of variables on which you desire an exact match. For nearest neighbor matching you can specify the distance metric used, e.g., `metric(euclidean)`. Mahalanobis is the default.

There are lots of other options!

Matching: objectives

Again, there are lots of approaches to creating matched comparison groups. However, there are a few basic principles:

- 1 You are appealing to the **conditional independence** assumption. So choose matching variables that make this plausible.
- 2 Given a choice of X , you want to see **balance** in your matched comparison groups. Ideally, you want to see balance in the full distributions of X , not just the means.
- 3 You want **common support**: treated and untreated cases throughout your distribution of X .
- 4 You want **efficient** estimators (smaller standard errors). Use more of the data when possible, but there is a bias-efficiency tradeoff..

Staying honest with teffects

teffects will automatically give you a treatment effect estimate based on the procedure you request (e.g., `nnmatch`). The option `ate` or `atet` in the options will request the ATE or ATT, respectively.

A word of caution: matching often involves multiple iterations to obtain better balance. It is not good practice to allow estimates of the treatment effect to guide your decisions about matching!!

You can precede `teffects` with `quietly:` to suppress the output. It will do all of the necessary matching—allowing you to do balance diagnostics—without letting you “cheat” by seeing the ATE.

Alternative commands like `psmatch2` can perform matching without requesting a treatment effect estimate.

Stata's tebalance summarize

Can use tebalance summarize following teffects nnmatch:

```
. tebalance summarize
note: refitting the model using the generate() option
```

Covariate balance summary			
	Raw	Matched	
Number of obs =	200	168	
Treated obs =	84	84	
Control obs =	116	84	

	Standardized differences		Variance ratio	
	Raw	Matched	Raw	Matched
age	.5124947	.0095797	.8829962	1.011965
educ	.1125516	.20222	1.038685	1.08452

Note: the *standardized difference* is the difference in means between the treated and untreated groups, divided by the square root of a pooled variance. They can be interpreted in standard deviation units.

$$\frac{\bar{X}_1 - \bar{X}_0}{\sqrt{(s_0^2 + s_1^2)/2}}$$

Stata's tebalance summarize

Try tebalance summarize, baseline following teffects to see baseline (pre-matching) differences in covariates in the original units.

```
. tebalance summarize, baseline
note: refitting the model using the generate() option
```

Covariate balance summary			
	Raw	Matched	
Number of obs =	750	556	
Treated obs =	278	278	
Control obs =	472	278	

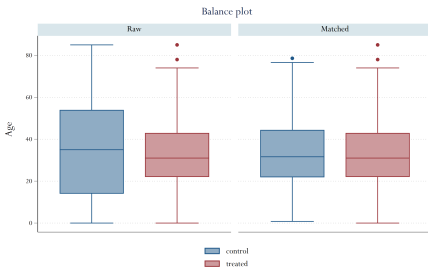
	Means		Variances	
	Control	Treated	Control	Treated
age	27.49364	30.3705	41.56259	38.57342

Stata's tebalance summarize

Note: when there are *multiple* nearest neighbor matches, they should be appropriately weighted so that the sum of the weights of one's neighbors equals one. (In other words, if one treatment observation has five matched untreated neighbors, they will each count as $1/5$). Stata should do this automatically in `tebalance`.

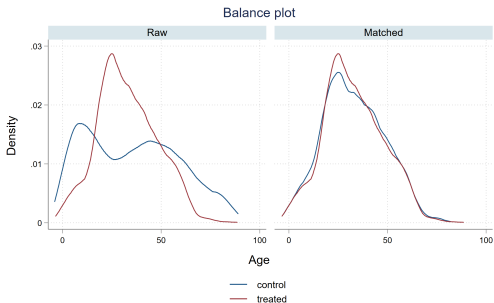
Stata's tebalance box

Can use `tebalance box` to get a fuller picture of the matched sample distributions:



Stata's tebalance density

Can use tebalance density to get a fuller picture of the matched sample distributions:



In-class example 1

See do-file: *Lecture 2 matching with simulated data*. In this example, potential earnings (y) are affected by age and education. There is also a treatment ($treat$) that is positively related to age and education. This do-file illustrates:

- Exact matching on one variable (age)
- Exact matching on multiple variables (age and education)
- Nearest neighbor matching (Euclidean and Mahalanobis)
- Balance checking
- Capturing the observation numbers of nearest neighbors (and distance to these neighbors); imputing “potential outcomes”

Refining nearest neighbor matches

There are a number of things you can do to control the number and quality of nearest neighbor matches:

- Choose a **caliper** or bandwidth for acceptable matches, in terms of distance. The option `caliper(#)` sets the max distance allowed.
- Can choose the number of nearest neighbors desired with `nneighbor(#)` option (the default is 1). Note ties are used.
- Can take all neighbors within a given caliper (**radius** matching) or k nearest neighbors subject to being within the caliper.
- Can perform matching with or without replacement. (`teffects nnmatch` is with replacement).

Refining nearest neighbor matches

There is typically a **bias-efficiency tradeoff** in these decisions. More matches = larger sample size = less sampling variation. But more matches typically means allowing for “worse” matches, so more opportunity for bias.

Matching with replacement = “better” matches. But matching with replacement means fewer observations used in the estimator—the same observations may be used over and over again as nearest neighbors.

Abadie & Imbens (2011) bias correction

When the conditional independence assumption holds, the only source of bias when matching comes from imbalance in the covariates (i.e., imperfect matches).

When there is imperfect matching, the treatment effect estimator is a combination of the “true” effect and differences in Y that are a byproduct of the—hopefully small—imbalance in covariates.

Abadie & Imbens (2011) propose a consistent bias-corrected estimator. The idea here is that one can use OLS to estimate the relationship between Y and covariates X . The difference in (predicted) Y due to the differences in X (between the perfect and actual match) is used to adjust the treatment effect estimate. In `teffects`: use `biasadj(varnames)` option with `varnames` the list of continuous covariates.

Post-matching predictions

After `teffects nnmatch` you can predict new variables that contain each unit's “potential outcomes” (`po`) and “treatment effect” (`te`). Obviously, we can't know these! These are imputed based on the matches.

- Need to specify which potential outcome condition you want (e.g., Y_{i0} or Y_{i1}). Let's call `po0` the potential outcome in the untreated state and `po1` the potential outcome in the treated state.
- For treated observations, `po0` is the mean outcome of their matched untreated observations. `te` is the difference between their actual y and this imputed counterfactual.
- For untreated observations, `po1` is the mean outcome of their matched treated observations. `te` is the difference between their actual y and this imputed counterfactual.

Using mahapick for Mahalanobis matching

Sometimes we want to use Mahalanobis matching to identify nearest neighbors for other reasons (e.g., identifying similar schools for participation in a study). An alternative command for identifying k nearest neighbors (with replacement) using Mahalanobis distance is `mahapick`. It automatically creates the list of matches and can output them to a file.

```
mahapick x1 x2 x3..., idvar(id) treated(treat)  
nummatches(#) genfile(filename) score
```

The $x1, x2, x3...$ are the matching variables, id is the unique observation ID, $treat$ is the treatment indicator, and $filename$ is where you want to save the resulting list of matches. `score` tells Stata to include the distance score in the output file. There are lots of other options. See also `mahascore` which gives you distance measures between all pairs.

As always with nearest neighbor matching, be aware of how ties are handled, and whether and how sort order matters.

Using psmatch2 for Mahalanobis matching

Another alternative for Mahalanobis matching is `psmatch2`, which is an older command used for propensity score matching. (More on this later).

```
psmatch2 treat , mahalanobis(x1 x2 x3...) neighbor(#)
```

The $x1, x2, x3...$ are the matching variables, and $treat$ is the treatment indicator. There are lots of options, including radius matching, matching *without* replacement, and more.

As always with nearest neighbor matching, be aware of how ties are handled, and whether and how sort order matters.

Coarsened exact matching

Iacus, King, and Porro (2012) introduced **coarsened exact matching**, in which exact matches are required on continuous variables that have been binned (“coarsened”). See the user-written Stata command `cem`. Ex:

```
cem x1 (#), treatment(treat) showbreaks
```

The option `(#)` is the number of cutpoints for variable `x1`. For example, `(#5)` will use 5 equally-spaced cutpoints. This can be omitted and `cem` will automatically coarsen the data based on a binning algorithm.

Coarsened exact matching

`cem` performs the coarsening and matching and creates weights, but does not estimate the treatment effect. You can do this yourself using the weights created by `cem` (`cem_weights`):

```
reg y treat [iweight=cem_weights]
```

Curse of dimensionality, revisited

The curse of dimensionality comes up again when trying to match on multiple variables. The more matching variables you have, the less likely it is you will find a “close” match on all variables. Getting a better match on one variable X_1 may result in a worse match on X_2 .

Propensity scores

An alternative to matching on multiple variables is the **propensity score**. Think of the propensity score as a “one-number summary” capturing the relationship between a binary treatment and X : $Pr(D_i = 1|X_i)$. It is the **probability of treatment** for unit i given X_i .

The propensity score can be estimated using a logistic model:

$$P(D_i|X_i) = \frac{1}{1 + e^{-X_i\beta}}$$

where the log odds $P/(1 - P)$ is a linear function of X . Other options for estimating propensity scores include probit, LPM, machine learning methods like regularized regression and boosted regression.

Propensity scores

Rosenbaum & Rubin (1983) showed that if potential outcomes Y_{0i}, Y_{1i} are independent of D conditional on X , then they are also independent of D conditional on the probability of treatment given X (the propensity score).

- Rather than stratifying or matching on all of the variables in X , it is sufficient to use the “one-number summary” of the relationship between treatment and X : $Pr(D_i = 1|X_i)$
- Let $P(X)$ be shorthand for the propensity score $Pr(D_i = 1|X_i)$.

Propensity scores

The propensity score estimator for ATT can be written as:

$$\widehat{ATT} = E_{P(X)|D=1} \left(\underbrace{E[Y(1)|D=1, P(X)]}_{\text{treated}} - \underbrace{E[Y(0)|D=0, P(X)]}_{\text{untreated}} \right)$$

In theory, *for each propensity score* we calculate the difference in mean outcomes for the treated and untreated with that $P(X)$. We then take a weighted average of these over the different propensity score values. The subscript $P(X)|D=1$ means we are taking a weighted average over the area of common support (same propensities to be treated).

Compare to Example 2 where we averaged the group differences in earnings across two groups with common support (A and C), weighting as appropriate.

Propensity scores

In practice $P(X)$ takes on a continuum of values and thus stratifying on specific values of $P(X)$ —in the manner we did with subclassification on X (Examples 1 and 2)—is not feasible.

However, we can do other things with the propensity score, including nearest neighbor matching, stratification (on the propensity score) or re-weighting. Even when propensity scores are not used to estimate treatment effects, they can be useful diagnostic tools since they force you to think about balance between the treated and untreated groups, and the model of selection into treatment.

Note: King & Nielson (2019) advise against using propensity scores for matching if estimating treatment effects. (See link to video on Github). They argue that p-scores should just be used for weighting. We introduce propensity score matching here since it is still widely seen in the literature.

Propensity score matching estimator

With nearest neighbor matching based on propensity scores, we can estimate ATT as:

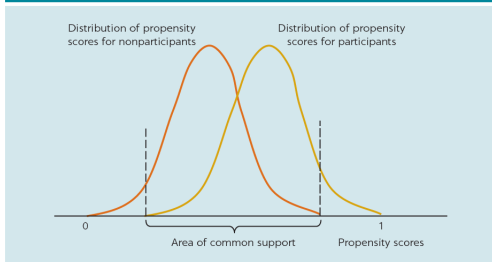
$$\widehat{ATT} = \frac{1}{N_T} \sum_{D_i=1} (Y_i - Y_{j(i)})$$

where $Y_{j(i)}$ represents the average Y for the matched case(s) for treated observation i .

- This sum would only be over the area of common support. Some treated cases may lack untreated cases with “close enough” p-scores.
- One could also find matches for untreated cases and calculate \widehat{ATU} .
- Later: can also assign more weight to neighbors that are “closer.”

Propensity scores: common support

FIGURE 13.1 Propensity scores and the area of common support



Source: Original figure for this publication.

Source: Glewwe & Todd chapter 13

Stata's `teffects psmatch`

`teffects psmatch` estimates propensity scores and can produce ATT and ATE using nearest neighbor matches.

```
teffects psmatch (y) (t x, tmodel), options
```

y is the outcome, x are the covariates, and t is the treatment indicator. $tmodel$ is the type of propensity score model you would like to estimate (e.g., logit, probit). In the options one can specify `ate` or `atet` for the treatment effect estimation, the number of nearest neighbors, the caliper, etc. (like `nnmatch`)

Again, it is best practice to *not* look at the treatment effect estimate until you have settled on a matching model/matched sample! Use quietly until you are ready. (Or see `psmatch2` later).

Checking for balance after teffects psmatch

If matching on propensity scores, it makes sense that our matched samples should be balanced on these. Can predict and inspect estimated propensity scores $\widehat{P(X)}$, to check for common support and balance.

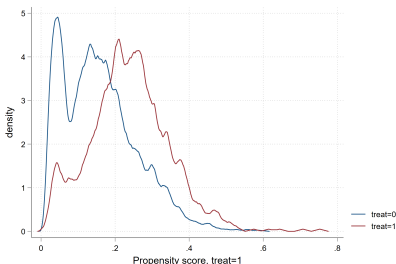
- Can compare the maxima and minima of $\widehat{P(X)}$ for the two groups
- Can compare the density distributions for each

The command `teoverlap` (following `teffects psmatch`) produces densities of propensity scores, although it uses the *full sample* rather than the matched sample (not ideal). Could save propensity scores and plot them yourself for matched sample. (See sample do-file).

You can also check for balance on the covariates themselves (with `tebalance summarize`)

Stata's teoverlap

`teoverlap` to see overlapping distribution of propensity scores:



Note: need to specify which value of the treatment variable you are interested in the propensity of (option `ptlevel(1)` if `treatment=1`).

Getting estimated propensity scores

Can obtain predicted propensity scores after `teffects psmatch` using `predict`. Requires the `gen()` option in the `teffects psmatch` command, which creates variables containing the IDs of the nearest neighbor(s):

```
predict (newvar), ps tlevel(1) options
```

The option `tlevel(1)` is needed to estimate $Pr(D = 1|X)$

As with `nnmatch` can also predict *potential outcomes* (`po`), individual treatment effects given potential outcomes (`te`), and distance to nearest neighbor (`distance`).

In-class example 2

See do-file: *Lecture 2 propensity score example*. This example uses a dataset with 4,727 community college students enrolled in an English I course. The aim is to estimate the effect of enrollment in a writing center class. This do-file illustrates:

- Propensity score estimation and nearest neighbor matching
- Balance checking and checking for common support
- Capturing the estimated propensity scores and distances
- Use of the older `psmatch2` command

Using psmatch2 for propensity score matching

I have also used the older user-written package `psmatch2`, which is useful for refining your propensity score model *before* requesting the treatment effect estimate. It has some other nice features that `teffects` doesn't.

This package should not be used for treatment effect estimation, however, as the standard errors are incorrect. Use `teffects` for the final treatment effect estimation, after you have decided on your propensity score model.

Using psmatch2 for propensity score matching

`psmatch2` can estimate propensity scores, find matches, and estimate treatment effects:

```
psmatch2 t x , outcome(y) ate logit ties
```

`t` is the treatment indicator, `x` are the covariates, and `y` is the outcome variable. There are lots of options, including type of propensity score model (probit is the default, can type `logit` for logit), number of nearest neighbors, caliper, etc. By default, finds 1 nearest neighbor and does not keep ties unless you use the `ties` option.

By default, `psmatch2` shows you the result of the propensity score model itself (logit or probit).

Using psmatch2 for propensity score matching

psmatch2 creates several variables in your dataset: `_pscore`, `_treated`, `_support`, `_weight`, `_id`, `_n1`, `_nn`, `_pdif`

- `_pscore`: estimated $P(X)$
- `_treated`: flags observations Stata recognized as treated
- `_support`: flags observations on common support
- `_weight`: weight for matched untreated obs (1 for treated)
- `_id`: id number assigned for identifying matches
- `_n1`: id of nearest neighbor (treated obs only)
- `_nn`: number of matched neighbors
- `_pdif`: absolute value of diff between $P(X)$ and $P(X)$ of NN

As noted earlier, `teffects psmatch` can be augmented with options (and used with the `predict` command to get similar information)

Checking covariate balance after psmatch2

After `psmatch2` can do covariate balance check using `pstest`:

```
. pstest age educ black hisp re74t re75t,both
```

Variable	Unmatched Matched	Mean Treated Control	%bias (bias)	%reduct (bias)	t-test p> t	V(T)/ V(C)	
age	U	25.816	33.444	-82.3	-9.43	0.000	0.42*
	M	25.816	24.989	8.9	0.95	0.342	0.58*
education	U	10.346	12.04	-67.9	-7.92	0.000	0.48*
	M	10.346	10.811	-18.6	-1.95	0.053	0.62*
black	U	.84324	.09739	224.5	33.96	0.000	.
	M	.84324	.84865	-1.6	-0.14	0.886	.
hispanic	U	.05946	.06671	-3.0	-0.39	0.694	.
	M	.05946	.03784	8.9	0.97	0.335	.
re74t	U	2.0956	14.746	-156.5	-16.63	0.000	0.22*
	M	2.0956	1.7488	4.3	0.79	0.433	1.96*
re75t	U	1.5321	14.38	-170.9	-17.24	0.000	0.10*
	M	1.5321	1.5778	-0.6	-0.14	0.891	1.03

* if variance ratio outside [0.75; 1.34] for U and [0.75; 1.34] for M

Sample	Pt B2	LR chi2	p>chi2	MeanBias	MedBias	B	R	%Var
Unmatched	0.463	961.39	0.000	117.5	119.4	266.1*	0.24*	100
Matched	0.013	6.66	0.354	7.2	6.6	27.0*	0.69	75

* if B>25%, R outside [0.5; 2]

Checking covariate balance after psmatch2

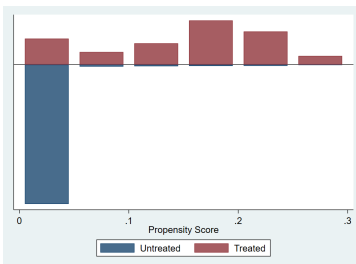
The column %bias above provides the standardized difference: the difference in sample means between the treated and untreated observations as a percentage of the square root of the average of the sample variances in the treated and untreated groups.

$$\Delta_X = \frac{\bar{X}_1 - \bar{X}_0}{\sqrt{(s_0^2 + s_1^2)/2}}$$

Same as `tebalance` summarize

Checking common support after psmatch2

You can compare histograms of propensity scores for the treated and untreated observations using `psgraph` (uses *all* of the data, not just the matched sample)



You can use the `_weight` variable to generate your own plots of propensity scores for the matched sample.

Checking for balance after propensity score matching

Dehejia & Wahba (2002): conditional on propensity score (binned for practicality), matching variables should not be related to treatment (a “stratification test”).

This goes further than just comparing the means and variances of the predictor variables in the matched sample.

See user-written commands `pstrata` and `covbal` for optimal stratification by propensity score and testing balance within strata.

What to do when there is imbalance?

If matched samples are *not* sufficiently balanced, you may need to tweak your matching criteria or propensity score model. In the propensity score model, interaction terms, quadratic or higher-order terms, or additional (or fewer) covariates may help.

If there remains imbalance after matching, can do regression analysis on the matched sample with covariates included for regression adjustment.

Practical advice on propensity score estimation

Choice of model:

- For binary treatment, whether one uses a logit, probit, or LPM model is probably not that consequential.
- For multiple treatments, the choice may be more important (see Caliendo & Kopeinig, 2008). Note: matching can be used for multi-level treatments (not covered here)

Covariate selection:

- Goal: choose X 's such that the unconfoundedness holds—should promote covariate balance.
- Should be correlated with treatment (D) and the outcome Y .
- Selection should be based on theory and contextual knowledge.
- X should be measured *before* treatment, and not affected by it (or by the anticipation of treatment).
- X 's should not be “too good” at predicting treatment—we are relying on common support.

On standard errors

The standard errors for matching estimators are complicated. Why?

- The matching procedure might involve multiple decision points, judgment calls—it's hard to think about these over repeated sampling.
- Propensity scores (and weights) have to be *estimated*, and some analysts may trim observations based on these.

Bootstrapping for standard errors is an option—but only recommended in the weighted matched sample case (not selecting specific matches).

For now, just use default standard errors in `teffects` but keep in mind their limitations.

Inverse probability weighting

Matching methods recap

All of the matching techniques illustrated thus far involve identifying specific matches. Ex:

- Exact matching: each treated case is matched to one or more untreated cases with the same X .
- Nearest neighbor matching: each treated case is matched to one or more untreated cases with the *closest* X .
- Propensity score matching: each treated case is matched to one or more untreated cases with the *closest* $\widehat{P}(X)$.

The matched sample is used to calculate the ATE or ATT.

Disadvantages: the matching process involves a lot of ad hoc decisions, including “pruning” observations that aren’t matched.

Inverse probability weighting (IPW)

Inverse probability weighting (IPW) reweights observations to create desired balance. Weights use the propensity scores:

$$w_{ATT} = D_i + (1 - D_i) \frac{\widehat{P(X)}}{1 - \widehat{P(X)}}$$

$$w_{ATE} = \frac{D_i}{\widehat{P(X)}} + \frac{(1 - D_i)}{1 - \widehat{P(X)}}$$

IPW doesn't rely on matching but uses all of the available data. (Some observations may be pruned if there is no or too little overlap).

Inverse probability weighting (IPW)

Intuition using a simple example:

	Treated ($D = 1$)	Untreated ($D = 0$)	N	$P(D X)$	Y_1	Y_0
$X = 1$	1	9	10	0.1	45	40
$X = 0$	4	1	5	0.8	38	25

- Conditional on X , treatment assignment D is unrelated to potential outcomes (Y_0, Y_1). X is the only confounder.
- Treatment propensity $P(D|X)$ is strongly related to X . The treated are disproportionately $X = 0$ types.

Inverse probability weighting (IPW)

	Treated ($D = 1$)	Untreated ($D = 0$)	N	$P(D X)$	Y_1	Y_0
$X = 1$	1	9	10	0.1	45	40
$X = 0$	4	1	5	0.8	38	25

Consider the simple difference in mean observed Y : $\bar{Y}_{D=1} - \bar{Y}_{D=0} = 39.4 - 38.5 = 0.9$. There is selection bias because the untreated group is a poor comparison group for the treated. Why? They are imbalanced on X and thus potential outcomes.

The goal of IPW is to construct weights for the treated and untreated so that they are balanced on X . For ATT, choose weights so the untreated “look like” the treated. For ATE, choose weights so that both groups “look like” the full sample.

Inverse probability weighting (IPW): ATT

	Treated ($D = 1$)	Untreated ($D = 0$)	N	$P(D X)$	IPW-ATT Treated	IPW-ATT Untreated
$X = 1$	1	9	10	0.1	1	0.1111
$X = 0$	4	1	5	0.8	1	4

ATT: all treated units get a weight of 1 while untreated units get a weight of $P(D|X)/(1 - P(D|X))$. For each X the effective sample size is the same.

We “up-weight” untreated cases with a high $P(X)$ and “down-weight” untreated cases with a low $P(X)$.

Inverse probability weighting (IPW): ATT

Use the weights to calculate a difference in *weighted* means to estimate the ATT:

$$\frac{\sum_{i \in D=1} w_i Y_i}{\sum_{i \in D=1} w_i} - \frac{\sum_{i \in D=0} w_i Y_i}{\sum_{i \in D=0} w_i}$$

where the w_i are the ATT weights. In this example, the ATT is $39.4 - 28 = 11.4$.

Inverse probability weighting (IPW): ATE

	Treated ($D = 1$)	Untreated ($D = 0$)	N	$P(D X)$	IPW-ATE Treated	IPW-ATE Untreated
$X = 1$	1	9	10	0.1	10.00	1.11
$X = 0$	4	1	5	0.8	1.25	5.00

ATE: Weight the treated by $1/P(D|X)$ and the untreated by $1/(1 - P(D|X))$.

Inverse probability weighting (IPW): ATE

Use the weights to calculate a difference in *weighted* means to estimate the ATE:

$$\frac{\sum_{i \in D=1} w_i Y_i}{\sum_{i \in D=1} w_i} - \frac{\sum_{i \in D=0} w_i Y_i}{\sum_{i \in D=0} w_i}$$

where the w_i are the ATE weights. In this example, the ATE is $42.7 - 35 = 7.7$.

Inverse probability weighting (IPW)

Note: IPW estimators can become unstable when there is low overlap (cases with very low probability of treatment). Re: these observations get extremely high weight when using inverse probability.

Connection to propensity score approach

In the simple example above:

- $P(D|X)$ is the propensity score—the probability of treatment given X .
- If we didn't already know the propensity score, we could estimate it easily from the data.
- The propensity score “model” in this case is pretty trivial, as there is only one binary X .
- If the conditional independence assumption holds with respect to this one X , we can use $P(D|X)$ and the resulting weights to balance X and get an unbiased estimate of treatment effects.

Connection to propensity score approach

In reality, there are more than one confounding X . But the same logic applies:

- There is a “true” propensity score for every combination of X such that—if you knew this propensity score—you could do the same procedure as above (construct weights and estimate treatment effects). Again, presuming CIA holds.
- In practice we need to know how the propensity for treatment varies with all confounders together (X_1, X_2, \dots)
- This could be challenging if the relationship is complicated (nonlinearities, interactions).

IPW in practice involves specifying a treatment model that we hope is a “good enough” estimate of the true propensity score model such that reweighting provides balance in the distributions of X .

Stata's `teffects ipw`

`teffects ipw` can estimate ATT and ATE using inverse probability weighting. Propensity scores (probability of treatment) are used in the weights. The syntax is very similar to `teffects psmatch`:

```
teffects ipw (y) (t x, tmodel), options
```

y is the outcome, *x* are the covariates, and *t* is the treatment indicator. *tmodel* is the type of propensity score model you would like to estimate (e.g., `logit`, `probit`). In the options can specify `ate`, `atet`, or the potential outcome means `po`.

Stata's `teffects ipw`

After estimating the propensity score model using `teffects ipw`, one should do a check for common support with `teoverlap`.

Trimming observations is an option if there are cases outside the common support, or that have very low or high propensities for treatment. (Treated cases with very low propensities and untreated cases with very high propensities will get large weights).

Other approaches to propensity score estimation

It is important to get the propensity score model “right” if propensity scores are going to be used for matching or weighting. What is the “right” model? Again, we want to be able to appeal to the conditional independence assumption in theory and demonstrate balance in practice.

Ad hoc approaches to estimating propensity scores are unsatisfying, which has led to more algorithmic approaches to model specification. Examples: the Imbens & Rubin (2015) algorithm, machine learning (regression trees, boosted regression).

Imbens & Rubin (2015) algorithm: `psestimate`

`psestimate` takes a stepwise approach to adding predictor variables and quadratic terms in search of the best fit.

```
psestimate treat [x1], tottry(x2) genpscore(varname)
```

treat is the treatment indicator, *x1* is the base model list of covariates and *x2* is a list of other variables that will be added in a stepwise fashion to the model. The option *genpscore* allows you to save the estimated propensity scores. A logit model is used for estimation. Note this command takes a long time to run.

TWANG (Toolkit for Weighting and Analysis of Nonequivalent Groups) is a package developed by RAND that uses generalized boosted regression (GBM) to estimate propensity scores and construct ATT or ATE weights. *Note this package calls R from Stata, so R is required.*

- `ps` command: implements GBM to estimate propensity scores.
- `balance` command: balance tables given weights
- `psplot`: diagnostic plots
- `dxwts`: balance assessment using weights generated using any method, not just `ps`.

Optimal propensity score stratification: `pstrata`

`pstrata` uses your estimated propensity scores and stratifies them in a way that minimizes mean differences in propensity scores within strata.

```
pstrata treat , pscore(var) plevel(#)
```

`treat` is the treatment indicator, `var` is the name of the propensity score variable (you estimate this separately), and `plevel()` is an option for a threshold significance level for balance on propensity scores. After running this can test balance on covariates using accompanying command `covbal`.

Matching vs. regression

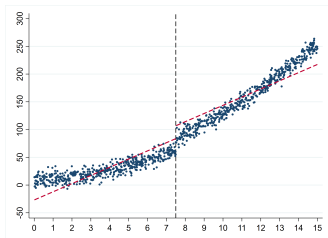
If potential outcomes are independent of treatment conditional on X , why not just estimate a regression controlling for X ?

- Matching does not require assumptions about functional form for the outcome model (e.g., a linear relationship between Y and X).
- Regression runs the risk of extrapolating onto a space where there is little common support.
- Matching focuses our attention on balance and the degree of common support.
- Can estimate treatment effects for different groups.

That said, matching or weighting using propensity scores “shifts the problem to the task of estimating the propensity score.” If the model for the propensity score is poor, the propensity score matching estimator will be biased.

Matching vs. regression

By making strong functional form assumptions, one can use regression to estimate treatment effects even when there is little overlap in X between the treated and untreated cases. But getting the functional form wrong can lead to poor inferences. We'll see this later with regression discontinuity:



Imbens (2015)

Useful guidance from Imbens (2015) on matching methods, with examples.

- Cases in which OLS estimators are likely to be especially problematic for estimating causal effects with non-experimental data
- Two recommended methods: (1) subclassification and regression; or (2) matching
- Trimming and other pre-processing steps to improve balance
- Supplementary analyses for assessing plausibility of conditional independence assumption

A key takeaway: “there are no, and will not be, general results implying that in general some estimators are superior to all others”

Imbens (2015) on OLS

Imbens provides an example illustrating why and when OLS can be problematic. Key takeaway points:

- 1 The OLS regression aims to provide the average of the potential control outcomes for the treated
- 2 Functional form assumptions can matter a lot: extrapolation and misspecification
- 3 This is especially true when the distribution of covariates differs between the treated and untreated cases
- 4 Extreme values can have a large influence on the OLS estimates: “regression models are not fundamentally robust to the substantial differences between treatment and control groups”

Imbens (2015) on analytic methods

Stages:

- ➊ **Design stage:** trimming the full sample and balancing on covariates
- ➋ **Supplementary analysis stage:** assessing balance
- ➌ **Analysis stage:** estimating treatment effect

Important: the outcome data are not used until the last stage.

Imbens (2015) advice

- **Normalized differences:** for assessing balance, use *normalized differences in mean covariates*. In Stata, this is the “% bias” in `pstest`. This is preferable to *t*-tests of significant differences.
- **Propensity score:** Imbens uses logit, but notes the choice of probit or logit matters more when there are cases with `pscores` close to 0 or 1
 - ▶ “the propensity score plays a mechanical role in balancing the covariates ... In choosing a specification, there is therefore little role for theoretical substantive arguments. We are mainly looking for a specification that leads to an accurate approximation to the conditional expectation.”
 - ▶ There is no harm in specification searches at this stage
 - ▶ Interactions and non-linearities are often important
 - ▶ There are data-driven algorithms for selecting covariates (e.g., stepwise approach of Imbens & Rubin, 2015; lasso methods)

- **Propensity score**

- ▶ “However, the point is again not to find a single method for estimating the propensity score that will outperform all others. Rather, the goal is to find a reasonable method for estimating the propensity score that will, in combination with the subsequent adjustment methods, lead to estimates for the treatment effects of interest that are similar to those based on other reasonable methods for estimating the propensity score”
- ▶ Stepwise approach of Imbens & Rubin: first choose a set of predictors that will be in the model, regardless of other decisions (e.g., lagged measures of the outcome). Then determined a threshold for inclusion of other linear and quadratic terms. Finally, successively add predictors and compare to this threshold.

Imbens (2015) on analytic methods

- **Blocking method:** one recommended estimator uses the propensity score to block (or subclassify) observations and then use regression within blocks.
 - ▶ Partition the range of the propensity score into J intervals
 - ▶ Within each interval, estimate a linear regression with some covariates (all or a subset of those thought to be most important)
 - ▶ The J estimates of the treatment effects are then combined into one overall effect
 - ▶ 5 blocks is common, but Imbens & Rubin (2015) propose an algorithm for selecting this
- **Matching method:** with replacement. Rather than using propensity scores, they use the Mahalanobis distance metric:

Additional resources

- Murnane & Willett ch. 12 on the differences between matching strategies and regression (pp. 304-ff).
- Guo & Fraser (2015) textbook: all things propensity scores.
- See Caliendo & Kopeinig (2008) for practical guidance on propensity score matching.
- See Imbens (2015) for guidance on matching and subclassification.
- There are many studies comparing impact estimates from randomized experiments to those using matching methods. E.g., Wilde & Hollister, 2007 using Tennessee STAR experiment; Dehejia & Wahba (1999, 2002); Smith & Todd (2005); Agodini & Dynarski (2004); Diaz & Handa (2006); Michalopoulos, Bloom & Hill (2004)