

Matching

EC 425/525, Set 7

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Prologue

Schedule

Last time

- The conditional independence assumption: $(Y_{0i}, Y_{1i}) \perp\!\!\!\perp D_i | X_i$
- Omitted variable bias
- Good vs. bad controls

Today

- Return first round of project proposals.
- Matching estimators (*MHE* 3.2 and Cameron and Trivedi 25.4).

Upcoming

- Admin: Assignment and midterm
- Next round of the project proposal

Follow up

OLS weighting

At the beginning of the lecture, we discussed OLS weights—especially for heterogeneous treatment effects.

We should keep our questions clear.

1. Which weights on β_1 and β_2 recover β_{12} , where β_i comes from a regression using observations in group i ?
2. What does β represent when the treatment effect is heterogeneous?

More soon.

Matching

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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?

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$$\tau(x) = E[Y_{1i} - Y_{0i} | X_i = x]$$

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The idea: Estimate a treatment effect only using observations with (nearly?) identical values of X_i .

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

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Goals

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

1. We want/need to know $\tau_i = Y_{1i} - Y_{0i}$.
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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.

Matching

More formally

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

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

Assume $\sum_j w_i(j) = 1$. Our estimate for the counterfactual of treated i is

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

Matching

More formally

If our estimated counterfactual for treated individual i is

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

then our estimated treatment effect (for individual i) is

$$\hat{\tau}_i = Y_{1i} - \widehat{Y}_{0i} = Y_{1i} - \sum_j w_i(j) Y_j$$

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

$$\hat{\tau}_M = \frac{1}{N_T} \sum_{i \in (D=1)} \left(Y_{1i} - \widehat{Y}_{0i} \right) = \frac{1}{N_T} \sum_{i \in (D=1)} \left(Y_{1i} - \sum_{j \in (D=0)} w_i(j) Y_j \right)$$

Matching

Weight for it[†]

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

 Plus an interesting, policy-relevant setting with valid conditional independence. And data.

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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.

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The plan Choose weights $w_i(j)$ that indicate **how close** \mathbf{X}_j is to \mathbf{X}_i .

 Plus an interesting, policy-relevant setting with valid conditional independence. And data.

Matching

Proximity

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

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If \mathbf{X} is **continuous**, then we need *proximity* rather than *equality*.

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If \mathbf{X} is **continuous**, then we need *proximity* rather than *equality*.

Nearest-neighbor matching chooses the single closest control observation using the Euclidean distance between \mathbf{X}_i and \mathbf{X}_j , i.e.,

$$d_{i,j} = (\mathbf{X}_i - \mathbf{X}_j)' (\mathbf{X}_i - \mathbf{X}_j)$$

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- $\hat{\tau}_i = \mathbf{Y}_{1i} - \mathbf{Y}_{0j}^i$, where \mathbf{Y}_{0j}^i 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.

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Nearest-neighbor matching with Mahalanobis distance chooses the single closest control using **Mahalanobis** distance between \mathbf{X}_i and \mathbf{X}_j , i.e.,

$$d_{i,j} = (\mathbf{X}_i - \mathbf{X}_j)' \Sigma_X^{-1} (\mathbf{X}_i - \mathbf{X}_j)$$

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

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

Matching

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.

Matching

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** $K(\cdot)$,

$$w_i(j) = \frac{K\left(\frac{\mathbf{X}_j - \mathbf{X}_i}{h}\right)}{\sum_{j \in (D=0)} K\left(\frac{\mathbf{X}_j - \mathbf{X}_i}{h}\right)}$$

Matching

More neighbors!

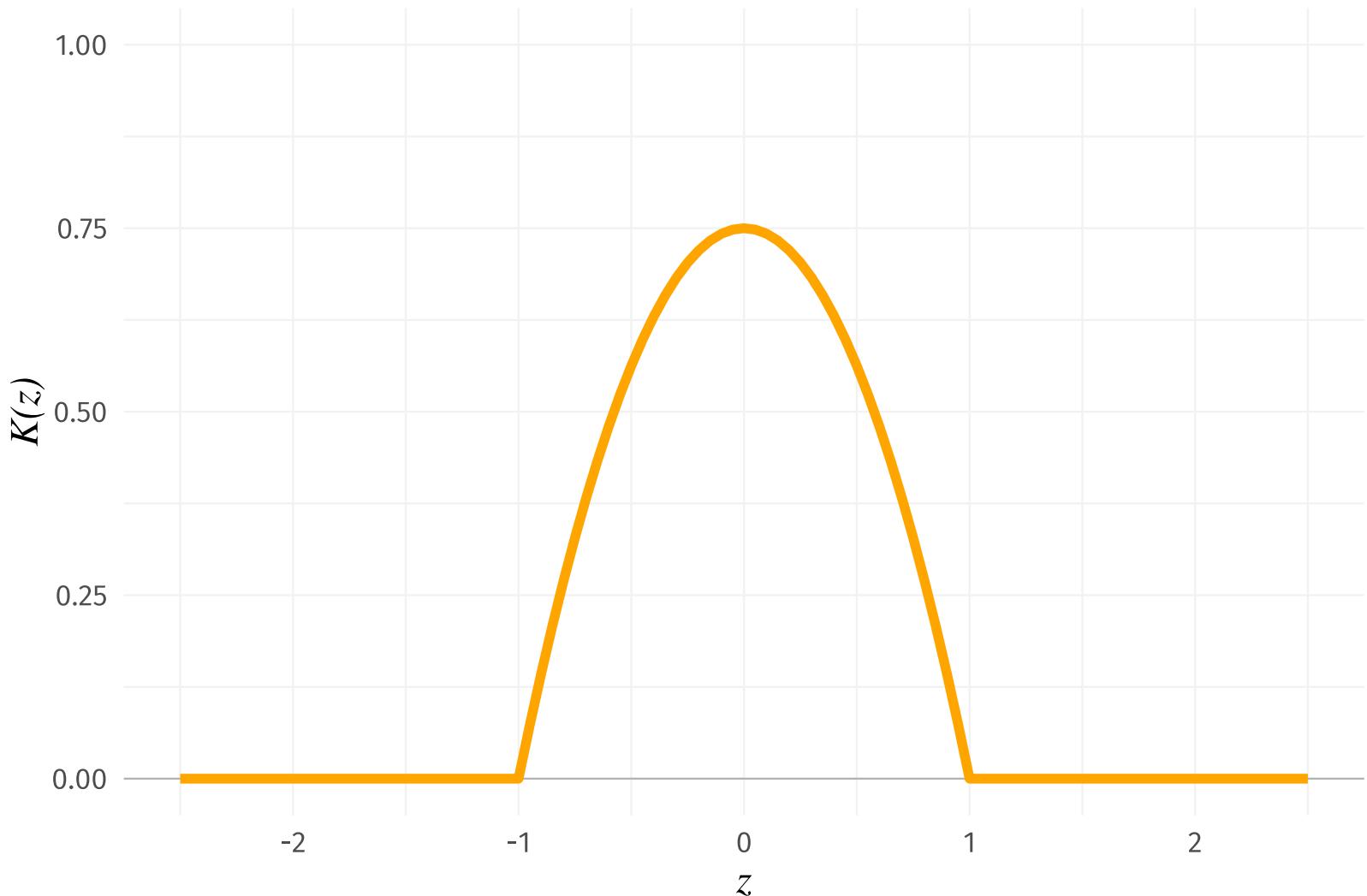
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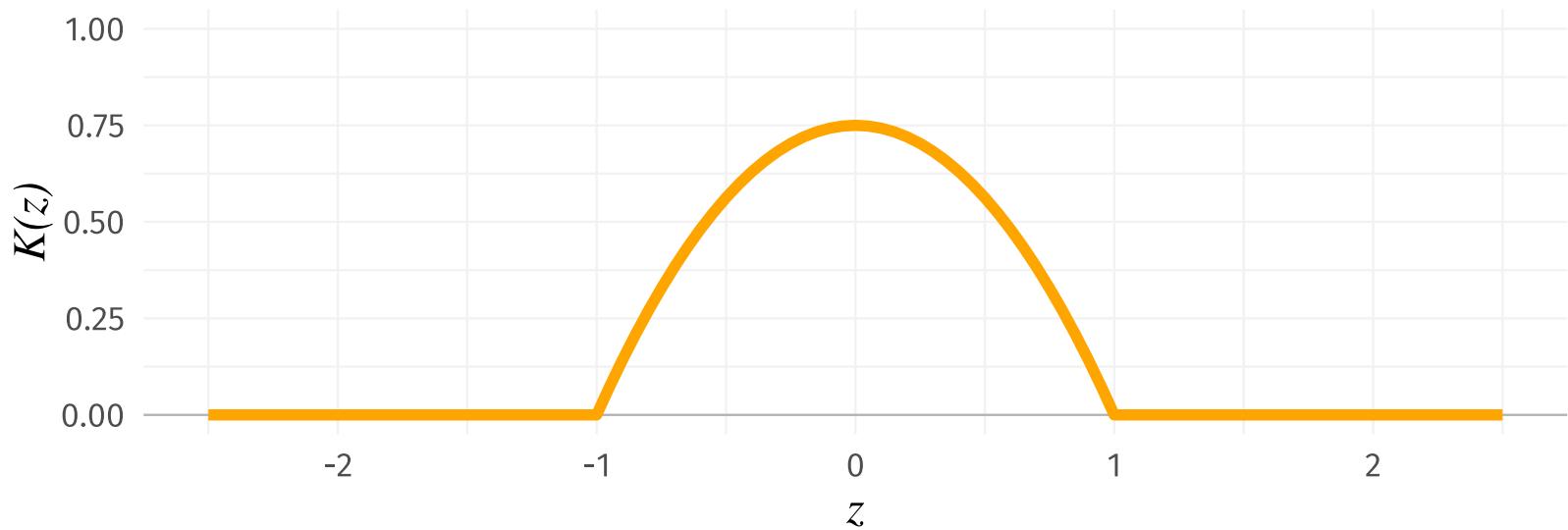
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Example The *Epanechnikov kernel* is defined as

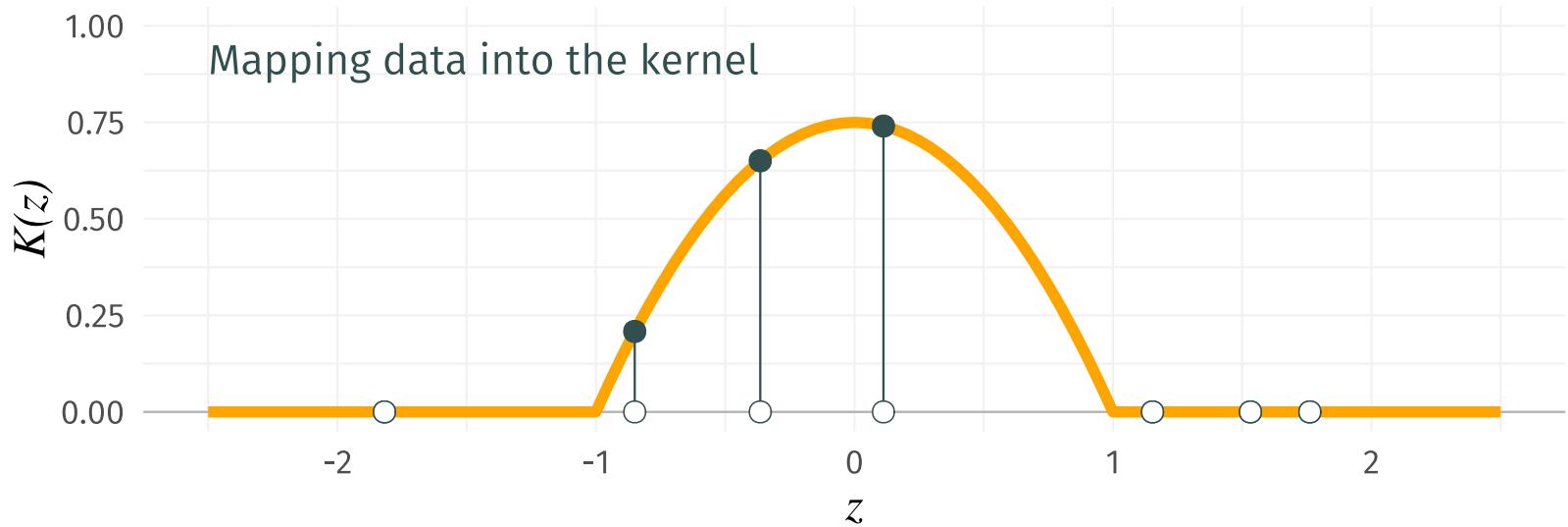
$$K(z) = \frac{3}{4}(1 - z^2) \times \mathbb{I}(|z| < 1)$$

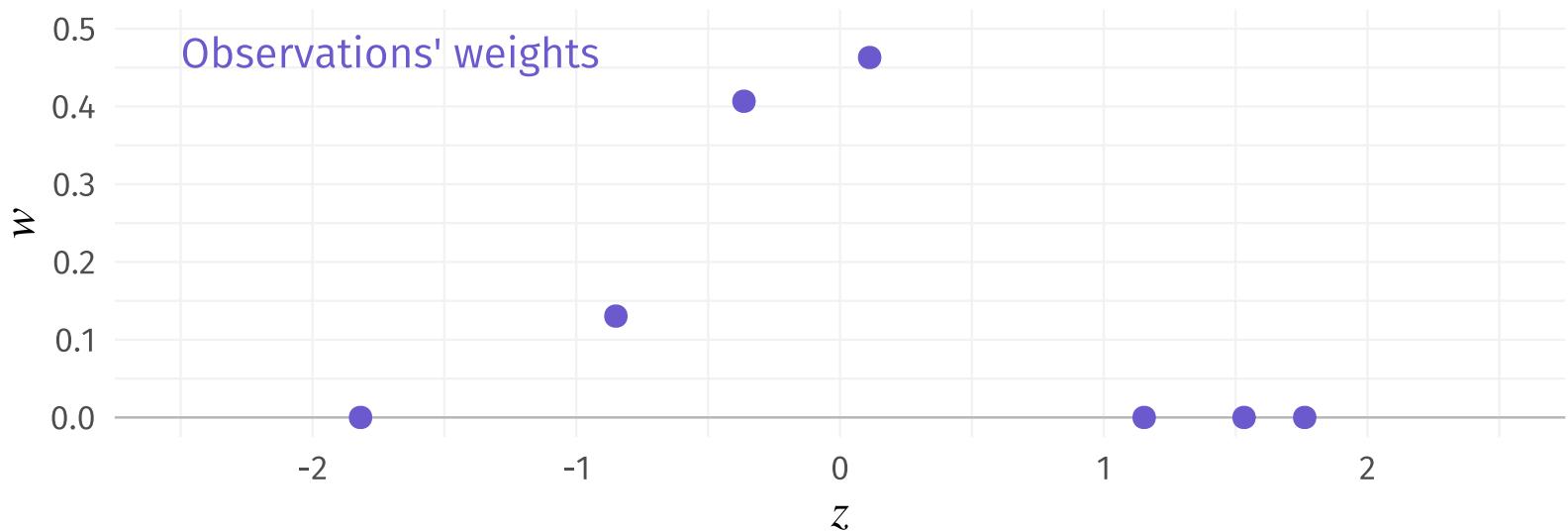
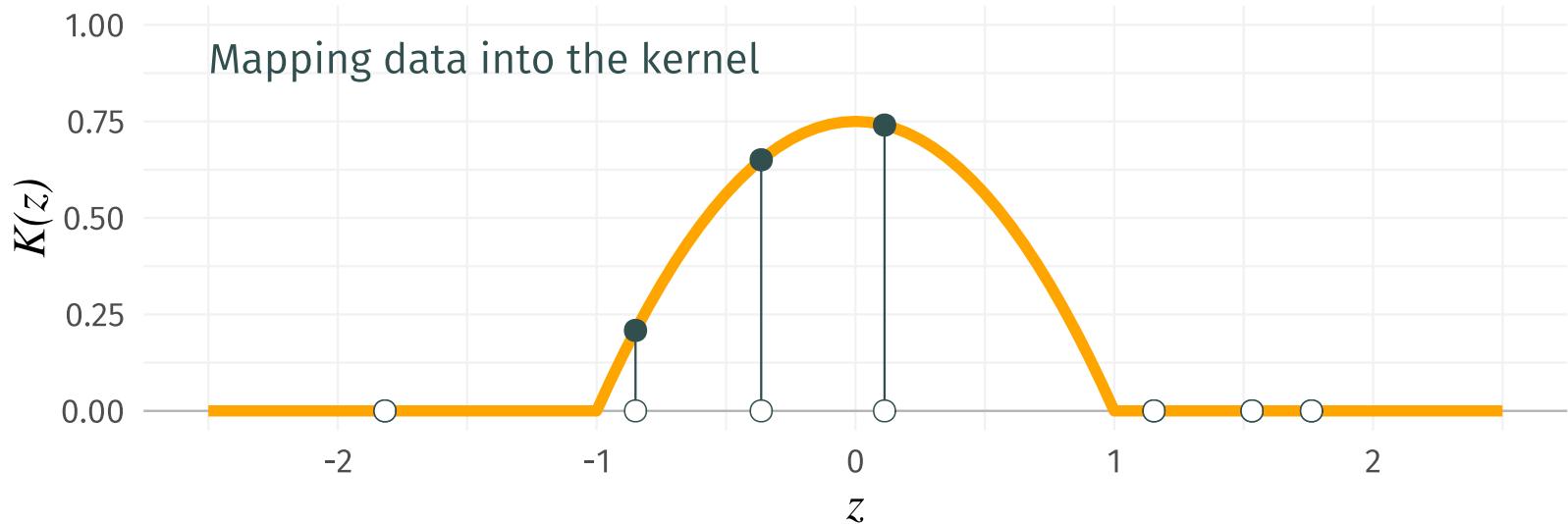
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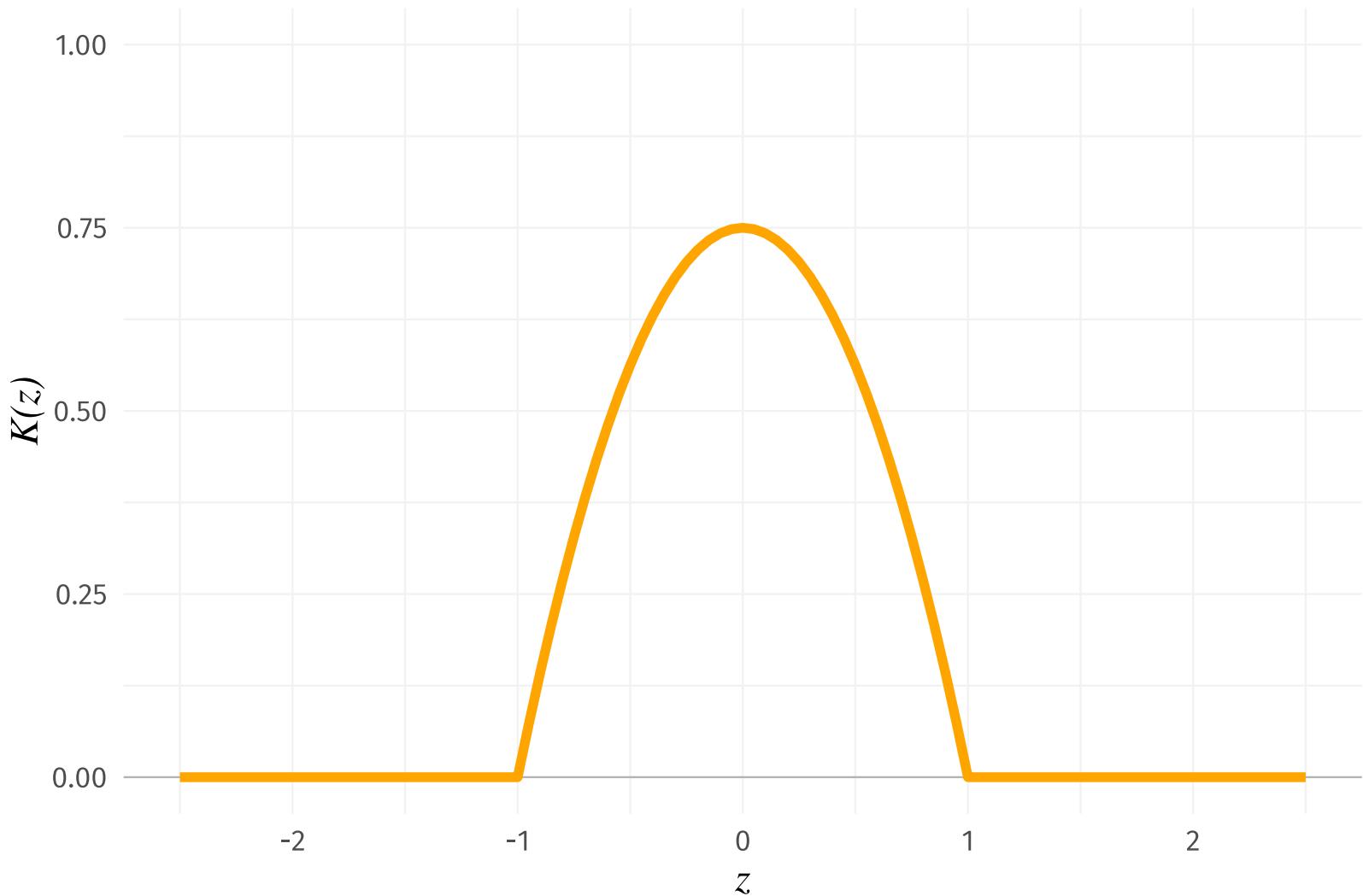


Mapping data into the kernel

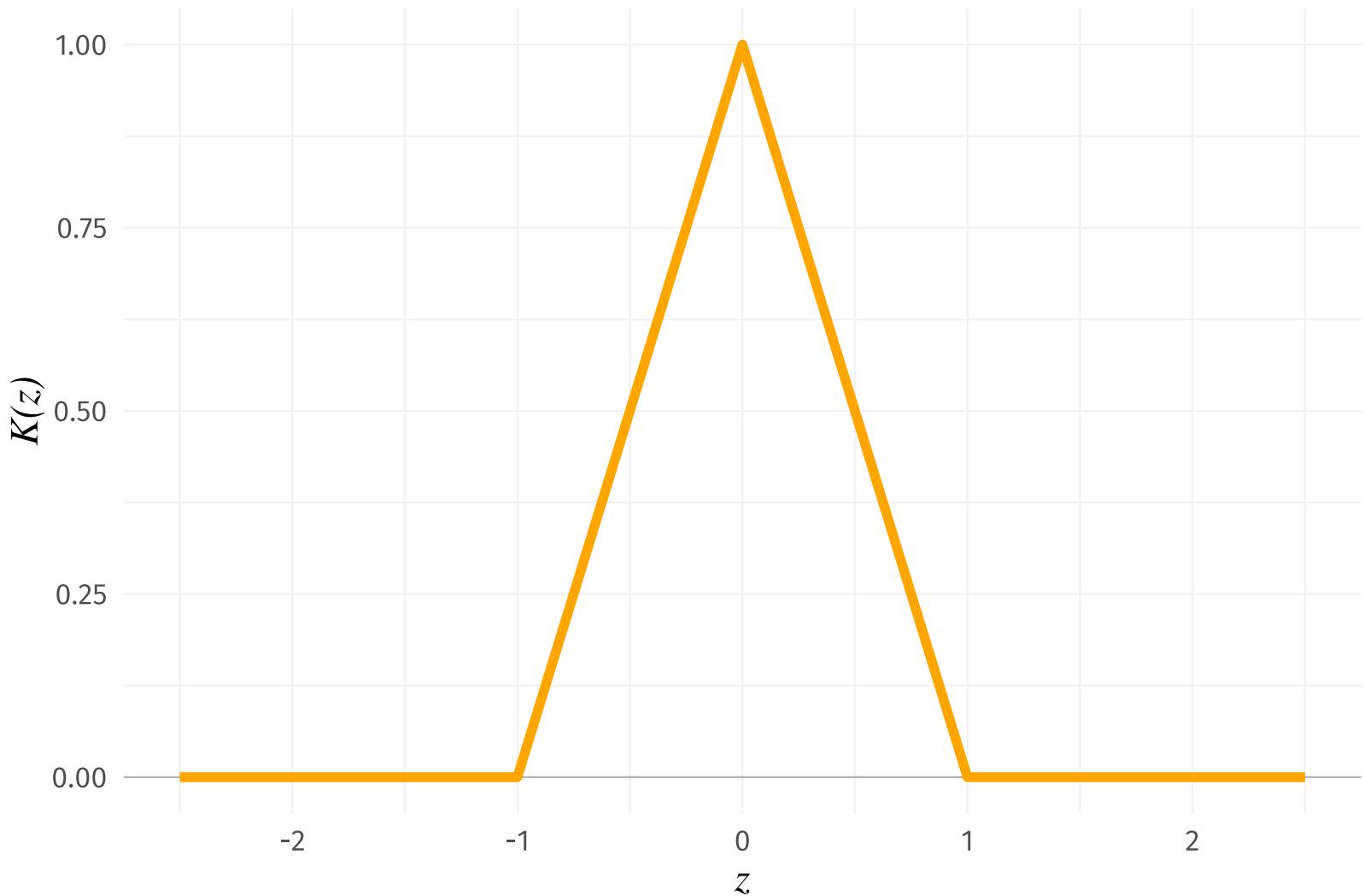




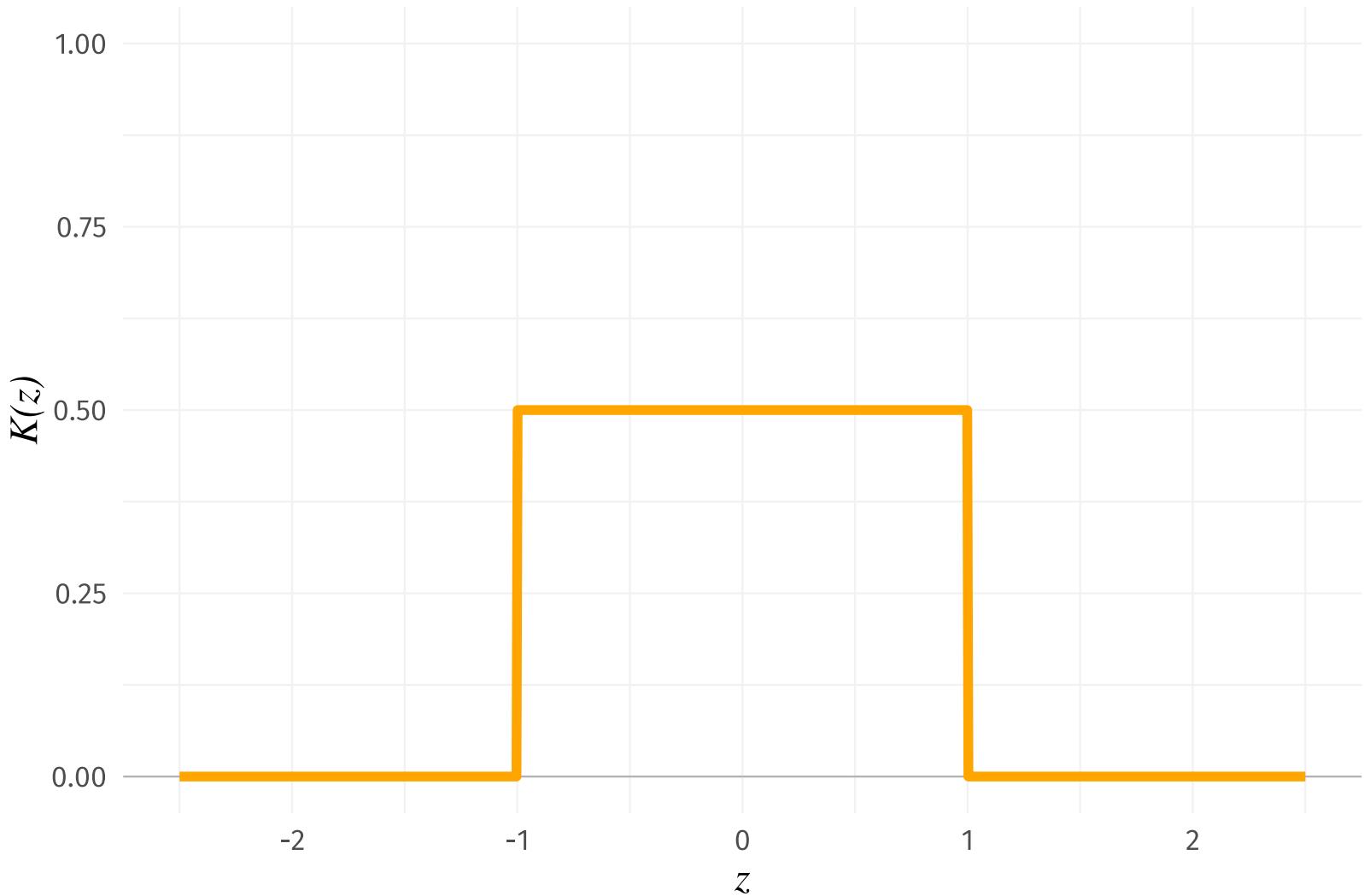
The Epanechnikov kernel $K(z) = \frac{3}{4}(1 - z^2) \times \mathbb{I}(|z| < 1)$



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



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



The Gaussian kernel $K(z) = (2\pi)^{-1/2} \exp(-z^2/2)$



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.

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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").

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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 bandwidth-choosing function called `bw.nrd0()`.

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Adding neighbors

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

Matching

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

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We need a way to shrink the dimensionality of \mathbf{X} .

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Propensity-score methods

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Setup

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

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

Propensity-score methods

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

Propensity scores provide a solution to this mess.

Propensity-score methods

The magic

It turns out that if $(Y_{0i}, Y_{1i}) \perp\!\!\!\perp D_i | X_i$, then we actually only need to match/condition on $p(X_i) = E[D_i | X_i]$.

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

Propensity-score methods

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

Proof

Propensity-score methods

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

Proof

To prove this theorem, we will show $\Pr(D_i = 1 | Y_{0i}, Y_{1i}, p(X_i)) = p(X_i)$,
i.e., D_i is independent of (Y_{0i}, Y_{1i}) after conditioning on $p(X_i)$.

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$$\Pr[D_i = 1 | Y_{0i}, Y_{1i}, p(X_i)]$$

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$$\Pr[D_i = 1 | Y_{0i}, Y_{1i}, p(X_i)]$$

$$= E[D_i | Y_{0i}, Y_{1i}, p(X_i)]$$

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$$\begin{aligned} & \Pr[D_i = 1 | Y_{0i}, Y_{1i}, p(X_i)] \\ &= E[D_i | Y_{0i}, Y_{1i}, p(X_i)] \\ &= E\left[E\left(D_i | Y_{0i}, Y_{1i}, p(X_i), X_i\right) | Y_{0i}, Y_{1i}, p(X_i)\right] \end{aligned}$$

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

Proof

$$\begin{aligned} \Pr[D_i = 1 | Y_{0i}, Y_{1i}, p(X_i)] &= \dots = E \left[E \left(D_i | Y_{0i}, Y_{1i}, X_i \right) | Y_{0i}, Y_{1i}, p(X_i) \right] \\ &= E \left[E \left(D_i | X_i \right) | Y_{0i}, Y_{1i}, p(X_i) \right] \\ &= E \left[p(X_i) | Y_{0i}, Y_{1i}, p(X_i) \right] \\ &= p(X_i) \\ \therefore (Y_{0i}, Y_{1i}) \perp\!\!\!\perp D_i | X_i &\implies (Y_{0i}, Y_{1i}) \perp\!\!\!\perp D_i | p(X_i) \quad \checkmark \end{aligned}$$

Propensity-score methods

Intuition

Q What's going on here?

\mathbf{X}_i carries way more information than $p(\mathbf{X}_i)$, so how can we still get conditional independence of treatment by only conditioning on $p(\mathbf{X}_i)$?

Propensity-score methods

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A₁ Conditional independence of treatment isn't about extracting all of the information possible from \mathbf{X}_i . We actually only care about creating a situation in which $\mathbf{D}_i | \text{something}$ is independent of $(\mathbf{Y}_{0i}, \mathbf{Y}_{1i})$.

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A₂ Back to our main concern: **selection bias**. People select into treatment. If \mathbf{X} says two people were equally likely to be treated, and if \mathbf{X}_i explains all of selection (CIA), then there cannot be selection between these two people.

Propensity-score methods

Estimation

So where do propensity scores come from?

Propensity-score methods

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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.*

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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 \mathbf{X}_i .

Propensity-score methods

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...

Propensity-score methods

Application

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

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Option 1 Conditioning via regression

Propensity-score methods

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Option 1 Conditioning via regression

Option 1a Use a **regression to condition** on $p(\mathbf{X}_i)$, i.e.,

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

Propensity-score methods

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

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

Propensity-score methods

Heterogeneity with regression

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

$$\begin{aligned} Y_{0i} &= \alpha + \beta X_i + u_i \\ Y_{1i} &= Y_{0i} + \delta_1 + \delta_2 X_i \end{aligned}$$

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

Propensity-score methods

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

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$$\begin{aligned} Y_i &= D_i Y_{1i} + (1 - D_i) Y_{0i} \\ &= D_i \left(Y_{0i} + \delta_1 + \delta_2 X_i \right) + (1 - D_i) Y_{0i} \end{aligned}$$

Propensity-score methods

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Propensity-score methods

Heterogeneity

This final equation

$$Y_i = \alpha + \delta_1 D_i + \delta_2 D_i X_i + \beta X_i + u_i$$

Propensity-score methods

Heterogeneity

This final equation

$$Y_i = \alpha + \delta_1 D_i + \delta_2 D_i X_i + \beta X_i + u_i$$

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

$$Y_i = \alpha + \delta_1 D_i + \delta_2 D_i p(X_i) + \beta p(X_i) + u_i \quad (1b)$$

Propensity-score methods

Heterogeneity

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

$$Y_i = \alpha + \delta_1 D_i + \delta_2 D_i p(X_i) + \beta p(X_i) + u_i \quad (1b)$$

which yields

1. a **group-specific treatment effect** $\delta_1 + \delta_2 X_i$ for each X_i
2. an **average treatment effect** $\delta_1 + \delta_2 \bar{p}(X_i)$

Propensity-score methods

More flexibility

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

Adding $p(\mathbf{X}_i)$ and $\mathbf{D}_i p(\mathbf{X}_i)$ as covariates in a linear regression doesn't quite exhaust our potential for flexible/nonparametric estimation.

Propensity-score methods

Blocking

Option 2 Block (stratify) on propensity scores.

Propensity-score methods

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.*,

$$\hat{\tau}_{\text{Block}} = \sum_{k=1}^K \hat{\tau}_k \frac{N_{1k} + N_{0k}}{N}$$

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Note Blocking is similar to NN/kernel matching using $p(\mathbf{X}_i)$ as distance.

Propensity-score methods

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.

Propensity-score methods

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 $\hat{\tau}_k$.

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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 $\hat{p}(X_i)$ above the maximum propensity score in the control group.

Propensity-score methods

Weighting

Option 3 Weight observations by the inverse propensity score.

Propensity-score methods

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Propensity-score methods

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

$$\hat{\tau}_{\text{Diff}} = \bar{Y}_T - \bar{Y}_C = \frac{\sum_i D_i Y_i}{\sum_i D_i} - \frac{\sum_i (1 - D_i) Y_i}{\sum_i (1 - D_i)}$$

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

$$E[Y_{0i}|D_i = 1] \neq E[Y_{0i}]$$

Propensity-score methods

Weighting, justified

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

Propensity-score methods

Weighting, justified

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

$$E\left[\frac{D_i Y_i}{p(\mathbf{X}_i)}\right]$$

Propensity-score methods

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

$$E\left[\frac{D_i Y_i}{p(\mathbf{X}_i)}\right] = E\left[\frac{D_i (D_i Y_{1i} + (1 - D_i) Y_{0i})}{p(\mathbf{X}_i)}\right]$$

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

$$E\left[\frac{(1 - D_i) Y_i}{1 - p(\mathbf{X}_i)}\right] = \textcolor{blue}{E[Y_{0i}]}$$

Propensity-score methods

Weighting: The estimator

Thus, we can estimate an unbiased treatment effect via

$$\hat{\tau}_{p\text{Weight}} = \frac{1}{N} \sum_{i=1}^N \left[\frac{D_i Y_i}{p(X_i)} - \frac{(1 - D_i Y_i)}{1 - p(X_i)} \right]$$

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

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

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

So we upweight (1) **treated** individuals with low $p(\mathbf{X}_i)$ and (2) **control** observations with high $p(\mathbf{X}_i)$.

Propensity-score methods

Weighting: The example

Suppose for some individual i , $p(\mathbf{X}_i) = 0.80$.

Propensity-score methods

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Suppose for some individual i , $p(\mathbf{X}_i) = 0.80$.

This propensity score says someone with this set of \mathbf{X}_i was four-times more likely to be **treated** than **control**.

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

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And guess what $5/1.25$ is...

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And guess what $5/1.25$ is... 4! This weighting scheme gets us back to equal representation for each set of \mathbf{X}_i .

Propensity-score methods

Weighting: Last issue

Practical issue Nothing guarantees $\sum_i \hat{p}(X_i) = 1$.

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Solution Normalize weights by their total sum.

Propensity-score methods

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Applying the normalized (and estimated) propensity scores

$$\hat{\tau}_{p\text{Weight}} = \sum_{i=1}^N \frac{\frac{D_i Y_i}{\hat{p}(\mathbf{X}_i)}}{\sum_i \frac{D_i}{\hat{p}(\mathbf{X}_i)}} - \sum_{i=1}^N \frac{\frac{(1 - D_i) Y_i}{1 - \hat{p}(\mathbf{X}_i)}}{\sum_i \frac{(1 - D_i)}{1 - \hat{p}(\mathbf{X}_i)}}$$

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

Propensity-score methods

Why choose one?

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

Thus, a **regression-based estimate**

$$Y_i = \alpha + X_i\beta + \tau D_i + u_i$$

Propensity-score methods

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

Thus, a **regression-based estimate**

$$Y_i = \alpha + X_i\beta + \tau D_i + u_i$$

with **weights**

$$w_i = \sqrt{\frac{D_i}{\hat{p}(X_i)} + \frac{(1 - D_i)}{1 - \hat{p}(X_i)}}$$

Propensity-score methods

Why choose one?

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

Propensity-score methods

Why choose one? Part two

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

Propensity-score methods

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Step 1 For each block k , we run the regression

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Propensity-score methods

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

$$Y_i = \alpha_k + X_i\beta_k + \tau_k D_i + u_i$$

Step 2 Aggregate block-level treatment-effect estimates

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

Propensity-score methods

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?

Propensity-score methods

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Propensity-score methods

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1. Is the **conditional-independence assumption** true?
2. Do we have **overlap** between treatment and control units.

Propensity-score methods

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?

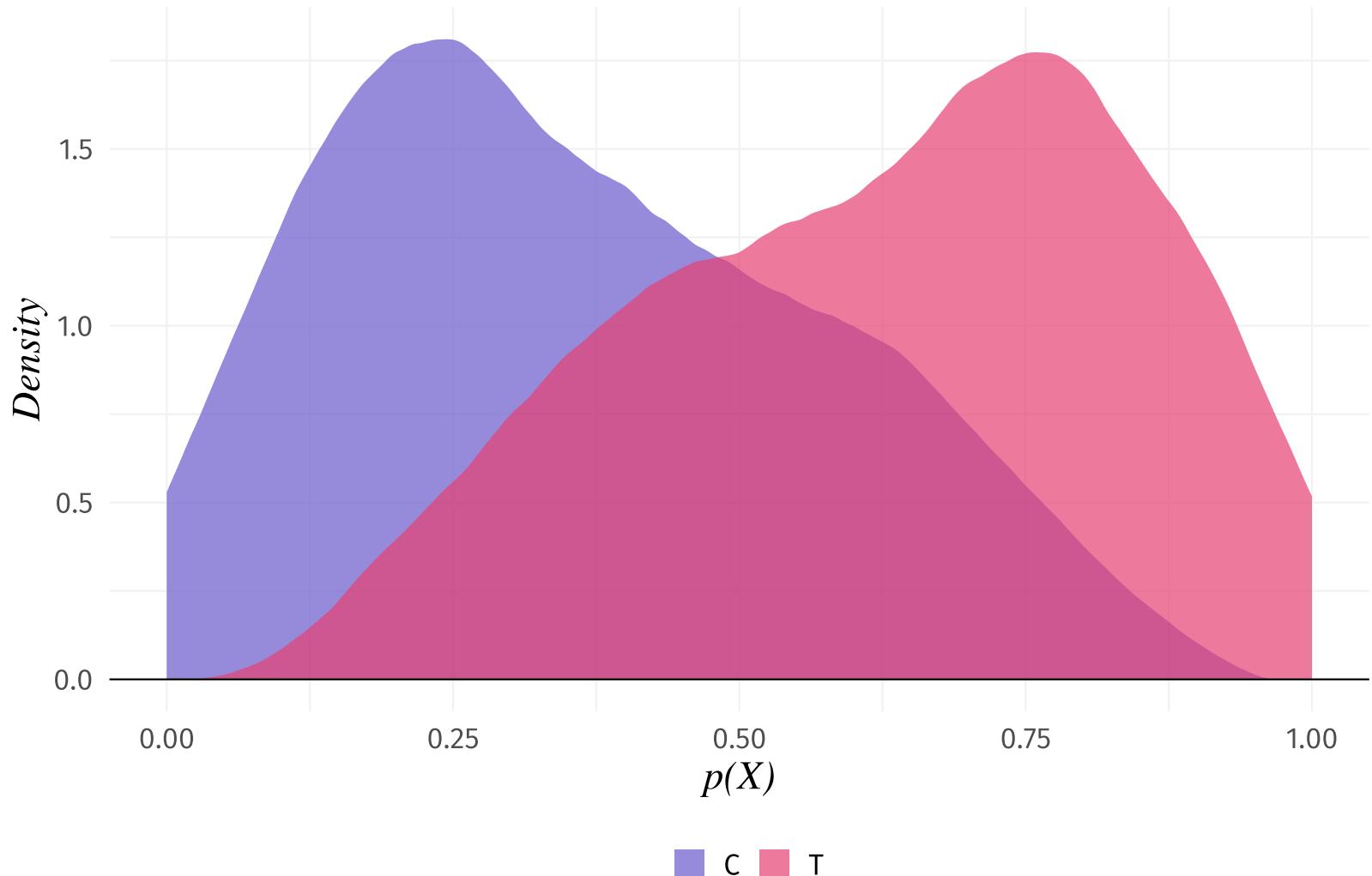
1. Is the **conditional-independence assumption** true?
2. Do we have **overlap** between treatment and control units.

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

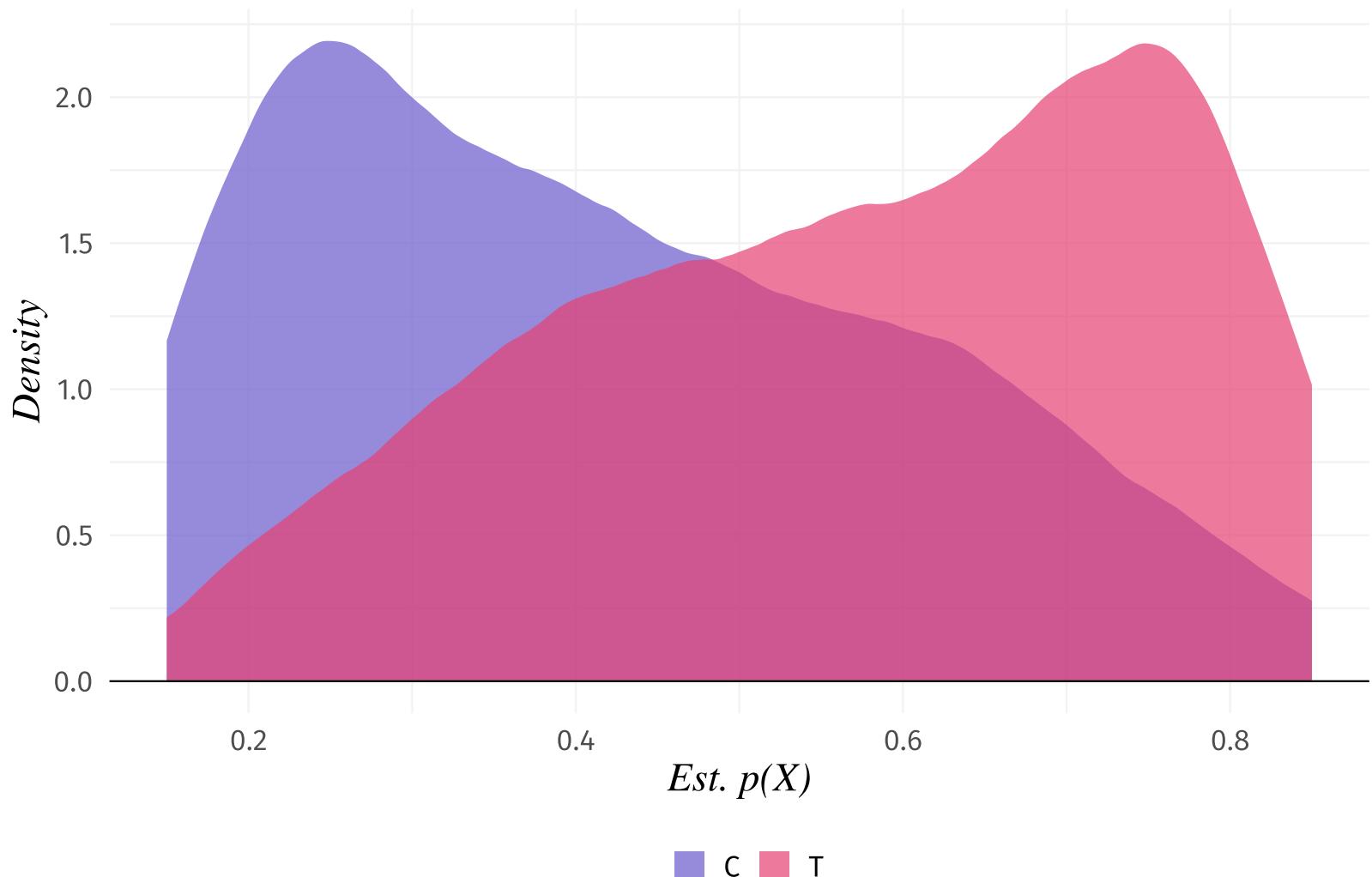
How? Plot the distributions of $p(\mathbf{X}_i)$ for **T** and **C**.

[†] Checking for overlap in \mathbf{X} -space, can be tough as the dimensions of \mathbf{X} expand.

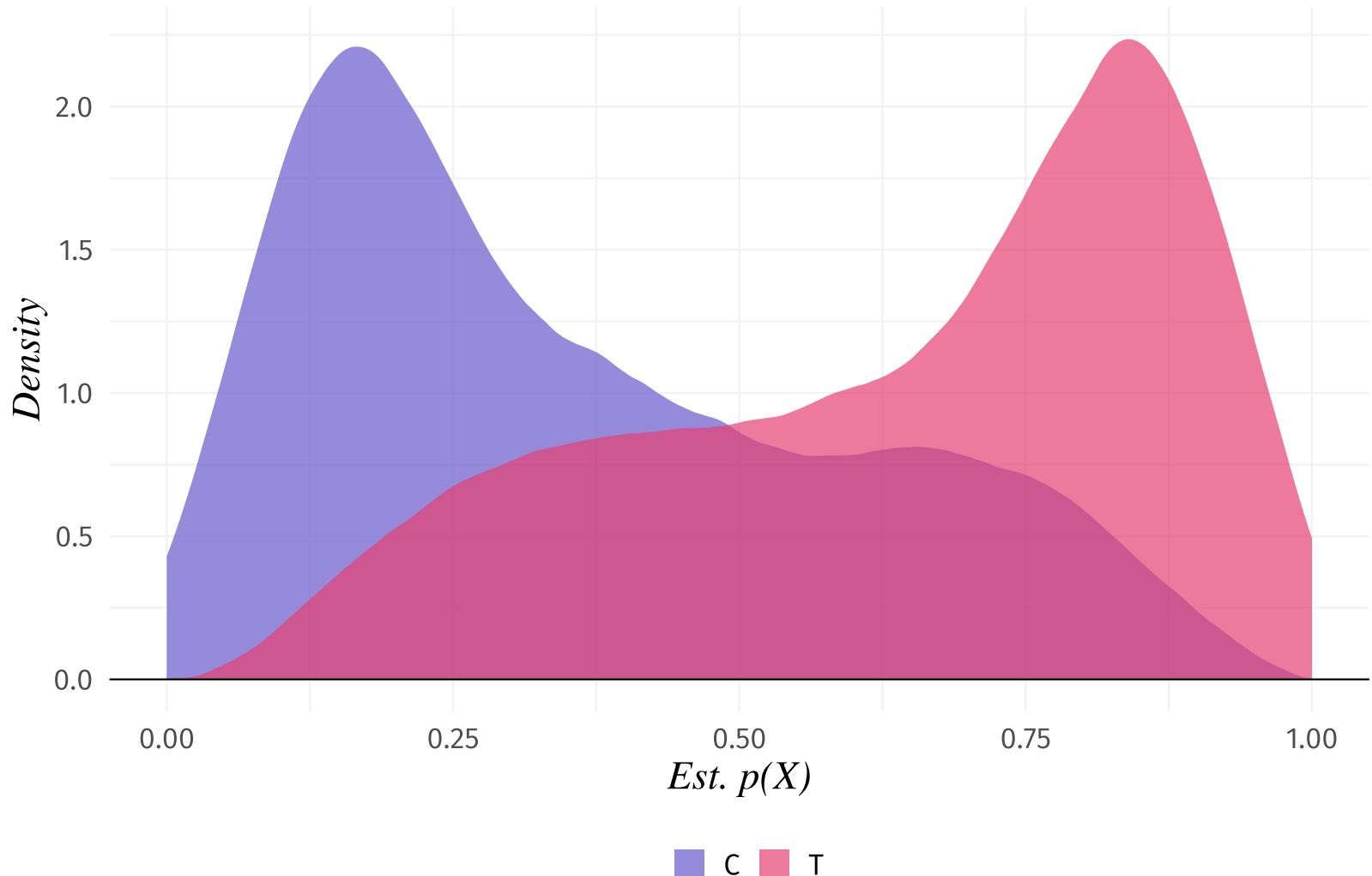
Missing overlap in $p(\mathbf{X}_i)$



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



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



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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