

Tutorial Week 8 and 9: Matching and Entropy Balancing

Problem Set - Propensity Scores, Matching, and Synthetic Control

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1 Load Packages

```
library(tidyverse)
library(haven)
library(MatchIt)
library(cobalt)
library(randomForest)
library(lmtest)
```

```

library(sandwich)
library(knitr)
library(kableExtra)
library(broom)
library(Synth)
library(stargazer)

```

2 Part I: Propensity Scores, Matching, and Robust Post-Matching Inference

2.1 Background

Research Question: Do United Nations interventions help shorten the duration of civil wars?

Gilligan and Sergenti (2008) use matching methods to re-evaluate earlier findings suggesting that UN interventions *prolong* conflict. They argue that this conclusion stems from **selection bias** — the UN tends to intervene in the *worst* conflicts.

Dataset: war_pre_snapshots.dta

Each row represents a **conflict episode** observed before a potential UN intervention. Our goal is to estimate the causal effect of UN involvement (UN) on the length of the conflict ($t_1 - t_0$), while balancing on key pre-treatment covariates.

2.2 Load Data

```

# Read the UN intervention dataset
data_un <- read_dta("war_pre_snapshots.dta")

# Display structure and summary
#glimpse(data_un)
#summary(data_un)

```

2.3 Define Treatment and Covariates

```

# Treatment variable: UN intervention (1 = Yes, 0 = No)
treat <- data_un$UN

# Outcome variable: conflict duration (t1 - t0)
outcome <- data_un$t - data_un$t0

# Covariates for propensity score model
covar <- c(
  "inter", "deaths", "couperv", "sos", "drugs", "t0",
  "ethfrac", "pop", "lmtnest", "milper",
  "eeurop", "lamerica", "asia", "ssafrika"
)

# Create covariate matrix
X <- data_un[, covar]

dta_new <- data.frame(
  treat = treat,

```

```
outcome = outcome,  
X)
```

2.4 Q0. First Check of the Data

2.4.1 Tasks:

1. Why might UN interventions **not** be randomly assigned across conflicts?
2. Which of the listed variables are most likely to confound the relationship between UN and conflict duration? Run a quick logistic regression and check.

```
# Logistic regression: UN intervention as function of covariates  
logit_selection <- glm(treat ~ inter + deaths + couprev + sos + drugs + t0 +  
                         ethfrac + pop + lmtnest + milper +  
                         eeurop + lamERICA + asia + ssafrica,  
                         data = dta_new,  
                         family = binomial(link = "logit"))  
  
# Create stargazer table  
stargazer(  
  logit_selection,  
  type = "latex",  
  title = "Selection into UN Intervention: Logistic Regression",  
  header = FALSE,  
  dep.var.labels = "UN Intervention (1 = Yes)",  
  covariate.labels = c(  
    "Internationalized Conflict",  
    "Battle Deaths",  
    "Coup/Revolution",  
    "Strategic Oil Supply",  
    "Drug Activity",  
    "Conflict Start Year",  
    "Ethnic Fractionalization",  
    "Log Population",  
    "Log Mountainous Terrain",  
    "Military Personnel per Capita",  
    "Eastern Europe",  
    "Latin America",  
    "Asia",  
    "Sub-Saharan Africa",  
    "Constant"  
,  
  no.space = TRUE,  
  omit.stat = c("aic", "ll"),  
  notes = "Standard errors in parentheses.",  
  notes.align = "l",  
  digits = 3  
)
```

Discussion:

Why might UN interventions not be randomly assigned?

Cases where the UN intervenes are quite different from where they do not, which is why UN missions are

Table 1: Selection into UN Intervention: Logistic Regression

| | <i>Dependent variable:</i> |
|-------------------------------|----------------------------|
| | UN Intervention (1 = Yes) |
| Internationalized Conflict | 0.929 (0.846) |
| Battle Deaths | −0.156 (0.218) |
| Coup/Revolution | −0.113 (1.234) |
| Strategic Oil Supply | −1.075 (0.994) |
| Drug Activity | 3.238*** (1.087) |
| Conflict Start Year | 0.005 (0.003) |
| Ethnic Fractionalization | −0.016 (0.017) |
| Log Population | −0.271 (0.471) |
| Log Mountainous Terrain | 0.271 (0.321) |
| Military Personnel per Capita | −1.123*** (0.351) |
| Eastern Europe | 2.186 (1.485) |
| Latin America | −3.775** (1.731) |
| Asia | −2.440 (1.630) |
| Sub-Saharan Africa | −1.337 (1.521) |
| Constant | 3.002 (4.006) |
| Observations | 1,227 |

Note:

*p<0.1; **p<0.05; ***p<0.01
Standard errors in parentheses.

not randomly assigned.

The decision to intervene depends on a number of factors, including the UN Security Council's selection process and whether the UN even pays attention to these cases.

Because the “treated” (intervention) and “untreated” (no intervention) cases are so different on so many variables, it becomes almost impossible to distinguish whether a causal effect is due to the treatment itself or some function of these other confounding variables. When you have differences that large between units, you face an extreme problem of constructing the counterfactual—that is, figuring out what would have happened had the UN not intervened.

Which variables show strong associations with UN intervention?

Looking at the results in table 1, we can see a high positive coefficient for **Drug Activity** ($\text{coef}=3.238$, $p<0.01$), meaning that the UN is more likely to intervene where there is drug-related activity present. This also makes intuitive sense, as interventions are fostered by international drug trafficking concerns. This is a prime example of why it is hard to compare regimes across regions that could potentially receive UN intervention. Since regimes, rebel groups, or terrorist groups in politically unstable systems often build their economic foundation on scarce resources (e.g., diamonds, oil, etc.) and also drugs, their different capabilities (e.g., geographic location, natural resources) offer different incentives for the UN to intervene, especially when their economic activities pose a global threat, as is the case with drugs.

Similarly, in countries with high per capita **military personnel** ($\text{coef}=-1.123$, $p<0.01$), countries with higher military capacity are less likely to receive UN intervention. This also makes intuitive sense, as weak military states would make it easier and safer to intervene, and they have more need for external support.

Furthermore, countries located in **Latin America** are less likely to receive UN intervention ($\text{coef}=-3.775$, $P<0.05$). This finding might be slightly confusing at first glance, although it might be explained by a couple of points. As many states in Latin America have faced high levels of political instability and also have a long history with drug trafficking, we might assume that they would be more likely to receive UN interventions. The absence of this finding might be due to the fact that Latin America has always been undisputedly located in the sphere of influence of the United States—historically articulated in the Monroe Doctrine (1823)—which has carried out its own “peacekeeping missions” across the continent, not based on decisions of the UN or other multilateral actors after the Cold War. Looking at recent developments around the coast of Venezuela, this dynamic still dominates today.

Overall, this regression table gives some preliminary hints that UN interventions are indeed not randomly assigned. It demonstrates clear selection bias: the UN systematically intervenes in cases with specific, observable characteristics (like high drug activity or weak militaries) and avoids others (like those in Latin America). While other factors like battle deaths or ethnic fractionalization don't show a significant effect in this model, the strong predictors identified here are exactly why it is extremely useful to use matching to create a valid comparison group.

2.5 Q1. Estimating Propensity Scores

2.5.1 Theoretical Background

Let $T_i \in \{0, 1\}$ be the treatment indicator and $X_i = (X_{i1}, X_{i2}, \dots, X_{ip})$ the vector of pre-treatment covariates.

The **propensity score** is defined as:

$$e(X_i) = P(T_i = 1 | X_i)$$

A **propensity score** is the probability that a unit with certain characteristics will be assigned to the treatment group. It ranges between 0 and 1.

2.5.2 Tasks:

1. Define the propensity score
2. Estimate $\hat{e}(X_i)$ in two ways:
 - (a) Logistic regression: $\text{logit}(e(X_i)) = X'_i \beta$
 - (b) Random forest classifier
3. Report mean, SD, and range of $\hat{e}(X_i)$ for treated and control
4. Create histogram/density plot by treatment status

```
# (a) Logistic regression propensity score
```

```
ps_logistic <- glm(treat ~ inter + deaths + couprev + sos + drugs + t0 +
                     ethfrac + pop + lmtnest + milper +
                     eeurop + lamerica + asia + ssafrica,
                     data = dta_new,
                     family = binomial(link = "logit"),
                     na.action = na.exclude)
```

```
dta_new$ps_logistic_probs <- predict(ps_logistic,
                                         newdata = dta_new,
                                         type = "response")
```

```
# (b) Random forest propensity score
```

```
ps_random_forest <- randomForest(
  factor(treat) ~ inter + deaths + couprev + sos + drugs + t0 +
  ethfrac + pop + lmtnest + milper +
  eeurop + lamerica + asia + ssafrica,
  data = dta_new,
  ntree = 1000,
  importance = TRUE
)
```

```
# Extract predicted probabilities
```

```
dta_new$ps_random_forest <- predict(ps_random_forest, type = "prob")[, "1"]
```

```
# Summary statistics of propensity scores
```

```
# Logistic Regression Stats
logit_control_mean <- mean(dta_new$ps_logistic_probs[dta_new$treat == 0], na.rm = TRUE)
logit_control_sd <- sd(dta_new$ps_logistic_probs[dta_new$treat == 0], na.rm = TRUE)
logit_control_min <- min(dta_new$ps_logistic_probs[dta_new$treat == 0], na.rm = TRUE)
logit_control_max <- max(dta_new$ps_logistic_probs[dta_new$treat == 0], na.rm = TRUE)
```

```
logit_treated_mean <- mean(dta_new$ps_logistic_probs[dta_new$treat == 1], na.rm = TRUE)
logit_treated_sd <- sd(dta_new$ps_logistic_probs[dta_new$treat == 1], na.rm = TRUE)
logit_treated_min <- min(dta_new$ps_logistic_probs[dta_new$treat == 1], na.rm = TRUE)
logit_treated_max <- max(dta_new$ps_logistic_probs[dta_new$treat == 1], na.rm = TRUE)
```

```
# Random Forest Stats
```

```
rf_control_mean <- mean(dta_new$ps_random_forest[dta_new$treat == 0], na.rm = TRUE)
rf_control_sd <- sd(dta_new$ps_random_forest[dta_new$treat == 0], na.rm = TRUE)
rf_control_min <- min(dta_new$ps_random_forest[dta_new$treat == 0], na.rm = TRUE)
```

```

rf_control_max <- max(dta_new$ps_random_forest[dta_new$treat == 0], na.rm = TRUE)

rf_treated_mean <- mean(dta_new$ps_random_forest[dta_new$treat == 1], na.rm = TRUE)
rf_treated_sd <- sd(dta_new$ps_random_forest[dta_new$treat == 1], na.rm = TRUE)
rf_treated_min <- min(dta_new$ps_random_forest[dta_new$treat == 1], na.rm = TRUE)
rf_treated_max <- max(dta_new$ps_random_forest[dta_new$treat == 1], na.rm = TRUE)

# Data Frame with results
summary_stats_q1 <- data.frame(
  Statistic = c("Mean", "Std. Deviation", "Min", "Max"),
  Logit_Control = c(logit_control_mean, logit_control_sd, logit_control_min, logit_control_max),
  Logit_Treated = c(logit_treated_mean, logit_treated_sd, logit_treated_min, logit_treated_max),
  RF_Control = c(rf_control_mean, rf_control_sd, rf_control_min, rf_control_max),
  RF_Treated = c(rf_treated_mean, rf_treated_sd, rf_treated_min, rf_treated_max)
)

kable(summary_stats_q1,
      digits = 3,
      col.names = c("Statistic", "Control (T=0)", "Treated (T=1)", "Control (T=0)", "Treated (T=1)"),
      caption = "Table 1: Propensity Score Distribution by Model and Treatment Status"
) %>%
  add_header_above(c(" " = 1, "Logistic Regression" = 2, "Random Forest" = 2)) %>%
  kable_styling(bootstrap_options = "striped", full_width = FALSE)

```

Table 2: Table 1: Propensity Score Distribution by Model and Treatment Status

| Statistic | Logistic Regression | | Random Forest | |
|----------------|---------------------|---------------|---------------|---------------|
| | Control (T=0) | Treated (T=1) | Control (T=0) | Treated (T=1) |
| Mean | 0.010 | 0.213 | 0.002 | 0.233 |
| Std. Deviation | 0.026 | 0.253 | 0.010 | 0.162 |
| Min | 0.000 | 0.000 | 0.000 | 0.000 |
| Max | 0.194 | 0.884 | 0.190 | 0.521 |

```
# Density plot of propensity scores by treatment status
```

Interpretation:

[Discuss the distribution of propensity scores. Are there regions of poor overlap?]

2.6 Q2. Implement 1:1 Nearest-Neighbor Matching

2.6.1 Matching Setup

For each estimated propensity score $\hat{e}(X_i)$, match each treated unit to the nearest control on the **logit of the propensity score**:

$$\ell_i = \log \left(\frac{\hat{e}(X_i)}{1 - \hat{e}(X_i)} \right)$$

Use **replacement** and a **caliper** of $0.2 \times \text{SD}(\ell_i)$ to restrict poor matches.

2.6.2 Tasks:

1. Implement matching using both logit and RF propensity scores
2. Report how many treated units fail to find a match
3. How does this change the estimand?

```
# Matching using logit propensity scores
```

```
# Number of matched treated units
```

```
# Matching using RF propensity scores
```

```
# Number of matched treated units
```

Discussion:

[How many treated units were dropped? What does this mean for the target estimand (ATT)?]

2.7 Q3. Standardized Mean Differences (SMDs)

2.7.1 SMD Formulas

For each covariate X^k :

Before matching (ATT version):

$$\text{SMD}_{\text{raw}}(k) = \frac{\bar{X}_{T=1}^k - \bar{X}_{T=0}^k}{\sqrt{s_{T=1}^{2,k}}}$$

After matching:

$$\text{SMD}_{\text{match}}(k) = \frac{\bar{X}_{\text{match}}^{k,\text{treated}} - \bar{X}_{\text{match}}^{k,\text{control}}}{\sqrt{s_{T=1}^{2,k}}}$$

2.7.2 Tasks:

1. Compute SMDs before and after matching for all covariates
2. Create a Love plot showing balance before and after matching (both methods)
3. Add vertical line at 0.1 (acceptable threshold)
4. Comment on which design achieves better covariate balance
5. Create two additional Love plots including interactions and squared terms

```
# Calculate SMDs before matching
```

```
# Calculate SMDs after matching (logit)
```

```
# Calculate SMDs after matching (RF)
```

```
# Love plot: Base covariates
```

```
# Love plot: Including interactions  
# Love plot: Including squared terms
```

Interpretation:

[Which method achieves better balance? Are all SMDs below 0.1?]

2.8 Q4. Overlap

2.8.1 Tasks:

1. For each method, report:
 - Min and max of $\hat{e}(X_i)$ for treated and controls
 - Proportion of treated units whose $\hat{e}(X_i)$ lies inside the support of controls (and vice versa)
2. Plot distributions of $\hat{e}(X_i)$ for treated and controls
3. Identify regions of poor overlap or extreme propensities
4. (Optional) Trim observations outside common support and re-compute ATT
5. Examine matched subsets - do matches seem like fair counterfactuals?

```
# Min/max propensity scores by treatment group  
  
# Proportion in common support  
  
# Plot propensity score distributions  
  
# Optional: Trim and re-estimate
```

Discussion:

[Is there good overlap? Which observations are on the edge of common support?]

2.9 Q5. Matched-Pair ATT

2.9.1 ATT Estimator

Let each matched pair be denoted by $(i, j(i))$ where i is treated and $j(i)$ is its matched control.

The **average treatment effect on the treated** is:

$$\hat{\tau}_{ATT} = \frac{1}{N_T^*} \sum_{i \in \mathcal{T}^*} (Y_i - Y_{j(i)})$$

where \mathcal{T}^* is the set of treated units with a valid match.

2.9.2 Task:

Compute the ATT for both matching methods.

```
# ATT using logit matching  
  
# ATT using RF matching
```

Interpretation:

[What is the estimated effect of UN intervention on conflict duration?]

2.10 Q5.5. Bias–Variance Tradeoff in Matching Ratios

2.10.1 (a) Conceptual Question

For 1-to- m nearest-neighbor matching without replacement, the ATT estimator is:

$$\hat{\tau}_{\text{ATT}}^{(m)} = \frac{1}{N_T^*} \sum_{i \in \mathcal{T}^*} \left(Y_i - \frac{1}{m} \sum_{j \in \mathcal{J}(i)} Y_j \right)$$

where $\mathcal{J}(i)$ is the set of the m closest control matches for treated unit i .

Tasks:

1. Explain why increasing m tends to:
 - Decrease variance
 - Increase bias
2. Discuss how this relates to distance in covariate space
3. If overlap is weak, which risk dominates as m grows?

Discussion:

[Your explanation of the bias-variance tradeoff here]

2.10.2 (b) Practical Exercise

Tasks:

1. Re-run matching for 1:1, 2:1, and 3:1 ratios (with replacement and same caliper)
2. Record: number matched, mean distance, ATT estimate
3. Compute cluster-robust standard errors for each design
4. Create results table
5. Plot ATT vs. m with ± 1.96 SE error bars

```
# 1:1 matching
```

```
# 2:1 matching
```

```
# 3:1 matching
```

```
# Create comparison table
```

```
# Plot ATT by matching ratio with error bars
```

Interpretation:

[Do results display expected bias-variance pattern?]

2.10.3 (c) Discussion

Tasks:

- Which design (1:1, 2:1, or 3:1) is most appropriate?
- How does observed pattern relate to Abadie & Imbens (2006)?
- What would happen with infinite data and perfect overlap?

Discussion:

[Your analysis here]

2.11 Q6. Robust Post-Matching Inference (Abadie & Spiess, 2021)

2.11.1 Regression with Cluster-Robust Standard Errors

After matching, fit the regression:

$$Y_i = \alpha + \tau T_i + \varepsilon_i$$

using only matched data.

Let $s(i)$ denote the **subclass (pair id)** of observation i .

Compute **cluster-robust standard errors** for $\hat{\tau}$ by clustering on $s(i)$:

$$\widehat{V}_{\text{CR}}(\hat{\tau}) = (X' X)^{-1} \left(\sum_s X_s' \hat{\varepsilon}_s \hat{\varepsilon}_s' X_s \right) (X' X)^{-1}$$

2.11.2 Tasks:

1. Report $\hat{\tau}$ and its cluster-robust standard error
2. Compare results for logit-matched and RF-matched samples

```
# Regression on logit-matched data with cluster-robust SE
```

```
# Regression on RF-matched data with cluster-robust SE
```

```
# Compare results
```

Interpretation:

[Compare point estimates and standard errors across methods]

2.12 Q7. (Optional) Bootstrap Check

2.12.1 Matched-Pair Bootstrap

Warning: Bootstraps are not theoretically valid for matching estimators, but this serves as a check.

Tasks:

1. Resample matched pairs (subclasses) with replacement
2. Recompute $\hat{\tau}^{(b)}$ for each bootstrap sample $b = 1, \dots, B$
3. Report bootstrap mean, SD, and percentile 95% CI
4. Compare to cluster-robust results

```
# Bootstrap procedure
```

Discussion:

[Do bootstrap and cluster-robust results tell a similar story?]

2.13 Q8. Reflection

2.13.1 Tasks:

1. Why does the propensity score $e(X_i)$ act as a **balancing score**?
2. How does random-forest estimation of $e(X_i)$ change matching results compared to logistic regression?
3. Why is overlap ($0 < e(X_i) < 1$) necessary for identifying the ATT?

Discussion:

[Your reflection here]

3 Part II: Synthetic Control - German Reunification Study

3.1 Background

In 1990, West Germany underwent reunification with East Germany. The question: *What was the economic cost (or benefit) of this event on West Germany's GDP per capita?*

Using the synthetic control method, we construct a counterfactual “synthetic West Germany” from a weighted combination of other OECD countries.

Paper: Abadie, Diamond & Hainmueller (2015), *Comparative Politics and the Synthetic Control Method*, AJPS.

Dataset: Available via Harvard Dataverse (doi:10.7910/DVN/24714)

3.2 Load Data

```
# Read German reunification dataset
# Load the replication dataset
load("repgermany.RData")
repgermany <- x

# Display structure
#str(repgermany)
#head(repgermany)
```

3.3 (a) Conceptual Questions

3.3.1 Tasks:

1. Explain the intuition behind the synthetic control method. What kind of assignment problem does it address?
2. Why is it particularly suitable for the West Germany case?
3. What is the key identification assumption?

Discussion:

1.)

The synthetic control method serves as a way to bridge the quantitative/qualitative divide in comparative politics and possibly all of social science research, helping to make precise quantitative inference in small-sample studies. Usually, small sample comparative case studies uncover evidence at a level of high granularity that is impossible to establish in large-scale quantitative studies, which in turn provide precise numerical results that can be compared across studies. For these reasons calls became loud to combine the best of both worlds.

The goal of most study designs is to compare outcomes between treated units, that are the main objects of study, and similar but unaffected control units. The comparison units are intended to reproduce the counterfactual of the treated units in the absence of the treatment. When we try to make quantitative causal inference in small sample comparative studies, we usually do not fail due to the small sample size itself, but due to the missing mechanism of how to find suitable comparison units for the small pool of units we have.

Especially in comparative political science, we often look at aggregate entities, such as states, countries or regions, for which obvious single comparisons often do not exist. In that case we often have one aggregate unit receiving treatment, where we try to select a set of similar untreated units from all of the untreated

cases (“donor pool”) to approximate the counterfactual. Standard designs based on selection on observables try to remove bias by controlling for measurable covariates. This approach fails when unobserved factors confound the result. This is particularly prevalent in comparative political research, as we study large entities as treatment units and control units, being made up of many individuals.

However, the synthetic control method is based on the premise that when the units of analysis are a few aggregate entities, a combination of comparison units (the synthetic control) often does a better job of reproducing the characteristics of the unit or units representing the case of interest than any single comparison unit alone. The comparison unit in the synthetic control method is selected as the weighted average of all potential comparison units. This weighted average is optimized to best reproduce the treated unit’s pre-treatment outcome path and other key predictors. The optimally-weighted average is what serves as the counterfactual, which is custom-built to be the treated unit’s ‘doppelgänger’ before treatment occurred. The core intuition is that by replicating the pre-treatment trend, the synthetic control also replicates the unobserved factors that drove that trend, thus creating a valid counterfactual.

2.)

3.)

3.4 (b) Mathematical/Optimization Questions

3.4.1 The Optimization Problem

The synthetic control method solves:

$$\min_w \sum_{t \leq T_0} \left(Y_{1t} - \sum_{j=2}^{J+1} w_j Y_{jt} \right)^2$$

subject to:

$$w_j \geq 0, \quad \sum_j w_j = 1$$

3.4.2 Tasks:

1. Write and explain each term in the optimization problem
2. What role do w -weights play in predictor balancing?
3. Why is the convex-combination constraint important? What if weights could be negative or sum $\neq 1$?

Mathematical Discussion:

The optimization problem in synthetic control is the problem to find the ideal set of weights to build the most similar looking control group to the treatment group pre-treatment. To answer the above questions in more detail, we first have to define the meaning of the underlying terminology.

The Synthetic Control

First of all, we have a sample of $J + 1$ units of analysis (e.g. countries), where $j = 1$ is the case of interest (“treated unit”), and the units ($j = 2$ to $j = J$) define the “Donor pool” of potential comparison units. The goal of the Synthetic control is to find a weighted average of the donor pool, that is a $(J \times 1)$ vector of weights $W = (w_2, \dots, w_{J+1})'$, with $0 \leq w_j \leq 1$ for $j = 2, \dots, J$ and $w_2 + \dots + w_{J+1} = 1$, that the characteristics of the treated unit is best resembled by the synthetic control. Let X_1 be a $(k \times 1)$ vector of preintervention characteristics for the treated unit and X_0 be a $(k \times J)$ matrix of the same variables for the donor pool ($j = 2$ to $j = J$). The optimization problem is defined as to find a W^* that minimizes $\|X_1 - X_0 W\|$, the difference between the treated unit’s characteristics and the synthetic control.

Operationalization Given $m = 1, \dots, k$, X_{1m} is the value of the m -th variable for the treated unit and X_{0m} : is a $(1 \times J)$ vector containing the values of the m -th variable for the units in the donor pool. W^* is chosen to minimize:

$$\sum_{m=1}^k v_m (X_{1m} - X_{0m} W)^2$$

Regarding Question 2.)

v_m is a weight that reflects the relative importance assigned to each of the predictor variables, when the difference between X_1 and $X_0 W$ is measured. It is crucial that the synthetic control closely reproduces the values that variables with a large predictive power on the outcome of interest take for the unit affected by the intervention. Therefore, variables with high predictive power, logically receive high v -weights.

The v -weights therefore scale the influence of a potential mismatch between the pre-treatment case of interest and the synthetic control. The unit weights w_j however, determine the contribution of each single unit ($j = 2$ to $j = J$) in our data (or donor pool), to the overall value of that value in the synthetic control condition. Therefore, the v -weights, scale the relative importance of the w_j weights for a variable m . Selecting a high value for v , also leads to a stronger influence of an individual units (j) difference between treatment and synthetic control. Where variables are assumed to receive a higher weight, values of w_j should also be chosen more carefully. Selecting low values for v , leads to lower influence of difference in pre-treatment characteristics of the treated and the synthetic control.

The Synthetic Control Estimator

Let Y_{it} denote the outcome of unit j at time t , Y_1 be a $(T_1 \times 1)$ vector collecting the post-intervention values of the outcome for the treated unit, and Y_0 be a $(T_1 \times J)$ matrix where column j contains the post-intervention values of the outcome for unit $j + 1$. For a post-intervention period t (with $t \geq T_0$), the synthetic control estimator of the effect of the treatment is given by the difference between the outcome of the treated unit (Y_{1t}) and the outcome for the synthetic control at that period, which equals the difference between the post-intervention outcomes of the treated unit and the synthetic control $Y_1 - Y_0 W^*$:

$$Y_{1t} - \sum_{j=2}^{J+1} w_j^* Y_{jt}$$

Regarding Question 3:

Recalling the premise and the motivation of the synthetic control method, the goal is to construct a combination of comparison units to reproduce the characteristics of the unit or units representing the case of interest. By definition, the comparison units are constructed by taking the weighted average of all potential comparison units that best resemble the characteristics of the case of interest.

Therefore we can answer the question of what would happen if the weights “could” be negative or sum up to different than one, using just some basic statistics definitions. A mathematical average (or mean) is the sum of all numbers in a set divided by the count of how many numbers are in that set. A weighted average is the sum of a set of numbers where each number has first been multiplied by its own “importance” or “weight” (as explained in the paper). When the weights are negative or greater than one, it is no longer a weighted average, but extrapolation, where a weight greater than one helps to project beyond, and a negative weight means to subtract that unit’s contribution, creating a value that has a negative effect.

According to the authors, this is exactly what we see in regression-based approaches. Although they use synthetic comparison units having coefficients that sum up to one as well, the regression approach **does not** restrict the coefficients of the linear combination that define the comparison unit to be between zero and one. If we now assume that our individual weights (w_j) in the synthetic control term could also have values beyond $[0, 1]$, this would mean that we would just have a simple linear regression model, whose weights extrapolate to produce a perfect fit, in order to minimize the error. The findings of the paper show, however, that extrapolation is not necessary in the context of this study, since there exists a synthetic control that closely fits the values of the characteristics of the units and that does not extrapolate outside of the support of the data.

3.5 (c) Estimation, Balance Before & After

3.5.1 Tasks:

1. Estimate synthetic control for West Germany over pre-treatment period
2. Compute balance table of key predictors (GDP, trade openness, inflation, schooling, investment) showing treated vs. synthetic mean **before treatment**
3. Report non-zero weights w_j
4. Interpret: which donor countries dominate and why?
5. Assess whether pre-treatment fit is acceptable for credible inference

```
# Prepare data for Synth package
```

```
# Run synthetic control estimation
```

```
# Create balance table for pre-treatment predictors
```

```
# Report unit weights
```

Interpretation:

[Which countries contribute most to synthetic West Germany? Is pre-treatment balance good?]

3.6 (d) Effect Size & Permutation Test

3.6.1 Tasks:

1. Plot actual vs. synthetic GDP per capita trajectory (pre- and post-treatment)
2. Calculate estimated effect (gap) in first few post-treatment years and average post-treatment gap
3. Perform **permutation (placebo) test** by reassigning treatment to each control country
4. Report where treated unit's gap falls in the distribution (approximate p-value)
5. Interpret: What does this suggest about the economic impact of reunification?

```
# Plot actual vs synthetic West Germany
```

```
# Calculate treatment effect (gap)
```

```
# Permutation test: assign treatment to each control
```

```
# Calculate p-value
```

Interpretation:

[What is the estimated effect? Is it statistically significant based on permutation test?]

3.7 (e) Placebo Test on Earlier Years

3.7.1 Tasks:

1. Conduct placebo treatment year **before** actual 1990 treatment (e.g., 1975)
2. Re-estimate synthetic control and plot the gap
3. What does pre-treatment gap behavior tell you about parallel-trajectory assumption?

4. Comment on how convincing you find the main causal estimate

```
# Placebo test with fake treatment year
```

```
# Plot placebo gap
```

Interpretation:

[Does the placebo test support the validity of the main estimate?]

4 Conclusion

[Optional: Summarize key findings from both parts]

5 References

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