

Causal Data Analysis

Study Questions – Solution Key

Short Questions

1. **Statement:** “In experimental data ATE will always be equal to ITE.” (Assume that the randomization was done correctly.)

Answer: *False / it depends.*

Let the individual potential outcomes be:

$$Y_i(1) \quad \text{and} \quad Y_i(0),$$

and the *individual treatment effect* (ITE):

$$\tau_i = ITE_i = Y_i(1) - Y_i(0).$$

The *average treatment effect* is:

$$ATE = \mathbb{E}[Y(1) - Y(0)] = \mathbb{E}[\tau_i].$$

In randomized experiments, random assignment ensures that

$$D_i \perp (Y_i(0), Y_i(1)),$$

therefore the difference in sample means between treated and control units identifies the ATE:

$$\widehat{ATE} = \bar{Y}_1 - \bar{Y}_0.$$

However, we can never observe both $Y_i(1)$ and $Y_i(0)$ for the same individual. Hence the ITE is never identified, only its expectation (the ATE) is.

$ATE = ITE$ holds only under *treatment effect homogeneity*:

$$\tau_i = \tau \quad \forall i.$$

In general, treatment effects are heterogeneous, so the statement is false except in special cases.

2. **Statement:** “In experimental data, a simple regression conveys the same information as a simple mean comparison between the treated and control groups.”

Answer: *True (under the usual setup).*

Consider the simple OLS regression:

$$Y_i = \alpha + \beta D_i + u_i,$$

where D_i is a binary treatment (1=treated, 0=control). Under OLS:

$$\hat{\beta} = \bar{Y}_1 - \bar{Y}_0,$$

i.e., the difference between treated and control sample means.

The test statistics and p -values are identical to those from a two-sample t -test.

Therefore, with a constant term and only the treatment dummy as regressor, the regression and the mean comparison contain exactly the same information.

3. **Statement:** “Exact matching produces the same estimated effect as running an OLS regression with controls for the variables which were used in the exact matching.”

Answer: *Generally false / it depends.*

Exact matching keeps only those treated–control pairs whose covariates match *exactly*. The treatment effect is then an average difference computed on the matched sample.

In contrast, OLS:

$$Y_i = \alpha + \tau D_i + X_i' \gamma + u_i$$

uses all observations and imposes a linear functional form. OLS may also extrapolate into regions with poor or no overlap.

The two methods differ in:

- the sample used (matching discards some observations),
- weighting (OLS assigns weights based on variance and covariate distribution),
- functional form assumptions (OLS imposes linearity; matching does not).

Only in special cases (same sample, same weighting, fully interacted specification) will the two methods coincide.

4. **Statement:** “A simple OLS regression will always provide biased estimates.”

Answer: *False.*

OLS is unbiased under the standard exogeneity assumption:

$$\mathbb{E}[u_i \mid X_i] = 0.$$

In experimental data, this often holds due to random assignment. In observational data it may fail, but not always. Hence the statement is false.

Long Questions

1. What is the ideal way to measure the causal effect of x on y (ITE), and how to do this in reality (ATE)? Define the two measures and provide the conditions when $ATE=ITE$. Give an example when they are not equal.

The *ideal* causal effect is the *individual treatment effect*:

$$ITE_i = Y_i(1) - Y_i(0).$$

This is fundamentally unobservable because we never see both potential outcomes for the same individual.

In practice, we estimate the *average treatment effect*:

$$ATE = \mathbb{E}[Y(1) - Y(0)].$$

In randomized experiments, the ATE is identified by comparing sample means.

When is $ATE = ITE$?

When the treatment effect is homogeneous:

$$Y_i(1) - Y_i(0) = \tau \quad \forall i.$$

Example when they differ

Suppose high-ability individuals benefit more from a training program:

$$\tau_i = \begin{cases} 5, & \text{high ability} \\ 1, & \text{low ability} \end{cases}$$

Then ITE varies by person, but ATE is only the average of these values.

2. Explain the three types of endogeneity which can arise in observational data. Draw the causal map and give an example for each type.

The three types:

- (a) **Omitted variable bias (confounding).** A third factor affects both X and Y :

$$X \leftarrow U \rightarrow Y.$$

Example: ability affects both education (X) and earnings (Y).

(b) **Reverse causality.** Causality runs both ways:

$$X \rightarrow Y \quad \text{and} \quad Y \rightarrow X.$$

Example: health and income mutually influence each other.

(c) **Measurement error in X .** Observed X is measured with noise:

$$X^{obs} = X^{true} + \varepsilon.$$

Example: self-reported income contains random errors, biasing OLS estimates of its effect on consumption.

3. An analyst runs the regression:

$$quality_i = \alpha + \beta \cdot family_i,$$

where quality is rated 1–5 and family is a dummy (1 if family-owned). The OLS regression with controls gives $\beta = -0.10^{**}$, $N = 8440$; exact matching on the same controls gives $\beta = -0.16^{**}$, $N = 1207$.

- **Meaning of β :** The estimated causal (or associational) effect of being family-owned on quality. A negative coefficient means family firms have lower average quality scores.
- **Why different N ?** OLS uses all observations; exact matching keeps only treated–control pairs with identical covariates, discarding cases without exact matches. Therefore matching yields a smaller sample.
- **Why is β different across methods?** Because:
 - the sample is different,
 - the weighting is different (OLS imposes variance-based weights),
 - matching avoids functional form assumptions while OLS assumes linearity,
 - OLS may extrapolate outside regions with support.
- **Limits of matching; example of bias:** Matching relies on overlap. If treated units exist only in covariate regions where no controls exist, matched estimates become biased or impossible. Example: if all large firms are treated and all small firms are controls, matching cannot separate treatment effects from firm size.

4. **Describe how to do propensity score matching. Explain the differences between this method and exact matching.**

Steps of PSM:

- (a) Estimate the propensity score:

$$p(X_i) = \Pr(D_i = 1 \mid X_i),$$

typically via logit or probit.

- (b) Match treated and control units with similar $p(X)$ values.
(c) Check balance (standardized differences).
(d) Estimate the treatment effect on the matched sample.

Differences from exact matching:

- Exact matching requires identical covariate values; PSM matches on a single scalar index.
- Exact matching suffers severely from curse of dimensionality; PSM does not.
- PSM ensures similarity in predicted probabilities, not exact covariates.
- PSM typically keeps a larger sample than exact matching.