

Stats 201A: Midterm Study Guide

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Note: The intent of this was to create a Sparknotes, condensed version of topics we have gone over in lecture. Having typed it all up, it seems as if this isn't very concise or condensed. Additionally, this is still a work in progress, so I will incrementally update it as I type up more of my notes.

1. Sampling Design

Our goal in this portion is to understand different sampling designs. We care about sampling because in the real world, it is generally infeasible to survey the entire population (due to a variety of reasons, i.e., costs, non-response, etc.). Therefore, we are stuck with sampling from the population in hopes of using the information we gather from the sample to extrapolate larger information about the population.

More formally, the primary goal within sampling is to estimate summary characteristic of population by using only the information we have from the sample. We want to also assess how accurate (or precise) these estimates are. Different sampling designs will yield estimates that are more or less precise than others.

1.1. Notation

We begin by introducing a lot of notation.

We denote the population as U . \mathcal{F} is all possible combinations of subsets that can exist from the universe U . A represents all the subsets of F that fit within our sampling design. a represents one set from A . $A_{(i)}$ is defined as the set of all A 's that contain the unit i .

Summarizing all of this:

$$a \in A \subseteq \mathcal{F} \subseteq U$$

In other words: a is the actual sample we have. A are the other permutations of the sample that we could have gotten under our sampling design. The distinction between \mathcal{F} and U remains mostly unclear, but based on the slides, we assume that they are vaguely interchangeable and both represent the population.

1.2. Design Linear Estimators

Definition 1.1 *Sampling Design*

Sampling design is the procedure by which the sample of units is selected from the population.

In most of the sampling designs we discuss, we usually select units to be in our sample by assigning a probability to each unit of being chosen for the sample. This is known as the **inclusion probability**.

Definition 1.2 *Inclusion Probability*

The inclusion probability for a unit i is:

$$\pi_i = P(i \in A) = \sum_{a \in A_{(i)}} p(a)$$

Additionally, by introducing an indicator variable I_i that denotes whether the i -th unit is included in our population, we can think of the inclusion probability as:

$$\pi_i = \mathbb{E}(I_i),$$

where

$$I_i = \begin{cases} 1, & \text{if unit } i \text{ is in the sample} \\ 0, & \text{else} \end{cases}$$

We note that $\pi_{ii} = \pi_i$, and $\pi_{ik} = P(\text{Both unit } i \text{ and } k \text{ are included in sample}) = \mathbb{E}(I_i I_k)$.¹

Recall that our goal in sampling is to be able to estimate some population estimand using an estimator. For now, we keep the estimand of interest relatively general, and denote it as θ .

Definition 1.3 *Linear Estimator*

$$\hat{\theta} = \sum_{i \in A} w_i y_i$$

We can think of this as a weighted combination of all the y_i 's in a sample. The weights w_i must not be a function of y_i . Extending the concept of a linear estimator into the design-based framework, we can re-formulate the linear estimator to be summed not just over the A , but the entire population U . This is known as the **design linear estimator**.

¹This should look vaguely familiar as the example from Lecture 1, in which we introduced the indicator Z_i in explaining design-based inference.

Definition 1.4 *Design Linear Estimator*

$$\hat{\theta} = \sum_{i \in U} I_i w_i y_i$$

The weights in the design linear estimators are always *pre-determined and fixed*, with respect to the sampling design. The great thing about design linear estimators is that so long as w_i and y_i are both fixed, we have a representation of the expectation, variance, and design-unbiased estimator for the variance for $\hat{\theta}$. This is more formally stated as two theorems (we refer to them in class as **Theorem 1** and **Theorem 2**).

Theorem 1.1 *Design Linear Estimators*

Assume N total units in a finite population. Let y_i represent a real-valued element for unit i from within the population, and let w_i be fixed. Then, given a design linear estimator of the following form:

$$\hat{\theta} = \sum_{i \in U} I_i w_i y_i,$$

the *expectation of $\hat{\theta}$* will be:

$$\mathbb{E}(\hat{\theta} \mid \mathcal{F}) = \sum_{i=1}^N w_i \pi_i y_i$$

and the *variance of $\hat{\theta}$* is:

$$\mathbb{V}(\hat{\theta} \mid \mathcal{F}) = \sum_{i=1}^N \sum_{k=1}^N (\pi_{ik} - \pi_i \pi_k) w_i y_i w_k y_k$$

Theorem 1.2 *Variance Estimator (for Design Linear Estimators)*

Once again, assume N total units in a finite population. Consistent with before, let y_i represent a real-valued element for unit i from within the population, and let w_i be fixed. Now, assuming that all joint inclusion probabilities are non-zero (i.e., $\pi_{ik} > 0 \forall i, k \in U$), then:

$$\hat{\mathbb{V}}(\hat{\theta} \mid \mathcal{F}) = \sum_{i,j \in A} \pi_{ik}^{-1} (\pi_{ik} - \pi_i \pi_k) w_i y_i w_k y_k$$

Furthermore, $\hat{\mathbb{V}}(\hat{\theta} \mid \mathcal{F})$ will be a *design-unbiased estimator* for $\mathbb{V}(\hat{\theta} \mid \mathcal{F})$.

Different estimators can be derived using this framework by changing how we formulate the weights. The main family of estimators we care about are known as the **Horovitz-Thompson Estimator**.

1.3. Horovitz-Thompson Estimator

The Horvitz-Thompson Estimator defines the weights in our design linear estimator as being the inverse of inclusion probability:

$$w_i = \frac{1}{\pi_i}$$

Up until now, we have kept the estimand of interest very general (specified as θ). We will now introduce two common estimands that are of interest:

- Finite Population Total:

$$T_y = \sum_{i \in U} y_i = \sum_{i=1}^N y_i$$

- Finite Population Mean:

$$\bar{y}_N = \frac{1}{N} T_y = \frac{1}{N} \sum_{i=1}^N y_i$$

The Horovitz-Thompson Estimator can be used to derive an *unbiased estimate for both the population total and mean*:

- Estimator for Finite Population Total:

$$\begin{aligned} \hat{T}_y &= \sum_{i \in U} I_i \pi_i^{-1} y_i = \sum_{i \in A} \pi_i^{-1} y_i \\ \hat{\mathbb{V}}(\hat{T}_y) &= \sum_{i,j \in A} \sum \pi_{ik}^{-1} (\pi_{ik} - \pi_i \pi_k) \pi_i^{-1} y_i \pi_k^{-1} y_k \end{aligned}$$

- Estimator for Finite Population Mean:

$$\begin{aligned} \bar{y}_{HT} &= \frac{1}{N} \hat{T}_y = \frac{1}{N} \sum_{i \in A} \pi_i^{-1} y_i \\ \hat{\mathbb{V}}(\bar{y}_{HT}) &= \frac{1}{N^2} \sum_{i,j \in A} \sum \pi_{ik}^{-1} (\pi_{ik} - \pi_i \pi_k) \pi_i^{-1} y_i \pi_k^{-1} y_k \end{aligned}$$

This will serve as the basis for all of our estimators that are derived from various sampling schemes. The basic estimator specification will remain the same, but we will change the inclusion probabilities (π_i) to fit with the sampling design.

Some potential drawbacks of the Horovitz-Thompson estimator are that it can be very high variance² (i.e., Basu’s elephant), and that it will be location variant.

Alternative Estimator: Hajek Estimator³

An alternative estimator to the Horovitz-Thompson estimator is the Hajek estimator. The Hajek estimator weights events that are more probable, such that the estimator is less susceptible to outliers or “weird” events that could be drawn. The Hajek estimator is more precise than the Horovitz-Thompson estimator; however, it will be biased. It takes on the following form:

$$\hat{T}_{Hajek} = \left(\sum_{i \in A} \pi_i^{-1} \right) \sum_{i \in A} \pi_i^{-1} y_i$$

1.4. Sampling Schemes

1. Simple Random Sampling

- Assume random draws (sampling without replacement)
- $\binom{N}{n}$ total possible samples
- $\pi_i = \frac{n}{N}$ for all i
- $\pi_{ik} = \frac{n}{N} \cdot \frac{n-1}{N-1}$
- Estimated Population Total:

$$\hat{T}_y = N \cdot \frac{1}{n} \sum_{i \in A} y_i$$

– Variance of Population Total:

$$\mathbb{V}(\hat{T}_y \mid \mathcal{F}) = N^2 \left(1 - \frac{n}{N} \right) \frac{1}{n} \left[\frac{1}{N-1} \sum_{i=1}^N (y_i - \bar{y}_N)^2 \right]$$

²Horovitz and Thompson showed that there exists an optimal weight assignment (or, optimal way to set up our inclusion probabilities) such that the high variance problem could be mitigated:

$$\frac{1}{n-1} \sum_{k=1}^N \pi_{ik} = \pi_i = \frac{ny_i}{\sum_{i=1}^N y_i}$$

As it turns out, this is profoundly unhelpful since we don’t know what $\sum_{i=1}^N y_i$ is, and if we did know it, we wouldn’t have to sample and use an estimator to begin with. It does, however, imply that we should attempt to find a good proxy for y and try to sample proportionally in accordance with this auxiliary variable.

³We never really mention Hajek after this, and all the sampling designs are discussed in the context of the HT estimator framework, but I’ve included this section as a fun fact in case it ever shows up as a Jeopardy question!

– Estimated Variance of Population Total:

$$\hat{\mathbb{V}}(\hat{T}_y \mid \mathcal{F}) = N^2 \left(1 - \frac{n}{N}\right) \frac{1}{n} \left[\frac{1}{n-1} \sum_{i \in A} (y_i - \bar{y}_n)^2 \right]$$

2. Poisson Sampling (a.k.a. p-coin flipping)

- Series of N independent Bernoulli trials
- Each unit has a probability of π_i (π_i can be different across units)
- $\pi_{ik} = \pi_i \pi_k$

3. Stratified Sampling

- We assume H total strata
- The h -th strata contains N_h element
- Each element is in only one strata
- We then have a sampling scheme that we apply to each strata.
- Estimate of population mean (assuming SRS of strata):

$$\bar{y} = \sum_{h=1}^H \frac{N_h}{N} \bar{y}_h,$$

where \bar{y}_h is the mean within each strata (i.e., $\bar{y}_h = \frac{1}{n_h} \sum_{i \in A_h} y_{h,i}$)

– The variance of the estimated population mean is:

$$\mathbb{V}(\bar{y} \mid \mathcal{F}) = \sum_{h=1}^H \left(\frac{N_h}{N} \right)^2 \left(1 - \frac{n_h}{N_h} \right) \frac{S_h^2}{n_h}$$

– The estimated variance of the estimated population mean is:

$$\hat{\mathbb{V}}(\bar{y} \mid \mathcal{F}) = \sum_{h=1}^H \left(\frac{N_h}{N} \right)^2 \left(1 - \frac{n_h}{N_h} \right) \frac{s_h^2}{n_h}$$

4. Cluster Sampling

1.5. Dealing with Non-Response

We care about non-response because there may exist biases that arise from persistent non-response. This can be problematic, because then we cannot estimate things accurately for the population.

Definition 1.5 *Non-Response*

Non-response is defined as when a unit does not respond.

We use an indicator variable R_i to represent this.

$$R_i = \begin{cases} 1 & \text{if unit } i \text{ responds} \\ 0 & \text{if unit } i \text{ does not respond} \end{cases}$$

2. Causality

Our goal is to measure the causal effect⁴ of a particular *unit*. For example, assume you have a headache from studying for your midterm. We want to see if, after you take aspirin, the aspirin mitigates your headache so you may return back to productively studying. In order to discuss causal effects, we must introduce something known as the **potential outcomes framework**.

2.1. The Potential Outcomes Framework

Within the potential outcomes framework, we assume many different units i exist. We can assign a unit i to treatment, or we can assign the unit to control. This is denoted by an indicator variable T_i . Each unit has its own potential outcomes: $Y_i(T_i)$. Our goal is to find the treatment effect of a particular unit i , i.e., the effect that being in treatment had.

More formally:

Definition 2.1 *Treatment Assignment*

$$T_i = \begin{cases} 1, & \text{unit } i \text{ is assigned to treatment} \\ 0, & \text{else} \end{cases}$$

Definition 2.2 *Potential Outcomes*

$$Y_i(T_i) = \begin{cases} Y_i(1), & \text{potential outcome for unit } i, \text{ if unit } i \text{ is assigned to treatment} \\ Y_i(0), & \text{potential outcome for unit } i, \text{ if unit } i \text{ is assigned to control} \end{cases}$$

The potential outcomes for each unit i is *fixed*. The only random component within our design is the treatment assignment.

⁴Rubin uses the term ‘causal effect’. We use the term ‘treatment effect’ in class.

Definition 2.3 *Treatment (Causal) Effect*

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

The above is the unit level treatment effect. Other estimands that are often of interest to us within a study are:

- Sample Average Treatment Effect (SATE):

$$\frac{1}{n} \sum_{i=1}^n (Y_i(1) - Y_i(0))$$

- Population Average Treatment Effect (PATE):

$$\frac{1}{N} \sum_{i=1}^N (Y_i(1) - Y_i(0))$$

- Population Average Treatment Effect for the Treated (PATT):

$$\mathbb{E}(Y_i(1) - Y_i(0) | T_i = 1)$$

- Population Conditional Average Treatment Effect (CATT):

$$\mathbb{E}(Y_i(1) - Y_i(0) | X_i = x)$$

Notice that by definition, all of the estimands that represent the treatment effect compare the potential outcomes for the same unit, at the same moment in time, following treatment assignment. In reality, we can never observe both $Y_i(0)$ and $Y_i(1)$, because a unit cannot both be simultaneously in the treatment and control group, such that both potential outcomes are realized. Therefore, we can only see something known as the **observed outcome**.

Definition 2.4 *Observed Outcome*

$$Y_i = T_i Y_i(1) + (1 - T_i) Y_i(0)$$

This brings to light the fundamental problem within causal inference: we want to see the specific effect of treatment on unit i by comparing what would have occurred if unit i had been under treatment, versus what would have occurred if unit i had received no treatment. However, by putting unit i in either treatment or control, we can no longer observe the other potential outcome! Therefore, we have a missing data problem. In other words, we can only compare observed outcomes, since there exists only one realized potential outcome per unit.

To bypass the fundamental problem of missing data, we can try to predict (or impute) the missing potential outcomes at an individual level. Alternatively, we can observe different units i that are assigned to treatment or control within the same time period and see if we can estimate the treatment effects. In order to perform any form of identification, the assignment mechanism must be *random*. In the next portion, we will talk about the necessary assumptions that need to be met within our designed experiment in order to estimate treatment effects.

Long story short: we cannot measure the causal effect of a treatment due to the missing data problem. Therefore, we must randomize our treatment assignments to *estimate* the treatment effects, such that the outcome of one unit is independent of the treatment assignments of other units. This primary assumption is known as SUTVA.

2.2. Stable Unit Treatment Value Assumption

The Stable Unit Treatment Value Assumption (SUTVA) is that the outcome of a single unit does not depend on the assignments of other units. This can be written as three explicit assumptions that must be met in order for us to make claims about causal effects.

The assumptions are:

1. No simultaneity (outcome cannot cause treatment)
2. No interference (a unit's outcome does not depend on whether other units are assigned treatments)
3. Same version of treatment is being applied

SUTVA is easily violated in the context of spillover effects, as well as scenarios in which there may be variation in which treatments are administered. In these cases, we often try to re-define the treatment or our framework. For example, we may change how we define a unit. Whereas before, a unit i may be an individual person, we may opt to define a unit as an entire neighborhood.

2.3. Assignment Mechanism

The assignment mechanism is formally defined as the process by which units receive treatment. There exist three basic restrictions on how our assignment mechanism works:

1. Individual Assignment: No dependence of a unit's assignment probability on the values of the covariates and potential outcomes for other units

2. Probabilistic Assignment: Non-zero probability for all units to be assigned treatment
3. Unconfounded Assignment: No dependence of assignment mechanism on potential outcomes

There are different types of assignment mechanisms, depending on the experiment design and question we are trying to answer. In particular:

1. Classical Randomized Experiments:⁵

In a classical randomized experiment, the researcher controls who receives treatment at a unit level. These experiments have *high internal validity*, since assignment probabilities are fully controlled and determined by the researcher. However, they may have *lower external validity* due to the fact that the results within an experiment can be difficult to generalize to the wider population.

2. Regular Assignment Mechanism

A regular assignment mechanism is where the actual assignment mechanism is unknown to the researcher, but the process by which units are assigned to treatment or control still meets the necessary assumptions. This is common within observational studies. It is usually up to the researcher to prove that the required assumptions are safe to make within this context. In these studies, findings may have *low internal validity*, but *high external validity*.

3. Irregular Assignment Mechanism

With an irregular assignment mechanism, there are issues with confoundment. There may be differences within the treatment group, due to the fact that receipt of treatment may vary amongst units. In order to perform causal inference here, we must make additional assumptions.

2.4. Design-Based Inference with Randomization

By randomization, we are able to justify causal inference, despite of the missing data problem. This is because we assume that, since there exists randomization, there exist no relationship between the treatment and underlying characteristics of each unit. In other words, we assume:

$$\{Y_i(0), Y_i(1)\} \perp\!\!\!\perp T_i$$

This would allow us to perform *identification by randomization*. This is often times referred to as *ignorability*.

When we cannot assume that the potential outcomes and the treatments are independent, we

⁵This is what our class is primarily focused on.

may find covariates X_i (i.e., some pre-treatment characteristics) that serve as a backdoor criterion⁶ such that:

$$\{Y_i(0), Y_i(1)\} \perp\!\!\!\perp T_i \mid X_i$$

This would render $Y_i(\cdot)$ and T_i conditionally independent, and we can then perform *identification by conditional independence* (this is referred to as *conditional ignorability*).

For the purposes of this course, we focus primarily on identification by randomization. In identification by randomization, we follow the following framework. We begin by fixing the number of units that must be assigned to treatment (call this n_1). There are n units in total. This means that the probability of treatment is:

$$P(T_i = 1) = \frac{n_1}{n}$$

Our estimands of interest are the SATE or PATE. The estimator that is used for SATE and PATE is known as the *difference in means estimator*.

Definition 2.5 *Difference-in-Means Estimator*

$$\hat{\tau} = \frac{1}{n_1} \sum_{i=1}^n T_i Y_i - \frac{1}{n_0} \sum_{i=1}^n (1 - T_i) Y_i$$

The difference-in-means estimator is an unbiased estimator of SATE. It is also an unbiased estimator of PATE, given certain assumptions. We will discuss properties of the difference-in-means estimator below.

Properties of the Difference-in-Means Estimator

Property 1. The difference-in-means estimator is an unbiased estimator of SATE

Property 2. Under certain assumptions, the difference-in-means estimator is an unbiased estimator of PATE.

Property 3. To estimate the variance of the difference-in-means estimator for SATE, we must use the Neyman Estimator as an upper bound for variance.

Property 4. The Neyman Estimator will be an unbiased estimator for the variance of the difference-in-means estimator for PATE.

⁶For all the homies out there who, like me, don't know what backdoor criterion is: assume we are trying to figure out the causal effect of a on y (i.e. $a \rightarrow y$) However, there's a confounding variable x , such that $x \rightarrow a$, and $x \rightarrow y$. This makes it hard to tease out the actual effect of a on y since there is a backdoor path that exists ($x \rightarrow a \rightarrow y$). Therefore, in order to tease out the effect of a on y , we have to first condition on x to "block" the pathway formed between x and a , and x and y . (This makes a lot more sense when looking at a DAG, but since I'm too lazy to draw one, words will have to suffice.) **TL;DR:** backdoor criterion is a Fancy Way of saying that we've conditioned on all potential confounding variables, such that there exists no confounding effects, so we can perform causal inference.

Each property will be expanded upon in more detail.

Property 1. Unbiased Estimator of SATE

Above, we stated that the difference-in-means estimator $\hat{\tau}$ is an unbiased estimator of SATE. To show this, we can take the expectation of $\hat{\tau}$. Let's define Ω as the set of potential outcomes in our sample of n units (formally: $\Omega = \{Y_i(0), Y_i(1)\}_{i=1}^n$):

$$\mathbb{E}(\hat{\tau} \mid \Omega) = \mathbb{E}\left(\underbrace{\frac{1}{n_1} \sum_{i=1}^n T_i Y_i}_{(1)} - \frac{1}{n_0} \sum_{i=1}^n (1 - T_i) Y_i \mid \Omega\right)$$

Recall that the observed outcome is defined as $Y_i = T_i Y_i(1) - (1 - T_i) Y_i(0)$. We can substitute this in for Y_i . Let's begin by looking at just the first term (denoted as (1)):

$$\begin{aligned} \frac{1}{n_1} \sum_{i=1}^n T_i Y_i &= \frac{1}{n_1} \sum_{i=1}^n T_i (T_i Y_i(1) - (1 - T_i) Y_i(0)) \\ &= \frac{1}{n_1} \sum_{i=1}^n (T_i^2 Y_i(1) - T_i(1 - T_i) Y_i(0)) \\ &= \frac{1}{n_1} \sum_{i=1}^n (T_i^2 Y_i(1) - (T_i - T_i^2) Y_i(0)) \end{aligned}$$

Notice that $T_i^2 = T_i$ (Since $T_i = 1$ or 0 , so the squared of this value will remain the same). We can rewrite the above as:

$$\begin{aligned} &= \frac{1}{n_1} \sum_{i=1}^n (T_i Y_i(1) - (T_i - T_i) Y_i(0)) \\ &= \frac{1}{n_1} \sum_{i=1}^n T_i Y_i(1) \end{aligned}$$

Now if we take the expectation across this sum, the only random component to this equation is T_i , since the potential outcomes $Y_i(\cdot)$ are fixed, as is n_1 . Therefore:

$$\begin{aligned} \mathbb{E}\left[\frac{1}{n_1} \sum_{i=1}^n T_i Y_i \mid \Omega\right] &= \frac{1}{n_1} \sum_{i=1}^n \mathbb{E}(T_i \mid \Omega) Y_i(1) \\ &= \frac{1}{n_1} \sum_{i=1}^n \frac{n_1}{n} Y_i(1) \\ &= \frac{1}{n} \sum_{i=1}^n Y_i(1) \end{aligned}$$

A similar substitution can be made on the second term. This gives us:

$$\begin{aligned}\mathbb{E}(\hat{\tau} \mid \Omega) &= \frac{1}{n} \sum_{i=1}^n Y_i(1) - \frac{1}{n_0} \sum_{i=1}^n \left(1 - \frac{n_1}{n}\right) Y_i(0) \\ &= \frac{1}{n} \sum_{i=1}^n (Y_i(1) - Y_i(0)) \\ &= \text{SATE}\end{aligned}$$

Property 2. Unbiased Estimator of PATE

The difference-in-means estimator can also be used as an unbiased estimator for PATE. However, in order to do so, we must assume that all units are randomly sampled from an infinite population. Recall that SATE is conditioned on the set Ω . Therefore, we can think Ω as one sub-group that serves as one realization of infinitely many other samples that could have been drawn. Using Law of Iterated Expectation, we find:

$$\mathbb{E}(\hat{\tau}) = \mathbb{E}(\mathbb{E}(\hat{\tau} \mid \Omega)) = \mathbb{E}(\text{SATE})$$

This is where things get a little weird. Before, we said that SATE and PATE were both estimands of interest. They technically are. However, SATE is also an estimator for PATE (much like how the sample mean is an estimator for the population mean). We can rewrite SATE in a way such that this makes more sense. In particular, we can introduce an indicator variable Z_i . Z_i takes on a value of 1 when the i -th unit is in our sample, and 0 otherwise (this is consistent with before, when we first introduced design based estimators). By introducing Z_i , we may rewrite the summation to now be from $i = 1, \dots, N$,⁷ instead of $i = 1, \dots, n$. Additionally, like before, the probability that $Z_i = 1$ is simply the number of units in the sample divided by the total number of units in the population.

In math speak:

$$\begin{aligned}\text{SATE} &= \frac{1}{n} \sum_{i=1}^n Y_i(1) - Y_i(0) \\ &= \frac{1}{n} \sum_{i=1}^N Z_i Y_i(1) - Z_i Y_i(0) \\ &= \frac{1}{n} \sum_{i=1}^N Z_i (Y_i(1) - Y_i(0))\end{aligned}$$

⁷We are going to ignore the fact that we’ve just assumed there exists an infinite population, in which case $N \rightarrow \infty$; instead, let’s reframe infinity to be something akin to a Very Large Number, like a million in accordance with Wu, 2018.

Taking the expectation of SATE:

$$\begin{aligned}\mathbb{E}(\text{SATE}) &= \mathbb{E}\left(\frac{1}{n} \sum_{i=1}^N Z_i(Y_i(1) - Y_i(0))\right) \\ &= \frac{1}{n} \sum_{i=1}^N \mathbb{E}(Z_i)(Y_i(1) - Y_i(0)) \\ &= \frac{1}{n} \sum_{i=1}^N \frac{n}{N} (Y_i(1) - Y_i(0)) \\ &= \frac{1}{N} \sum_{i=1}^N Y_i(1) - Y_i(0)\end{aligned}$$

As such, SATE is an unbiased estimator for PATE.

Therefore, after this fun digression, we can return back to the expectation of $\hat{\tau}$ to show that:

$$\mathbb{E}(\hat{\tau}) = \mathbb{E}(\mathbb{E}(\hat{\tau} \mid \Omega)) = \mathbb{E}(\text{SATE}) = \text{PATE}$$

Property 3. Estimating the Variance of $\hat{\tau}$ for SATE

What about the variance of $\hat{\tau}$?

We once again begin by conditioning on just our sample's potential outcomes.

$$\text{var}(\hat{\tau} \mid \Omega) = \frac{S_1^2}{n_1} + \frac{S_0^2}{n_0} - \frac{S_{01}^2}{n}$$

Above, S_t^2 is the equivalent of the sample variance for $Y_i(t)$. More formally:

$$S_t^2 = \frac{1}{n-1} \sum_{i=1}^n (Y_i(t) - \overline{Y(t)})^2$$

(This is equivalent to if you took your entire column of $Y_i(1)$'s and typed in `var(y1)` into R).

S_{01}^2 is the sample variance of τ_i (the treatment effect)⁸:

$$S_{01}^2 = \frac{1}{n-1} \sum_{i=1}^n (\tau_i - \text{SATE})^2$$

We can rewrite this as:

$$S_{01}^2 = \frac{1}{n-1} \sum_{i=1}^n (Y_i(1) - Y_i(0) - \text{SATE})^2$$

⁸Brief note on notation: in the lecture notes, we use τ to denote SATE. This is mildly confusing, because there also exists PATE, which could arguably also be represented by τ . In formulating the sample variance, the Rubin book denotes the term as τ_{fs} . To bypass introducing more symbols, I simply write in SATE.

This is mildly problematic, because in order to estimate the variance of the estimator, we would need to somehow estimate the sample variance of the treatment effect. This would require us to know both potential outcomes, which we don't know, or we wouldn't be down this rabbit hole of inference through randomization to begin with!

Since we have no means of ever really measuring S_{01}^2 without entering the alternate universe in which the treatment and control groups were flipped, the next best option is to bound the variance. We can do this relatively trivially by noting the following:

$$\text{var}(\hat{\tau} \mid \Omega) = \frac{S_1^2}{n_1} + \frac{S_0^2}{n_0} - \frac{S_{01}^2}{n} \leq \frac{S_1^2}{n_1} + \frac{S_0^2}{n_0}$$

Neyman was the first to observe this⁹, so we call this bound the **Neyman estimator**:

$$\hat{\mathbb{V}}_{\text{Neyman}}(\hat{\tau} \mid \Omega) = \frac{S_1^2}{n_1} + \frac{S_0^2}{n_0}$$

The Neyman estimator can serve as an estimator for the variance of SATE. If the treatment effect is constant for all units i (i.e., $\tau_i = c$, $\forall i$, which would imply that SATE is also equal to c , thereby making the entire S_{01}^2 term go to zero), then the Neyman estimator will be unbiased (in the lecture notes, this is referred to explicitly as the *constant treatment assumption*). However, this constant treatment effect assumption is usually not met. This is actually okay. While the Neyman estimator may not be unbiased under non-constant treatment effect scenarios, it serves as a conservative estimate for what the actual variance is. This means that while we may have overcoverage, we can assume at least nominal coverage.

Property 4. Estimating the Variance of $\hat{\tau}$ for PATE

For the variance of the estimator when looking at PATE, we have the following:

$$\mathbb{V}(\hat{\tau}) = \mathbb{V}(\mathbb{E}(\hat{\tau} \mid \Omega)) + \mathbb{E}(\mathbb{V}(\hat{\tau} \mid \Omega)) \approx \frac{\sigma_1^2}{n_1} + \frac{\sigma_0^2}{n_0}$$

Therefore, for PATE, there exists no need to know both potential outcomes! We can rejoice, because this implies that we can use the Neyman estimator as an unbiased estimate for the variance:

$$\hat{\mathbb{V}}(\hat{\tau}) = \hat{\mathbb{V}}_{\text{Neyman}}(\hat{\tau})$$

2.5. Randomization Inference

Up until this point, we have talked a lot about estimating the average treatment effects within both samples and populations. However, estimating an average treatment effect of zero does not

⁹I'm not sure if that's actually true, but I'd imagine so, since this is named after him. "Did you ever think about what a coincidence it is that Lou Gehrig had Lou Gehrigs disease?" (Kresin, 2018).

necessarily imply a lack of treatment effect across all units. A very simple example could be that one sub-group within our sample has a positive treatment effect, while another sub-group has a negative treatment effect. Therefore, this could lead to an averaged zero treatment effect.

We want to examine the question whether or not a treatment effect is zero for every unit. To do this, we must introduce something known as **randomization inference**, also referred to as **permutation (or Fisherian) inference**.

To begin, we define something known as the Sharp Null Hypothesis.

Definition 2.6 *Sharp Null Hypothesis*

The Sharp Null Hypothesis assumes that the estimated treatment effect is zero for all units i . This can be extended such that we may choose the estimated treatment effect to be any constant c under the sharp null.

The Sharp Null Hypothesis allows us to bypass the missing data problem, because under the sharp null, we can easily calculate the missing potential outcomes that are unobserved by using the observed outcomes. In other words, because we are assuming there is no treatment effect, $Y_i(0) = Y_i(1) = Y_i$.

Under the randomized inference framework, we calculate all the possible treatment assignments. This is possible because within the classical randomized experiment setting, we (the researcher) have full control over the assignment mechanism, and therefore know the randomization scheme that is being used to assign units to treatment or control. For some test statistic t , we can generate a distribution of all possible values of t under our assignment mechanism.¹⁰ (It is important to reiterate that we can create such a distribution because all the potential outcomes are known, since we assume the only thing that changes under the Sharp Null is the label of whether or not a unit is in treatment or control; the outcome never changes under the Sharp Null.) As such, by generating such a distribution, we can directly calculate the probability of observing our actual test statistic under the sharp null.

The probability of observing our test statistic, or a more extreme value, under the sharp null is what is formally known as **Fisher’s exact p-values**.

Definition 2.7 *Fisher’s Exact P-Values*

Let Ω be defined as the set of all possible randomizations under our assignment mechanism. For a one-sided test, Fisher’s exact p-values are defined as:

$$P = \frac{1}{|\Omega|} \sum_i^{|\Omega|} \mathbb{I}(t_i \geq t_{obs})$$

¹⁰ t is never defined as anything explicit, but Rubin makes a note that the test statistic we have in question is going to be the function of the assignment variable T_i , the observed outcome Y_i , and potentially some covariates.

For a two-sided test, the p-value would take on the following form:

$$P = \frac{1}{|\Omega|} \sum_i^{|\Omega|} \mathbb{I}(|t_i| \geq |t_{obs}|)$$

Essentially, we simply count up the total number of test statistics that, under all the various permutations of assignments, could be more extreme than the observed test statistic, and divide it by the total number of different permutations of assignments that exist in our design. If the probability of getting a test statistic like the one we observed is very low under the sharp null, then we have reason to believe that there may not necessarily be a zero treatment effect for all units, and, as such, can opt to reject the Sharp Null.

Confidence intervals? Don't currently understand what this is all about ... TBD