Propensity score with continuous treatments

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1 Introduction

Causal inference, the discipline concerned with estimating causal effects from non-randomized observational data, has traditionally focused on scenarios involving binary treatment variables. However, not all interventions are binary and sometimes we are interested in continuous treatments. For example, instead of the binary treatment of taking a drug versus not taking a drug, we can perhaps gain more insight if we consider the treatment dose to be continuous to help us decide whether taking too much of a drug may lose effectiveness and become harmful. Since we already have an established framework for binary treatment cases that utilizes propensity score to estimate causal effects, we hope to extend the same intuition into the continuous case as well. This is exactly the proposal put forth by Hirano and Imbens in their paper on the Generalized Propensity Score.

2 Methodology

2.1 Notations

To begin, we define the notations we will be using throughout the paper. We have random sample of units, indexed by $i = 1, \dots, N$. Each unit i assumes the existence of a set of potential outcomes denoted $Y_i(t)$. Unlike in binary treatment case in which **T** is either 0 or 1, in the continuous treatment case, **T** is an interval $[t_0, t_1]$. We are interested in the expected potential outcome, or average dose-response function defined as $\mu(t) = E[Y_i(t)]$.

Each unit i has a vector of covariates X_i and treatment received T_i. The potential outcome corresponding to the level of treatment received is denoted by $Y_i = Y_i(T_i)$. We simplify the notation by dropping subscript i and assume that there is a collection of all potential outcomes for all individuals in the sample across all possible treatment levels between the previously defined interval $\{Y(t)\}_{t \in \boldsymbol{T}}$. Note that T, X are defined on a common probability space and T is continuously distributed with respect to Lebesgue measure on **T.**

2.2 Weak Unconfoundedness and Generalized Propensity Score

The key assumption made in Hirano and Imbens' work generalizes the unconfoundedness assumption previously introduced by Rosenbaum and Rubin (1983) for binary treatments to the continuous case. This assumption is called the "**Weak Unconfoundedness**":

$$Y(t) \perp T|X \; for \; all \; t \in \mathbf{T} \qquad (Assumption \; 1)$$

It assumes that by adjusting for the difference in a set of covariates, the outcome will be independent to the treatment which allows us to estimate the causal effect of the treatment on outcome.

Weak unconfoundedness does not require joint independence of all potential outcomes but require conditional independence for each value of the treatment. Weak unconfoundedness differs from strong ignorability of treatment assignment because there is no requirement for joint independence for all treatment doses.

Next, we will introduce the definition of "**Generalized Propensity Score (GPS)**" which helps us adjust for differences in covariate distributions between treatment groups and ensure the weak unconfoundedness assumption holds.

Let $r(t,x)$ be the conditional density of the treatment given the covariates:

$$\mathrm{r}(t,x) = f_{T|x}(t|x) \qquad (Definition 1)$$

The Generalized Propensity Score is $R = r(T,X)$.

GPS is a function that predicts the probability of receiving a particular treatment value t given a set of pretreatment variable X (covariates). Like the standard propensity score, GPS also has a balancing property: $X \perp \mathbf{1}\{T = t\}|r(t,X)$. If we condition on $r(t,X)$ then whether a unit receives the treatment at level t or not is not related to their covariates.

In the original research paper, Hirano and Imbens provided a mathematical proof that provides a theoretical justification for using GPS to adjust for differences in covariate distributions between treatment groups. Theorem 1 in the paper states that if assignment to treatment is weakly unconfounded given pretreatment variables X. Then for every t

$$f_T\big(t\big|r(t,X),Y(t)\big) = f_T\big(t\big|r(t,X)\big) \qquad (Theorem\ 1)$$

The result of the proof shows that by conditioning on the GPS, the treatment assignment mechanism does not depend on the potential outcomes beyond what is already captured by the pretreatment variables X. In other words, once we condition on GPS, there are no other confounding variables that affects both treatment assignment and potential outcomes.

2.3 Bias Removal using GPS

Hirano and Imbens provided a two-step approach to show that GPS can be used to eliminate any biases associated with differences in the covariates. The first step is to estimate the conditional expectation of the outcome as a function of two scalar variables: the treatment level T and the GPS R. This is represented by $\beta(t,r) = E[Y|T = t, R = r]$. Second, we estimate the dose-response function at a particular level of the treatment. We do this by averaging the conditional expectation over the GPS at that level of the treatment, $\mu(t) = E\big[\beta\big(t,r(t,X)\big)\big]$. We do not average over the entire GPS $R = r(T,X)$ instead we only average GPS scores that are relevant to the specific treatment level we are interested in analyzing, $r(t,X)$.

Theorem 2 states that if assignment to treatment is weakly unconfounded given pretreatment variables X. Then the following is true.

$$\beta(t,r) = E[Y(t)|r(t,X) = r] = E[Y|T = t, R = r] \qquad (i)$$

$$\mu(\mathrm{t}) \;=\; \mathrm{E}[\beta(\mathrm{t},\mathrm{r}(\mathrm{t},\mathrm{X})] \qquad (ii)$$

According to the proof, by applying Bayes rule and the results from Theorem 1, the conditional density (with respect to some measure) of $Y(t)$ given $T = t$ and $r(t,X) = r$:

$$f_{Y(t)|T,r(t,X)}(y|t,r) = f_{Y(t)|r(t,X)}(y|r) \qquad (1)$$

Then taking the expectation:

$$E[Y(t)|T = t, r(t,X) = r] = E[Y(t)|r(t,X) = r] \quad (2)$$

After some simplifying, we also have the result:

$$E[Y(t)|T = t, R = r] = E[Y(t)|r(t,X) = r] = \beta(t,r) \quad (3)$$

Lastly, applying iterated expectations

$$E\big[\beta\big(t, r(t,X)\big)\big] = E\big[E[Y(t)|r(t,X)]\big] = E[Y(t)] \quad (4)$$

Note that $\beta(t,r)$ does not have any intrinsic causal interpretation because we are still conditioning on treatment and GPS. We need to take the expectation again to represent the population. Generalized Propensity score is a scalar function of the covariates that predicts the probability of receiving treatment. If there are differences in covariates between treatment groups, then the GPS may not be balanced across these groups, which can lead to biased estimates of causal effects. Therefore, when using GPS to estimate causal effects, it is important to weight observations appropriately to account for differences in covariates between treatment groups. In continuous treatment, the weights must be "stabilized" by the marginal probabilities of treatment.

Hirano and Imbens (2004) discussed a separate method called Marginal Structural Model (MSM) proposed by Robins, Hernan, and Brumback (2000), that parameterize the form of the $Y(t)$ process, the form of $\mu(t)$, to address the issue. The parameters of the MSM are estimated using a weighted scheme based on the GPS. However, in the paper they decided to employ parametric assumptions on $\beta(t,r)$ instead of $\mu(t)$ to avoid the need to reweight observations.

3 Estimation and Inference (Implementation)

We will walk through a simple example to demonstrate how to practically implement the method. Given a parametric model for the relationship between the treatment T and the covariates X, the conditional density of the treatment given covariates is $r(t,x) = f_{T|X}(t|x)$. For each individual i, the Generalized Propensity Score $r_i(T,X)$ represents the treatment's conditional density evaluated based on the individual's specific treatment and covariate value. The method proposed by Hirano and Imbens (2004) to estimate the effects of continuous treatments can be generalized in the following steps with examples attached:

1. Model the continuous treatment variable as a function of covariates.
    a. We first fit the linear regression model: $T_i = \beta_0 + \beta X_i + \epsilon_i$ with $e_i \sim N(0, \sigma_i^2)$ to represent normal distribution for the treatment given covariates.

$$T_i|X \sim N(\beta_0 + \beta X_i,\ \sigma^2) \quad (5)$$

2. Obtain the GPS
    a. Then estimate β_0, β, and σ^2 by maximum likelihood. The Estimated GPS can be calculated as follows:

$$\hat{R}_i = r_i(T, X) = \frac{1}{\sqrt{2\pi\hat{\sigma}^2}} exp\left(-\frac{1}{2\sigma^2}\left(T_i - \hat{\beta}_0 - \hat{\beta}X_i\right)^2\right) \quad (6)$$

3. Model the outcomes as a function of the treatment and GPS
    a. In the next stage, we model conditional expectation of Y_i given T_i and R_i as a flexible function of its two arguments (Quadratic Approximation):

$$E[Y_i|T_i, R_i] = \alpha_0 + \alpha_1 T_i + \alpha_2 T_i^2 + \alpha_3 R_i + \alpha_4 R_i^2 + \alpha_5 T_i R_i \quad (7)$$

    b. Note that $\beta(t, r) = E[Y|T, R]$ does not have a causal interpretation as discussed previously and we estimate the above parameters by ordinary least squares using the estimated GPS $\hat{R}_i$.

4. Estimate the average potential outcome at each treatment dose of interest and plot the dose-response function.
    a. Finally, we estimate the average potential outcome at treatment level t as

$$\widehat{E[Y(t)]} = \frac{1}{N}\sum_{i=1}^{N}(\hat{\alpha}_0 + \hat{\alpha}_1 t + \hat{\alpha}_2 t^2 + \hat{\alpha}_3 \hat{r}(t, X_i) + \hat{\alpha}_4 \hat{r}(t, X_i)^2 + \hat{\alpha}_5 t\hat{r}(t, X_i)) \quad (8)$$

    b. Repeat the calculation for each level of the treatment we are interested in, to obtain an estimate of the entire dose-response function.

4 Data Analysis

In this section, we conduct a short analysis with the previously proposed method of estimating the effects of continuous treatments using data from Math Nation provided by Walter Leite in Propensity Score Methods for Continuous Treatment Doses. We aim to replicate the analysis in the book while offering additional insights to enhance our understanding of this particular implementation of the Generalized Propensity Score.

4.1 Background

Math Nation (formerly known as Algebra Nation in 2013) is a virtual learning platform that aimed to improve Algebra I achievement in Florida. Florida's Algebra I End-of-Course (EOC) assessment is a mandatory standardized test administered to students who recently completed their Algebra I course. The introduction of the Algebra I assessment in the 2011-2012 academic year marked a significant step in the state's educational reform initiatives. Algebra I serves as a crucial foundation for higher-level math courses that often pave the way to promising career paths with notable growth potential. By enhancing students' proficiency rates in this subject, the assessment plays a vital role in shaping their long-term career trajectories. The data obtained from the platform presents us with opportunities to assess the impact of virtual learning environments on teaching and learning outcomes. "The study's population consisted of high schools, middle/high schools, and senior high schools in all school districts in Florida. The data was collected from February to April 2014 and contains observations for 448 schools." - Walter Leite. The purpose of this example is to assess the impact of school engagement with the Math

Nation virtual learning platform on the average student scores in Florida's Algebra I End-of-Course (EOC) assessment at the school level.

4.2 Procedures

We established in the previous section that we are interested in school-level means of student scores for spring 2014 Algebra I EOC exam as our outcome measure. The treatment dose defined in the example is the ratio of number of logins and number of examinees in each school. The total number of logins for a school includes both teacher and student logins from all sources, while the number of examinees refers to the count of students from that school who participated in the Algebra I EOC assessment during the spring of 2014. We also have 13 covariates that are related to treatment dosage and the outcome. The covariates encompass various factors, such as the school-level mean scores on the Algebra I EOC exam in 2012, student enrollment numbers, the percentage of students eligible for free lunch, the percentage of students eligible for reduced-price lunch, and binary indicators representing whether the school was classified as a charter, magnet, or Title I institution in 2012.

In order to obtain the Generalized Propensity Score, we will first regress our treatment variable with the covariates. Since the method proposed by Hirano and Imben (2004) employ parametric assumptions on $\beta(t,r)$, we must make sure that the distribution $T_i|X$ satisfy normality assumptions as well. The original distribution of logins per examinee was highly skewed and a log transformation was applied to carry out the rest of the analysis.

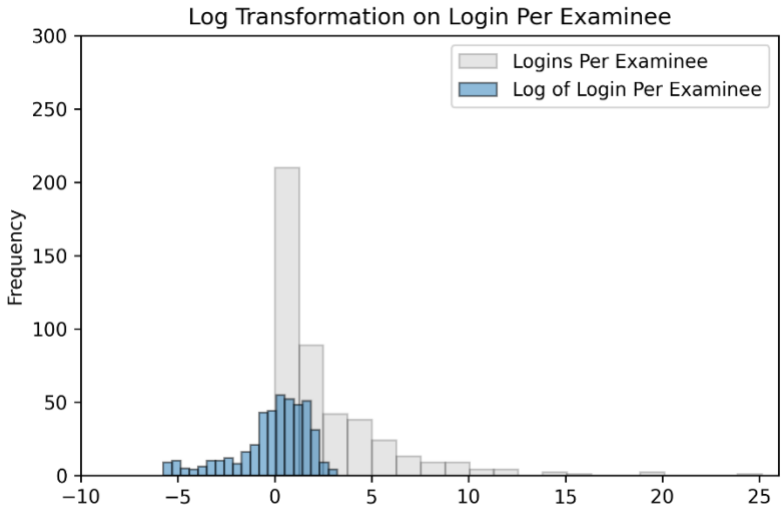

Figure 1. Before and after applying Log Transformation onto Logins Per Examinee

After completing the first step of modeling the continuous treatment variable as a function of covariates, we used the conditional Gaussian density described in (6) to calculate the GPS. An approach to evaluate our covariate balance involves stratifying by GPS and conducting separate regressions for each covariate. “In each regression, the treatment dose is the outcome variable while the GPS strata and covariates serve as the predictors. The standardized regression coefficients can then be utilized as a measure of the effect size of covariates on treatment dose” (Leite, 2019). We see in Table 1 below, that the covariate balancing has improved compared to

baseline covariates but not all achieved the desired level of balance (<0.1). The covariate balance results could be improved if we introduce a different GPS model, but we will continue the rest of the steps using the same model for this demonstration.

Table 1. Standardized Coefficients of Regressions of Treatment Dose on Covariates: There is significant improvement in covariate balance. Only Charter, Magnet, and location size did not achieve desired level of balance.

Variables	Baseline	GPS Strata
Charter	0.322	0.346
Magnet	0.460	0.319
Title_I_School	0.105	0.024
locationRural	0.322	0.034
locationSize	0.398	0.215
Students	0.000	0.000
SeniorHigh	0.064	0.000
numOfStud2014	0.106	0.038
meanScale2012	0.117	0.077
lev1Perc2012	0.108	0.064
lev5Perc2012	0.084	0.093
perc_free_lunch	0.037	0.015
perc_reduced_lunch	0.056	0.067

With the assistance of the *survey* package in R, we estimated the outcome model using the identical formulation described in (7). It is crucial to emphasize that this model lacks any causal interpretation and serves solely as an intermediate step towards deriving individual treatment effects. Lastly, leveraging the same package, we acquire the individual treatment effects outlined in (8), commonly referred to as the average potential outcomes at treatment dose T based on the coefficients of the outcome model.

4.3 Results

We calculated estimates for the average treatment effects across the range of treatment doses from the 1st percentile to the 100th percentile.

Table 2 shows the treatment dose effects and confidence intervals corresponding to every 10^{th} percentile treatment dosage.

Percentile	logLoginsPerExaminee	meanScale2014	SE	lowerCL	upperCL
10%	-2.9014	395.2411	2.6808	389.9867	400.4955
20%	-1.2459	397.0403	0.9809	395.1177	398.9629
30%	-0.5532	397.5792	1.0230	395.5741	399.5844
40%	-0.1112	397.8572	1.0491	395.8010	399.9133
50%	0.2966	398.0680	1.0185	396.0717	400.0643
60%	0.6664	398.2214	0.9445	396.3701	400.0727
70%	1.0840	398.3514	0.8429	396.6992	400.0035
80%	1.4577	398.4288	0.8179	396.8257	400.0318
90%	1.8349	398.4697	0.9671	396.5742	400.3651
100%	3.2243	398.2975	3.0402	392.3387	404.2563

"The treatment dose effects can be interpreted as the expected values of the outcome if all the participants received each specific dose of treatment. " (Leite, 2019). By comparing the confidence intervals of treatment effects, it is possible to determine if the difference between two doses is statistically significant. It appears that all confidence intervals overlap, suggesting that there are no significant distinctions between the doses. Upon plotting the dose response function, we observed that the confidence intervals in the middle of the plot were narrower. This narrower width indicates a higher level of certainty regarding the treatment effects for those specific doses. The reason behind this increased certainty is the availability of greater number of schools at the middle region of the distribution, which facilitates more accurate estimation of the treatment effects.

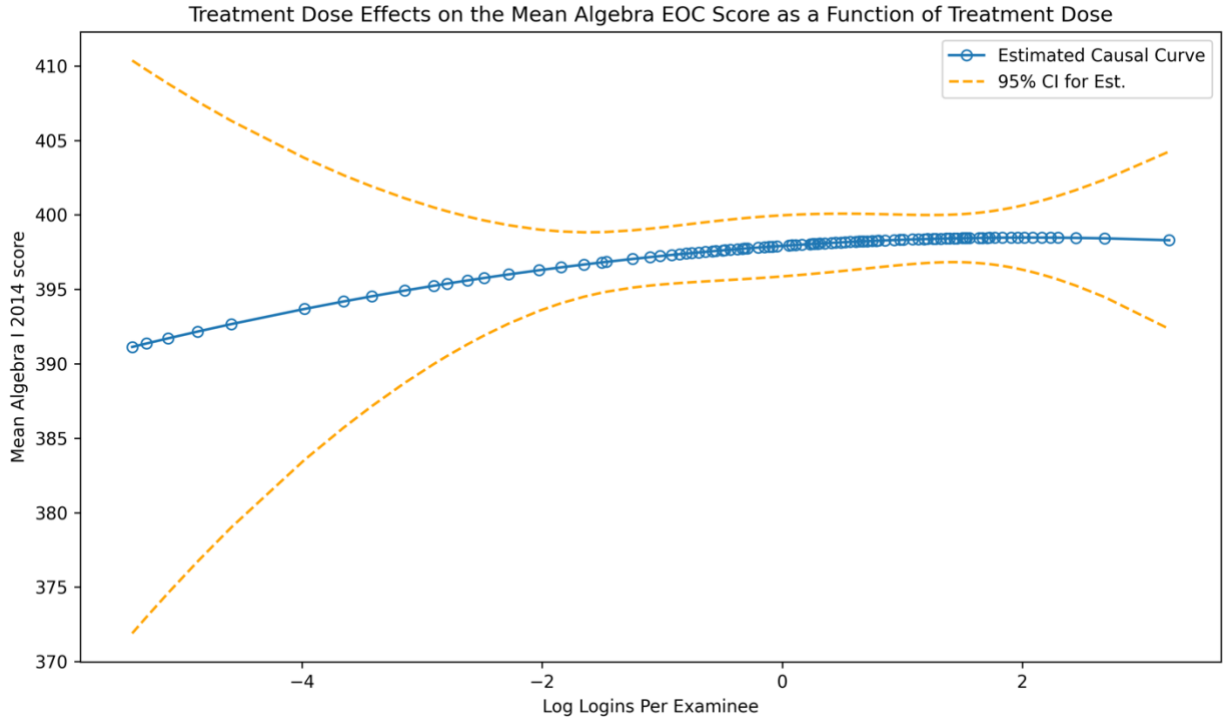

Figure 2. Dose Response function (Treatment vs Outcome): notice narrower confidence interval in the middle indicating more certainty due to having more schools available to estimate the treatment dose effects at that range. We may also see that increase in treatment dose increase our mean average score.

As mentioned above, we followed Hirano and Imben's exact method to estimate the dose response function including using the quadratic terms during the third step of modeling outcome. Here, we provide the results of modeling outcome if we only use linear terms.

Table 3 shows the treatment dose effects and confidence intervals corresponding to every 10^{th} percentile treatment dosage. This time using $E[Y_i|T_i, R_i] = \alpha_0 + \alpha_1 T_i + \alpha_2 R_i$ to estimate the outcome model. There are still overlapping in the intervals indicating no significant distinctions between doses.

Percentile	logLoginsPerExaminee	meanScale2014	SE	lowerCL	upperCL
10%	-2.9014	396.1257	1.0145	394.1372	398.1141
20%	-1.2459	396.8616	0.6300	395.6268	398.0965
30%	-0.5532	397.1696	0.5374	396.1163	398.2228
40%	-0.1112	397.3661	0.5177	396.3514	398.3807
50%	0.2966	397.5473	0.5312	396.5063	398.5884
60%	0.6664	397.7117	0.5680	396.5985	398.8250
70%	1.0840	397.8974	0.6324	396.6579	399.1369
80%	1.4577	398.0635	0.7054	396.6810	399.4461
90%	1.8349	398.2312	0.7895	396.6838	399.7787
100%	3.2243	398.8489	1.1501	396.5947	401.1031

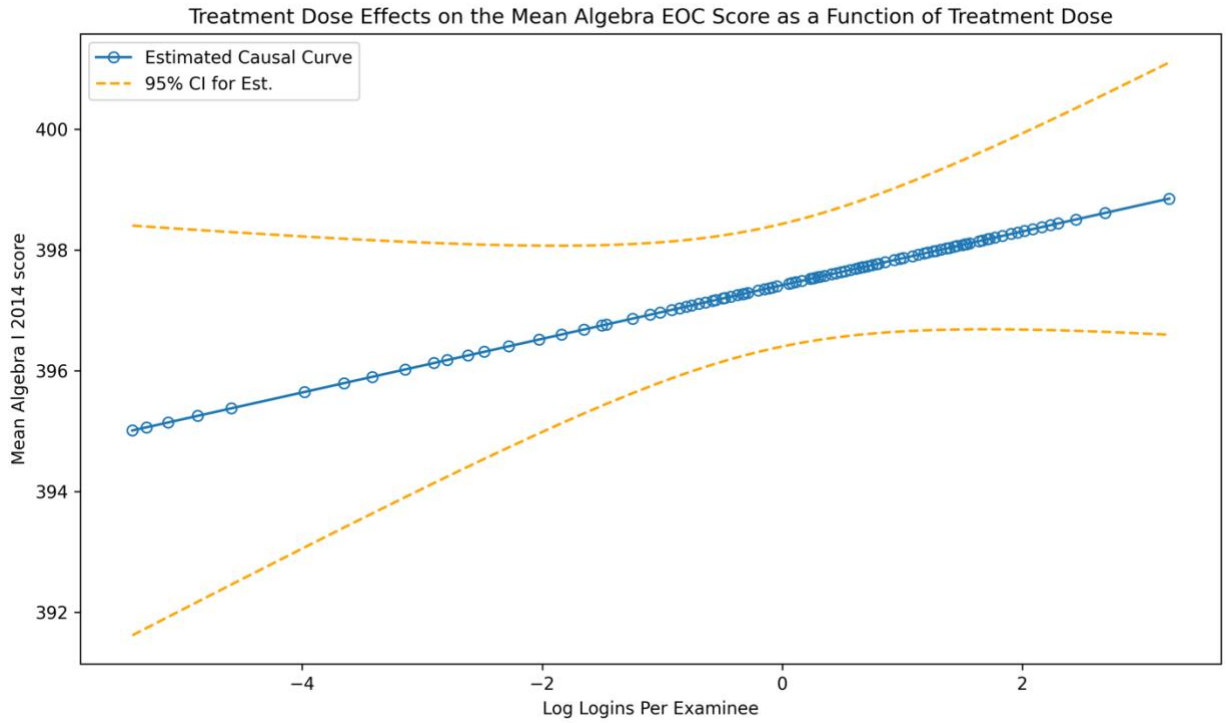

Figure 3. Dose Response function (Treatment vs Outcome): notice compared to Figure 2; the middle section has become wider but the confidence interval at the ends are smaller. Since linearity assumption holds for the model without quadratic term, we believe this is a better model.

5 Discussion and Conclusion

In conclusion, we have summarized Hirano and Imben's paper on Generalized Propensity Score and explained the main ideas of why the proposed method works. Generalized Propensity Score extends the nice properties of propensity scores from the binary treatment case to continuous treatment situations. Although this paper is more related to formally defining GPS, as briefly mentioned by Hirano and Imben, there are other methods that utilize the GPS such as the Marginal Structural Model (MSM). We also recreated the example from Leite (2019) to demonstrate Hirano and Imben's parametric approach to using GPS to estimate causal effects. Overall, this report has shed light on the significance of the Generalized Propensity Score and its potential for improving causal effect estimation in various research contexts.

6 References

Hirano, K., & Imbens, G. W. (2004). The Propensity Score with Continuous Treatments. Wiley Online Library. https://scholar.harvard.edu/imbens/files/hir_07feb04.pdf

Leite, W. (2019, December 20). Practical propensity score methods using R. Sage Research Methods. https://methods.sagepub.com/book/practical-propensity-score-methods-using-r/i852.xml

Link to Github: https://github.com/jttsai99/STA250_Final_Project