# Project 4: Causal Inference Algorithms Evaluation Group 4

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## Basic Setup

```
#Test Branch created
if(!require("readr")){
  install.packages("readr")
}
if(!require("tidyverse")){
  install.packages("tidyverse")
if(!require("glmnet")){
  install.packages("glmnet")
if(!require("pryr")){
  install.packages("pryr")
if(!require("viridis")){
  install.packages("viridis")
}
library(viridis)
library(readr)
library(tidyverse)
library(glmnet)
library(pryr)
lowDim_raw <- read_csv('../data/lowDim_dataset.csv')</pre>
highDim_raw <- read_csv('../data/highDim_dataset.csv')</pre>
lowDim <- lowDim_raw</pre>
highDim <- highDim_raw
```

## **Project Overview**

Since the 1900's, associations between tobacco, lung cancer and cardiovascular diseases have been made. Many reports have asserted that tobacco users would live shorter lives, or that tobacco would be a root cause of lung cancer having higher impact on men than women. However, associations had just been made, and causality had never been proven.

This is a concrete example where causal inference is crucial in every-day life. To prove a cause-to-effect relation of a particular treatment, two main studies are conductible: observational and experimental studies.

In experimental studies, it is easy to impose that the probability of being treated or untreated to be the same. Therefore, a subject's treatment status is random. This equal probability of treatment enables to make a crucial assumption for causal inference, which is that the treated and control groups are alike on all factors except their treatment. This assumption is called the exchangeability between groups. However, there are many times when experimental studies are not feasible or not ethical.

Therefore, most of causal inference studies conduct observational studies instead of experimental ones. However, one of the biggest challenges with observational studies is that the probability of being treated and untreated is not random.

Hence, to use observational studies and satisfy the exchangeability condition, the propensity score metric is introduced. The propensity score is a probability, the conditional probability of receiving a treatment given a set of covariates. Using the propensity score computed for each subject regardless of their actual exposure to a treatment to match treated and untreated subjects with similar scores can help mimic experimental data from observational studies.

For the purpose of this project, we are given low and high dimensional data, where coviariates are already predefined for each individual. After evaluating the propensity scores based on logistic regression for each individual, we will evaluate four inference algorithms, which are Inverse Propensity Weighting (IPW) + Logistic Regression, Regression Estimate, Stratification + Logistic Regression, and Regression Adjustment + Logistic Regression. Using these algorithms on the two distinct datasets (low dimensional and high dimensional), we will compute the Average Treatment Effect (ATE) and will compare their performance and computational efficiency.

## **Propensity Scores**

By definition, a **propensity score** is the probability of a unit being assigned to a particular treatment given a set of observed covariates, and the purpose to use propensity scores is to reduce selection bias by equating groups based on those covariates. In this project, we estimate the propensity scores using logistic regression:

$$logit[Pr(T=1|X)] = \beta_0 + \beta_1 x_1 + \dots + \beta_p x_p$$
$$Pr(T=1|X) = \frac{1}{1 + e^{-(\beta_0 + \beta_1 x_1 + \dots + \beta_p x_p)}}$$

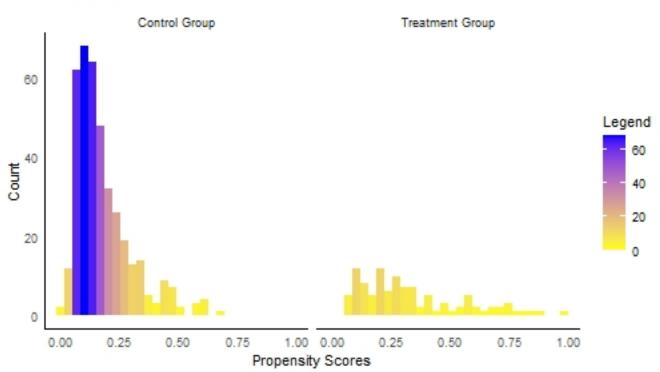
#### Getting PS

In R, we used the **glm** function to build the logistic regression model based on both High-Dimensional and Low-Dimensional datasets. Then, based on the logistic regression model obtaines, we estimated propensity scores for all individuals present in both datasets. Finally, we conducted some exploratory data analysis on the propensity scores distribution among each treated and control groups of each low and high dimensional data.

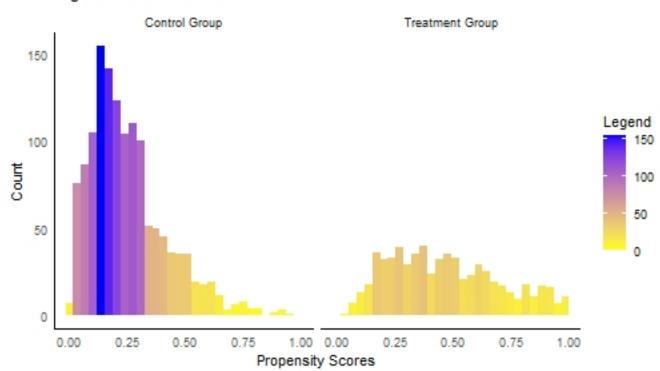
```
# High Dimentional Data
ps_high_estimate <- glm(data = highDim,</pre>
                         formula = A \sim . -Y,
                         family=binomial())
ps_high_data <- data.frame(ps = predict(ps_high_estimate,type="response"),</pre>
                            treatment = ps_high_estimate$model$A)
# Low Dimentional Data
ps_low_estimate <- glm(data = lowDim,</pre>
                        formula = A \sim . -Y,
                        family=binomial())
ps_low_data <- data.frame(ps = predict(ps_low_estimate,type="response"),</pre>
                           treatment = ps low estimate$model$A)
# Show PS means
(ps_means <- data.frame(high_treat_ps_mean = mean(ps_high_data[ps_high_data$treatment==1,]$ps),
                        high_control_ps_mean = mean(ps_high_data[ps_high_data$treatment==0,]$ps),
                        low_treat_ps_mean = mean(ps_low_data[ps_low_data$treatment==1,]$ps),
                        low control ps mean = mean(ps low data[ps low data$treatment==0,]$ps)))
    high treat ps mean high control ps mean low treat ps mean
##
## 1
              0.4663911
                                    0.2528449
                                                       0.3290442
##
    low control ps mean
## 1
               0.1805109
```

Visualize PS

## Low Dimentional Data



# High Dimentional Data



First, it is important to mention that high-dimensional data and low dimensional data have the same distribution for propensity scores regarding both treatment and control groups. For the high-dimensional data, there is a large number of individuals within the control group with propensity score around 0.1, rather than the treatment group have a uniform distribution. Samely, for the low-dimensional data, the control group follows a gaussian-like distribution around 0.10, rather than the treatment group is more uniform across propensity scores. Therefore, in order to evaluate the average true effect of the treatment on these populations, we need to reduce the bias present in the data by using several different algorithms presented below.

## Algorithm 1: Inverse Propensity Weighting and Logistic Regression

Inverse Propensity score is one method of propensity score weighting, which is is an alternative way to propensity score matching. It can effectively remove the bias by re-weighting the data with weights inversely proportional to the probability of selection. Averaging with known inverse sampling weights was introduced by Horvitz and Thompson in 1952 and has been further studied in recent KDD papers.

$$w_i = \frac{T_i}{\hat{e}_i} + \frac{1 - T_i}{1 - \hat{e}_i}$$

the sum of the weights over the controls equals one. where  $\hat{e}_i$  is the estimated propensity score for individual i.

Estimate ATE:

## ATE

## Running Time (secs)

$$\hat{\triangle}_{IPW} = N^{-1} \left( \sum_{i \in Treated} w_i Y_i - \sum_{i \in Controlled} w_i Y_i \right)$$

where the first summation is from the treated group, and the second summation is from the controlled group.

This method presents the advantage of using all the data available, and being very efficient in reducing the bias of the data. However, the main drawback of this method is that the IPW will have high variance if p (X) is close to one for some controls. The propensities close to one arise if X nearly separates the controls and exposed or the algorithm used to estimate the propensities is unstable.

```
set.seed(0)
# Write Algorithm
IPW <- function(df,ps){</pre>
  start <- Sys.time()</pre>
  ps['weights'] \leftarrow df$A/ps$ps+(1-df$A)/(1-ps$ps)
  treatment <- sum(ps[ps$treatment==1,]$weights*df$Y[df$A==1])</pre>
  controll <- sum(ps[ps$treatment==0,]$weights*df$Y[df$A==0])</pre>
  ATE <- (treatment-controll)/nrow(df)
  end <- Sys.time()</pre>
  runtime = end - start
  return(list(ATE=ATE,runtime=runtime))
}
# Output Performance
matrix(c(IPW(highDim,ps_high_data)$ATE,
         IPW(lowDim,ps_low_data)$ATE,
         IPW(highDim,ps_high_data)$runtime,
         IPW(lowDim,ps_low_data)$runtime),
       nrow = 2,byrow = TRUE,
       dimnames = list(c("ATE", "Running Time (secs)"), c("High Dimension", "Low Dimension")))
##
                        High Dimension Low Dimension
```

1.689329

0.000000

-57.573028669

0.001991987

## Algorithm 2: Regression Estimate

In this section, we estimated ATE by using a simple regression, not based on the propensity score. The formulas below were exctracted from the research paper written by Chan et al. (2010),

$$\hat{\triangle}_{reg_{ATT}} = n_1^{-1} \sum_{exposed} (\hat{m}_1(X_i) - \hat{m}_0(X_i)), n_1 = \sum_{i=1}^n Z_i$$

The formula above is used for ATT calcultions. However, for our purpose, we are more intereste in computing the Average True Effect - ATE, which is why we used the formula below instead:

$$\hat{\triangle}_{reg_{ATE}} = N^{-1} \sum_{i=1}^{N} (\hat{m}_1 - \hat{m}_0(X_i))$$

where  $\hat{m}_1$  is the model that using A=1 (Treatment Group), and  $\hat{m}_1$  is the model that using A=0 (Controlled Group). This algorithm has the best performance in terms of the lowest squared error, but it will take more computational cost.

```
set.seed(0)
# Write Algorithm
Regression_Estimate <- function(df){</pre>
  start <- Sys.time()</pre>
  model_0 <- glm(Y ~ ., data = subset(df[df$A==0,], select = -A))</pre>
  model_1 <- glm(Y ~ ., data = subset(df[df$A==1,], select = -A))</pre>
  ATE = 1/nrow(df) * sum(predict(model_1, newdata = df%>% select(-Y,-A)) -
                            predict(model_0, newdata = df%>% select(-Y,-A)))
  end <- Sys.time()</pre>
  runtime = end - start
  return(list(ATE = ATE, runtime = runtime))
# Output Performance
matrix(c(Regression Estimate(highDim)$ATE,
         Regression_Estimate(lowDim)$ATE,
         Regression_Estimate(highDim)$runtime,
         Regression_Estimate(lowDim)$runtime),
         nrow = 2,
         byrow = TRUE
         dimnames = list(c("ATE", "Running Time (secs)"), c("High Dimension", "Low Dimension")))
##
                        High Dimension Low Dimension
## ATE
                           -57.4265873
                                           2.12513807
## Running Time (secs)
                             0.3098102
                                           0.02273798
```

## Algorithm 3: Stratification and Logistic Regression

$$\hat{\triangle}_S = \sum_{i=1}^K \frac{N_j}{N} (N_{1j}^{-1} \sum_{i=1}^N T_i Y_i I(\hat{e}_i \in \hat{Q}_j) - N_{0j}^{-1} \sum_{i=1}^N (1 - T_i) Y_i I(\hat{e}_i \in \hat{Q}_j))$$

where K is the number of strata (K=5).  $N_j$  is the number of individuals in stratum j.

 $N_{1j}$  is the number of "treated" individuals in stratum j, and  $N_{0j}$  is the number of "controlled" individuals in stratum j.

 $\hat{Q}_j = (\hat{q}_{j-1}, \hat{q}_j)$  where  $\hat{q}_j$  is the jth sample quantile of the estimated propensity scores.

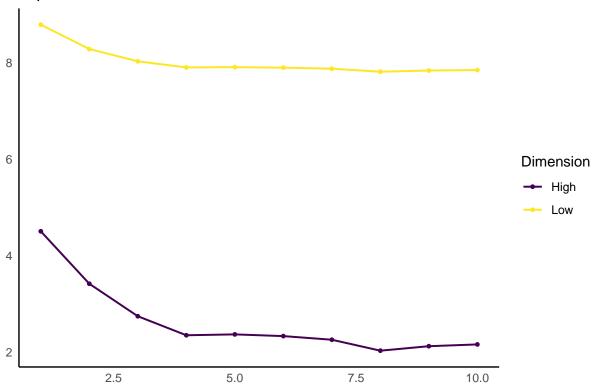
Estimation of treatment effects with causal interpretation from raw data is hard because exposure to treatment may be confounded with subject characteristics. Therefore, we need to use stratification to adjust for confounding.

In our implementation, we can select k where k represented the number of the strata. With the help of strata, we can fist cauculate the data which have similar propeties from the propensity score aspect. We also tested which K value in the formula above has the best performance. In the last part of this section, from the graph, we can see that K=8 should provide the best result (lowest squared error for both high and low dimensional datasets).

```
set.seed(0)
# Write Algorithm
Strat <- function(df, ps, k=8){
  start = Sys.time()
  ATE <- 0
  prev <- 0
  cancatdata <- cbind(df, ps)</pre>
  for (i in 1:k) {
    ps_str = as.numeric(ps$ps)
    str_q <- quantile(ps_str, (1/k)*i)</pre>
    str_data <- cancatdata[cancatdata$ps>=prev,]
    str_data <- str_data[str_q>=str_data$ps,]
    prev = str_q
    temp_t <- sum(str_data$Y[str_data$A==1])</pre>
    temp_t <- 1/nrow(str_data[str_data$A==1,])*temp_t</pre>
    temp_c <- sum(str_data$Y[str_data$A==0])</pre>
    temp_c <- 1/nrow(str_data[str_data$A==0,])*temp_c</pre>
    str_e <- temp_t - temp_c</pre>
    ATE <- ATE + str_e*nrow(str_data)/nrow(df)
  end = Sys.time()
  runtime = end - start
  return(list(ATE = ATE, runtime = runtime))
# Output Performance
stra_result_high <- matrix(0, nrow = 10, ncol = 4,
                     dimnames = list(c("1","2","3","4","5","6","7","8","9","10"),
                                  c("K", "ATE", "Running Time (secs)", "Sq Error")))
stra_result_low <- matrix(0, nrow = 10, ncol = 4,</pre>
                     dimnames = list(c("1","2","3","4","5","6","7","8","9","10"),
                                  c("K","ATE","Running Time (secs)","Sq_Error")))
for (i in 1:10){
  stra_result_high[i,] <- c(i,</pre>
                              Strat(highDim,ps_high_data, i)$ATE,
                              Strat(highDim,ps_high_data, i)$runtime,
                              sqrt(abs(-54.8558-Strat(highDim,ps_high_data, i)$ATE)))
  stra_result_low[i,] <- c(i,</pre>
                            Strat(lowDim,ps_low_data, i)$ATE,
                            Strat(lowDim,ps_low_data, i)$runtime,
                            sqrt(abs(2.0901-Strat(highDim,ps_high_data, i)$ATE)))
}
```

```
## High Dimension Low Dimension
## ATE -58.98685168 2.02639488
## Running Time (secs) 0.09974003 0.01193094
```

#### Squared Error of Different Ks in Stratification



# Algorithm 4: Regression Adjustment and Logistic Regression

Regress the outcome variable Y on treatment indicator variable T and the estimated propensity score. Then, the estimated coefficient on the treatment indicator variable would be an estimate of ATE. In this method, we regress the response variable (Y) with the treatment variable (A) and the propensity scores estimated using our model above, in this case, logistic regression. The estimated coefficient of the treatment variable (A) is then an estimate of the ATE.

D'Agostino (1998) and Austin (2011) compare regression adjustment with more traditional propensity score methods. One of the main advantages of the regression adjustment is in its simplicity in execution, in which one performs a somewhat basic linear regression model on two covariates and one response variable.

However, depending on the size of the dataset, this may run into computation issues as linear regression involves finding the inverse of a matrix. Additionally, regression adjustment may also not be helpful in cases where there is a strong separation between the two groups.

No such issues were present in this setup given that both datasets had a relatively small number of observations and there is no clear separation between the two groups

```
set.seed(0)
# Write Algorithm
Reg_adj <- function(df,ps_data){</pre>
  df<- data.frame(cbind(Y=df$Y,A=df$A,ps=ps_data$ps))</pre>
  start <- Sys.time()</pre>
  m < -lm(Y \sim A + ps, data = df)
  ATE = m$coefficients[2]
  end <- Sys.time()</pre>
  runtime = end - start
  return(list(ATE = ATE, runtime = runtime))
}
# Output Performance
matrix(c(Reg_adj(highDim,ps_high_data)$ATE,
         Reg adj(lowDim,ps low data)$ATE,
         Reg_adj(highDim,ps_high_data)$runtime,
         Reg_adj(lowDim,ps_high_data)$runtime),
       nrow = 2,
       byrow = TRUE,
       dimnames = list(c("ATE", "Running Time (secs)"),
                        c("High Dimension","Low Dimension")))
```

```
## ATE High Dimension Low Dimension
## ATE -59.416700238 2.418086454
## Running Time (secs) 0.001967907 0.003038168
```

## **Model Comparisons**

Performance is measured by the squared differences of true ATEs and estimated ATEs for all four algorithms, and run-times are measured in seconds.

## Low Dimension Dataset

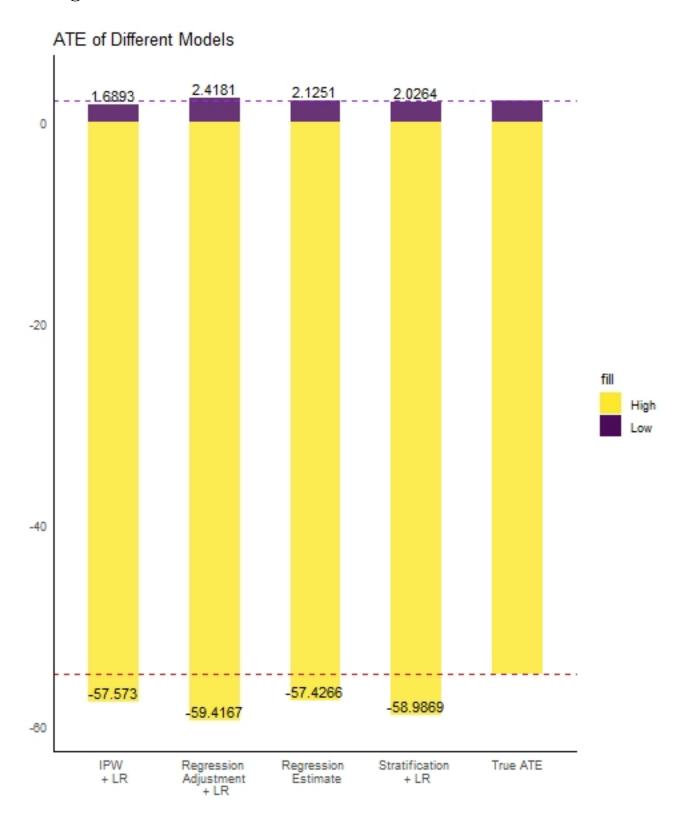
Algorithm	ATE	Run_Time	Squared_Error
True ATE	2.0901	N.A	N.A.
IPW + LR	1.6893	0	0.6331
Regression Estimate	2.1251	0.02094102	0.1872
Stratification + LR	2.0264	0.01596093	0.2524
Regression Adjustment $+$ LR	2.4181	0.00199199	0.5727

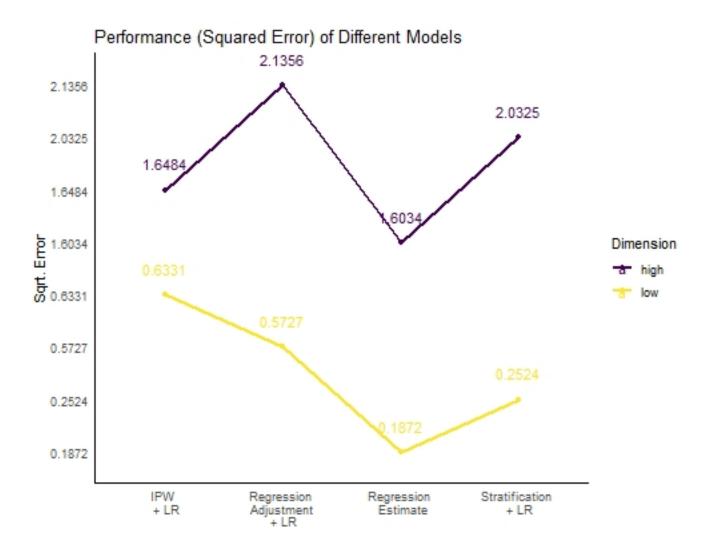
## **High Dimension Dataset**

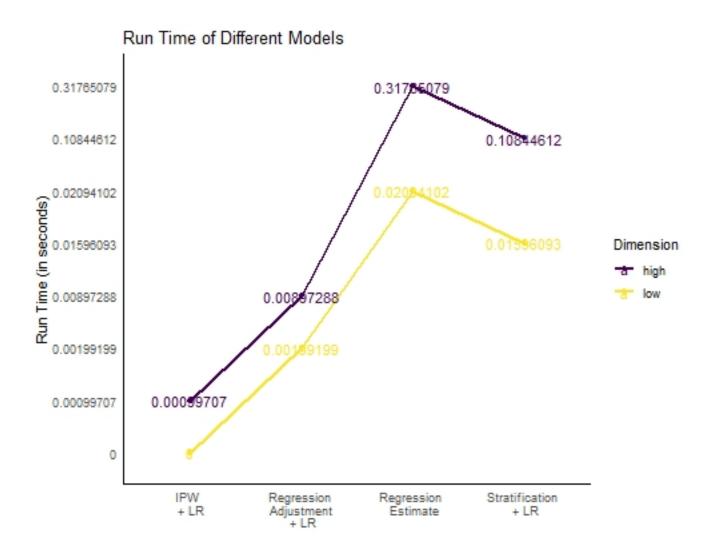
Algorithm	ATE	Run_Time	Squared_Error
True ATE	-54.8558	N.A	N.A.
IPW + LR	-57.5730	0.00099707	1.6484
Regression Estimate	-57.4266	0.31765079	1.6034
Stratification + LR	-58.9869	0.10844612	2.0325
Regression Adjustment $+$ LR	-59.4167	0.00897288	2.1356

From the tables above, we can find that Regression Estimate has the best performance on both Low and High Dimensional Datasets in terms of the lowest squared errors in both cases.

# Plotting the Result







#### Conclusion

Among four different algorithms, IPW (with Logistic Regression (LR)), Regression Estimate, Stratification (with LR), and Regression Adjustment (with LR), Regression Estimate has the best performances with both high and low dimension datasets since it has the lowest squared errors in both cases. However, Regression Adjustment algorithm will take more computational cost than other algorithms (longer run-time). Besides, the squared errors for high dimensional dataset are all larger than the squared errors of low dimensional dataset for all four algorithms. In terms of computational cost, for Regression Estimate and Stratification (with LR), the run-times of high dimensional dataset is longer than the run-times for low dimensional dataset, but for Regression Adjustment (with LR) and IPW (with LR), the run-times of both datasets are similar. However, since the run-times for all four algorithms are lower than 0.18 seconds even when calculating the ATE for high dimentianl dataset, we can ignore their differences in computational costs. Therefore, we think Regression Estimate is the best algorithm when estimating the ATE of both datasets.

#### Reference

Austin, Peter C. 2011. "An Introduction to Propensity Score Methods for Reducing the Effects of Confounding in Observational Studies." Multivariate Behavioral Research 46 (3): 399–424.

Chan, David, Rong Ge, Ori Gershony, Tim Hesterberg, and Diane Lambert. 2010. "Evaluating Online Ad Campaigns in a Pipeline: Causal Models at Scale." In Proceedings of the 16th Acm Sigkdd International Conference on Knowledge Discovery and Data Mining, 7–16.

Kuei-Fung Lin and Wei-Chun Huang, 2017. "Propensity score analysis of lung cancer risk in a population with high prevalence of non-smoking related lung cancer". National Library of Medicine.

Lunceford, Jared K, and Marie Davidian. 2004. "Stratification and Weighting via the Propensity Score in Estimation of Causal Treatment Effects a Comparative Study." Statistics in Medicine 23 (19): 2937–60.

Stuart, Elizabeth A. 2010. "Matching Methods for Causal Inference: A Review and a Look Forward." Statistical Science: A Review Journal of the Institute of Mathematical Statistics 25 (1): 1.