ML Techniques to Recover Treatment Effects from Karlan and List (2007) HW1, Econ 293

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1 Setup

Here, we investigate the treatment effect of offering any sort of "match" promise on charitable giving. Specifically, we look at Karlan & List's 2007 AER paper : "Does Price Matter in Charitable Giving? Evidence from a Large-Scale Natural Field Experiment", which randomly selected 2/3 of the population of prior donors of a large charity to recieve one of nine treatments that were a variation of promising to match any donations for a limited time via an outside donor. The control group recieved a similar letter soliciting donations with no promise of a matching donation to theirs.

Our focus is on the average treatment effect (ATE) of promising to match donations on expected dollars donated in response to the solicitation. First, we recreate the average treatment effect documented in the paper. This is column (1) of Table 4 in the paper. We estimate an average treatment effect of an additional \$0.1536 from the matching promise in the solicitation, consistent with the paper. The confidence interval is estimated to be $[-0.008, 0.315]$.We then transform the paper's randomized experiment into an observational study by implementing a selection rule of the sample that depends on an interaction of treatment with baseline covariates of the donors. Specifically, let K_i be an indicator for whether an individual is kept in the sample for the "observational" study, and let $\psi(X_i) = Pr(K_i = 1|X_i)$ be the respondent's "propensity" to be kept in the sample. We define this differentially between donors depending on their assignment to treatment and control:

$$\psi(X_i) = \begin{cases} (.01 + -0.01 * \text{arcsinh}(X_{3,i})^5 + \text{arcsinh}(X_{3,i})^3)/300 & \text{if } W_i = 1 \\ (X_{2,s} + 1) * (\arccos(X_{1,s}) * \arctan(X_{1,s}))/3 & \text{if } W_i = 0 \\ 0.5 & \text{if missing one of } X_{1,s}, X_{2,s}, X_{3,i} \end{cases}$$

*Coding done in colloboration with Jack Blundell

Where W_i is a treatment indicator, $X_{3,i}$ is the highest previous donation by the donor, $X_{2,s}$ is the number of cases the charity undertook in the state s from 2004-05, and $X_{2,s}$ is the state's voting share for George Bush in the 2004 presidential election, and arcsinh is the inverse hyperbolic sine function (e.g. $\text{arcsinh}(X) = \log(X_{3,i} + \sqrt{X_{3,i}^2 + 1})$. Figures 1 and 2 contain plots of this function for treatment/control groups. So a treatment group individual's selection propbability is proportional to the inverse-hyperbolic sine (IHS) of their highest previous donation, while a control group individual's selection probability is proportional to a complex trigonometric function of state characteristics, specifically, the caseload of the organization in the state and their conservative ideological tendency, as measured by the bush vote share. Those with missing covariates are given 50-50 odds of being kept in the sample.

From our selection rule, we can directly use Bayes' rule to back out the true propensity score for treatment, conditional on the covariates,X_i. for the non-random selected subsample. Specifically, the propensity score is:

$$\begin{aligned}\rho(X_i) = P(W_i = 1|K_i = 1) &= \frac{P(K_i = 1|W_i = 1)P(W_i = 1)}{P(K_i = 1|W_i = 1)P(W_i = 1) + P(K_i = 1|W_i = 0)P(W_i = 0)} \\ &= \frac{\psi(X_i|W_i = 1)\frac{2}{3}}{\psi(X_i|W_i = 1)\frac{2}{3} + \psi(X_i|W_i = 0)\frac{1}{3}}\end{aligned}$$

and $\rho(X_i) = \frac{2}{3}$ for those with missing relevant covariates. implicitly

After performing the selection, we are left with 15,169 observations. A naive regression on an intercept and treatment indicator yields a treatment effect of 0.403, so about 3 times larger in magnitude. This is at least in part due to the selection rule of keeping only treatment individuals who in the past donated larger amounts (and hence were more likely to donate large amounts in response to the treatment, regardless of the contents of the solicitation). Overlap is preserved, as is evident in the CDFs of propensity scores for each treatment group (Figure 4).

In order to compute the bias function from Athey et al. (2017), we plug in our recovered propensity scores, along with treatment group outcome means. To calculate the conditional means ($\mu(W, X)$), we calculate outcome means within neighborhoods for each observation of the X-space of relevant covariates $X_{1,s}, X_{2,s}, X_{3,i}$,since our dropout rule selected on continuous covariates, . These neighborhoods are determined by measured by the maximum Mahalanobis distance required to get at least one observation of each treatment group in an X neighborhood. The covariates are normalized to each have mean 0 and SD 1 when calculating these distances. Figure 5 plots the histogram of the bias functions, and shows a clear right skew, as might be expected since our naive estimation vastly overstated the treatment effect. The expected bias is estimated to be 0.044.

2 Recovering the ATE

For code and output details, please see the R markdown document included in the submission. In all of the estimation that follows, we consider the feature space of all the original 48 covariates included in the original dataset, along with two-way interactions between all the 9 individual-level features, and two-way interactions between the 20 state-level features. We do not consider interactions for the 18 missing dummies that are included. The advantage of this choice of features is it is sufficiently complex that it may be able to capture most of the true relationships in the propensity score (such as the positive interaction between vote share and case load), while being sufficiently simple that I can run all of the methods such as OLS and Approximate Residual Balancing on the full feature space, without worrying about intractability. This way, I can compare the methods directly, as opposed to enriching the feature space for regularized regressions and not being sure if they perform better due to the selection algorithm or including the right variables in the choice set.

Our first set of estimations are the "classic" methods for recovering treatment effects via propensity scores / linear regression. Here, we estimate the propensity scores via OLS regression on all covariates, and predicting the probability of treatment $\hat{p}(X_i)$ (censoring it to be between 0 and 1, which is required very infrequently). After estimating these p-scores, we calculate the propensity-weighted ATE as:

$$\widehat{ATE}_{ps} = \frac{1}{N} \sum_i \frac{Y_i(W_i - \hat{p}(X_i))}{\hat{p}(X_i)(1 - \hat{p}(X_i))}$$

which yields an estimate of $\widehat{ATE}_{ps} = 0.269$, a notable improvement over the naive estimate, but still somewhat far away from the true ATE. Conversely, an OLS linear regression of the outcome on the treatment variable plus all the X's in our feature space yields an estimate of $\widehat{ATE}_{OLS} = 0.196$, even better than the previous method. And finally, combining the two above methods by running OLS with inverse propensity score (IPS) weights

$$w_i = \frac{1}{W_i\hat{p}(X_i) + (1 - W_i)(1 - \hat{p}(X_i))}$$

, the traditional double robust methodology, we get an estimate of 0.1751665. Thus, the traditional Double Robust performs extremely well in recovering the original treatment effect estimated randomized experiment.

Next, we use LASSO to estimate the propensity score, selecting the LASSO penalty via 10-fold cross validation (CV) on minimizing mean squared error (MSE), and re-do the propensity score weighted methods discussed in the previous section. For all the LASSO regressions that follow, we follow this procedure by choosing the specification with the lowest MSE (based on 10-fold CV). With our re-estimated propensity scores via regularized regression. Figure 6 contains a density plot of both the LASSO and OLS estimated propensity scores along with the true propensity score recovered from the dropout rule. We see that relative to the true scores, the OLS are too smooth, which is a consequence of

being a weighted mixture of 300 covariates. It does, however, do a good job of capturing the overall mass distribution of the propensity score, despite the highly nonlinear underlying propensity scores. On the other hand, the lasso propensity scores do a relatively good job of capturing the nonlinearities of the propensity scores, as is evident by the large "humps" in the distribution. but it is too "concentrated" relative to the true propensity score distribution, possibly due to including too *few* features in the final specification. Using the lasso propensity scores, we get an estimate of $\widehat{ATE}_{ps} = 0.2764256$ from IPS weighted means, and an estimate of $\widehat{ATE}_{DR} = 0.230039$ from the double robust method of OLS with lasso-estimated IPS weights. While we see here again that the double robust method performs better than just the weighted means, both versions with the regularized propensity scores perform worse than those with propensity scores estimated via OLS. If we do a direct lasso regression on the outcome with a treatment indicator and all of our covariates, while setting the penalty factor on the treatment coefficient to zero [1], we get a treatment effect of $\widehat{ATE}_{LASSO} = 0.2283$. If we look at the path of the treatment effect coefficient as the LASSO penalty increases (as in Figure 7), we see that it increases steadily, until it flattens out around $\lambda = .9$ to the naive OLS estimator (where presumably all other parameters have been zeroed out). It is somewhat intuitive that as lambda increases, the treatment coefficient increases (even though at face value it is counterintuitive since a higher lambda is going to penalize a large coefficient more). The reason may be that as lambda increases, the LASSO is zeroing out more and more features that explain the selection done in the observational study, which is causing the treatment coefficient to improperly increase.

Next, we perform the double-selection procedure outlined in Belloni et al. (2014), where we select features to include in a regression from the union of separate regressions of Y and W on the full set of features. This leads to a treatment effect estimation of $\widehat{ATE}_{DS} = 0.2026$, which is actually noticeably closer to the true ATE than the other methods using regularization, although it still performs worse than both simple OLS and traditional Double robust OLS with IPS weights.

In addition, we perform a double machine learning (lasso residual-on-residual) regression obtained by separate lasso regressions Y and W on the full set of features, choosing the optimal prediction for each separate regularized regression (in terms of MSE), calculate the residuals for both Y and W then regress residual of Y on the residual of W, to obtain an estimate of the treatment coefficient of $\widehat{ATE}_{DML} = 0.2269$, which is quite close to both the traditional DR method with regularized IPS weights, and the direct lasso regression.

Finally, we use the balanceHD package to perform approximate residual balancing on the sample to estimate an average treatment effect. The algorithm performs somewhat poorly ($\widehat{ATE}_{ARB} = 0.2462$), possibly due to the large sample size which makes optimization significantly more difficult.

In general, we see in our concocted observational study a relative advantage

[1] so it is never selected out of the regression equation

in the classic approaches for estimating ATEs, such as OLS and traditional DR, in calculating the true ATE. I think that this is probably a consequence of the relatively low $p/n = 0.02$ ratio, since the relative advantageous of the ML methods discussed in class seem to kick in when p is close to or greater than n. Realistically, a standard OLS regression with 300 controls is not outrageous for a sample of 10,000 observations. This finding I think highlights the fact that one must be careful not to fall into the overpromise of ML algorithms as a "be-all end-all" solution to our traditional data problems. One must pay careful attention to the specific setting of the problem and make sure the ML methods considered are appropriate solutions to the problem at hand, as was evident in this setting, where, despite creating a rich feature space to test out all the methods against, the ML estimations often performed worse than traditional methods.

Table 1: Estimated treatment effects

Estimator	Estimated ATE
(1) Full-sample simple OLS (*'true ATE'*)	0.1536
(2) Restricted-sample Naive OLS	0.4027
(3) PS Weighting	0.2685
(4) OLS with Controls	0.1961
(5) Traditional DR OLS with IPS Weights	0.1752
(6) Regularized PS Weighted	0.2764
(7) Classic Double Robust with Regularized PS	0.2300
(8) Direct Lasso on Outcome	0.2283
(9) Double Selection	0.2026
(10) Lasso Residual-on-Residual	0.2269
(11) Residual balancing	0.2462

Notes: This table shows the various treatment effects estimated throughout this homework

Figure 1:

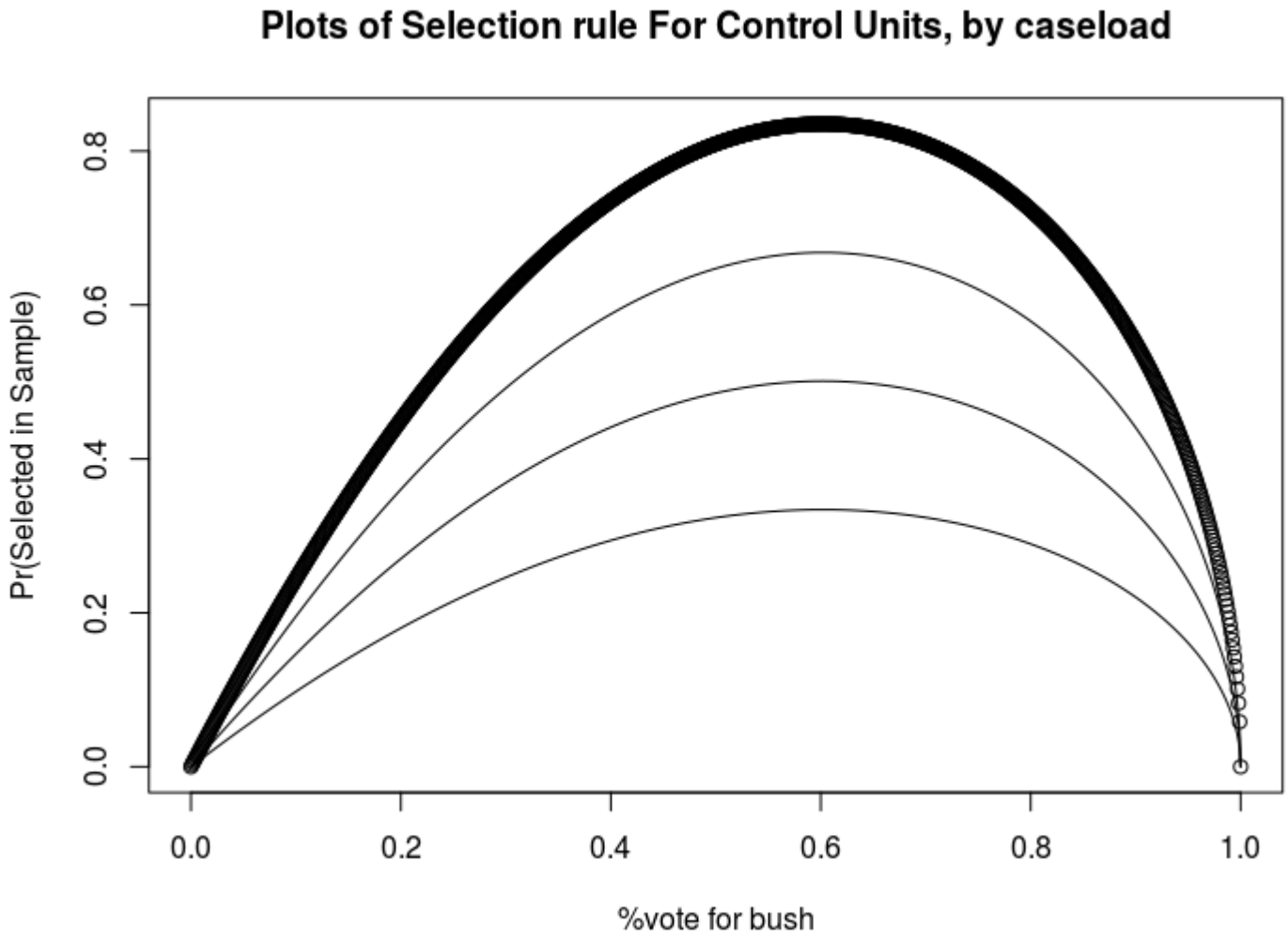
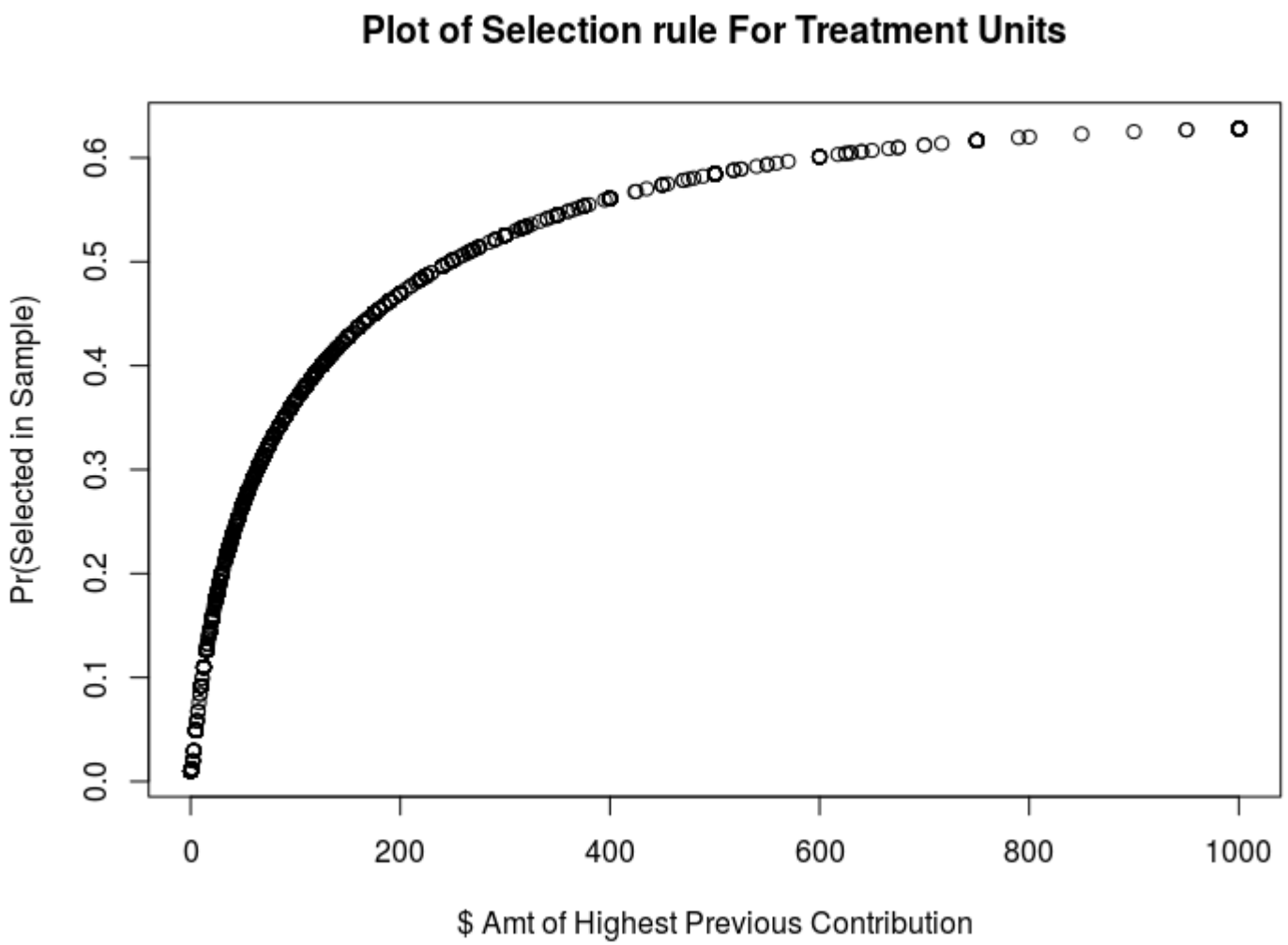

Plots of Selection rule For Control Units, by caseload
Pr(Selected in Sample)
0.0
0.2
0.4
0.6
0.8
0.0
0.2
0.4
0.6
0.8
1.0
%vote for bush

Figure 2:


Plot of Selection rule For Treatment Units
Pr(Selected in Sample)
0.0
0.1
0.2
0.3
0.4
0.5
0.6
0
200
400
600
800
1000
$ Amt of Highest Previous Contribution

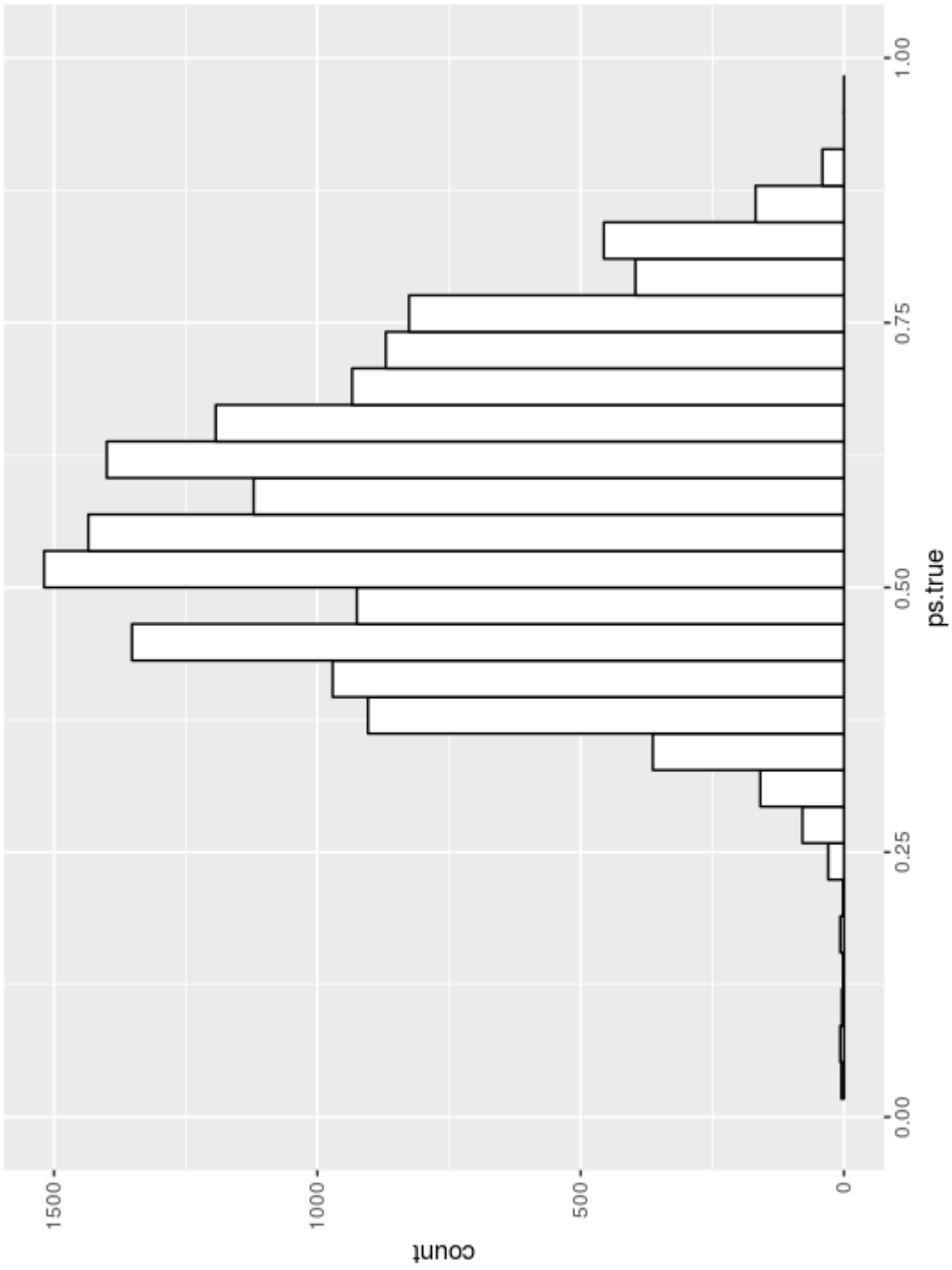
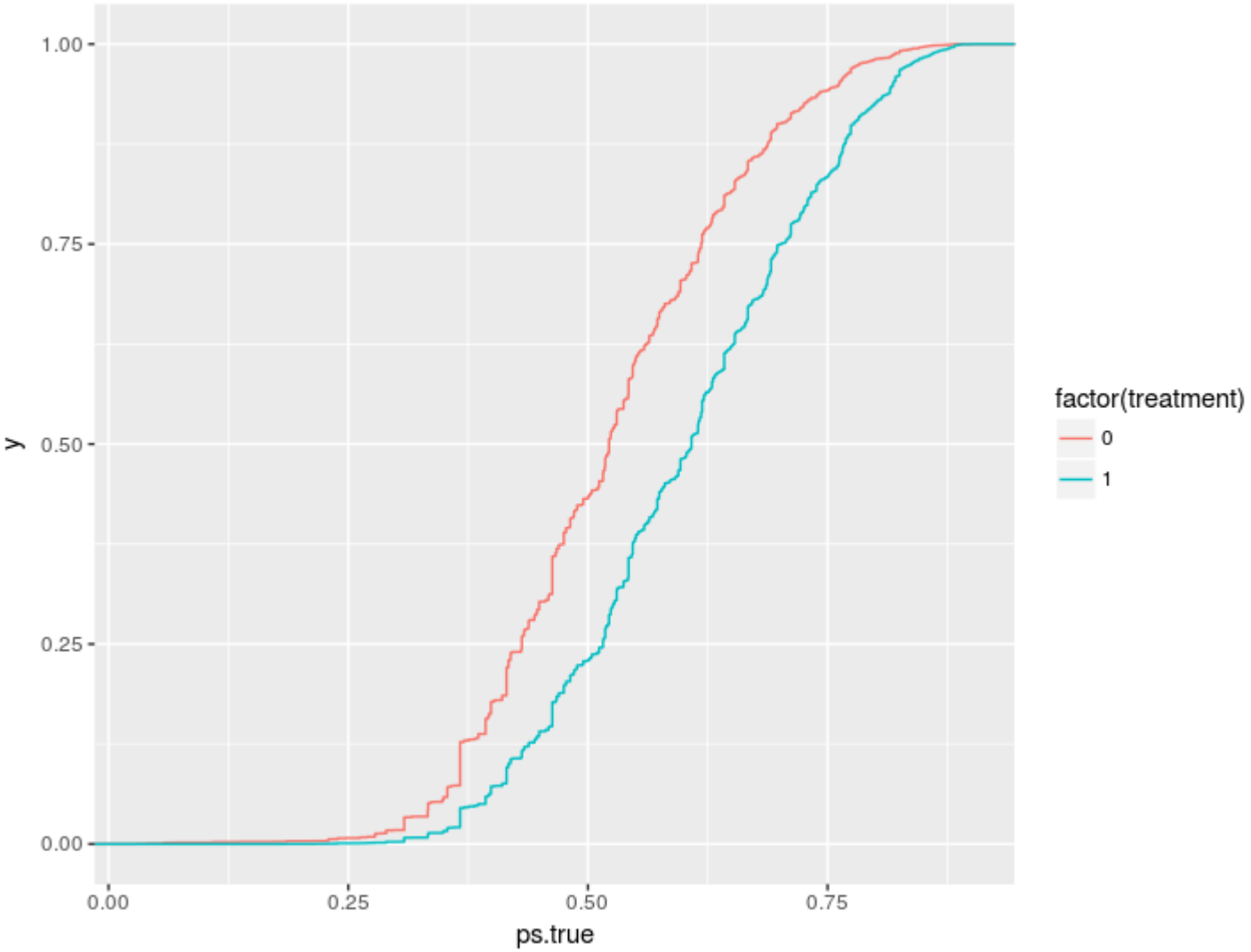


Figure 3: Histogram of propensity scores

Figure 4: Propensity Score Overlap by Treatment Group



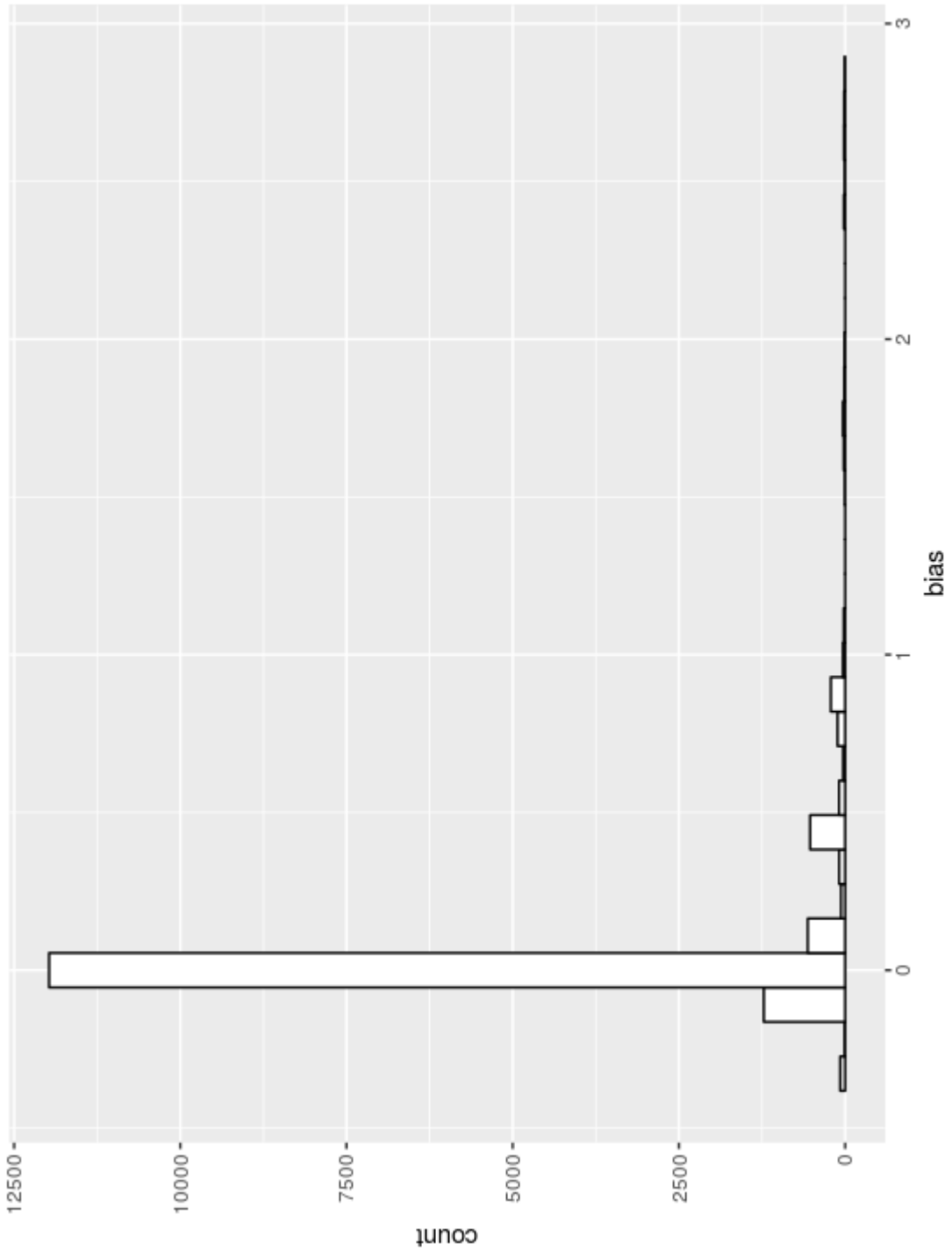


Figure 5: Distribution of bias functions

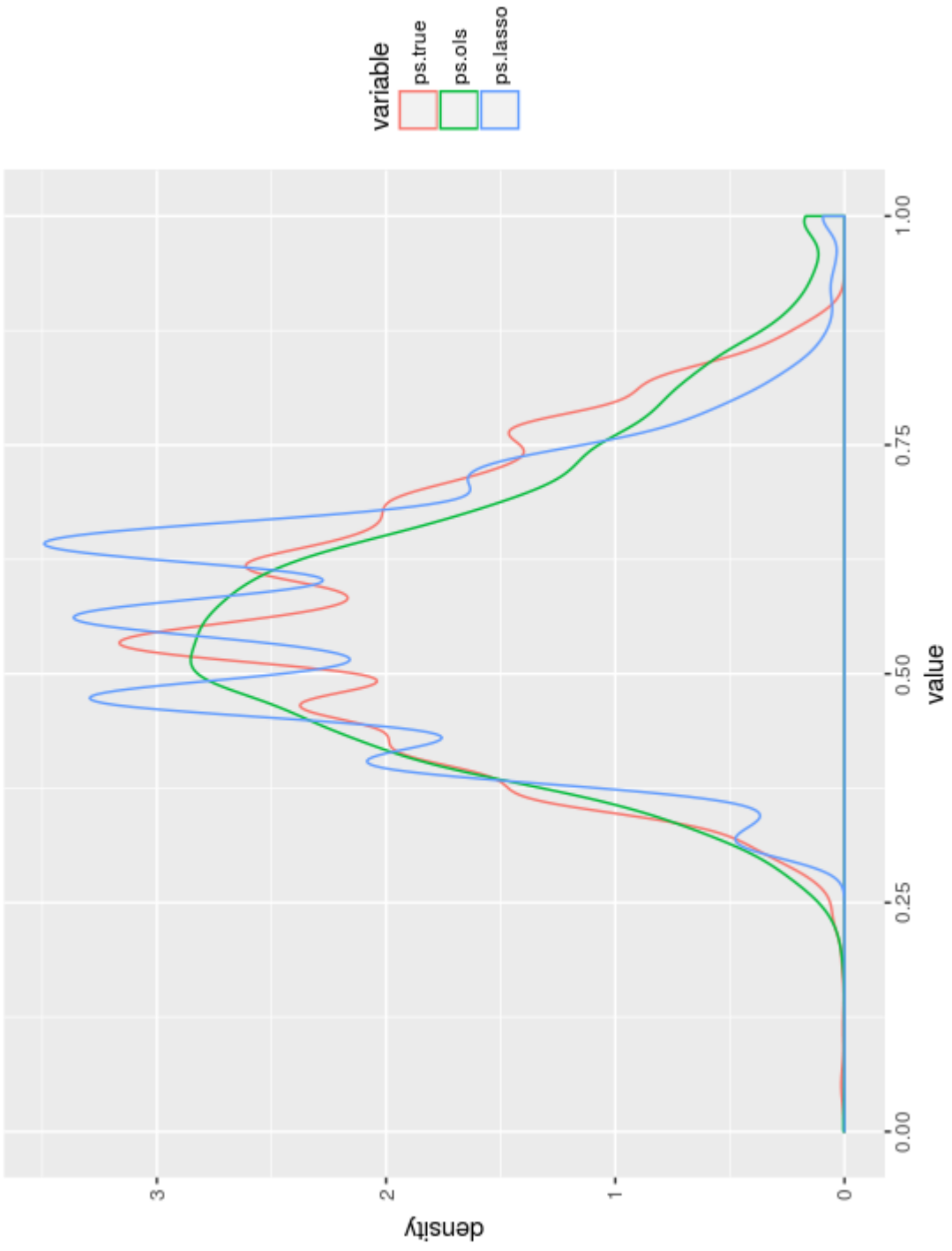


Figure 6: Estimated Propensity Scores and True Propensity Score

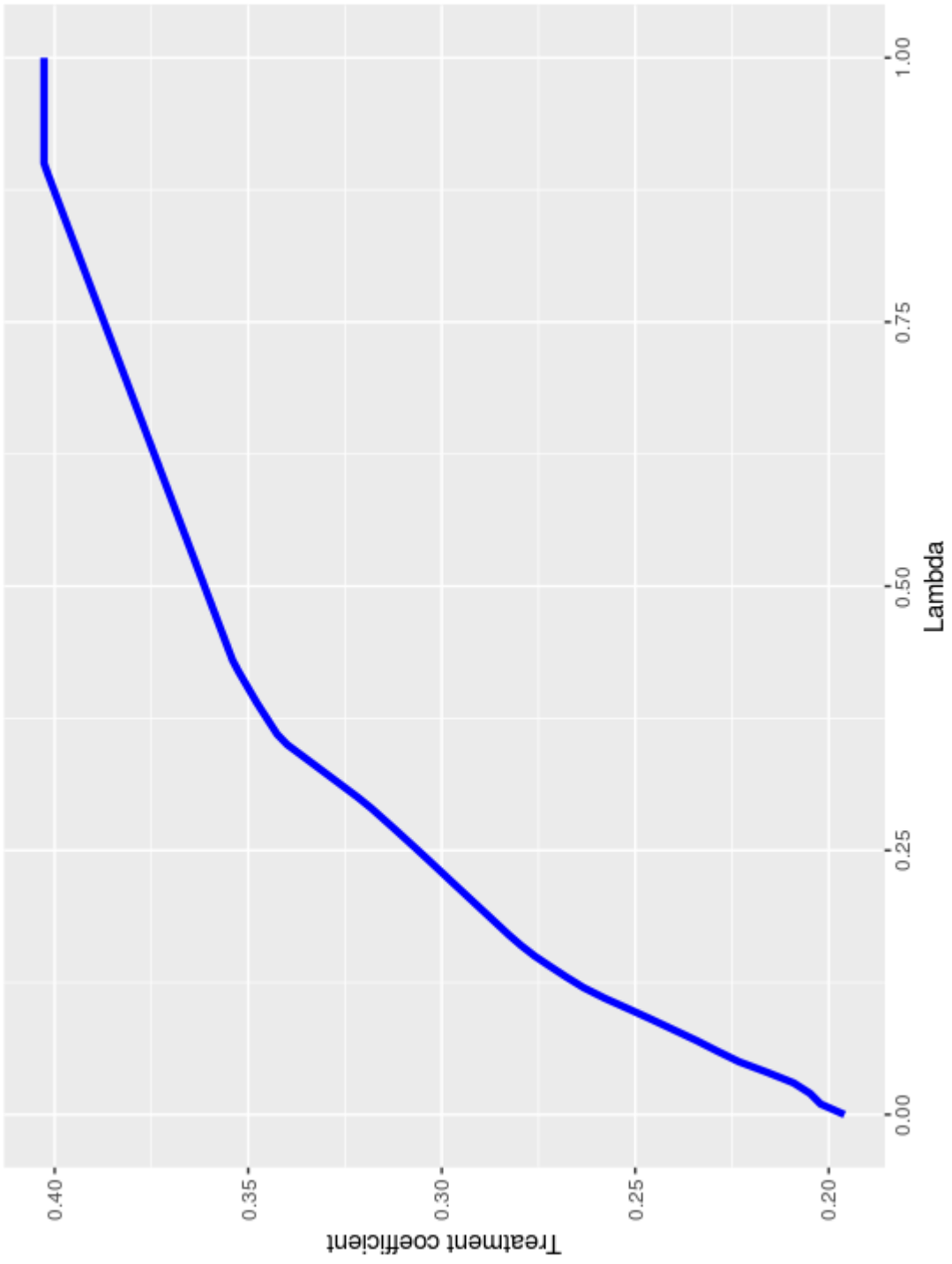


Figure 7: Treatment coefficient by lasso penalty