Toolkit for Weighting and Analysis of Nonequivalent Groups: A tutorial for the `twang` package

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1 Introduction

The Toolkit for Weighting and Analysis of Nonequivalent Groups, `twang`, contains a set of functions and procedures to support causal modeling of observational data through the estimation and evaluation of propensity scores and associated weights. This package was developed in 2004. After extensive use, it received a major update in 2012.

The propensity score is the probability that a particular case would be assigned or exposed to a treatment condition. Rosenbaum & Rubin (1983) showed that knowing the propensity score is sufficient to separate the effect of a treatment on an outcome from observed confounding factors that influence both treatment assignment and outcomes, provided the necessary conditions hold. The propensity score has the balancing property that given the propensity score the distribution of features for the treatment cases is the same as that for the control cases. While the treatment selection probabilities are generally not known, good estimates of them can be effective at diminishing or eliminating confounds between pretreatment group differences and treatment outcomes in the estimation of treatment effects.

There are now numerous propensity scoring methods in the literature. They differ in how they estimate the propensity score (e.g. logistic regression, CART), the target estimand (e.g. treatment effect on the treated, population treatment effect), and how they utilize the resulting estimated propensity scores (e.g. stratification, matching, weighting, doubly robust estimators). We originally developed the `twang` package with a particular process in mind, generalized boosted regression, to estimate the propensity scores and weighting of the comparison cases to estimate a treatment effect on the treated. However, we have updated the package to also meaningfully handle the case where interest lies in using the population weights (e.g., weighting of comparison and treatment cases to estimate the population average treatment effect.) The main workhorse of `twang` is the `ps()` function which implements generalized boosted regression modeling to estimate the propensity scores. However, the framework of the package is flexible enough to allow the user to use propensity score estimates from other methods and to assess the usefulness of those estimates for ensuring equivalence (or "balance") in the pretreatment covariate distributions of treatment and control groups using tools from the package. The same set of functions is also useful for other tasks such as non-response weighting, as discussed in Section 4.

The `twang` package aims to compute from the data estimates of the propensity scores which yield accurate causal effect estimates, check the quality of the weights by assessing whether or

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not they have the balancing properties that we expect in theory, and use them in computing treatment effect estimates.

2 An ATT example to start

If you have not already done so, install `twang` by typing `install.packages("twang")`. `twang` relies on other R packages, especially `gbm`, `survey`, and `lattice`. You may have to run `install.packages()` for these as well if they are not already installed. You will only need to do this step once. In the future running `update.packages()` regularly will ensure that you have the latest versions of the packages, including bug fixes and new features.

To start using `twang`, first load the package. You will have to do this step once for each R session that you run. We also set the seed of R's pseudo random number generator so that the results are exactly replicable. (There is a stochastic element to the fitting of the propensity score models.)

```
> library(twang)

Loaded gbm 1.6-3.1

> set.seed(1)
```

To demonstrate the package we utilize data from Lalonde's National Supported Work Demonstration analysis (Lalonde 1986, Dehejia & Wahba 1999, `http://www.columbia.edu/~rd247/nswdata.html`). This dataset is provided with the `twang` package.

```
> data(lalonde)
```

R can read data from many other sources. The manual "R Data Import/Export," available at `http://cran.r-project.org/doc/manuals/R-data.pdf`, describes that process in detail.

For the `lalonde` dataset, the variable `treat` is the 0/1 treatment indicator, 1 indicates "treatment" by being part of the National Supported Work Demonstration and 0 indicates "comparison" cases drawn from the Current Population Survey. In order to estimate a treatment effect for this demonstration program that is unbiased by pretreatment group differences on other observed covariates, we include these covariates in a propensity score model of treatment assignment: age, education, black, Hispanic, having no degree, married, earnings in 1974 (pretreatment), and earnings in 1975 (pretreatment). Note that we specify no outcome variables at this time. The `ps()` function is the primary method in `twang` for estimating propensity scores. This step is computationally intensive and can take a few minutes.

```
> ps.lalonde <- ps(treat ~ age + educ + black + hispan + nodegree +
+                            married + re74 + re75,
+                  data = lalonde,
+                  n.trees=5000,
+                  interaction.depth=2,
+                  shrinkage=0.01,
+                  perm.test.iters=0,
+                  stop.method=c("es.mean","ks.max"),
+                  estimand = "ATT",
+                  verbose=FALSE)
```

The arguments to `ps()` require some discussion. The first argument specifies a formula indicating that `treat` is the 0/1 treatment indicator and that the propensity score model should predict `treat` from the eight covariates listed there separated by "+". The "+" does *not* mean that these variables are being summed *nor* does it mean that the model is linear. This is just R's notation for including predictor variables in the model. There is no need to specify interaction terms in the formula. There is also no need — and it can be counterproductive — to create indicator, or "dummy coded," variables to represent categorical covariates, provided the categorical variables are stored as a `factor` or as `ordered` (see `help(factor)` for more details).

The next argument, `data`, indicates the dataset. `n.trees`, `interaction.depth`, and `shrinkage` are parameters for the `gbm` model that `ps()` computes and stores. The resulting `gbm` object describes a family of candidate propensity score models indexed by the number of GBM iterations from one to `n.trees`. The argument `n.trees` is the maximum number of iterations that `gbm` will run. `ps()` will issue a warning if the estimated optimal number of iterations is too close to the bound selected in this argument because it indicates that balance may improve if more complex models (i.e., those with more trees) are considered. The user should increase `n.trees` or decrease `shrinkage` if this warning appears.

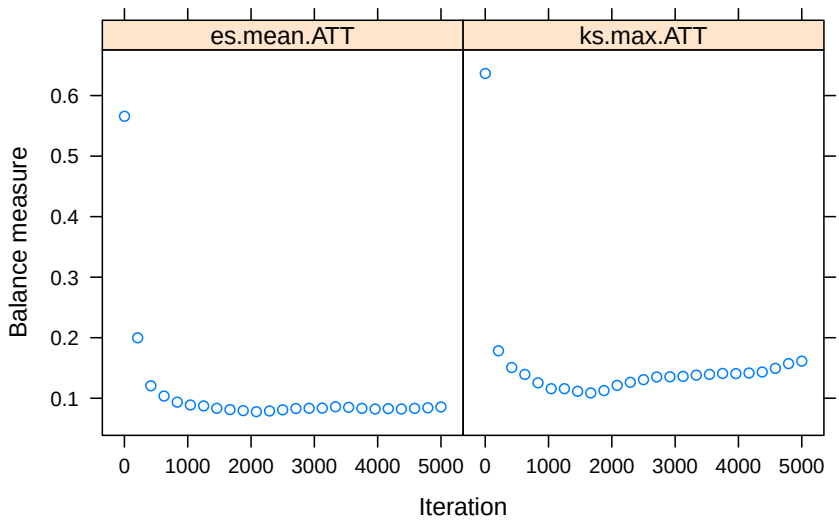
`perm.test.iters` specifies whether p-values for KS statistics should be calculated using Monte Carlo methods, which is slow but can be accurate, or estimated using an analytic approximation that is fast, but produces poor estimates in the presence of many ties. If `perm.test.iters=0` is called, then analytic approximations are used. If `perm.test.iters=500` is called, then 500 Monte Carlo trials are run to establish the reference distribution of KS statistics for each covariate. Higher numbers of trials will produce more precise p-values.

The `estimand` argument is used to indicate whether the analyst is interested in estimating the average treatment effect (ATE) or the average treatment effect on the treated (ATT), as we do above. ATE addresses the question of how outcomes would differ if everyone in the sample were given the treatment versus everyone being given the control (Wooldridge, 2002). ATT, on the other hand, estimates the analogous quantity averaging only over the subjects who were actually treated. The `estimand` argument was added to the 2012 revision of the package which integrated ATE weighting into the package and the `ps` function estimate of the propensity score.

The `stop.method` argument specifies a set (or sets) of rules and measures for assessing the balance, or equivalence, established on the pretreatment covariates of the treatment and weighted control group. The `ps` function selects the optimal number of GBM iterations to minimize the differences between the treatment and control groups as measured by the rules of the given `stop.method` object. The package includes four built-in `stop.method` objects. They are `es.mean`, `es.max`, `ks.mean`, and `ks.max`. The four stopping rules are defined by two components: a balance metric for covariates and rule for summarizing across covariates. The balance metric summarizes the difference between two univariate distributions of a single pre-treatment variable (e.g., age). The default stopping rules in `twang` use two balance metrics: absolute standardized bias (also referred to as the absolute standardized mean difference of the *E*ffect *S*ize) and the Kolmogorov-Smirnov (KS) statistic. The stopping rule use two different rules for summarizing across covariates: the mean of the covariate balance metrics ("mean") or the maximum of the balance metrics ("max"). The first piece of the stopping rule name identifies the balance metric (ES or KS) and the second piece specifies the method for summarizing across balance metrics. For instance, `es.mean` uses the effect size or the absolute standardized bias and summarizes across variables with the mean and the `ks.max` uses the KS statistics to assess balances and summarizes using the maximum across variables and the other two stopping rules use the remaining two combinations of balance metrics and summary statistics. The variable distributions used in the balance metrics depend on whether we are interested in estimating the ATT or ATE, and correct specification of these distributions is set automatically by the specification of the `estimand` in the `ps()` function.

Having fit the `ps` object, the analyst should perform several diagnostic checks before estimating the causal effect in question. The first of these diagnostic checks makes sure that the specified value of `n.trees` allowed GBM to explore sufficiently complicated models. We can do this quickly with the `plot()` function.[1] As a default, the `plot()` function applied to a `ps` object gives the balance measures as a function of the number of iterations in the GBM algorithm, with higher iterations corresponding to more complicated fitted models. In the example below, 2127 iterations minimized the average effect size difference and 1756 iterations minimized the largest of the eight Kolmogorov-Smirnov (KS) statistics computed for the covariates. If it appears that additional iterations would be likely to result in lower values of the balance statistic, `n.trees` should be increased. However, after a point, additional complexity typically makes the balance worse, as in the example below. This figure also gives information on how compatible two or more stopping rules are: if the minima for multiple stopping rules under consideration are near one another, the results should not be sensitive to which stopping rule one uses for the final analysis. See Section 5.3 for a discussion of these and other balance measures.

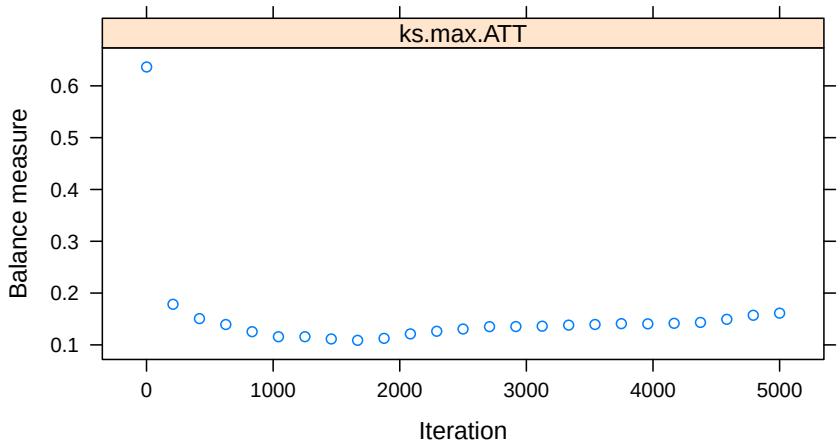
```
> plot(ps.lalonde)
```



If we wish to focus on only one stopping rule, the plotting commands also take a `subset` argument.

[1] In versions 1.0.x of the `twang` package, the `ps` function itself included some plotting functions. This is no longer the case (and the function no longer includes a `plots` argument); these functions have been moved to the generic `plot()` function.

```
>     plot(ps.lalonde, subset = 2)
```



The `gbm` package has various tools for exploring the relationship between the covariates and the treatment assignment indicator if these are of interest. `summary()` computes the relative influence of each variable for estimating the probability of treatment assignment. The gbm estimates depend on the number of iterations at which the gbm model is evaluated, which is specified by the `n.trees` argument in the `summary` method for gbm. In this example, we choose the number of iterations to be the optimal number for minimizing the largest of the KS statistics. This value can be found in the `ps.lalonde$desc$ks.max.ATT$n.trees`. Figure 1 shows the barchart of the relative influence and is produced when `plot=TRUE` in the call to `summary()`.

```
> summary(ps.lalonde$gbm.obj,
+         n.trees=ps.lalonde$desc$ks.max.ATT$n.trees,
+         plot=FALSE)

       var     rel.inf
1    black 57.83094183
2      age 16.51591133
3     re74 15.61359263
4     re75  3.58011684
5  married  3.02141880
6     educ  2.91846328
7 nodegree  0.43884552
8   hispan  0.08070978
```

2.1 Assessing "balance" using balance tables

Having estimated the propensity scores, `bal.table()` produces a table that shows how well the resulting weights succeed in manipulating the control group so that its weighted pretreatment characteristics match, or balance, those of the unweighted treatment group if `estimand = "ATT"` or the control and treatment groups so that the weighted pretreatment characteristics match, or balance, with one another if `estimand = "ATE"`. By default, the `bal.table()` function uses the value of `estimand` set with the `ps()` function call. For example, in the analysis we

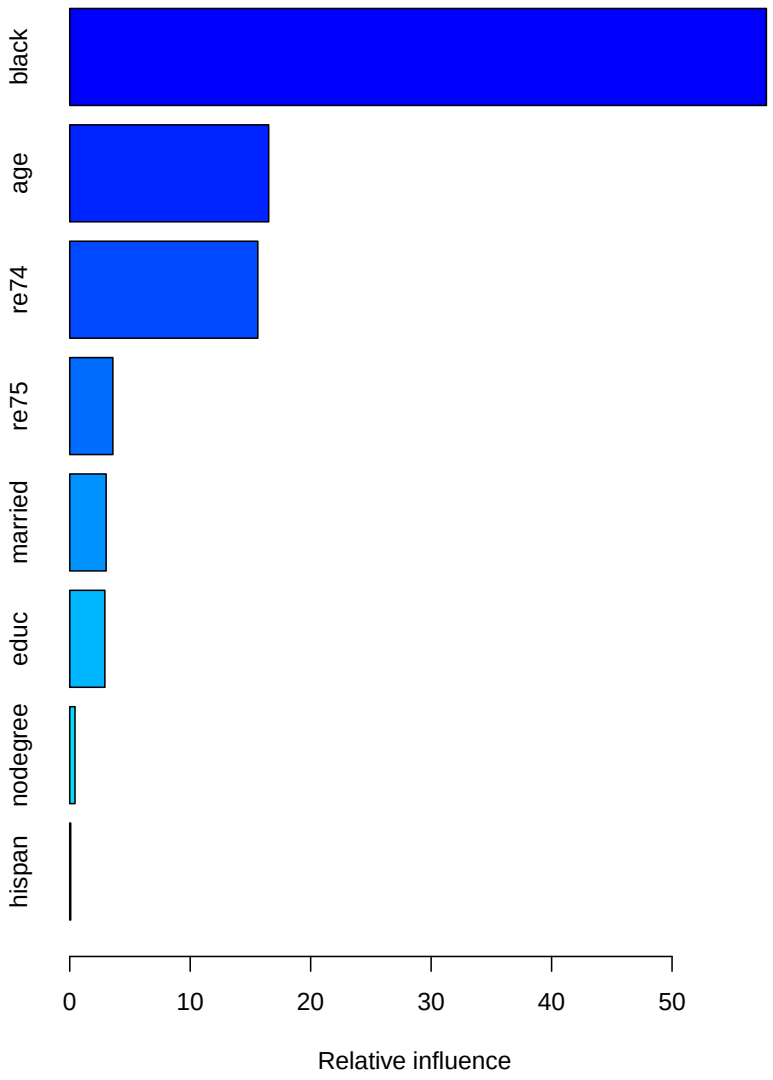


Figure 1: Relative influence of the covariates on the estimated propensity score

set `estimand = "ATT"` when calling `ps()` to estimate the propensity scores and the resulting `ps.object`, `ps.lalonde`, contains an element "estimand" which takes the value `"ATT"`. The function `bal.table()` checks this value and automatically uses ATT weights when checking balance and comparing the distributions of pre-treatment variables for the weighted control group with those from the unweighted treatment group.

```
> lalonde.balance <- bal.table(ps.lalonde)
> lalonde.balance

$unw
            tx.mn    tx.sd    ct.mn    ct.sd std.eff.sz   stat     p    ks ks.pval
age        25.816    7.155   28.030   10.787     -0.309 -2.994 0.003 0.158   0.003
educ       10.346    2.011   10.235    2.855      0.055  0.547 0.584 0.111   0.074
black       0.843    0.365    0.203    0.403      1.757 19.371 0.000 0.640   0.000
hispan      0.059    0.237    0.142    0.350     -0.349 -3.413 0.001 0.083   0.317
nodegree    0.708    0.456    0.597    0.491      0.244  2.716 0.007 0.111   0.074
married     0.189    0.393    0.513    0.500     -0.824 -8.607 0.000 0.324   0.000
re74     2095.574 4886.620 5619.237 6788.751     -0.721 -7.254 0.000 0.447   0.000
re75     1532.055 3219.251 2466.484 3291.996     -0.290 -3.282 0.001 0.288   0.000

$es.mean.ATT
            tx.mn    tx.sd    ct.mn    ct.sd std.eff.sz   stat     p    ks ks.pval
age        25.816    7.155   25.802    7.279      0.002  0.015 0.988 0.122   0.892
educ       10.346    2.011   10.573    2.089     -0.113 -0.706 0.480 0.099   0.977
black       0.843    0.365    0.842    0.365      0.003  0.027 0.978 0.001   1.000
hispan      0.059    0.237    0.042    0.202      0.072  0.804 0.421 0.017   1.000
nodegree    0.708    0.456    0.609    0.489      0.218  0.967 0.334 0.099   0.977
married     0.189    0.393    0.189    0.392      0.002  0.012 0.990 0.001   1.000
re74     2095.574 4886.620 1556.930 3801.566      0.110  1.027 0.305 0.066   1.000
re75     1532.055 3219.251 1211.575 2647.615      0.100  0.833 0.405 0.103   0.969

$ks.max.ATT
            tx.mn    tx.sd    ct.mn    ct.sd std.eff.sz   stat     p    ks ks.pval
age        25.816    7.155   25.764    7.408      0.007  0.055 0.956 0.107   0.919
educ       10.346    2.011   10.572    2.140     -0.113 -0.712 0.477 0.107   0.919
black       0.843    0.365    0.835    0.371      0.022  0.187 0.852 0.008   1.000
hispan      0.059    0.237    0.043    0.203      0.069  0.779 0.436 0.016   1.000
nodegree    0.708    0.456    0.601    0.490      0.235  1.100 0.272 0.107   0.919
married     0.189    0.393    0.199    0.400     -0.024 -0.169 0.866 0.010   1.000
re74     2095.574 4886.620 1673.666 3944.600      0.086  0.800 0.424 0.054   1.000
re75     1532.055 3219.251 1257.242 2674.922      0.085  0.722 0.471 0.094   0.971
```

`bal.table()` returns information on the pretreatment covariates before and after weighting. The object is a list with named components, one for an unweighted analysis (named `unw`) and one for each `stop.method` specified, here `es.mean` and `ks.max`. McCaffrey et al (2004) essentially used `es.mean` for the analyses, but our more recent work has sometimes used `ks.max`. See Section 5.3 for a more detailed description of these choices.

If there are missing values (represented as `NA`) in the covariates, `twang` will attempt to construct weights that also balance rates of missingness in the treatment and control arms. In this case, the `bal.table()` will have an extra row for each variable that has missing entries.

The columns of the table consist of the following items:

tx.mn, ct.mn The treatment means and the control means for each of the variables. The unweighted table (unw) shows the unweighted means. For each stopping rule the means are weighted using weights corresponding to the gbm model selected by `ps()` using the stopping rule. When `estimand = "ATT"` the weights for the treatment group always equal 1 for all cases and there is no difference between unweighted and propensity score weighted tx.mn.

tx.sd, ct.sd The propensity score weighted treatment and control groups' standard deviations for each of the variables. The unweighted table (unw) shows the unweighted standard deviations

std.eff.sz The standardized effect size, defined as the treatment group mean minus the control group mean divided by the treatment group standard deviation if `estimand = "ATT"` or divided by the pooled sample (treatment and control) standard deviation if `estimand = "ATE"`. (In discussions of propensity scores this value is sometimes referred to as "standardized bias".) Occasionally, lack of treatment group or pooled sample variance on a covariate results in very large (or infinite) standardized effect sizes. For purposes of analyzing mean effect sizes across multiple covariates, we set all standardized effect sizes larger than 500 to `NA` (missing values).

stat, p Depending on whether the variable is continuous or categorical, `stat` is a t-statistic or a χ^2 statistic. `p` is the associated p-value

ks, ks.pval The Kolmogorov-Smirnov test statistic and its associated p-value. P-values for the KS statistics are either derived from Monte Carlo simulations or analytic approximations, depending on the specifications made in the `perm.test.iters` argument of the `ps` function. For categorical variables this is just the χ^2 test p-value

Components of these tables are useful for demonstrating that pretreatment differences between groups on observed variables have been eliminated using the weights. The `xtable` package aids in formatting for LaTeX and Word documents. Table 1 shows the results for `ks.max` reformatted for a LaTeX document. For Word documents, paste the LaTeX description of the table into a Word document, highlight it and use Word tools to convert the text to a table using "&" as the separator.

```
> library(xtable)
> pretty.tab <- lalonde.balance$ks.max.ATT[,c("tx.mn","ct.mn","ks")]
> pretty.tab <- cbind(pretty.tab, lalonde.balance$unw[,"ct.mn"])
> names(pretty.tab) <- c("E(Y1|t=1)","E(Y0|t=1)","KS","E(Y0|t=0)")
> xtable(pretty.tab,
+        caption = "Balance of the treatment and comparison groups",
+        label = "tab:balance",
+        digits = c(0, 2, 2, 2, 2),
+        align=c("l","r","r","r","r"))
```

	E(Y1|t=1)	E(Y0|t=1)	KS	E(Y0|t=0)
age	25.82	25.76	0.11	28.03
educ	10.35	10.57	0.11	10.23
black	0.84	0.83	0.01	0.20
hispan	0.06	0.04	0.02	0.14
nodegree	0.71	0.60	0.11	0.60
married	0.19	0.20	0.01	0.51
re74	2095.57	1673.67	0.05	5619.24
re75	1532.06	1257.24	0.09	2466.48

Table 1: Balance of the treatment and comparison groups

The `summary()` method for `ps` objects offers a compact summary of the sample sizes of the groups and the balance measures. If `perm.test.iters>0` was used to create the `ps` object, then

Monte Carlo simulation is used to estimate p-values for the maximum KS statistic that would be expected across the covariates, had individuals with the same covariate values been assigned to groups randomly. Thus, a p-value of 0.04 for `max.ks.p` indicates that the largest KS statistic found across the covariates is larger than would be expected in 96% of trials in which the same cases were randomly assigned to groups.

```
> summary(ps.lalonde)
```

```
            n.treat n.ctrl ess.treat  ess.ctrl    max.es
unw             185    429       185 429.00000 1.7567745
es.mean.ATT     185    429       185  22.96430 0.2177817
ks.max.ATT      185    429       185  27.05472 0.2348846
                mean.es    max.ks max.ks.p    mean.ks iter
unw         0.56872589 0.6404460       NA 0.27024507   NA
es.mean.ATT 0.07746175 0.1223384       NA 0.06361021 2127
ks.max.ATT  0.08025994 0.1070761       NA 0.06282432 1756
```

In general, weighted means can have greater sampling variance than unweighted means from a sample of equal size. The effective sample size (ESS) of the weighted comparison group captures this increase in variance as

$$\text{ESS} = \frac{\left(\sum_{i \in C} w_i\right)^2}{\sum_{i \in C} w_i^2}. \tag{1}$$

The ESS is approximately the number of observations from a simple random sample that yields an estimate with sampling variation equal to the sampling variation obtained with the weighted comparison observations. Therefore, the ESS will give an estimate of the number of comparison participants that are comparable to the treatment group when `estimand = "ATT"`. The ESS is an accurate measure of the relative size of the variance of means when the weights are fixed or they are uncorrelated with outcomes. Otherwise the ESS underestimates the effective sample size (Little & Vartivarian, 2004). With propensity score weights, it is rare that weights are uncorrelated with outcomes. Hence the ESS typically gives a lower bound on the effective sample size, but it still serves as a useful measure for choosing among alternative models and assessing the overall quality of a model, even if it provides a possibly conservative picture of the loss in precision due to weighting.

The `ess.treat` and `ess.ctrl` columns in the summary results shows the ESS for the estimated propensity scores. Note that although the original comparison group had 429 cases, the propensity score estimates effectively utilize only 23 or 27.1 of the comparison cases, depending on the rules and measures used to estimate the propensity scores. While this may seem like a large loss of sample size, this indicates that many of the original cases were unlike the treatment cases and, hence, were not useful for isolating the treatment effect. Moreover, similar or even greater reductions in ESS would be expected from alternative approaches to using propensity scores, such as matching or stratification. Since the estimand of interest in this example is ATT, `ess.treat = n.treat` throughout (i.e., all treatment cases have a weight of 1).

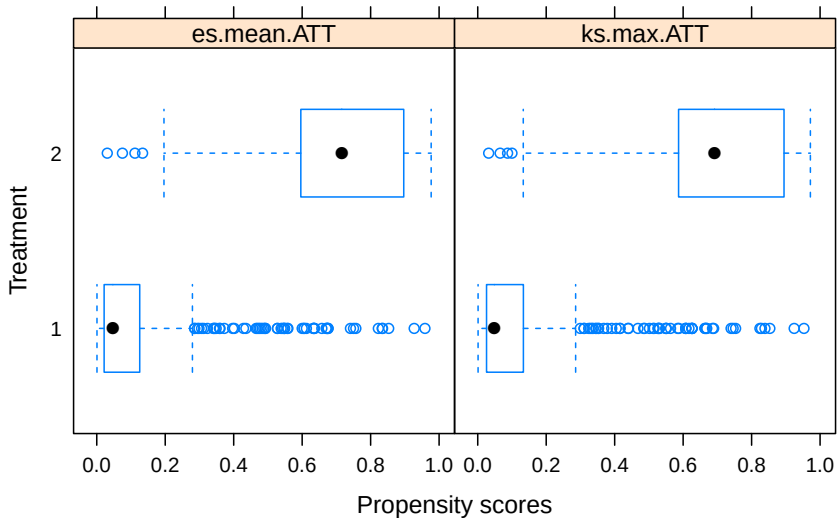
2.2 Graphical assessments of balance

The `plot()` method can generate useful diagnostic plots from the propensity score objects. The full set of plots available in `twang` and the argument value of `plot` to produce each one are given in Table 2. The convergence plot — the default — was discussed above.

The `plot()` function takes a `plots` argument in order to produce other diagnostic plots. For example, specifying `plots = 2` or `plots = "boxplot"` produces boxplots illustrating the spread

of the estimated propensity scores in the treatment and comparison groups. Whereas propensity score stratification requires considerable overlap in these spreads, excellent covariate balance can often be achieved with weights, even when the propensity scores estimated for the treatment and control groups show little overlap.

```
> plot(ps.lalonde, plots=2)
```

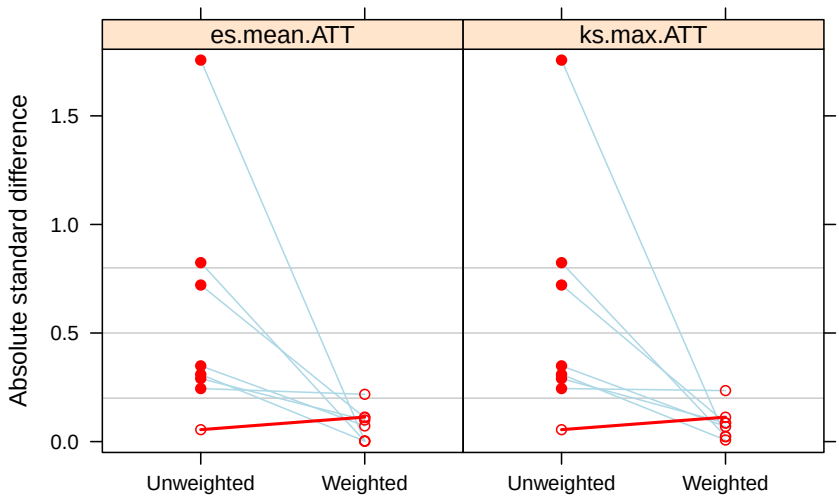


Descriptive argument	Numeric argument	Description
`"optimize"`	1	Balance measure as a function of GBM iterations
`"boxplot"`	2	Boxplot of treatment/control propensity scores
`"es"`	3	Standardized effect size of pretreatment variables
`"t"`	4	t-test p-values for weighted pretreatment variables
`"ks"`	5	Kolmogorov-Smirnov p-values for weighted pretreatment variables
`"histogram"`	6	Histogram of weights for treatment/control

Table 2: Available options for `plots` argument to `plot()` function.

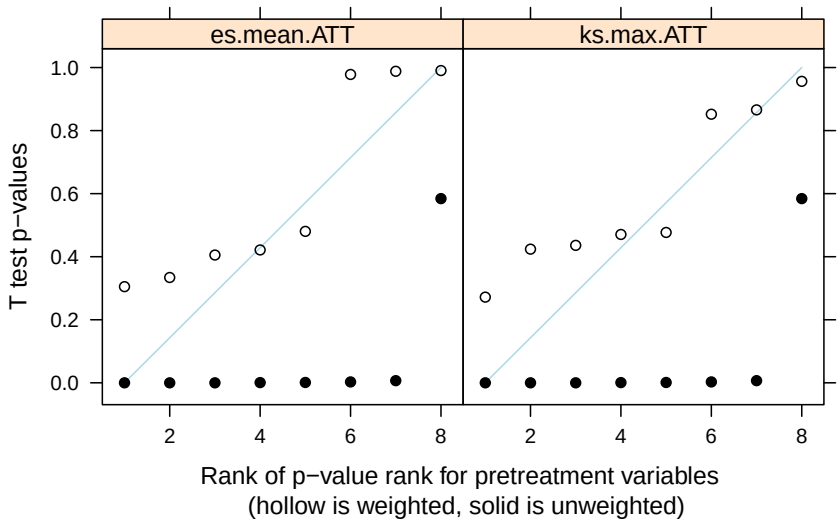
The effect size plot illustrates the effect of weights on the magnitude of differences between groups on each pretreatment covariate. These magnitudes are standardized using the standardized effect size described earlier. In these plots, substantial reductions in effect sizes are observed for most variables (blue lines), with only one variable showing an increase in effect size (red lines), but only a seemingly trivial increase. Closed red circles indicate a statistically significant difference, many of which occur before weighting, none after. In some analyses variables can have very little variance in the treatment group sample or the entire sample and group differences can be very large relative to the standard deviations. In these situations, the user is warned that some effect sizes are too large to plot.

```
> plot(ps.lalonde, plots=3)
```



P-values from independent tests in which the null hypothesis is true have a uniform distribution. Therefore, a QQ plot comparing the quantiles of the observed p-values to the quantiles of the uniform distribution illustrate whether group differences observed before and after weighting are consistent with what we would expect to see had groups been formed by random assignment (and hence the null hypothesis would be true). Setting `plots = 4` or `plots="t"` generates such QQ plots.

```
> plot(ps.lalonde, plots = 4)
```

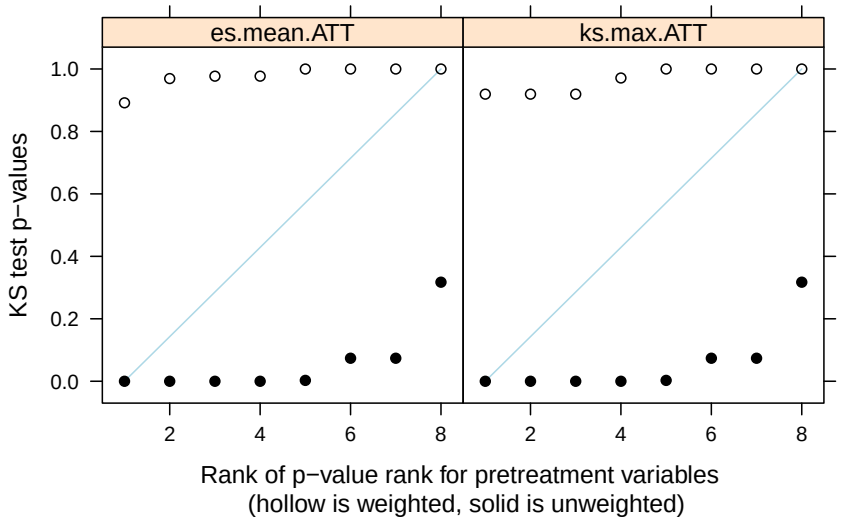


Before weighting (closed circles), the groups have statistically significant differences on many variables (i.e., p-values are near zero). After weighting (open circles) the p-values are generally above the 45-degree line, which represents the cumulative distribution of a uniform variable on

[0,1]. This indicates that the p-values are even larger than would be expected in a randomized study.

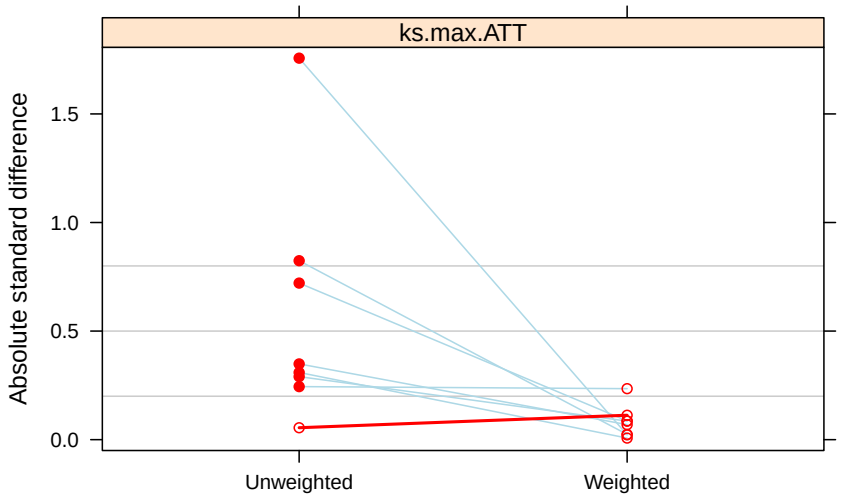
One can inspect similar plots for the KS statistic with the argument `plots = "ks"` or

```
> plot(ps.lalonde, plots = 5)
```



In all cases, the `subset` argument can be used if we wish to fous on results from one stopping rule.

```
> plot(ps.lalonde, plots = 3, subset = 2)
```



2.3 Analysis of outcomes

A separate R package, the `survey` package, is useful for performing the outcomes analyses using weights. Its statistical methods account for the weights when computing standard error estimates. It is not a part of the standard R installation but installing `twang` should automatically install `survey` as well.

```
> library(survey)
```

The `get.weights()` function extracts the propensity score weights from a `ps` object. Those weights may then be used as case weights in a `svydesign` object. By default, it returns weights corresponding to the estimand (ATE or ATT) that was specified in the original call to `ps()`. If needed, the user can override the default via the optional `estimand` argument.

```
> lalonde$w <- get.weights(ps.lalonde, stop.method="es.mean")
> design.ps <- svydesign(ids=~1, weights=~w, data=lalonde)
```

The `stop.method` argument specifies which GBM model, and consequently which weights, to utilize.

The `svydesign` function from the `survey` package creates an object that stores the dataset along with design information needed for analyses. See `help(svydesign)` for more details on setting up `svydesign` objects.

The aim of the National Supported Work Demonstration analysis is to determine whether the program was effective at increasing earnings in 1978. The propensity score adjusted test can be computed with `svyglm`.

```
> glm1 <- svyglm(re78 ~ treat, design=design.ps)
> summary(glm1)

Call:
svyglm(re78 ~ treat, design = design.ps)

Survey design:
svydesign(ids = ~1, weights = ~w, data = lalonde)

Coefficients:
            Estimate Std. Error t value Pr(>|t|)
(Intercept)   5616.6      884.9   6.347 4.28e-10 ***
treat          732.5     1056.6   0.693    0.488
---
Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1

(Dispersion parameter for gaussian family taken to be 49804197)

Number of Fisher Scoring iterations: 2
```

The analysis estimates an increase in earnings of $733 for those that participated in the NSW compared with similarly situated people observed in the CPS. The effect, however, does not appear to be statistically significant.

Some authors have recommended utilizing both propensity score adjustment and additional covariate adjustment to minimize mean square error or to obtain "doubly robust" estimates of the treatment effect (Huppler-Hullsiek & Louis 2002, Bang & Robins 2005). These estimators

are consistent if either the propensity scores are estimated correctly *or* the regression model is specified correctly. For example, note that the balance table for `ks.max.ATT` made the two groups more similar on `nodegree`, but still some differences remained, 70.8% of the treatment group had no degree while 60.1% of the comparison group had no degree. While linear regression is sensitive to model misspecification when the treatment and comparison groups are dissimilar, the propensity score weighting has made them more similar, perhaps enough so that additional modeling with covariates can adjust for any remaining differences. In addition to potential bias reduction, the inclusion of additional covariates can reduce the standard error of the treatment effect if some of the covariates are strongly related to the outcome.

```
> glm2 <- svyglm(re78 ~ treat + nodegree, design=design.ps)
> summary(glm2)

Call:
svyglm(re78 ~ treat + nodegree, design = design.ps)

Survey design:
svydesign(ids = ~1, weights = ~w, data = lalonde)

Coefficients:
            Estimate Std. Error t value Pr(>|t|)
(Intercept)   6768.4     1471.0   4.601 5.11e-06 ***
treat          920.3     1082.8   0.850    0.396
nodegree     -1891.8     1261.9  -1.499    0.134
---
Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1

(Dispersion parameter for gaussian family taken to be 49013778)

Number of Fisher Scoring iterations: 2
```

Adjusting for the remaining group difference in the `nodegree` variable slightly increased the estimate of the program's effect to $920, but the difference is still not statistically significant. We can further adjust for the other covariates, but that too in this case has little effect on the estimated program effect.

```
> glm3 <- svyglm(re78 ~ treat + age + educ + black + hispan + nodegree +
+                        married + re74 + re75,
+                design=design.ps)
> summary(glm3)

Call:
svyglm(re78 ~ treat + age + educ + black + hispan + nodegree +
    married + re74 + re75, design = design.ps)

Survey design:
svydesign(ids = ~1, weights = ~w, data = lalonde)

Coefficients:
              Estimate Std. Error t value Pr(>|t|)
(Intercept) -2.459e+03  4.289e+03  -0.573  0.56671
```

```
treat        7.585e+02  1.019e+03   0.745  0.45674
age          3.005e+00  5.558e+01   0.054  0.95691
educ         7.488e+02  2.596e+02   2.884  0.00406 **
black       -7.627e+02  1.012e+03  -0.753  0.45153
hispan       6.106e+02  1.711e+03   0.357  0.72123
nodegree     5.350e+02  1.626e+03   0.329  0.74227
married      4.918e+02  1.072e+03   0.459  0.64660
re74         5.699e-02  1.801e-01   0.316  0.75176
re75         1.568e-01  1.946e-01   0.806  0.42076
---
Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1

(Dispersion parameter for gaussian family taken to be 47150852)

Number of Fisher Scoring iterations: 2
```

2.4 Estimating the program effect using linear regression

The more traditional regression approach to estimating the program effect would fit a linear model with a treatment indicator and linear terms for each of the covariates.

```
> glm4 <- lm(re78 ~ treat + age + educ + black + hispan + nodegree +
+                   married + re74 + re75,
+            data=lalonde)
> summary(glm4)

Call:
lm(formula = re78 ~ treat + age + educ + black + hispan + nodegree +
    married + re74 + re75, data = lalonde)

Residuals:
   Min     1Q Median     3Q    Max
-13595  -4894  -1662   3929  54570

Coefficients:
             Estimate Std. Error t value Pr(>|t|)
(Intercept)  6.651e+01  2.437e+03   0.027   0.9782
treat        1.548e+03  7.813e+02   1.982   0.0480 *
age          1.298e+01  3.249e+01   0.399   0.6897
educ         4.039e+02  1.589e+02   2.542   0.0113 *
black       -1.241e+03  7.688e+02  -1.614   0.1071
hispan       4.989e+02  9.419e+02   0.530   0.5966
nodegree     2.598e+02  8.474e+02   0.307   0.7593
married      4.066e+02  6.955e+02   0.585   0.5590
re74         2.964e-01  5.827e-02   5.086 4.89e-07 ***
re75         2.315e-01  1.046e-01   2.213   0.0273 *
---
Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1

Residual standard error: 6948 on 604 degrees of freedom
```

```
Multiple R-squared: 0.1478,        Adjusted R-squared: 0.1351
F-statistic: 11.64 on 9 and 604 DF,  p-value: < 2.2e-16
```

This model estimates a rather strong treatment effect, estimating a program effect of $1548 with a p-value=0.048. Several variations of this regression approach also estimate strong program effects. For example using square root transforms on the earnings variables yields a p-value=0.016. These estimates, however, are very sensitive to the model structure since the treatment and control subjects differ greatly as seen in the unweighted balance comparison ($unw) from `bal.table(ps.lalonde)`.

2.5 Propensity scores estimated from logistic regression

Propensity score analysis is intended to avoid problems associated with the misspecification of covariate adjusted models of outcomes, but the quality of the balance and the treatment effect estimates can be sensitive to the method used to estimate the propensity scores. Consider estimating the propensity scores using logistic regression instead of `ps()`.

```
> ps.logit <- glm(treat ~ age + educ + black + hispan + nodegree +
+                           married + re74 + re75,
+                 data = lalonde,
+                 family = binomial)
> lalonde$w.logit <- rep(1,nrow(lalonde))
> lalonde$w.logit[lalonde$treat==0] <- exp(predict(ps.logit,subset(lalonde,treat==0)))
```

`predict()` for logistic regression model produces estimates on the log-odds scale by default. Exponentiating those predictions for the comparison subjects gives the ATT weights $p/(1-p)$. `dx.wts()` from the `twang` package diagnoses the balance for an arbitrary set of weights producing a balance table. This function requires the user to specify the estimand argument in order to perform the appropriate calculations relative to the target group on which we are drawing inferences.

```
> bal.logit <- dx.wts(x = lalonde$w.logit,
+                     data=lalonde,
+                     vars=c("age","educ","black","hispan","nodegree",
+                       "married","re74","re75"),
+                     treat.var="treat",
+                     perm.test.iters=0, estimand = "ATT")
```

```
> bal.logit

  type n.treat n.ctrl ess.treat  ess.ctrl    max.es
1  unw     185    429       185 429.00000 1.7567745
2          185    429       185  99.81539 0.1188496
    mean.es    max.ks    mean.ks iter
1 0.5687259 0.6404460 0.27024507   NA
2 0.0318841 0.3078039 0.09302319   NA
```

Applying the `bal.table()` function to this object returns a variable-by-variable summary of balance, just like it did for the `ps` object.

```
> bal.tab.logit <- bal.table(bal.logit)
> bal.tab.logit

$unw
            tx.mn    tx.sd    ct.mn    ct.sd std.eff.sz
age        25.816    7.155   28.030   10.787     -0.309
educ       10.346    2.011   10.235    2.855      0.055
black       0.843    0.365    0.203    0.403      1.757
hispan      0.059    0.237    0.142    0.350     -0.349
nodegree    0.708    0.456    0.597    0.491      0.244
married     0.189    0.393    0.513    0.500     -0.824
re74     2095.574 4886.620 5619.237 6788.751     -0.721
re75     1532.055 3219.251 2466.484 3291.996     -0.290
           stat     p    ks ks.pval
age      -2.994 0.003 0.158   0.003
educ      0.547 0.584 0.111   0.074
black    19.371 0.000 0.640   0.000
hispan   -3.413 0.001 0.083   0.317
nodegree  2.716 0.007 0.111   0.074
married  -8.607 0.000 0.324   0.000
re74     -7.254 0.000 0.447   0.000
re75     -3.282 0.001 0.288   0.000

[[2]]
            tx.mn    tx.sd    ct.mn    ct.sd std.eff.sz
age        25.816    7.155   24.966   10.535      0.119
educ       10.346    2.011   10.403    2.459     -0.028
black       0.843    0.365    0.845    0.362     -0.006
hispan      0.059    0.237    0.059    0.236      0.001
nodegree    0.708    0.456    0.690    0.463      0.040
married     0.189    0.393    0.171    0.377      0.047
re74     2095.574 4886.620 2106.045 4235.832     -0.002
re75     1532.055 3219.251 1496.541 2716.258      0.011
           stat     p    ks ks.pval
age       0.739 0.460 0.308   0.000
educ     -0.219 0.827 0.036   1.000
black    -0.069 0.945 0.002   1.000
hispan    0.008 0.993 0.000   1.000
nodegree  0.332 0.740 0.018   1.000
married   0.456 0.649 0.019   1.000
re74     -0.022 0.983 0.228   0.002
re75      0.107 0.915 0.133   0.185
```

For weights estimated with logistic regression, the largest KS statistic was reduced from the unweighted sample's largest KS of 0.64 to 0.31, which is still quite a large KS statistic. Table 3 shows the details of the balance of the treatment and comparison groups. The means of the two groups appear to be quite similar while the KS statistic shows substantial differences in their distributions.

```
> pretty.tab <- bal.table(bal.logit)[[2]][,c("tx.mn","ct.mn","ks")]
> pretty.tab <- cbind(pretty.tab, bal.table(bal.logit)[[1]]$ct.mn)
```

```
> names(pretty.tab) <- c("E(Y1|t=1)","E(Y0|t=1)","KS","E(Y0|t=0)")
> xtable(pretty.tab,
+        caption = "Logistic regression estimates of the propensity scores",
+        label = "tab:balancelogit",
+        digits = c(0, 2, 2, 2, 2),
+        align=c("l","r","r","r","r"))
```

	E(Y1|t=1)	E(Y0|t=1)	KS	E(Y0|t=0)
age	25.82	24.97	0.31	28.03
educ	10.35	10.40	0.04	10.23
black	0.84	0.84	0.00	0.20
hispan	0.06	0.06	0.00	0.14
nodegree	0.71	0.69	0.02	0.60
married	0.19	0.17	0.02	0.51
re74	2095.57	2106.05	0.23	5619.24
re75	1532.06	1496.54	0.13	2466.48

Table 3: Logistic regression estimates of the propensity scores

Table 4 compares the balancing quality of the weights directly with one another.

	n.treat	ess.ctrl	max.es	mean.es	max.ks	mean.ks
unw	185	429.00	1.76	0.57	0.64	0.27
logit	185	99.82	0.12	0.03	0.31	0.09
es.mean.ATT	185	22.96	0.22	0.08	0.12	0.06
ks.max.ATT	185	27.05	0.23	0.08	0.11	0.06

Table 4: Summary of the balancing properties of logistic regression and gbm

```
> design.logit <- svydesign(ids=~1, weights=~w.logit, data=lalonde)
> glm6 <- svyglm(re78 ~ treat, design=design.logit)
> summary(glm6)

Call:
svyglm(re78 ~ treat, design = design.logit)

Survey design:
svydesign(ids = ~1, weights = ~w.logit, data = lalonde)

Coefficients:
            Estimate Std. Error t value Pr(>|t|)
(Intercept)   5135.1      588.9   8.719   <2e-16 ***
treat         1214.1      824.7   1.472    0.142
---
Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1

(Dispersion parameter for gaussian family taken to be 49598072)

Number of Fisher Scoring iterations: 2
```

The analysis estimates an increase in earnings of $1214 for those that participated in the NSW compared with similarly situated people observed in the CPS. Table 5 compares all of the treatment effect estimates.

Treatment effect	PS estimate	Linear adjustment
$733	GBM, minimize KS	none
$920	GBM, minimize KS	nodegree
$758	GBM, minimize KS	all
$1548	None	all
$1214	Logistic regression	none
$1237	Logistic regression	all

Table 5: Treatment effect estimates by various methods

3 An ATE example

In the analysis of Section 2, we focused on estimating ATT for the `lalonde` dataset. In this situation, the ATE is not of great substantive interest because not all people who are offered entrance into the program could be expected to take advantage of the opportunity. Further, there is some evidence that the treated subjects were drawn from a subset of the covariate space. In particular, in an ATE analysis, we see that we are unable to achieve balance, especially for the "black" indicator.

We now turn to an ATE analysis that is feasible and meaningful. We focus on the `lindner` dataset, which was included in the `USPS` package (Obenchain 2011), and is now included in `twang` for convenience. A tutorial by Helmreich and Pruzek (2009; HP) for the `PSAgraphics` package also uses propensity scores to analyze a portion of these data. HP describe the data as follows on p. 3 with our minor recodings in square braces:

> The `lindner` data contain data on 996 patients treated at the Lindner Center, Christ Hospital, Cincinnati in 1997. Patients received a Percutaneous Coronary Intervention (PCI). The data consists of 10 variables. Two are outcomes: [`sixMonthSurvive`] ranges over two values... depending on whether patients surved to six months post treatment [denoted by `TRUE`] or did not survive to six months [`FALSE`]... Secondly, `cardbill` contains the costs in 1998 dollars for the first six months (or less if the patient did not survive) after treatment... The treatment variable is `abcix`, where 0 indicates PCI treatment and 1 indicates standard PCI treatment and additional treatment in some form with abciximab. Covariates include `acutemi`, 1 indicating a recent acute myocardial infarction and 0 not; `ejecfrac` for the left ventricle ejection fraction, a percentage from 0 to 90; `ves1proc` giving the number of vessels (0 to 5) involved in the initial PCI; `stent` with 1 indicating coronary stent inserted, 0 not; `diabetic` where 1 indicates that the patient has been diagnosed with diabetes, 0 not; `height` in centimeters and `female` coding the sex of the patent, 1 for female, 0 for male.

HP focus on `cardbill` — the cost for the first months after treatment — as their outcome of interest. However, since not all patients survived to six months, it is not clear whether a lower value of `cardbill` is good or not. For this reason, we choose six-month survival (`sixMonthSurvive`) as our outcome of interest.

Ignoring pre-treatment variables, we see that `abcix` is associated with lower rates of 6-month mortality:

```
> data(lindner)
> table(lindner$sixMonthSurvive, lindner$abcix)

         0   1
  FALSE  15  11
  TRUE  283 687

> chisq.test(table(lindner$sixMonthSurvive, lindner$abcix))

        Pearson's Chi-squared test with Yates' continuity
        correction

data:  table(lindner$sixMonthSurvive, lindner$abcix)
X-squared = 8.5077, df = 1, p-value = 0.003536
```

The question is whether this association is causal. If health care policies were to be made on the basis of these data, we would wish to elicit expert opinion as to whether there are likely to be other confounding pretreatment variables. For this tutorial, we simply follow HP in choosing the pre-treatment covariates. The `twang` model is fit as follows

```
> set.seed(1)
> ps.lindner <- ps(abcix ~ stent + height + female + diabetic +
+                  acutemi + ejecfrac + ves1proc, data = lindner,
+                  verbose = FALSE, estimand = "ATE")
```

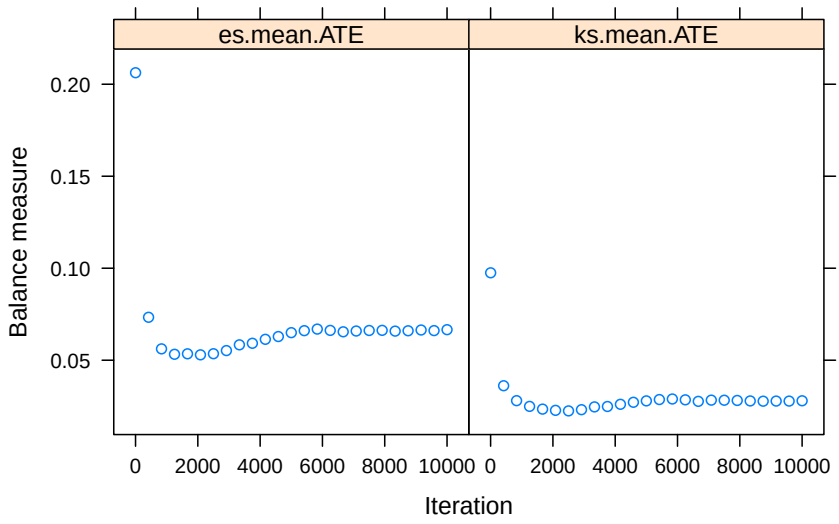
We set `estimand = "ATE"` because we are interested in the effects of abciximab on everyone in the population. We do not specify the stopping rules. Consequently `ps()` uses the defaults: `es.mean` and `ks.max`. We then inspect pre- and post-weighting balance with the command

```
> bal.table(ps.lindner)

$unw
           tx.mn  tx.sd   ct.mn  ct.sd std.eff.sz   stat     p    ks ks.pval
stent      0.705  0.456   0.584  0.494      0.259  3.624 0.000 0.121   0.004
height   171.443 10.695 171.446 10.589      0.000 -0.005 0.996 0.025   0.999
female     0.331  0.471   0.386  0.488     -0.115 -1.647 0.100 0.055   0.531
diabetic   0.205  0.404   0.268  0.444     -0.153 -2.127 0.034 0.064   0.349
acutemi    0.179  0.384   0.060  0.239      0.342  5.923 0.000 0.119   0.005
ejecfrac  50.403 10.419  52.289 10.297     -0.182 -2.640 0.008 0.114   0.008
ves1proc   1.463  0.706   1.205  0.480      0.399  6.693 0.000 0.188   0.000

$ks.mean.ATE
           tx.mn  tx.sd   ct.mn  ct.sd std.eff.sz   stat     p    ks ks.pval
stent      0.683  0.466   0.657  0.475      0.054  0.718 0.473 0.025   1.000
height   171.470 10.550 171.591 10.589     -0.011 -0.155 0.877 0.015   1.000
female     0.338  0.473   0.345  0.476     -0.015 -0.200 0.841 0.007   1.000
diabetic   0.215  0.411   0.229  0.421     -0.033 -0.432 0.666 0.014   1.000
acutemi    0.148  0.355   0.107  0.310      0.116  1.331 0.183 0.040   0.935
ejecfrac  51.052 10.333  51.604  9.110     -0.053 -0.798 0.425 0.027   0.999
ves1proc   1.395  0.666   1.337  0.573      0.091  1.203 0.229 0.028   0.999

$es.mean.ATE
```

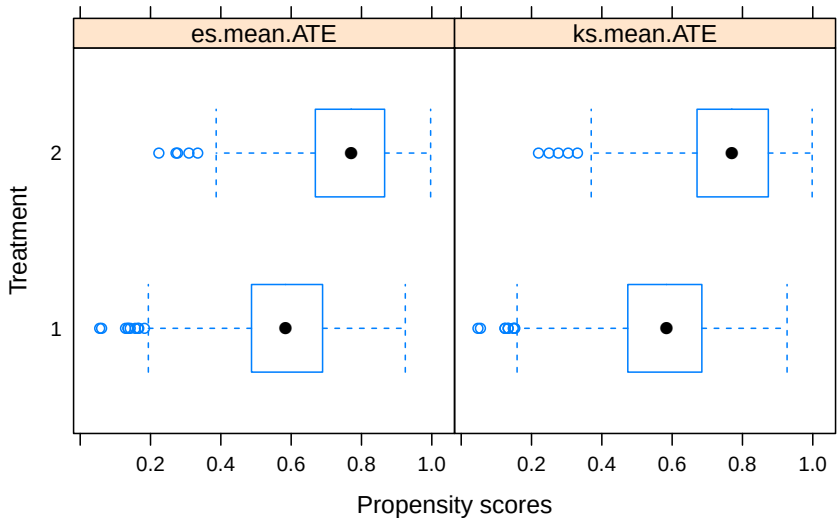
	tx.mn	tx.sd	ct.mn	ct.sd	std.eff.sz	stat	p	ks	ks.pval
stent	0.683	0.466	0.656	0.476	0.057	0.752	0.452	0.027	0.999
height	171.466	10.542	171.584	10.664	-0.011	-0.149	0.882	0.016	1.000
female	0.338	0.473	0.345	0.476	-0.016	-0.209	0.834	0.007	1.000
diabetic	0.215	0.411	0.231	0.422	-0.039	-0.512	0.609	0.016	1.000
acutemi	0.148	0.355	0.108	0.311	0.115	1.327	0.185	0.040	0.938
ejecfrac	51.037	10.350	51.544	9.172	-0.049	-0.730	0.466	0.027	0.999
ves1proc	1.396	0.666	1.342	0.579	0.084	1.089	0.276	0.026	1.000

This balance table shows that `stent`, `acutemi`, `ejectfrac` and `ves1proc` were all significantly imbalanced before weighting. After weighting (using either `stop.method` considered) we do not see problems in this regard. Examining `plot(ps.lindner, plots = x)` for `x` running from 1 to 5 does not reveal problems, either. In regard to the optimize plot, we note that the scales of the KS and ES statistics presented in the optimize plots are not necessarily comparable. The fact that the KS values are lower than the ES values in the optimize plot does not suggest that the KS stopping rule is finding superior models. Each panel of the optimize plot indicates the gbm model that minimizes each stopping rule. The panels should not be compared other than to compare the number of iterations selected by each rule.

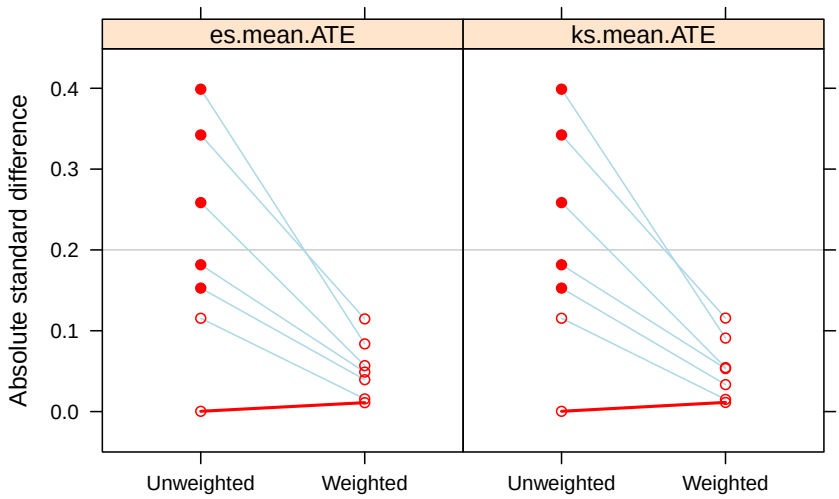
```
> plot(ps.lindner, plots = 1)
```



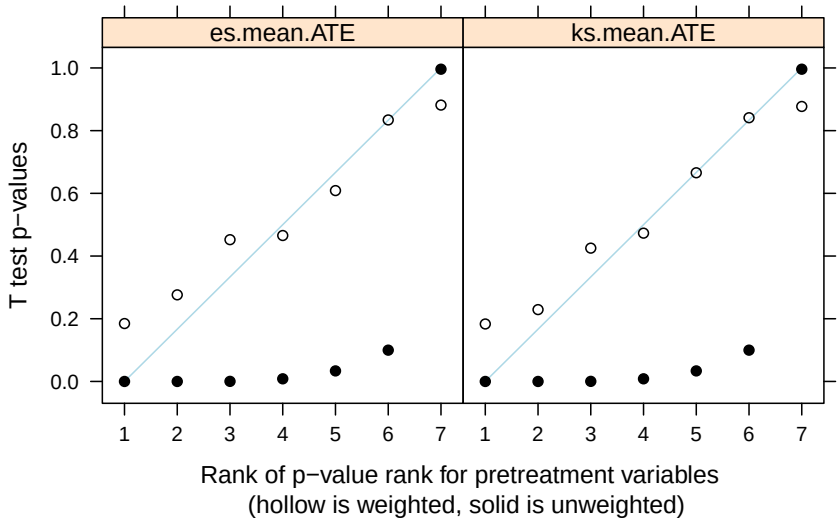
```
> plot(ps.lindner, plots = 2)
```



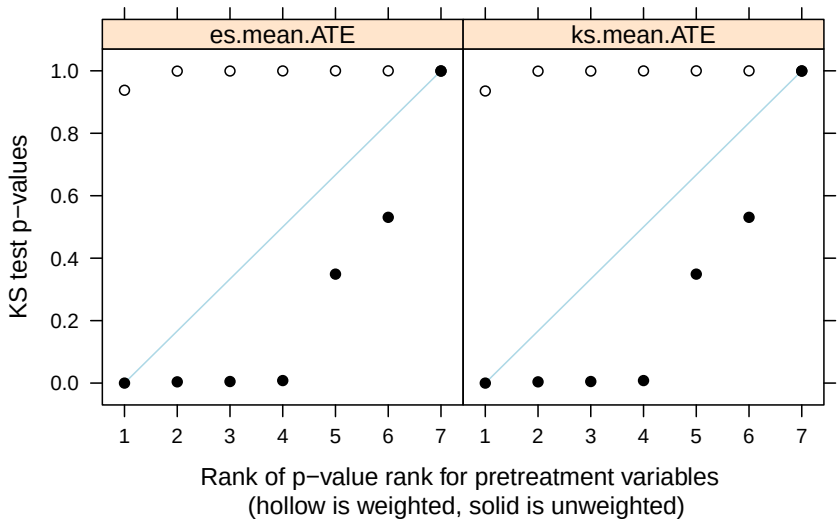

```
> plot(ps.lindner, plots = 3)
```



```
> plot(ps.lindner, plots = 4)
```




```
> plot(ps.lindner, plots = 5)
```



From a call to `summary()`, we see that the `es.mean.ATE` stopping rule results in a slightly higher ESS with comparable balance measures, so we proceed with those weights. Also, we note that `ess.treat` is no longer equal to `n.treat` since we are focusing on ATE rather than ATT.

```
> summary(ps.lindner)
```

```
           n.treat n.ctrl ess.treat ess.ctrl    max.es
unw            698    298  698.0000 298.0000 0.3988701
```

```
ks.mean.ATE     698    298  655.6754 228.8501 0.1157184
es.mean.ATE     698    298  658.4838 230.7292 0.1146893
                mean.es     max.ks max.ks.p    mean.ks iter
unw         0.20712864 0.18841945       NA 0.09791845   NA
ks.mean.ATE 0.05344346 0.04012757       NA 0.02235951 2603
es.mean.ATE 0.05291540 0.03977072       NA 0.02273098 2096
```

As before, we use the `survey` package to reweight our sample and perform the analysis.

```
> lindner$w <- get.weights(ps.lindner, stop.method = "es.mean")
> design.ps <- svydesign(ids=~1, weights = ~w, data = lindner)
> svychisq(~sixMonthSurvive + abcix, design = design.ps)

        Pearson's X^2: Rao & Scott adjustment

data:  svychisq(~sixMonthSurvive + abcix, design = design.ps)
F = 9.3894, ndf = 1, ddf = 995, p-value = 0.002241
```

The reweighting does not diminish the association between the treatment and the outcome. Indeed, it is marginally more significant after the reweighting.

4 Non-response weights

The `twang` package was designed to estimate propensity score weights for the evaluation of treatment effects in observational or quasi-experimental studies. However, we find that the package includes functions and diagnostic tools that are highly valuable for other applications, such as for generating and diagnosing nonresponse weights for survey nonresponse or study attrition. We now present an example that uses the tools in `twang`. This example uses the subset of the US Sustaining Effects Study data distributed with the HLM software (Bryk, Raudenbush, Congdon, 1996) and also available in the R package `mlmRev`. The data include mathematics test scores for 1721 students in kindergarten to fourth grade. They also include student race (black, Hispanic, or other), gender, an indicator for whether or not the student had been retained in grade, the percent low income students at the school, the school size, the percent of mobile students, the students' grade-levels, student and school IDs, and grades converted to year by centering. The study analysis plans to analyze growth in math achievement from grade 1 to grade 4 using only students with complete data. However, the students with complete data differ from other students. To reduce bias that could potentially result from excluding incomplete cases, our analysis plan is to weight complete cases with nonresponse weights.

The goal of nonresponse weighting is to develop weights for the respondents that make them look like the entire sample — both the respondents and nonrespondents. Since the respondents already look like themselves, the hard part is to figure out how well each respondent represents the nonrespondents. Nonresponse weights equal the reciprocal of the probability of response and are applied only to respondents.

Note that the weights p are equivalent to the propensity score if we consider subjects with an observed outcome to be the "treated" group, and those with an unobserved outcome to be the "controls". We wish to reweight the sample to make it equivalent to the population from which the sample was drawn, so ATE weights are more appropriate in this case. Further, recall that the weights for the treated subjects are $1/p$ in an ATE analysis. Therefore we can reweight the sample of respondents using the `get.weights()` function.

Before we can generate nonresponse weights, we need to prepare the data using the following commands. First we load the data.

```
> data(egsingle)
```

Next we create the patterns of grades for which students have responses

```
> tmp <- sapply(split(egsingle,egsingle$childid),function(x){
+               paste(as.character(x$grade),collapse="")})
```

identify students with test scores for every grade from 1 to 4

```
> tmp <- data.frame(childid=names(tmp), gpatt=tmp,
+                   resp=as.numeric((1:length(tmp)) %in%
+                           grep("1234",as.character(tmp))))
```

and merge this back to create a single data frame

```
> egsingle <- merge(egsingle, tmp)
```

Because nonresponse is a student-level variable rather than a student-by-year-level variable we create one record per student.

```
> egsingle.one <-unique(egsingle[,-c(3:6)])
```

We also create a race variable

```
> egsingle.one$race <- as.factor(race <- ifelse(egsingle.one$black==1, 1,
+                                          ifelse(egsingle.one$hispanic==1, 2, 3)))
```

As discussed above, to use `ps()` to estimate nonresponse, we need to let respondents be the treatment group by modeling an indicator of response.

```
> egsingle.ps <-
+    ps(resp ~ race + female + size + lowinc + mobility,
+       data=egsingle.one,
+       stop.method=c("es.mean","ks.max"),
+       n.trees=2500,
+       verbose=FALSE,
+       estimand = "ATE")
```

The optimal number of iterations for gbm to minimize the maximum KS statistic is 2468 and the optimal number of iterations for gbm to minimize the average effect size is 2379. The weights do an excellent job matching the distribution of the respondent group covariates to those of the nonrespondents as shown in Table 6.

```
> pretty.tab<-bal.table(egsingle.ps)$ks.max.ATE[,c("tx.mn","ct.mn","std.eff.sz","ks")]
> names(pretty.tab) <- c("Non-responders","Weighted responders","Std ES","KS")
> xtable(pretty.tab,
+        caption = "Balance of the nonrespondents and respondents",
+        label = "tab:balance2",
+        digits = c(0, 2, 2, 2, 2),
+        align=c("l","r","r","r","r"))
```

The final steps are to find the ATE weights

```
> egsingle.one$wgt <- get.weights(egsingle.ps, stop.method="ks.max")
```

and select only the records with an observed outcome.

```
>  egsinge.resp <- merge(subset(egsingle, subset=resp==1),
+                       subset(egsingle.one, subset=resp==1,
+                              select=c(childid, wgt)) )
```

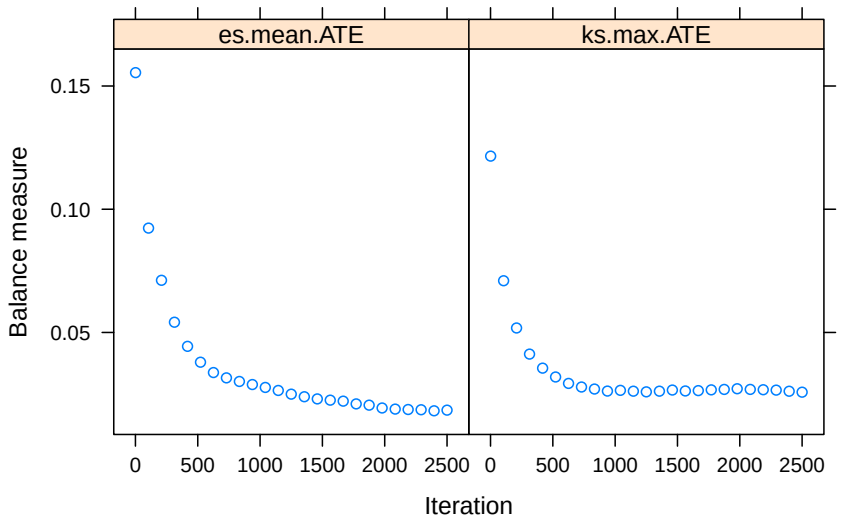


Figure 2: Optimization of `es.mean.ATE` and `ks.max.ATE` for nonresponse weighting of egsingle data. The horizontal axes indicate the number of iterations and the vertical axes indicate the measure of imbalance between the two groups. For `es.mean.ATE` the measure is the average effect size difference between the two groups and for `ks.max.ATE` the measure is the largest of the KS statistics

	Non-responders	Weighted responders	Std ES	KS
race:1	0.69	0.70	-0.03	0.01
race:2	0.14	0.14	-0.01	0.00
race:3	0.17	0.15	0.04	0.02
female:Female	0.49	0.49	-0.01	0.00
female:Male	0.51	0.51	0.01	0.00
size	756.72	758.89	-0.01	0.02
lowinc	78.52	78.48	0.00	0.03
mobility	34.23	34.79	-0.04	0.02

Table 6: Balance of the nonrespondents and respondents

5 The details of `twang`

5.1 Propensity scores and weighting

Propensity scores can be used to reweight comparison cases so that the distribution of their features match the distribution of features of the treatment cases, for ATT, or cases from both treatment and control groups to match each other, for ATE (Rosenbaum 1987, Wooldridge 2002, Hirano and Imbens 2001, McCaffrey *et al.* 2004) Let $f(\mathbf{x}|t=1)$ be the distribution of features for the treatment cases and $f(\mathbf{x}|t=0)$ be the distribution of features for the comparison cases. If treatments were randomized then we would expect these two distributions to be similar. When they differ for ATT we will construct a weight, $w(\mathbf{x})$, so that

$$f(\mathbf{x}|t=1) = w(\mathbf{x})f(\mathbf{x}|t=0). \tag{2}$$

For example, if $f(\text{age=}65, \text{sex=F}|t=1) = 0.10$ and $f(\text{age=}65, \text{sex=F}|t=0) = 0.05$ (i.e. 10% of the treatment cases and 5% of the comparison cases are 65 year old females) then we need to give a weight of 2.0 to every 65 year old female in the comparison group so that they have the same representation as in the treatment group. More generally, we can solve (2) for $w(\mathbf{x})$ and apply Bayes Theorem to the numerator and the denominator to give an expression for the propensity score weight for comparison cases,

$$w(\mathbf{x}) = K\frac{f(t=1|\mathbf{x})}{f(t=0|\mathbf{x})} = K\frac{P(t=1|\mathbf{x})}{1-P(t=1|\mathbf{x})}, \tag{3}$$

where K is a normalization constant that will cancel out in the outcomes analysis. Equation (3) indicates that if we assign a weight to comparison case i equal to the odds that a case with features $\mathbf{x}_i$ would be exposed to the treatment, then the distribution of their features would balance. Note that for comparison cases with features that are atypical of treatment cases, the propensity score $P(t=1|\mathbf{x})$ would be near 0 and would produce a weight near 0. On the other hand, comparison cases with features typical of the treatment cases would receive larger weights.

For ATE, each group is weighted to match the population. The weight must satisfy:

$$f(\mathbf{x}|t=1) = w(\mathbf{x})f(\mathbf{x}), \text{ and} \tag{4}$$

$$f(\mathbf{x}|t=0) = w(\mathbf{x})f(\mathbf{x}), \text{ and} \tag{5}$$

Again using Bayes Theorem we obtain $w(\mathbf{x}) = 1/f(\mathbf{x}|t=1)$ for the treatment group and $w(\mathbf{x}) = 1/f(\mathbf{x}|t=0)$ for the control group.

5.2 Estimating the propensity score

In randomized studies $P(t = 1|\mathbf{x})$ is known and fixed in the study design. In observational studies the propensity score is unknown and must be estimated, but poor estimation of the propensity scores can cause just as much of a problem for estimating treatment effects as poor regression modeling of the outcome. Linear logistic regression is the common method for estimating propensity scores, and can suffice for many problems. Linear logistic regression for propensity scores estimates the log-odds of a case being in the treatment given $\mathbf{x}$ as

$$\log \frac{P(t = 1|\mathbf{x})}{1 - P(t = 1|\mathbf{x})} = \beta'\mathbf{x} \tag{6}$$

Usually, β is selected to maximize the logistic log-likelihood

$$\ell\beta = \frac{1}{n}\sum_{i=1}^{n} t_i\beta'\mathbf{x}_i - \log\left(1 + \exp(\beta'\mathbf{x}_i)\right) \tag{7}$$

Maximizing (7) provides the maximum likelihood estimates of β. However, in an attempt to remove as much confounding as possible, observational studies often record data on a large number of potential confounders, many of which can be correlated with one another. Standard methods for fitting logistic regression models to such data with the iteratively reweighted least squares algorithm can be statistically and numerically unstable. To improve the propensity score estimates we might also wish to include non-linear effects and interactions in $\mathbf{x}$. The inclusion of such terms only increases the instability of the models.

One increasingly popular method for fitting models with numerous correlated variables is the lasso (least absolute subset selection and shrinkage operator) introduced in statistics in Tibshirani (1996). For logistic regression, lasso estimation replaces (7) with a version that penalizes the absolute magnitude of the coefficients

$$\ell\beta = \frac{1}{n}\sum_{i=1}^{n} t_i\beta'\mathbf{x}_i - \log\left(1 + \exp(\beta'\mathbf{x}_i)\right) - \lambda\sum_{j=1}^{J}|\beta_j| \tag{8}$$

The second term on the right-hand side of the equation is the penalty term since it decreases the overall of $\ell\beta$ when there are coefficient that are large in absolute value. Setting $\lambda = 0$ returns the standard (and potentially unstable) logistic regression estimates of β. Setting λ to be very large essentially forces all of the β_j to be equal to 0 (the penalty excludes β_0). For a fixed value of λ the estimated $\hat{\beta}$ can have many coefficients exactly equal to 0, not just extremely small but precisely 0, and only the most powerful predictors of t will be non-zero. As a result the absolute penalty operates as a variable selection penalty. In practice, if we have several predictors of t that are highly correlated with each other, the lasso tends to include all of them in the model, shrink their coefficients toward 0, and produce a predictive model that utilizes all of the information in the covariates, producing a model with greater out-of-sample predictive performance than models fit using variable subset selection methods.

Our aim is to include as covariates all piecewise constant functions of the potential confounders and their interactions. That is, in $\mathbf{x}$ we will include indicator functions for continuous variables like $I(\text{age} < 15), I(\text{age} < 16), \ldots, I(\text{age} < 90)$, etc., for categorical variables like $I(\text{sex} = \text{male}), I(\text{prior MI} = \text{TRUE})$, and interactions among them like $I(\text{age} < 16)I(\text{sex} = \text{male})I(\text{prior MI} = \text{TRUE})$. This collection of basis functions spans a plausible set of propensity score functions, are computationally efficient, and are flat at the extremes of $\mathbf{x}$ reducing the likelihood of propensity score estimates near 0 and 1 that can occur with linear basis functions of $\mathbf{x}$. Theoretically with the lasso we can estimate the model in (8), selecting a λ small enough so that it will eliminate most of the irrelevant terms and yield a sparse model with only the

most important main effects and interactions. Boosting (Friedman 2001, 2003, Ridgeway 1999) effectively implements this strategy using a computationally efficient method that Efron *et al.* (2004) showed is equivalent to optimizing (8). With boosting it is possible to maximize (8) for a range of values of λ with no additional computational effort than for a specific value of λ. We use boosted logistic regression as implemented in the generalized boosted modeling (`gbm`) package in R (Ridgeway 2005).

5.3 Evaluating the weights

As with regression analyses, propensity score methods cannot adjust for unmeasured covariates that are uncorrelated with the observed covariates. Nonetheless, the quality of the adjustment for the observed covariates achieved by propensity score weighting is easy to evaluate. The estimated propensity score weights should equalize the distributions of the cases' features as in (2). This implies that weighted statistics of the covariates of the comparison group should equal the same statistics for the treatment group. For example, the weighted average of the age of comparison cases should equal the average age of the treatment cases. To assess the quality of the propensity score weights one could compare a variety of statistics such as means, medians, variances, and Kolmogorov-Smirnov statistics for each covariate as well as interactions. The `twang` package provides both the standardized effect sizes and KS statistics and p-values testing for differences in the means and distributions of the covariates for analysts to use in assessing balance.

5.4 Analysis of outcomes

With propensity score analyses the final outcomes analysis is generally straightforward, while the propensity score estimation may require complex modeling. Once we have weights that equalize the distribution of features of treatment and control cases by reweighting. For ATT, we give each treatment case a weight of 1 and each comparison case a weight $w_i = p(\mathbf{x}_i)/(1-p(\mathbf{x}_i))$. To estimate the ATE, we give control cases weight $w_i = 1/p(\mathbf{x}_i)$ and we give the treatment cases $w_i = 1/(1-p(\mathbf{x}_i)$. We then estimate the treatment effect estimate with a weighted regression model that contains only a treatment indicator. No additional covariates are needed if the weights account for differences in $\mathbf{x}$.

A combination of propensity score weighting and covariate adjustment can be useful for several reasons. First, the propensity scores may not have been able to completely balance all of the covariates. The inclusion of these covariates in addition to the treatment indicator in a weighted regression model may correct this if the imbalance is relatively small. Second, in addition to exposure, the relationship between some of the covariates and the outcome may also be of interest. Their inclusion can provide coefficients that can estimate the direction and magnitude of the relationship. Third, as with randomized trials, stratifying on covariates that are highly correlated with the outcome can improve the precision of estimates. Lastly, the some treatment effect estimators that utilize an outcomes regression model and propensity scores are "doubly robust" in the sense that if either the propensity score model is correct or the regression model is correct then the treatment effect estimator will be unbiased (Bang & Robins 2005).

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