

Deep Propensity Network using Sparse Autoencoders

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Abstract

Understanding causality is critical, especially in biomedical and social science; and the gold-standard solution is to perform randomized experiments, enacting interventional probabilities as opposed to conditional ones, but it is not often feasible due to operational or ethical constraints. On the other hand, drawing causal estimates from pre-existing, observational data is problematic, because datasets are often littered with underlying bias. Identifying the true causal effects however is important to evaluate what-if scenarios, i.e. counterfactuals. In fact, a prediction model based only on conditional probabilities, even with perfect accuracy, is neither guaranteed to estimate the correct causal effect, in terms of individual or average treatment effects (ITE, ATE), nor to calculate the correct counterfactuals. Propensity score matching (PSM) is a popular statistical approach for observational data that attempts to estimate the effect of an intervention, e.g. a medical treatment, by taking into account other factors that may bias the chance to undertake the intervention itself, e.g. social discrimination. PSM is usually implemented with logistic regression, but its performance can be limited due to linearity, high-dimensionality, and residual confounding in the feature space. Recently, deep counterfactual neural networks with propensity dropout (DCN-PD) have been introduced, enabling nonlinear PSM with advantage over classical methods in terms of treatment assignment error and ATE. In this work, we propose a deep propensity network using a sparse autoencoder (DPN-SA), a novel deep learning architecture for PSM to tackle the problem of high-dimensionality and residual confounding. DPN-SA takes advantage of a sparse autoencoder to reduce the dimension of the feature space. Tests performed on real-world observational data showed that the DPN-SA outperforms logistic regression-, LASSO-, and DCN-PD in estimating treatment assignment probability, ITE and ATE. The code is available under the MIT license on Github at: <https://github.com/Shantanu48114860/>

1 Introduction

In many research fields, such as biomedical and social sciences, preexisting (i.e. observational) data may contain underlying bias, arising in various steps of the data generation or collation process, for which datasets cannot be used

seamlessly to draw causal claims (Prospero et al. 2020). For instance, one could be interested in studying the effectiveness of certain medical treatments or interventions in the population, but the way in which people access the healthcare system could be different (e.g. due to social inequality, systemic racism). Therefore, due to such bias, the causal effects of the treatments could not be estimated properly. One solution would be to force the interventions to be non-discriminated, performing randomized controlled experiments or trials (RCT) (Sibbald and Roland 1998) (Concato, Shah, and Horwitz 2000). In RCT, individuals are assigned to different treatment (or control, no treatment) groups at random, regardless their background characteristics (i.e. the covariate or feature space). The mean difference between the observed treatment outcomes of two different groups is called the average treatment effect (ATE). Note that the individual treatment effect (ITE) is a missing data problem (Pearl, Glymour, and Jewell 2016) (Hernan and Robins 2019), because only one factual outcome can be observed (a person cannot be assigned to both treatment and control groups). Since RCT are not always feasible due to ethical or operational constraints, e.g. conducting RCT to assess the effect of smoking toward development of lung cancer, observational data are often used, and one must account for the possible types of underlying bias, including confounders and colliders (Greenland and Morgenstern 2001).

Propensity score matching (PSM) is a popular statistical approach for observational data that attempts to estimate the causal effect of a treatment variable with respect to an outcome, taking into account possible confounding bias from other pre-treatment characteristics (Rosenbaum and Rubin 1983) (Rosenbaum and Rubin 1984). The propensity score is a scalar estimate $\pi(\mathbf{x})$ representing the conditional probability of receiving a certain treatment $T = 1$, versus the control group or no treatment $T = 0$, given a set of measured pre-treatment covariates \mathbf{X} , denoted as

$$\pi(\mathbf{x}) = P(T = 1 | \mathbf{X} = \mathbf{x}), \quad (1)$$

Hence, PSM balances the pre-treatment potential confounders by achieving a quasi-randomization of the different treatment group assignments, allowing a better estimation of the treatment effect. However, PSM accounts only for mea-

sured (and measurable) covariates, and latent bias may remain after matching (Garrido and others 2014).

PSM has been implemented historically through logistic regression, which calculates the probability of treatment assignment given the pre-treatment covariates (Kurth et al. 2005). In presence of high-dimensional datasets, compiled from large electronic health record (EHR) databases (Prosperi et al. 2018), different feature selection methods within PSM have been employed, such as the high-dimensional propensity score (hdPS) (Schneeweiss et al. 2009) or L1-logistic regression (Tian, Schuemie, and Suchard 2018). However, logistic regression is limited because it calculates a mere linear combination of input variables, thus it is not able to capture complex relationships between the pre-treatment covariates and the treatment assignment. This is particularly true in high-dimensional settings, where it is problematic to define explicitly variable-to-variable interactions, e.g. as higher-order terms in the logistic function, and computationally burdensome to scan all of them.

An artificial neural network is a universal approximator, and can smooth polynomial functions regardless of the order of the polynomial or the number of interaction terms (Barron 1994) (Bishop and others 1995) (Mhaskar 1996). In addition, it does not require *a priori* knowledge of what interactions and functional forms are likely to be relevant among covariates. Therefore, it is suited to overcome the issues of PSM by logistic regression. The network can provide estimation of probability of treatment group assignment (e.g. through *softmax*), and consequently of ATE and ITE.

Alaa, Weisz, and van Der Schaar designed a multitask deep counterfactual network with propensity dropout (DCN-PD) (Alaa, Weisz, and Van Der Schaar 2017) which is a feed-forward network with a set of shared layers is used to calculate the counterfactual outcomes, being regularized by another network that calculates propensity scores and a dropout probability for training examples (Gal and Ghahramani 2016), through alternate training phases (treatment vs. control groups) where the shared and the outcome layers' weights are updated. The DCN-PD led to lower mean squared error (MSE) in treatment assignments and more accurate ATE than other classic PSM methods.

In this work, we propose a novel deep neural architecture—the deep propensity network using a sparse autoencoder (DPN-SA)—that addresses the problem of high-dimensional PSM, yet maintains the MSE advantage of the DCN-PD approach as compared to others. The DPN-SA estimates the propensity score using a sparse autoencoder (Ng and others 2011) which at the same time learns a nonlinear feature representation and reduces the dimensionality of the pre-treatment covariate space. The sparse autoencoder is learnt in an end-to-end manner, while the counterfactual prediction follows the DCN-PD layout.

2 Methodology

2.1 Problem Formulation

Let assume a population sample (independent and identically distributed) of N ($1 \dots i \dots n$) individuals, given background set of pre-treatment covariates \mathbf{X} , a treatment T

(binary, for simplicity) and a health outcome Y . Each subject i is represented by the tuple $\{\mathbf{X}_i, T_i, Y_i\}$. Let Y_i^0 and Y_i^1 the potential outcome for individual i under treatment $T_i = 0$ and $T_i = 1$, respectively (Rubin 1974) (Rubin 2005). Given $\mathbf{X}_i = \mathbf{x}$, the ITE $\tau(\mathbf{x})$ is defined as the difference in the mean potential outcomes for the individual i under both treatments, conditional on the observed covariate vector \mathbf{x}

$$\tau(\mathbf{x}) = \mathbb{E}[Y_i^1 - Y_i^0 \mid \mathbf{X}_i = \mathbf{x}] \quad (2)$$

The ITE formulation as $\tau(\mathbf{x})$ —called the counterfactual framework—is usually incalculable in reality, since people cannot be assigned two different treatments at the same time. However, under the assumption of strongly ignorable treatment assignment (SITA), which means that the potential outcomes are independent of treatment conditional on background variables, i.e. $\{Y_i^1, Y_i^0\} \perp T \mid \mathbf{X}$ (Imbens 2000) (Lechner 2001) (Pearl, Glymour, and Jewell 2016) (Peters, Janzing, and Schölkopf 2017).

Under the assumption of SITA, the ITE can be calculated as $\tau(\mathbf{x}) = \mathbb{E}[Y^1 \mid T = 1, \mathbf{X} = \mathbf{x}] - \mathbb{E}[Y^0 \mid T = 0, \mathbf{X} = \mathbf{x}] = \mathbb{E}[Y \mid T = 1, \mathbf{X} = \mathbf{x}] - \mathbb{E}[Y \mid T = 0, \mathbf{X} = \mathbf{x}]$.

Further, under SITA and by averaging over the distribution of \mathbf{X} , the ATE τ_{01} can be calculated as

$$\tau_{01} = \mathbb{E}[\tau(\mathbf{X})] = \mathbb{E}[Y \mid T = 1] - \mathbb{E}[Y \mid T = 0] \quad (3)$$

However, by assuming SITA, ITE and ATE can be calculated only with \mathbf{x} being the same in treatment groups, which becomes quickly unfeasible when the dimension of \mathbf{x} grows. PSM, through the conditional probability $\pi(\mathbf{x})$ (see eq. 1) attempts at balancing the probability of receiving T given $\mathbf{X} = \mathbf{x}$.

Once propensity scores are obtained for a population sample, the individuals in the treatment group must be matched those in the control group to make sure that they are balanced with respect to the background covariates; this is a second problem for which a number of (approximate) solutions can be used, including k -nearest neighbor, Caliper matching (Austin 2014), and propensity weighing (Abadie and Imbens 2016) (on which the DCN-PD was based).

2.2 Proposed Approach

The DCN-PD method (Alaa, Weisz, and Van Der Schaar 2017) offered a novel and effective way to calculate nonlinear propensity scores as well as to match individuals, providing better ITE/ATE estimation over classical PSM methods. However, DCN-PD might be affected by curse of dimensionality, with associated residual confounding in study settings where the covariate space has potentially very high cardinality, e.g. in target trial designs that use EHR (Zhang et al. 2018) (Li et al. 2020). Feature selection and shrinkage has been proven effective for PSM by logistic regression (Schneeweiss et al. 2009) (Tian, Schuemie, and Suchard 2018), and in the same way dimensionality reduction can be implemented in deep neural architectures (Hinton and Salakhutdinov 2006). An autoencoder is a neural network that learns to copy its input to its output (encoder-decoder), but the input is coded into a lower dimension within the hidden layer(s) (Hinton and Zemel 1993). In its simplest form,

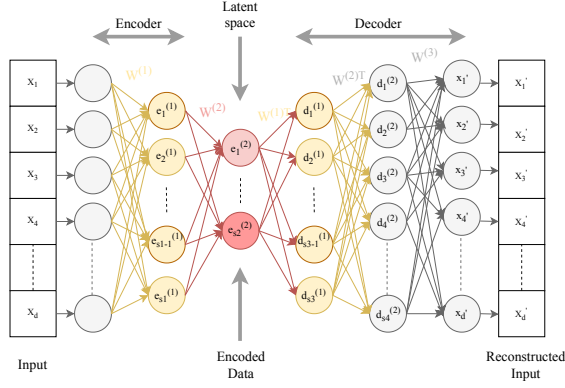


Figure 1: Architecture of a deep stacked sparse autoencoder with the sparsity constraint (brighter neurons are active) enforced in the 2^{nd} layer

i.e. a single layer with linear/sigmoid activations, the autoencoder is closely related to principal component analysis (PCA), while highly nonlinear codes can be achieved by augmenting the layer architecture, e.g. deep beliefs networks (Zhou et al. 2014). Autoencoders have been employed in several applications, from machine translation to drug discovery (Cho et al. 2014) (Zhavoronkov et al. 2019). The sparse autoencoder is an approach that includes extra units (more than inputs) in the hidden layer, but only a small number of them units are allowed to be activated depending on the input (Ng and others 2011) (Makhzani and Frey 2013). It has been also broadly applied in biomedical studies, including imaging and -omics datasets (Yu et al. 2017) (Mao et al. 2018) (Praveen et al. 2018) (Lemsara, Ouadfel, and Fröhlich 2020).

The DPN-SA that we propose here exploits a deep stacked sparse autoencoder to encode the covariate space \mathbf{X} into a lower dimensional, nonlinear feature representation. After training, the decoder is replaced by a *softmax* classifier which calculates the probability of treatment T assignment, thus estimating the propensity score $\pi(\mathbf{X})$. The matching uses the propensity dropout component of the DCN-PD, also used for ITE/ATE calculation. Different training (end-to-end vs greedy stacked) procedures and layer (multiple vs single) architectures are tested on real and synthetic high-dimensional data, as detailed in the next sections.

2.3 Architecture and Training of DPN-SA

A deep stacked sparse autoencoder consists of multiple encoders and decoders to learn the identity function of the feature vectors, stacked up to each other, with the setup of sparsity constraints in the hidden layers, whose neurons can be activated or not depending on the input, as shown in Figure 1.

Let $\mathbf{X} = \{x(1), x(2), \dots, x(N)\}$ represent the population of $x(i)$ individuals ($j = 1 \dots N$ i.i.d samples). Each $x(i) \in \mathbb{R}^d$ is a d -dimensional vector, where d represents the number of pre-treatment covariates, i.e. the number of features. The DPN-SA uses up to l encoder and l decoder layers stacked together one after the other, with an additional lin-

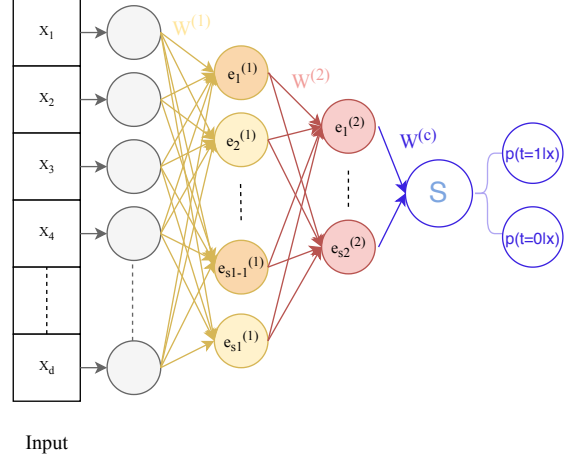


Figure 2: The deep propensity network, stacking the multi-layer sparse autoencoder next to the *softmax* classifier

ear layer at the end of the last decoder. The activation function of each encoder and decoder is the hyperbolic tangent *layertanh*. As $x(i) \in \mathbb{R}^d$, the reconstructed output also needs to be $x'(i) \in \mathbb{R}^d$. Supposing we set $l = 2$, the sample input covariate vector $x(i)$ is reconstructed in the forward propagation as follows:

$$e^{(1)}(i) = f(W^{(1)}x(i) + b^{(1)}) \quad (4)$$

$$e^{(2)}(i) = f(W^{(2)}e^{(1)}(i) + b^{(2)}) \quad (5)$$

$$d^{(1)}(i) = f(W^{(2)T}e^{(2)}(i) + b^{(3)}) \quad (6)$$

$$d^{(2)}(i) = f(W^{(1)T}d^{(1)}(i) + b^{(4)}) \quad (7)$$

$$x'(i) = W^{(3)}d^{(2)}(i) + b^{(5)} \quad (8)$$

where, $e^{(l)}(i)$, $d^{(l)}(i)$, $x'(i)$ are the activations of the encoder, decoder and the reconstructed input of l^{th} layer for the i^{th} sample, respectively. $W^{(l)}$ and $W^{(2)}$ are the weight matrices of encoder 1 and encoder 2, having sizes $s1 \times d$ and $s2 \times s1$, $W^{(2)T}$ and $W^{(1)T}$ are the weight matrices of decoder 1 and decoder 2 having sizes $s1 \times s2$ and $d \times s1$. $s(l)$ denotes size or number of neurons of l^{th} layer and $f(\cdot)$ is the activation function (*tanh*).

After reconstructing the a sample input feature $x(i)$, the objective function of the sparse autoencoder $J_{sparse}(W, b)$ has to be minimized, as described in (Ng and others 2011):

$$J(W, b) = \left[\frac{1}{2N} \sum_{i=1}^N \|x'_{W,b}(i) - x(i)\|^2 \right] + \frac{\lambda}{2N} \sum_{l=1}^L \|W^{(l)}\|_F^2 \quad (9)$$

$$J_{sparse}(W, b) = J(W, b) + \beta \sum_{j=1}^{s2} KL(\rho || \rho_j) \quad (10)$$

where λ is the regularization parameter, $W^{(l)}$ is the weight matrix corresponding to l^{th} layer of the network,

s_2 is the number of neurons in the 2^{nd} hidden layer, β is the weight of the sparsity penalty, KL is the Kullback-Leibler divergence, and the subscript F is the Frobenius norm (equivalent to the squared norm of the weight matrix). The $J_{sparse}(W, b)$ is minimized using backpropagation, for K_{sa} number of epochs.

The DPN-SA is trained in two phases. In the first phase the sparse autoencoder is trained and optimized (K_{sa} epochs). The parameters (W, b) are updated by the Adam optimizer in each iteration. After the sparse autoencoder has learnt the latent representation of the covariates, the decoder part is removed and a *softmax* classifier is attached to the end of the last encoder layer, as shown in 2. The *softmax* classifier is trained for K_c number of epochs. The final network gives the estimation of the propensity score $\pi(x)$. Algorithm 1 describes the two-phase procedures to obtain the final DPN-SA.

Algorithm 1 Training DPN-SA

Input: Batch B of random samples with assigned treatment T , training set X_{train} , number of epochs K_s , K_c , learning rates lr_{SA} , lr_c for the sparse autoencoder (SA) and the *softmax* classifier, respectively.

Output: PS_{out} consisting of propensity score for each sample i , where i ranges from 1 to N .

```

1: procedure DPN-SA TRAINING
2:   Initialise  $W^{(l)}, b^{(l)}$  of the SA.
3:   for  $epochs = 1, 2, \dots, K_s$  do
4:     for  $batches = 1, 2, \dots, B$  in  $X_{batch}$  do
5:       Compute  $x'_{batch}$  using forward propagation.
6:       Compute  $J_{sparse}$ .
7:       Compute Gradient:  $\nabla(J_{sparse})$ 
8:        $(W^{(l)}, b^{(l)}) \leftarrow \text{Adam}(X_{batch}, W^{(l)}, b^{(l)})$ 
9:     end for
10:  end for
11:  Remove the decoder from the SA
12:  Attach a softmax classifier to the last encoder
13:  Initialize  $W^{(c)}, b^{(c)}$  of the classifier
14:   $PS_{out} \leftarrow \text{Empty}$ 
15:  for  $epochs = 1, 2, \dots, K_c$  do
16:    for  $batches = 1, 2, \dots, B$  in  $X_{batch}$  do
17:       $(W^{(l)}, b^{(l)}, W^{(c)}, b^{(c)}) \leftarrow \text{Adam}(X_{batch},$ 
18:         $W^{(l)}, b^{(l)}, W^{(c)}, b^{(c)})$ 
19:      Get the propensity score  $\pi(T|X_{batch})$  and
20:      add to  $PS_{out}$ 
21:    end for
22:  end for
23: end procedure

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2.4 Experimental Setup

Datasets. We used the Infant Health and Development Program (IHDP) dataset, a multi-site, longitudinal, randomized, clinical trial designed to evaluate the efficacy of comprehensive early intervention in enhancing the outcomes of low birth weight, prematurely born infants in the United States. The dataset consists of 747 subjects (139 in the treatment group and 608 controls), with 25 associated covari-

ates, describing characteristics of the infants and their mothers. The datasets has been used as a benchmark in several causal inference methodology and modelling studies, including (Hill 2011) and (Alaa, Weisz, and Van Der Schaar 2017).

In addition to IHDP, we also created a synthetic data set with larger covariate space, first by doubling the original feature set through the creation of 25 random variables (shuffling the original ones), and then triplicating it by creating another set of 25 covariates partially correlated to the original ones (apprx. $\rho=0.4$), using a Gaussian noise addition $\mathcal{N}(0, 2\sigma(x_k))$ to each original variable x_k .

DPN-SA and Other Comparison Methods. Four different configurations of the DPN-SA were tested –on both real and synthetic datasets: (i) 25-20-10-20-25; (ii) 25-10-25, which is similar to PCA; (iii) 25-1-25, which is similar to regularized logistic regression. For all four, both end-to-end and stacked greedy training were executed. The greedy strategy is a layer-wise training where each layer is trained independently, and then stacked to the others. We used 200 epochs (K_s) for the sparse autoencoder, and the Adam optimizer. The learning rates for the sparse autoencoder and for the *softmax* were 0.0001 and 0.01, respectively. We set weight decay (λ), sparsity parameter (ρ), and sparse penalty (β) to 0.0003, 0.08, and 0.4, respectively, with a batch size of 32. The *softmax* classifier was ran for 100 epochs (K_c) with a batch size of 32. The DPN-SA was implemented using the *Pytorch* framework (<https://pytorch.org/>).

In addition to the DPN-SA, we run and compared: (i) the original DCN-PD, (ii) a standard logistic regression, and (iii) logistic regression with LASSO regularization.

Testing. The models were trained on 80% of the data and validated on the remaining 20%, repeating the procedure for 100 times, calculating MSE, ITE and ATE in the same way as (Alaa, Weisz, and Van Der Schaar 2017). The error distributions were compared by means of a t-test with sample overlap adjustment (Nadeau and Bengio 2003).

3 Results

Figures 3 and 4 shows the histogram, quantile-quantile, and correlation plot of the $\pi(X)$ distribution (stratified by treatment group) of the PDN-SA and the other models (real dataset). Table 1 shows the MSE results for all models (real and synthetic data), while Figure 5 shows the violin-plots of each model's ATE estimation (real dataset), compared to the true ATE.

On the real dataset, the DPN-SA configured with the 25-20-10-20-25 layer stacking, trained in an end-to-end manner, yielded the best performance in terms of MSE, with an improvement of 7% over the DCN-PD, and 32% over the LASSO logistic regression. The one-neuron DPN-SA 25-1-25 configuration was better than LASSO, and the PCA-like DPN-SA 25-10-25 . .

On the synthetic dataset, performance decreased due to the artificial noise addition, but the regularization/sparsity constraints demonstrated to robust against such noise and the additional correlated variables. The DPN-SA maintained the advantage over the other models.

Dataset	# Covariates	Model	MSE (SD)	Bengio's p-value	Raw p-value
Real	25	DPN-SA (25-20-10-20-25) end-to-end	1.92 (0.09)	Ref.	Ref.
		DPN-SA (25-20-10-20-25) stacked	2.12 (0.23)	0.096	1.17e-13
		DPN-SA (25-1-25)	2.32 (0.18)	0.001	5.18e-34
		DPN-SA (25-10-25)	? (?)	?	?
		DCN-PD	2.06 (0.15)	0.099	1.75e-13
		Logistic Regression	2.74 (0.49)	0.003	1.47e-29
		LASSO Logistic Regression	2.54 (0.34)	0.001	3.02e-33
Real + Synthetic	75	DPN-SA (25-20-10-20-25) end-to-end	? (?)	Ref.	Ref.
		DPN-SA (25-20-10-20-25) stacked	? (?)	?	?
		DPN-SA (25-1-25)	? (?)	?	?
		DPN-SA (25-10-25)	? (?)	?	?
		DCN-PD	? (?)	?	?
		Logistic Regression	? (?)	?	?
		LASSO Logistic Regression	? (?)	?	?

Table 1: Performance of the models against IHDP dataset

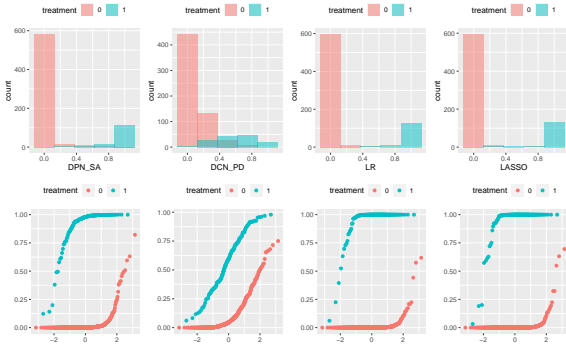


Figure 3: Histograms and quantile-quantile plots of the propensity score distributions (stratified by treatment group) for the DPN-SA, DCN-PD, Logistic Regression, and LASSO.

The end-to-end training in general yielded lower MSE, but due to the limited sample size, the null hypothesis of no difference could not be rejected at the 0.05 significance level.

The propensity scores of the DPN-SA exhibited a more polarized distribution toward the extremes as compared to DCN-PD, more similar to logistic regression and LASSO. In terms of ATE, the DPN-SA was as close at the DCN-PD to the true value, and showed lower variance. Both logistic regression and LASSO substantially underestimated the ATE.

4 Discussion

The DPN-SA architecture conjugates the ability to calculate nonlinear propensity scores with feature regularization, and demonstrates advantage over other methods in MSE and treatment effect estimation. The multi-layer deep architecture also improves over less complex autoencoder configurations that are akin to LASSO and PCA. The advantage of DPN-SA over the DCN-PD is also appreciable, especially ...

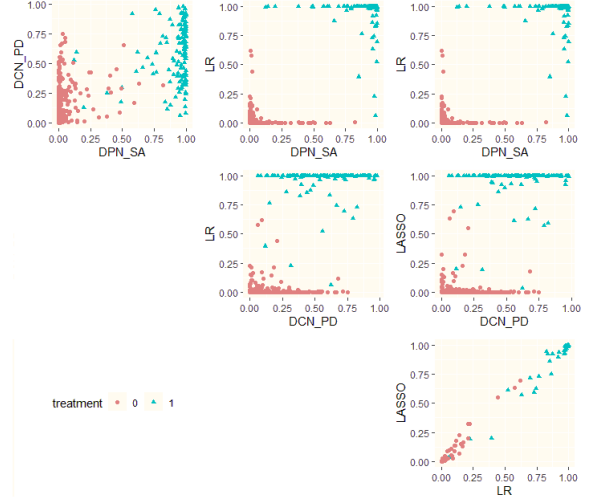


Figure 4: Correlation plots of the propensity score distributions (stratified by treatment group) comparing the DPN-SA, DCN-PD, Logistic Regression, and LASSO.

This work has some limitations. First, the choice of a *softmax* classifier as a replacement to the decoder is relatively simplistic, nonetheless, it provided lower MSE and lower-variance ATE. Other solutions for embedding the sparse autoencoder within the DCN-PD framework or other approaches, such as ITE estimation with generalized adversarial networks (Yoon, Jordon, and van der Schaar 2018), could be devised. Another limitation is that the benchmark data set has a limited sample size, and therefore the differences in average performance between models are subject to uncertainty. Finally, the distribution of propensity scores among treatment and control groups is often highly dependent on the dataset, and can be highly imbalanced, and therefore the results obtained with one experimental data set could not be replicated with others.

In conclusion, the DPN-SA provides a valid, possibly im-

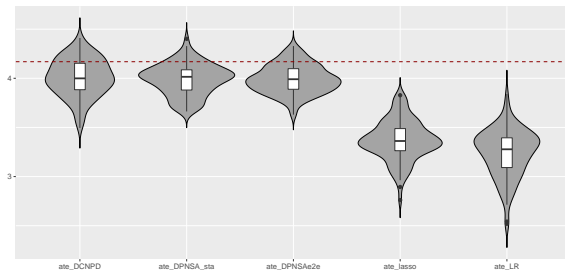


Figure 5: Boxplots of average treatment effect estimation (dashed line is the true effect).

proved, alternative to DCN-PD and to more traditional PSM methods.

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