Domain Adaptation by Using Causal Inference to Predict Invariant Conditional Distributions

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Abstract

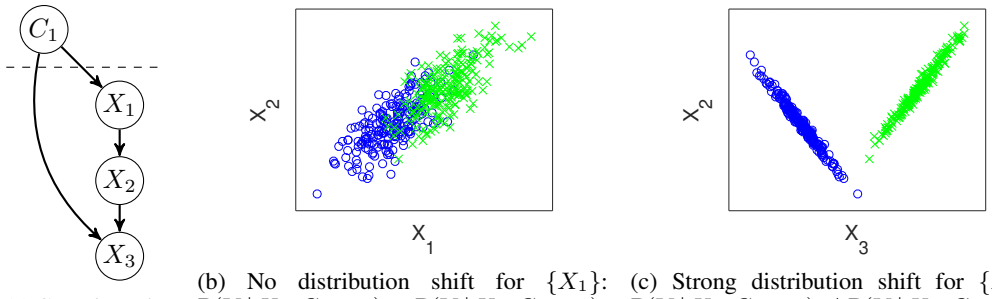
An important goal common to domain adaptation and causal reasoning is to make accurate predictions when the distributions for the target domain(s) and the source domain(s) differ. We consider the case in which the domains correspond to different contexts in which a system has been measured, for example, a purely observational context and several interventional contexts in which the system has been perturbed by external interventions. We consider a class of such *causal* domain adaptation problems, where data for multiple source domains are given, and the task is to predict the distribution of a certain target variable from measurements of other variables in one or more target domains. We propose an approach for solving these problems that exploits causal inference and does not rely on prior knowledge of the causal graph, nor of the type of the interventions or the intervention targets. We propose a practical implementation of the approach and evaluate it on simulated and real world data.

1 Introduction

Predicting unknown values based on observed data is a problem central to many sciences, and well studied in statistics and machine learning. This problem becomes significantly harder if the training and test data do not have the same distribution because they come from different domains. Such a distribution shift will happen in practice whenever the circumstances under which the training data were gathered are different from those for which the predictions are to be made. A rich literature exists on this problem of *domain adaptation*, a particular task in the field of *transfer learning*; see e.g. Quiñonero-Candela et al. (2009); Pan and Yang (2010) for overviews.

When the domain changes, so may the relations between the different variables under consideration. While for some (sets of) variables $\boldsymbol{A}$, a function $f : \boldsymbol{\mathcal{A}} \to \mathcal{Y}$ learned in one domain may continue to offer good predictions for $Y \in \mathcal{Y}$ in a different domain, this may not be true of other sets $\boldsymbol{A}'$ of variables. *Causal graphs* (e.g., Pearl, 2009; Spirtes et al., 2000) allow us to reason about this in a principled way when the domains correspond to different external *interventions* on the system, or more generally, to different contexts in which a system has been measured. Knowledge of the causal graph that describes the data generating mechanism, and of which parts of the model are invariant

32nd Conference on Neural Information Processing Systems (NIPS 2018), Montréal, Canada.



(a) Causal graph

(b) No distribution shift for $\{X_1\}$: $\mathbb{P}(Y \mid X_1, C_1 = 0) = \mathbb{P}(Y \mid X_1, C_1 = 1)$

(c) Strong distribution shift for $\{X_3\}$: $\mathbb{P}(Y \mid X_3, C_1 = 0) \neq \mathbb{P}(Y \mid X_3, C_1 = 1)$

Figure 1: Example of a situation where an intervention C_1 leads to a shift of distribution between source domain and target domain (see also Example 1). Green crosses show source domain data ($C_1 = 0$), blue circles show target domain data ($C_1 = 1$). A standard feature selection method that does not take into account the causal structure but would use X_3 to predict $Y := X_2$ (because X_3 is a good predictor of Y in the source domain) would obtain extremely biased predictions in the target domain. Using X_1 instead yields less accurate predictions in the source domain, but more accurate ones in the target domain.

across the different domains, allows one to transfer knowledge from one domain to the other in order to address the problem of domain adaptation (Spirtes et al., 2000; Storkey, 2009; Schölkopf et al., 2012; Bareinboim and Pearl, 2016).

Over the last years, various methods have been proposed to exploit the causal structure of the data generating process in order to address certain domain adaptation problems, each relying on different assumptions. For example, Bareinboim and Pearl (2016) provide theory for identifiability under transfer ("transportability") assuming that the causal graph is known, that interventions are perfect, and that the intervention targets are known. Hyttinen et al. (2015) also assume perfect interventions with known targets but do not rely on complete knowledge of the causal graph, instead inferring the relevant aspects of it from the data. Rojas-Carulla et al. (2016) make the assumption that if the conditional distribution of the target given some subset of covariates is invariant across different source domains, then this conditional distribution must also be the same in the target domain. The methods proposed in (Schölkopf et al., 2012; Zhang et al., 2013, 2015; Gong et al., 2016) all address challenging settings in which conditional independences that follow from the usual Markov and faithfulness assumptions alone do not suffice to solve the problem, but additional assumptions on the data generating process have to be made.

In this work, we will make no such additional assumptions, and address the setting in which both the causal graph *and* the intervention types and targets may be (partially) unknown. Our contributions are the following. We consider a set of relatively weak assumptions that make the problem well-posed. We propose an approach to solve this class of causal domain adaptation problems that can deal with the presence of latent confounders. The main idea is to select the subset of features $\boldsymbol{A}$ that leads to the best predictions of Y in the source domains, while satisfying *invariance* (i.e., $\mathbb{P}(Y \mid \boldsymbol{A})$ is the same in the source and target domains). To test whether the invariance condition is satisfied, we apply the recently proposed Joint Causal Inference (JCI) framework (Mooij et al., 2018) to exploit the information provided by multiple domains corresponding to different interventions. We propose an implementation of this method based on a causal discovery algorithm by (Hyttinen et al., 2014). The basic idea is as follows. First, a standard feature selection method is applied to source domains data to find sets of features that are predictive of a target variable, trading off bias and variance, but unaware of changes in the distribution across domains. A causal reasoning method then draws conclusions from all given data about the causal graph, avoiding sets of features for which the predictions would not transfer to the target domains. We evaluate the method on synthetic data and a real-world example.

2 Theory

Before giving a precise definition of the class of domain adaptation problems that we consider in this work, we begin with a motivating example.

Example 1. *Given three variables X_1, X_2, X_3 describing different aspects of a system (for example, certain blood cell phenotypes in mice). We have observational measurements of these three variables (the source domain, designated with $C_1 = 0$), and in addition, measurements of X_1 and X_3 under an intervention (the target domain, designated with $C_1 = 1$), e.g., in which the mice have been exposed to a certain drug. The domain adaptation task is to predict the values of $Y := X_2$ in the interventional target domain (i.e., when $C_1 = 1$). Let us assume for this example that the causal graph in Figure 1(a) applies, i.e., we assume that X_2 is affected by X_1 and affects X_3, while C_1 (the intervention) affects both X_1 and X_3. Suppose further that the relation between X_1 and X_2 is about equally strong as the relation between X_2 and X_3, but considerably more noisy. Then a feature selection method using only available source domain data, and aiming to select the best subset of features to use for prediction of Y will prefer both $\{X_3\}$ and $\{X_1, X_3\}$ over $\{X_1\}$ (because predicting Y from X_1 leads to larger variance than predicting Y from X_3, and to a larger bias than predicting Y from both X_1 and X_3). However, under the intervention ($C_1 = 1$), $\mathbb{P}(Y \mid X_3)$ and $\mathbb{P}(Y \mid X_1, X_3)$ both change,[1] so that using those features to predict Y in the target domain could lead to extreme bias, as illustrated in Figure 1(c). Because the conditional distribution of Y given X_1 is invariant across domains, i.e., $\mathbb{P}(Y \mid X_1, C_1 = 0) = \mathbb{P}(Y \mid X_1, C_1 = 1)$, as illustrated in Figure 1(b), predictions of Y based only on X_1 can be safely transferred to the target domain.*

This example provides an instance of a domain adaptation problem where feature selection methods that do not take into account the causal structure would pick a set of features that does not generalize to the target domain, and may lead to arbitrarily bad predictions (even asymptotically, as the number of data points tends to infinity). On the other hand, correctly taking into account the causal structure and the possible distribution shift from source to target domain allows to upper bound the prediction error in the target domain, as we will see in Section 2.3.

2.1 Problem Setting

We now formalize the domain adaptation problems that we address in this paper. We will make use of the terminology of the recently proposed Joint Causal Inference (JCI) framework (Mooij et al., 2018).

Let us consider a system of interest described by a set of *system variables* $\{X_j\}_{j \in \mathcal{J}}$. In addition, we model the domain in which the system has been measured by *context variables* $\{C_i\}_{i \in \mathcal{I}}$ (we will use "context" as a synonym for "domain"). We will denote the tuple of all system and context variables as $\boldsymbol{V} = ((X_j)_{j \in \mathcal{J}}, (C_i)_{i \in \mathcal{I}})$. System and context variables can be discrete or continuous. As a concrete example, the system of interest could be a mouse. The system variables could be blood cell phenotypes such as the concentration of red blood cells, the concentration of white blood cells, and the mean red blood cell volume. The context variables could indicate for example whether a certain gene has been knocked out, the dosage of a certain drug administered to the mice, the age and gender of the mice, or the lab in which the measurements were done. The important underlying assumption is that context variables are *exogenous* to the system, whereas system variables are *endogenous*. The interventions are not limited to the perfect ("surgical") interventions modeled by the do-operator of Pearl (2009), but can also be other types of interventions such as mechanism changes (Tian and Pearl, 2001), soft interventions (Markowetz et al., 2005), fat-hand interventions (Eaton and Murphy, 2007), activity interventions (Mooij and Heskes, 2013), and stochastic versions of all these. Knowledge of the intervention *targets* is not necessary (but is certainly helpful). For example, administering a drug to the mice may have a direct causal effect on an unknown subset of the system variables, but we can simply model it as a binary exogenous variable (indicating whether or not the drug was administered) or a continuous exogenous variable (describing the dosage of the administered drug) without specifying in advance on which variables it has a direct effect. We can now formally state the domain adaptation task that we address in this work:

Task 1 (Domain Adaptation Task)**.** *Given are data for a single or multiple source domains, in each of which $C_1 = 0$, and for a single or multiple target domains, in each of which $C_1 = 1$. Assume the source domains data is complete (i.e., no missing values), and the target domains data is complete with the exception of all values of a certain target variable $Y = X_j$. The task is to predict these missing values of the target variable Y given the available source and target domains data.*

[1] More precisely, we should say that $\mathbb{P}(Y \mid X_3, C_1 = 1)$ may differ from $\mathbb{P}(Y \mid X_3, C_1 = 0)$, and similarly when conditioning on $\{X_1, X_3\}$.

	Context variables		System variables		
	C_1	C_2	X_1	X_2	X_3
source domains	0	0.1	0.1	0.2	0.5
	0	0.2	0.13	0.21	0.49
	0	0.4	0.23	0.21	0.51
	0	1.5	0.5	0.19	0.52
	0	1.7	0.6	0.18	0.51
target domains	1	0.2	0.2	**?**	0.92
	1	0.1	0.23	**?**	0.99
	1	1.6	0.53	**?**	0.95
	1	1.8	0.61	**?**	0.90
	1	1.7	0.55	**?**	0.97

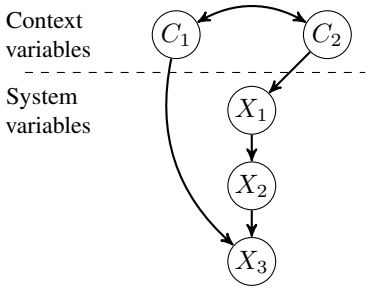


Figure 2: Example of a causal domain adaptation problem. The causal graph is depicted on the right, the corresponding data on the left. The task is to predict the missing values of $Y = X_2$ in the target domains ($C_1 = 1$), based on the observed data from the source domains and the target domains, without knowledge of the causal graph. See also Example 2.

An example is provided in Figure 2. In the next subsection, we will formalize our assumptions to make this task a well-posed problem.

2.2 Assumptions

Our first main assumption is that the data generating process (on both system and context variables) can be represented as a Structural Causal Model (SCM) (see e.g., (Pearl, 2009)):

$$\mathcal{M}: \begin{cases} C_i & = g_i(\boldsymbol{E}_{\text{PA}(i)\cap\mathcal{K}}), \qquad i \in \mathcal{I} \\ X_j & = f_j(\boldsymbol{X}_{\text{PA}(j)\cap\mathcal{J}}, \boldsymbol{C}_{\text{PA}(j)\cap\mathcal{I}}, \boldsymbol{E}_{\text{PA}(j)\cap\mathcal{K}}) \qquad j \in \mathcal{J} \\ p(\boldsymbol{E}) & = \prod_{k\in\mathcal{K}} p(E_k). \end{cases} \tag{1}$$

Here, we introduced exogenous latent independent "noise" variables $(E_k)_{k\in\mathcal{K}}$ that model any latent causes of the context and system variables. The parents of each variable are denoted by $\text{PA}(\cdot)$. There could be cyclic dependencies, for example due to feedback loops, but for simplicity of exposition we will discuss only the acyclic case here, noting that the extension to the cyclic case is straightforward. This SCM provides a causal model for the distributions of the various domains, and in particular, it induces a distribution $\mathbb{P}(\boldsymbol{V})$ on the context and system variables.

The SCM $\mathcal{M}$ can be represented graphically by its causal graph $\mathcal{G}(\mathcal{M})$, a graph with nodes $\mathcal{I} \cup \mathcal{J}$ (i.e., the labels of both system and context variables), directed edges $l_1 \to l_2$ for $l_1, l_2 \in \mathcal{I} \cup \mathcal{J}$ iff $l_1 \in \text{PA}(l_2)$, and bidirected edges $l_1 \leftrightarrow l_2$ for $l_1, l_2 \in \mathcal{I} \cup \mathcal{J}$ iff there exists a $k \in \text{PA}(l_1) \cap \text{PA}(l_2) \cap \mathcal{K}$. In the acyclic case, this causal graph is an Acyclic Directed Mixed Graph (ADMG), and $\mathcal{M}$ is also known as a Semi-Markov Causal Model (see e.g., (Pearl, 2009)). The directed edges represent direct causal relationships, and the bidirected edges represent hidden confounders (both relative to the set of variables in the ADMG). The (causal) *Markov assumption* holds (Richardson, 2003), i.e., any d-separation $\boldsymbol{A} \perp \boldsymbol{B} \mid \boldsymbol{S}\ [\mathcal{G}(\mathcal{M})]$ between sets of random variables $\boldsymbol{A}, \boldsymbol{B}, \boldsymbol{S} \subseteq \boldsymbol{V}$ in the ADMG $\mathcal{G}(\mathcal{M})$ implies a conditional independence $\boldsymbol{A} \perp\!\!\!\perp \boldsymbol{B} \mid \boldsymbol{S}\ [\mathbb{P}(\boldsymbol{V})]$ in the distribution $\mathbb{P}(\boldsymbol{V})$ induced by the SCM $\mathcal{M}$. A standard assumption in causal discovery is that the joint distribution $\mathbb{P}(\boldsymbol{V})$ is *faithful* with respect to the ADMG $\mathcal{G}(\mathcal{M})$ (i.e., that there are no other conditional independences in the joint distribution than those implied by d-separation).

We will make the following assumptions on the causal structure (where henceforth we will simply write $\mathcal{G}$ instead of $\mathcal{G}(\mathcal{M})$), which are discussed in detail by Mooij et al. (2018):

Assumption 1 (JCI Assumptions)**.** *Let $\mathcal{G}$ be a causal graph with variables $\boldsymbol{V}$ (consisting of system variables $\{X_j\}_{j\in\mathcal{J}}$ and context variables $\{C_i\}_{i\in\mathcal{I}}$).*

(i) No variable directly causes any context variable ("exogeneity")
($\forall j \in \mathcal{J}, \forall i \in \mathcal{I} : X_j \to C_i \notin \mathcal{G}, \quad \forall i, i' \in \mathcal{I} : C_{i'} \to C_i \notin \mathcal{G}$);
(ii) No system variable is confounded with a context variable ("randomization")
($\forall j \in \mathcal{J}, \forall i \in \mathcal{I} : X_j \leftrightarrow C_i \notin \mathcal{G}$);
(iii) Every pair of context variables is confounded ("genericity")
($\forall i, i' \in \mathcal{I} : C_i \leftrightarrow C_{i'} \in \mathcal{G}$).

In addition, in order to be able to address the causal domain adaptation task, we will assume:

Assumption 2. *Let $\mathcal{G}$ be a causal graph with variables $\boldsymbol{V}$ (consisting of system variables $\{X_j\}_{j\in\mathcal{J}}$ and context variables $\{C_i\}_{i\in\mathcal{I}}$), and $\mathbb{P}(\boldsymbol{V})$ be the corresponding distribution on $\boldsymbol{V}$. Let C_1 be the source/target domains indicator and $Y = X_j$ the target variable.*

(i) The distribution $\mathbb{P}(\boldsymbol{V})$ is Markov and faithful w.r.t. $\mathcal{G}$;

(ii) Any conditional independence involving Y in the source domains also holds in the target domains, i.e., if $\boldsymbol{A} \cup \boldsymbol{B} \cup \boldsymbol{S}$ contains Y but not C_1 then:[2]

$$\boldsymbol{A} \perp\!\!\!\perp \boldsymbol{B} \mid \boldsymbol{S}\ [C_1 = 0] \implies \boldsymbol{A} \perp\!\!\!\perp \boldsymbol{B} \mid \boldsymbol{S}\ [C_1 = 1];$$

(iii) C_1 has no direct *effect on Y w.r.t. $\boldsymbol{V}$, i.e., $C_1 \to Y \notin \mathcal{G}$.*

The Markov and faithfulness assumption is standard in constraint-based causal discovery on a single domain; we apply it here on the "meta-system" composed of system and context. Note that Assumption 2(ii) does not exclude the possibility of additional *independences* holding in the target domains, e.g., when C_1 models a perfect surgical intervention. Note further that Assumption 2(iii) gets weaker the more relevant system variables are observed.[3] In the next subsections, we will discuss how these assumptions enable us to address the domain adaptation task.

2.3 Separating Sets of Features

Our approach to addressing Task 1 is based on finding a *separating set* $\boldsymbol{A} \subseteq \boldsymbol{V} \setminus \{C_1, Y\}$ of (context and system) variables that satisfies $C_1 \perp Y \mid \boldsymbol{A}\ [\mathcal{G}]$. If such a separating set $\boldsymbol{A}$ can be found, then the distribution of Y conditional on $\boldsymbol{A}$ is *invariant* under transferring from the source domains to the target domains, i.e., $\mathbb{P}(Y \mid \boldsymbol{A}, C_1 = 0) = \mathbb{P}(Y \mid \boldsymbol{A}, C_1 = 1)$. As the former conditional distribution can be estimated from the source domains data, we directly obtain a prediction for the latter, which then enables us to predict the values of Y from the observed values of $\boldsymbol{A}$ in the target domains.[4]

We will now discuss the effect of the choice of $\boldsymbol{A}$ on the quality of the predictions. For simplicity of the exposition, we make use of the squared loss function and ignore finite-sample issues. When predicting Y from a subset of features $\boldsymbol{A} \subseteq \boldsymbol{V} \setminus \{Y, C_1\}$, the optimal predictor is defined as the function $\hat{Y}$ mapping the domain of $\boldsymbol{A}$ to the domain of Y that minimizes the *target domains risk* $\mathbb{E}\big((Y - \hat{Y})^2 \mid C_1 = 1\big)$, and is given by the conditional expectation (regression function) $\hat{Y}^1_{\boldsymbol{A}}(\boldsymbol{a}) := \mathbb{E}(Y \mid \boldsymbol{A} = \boldsymbol{a}, C_1 = 1)$. Since Y is not observed in the target domains, we cannot directly estimate this regression function from the data.

One approach that is often used in practice is to ignore the difference in distribution between source and target domains, and use instead the predictor $\hat{Y}^0_{\boldsymbol{A}}(\boldsymbol{a}) := \mathbb{E}(Y \mid \boldsymbol{A} = \boldsymbol{a}, C_1 = 0)$, which minimizes the *source domains risk* $\mathbb{E}\big((Y - \hat{Y})^2 \mid C_1 = 0\big)$. This approximation introduces a bias $\hat{Y}^1_{\boldsymbol{A}} - \hat{Y}^0_{\boldsymbol{A}}$ that we will refer to as the *transfer bias* (when predicting Y from $\boldsymbol{A}$). When ignoring that source domains and target domains have different distributions, any standard machine learning method can be used to predict Y from $\boldsymbol{A}$. As the transfer bias can become arbitrarily large (as we have seen in Example 1), the prediction accuracy by this solution strategy may be arbitrarily bad (even in the infinite-sample limit).

Instead, we propose to only predict Y from $\boldsymbol{A}$ when the set $\boldsymbol{A}$ of features satisfies the following *separating set* property:

$$C_1 \perp Y \mid \boldsymbol{A}\ [\mathcal{G}], \tag{2}$$

i.e., it d-separates C_1 from Y in $\mathcal{G}$. By the Markov assumption, this implies $C_1 \perp\!\!\!\perp Y \mid \boldsymbol{A}\ [\mathbb{P}(\boldsymbol{V})]$. In other words, for separating sets, the distribution of Y conditional on $\boldsymbol{A}$ is *invariant* under transferring from the source domains to the target domains, i.e., $\mathbb{P}(Y \mid \boldsymbol{A}, C_1 = 0) = \mathbb{P}(Y \mid \boldsymbol{A}, C_1 = 1)$. By

[2] Here, with $\boldsymbol{A} \perp\!\!\!\perp \boldsymbol{B} \mid \boldsymbol{S}\ [C_1 = 0]$ we mean $\boldsymbol{A} \perp\!\!\!\perp \boldsymbol{B} \mid \boldsymbol{S}\ [\mathbb{P}(\boldsymbol{V} \mid C_1 = 0)]$, i.e., the conditional independence of $\boldsymbol{A}$ from $\boldsymbol{B}$ given $\boldsymbol{S}$ in the mixture of the source domains $\mathbb{P}(\boldsymbol{V} \mid C_1 = 0)$, and similarly for the target domains.

[3] This assumption can be weakened further: in some circumstances one can infer from the data and the other assumptions that C_1 cannot have a direct effect on Y. For example: if there exists a descendant $D \in \mathrm{DE}(Y)$, and if there exists a set $\boldsymbol{S} \subseteq \boldsymbol{V} \setminus (\{C_1, Y\} \cup \mathrm{DE}(Y))$, such that $C_1 \perp\!\!\!\perp D \mid \boldsymbol{S}$, then C_1 is not a direct cause of Y w.r.t. $\boldsymbol{V}$. For some proposals on alternative assumptions that can be made when this assumption is violated, see e.g., (Schölkopf et al., 2012; Zhang et al., 2013, 2015; Gong et al., 2016).

[4] This trivial observation is not novel; see e.g. (Ch. 7, p. 164, Spirtes et al., 2000). It also follows as a special case of (Theorem 2, Pearl and Bareinboim, 2011). The main novelty of this work is the proposed strategy to identify such separating sets.

virtue of this invariance, regression functions are identical for the source domains and target domains, i.e., $\hat{Y}^0_{\boldsymbol{A}} = \hat{Y}^1_{\boldsymbol{A}}$, and hence also the source domains and target domains risks are identical when using the predictor $\hat{Y}^0_{\boldsymbol{A}}$:

$$C_1 \perp Y \,|\, \boldsymbol{A}\ [\mathcal{G}] \implies \mathbb{E}\big((Y - \hat{Y}^0_{\boldsymbol{A}})^2 \,|\, C_1 = 1\big) = \mathbb{E}\big((Y - \hat{Y}^0_{\boldsymbol{A}})^2 \,|\, C_1 = 0\big). \tag{3}$$

The r.h.s. can be estimated from the source domains data, and the l.h.s. equals the generalization error to the target domains when using the predictor $\hat{Y}^0_{\boldsymbol{A}}$ trained on the source domains (which equals the predictor $\hat{Y}^1_{\boldsymbol{A}}$ that one could obtain if all target domains data, including the values of Y, were observed).[5] Although this approach leads to zero transfer bias, it introduces another bias: by using only a subset of the features $\boldsymbol{A}$, rather than *all available* features $\boldsymbol{V} \setminus \{C_1, Y\}$, we may miss relevant information to predict Y. We refer to this bias as the *incomplete information bias*, $\hat{Y}^1_{\boldsymbol{V}\setminus\{Y,C_1\}} - \hat{Y}^1_{\boldsymbol{A}}$.

The total bias when using $\hat{Y}^0_{\boldsymbol{A}}$ to predict Y is the sum of the transfer bias and the incomplete information bias:

$$\underbrace{\hat{Y}^1_{\boldsymbol{V}\setminus\{Y,C_1\}} - \hat{Y}^0_{\boldsymbol{A}}}_{\text{total bias}} = \underbrace{(\hat{Y}^1_{\boldsymbol{A}} - \hat{Y}^0_{\boldsymbol{A}})}_{\text{transfer bias}} + \underbrace{(\hat{Y}^1_{\boldsymbol{V}\setminus\{Y,C_1\}} - \hat{Y}^1_{\boldsymbol{A}})}_{\text{incomplete information bias}}.$$

For some problems, one may be better off to simply ignore the transfer bias and minimize the incomplete information bias, while for other problems, it is crucial to take the transfer into account to obtain small generalization errors. In that situation, we could use any subset $\boldsymbol{A}$ for prediction that satisfies the separating set property (2), implying zero transfer bias; obviously, the best predictions are then obtained by selecting a separating subset that also minimizes the source domains risk (i.e., minimizes the incomplete information bias). We conclude that this strategy of selecting a subset $\boldsymbol{A}$ to predict Y may yield an asymptotic guarantee on the prediction error by (3), whereas simply ignoring the shift in distribution may lead to unbounded prediction error, since the transfer bias could be arbitrarily large in the worst case scenario.

2.4 Identifiability of Separating Feature Sets

For the strategy of selecting the best separating sets of features as discussed in Section 2.3, we need to find one or more sets $\boldsymbol{A} \subseteq \boldsymbol{V} \setminus \{C_1, Y\}$ that satisfy (2). Of course, the problem is that we cannot directly test this in the data, because the values of Y are missing for $C_1 = 1$. Note that also Assumption 2(ii) cannot be directly used here, because it only applies when C_1 is *not* in $\boldsymbol{A} \cup \boldsymbol{B}$. When the causal graph $\mathcal{G}$ is known, it is easy to verify whether (2) holds directly using d-separation. Here we address the more challenging setting in which the causal graph and the targets of the interventions are (partially) unknown.[6] Conceptually, what one could do is first estimate the causal graph by using a causal discovery algorithm, and then read off separating sets from the estimated causal graph. However, it is not necessary to estimate the complete causal graph: we only need to know enough about it to verify or falsify whether a given set of features separates C_1 from Y.

Example 2. *Assume that Assumptions 1 and 2 hold for two intervention variables C_1, C_2 and three system variables X_1, X_2, X_3 with $Y = X_2$. If the following conditional (in)dependences all hold in the source domains:*

$$C_2 \perp\!\!\!\perp X_2 \,|\, X_1\ [C_1 = 0], \qquad C_2 \not\perp\!\!\!\perp X_2 \,|\, \emptyset\ [C_1 = 0], \qquad C_2 \perp\!\!\!\perp X_3 \,|\, X_2\ [C_1 = 0],$$

then $C_1 \perp X_2 \,|\, X_1\ [\mathcal{G}]$, i.e., $\{X_1\}$ is a separating set for C_1 and Y. One possible causal graph leading to those (in)dependences is provided in Figure 2 (others are shown in Figure 4c). For that ADMG, and given enough data, feature selection applied to the source domains data will generically select $\{X_1, X_3\}$ as the optimal set of features for predicting Y, which can lead to an arbitrarily large prediction error. On the other hand, using the separating set $\{X_1\}$ to predict Y leads to zero transfer bias, and therefore provides a guarantee on the target domains risk (i.e., it provides an upper bound on the optimal target domains risk, which can be estimated from the source domains data).

[5] Note that this equation only holds asymptotically; for finite samples, in addition to the transfer from source domains to target domains, we have to deal with the generalization from empirical to population distributions and from the covariate shift if $\mathbb{P}(\boldsymbol{A} \,|\, C_1 = 1) \neq \mathbb{P}(\boldsymbol{A} \,|\, C_1 = 0)$ (see e.g. Mansour et al., 2009).

[6] Another option, proposed by Rojas-Carulla et al. (2016), would be to *assume* that if $p(Y \,|\, \boldsymbol{A})$ is invariant across all source domains (i.e., $p(Y \,|\, \boldsymbol{A}, C_1 = 0, C_{\setminus 1} = c) = p(Y \,|\, \boldsymbol{A}, C_1 = 0)$ for all c), then the same holds across all source *and* target domains (i.e., $p(Y \,|\, \boldsymbol{A}, C_1 = 1) = p(Y \,|\, \boldsymbol{A}, C_1 = 0)$). This is a different assumption than the ones we are making here, and Example 2 shows a simple case in which it would be violated.

Rather than characterising by hand all possible situations in which a separating set can be identified (like in Example 2), in this work we delegate the causal reasoning to an automatic theorem prover. Intuitively, the idea is to provide the automatic theorem prover with the conditional (in)dependences that hold in the data, in combination with an encoding of Assumptions 1 and 2 into logical rules, and ask the theorem prover whether it can prove that $C_1 \perp Y \mid \boldsymbol{A}$ holds for a candidate set $\boldsymbol{A}$ from the assumptions and provided conditional (in)dependences. There are three possibilities: either it can prove the query (and then we can proceed to predict Y from $\boldsymbol{A}$ and get an estimate of the target domains risk), or it can disprove the query (and then we know $\boldsymbol{A}$ will generically give predictions that suffer from an arbitrarily large transfer bias), or it can do neither (in which case hopefully another subset $\boldsymbol{A}$ can be found that does provably satisfy (2)).

2.5 Implementation Details

A simple (brute-force) implementation of our proposed approach is the following. By using a standard feature selection method, produce a ranked list of subsets $\boldsymbol{A} \subseteq \boldsymbol{V} \setminus \{Y, C_1\}$, ordered ascendingly with respect to the empirical source domains risks. Going through this list of subsets (starting with the one with the smallest empirical source domains risk), test whether the separating set property can be inferred from the data by querying the automated theorem prover. If (2) can be shown to hold, use that subset $\boldsymbol{A}$ for prediction of Y and stop; if not, continue with the next candidate subset $\boldsymbol{A}$ in the list. If no subset satisfies (2), abstain from making a prediction.[7]

An important consequence of Assumption 2(ii) is that it enables us to transfer conditional independence involving the target variable from the source domains to the target domains (proof provided in the Supplementary Material):

Lemma 1. *Under Assumption 2,*

$$\boldsymbol{A} \perp\!\!\!\perp \boldsymbol{B} \mid \boldsymbol{S}\ [C_1 = 0] \iff \boldsymbol{A} \perp\!\!\!\perp \boldsymbol{B} \mid \boldsymbol{S} \cup \{C_1\} \iff \boldsymbol{A} \perp \boldsymbol{B} \mid \boldsymbol{S} \cup \{C_1\}\ [\mathcal{G}]$$

for subsets $\boldsymbol{A}, \boldsymbol{B}, \boldsymbol{S} \subseteq \boldsymbol{V}$ such that their union contains Y but not C_1.

To test the separating set condition (2), we use the approach proposed by Hyttinen et al. (2014), where we simply add the JCI assumptions (Assumption 1) as constraints on the optimization problem, in addition to the domain-adaptation specific assumption that $C_1 \to Y \notin \mathcal{G}$ (Assumption 2(iii)). As inputs we use all directly testable conditional independence test p-values $p_{\boldsymbol{A} \perp\!\!\!\perp \boldsymbol{B} \mid \boldsymbol{S}}$ in the pooled data (when $Y \notin \boldsymbol{A} \cup \boldsymbol{B} \cup \boldsymbol{S}$) and all those resulting from Lemma 1 from the source domains data only (if $Y \in \boldsymbol{A} \cup \boldsymbol{B} \cup \boldsymbol{S}$). If background knowledge on intervention targets or the causal graph is available, it can easily be added as well. We use the method proposed by Magliacane et al. (2016) to query for the confidence of whether some statement (e.g., $Y \perp\!\!\!\perp C_1 \mid \boldsymbol{A}$) is true or false. The results of Magliacane et al. (2016) show that this approach is sound under oracle inputs, and asymptotically consistent whenever the statistical conditional independence tests used are asymptotically consistent. In other words, in this way the probability of wrongly deciding whether a subset $\boldsymbol{A}$ is a separating set converges to zero as the sample size increases. We chose this approach because it is simple to implement on top of existing open source code.[8] Note that the computational cost quickly increases with the number of variables, limiting the number of variables that can be considered simultaneously.

One remaining issue is how to predict Y when an optimal separating set $\boldsymbol{A}$ has been found. As the distribution of $\boldsymbol{A}$ may shift when transferring from source domains to target domains, this means that there is a *covariate shift* to be taken into account when predicting Y. Any method (e.g., least-squares regression) could in principle be used to predict Y from a given set of covariates, but it is advisable to use a prediction method that works well under covariate shift, e.g., (Sugiyama et al., 2008).

[7] Abstaining from predictions can be advantageous when trading off recall and precision. If a prediction *has* to be made, we can fall back on some other method or simply accept the risk that the transfer bias may be large.

[8] We build on the source code provided by Magliacane et al. (2016) which in turn extends the source code provided by Hyttinen et al. (2014). The full source code of our implementation and the experiments will be made available under an open source license on publication.

3 Evaluation

We perform an evaluation on both synthetic data and a real-world dataset based on a causal inference challenge.[9] The latter dataset consists of hematology-related measurements from the International Mouse Phenotyping Consortium (IMPC), which collects measurements of phenotypes of mice with different single-gene knockouts.

In both evaluations we compare a standard feature selection method (which uses Random Forests) with our method that builds on top of it and selects from its output the best separating set. First, we score all possible subsets of features by their out-of-bag score using the implementation of Random Forest Regressor from `scikit-learn` (Pedregosa et al., 2011) with default parameters. For the baseline we then select the best performing subset and predict Y. Instead, for our proposed method we try to find a subset of features $\boldsymbol{A}$ that is also a separating set, starting from the subsets with the best scores. To test whether $\boldsymbol{A}$ is a separating set, we use the method described in Section 2.5, using the ASP solver `clingo 4.5.4` (Gebser et al., 2014). We provide as inputs the independence test results from a partial correlation test with significance level $\alpha = 0.05$ and combine it with the weighting scheme from (Magliacane et al., 2016). We then use the first subset $\boldsymbol{A}$ in the ranked list of predictive sets of features found by the Random Forest method for which the confidence that $C_1 \perp Y \mid \boldsymbol{A}$ holds is positive. If there is no set $\boldsymbol{A}$ that satisfies this criterium, then we abstain from making a prediction.

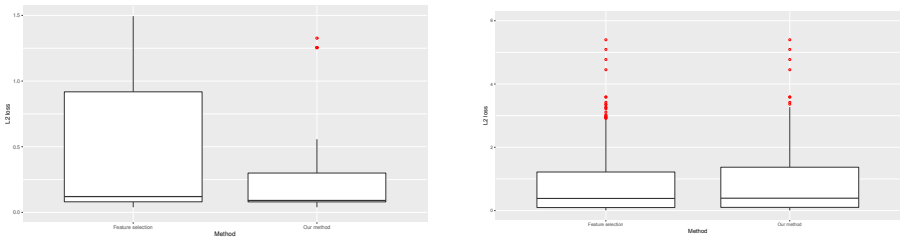
For the synthetic data, we generate randomly 200 linear acyclic models with latent variables and Gaussian noise, each with three system variables, and sample N data points each for the observational and two experimental domains, where we simulate soft interventions on randomly selected targets, focusing on small, medium and large perturbations. We randomly select which intervention variable will be C_1 and which system variable will be Y. We disallow direct effects of C_1 on Y, and enforce that no intervention can directly affect all variables simultaneously. Figure 3a shows a boxplot of the L_2 loss of the predicted Y values with respect to the true values for both the baseline and our method, considering the 120 cases out of 200 in which our method does produce an answer. In particular, Figure 3a considers the case of $N = 1000$ samples per regime and interventions that all produce a large perturbation. In the Supplementary Material we show that results improve with more samples, both for the baseline, but even more so for our method, since the quality of the conditional independence tests improves. We also show that, according to expectations, if the target distribution is very similar to the source distributions, i.e., the transfer bias is small, our method does not provide any benefit and seems to perform worse than the baseline. Conversely, the larger the intervention effect, the bigger the advantage of using our method.

For the real-world dataset, we select a subset of the variables considered in the CRM Causal Inference Challenge. Specifically, for simplicity we focus on 16 phenotypes that are not deterministically related to each other. The dataset contains measurements for 441 "wild type" mice and for about 10 "mutant" mice for each of 13 different single gene knockouts. We then generate 1000 datasets by randomly selecting subsets of 3 variables and 2 gene knockouts interventions, and always include also "wild type" mice. For each dataset we randomly choose Y and C_1, and remove the values of Y for $C_1 = 1$. Figure 3b shows a boxplot of the L_2 loss of the predicted Y values with respect to the real values for the baseline and our method. Given the small size of the datasets, this is a very challenging problem. In this case, our method does not perform better than the baseline, and abstains from making a prediction for 170 cases out of 1000.

4 Discussion and Conclusion

We have defined a general class of causal domain adaptation problems and proposed a method that can identify sets of features that lead to transferable predictions. Our assumptions are very general and do not require the causal graph or the intervention targets to be known. The method gives promising results on simulated and real-world data. More work remains to be done on the implementation side, for example, scaling up to more variables. We hope that this work will also inspire further research on the interplay between bias, variance and causality from a statistical learning theory perspective.

[9] Part of the CRM workshop on Statistical Causal Inference and Applications to Genetics, Montreal, Canada (2016). See also `http://www.crm.umontreal.ca/2016/Genetics16/competition_e.php`



(a) Synthetic data with $N = 1000$ samples and a large perturbation

(b) Real-world data

Figure 3: Evaluation results (see main text and Supplementary Material for details).

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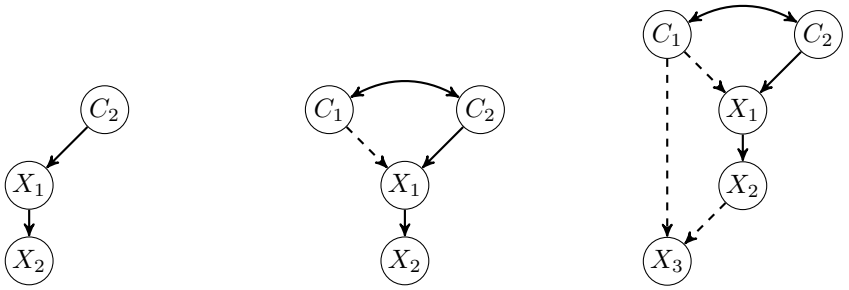


(a) Marginal ADMG $\mathcal{G}'$. (b) Marginal ADMG $\mathcal{G}''$. (c) ADMG $\mathcal{G}$.

Figure 4: ADMGs for proof of Example 2. Each dashed edge can either be present or absent.

Supplementary material

Proofs

Proof of Lemma 1. First of all, $\boldsymbol{A} \not\perp\!\!\!\perp \boldsymbol{B} \mid \boldsymbol{S}\ [C_1 = 0]$ implies (by definition) $\boldsymbol{A} \not\perp\!\!\!\perp \boldsymbol{B} \mid \boldsymbol{S} \cup \{C_1\}$. Second, $\boldsymbol{A} \perp\!\!\!\perp \boldsymbol{B} \mid \boldsymbol{S}\ [C_1 = 0]$ implies (by assumption) $\boldsymbol{A} \perp\!\!\!\perp \boldsymbol{B} \mid \boldsymbol{S}\ [C_1 = 1]$, and taken together, we get $\boldsymbol{A} \perp\!\!\!\perp \boldsymbol{B} \mid \boldsymbol{S} \cup \{C_1\}$. By the Markov and faithfulness assumption (Assumption 2(i)), this holds iff $\boldsymbol{A} \perp \boldsymbol{B} \mid \boldsymbol{S} \cup \{C_1\}\ [\mathcal{G}]$. □

Proof of Example 2. In the JCI setting, we assume that in the full ADMG $\mathcal{G}$ over variables $\{C_1, C_2, X_1, X_2, X_3\}$, C_1 and C_2 are confounded and not caused by system variables X_1, X_2, X_3. Furthermore, no pair of system variable and intervention variable is confounded.

In the context $[C_1 = 0]$, if the conditional independences $C_2 \perp\!\!\!\perp X_2 \mid X_1\ [C_1 = 0]$ and $C_2 \not\perp\!\!\!\perp X_2 \mid \emptyset\ [C_1 = 0]$ hold, then we can also derive that $C_2 \not\perp\!\!\!\perp X_1 \mid \emptyset\ \ [C_1 = 0]$, for example using Rule (9) from Magliacane et al. (2016). Moreover, we know that C_2 is not caused by X_1 and X_2, or in other words $X_1 \not\dashrightarrow C_2$ and $X_2 \not\dashrightarrow C_2$. Thus we conclude that (C_2, X_1, X_2) is an LCD triple (Cooper, 1997) in the context $C_1 = 0$. Since in addition, in this case C_2 and X_1 are unconfounded, the marginal ADMG $\mathcal{G}'$ on $\{C_2, X_1, X_2\}$ (in the context $C_1 = 0$, and hence by Lemma 1 in all contexts) must be given by Figure 4a.

Therefore, the extended marginal ADMG $\mathcal{G}''$ on variables $\{C_1, C_2, X_1, X_2\}$ must also have a directed path from C_2 to X_1 and from X_1 to X_2. C_1 cannot be on these paths, as none of the variables causes C_1, and therefore $\mathcal{G}''$ also contains the directed edges $C_2 \to X_1$ and $X_1 \to X_2$. Moreover, $\mathcal{G}''$ cannot contain any edge between C_2 and X_2, nor a bidirected edge between X_1 and X_2, because that would violate the conditional independence. By construction, in the JCI setting there is a bidirected edge between C_1 and C_2, and that is the only bidirected edge connecting to C_1 or C_2. As we assumed there is no direct effect of C_1 on target X_2, there is no edge between C_1 and X_2 in $\mathcal{G}''$. There is also no directed edge $X_1 \to C_1$ in $\mathcal{G}''$, as the JCI assumption implies none of the other variables causes C_1. Therefore, the marginal ADMG $\mathcal{G}''$ is given by Figure 4b, either with the directed edge $C_1 \to X_1$ present, or without that edge.

If it additionally holds that $C_2 \perp\!\!\!\perp X_3 \mid X_2\ [C_1 = 0]$, we have two possibilities:

1. if $C_2 \perp\!\!\!\perp X_3 \mid \emptyset\ [C_1 = 0]$ holds, then X_3 is not caused by C_2. This means it cannot be on any directed path from C_2 to X_1, from X_1 to X_2, or be a descendant of X_2. Therefore the full ADMG $\mathcal{G}$ also necessarily contains the directed edges $C_2 \to X_1$ and $X_1 \to X_2$.
2. if $C_2 \not\perp\!\!\!\perp X_3 \mid \emptyset\ [C_1 = 0]$ holds, then in conjunction with $C_2 \perp\!\!\!\perp X_3 \mid X_2\ [C_1 = 0]$ we can derive $X_2 \dashrightarrow X_3$, for example using Rule (5) from (Magliacane et al., 2016). This means X_3 must be a descendant of X_2 in the full ADMG $\mathcal{G}$, which implies it cannot be on the directed path from C_2 to X_1, or on the one from X_1 to X_2. Therefore the full ADMG $\mathcal{G}$ also necessarily contains the directed edges $C_2 \to X_1$ and $X_1 \to X_2$.

Because of the independence statements and JCI assumptions, there cannot be a bidirected edge between X_3 and X_1, X_2, C_1 or C_2. Similarly, there cannot be directed edges from X_3 to one of those nodes. The edges $X_1 \to X_3$ and $C_2 \to X_3$ must also be absent.

In both cases, there can be a directed edge from C_1 to X_3. Therefore, the full ADMG $\mathcal{G}$ is given by Figure 4c. In all cases we see that $C_1 \perp X_2 \mid X_1\ [\mathcal{G}]$, and we conclude that $\{X_1\}$ is a valid separating set.

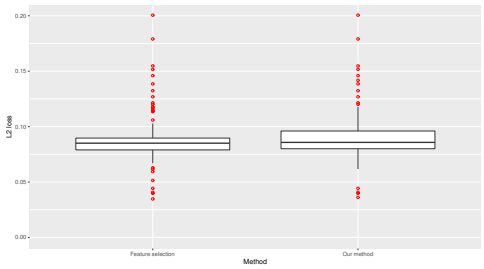
If the ADMG is as in Figure 2, then a standard feature selection method would asymptotically prefer the subset $\{X_1, X_3\}$ to predict X_2 over the subset $\{X_1\}$ (note that the Markov blanket of X_2 in context $[C_1 = 0]$ is $\{X_1, X_3\}$). As a result, any prediction method trained on all available features using source domain data (i.e., in context $[C_1 = 0]$) may incur a possibly unbounded prediction error when used to predict X_2 in the target domain $[C_1 = 1]$ (for example, if X_3 is an almost deterministic copy of X_2 if $C_1 = 0$, but has a drastically different distribution if $C_1 = 1$). □

Additional results on synthetic data

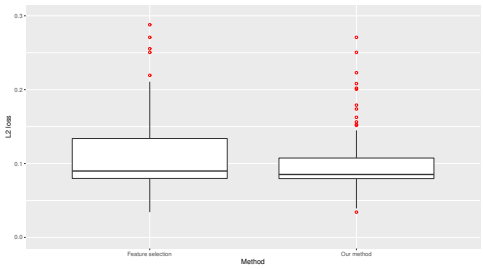
We provide some additional results on the synthetic data. We generate randomly 200 linear acyclic models with a small random number of latent variables and Gaussian noise, each with three system variables. Similarly to the evaluation in the main paper, we sample *N* data points each for the observational and two experimental domains, and simulate soft interventions on randomly selected targets. These interventions have linear coefficients sampled from $\mathcal{N}(0.2, 0.8)$, for which we randomly select the sign. In order to scale the effect of these interventions we multiply the coefficients for all interventions by the parameter *IFactor*, varying it from 0.1 to 100. Moreover, we randomly select C_1 and Y from intervention and system variables respectively. We disallow direct effects of C_1 on Y, and enforce that no intervention can directly affect all variables simultaneously.

As expected, our method performs well when the target distribution is significantly different than the source distributions. Figure 5 shows different settings with different scales of intervention effects. In Figure 5a the intervention effects are all scaled by 0.1, resulting in very similar distributions in all domains. In this case, using our method does not offer any advantage with respect to the baseline and it actually performs worse. In the other cases, using our method starts to pay off in terms of prediction accuracy, and the difference increases with the scale of the interventions, as seen in Figure 5d.

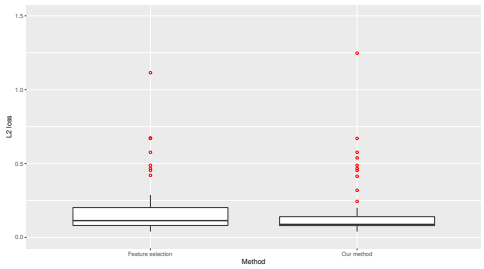
In Figure 6, we vary the number of samples N for each regime. The results improve with more samples, especially for our method, since the quality of the conditional independence test improves, but also for the baseline. In particular, as shown in Figure 6a, the accuracy is low for N=100 samples, but it improves substantially with N=1000 samples (Figure 5b).



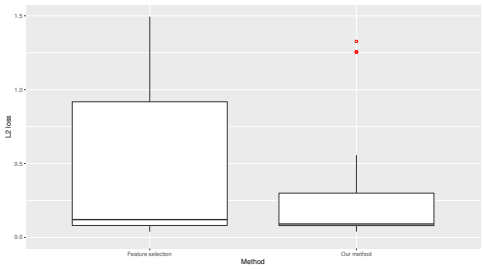
(a) Synthetic data with a very small perturbation (IFactor=0.1) and N=1000 samples (zoomed version)



(b) Synthetic data with a small perturbation (IFactor=1) and N=1000 samples (zoomed version)

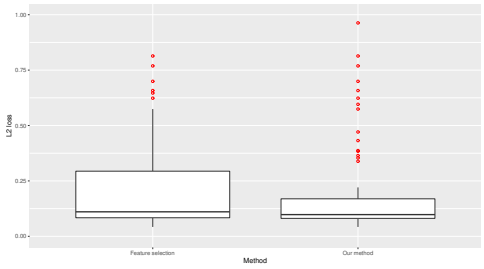


(c) Synthetic data with a medium perturbation (IFactor=10) and N=1000 samples

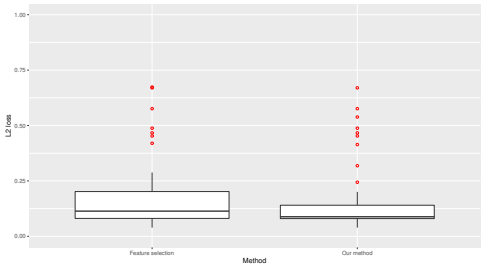


(d) Synthetic data with a large perturbation (IFactor=100) and N=1000 samples

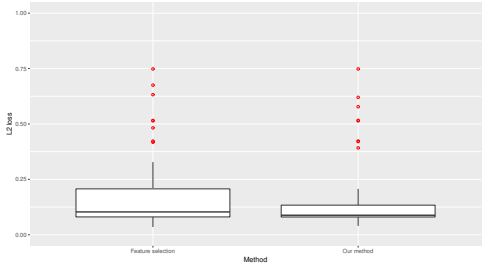
Figure 5: Additional results when varying the causal effect of all interventions (IFactor).



(a) Synthetic data with N=100 samples per regime and a medium perturbation (IFactor=10)



(b) Synthetic data with N=1000 samples per regime and a medium perturbation (IFactor=10)



(c) Synthetic data with N=5000 samples per regime and a medium perturbation (IFactor=10)

Figure 6: Additional results when varying the sample size per regime (N).