A Bayesian semiparametric framework for causal inference in high-dimensional data

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Abstract

We introduce a Bayesian framework for estimating causal effects of binary and continuous treatments in high-dimensional data. The proposed framework extends to high-dimensional settings many of the existing semiparametric estimators introduced in the causal inference literature. Our approach has the following features: it 1) considers semiparametric estimators that reduce model dependence; 2) introduces flexible Bayesian priors for dimension reduction of the covariate space that accommodates non linearity; 3) provides posterior distributions of any causal estimator that can broadly be defined as a function of the treatment and outcome model (e.g. standard doubly robust estimator or the inverse probability weighted estimator); 4) provides posterior credible intervals with improved finite sample coverage compared to frequentist measures of uncertainty which rely on asymptotic properties. We show that the posterior contraction rate of the proposed doubly robust estimator is the product of the posterior contraction rates of the treatment and outcome models, allowing for faster posterior contraction. Via simulation we illustrate the ability of the proposed estimators to flexibly estimate causal effects in high-dimensions, and show that it performs well relative to existing approaches. Finally, we apply our proposed procedure to estimate the effect of continuous environmental exposures.

1 Introduction

In observational studies, the goal is often to estimate the causal effect of a treatment (T) on an outcome (Y), adjusted for a set of pre-treatment covariates ($\boldsymbol{X}$) to account for imbalances between covariates in the treated and control groups. In this paper we introduce a new class of causal inference estimators that can perform well when the number of covariates (p) is large relative to the sample size (n), and the conditional relationships between the covariates and the treatment/outcome can be nonlinear.

There has been a rapid growth in the interest of estimating causal effects when the dimension of the covariate space grows with the sample size (Belloni *et al.* , 2014; Ertefaie *et al.* , 2015; Farrell, 2015; Athey *et al.* , 2016; Chernozhukov *et al.* , 2016; Antonelli *et al.* , 2016; Shortreed & Ertefaie, 2017; Antonelli *et al.* , 2017). In high-dimensions, some form of dimension reduction or variable selection is required, and most approaches utilize both the treatment and outcome to reduce the dimension of the parameter space in a way that eliminates confounding bias. Belloni *et al.* (2014) and Farrell (2015) estimate treatment and outcome models using the lasso (Tibshirani, 1996), which identifies covariates to be used in a post-selection regression or doubly robust estimator, respectively. Antonelli *et al.* (2016) also utilized lasso models to estimate a propensity and prognostic score, and showed that matching on both quantities leads to doubly robust estimates of treatment effects. Athey *et al.* (2016) use the lasso or elastic net (Zou & Hastie, 2005) in a first step outcome regression model, then re-weight the residuals using the balancing weights of Zubizarreta (2015) to adjust for any remaining imbalances among the covariates. (Ertefaie *et al.* , 2018) derived a new penalization estimator that incorporates information from both the treatment and outcome to identify confounders. Shortreed & Ertefaie (2017) used the adaptive lasso to estimate propensity score models that reduce shrinkage of coefficients for covariates also associated with the outcome. Antonelli *et al.* (2017) used similar ideas but used information from the treatment model to reduce shrinkage of coefficients in an outcome model. Finally, Hahn *et al.* (2016) utilized horseshoe priors on a re-parameterized outcome and treatment model to tailor shrinkage of coefficients towards estimating treatment effects. All of these approaches rely either on linearity of treatment/outcome models, asymptotic confidence intervals for inference, or both. We will show that in small sample sizes, inference based on these confidence intervals will be anti-conservative, particularly as the number of covariates grows.

Nonlinear models have been adopted in the causal inference framework to flexibly estimate treatment effects. A large literature exists based on targeted maximum likelihood (TMLE), which allows for the use of super learners or flexible machine learning techniques to estimate functions of the covariates necessary to estimate treatment effects (Van Der Laan & Rubin, 2006; Van der Laan & Rose, 2011). Recently, flexible Bayesian methods such as Bayesian additive regression trees (BART) (Chipman *et al.* , 2010) have been utilized to flexibly model potential outcomes to estimate treatment effects (Hill, 2011). BART has also been utilized to flexibly estimate heterogeneous treatment effects (Hahn *et al.* , 2017). While all of these approaches do not rely on modeling assumptions of linearity in either the treatment or outcome model, they

do not immediately extend to the high-dimensional regime where $p \geq n$. Methods that allow to estimate causal effects in the context of high-dimensional data and that at the same time do not rely on strong modeling assumptions are lacking.

Doubly robust estimation was first introduced in the Bayesian framework in Saarela *et al.* (2016); Graham *et al.* (2016). In this paper we will build upon these ideas and extend them in several directions. We introduce a fully Bayesian framework for estimating causal effects of binary and continuous treatments in the context of high-dimensional data. We propose doubly robust estimates of treatment effects that incorporate nonparametric Bayesian methods to relax modeling assumptions, coupled with sparsity inducing priors to reduce the dimension of the covariate space in high-dimensional scenarios. We will show that - by calculating the posterior distribution of both the treatment and outcome models - we can obtain posterior distributions of a large class of causal estimators such as the standard doubly robust estimator (Bang & Robins, 2005). We will estimate the finite sample posterior variance of the causal effect estimator by combining posterior samples with an efficient resampling procedure that will only slightly increase computation time, while giving credible intervals that have good frequentist properties in finite samples. Finally, we calculate posterior contraction rates of our estimators, and show that the posterior contraction rate of our doubly robust estimator is the product of the posterior contraction rates for the treatment and outcome model. This is advantageous as it allows us to use flexible or high-dimensional models and still maintain fast posterior contraction rates.

2 Flexible doubly robust estimator

Throughout, we will assume that the observed data is $(Y_i, T_i, \boldsymbol{X}_i)$ for $i = 1 \dots n$. Y and T are the outcome and treatment of interest, respectively, while $\boldsymbol{X}_i$ is a $p-$dimensional vector of potential confounders. We will be working under the high-dimensional situation where the number of covariates exceeds the sample size, and is potentially growing with the sample size. Our framework will be quite general in the sense that the ideas presented will be applicable to a wide variety of causal estimands. One such estimand is the average treatment effect (ATE), which we will focus on for clarity, and is defined for a binary treatment as $\Delta(1,0) = E(Y(1) - Y(0))$. Here, $Y(t)$ is the potential outcome one would have received under treatment $T = t$.

For binary treatments, identification of the average treatment effect relies on the stable unit treatment value assumption (SUTVA) (Little & Rubin, 2000), strong ignorability, and positivity. SUTVA implies that the treatment received by one observation or unit does not affect the outcomes of other units and the potential outcomes are well-defined in the sense that there are not different versions of the treatment that lead to different potential outcomes. Strong ignorability and positivity can be defined as:

Strong Ignorability: $Y(1), Y(0) \perp\!\!\!\perp T | \boldsymbol{X}$

Positivity: $0 < \delta < P(T = 1|\boldsymbol{X}) < 1 - \delta < 1$ for all $\boldsymbol{X}$ and some constant δ

where $P(T = 1|\boldsymbol{X})$ denotes the propensity score (Rosenbaum & Rubin, 1983). There exists analagous assumptions when estimating the effect of a continuous treatment on an outcome, though we refer readers to previous literature on the topic for more details (Gill & Robins, 2001; Kennedy *et al.* , 2017).

2.1 Modeling framework

Throughout the manuscript we will posit a model for both the treatment and outcome as many causal inference estimators can be defined as a function of one or the both of them. While we will always be utilizing both models, if an estimator relies on only one of the two models, then the other model can be dropped from estimation as it would not impact the results, since we will be factorizing the likelihood via $P(Y, T|\boldsymbol{X}) = P(Y|T, \boldsymbol{X})P(T|\boldsymbol{X})$. We introduce:

$$h_y^{-1}(E(Y_i|T_i, \boldsymbol{X}_i)) = \beta_0 + f_t(T_i) + \sum_{j=1}^{p} f_j(X_{ji}) \tag{1}$$

$$h_t^{-1}(E(T_i|\boldsymbol{X}_i)) = \alpha_0 + \sum_{j=1}^{p} g_j(X_{ji}). \tag{2}$$

For now we have left the functional form of the relationships between the covariates and the treatment or outcome to be unknown, and we will detail how we estimate them in the following sections. We use this formulation, because many well-known estimators can be obtained from the fitted values of these two models. For instance, a doubly robust estimator can be constructed as

$$\Delta(1,0) = \frac{1}{n}\left[\sum_{i=1}^{n} \frac{T_i Y_i}{e_i} - \frac{T_i - e_i}{e_i} m_{1i}\right] - \frac{1}{n}\left[\sum_{i=1}^{n} \frac{(1-T_i)Y_i}{1-e_i} + \frac{T_i - e_i}{1-e_i} m_{0i}\right], \tag{3}$$

where $e_i = P(T_i = 1|\boldsymbol{X_i})$, $m_{0i} = E(Y_i|T_i = 0, \boldsymbol{X_i})$ and $m_{1i} = E(Y_i|T_i = 1, \boldsymbol{X_i})$. If we have posterior samples of the parameters for the outcome and treatment models then we automatically have posterior samples of the doubly robust estimator as well. One might expect that by having posterior samples of the above estimator, we can automatically create credible regions and perform inference, but these posterior samples would only account for the uncertainty in the parameters, and not any additional uncertainty from the treatment and outcome that is not captured by the variability in the parameters. In Cefalu *et al.* (2016), the authors built a doubly robust estimator based on Bayesian model averaging, and accounted for this additional uncertainty using the bootstrap, which requires many MCMC runs. We will see in Section 4 that a small resampling procedure can account for this additional source of uncertainty with only one MCMC run, thereby improving computation time.

Further, we are in a high-dimensional setting and therefore some form of variable selection or dimension reduction is necessary, so we will imbed spike and slab priors (George & McCulloch, 1993) into these models to reduce the parameter space and allow for more efficient estimation when the number of covariates is large. In the following sections we will highlight two different flexible approaches to estimating the unknown functions, and how to utilize variable selection within each formulation.

2.2 Nonparametric prior specification

Here we will adopt fully nonparametric priors for the unknown regression functions, $f_j()$ and $g_j()$ for $j = 1, \ldots, p$. We will present the prior specification here for the outcome model only, but analagous representations will be used for the treatment model. We will use a Gaussian process prior with kernel function $K(\cdot, \cdot)$, which means we can represent our prior as follows:

$$
\begin{aligned}
f_j(X_j) &\sim (1 - \gamma_j)\delta_{\mathbf{0}} + \gamma_j \mathcal{N}(\mathbf{0}_n, \sigma^2\tau_j^2\boldsymbol{\Sigma}_j) && (4)\\
\gamma_j &\sim \text{Bernoulli}(\theta) && (5)\\
\theta &\sim \mathcal{B}(a_\theta, b_\theta) && (6)\\
\tau_j^2 &\sim \text{Gamma}(1/2, 1/2) && (7)\\
\sigma^2 &\sim \text{InvGamma}(a_{\sigma^2}, b_{\sigma^2}). && (8)
\end{aligned}
$$

Here, σ^2 is the residual variance of the model when the outcome is normally distributed, otherwise it is simply fixed to 1. Our prior formulation resembles that of Reich *et al.* (2009), although they performed variable selection within Gaussian processes by placing spike and slab priors on the variance of the Gaussian process. We utilize a latent variable, γ_j, which indicates whether variable j is important for predicting the outcome. If $\gamma_j = 0$ then the predictor is eliminated from the model completely. We will assume the prior on the variance τ_j^2 to be a gamma$(1/2, 1/2)$ similarly to Mitra & Dunson (2010). Finally, $\boldsymbol{\Sigma}_j$ is a covariance matrix with the (i, i') component being $K(X_{ji}, X_{ji'})$. Any number of kernels can be chosen for $K(\cdot, \cdot)$, though we will proceed with $K(z, z') = \exp -\frac{|z-z'|}{\phi}$, where ϕ is a bandwidth parameter that must be chosen.

The formulation above is fully nonparametric in the sense that we assume no parametric form about the response functions $f_j(X_j)$. This removes the burden from the user of having to pre-specify any functional forms between the covariates and the outcome. One criticism of using Gaussian processes is that they can be very computationally burdensome, particularly as the sample size increases, because at each iteration of an MCMC one must invert an n by n matrix. Reich *et al.* (2009) showed that this can be avoided by using a singular value decomposition on the kernel covariance matrices before running the MCMC. Details of this can be found in their paper or in our appendix. This allows for us to utilize Gaussian processes in reasonably sized data sets, but the computation can still be slow for large sample sizes, so now we will introduce semi-parametric models to alleviate these concerns.

2.3 Semiparametric prior specification

In this section, we present an analagous formulation of the problem, where we drop the fully nonparametric Gaussian process and replace it with a semi-parametric model based on basis functions, such as cubic splines. To do this, we must introduce some additional notation. We will let $\widetilde{\boldsymbol{X}}_j$ represent an n by q matrix of basis functions used to approximate the effect of X_j on the outcome. In the case of cubic splines this would represent modeling $f_j(X_j)$ with q-degrees of freedom splines. We can now write our prior specification as follows:

$$
\begin{aligned}
f_j(X_j) &= \widetilde{\boldsymbol{X}}_j\boldsymbol{\beta}_j && (9)\\
(\boldsymbol{\beta}_j|\gamma_j) &\sim (1 - \gamma_j)\delta_{\mathbf{0}} + \gamma_j\psi(\boldsymbol{\beta}_j) && (10)\\
\gamma_j &\sim \text{Bernoulli}(\theta) && (11)\\
\theta &\sim \text{Beta}(a_\theta, b_\theta) && (12)\\
\sigma^2 &\sim \text{InvGamma}(a_{\sigma^2}, b_{\sigma^2}). && (13)
\end{aligned}
$$

There are a couple of key differences to note here. We are now placing spike and slab priors on regression coefficients instead of on the entire function, $f_j(X_j)$. We have placed a multivariate spike and slab prior on the group of coefficients, $\boldsymbol{\beta}_j$, that will force all coefficients to zero and eliminate covariate j from the model if $\gamma_j = 0$. If $\gamma_j = 1$, then all elements of $\boldsymbol{\beta}_j$ will be nonzero and their prior distribution will be $\psi(\boldsymbol{\beta}_j)$, which we will set to be a multivariate normal distribution centered at $\mathbf{0}$ with covariance set to $\sigma^2\sigma^2_{\boldsymbol{\beta}} I_n$. We must select a value of $\sigma^2_{\boldsymbol{\beta}}$, which can be done either via empirical Bayes or by placing a hyper prior on $\sigma^2_{\boldsymbol{\beta}}$.

2.4 Estimation of treatment effects

In the previous sections we detailed how to model the nuisance parameters required to estimate average treatment effects, so now we will illustrate how to simultaneously estimate treatment effects while estimating the nuisance functions, $f_j(X_j)$ and $g_j(X_j)$. To do so, we will highlight how the doubly robust estimator in Equation 3 could be implemented within our modeling strategy. Letting $b = 1, \ldots, B$ represent the B iterations of an MCMC, once we have samples of the nuisance functions, $f_j(X_j)^{(b)}$ and $g_j(X_j)^{(b)}$, then we automatically obtain $e_i^{(b)}$,$m_{1i}^{(b)}$, and $m_{0i}^{(b)}$ as they are simply functions of the unknown regression functions. Given these quantities, we can estimate the posterior mean of the doubly robust estimator via

$$\widehat{\Delta}(1,0) = \frac{1}{Bn}\sum_{b=1}^{B}\left[\sum_{i=1}^{n}\frac{T_i Y_i}{e_i^{(b)}} - \frac{T_i - e_i^{(b)}}{e_i^{(b)}} m_{1i}^{(b)}\right] - \left[\sum_{i=1}^{n}\frac{(1-T_i)Y_i}{1-e_i^{(b)}} + \frac{T_i - e_i^{(b)}}{1-e_i^{(b)}} m_{0i}^{(b)}\right]. \tag{14}$$

Typically, by having a posterior distribution of a given quantity, we can also conduct inference since we can use the quantiles or standard deviation of the posterior samples to construct credible intervals. To understand why this is not the case, however, we can think of an extreme example of where we know the true values for e_i, m_{1i}, and m_{0i} and therefore there is no uncertainty in the posterior distribution of $\Delta(1,0)$. Clearly, there is still uncertainty in the estimation of the doubly robust estimator, which stems from the randomness in Y and T that is not accounted for across the posterior samples. In Section 4 we will detail how to supplement the MCMC samples with a bootstrapping procedure to estimate the standard error of our estimator without having to run any additional MCMC.

2.5 Treatment effect heterogeneity

So far we have assumed that the effect of the treatment on the outcome is shared across all subjects in the data, or all subsets of the covariate space. In practice, this may not be a realistic assumption, and ignoring this treatment effect heterogeneity can lead to bias of average treatment effects. We will briefly discuss how to extend the above ideas to heterogeneous treatment effects with binary treatments, however, it is important to note that we are not trying to estimate or identify subpopulations with heterogeneous treatment effects. Rather, we are simply trying to allow for the existence of such groups when estimating population level average treatment effects. Following the ideas of Hill (2011), we can simply propose separate outcome models for the treated and control groups utilizing the same ideas regarding flexible function estimation and variable selection as above. We would now have three separate models: A treatment model, an outcome model for the controls, and an outcome model for the treated. Using the posterior distribution of these three models allows us to estimate necessary quantities such as the conditional distribution of the potential outcomes in a manner that allows for heterogeneity. For instance, in the doubly robust estimator of Equation 3, m_{0i} and m_{1i} would come from the separate regressions on the control group and treated group, respectively.

3 Posterior contraction rate for treatment effect

In the frequentist literature, convergence rates of treatment effects are typically established, particularly in high-dimensional models when achieving $\sqrt{n}-$consistency is not trivial. Here we will establish analagous results for the posterior distribution, and show that the posterior contracts at similar rates to those seen in the semi-parametric causal inference literature. We will restrict attention to the doubly robust setting, which is particularly of interest in high-dimensional scenarios because it allows us to achieve better rates of convergence than approaches based on a single model. The intuition behind this is that the estimation error of the doubly robust estimator as written in Equation 3 can be written as the product of the errors from the outcome model and the treatment model. This can be used to our advantage in a number of ways. If one model is incorrect, we can still obtain consistent estimates of treatment effects if the other is correct (i.e double robustness). The other advantage is that if we are in a high-dimensional scenario where the treatment and outcome models converge more slowly than standard, low-dimensional models, then the convergence rate of the doubly robust estimator is the product of the convergence rates of the treatment and outcome model. In the semi-parametric causal inference literature this has been used to allow for machine learning approaches that converge at $n^{1/4}$ rates or high-dimensional models that converge at $\sqrt{n/\log p}$ rates Chernozhukov *et al.* (2016); Farrell (2015). Here, we establish that these same ideas carry over into posterior

contraction rates for the posterior distribution of the treatment effect. For this, we will restrict attention to estimating $E(Y(t)) = \mu_t^*$, and the extension to estimating $\Delta(1,0)$ is trivial.

3.1 Notation and assumptions

Before showing posterior contraction rates, we must introduce some new notation. Let $p_{ti} = P(T_i = t|\boldsymbol{X}_i)$ and $m_{ti} = E(Y(t)|\boldsymbol{X}_i)$, where each of these can be calculated from the parameters in our model specification above. Denote their true values by p_{ti}^* and m_{ti}^*, and let $\boldsymbol{D}$ represent the observed data. For a sequence of numbers $\epsilon_n \to 0$, and a constant M, which does not depend on ϵ_n, we will show that

$$\mathbb{P}_n(p_t, m_t : |\mu_t - \mu_t^*| > M\epsilon_n|\boldsymbol{D}) \to 0 \tag{15}$$

where ϵ_n defines the rate of contraction of the posterior. The faster that ϵ_n converges to zero while maintaining this result implies that our posterior distribution contracts at a faster rate. Our goal will be to show that if the treatment model contracts at rate η_t and the outcome model contracts at rate η_y, then the doubly robust estimator contracts at a rate of $\max(n^{-1/2}, \eta_t\eta_y)$. If only one of the models is consistent, then we obtain a rate of $\max(n^{-1/2}, \eta)$, where η is the rate of the correctly specified model. We will require assumptions on the treatment and outcome models that allow us to obtain posterior contract rates of the individual models, such as sparsity or conditions on the design matrix. These rates have been proved elsewhere and we defer to those papers for details of the assumptions required to obtain posterior contraction (van der Vaart & van Zanten, 2008; Castillo *et al.* , 2015). Our goal is simply to show what happens to our Bayesian doubly robust estimator, if one has posterior contraction rates for the treatment and/or outcome models (conditional on any assumptions required to achieve those rates).

3.2 Main result

Theorem 1: Assume positivity, no unmeasured confounding, SUTVA, and any additional assumptions required for the posterior contraction rates of either the treatment or outcome model. If both models are consistent, then Equation 15 is satisfied with $\epsilon_n = max(n^{-1/2}, \eta_t\eta_y)$. If only one model is correctly specified, then Equation 15 is satisfied with $\epsilon_n = max(n^{-1/2}, \eta)$, where η is the contraction rate for the correctly specified model.

A proof of this result can be found in The Appendix. This result has a number of implications for the choice of treatment and outcome models in both low-dimensional, and high-dimensional scenarios. In high-dimensional models where the posterior distributions of any nuisance functions will contract at slower rates than $n^{-1/2}$, we can still obtain a contraction rate of the treatment effect that is $n^{-1/2}$ if both models are correctly specified and the product of their contraction rates is less than or equal to $n^{-1/2}$. For instance, it is well understood that in high-dimensional linear regression, the posterior contraction rate of regression coefficients when using spike and slab priors is $\sqrt{\log p/n}$ (Castillo *et al.* , 2015). If both the treatment model and outcome model parameters contract at this rate, then the posterior distribution of the treatment effect still contracts at $n^{-1/2}$ as long as $\log p \leq n^{3/2}$. Our result has implications for low-dimensional models as well. If one is interested in using nonparametric priors, which contract more slowly than parametric models, but allow for flexible modeling of the nuisance functions, $n^{-1/2}$ contraction can still be obtained under the same logic. Of course, one of the main implications of this theorem, regardless of the covariate dimension, is that posterior consistency is achieved as long as one model is correctly specified.

4 Partial bootstrap for valid inference

As discussed in Section 2.4, the posterior samples of our estimator do not account for all the sources of uncertainty because they ignore the additional uncertainty from the data. To see how we can alleviate this issue we can write out the variance decomposition formula in our context where $\boldsymbol{\Psi}$ represents all parameters in our model:

$$\text{Var}(\widehat{\Delta}) = E_{Y,T}(\text{Var}_{\boldsymbol{\Psi}}(\widehat{\Delta}|Y,T)) + \text{Var}_{Y,T}(E_{\boldsymbol{\Psi}}(\widehat{\Delta}|Y,T)) \tag{16}$$

Now we have explicitly accounted for the variation in (Y,T), whereas if we simply looked at the posterior distribution of Δ we would obtain $\text{Var}_{\boldsymbol{\Psi}}(\widehat{\Delta}|Y,T)$ and would underestimate the variance. All expectations and variances with respect to $\boldsymbol{\Psi}$ can be calculated from the posterior samples, but now we will introduce a bootstrapping procedure to estimate the moments that are with respect to (Y,T).

We will adopt the standard nonparametric bootstrap (Efron & Tibshirani, 1994), which involves drawing n samples with replacement from our data. We will do this M times to create M resampled data sets. For each of the resampled data sets, we can calculate the estimator of interest for each of the $b = 1, \ldots, B$ posterior draws of the unknown parameters. This leaves us with MB estimates of the quantity of interest,

which is depicted by the matrix below. In the matrix, the rows correspond to the estimates for a given data set, while the columns represent estimates for a given MCMC iteration.

$$\begin{pmatrix} \widehat{\Delta}_{11} & \widehat{\Delta}_{12} & \dots & \widehat{\Delta}_{1B} \\ \widehat{\Delta}_{21} & \ddots & & \widehat{\Delta}_{2B} \\ \vdots & & \ddots & \vdots \\ \widehat{\Delta}_{M1} & \widehat{\Delta}_{M2} & \dots & \widehat{\Delta}_{MB} \end{pmatrix}$$

To calculate the expectations and variances needed, we can simply take the means and variances across the relevant rows and columns of the matrix of estimates. For instance, to calculate $E_{Y,T}(\text{Var}_{\mathbf{\Psi}}(\widehat{\Delta}|Y,T))$ we could calculate the variance of the estimates within each row, which will leave us with an $M-$dimensional vector of conditional variances. Then we can simply average across the M conditional variances to get their expectation with respect to (Y,T). An analagous operation could be performed to estimate $\text{Var}_{Y,T}(E_{\mathbf{\Psi}}(\widehat{\Delta}|Y,T))$ and then we would have an estimate of the standard error for $\widehat{\Delta}$. This could be used in conjuction with a normal approximation to construct credible intervals for Δ. Alternatively, one could simply take the 2.5 and 97.5 percentiles of the entire matrix of values and use this to construct a 95% credible interval for Δ. We have found that both of them work well, however, if the sampling distribution of Δ were far from normal, then the percentile approach would be preferred. This may seem as though it is computationally burdensome since we are calculating our estimator MB times, however, it is much faster than even one additional MCMC run, let alone M. For each data set, we do not have to perform MCMC, we simply have to take the output from the original MCMC and apply it to each data set separately, which takes a very small amount of time.

5 Illustration of how asymptotics suffer in high-dimensions

In this section we will compare the finite sample and asymptotic variance of our proposed estimator and the one proposed by Farrell (2015). We will simulate data from sparse, linear models for both the treatment and outcome. We will apply our doubly robust estimator with Bayesian linear models and sparsity inducing priors as described in Section 2.3. To build a doubly robust estimator, Farrell (2015) fit lasso (or group lasso) models (Tibshirani, 1996; Yuan & Lin, 2006) on both a treatment and outcome model to identify covariates that are associated with the treatment and outcome respectively. Then, they re-fit non-penalized estimators of the treatment and outcome models using only the covariates identified by the original lasso regressions. From these two regressions they can calculate the doubly robust estimator defined in Equation 3 where e_i, m_{0i}, and m_{1i} are estimated using the non-penalized regression models. The authors derived some important theoretical results that demonstrate that their proposed double robust estimator is consistent and asymptotically normal. Our goal of this brief illustration is to elucidate why Bayesian inference, which does not rely on asymptotics, can provide a more accurate assessment of the finite sample uncertainty, especially in high-dimensional scenarios. Here we focus on the estimator from Farrell (2015) as it uses the exact same doubly robust estimator, with the main difference coming in how inference is performed. We will see that these ideas extend to other estimators rooted in asymptotics in the following section.

For each of the two doubly robust estimators, we will plot two lines. First, we will show the sampling distribution of the estimator as taken by the empirical distribution of the estimators across a large number of simulated datasets. Next, we will plot a normal density centered at the mean of the estimates across all datasets with a standard deviation that is the average estimated standard error across all datasets. If the estimated standard errors are correct, then this average standard error should be the same as the standard deviation of the sampling distribution and the two curves should look similar. Figure 1 shows the results for $n = 100$ and $p \in \{100, 300, 500\}$. The top row shows the results for the estimator based on asymptotic confidence intervals and the dashed line has much smaller tails than the solid line, indicating that the asymptotic distribution used for inference is not properly accounting for the uncertainty in the estimator. This phenomenon gets worse as p grows larger, and we see that the coverage probabilities decrease from 88% to 80%. The Bayesian counterpart of the same estimator, however, maintains the correct coverage probabilities for any dimension of the data, and the dashed and solid lines are very similar, showing that the uncertainty in the estimator is fully accounted for.

6 Simulation studies

Here we present results on simulated data to assess the performance of our proposed approach in a number of settings. First we will present results using the doubly robust estimator discussed throughout the paper in the context of binary treatments and a high-dimensional vector of confounders. We will then turn attention to a continuous treatment and examine doubly robust estimators in that setting, as this will be the scenario that our data analysis of Section 7 will be in.

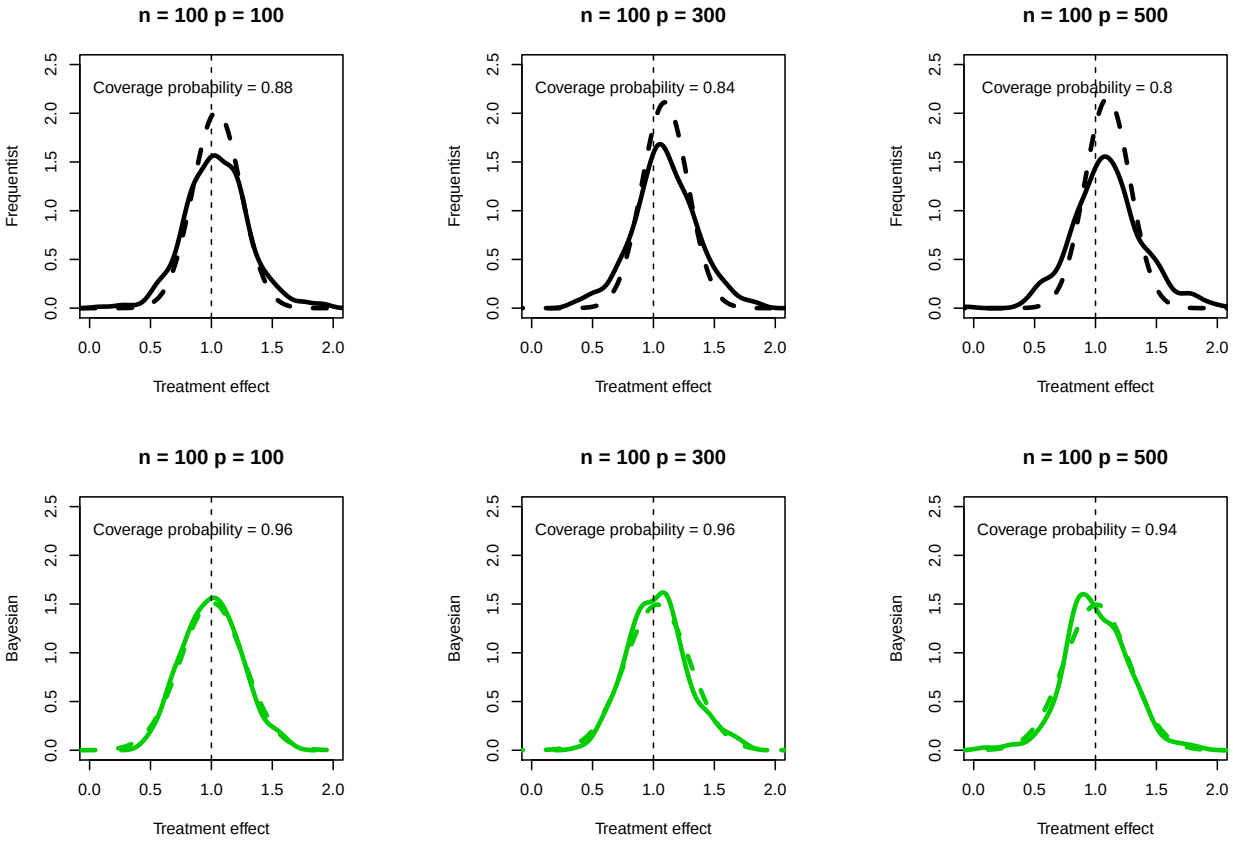

Figure 1: Comparison of empirical and assumed distributions for the doubly robust estimator of Farrell (2015) and our approach from Section 2. The solid lines are the empirical sampling distributions, while the dashed lines are normal distributions with standard deviation equal to the average estimated standard deviation across the simulations

6.1 Binary treatments

Here, we will restrict attention to $n = 100$ and $p = 500$, and we will generate data from the following setup:

$$Y_i|T_i, \boldsymbol{X}_i \sim \mathcal{N}(\mu_i, \boldsymbol{I}_n) \tag{17}$$
$$T_i|\boldsymbol{X}_i \sim \text{Bernoulli}(p_i) \tag{18}$$
$$\boldsymbol{X}_i \sim N(\boldsymbol{0}_p, \boldsymbol{\Sigma}). \tag{19}$$

Throughout, we will assume that $\Sigma_{ij} = 1$ if $i = j$ and $\Sigma_{ij} = 0.3$ if $i \neq j$. We will simulate data under two scenarios for the true propensity and outcome regressions:

Linear Simulation: (20)
$$\mu_i = T_i + 0.75X_{1i} + X_{2i} + 0.6X_{3i} - 0.8X_{4i} - 0.7X_{5i} \tag{21}$$
$$p_i = \Phi(0.15X_{1i} + 0.2X_{2i} - 0.4X_{5i}) \tag{22}$$
Nonlinear Simulation: (23)
$$\mu_i = T_i + 0.8X_{1i} + 0.4X_{2i}^3 + 0.25\text{exp}(\text{abs}(X_{2i})) + 0.8X_{5i}^2 - 1.5\text{sin}(X_{5i}) \tag{24}$$
$$p_i = \Phi(0.15X_{1i} - 0.4X_{2i} - 0.5X_{5i}) \tag{25}$$

We will be estimating the average treatment effect using: a) double post selection regression (Double PS) introduced in Belloni *et al.* (2014); b) doubly robust estimators (DR-lasso) introduced in Farrell (2015); and c) the residual de-biasing approach (De-biasing) of Athey *et al.* (2016). Using the Bayesian methods described above we will estimate the treatment effect using regression based estimators as well as doubly robust estimators. For the treatment and outcome models, we will consider linear models, models using 3 degree of freedom splines for each covariate, and models that use Gaussian process priors for each covariate. This will lead to 3 regression based estimators and 9 potential doubly robust estimators (all possible combinations of the treatment and outcome model). We will show the results for the doubly robust estimator that

uses the best treatment and outcome model as chosen by WAIC Watanabe (2010); Gelman *et al.* (2014). The regression estimators will be denoted by Reg-1, Reg-3, and Reg-GP, while the doubly robust estimator will be denoted by DR-Bayes.

Figure 2 shows the results from the two simulation studies across both scenarios examined. Estimators proposed in this paper are in grey, while the existing approaches can be found in black. In the linear scenario, the models that estimate treatment effects using linear outcome models do very well, as the Reg-1 and Double PS approaches do very well. Our DR-Bayes estimator does slightly worse than the Reg-1 estimator in terms of MSE, though the differences are not substantial. Interestingly, the DR-Bayes and Reg-1 estimators are the only estimators that achieve interval coverages near the nominal level. In the nonlinear simulation, we see that our estimators that allow for flexible modeling (Reg-3, Reg-GP, DR-Bayes) obtain the lowest MSE of all approaches. This is expected since the other approaches assume linearity of the outcome model, treatment model, or both. Again, our Bayesian estimators achieve the nominal coverage rate in small samples.

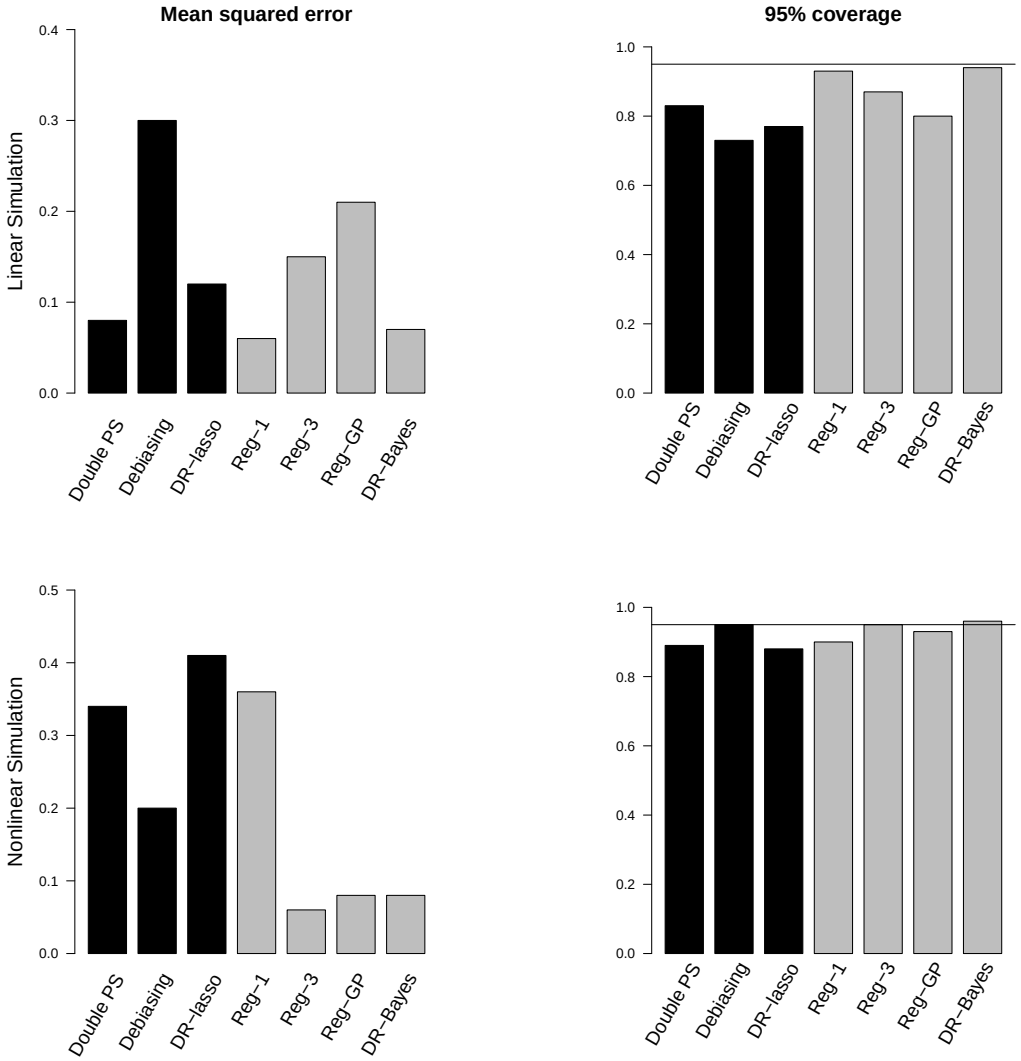

Figure 2: Results from simulations with binary treatments. The top panel shows results for the linear scenario, while the bottom panel shows results for the nonlinear scenario. The first column shows mean squared error, while the second column shows 95% interval coverages.

6.2 Continuous treatments

Here, we will restrict attention to $n = 200$ and $p = 200$, and we will generate data from the following setup:

$$Y_i | T_i, \boldsymbol{X}_i \sim \mathcal{N}(\mu_i^y, \boldsymbol{I}_n) \tag{26}$$

$$T_i | \boldsymbol{X}_i \sim \mathcal{N}(\mu_i^t, \boldsymbol{I}_n) \tag{27}$$

$$\boldsymbol{X}_i \sim N(\mathbf{0}_p, \boldsymbol{\Sigma}), \tag{28}$$

where

$$\mu_i^y = 5 + 0.05T_i^3 - 0.1T_i^2 + 0.6X_{1i} + 0.4\text{exp}(X_{1i}) + \log(\text{abs}(0.65X_{2i})) + 0.5(1 + X_{3i})^2 \tag{29}$$

$$\mu_i^t = 0.6X_{1i}^2 + 0.6X_{1i} + \exp(\text{abs}(0.65X_{2i})) - 0.8X_{3i}^2, \tag{30}$$

and $\Sigma_{ij} = 1$ if $i = j$ and $\Sigma_{ij} = 0.3$ if $i \neq j$. Our estimand of interest is now the entire exposure response curve, therefore we will be estimating $E(Y(t))$ for all t in the support of T. To estimate this quantity for all t we will use our models as described above and then apply either regression based estimators that marginalize over the covariate distribution, or the doubly robust estimator that was introduced in Kennedy *et al.* (2017). This estimator involves creating a pseudo-outcome:

$$\xi(\boldsymbol{D}_i, \boldsymbol{\Psi}) = \frac{Y_i - E(Y_i|T_i, \boldsymbol{X}_i)}{p(T_i = t|\boldsymbol{X}_i)} \int_{\mathcal{X}} p(T_i = t|\boldsymbol{X}_i) d\mathbb{P}_n(\boldsymbol{X}) + \int_{\mathcal{X}} E(Y_i|T_i, \boldsymbol{X}_i) d\mathbb{P}_n(\boldsymbol{X}) \tag{31}$$

,

and then regressing this pseudo-outcome against the treatment, potentially in a flexible manner so that the exposure-response curve can be nonlinear. We will use this estimator, where the treatment and outcome models are built using the Bayesian machinery above to reduce the dimension of the covariate space, and then perform inference using the resampling approach described in Section 4. To assess the performance of the various methods at estimating the whole curve, we will evaluate the performance of each method at 20 distinct locations on the curve and average relevant metrics such as bias or interval coverage across the 20 locations. We will use cubic polynomials to model the exposure-response curve, which encaptures the true curve. Any flexible approach would work here, however, we choose the correct model since the goal of our simulation study is not to assess the impact of the final stage model on inference, rather we want to assess how high-dimensional models work in this context to adjust for confounding and provide valid credible intervals.

Figure 3 shows the results averaged across 1000 simulations. The Reg-1 estimator does very poorly in terms of MSE and interval coverages, which is expected because it assumes linearity, when the true model is highly nonlinear. The Reg-3, Reg-GP, and DR-Bayes approaches all allow for nonlinear relationships between the covariates and treatment/outcome, and therefore these approaches perform well with respect to the metrics looked at. Again, our DR-Bayes estimator achieves interval coverages at or near the nominal level of 95%. The right panel of Figure 3 shows that the DR-Bayes estimator generally estimates the entire curve well, with very few simulations deviating from the true shape.

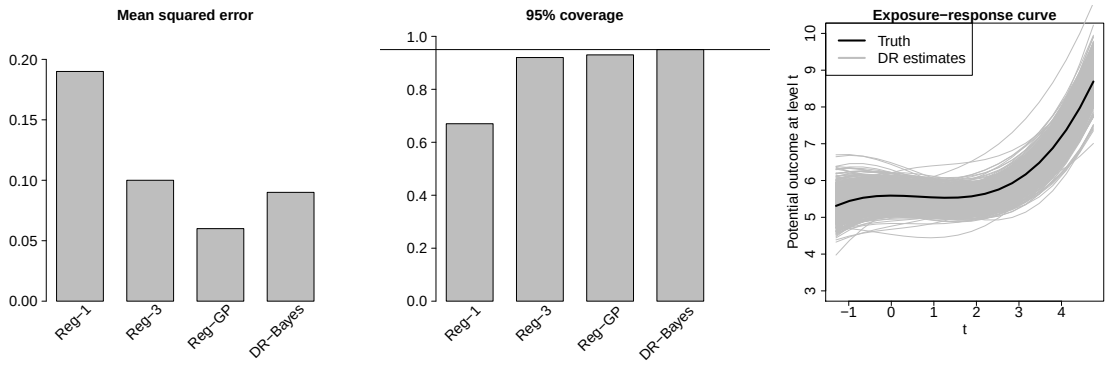

Figure 3: Simulation results for continuous treatments. The left panel presents the mean squared error, the middle panel shows the 95% credible interval coverage, and the right panel shows the estimates of the exposure-response curve across the 1000 simulations for the doubly robust estimator.

7 Application to EWAS

Environmental wide association studies (EWAS) have been increasingly common in recent years as scientists attempt to gain a better understanding of how various chemicals and toxins affect the biological processes in the human body (Wild, 2005; Patel & Ioannidis, 2014). In particular, EWAS look to study the effects of a large number of exposures that humans are invariably exposed to on disease or other functions in the body. The National Health and Nutrition Examination Survey (NHANES), is a cross-sectional data source

made publicly available by the Centers for Disease Control and Prevention (CDC). We will restrict attention to the 1999-2000, 2001-2002, 2003-2004, and 2005-2006 surveys, and we will aim to estimate the effects of environmental exposures on three different outcomes: HDL cholesterol levels, LDL cholesterol levels, and triglyceride levels in humans. We will use the data found in Wilson *et al.* (2018), which studied the impact of environmental agents from the NHANES data. The study contains a large number of potential confounders as participants fill out questionnaires regarding their health status, while a subset of patients receive clinical and laboratory tests that contain information on environmental factors such as pollutants, allergens, bacterial/viral organisms, chemical toxicants, and nutrients. In previous work, (Patel *et al.* , 2012), the environmental agents for which we want to estimate the causal effects of, were separated into different groups containing similar agents that might affect similar biological pathways. We will look at the effects of 14 different environmental agent groups on the three outcomes, leading to 42 different analyses. Each exposure we look at is defined as the average exposure level across all agents within the same grouping. In the NHANES data, different subjects had different environmental agents measured, leading to different populations, covariate dimensions, and sample sizes for each of the 14 different exposures. We apply our Bayesian models in conjuction with the doubly robust estimator of Kennedy *et al.* (2017) to estimate the exposure response curves for each of the 42 analyses. Both p and n vary for each of the 14 data sets, however, there is a wide range of p/n ratios from 0.08 to 0.51, with a mean of 0.25. In the following sections we will highlight different aspects of the proposed approach across all 42 analyses, such as the amount of sparsity and nonlinearity found in the data, and then we will highlight the exposure response curves of a few of the exposures considered.

7.1 Differing levels of nonlinearity

To analyze the data we fit a treatment model and an outcome model under each of the three levels of flexibility that we used in the simulation study. This includes a linear function of the covariates, three degree of freedom splines, and Gaussian processes. For each data set we looked at the WAIC of both the treatment and outcome model, and used the model with the minimum WAIC for the doubly robust estimate of the exposure response curve. Figure 4 shows histograms of the ratio of the WAIC values with the minimum WAIC within a given dataset across the three models. A value of one indicates that a particular model had the best WAIC, while larger values indicate worse fits to the data. We see that for the treatment model, the Gaussian process prior does the best as it is selected more than any other model and most of the values in the histogram are less than 1.05. Linear models do the next best and have the lowest WAIC for a number of datasets, while the spline model does the worst overall. For the outcome model, the linear model does best, followed by the Gaussian process prior and spline model, which do similarly well. Overall, these plots suggest that differing amounts of flexibility were required in these analyses, and our flexible approach might be more accurately depicting the true data generating processes.

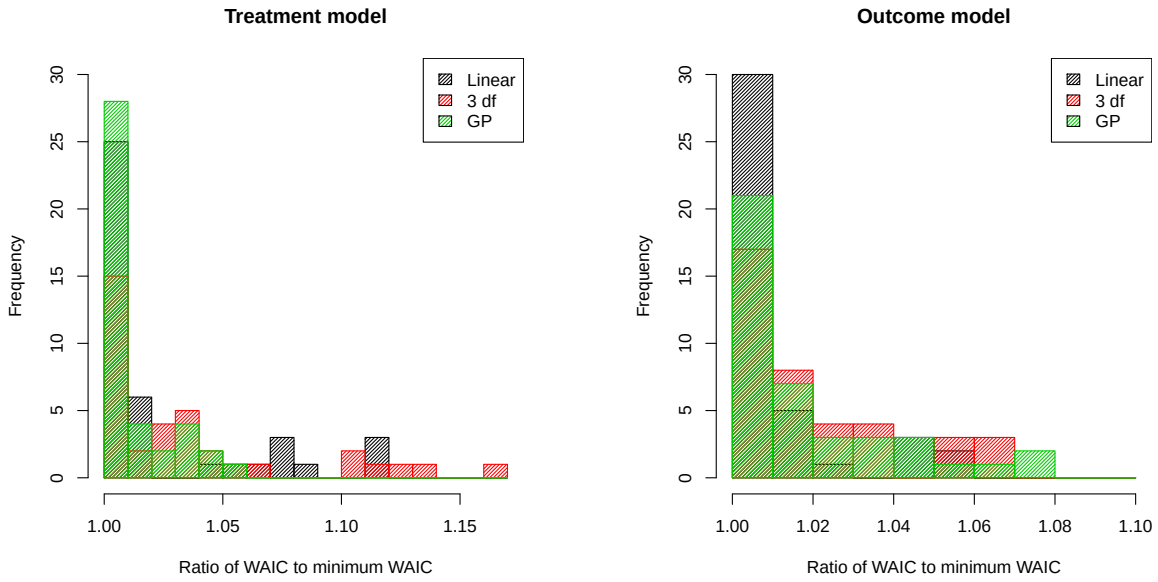

Figure 4: Ratio of WAIC values to the minimum values for each of the three models considered. The left panel shows the treatment model WAIC values, while the outcome model shows the WAIC for the outcome models.

7.2 Amount of sparsity

Now we can also examine the extent to which our sparsity inducing priors reduced the dimension of the covariate space. While our models provide posterior inclusion probabilities, we can report a binary assessment of a variable's importance by reporting whether the posterior inclusion probability is greater than 0.5. Figure 5 shows the percentage of covariates that have a posterior inclusion probability greater than 0.5 in the treatment model, outcome model, and in both models. It is clear from both the treatment and outcome models that the spike and slab priors are greatly reducing the number of covariates in the model as all datasets have less than 30% of the covariates in the models, and many are less than 10%. Looking at the right panel of Figure 5, which shows the percentage of covariates in both the treatment and outcome models, we can see that very few covariates are in both models and many data sets have 0 covariates in both models. This suggests that there is not a lot of strong confounding within these datasets. This is further supported by the fact that many of the estimated exposure response curves are very similar to the curves one would get by not controlling for any covariates.

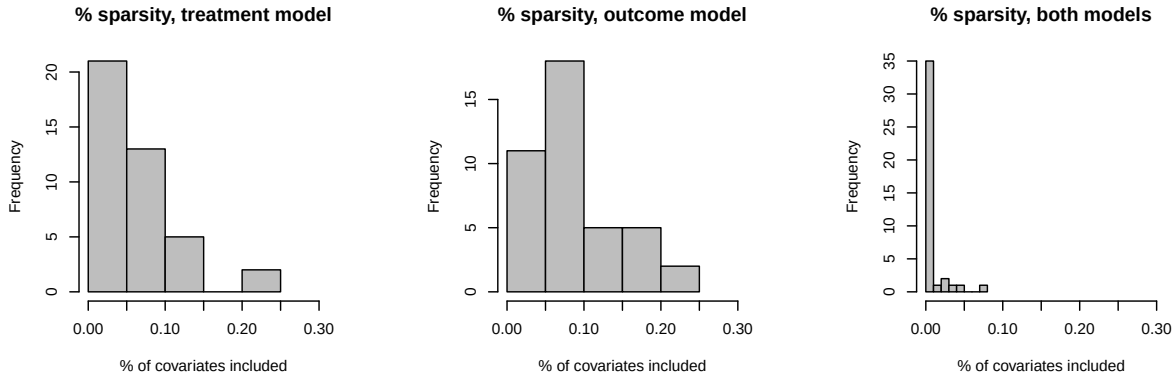

Figure 5: Percentage of covariates included in the chosen treatment models, outcome models, or both.

7.3 Exposure response curves

Here, we will highlight the estimation of the exposure response curves for three of the exposures in the analysis: Dioxins, Organochlorine pesticides, and Diakyl. The p/n ratio for these three analyses was 0.41, 0.18, and 0.34, respectively. Figure 6 shows the doubly robust estimate of the exposure response curve along with the naive curve one would get by not including any covariates in the analysis. The two estimated curves are fairly similar with a couple of exceptions. The effect of OC pesticides on Triglycerides has a much smaller slope when adjusting for covariates, and the effect of Diakyl on Triglycerides is much larger at lower levels of exposure when adjusting for covariates. In some areas of the curves there is less uncertainty in the doubly robust estimate, however, in general the naive curves are somewhat more efficient. This is not entirely surprising as the doubly robust estimators are adjusting for a large number of covariates, which can decrease efficiency unless the covariates are highly predictive of the outcome. Importantly, however, the credible intervals of the doubly robust estimator are not much wider than the naive curves, indicating that the dimension reduction from the spike and slab priors is helping with efficiency.

8 Discussion

We have introduced a Bayesian approach for causal inference that has a number of desirable features. Our approach can be applied to semiparametric estimators of causal effects that rely on a treatment or outcome model, in the context of binary, categorical, or continuous treatments. This is particularly important as the literature on estimating the causal effect curve for continuous treatments is small, and has not been extended to high-dimensional scenarios. We showed our approach maintains asymptotic properties such as double robustness and posterior contraction rates, while showing improved performance in finite samples. In particular, our approach to inference is able to capture all of the uncertainty in the data, leading to nominal credible interval coverages when frequentist counterparts that rely on asymptotics have decreased interval coverage. Further, flexible Bayesian methods allow our approach to capture nonlinear relationships in the treatment and outcome models, reducing the impact of model misspecification. Our approach has widespread applicability, as many causal estimators can be written as functions of treatment and outcome models, and the ideas seen here will apply directly. This allows users to estimate causal effects using many desirable Bayesian tools such as nonparametric priors and spike and slab priors, to name a few. While we

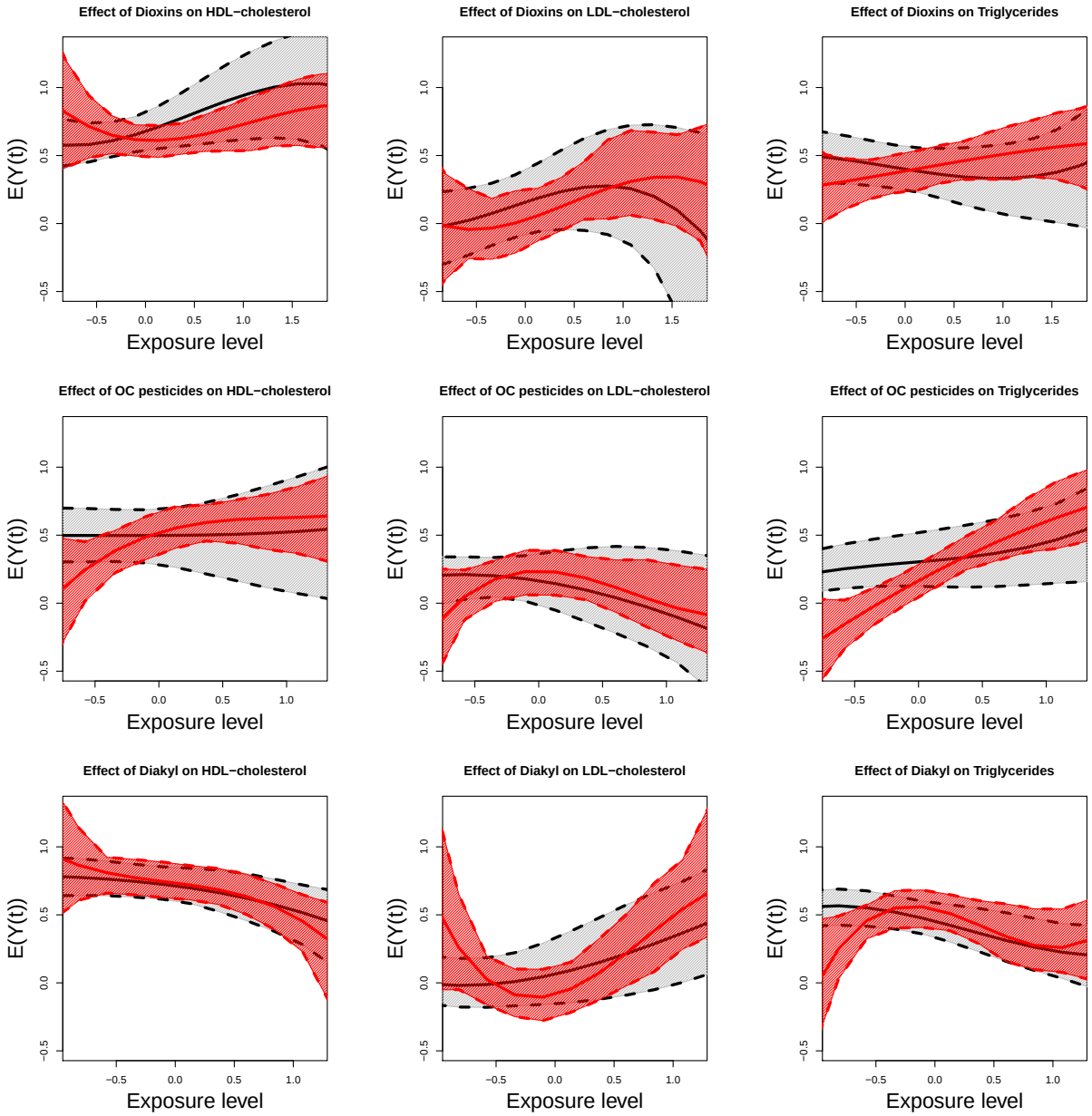

Figure 6: Estimated exposure response curves from the doubly robust estimator (black line) as well as the naive curve (red line), which does not adjust for any covariates.

focused on high-dimensional scenarios with spike and slab priors in this paper, the ideas presented apply to any type of modeling framework for the treatment and outcome models.

One of the main drawbacks of our approach is the computational burden that comes with performing Bayesian inference in high-dimensions. We have found that parametric and semi-parametric models are surprisingly fast at converging, however, fully nonparametric Gaussian process priors are slow as the sample size increases even into the 500-1000 range. While this is certainly not ideal, we have shown that reasonable amounts of flexibility can be obtained using flexible parametric structures such as splines, and this computational burden can be alleviated when the dimension of the data can overwhelm nonparametric approaches. Furthermore, we have shown that the greatest advantages of our approach over existing estimators lies in small sample sizes when the computational burden is not large and posterior samples can be easily obtained. An R package for implementing our approach can be found at github.com/jantonelli111/DoublyRobustHD

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Appendix A: Proof of posterior contraction rates

Proof: For simplicity we will drop the p_t, m_t notation in Equation 15 and write our estimator as follows:

$$\begin{aligned}
\mathbb{P}_n(|\mu_t - \mu_t^*| > M\epsilon_n|\boldsymbol{D}) &= \mathbb{P}_n\left(\frac{1}{n}\left|\sum_{i=1}^n \frac{1(T_i=t)}{p_{ti}}(y_i - m_{ti}) + m_{ti} - \mu_t^*\right| > M\epsilon_n|\boldsymbol{D}\right) \\
&= \mathbb{P}_n\left(\frac{1}{n}\left|\sum_{i=1}^n \frac{1(T_i=t)}{p_{ti}}(y_i - m_{ti}) + m_{ti} - \mu_t^*\right| > M\epsilon_n|\boldsymbol{D}\right) \\
&= \mathbb{P}_n\left(\frac{1}{n}\left|\sum_{i=1}^n \frac{1(T_i=t)}{p_{ti}}(y_i - m_{ti}) + m_{ti} - \frac{1(T_i=t)}{p_{ti}^*}(y_i - m_{ti}^*) - m_{ti}^*\right.\right. \\
&\left.\left. + \frac{1(T_i=t)}{p_{ti}^*}(y_i - m_{ti}^*) + m_{ti}^* - \mu_t^*\right| > M\epsilon_n|\boldsymbol{D}\right) \\
&= \mathbb{P}_n\left(\left|A + B\right| > M\epsilon_n|\boldsymbol{D}\right).
\end{aligned}$$

where we can define A and B as follows:

$$\begin{aligned}
A &= \frac{1}{n}\sum_{i=1}^n \frac{1(T_i=t)}{p_{ti}}(y_i - m_{ti}) + m_{ti} - \frac{1(T_i=t)}{p_{ti}^*}(y_i - m_{ti}^*) - m_{ti}^* \\
B &= \frac{1}{n}\sum_{i=1}^n \frac{1(T_i=t)}{p_{ti}^*}(y_i - m_{ti}^*) + m_{ti}^* - \mu_t^*
\end{aligned}$$

After re-arranging some terms, we can further decompose A into three separate parts such that $A = A_1 + A_2 + A_3$ where each are defined below:

$$\begin{aligned}
A_1 &= \frac{1}{n}\sum_{i=1}^n (m_{ti} - m_{ti}^*)\left(\frac{1(T_i=t)}{p_{ti}^*} - 1\right) \\
A_2 &= \frac{1}{n}\sum_{i=1}^n \frac{1(T_i=t)(p_{ti} - p_{ti}^*)(y_i - m_{ti}^*)}{p_{ti}p_{ti}^*} \\
A_3 &= \frac{1}{n}\sum_{i=1}^n \frac{1(T_i=t)(p_{ti} - p_{ti}^*)(m_{ti} - m_{ti}^*)}{p_{ti}p_{ti}^*}.
\end{aligned}$$

We can now write the probability as

$$\begin{aligned}
\mathbb{P}_n(|\mu_t - \mu_t^*| > M\epsilon_n|\boldsymbol{D}) &= \mathbb{P}_n(|A_1 + A_2 + A_3 + A_4| > M\epsilon_n|\boldsymbol{D}) \\
&\leq \mathbb{P}_n(|A_1| > \frac{M}{4}\epsilon_n|\boldsymbol{D}) + \mathbb{P}_n(|A_2| > \frac{M}{4}\epsilon_n|\boldsymbol{D}) + \\
&\quad \mathbb{P}_n(|A_3| > \frac{M}{4}\epsilon_n|\boldsymbol{D}) + \mathbb{P}_n(|B| > \frac{M}{4}\epsilon_n|\boldsymbol{D}),
\end{aligned}$$

so it now suffices to show that each of the four components above contracts at the ϵ_n rate. We will begin with the B component, which does not depend on either the posterior distribution of the treatment or outcome model as it is simply the doubly robust estimator of μ_1 evaluated at the true values for the propensity score and outcome regression minus the parameter of interest. It is easily shown that $E(B) = 0$ and therefore we can apply Chebyshevs inequality to see that

$$\begin{aligned}
\mathbb{P}_n(|B| > \frac{M}{4}\epsilon_n|\boldsymbol{D}) &\leq \frac{16\text{var}(B)}{M^2\epsilon_n^2} \\
&= \frac{16\sigma_B^2}{M^2\epsilon_n^2 n}
\end{aligned}$$

where σ_B^2 is the variance of $\frac{1(T_i=t)}{p_{ti}^*}(y_i - m_{ti}^*)$. Clearly if $\epsilon_n > n^{-1/2}$ then this probability goes to zero and we have the desired result. Now we can turn to A_1:

$$\mathbb{P}_n(|A_1| > \frac{M}{4}\epsilon_n|\boldsymbol{D}) = \mathbb{P}_n(A_1 > \frac{M}{4}\epsilon_n|\boldsymbol{D}) + \mathbb{P}_n(A_1 < -\frac{M}{4}\epsilon_n|\boldsymbol{D}).$$

We will show that $\mathbb{P}_n(A_1 > \frac{M}{4}\epsilon_n|\boldsymbol{D}) \to 0$, and an analagous result follows trivially for $\mathbb{P}_n(A_1 < -\frac{M}{4}\epsilon_n|\boldsymbol{D})$.

$$\begin{aligned}
\mathbb{P}_n(A_1 > \frac{M}{4}\epsilon_n|\boldsymbol{D}) &= \mathbb{P}_n\left(\frac{1}{n}\sum_{i=1}^{n}(m_{ti} - m^*_{ti})\left(\frac{1(T_i = t)}{p^*_{ti}} - 1\right) > \frac{M}{4}\epsilon_n|\boldsymbol{D}\right) \\
&\leq \mathbb{P}_n\left(\frac{1}{n}\sum_{i=1}^{n}K_1\left(\frac{1(T_i = t)}{p^*_{ti}} - 1\right) > \frac{M}{4}\epsilon_n|\boldsymbol{D}\right) \\
&= \mathbb{P}_n\left(\frac{1}{n}\sum_{i=1}^{n}\left(\frac{1(T_i = t)}{p^*_{ti}} - 1\right) > \frac{M}{4K_1}\epsilon_n|\boldsymbol{D}\right) \\
&\leq \frac{16K_1^2\text{var}(\frac{1(T_i=t)}{p^*_{ti}})}{M^2n\epsilon_n^2}.
\end{aligned}$$

The first inequality comes from the assumption that $\mathbb{P}_n(m_{ti} - m^*_{ti} > K_1|\boldsymbol{D}) \to 0$, which essentially states that the posterior distribution of m_1 only assigns positive probability to finite values and is not very restrictive. The last inequality comes from Chebyshevs inequality. We arrive at a similar probability to the one encountered for the B component, so we can see by similar logic that if $\epsilon_n > n^{-1/2}$, then this probability goes to zero and we have the desired result.

Now we can look at A_2, which has a similar construction as A_1.

$$\begin{aligned}
\mathbb{P}_n(A_2 > \frac{M}{4}\epsilon_n|\boldsymbol{D}) &= \mathbb{P}_n\left(\frac{1}{n}\sum_{i=1}^{n}\frac{1(T_i = t)(p_{ti} - p^*_{ti})(y_i - m^*_{ti})}{p_{ti}p^*_{ti}} > \frac{M}{4}\epsilon_n|\boldsymbol{D}\right) \\
&\leq \mathbb{P}_n\left(\frac{1}{n}\sum_{i=1}^{n}K_2 1(T_i = t)(y_i - m^*_{ti}) > \frac{M}{4}\epsilon_n|\boldsymbol{D}\right) \\
&= \mathbb{P}_n\left(\frac{1}{n}\sum_{i=1}^{n}1(T_i = t)(y_i - m^*_{ti}) > \frac{M}{4K_2}\epsilon_n|\boldsymbol{D}\right) \\
&\leq \frac{16K_2^2\text{var}(1(T_i = t)y_i)}{Mn\epsilon_n^2}.
\end{aligned}$$

We can easily see that if $\epsilon_n > n^{-1/2}$, then this probability goes to zero and we have the desired result. The first inequality relied on the assumption that $\mathbb{P}_n(\frac{p_{ti}-p^*_{ti}}{p_{ti}p^*_{ti}} > K_2|\boldsymbol{D}) \to 0$, which effectively assumes that our estimate of the propensity score is bounded away from 0 and 1. To prove the contraction rate formally for A_2, we must also show that $\mathbb{P}_n(A_2 < -\frac{M}{4}\epsilon_n|\boldsymbol{D})$, but it is trivial using the same argument as above.

Finally, we need to show contraction rates for A_3, which is where the double robustness property can be seen for the posterior distribution of μ_t.

$$\begin{aligned}
\mathbb{P}_n(|A_3| > \frac{M}{4}\epsilon_n|\boldsymbol{D}) &= \mathbb{P}_n\left(\left|\frac{1}{n}\sum_{i=1}^{n}\frac{1(T_i = t)(p_{ti} - p^*_{ti})(m_{ti} - m^*_{ti})}{p_{ti}p^*_{ti}}\right| > \frac{M}{4}\epsilon_n|\boldsymbol{D}\right) \\
&\leq \mathbb{P}_n\left(\left|\frac{1(T_i = t)(p_{ti} - p^*_{ti})(m_{ti} - m^*_{ti})}{p_{ti}p^*_{ti}}\right| > \frac{M}{4}\epsilon_n|\boldsymbol{D}\right) \\
&= \mathbb{P}_n\left(\left|\frac{1(T_i = t)(p_{ti} - p^*_{ti})}{p_{ti}p^*_{ti}}\right|\left|(m_{ti} - m^*_{ti})\right| > \frac{M}{4}\epsilon_n|\boldsymbol{D}\right) \\
&\leq \mathbb{P}_n\left(\left|K_3(p_{ti} - p^*_{ti})\right|\left|(m_{ti} - m^*_{ti})\right| > \frac{M}{4}\epsilon_n|\boldsymbol{D}\right) \\
&= \mathbb{P}_n\left(\left|(p_{ti} - p^*_{ti})\right|\left|(m_{ti} - m^*_{ti})\right| > \frac{M}{4K_3}\epsilon_n|\boldsymbol{D}\right) \\
&= \mathbb{P}_n\left(\left|(p_{ti} - p^*_{ti})\right| > \frac{M}{4K_3\left|(m_{ti} - m^*_{ti})\right|}\epsilon_n|\boldsymbol{D}, \left|(m_{ti} - m^*_{ti})\right| > \epsilon_n^{\nu_1}\right) \\
&\qquad \mathbb{P}_n\left(\left|(m_{ti} - m^*_{ti})\right| > \epsilon_n^{\nu_1}|\boldsymbol{D}\right) + \\
&\mathbb{P}_n\left(\left|(p_{ti} - p^*_{ti})\right| > \frac{M}{4K_3\left|(m_{ti} - m^*_{ti})\right|}\epsilon_n|\boldsymbol{D}, \left|(m_{ti} - m^*_{ti})\right| \leq \epsilon_n^{\nu_1}\right)
\end{aligned}$$

$$\mathbb{P}_n\left(\left|(m_{ti} - m^*_{ti})\right| \leq \epsilon_n^{\nu_1} | \boldsymbol{D}\right).$$

Here, ν_1 is chosen to lie between 0 and 1. We will now break down this probability under two cases: When the outcome model contracts at rate $\epsilon_n^{\nu_1}$, and when it does not. Intuitively these two cases are when the outcome model is correctly specified, and when it is incorrectly specified. Although it is possible for the outcome to be correctly specified and simply contract at a slower rate than ν_1 we will use this to show the robustness to model specification. First let's look at the case where the outcome model contracts at rate $\epsilon_n^{\nu_1}$. In this case $\mathbb{P}_n(\left|(m_{ti} - m^*_{ti})\right| \leq \epsilon_n^{\nu_1} | \boldsymbol{D}) \to 1$. Therefore in this setting we have

$$\begin{aligned}
\mathbb{P}_n(|A_3| > \frac{M}{4}\epsilon_n | \boldsymbol{D}) &\leq \mathbb{P}_n\left(\left|(p_{ti} - p^*_{ti})\right| > \frac{M}{4K_3\left|(m_{ti} - m^*_{ti})\right|}\epsilon_n | \boldsymbol{D}, \left|(m_{ti} - m^*_{ti})\right| \leq \epsilon_n^{\nu_1}\right) \\
&\leq \mathbb{P}_n\left(\left|(p_{ti} - p^*_{ti})\right| > \frac{M}{4K_3\epsilon_n^{\nu_1}}\epsilon_n | \boldsymbol{D}\right) \\
&= \mathbb{P}_n\left(\left|(p_{ti} - p^*_{ti})\right| > M'\epsilon_n^{1-\nu_1} | \boldsymbol{D}\right)
\end{aligned}$$

And clearly, by the definition of posterior contraction, this goes to zero if the treatment model contracts at rate $\epsilon_n^{1-\nu_1}$. This means that the entire treatment effect contracts at rate ϵ_n if the product of the contraction rates for the outcome and treatment model is less than or equal to ϵ_n. This is a Bayesian analog to results seen in Farrell (2015), and shows that we can use flexible or high-dimensional models and obtain fast posterior contraction rates due to the double robustness property. This of course assumes that both models are correctly specified. Now we will look at the second case, where the outcome model is misspecified.

$$\begin{aligned}
\mathbb{P}_n(|A_3| > \frac{M}{4}\epsilon_n | \boldsymbol{D}) &\leq \mathbb{P}_n\left(\left|(p_{ti} - p^*_{ti})\right|\left|(m_{ti} - m^*_{ti})\right| > \frac{M}{4K_3}\epsilon_n | \boldsymbol{D}\right) \\
&\leq \mathbb{P}_n\left(\left|(p_{ti} - p^*_{ti})\right| K_4 > \frac{M}{4K_3}\epsilon_n | \boldsymbol{D}\right) \\
&\leq \mathbb{P}_n\left(\left|(p_{ti} - p^*_{ti})\right| > \frac{M}{4K_3K_4}\epsilon_n | \boldsymbol{D}\right).
\end{aligned}$$

This quantity goes to zero if the treatment model contracts at rate ϵ_n. If $\epsilon_n = \eta$, then the entire treatment effect contracts at rate η unless $\eta \leq n^{-1/2}$, in which case the treatment effect contracts at rate $n^{-1/2}$, since this is the contraction rate for the other components. So now we have shown a double robustness property, in that the treatment effect still contracts around the truth if the treatment model is correctly specified and the outcome model is misspecified. We have also shown that the contraction rate can be much faster if both are specified correctly. The reverse is also true: If the outcome model is correctly specified and the treatment model is misspecified, we still obtain posterior contraction of the treatment effect and it is at the rate at which the outcome model contracts. The proof of this is nearly identical to the one we just showed so we leave it out for brevity.

Appendix B: Details of posterior sampling

Here we will present the details required for posterior sampling from both the semiparametric and nonparametric priors utilized. Throughout we will denote the full observed data as $\boldsymbol{D}_i = (Y_i, T_i, \boldsymbol{X}_i)$. First we will present the posterior sampling for the semiparametric prior that models the conditional associations between the treatment/outcome and covariates using splines with d degrees of freedom. We will be always be working with $\boldsymbol{X}$ being standardized to have mean zero and variance 1, which is crucial when using spike and slab priors. Throughout, we will show how to estimate the outcome model, but sampling from the treatment model is analagous with straightforward alterations. Finally, we will be working with the latent outcome Y_i^*, where in the case of continuous data, $Y_i^* = Y_i$. If Y_i is binary, then at every iteration of our MCMC we draw Y_i^* from a truncated normal distribution with mean set to $\beta_0 + f_t(T_i) + \sum_{j=1}^p f_j(X_{ji})$ and variance set to 1. If $Y_i = 1$ then this distribution is truncated below by 0 and if $Y_i = 0$ then it is truncated above by 0. Once we have obtained Y_i^*, then posterior sampling can continue using the latent outcome as if we had linear regression, even if the outcome is binary.

MCMC sampling for semiparametric prior

Below we detail the full conditional updates for all parameters in the model.

1. If Y_i is binary then set $\sigma^2 = 1$, and if the outcome is continuous draw σ^2 from an inverse-gamma distribution with parameters a^* and b^*, defined as:

$$a^* = a_{\sigma^2} + \frac{n}{2} + \frac{d\sum_{j=1}^{p}\gamma_j}{2}$$
$$b^* = b_{\sigma^2} + \frac{\sum_{i=1}^{n}\left(Y_i^* - \beta_0 - f_t(T_i) - \sum_{j=1}^{p} f_j(\boldsymbol{X_{ji}})\right)^2}{2} + \sum_{j=1}^{p}\sum_{k=1}^{d}\frac{\beta_{jk}^2}{2\sigma_{\boldsymbol{\beta}}^2}$$

2. While not discussed in the main text, we will be placing a $\mathcal{IG}(a_{\sigma_{\boldsymbol{\beta}}^2}, b_{\sigma_{\boldsymbol{\beta}}^2})$ prior on $\sigma_{\boldsymbol{\beta}}^2$ and therefore we can update from the full conditional:

$$\sigma_{\boldsymbol{\beta}}^2|\bullet \sim \mathcal{IG}\left(a_{\sigma_{\boldsymbol{\beta}}^2} + \frac{d\sum_{j=1}^{p}\gamma_j}{2}, b_{\sigma_{\boldsymbol{\beta}}^2} + \sum_{j=1}^{p}\sum_{k=1}^{d}\frac{\beta_{jk}^2}{2\sigma^2}\right)$$

3. Update θ from the full conditional:

$$\theta|\bullet \sim \mathcal{B}\left(a_\theta + \sum_{j=1}^{p}\gamma_j, b_\theta + \sum_{j=1}^{p}(1-\gamma_j)\right)$$

4. To update γ_j for $j = 1 \dots p$ we need to look at the conditional posterior that has marginalized over $\boldsymbol{\beta}_j$. Specifically, if we allow $\boldsymbol{\Lambda}$ to represent all parameters in the model except for $(\gamma_j, \boldsymbol{\beta}_j)$ then we can update γ_j from the following conditional distribution:

$$\begin{aligned}
p(\gamma_j = 1|\boldsymbol{D}, \boldsymbol{\Lambda}) &= \frac{p(\boldsymbol{\beta_j} = \boldsymbol{0}, \gamma_j = 1|\boldsymbol{D}, \boldsymbol{\Lambda})}{p(\boldsymbol{\beta_j} = \boldsymbol{0}|\gamma_j = 1, \boldsymbol{D}, \boldsymbol{\Lambda})} \\
&= \frac{p(\boldsymbol{D}, \boldsymbol{\Lambda}|\boldsymbol{\beta_j} = \boldsymbol{0}, \gamma_j = 1)p(\boldsymbol{\beta_j} = \boldsymbol{0}, \gamma_j = 1)}{p(\boldsymbol{D}, \boldsymbol{\Lambda})p(\boldsymbol{\beta_j} = \boldsymbol{0}|\gamma_j = 1, \boldsymbol{D}, \boldsymbol{\Lambda})} \\
&= \frac{p(\boldsymbol{D}, \boldsymbol{\Lambda}|\boldsymbol{\beta_j} = \boldsymbol{0})p(\boldsymbol{\beta_j} = \boldsymbol{0}, \gamma_j = 1)}{p(\boldsymbol{D}, \boldsymbol{\Lambda})p(\boldsymbol{\beta_j} = \boldsymbol{0}|\gamma_j = 1, \boldsymbol{D}, \boldsymbol{\Lambda})} \\
&\propto \frac{p(\boldsymbol{\beta_j} = \boldsymbol{0}, \gamma_j = 1)}{p(\boldsymbol{\beta_j} = \boldsymbol{0}|\gamma_j = 1, \boldsymbol{D}, \boldsymbol{\Lambda})} \\
&= \frac{\theta\ \Phi(\boldsymbol{0}; \boldsymbol{0}, \boldsymbol{\Sigma_\beta})}{\Phi(\boldsymbol{0}; \boldsymbol{M}, \boldsymbol{V})}
\end{aligned}$$

   where $\Phi()$ represents the multivariate normal density function. $\boldsymbol{M}$ and $\boldsymbol{V}$ represent the conditional posterior mean and variance for $\boldsymbol{\beta}_j$ when $\gamma_j = 1$ and can be defined as

$$\boldsymbol{M} = \left(\frac{\widetilde{\boldsymbol{X}}_j^T\widetilde{\boldsymbol{X}}_j}{\sigma^2} + \boldsymbol{\Sigma}_{\boldsymbol{\beta}}^{-1}\right)^{-1}\widetilde{\boldsymbol{X}}_j^T\widetilde{\boldsymbol{Y}}, \quad \boldsymbol{V} = \left(\frac{\widetilde{\boldsymbol{X}}_j^T\widetilde{\boldsymbol{X}}_j}{\sigma^2} + \boldsymbol{\Sigma}_{\boldsymbol{\beta}}^{-1}\right)^{-1}, \tag{32}$$

   where $\widetilde{\boldsymbol{Y}} = \boldsymbol{Y}^* - \beta_0 - f_t(\boldsymbol{T}) - \sum_{k\neq p} f_k(\boldsymbol{X}_k)$ and $\boldsymbol{\Sigma_\beta}$ is a $d-$dimensional diagonal matrix with $\sigma^2\sigma_{\boldsymbol{\beta}}^2$ on the diagonals.

5. For $j = 1 \dots p$, if $\gamma_j = 1$ update $\boldsymbol{\beta}_j$ from a multivariate normal distribution with mean $\boldsymbol{M}$ and variance $\boldsymbol{V}$ as defined above. If $\gamma_j = 0$, then set $\boldsymbol{\beta}_j = \boldsymbol{0}$.

6. We will jointly update β_0 and $f_t(\boldsymbol{T})$. For now we will let $f_t(\boldsymbol{T}) = \beta_t T$, though the full conditional will take the same form even if we model $f_t(\boldsymbol{T})$ with polynomials or splines. Define $\boldsymbol{Z}_t = [\boldsymbol{1}', \boldsymbol{T}]$, then the full conditional is of the form

$$(\beta_0, \beta_t)|\bullet \sim MVN\left(\left(\frac{\boldsymbol{Z}_t^T\boldsymbol{Z}_t}{\sigma^2} + \boldsymbol{\Sigma}_t^{-1}\right)^{-1}\boldsymbol{Z}_t^T\widetilde{\boldsymbol{Y}}, \left(\frac{\boldsymbol{Z}_t^T\boldsymbol{Z}_t}{\sigma^2} + \boldsymbol{\Sigma}_t^{-1}\right)^{-1}\right)$$

   where $\widetilde{\boldsymbol{Y}} = \boldsymbol{Y}^* - \sum_{j=1}^{p} f_j(\boldsymbol{X}_j)$ and $\boldsymbol{\Sigma_t}$ is a diagonal matrix with K on the diagonals, with K large so that the treatment effect is not heavily shrunk towards zero.

MCMC sampling with gaussian process priors

Now we will detail the posterior sampling for the model defined in Section 2.2.

1. Update $(\theta, \beta_0, \beta_t)$ using the same updates as above for the semiparametric prior specification.
2. To update γ_j for $j = 1 \dots p$ we need to look at the conditional posterior that has marginalized over $f_j(\boldsymbol{X}_j)$. Specifically, if we allow $\boldsymbol{\Lambda}$ to represent all parameters in the model except for $(\gamma_j, f_j(\boldsymbol{X}_j))$ then we can update γ_j from the following conditional distribution:

$$\begin{aligned} p(\gamma_j = 1|\boldsymbol{D}, \boldsymbol{\Lambda}) &= \frac{p(f_j(\boldsymbol{X}_j) = \boldsymbol{0}, \gamma_j = 1|\boldsymbol{D}, \boldsymbol{\Lambda})}{p(f_j(\boldsymbol{X}_j) = \boldsymbol{0}|\gamma_j = 1, \boldsymbol{D}, \boldsymbol{\Lambda})} \\ &= \frac{p(\boldsymbol{D}, \boldsymbol{\Lambda}|f_j(\boldsymbol{X}_j) = \boldsymbol{0}, \gamma_j = 1)p(f_j(\boldsymbol{X}_j) = \boldsymbol{0}, \gamma_j = 1)}{p(\boldsymbol{D}, \boldsymbol{\Lambda})p(f_j(\boldsymbol{X}_j) = \boldsymbol{0}|\gamma_j = 1, \boldsymbol{D}, \boldsymbol{\Lambda})} \\ &= \frac{p(\boldsymbol{D}, \boldsymbol{\Lambda}|f_j(\boldsymbol{X}_j) = \boldsymbol{0})p(f_j(\boldsymbol{X}_j) = \boldsymbol{0}, \gamma_j = 1)}{p(\boldsymbol{D}, \boldsymbol{\Lambda})p(f_j(\boldsymbol{X}_j) = \boldsymbol{0}|\gamma_j = 1, \boldsymbol{D}, \boldsymbol{\Lambda})} \\ &\propto \frac{p(f_j(\boldsymbol{X}_j) = \boldsymbol{0}, \gamma_j = 1)}{p(f_j(\boldsymbol{X}_j) = \boldsymbol{0}|\gamma_j = 1, \boldsymbol{D}, \boldsymbol{\Lambda})} \\ &= \frac{\theta\ \Phi(\boldsymbol{0}; \boldsymbol{0}, \sigma^2\tau^2\boldsymbol{\Sigma_j})}{\Phi(\boldsymbol{0}; \boldsymbol{M}, \boldsymbol{V})} \end{aligned}$$

   where $\Phi()$ represents the multivariate normal density function. $\boldsymbol{M}$ and $\boldsymbol{V}$ represent the conditional posterior mean and variance for $f_j(\boldsymbol{X}_j)$ when $\gamma_j = 1$ and can be defined as

$$\boldsymbol{M} = \left(\boldsymbol{I}_n + \frac{1}{\tau_j^2}\boldsymbol{\Sigma_j}^{-1}\right)^{-1}\widetilde{\boldsymbol{Y}}, \quad \boldsymbol{V} = \left(\boldsymbol{I}_n + \frac{1}{\tau_j^2}\boldsymbol{\Sigma_j}^{-1}\right)^{-1}, \tag{33}$$

   where $\widetilde{\boldsymbol{Y}} = \boldsymbol{Y}^* - \beta_0 - f_t(\boldsymbol{T}) - \sum_{k \neq p} f_k(\boldsymbol{X}_k)$.
3. For $j = 1 \dots p$, if $\gamma_j = 1$ update $f_j(\boldsymbol{X}_j)$ from a multivariate normal distribution with mean $\boldsymbol{M}$ and variance $\boldsymbol{V}$ as defined above. If $\gamma_j = 0$, then set $f_j(\boldsymbol{X}_j) = \boldsymbol{0}$.
4. If $\gamma_j = 0$, update τ_j^2 from it's prior distribution, which is a Gamma$(1/2, 1/2)$. If $\gamma_j = 1$, update τ_j^2 from the following distribution:

$$\mathcal{IG}\left(\frac{n+1}{2}, \frac{1}{2} + \frac{f_j(\boldsymbol{X}_j)^T\boldsymbol{\Sigma}_j^{-1}f_j(\boldsymbol{X}_j)}{2\sigma^2}\right)$$

5. If Y_i is binary then set $\sigma^2 = 1$, and if the outcome is continuous draw σ^2 from an inverse-gamma distribution with parameters a^* and b^* defined as:

$$\begin{aligned} a^* &= a_{\sigma^2} + \frac{n(1 + \sum_{j=1}^p \gamma_j)}{2} \\ b^* &= b_{\sigma^2} + \frac{\sum_{i=1}^n \left(Y_i^* - \beta_0 - f_t(T_i) - \sum_{j=1}^p f_j(\boldsymbol{X_{ji}})\right)^2}{2} + \sum_{j=1}^p \frac{\gamma_j f_j(\boldsymbol{X}_j)^T\boldsymbol{\Sigma}_j^{-1}f_j(\boldsymbol{X}_j)}{2\tau_j^2} \end{aligned}$$

One thing to note is that in the conditional updates for $(\gamma_j, f_j(\boldsymbol{X}_j))$, we must calculate $\left(\boldsymbol{I}_n + \frac{1}{\tau_j^2}\boldsymbol{\Sigma_j}^{-1}\right)^{-1}$, which means inverting an n by n matrix at every MCMC iteration. To avoid this, we can first compute the singular value decomposition, $\boldsymbol{\Sigma}_j = \boldsymbol{ABA}^T$, where $\boldsymbol{A}$ is a matrix of eigenvectors and $\boldsymbol{B}$ is a diagonal matrix of eigenvalues. From this, it can be shown that $\left(\boldsymbol{I}_n + \frac{1}{\tau_j^2}\boldsymbol{\Sigma_j}^{-1}\right)^{-1} = \boldsymbol{A}\left(\boldsymbol{I}_n + \frac{\boldsymbol{B}^{-1}}{\tau_j^2}\right)^{-1}\boldsymbol{A}^T$, which only requires inverting a diagonal matrix and can be computed much faster.