

Supplement to "Flexible sensitivity analysis for observational studies without observable implications"

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1 Theory

Proof of Theorem 1: τ^{ATE} , τ^{ATT} , τ^{ATC} and τ^{OR} are all only functions $f(Y(t))$ $t \in \{0, 1\}$ (or $f(Y(t) | X)$ for conditional treatment effects). Thus it suffices to show that $f(Y(t) | T)$ are independent of any copula parameters. Note that in the extrapolation factorization we model $f(Y(t) | T = t)$ directly and thus, this conditional expectation is independent of copula parameters by definition. Thus it suffices to show that $f(Y(t) | T = (1-t))$ is independent of copula parameters.

$$\begin{aligned}
 f(Y(t) | T = (1-t)) &= \int f(Y(t), Y(1-t) | T = (1-t)) dY(1-t) \\
 &= \int f(Y(t) | Y(1-t), T = (1-t)) f(Y(1-t) | T = (1-t)) dY(1-t) \\
 &\propto \int f(Y(t) | T = t) \frac{f(T = (1-t) | Y(t))}{f(T = t | Y(t))} c(F(Y(t) | T), F(Y(1-t) | T)) \times \\
 &\quad f(Y(1-t) | T = (1-t)) dY(1-t) \\
 &= f(Y(t) | T = t) \frac{f(T = (1-t) | Y(t))}{f(T = t | Y(t))} \times \\
 &\quad \int c(F(Y(t) | T), F(Y(1-t) | T) | T) f(Y(1-t) | T = (1-t)) dY(1-t) \\
 &= f(Y(t) | T = t) \frac{f(T = (1-t) | Y(t))}{f(T = t | Y(t))}
 \end{aligned}$$

Where the last equality holds by using the definition of the copula density:

$$\begin{aligned}
 &\int c(F(Y(t) | T), F(Y(1-t) | T) | T) f(Y(1-t) | T = (1-t)) dY(1-t) = \\
 &= \int \frac{f(Y(t), Y(1-t) | T = (1-t))}{f(Y(t) | T = (1-t)) f(Y(1-t) | T = (1-t))} f(Y(1-t) | T = (1-t)) dY(1-t) \\
 &= \int f(Y(1-t) | Y(t), T) dY(1-t) \\
 &= 1
 \end{aligned}$$

Proof of Proposition 3: We seek to find the value of γ_t such that the model (17) implies $\rho_{Y|X}^2$ achieves a

particular value, ρ_*^2 .

$$m(X, Y(t)) =: \text{logit}(e(X, Y(t))) = \alpha_t(X) + \gamma_t Y(t) \quad (1)$$

$$= \alpha_t(X) + \gamma_t \mu_t(X) + \gamma_t (Y(t) - \mu_t(X)) \quad (2)$$

$$= \alpha_t^*(X) + \tilde{\gamma}_t \tilde{R}(t) \quad (3)$$

$$= m(X, \tilde{R}(t)) \quad (4)$$

where $\tilde{R}(t) = \frac{R(t)}{\sigma_{rt}}$ is the unit-scaled complete data residual, $\tilde{\gamma} =: \sigma_{rt}\gamma$ and $\sigma_{rt} = \sqrt{E[\text{Var}(Y(t) | X)]}$. We define $\alpha_t^*(X) =: \alpha_t(X) + \gamma_t \mu_t(X)$. Importantly, since $m(X, Y(t)) = m(X, \tilde{R}(t))$ the above implies that $\rho_{Y(t), X}^2 = \rho_{\tilde{R}(t), X}^2$. Since $\tilde{R}(t)$ is orthogonal to $\alpha_t^*(X)$ and has unit variance, we have $\text{Var}(m(X, \tilde{R}(t))) = \text{Var}(m(X) + \tilde{\gamma}_t \tilde{R}(t)) = \text{Var}(m(X)) + \tilde{\gamma}_t^2$. Thus,

$$\rho_{X, Y(t)}^2 = \rho_{X, \tilde{R}(t)}^2 = \frac{\text{Var}(m(X)) + \tilde{\gamma}_t^2}{\text{Var}(m(X)) + \tilde{\gamma}_t^2 + \pi^2/3}. \quad (5)$$

Using the definition of ‘‘implicit R-squared’’ from Section 5, we have

$$\rho_{\tilde{Y}(t)|X}^2 = \frac{\rho_{X, \tilde{Y}(t)}^2 - \rho_X^2}{1 - \rho_X^2} \quad (6)$$

$$= \frac{\frac{\pi^2/3}{\text{Var}(m(X)) + \pi^2/3} - \frac{\pi^2/3}{\text{Var}(m(X)) + \pi^2/3 + \tilde{\gamma}_t^2}}{\frac{\pi^2/3}{\text{Var}(m(X)) + \pi^2/3}} \quad (7)$$

$$= 1 - \frac{\text{Var}(m(X)) + \pi^2/3}{\text{Var}(m(X)) + \pi^2/3 + \tilde{\gamma}_t^2} \quad (8)$$

Solving the above equation for $\tilde{\gamma}_t$ such that $\rho_{\tilde{Y}(t)|X}^2 = \rho_*^2$, yields

$$|\tilde{\gamma}_t| = \sqrt{\frac{\rho_*^2}{1 - \rho_*^2} (\text{Var}(m(X)) + \pi^2/3)} \quad (9)$$

We complete the result by using the fact that $\tilde{\gamma}_t = \sigma_{rt}\gamma_t$

2 Additional Results from Section 5.1

In Section 5.1, we focus on one particular potential outcomes model, although many plausible models are possible. In this section, we provide results for two variations of the observed potential outcome model. This plot highlights that the ATE estimates vary as a function of both model specification (model checking) and the strength of confounding in both treatment arms (sensitivity analysis).

First, we posit a *pooled* model for the mean surface and residual variance $(\mu_t(X), \sigma_t^2) \sim \text{BART}(X, T)$ with $\mu_t(X) = \mu(t, X)$ and $\sigma_t^2 = \sigma_{1-t}^2$. In Figure 2a we show the results for this model, which shows has the largest estimated effect size under unconfoundedness of any of the models considered. Under unconfoundedness, the posterior mean ATE is approximately -2.5 mmHg under this model, and unlike the model proposed in Section 6.1 appears significantly different from 0.

We also show the results for the Bayesian Causal Forest (BCF) model recently introduced by (Hahn et al., 2017). In this model, the observed propensity score is included as a covariate and independent BART prior distributions are specified for the control and for the heterogeneous treatment effect and one is used for the the control outcome surface. In this model, under unconfoundedness the posterior mean for the ATE is approximately -1.73 mmHg but in contrast to the other observed data models, yields ATEs with large posterior uncertainty.

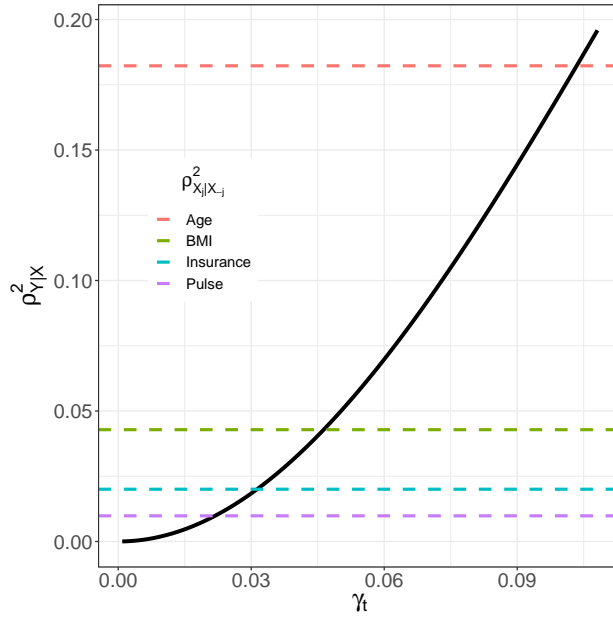
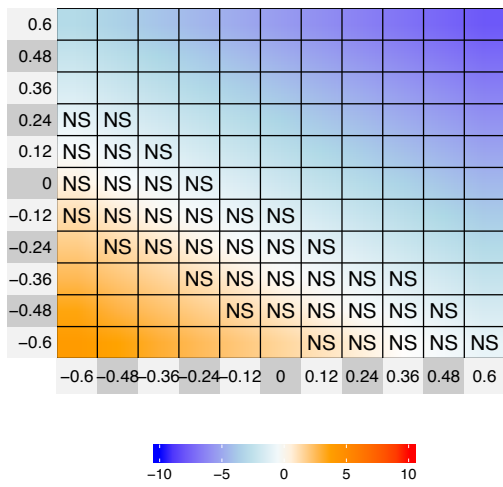
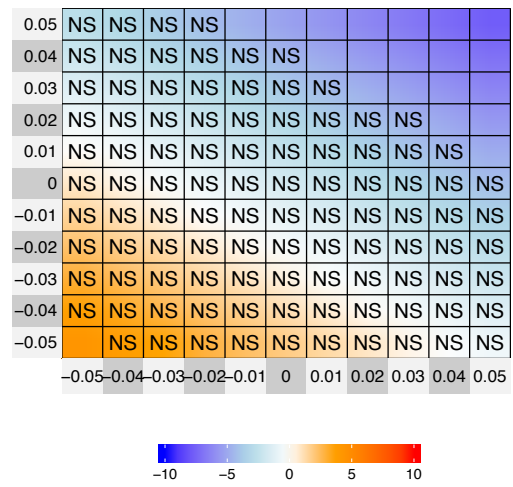


Figure 1: γ_t vs $\rho_{Y|X}^2$, calibration for the NHANES data. The magnitude of the sensitivity parameter γ_t is increasing with the residual coefficient of determination, $\rho_{Y|X}^2$. For comparison, we plot the partial coefficients of variation from covariates, $\rho_{X_j|X_{-j}}^2$, for the most important predictors: age, BMI, insurance and pulse. We calibrate the magnitude of γ_t in Section 6.1 based on BMI.



(a) ATE for pooled model



(b) ATE for BCF model

Figure 2: Average treatment effect measured in units of millimeters of mercury (mmHg). NS denotes “not significant”. a) Average treatment effect in the pooled model. Under unconfoundedness, the effect size is significantly negative. This model has the smallest posterior uncertainty. b) Average treatment effect in the Bayesian Causal Forest model. Although the effect sizes are comparable, the posterior uncertainty is significantly larger.

References

Hahn, P. R., J. S. Murray, and C. M. Carvalho (2017). Bayesian regression tree models for causal inference: regularization, confounding, and heterogeneous effects.