Bayesian Propensity Score Analysis: Simulation and Case Study

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Introduction

- This talk considers the use of propensity scores for equating groups on the basis of pre-treatment variables with the goal of strengthening causal inferences in observational studies.
- Propensity score analysis has been used extensively in fields such epidemiology, education, and sociology.
- Typically, however, propensity score analysis has been implemented within the conventional frequentist perspective of statistics.
- This perspective does not allow for encoded prior information regarding either the parameters of the model that generates the propensity scores or the model that provides the causal estimand (the outcomes model).
- A Bayesian approach to propensity score analysis provides a solution to the problem.

Bayesian Propensity Score Approaches

- A review of the extant literature reveals very few studies examining Bayesian approaches to propensity score analysis.
- An earlier paper by Rubin (1985) argued that because propensity scores are, in fact, randomization probabilities, these should be of great interest to the applied Bayesian analyst.
- In the context of propensity score analysis, Rubin (1985) argued that under the condition of strong ignorability and assuming that the estimated propensity score $\hat{e}(z)$ is an adequate summary of the observed covariates z, then the applied Bayesian will be *well-calibrated* (Dawid, 1982), in the sense that posterior predictions should match up with what happens in reality.
- Although Rubin (1985) provides a justification for why an applied Bayesian should be interested in propensity scores, his analysis does not address the actual estimation of the propensity score equation or the subsequent outcomes equation from a Bayesian perspective.

Bayesian Propensity Score Approaches

- A paper by McCandless et al. (2009) provides an approach to Bayesian propensity score analysis for observational data.
- Their approach involves treating the propensity score as a latent variable and modeling the joint distribution of the data and the parameters for the propensity score and outcomes equations simultaneously via an MCMC algorithm.
- From there, the marginal posterior probability of the treatment effect that directly incorporates uncertainty in the propensity score can be obtained.
- A recent paper by An (2010) also puts forth a joint modeling approach to Bayesian propensity score analysis.
- Gelman, et al. (2003) have argued that the propensity score should provide information only regarding study design and not regarding the treatment effect, as is the case with the Bayesian procedure advocated by McCandless et al. (2009) and An (2010).

Bayesian Propensity Score Approaches

- Recent work by Kaplan and Chen (2010) examined conventional and Bayesian propensity score approaches in a real data setting.
- The Bayesian propensity score was implemented via strata subclassification, weighting, and optimal matching and compared to the conventional propensity score.
- Results indicated that estimates of causal effects were similar across methods, but posterior probability intervals were wider, as expected.
- Kaplan and Chen (2010) did not examine these approaches in a controlled simulation setting, nor did they examine the combination of a Bayesian propensity score equation with a Bayesian outcomes equation.
- We extend Kaplan and Chen (2010) by examining a Bayesian approach to propensity score analysis in a comprehensive simulation study. We also examine the implications of specifying a Bayesian model for the treatment effect.

Strata Sub-classification on $e(z)$

1. Create strata on the estimated propensity score.
2. Estimate treatment effect within each strata.
3. Average the treatment effect and standard errors across strata using the “Rubin” approach.

- Cochran (1968) and also Rosenbaum and Rubin (1983) found that subclassification into five strata on continuous distributions such as the propensity score has been observed to remove approximately 90% of the bias due to non-random selection effects (Cochran, 1968).
- Rosenbaum and Rubin (1983) proved that when subclass units are homogeneous with respect to $\hat{e}(z)$ then the treatment and control units in the same subclass will have the same distribution on z.

Propensity Score Weighting

- Propensity score weighting is based on the idea of Horvitz-Thompson sampling weights and is designed to reweight the treatment and control group participants in terms of their propensity scores.
- Let $\hat{e}(z)$ be the estimated propensity score, and let T indicate whether an individual is treated ($T = 1$) or not ($T = 0$).
- The weight used to estimate the ATE can be defined as

$$\omega_1 = \frac{T}{\hat{e}(z)} + \frac{1 - T}{1 - \hat{e}(z)}. \tag{1}$$

- We see that when $T = 1$, $\omega_1 = 1/\hat{e}(z)$ and when $T = 0$, $\omega_1 = 1/[1 - \hat{e}(z)]$.
- This approach weights the treatment and control group up to the full sample.

Optimal Matching on the Propensity Score

- Optimal matching is an improvement on the so-called *greedy matching* algorithm.
- Greedy matching essentially does not revisit the match. It does not attempt to provide the lowest overall “cost” for the match.
- Optimal matching might reconsider a match if the total distance across all matches is less than if the algorithm proceeded.
- Optimal matching is as good and often better than greedy matching.
- Greedy matching can provide a good answer, but there is no guarantee that the answer will be tolerable - and it can be quite bad.

Design and Results of Simulation Study I

- We propose a two-step Bayesian propensity score analysis approach, with a Bayesian propensity score model in the first step and Bayesian outcomes model in the second step, and compare it with the conventional propensity score analysis (PSA).
- Also, we fit the simple linear regression and Bayesian simple regression without any propensity score adjustment for comparative purposes.
- The Bayesian simple regression utilizes the Gibbs sampler within the *MCMCregress* package in R to simulate the posterior distribution of the outcomes model.

- The simple outcomes model is written as

$$Y = \mu + \gamma T + \epsilon_1, \tag{2}$$

  where μ is the intercept, Y is the outcome, γ is the causal effect, and T is the treatment indicator.

- We assume $\epsilon_1 \sim N(0, \sigma_1^2 I_n)$ where I_n is the n dimensional identity matrix.

- Noninformative uniform priors are used for Bayesian simple regression and an inverse gamma prior is used for σ_1^2, with shape parameter and scale parameter both 0.001.

- For both PSA and BPSA, two models are specified. The first is the propensity score model, specified as a logit model.

$$Log\left(\frac{e(z)}{1-e(z)}\right) = \alpha + \beta' Z, \tag{3}$$

where α and β are unknown parameters, Z represents a design matrix of chosen covariates.

- For BPSA, we utilized the R function *MCMClogit* to simulate from the posterior distribution of a logistic regression using a random walk Metropolis algorithm.

- After estimating the conventional or Bayesian propensity scores, we use the outcomes model in the second step to estimate the causal effect via the three approaches: stratification, weighting, and matching.

- In the two simulation studies and the case study, the frequentist based average treatment effect $\hat{\gamma}_{ate}$ and standard error $\hat{\sigma}$ are estimated via ordinary least squares regression (OLS).
- For the conventional PSA, propensity score stratification is conducted by forming strata on the propensity score, calculating the OLS treatment effect within stratum, and averaging over the strata using “Rubin’s” rules.
- Propensity score weighting is performed by fitting a weighted regression with $1/\hat{e}(z)$ and $1/[1-\hat{e}(z)]$ as the weights for the treated and control group, respectively. These are the ATE weights.
- Propensity score matching utilizes the full optimal matching method proposed by Rosenbaum (1989).

Steps in Study I

- Study I examines the effects of the Bayesian propensity score model and OLS outcomes model via different sample sizes, true treatment effects and priors. Data are generated according to the following steps:
  - 1. Randomly generate three covariates z_1, z_2 and z_3 with sample sizes $n = 100$ and $n = 250$, respectively as

$$\begin{aligned} z_1 &\sim Normal(1, 1) \\ z_2 &\sim Poisson(2) \\ z_3 &\sim Binomial(n, 0.5). \end{aligned}$$

  - 2. Obtain propensity score "true model" by

$$e(z) = \frac{\exp(0.2z_1 + 0.3z_2 - 0.2z_3)}{1 - \exp(0.2z_1 + 0.3z_2 - 0.2z_3)}, \tag{4}$$

    that is, the propensity score generating model has true $\alpha = 0$ and true $\beta = (0.2, 0.3, -0.2)$.

Steps in Study I

- 3. Calculate a treatment assignment vector T by comparing the estimated propensity score $\hat{e}_i(z)$ to a random variable U_i generated from the $Uniform(0,1)$ distribution, where $i = 1, \ldots, n$. Assign $T_i = 1$ if $U_i <= \hat{e}_i(z)$, $T_i = 0$ otherwise.

- 4. Generate the outcome $Y_1, \ldots, Y_n$ using the causal model:

$$Y = 0.4z_1 + 0.3z_2 + 0.2z_3 + \gamma T + \epsilon_3, \tag{5}$$

  where $\epsilon_3 \sim N(0, 0.1)$ and γ is the true treatment effect taking two different values 0.25 and 1.25.

- 5. Data = $\{(Y_i, Z_i, T_i), i = 1, \ldots, n, n = 100 \text{ or } 250\}$.

- 6. Perform 100 replications for the PSA model.

Steps in Study I

- We specify a uniform prior on the intercept α and a multivariate normal prior on β:

$$\beta \sim Normal(b_\beta, B_\beta^{-1}),$$

with b_β as the prior mean vector and B_β as the prior precision matrix.

- For Study 1, b_β is set to 0 to imitate the case of having little information on the mean, the same as what McCandless et al. (2009) chose in their study.

- Furthermore, we examine different prior precisions at $B_\beta = 1, 10,$ and 100 to explore the relation between the choice of prior precisions and the causal effects.

Steps in Study I

- The MCMC sampling of the Bayesian propensity score model has 10^4 iterations with 1000 burnin and thinning interval of 10.
- There are $m = 1000$ sets of propensity scores $\hat{e}_{1i}(z), \ldots, \hat{e}_{mi}(z)$ generated from the predictive distribution of responses.
- For $j = 1, \ldots, m$, a treatment effect estimate $\hat{\gamma}_j$ is obtained using the jth generated Bayesian propensity score $\hat{e}_j(z)$ by the conventional stratification, weighting and optimal matching method as in the traditional PSA.

Steps in Study I

- The final estimate of the treatment effect is

$$\hat{\gamma} = \frac{\sum_{j=1}^{m} \hat{\gamma}_j}{m}. \tag{6}$$

- We show in our paper a new variance estimation formula of the estimated treatment effects for the two-stage BPSA approach,

$$Var(\hat{\gamma}) = \frac{m^{-1} \sum_{j=1}^{m} \hat{\sigma}_j^2 + (m-1)^{-1} \sum_{j=1}^{m} \left(\hat{\gamma}_j - m^{-1} \sum_{j=1}^{m} \hat{\gamma}_j \right)^2}{m}. \tag{7}$$

which accounts for variation in the treatment effect and variation in the propensity scores.

Table 1:

The $\hat{\gamma}_{ATE}$s & $S.E.$s for Conventional PSA and BPSA
with Different True γ_{ATE}s, Sample Sizes and Prior Precisions in Simulation Study 1

		$N = 100$		$N = 250$	
First Step	Second Step	γ_{ATE} =.25	γ_{ATE}=1.25	γ_{ATE} =.25	γ_{ATE} =1.25
PSA-1rep	Stratification	.30 (.13)	1.30 (.13)	.29 (.07)	1.29 (.07)
	Weighting	.31 (.12)	1.31 (.12)	.28 (.08)	1.28 (.08)
	Matching	.23 (.11)	1.23 (.11)	.29 (.07)	1.29 (.07)
PSA-100rep	Stratification	.28 (.10)	1.28 (.10)	.28 (.06)	1.28 (.06)
	Weighting	.25 (.12)	1.25 (.12)	.25 (.08)	1.25 (.08)
	Matching	.27 (.09)	1.27 (.09)	.25 (.05)	1.25 (.05)
BPSA $B_\beta = 0$	Stratification	.32 (.13)	1.32 (.13)	.32 (.09)	1.32 (.09)
	Weighting	.31 (.18)	1.31 (.18)	.27 (.13)	1.27 (.13)
	Matching	.27 (.13)	1.27 (.13)	.31 (.09)	1.31 (.09)
BPSA $B_\beta = 1$	Stratification	.31 (.13)	1.31 (.13)	.31 (.09)	1.31 (.09)
	Weighting	.29 (.17)	1.29 (.17)	.26 (.13)	1.26 (.13)
	Matching	.26 (.13)	1.26 (.13)	.30 (.08)	1.30 (.08)
BPSA $B_\beta = 10$	Stratification	.29 (.12)	1.29 (.12)	.29 (.07)	1.29 (.07)
	Weighting	.27 (.15)	1.27 (.15)	.27 (.11)	1.27 (.11)
	Matching	.24 (.12)	1.24 (.12)	.27 (.07)	1.27 (.07)
BPSA $B_\beta = 100$	Stratification	.29 (.11)	1.29 (.11)	.29 (.06)	1.29 (.06)
	Weighting	.28 (.13)	1.28 (.13)	.35 (.09)	1.35 (.09)
	Matching	.25 (.11)	1.25 (.11)	.27 (.06)	1.27 (.06)
No Adjustment	SLR-1rep	.35 (.12)	1.35 (.12)	.54 (.09)	1.54 (.09)
	SLR-100rep	.48 (.13)	1.48 (.13)	.47 (.08)	1.47 (.08)
	Bayes SLR	.35 (.12)	1.35 (.12)	.54 (.09)	1.54 (.09)

Design and Results of Simulation Study II

- We conduct the second simulation study for the BPSA with both Bayesian propensity score model and Bayesian outcomes model, in which uniform priors were compared to normal priors with varying precision.
- Also, the effects of different sample sizes and true γ_{ate} on the causal inference are studied.
- The generated data for this study is the same as the one in Simulation Study 1.
- In addition, a Bayesian outcomes model equation is developed according to equation 2 using MCMCregress function in R, which replaces the regular OLS outcomes model for stratification and optimal matching in simulation study 1.
- We are not aware of a program to conduct Bayesian weighted regression. Possible to program it in R.

Steps in Study II

- For the Bayesian propensity score model, multivariate normal priors are chosen for both α and β:

$$\begin{aligned} \alpha &\sim Normal(b_\alpha, B_\alpha^{-1}) \\ \beta &\sim Normal(b_\beta, B_\beta^{-1}), \end{aligned}$$

  where b_α and b_β are prior means, and B_α and B_β are prior precisions.

- In the Bayesian outcomes model, we also assume multivariate normal priors on the intercept μ and the treatment effect γ:

$$\begin{aligned} \mu &\sim Normal(b_\mu, B_\mu^{-1}) \\ \gamma &\sim Normal(b_\gamma, B_\gamma^{-1}), \end{aligned}$$

  with b_μ and b_γ as the prior mean, and B_μ and B_γ as the prior precisions.

- Note that when a prior precision takes value 0, *MCMCregress* uses a uniform prior.

Steps in Study II

- Study II contains two conditions to examine the performance of BPSA when there is little prior information or abundant information, respectively.

- Condition A: Little prior information
  - b_α,b_β,b_μ and b_γ set to 0.
  - B_α = B_β = 0, 1, 10, 100 for PSA model.
  - B_μ = B_γ = 0, 1, 10, 100 for outcomes model.

- Condition B: Abundant prior information. Hyperparameter values based on data generating parameter values
  - $b_\alpha = 0$ and $b_\beta = (0.2, 0.3, -0.2)$ for PSA model.
  - $b_\mu = B_\mu = 0$ for outcomes model to indicate no prior information on μ.
  - $b_\gamma = 0.25$ or 1.25 for the treatment effect.
  - $B_\alpha = B_\beta = 0, 1, 10, 100$ in the PSA model.
  - $B_\gamma = 0, 1, 10, 100$ for outcomes model.

Table 2:

The $\hat{\gamma}_{ATE}$s & $S.E.$s for Conventional and Bayesian Stratification and Matching of Design I with True γ_{ATE}=0.25 in Simulation Study 2

		$N = 100$		$N = 250$	
First Step	Second Step	Stratification	Matching	Stratification	Matching
B_β=0	B_γ = 0	.32 (.14)	.27 (.13)	.32(.09)	.31 (.09)
	$B_\gamma = 1$	.34 (.13)	.28 (.13)	.33 (.09)	.32 (.08)
	$B_\gamma = 10$	.39 (.09)	.31 (.11)	.40 (.08)	.39 (.08)
	$B_\gamma = 100$	.11 (.05)	.38 (.08)	.29 (.04)	.52 (.06)
B_β=1	B_γ = 0	.31 (.14)	.26 (.13)	.31 (.09)	.30 (.08)
	$B_\gamma = 1$	.33 (.13)	.26 (.13)	.32 (.09)	.31 (.08)
	$B_\gamma = 10$	.38 (.09)	.30 (.11)	.39 (.08)	.38 (.08)
	$B_\gamma = 100$	.11 (.05)	.38 (.08)	.29 (.04)	.52 (.06)
B_β=10	B_γ = 0	.29 (.13)	.24 (.12)	.29 (.07)	.27 (.07)
	$B_\gamma = 1$	.31 (.12)	.24 (.12)	.30 (.07)	.28 (.07)
	$B_\gamma = 10$	.37 (.09)	.29 (.10)	.36 (.07)	.34 (.07)
	$B_\gamma = 100$	.11 (.05)	.38 (.07)	.29 (.04)	.51 (.06)
B_β=100	B_γ = 0	.28 (.12)	.25 (.11)	.29 (.07)	.27 (.06)
	$B_\gamma = 1$	.30 (.11)	.25 (.11)	.30 (.06)	.28 (.06)
	$B_\gamma = 10$	.37 (.08)	.29 (.10)	.35 (.06)	.32 (.06)
	$B_\gamma = 100$	.12 (.05)	.38 (.07)	.31 (.04)	.51 (.06)
PSA-1rep		.30 (.13)	.23 (.11)	.29 (.07)	.29 (.07)
PSA-100rep		.28 (.10)	.27 (.09)	.28 (.06)	.25 (.05)

Table 3:

The $\hat{\gamma}_{ATE}$s & $S.E.$s for Conventional and Bayesian Stratification and Matching of Design I with True γ_{ATE}=1.25 in Simulation Study 2

		$N = 100$		$N = 250$	
First Step	Second Step	Stratification	Matching	Stratification	Matching
B_β=0	B_γ = 0	1.32 (.14)	1.27 (.13)	1.32 (.09)	1.31 (.09)
	$B_\gamma = 1$	1.27 (.13)	1.27 (.13)	1.31 (.09)	1.32 (.08)
	$B_\gamma = 10$	1.00 (.12)	1.22 (.11)	1.22 (.06)	1.35 (.07)
	$B_\gamma = 100$	.08 (.05)	.62 (.11)	.27 (.06)	1.15 (.06)
B_β=1	B_γ = 0	1.31 (.14)	1.26 (.13)	1.31 (.09)	1.30 (.08)
	$B_\gamma = 1$	1.26 (.13)	1.25 (.13)	1.30 (.08)	1.31 (.08)
	$B_\gamma = 10$	1.00 (.12)	1.21 (.11)	1.22 (.06)	1.34 (.07)
	$B_\gamma = 100$	.08 (.05)	.62 (.11)	.27 (.06)	1.15 (.06)
B_β=10	B_γ = 0	1.29 (.13)	1.24 (.12)	1.29 (.07)	1.27 (.07)
	$B_\gamma = 1$	1.25 (.12)	1.23 (.12)	1.28 (.07)	1.28 (.07)
	$B_\gamma = 10$	1.00 (.12)	1.20 (.10)	1.22 (.06)	1.31 (.06)
	$B_\gamma = 100$	.08 (.05)	.62 (.11)	.30 (.07)	1.15 (.06)
B_β=100	B_γ = 0	1.28 (.12)	1.25 (.11)	1.29 (.07)	1.27 (.06)
	$B_\gamma = 1$	1.25 (.11)	1.25 (.11)	1.29 (.06)	1.27 (.05)
	$B_\gamma = 10$	1.06 (.13)	1.22 (.10)	1.25 (.05)	1.30 (.05)
	$B_\gamma = 100$	.08 (.05)	.62 (.11)	.34 (.09)	1.16 (.06)
PSA-1rep		1.30 (.13)	1.23 (.11)	1.29 (.07)	1.29 (.07)
PSA-100rep		1.28 (.10)	1.27 (.09)	1.28 (.06)	1.25 (.05)

Table 4:

The $\hat{\gamma}_{ATE}$s & $S.E.$s for Conventional and Bayesian Stratification and Matching of Design II with True γ_{ATE}=0.25 in Simulation Study 2

		$N = 100$		$N = 250$	
First Step	Second Step	Stratification	Matching	Stratification	Matching
B_β=0	B_γ = 0	.32 (.14)	.27 (.13)	.32(.09)	.31 (.09)
	$B_\gamma = 1$	.31 (.13)	.27 (.13)	.32 (.09)	.31 (.09)
	$B_\gamma = 10$	.29 (.10)	.27 (.12)	.30 (.08)	.31 (.08)
	$B_\gamma = 100$	.26 (.04)	.26 (.08)	.26 (.04)	.29 (.07)
B_β=1	B_γ = 0	.30 (.14)	.25 (.13)	.31 (.09)	.30 (.08)
	$B_\gamma = 1$	.30 (.13)	.25 (.13)	.31 (.09)	.30 (.08)
	$B_\gamma = 10$	.28 (.10)	.25 (.12)	.30 (.07)	.30 (.08)
	$B_\gamma = 100$	.26 (.04)	.25 (.08)	.26 (.04)	.28 (.06)
B_β=10	B_γ = 0	.28 (.13)	.23 (.11)	.28 (.07)	.27 (.07)
	$B_\gamma = 1$	.27 (.12)	.23 (.11)	.28 (.07)	.27 (.07)
	$B_\gamma = 10$	.26 (.09)	.23 (.11)	.27 (.06)	.27 (.07)
	$B_\gamma = 100$	.25 (.04)	.24 (.07)	.25 (.04)	.26 (.06)
B_β=100	B_γ = 0	.24 (.09)	.23 (.09)	.26 (.06)	.24 (.05)
	$B_\gamma = 1$	.24 (.09)	.23 (.09)	.26 (.06)	.24 (.05)
	$B_\gamma = 10$	.25 (.08)	.23 (.08)	.25 (.06)	.24 (.05)
	$B_\gamma = 100$	.25 (.04)	.24 (.06)	.24 (.04)	.24 (.05)
PSA-1rep		.30 (.13)	.23 (.11)	.29 (.07)	.29 (.07)
PSA-100rep		.28 (.10)	.27 (.09)	.28 (.06)	.25 (.05)

Table 5:

The $\hat{\gamma}_{ATE}$s & $S.E.$s for Conventional and Bayesian Stratification and Matching of Design II with True γ_{ATE}=1.25 in Simulation Study 2

		$N = 100$		$N = 250$	
First Step	Second Step	Stratification	Matching	Stratification	Matching
B_β=0	B_γ = 0	1.32 (.14)	1.27 (.13)	1.32(.09)	1.31 (.09)
	$B_\gamma = 1$	1.31 (.13)	1.27 (.13)	1.32 (.09)	1.31 (.09)
	$B_\gamma = 10$	1.29 (.10)	1.27 (.12)	1.30 (.08)	1.31 (.08)
	$B_\gamma = 100$	1.26 (.04)	1.26 (.08)	1.26 (.04)	1.29 (.07)
B_β=1	B_γ = 0	1.30 (.14)	1.25 (.13)	1.31 (.09)	1.30 (.08)
	$B_\gamma = 1$	1.30 (.13)	1.25 (.13)	1.31 (.09)	1.30 (.08)
	$B_\gamma = 10$	1.28 (.10)	1.25 (.12)	1.30 (.07)	1.30 (.08)
	$B_\gamma = 100$	1.26 (.04)	1.25 (.08)	1.26 (.04)	1.28 (.06)
B_β=10	B_γ = 0	1.28 (.13)	1.23 (.11)	1.28 (.07)	1.27 (.07)
	$B_\gamma = 1$	1.27 (.12)	1.23 (.11)	1.28 (.07)	1.27 (.07)
	$B_\gamma = 10$	1.26 (.09)	1.23 (.11)	1.27 (.06)	1.27 (.07)
	$B_\gamma = 100$	1.25 (.04)	1.24 (.07)	1.25 (.04)	1.26 (.06)
B_β=100	B_γ = 0	1.24 (.09)	1.23 (.09)	1.26 (.06)	1.24 (.05)
	$B_\gamma = 1$	1.24 (.09)	1.23 (.09)	1.26 (.06)	1.24 (.05)
	$B_\gamma = 10$	1.25 (.08)	1.23 (.08)	1.25 (.06)	1.24 (.05)
	$B_\gamma = 100$	1.25 (.04)	1.24 (.06)	1.24 (.04)	1.24 (.05)
PSA-1rep		1.30 (.13)	1.23 (.11)	1.29 (.07)	1.29 (.07)
PSA-100rep		1.28 (.10)	1.27 (.09)	1.28 (.06)	1.25 (.05)

Conclusion

- To summarize, Study I reveals that greater precision in the propensity score equation yields better recovery of the frequentist-based causal effect compared to traditional PSA and compared to no adjustment.
- Study I also reveals a very small advantage to the Bayesian approach for $N = 100$ versus $N = 250$.
- Study II-A reveals that greater precision around the wrong causal effect can lead to seriously distorted results.
- Study II-B reveals that greater precision around the correct causal parameter yeilds quite good results, with slight improvement seen with greater precision in the propensity score equation.
- This study also suggests that optimal matching is preferred in terms of bias and precision.

- To conclude, we propose a simple and reasonable strategy for Bayesian propensity score analysis based on a two step approach.
- This approach preserves the basic idea that the propensity score should provide information only regarding study design and not regarding the treatment effect.
- Bayesian PSA is easy to implement and addresses the encoding of prior information in both the propensity score equation and outcomes model equation.
- Research on the elicitation of priors will be essential to further demonstrate the value of the Bayesian approach (O'Hagan et al., 2006).