Bayesian Causal Inference

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Potential Outcome Setup

- Outcome $Y_i(Z_i)$, treatment $Z_i = 0, 1$ and covariates X_i
- Each unit i, two potential outcomes $Y_i(0)$ and $Y_i(1)$

- Individual Treatment Effect (ITE): $\tau_i = Y_i(1) - Y_i(0)$
- Sample Average Treatment Effect (SATE): $\tau^S = N^{-1} \sum_{i=1}^N (Y_i(1) - Y_i(0))$
- Conditional Average Treatment Effect (CATE): $\tau(x) = E(Y_i(1) - Y_i(0) \mid X_i = x)$
- Population Average Treatment Effect (PATE): $\tau^P = E(Y_i(1) - Y_i(0)) = E(\tau(X_i)) = \int \tau(x) F_X(dx)$
- Mixed Average Treatment Effect (MATE): $\tau^M = \int \tau(x) \hat{F}_X(dx) = N^{-1} \sum_{i=1}^N \tau(X_i)$

Identification Assumption: Ignorability

- Unconfoundedness
  $\Pr(Z_i \mid Y_i(0), Y_i(1), X_i) = \Pr(Z_i \mid X_i)$
  no unmeasured confounding
- Overlap
  $0 < \Pr(Z_i \mid Y_i(0), Y_i(1), X_i) = \Pr(Z_i \mid X_i) < 1$
  Propensity score $e(X_i) \equiv \Pr(Z_i = 1 \mid X_i)$
- Outcome modeling identifiable
  $\mu_z(x) \equiv E(Y_i(z) \mid X_i = x) = E(Y_i \mid Z_i = z, X_i = x)$

Example 3.1

- Completely randomized experiment with covariates X
- $$\begin{pmatrix} Y_i(1) \\ Y_i(0) \end{pmatrix} \mid (X_i, \beta_1, \beta_0, \sigma_1^2, \sigma_0^2, \rho) \sim \mathrm{N}(\begin{pmatrix} \beta_1' X_i \\ \beta_0' X_i \end{pmatrix}, \begin{pmatrix} \sigma_1^2 & \rho\sigma_1\sigma_0 \\ \rho\sigma_1\sigma_0 & \sigma_0^2 \end{pmatrix})$$
- ITE: $\tau_i = Y_i(1) - Y_i(0)$
- SATE: $\tau^S = N^{-1} \sum_{i=1}^N (Y_i(1) - Y_i(0))$
- CATE: $\tau(x) = E(Y_i(1) - Y_i(0) \mid X = x) = (\beta_1 - \beta_0)' x$
- PATE: $\tau^P = E(Y_i(1) - Y_i(0)) = (\beta_1 - \beta_0)' \mathbb{E}(X_i)$
- MATE: $\tau^M = N^{-1} \sum_{i=1}^N \tau(X_i) = (\beta_1 - \beta_0)' \bar{X}$

Bayesian Causal Inference

- Potential outcome frame is essentially a missing data problem
- Ignorable assignment mechanism ⇔ missing at random

- $Y_i(0), Y_i(1), X_i, Z_i$ only three observed
- $Y_i^{\text{obs}} = Y_i(Z_i)$, $Y_i^{\text{miss}} = Y_i(1 - Z_i)$
- Complete likelihood given parameter $\theta = (\theta_X, \theta_Y, \theta_Z)$:

$$\Pr(\mathbf{Y}(0), \mathbf{Y}(1), \mathbf{X}, \mathbf{Z} \mid \theta) = \prod_{i=1}^{N} \Pr(Y_i(0), Y_i(1), X_i, Z_i \mid \theta)$$

$$= \prod_{i=1}^{N} \underbrace{\Pr(Z_i \mid Y_i(0), Y_i(1), X_i; \theta_Z)}_{\text{Propensity score } \Pr(Z_i|X_i;\theta_Z)} \underbrace{\Pr(Y_i(0), Y_i(1) \mid X_i; \theta_Y)}_{\text{Potential outcomes}} \underbrace{\Pr(X_i; \theta_X)}_{\text{Covariates}}$$

Prior

- (Prior Independence): The prior of $\theta_X, \theta_Y, \theta_Z$ are distinct and independent.
- Under independent priors

$$\begin{aligned} \Pr(\theta_X, \theta_Y, \theta_Z \mid \cdot) \propto \Pr(\theta_X) &\prod_{i=1}^{N} \Pr(X_i \mid \theta_X) \\ \Pr(\theta_Y) &\prod_{i=1}^{N} \Pr(Y_i(1), Y_i(0) \mid \theta_X; \theta_Y) \\ \Pr(\theta_Z) &\prod_{i=1}^{N} \Pr(Z_i \mid X_i; \theta_Z). \end{aligned}$$

- $\hat{\theta}_X^{\text{pos}}, \hat{\theta}_Y^{\text{pos}}$ does not dependent on propensity score (ignorable)

Revisit ATEs

- MATE: $\tau^M = \int \tau(x;\theta_Y)\hat{F}_X(dx)$
  A convenient approximation of PATE.
  Not depend on the association between $Y_i(1)$ and $Y_i(0)$; $\hat{\theta}_Y^{\text{pos}}$.
  $(\hat{\beta}_1^{\text{pos}} - \hat{\beta}_0^{\text{pos}})'\bar{X}$

- PATE: $\tau^P = E(Y_i(1) - Y_i(0)) = E[E(Y_i(1) - Y_i(0) \mid X)] = E[E(Y_i(1) \mid X) - E(Y_i(0) \mid X)] = \int \tau(x;\theta_Y)F(dx;\theta_X)$
  Function of distribution of potential outcomes in a population;
  Not depend on the association between $Y_i(1)$ and $Y_i(0)$;
  need $\hat{\theta}_X^{\text{pos}}, \hat{\theta}_Y^{\text{pos}}$.
  Need additional model on $\Pr(X_i \mid \theta_X)$ or draw X from a Bayesian bootstrap (Rubin, 1985) $(\hat{\beta}_1^{\text{pos}} - \hat{\beta}_0^{\text{pos}})'\mathbb{E}(X_i)$

Posterior inference of ATEs

- SATE: $\tau^S = N^{-1} \sum_{i=1}^{N} (Y_i(1) - Y_i(0))$
  Average of ITE in finite samples;
  draw Y^{miss} from posterior inference;
  depend on the association between $Y_i(1)$ and $Y_i(0)$.
  Besides θ_Y, we need to impute $\mathbf{Y}^{\text{miss}}$.

$$\Pr(\mathbf{Y}^{\text{miss}} \mid \mathbf{Y}^{\text{obs}}, \mathbf{Z}, \mathbf{X}; \theta_Y) \propto \prod_{i:Z_i=1} \Pr(Y_i(0) \mid Y_i(1), X_i; \theta_Y) \prod_{i:Z_i=0} \Pr(Y_i(1) \mid Y_i(0), X_i; \theta_Y)$$

Outcome model

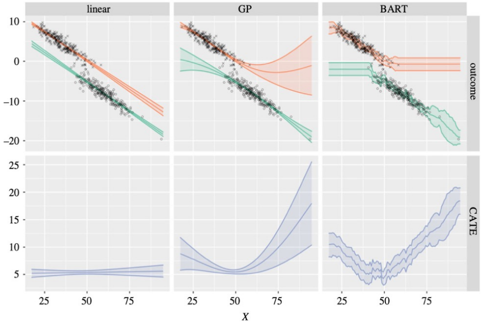
- Specify the outcome model $\mu_z(x) = \mu(x, z)$
- Two general categories:
  1. S(ingle) learner: $\mu_Z(x) = x + z + xz$
  2. T(wo) learner: two separate model for each group $\mu_1(x)$ and $\mu_2(x)$
- Popular Bayesian choice: BART (Hill 2011), Bayesian random forest (Hahn et al 2020), GP (Ray & van der Vaart 2020), DP mixture (Karabatsos G., & Walker S. G. 2012)

High dimensional

- When the two groups are poorly overlapped, and not all priors can adaptively capture the uncertainty according to the degree of overlap.
- Standard Bayesian non-parametric priors inadequate

Example 4.1

- $X_i \mid Z_i = 1 \sim \text{Ga}(\text{mean} = 35, \text{sd} = 8)$, 250 units,
  $X_i \mid Z_i = 0 \sim \text{Ga}(\text{mean} = 60, \text{sd} = 8)$, 250 units,
  $Y_i(z) = 10 + 5z - 0.3X_i + \epsilon_i$, $\epsilon_i \sim \text{N}(0, 1)$, CATE$\tau(x) = 5$
- Bayesian outcome model $\mu_z(x) = f_z(x) + \epsilon_i, \epsilon_i \sim \text{N}(0, \sigma^2)$
- (i) LM: $f_z(x) = \alpha_z + \beta_z x$
  (ii) GP: $(f_z(x_1), \dots, f_z(x_N))' \sim \text{N}(0, \Sigma)$, exponential
  (iii) BART: $\mu_z(x) = \sum_{t=1}^{T} g(z, x; \text{Tree}_t, M_t)$



High dimensional

- Traditional sparsity-inducing priors will act as an informative prior

Paradoxical Role of PS in Bayesian Causal Inference

- Under the Frequentist domain, Propensity Score (PS) plays a central role in causal inference. However, under ignorability and prior independence, Bayesian inference of causal effects does not depend on PS.
- Bayesian causal inference is based on the outcome model, which dose not account for overlapping and balance.
- The result is that Bayesian causal inference is sensitive to the outcome model specification, and may fail to quantify uncertaincities accordingly.
- PS is essential in ensuring overlap and balance. Hence, consider it in either the design stage or the analysis stage increases the robustness of Bayesian causal inference.
- Focus on the analysis stage: directly incorporating PS into the outcome model.

Approach 1: PS as an additional covariate

- Use PS as an additional covariate in the outcome model $\mu(x, z) = \mu(x, z, e(x))$.

- Two-stage implementation: (i) estimate PS $\hat{e}_i$; (ii) plug in $\hat{e}_i$ into the outcome model.

- A Bayesian analogue of doubly-robust: (i) if $\mu(x)$ is correctly specified, $e(x)$ is redundant; (ii) if $\mu(x)$ is misspecified, results are less sensitive to model (because the covariates are balanced within a value of PS).

- Controversies: (i) not dogmatically Bayesian; (ii) Why dose true outcome generating mechanism depend on the assignment mechanism (PS)?

Approach 2: Dependent priors

- Replacing the independent prior assumption: specify priors of outcome model that are dependent on PS.

- Examples:
  - Wang et al. (2012): dependent prior for simultaneous variable selection in the PS and outcome models. Assume logistic PS model with coefficients $\boldsymbol{\alpha}$, and linear outcome model with coefficients $\boldsymbol{\beta}$. Putting a spike-and-slab prior on $\boldsymbol{\alpha}$ and $\boldsymbol{\beta}$, with latent indicators $\boldsymbol{\gamma}^{\alpha}$ and $\boldsymbol{\gamma}^{\beta}$. Make them dependent a priori.

  - Little (2004): include PS in the outcome model through the conditional variance. Assume $Y_i(1)|X_i \sim N(\mu_1, \sigma_1^2 e(X_i))$ and $Y_i(0)|X_i \sim N(\mu_0, \sigma_0^2 e(X_i))$, with flat priors on μ_1 and μ_0. The PATE in that case is closely related to the IPW estimator.

- Limitations: specification of such prior is case-dependent, no general solution.

Approach 3: Posterior predictive estimation

- Motivated from double-robust estimation.
- Procedure:
  1. Specify a separate Bayesian PS model and outcome model;
  2. Draw PS $\hat{e}_i$ and missing potential outcomes $\hat{Y}_i^{\text{mis}}$ from their respective posterior predictive distributions;
  3. Plug these into the double-robust estimator of ATE.
- Advantage: easy to implement, flexible choice of models, proper uncertainty quantification.
- Problem (conceptual): not dogmatically Bayesian.

Sensitivity analysis in observational studies

- *Sensitivity analysis*: assessing the sensitivity of the results w.r.t. unmeasured confoudning in an observational study.

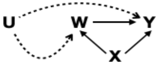
- Performing sensitivity analysis: (i) obtain the result over a plausible range of values of the sensitivity parameters. (ii) derive theoretical threshold for the sensitivity parameters that would explain away the observed treatment-outcome association.

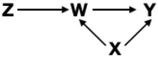
- Methods have been proposed, characterized by the specific parameterization of confounding: (i) involve unmeasured confounders; (ii) involve distributions of potential outcomes (because unconfoundedness means $\Pr(Y(z)|Z = 1, X) = \Pr(Y(z)|Z = 0, X)$, for $z = 0, 1$).

- Criticism: to assess untestable unconfoundedness, one make even more untestable assumptions.

Instrumental variable (IV)

- IVs are used when we have unmeasured confoundings.





- IVs affects the outcome only through its effect on the treatment (assignment).
- IVs are usually the variable that is believed to be randomized in nature, or can be thought of as a randomized encouragement to receive treatment.
- Given a valid IV, we can extract the causal effects of a treatment by a two-stage least-squares (2SLS) estimator.
- Link IV to potential outcome (Angrist et al. (1996)): randomized trials with binary treatment and noncompliance.
- The treatment assignment (Z) is used as the IV.

Randomized trials with noncompliance

- For a given subject, it is assigned treatment Z, and received treatment W.
- Potential treatment, $\mathcal{U} = (W_i(1), W_i(0))$, lead to principal strata, always-takers (at), compliers (co), defiers (df), and never-takers (nt).
- Principal causal effect $\tau_u = \mathrm{E}[Y_i(1) - Y_i(0)|\mathcal{U}_i = u]$ lead to intention-to-treat effect $\mathrm{E}[Y_i(1) - Y_i(0)] = \sum_u \Pr(\mathcal{U}_i = u)\tau_u$, which is nonidentifiable. Global
- The compiler average causal effect $\tau_{co} = \mathrm{E}[Y(1) - Y(0)|\mathcal{U}_i = co]$ is identifiable. Also for compliers, intention-to-treat effect is the same as treatment-received effect. Local
- Intuition: with unmeasured confounding, there is no hope for estimating a global causal effect. With reasonable assumptions, local is good enough.

Bayesian inference of the IV set-up: an example

- Quantities associated with each unit are $\{Y_i(1), Y_i(0), W_i(1), W_i(0), Z_i\}$, three observed $\{Y_i(Z_i), W_i(Z_i), Z_i\}$, and two missing $\{Y_i(1-Z_i), W_i(1-Z_i)\}$. (no covariates)
- Consider binary outcome, and control units have no access to the treatment, $W_i(0) = 0, \forall i$, resulting in only co and nt.
- The joint model $P(\boldsymbol{\theta}) \prod_{i=1}^{N} P(Y_i(0), Y_i(1)|\mathcal{U}_i; \theta_Y) P(\mathcal{U}_i|\theta_{\mathcal{U}})$.
- Outcome model: $Y_i(z)|\mathcal{U}_i = co \sim Bern(p_{co,z})$, and $Y_i(z)|\mathcal{U}_i = nt \sim Bern(p_{nt})$, for $z = 1, 2$. Compliance type model: $Pr(\mathcal{U}_i = co) = \pi_{co}$, $Pr(\mathcal{U}_i = nt) = 1 - \pi_{co}$. Assume conjugate prior for hyperparameters.
- Causal effect $\tau_{co} = p_{co,1} - p_{co,0}$.
- If $Z_i = 0$, $W_i = 0$, $\mathcal{U}_i$ is not observed. Impute based on $\pi_{co} \cdot p_{co,0}^{Y_i}(1 - p_{co,0})^{1-Y_i}$ and $\pi_{nt} \cdot p_{nt}^{Y_i}(1 - p_{nt})^{1-Y_i}$.

Time-varying treatment and confounding: setup

- Suppose treatments are assigned at T time points. Let Z_{it} denote the treatment at time t for unit i, and $\bar{Z}_{it} = (Z_{i1}, \cdots, Z_{iT})$ denote the observed sequence of treatment.
- Let L_i0 denote the time-invariant covariates, $L_{i,t-1}$ denote the time-varying confounders, and $\bar{L}_{it} = (L_{i1}, \cdots, L_{iT})$.
- Causal estimand: $\tau_{\bar{z}_T, \bar{z}'_T} = \mathrm{E}[Y_i(\bar{z}_T) - Y_i(\bar{z}'_T)]$.
- The central question: role of the time-varying confounders L_t in the assignment mechanism.
- Sequentially ignorable assignment mechanism: $\Pr(Z_t | \bar{Z}_{t-1}, \bar{L}_{t-1}, Y(\bar{z}_t) \forall \bar{z}_t) = \Pr(Z_t | \bar{Z}_{t-1}, \bar{L}_{t-1})$, for $t = 1, \cdots, T$.
- A full Bayesian approach would specify a joint model for Z_t, L_t at all time points and $Y(\bar{Z}_T)$. It becomes intractable quickly.

Time-varying treatment and confounding: an example

- Consider $N = 5000$ subjects, with two time points ($t = 1, 2$), and also binary treatment ($Z_{it} = 0, 1$), binary covariate ($X_{it} = 0, 1$), binary final outcome ($Y_i = 0, 1$).
- Each individual's data is simulated from the following model

$$X_1 \sim Bern(0.25), \quad Z_1 \sim Bern(0.05 + \kappa_1 X_1)$$

$$X_2 \sim \begin{cases} Bern(0.25 - \kappa_2 Z_1) & X_1 = 0 \\ Bern(0.95) & X_1 = 1 \end{cases}$$

$$Z_2 \sim \begin{cases} Bern(0.1 + \kappa_1 X_2) & Z_1 = 0 \\ Bern(0.9) & Z_1 = 1 \end{cases}$$

$$Y \sim MaxBern(0.1 - \kappa_3 Z_1 + \kappa_4 X_1, 0.2 - \kappa_3 Z_2 + \kappa_4 X_2)$$

where $MaxBern(a, b)$ is the maximum of independent $Bern(a)$ and $Bern(b)$ random variables.

- Hyperparameters $\kappa_1 = 0.25$, $\kappa_2 = 0.075$, $\kappa_3 = 0.03$, $\kappa_4 = 0.15$.

Bayesian inference approach 1: Bayesian g-formula (BGF)

- The g-formula approach probabilistically express the time-evolution of all variables.
- For this specific example, we care about

$$\theta_{\zeta\zeta'} = E(Y(Z_{\zeta\zeta'})) = \sum_{x_1=0}^{1}\sum_{x_2=0}^{1}[Pr(X_1 = x_1)Pr(X_2 = x_2|X_1 = x_1, Z_1 = \zeta)Pr\{Y = 1|X = (x_1, x_2), Z = (\zeta, \zeta')\}]$$

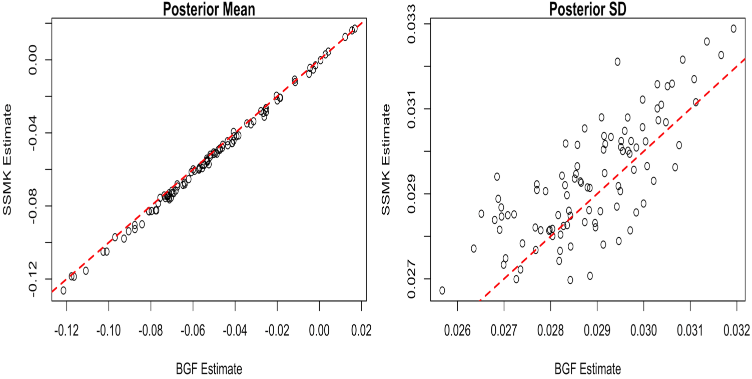
- Bayesian saturated binary regression (BSAT) models are fitted for (X_1), $(X_2|X_1, Z_1)$ and $(Y|X_1, Z_1, X_2, Z_2)$.
- BSAT: for a binary response A and binary covariates $\mathbf{B} \in \mathbb{R}^p$, model $\Pr(A = 1|\mathbf{B} = \mathbf{b}) = \lambda_{\mathbf{b}}$ for all 2^p possible values of $\mathbf{b}$, with prior $\lambda_{\mathbf{b}} \overset{i.i.d.}{\sim} Unif(0, 1)$.
- Obtain posterior inference of functions involving $\theta_{\zeta\zeta'}$ is simple based on simulated posterior samples.

Bayesian inference approach 2: Saarela et al. (2015)

- Saarela et al. devised a Bayesian version of the marginal structural model via the Bayesian bootstrap (referred as SSMK).
- SSMK can be viewed as a generalization of IPW in time-varying treatment settings. The key is to estimate the propensity scores and ensure overlap at each time point.
- The procedure (specific to the example) is:
  1. Fit BSAT for (Z_1), $(Z_2|Z_1)$, $(Z_1|X_1)$ and $(Z_2|X_1, Z_1, X_2)$, and obtain L posterior samples of model parameters $\phi^{(\ell)}$.
  2. Calculate the estimate weight $\hat{\omega}_i = \sum_L P(Z_{i1}; \phi^{(\ell)}) P(Z_{i2}|Z_{i1}; \phi^{(\ell)}) / \, [\sum_L P(Z_{i1}|X_{i1}; \phi^{(\ell)}) P(Z_{i2}|X_{i1}, Z_{i1}, X_{i2}; \phi^{(\ell)})]$.
  3. Obtain posterior samples of $\theta^{(\ell)}_{\zeta\zeta'}$ by calculating the IPW estimator with weight factor $\pi_i^{(\ell)}$ for each subject drawing from a Dirichlet distribution.
- Full posterior inference based on posterior samples of $\theta_{\zeta\zeta'}$.

Result

- We obtain posterior mean and standrad deviation of $\theta_{11} - \theta_{00}$, using both BGF and SSMK.
- Perform the inference on 100 simulated data set to provide the following plots for comparison.



Why (and When) Bayesian?

- Usual arguments: uncertainty quantification, not rely on large sample asymptotics.
- Specific to causal inference:
  - Impute all missing potential outcomes, thus allows straightforward inference of any causal estimand;
  - Automatic uncertainty quantification of any estimands; can combine with decision theory for dynamic decision making;
  - Particularly suitable for complex settings: post-treatment confounding, sequential treatments, spatial and temporal data;
  - Advanced Bayesian models and methods bring new tools: Bayesian nonparametrics, spatial-temporal models, Bayesian variable selection $\cdots$

Final words

- The fundamental problem of causal inference: $Y_i(0)$ and $Y_i(1)$ can never be jointly observed. In other words, the observed data provides no information about their pair-wised correlation ρ.
- Frequentist method try to establish "balance", and causal effects is identifiable on balanced design. Their identifiability is all-or-nothing.
- Bayesian puts a prior model on ρ, hence the identifiability is a continuum between weak to strong identification.
- Lack of overlap, little learning of ρ from data. Result will be sensitive to the choice of priors and the outcome model. Enough overlap, some hope to learn ρ from data and obtain robust result.
- Proper Bayesian causal inference must take into account assignment mechanism or propensity scoire in either the deisgn or the analysis stage.

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Thanks!