A Joint Bayesian Framework for Causal Inference and Bipartite Matching for Record Linkage

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Abstract

The recent proliferation in the use of digital health data has opened enormous possibilities for researchers to gather information on a common set of entities from various government and non-government sources and make causal inferences about important health outcomes. In such scenarios, the response of interest may be obtained from a source different than the one from which the treatment assignment and covariates are obtained. In absence of error free direct identifiers (e.g., social security numbers), straightforward merging of separate files based on these identifiers is not feasible, giving rise to the need for matching on imperfect linking variables (e.g., names, birth years). Causal inference in such situations generally follows using a two-stage procedure, wherein the first stage involves linking two files using a probabilistic linkage technique with the imperfect linking variables common to both files, followed by causal inference on the linked dataset in the second stage. Rather than sequentially performing record linkage and causal inference, this article proposes a novel framework for simultaneous Bayesian inference on probabilistic linkage and the causal effect. In contrast with the two-stage approach, our proposed methodology facilitates borrowing of information between the models employed for causal inference and record linkage, thus improving accuracy of inference in both models. Importantly, the joint modeling framework offers characterization of uncertainty, both in causal inference and in
record linkage. An efficient computational template using Markov chain Monte Carlo (MCMC) is developed for the joint model. Simulation studies and real data analysis provide evidence of both improved accuracy in estimates of treatment effects, as well as more accurate linking of two files in the joint modeling framework over the two-stage modeling option. The conclusion is further buttressed by theoretical insights presented in this article.

Keywords: Causal Inference, Record Linkage.

1 Introduction

In recent times, the burgeoning presence of digital data platforms has opened new possibilities for health researchers to link patient data from multiple sources, such as electronic health records and Medicare claims data, in order to answer important causal questions. This linking is useful because it allows researchers to make inferences about outcomes not measured in planned studies, and long-term outcomes, without having to incur the substantial costs to collect new primary data. When perfectly measured unique identifiers like social security numbers or Medicare patient ids are available in all files, it is reasonably straightforward to link multiple files and match individuals (based on these identifiers) in order to carry out causal estimation. However, these direct identifiers may often be missing from one or more files, or not be made available due to patient privacy restrictions. In such situations, data files have to be linked based on indirect identifiers, such as patient initials, birth dates, zip codes, diagnosis codes and dates of encounter, which are inherently imperfect. This can cause erroneous links, which in turn can lead to fallacious causal inference. Even when direct personal identifiers are available for linkage, they might often be impacted by various data quality issues, such as missing data and typographical errors. Such challenges have propelled a large literature on record linkage in the fields of statistics and data mining (Larsen and Rubin, 2001; Raghunathan et al., 2003; Reiter, 2005; Singla and Domingos, 2006).

This article specifically considers bipartite record linkage, where two datafiles are merged assuming that there is no duplication within each file. A corollary to this assumption comes
in the form of a maximum one-to-one restriction in the linkage, i.e., a record in one file can be linked with a maximum of one record in the other file. Although a substantial part of the statistical literature on record linkage deals with this scenario (Fellegi and Sunter, 1969; Jaro, 1989; Winkler, 1993; Belin and Rubin, 1995; Larsen and Rubin, 2001; Herzog et al., 2007), the common practice is to enforce one-to-one linkage as a post-processing step. Perhaps a more desirable idea is to incorporate this bipartite matching constraint into the model (Fortini et al., 2002; Tancredi and Liseo, 2011; Larsen, 2010; Gutman et al., 2013; Sadinle, 2017), rather than forcing it in the post-processing step. We will subsequently adopt the model based approach in developing our framework.

Historically, record linkage and causal inference have been carried out as a two-stage process, where records are first linked using a probabilistic record linkage model fitted on indirect identifiers, not taking into account any available information on the outcome, covariate or treatment status. Subsequently, inference on the causal effect follows on the linked records. Since estimation of the causal effect is affected by incorrect record linkage, it may turn out to be beneficial to iteratively seek feedback from the causal model to improve upon the linkage procedure.

In a related problem of fitting a generalized linear model (GLM) with the response and predictors lying in different files with no unique identifiers, Solomon and O’Brien (2019) develop a new framework for linking records with a goal to maximize the probability that the point estimate of the parameter of interest in the GLM will have the correct sign and that the confidence interval around this estimate will correctly exclude the null value of zero. They further show that maximizing the asymptotic power of testing the null hypothesis of no predictor effect in the regression requires declaring all pairs with greater than 50% probability of being a true match to be links. A more direct approach to simultaneous record linkage and regression analysis using a Bayesian hierarchical model is proposed in Dalzell and Reiter (2018).

In the context of simultaneous causal inference and record linkage, the first (and arguably the only till date) effort has come from Wortman and Reiter (2018), where in the first step,
record pairs identified as links by the record linkage procedure are ordered using some linking scores, with a subset of these pairs treated as certain links and the rest deemed as uncertain links. Starting with the set of certain links, the method sequentially concatenates new links from the set of uncertain links to estimate the treatment effect, each time computing some criterion intended to increase when adding inexact matches. Finally, the set of links minimizing this criterion is used to estimate the final treatment effect. Their minimum estimated variance (MEV) criterion selects the set of links that minimizes the estimated variance of the estimated average treatment effect (ATE). Alternatively, their estimate-tethered stopping rule (ETSR) chooses the threshold that minimizes the estimated variance of the ATE subject to the constraint that the ATE must be within $k$ standard errors of the ATE estimate obtained when the analysis is restricted to pairs of records for which the link is known to be correct. This approach has introduced the concept of drawing feedback between a causal model and a record linkage procedure to improve the estimation of causal effects, providing point estimates of the causal effect (and its variance), based on the final set of ascertained links. Nevertheless, there is still a requirement for methods that can provide uncertainty quantification in both the causal model and the probabilistic model for record linkage.

This article proposes a direct way to embed the causal inference and bipartite record linkage simultaneously within a novel Bayesian hierarchical framework. In particular, our proposed model posits that the vector of similarity measures comparing linking variables appearing in both files and the outcome variable for causal inference jointly follow a two component mixture model. The two components of the mixture correspond to the joint distributions for matches and non-matches. The Bayesian implementation of the hierarchical model generates multiple imputations of the linked files and corresponding causal effects. By implementing record linkage and causal inference simultaneously, this approach provides researchers with tools to account for uncertainty in the causal effect as well as in the probabilistic linking of records in two files, thereby improving the validity of causal conclusions. Further, the framework allows the causal model and the record linkage model to take advan-
tage of shared information across both files, which improves the accuracy of causal inference, along with the accuracy of linking between files. In fact, the article provides theoretical insight to show that joint modeling is more prone to identifying correct links compared to the two-stage model which executes record linkage and causal inference sequentially. Additionally, the joint modeling approach allows more robustness to mis-specification of the causal model, as illustrated in the simulation studies. For a comprehensive study, we carry out our investigations with both parametric and non-parametric causal models and also extend our joint modeling framework to accommodate scenarios with missing outcomes and correlated covariates.

The remainder of the article proceeds as follows. Section 2 discusses the background, notations and the formulation of the joint modeling framework. Section 2 also discusses theoretical results arguing better inference on record linkage from joint model compared to the two-stage model. Section 3 describes posterior computation of the proposed model. In Section 4, we provide results from the simulation studies used to assess the effectiveness of the method vis-a-vis competitors.

2 Model and Prior Formulation

We begin by defining a few key concepts and assumptions related to causal inference in Section 2.1. We then describe probabilistic record linkage with one-one bipartite matching in Section 2.2, followed by proposing our joint modeling approach with simultaneous bipartite record linkage and causal inference in Section 2.3.

2.1 Some Background and Notation for Causal Inference

We assume a binary treatment, and let $w_i \in \{0, 1\}$ be a binary indicator, with $w_i = 1$ and $w_i = 0$ indicating treatment and control assignment to individual $i$, respectively. Let $x_i$ be the $p \times 1$ covariate for individual $i$. Let $y_i$ be a continuous outcome, for example, an index of patient health. Each unit or observation is assumed to have two potential outcomes (Rubin,
1974), one under each value of the treatment. We denote $y_i(1)$ and $y_i(0)$ as the potential outcomes for individual $i$ when $w_i = 1$ or $w_i = 0$, respectively. The treatment effect for the $i$th individual is given by $T_i = y_i(1) - y_i(0)$. Note that we observe only one of $y_i(1)$ and $y_i(0)$ in reality, and define $y_i = w_i y_i(1) + (1 - w_i) y_i(0)$ as the observed outcome for the $i$th individual.

Additionally, we make the following assumptions in our causal framework:

1. **Stable unit treatment value assumption (SUTVA):** The SUTVA contains two sub-assumptions, no interference between units (i.e., the treatment applied to one unit does not affect the outcome for another unit) and no different versions of a treatment (Rubin, 1974).

2. **Strong ignorability:** Strong ignorability stipulates that (a) $(y_i(0), y_i(1)) \perp w_i | x_i$ for all $i$, which means that there is no confounded effect in assigning the treatment and, (b) $0 < P(w_i = 1 | x_i) < 1$, i.e., there is a positive probability of assigning treatment to every unit.

We will also utilize the *propensity score* for a unit, defined as $e(x_i) = P(w_i = 1 | x_i)$, i.e., the probability of being assigned a treatment given the covariate $x_i$. As postulated by Rosenbaum and Rubin (1983), the treatment assignment is independent of $x_i$ given $e(x_i)$ under the aforesaid assumptions. Throughout, $I(E)$ represents the indicator for an event $E$.

### 2.2 Record Linkage with One-One Bipartite Matching

Consider the scenario where we seek to link two files, File A containing the response or outcome, and File B containing the predictors and the treatment status for subjects, using imperfect linking variables. File A and File B are comprised of $n_A$ and $n_B$ records, respectively, and without loss of generality we assume that $n_A \geq n_B$. Suppose each individual or entity is recorded at most once in each datafile, i.e., the datafiles contain no duplicates. This article focuses on *bipartite record linkage*, which means that a record in one file can
be linked (also interchangeably referred to as *matched*) to a maximum of one record in the other file. Note that bipartite record linkage with the assumption of no duplication within a datafile implies that there is no common record between any two linked pairs. Thus, under this setting, the idea of record linkage is to identify which records in File A and B refer to the same subject.

Following Sadinle (2017), we introduce *labelling* $z = (z_1, ..., z_{n_B})'$ for the records in File B to encode a particular matching status between the two files. Specifically, $z_j$ is defined as

$$z_j = \begin{cases} 
  i, & \text{if record } i \text{ in File A and record } j \text{ in File B is a match} \\
  n_A + j, & \text{if record } j \text{ in File B has no match in File A}
\end{cases}$$

In the context of bipartite matching, one enforces $z_j \neq z_{j'}$, when $j \neq j'$.

In probabilistic record linkage applications, two records that refer to the same entity should be very similar, otherwise the amount of error in the datafiles may be too large for the record linkage task to be feasible. On the other hand, two records that refer to different entities should generally be very different. Suppose we have $F$ imperfect linking variables (also referred to as *fields* in the record linkage parlance). For each pair of records $(i, j)$ in File A×File B, we define a $F$-dimensional vector of observations $\gamma_{ij} = (\gamma_{1,ij}, ..., \gamma_{F,ij})'$, where $\gamma_{f,ij}$ is the score reflecting the similarity in the field $f$ for the record pair. In this article we set the comparison score $\gamma_{f,ij}$ as 0 or 1, depending on whether the records $i$ and $j$ are identical or within an acceptable tolerance level in field $f$. To be more precise, for unstructured nominal information (e.g., age, birth year, gender) it is straightforward to compare the two records, and set $\gamma_{f,ij}$ equal to 1 or 0 according to whether the values are equal in the two files or not. To take into account partial agreement among string fields (e.g., names), we calculate the normalized Levenshtein Similarity measure between two strings (Winkler, 1990). This metric ranges between 0 (no agreement) and 1 (full agreement) and is obtained using the “levenshteinSim” function in the RecordLinkage package in R. This metric can be converted into a binary variable $\gamma_{f,ij}$ by setting $\gamma_{f,ij}$ as 1 or 0 depending on whether the distance metric exceeds a predetermined threshold (for e.g., 0.95) or not.
Following Fellegi and Sunter (1969) and related literature, we assume that \( \gamma_{ij} \) is a random realization from a mixture of two distributions, one for true links and the other for nonlinks, i.e.,

\[
\gamma_{ij} | (z_j = i) \overset{iid}{\sim} g(\theta_m), \quad \gamma_{ij} | (z_j \neq i) \overset{iid}{\sim} g(\theta_u),
\]

(1)

where \( \theta_m = (\theta_{1,m}, \ldots, \theta_{F,m})' \) and \( \theta_u = (\theta_{1,u}, \ldots, \theta_{F,u})' \) are parameters specific to each mixture component. Setting aside more complex log-linear models for \( g(\cdot) \) (Larsen and Rubin, 2001) for any future exploration, we posit conditional independence across fields to compute,

\[
g(\theta_m) = P(\gamma_{ij} | z_j = i) = \prod_{f=1}^{F} P(\gamma_{f,ij} | z_j = i) = \prod_{f=1}^{F} \theta_{f,m}^{\gamma_{f,ij}} (1 - \theta_{f,m})^{1 - \gamma_{f,ij}}
\]

\[
g(\theta_u) = P(\gamma_{ij} | z_j \neq i) = \prod_{f=1}^{F} P(\gamma_{f,ij} | z_j \neq i) = \prod_{f=1}^{F} \theta_{f,u}^{\gamma_{f,ij}} (1 - \theta_{f,u})^{1 - \gamma_{f,ij}}
\]

(2)

To propose a prior distribution on the \( z_j \)’s with the constraint \( z_j \neq z_{j'} \) for any \( j \neq j' \), we follow a construct used in the bipartite record linkage literature, including Fortini et al., 2002, Larsen, 2010 and Sadinle, 2017. Specifically, let \( I(z_j \leq n_A) \sim Ber(\pi) \), where \( \pi \) represents the proportion of matches expected a-priori as a fraction of the smallest file. We assume \( \pi \) to be distributed according to a Beta(\( \alpha_\pi, \beta_\pi \)) a-priori. Marginalizing over \( \pi \), the total number of matches between Files A and B, given by \( n_{AB}(z) = \sum_{j=1}^{n_B} I(z_j \leq n_A) \), is distributed according to a Beta-binomial \( (n_B, \alpha_\pi, \beta_\pi) \) distribution. Conditioning on the knowledge of which records in File B have a match, all possible bipartite matchings are taken as equally likely. The final form of the prior distribution of \( z \), marginalizing over \( \pi \), is given by

\[
P(z | a_\pi, b_\pi) = \frac{(n_A - n_{AB}(z))!}{n_A!} \frac{B(n_{AB}(z) + \alpha_\pi, n_B - n_{AB}(z) + \beta_\pi)}{B(\alpha_\pi, \beta_\pi)}.
\]

(3)

The choice of the hyper-parameters \( \alpha_\pi \) and \( \beta_\pi \) provides prior information on the number of overlaps between the two files. We discuss the specific choices of \( \alpha_\pi \) and \( \beta_\pi \) for model fitting in the posterior computation section. Finally, the parameters \( \theta_{f,m} \) and \( \theta_{f,u} \) follow i.i.d
Beta\((a, b)\) distribution for all \(f = 1, \ldots, F\).

### 2.3 A Joint Modeling Framework for Causal Inference and Record Linkage

In our endeavor to develop a joint model for causal inference and record linkage, we specify the distribution of an outcome in File A depending on whether it is linked to any covariate and treatment in File B or not. Given that the \(i\)th record in the outcome file (File A) is matched with the \(j\)th record in the covariate and treatment status file (File B), we specify the conditional distribution of \(y_i|\left(x_j, w_j\right)\) by a causal model of our choice. When the \(i\)th record in File A finds no match with any record in File B, a probabilistic model is specified for the marginal distribution of \(y_i\). More specifically, the contribution of the likelihood function from the \(i\)th record in File A is

\[
\prod \left\{ \frac{f_1(y_i|x_j, w_j, \theta_c)}{\prod_{f=1}^F \theta_{f,m} \gamma_{f,ij} (1 - \theta_{f,m})^{1 - \gamma_{f,ij}}} \right\}_I(z_j = i) \times \left\{ \frac{f_2(y_i|\theta_d)}{\prod_{f=1}^F \theta_{f,u} \gamma_{f,ij} (1 - \theta_{f,u})^{1 - \gamma_{f,ij}}} \right\}_I(z_j \neq i)
\]

\(I(z_j \neq z_j', \text{ whenever } j \neq j')\). 

(4)

Carrying out causal inference and record linkage jointly can potentially lead to a number of advantages. First, the simultaneous Bayesian exercise of record linkage and causal inference can provide researchers with tools to account for uncertainty in the causal effect as well
as in the probabilistic linking of the records in two files, thereby improving the validity of causal conclusions. Furthermore, this framework allows sharing of information amongst the causal model and the record linkage model, which can significantly improve the accuracy of causal inference along with the accuracy of linking between files. Additionally, the joint modeling approach may be more robust in presence of mis-specification of the causal model or correlated covariates. These conjectures will be examined in the simulation studies to assess the potential advantages of adopting a joint approach.

To theoretically observe the benefit of joint modeling over the two-stage modeling in terms of linking the two files, we resort to the classical Felligi-Sunter approach and compare the likelihood ratio of recognizing a pair of records as matched vs. unmatched for both these modeling approaches. To elaborate, consider the task of matching record \( i \in \{1, \ldots, n_A\} \) in File A with the record \( j \in \{1, \ldots, n_B\} \) in File B. Under the joint model, the likelihood ratio of \( i \sim j \) (i.e., record \( i \) is linked to record \( j \)) and \( i \not\sim j \) is given by

\[
\text{Ratio}_{\text{Joint}} = \frac{L(\theta_d, \theta_e, \theta_m, \theta_u, z|\{\gamma_{ij}: 1 \leq i \leq n_A, 1 \leq j \leq n_B\}, y, w, X, i \sim j)}{L(\theta_d, \theta_e, \theta_m, \theta_u, z|\{\gamma_{ij}: 1 \leq i \leq n_A, 1 \leq j \leq n_B\}, y, w, X, i \not\sim j)}
\] (5)

Notably, under the joint model, the likelihood ratio for linking records \( i \) and \( j \) does depend on the likelihood from the causal model. In contrast, the likelihood ratio for linking records in the traditional two-stage model (in which records are linked first, and causal inference is carried out on the records linked in the initial step, i.e., the exercise is sequential as opposed to joint) only involves the likelihood from the assumed probabilistic record linkage model. To be more precise, the likelihood ratio of \( i \sim j \) vs. \( i \not\sim j \) under the two-stage model is given by

\[
\text{Ratio}_{\text{2Stage}} = \prod_{f=1}^{F} \left( \frac{\theta_{f,m}}{\theta_{f,u}} \right)^{\gamma_{f,ij}} \left( \frac{1 - \theta_{f,m}}{1 - \theta_{f,u}} \right)^{1-\gamma_{f,ij}}
\] (6)

The following theorem provides an insight into the behavior of \( \text{Ratio}_{\text{Joint}} \) and \( \text{Ratio}_{\text{2Stage}} \).

**Theorem 2.1** Assuming \( f_1(y|\theta_1, \theta_d) \) and \( f_2(y|\theta_d) \) is bounded away from 0 and \( \infty \) in their supports, we have
(a) $E_{i \sim j}[{\text{Ratio}}_{\text{Joint}}] \geq E_{i \sim j}[{\text{Ratio}}_{\text{2Stage}}]$

(b) $E_{i \not\sim j}[{\text{Ratio}}_{\text{Joint}}] \leq E_{i \not\sim j}[{\text{Ratio}}_{\text{2Stage}}]$.

The theorem indicates that the likelihood ratio of the joint model is more extreme than the two stage model, which facilitates more accurate identification of link or no link between records $i$ and $j$. The proof of the theorem is provided in Appendix B.

We assume $y_i \in \mathbb{R}$ and $f_2(y_i|\theta_d) = N(y_i|\mu_1,\sigma_1^2)$ for simplicity, i.e., $\theta_d = (\mu_1,\sigma_1^2)'$, though setting either a more complicated distributional form for $f_2$ or extending our approach to a categorical $y_i$ is relatively straightforward. The conditional density $f_1(y_i|w_j, x_j, \theta_c)$ is defined through the choice of the causal model, which we elaborate on in the next section. Finally, in the context of causal inference, our eventual goal is to draw posterior inference on the causal estimand known as the Average Treatment Effect (ATE), defined as

$$\text{ATE} = \frac{\sum_{i \in A} (y_i(1) - y_i(0))}{\#A} = \frac{\sum_{i \in A} T_i}{\#A},$$

(7)

where $A = \{i : z_j = i, \text{for some } j\}$ and $\#A$ denotes the cardinality of $A$.

### 2.3.1 Causal Model

When the record pair $(i, j)$ is a match, we assume that the causal model for the outcome is represented in the following general mean-zero additive error representations form:

$$y_i = m(x_j, w_j) + \epsilon_{i,j}, \quad \epsilon_{i,j} \sim N(0, \sigma^2).$$

(8)

To have an explicit control over how $m(\cdot, \cdot)$ varies with the treatment status $w$, we propose a sufficiently expressive yet well interpretable structure on $m(\cdot, \cdot)$. To elaborate, we assume

$$m(x_j, w_j) = m_1(x_j) + m_2(x_j)w_j.$$ 

(9)

Equations (8) and (9) together propose a causal model in $w$ with covariate-dependent functions for the slope and the intercept. In this article, we investigate both parametric and non-
parametric choices of functions $m_1(\cdot)$ and $m_2(\cdot)$. For both the parametric and nonparametric approaches, $m_1(\cdot)$ and $m_2(\cdot)$ assume the form, $m_1(x_j) = m_1(\hat{e}(x_j))$ and $m_2(x_j) = m_2(\hat{e}(x_j))$, where $\hat{e}(x_j)$ is the estimated propensity score given by $\hat{e}(x_j) = h^{-1}(x_j^T \hat{\eta})$, $h(\cdot)$ being the logit link function. Here, $\hat{\eta}$ is the MLE of $\eta$ obtained by fitting a binary regression model of $x_j$ on $w_j$ for all $j \in B = \{j : z_j = i, \text{ for some } i, 1 \leq i \leq n_A\}$ with the logit link $h(\cdot)$. The specific structures of $m_1(\cdot)$ and $m_2(\cdot)$ under parametric and nonparametric approaches are given below.

**Parametric causal model.** The parametric forms of $m_1(\cdot)$ and $m_2(\cdot)$ are, $m_1(\hat{e}(x_j)) = \beta_0 + \hat{e}(x_j)\beta_1$ and $m_2(\hat{e}(x_j)) = \alpha$, so that $\theta_c = (\beta_0, \beta_1, \alpha, \sigma^2)'$. The parameters $\beta = (\beta_0, \beta_1)'$ and $\alpha$ are jointly assigned a multivariate normal prior distribution, $(\beta, \alpha)' \sim N(0, I)$ and the error variance $\sigma^2$ is assigned an $IG(a, b)$ prior for model computation.

**Nonparametric causal model.** The non-parametric approach of modeling $m_1(\cdot)$ and $m_2(\cdot)$ employs a penalized spline approach (Ruppert et al., 2003) for model estimation as described below. Let $\kappa_1 < \kappa_2 < \cdots < \kappa_m$ be a set of $m$ fixed knot points in $(0, 1)$. The functions $m_1(\cdot)$ and $m_2(\cdot)$ are represented using spline basis functions as following:

\[
\begin{align*}
    m_1(\hat{e}(x_j)) &= \beta_0 + \sum_{l_1=1}^s \beta_{l_1} \hat{e}(x_j)^{l_1} + \sum_{l_2=1}^m \beta_{s+l_2}(\hat{e}(x_j) - \kappa_{l_2})^{l_2}_+ \\
    m_2(\hat{e}(x_j)) &= \sum_{l_1=1}^s \gamma_{l_1} \hat{e}(x_j)^{l_1} + \sum_{l_2=1}^m \gamma_{s+l_2}(\hat{e}(x_j) - \kappa_{l_2})^{l_2}_+.
\end{align*}
\] (10)

so that the parameters for the causal model are $\theta_c = (\beta_0, \beta_1, \ldots, \beta_{s+m}, \gamma_1, \ldots, \gamma_{s+m}, \sigma^2)'$. The general modeling framework for non-parametric functions is motivated by the penalized spline regression approaches in the Bayesian survey sampling literature (Zheng and Little, 2003, 2005), with survey weights replaced by propensity scores. As a strategy to accurately estimate the non-parametric functions, we suggest placing a large number of knots. However, even a moderately large choice of $m$ may result in model over-fitting. To avoid the issue related to over-fitting, this article adopts regularizing the spline coefficients $\beta_{s+1}, \ldots, \beta_{s+m}$ and $\gamma_{s+1}, \ldots, \gamma_{s+m}$. In particular, $\beta_{j+s}$ and $\gamma_{j+s}$ follow the Bayesian Lasso shrinkage priors,
for \( j = 1, \ldots, m \). Following Park and Casella (2008), a scale-mixture representation of the Bayesian Lasso shrinkage prior is given by

\[
\beta_{j+s} \mid \tau_{1,j}^2 \sim N(0, \sigma^2 \tau_{1,j}^2), \quad \gamma_{j+s} \mid \tau_{2,j}^2 \sim N(0, \sigma^2 \tau_{2,j}^2) \\
\tau_{1,j}^2 \overset{iid}{\sim} Exp(\lambda_1^2), \quad \tau_{2,j}^2 \overset{iid}{\sim} Exp(\lambda_2^2), \quad j = 1, \ldots, m \\
\lambda_1^2 \sim Gamma(r_1, \delta_1), \quad \lambda_2^2 \sim Gamma(r_1, \delta_1).
\]

(11)

The choice of hyper-parameters is elicited in Section 3. The parameters \( \beta_j, \gamma_j \) are assigned i.i.d. \( N(0,1) \) priors for \( j = 1, \ldots, s \). We also assign \( \beta_0 \sim N(0,1) \) and \( \sigma^2 \sim IG(a, b) \) a-priori. The prior specification is completed by setting prior distributions on parameters \( \theta_d = (\mu_1, \sigma_1^2)' \) as \( \mu_1 \sim N(0,1) \) and \( \sigma_1^2 \sim IG(a_{\sigma_1}, b_{\sigma_1}) \) a-priori.

### 3 Posterior Computation

Incorporating the prior information, the full posterior for the parametric causal model is proportional to

\[
L(\theta_d, \theta_c, \theta_m, \theta_u, z \mid \{\gamma_{ij} : 1 \leq i \leq n_A, 1 \leq j \leq n_B\}, \mathbf{y}, \mathbf{w}, \mathbf{X}) \times P(z \mid \alpha_\pi, \beta_\pi) \\
\times \prod_{f=1}^{F} \theta_{f,m}^{a-1}(1 - \theta_{f,m})^{b-1} \times \prod_{f=1}^{F} \theta_{f,u}^{a-1}(1 - \theta_{f,u})^{b-1} \times IG(\sigma^2 | a, b) \times N ((\beta, \alpha)'|0, I) \\
\times N(\mu_1|0,1) \times IG(\sigma_1^2|a_1, b_1),
\]

(12)
Similarly, the full posterior for the non-parametric causal model, incorporating the prior information, is proportional to

\[
L(\theta_d, \theta_c, \theta_m, \theta_u, z; \{\gamma_{ij} : 1 \leq i \leq n_A, 1 \leq j \leq n_B\}, y, w, X) \times P(z | \alpha, \beta)
\]

\[
\times \prod_{j=1}^{F} \theta_{f,m}^{\alpha-1} (1 - \theta_{f,m})^{b-1} \times \prod_{j=1}^{F} \theta_{f,u}^{\alpha-1} (1 - \theta_{f,u})^{b-1} \times IG(\sigma^2 | a, b) \times N(\beta_0 | 0, 1)
\]

\[
\times \prod_{j=1}^{s} N((\beta_j, \gamma_j)' | 0, I) \times \prod_{j=1}^{m} \left[ N(\beta_{j+s} | 0, \sigma^2 \tau_1^{2,j}) \times N(\gamma_{j+s} | 0, \sigma^2 \tau_2^{2,j}) \right]
\]

\[
\times \prod_{j=1}^{m} \left[ Exp(\lambda_1^{2,j} | r_1, \delta_1) \times Gamma(\lambda_1^{2,j} | r_1, \delta_1) \times Gamma(\lambda_2^{2,j} | r_2, \delta_2) \right]
\]

\[
\times N(\mu_1 | 0, 1) \times IG(\sigma_1^2 | a_1, b_1).
\]

Although summaries of the posterior distribution cannot be computed in closed form, full conditional distributions for all the parameters are available, both in the cases of parametric and non-parametric causal models and correspond to standard families. Thus, posterior computation can proceed through a Markov chain Monte Carlo algorithm.

While fitting the joint model using MCMC, in each iteration, we take the model-determined matched pairs and fit the causal model only taking the linked data subset, thus updating the parameters in the iteration, and repeat the process. We also emphasize that the estimated propensity score \( \hat{e}(x_j) \) is computed in each iteration for only those records in File B which have been matched to some records in File A. Details of the full conditional posterior distributions of parameters are provided in Appendix A.

We let the MCMC chain run for 2000 iterations and discard the first 1500 as burn-in and draw inference on both the average treatment effect (ATE) (occasionally referred to as the causal effect) and record linkage based on the post burn-in iterates. To draw inference on record linkage, let \( z_{j}^{(1)}, ..., z_{j}^{(L)} \) be the \( L \) post burn-in MCMC iterates of \( z_j, j = 1, ..., n_B \). For each \( j \), we empirically estimate \( P(z_j = q | -) \) using the proportion of post burn-in samples where \( z_j \) takes the value \( q \), i.e., \( \hat{P}(z_j = q | -) = \frac{\#\{z_{j}^{(l)} = q\}}{L} \), for \( q \in \mathcal{J}_j = \{1, ..., n_A, n_A + j\} \).

If \( 1 \leq q^* = \arg \max_{q \in \mathcal{J}_j} \hat{P}(z_j = q | -) \leq n_A \), we declare that the \( q^* \)th record of File A has a
match with the \( j \)th record of File B, and denote \( \hat{z}_j = q \). On the other hand, if \( q^* = n_A + j \), we declare no match between the \( j \)th record of File B with any record in File A. Thus the point estimates and uncertainties of linkage between records in two files can be readily estimated empirically. In particular, the estimated uncertainty of determining a link between \( q^* \)th record in File A and \( j \)th record in File B is given by \( \hat{P}(z_j = q^* | -) \).

To draw posterior inference on the average treatment effect (ATE) given in (7), define \( y_{\text{miss},i} = (1 - w_i)y_i(1) + w_iy_i(0) \) as the counterfactual outcome for the \( i \)th observation in File A, \( i = 1, ..., n_A \). At the \( l \)-th post burn-in iteration, the counterfactual outcomes \( y_{\text{miss},i}^{(l)} \)'s for all \( i \in A^{(l)} = \{ i : z_j^{(l)} = i, \text{ for some } j \} \) are imputed from their posterior predictive distributions. In particular, we perform one-one sampling from the following posterior predictive distribution,

\[
p(y_{\text{miss},i}|y_1, ..., y_{n_A}) = \int f_1(y_{\text{miss},i}|1 - w_j, x_j, \theta_c) p(\theta_c|y_1, ..., y_{n_B}) d\theta_c.
\]

Thus, for each post burn-in \( \theta_c^{(l)} \), \( y_{\text{miss},i}^{(l)} \) is simulated from the causal model of interest, with \( w_j \) replaced by \( (1 - w_j) \). Depending on whether \( y_i \) represents \( y_i(1) \) or \( y_i(0) \), the \( l \)-th post burn-in iterate for the ATE is obtained from \( y_i \) and \( y_{\text{miss},i}^{(l)} \) over all \( i \in A^{(l)} \) using the formula in (7). Throughout the analysis we choose the values of the hyperparameters as \( a = 1, b = 1, \alpha_\pi = 1, \beta_\pi = 1, a_1 = 1, b_1 = 1, r_1 = r_2 = \delta_1 = \delta_2 = 1 \).

4 Simulation Studies

We carry out simulation studies to assess the performance of our method (4), which we refer to as the joint model, vis-a-vis competitors under various simulation settings. We consider simulation scenarios in which we vary (a) the proportion of overlap between the two files; (b) the true causal model generating the data overlapping between the two files, and (c) the number of linking variables. We additionally consider simulation scenarios with a mis-specified causal model, correlated covariates and with missing outcomes.

For the simulation studies, we work with the dataset ‘RLdata10000’ contained in the R
package RecordLinkage. The dataset contains artificial personal data with 10000 records, in which there are two sets of 1000 duplicate records corresponding to the same set of 1000 subjects, but with randomly generated errors in their linking fields. From this synthetic data, we construct 2 files, File A and File B, with a pre-specified number of overlaps and choose the overlapping records in Files A and B from these two sets of duplicate records. The non-overlapping records in Files A and B are taken from the rest 8000 records in ‘RLdata10000,’ after ensuring that the non-overlapping records correspond to different subjects. In all simulations, the size of Files A and B are taken to be equal with \( n_A = n_B = 1000 \). Various simulation studies investigate the proposed model and its competitors with different degrees of overlap between these two files. In each file, we have information on four error prone linking fields, namely, first name, last name, birth year and birth date. Since it would be expected for a record linkage methodology to perform well when the records have a lot of identifying information, we restrict ourselves to more challenging scenarios where decisions have to be made based on only a small number of fields.

Next, we simulate the \( p \) predictors in File B. The \( l \)-th predictor for the \( n_B \) records are simulated as \((x_{l,1}, \ldots, x_{l,n_B})' \sim N(0, \Sigma)\), where \( \Sigma \) is a \( n_B \times n_B \) matrix crucially accounting for the correlation of the \( l \)-th predictor observed over different records, \( l = 1, \ldots, p \). The binary treatment assignments \( w_j \)'s, for \( j = 1, \ldots, n_B \), are generated using the propensity score model specified as follows:

\[
e(\mathbf{x}_j) = P(w_j = 1|\mathbf{x}_j) = \frac{e^{\alpha_0 + \sum_{l=1}^{p} \alpha_l x_{l,j}}}{1 + e^{\alpha_0 + \sum_{l=1}^{p} \alpha_l x_{l,j}}}, \quad (14)
\]

Corresponding to the records in File A which are true matches to the records in File B (i.e., which have overlapping records from File B), we simulate the outcome from the causal model. To be more specific, when the \( i \)-th record in File A has a true match with the \( j \)-th record in File B, we simulate the outcome \( y_i \) from the following model similar to (9),

\[
y_i = m_1^0(\mathbf{x}_j) + m_2^0(\mathbf{x}_j)w_j + \epsilon_{i,j}, \quad \epsilon_{i,j} \sim N(0, 1). \quad (15)
\]
We adopt two specific choices for \( m_1^0 \) and \( m_2^0 \) as follows:

(A) **Linear function in propensity score:** \( m_1^0(x_j) = 1 + 2e(x_j) \) and \( m_2^0(x_j) = 4 \).

(B) **Nonlinear function in propensity score:** \( m_1^0(x_j) = 5 - 1.5e(x_j) \) and \( m_2^0(x_j) = e^{-0.8+2.6e(x_j)} \).

The outcome \( y_i \) corresponding to the records in File A which do not have any link to File B are drawn i.i.d. from \( N(0, 1) \). For all simulations, we set \( p = 2 \) and \( \alpha_0 = 1 \), \( \alpha_1 = 1.5 \), \( \alpha_2 = -1 \).

**Competitors.** We subsequently compare the performance of the *joint* model with the following two competitors:

*Two-Stage Model:* In the two-stage model, records are linked first, and causal inference is carried out on the records linked in the initial step, i.e., the exercise is sequential as opposed to joint. Comparisons with this model reveals if the concurrent flow of information between the record linkage and causal inference exercises offers any inferential advantages.

*Known Link Model:* The causal model (parametric or non-parametric) is run on records which are *true links*. In this approach, there is no error in the causal model fitting due to any incorrect matching between the two files. Hence, if the true model and the fitted model are the same (i.e., there is no model mis-specification), the causal inference obtained from this approach can be considered the *gold standard* in different simulation scenarios.

**Metrics for comparison.** For both the joint and two-stage models, we compare performance accuracy in terms of both causal inference and record linkage. For these two competitors, we look at two measures of accuracy for our linkage and non-linkage decisions, namely the **positive predictive value** (PPV) and the **negative predictive value** (NPV), respectively. Following the notations in Section 3, let \( \hat{z} \) be the point estimate of \( z \). The PPV is the proportion of links that are actual matches, that is \( \sum_{j=1}^{n_B} I(\hat{z}_j = z_j \leq n_A) / \sum_{j=1}^{n_B} I(z_j \leq n_A) \). On the other hand, the NPV is the proportion of non-links that are actual non-matches, \( \sum_{j=1}^{n_B} I(\hat{z}_j = z_j = n_A + j) / \sum_{j=1}^{n_B} I(z_j = n_A + j) \). A perfect record linkage procedure would result in PPV=NPV=1. Additionally, in order to assess the quality of causal inference, we use the mean squared error (MSE) of the post burn-in causal effects from any competitor...
with the true causal effect, i.e., $\text{MSE} = \frac{1}{L} \sum_{l=1}^{L} (\text{ATE}^{(l)} - \text{ATE}_0)^2$, where $\text{ATE}_0$ is the true value of ATE (defined in (7)) and $\text{ATE}^{(l)}$ is the $l$th post burn-in estimate of ATE. Since the known link model operates on only true links, we will report its MSE for causal inference. We also report the posterior distributions as well as the 95% credible intervals of the causal effects (ATE) from the competitors vis-a-vis the true causal effect.

The next few sections demonstrate performance of all competitors under different simulation settings.

### 4.1 Simulation 1: Performance under Uncorrelated Predictors

Throughout the first set of simulations, we set $\Sigma = I$, i.e., the covariates are i.i.d. across records in File B. This section performs two sets of simulations assuming (i) no model mis-specification and (ii) model mis-specification.

**(i) Performance under no model mis-specification.**

Under no model mis-specification, a parametric or non-parametric causal model (described in Section 2.3.1) is fitted, depending on whether the choices of $m_0^1(\cdot)$ and $m_0^2(\cdot)$ in the true causal model (15) are linear (Scheme (A)) or nonlinear (Scheme (B)) in nature, respectively. For either parametric or non-parametric choices of the true causal model, we simulate three datafiles with 10%, 50% and 90% overlaps, respectively.

Both the joint model and the two-stage models are fitted with three linking fields, *first name*, *last name* and *birth year*. Table 1 records the measures of accuracy of record linkage in terms of PPV and NPV for the two competitors. A few interesting patterns emerge from Table 1. First, for data generated both under Schemes (A) and (B), the joint model shows a decrease in PPV and an increase in NPV as the percentage of overlap between Files A and B decreases. The two-stage model also demonstrates a similar pattern in all cases except in the case of 10% overlap under Scheme (A), where NPV drops marginally compared to the case with 50% overlap under Scheme (A). Notably, with a higher percentage of overlaps, the PPV of the two-stage method is marginally higher than the joint model, whereas the NPV of the two-stage method is substantially lower than that of the joint model. As the
percentage of overlaps decreases, the situation gets reversed, with the PPV of the two-stage method substantially deteriorating compared to the joint. Digging a bit deeper, we observe that under 90% overlap, the two-stage method shows the tendency of identifying many more links over and above the set of true links, which explains the higher PPV and lower NPV observed. However, with a 10% overlap, it estimates most records in File B being not linked to any record in File A, which explains the higher NPV and lower PPV.

The superior performance of the joint model over the two-stage model has a positive impact on the estimation of the causal effect. The MSE measure of estimating the true causal effect by the competitors provided in Table 2 reveals that the joint model performs significantly better than the two-stage model. In fact, the performance gap becomes more drastic as the percentage of overlap decreases. Notably, the Known Link model is the gold standard under these simulation scenarios and the joint model performs very close to the Known Link model.

| Scheme | Percentage of Overlaps | PPV (Joint) | NPV (Joint) | PPV (2-Stage) | NPV (2-Stage) |
|--------|------------------------|-------------|-------------|---------------|---------------|
| (A)    | 90                     | 0.916       | 0.656       | 0.947         | 0.500         |
|        | 50                     | 0.822       | 0.926       | 0.900         | 0.957         |
|        | 10                     | 0.758       | 0.998       | 0.280         | 0.925         |
| (B)    | 90                     | 0.919       | 0.720       | 0.953         | 0.010         |
|        | 50                     | 0.874       | 0.927       | 0.899         | 0.721         |
|        | 10                     | 0.834       | 0.992       | 0.240         | 1.000         |

Table 1: Table presents the positive predictive values (PPV) and the negative predictive values (NPV) for the joint and the 2-stage models, corresponding to different overlap levels. The two schemes correspond to the two data generation schemes (A) and (B). The known link model already has the true links incorporated, and hence any test of accuracy of record linkage is not applicable therein.

(ii) Performance under model mis-specification.
In order to assess the impact of model mis-specification on the performance of both causal inference and record linkage, we fit a non-parametric causal model with data generated under Scheme (A) and parametric causal model with data generated under Scheme (B). In each case, the performances of the joint and two-stage models under mis-specification
### Table 2: MSE of estimating the true causal effect ($ATE_0$) for each of the competing models, namely the joint, the 2-stage and the known link models, corresponding to different overlap levels.

| Scheme   | Percentage of Overlaps | Joint Model | 2-Stage Model | Known Link Model |
|----------|------------------------|-------------|---------------|------------------|
| (A)      | 90                     | 0.016       | 0.734         | 0.013            |
|          | 50                     | 0.048       | 2.200         | 0.016            |
|          | 10                     | 0.058       | 0.403         | 0.053            |
| (B)      | 90                     | 0.014       | 0.699         | 0.012            |
|          | 50                     | 0.027       | 1.269         | 0.028            |
|          | 10                     | 0.528       | 11.317        | 0.145            |

Table 2: Table presents the MSE of estimating the true causal effect ($ATE_0$) for each of the competing models, namely the joint, the 2-stage and the known link models, corresponding to different overlap levels. Results under both data generating schemes (A) and (B) are presented.

are compared to those of the corresponding models under no mis-specification to assess the impact of mis-specification. Table 3 presents the PPV, NPV and MSE for the competing models. Although we do not observe much deterioration of record linkage in terms of PPV and NPV, MSE of estimating ATE worsens under model mis-specification.

| Scheme   | Fitted Model     | PPV Joint | PPV 2-Stage | NPV Joint | NPV 2-Stage | MSE Joint | MSE 2-Stage |
|----------|------------------|-----------|-------------|-----------|-------------|-----------|-------------|
| (A)      | Parametric       | 0.822     | 0.900       | 0.926     | 0.957       | 0.048     | 2.200       |
|          | Nonparametric    | 0.822     | 0.901       | 0.936     | 0.719       | 0.218     | 2.596       |
| (B)      | Parametric       | 0.855     | 0.902       | 0.931     | 0.962       | 0.245     | 1.757       |
|          | Nonparametric    | 0.874     | 0.899       | 0.927     | 0.721       | 0.027     | 1.269       |

Table 3: Table presents the PPV, NPV and MSE of estimating the true causal effect ($ATE_0$) for the joint and the 2-stage models, for both causal model mis-specification and correct model specification. Results under both data generating schemes (A) and (B) are presented.

### 4.2 Simulation 2: Performance under correlated predictors

In this section, we assess the performance of both the joint and the two-stage models when each covariate across all records in File B are highly correlated, i.e., $\Sigma \neq I$, unlike in Simulation 1. More specifically, we simulate predictors with $\Sigma = 0.5I + 0.5J$, where $J$ is a $n_B \times n_B$ matrix with each entry being 1. Thus the $l$th predictor values corresponding
to the $j$th and $j'$th records have a correlation of 0.5, for all $1 \neq j \neq j' \leq n_B$, $l = 1, ..., p$.

The simulations in this section assume no model mis-specification. Thus, a parametric or non-parametric causal model (described in Section 2.3.1) is fitted, depending on whether the choices of $m_1^0()$ and $m_2^0()$ in the true causal model (15) are linear (Scheme (A)) or nonlinear (Scheme (B)) in nature, respectively. For either parametric or non-parametric choices of the true causal model, we simulate datafiles with 10% and 90% overlaps, respectively. Unlike Section 4.1, we do not present results for the 50% overlap since the trend becomes evident from the results corresponding to 10% and 90% overlaps. PPV, NPV and MSE for the joint model as well for the two-stage model are presented in Table 4. The overall trend does appear to change significantly in comparison with Section 4.1. In particular, the joint model produces a higher PPV and a lower NPV with a 90% overlap compared to a 10% overlap.

Contrasting results in this section with Section 4.1, we observe a more sharp decrease in PPV for the joint model with lesser overlaps when predictors in File B are correlated amongst records. The MSE of estimating ATE remains significantly smaller in the joint model.

### 4.3 Simulation 3: Performance under Missing Data

Finally, in this section, we present the performance of the joint and two-stage models in the presence of missing responses in File A. While fitting the joint model with missing data, we impute the missing observations by sampling them from their posterior predictive...
distributions in each MCMC iteration. For the *two-stage* model, the missing responses in File A corresponding to linked pairs are imputed similarly from their posterior predictive distributions. Similar to Section 4.2, this section also assumes no model mis-specification. For either parametric or non-parametric choices of the true causal model, we present the PPV, NPV and MSE of competing models with 5% and 10% missing observations in File A. All simulations have 90% overlap of records between File A and File B.

| Scheme | Fitted Model | Missing % | PPV       | NPV       | MSE       |
|--------|--------------|-----------|-----------|-----------|-----------|
| (A)    | Parametric   | 5%        | 0.922     | 0.946     | 0.645     | 0.500     | 0.326     | 0.770     |
| (A)    | Parametric   | 10%       | 0.917     | 0.949     | 0.615     | 0.333     | 0.893     | 0.781     |
| (B)    | Nonparametric| 5%        | 0.917     | 0.946     | 0.740     | 0.010     | 0.011     | 0.718     |
| (B)    | Nonparametric| 10%       | 0.902     | 0.944     | 0.650     | 0.010     | 0.023     | 0.769     |

Table 5: Table presents the PPV, NPV and MSE of estimating the true causal effect ($ATE_0$) for the *joint* and the *2-stage* models, for 5% and 10% missing records in File A. Results under both data generating schemes (A) and (B) are presented.

As expected, the results in Table 5 show a bit of deterioration in the performance of *joint* model when the percentage of missing data increases in File A. This can be seen in the form of a lower PPV, NPV and higher MSE in the case corresponding to 10% missing observations. A similar trend is observed for the *two-stage* model as well. Consistent with our observation for the cases with 90% overlap between Files A and B in earlier sections, we find the *two-stage* model delivering marginally higher PPV and considerably lower NPV than the *joint* model, i.e., it shows a tendency to declare a lot more records as links. This provides a considerable edge to the *joint* model over the *two-stage* model in terms of estimating the average treatment effect. In fact, the MSE of estimating ATE is found to be considerably lower in the *joint* model compared to the *two-stage* model for all cases under Scheme (B). Under Scheme (A) with 5% missing responses, the *joint* model shows superior performance over the *two-stage* model, though their performance becomes competitive in the case with 10% missing records in File A. This may appear to be surprising since the *joint* model demonstrates a better record linkage performance than the *two-stage* model under this scenario. However, note that under
Scheme (A), both the linked and non-linked responses follow normal distributions (albeit with different means and variances). Perhaps the differences between these two distributions are not stark under Scheme (A), which can explain the result.

5 Real Data Application

6 Conclusion and Future Work

In a variety of digital health data, it is of interest to draw causal inferences with the response in one file and the treatment status and predictors in a separate file. A common approach in such scenarios is to first implement a record linkage procedure followed by causal inference on the linked records between two files. Rather than following the sequential two stage procedure, this article proposes a joint Bayesian model that simultaneously draws posterior inference on the linked datafiles and the causal effect. Intuitively, the joint framework borrows information between the models deployed for causal inference and probabilistic record linkage and thus should improve the inference in both. Empirical investigations of our proposed joint model vis-a-vis the sequential procedure reveal more precise record linkage and causal inference from the former. We demonstrate the performance of these two models under various simulation scenarios including model mis-specification, correlated predictors and missing records in the response file.

As a first step towards joint modeling of causal inference and probabilistic record linkage, our results suggest many interesting future directions. For example, many health record data applications suggest predictors and treatment status data residing in different files. This requires significant modifications of our existing approach. Another important future direction is to develop our approach with causal inference models employed in social science in the context of relational data. We are currently pursuing some of this research threads as our future work.
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7 Appendix

7.1 Appendix A

7.1.1 Parametric Causal Model

The full conditionals for the *parametric* causal inference model with record linkage are:

- \( \theta_{f,m} | - \sim \text{Beta} \left[ \left( \sum_{i,j} \gamma_{f,ij} I(z_j = i) + a \right), \left( \sum_{i,j} (1 - \gamma_{f,ij}) I(z_j = i) + b \right) \right] \)
- \( \theta_{f,u} | - \sim \text{Beta} \left[ \left( \sum_{i,j} \gamma_{f,ij} I(z_j \neq i) + a \right), \left( \sum_{i,j} (1 - \gamma_{f,ij}) I(z_j \neq i) + b \right) \right] \)

- Find all \((i, j)\) s.t. \(z_j = i\), where \(j = 1, \ldots, n_B\) and \(i = 1, \ldots, n_A\). Stacking all \(y\)'s corresponding to these pairs, we obtain \(\tilde{y}\), a vector of dimension \(n_{A,B}\), where \(n_{A,B}\) is the number of matches between two files. Note that, the set of matched pairs (and hence \(n_{A,B}\)) is updated in every MCMC iteration. We construct a \(n_{A,B} \times 3\) matrix \(\tilde{K} = [1 : \tilde{E} : \tilde{W}]\), where \(\tilde{E}\) and \(\tilde{W}\) are propensity scores and treatment status corresponding to the \(n_{A,B}\) matches. Hence, \(\pi((\beta, \alpha)' | -) \propto N(\tilde{y} | \tilde{K}(\beta, \alpha)', \sigma^2 I_{n_{A,B}}) \times N((\beta, \alpha)' | 0, I_3)\), which leads to \((\beta, \alpha)' | - \sim N(\mu_{\beta|}, \Sigma_{\beta|})\), where

\[
\Sigma_{\beta|} = \left( \frac{\tilde{K}' \tilde{K}}{\sigma^2} + I \right)^{-1}, \quad \mu_{\beta|} = \Sigma_{\beta|} \tilde{K}' \tilde{y} \frac{1}{\sigma^2}.
\]
• The full conditional for $z$ is given below.

\[
P(z_j = q | \cdot) \propto \exp(\omega_{1,qj} + \omega_{2,qj})I(z_j' \neq q, \forall j' \neq j); \ q = 1, ..., n_A
\]

\[
P(z_j = n_A + j | \cdot) \propto (n_A - n_{AB}(z_{-j}))^{n_B - n_{AB}(z_{-j}) - 1 + \beta_a}n_{AB}(z_{-j})^{\alpha_a - 1},
\]

where $\omega_{1,qj} = \sum_{f=1}^F \{\gamma_{f,qj} \log(\frac{\theta_{f,m}}{f_{f,a}}) + (1 - \gamma_{f,qj}) \log(\frac{1 - \theta_{f,m}}{1 - f_{f,a}})}\}$, $\omega_{2,qj} = \log(\frac{f_{yq}(y|u,w)}{f_{yq}})$ and $z_{-j}$ represents the vector of $z$ without $z_j$.

### 7.1.2 Nonparametric Causal Model

The full conditionals for the nonparametric causal inference model with record linkage are:

- $\theta_{f,m} | \sim \text{Beta} \left( (\sum_{i,j} \gamma_{f,ij} I(z_j = i) + a), (\sum_{i,j} (1 - \gamma_{f,ij}) I(z_j = i) + b) \right)$

- $\theta_{f,u} | \sim \text{Beta} \left( (\sum_{i,j} \gamma_{f,ij} I(z_j = i) + a), (\sum_{i,j} (1 - \gamma_{f,ij}) I(z_j = i) + b) \right)$

- Find all $(i, j)$ s.t. $z_j = i$, where $j = 1, ..., n_B$ and $i = 1, ..., n_A$. Stacking all $y$'s corresponding to these pairs, we obtain $\tilde{y}$, a vector of dimension $n_{A,B}$, where $n_{A,B}$ is the number of matches between two files. Note that, the set of matched pairs (and hence $n_{A,B}$) is updated in every MCMC iteration. We construct a $n_{A,B} \times (2(s + m) + 1)$ matrix $\tilde{K} = [1, \tilde{E}_1, \tilde{B}_1, \tilde{E}_2, \tilde{B}_2]$, where $\tilde{E}_1$ and $\tilde{E}_2$ are $n_{A,B} \times s$ matrices and $\tilde{B}_1, \tilde{B}_2$ are $n_{A,B} \times m$ matrices. Now, $\pi(\beta, \gamma | \cdot) \propto N(\tilde{y} | \tilde{K}(\beta, \gamma)'', \sigma^2 I) \times N((\beta, \gamma)' | \mu_{\beta,\gamma}, \Sigma_{\beta,\gamma})$

Hence, $(\beta, \gamma)' \sim N(\mu_{\beta,\gamma}', \Sigma_{\beta,\gamma}')$, where

\[
\Sigma_{\beta,\gamma}' = \left( \frac{\tilde{K}' \tilde{K}}{\sigma^2} + \Sigma_{\beta,\gamma}^{-1} \right)^{-1}, \ \mu_{\beta,\gamma}' = \Sigma_{\beta,\gamma}' \left( \frac{\tilde{K}' \tilde{y}}{\sigma^2} + \Sigma_{\beta,\gamma}^{-1} \mu_{\beta,\gamma} \right).
\]

Here, $\mu_{\beta,\gamma} = 0$, $\Sigma_{\beta,\gamma} = \text{diag}(I_{s+1}, \sigma^2 \text{diag}(\tau_1^2), I_s, \sigma^2 \text{diag}(\tau_2^2))$

- $\frac{1}{\tau_{1,j}} \sim \text{Inverse - Gaussian} \left( \sqrt{\frac{\lambda_1^2 \sigma^2}{\beta_{j+s}}, \lambda_1^2} \right)$

- $\frac{1}{\tau_{2,j}} \sim \text{Inverse - Gaussian} \left( \sqrt{\frac{\lambda_2^2 \sigma^2}{\beta_{j+s}}, \lambda_2^2} \right)$

- $\lambda_1^2 \sim \text{Gamma}(r_1 + m, \delta_1 + \sum_{j=1}^m \tau_{1,j}^2/2)$

- $\lambda_2^2 \sim \text{Gamma}(r_2 + m, \delta_2 + \sum_{j=1}^m \tau_{2,j}^2/2)$
• The full conditional for \( z \) is given below.

\[
P(z_j = q|\cdot) \propto \exp(\omega_{1,qj} + \omega_{2,qj}) I(z_j \neq q, \forall j' \neq j); \quad q = 1, \ldots, n_A
\]

\[
P(z_j = n_A + j|\cdot) \propto (n_A - n_{AB}(z_j)) \frac{n_B - n_{AB}(z_{-j}) - 1 + \beta_{n_A}}{n_{AB}(z_{-j}) + \alpha_{n_A}};
\]

where \( \omega_{1,qj} = \sum_{f=1}^{F} \{ \gamma_{f,qj} \log(\frac{\theta_{f,m}}{\theta_{f,u}}) + (1 - \gamma_{f,qj}) \log(\frac{1 - \theta_{f,m}}{1 - \theta_{f,u}}) \} \), \( \omega_{2,qj} = \log(\frac{f(y_j|x_j,w_j)}{f(y_j)}) \) and \( z_{-j} \) represents the vector of \( z \) without \( z_j \).

### 7.2 Appendix B

#### 7.2.1 Proof of Theorem 2.1

The likelihood ratio \( \text{Ratio}_{\text{Joint}} \) under the joint model can be expressed as following.

\[
\text{Ratio}_{\text{Joint}} = \prod_{(k,l):z_k=l, k \neq j} f_1(y_l|x_k, w_k, \theta_c) \times \prod_{f:z_k \neq l} f_2(y_l|\theta_d) \times \prod_{f:z_k \neq l} f_2(y_l|\theta_d) \times \prod_{f=1}^{F} \theta_{f,m}^{\gamma_{f,sj}^j} (1 - \theta_{f,m})^{1 - \gamma_{f,sj}^j}
\]

\[
= \frac{f_1(y_i|x_j, w_j, \theta_c)}{f_2(y_i|\theta_d)} \prod_{f=1}^{F} \left( \frac{\theta_{f,m}}{\theta_{f,u}} \right)^{\gamma_{f,sj}^j} \left( \frac{1 - \theta_{f,m}}{1 - \theta_{f,u}} \right)^{1 - \gamma_{f,sj}^j}
\]

(16)

Similarly,

\[
\text{Ratio}_{2\text{Stage}} = \prod_{f=1}^{F} \left( \frac{\theta_{f,m}}{\theta_{f,u}} \right)^{\gamma_{f,sj}^j} \left( \frac{1 - \theta_{f,m}}{1 - \theta_{f,u}} \right)^{1 - \gamma_{f,sj}^j}
\]

(17)

Let \( h(\theta_{f,m}, \theta_{f,u}) = \prod_{f=1}^{F} \left( \frac{\theta_{f,m}}{\theta_{f,u}} \right)^{\gamma_{f,sj}^j} \left( \frac{1 - \theta_{f,m}}{1 - \theta_{f,u}} \right)^{1 - \gamma_{f,sj}^j} \).

Thus,

\[
\log(\text{Ratio}_{\text{Joint}}) = \log(h(\theta_{f,m}, \theta_{f,u})) + \log \left[ \frac{f_1(y_i|x_j, w_j, \theta_c)}{f_2(y_i|\theta_d)} \right]
\]

and,

\[
\log(\text{Ratio}_{2\text{Stage}}) = \log(h(\theta_{f,m}, \theta_{f,u}))
\]

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\[ E_{e \sim j}[\log(\text{Ratio}_{\text{Joint}}) - \log(\text{Ratio}_{2\text{Stage}})] \]

\[ = \int \int \left[ \prod_{f=1}^{F} \theta_{f,m}^{\gamma_{f,i}^{j}}(1 - \theta_{f,m})^{1 - \gamma_{f,i}^{j}} \right] f_1(y_i | x_j, w_j, \theta_c) \log \left[ \frac{f_1(y_i | x_j, w_j, \theta_c)}{f_2(y_i | \theta_d)} \right] \geq 0 \]

as a consequence of this expression being a Kullback-Leibler divergence. Again,

\[ E_{e \neq j}[\log(\text{Ratio}_{\text{Joint}}) - \log(\text{Ratio}_{2\text{Stage}})] \]

\[ = \int \int \left[ \prod_{f=1}^{F} \theta_{f,a}^{\gamma_{f,i}^{j}}(1 - \theta_{f,a})^{1 - \gamma_{f,i}^{j}} \right] f_2(y_i | \theta_d) \log \left[ \frac{f_1(y_i | x_j, w_j, \theta_c)}{f_2(y_i | \theta_d)} \right] \leq 0, \]

where the last inequality follows by the fact that the expression is negative of the Kullback-Leibler divergence between the two densities \( f_1 \) and \( f_2 \).