A Bayesian Analysis of Two-Stage Randomized Experiments in the Presence of Interference and Treatment Nonadherence

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Abstract

Two crucial assumptions for causal inference that are often violated in modern, complicated experiments are “no interference” and treatment adherence. There are very limited methodologies that have been developed to address the interference and nonadherence issues simultaneously. Methodologies for performing causal inferences in the presence of both interference and nonadherence under complicated outcome generating mechanisms do not exist. We propose a Bayesian causal inference methodology to address this gap. Our methodology extends existing causal frameworks and methods, specifically, two-staged randomized experiments and the principal stratification framework. In contrast to existing methods that invoke strong structural assumptions to identify principal causal effects, our Bayesian approach uses flexible distributional models that can accommodate the complexity of interference and that ensure that principal causal effects are identifiable. We illustrate our methodology via extensive simulation studies and a re-analysis of real-life data from an evaluation of India’s National Health Insurance Program. In the latter case study our methodology enables us to identify new significant causal effects that were not identified in past analyses. Ultimately, our simulation studies and case study demonstrate how our methodology can yield more informative analyses in modern experiments with interference, treatment nonadherence, and complicated outcome generation mechanisms.

Keywords: Bayesian causal inference; Noncompliance; Principal stratification; Rubin Causal Model; Two-stage randomized design
1 Introduction

Causal inference is a fundamental consideration across a wide range of domains in science, technology, engineering, and medicine (Pearl, 2009; Imbens and Rubin, 2015). A traditional gold standard for performing causal inference is the classical randomized experiment (Rubin, 2008; Imbens and Rubin, 2015). In this experiment, a great deal of control and precautions can be taken so as to eliminate events that would introduce instabilities and biases in causal inferences. It is becoming more difficult to institute such control and precautions in modern, complicated experiments. Two significant sources of complications that are increasingly of interest are interference among experimental units and nonadherence/noncompliance of experimental units to their assigned treatments. Interference exists if the outcome of an experimental unit depends not only on its assigned treatment, but also on the assigned treatments for other units. It arises when limited controls are placed on the interactions of experimental units with one another during the course of an experiment. Treatment nonadherence frequently occurs in human subject experiments. Clinical trials in particular inevitably have subjects that do not adhere to their assigned treatments due to adverse side effects or intercurrent events (Little et al., 2012). Failing to account for interference and nonadherence in modern experiments will generally yield unstable and biased inferences on treatment effects for the latent stratum of experimental units that will adhere to their assigned treatments, i.e., the compliers.

A great deal of research efforts have been dedicated over the past three decades to develop causal inferential methodologies that address the issues of interference and nonadherence separately. Hudgens and Halloran (2008) first introduced the concept of the two-stage randomized design for performing causal inference in the presence of interference when all experimental units adhere to their assigned treatments. In this design, experimental units belong to clusters, and randomizations are performed at both the cluster-level
and experimental unit-level. Specifically, the clusters are first randomly assigned different probabilities for treatment assignment of their constituent experimental units, and then treatments are randomly assigned to the units within the clusters (with treatment assignment performed independently across clusters) based on the cluster-specific assignment probabilities. Hudgens and Halloran (2008) demonstrated how both direct treatment effects and indirect treatment effects (i.e., those effects that can be attributed to the treatments received by other units) can be inferred under this design in the presence of interference. This design and the corresponding causal inference methods that can be performed under it have been further studied by VanderWeele and Tchetgen (2011), Tchetgen and Vanderweele (2012), Liu and Hudgens (2014), and Basse and Feller (2018).

For experiments that have treatment nonadherence but not interference, one standard methodology that has been considered for their analyses is the intention-to-treat (ITT) method. Under this approach, the treatment received by an experimental unit is ignored, and instead only the treatment assigned is considered. This method follows the traditional principle of analyzing an experiment according to its physical randomization (Cox and Reid, 2000, p. 14) mechanism, and can yield valid causal inferences on the effects of treatment assigned in certain situations. However, the ITT method will generally yield biased inferences on the effects of treatment received because it does not account for the latent stratification of experimental units defined according to their adherence behaviors to the different treatment assignments, and consequently does not provide inferences for the target stratum of compliers. Angrist et al. (1996) and Imbens and Rubin (1997) developed a framework to provide a more principled approach for the analyses of experiments with nonadherence, and Frangakis and Rubin (2002) extended those frameworks to develop the more general principal stratification framework. This framework has been applied to a wide variety of real-life problems involving complications such as censoring or truncation due to death (Zhang and Rubin, 2003) and the occurrence of intermediate variables.
that are thought to mediate the effects of treatments on the outcome (Gallop et al., 2009). VanderWeele (2011) provides a detailed review of principal stratification.

Until very recently, interference and nonadherence have typically been studied separately. However, in modern, real-life experiments it is common for both issues to exist simultaneously. Imai et al. (2021) presented a nonparametric identification of the complier average direct and indirect effects, and proposed consistent estimators for them under the two-stage randomized design in the presence of interference and nonadherence. They derived large-sample nonparametric bounds for the causal effects, but their results are not necessarily valid for the finite-sample regime. In addition, the estimators of Imai et al. (2021) are sensitive to outliers. Vazquez-Bare (2022) built on the work of Imai et al. (2021) and analyzed spillover effects using instrumental variables in the two-stage randomized experiment. They considered the identification of causal direct and spillover effects under one-sided noncompliance and demonstrated that these effects can be estimated using two-stage least squares (2SLS). However, their methods do not work in general under two-sided noncompliance or when units have multiple peers without a strong structural assumption about peers’ compliance types. Kang and Imbens (2016) considered the peer encouragement design to study network treatment effects when treatment randomization cannot feasibly be forced on experimental units, and presented identification results only for the case of one-sided noncompliance. Forastiere et al. (2016) developed a Bayesian principal stratification method for causal inference in clustered encouragement designs (CEDs), where the assignment of treatment encouragement is performed at the cluster level. The CED is effectively a special case of the two-stage randomized experiment with no randomization within clusters, i.e., with treatment assignment probability for units within clusters being either zero or one. As there is no randomization performed at the unit level within clusters, their methodology cannot capture the complexity of adherence behaviors in the two-stage experiments, where units could possibly change their adherence behaviors depending on
what assignment probabilities their clusters are assigned to.

We develop a new Bayesian causal inferential methodology for two-stage randomized experiments with both interference and noncompliance. Our methodology utilizes the principal stratification framework to address the identification issues arising from treatment nonadherence in the two-stage randomized design. To the best of our knowledge, none of the existing causal inference methods have applied Bayesian principal stratification to two-stage randomized designs as our method does. Our use of the Bayesian approach enables more informative inferences in the presence of nonadherence because it addresses the identification issues of causal estimands in a more powerful manner compared to existing frequentist approaches that rely on strong structural assumptions. Furthermore, our Bayesian method enables us to infer principal causal effects under two-sided nonadherence and define new types of interpretable and informative causal estimands, such as the complier spillover and overall treatment effects. Our estimands are always weakly identifiable (Imbens and Rubin, 1997) without the need for strong structural assumptions, and would be of great interest to policy makers. In addition, our methodology highlights new assumptions on compliance types for making causal inferences more efficient and stable in experiments with interference and nonadherence. The use of Bayesian models in our methodology enables us to accommodate complicated outcome generation mechanisms that frequently occur in modern experiments, such as heavy-tailed, skewed, zero-inflated, and/or multi-modal outcome distributions.

We proceed in Section 2 to review the Rubin Causal Model (RCM, Rubin, 1974), the principal stratification framework, and relevant assumptions and causal estimands for the two-stage randomized experiment. Section 3 introduces the models, and computational algorithms involved in our Bayesian methodology. In Section 4 we perform extensive simulation studies to investigate the frequentist performance of our method for a heavy-tailed distribution with an excess of zeros values. These simulation studies effectively validate
our methodology in a situation where the existing frequentist approach performs poorly in terms of bias and mean squared error (MSE). Finally, in Section 5 we apply our methodology to the real-life data from the evaluation of India’s National Health Insurance Program (RSBY) (Nandi et al., 2015). In our analysis of this case study we are able to uncover more definitive evidence of causal effects that were previously found to be insignificant in past analyses. Our concluding remarks are in Section 6.

2 Background

2.1 Two-Stage Randomized Experiments with Interference and Noncompliance

Throughout this manuscript we consider two-stage randomized experiments involving two treatments and $J$ clusters, with $N_j$ experimental units in cluster $j = 1, \ldots, J$ (each experimental unit belongs to only one cluster). We let $N = \sum_{j=1}^{J} N_j$ denote the total number of experimental units. The assignment mechanism in the two-stage randomized experiment is performed sequentially, with each stage involving a type of completely randomized design. In the first stage, $J_1$ clusters are randomly chosen to have a treatment assignment probability of $a_1 \in (0, 1)$ for their constituent experimental units, and the remaining $J - J_1$ clusters have a treatment assignment probability of $a_0 \in (0, 1)$ for their units. For each $j = 1, \ldots, J$ we let $A_j$ be the indicator for whether cluster $j$ was assigned $a_1$ ($A_j = 1$) or $a_0$ ($A_j = 0$). We let $\mathbf{A} = (A_1, \ldots, A_J)^T$, and without loss of generality we let $a_1 > a_0$. In the second stage, the experimental units within the clusters are randomly assigned treatment and control according to the treatment assignment probabilities assigned to their clusters, with the treatment assignment performed independently across clusters. For each cluster $j$ with $A_j = 1$, $N_j a_1$ of their experimental units are randomly assigned treatment and the
remaining $N_j(1 - a_1)$ are assigned control. Similarly, for each cluster $j'$ with $A_{j'} = 0$, $N_{j'}a_0$ of their experimental units are randomly assigned treatment and the remaining $N_{j'}(1 - a_0)$ are assigned control. We assume that $N_ja_1$ and $N_ja_0$ are integers for all $j = 1, \ldots, J$. We let $Z_{i,j}$ denote the treatment assignment indicator for unit $i$ in cluster $j$, with $Z_{i,j} = 1$ if it is assigned treatment and $Z_{i,j} = 0$ otherwise. We let $Z_j = (Z_{1,j}, \ldots, Z_{N_j,j})^T$ denote the vector of treatment assignment indicators for all units in cluster $j$, and $Z_{-i,j}$ denote the subvector of $Z_j$ with the $i$th entry removed. Other assignment mechanisms for two-stage randomized experiments are provided by VanderWeele and Tchetgen (2011), but we do not consider them here.

As we consider two-stage randomized experiments with nonadherence and interference under the RCM, we must introduce two types of potential outcomes for the treatment received by an experimental unit and the final outcome of interest that are functions of the experimental units’ treatment assignments. We let $D_{i,j}(z)$ denote the treatment received for unit $i$ in cluster $j$ under treatment assignment $z \in \{0, 1\}^N$, $D_j(z) = (D_{1,j}(z), \ldots, D_{N_j,j}(z))^T$ be the vector of treatments received by the units in cluster $j$, and $D(z) = (D_1(z), \ldots, D_J(z))^T$. Furthermore, we let $Y_{i,j}(z, D(z))$ denote the potential outcome for unit $i$ in cluster $j$ under treatment assignment vector $z$ and treatment received vector $D(z)$. Although the potential outcomes can be written solely as a function of $z$ (because $D(z)$ is a function of $z$) we include $D(z)$ in the notation for $Y_{i,j}(z, D(z))$ to emphasize the existence of nonadherence.

Besides treatments and potential outcomes, we assume covariates are measured for the experimental units. These covariates are either measured prior to treatment assignment, or are measured afterwards but are not affected by treatment assignment. We denote the vector of covariates for unit $i$ in cluster $j$ by $x_{i,j}$. 

2.2 Assumptions on the Structure of Interference

Following Imai et al. (2021) we assume the exclusion restriction with interference between units in the two-stage randomized experiment. In this case, the potential outcome of a unit \(i\) in cluster \(j\) only depends on the treatments received by units within cluster \(j\).

**Assumption 1.** For any treatment assignment vectors \(z, z' \in \{0, 1\}^N\) with \(D_j(z) = D_j(z')\), \(Y_{i,j}(z, D(z)) = Y_{i,j}(z', D(z'))\) for all experimental units \(i\) in cluster \(j\).

We also invoke the partial interference assumption in which units in different clusters do not interact or affect one another. This assumption was first formulated by Hudgens and Halloran (2008), and was extended to the noncompliance setting by Imai et al. (2021). Partial interference facilitates causal inference in our setting of interest because an experimental unit \(i\)’s treatment received and final outcome will only be functions of treatment assignments for other units within the same cluster \(j\) as unit \(i\).

**Assumption 2.** If \(z, z' \in \{0, 1\}^N\) such that \(z_j = z'_j\) for a cluster \(j\), then \(D_j(z) = D_j(z')\) and \(Y_{i,j}(z, D(z)) = Y_{i,j}(z', D(z'))\) for all experimental units \(i\) in cluster \(j\).

The final assumption that we consider is the stratified interference assumption of Hudgens and Halloran (2008). This assumption imposes further structure on interference by having the treatment received and the potential outcome for an experimental unit being a function of just the number of experimental units assigned treatment within the cluster. This assumption was also considered by Forastiere et al. (2016) and Imai et al. (2021). It is important to recognize that a great deal of work has been conducted to move beyond this condition and consider more flexible structures of interference (Aronow, 2012; Manski, 2013; Basse and Airoldi, 2018a,b; Aronow and Samii, 2017; Baird et al., 2018; Basse and Airoldi, 2018a; Athey et al., 2018; Basse et al., 2019; Leung, 2020; Forastiere et al., 2021; Sävje et al., 2021).
Assumption 3. For a cluster \( j \) and experimental unit \( i \) in cluster \( j \), if \( \mathbf{z}, \mathbf{z}' \in \{0, 1\}^N \) such that \( z_{i,j} = z'_{i,j} \) and \( \mathbf{z}_j^\top \mathbf{1} = (\mathbf{z'}_j^\top \mathbf{1} \), then \( D_{i,j}(\mathbf{z}) = D_{i,j}(\mathbf{z'}) \) and \( Y_{i,j}(\mathbf{z}, D(\mathbf{z})) = Y_{i,j}(\mathbf{z}', D(\mathbf{z}')) \).

These three assumptions imply that for two-stage randomized experiments in which the number of treated units within any cluster is fixed by design, the potential outcomes for an experimental unit are a function of its own treatment assignment and the treatment assignment probability for its cluster. Specifically, we slightly abuse the potential outcomes notation to write \( D_{i,j}(\mathbf{z}) \) and \( Y_{i,j}(\mathbf{z}, D(\mathbf{z})) \) as \( D_{i,j}(\mathbf{z}, a) \) and \( Y_{i,j}(\mathbf{z}, a) \), respectively, where \( z \) denotes the treatment assignment for the experimental unit and \( a \) denotes the treatment assignment probability for the unit’s cluster.

2.3 Principal Strata, Monotonicity, and the Exclusion Restriction for Two-Stage Randomized Experiments

Under the principal stratification framework, we stratify the experimental units according to their values of \( D_{i,j}(z, a) \) under the different possible treatment assignments \( z \in \{0, 1\} \) and treatment assignment probabilities \( a \in \{a_0, a_1\} \) for the clusters. There exist four such potential values: \( D_{i,j}(0, a_0), D_{i,j}(1, a_0), D_{i,j}(0, a_1), \) and \( D_{i,j}(1, a_1) \). A unique feature of our consideration of nonadherence for the two-stage randomized design is that, according to Assumption 3, units can have different compliance behaviors under different assignment probabilities for their clusters. We formally define the compliance behavior of unit \( i \) in cluster \( j \) under each treatment assignment probability \( a \in \{a_0, a_1\} \) for the cluster as

\[
G_{i,j}(a) = \begin{cases} 
  n & \text{if } D_{i,j}(0, a) = D_{i,j}(1, a) = 0, \\
  c & \text{if } D_{i,j}(0, a) = 0, D_{i,j}(1, a) = 1, \\
  d & \text{if } D_{i,j}(0, a) = 1, D_{i,j}(1, a) = 0, \\
  a & \text{if } D_{i,j}(0, a) = D_{i,j}(1, a) = 1,
\end{cases}
\]

where \( n, c, d, \) and \( a \) denote never-takers, compliers, defiers, and always-takers, respectively.

Finally, the compliance behavior for unit \( i \) in cluster \( j \) is defined according to the pair of compliance indicators \( G_{i,j} = (G_{i,j}(a_0), G_{i,j}(a_1)) \).
A standard assumption for the compliance behavior is monotonicity.

**Assumption 4.** For all units $i = 1, \ldots, N_j$ in cluster $j = 1, \ldots, J$ and any $a \in \{a_0, a_1\}$, $D_{i,j}(1, a) \geq D_{i,j}(0, a)$, and strict inequality exists for at least one experimental unit.

This assumption was also considered by Imai et al. (2021) and Forastiere et al. (2016). Monotonicity eliminates the possibility of defiers under either treatment assignment probabilities $a_0$ and $a_1$. It also reduces the number of principal strata from sixteen to nine.

In addition to this existing monotonicity assumption, we also consider the following new assumption on compliance behaviors across the different treatment assignment probabilities that can be assigned to the clusters.

**Assumption 5.** The two sets \{\text{n, c, a}\} and \{\text{a_0, a_1}\} are partial order sets. For $a_0 < a_1$, units whose clusters have been assigned $a_1$ would have a non-strictly lower compliance type of \{n, c, a\} than the current compliance type if their clusters were assigned $a_0$. Units whose clusters have been assigned $a_0$ would have a non-strictly greater compliance type of \{n, c, a\} than the current compliance type if their clusters were assigned $a_1$.

This assumption further reduces the number of principal strata. To see this, we first recognize that there are only six possible definitions of partial orders for \{n, c, a\}: $n \leq c \leq a$, $n \leq a \leq c$, $c \leq n \leq a$, $c \leq a \leq n$, $a \leq n \leq c$, and $a \leq c \leq n$. We adopt the partial orders $n \leq c \leq a$ as well as $a_0 \leq a_1$ throughout. These correspond to units being more likely to take treatment if a larger proportion of their neighbors take treatment. Alternatively, if a cluster is assigned $a_1$ then its units are more likely to receive treatment, no matter what the units are assigned, compared to the case if the cluster is assigned $a_0$. The combination of Assumptions 4 and 5 thus reduces the number of principal strata to six: \{(n, n), (c, c), (a, a), (n, c), (n, a), (c, a)\}. Table 1 contains the one-to-one correspondence between the six possible principal strata and the values of $D_{i,j}(z, a)$ for $z \in \{0, 1\}, a \in \{a_0, a_1\}$. Without loss of generality, these orderings can be rearranged.
Table 1: Summary of the six principal strata defined according to the combinations of $D_{i,j}(z, a)$ for $z \in \{0, 1\}$ and $a \in \{a_0, a_1\}$ under Assumptions 4 and 5.

|              | $D_{i,j}(0, a_0)$ | $D_{i,j}(1, a_0)$ | $D_{i,j}(0, a_1)$ | $D_{i,j}(1, a_1)$ |
|--------------|-------------------|-------------------|-------------------|-------------------|
| $(n, n)$     | 0                 | 0                 | 0                 | 0                 |
| $(c, c)$     | 0                 | 1                 | 0                 | 1                 |
| $(a, a)$     | 1                 | 1                 | 1                 | 1                 |
| $(n, c)$     | 0                 | 0                 | 0                 | 1                 |
| $(n, a)$     | 0                 | 0                 | 1                 | 1                 |
| $(c, a)$     | 0                 | 1                 | 1                 | 1                 |

dependning on the context. The plausibility of an adopted ordering should be judged either by expert knowledge or by some established domain knowledge.

Our definition of principal strata based on Assumptions 4 and 5 is more general than existing definitions. For example, Forastiere et al. (2016) only defined three principal strata based on the treatment uptake status, excluding defiers. Also, Vazquez-Bare (2022) defined five principal strata by posing monotonicity directly on treatment received, which led to the removal of strata $(n, a)$ from consideration. These distinctions exist because we consider the two-stage randomized design, and we define the monotonicity assumptions on the compliance behaviors with respect to the treatment probabilities for clusters. Forastiere et al. (2016) considered clustered encouragement designs (CEDs) in which encouragement is randomized at the level of clusters but with no randomization carried out within clusters. In contrast, the two-stage randomized design has encouragement randomized at the level of units within clusters, with clusters assigned a treatment probability that governs the proportion of treated units within the cluster. The latter design generates more complicated structures for the units’ compliance behaviors because some units might behave differently based on the treatment probabilities assigned to their clusters. This distinction is critical because of our need to capture behavioral shifts of units between such treatment probabilities, and because of the direct and spillover causal estimands of interest that are defined in Section 2.4.
In addition to monotonicity, we also consider the exclusion restriction for certain principal strata in the case of nonadherence.

**Assumption 6.** For any unit \( i = 1, \ldots, N_j \) in cluster \( j = 1, \ldots, J \):

- if \( G_{i,j} \in \{(a,a),(n,n),(n,a)\} \), then \( Y_{i,j}(0,a) = Y_{i,j}(1,a) \) for \( a \in \{a_0, a_1\} \),
- if \( G_{i,j} = (c,a), Y_{i,j}(0,a_1) = Y_{i,j}(1,a_1) \), and
- if \( G_{i,j} = (n,c), Y_{i,j}(0,a_0) = Y_{i,j}(1,a_0) \).

This assumption captures the idea that treatment assignment has no effect on the outcome if the unit is either an always-taker or a never-taker under each treatment assignment probability. It also corresponds to Assumption 1 from which the outcome of an unit is determined only through the treatment received by units within the same cluster.

2.4 Direct, Spillover, and Overall Causal Estimands

We consider the finite-population framework for causal estimands under the RCM. In this case, estimands are defined in terms of comparisons of potential outcomes for the \( N \) experimental units. Our approach for defining direct, spillover, and overall causal estimands in two-stage randomized experiments with interference and nonadherence follows that of Hudgens and Halloran (2008) and Imai et al. (2021).

To simplify our expressions of our causal estimands, we first write the potential outcomes for unit \( i \) in cluster \( j \) under treatment assignment \( z \) as \( D_{i,j}(z), Y_{i,j}(z, D(z)) \), respectively. We let \( K_j(a) \) denote the number of treated units in cluster \( j \) under treatment assignment probability \( a \in \{a_0, a_1\} \), and \( \mathcal{Z}_{-ij} \) denote the set of all subvectors of \( z_j \in \{0,1\}^{N_j} \) with the \( i \)th element removed such that \( \sum_{i=1}^{N_j} z_{i,j} = K_j(a) \). Under Assumptions 2 and 3 we
recognize that
\[
D_{i,j}(z,a) = \sum_{z_{-i,j} \in Z_{-i,j}} D_{i,j}(z)p(Z_{-i,j} = z_{-i,j} \mid Z_{i,j} = z, A_j = a) = D_{i,j}(z, a)
\]
\[
Y_{i,j}(z,a) = \sum_{z_{-i,j} \in Z_{-i,j}} Y_{i,j}(z, D(z))p(Z_{-i,j} = z_{-i,j} \mid Z_{i,j} = z, A_j = a) = Y_{i,j}(z, a).
\]

One set of causal estimands that we consider are the unit-level direct intention-to-treat (ITT) effects of treatment assignment on treatment received and the final outcome under treatment assignment probability \(a \in \{a_0, a_1\}\), i.e., ITT_{D,i,j}(a) = \bar{D}_{i,j}(1,a) - \bar{D}_{i,j}(0,a) and ITT_{Y,i,j}(a) = \bar{Y}_{i,j}(1,a) - \bar{Y}_{i,j}(0,a), respectively. These estimands capture the adherence behavior and changes in the final outcome under the same treatment assignment probability \(a\) when unit \(i\) in cluster \(j\) is assigned treatment as opposed to control. We average the unit-level effects to define the cluster-level and finite population-level ITT effects as ITT_{D,j}(a) = \sum_{i=1}^{N_j} ITT_{D,i,j}(a)/N_j, ITT_{D,\cdot}(a) = \sum_{j=1}^{J} N_j ITT_{D,j}(a)/N, ITT_{Y,j}(a) = \sum_{i=1}^{N_j} ITT_{Y,i,j}(a)/N_j, ITT_{Y,\cdot}(a) = \sum_{j=1}^{J} N_j ITT_{Y,j}(a)/N.

Another causal estimand of interest in the presence of interference is the spillover (indirect) effect of treatments assigned to other units on the potential outcomes for a particular experimental unit. Following Imai et al. (2021) we define unit-level spillover effects on treatment receipt and outcome as \(S_{D,i,j}(z) = \bar{D}_{i,j}(z, a_1) - \bar{D}_{i,j}(z, a_0)\) and \(S_{Y,i,j}(z) = \bar{Y}_{i,j}(z, a_1) - \bar{Y}_{i,j}(z, a_0)\). In these two estimands, we consider the average potential outcomes corresponding to the two different treatment assignment probabilities \(a_0\) and \(a_1\) for cluster \(j\) but the same treatment assignment \(z\) for unit \(i\) in cluster \(j\). These estimands quantify the intuition that, if differences exist in potential outcomes under the same treatment assignment \(z\), then they can be attributed to the first stage of the experiment that governs the proportion of treated units in each cluster. This is because under Assumption the treated units in a particular unit \(i\)'s cluster are the only factor besides the assigned treatment that can affect unit \(i\)'s outcomes, and so the \(S_{D,i,j}(z)\) and \(S_{Y,i,j}(z)\) can be reasonably regarded as spillover effects. We define cluster-level and population-level spillover effects by averaging
the $S_{D,i,j}(z)$ and $S_{Y,i,j}(z)$ across $i$ and $j$, respectively.

The final estimand that we consider is the overall effect of the first stage of the experiment, i.e., the effect of having treatment assignment probability $a_1$ versus $a_0$ for a cluster. This effect is usually of greatest interest for policy makers. For example, infectious disease experts may be interested in comparing infection rates under two different vaccine allocation plans (e.g., 40% and 80%) within each cluster. We define unit-level overall effects of the first stage on treatment receipt and outcome as $O_{D,i,j} = D_{i,j}(a_1) - D_{i,j}(a_0)$ and $O_{Y,i,j} = Y_{i,j}(a_1) - Y_{i,j}(a_0)$, respectively, where

$$
\bar{Y}_{i,j}(a) = \sum_{z_j \in Z_j} Y_{i,j}(z) p(Z_j = z_j | A_j = a).
$$

$Y_{i,j}(a)$ is effectively the average value of the individual’s outcome under the treatment assignment probability $a \in \{a_0, a_1\}$. It is decomposed into

$$
\bar{Y}_{i,j}(a) = \left( \frac{K_j(a)}{N_j} \right) \bar{Y}_{i,j}(1,a) + \left( \frac{N_j - K_j(a)}{N_j} \right) \bar{Y}_{i,j}(0,a). \tag{1}
$$

The proof of (1) is provided in the supplementary material. Plugging into $O_{Y,i,j}$, we have

$$
O_{Y,i,j} = \left\{ \frac{K_j(a_1)}{N_j} \right\} \text{ITT}_{Y,i,j}(a_1) - \left\{ \frac{K_j(a_0)}{N_j} \right\} \text{ITT}_{Y,i,j}(a_0) + S_{Y,i,j}(0).
$$

We average the $O_{Y,i,j}$ to define the cluster-level and population-level overall effects as $O_{Y,:j} = \sum_{i=1}^{N_j} O_{Y,i,j}/N_j$ and $O_{Y,:} = \sum_{j=1}^{J} N_j O_{Y,:j}/N$. VanderWeele and Tchetgen (2011) provided the same decomposition as ours, but with a slightly different proof. The overall effect is expressed as the sum of the spillover effect and the contrast of two ITT effects under the treatment assignment probability $a_1$ and under the treatment assignment probability $a_0$, each of which are multiplied by the proportion of treated units under that assignment probability.
2.5 Principal Causal Estimands

In addition to defining direct, spillover, and overall causal estimands, we define new principal causal estimands. These estimands extend those in Section 2.4 to account for compliers under the different treatment probabilities $a_0$ and $a_1$ that can be assigned to clusters.

Imai et al. (2021) used Assumption 3 to define the complier average direct effect under treatment probability $a \in \{a_0, a_1\}$ as

$$\frac{\sum_{j=1}^{J} \sum_{i=1}^{N_j} \left( Y_{i,j}(1, a) - Y_{i,j}(0, a) \right) \mathbb{1}(G_{i,j}(a) = c) }{\sum_{j=1}^{J} \sum_{i=1}^{N_j} \mathbb{1}(G_{i,j}(a) = c) }.$$  \hspace{1cm} (2)

where $\mathbb{1}(\cdot)$ denotes the indicator for an event. We define a new complier average direct effect by generalizing the estimand in equation (2) to account for how experimental units comply under the different treatment probabilities assigned to clusters. This new definition is motivated by the practical interest in modifying the effect of $a_1$ to account for the units who would comply under $a_0$, and vice versa. To define this new complier average direct effect, we introduce the notions of the “base cluster assignment” as the treatment probability for a cluster under which compliers are defined, and the “target cluster assignment” as the treatment probability for a cluster under which causal effects are considered. It is important to separate the base from the target cluster assignments because the treatments received by units are influenced by the treatments assigned to other units, and because compliance behaviors vary with the treatment probabilities $a_0$ and $a_1$. For a target cluster assignment $a$ and base cluster assignment $a'$, our new population-level complier average direct effect is defined as

$$\text{CADE}(a, a') = \left[ \sum_{j=1}^{J} \sum_{i=1}^{N_j} \left( Y_{i,j}(1, a) - Y_{i,j}(0, a) \right) \mathbb{1}(G_{i,j}(a') = c) \right] \left/ \sum_{j=1}^{J} \sum_{i=1}^{N_j} \mathbb{1}(G_{i,j}(a') = c) \right.$$  \hspace{1cm} (3)

Equation (2) is a special case of equation (3) with $a = a'$.

The complier average spillover effect for treatment $z$ defined by Imai et al. (2021) is
expressed as
\[
\left[ \sum_{j=1}^{J} \sum_{i=1}^{N_j} \{Y_{i,j}(z, a_1) - Y_{i,j}(z, a_0)\} \mathbb{1}(G_{i,j} \in \{(c, a), (n, a)\}) \right] / \left[ \sum_{j=1}^{J} \sum_{i=1}^{N_j} \mathbb{1}(G_{i,j} \in \{(c, a), (n, a)\}) \right].
\]
This estimand is interpreted as a local average treatment effect for units who comply with the treatment probability assigned to the cluster. These estimands are not clearly interpretable as complier spillover effects because the compliance is defined for the compliance behaviors with respect to treatment probability, not the actual treatment assignment. We define a new complier average spillover effect using base and target cluster assignments.

For treatment \( z \) and base cluster assignment \( a' \), we define
\[
\text{CASE}(z, a') = \left[ \sum_{j=1}^{J} \sum_{i=1}^{N_j} \{Y_{i,j}(z, a_1) - Y_{i,j}(z, a_0)\} \mathbb{1}(G_{i,j}(a') = c) \right] / \left[ \sum_{j=1}^{J} \sum_{i=1}^{N_j} \mathbb{1}(G_{i,j}(a') = c) \right].
\]
Similar to the population-level spillover effect on outcome defined in Section 2.4, \( \text{CASE}(z, a') \) captures the compliers’ local average spillover effect by comparing the two sets of potential outcomes under the two cluster assignments \( a_0 \) and \( a_1 \) and treatment \( z \). In this estimand, the compliers are defined under the base cluster assignment \( a' \), and it is interpretable as an average effect for the compliers of the treatment received by others in the same cluster.

We finally define the finite-population complier average overall effect by combining equation (1) with the unit-level complier average overall effect under the base cluster assignment \( a' \), \( \{\tilde{Y}_{i,j}(a_1) - \tilde{Y}_{i,j}(a_0)\} \mathbb{1}(G_{i,j}(a') = c) \), and decomposing the effect according to the sum of the unit-level complier average direct and spillover effects. Specifically, we have
\[
\text{CAOE}_{i,j}(a') = \left\{ \frac{K_j(a_1)}{N_j} \right\} \{Y_{i,j}(1, a_1) - Y_{i,j}(0, a_1)\} \mathbb{1}(G_{i,j}(a') = c) - \left\{ \frac{K_j(a_0)}{N_j} \right\} \{Y_{i,j}(1, a_0) - Y_{i,j}(0, a_0)\} \mathbb{1}(G_{i,j}(a') = c) + \{Y_{i,j}(0, a_1) - Y_{i,j}(0, a_0)\} \mathbb{1}(G_{i,j}(a') = c).
\]
The population-level effect is then the average of the unit-level \( \text{CAOE}_{i,j}(a') \) over compliers under the base cluster assignment \( a' \):
\[
\text{CAOE}(a') = \sum_{j=1}^{J} \sum_{i=1}^{N_j} \text{CAOE}_{i,j}(a') / \left( \sum_{j=1}^{J} \sum_{i=1}^{N_j} \mathbb{1}(G_{i,j}(a') = c) \right).
\]
3 Bayesian Model and Inferential Approach

3.1 Overview of Methodology

Our Bayesian methodology for performing causal inferences on two-stage randomized experiments with interference and nonadherence is based on model-based imputation of missing potential outcomes to derive the posterior distributions of the finite-population causal estimands. Formally, if we let $\tau$ denote one of the causal estimands from Sections 2.4 and 2.5, $X$ the $N \times P$ matrix of covariates for all experimental units, $A^{\text{obs}}$ the $N \times 1$ vector of treatment probabilities assigned to the clusters for the units, $Z^{\text{obs}}$ the $N \times 1$ vector of treatments assigned to the units, $D^{\text{obs}}$ the $N \times 1$ vector of treatments received by the units in the realized experiment, and $Y^{\text{obs}}$ the observed outcomes in the experiment, then the Bayesian methodology calculates the distribution $p(\tau \mid X, A^{\text{obs}}, Z^{\text{obs}}, D^{\text{obs}}, Y^{\text{obs}})$. As causal estimands are functions of the observed and missing outcomes, we calculate the posterior distribution above by integrating over the missing data according to

$$p(\tau \mid X, A^{\text{obs}}, Z^{\text{obs}}, D^{\text{obs}}, Y^{\text{obs}}) = \int \left\{ p(\tau \mid X, A^{\text{obs}}, Z^{\text{obs}}, D^{\text{obs}}, G^{\text{mis}}, Y^{\text{obs}}, Y^{\text{mis}}) \times p(G^{\text{mis}}, Y^{\text{mis}} \mid X, A^{\text{obs}}, Z^{\text{obs}}, D^{\text{obs}}, Y^{\text{obs}}) \right\} dG^{\text{mis}} dY^{\text{mis}},$$

where $G$ is the $N \times 2$ matrix of (latent) principal strata memberships for the experimental units and $G^{\text{mis}}$ is the submatrix of $G$ that corresponds to those experimental units whose principal strata are not observed. This motivates our Bayesian methodology of first deriving a Monte Carlo approximation for the posterior predictive distribution of the missing principal strata and outcomes conditional on the observed data, and then using that posterior predictive distribution to derive a Monte Carlo approximation for the posterior distribution of $\tau$. Our methodology is distinct from existing methods for two-stage ran-
domized experiments in that we impute missing principal strata memberships and at most three missing potential outcomes for each experimental unit according to the assumptions in Section 2.

In Section 3.2 we describe how imputation of the missing principal strata is facilitated by the assumptions in Section 2. Our Bayesian modeling approach, and the unconfoundedness condition underlying it that is justified by the design of the two-stage randomized experiment, is outlined in Section 3.3. The Gibbs sampling algorithm that we use to derive a Monte Carlo approximation to the posterior distribution of the causal estimand is in Section 3.4. Section A.2 discusses the role of exchangeability in our Bayesian methodology.

3.2 Imputation of Missing Values

By virtue of Assumptions 4 and 5 any experimental units in the two-stage randomized experiment belong to one of six principal strata. Table 2 presents the correspondence between the possible observed values of \((A_{i,j}^{\text{obs}}, Z_{i,j}^{\text{obs}}, D_{i,j}^{\text{obs}})\) and principal strata memberships.

Let \(G(a, z, d)\) denote a set of possible principal strata for units with \((A_{i,j}^{\text{obs}}, Z_{i,j}^{\text{obs}}, D_{i,j}^{\text{obs}}) = (a, z, d)\). Assumption 5 in particular helps to narrow down the possible strata that a unit could belong to based on the observed data. For example, if an experimental unit \(i\) in cluster \(j\) has \(A_{j}^{\text{obs}} = a_0, Z_{i,j}^{\text{obs}} = 0, D_{i,j}^{\text{obs}} = 1\), then \(G(a_0, 0, 1) = \{(a, a)\}\). Hence, we can immediately conclude that \(G_{i,j} = (a, a)\). We note that four potential values of treatment receipt are associated with each individual: \(D_{i,j}(0, a_0), D_{i,j}(1, a_0), D_{i,j}(0, a_1),\) and \(D_{i,j}(1, a_1)\). Once \(G_{i,j}\) is fixed, we can immediately impute all missing \(D_{i,j}^{\text{mis}}\).

We also impute at most three missing potential outcomes, \(Y_{i,j}^{\text{mis}}\), for each unit. For example, if the principal stratum for unit \(i\) in cluster \(j\) is \(G_{i,j} = (c, c)\), there are four potential outcomes with one of them being observed. Therefore, the remaining three missing potential outcomes need to be imputed by the outcome models. If \(G_{i,j} = (a, a)\), however, we only need to impute one missing potential outcome because we can restrict the potential
Table 2: Summary of the possible principal strata that an experimental unit can belong to under different values of $A_{ij}^{\text{obs}}, Z_{ij}^{\text{obs}},$ and $D_{ij}^{\text{obs}}$.

| $A_{ij}^{\text{obs}}$ | $Z_{ij}^{\text{obs}}$ | $D_{ij}^{\text{obs}}$ | $G(A_{ij}^{\text{obs}}, Z_{ij}^{\text{obs}}, D_{ij}^{\text{obs}})$ |
|--------------------|--------------------|--------------------|-----------------------------|
| a_0                | 0                  | 0                  | (c, c), (n, n), (c, a), (n, a) |
| a_0                | 1                  | 0                  | (n, n), (n, c), (n, a)     |
| a_0                | 0                  | 1                  | (a, a)                     |
| a_0                | 1                  | 1                  | (c, c), (a, a), (c, a)    |
| a_1                | 0                  | 0                  | (c, c), (n, n), (n, c)    |
| a_1                | 1                  | 0                  | (n, n)                     |
| a_1                | 0                  | 1                  | (a, a), (c, a), (n, a)    |
| a_1                | 1                  | 1                  | (c, c), (a, a), (c, a), (n, c), (n, a) |

outcomes by invoking Assumption 3.

3.3 Bayesian Model Building

Following the Bayesian paradigm of Imbens and Rubin (2015) and Gelman et al. (2013), four inputs are necessary for deriving the posterior distributions of the causal estimands.

Note that $D$ and $Y$ are the $N \times 4$ matrices for potential values of treatment receipts and potential values for all experimental units respectively. The first is knowledge of the assignment mechanisms, as encoded in the probability mass functions $p(A \mid X)$ and $p(Z \mid X, A)$. By virtue of the known design of the two-stage randomized experiment, these two probability mass functions are obtained immediately. In addition, these two probability mass functions are independent of the experimental units’ covariates $X$, so that $p(A \mid X) = p(A)$ and $p(Z \mid X, A) = p(Z \mid A)$. The second input is the model for treatment received conditional on $X, A, Z$. We denote this input by the probability mass function $p(D \mid X, A, Z, \psi)$ with parameter vector $\psi$. An equivalent input is the model for principal strata memberships of all experimental units, $G$, conditional on $X, A, Z$ because there is a one-to-one mapping between $G$ and $D$. To simplify our notation we denote this input as $p(G \mid X, A, Z, \psi)$ with the understanding that $\psi$ is generic notation for the parameter vector associated with either of these two models. The third input is the
model for the potential outcomes conditional on $X, A, Z, D$. We denote this model via a probability density function $p(Y | X, A, Z, D, \theta)$ with parameter vector $\theta$. This model can also be equivalently formulated using $G$ as $p(Y | X, A, Z, G, \theta)$. The final input is the prior distribution for $\psi$ and $\theta$, denoted by $p(\psi, \theta)$. We assume throughout that $\psi$ and $\theta$ are distinct and do not share any common parameters.

Specifying the second and third inputs is facilitated by the fact that the two-stage randomized experiment has an unconfounded assignment mechanism at both cluster and individual experimental unit levels. Noting that $D_{i,j} = (D_{i,j}(0, a_0), D_{i,j}(0, a_1), D_{i,j}(1, a_0), D_{i,j}(1, a_1))$ and $Y_{i,j} = (Y_{i,j}(0, a_0), Y_{i,j}(0, a_1), Y_{i,j}(1, a_0), Y_{i,j}(1, a_1))$ are 4 dimensional vectors of potential values of treatment receipts and potential outcomes for each unit, unconfoundedness is formally expressed via the following assumption.

**Assumption 7.** For experimental unit $i = 1, \ldots, N_j$ in cluster $j = 1, \ldots, J$,

$$p(D_{i,j} | X, A, Z) = p(D_{i,j} | X), \quad p(Y_{i,j} | X, A, Z, D_{i,j}) = p(Y_{i,j} | X, D_{i,j})$$

Assumption [7] also implies $p(Y_{i,j} | X, A, Z, G_{i,j}) = p(Y_{i,j} | X, G_{i,j})$. Assumption [7] implies that it is not necessary to incorporate the actual treatment assignment mechanism in the likelihood function. This effectively helps to automate the model fitting process. In addition, unconfoundedness and unit exchangeability imply de Finetti’s Theorem that we can specify models on the individual experimental unit-level for the principal strata memberships $G_{i,j}$ and the potential outcomes $Y_{i,j}(z, a)$ to derive the joint model for $G$ and $Y$ as in (3.3), which further helps to simplify Bayesian modeling. A justification for the assumption of unit exchangeability in the two-stage randomized experiment is in the supplementary material A.2. By appealing de Finetti’s Theorem, we have

$$p(Y, D | X) = \int \prod_{i,j} p(D_{i,j} | X_{i,j}, \psi) p(Y_{i,j} | X_{i,j}, D_{i,j}, \theta) p(\psi, \theta) d\psi d\theta$$

(4)
The posterior distribution of \( \psi \) and \( \theta \) can be written as

\[
p (\psi, \theta | X, A^{obs}, Z^{obs}, D^{obs}, Y^{obs}) \propto p(\psi, \theta) \int \prod_{i,j} p(D_{i,j} | X_{i,j}, \psi) p(Y_{i,j} | X_{i,j}, D_{i,j}, \theta) dY^{mis} dD^{mis}
\]

Let \( O(A^{obs}_{i,j}, Z^{obs}_{i,j}, D^{obs}_{i,j}) \) denote the observed group defined by the observed variables \( A^{obs}_{i,j} \), \( Z^{obs}_{i,j} \) and \( D^{obs}_{i,j} \). Define \( w^g_{i,j} = p (G_{i,j} = g | X_{i,j}, \psi) \) and let \( f^{g,a,z}_{i,j} \) be the probability mass/density function of \( Y_{i,j}(z, a) \) for \( g = (n, n), (c, c), (a, a), (n, c), (n, a), (c, a) \). To perform the above integration under Assumptions \( \Delta \| \) the posterior distribution of \( \psi \) and \( \theta \) can be written as follows.

\[
p (\psi, \theta | X, A^{obs}, Z^{obs}, D^{obs}, Y^{obs}) \propto p(\psi, \theta)
\]

\[
\times \prod_{(i,j) \in O(a_0, 0, 0)} \left( w^{(c,c)}_{i,j} f^{(c,c)}_{i,j,0,0} + w^{(n,n)}_{i,j} f^{(n,n)}_{i,j,0,0} + w^{(c,a)}_{i,j} f^{(c,a)}_{i,j,0,0} + w^{(n,c)}_{i,j} f^{(n,c)}_{i,j,0,0} + w^{(n,a)}_{i,j} f^{(n,a)}_{i,j,0,0} \right)
\]

\[
\times \prod_{(i,j) \in O(a_0, 1, 0)} \left( w^{(c,c)}_{i,j} f^{(c,c)}_{i,j,1,0} + w^{(n,n)}_{i,j} f^{(n,n)}_{i,j,1,0} + w^{(c,a)}_{i,j} f^{(c,a)}_{i,j,1,0} \right) \times \prod_{(i,j) \in O(a_0, 0, 1)} \left( w^{(a,a)}_{i,j} f^{(a,a)}_{i,j,0,1} \right)
\]

\[
\times \prod_{(i,j) \in O(a_1, 0, 0)} \left( w^{(c,c)}_{i,j} f^{(c,c)}_{i,j,0,0} + w^{(n,n)}_{i,j} f^{(n,n)}_{i,j,0,0} + w^{(n,c)}_{i,j} f^{(n,c)}_{i,j,0,0} \right) \times \prod_{(i,j) \in O(a_1, 1, 0)} \left( w^{(n,n)}_{i,j} f^{(n,n)}_{i,j,1,0} \right)
\]

\[
\times \prod_{(i,j) \in O(a_1, 0, 1)} \left( w^{(a,a)}_{i,j} f^{(a,a)}_{i,j,0,1} + w^{(c,a)}_{i,j} f^{(c,a)}_{i,j,0,1} + w^{(n,a)}_{i,j} f^{(n,a)}_{i,j,0,1} \right)
\]

\[
\times \prod_{(i,j) \in O(a_1, 1, 1)} \left( w^{(c,c)}_{i,j} f^{(c,c)}_{i,j,1,1} + w^{(n,c)}_{i,j} f^{(n,c)}_{i,j,1,1} + w^{(n,a)}_{i,j} f^{(n,a)}_{i,j,1,1} + w^{(n,a)}_{i,j} f^{(n,a)}_{i,j,1,1} \right)
\]

Therefore, our model-based Bayesian inference requires to specify two models: \( w^g_{i,j} \) and \( f^{g,a,z}_{i,j} \). Our models and prior distributions are provided in Section \( \| \) and \( 5 \).

An important advantage of our Bayesian methodology compared to existing frequentist methods is that it clarifies what can be learned when causal estimands are not identifiable but are instead weakly identifiable, in the sense that the likelihood functions of parameters and causal estimands have substantial regions of flatness. In addition, issues of identifiability under the Bayesian paradigm are distinct from those under the frequentist paradigm because the specification of proper prior distributions always yields proper posterior distributions (Imbens and Rubin, 1997). Accordingly, our Bayesian methodology enables us
to infer principal causal effects under two-sided noncompliance, whereas existing methods requires additional strict assumptions to perform inferences for principal causal effects under two-sided noncompliance including always-takers and social-interaction compliers (Vazquez-Bare, 2022) (i.e., the strata \((a, a)\) and \((c, a)\)).

3.4 Gibbs Sampling Algorithm

We utilize the Gibbs sampling algorithm (Geman and Geman, 1984; Gelfand and Smith, 1990; Imbens and Rubin, 1997) to derive the joint posterior distribution of \(G^{\text{mis}}\) and \(Y^{\text{mis}}\) given the observed data, so as to impute the missing data and derive the posterior distributions for the causal estimands. Specifically, we iterate between drawing from the conditional distributions of \((\psi, \theta)\) and \(G^{\text{mis}}\) given the other variables respectively. Then for each iteration we impute \(Y^{\text{mis}}\) and calculate the causal estimands of interest to effectively get a draw from their posterior distribution. The algorithm proceeds as follows.

1. Initialize parameters \(\psi^{(0)}, \theta^{(0)}\).

2. For \(t = 0, 1, \ldots:\)

   (a) Draw \(G^{\text{mis},(t+1)} \sim p\left(G^{\text{mis}} \mid X, A^{\text{obs}}, Z^{\text{obs}}, D^{\text{obs}}, Y^{\text{obs}}, \psi^{(t)}, \theta^{(t)}\right)\).

   (b) Draw \(Y^{\text{mis},(t+1)} \sim p\left(Y^{\text{mis}} \mid X, A^{\text{obs}}, Z^{\text{obs}}, Y^{\text{obs}}, G^{\text{mis},(t+1)}, \psi^{(t)}, \theta^{(t)}\right)\).

   (c) Calculate the causal estimand based on the observed and imputed data.

   (d) Draw \(\left(\psi^{(t+1)}, \theta^{(t+1)}\right) \sim p\left(\psi, \theta \mid X, A^{\text{obs}}, Z^{\text{obs}}, Y^{\text{obs}}, G^{\text{mis},(t+1)}\right)\).

   (e) Repeat steps (a) - (e).

This Gibbs sampler enables us to obtain posterior draws of all the missing potential outcomes and principal strata for all units. We then perform causal inferences by means of the posterior distributions. For example, points estimates of the estimands can be obtained via their posterior means or medians, and intervals for the estimands can be obtained via
the central credible intervals. If tractable prior distributions and models are used, it will be possible to derive all the conditional posterior distributions involved in the Gibbs sampler in closed-form. Otherwise, Metropolis-within-Gibbs or Hamiltonian Monte Carlo-within-Gibbs steps will need to be considered in the algorithm. Detailed derivations for each step of the Gibbs sampler are provided in the supplementary material.

4 Simulation Studies

4.1 Evaluation Metrics and Data Generating Mechanisms

We evaluate the frequentist properties of our Bayesian methodology with respect to those of the method of Imai et al. (2021), which we implement using its R package experiment (Imai et al., 2019). The specific evaluation metrics that we consider are bias and mean square error (MSE) in estimating a causal estimand, coverage of an interval estimator for a causal estimand, and the interval length. Bias and MSE are generally defined as $\sum_{m=1}^{M} (\tau - \hat{\tau}_m) / M$ and $\sum_{m=1}^{M} (\tau - \hat{\tau}_m)^2 / M$ respectively, where $M$ denotes the number of simulated datasets, $\tau$ denotes the true causal estimand, and $\hat{\tau}_m$ denotes the estimate of the causal estimand in dataset $m = 1, \ldots, M$. For our Bayesian method, the point estimator is the median of the posterior distribution of a causal estimand, and the interval estimator is the 95% central credible interval. The interval length is the median of the credible intervals computed from $M$ simulated datasets. The data generating mechanisms that we consider in our study are specified to simulate data resembling those in our case study in Section 5.

For the two-stage randomized experiments in our simulation study, the clusters have equal numbers of units (i.e., $N_j = N/J$), and we specify $p(A_j = a_0) = p(A_j = a_1) = 0.5$, $p(Z_{i,j} = 1 \mid A_j = a_0) = 0.4$, and $p(Z_{i,j} = 1 \mid A_j = a_1) = 0.8$ for any experimental unit $i = 1, \ldots, N_j$ in cluster $j = 1, \ldots, J$. Each experimental unit belongs to just one of the latent principal strata $\{(n,n), (c,c), (a,a), (n,c), (n,a), (c,a)\}$. The principal strata member-
ships are generated according to $G_{i,j} \sim \text{Multinomial}\left(\pi_{(n,n)}, \pi_{(c,c)}, \pi_{(a,a)}, \pi_{(n,c)}, \pi_{(n,a)}, \pi_{(c,a)}\right)$.

The potential outcomes in our simulation study are continuous and are generated to have a right-skewed distribution with an excess of zeros. The specific generation mechanism that we implement is a zero-inflated Log-Normal distribution, with the parameters of the underlying Bernoulli and Log-Normal random variables (representing the excess zeros and heavy tail of the outcomes, respectively) specified to be distinct for each strata and treatment.

More formally, for $a \in \{a_0, a_1\}$ and $z \in \{0, 1\}$, the potential outcomes for unit $i$ in cluster $j$ are generated by first sampling $W_{i,j}(z, a) \sim \text{Bernoulli}(p_{z,a,G_{i,j}})$, then sampling $\tilde{Y}_{i,j}(z, a) \sim \text{Log-Normal}(\mu_{z,a,G_{i,j}}, \sigma^2_{z,a,G_{i,j}})$, and finally generating $Y_{i,j}(z, a) = \{1 - W_{i,j}(z, a)\} \tilde{Y}_{i,j}(z, a)$.

For this simulation study, we assume that potential outcomes are generated independently of one another. In addition, Assumption 6 applies throughout the data generating mechanism, including for $W_{i,j}(z, a)$ and $\tilde{Y}_{i,j}(z, a)$. The generated $W_{i,j}(z, a)$ and $\tilde{Y}_{i,j}(z, a)$ are not recorded as data. Finally, we use conjugate prior distributions for all parameters, that is, $\left(\pi_{(n,n)}, \pi_{(c,c)}, \pi_{(a,a)}, \pi_{(n,c)}, \pi_{(n,a)}, \pi_{(c,a)}\right) \sim \text{Dirichlet}(2, 2, 2, 2, 2, 2)$, $p_{z,a,G_{i,j}} \sim \text{Beta}(1, 1)$, $\mu_{z,a,G_{i,j}} \sim \text{Normal}(0, 10^2)$ and $\sigma^2_{z,a,G_{i,j}} \sim \text{InverseGamma}(1, 1)$. Detailed algorithms for the Gibbs sampler are provided in the appendix. All parameter values that are utilized in our simulation study are provided in Table 7 in the appendix. We simulate 500 datasets for each $N = 5000, 10000, 50000$ with a fixed cluster size of $J = 100$.

We also present an additional simulation study using the Gamma distribution for the outcome model instead of the Log-Normal distribution, namely, $\tilde{Y}_{i,j}(z, a) \sim \text{Gamma}(\alpha_{z,a,G_{i,j}}, \theta_{z,a,G_{i,j}})$, where $\alpha$ and $\theta$ are the shape and scale parameters with prior distributions $\alpha_{z,a,G_{i,j}} = \tilde{\alpha}_{z,a,G_{i,j}} \mathbb{1}(\tilde{\alpha}_{z,a,G_{i,j}} > 0)$ where $\tilde{\alpha}_{z,a,G_{i,j}} \sim \text{Normal}(1, \sqrt{1000})$ and $\theta_{z,a,G_{i,j}} \sim \text{InverseGamma}(1, 100)$ respectively. This additional scenario serves to check if our approach consistently provides comparable results with the frequentist approach under different data generating processes, in which the frequentist approach is supposed to perform well since the gamma distribution with the specific parameterizations is not heavily skewed and does not yield many outliers.
For the frequentist evaluations, we also consider the super-population versions of the causal estimands that we defined in Sections 2.4 and 2.5. Additional investigation into the robustness of our Bayesian methodology to model misspecifications is left for future study.

4.2 Results

Table 3 summarizes the results of our simulation study using the Log-Normal distribution. The method of Imai et al. (2021) is abbreviated as “IJM” in this table. We observe that both methods perform well with respect to coverage and bias, with the IJM method exhibiting less bias than our Bayesian method for small sample sizes. This difference in bias can be attributed to the effect of the prior distributions, which is not negligible for small \( N \) given the small number \( N_j = 50 \) of units in each cluster. In addition, the posterior median is not an unbiased estimator of the mean of the Log-Normal distribution, although it is arguably a desirable estimator as it should be more robust for the chosen data generating mechanism. This difference in bias should also be expected as the emphasis of the Bayesian approach is on the posterior distribution and whether it is well-calibrated. Finally, we recognize that our Bayesian methodology outperforms the IJM method with respect to MSE under all conditions. The differences in MSE imply that the Bayesian point estimator is less likely to deviate from the true causal effect over multiple experiments as compared to the IJM method, that the Bayesian estimator has less variability, and that the Bayesian intervals have smaller widths. Ultimately, the Bayesian methodology yields more precise causal inferences. The IJM method is sensitive to the shape of the distribution and exhibits greater variability and MSEs as it attempts to accommodate outliers.

The table also summarizes the results of our simulation study using the Gamma distribution. The Bayesian approach is well-calibrated in the sense that it yields nearly 95% coverages across all conditions. It also has the same level of Bias as the frequentist model, however, it sometimes yields slightly larger bias due to the same reasons as discussed above.
Table 3: Evaluation metrics for our Bayesian methodology versus the method of Imai et al. (2021) (abbreviated as “IJM”) for the Log-Normal (left) and Gamma (right) data-generating processes.

| Coverage | Bias | MSE | Interval Width | Coverage | Bias | MSE | Interval Width |
|----------|------|-----|----------------|----------|------|-----|----------------|
| Log-Normal |       |     |                | Gamma    |      |     |                |
| N | IJM Bayes | IJM Bayes | IJM Bayes | IJM Bayes | IJM Bayes | IJM Bayes | IJM Bayes | IJM Bayes | IJM Bayes | IJM Bayes | IJM Bayes | IJM Bayes | IJM Bayes | IJM Bayes | IJM Bayes | IJM Bayes | IJM Bayes |
| 5000 | 96% | 97% | 4.34E+02 | 5.02E+02 | 3.69E+00 | 4.75E+00 | 1.91E+04 | 9.14E+04 | 90% | 95% | 3.26E+01 | -1.93E+00 | 1.22E+01 | 1.76E+01 | 1.82E+01 | 1.67E+02 |
| IJM | 5000 | 96% | 97% | -2.52E+02 | 2.93E+02 | 1.35E+07 | 1.46E+07 | 1.84E+04 | 5.41E+04 | 92% | 96% | 1.54E+01 | -2.37E+00 | 1.34E+01 | 9.64E+01 | 1.28E+02 | 1.16E+02 |
| CADE(0, a) | 10000 | 95% | 97% | 2.12E+02 | 8.92E+02 | 5.97E+00 | 4.53E+00 | 6.64E+03 | 2.90E+03 | 96% | 96% | 1.54E+01 | -2.37E+00 | 1.34E+01 | 9.64E+01 | 1.28E+02 | 1.16E+02 |
| IJM | 50000 | 95% | 97% | 4.81E+02 | 2.98E+02 | 2.18E+07 | 7.75E+05 | 1.68E+05 | 4.81E+03 | 95% | 95% | 1.54E+01 | -2.37E+00 | 1.34E+01 | 9.64E+01 | 1.28E+02 | 1.16E+02 |
| CADE(1, a) | 10000 | 95% | 97% | 1.68E+02 | 2.46E+02 | 9.11E+06 | 1.20E+06 | 9.63E+03 | 4.68E+03 | 91% | 95% | 1.68E+02 | -2.40E+00 | 6.14E+02 | 3.31E+02 | 7.35E+01 | 6.88E+01 |
| IJM | 5000 | 96% | 98% | 1.68E+02 | 2.46E+02 | 9.11E+06 | 1.20E+06 | 9.63E+03 | 4.68E+03 | 91% | 95% | 1.68E+02 | -2.40E+00 | 6.14E+02 | 3.31E+02 | 7.35E+01 | 6.88E+01 |
| IJM | 10000 | 96% | 98% | -1.21E+02 | 1.46E+02 | 3.49E+06 | 4.86E+05 | 6.86E+03 | 2.80E+03 | 92% | 94% | 6.96E+00 | -1.42E+00 | 2.78E+02 | 2.02E+02 | 5.80E+01 | 5.20E+00 |
| IJM | 50000 | 93% | 96% | -5.61E+01 | 8.06E+01 | 9.08E+05 | 7.39E+04 | 3.32E+04 | 1.08E+03 | 90% | 94% | -3.46E+00 | 7.52E-01 | 6.25E+01 | 5.98E+01 | 2.58E+01 | 2.31E+01 |
| IJM | 5000 | 96% | 98% | -4.54E+02 | 6.00E+02 | 4.62E+07 | 1.42E+07 | 2.03E+04 | 7.74E+03 | 92% | 96% | -3.85E+00 | 2.29E+00 | 3.39E+02 | 2.88E+02 | 7.17E+01 | 6.57E+01 |
| IJM | 10000 | 94% | 97% | 1.18E+02 | 4.41E+02 | 2.64E+07 | 8.34E+05 | 1.52E+04 | 5.30E+03 | 96% | 96% | -3.85E+00 | 2.29E+00 | 3.39E+02 | 2.88E+02 | 7.17E+01 | 6.57E+01 |
| IJM | 50000 | 94% | 97% | 1.58E+02 | 6.00E+02 | 5.03E+05 | 1.21E+05 | 1.74E+04 | 5.28E+03 | 92% | 96% | -3.85E+00 | 2.29E+00 | 3.39E+02 | 2.88E+02 | 7.17E+01 | 6.57E+01 |
| IJM | 5000 | 96% | 97% | -7.57E-02 | 4.34E-03 | 3.25E-04 | 3.12E-04 | 8.96E-02 | 7.45E-02 | 95% | 91% | 7.29E-01 | -2.51E-03 | 1.74E-02 | 1.63E-02 | 5.13E-02 | 4.47E-02 |
| IJM | 10000 | 93% | 94% | -2.26E-04 | 4.41E-03 | 1.74E-04 | 2.12E-04 | 4.32E-02 | 4.68E-02 | 95% | 93% | 1.52E-01 | -2.51E-03 | 1.74E-02 | 1.63E-02 | 5.13E-02 | 4.47E-02 |
| IJM | 50000 | 94% | 96% | -6.32E-04 | 4.34E-03 | 3.12E-04 | 3.12E-04 | 8.96E-02 | 7.45E-02 | 95% | 96% | 7.29E-01 | -2.51E-03 | 1.74E-02 | 1.63E-02 | 5.13E-02 | 4.47E-02 |
| IJM | 5000 | 94% | 94% | -7.57E-01 | 4.34E-03 | 3.25E-04 | 3.12E-04 | 8.96E-02 | 7.45E-02 | 95% | 91% | 7.29E-01 | -2.51E-03 | 1.74E-02 | 1.63E-02 | 5.13E-02 | 4.47E-02 |
| IJM | 10000 | 93% | 93% | -2.26E-04 | 4.41E-03 | 1.74E-04 | 2.12E-04 | 4.32E-02 | 4.68E-02 | 95% | 93% | 1.52E-01 | -2.51E-03 | 1.74E-02 | 1.63E-02 | 5.13E-02 | 4.47E-02 |
| IJM | 50000 | 94% | 96% | -6.32E-04 | 4.34E-03 | 3.12E-04 | 3.12E-04 | 8.96E-02 | 7.45E-02 | 95% | 96% | 7.29E-01 | -2.51E-03 | 1.74E-02 | 1.63E-02 | 5.13E-02 | 4.47E-02 |

The Bayesian approach performs better than the frequentist approach in terms of MSE, even though the Gamma distribution does not produce as many outliers as the Log-Normal distribution does.

5 Case Study: The Rastriya Swasthya Bima Yojana Health Insurance Dataset

Approximately 63 million people are below the poverty line due to health care expenditures. In 2008 a large-scale national hospital insurance plan for the poor was launched. This plan is known as the Rastriya Swasthya Bima Yojana (RSBY). It is a large-scale national hospital insurance plan that households below the poverty line can join with a nominal co-payment. Under RSBY, these households can be covered for up to five people and more than 700 medical treatments and procedures, with the price set by the
government. Medical services are provided nationwide by government-contracted public and private hospitals. Beneficiaries use their RSBY biometric ID cards, eliminating the need for cash transactions and insurance claims. Additional information and references for RSBY are provided by Nandi et al. (2015).

Imai et al. (2021) conducted a two-stage randomized experiment to determine whether access to the national insurance plan provided by RSBY increases access to hospitalization and reduces impoverishment due to high medical expenses. This experiment consisted of \( N = 10,072 \) households in \( J = 435 \) villages, with \( J_1 = 219 \) villages assigned treatment probability \( a_1 = 0.8 \) and the remaining \( 216 \) villages assigned treatment probability \( a_0 = 0.4 \). Of concern in their experiment was the spillover effects between households, because one household’s enrollment in RSBY may depend on the treatments assigned to other households. Another concern is that some households assigned treatment may decide to not enroll in RSBY, and some households assigned control may ultimately manage to enroll in RSBY. We utilize our Bayesian methodology to infer the direct and spillover effects, accounting for interference and treatment nonadherence, on the annual household hospital expenditure outcome (which ranges from 0 to INR 500,000). We use the same mixture model as in Section 4.1 to analyze the RSBY dataset. The model for principal strata memberships is the Multinomial distribution, \( G_{i,j} \sim \text{Multinomial} \left( \pi_{(n,n)}, \pi_{(c,c)}, \pi_{(a,a)}, \pi_{(n,a)}, \pi_{(c,a)} \right) \). For the potential outcomes, we specify a zero-inflated Log-Normal distribution for each principal stratum, that is, for \( a \in \{a_0, a_1\} \) and \( z \in \{0, 1\} \), the potential outcomes for unit \( i \) in cluster \( j \) are determined by \( Y_{i,j}(z, a) = (1 - W_{i,j}(z, a)) \tilde{Y}_{i,j}(z, a) \) where \( W_{i,j}(z, a) \sim \text{Bernoulli}(p_{z,a,G_{i,j}}) \) and \( \tilde{Y}_{i,j}(z, a) \sim \text{Log-Normal}(\mu_{z,a,G_{i,j}}, \sigma_{z,a,G_{i,j}}^2) \). Finally, we use conjugate prior distributions for all parameters. \( \left( \pi_{(n,n)}, \pi_{(c,c)}, \pi_{(a,a)}, \pi_{(n,a)}, \pi_{(c,a)} \right) \sim \text{Dirichlet}(\alpha_{(n,n)}, \alpha_{(c,c)}, \alpha_{(a,a)}, \alpha_{(n,a)}, \alpha_{(c,a)}) \), \( p_{z,a,G_{i,j}} \sim \text{Beta}(a_0, b_0) \), \( \mu_{z,a,G_{i,j}} \sim \text{Normal}(\mu_0, \sigma_0^2) \) and \( \sigma_{z,a,G_{i,j}}^2 \sim \text{InverseGamma}(k_0, \theta_0) \) where \( \theta_0 \) is a scale parameter. For hyperparameters, we choose \( \alpha_{(n,n)} = \alpha_{(c,c)} = \alpha_{(a,a)} = \alpha_{(n,a)} = \alpha_{(c,a)} = 1 \), \( a_0 = b_0 = 1 \), \( \mu_0 = 0 \), \( \sigma_0^2 = 5 \), \( k_0 = 0.1 \) and \( \theta_0 = 1 \). Note that
\( \sigma_0^2 = 5 \) is sufficiently large on a log scale. Our MCMC algorithm was performed for 100,000 iterations with a burn-in of 50,000 draws.

We consider the finite-population inference following Imai et al. (2021). On the other hand, a salient feature of the super-population estimands is that they are free of the association parameters of potential outcomes. This is an advantage since the data are not informative about the association between potential outcomes because they are never jointly observed due to the fundamental problem of causal inference. Ding and Li (2018) suggested isolating the parameters, denoted by \( \theta_m \), that governs the marginal distributions from the parameters, denoted by \( \theta_a \), that governs the association between potential outcomes, and perform the sensitivity analysis about the association parameters. We omit the sensitivity analysis in this study but it is of our future interest to verify how sensitive our method is with respect to the association parameters.

Table 4 compares the results obtained from our Bayesian methodology with those obtained by the method of Imai et al. (2021). We do not consider the complier average spillover effects because we defined these estimands differently from Imai et al. (2021), and because Imai et al. (2021) mentioned that these estimands were imprecisely estimated in their analyses. We observe in Table 4 that our results are generally consistent with those

|                         | Mean | SD  | Median | 95% interval | IJM Est. | IJM SD |
|-------------------------|------|-----|--------|--------------|----------|--------|
| CADE\((a_1, a_1)\)      | −2041| 7247| −1813  | (−3782, −256) | −1649    | 1061   |
| CADE\((a_0, a_0)\)      | 298  | 3912| 158    | (−1356, 1982) | 1984     | 1215   |
| ITT\(Y, r, r, (a_1)\)  | −853 | 3040| −759   | (−1586, −106) | −795     | 514    |
| ITT\(Y, r, r, (a_0)\)  | 139  | 1811| 74     | (−632, 913)   | 875      | 530    |
| ITT\(D, r, r, (a_1)\)  | 0.418| 0.010| 0.418  | (0.397, 0.438) | 0.482    | 0.023  |
| ITT\(D, r, r, (a_0)\)  | 0.465| 0.009| 0.465  | (0.446, 0.483) | 0.441    | 0.021  |
| SY\(r, r, (1)\)        | −1129| 1795| −1071  | (−1741, −459) | −1374    | 823    |
| SY\(r, r, (0)\)        | −136 | 3044| −222   | (−1003, 666)  | 297      | 858    |
| SD\(r, r, (1)\)        | 0.030| 0.007| 0.029  | (0.018, 0.047) | 0.086    | 0.053  |
| SD\(r, r, (0)\)        | 0.077| 0.009| 0.077  | (0.060, 0.095) | 0.045    | 0.028  |
Table 5: Additional causal estimands and their estimates for the RSBY data.

| Estimand                   | Post. Mean | Post. Median | 95% Credible Interval |
|----------------------------|------------|--------------|-----------------------|
| $O_{Y,\cdot}$              | $-731$     | $-739$       | $(-1280, -181)$       |
| CAOE($a_0$)                | $-1242$    | $-1154$      | $(-2334, -30)$        |
| CAOE($a_1$)                | $-1234$    | $-1328$      | $(-2600, -75)$        |
| CADE($a_1, a_0$)           | $-1647$    | $-1626$      | $(-3329, -230)$       |
| CADE($a_0, a_1$)           | $259$      | $237$        | $(-1444, 2162)$       |
| CASE($0, a_0$)             | $-33$      | $-64$        | $(-1691, 1797)$       |
| CASE($1, a_0$)             | $-2004$    | $-1870$      | $(-3484, -548)$       |
| CASE($0, a_1$)             | $128$      | $-76$        | $(-1903, 1981)$       |
| CASE($1, a_1$)             | $-2172$    | $-2160$      | $(-3831, -694)$       |

of Imai et al. (2021). However, differences exist because our Bayesian method is able to detect significance effects in $CADE(a_1, a_1)$, $ITT_{Y,\cdot}(a_1)$, and $S_{Y,\cdot}(1)$. The negative spillover effects that our method detects indicates that treated households are more likely to be negatively affected by the shift from $a_0$ to $a_1$. Alternatively, assigning a greater proportion of households to treatment will cause another treated household in the same village to spend less. We also observe large posterior standard deviations, but the posterior intervals always have smaller widths than the IJM’s confidence intervals. The new effects that our methodology detects can be attributed to the greater precision (hence power) that follow from the use of the Bayesian model. Furthermore, our Bayesian methodology’s ability to consider a point estimator based on the median, and a model that accommodates both an abundance of zeros and heavy tails, is advantageous for analyzing the data as the inferences would be robust to outlying observations. In particular, there are 36 observations greater than INR 100,000, with the two largest ones being INR 403,000 and 500,000. For comparison, the median in the dataset is INR 1,000.

Table 5 summarizes the results for the other causal estimands. Interestingly, the credible intervals of the overall effects for all units, as well as for compliers, lie below zero. The overall effect is of greatest interest to policy makers as it captures a pure impact of the intervention on all units. Our inferences on compliers imply that the overall effects are negative for units who comply with the assignment regardless of which treatment probabil-
ity $a_0$ or $a_1$ is assigned to their respective cluster. We can also conclude from both Tables 4 and 5 that the direct effect of the treatment assignment under treatment probability $a_1$ is negative for compliers, regardless of their base cluster assignment. Finally, we can conclude that the spillover effects for compliers are negative when all units are assigned to treatment, regardless of whether they are compliers under $a_0$ or $a_1$. Combined with our inferences on $S_{Y_{i,j}}(1)$, we have that the spillover effect of treatment assignment on units is negative, regardless of their principal strata.

### 5.1 Sensitivity Analysis to Prior Specifications

When performing Bayesian analyses with weakly identifiable models, it is important to investigate the robustness of the results with respect to the prior specifications, so as to make inferences more reliable. The results in this section are derived using proper, weakly informative prior distributions. In particular, we use $\mu_{z,a,G_{i,j}} \sim \text{Normal}(0,5)$, $\sigma^2_{z,a,G_{i,j}} \sim \text{InverseGamma}(0.1,1)$, $(\pi_{(n,a)}, \pi_{(c,a)}, \pi_{(a,a)}, \pi_{(n,c)}, \pi_{(c,a)}) \sim \text{Dirichlet}(1,1,1,1,1,1)$, and $p_{z,a,G_{i,j}} \sim \text{Beta}(1,1)$ as the baseline prior distributions. We investigate the robustness of the results with respect to the prior specifications using more informative and less informative priors for the variance parameters. In particular, the less informative priors (Case 1) are specified by $\mu_{z,a,G_{i,j}} \sim \text{Normal}(0,\sqrt{7}^2)$ and $\sigma^2_{z,a,G_{i,j}} \sim \text{InverseGamma}(0.01,1)$. The more informative priors (Case 2) are specified by $\mu_{z,a,G_{i,j}} \sim \text{Normal}(0,\sqrt{3}^2)$ and $\sigma^2_{z,a,G_{i,j}} \sim \text{InverseGamma}(1,1)$. Note that $\sigma^2_0 = 3$ and $\sigma^2_0 = 7$ are substantially different from $\sigma^2_0 = 5$ of the baseline model on a log scale. We also investigate the sensitivity with respect of other parameters, namely, we consider Case 3 specified by $(\pi_{(n,a)}, \pi_{(c,a)}, \pi_{(a,a)}, \pi_{(n,c)}, \pi_{(c,a)}) \sim \text{Dirichlet}(2,2,2,2,2)$, and Case 4 specified by $p_{z,a,G_{i,j}} \sim \text{Beta}(2,2)$. Cases 3 and 4 correspond to more informative priors on $\pi$ and $p$, respectively. Table 6 reports the results for CADE. We see that different prior specifications lead to only slight changes in the results.
Table 6: Sensitivity analysis to prior specifications. Posterior medians and 95% interval of causal estimands are presented.

| Case     | CADE($a_0$, $a_0$)      | CADE($a_1$, $a_0$)      | CADE($a_0$, $a_1$)      | CADE($a_1$, $a_1$)      |
|----------|-------------------------|-------------------------|-------------------------|-------------------------|
| Baseline | $(-1356, 158, 1982)$    | $(-3328, -1626, -229)$ | $(-1444, 236, 2162)$   | $(-3782, -1813, -256)$ |
| Case 1   | $(-1265, 165, 2134)$    | $(-3431, -1679, -170)$ | $(-1357, 212, 2084)$   | $(-3845, -1873, -241)$ |
| Case 2   | $(-1346, 125, 1773)$    | $(-3329, -1593, -161)$ | $(-1423, 210, 2034)$   | $(-3691, -1770, -181)$ |
| Case 3   | $(-1313, 192, 1944)$    | $(-3322, -1579, -179)$ | $(-1399, 262, 2094)$   | $(-3742, -1764, -205)$ |
| Case 4   | $(-1351, 143, 2013)$    | $(-3458, -1682, -308)$ | $(-1458, 190, 2120)$   | $(-3905, -1881, -353)$ |

6 Concluding Remarks

We presented a Bayesian model-based methodology to address both interference and non-adherence in two-stage randomized experiments. Our methodology provides three contributions. First, we clarified and formalized assumptions about adherence behaviors within and across clusters. Second, we defined new causal estimands, including the overall effects of intervention and interpretable spillover effects, that can be inferred by means of our flexible Bayesian models. Our Bayesian methodologies address the issues of identifiability under two-sided nonadherence. Finally, we demonstrated via simulation studies how our methodology can enable more precise causal inferences compared to existing methods for complex, non-standard data-generating mechanisms. We further illustrated the utility of our methodology via its application to the RSBY dataset. In particular, our methodology was able to uncover more definitive evidence of spillover and overall effects of the intervention, which were not found in the previous analyses by Imai et al. (2021). Our results are further validated by sensitivity analyses to prior specifications.

Of great interest for future research on the Bayesian methodology is to relax the assumptions of the interference structure. In certain real-life contexts it may be too restrictive to employ the two-stage randomized design and assume stratified interference. A great deal of research has been conducted on inferring causal effects without using special designs such as the two-stage randomized design or clustered encouragement design. The work of Aronow and Samii (2017) and Sävie et al. (2021) provide one possible path for future
research based on the network structure and exposure mapping.

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A Supplementary Material

A.1 Derivation of $\tilde{D}_{i,j}(z, a)$ and $\tilde{Y}_{i,j}(a)$

Under Assumptions [2] and [3] we have

$$\tilde{D}_{i,j}(z, a) = \sum_{z_{-i,j} \in Z_{-i,j}} D_{i,j}(z, z_{-i,j})p(Z_{-i,j} = z_{-i,j} | Z_{i,j} = z, A_j = a)$$

$$= \sum_{z_{-i,j} \in Z_{-i,j}, Z_{i,j} = z, A_j = a} D_{i,j}(z, a)p(Z_{-i,j} = z_{-i,j}|Z_{i,j} = z, A_j = a)$$

$$= \sum_{z_{-i,j} \in Z_{-i,j}, Z_{i,j} = z, A_j = a} D_{i,j}(z, a)\left(\frac{N_j - 1}{K_j(a) - z}\right) = D_{i,j}(z, a).$$

where the second line follows from Assumption [3] and the third line follows from a simple probability calculation.

$$\tilde{Y}_{i,j}(a) = \sum_{z_j \in Z_j} Y_{i,j}(z)p(Z_j = z_j | A_j = a) = \sum_{z_j \in Z_j, A_j = a} Y_{i,j}(z_j)p(Z_j = z_j | A_j = a)$$

$$= \sum_{z_{-i,j} \in Z_{-i,j}, Z_{i,j} = 1, A_j = a} Y_{i,j}(Z_{i,j} = 1, Z_{-i,j} = z_{-i,j})p(Z_{i,j} = 1, Z_{-i,j} = z_{-i,j} | A_j = a)$$

$$+ \sum_{z_{-i,j} \in Z_{-i,j}, Z_{i,j} = 0, A_j = a} Y_{i,j}(Z_{i,j} = 0, Z_{-i,j} = z_{-i,j})p(Z_{i,j} = 0, Z_{-i,j} = z_{-i,j} | A_j = a)$$

$$= \sum_{z_{-i,j} \in Z_{-i,j}, Z_{i,j} = 1, A_j = a} Y_{i,j}(Z_{i,j} = 1, Z_{-i,j} = z_{-i,j})p(Z_{-i,j} = z_{-i,j} | Z_{i,j} = 1, A_j = a)p(Z_{i,j} = 1 | A_j = a)$$

$$+ \sum_{z_{-i,j} \in Z_{-i,j}, Z_{i,j} = 0, A_j = a} Y_{i,j}(Z_{i,j} = 0, Z_{-i,j} = z_{-i,j})p(Z_{-i,j} = z_{-i,j} | Z_{i,j} = 0, A_j = a)p(Z_{i,j} = 0 | A_j = a)$$

$$= \sum_{z_{-i,j} \in Z_{-i,j}, Z_{i,j} = 1, A_j = a} Y_{i,j}(Z_{i,j} = 1, Z_{-i,j} = z_{-i,j})p(Z_{-i,j} = z_{-i,j} | Z_{i,j} = 1, A_j = a)\frac{K_j(a)}{N_j}$$

$$+ \sum_{z_{-i,j} \in Z_{-i,j}, Z_{i,j} = 0, A_j = a} Y_{i,j}(Z_{i,j} = 0, Z_{-i,j} = z_{-i,j})p(Z_{-i,j} = z_{-i,j} | Z_{i,j} = 0, A_j = a)\frac{N_j - K_j(a)}{N_j}$$

$$= \frac{K_j(a)}{N_j} \sum_{z_{-i,j} \in Z_{-i,j}, Z_{i,j} = 1, A_j = a} Y_{i,j}(Z_{i,j} = 1, Z_{-i,j} = z_{-i,j})\left(\frac{N_j - 1}{K_j(a) - 1}\right)^{-1}$$

$$+ \frac{N_j - K_j(a)}{N_j} \sum_{z_{-i,j} \in Z_{-i,j}, Z_{i,j} = 0, A_j = a} Y_{i,j}(Z_{i,j} = 0, Z_{-i,j} = z_{-i,j})\left(\frac{N_j - 1}{K_j(a)}\right)^{-1}$$

$$= \frac{K_j(a)}{N_j} \tilde{Y}_{i,j}(1, a) + \frac{N_j - K_j(a)}{N_j} \tilde{Y}_{i,j}(0, a)$$
A.2 The Role of Exchangeability

Exchangeability can be justified for the two-stage randomized experiment in the presence of interference between units. First, consider two units that belong to different clusters. Assumption 2 implies that they do not interfere with each other. As such, it is plausible that their outcomes are independent. Also, as covariates are independent of treatment assignments, the treatments received and potential outcomes under different treatment assignments would be identical if the units’ cluster labels were permuted. Thus exchanging units that belong to different clusters in this manner does not affect the joint distribution for all units, and so they are exchangeable. We next consider two units $i_1$ and $i_2$ in the same cluster $j$. These units are not independent because their outcomes can be affected by interference. If both $i_1$ and $i_2$ are assigned treatment, then as Assumption 3 implies that interference is determined by the total number of treated units within cluster $j$, the joint distribution of all units in the cluster doesn’t change even if their labels are permuted because the number of treated units stays the same. Hence, the units are exchangeable. The same argument applies to the case in which both $i_1$ and $i_2$ are assigned control. Finally, if one is assigned treatment and the other is assigned control, then the total number of treated units in cluster $j$ still remains the same, and so the effect on the rest of the units within cluster $j$ would not change. The joint distribution of $i_1$ and $i_2$ will not change because only one of them is assigned treatment and the other is assigned control. Thus, exchangeability holds in such a case as well.

Ultimately, Assumptions 2 and 3 justify de Finetti’s Theorem in the two-stage randomized experiment and the use of parametric models under our methodology. If these assumptions did not hold, unit exchangeability would be questionable because the interference structure may collapse if the labels of the experimental units were permuted, which would affect the joint distribution of all units.
A.3 Derivations of Gibbs Samplers

The algorithm proceeds as in Section 3.4. For example, for the units with \((A_{ij}^{\text{obs}}, Z_{ij}^{\text{obs}}, D_{ij}^{\text{obs}}) = (a_0, 0, 0)\), there are five possible compliance behaviors for them, i.e., \(G_{ij} \in \mathcal{G}(a_0, 0, 0) = \{(c, c), (n, n), (c, a), (n, c), (n, a)\}\). Recall that \(\mathcal{G}(a, z, d)\) is a set of possible compliance types for units with \((A_{ij}^{\text{obs}}, Z_{ij}^{\text{obs}}, D_{ij}^{\text{obs}}) = (a, z, d)\). As defined in Section 4, \(G_{ij} \sim \text{Multinomial} \left(\pi_{(a,a)}, \pi_{(c,a)}, \pi_{(a,c)}, \pi_{(c,c)}, \pi_{(c,a)}\right)\) and \(Y_{ij}(z, a) = \{1 - W_{ij}(z, a)\} \tilde{Y}_{ij}(z, a)\) where \(W_{ij}(z, a) \sim \text{Bernoulli}(p_{z,a,G_{ij}})\) and \(\tilde{Y}_{ij}(z, a) \sim \text{Log-Normal}(\mu_{z,a,G_{ij}}, \sigma_{z,a,G_{ij}}^2)\). Recall that we defined \(w_{ij}^g\) as the probability mass function of \(G_{ij}\) and \(f_{ij}^{g,a,z}\) as the probability mass/density function of \(Y_{ij}(a, z)\) for \(g\) in Section 3.3. So, \(w_{ij}^g = \pi_g\) and \(f_{ij}^{g,a,z} = (p_{z,a,g} \mathbb{I}(y=0) \frac{1}{(1-p_{z,a,g}) h(y | \mu_{z,a,g}, \sigma_{z,a,g}^2)})^{1(y>0)}\) where \(h(y | \mu_{z,a,g}, \sigma_{z,a,g}^2)\) is the probability density function for the log-normal distribution with parameters \(\mu_{z,a,g}\) and \(\sigma_{z,a,g}^2\). The conditional distribution of \(G_{ij} = g\) given \(\mathcal{O}^{\text{obs}} = (A_{ij}^{\text{obs}}, Z_{ij}^{\text{obs}}, D_{ij}^{\text{obs}}, Y_{ij}^{\text{obs}}) = (a, z, d, y)\) is

\[
p(G_{ij}^{(t+1)} = g | \mathcal{O}^{\text{obs}}, y^{(t)}, \theta^{(t)}) = \frac{\pi_g^{(t)}(p_{z,a,g}^{(t)} \mathbb{I}(y=0)) (1-p_{z,a,g}^{(t)}) h(y | \mu_{z,a,g}^{(t)}, \sigma_{z,a,g}^{(t)2}))^{1(y>0)}}{\sum_{g \in \mathcal{G}(a,z,d)} \pi_g^{(t)}(p_{z,a,g}^{(t)} \mathbb{I}(y=0)) (1-p_{z,a,g}^{(t)}) h(y | \mu_{z,a,g}^{(t)}, \sigma_{z,a,g}^{(t)2}))^{1(y>0)}}
\]

for \(g \in \mathcal{G}(a, z, d)\).

In Step 2(b), we impute the missing potential outcomes for unit \(i\) in cluster \(j\) using the parameter \(\theta^{(t)}\), the compliance behaviors \(G_{ij}^{(t+1)} = g\) and the observed variables \((A_{ij}^{\text{obs}}, Z_{ij}^{\text{obs}}, D_{ij}^{\text{obs}}) = (a, z, d)\). We need to impute the missing values such that Assumption 6 holds. The imputation is two-fold. First, we sample \(W_{ij}^{(t+1)} \sim \text{Bernoulli}(p_{z,a,g}^{(t)})\). Then, if \(W_{ij}^{(t+1)} = 0\), we sample \(\tilde{Y}_{ij}^{(t+1)} \sim \text{LogNormal}(\mu_{z, a, g}^{(t)}, \sigma_{z, a, g}^{(t)2})\) for missing potential outcomes where \((a, z) \neq (a', z')\). For example, if we observe \((A_{ij}^{\text{obs}}, Z_{ij}^{\text{obs}}, D_{ij}^{\text{obs}}) = (a_0, 0, 0)\) and are given \(G_{ij}^{(t+1)} = (c, c)\) in the previous step of the Gibbs sampler, we need to impute three missing potential outcomes \(Y_{ij}(1, a_0), Y_{ij}(0, a_1), Y_{ij}(1, a_1)\) using corresponding parameters \((p_0^{(t)}, \mu_0^{(t)}, \sigma_0^{(t)})\), \((p_1^{(t)}, \mu_1^{(t)}, \sigma_1^{(t)})\), \((p_0^{(t)}, \mu_0^{(t)}, \sigma_0^{(t)})\), \((p_1^{(t)}, \mu_1^{(t)}, \sigma_1^{(t)})\).
If $G_{i,j}^{(t+1)} \in \{(a,a), (n,n), (n,a)\}$, we can invoke Assumption 6 and only need to impute either $Y_{ij}(0,a_0)$ or $Y_{ij}(0,a_1)$, depending on the observed treatment assignment for the unit.

If $A_{i,j}^{\text{obs}} = a_0$, we impute $Y_{ij}(0,a_1)$, otherwise, we impute $Y_{ij}(0,a_0)$. If $G_{i,j}^{(t+1)} = (n,c)$ and $A_{i,j}^{\text{obs}} = a_0$, we need to impute two missing potential outcomes $Y_{ij}(0,a_0), Y_{ij}(1,a_1)$.

If $A_{i,j}^{\text{obs}} = a_1$, we need to impute two missing potential outcomes $Y_{ij}(0,a_0), Y_{ij}(1,a_0)$. If $G_{i,j}^{(t+1)} = (c,a)$ and $A_{i,j}^{\text{obs}} = a_1$, we impute $Y_{ij}(0,a_0)$ and $Y_{ij}(0,a_1)$. If $(A_{i,j}^{\text{obs}}, Z_{i,j}^{\text{obs}}) = (a_1,0)$, we impute $Y_{ij}(0,a_0)$ and $Y_{ij}(1,a_1)$.

Now, assuming appropriate conjugate prior distributions for each parameter, we use the corresponding factor of the complete-data likelihood function to derive the posterior update for each parameter.
**Update of \( \pi \):** Consider a prior distribution \( (\pi_{(c,c)}, \pi_{(a,a)}, \pi_{(n,n)}, \pi_{(c,a)}, \pi_{(n,c)}, \pi_{(a,n)}) \sim \text{Dirichlet}(\alpha) \)
where \( \alpha = (1,1,1,1,1,1) \). Then, using the corresponding factor of \( \mathcal{L}_{\text{comp}} \), we draw from \( \pi^{(t+1)} \sim \text{Dirichlet}(N_{(c,c)}^{(t)} + 1, N_{(a,a)}^{(t)} + 1, N_{(n,n)}^{(t)} + 1, N_{(c,a)}^{(t)} + 1, N_{(n,c)}^{(t)} + 1, N_{(a,n)}^{(t)} + 1) \) where \( N_{g}^{(t)} \) is the number of compliance type \( g \) at the \( t \)-th MCMC step.

**Update of \( p \):** Consider a prior distribution \( p_{g,a}^{(t+1)} \sim \text{Beta}(1,1) \). Then, using the corresponding factor of \( \mathcal{L}_{\text{comp}} \), we have the posterior distribution such that, for \( z = 0,1 \) and \( a = a_0, a_1 \),

\[
\begin{align*}
    p_{z,a_0,a_1}^{(t+1)} & \sim \text{Beta} \left( \sum_{(i,j):G_{ij}^{(t+1)}=(c,c),Z_{ij}=z,A_j=a_0} 1(Y_{i,j}^{\text{obs}} = 0) + 1, \sum_{(i,j):G_{ij}^{(t+1)}=(c,c),Z_{ij}=z,A_j=a_1} 1(Y_{i,j}^{\text{obs}} \neq 0) + 1 \right) \\
    p_{0,a_0,a_1}^{(t+1)} & \sim \text{Beta} \left( \sum_{(i,j):G_{ij}^{(t+1)}=(a,a),A_j=a_0} 1(Y_{i,j}^{\text{obs}} = 0) + 1, \sum_{(i,j):G_{ij}^{(t+1)}=(a,a),A_j=a_1} 1(Y_{i,j}^{\text{obs}} \neq 0) + 1 \right) \\
    p_{0,a_0,(c,a)}^{(t+1)} & \sim \text{Beta} \left( \sum_{(i,j):G_{ij}^{(t+1)}=(c,a),A_j=a_0} 1(Y_{i,j}^{\text{obs}} = 0) + 1, \sum_{(i,j):G_{ij}^{(t+1)}=(c,a),A_j=a_1} 1(Y_{i,j}^{\text{obs}} \neq 0) + 1 \right) \\
    p_{0,a_0,(n,a)}^{(t+1)} & \sim \text{Beta} \left( \sum_{(i,j):G_{ij}^{(t+1)}=(n,a),A_j=a_0} 1(Y_{i,j}^{\text{obs}} = 0) + 1, \sum_{(i,j):G_{ij}^{(t+1)}=(n,a),A_j=a_1} 1(Y_{i,j}^{\text{obs}} \neq 0) + 1 \right) \\
    p_{z,a_1,(c,a)}^{(t+1)} & \sim \text{Beta} \left( \sum_{(i,j):G_{ij}^{(t+1)}=(c,c),Z_{ij}=z,A_j=a_1} 1(Y_{i,j}^{\text{obs}} = 0) + 1, \sum_{(i,j):G_{ij}^{(t+1)}=(c,c),Z_{ij}=z,A_j=a_0} 1(Y_{i,j}^{\text{obs}} \neq 0) + 1 \right) \\
    p_{0,a_1,(c,a)}^{(t+1)} & \sim \text{Beta} \left( \sum_{(i,j):G_{ij}^{(t+1)}=(a,a),A_j=a_1} 1(Y_{i,j}^{\text{obs}} = 0) + 1, \sum_{(i,j):G_{ij}^{(t+1)}=(a,a),A_j=a_0} 1(Y_{i,j}^{\text{obs}} \neq 0) + 1 \right) \\
    p_{0,a_1,(n,a)}^{(t+1)} & \sim \text{Beta} \left( \sum_{(i,j):G_{ij}^{(t+1)}=(n,a),A_j=a_1} 1(Y_{i,j}^{\text{obs}} = 0) + 1, \sum_{(i,j):G_{ij}^{(t+1)}=(n,a),A_j=a_0} 1(Y_{i,j}^{\text{obs}} \neq 0) + 1 \right)
\end{align*}
\]

Note that, by Assumption \( \text{[9]} \) the number of parameters to be estimated varies with compliance behavior. This can be seen in the complete-data likelihood as well. That is, if the parameter is not defined due to Assumption \( \text{[9]} \) the corresponding observations are used for the update of the counterpart parameter. For example, \( p_{1,a_0,(a,a)} \) is not defined due to Assumption \( \text{[6]} \). In such a case, all the observations with \( A_{ij}^{\text{obs}} = a_0 \) are used for updating \( p_{0,a_0,a_1} \) regardless of the observed value of \( Z_{i,j}^{\text{obs}} \).

**Update of \( \mu \) and \( \sigma \):** We only show the update of \( \mu_{0,a_0,(c,c)} \) and \( \sigma_{0,a_0,(c,c)} \) but we can follow
the same procedures for the other compliance types and observed values. For the notational convenience, $\sigma_{0,a_0,(c,c)}$ denotes the variance parameter. We should note the fact that the number of target units varies with compliance behavior, which we saw in the update of $\mu$.

For conjugate priors $\mu_{z,a,g} \sim N(0, \sqrt{5})$ and $\sigma_{z,a,g} \sim IG(0.1, 1)$ for $\forall g, z, a$, we have

$$
\sigma_{0,a_0,(c,c)}^{(t+1)} \sim IG\left(0.1 + S_0, 1 + S_1\right), \quad \mu_{0,a_0,(c,c)}^{(t+1)} \sim N\left(\frac{S_2}{S_0 + \sigma_{0,a_0,(c,c)}^{(t+1)}}, \frac{\sigma_{0,a_0,(c,c)}^{(t+1)}}{S_0 + \sigma_{0,a_0,(c,c)}^{(t+1)}}\right)
$$

$$
S_0 = \sum_{(i,j):G_{ij}^{(t+1)}=(c,c),Z_{i,j}=0,A_j=a_0} 1(Y_{i,j}^{obs} > 0)
$$

$$
S_1 = \frac{\sum_{(i,j):G_{ij}^{(t+1)}=(c,c),Z_{i,j}=0,A_j=a_0,Y_{i,j}^{obs} > 0} \left(\log Y_{i,j}^{obs} - \mu_{0,a_0,(c,c)}^{(t)}\right)^2}{2}
$$

$$
S_2 = \sum_{(i,j):G_{ij}^{(t+1)}=(c,c),Z_{i,j}=0,A_j=a_0,Y_{i,j}^{obs} > 0} \log Y_{i,j}^{obs}
$$

A.4 Parameters for Simulation Studies and Super-population Evaluation

Table 7 presents the parameters used for simulation studies. For the frequentist evaluations, we also consider the super-population versions of the causal estimands that we defined in Sections 2.4 and 2.5. This is done to eliminate a source of variation in our
simulations corresponding to the different values of the finite-population estimands across the simulated datasets. The specific super-population estimands that we focus in our evaluations are CADE_{sp}(a_0) = \mathbb{E}[Y_{i,j}(1, a_0) - Y_{i,j}(0, a_0) | G_{i,j} \in \{(c, c), (c, a)\}], CADE_{sp}(a_1) = \mathbb{E}[Y_{i,j}(1, a_1) - Y_{i,j}(0, a_1) | G_{i,j} \in \{(c, c), (c, a)\}], ITT_{Y,sp}(a_0) = \mathbb{E}[Y_{i,j}(1, a_0) - Y_{i,j}(0, a_0)], ITT_{Y,sp}(a_1) = \mathbb{E}[Y_{i,j}(1, a_1) - Y_{i,j}(0, a_1)], ITT_{D,sp}(a_0) = \mathbb{E}\{D_{i,j}(1, a_0) - D_{i,j}(0, a_0)\], ITT_{D,sp}(a_1) = \mathbb{E}\{D_{i,j}(1, a_1) - D_{i,j}(0, a_1)\].

\[
\begin{align*}
CADE_{sp}(0, a_0) &= \mathbb{E}[Y_{i,j}(1, a_0) - Y_{i,j}(0, a_0) | G_{i,j} \in \{(c, c), (c, a)\}] \\
&= (1 - \mathbb{E}[W_{i,j}(1, a_0) | G_{i,j} \in \{(c, c), (c, a)\}]) \mathbb{E}[\hat{Y}_{i,j}(1, a_0) | G_{i,j} \in \{(c, c), (c, a)\}]
- (1 - \mathbb{E}[W_{i,j}(0, a_0) | G_{i,j} \in \{(c, c), (c, a)\}]) \mathbb{E}[\hat{Y}_{i,j}(0, a_0) | G_{i,j} \in \{(c, c), (c, a)\}]
\end{align*}
\]

\[
= \left(\frac{\pi_{(c,c)}}{\pi_{(c,c)} + \pi_{(c,a)}}(1 - p_{(c,c)}^{1.0}) + \frac{\pi_{(c,a)}}{\pi_{(c,c)} + \pi_{(c,a)}}(1 - p_{(c,a)}^{1.0})\right)
\times \left(\frac{\pi_{(c,c)}}{\pi_{(c,c)} + \pi_{(c,a)}} \exp (\mu_{(c,c)}^{1.0} + \frac{\sigma_{(c,c)}^{1.0^2}}{2}) + \frac{\pi_{(c,a)}}{\pi_{(c,c)} + \pi_{(c,a)}} \exp (\mu_{(c,a)}^{1.0} + \frac{\sigma_{(c,a)}^{1.0^2}}{2})\right)
- \left(\frac{\pi_{(c,c)}}{\pi_{(c,c)} + \pi_{(c,a)}}(1 - p_{(c,c)}^{0.0}) + \frac{\pi_{(c,a)}}{\pi_{(c,c)} + \pi_{(c,a)}}(1 - p_{(c,a)}^{0.0})\right)
\times \left(\frac{\pi_{(c,c)}}{\pi_{(c,c)} + \pi_{(c,a)}} \exp (\mu_{(c,c)}^{0.0} + \frac{\sigma_{(c,c)}^{0.0^2}}{2}) + \frac{\pi_{(c,a)}}{\pi_{(c,c)} + \pi_{(c,a)}} \exp (\mu_{(c,a)}^{0.0} + \frac{\sigma_{(c,a)}^{0.0^2}}{2})\right)
\]

= 4765.78

The rest can be calculated in the same way. CADE_{sp}(1, a_1) = 5156.41, ITT_{Y,sp}(a_0) = 2382.89, ITT_{Y,sp}(a_1) = 2324.13, ITT_{D,sp}(a_0) = \pi_{cc} + \pi_{ca} = 0.5, ITT_{D,sp}(a_1) = \pi_{cc} + \pi_{nc} = 0.45. Table 8 presents the simulation results under the super-population perspective. We can see the results change only slightly from the finite-population evaluation.
Table 8: Evaluation metrics for our Bayesian methodology versus the method of Imai et al. (2021) (abbreviated as “IJM”) for the Log-Normal (left) and Gamma (right) data-generating processes under the super-population perspective.

|                | Coverage | Bias   | MSE    | Interval Width | Coverage | Bias   | MSE    | Interval Width |
|----------------|----------|--------|--------|----------------|----------|--------|--------|----------------|
|                | N        | IJM    | Bayes  | IJM            | Bayes    | IJM    | Bayes  | IJM            |
| Log-Normal     |          |        |        |                |          |        |        |                |
|                | 5000     | 96%    | 98%    | 2.40E+02      | 5.18E+02 | 3.60E+07| 4.59E+06| 1.91E+04      |
|                | 10000    | 97%    | 98%    | 2.72E+02      | 5.19E+02 | 3.93E+07| 1.93E+04| 5.41E+04      |
|                | 50000    | 94%    | 95%    | 2.90E+00      | -9.78E-01| 2.20E+03| 2.25E+03| 2.09E+03      |
| CADReg(0, a)   | 10000    | 97%    | 98%    | -2.72E+02     | 2.72E+02 | 1.39E+07| 1.91E+04| 5.41E+04      |
|                | 50000    | 95%    | 96%    | -6.53E+01     | -1.22E+00| 2.45E+02| 2.40E+02| 5.72E+01      |
|                | 10000    | 96%    | 95%    | -8.67E+02     | 1.22E+03 | 3.58E+08| 5.87E+06| 1.20E+06      |
|                | 50000    | 94%    | 95%    | -3.28E+02     | 3.12E+02 | 2.18E+07| 7.77E+05| 4.81E+03      |
|                | 50000    | 95%    | 98%    | 6.08E+01      | 4.28E+02 | 2.64E+07| 8.15E+05| 1.52E+04      |
|                | 10000    | 94%    | 95%    | -3.41E-04     | 2.68E-03 | 1.74E-04| 1.85E-04| 5.32E-05      |
|                | 50000    | 95%    | 98%    | -1.34E-04     | 5.48E-04 | 4.74E-05| 4.30E-05| 2.86E-02      |
|                | 5000    | 96%    | 94%    | -1.58E-03     | 4.36E-04 | 4.36E-04| 9.68E-02| 7.64E-02      |
|                | 10000   | 93%    | 92%    | -7.23E-04     | 3.53E-04 | 2.80E-04| 2.65E-04| 6.40E-02      |
|                | 50000   | 96%    | 94%    | -1.34E-04     | 3.44E-04 | 4.74E-05| 4.36E-05| 2.80E-02      |

|                | Coverage | Bias   | MSE    | Interval Width | Coverage | Bias   | MSE    | Interval Width |
|----------------|----------|--------|--------|----------------|----------|--------|--------|----------------|
|                | N        | IJM    | Bayes  | IJM            | Bayes    | IJM    | Bayes  | IJM            |
| Gamma          |          |        |        |                |          |        |        |                |
|                | 5000     | 96%    | 98%    | 2.40E+02      | 5.18E+02 | 3.60E+07| 4.59E+06| 1.91E+04      |
|                | 10000    | 97%    | 98%    | 2.72E+02      | 5.19E+02 | 3.93E+07| 1.93E+04| 5.41E+04      |
|                | 50000    | 94%    | 95%    | 2.90E+00      | -9.78E-01| 2.20E+03| 2.25E+03| 2.09E+03      |
| CADReg(1, a)   | 10000    | 97%    | 98%    | -2.72E+02     | 2.72E+02 | 1.39E+07| 1.91E+04| 5.41E+04      |
|                | 50000    | 95%    | 96%    | -6.53E+01     | -1.22E+00| 2.45E+02| 2.40E+02| 5.72E+01      |
|                | 10000    | 96%    | 95%    | -8.67E+02     | 1.22E+03 | 3.58E+08| 5.87E+06| 1.20E+06      |
|                | 50000    | 94%    | 95%    | -3.28E+02     | 3.12E+02 | 2.18E+07| 7.77E+05| 4.81E+03      |
|                | 50000    | 95%    | 98%    | 6.08E+01      | 4.28E+02 | 2.64E+07| 8.15E+05| 1.52E+04      |
|                | 50000    | 95%    | 98%    | -3.41E-04     | 2.68E-03 | 1.74E-04| 1.85E-04| 5.32E-05      |
|                | 5000    | 96%    | 94%    | -1.58E-03     | 4.36E-04 | 4.36E-04| 9.68E-02| 7.64E-02      |
|                | 10000   | 93%    | 92%    | -7.23E-04     | 3.53E-04 | 2.80E-04| 2.65E-04| 6.40E-02      |
|                | 50000   | 96%    | 94%    | -1.34E-04     | 3.44E-04 | 4.74E-05| 4.36E-05| 2.80E-02      |