Bayesian Approach to Two-Stage Randomized Experiments in the Presence of Interference and Noncompliance

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October 22, 2021

Abstract

No interference between experimental units is a critical assumption in causal inference. Over the past decades, there have been significant advances to go beyond this assumption using the design of experiments; two-stage randomization is one such. The researchers have shown that this design enables us to estimate treatment effects in the presence of interference. On the other hand, the noncompliance behavior of experimental units is another fundamental issue in many social experiments, and researchers have established methods to deal with noncompliance under the assumption of no interference between units. In this article, we propose a Bayesian approach to analyze a causal inference problem with both interference and noncompliance. Building on previous work on two-stage randomized experiments and noncompliance, we apply the principal stratification framework to compare treatments adjusting for posttreatment variables yielding special principal effects in the two-stage randomized experiment. We illustrate the proposed methodology by conducting simulation studies and reanalyzing the evaluation of India’s National Health Insurance Program, where we draw more definitive conclusions than existing results.

Keywords: Bayesian causal inference; Interference; Noncompliance; Principal stratification; Two-stage randomized design


1 Introduction

Causal inference is an important issue in any scientific problem. The Rubin’s Causal Model (RCM) has been developed over the past decades as a statistical approach to analyzing the causal effects based on the framework of potential outcomes. The Stable Unit Treatment Value Assumption (SUTVA) is a key set of assumptions that are usually made in the Rubin’s causal model. The SUTVA consists of two assumptions.

1. No interference

2. No hidden variations of treatments

The first component of SUTVA implies that there is no interference between experimental units. To put it another way, the potential outcomes of a unit \(i\) depend only on its treatment assignment and are not affected by the treatment assignments of other units. The second component of SUTVA requires that an individual receiving a specific treatment level cannot receive different forms of that treatment. Even under these assumptions, the RCM is a very powerful framework and has solved a wide variety of questions in various study fields, such as economics, social science, business (Imbens and Rubin (2015)). However, in real-world data analysis, it is not always easy to guarantee that the SUTVA holds, even worse it is often violated. For example, one unit’s treatment assignment may affect another unit’s decision of the treatment receipt. This is a violation of the first assumption of SUTVA. Over the last few decades, massive research efforts have been put into dealing with causal inference in the presence of interference between units. In the growing literature on interference, Hudgens and Halloran (2008) first introduced the two-stage randomized design in the context of interference. It is an experimental design where clusters to which each experimental unit belongs are randomly assigned different assignment probabilities, and then the treatment is randomly assigned to the units within the clusters based on the assignment
probability for the cluster to which the units belong. They estimated both direct effects of treatment assignment and indirect effects through the treatment receipts of other units in the presence of interference. A lot of authors have studied causal inference with interference under this design (VanderWeele and Tchetgen (2011); Tchetgen and Vanderweele (2012); Liu and Hudgens (2014); Basse and Feller (2018)). Another complication of the causal inference problem is the possible existence of noncompliers who would not necessarily comply with the treatment assignment. For example, some units who are assigned treatment may refuse to receive the treatment, or control units may take the treatment even though they are not assigned treatment. One approach to analyzing the randomized experiments with noncompliance is an intention-to-treat (ITT) analysis which discards entirely any information about the treatment. There is also a rich history in analyzing the noncompliance behavior in causal inference problems.

Until very recently, interference and noncompliance have been studied independently in the causal inference literature, assuming the other assumption holds. However, it may be unrealistic because both assumptions can often be violated at the same time especially in social experiments. Very little literature addresses these issues simultaneously. A notable work, Imai et al. (2021), presented a nonparametric identification of the complier average direct and indirect effects and proposed consistent estimators for them in the two-stage randomized design with interference and noncompliance. They applied their methodology to the randomized evaluation of the Indian health insurance program (RSBY). Kang and Imbens (2016) also analyzed the two-stage randomized experiment with interference and noncompliance by proposing a new experimental design, called the peer encouragement design. Forastiere et al. (2016) applied the Bayesian principal stratification (Frangakis and Rubin (2002)) to causal inference in clustered encouragement designs (CEDs). In the CEDs, the assignment of treatment encouragement is at the level of clusters where the compliance with the assignment varies across the units within clusters. There-
fore, this design can be seen as a special case of the two-stage randomized experiment with no randomization within clusters, that is, the treatment assignment probability for units within clusters is either zero or one. Vazquez-Bare (2021) built on Imai et al. (2021) and analyzed spillover effects using instrumental variables in the two-stage randomized experiment. They analyzed the identification of causal direct and spillover effects under one-sided noncompliance, and showed that these effects can be estimated by the two-Stage least squares regression analysis (2SLS).

Building on Imai et al. (2021), we extend their work and propose a Bayesian model-based approach to the two-stage randomized experiment with both interference and noncompliance. Relying on the flexibility of Bayesian modeling, we can flexibly customize our model to make the inference more robust to unwieldy distributions, e.g., heavy-tailed distributions with zero-inflation and outliers. We use a Bayesian principal stratification to deal with the difficulties that arise in a two-stage randomized design. To the best of our knowledge, none of the previous work has attempted to apply the Bayesian principal stratification approach to causal inference in two-stage randomized designs with interference and noncompliance. The Bayesian principal stratification approach allows us to define various types of causal estimands that the previous work was not able to consider. Specifically, we make the following three contributions to the literature. First, we propose a new Bayesian approach to causal inference in the two-stage randomized design with interference and noncompliance. The Gibbs sampling based posterior inference is developed. Second, we present new sets of assumptions on compliance types that make our inference efficient and stable, and propose new causal estimands including the complier spillover and overall effects of intervention, which would be of the greatest interest for policy makers. Many of the estimands are new or are defined differently from previous work to provide clearer interpretability. Finally, we revisit the evaluation of India’s National Health Insurance Program (RSBY) dataset (Nandi et al. (2015)), and find more definitive evidence of some
causal effects which were found insignificant in the previous research. Our methodology is validated by conducting extensive simulation studies to investigate the frequentist properties in a simple situation where the existing frequentist approach performs poorly in terms of bias and MSE.

The rest of the paper is organized as follows. In Section 2, we briefly review the preliminary concepts of the RCM for the two-stage randomized experiment. We also provide some notations and setup therein. Section 3 introduces some fundamental assumptions of our analysis and presents our model-based Bayesian approach by the principal stratification. In Section 4, we perform simulation studies to investigate the performance of our method for a heavy-tailed distribution with an excess of zeros values. In Section 5, we apply our methodology to the RSBY dataset. Section 6 concludes the paper.

2 Setup and Assumptions

In this section, we will briefly review the two-stage randomized experiment and give the formal definition of causal quantities of interest. The two-stage randomized experiment in the context of causal inference allowing for interference was first proposed by Hudgens and Halloran (2008). A lot of authors have built upon and extend their work. We also discuss sets of assumptions made throughout this article.

2.1 Potential Outcomes Framework in a Two-stage Randomized Experiments with Interference and Noncompliance

We use the potential outcomes framework of causal inference Rubin (1974) to describe our problem in which we consider a finite-population model, where the only source of randomness is treatment assignment. We consider $J$ clusters $j = 1,...,J$ with $n_j$ units in cluster $j$. Each experimental unit belongs to one of the clusters. We use $i = 1,...,n_j$
to index the experimental units in cluster $j$, and where $N$ is the number of the total experimental units in the experiment $N = \sum_{j=1}^{J} n_j$. A pair of indices $ij$ uniquely denotes the experimental unit $i$ in cluster $j$.

The two-stage randomized experiment considers a sequential randomization procedure in which each stage of randomization follows complete randomization; that is, a fixed number of units are assigned to treatment at each stage [Imbens and Rubin (2015)]. Other randomization schemes in the context of two-staged randomized experiments are discussed in [VanderWeele and Tchetgen (2011)]. First, we randomly assign each cluster one of the treatment assignment mechanisms $A_j \in \{a_0, a_1\}$, where $A_j = a_1$ ($A_j = a_0$) indicates that a high (low) proportion of units are assigned to the treatment within cluster $j$. For example, 80% of units within cluster are assigned treatment if $A_j = a_1$, and 40% of units within cluster are assigned treatment if $A_j = a_0$. We assign $J_1$ clusters to $A_j = a_1$ and $A = (A_1, ..., A_J)$ denote the assignment mechanism vector for all clusters. Thus, in the first stage of the complete randomization, we have $Pr(A) = \left(\frac{J}{J_1}\right)^{-1}$ for all $A$ such that $\sum_{j=1}^{J} A_j = J_1$.

For the second stage of randomization, units in cluster $j$ are assigned to treatment based on the assignment mechanism $A_j$. Let $Z_{ij}$ denote the binary treatment assignment for unit $i$ in cluster $j$ and $Z_j = (Z_{1j}, ..., Z_{nj})$ denote the assignment vector for the $n_j$ units in cluster $j$, where $Z_{ij} = 1$ if unit $i$ in cluster $j$ is assigned treatment, and $Z_{ij} = 0$ otherwise. Similarly, we define $Z_{-ij}$ as a sub-vector of $Z_j$ with the $i$th element removed. Since we assume the complete randomization for the second stage too, a fixed proportion of units $K_j(a)$ are assigned treatment in cluster $j$ conditioned on $A_j = a$. Thus, we have $Pr(Z_j | A_j = a) = \left(\frac{n_j}{K_j(a)}\right)^{-1}$ for all $Z_j$ such that $\sum_{i=1}^{n_j} Z_{ij} = K_j(a)$ under $A_j = a$. The number of treated units $K_j(a)$ varies with the assignment mechanism $a$.

Next, we introduce the notations for treatment receipt of units. Since we consider an experiment where both interference and noncompliance exist, both potential outcomes and
treatment receipts for all experimental units can be influenced by treatment assignments of other units. Let \( D_{ij} \) denote the treatment receipt for unit \( i \) in cluster \( j \) and \( D_j = (D_{1j}, ..., D_{nj}) \) be the treatment receipt vector for cluster \( j \). Similarly, let \( Y_{ij} \) denote the observed outcome for unit \( i \) in cluster \( j \) and \( Y_j = (Y_{1j}, ..., Y_{nj}) \) be the vector of observed outcomes for all units in cluster \( j \). For the treatment assignment vector \( z \) for all \( N \) units, we write \( D_{ij}(z) \) as the potential value of treatment receipt of unit \( i \) in cluster \( j \). Furthermore, \( Y_{ij}(z; d) \) denotes the potential outcomes for the unit, where \( d \) denotes the treatment receipt vector for all \( N \) units. The potential outcomes can also be written as \( Y_{ij}(z) = Y_{ij}(z; D_{ij}(z)) \) for the treatment assignment vector \( z \) since the treatment receipt \( D_{ij} \) is determined by \( z \).

Following Imai et al. (2021), we assume the exclusion restriction with interference between units. This assumption states that the outcome of a unit only depends on the treatment receipts of units within the same cluster.

**Assumption 1.** *(Exclusion restriction with interference between units).*

For any \( z_j, z'_j \) and \( d_j \),

\[
Y_{ij}(z_j; d_j) = Y_{ij}(z'_j; d_j)
\]

In a usual setting of causal inference without interference, by invoking the SUTVA, we can write \( Y_{ij}(z) = Y_{ij}(z_{ij}) \) because the potential outcomes for unit \( i \) in cluster \( j \) are determined by its own treatment assignment \( z_{ij} \) alone under no interference between units. With interference, however, there are \( 2^N \) patterns for the potential values of treatment receipt and outcomes for every experimental unit. To reduce the complexity of the problem, Hudgens and Halloran (2008) made the following plausible assumption, called *Partial interference*, which indicates that units are not affected by each other if they are in different clusters. Imai et al. (2021) further apply this assumption to the potential values of treatment receipt in the noncompliance setting. With this assumption, the treatment receipt and outcome are influenced by the treatment assignment of other units within the same cluster. Following the literature, we make the same assumption.
Assumption 2. (Partial interference)

\[ Y_{ij}(z) = Y_{ij}(z') \text{ and } D_{ij}(z) = D_{ij}(z') \]
for all \( z \) and \( z' \) such that \( z_j = z'_j \).

[Hudgens and Halloran (2008)] claimed that unbiased estimation of the variances is impossible without making further assumptions in addition to the partial interference assumption. **Stratified interference** is another key assumption about the structure of interference proposed by [Hudgens and Halloran (2008)], which makes the inference of variance feasible.

Assumption 3. (Stratified interference)

\[ Y_{ij}(z) = Y_{ij}(z') \text{ and } D_{ij}(z) = D_{ij}(z') \]
if \( z_{ij} = z'_{ij} \) and \( \sum_{i=1}^{n_j} z_{ij} = \sum_{i=1}^{n_j} z'_{ij} \).

This assumption states that the potential values of treatment receipt and outcome are only influenced by the number of individuals assigned to treatment conditions within the same cluster. This is a fundamental assumption made in the literature ([Hudgens and Halloran (2008); Imai et al. (2021); Forastiere et al. (2016)]). In this article, we also make this assumption, however, it is noteworthy that enormous recent research effort has been put into going beyond this condition to deal with more flexible structures of interference ([Aronow (2012); Manski (2013); Toulis and Kao (2013); Basse and Airoldi (2018b); 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); Sävje (2021)]).

With this assumption and the nature of the complete randomized experiment, we can simply write the potential values of treatment receipt and outcomes of one unit as the function of its own treatment assignment and the assignment mechanism for the cluster to which the unit belongs; that is, we can write \( D_{ij}(z_j) \) and \( Y_{ij}(z_j) \) as \( D_{ij}(z_{ij}, a) \) and
Y_{ij}(z_{ij}, a) respectively since the number of treated units is the only factor that affects others’ treatment receipt and potential outcomes, and the number of treated units is fixed due to the completely randomized design.

2.2 Causal Estimands

We next discuss causal quantities of interest, closely following Hudgens and Halloran (2008); Imai et al. (2021). A fundamental problem of causal inference is that it is not possible to observe more than one potential outcome for an individual. The causal estimand of interest can be written as a function of observed potential outcomes and missing potential outcomes with the treatment assignment (receipt). We begin by writing the average potential values of treatment receipt and outcomes for unit i in cluster j under z_{ij} = z respectively as

\[ D_{ij}(Z_{ij} = z, Z_{-ij} = z_{-ij}), \ Y_{ij}(Z_{ij} = z, Z_{-ij} = z_{-ij}) \]

for z = 0, 1. Under Assumption 2 and 3, we define the individual average potential value of treatment receipt \( \bar{D}_{ij} \) under treatment assignment \( z \) and treatment assignment mechanism \( a \) as

\[ \bar{D}_{ij}(z, a) = \sum_{Z_{-ij}\in Z_{-ij}, Z_{ij}=z, A_j=a} D_{ij}(Z_{ij} = z, Z_{-ij} = z_{-ij}) Pr(Z_{-ij} = z_{-ij}|Z_{ij} = z, A_j = a) \]

\[ = \sum_{Z_{-ij}\in Z_{-ij}, Z_{ij}=z, A_j=a} D_{ij}(Z_{ij} = z, A_j = a) Pr(Z_{-ij} = z_{-ij}|Z_{ij} = z, A_j = a) \]

\[ = \sum_{Z_{-ij}\in Z_{-ij}, Z_{ij}=z, A_j=a} D_{ij}(Z_{ij} = z, A_j = a) \left( \frac{n_j - 1}{K_j(a) - z} \right)^{-1} \]

\[ = D_{ij}(Z_{ij} = z, A_j = a) \]

where \( Z_{-ij} \) is the set of all potential sub-vectors of \( Z_j \) with the i\textsuperscript{th} element removed, such that \( \sum_{i=1}^{n_j} Z_{ij} = K_j(a) \) under \( A_j = a \). Then, the second line follows from Assumption 3 and the third line follows from a simple probability calculation. We define the individual
average potential outcome $\bar{Y}_{ij}$ in the same manner.

$$\bar{Y}_{ij}(z, a) = \sum_{z_{-ij} \in \mathcal{Z}_{-ij}, z_{ij} = z, A_j = a} Y_{ij}(Z_{ij} = z, Z_{-ij} = z_{-ij}, A_j = a) Pr(Z_{-ij} = z_{-ij} | Z_{ij} = z, A_j = a) = Y_{ij}(Z_{ij} = z, A_j = a)$$

Given these individual average values of potential outcomes, we define the cluster-level and population-level average values of treatment receipt and outcome as

$$\bar{D}_j(z, a) = \frac{1}{n_j} \sum_{i=1}^{n_j} \bar{D}_{ij}(z, a), \quad \bar{D}(z, a) = \frac{1}{N} \sum_{j=1}^{J} n_j \bar{D}_j(z, a)$$

$$\bar{Y}_j(z, a) = \frac{1}{n_j} \sum_{i=1}^{n_j} \bar{Y}_{ij}(z, a), \quad \bar{Y}(z, a) = \frac{1}{N} \sum_{j=1}^{J} n_j \bar{Y}_j(z, a)$$

Using these quantities, we define the Intention-to-Treat (ITT) effects of treatment assignment on the treatment receipt and outcome under the assignment mechanism $a$.

$$DED_{ij}(a) = \bar{D}_{ij}(1, a) - \bar{D}_{ij}(0, a)$$

$$DEY_{ij}(a) = \bar{Y}_{ij}(1, a) - \bar{Y}_{ij}(0, a)$$

where $DED$ and $DEY$ stand for the average direct effect on $D$ and $Y$ respectively. Intuitively, these quantities represent how much the treatment receipt behavior and the outcome would change under the same assignment mechanism $a$ when a unit is assigned treatment. By averaging the unit-level ITT effects, $DED_{ij}$ and $DEY_{ij}$, we define the cluster-level and population-level ITT effects.

$$DED_j(a) = \frac{1}{n_j} \sum_{i=1}^{n_j} DED_{ij}(a), \quad DED(a) = \frac{1}{N} \sum_{j=1}^{J} n_j DED_j(a)$$

$$DEY_j(a) = \frac{1}{n_j} \sum_{i=1}^{n_j} DEY_{ij}(a), \quad DEY(a) = \frac{1}{N} \sum_{j=1}^{J} n_j DEY_j(a)$$
In causal inference problem with interference, another quantity of interest would be spillover (indirect) effect of treatment assignments of other units on the potential values of treatment receipt and outcome. Following Imai et al. (2021), We define the unit-level spillover effects on treatment receipt and outcome as follows.

\[
S\text{ED}_{ij}(z) = \bar{D}_{ij}(z, a_1) - \bar{D}_{ij}(z, a_0)
\]

\[
S\text{EY}_{ij}(z) = \bar{Y}_{ij}(z, a_1) - \bar{Y}_{ij}(z, a_0)
\]

where we compare the average potential values under two different assignment mechanisms, that is, \(A_j = a_1\) and \(A_j = a_0\) with treatment assignment fixed at \(Z_{ij} = z\). This definition quantifies our intuition that, if there exists any difference in potential values under the same treatment assignment \(z\), this difference is attributed to the treatment mechanism which governs the proportion of treated units. Under the stratified interference assumption, the treated units in the neighbor are the only factor that influences your outcome. Hence, it is reasonable to regard these quantities as the spillover effect. We define the cluster-level and population-level spillover effects in the same way as in the direct effects.

\[
S\text{ED}_j(z) = \frac{1}{n_j} \sum_{i=1}^{n_j} S\text{ED}_{ij}(z), \quad S\text{ED}(z) = \frac{1}{N} \sum_{j=1}^{J} n_j S\text{ED}_j(z)
\]

\[
S\text{EY}_j(z) = \frac{1}{n_j} \sum_{i=1}^{n_j} S\text{EY}_{ij}(z), \quad S\text{EY}(z) = \frac{1}{N} \sum_{j=1}^{J} n_j S\text{EY}_j(z)
\]

Finally, we discuss the overall effects of the assignment mechanism on the outcome. The overall effect is usually of the greatest interest for policy makers. Intuitively, the overall effects quantify the whole effect of intervention by comparing the assignment mechanism \(a_0\) with another assignment mechanism \(a_1\) in terms of outcomes. For example, policy makers may be interested in comparing infection rates under two different allocation plans of vaccinating 40% and 80% of each cluster. We define the unit-level overall effects of
treatment mechanism on treatment receipt and outcome as follows.

$$O E Y_{ij}(a_0, a_1) = \bar{Y}_{ij}(a_1) - \bar{Y}_{ij}(a_0)$$

where

$$\bar{Y}_{ij}(a) = \sum_{z_{ij} \in Z_{ij}, A_j = a} Y_{ij}(Z_{ij} = z_{ij}) Pr(Z_{ij} = z_{ij} | A_j = a)$$

$\bar{Y}_{ij}(a)$ can be seen as the average value on the individual’s outcome under the mechanism $a$. It is decomposed into

$$\bar{Y}_{ij}(a) = \sum_{z_{ij} \in Z_{ij}, A_j = a} Y_{ij}(Z_{ij} = z_{ij}) Pr(Z_{ij} = z_{ij} | A_j = a)$$

$$= \sum_{z_{-ij} \in Z_{-ij}, Z_{ij} = 1, A_j = a} Y_{ij}(Z_{ij} = 1, Z_{-ij} = z_{-ij}) Pr(Z_{ij} = 1, Z_{-ij} = z_{-ij} | A_j = a)$$

$$+ \sum_{z_{-ij} \in Z_{-ij}, Z_{ij} = 0, A_j = a} Y_{ij}(Z_{ij} = 0, Z_{-ij} = z_{-ij}) Pr(Z_{ij} = 0, Z_{-ij} = z_{-ij} | A_j = a)$$

$$= \sum_{z_{-ij} \in Z_{-ij}, Z_{ij} = 1, A_j = a} Y_{ij}(Z_{ij} = 1, Z_{-ij} = z_{-ij}) Pr(Z_{ij} = 1, Z_{-ij} = z_{-ij} | Z_{ij} = 1, A_j = a) Pr(Z_{ij} = 1 | A_j = a) \frac{K_j(a)}{n_j}$$

$$+ \sum_{z_{-ij} \in Z_{-ij}, Z_{ij} = 0, A_j = a} Y_{ij}(Z_{ij} = 0, Z_{-ij} = z_{-ij}) Pr(Z_{ij} = 0, Z_{-ij} = z_{-ij} | Z_{ij} = 0, A_j = a) n_j - \frac{K_j(a)}{n_j}$$

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

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

$$= \frac{K_j(a)}{n_j} \bar{Y}_{ij}(1, a) + \frac{n_j - K_j(a)}{n_j} \bar{Y}_{ij}(0, a)$$

(1)
Plugging into $OEY_{ij}(a_0, a_1)$, we have

$$OEY_{ij}(a_0, a_1) = \frac{K_j(a_1)}{n_j} DEY_{ij}(a_1) - \frac{K_j(a_0)}{n_j} DEY_{ij}(a_0) + SEY_{ij}(0)$$

which turns out to be the same decomposition as [VanderWeele and Tchetgen (2011)]. Intuitively, the overall effect is expressed as the sum of the spillover effect and the uplift of the direct effect weighted by the proportion of treated units in a cluster. The cluster-level and population-level effects are defined as follows.

$$OEY_j(a_0, a_1) = \frac{1}{n_j} \sum_{i=1}^{n_j} OEY_{ij}(a_0, a_1), \ OEY(a_0, a_1) = \frac{1}{N} \sum_{j=1}^{J} n_j OEY_j(a_0, a_1)$$

3 Model and Estimation

3.1 Principal Stratification

In general, Principal stratification ([Frangakis and Rubin, 2002]) refers to settings with latent unconfoundedness of the primary treatment assignment, where, conditional on an only partially observed covariate, unconfoundedness holds. The units can be stratified in subpopulation, principal strata, defined according to the potential values of the actual treatment assignment and receipt. As defined in the previous section, the potential value of treatment receipt for unit $i$ in cluster $j$, $D_{ij}(z, a)$, is a function of the treatment assignment $Z_{ij} = z$ and the assignment mechanism $A_j = a$. If we consider the binary treatment assignment $Z_{ij} \in \{0, 1\}$ and two assignment mechanisms $A_j \in \{a_0, a_1\}$, there exist four potential values of treatment receipt for each unit; i.e., $D_{ij}(0, a_0), D_{ij}(1, a_0), D_{ij}(0, a_1)$ and $D_{ij}(1, a_1)$. Since we cannot observe these potential values simultaneously, we define a latent variable that defines the compliance behavior of a unit when the unit is assigned to the treatment (control) condition under both mechanisms. What is unique with this design is that units could show different compliance behaviors under different mechanisms.
as we assume stratified interference on treatment receipt. In other words, if units were to belong to a cluster with a higher assignment probability, they might start to comply with the treatment assignment, even if they do not comply with it in a cluster with a lower assignment probability, or vice versa.

First, we define the compliance behavior of unit $i$ in cluster $j$ at each mechanism, $G_{ij}(a)$ for $a = a_0, a_1$.

$$G_{ij}(a) = \begin{cases} 
  n & \text{if } D_{ij}(0, a) = 0, D_{ij}(1, a) = 0 \\
  c & \text{if } D_{ij}(0, a) = 0, D_{ij}(1, a) = 1 \\
  d & \text{if } D_{ij}(0, a) = 1, D_{ij}(1, a) = 0 \\
  a & \text{if } D_{ij}(0, a) = 1, D_{ij}(1, a) = 1
\end{cases}$$

where $n, c, d, a$ stand for nevertakers, compliers, defiers and alwaystakers respectively, often referred to in the literature. Based on the compliance behavior on each mechanism, the compliance status for unit $i$ in cluster $j$ is defined as

$$G_{ij} = \{G_{ij}(a_0), G_{ij}(a_1)\}$$

Now, we consider the assumptions underlying the compliance behavior. We have defined 16 principal strata for each unit. First, we make the following monotonicity assumption at each assignment mechanism.

**Assumption 4. (Monotonicity under a fixed assignment mechanism)**

For the assignment mechanism $a = a_0, a_1$,

$$D_{ji}(1, a) \geq D_{ji}(0, a)$$

Similar assumptions are made in the literature [Imai et al. (2021); Forastiere et al. (2016)]. This assumption rules out the presence of defiers under both treatment assignment mechanisms $a = a_0, a_1$ or, in other words, restrict the sign of the effect of the treatment assignment on the treatment under a fixed mechanism. This assumption reduces the number of
principal strata from sixteen to nine strata. Additionally, we further make the following assumption about the compliance behavior across assignment mechanisms.

**Assumption 5. (Monotonicity of compliance behavior across mechanisms)**

\[ G = \{n, c, a\} \text{ and } A = \{a_0, a_1\} \text{ are partial order sets of compliance behaviors and assignment mechanisms with } a_0 \leq a_1, \text{ and } G \text{ is non-strictly monotonic with respect to } A. \]

This assumption further reduces the number of strata to consider. First, this assumption implicitly assumes that compliance behaviors are somewhat ordered. Then we define partial orders of the set of compliance behaviors and the set of assignment mechanisms. For example, there are possibly six orders for the set of compliance behaviors; i.e., \( n \leq c \leq a \), \( n \leq a \leq c \), \( c \leq n \leq a \), \( c \leq a \leq n \), \( a \leq n \leq c \) and \( a \leq c \leq n \). Throughout this article, we assume the partial order \( n \leq c \leq a \) for compliance behaviors, and \( a_0 \leq a_1 \) for the assignment mechanism, where \( a_0 \) and \( a_1 \) represent the mechanism with low and high assignment probabilities within cluster respectively. Intuitively, this order assumes that people are more likely to decide to take the treatment if a larger proportion of their neighbors is taking the treatment. In other words, the higher assignment probability within cluster drives units to receive the treatment no matter what they are assigned. Note that without loss of generality, these orderings can be rearranged depending on the context and its plausibility should be judged either by expert knowledge or by some related evidence in the data for each application. With these two assumptions about compliance, the number of principal strata reduces to six after all. Each unit is a member of one of the strata, \( G_{ij} \in \{cc, aa, nn, ca, nc, na\} \), where the first and second character of elements stands for the compliance status under the first and second assignment mechanism respectively.

It may be necessary here to comment on the difference between our definition of principal strata and that of previous work. As seen in our definition, we defined six principal strata, whereas Forastiere et al. (2016) defined three strata based on the treatment uptake status, excluding defiers. This difference is due to the randomization design. They considered
cluster randomized encouragement designs (CEDs), where encouragement is randomized at the level of a cluster of units. No randomization is carried out within the cluster. On the other hand, we consider the two-stage randomized design, where the encouragement is randomized at the level of a unit within clusters. All clusters are just assigned a treatment mechanism that governs the proportion of treated units within the cluster. This randomization procedure generates more complicated structures of the compliance behaviors for each unit since some units might behave differently under different assignment mechanisms. This complicated structure leads us to define the two-stage principal strata as previously defined.

This difference is critical in our analysis because (1) we are interested in the principal causal direct effect in each assignment mechanism, (2) the principal spillover effect is defined as the difference of the potential outcomes induced by the assignment mechanism, and (3) we need to capture the behavioral shift of units between assignment mechanisms. Also, it turns out that the Assumption 5 is a generalization of Assumption 3 of Vazquez-Bare (2021). The key difference is that we pose the monotonicity assumption on the compliance behaviors with respect to the assignment mechanism, whereas they posed the monotonicity directly on the treatment receipt. As a result, they reduced the compliance types to five instead of six. They also acknowledged that the ordering can be rearranged depending on the context, however, our definition of monotonicity can provide more flexible ordering of compliance types since we allow for all possible orderings for the compliance behavior.

We conclude this section with a discussion of another assumption, often referred to as exclusion restrictions. Let us first state the assumption.

**Assumption 6. (Exclusion Restrictions)**

For all units $i$ in cluster $j$ with $G_{ij} \in \{aa, nn, na\}$ under the assignment mechanism $a = a_0, a_1$,

$$Y_{ij}(0, a) = Y_{ij}(1, a)$$
For all units $i$ in cluster $j$ with $G_{ij} \in \{ca\}$,

$$Y_{ij}(0, a_1) = Y_{ij}(1, a_1)$$

For all units $i$ in cluster $j$ with $G_{ij} \in \{nc\}$,

$$Y_{ij}(0, a_0) = Y_{ij}(1, a_0)$$

This assumption captures the idea that there is no effect of the assignment on the outcome if the unit is either an always-taker or a never-taker under each assignment mechanism. We assume that they always (or never) receive the treatment no matter what condition they are assigned to, and, as stated in Assumption [1], the outcome of a unit is determined only through the treatment receipts of units within the same cluster.

### 3.2 Causal Estimands for Principal Causal Effects

We now define new causal estimands for principal causal effects. We argue that a little more attention must be paid on how we define the principal causal effects in a two-stage randomized experiment. Let us first look at how these quantities were defined in the literature. Imai et al. (2021) defined, under Assumption 3, the complier average direct effect ($CADE^I$) as follows.

$$CADE^I(a) = \frac{\sum_{j=1}^J \sum_{i=1}^{n_j} \{Y_{ij}(1, a) - Y_{ij}(0, a)\} \mathbb{1}\{D_{ij}(1, a) = 1, D_{ij}(1, a) = 0\}}{\sum_{j=1}^J \sum_{i=1}^{n_j} \mathbb{1}\{D_{ij}(1, a) = 1, D_{ij}(0, a) = 0\}}$$

for assignment mechanism $a$. Using our notations, the alternative to this quantity can be written as

$$CADE(a) = \frac{1}{N_c(a)} \sum_{(i,j):G_{ij}(a) = c} \{Y_{ij}(1, a) - Y_{ij}(0, a)\}$$

where $N_c(a) = \sum_{j=1}^J \sum_{i=1}^{n_j} \mathbb{1}(G_{ij}(a) = c)$.
The issue of the quantity in Equation (2) is that the treatment effect of the mechanism \( a \) is measured over the units who would comply under the same mechanism \( a \). However, it could be more of interest in practice if the treatment effect of the mechanism \( a_1 \) is defined over the units who comply under another mechanism \( a_0 \), or vice versa. In other words, we may be interested in the treatment effects under the target mechanism \( a_1 \) for compliers defined under the base mechanism \( a_0 \).

Let us define some terms to deal with this issue. We refer to the base mechanism as the mechanism at which compliers are defined. On the other hand, the target mechanism is referred to as the mechanism where the causal effects are measured. The issue of the quantities in Equation (2) is that it lacks the perspective of the base mechanism for compliers. It is important to separate the base mechanism from the target mechanism because the treatment receipt of units is influenced by treatment assignments of other units and the compliance behavior varies with the mechanisms. Note that the complier under the base mechanism may not be a complier under the target mechanism anymore, but it is still of our great interest. The importance of this notion becomes more apparent in the complier average spillover effect \( \text{CASE} \) and the complier average overall effect \( \text{CAOE} \). Taking these into account, let us define a new population-level \( \text{CADE} \) as follows.

\[
\text{CADE}(a; a^*) = \frac{1}{N_c(a^*)} \sum_{(i,j):G_{ij}(a^*)=c} \{Y_{ij}(1, a) - Y_{ij}(0, a)\}
\]

where \( a^* \) is the base mechanism and \( a \) is the target mechanism. If we set \( a^* = a \), then we obtain Equation (2). Thus, this is a generalization of Equation (2).

\( \text{CASE} \) was originally defined in Imai et al. (2021) as follows. For the treatment assignment \( z \),

\[
\text{CASE}'(z) = \frac{\sum_j \sum_{i=1}^{n_j} \{Y_{ij}(z, a_1) - Y_{ij}(z, a_0)\} 1\{D_{ij}(z, a_1) = 1, D_{ij}(z, a_0) = 0\}}{\sum_j \sum_{i=1}^{n_j} 1\{D_{ij}(z, a_1) = 1, D_{ij}(z, a_0) = 0\}}
\]

This quantity can be interpreted as a local average treatment effect for units who comply
with the treatment mechanism. Using our notations, the alternative to this estimand can be written as

\[
CASE(0) = \frac{1}{N_{ca} + N_{na}} \sum_{(i,j): G_{ij} \in \{ca, na\}} \{Y_{ij}(z, a_1) - Y_{ij}(z, a_0)\}
\]

\[
CASE(1) = \frac{1}{N_{nc} + N_{na}} \sum_{(i,j): G_{ij} \in \{nc, na\}} \{Y_{ij}(z, a_1) - Y_{ij}(z, a_0)\}
\]

Unfortunately, this estimand does not carry a clear interpretation as a spillover effect. Instead, we define a new complier average spillover effect using the idea of the base mechanism. For the treatment assignment \(z\) and the base mechanism \(a^*\),

\[
CASE(z, a^*) = \frac{1}{N_{c}(a^*)} \sum_{(i,j): G_{ij}(a^*) = c} \{Y_{ij}(z, a_1) - Y_{ij}(z, a_0)\}
\]

Similar to \(SEY(z)\) defined in Section 2.2, \(CASE(z, a^*)\) is the compliers’ local average spillover effects that compares the two potential values under two different assignment mechanisms, that is, \(a = a_1\) and \(a = a_0\) with treatment assignment fixed at \(z\). The compliers are defined under the base mechanism \(a^*\). Additionally, the unit-level \(CADE\) and \(CASE\) are defined as

\[
CADE_{ij}(a; a^*) = \{Y_{ij}(1, a) - Y_{ij}(0, a)\} \mathbb{1}(G_{ij}(a^*) = c)
\]

\[
CASE_{ij}(z; a^*) = \{Y_{ij}(z, a_1) - Y_{ij}(z, a_0)\} \mathbb{1}(G_{ij}(a^*) = c)
\]

Finally, we define the unit-level complier average overall effect (\(CAOE(a_0, a_1; a^*)\)) comparing two distinct assignment mechanisms under the base mechanism as follows.

\[
CAOE_{ij}(a_0, a_1; a^*) = \{Y_{ij}(a_1) - Y_{ij}(a_0)\} \mathbb{1}(G_{ij}(a^*) = c)
\] (3)

Plugging Equation (1) into (3), \(CAOE\) can be decomposed into the sum of the unit-level \(CADE\) and \(CASE\).

\[
CAOE_{ij}(a_0, a_1; a^*) = \frac{K_j(a_1)}{n_j} CADE_{ij}(a_1; a^*) - \frac{K_j(a_0)}{n_j} CADE_{ij}(a_0; a^*) + CASE_{ij}(0, a^*)
\]
Table 1: Abbreviations of causal estimands

| Abbreviation | Definition                                |
|--------------|-------------------------------------------|
| DEY          | Direct effect on the outcome              |
| DED          | Direct effect on the treatment receipt    |
| SEY          | Indirect effect on the outcome            |
| SED          | Indirect effect on the treatment receipt  |
| OEW          | Overall effect on the outcome             |
| CADE         | Complier average direct effect on the outcome |
| CASE         | Complier average indirect effect on the outcome |
| CAOE         | Complier average overall effect on the outcome |

The population-level $CAOE$ is defined as the average of the unit-level $CAOE$ over compliers under the base mechanism $a^*$.

$$CAOE(a_0, a_1; a^*) = \frac{1}{N_c(a^*)} \sum_{(i,j): G_{ij}(a^*)=c} CAOE_{ij}(a_0, a_1; a^*)$$

Table 1 presents the abbreviations of causal estimands we defined in Section 2.2 and 3.2 for reference purposes.

### 3.3 Estimation

We now formalize a model-based imputation approach for estimating treatment effects. We develop the likelihood function using a missing data approach, following Imbens and Rubin (2015). The key difference from the literature is that we need to impute the principal strata memberships and at most three missing potential outcomes for each unit since there are four potential outcomes, i.e., $Y_{ij}(0, a_0), Y_{ij}(1, a_0), Y_{ij}(0, a_1)$ and $Y_{ij}(1, a_1)$, and we can observe only one of them.

A general goal of the Bayesian approach in causal inference problem is to find a predictive distribution of the causal estimand $\tau$ given the observed outcomes $Y^{obs}$, treatment
assignment mechanism $A^{\text{obs}}$, treatment assignment $Z^{\text{obs}}$, treatment receipt $D^{\text{obs}}$ and covariates $X$, namely,

$$Pr(\tau|Y^{\text{obs}}, A^{\text{obs}}, Z^{\text{obs}}, D^{\text{obs}}, X)$$

Let $Y, A, Z, D, G$ and $X$ respectively denote the vectors of potential outcomes, treatment assignment mechanisms, treatment assignments, treatment receipts, compliance behaviors, and covariates for all units including all missing values. The covariates are a priori known to be unaffected by the two-stage treatment assignments. Any causal estimands of the form $\tau(Y, G, A, Z, X)$ can be written in terms of observed and missing variables as $\tau(Y^{\text{obs}}, Y^{\text{mis}}, D^{\text{obs}}, D^{\text{mis}}, A, Z, X)$ if $G$ is a one-to-one function of $(D^{\text{obs}}, D^{\text{mis}})$. We will discuss this correspondence in the following section. Now, we can see that these estimands are unknown because $(Y^{\text{mis}}, D^{\text{mis}})$ is unobservable. Therefore, in order to derive the posterior distribution of $\tau(Y^{\text{obs}}, Y^{\text{mis}}, D^{\text{obs}}, D^{\text{mis}}, A, Z, X)$, we need to derive the predictive distribution of the missing data $(Y^{\text{mis}}, D^{\text{mis}})$. In this section, we formalize the general structure of Bayesian inference in a two-stage experiments with interference and noncompliance and develop the inferential procedure based on the Gibbs sampling.

### 3.3.1 Correspondence between Principal Strata and Observed Data

Treatment receipt $D_{ij}(z, a)$ has four potential values for the treatment assignment $z$ and mechanism $a$. By Assumption 4 and 5, the number of principal strata (compliance behaviors) reduces from 16 to 6. The original 16 strata correspond to the $2^4$ patterns of possible potential values of treatment, where $D_{ij}(z, a) \in \{0, 1\}$ for $z = 0, 1$ and $a = a_0, a_1$. Table 2 shows the one-to-one correspondence between six compliance behaviors and potential treatment receipts. Table 3 presents the correspondence between observed data and principal strata that the units with the observation could belong to. With Assumption 5 we can narrow down the possible membership of units in the strata. For example, if we observe $A_j = a_0, Z_{ij} = 0, D_{ij} = 1$ for unit $i$ in cluster $j$, then its compliance type should be $aa$. 

21
Table 2: Compliance Behaviors and Treatment Receipts.

|       | $D_{ij}(0, a_0)$ | $D_{ij}(1, a_0)$ | $D_{ij}(0, a_1)$ | $D_{ij}(1, a_1)$ |
|-------|------------------|------------------|------------------|------------------|
| $cc$  | 0                | 1                | 0                | 1                |
| $aa$  | 1                | 1                | 1                | 1                |
| $nn$  | 0                | 0                | 0                | 0                |
| $ca$  | 0                | 1                | 1                | 1                |
| $nc$  | 0                | 0                | 0                | 1                |
| $na$  | 0                | 0                | 1                | 1                |

Without Assumption 5, its compliance type can be $ac, an$, which we excluded under the assumption.

### 3.3.2 Bayesian Inference

We will describe an overview of how Bayesian inference proceeds in our model and a set of assumptions about the unconfoundedness. Consider the quantities associated with each unit:

$(Y_{ij}(0, a_0), Y_{ij}(1, a_0), Y_{ij}(0, a_1), Y_{ij}(1, a_1), D_{ij}(0, a_0), D_{ij}(1, a_0), D_{ij}(0, a_1), D_{ij}(1, a_1), A_j, Z_{ij}, X_{ij})$

The first input into a model-based approach is the joint density of all these random variables.

$$Pr(Y(0, a_0), Y(1, a_0), Y(0, a_1), Y(1, a_1), D(0, a_0), D(1, a_0), D(0, a_1), D(1, a_1)|A, Z, X)$$

$$= Pr(Y(0, a_0), Y(1, a_0), Y(0, a_1), Y(1, a_1), D(0, a_0), D(1, a_0), D(0, a_1), D(1, a_1)|A, Z, X) Pr(Z|A, X) Pr(A|X) Pr(X)$$

$$= Pr(Y(0, a_0), Y(1, a_0), Y(0, a_1), Y(1, a_1), D(0, a_0), D(1, a_0), D(0, a_1), D(1, a_1)|A, Z, X) Pr(Z|A) Pr(A) Pr(X)$$
Table 3: Compliance Behaviors and Observed Data.

| $A_j$ | $Z_{ij}$ | $D_{ij}$ | Possible Compliance Behaviors |
|-------|---------|---------|------------------------------|
| $a_0$ | 0       | 0       | $cc, nn, ca, nc, na$         |
| $a_0$ | 1       | 0       | $nn, nc, na,$               |
| $a_0$ | 0       | 1       | $aa$                        |
| $a_0$ | 1       | 1       | $cc, aa, ca$                |
| $a_1$ | 0       | 0       | $cc, nn, nc$                |
| $a_1$ | 1       | 0       | $nn$                        |
| $a_1$ | 0       | 1       | $aa, ca, na$                |
| $a_1$ | 1       | 1       | $cc, aa, ca, nc, na$        |

$$
= Pr(Y(0, a_0), Y(1, a_0), Y(0, a_1), Y(1, a_1), D(0, a_0), D(1, a_0), D(0, a_1), D(1, a_1) | X)

Pr(Z|A)Pr(A)Pr(X)

$$

The second equality follows from the two-stage complete randomized design. At the first stage of randomization, the assignment mechanism for clusters is assumed to be independent of covariates. At the second stage of randomization, the treatment assignment for units just depends on the assignment mechanism. The third equality follows from the following unconfoundedness assumption.

**Assumption 7.** *(Unconfoundedness of the assignment mechanism and the treatment assignment.)*

$$(Z_{ij}, A_j) \perp \perp (Y_{ij}(z, a), D_{ij}(\tilde{z}, \tilde{a})) \mid X_{ij} = x$$

for $\forall x \in \mathcal{X}, z, \tilde{z} \in \{0, 1\}$, $a, \tilde{a} \in \{a_0, a_1\}$ and $\forall i, j$.

This assumption extends a standard unconfoundedness assumption to the two-stage randomization setting. It states that, conditional on each unit’s covariates $X_{ij}$, the assignment mechanism for clusters $A_j$ and the treatment assignment for units within cluster $Z_{ij}$
are independent of all the potential values of the treatment received and outcome. As shown in Table 2, there is a one-to-one mapping between the compliance behavior $G_{ij}$ and the treatment received $D_{ij}(z, a)$. Therefore, Assumption 7 implies the latent unconfoundedness shown below.

**Assumption 8.** (Unconfoundedness of the assignment mechanism and the treatment assignment.)

$$(Z_{ij}, A_j) \perp \!\!\!\!\perp (Y_{ij}(z, a)) \mid G_{ij} = g, X_{ij} = x$$

for $\forall x \in \mathcal{X}$, $z \in \{0, 1\}$, $a \in \{a_0, a_1\}$ and $\forall i, j$.

Note that Assumption 7 and 8 imply the same thing.

In what follows, we will condition on the observed distribution of covariates $X$, so that $Pr(X)$ does not need to be modeled. Assuming unit exchangeability and by appealing to de Finetti’s theorem,

$$Pr(Y(0, a_0), Y(1, a_0), Y(0, a_1), Y(1, a_1), D(0, a_0), D(1, a_0), D(0, a_1), D(1, a_1) \mid X)$$

$$= \int \prod_{i,j} Pr(Y_{ij}(0, a_0), Y_{ij}(1, a_0), Y_{ij}(0, a_1), Y_{ij}(1, a_1), D_{ij}(0, a_0), D_{ij}(1, a_0), D_{ij}(0, a_1), D_{ij}(1, a_1) \mid X_{ij}; \theta)p(\theta)d\theta$$

$$= \int \prod_{i,j} Pr(G_{ij} \mid X_{ij}; \theta)Pr(Y_{ij}(0, a_0), Y_{ij}(1, a_0), Y_{ij}(0, a_1), Y_{ij}(1, a_1) \mid X_{ij}, G_{ij}; \theta)p(\theta)d\theta$$

(4)

A justification for assuming unit exchangeability in a two-stage randomized experiment with interference will be discussed in the supplementary material A.3.

In order to obtain the posterior distribution of causal estimand $\tau(Y, G, A, Z, X)$, we need the posterior distribution of the missing data $(G, Y^{mis})$ given observed data $(X, A, Z, D^{obs}, Y^{obs})$. Generally speaking, it is difficult to specify the posterior, so we will begin with three models.

- The model for principal stratum membership $G_{ij}$ given the covariates and parameters:
  i.e., $Pr(G_{ij} \mid X_{ij}; \theta)$

24
- The model for the potential outcomes $Y_{ij}(z, a)$ given the covariates, parameters and principal stratum membership; i.e., $Pr(Y_{ij}(0, a_0), Y_{ij}(1, a_0), Y_{ij}(0, a_1), Y_{ij}(1, a_1)|X_{ij}, G_{ij} : \theta)$

- The prior distribution of $\theta$: i.e., $p(\theta)$

Then, we use the Gibbs sampling to obtain the joint posterior distribution $Pr(G, \theta|X, A, Z, D^{obs}, Y^{obs})$. Specifically, we iteratively draw between $Pr(G|X, A, Z, D^{obs}, Y^{obs}; \theta)$ and $Pr(\theta|X, A, Z, D^{obs}, Y^{obs}, G)$. Finally, using these draws, we can obtain the predictive distribution of $Y^{mis}$, and hence of $\tau(Y, G, A, Z, X)$. The specification of the models is subject to the analyst’s choice. We will present models tailored for our analysis in the following section.

### 3.4 Gibbs Sampling

We derive an MCMC algorithm to get the posterior distributions of the causal estimands. We follow the general procedure presented in Section 3.3.2. As described earlier, we will use the Gibbs sampling. The algorithm proceeds as follows.

**Step 1.** Initialize parameters $\theta^{(0)}$.

**Step 2.** Sample from $Pr(G^{(t+1)}|A, Z, D^{obs}, Y^{obs}; \theta^{(t)})$

**Step 3.** Sample from $Pr(Y^{mis^{(t+1)}}|A, Z, Y^{obs}, G^{(t+1)}; \theta^{(t)})$

**Step 4.** Sample from $Pr(\theta^{(t+1)}|A, Z, D^{obs}, Y^{obs}, G^{(t+1)}; \theta^{(t)})$

**Step 5.** Repeat Step 2-4.

By repeating the steps above, we can obtain the posterior draws of all the missing potential outcomes and compliance behaviors for all units. Then we can obtain our estimates by imputing these values into the causal estimands of interest defined in Section 2.2 and 3.2. Since we are using tractable distributions for our model, it is possible to derive all the posterior distributions in a closed-form with appropriate prior distributions. The supplementary material provides the detailed derivations for each step.
4 Simulation Studies

In this section, we will validate our proposed Bayesian model by comparing it with the one proposed in Imai et al. (2021) in terms of frequentist properties, such as bias, mean square error (MSE), and coverage. For comparisons, we will use the package that the authors have their methods published as an open-source software. First, we conduct simulation studies with the data generating process that mimics the observed data we analyze in Section 5. Additional simulation studies using other data generating processes are given in the supplementary material A.6 with a brief discussion on robustness to model misspecification.

First of all, we conduct two-stage randomization for $N$ units and $J$ clusters. Each cluster has an equal size of units, that is, $n_j = N/J$. The two-stage randomization is carried out based on the following assignment probabilities.

$$
Pr(A_j = a_0) = 0.5
$$
$$
Pr(Z_{ij} = 1 | A_j = a_0) = 0.4
$$
$$
Pr(Z_{ij} = 1 | A_j = a_1) = 0.8
$$

Now, we assume that each experimental unit belongs to one of latent principal strata $G_{ij} = g \in \{cc, aa, nn, ca, nc, na\}$ with unknown probabilities $\pi$.

$$
G_{ij} \sim \text{Categorical}(\pi)
$$

where $\pi = (\pi_{cc}, \pi_{aa}, \pi_{nn}, \pi_{ca}, \pi_{nc}, \pi_{na})$ such that $\sum_{g \in \{cc,aa,nn,ca,nc,na\}} \pi_g = 1$.

Our exploratory data analysis implies that the outcome values $Y_{ij}^{obs}$ has an atypical right-skewed distribution with an excess of zero values. To mimic the observed data, we assume that the observed and missing potential outcomes for each unit $Y_{ij}(z, a)$ follow a mixture distribution of Bernoulli $W_{ij}$ and Log-Normal distribution with parameters specific to each strata and assignments, which represent the excess of zero values and the heavy tail of the data. Specifically, we assume the following ”true” data-generating process for
both observed and missing potential outcomes of unit $i$ in cluster $j$. For $a \in \{a_0, a_1\}$ and $z \in \{0, 1\}$,

\[
W_{ij}(z, a) \mid A_j = a, Z_{ij} = z, G_{ij} = g \sim Bernoulli(p_{g}^{z,a})
\]

\[
X_{ij}(z, a) \mid A_j = a, Z_{ij} = z, G_{ij} = g \sim LogNormal(\mu_{g}^{z,a}, \sigma_{g}^{z,a^2})
\]

\[
Y_{ij}(z, a) = \{1 - W_{ij}(z, a)\} X_{ij}(z, a)
\]

where $W_{ij}$ is the latent variable such that the potential outcome $Y_{ij} = 0$ if $W_{ij} = 1$. For the sake of simplicity, we assume all missing potential outcomes are independent. We also take Assumption 6 into account. This assumption needs to apply to both $W_{ij}$ and $X_{ij}$. The parameter values above are provided in the supplementary material.

Recall that we defined our estimands in Section 2.2 and 3.2 under the finite-population perspective, which means the randomness of the data is only due to the treatment assignments, and we defined the estimands conditional on the finite sample. However, for the sake of the evaluation, we will take the super-population perspective in this section. All evaluations are based on the super-population versions of the estimands. There are several reasons why we are taking this perspective here. First, we can calculate the true values of the causal estimands since we know the true data-generating process. If we take the finite-sample perspective and the estimands are defined on the generated samples, which is random, thus we obtain different values of estimands in every round of simulation. This fluctuation over rounds makes the evaluation unstable within the finite rounds. Fortunately, since we can naturally define the super-population version of each estimand for this simulation, this perspective shift would not much hurt the validity of our simulation. For the sake of simplicity of comparison, we will only focus on $CADE(0; a_0)$, $CADE(1; a_1)$, $DEY(0)$, $DEY(1)$, $DED(0)$ and $DED(1)$, which were provided and precisely estimated in

27
CADE \(sp(0; a_0) = \mathbb{E}[Y_{ij}(1, a_0) - Y_{ij}(0, a_0) \mid G_{ij} \in \{cc, ca\}]\)

CADE \(sp(1; a_1) = \mathbb{E}[Y_{ij}(1, a_1) - Y_{ij}(0, a_1) \mid G_{ij} \in \{cc, nc\}]\)

DEY \(sp(0) = \mathbb{E}[Y_{ij}(1, a_0) - Y_{ij}(0, a_0)]\)

DEY \(sp(1) = \mathbb{E}[Y_{ij}(1, a_1) - Y_{ij}(0, a_1)]\)

DED \(sp(0) = \mathbb{E}[D_{ij}(1, a_0) - D_{ij}(0, a_0)]\)

DED \(sp(1) = \mathbb{E}[D_{ij}(1, a_1) - D_{ij}(0, a_1)]\)

The supplementary material provides details for the calculation of the above estimands.

Finally, note that our simulation studies consider a situation where the model is well-specified. We should be aware of the fact that the definitive conclusions in our analysis are drawn under the assumption of the model being well-specified even though we are using the simulated dataset that seems to possess some of the aspects of the observed data that we use in our analysis, i.e., an excess of zeros and heavy-tail. However, we believe that the effect of model misspecification would be marginal in our analysis. An empirical study on model misspecification is given in Section A.6. Detailed research of the robustness to model misspecification is left to future research.

4.1 Discussions

We generate \(N_{sim} = 500\) datasets and run simulation \(N_{sim}\) times for \(N = 5000, 10000, 50000\) with a fixed cluster size \(J = 100\). Table 4.1 shows the results for \(CADE(0, a_0)\) and \(CADE(1, a_1)\). Other results are provided in the supplementary material. We refer to the method in Imai et al. (2021) as the frequentist approach in this section. The coverage is calculated based on the 95% confidence interval. For the Bayesian approach, we use the posterior median as the point estimate for calculating bias and MSE, and 95% credible
Table 4: Simulation Results

| Coverage | Bias | MSE |
|----------|------|-----|
|          |      |     |

**CADE(0, a_0)**

| N  | Freq | Bayes | Freq Post. Median | Freq Post. Median |
|----|------|-------|-------------------|-------------------|
| 5000 | 96%  | 98%   | 2.40E+02          | 4.50E+02          |
| 10000 | 97%  | 98%   | -2.72E+02         | -2.16E+02         |
| 50000 | 94%  | 97%   | -6.51E+01         | 1.99E+02          |

**CADE(1, a_1)**

| N  | Freq | Bayes | Freq Post. Median | Freq Post. Median |
|----|------|-------|-------------------|-------------------|
| 5000 | 92%  | 98%   | -8.67E+02         | 1.18E+03          |
| 10000 | 93%  | 96%   | 7.40E+01          | -6.48E+02         |
| 50000 | 95%  | 97%   | 3.28E+02          | 1.85E+02          |

interval for calculating 95% coverage. Each metric is defined as follows.

\[
Coverage = \frac{1}{N_{sim}} \sum_{k=1}^{N_{sim}} 1(\hat{\mu}_{k,lower} \leq \mu \leq \hat{\mu}_{k,upper})
\]

\[
Bias = \frac{1}{N_{sim}} \sum_{k=1}^{N_{sim}} (\hat{\mu}_k - \mu)
\]

\[
MSE = \frac{1}{N_{sim}} \sum_{k=1}^{N_{sim}} (\hat{\mu}_k - \mu)^2
\]

where \( \mu, \hat{\mu}_k, \hat{\mu}_{k,lower} \) and \( \hat{\mu}_{k,upper} \) are the true estimand under the super-population perspective, the point estimate for the \( k \)th round of the simulation, the 95% lower and upper bounds of intervals respectively.

The frequentist approach and our Bayesian approach both achieve approximately 95% coverage. In terms of bias, both methods are working well. One might say that the
frequentist method sometimes seems to perform slightly better than the posterior median in terms of bias. This is due to two reasons. First, the effect of prior distributions is not negligible for a small sample size, \( N = 5000 \). Second, we choose the posterior median for the point estimate of our estimator due to its robustness, however, the sample median is not an unbiased estimator for the mean of the log-normal distribution. We believe this should not be a big issue since the Bayesian usually cares more about the posterior distribution with a well-calibrated coverage. Also, this superiority of the frequentist method comes with the price of high MSEs. In terms of MSE, the Bayesian method outperforms the frequentist approach under all conditions. The lower MSE implies that the estimate is less likely to deviate from the true causal effect over multiple experiments. It also implies a lower variance of the estimator and a narrower credible interval. In other words, by appealing to the model-based approach, we achieved a tighter estimation of the causal effect. We can also see that both bias and MSE decrease as the number of samples increases in the Bayesian model. This is a natural observation because, according to the Bernstein-von Mises theorem, the posterior distribution of parameters converges to a normal distribution centered at the true parameter values with the inverse Fisher information as variance in a well-specified setting (under some more regularity conditions), as the sample size \( N \) increases, i.e., the influence of the prior distribution vanishes. The frequentist estimators, however, are negatively biased with large MSEs. This shows that the frequentist approach is sensitive to the atypical shape of the distribution, i.e., the non-gaussian heavy-tailed shape with an excess of zero values. As a consequence, the variance estimator tends to be larger to deal with outliers. A simulation study on different data generating process with relatively fewer outliers is provided in the next section.
5 Application to RSBY Study

We apply our method to Rastriya Swasthya Bima Yojana (RSBY) health insurance dataset. About 63 million people in India are below the poverty line (BPL) due to health care expenditures. In 2008, a large-scale national hospital insurance scheme for the poor (RSBY) was launched. RSBY is a large-scale national hospital insurance scheme for poor households, which can be joined at a nominal co-payment. Families below BPL can cover up to five people for more than 700 medical treatments and procedures under RSBY at a price set by the government. Medical services are provided nationwide by government-contracted public and private hospitals, and beneficiaries use their RSBY biometric ID cards, eliminating the need for cash transactions and insurance claims, see Nandi et al. (2015) and references therein for more details.

Imai et al. (2021) analyzed this dataset and conducted a randomized controlled trial to determine whether RSBY increases access to hospitalization, and reduced impoverishment due to high medical expenses. In this evaluation, they are concerned about the spillover effects between households since whether or not one household enrolls in RSBY may depend on the treatment assignment of other households. On top of that, noncompliance must be addressed because some households in the treatment group may decide not to enroll in RSBY, while some households in the control group managed to join RSBY. Unfortunately, they could not draw strong conclusions about the direct treatment effects and spillover effects in the sense that very few of the estimators were statistically significant, making it difficult to draw a definite conclusion about whether or not the proportion of treated households in a village directly affects one’s outcome.

We also focus on the annual household hospital expenditure, which ranges from 0 to INR 500,000. The 10,072 households in 435 villages were included in a two-stage randomized experiment. Under the two-stage randomization design, $J_1 = 219$ villages were
Table 5: Comparisons with the literature.

| Case                | Mean | Median | 95% Interval       | Imai et al. (2021) |
|---------------------|------|--------|--------------------|--------------------|
| CADE($a_1; a^* = a_1$) | −1876 | −1766  | (−3703, −227)      | −1649(1061)        |
| CADE($a_0; a^* = a_0$) | 759   | 171    | (−1311, 2443)      | 1984(1215)         |
| DEY($a_1$)          | −786  | −737   | (−1559, −96)       | −795(514)          |
| DEY($a_0$)          | 355   | 79     | (−610, 1122)       | 875(530)           |
| DED($a_1$)          | 0.419 | 0.419  | (0.398, 0.439)     | 0.482(0.023)       |
| DED($a_0$)          | 0.464 | 0.464  | (0.445, 0.481)     | 0.441(0.021)       |
| SEY(1)              | −1344 | −1066  | (−1919, −480)      | −1374(823)         |
| SEY(0)              | −203  | −237   | (−1028, 660)       | 297(858)           |
| SED(1)              | 0.032 | 0.031  | (0.021, 0.049)     | 0.086(0.053)       |
| SED(0)              | 0.077 | 0.076  | (0.060, 0.096)     | 0.045(0.028)       |

assigned to the assignment mechanism $a_1$, with the assignment probability within the cluster being 80%, whereas the rest of the villages were assigned to the mechanism $a_0$ with 40% assignment probability.

We used the same mixture model as in (5) to analyze the RSBY dataset and ran the MCMC algorithm for 100,000 iterations using a burn-in of 50,000. The iteration numbers were chosen after experimentation to deliver stable results over multiple runs. Table 5 compares our estimates with the results of Imai et al. (2021). $CASE(0)$ and $CASE(1)$ are not shown here for several reasons. First, we defined $CASE$s differently from their definitions. As described earlier in 3.2, their definition is hard to interpret. Thus we redefined new complier spillover effects. Second, they also mentioned in their paper $CASE$s are imprecisely estimated. Basically, many of our results are consistent with existing results but there are a few things worth noting. For example, the 95% credible intervals of $CADE(a_1; a^* = a_1)$, $DEY(a_1)$ and $SEY(1)$ are negative. Since these
effects were not significant in Imai et al. (2021), we can say they are a little more definitive results. These results are explainable by the results of simulation studies in Section 4. By appealing to the model-based approach, we are able to make the tighter estimation of the causal effects, robust to some outlying observations, i.e., there are 36 observations greater than INR 100,000, including INR 403,000 and 500,000, the two largest ones, whereas the median is only INR 1,000.

Specifically, SEY(0) and SEY(1) measure the spillover effect for all units when units are assigned control and treatment respectively. The negative spillover effect is detected for SEY(1), even though it is not significant for SEY(0). This result indicates that treated people are more likely to get negatively affected by the shift of treatment mechanism. In other words, assigning a greater proportion of households to the treatment condition makes another treated household of the same village spend less expenditure.

Table 6 presents a summary of the rest of the results not shown in Table 5. Interestingly, the credible intervals of the overall effects for both all units and compliers are negative.

|                  | Post. mean | Post. median | 95% credible interval |
|------------------|------------|--------------|-----------------------|
| OY \((a_1, a_0)\) | -915       | -746         | (-1420, -197)         |
| CAOE \((a_1, a_0; a^* = a_0)\) | -1546      | -1159        | (-2664, -38)          |
| CAOE \((a_1, a_0; a^* = a_1)\) | -1274      | -1292        | (-2547, -69)          |
| CADE \((a_1; a^* = a_0)\) | -1642      | -1581        | (-3290, -192)         |
| CADE \((a_0; a^* = a_1)\) | 220        | 193          | (-1462, 2083)         |
| CASE \((0; a^* = a_0)\) | -43        | -109         | (-1612, 1771)         |
| CASE \((1; a^* = a_0)\) | -2444      | -1837        | (-3981, -564)         |
| CASE \((0; a^* = a_1)\) | -6         | -124         | (-1820, 1986)         |
| CASE \((1; a^* = a_1)\) | -2103      | -2055        | (-3768, -663)         |
Unarguably, the overall effects are of the greatest interest to policy makers since they are defined as the comparison of two policies. Specifically, $OYE(a_1, a_0)$ can be viewed as a pure impact on all units of intervention. That is, this is interpreted as how outcomes of interest would change when the policy changes from $a_0$ to $a_1$. On the other hand, $CAOE$s imply that the overall effects are negative for units who comply with the assignment regardless of which mechanism they belong to, $a_0$ or $a_1$.

The 95% interval of $CADE(a_1; a^* = a_0)$ is negative. Noting that $CADE(a_1; a^* = a_1)$ is also negative in Table 5, the direct effect of the treatment assignment is negative for compliers, regardless of which mechanism they belong to, $a_0$ or $a_1$.

Finally, the spillover effects of treatment assignment on complier’s outcomes, i.e., $CASE(1; a^* = a_0)$ and $CASE(1; a^* = a_1)$, are negative, which means that the spillover effect on compliers is significantly negative, regardless of whether they are a complier under $a_0$ or $a_1$. Moreover, together with the result of $SEY(1)$, the spillover effect of treatment assignment on units, regardless of whether they are a complier or not, is negative.

6 Conclusions

In this article, we made three contributions. First, we provided a Bayesian framework based on principal stratification for a two-stage randomized experiment with interference and noncompliance. The model was validated by simulation studies. The simulation study indicated that our model allows for tighter estimation of the causal effects when the data contains the mass on the tail or some outliers. Second, we present sets of assumptions about compliance behaviors within and across clusters. In addition, new causal estimands, including the overall effects of intervention and interpretable spillover effects, were proposed with the flexibility of Bayesian modeling. Finally, we applied our methodology to the RSBY dataset, and find more definitive evidence of the spillover effects and overall effects of the
intervention. These results were not found in the previous literature.

For future research, it is of the greatest interest to relax the assumptions of the interference structure. In some applications, it may be too restrictive to employ the two-stage randomized design and make the stratified interference assumption. A great deal of research has been done on estimating causal effects of intervention without using special cluster-based designs such as the two-stage randomized design or clustered encouragement design. Aronow and Samii (2017) and the line of their work provided one possible future path based on the network structure and the exposure mapping. It would be interesting to further explore how effectively we can draw causal conclusions by exploiting (or not even knowing) the network structure between experimental units.

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A Supplementary Material

A.1 Parameters for Simulation Studies

Table 7 and 10 present the parameters used for simulation studies.

A.2 Derivations of Causal Estimands in the Super-population Perspective.

\[
CADE_{sp}(0, a_0) = E[Y_{ij}(1, a_0) - Y_{ij}(0, a_0) \mid G_{ij} \in \{cc, ca\}]
= E[(1 - W_{ij}(1, a_0))X_{ij}(1, a_0) - (1 - W_{ij}(0, a_0))X_{ij}(0, a_0) | G_{ij} \in \{cc, ca\}]
=(1 - E[W_{ij}(1, a_0) | G_{ij} \in \{cc, ca\}]) E[X_{ij}(1, a_0) | G_{ij} \in \{cc, ca\}]
- (1 - E[W_{ij}(0, a_0) | G_{ij} \in \{cc, ca\}]) E[X_{ij}(0, a_0) | G_{ij} \in \{cc, ca\}]
\]

\[
= \left(\frac{\pi_{cc}}{\pi_{cc} + \pi_{ca}}(1 - p_{cc}^{1,0}) + \frac{\pi_{ca}}{\pi_{cc} + \pi_{ca}}(1 - p_{ca}^{1,0})\right)\left(\frac{\pi_{cc}}{\pi_{cc} + \pi_{ca}} \exp \left(\mu_{cc}^{1,0} + \frac{\sigma_{cc}^{1,0^2}}{2}\right) + \frac{\pi_{ca}}{\pi_{cc} + \pi_{ca}} \exp \left(\mu_{ca}^{1,0} + \frac{\sigma_{ca}^{1,0^2}}{2}\right)\right)
- \left(\frac{\pi_{cc}}{\pi_{cc} + \pi_{ca}}(1 - p_{cc}^{0,0}) + \frac{\pi_{ca}}{\pi_{cc} + \pi_{ca}}(1 - p_{ca}^{0,0})\right)\left(\frac{\pi_{cc}}{\pi_{cc} + \pi_{ca}} \exp \left(\mu_{cc}^{0,0} + \frac{\sigma_{cc}^{0,0^2}}{2}\right) + \frac{\pi_{ca}}{\pi_{cc} + \pi_{ca}} \exp \left(\mu_{ca}^{0,0} + \frac{\sigma_{ca}^{0,0^2}}{2}\right)\right)
= 4765.78
\]

The third equality follows from \(W_{ij}(z, a) \perp \perp X_{ij}(z, a)\). The fourth equality follows from a simple calculation of conditional expectation and the fact that \(W_{ij}\) follows the Bernoulli distribution and \(X_{ij}\) follows the log-normal distribution. \(CADE_{sp}(1; a_1)\) can be calculated...
in the same way. The rest is easy to compute.

\[ CADE_{sp}(1; a_1) = 5156.41 \]
\[ DEY_{sp}(0) = 2382.89 \]
\[ DEY_{sp}(1) = 2324.13 \]
\[ DED_{sp}(0) = \pi_{cc} + \pi_{ca} = 0.5 \]
\[ DED_{sp}(1) = \pi_{cc} + \pi_{nc} = 0.45 \]

### A.3 On Exchangeability

De Finetti’s theorem plays a fundamental role in Bayesian inference and unit exchangeability is a key assumption for the theorem. We briefly discuss the justification of exchangeability under interference between units and see how much more we can squeeze out of the common assumptions in the literature, Assumption 2 and 3.

First, consider two units that belong to different clusters. They are not interfered with each other due to Assumption 2. Therefore, it is plausible to assume their outcomes are independent if they are in different clusters. Also, noting that the covariates are independent of treatment assignments, one’s outcomes \((Y_{ij}(0, a_0), Y_{ij}(1, a_0), Y_{ij}(0, a_1), Y_{ij}(1, a_1))\) and \((D_{ij}(0, a_0), D_{ij}(1, a_0), D_{ij}(0, a_1), D_{ij}(1, a_1))\) would be identical even if they move to a different cluster. Thus exchanging units does not affect the joint distribution for all units, hence, they are exchangeable.

Let us now consider two units \(i_1\) and \(i_2\) within the same cluster \(j\). In such a case, they are not independent because their outcomes can be affected by each other through interference. Consider a situation where both \(i_1\) and \(i_2\) are assigned treatment. Assumption 3 states that the interference is determined by the total number of treated units within the same cluster. Therefore, if both \(i_1\) and \(i_2\) are treated, the joint distribution of all units in cluster \(j\) remains the same even if they are exchanged because the number of treated units
remains the same. So, they are exchangeable. The same argument applies to a case where both \(i_1\) and \(i_2\) are untreated within the same cluster. If either of them is treated and the other is untreated, then the total number of treated units with the cluster still remains the same, thus the effect on the rest of the units within the same cluster would not change. The joint distribution of units \(i_1\) and \(i_2\) will not change since it is still either of them being treated and the other being untreated. Therefore, the exchangeability holds in such a case as well.

Therefore, Assumption 2 and 3 justify de Finetti’s theorem in the two-stage randomized experiment, and further justifies the existence of parametric models defined in (4). Without these assumptions, however, the unit exchangeability may not hold because the interference structure may collapse if we exchange a unit with one another. This would affect the joint distribution of all units, hence, unit exchangeability is no longer valid.

A.4 Derivations of Gibbs Samplers

The algorithm steps are restated here.

**Step 1.** Initialize parameters \(\theta^{(0)}\).

**Step 2.** Sample from \(P_{r}(G^{(t+1)}|A, Z, D_{\text{obs}}, Y_{\text{obs}}; \theta^{(t)})\)

**Step 3.** Sample from \(P_{r}(Y_{\text{mis}}^{(t+1)}|A, Z, Y_{\text{obs}}, G^{(t+1)}; \theta^{(t)})\)

**Step 4.** Sample from \(P_{r}(\theta^{(t+1)}|A, Z, D_{\text{obs}}, Y_{\text{obs}}, G^{(t+1)}; \theta^{(t)})\)

**Step 5.** Repeat Step 2-4.

Assuming the compliance behaviors for units are independent in Step 2, we will first focus on the individual conditional probability.

\[
P_{r}(G_{ij}^{(t+1)} | A_j, Z_{ij}, D_{ij}^{\text{obs}}, Y_{ij}^{\text{obs}}, \theta^{(t)})
\]

Let \(\theta\) be a vector of all parameters shown in Table 7. We will sample from Equation (6) with reference to Table 3. For example, for the units with \((A_j, Z_{ij}, D_{ij}) = (a_0, 0, 1)\) observed,
their compliance behavior is \(aa\) with probability 1. So, there is no need to sample for them. For the units with \((A_j, Z_{ij}, D_{ij}) = (a_0, 0, 0)\), there are five possible compliance behaviors for them, i.e., \(G_{ij} \in \{cc, nn, ca, nc, na\}\). The conditional distribution of \(G_{ij} = g\) given \((A_j, Z_{ij}, D_{ij}) = (a_0, 0, 0)\) and \(\theta\) in Equation (6) is given by the Categorical distribution with probability \(p_g\) for \(g \in \{cc, nn, ca, nc, na\}\). Noting that \(Y_{ij}\) is a mixture of a Bernoulli random variable \(W_{ij}\) and a log-normal random variable \(X_{ij}\), we have

\[
p_g = Pr(G_{ij}^{(t+1)} = g \mid A_j = a_0, Z_{ij} = 0, D_{ij}^{obs} = 0, Y_{ij}^{obs} = y; \theta^{(t)})
\]

\[
= \frac{\pi_g(t) p_{g,0}^{(t)} I(y=0) ((1 - p_{g,0}^{(t)}) f(y; \mu_g^{0,0(t)}, \sigma_g^{0,0(t)}) )}{\sum_{g \in \{cc, nn, ca, nc, na\}} \pi_g(t) p_{g,0}^{(t)} I(y=0) ((1 - p_{g,0}^{(t)}) f(y; \mu_g^{0,0(t)}, \sigma_g^{0,0(t)}) )} I(y>0)
\]

where \(f(y; \mu_g^{0,0}, \sigma_g^{0,0})\) is a probability density function (pdf) of the log-normal distribution with parameters \(\mu_g^{0,0}\) and \(\sigma_g^{0,0}\). We can follow the same procedure to obtain samples of compliance behavior \(G_{ij}\) for any combination of observations, \((A_j, Z_{ij}, D_{ij})\).

In Step 3, we just need to impute the missing potential outcomes for unit \(i\) in cluster \(j\) using the parameter \(\theta^{(t)}\) and the compliance behaviors \(G_{ij}^{(t+1)}\), paying attention to Assumption 6. We need to impute the missing values such that Assumption 6 holds. Our imputation process is a little bit tricky because we are using a mixture model. The imputation is two-fold. First, we sample \(W_{ij}^{(t+1)} \sim Bernoulli(p^{(t)})\). Then, if \(W_{ij}^{(t+1)} = 0\), we sample \(X_{ij}^{(t+1)} \sim LogNormal(\mu^{(t)}, \sigma^{(t)})\) for missing potential outcomes. Assumption 6 must hold for both \(W_{ij}^{(t+1)}\) and \(X_{ij}^{(t+1)}\). For example, if we observe \((A_j, Z_{ij}, D_{ij}) = (a_0, 0, 0)\) and are given \(G_{ij}^{(t+1)} = cc\) 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. If \(G_{ij}^{(t+1)} = aa\), we can invoke Assumption 6 and just need to impute either \(Y_{ij}(0, a_1)\) or \(Y_{ij}(1, a_1)\), depending on the observed treatment assignment for the unit. On the other hand, if we for example observe \((A_j, Z_{ij}, D_{ij}) = (a_0, 1, 0)\) and are given \(G_{ij}^{(t+1)} = nc\), we need to impute two missing potential outcomes \(Y_{ij}(0, a_1), Y_{ij}(1, a_1)\) because \(Y_{ij}(1, a_0)\) is observed in this case and \(Y_{ij}(0, a_0) = Y_{ij}(1, a_0)\) from Assumption 6.
Finally, in Step 4, we use conjugate prior distributions for all variables. We refer readers to any statistics textbook for the posterior derivation using conjugate prior distributions. If the closed-form posteriors are not obtainable, then we may use some other MCMC methods for this step, such as Metropolis-Hasting and Hamiltonian Monte Carlo. We will update each parameter in $\theta$ one by one by the Gibbs sampling. Had we observed the full compliance
types of the units, the resulting complete-data likelihood is

\[ L_{\text{comp}}(\theta \mid A, Z, D^{\text{obs}}, Y^{\text{obs}}, G) = \prod_{(i,j): G_{ij} = cc, Z_{ij} = 0, A_j = a_0} f(Y(0, 0) \mid G_{ij} = cc, \mu_{cc}^{0,aa}, \sigma_{cc}^{0,aa}, p_{cc}^{0,aa}) \prod_{(i,j): G_{ij} = cc, Z_{ij} = 1, A_j = a_0} f(Y(1, 0) \mid G_{ij} = cc, \mu_{cc}^{1,aa}, \sigma_{cc}^{1,aa}, p_{cc}^{1,aa}) \prod_{(i,j): G_{ij} = cc, Z_{ij} = 0, A_j = a_1} f(Y(0, 1) \mid G_{ij} = cc, \mu_{cc}^{0,a1}, \sigma_{cc}^{0,a1}, p_{cc}^{0,a1}) \prod_{(i,j): G_{ij} = cc, Z_{ij} = 1, A_j = a_1} f(Y(1, 1) \mid G_{ij} = cc, \mu_{cc}^{1,a1}, \sigma_{cc}^{1,a1}, p_{cc}^{1,a1}) \prod_{(i,j): G_{ij} = aa, A_j = a_0} f(Y(0, 0) \mid G_{ij} = aa, \mu_{aa}^{0,aa}, \sigma_{aa}^{0,aa}, p_{aa}^{0,aa}) \prod_{(i,j): G_{ij} = aa, A_j = a_1} f(Y(0, 1) \mid G_{ij} = aa, \mu_{aa}^{0,a1}, \sigma_{aa}^{0,a1}, p_{aa}^{0,a1}) \prod_{(i,j): G_{ij} = nn, A_j = a_0} f(Y(0, 0) \mid G_{ij} = nn, \mu_{nn}^{0,aa}, \sigma_{nn}^{0,aa}, p_{nn}^{0,aa}) \prod_{(i,j): G_{ij} = nn, A_j = a_1} f(Y(0, 1) \mid G_{ij} = nn, \mu_{nn}^{0,a1}, \sigma_{nn}^{0,a1}, p_{nn}^{0,a1}) \prod_{(i,j): G_{ij} = ca, Z_{ij} = 0, A_j = a_0} f(Y(0, 0) \mid G_{ij} = ca, \mu_{ca}^{0,aa}, \sigma_{ca}^{0,aa}, p_{ca}^{0,aa}) \prod_{(i,j): G_{ij} = ca, Z_{ij} = 1, A_j = a_0} f(Y(1, 0) \mid G_{ij} = ca, \mu_{ca}^{0,a1}, \sigma_{ca}^{0,a1}, p_{ca}^{0,a1}) \prod_{(i,j): G_{ij} = ca, A_j = a_1} f(Y(0, 1) \mid G_{ij} = ca, \mu_{ca}^{0,a1}, \sigma_{ca}^{0,a1}, p_{ca}^{0,a1}) \prod_{(i,j): G_{ij} = nc, A_j = a_0} f(Y(0, 0) \mid G_{ij} = nc, \mu_{nc}^{0,aa}, \sigma_{nc}^{0,aa}, p_{nc}^{0,aa}) \prod_{(i,j): G_{ij} = nc, A_j = a_1} f(Y(0, 1) \mid G_{ij} = nc, \mu_{nc}^{0,a1}, \sigma_{nc}^{0,a1}, p_{nc}^{0,a1}) \prod_{(i,j): G_{ij} = nc, Z_{ij} = 0, A_j = a_1} f(Y(0, 1) \mid G_{ij} = nc, \mu_{nc}^{0,a1}, \sigma_{nc}^{0,a1}, p_{nc}^{0,a1}) \prod_{(i,j): G_{ij} = nc, Z_{ij} = 1, A_j = a_1} f(Y(1, 0) \mid G_{ij} = nc, \mu_{nc}^{0,a1}, \sigma_{nc}^{0,a1}, p_{nc}^{0,a1}) \prod_{(i,j): G_{ij} = na, A_j = a_0} f(Y(0, 0) \mid G_{ij} = na, \mu_{na}^{0,aa}, \sigma_{na}^{0,aa}, p_{na}^{0,aa}) \prod_{(i,j): G_{ij} = na, A_j = a_1} f(Y(0, 1) \mid G_{ij} = na, \mu_{na}^{0,a1}, \sigma_{na}^{0,a1}, p_{na}^{0,a1}) \prod_{(i,j): G_{ij} = na, Z_{ij} = 0, A_j = a_1} f(Y(0, 1) \mid G_{ij} = na, \mu_{na}^{0,a1}, \sigma_{na}^{0,a1}, p_{na}^{0,a1}) \prod_{(i,j): G_{ij} = na, Z_{ij} = 1, A_j = a_1} f(Y(1, 0) \mid G_{ij} = na, \mu_{na}^{0,a1}, \sigma_{na}^{0,a1}, p_{na}^{0,a1}) \prod_{(i,j): G_{ij} = cc} p(G_{ij} \mid \pi_{cc}) \prod_{(i,j): G_{ij} = aa} p(G_{ij} \mid \pi_{aa}) \prod_{(i,j): G_{ij} = nn} p(G_{ij} \mid \pi_{nn}) \prod_{(i,j): G_{ij} = ca} p(G_{ij} \mid \pi_{ca}) \prod_{(i,j): G_{ij} = nc} p(G_{ij} \mid \pi_{nc}) \prod_{(i,j): G_{ij} = na} p(G_{ij} \mid \pi_{na}) \tag{7} \]
where $f(Y(.,.))$ and $p(G(.,.))$ are a pdf of the mixture potential outcomes and the compliance behavior respectively. 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 $\pi \sim Dirichlet(\alpha)$ where $\pi = (\pi_{cc}, \pi_{aa}, \pi_{nn}, \pi_{ca}, \pi_{nc}, \pi_{na})$ and $\alpha = (1, 1, 1, 1, 1)$. Then, using the corresponding factor of $L_{comp}$, we have the posterior distribution such that,

$$\pi^{(t+1)} \sim Dirichlet(N^{(t)}_{cc} + 1, N^{(t)}_{aa} + 1, N^{(t)}_{nn} + 1, N^{(t)}_{ca} + 1, N^{(t)}_{nc} + 1, N^{(t)}_{na} + 1)$$

where $N^{(t)}_{g}$ is the number of compliance type $g \in \{cc, aa, nn, ca, nc, na\}$ at the $t$-th MCMC iteration.

**Update of $p$**

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

\[
\begin{align*}
\tilde{p}_{cc}^{(t+1)} &\sim \text{Beta} \left( \sum_{(i,j):G_{ij}^{(t+1)}=cc, Z_{ij}=z, A_j=a} 1(W_{ij}^{\text{obs}} = 1) + 1, \sum_{(i,j):G_{ij}^{(t+1)}=cc, Z_{ij}=z, A_j=a} 1(W_{ij}^{\text{obs}} = 0) + 1 \right) \\
\tilde{p}_{aa}^{(t+1)} &\sim \text{Beta} \left( \sum_{(i,j):G_{ij}^{(t+1)}=aa, A_j=a_0} 1(W_{ij}^{\text{obs}} = 1) + 1, \sum_{(i,j):G_{ij}^{(t+1)}=aa, A_j=a_0} 1(W_{ij}^{\text{obs}} = 0) + 1 \right) \\
\tilde{p}_{nn}^{(t+1)} &\sim \text{Beta} \left( \sum_{(i,j):G_{ij}^{(t+1)}=nn, A_j=a_0} 1(W_{ij}^{\text{obs}} = 1) + 1, \sum_{(i,j):G_{ij}^{(t+1)}=nn, A_j=a_0} 1(W_{ij}^{\text{obs}} = 0) + 1 \right) \\
\tilde{p}_{ca}^{(t+1)} &\sim \text{Beta} \left( \sum_{(i,j):G_{ij}^{(t+1)}=ca, Z_{ij}=z, A_j=a_0} 1(W_{ij}^{\text{obs}} = 1) + 1, \sum_{(i,j):G_{ij}^{(t+1)}=ca, Z_{ij}=z, A_j=a_0} 1(W_{ij}^{\text{obs}} = 0) + 1 \right) \\
\tilde{p}_{ab}^{(t+1)} &\sim \text{Beta} \left( \sum_{(i,j):G_{ij}^{(t+1)}=ca, A_j=a_1} 1(W_{ij}^{\text{obs}} = 1) + 1, \sum_{(i,j):G_{ij}^{(t+1)}=ca, A_j=a_1} 1(W_{ij}^{\text{obs}} = 0) + 1 \right) \\
\tilde{p}_{nc}^{(t+1)} &\sim \text{Beta} \left( \sum_{(i,j):G_{ij}^{(t+1)}=nc, A_j=a_0} 1(W_{ij}^{\text{obs}} = 1) + 1, \sum_{(i,j):G_{ij}^{(t+1)}=nc, A_j=a_0} 1(W_{ij}^{\text{obs}} = 0) + 1 \right) \\
\tilde{p}_{na}^{(t+1)} &\sim \text{Beta} \left( \sum_{(i,j):G_{ij}^{(t+1)}=na, A_j=a_0} 1(W_{ij}^{\text{obs}} = 1) + 1, \sum_{(i,j):G_{ij}^{(t+1)}=na, A_j=a_0} 1(W_{ij}^{\text{obs}} = 0) + 1 \right) \\
\tilde{p}_{na}^{(t+1)} &\sim \text{Beta} \left( \sum_{(i,j):G_{ij}^{(t+1)}=na, A_j=a_1} 1(W_{ij}^{\text{obs}} = 1) + 1, \sum_{(i,j):G_{ij}^{(t+1)}=na, A_j=a_1} 1(W_{ij}^{\text{obs}} = 0) + 1 \right)
\end{align*}
\]

Note that, by Assumption 8, the number of target units varies with compliance behavior. This can be seen in the complete-data likelihood (7) as well. That is, if the parameter
is not defined due to Assumption 6, the corresponding observations are absorbed into the update of the counterpart parameter. For example, $p_{aa}^{1,0}$ is not defined due to the exclusion restriction assumption $Y_{ij}(0, a_0) = Y_{ij}(1, a_0)$ for $G_{ij} = aa$. In such a case, all the observations with $A_j = 0$ are used for updating $p_{aa}^{0,0}$ regardless of the observed value of $Z_{ij}$.

**Update of $\mu$ and $\sigma$**

For the sake of simplicity, we will just show the update of $\mu_{cc}^{0,a_0}$ and $\sigma_{cc}^{0,a_0}$. For the notational convenience, $\sigma_{cc}^{0,a_0}$ denotes the variance parameter. We need to pay attention to the fact that the number of target units varies with compliance behavior, which we saw in the update of $p$.

We use conjugate priors $\mu_g^{z,a} \sim N(0, 100)$ and $\sigma_g^{z,a^2} \sim IG(0.01, 0.01)$ for $\forall g, z, a$. Then we have

$$
\sigma_{cc}^{0,a_0(t+1)} \sim IG \left( 0.01 + S_0, 0.01 + S_1 \right)
$$

$$
\mu_{cc}^{0,a_0(t+1)} \sim N \left( \frac{S_2}{S_0 + \sigma_{cc}^{0,a_0(t+1)}}, \frac{\sigma_{cc}^{0,a_0(t+1)}}{S_0 + \sigma_{cc}^{0,a_0(t+1)}} \right)
$$

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

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

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

### A.5 Simulation Results

Table A.5 shows the simulation results for $DEY(0)$, $DEY(1)$, $DED(0)$ and $DED(1)$. 

47
A.6 Additional Scenarios and Robustness to Model Misspecification

We now present an additional simulation study using the Gamma distribution for the outcome model instead of the Log-Normal distribution. We consider the following outcome model.

\[
W_{ij}(z, a) \mid A_j = a, Z_{ij} = z, G_{ij} = g \sim Bernoulli(p_{g}^{\alpha, a})
\]

\[
X_{ij}(z, a) \mid A_j = a, Z_{ij} = z, G_{ij} = g \sim Gamma(\alpha_{g}^{\alpha, a}, \theta_{g}^{\alpha, a})
\]

\[
Y_{ij}(z, a) = \{1 - W_{ij}(z, a)\} \cdot X_{ij}(z, a)
\]

where \(\alpha\) and \(\theta\) are the shape and scale parameters respectively. The simulation parameters are provided in Table 10. We generate \(N_{sim} = 200\) datasets with \(N = 5000, 10000\) experimental units in each dataset.

This additional scenario serves two purposes. The first purpose is to check if our approach consistently provides comparable results with the frequentist approach under different data generating processes. The "Well-specified" columns in Table A.6 represent a case where we know the true data generating process (8), thus we can fit the same model. The well-specified Bayesian model has the same level of accuracy as the frequentist model in coverage and MSE, however, it has a little bit larger bias due to the same reason as discussed in Section 4.1, that is, (1) the Bayes estimators are always biased and (2) the median of the Gamma distribution is not an unbiased estimator for the mean. We believe this is not a big issue because we would be less interested in the unbiasedness of the Bayesian inference. The reason that the Bayesian approach does not necessarily outperform the frequentist’s MSE as it did in Section 4.1 is due to the data generating process (8). The Gamma distribution does not produce as many outliers as the Log-Normal distribution does, thus the frequentist approach is not much affected by them and provides the same level of performance as the Bayesian approach.
The second purpose is to empirically check the robustness to model misspecification. The "Misspecified" columns present a case where we do not know the true data generating process, thus all we can do is to fit the best possible model that we can think of. Specifically, we fit the model (5) to the true data generating process (8). Even though the misspecified model has a little bit larger bias than the well-specified model in most cases, they have the same level of accuracy in terms of MSE, which implies the robustness of our approach to model misspecification. We can see a common trend in the Bayesian models that the bias and MSE decrease as the number of units increases. There have been recent methodological developments in general Bayesian updating to deal with model misspecification (Bissiri et al. (2016)). A more detailed study on model misspecification is left to future research.
Table 7: Parameters for simulation studies.

|                  | $Z_{ij} = 0$, $A_j = a_0$ | $Z_{ij} = 1$, $A_j = a_0$ | $Z_{ij} = 0$, $A_j = a_1$ | $Z_{ij} = 1$, $A_j = a_1$ |
|------------------|---------------------------|---------------------------|---------------------------|---------------------------|
| $\mu_{cc}$       | 5                         | 7.5                       | 5                         | 7.5                       |
| $\mu_{aa}$       | 10                        | -                         | 10                        | -                         |
| $\mu_{nn}$       | 3                         | -                         | 3                         | -                         |
| $\mu_{ca}$       | 5                         | 8                         | 10                        | -                         |
| $\mu_{nc}$       | 2                         | -                         | 4                         | 8                         |
| $\mu_{na}$       | 2                         | -                         | 10                        | -                         |
| $\sigma^2_{cc}$  | 1.5                       | 2.5                       | 1.5                       | 2.5                       |
| $\sigma^2_{aa}$  | 2                         | -                         | 2.5                       | -                         |
| $\sigma^2_{nn}$  | 2                         | -                         | 2.5                       | -                         |
| $\sigma^2_{ca}$  | 1.5                       | 1.5                       | 2.5                       | -                         |
| $\sigma^2_{nc}$  | 2                         | -                         | 2.5                       | 2.5                       |
| $\sigma^2_{na}$  | 1.5                       | -                         | 1.5                       | -                         |
| $p_{cc}$         | 0.1                       | 0.2                       | 0.1                       | 0.2                       |
| $p_{aa}$         | 0.05                      | -                         | 0.05                      | -                         |
| $p_{nn}$         | 0.03                      | -                         | 0.03                      | -                         |
| $p_{ca}$         | 0.1                       | 0.2                       | 0.1                       | -                         |
| $p_{nc}$         | 0.02                      | -                         | 0.08                      | 0.18                      |
| $p_{na}$         | 0.04                      | -                         | 0.06                      | -                         |
| $\pi_{cc}$       |                           | 0.4                       |                           |                           |
| $\pi_{aa}$       |                           | 0.2                       |                           |                           |
| $\pi_{nn}$       |                           | 0.2                       |                           |                           |
| $\pi_{ca}$       |                           | 0.1                       |                           |                           |
| $\pi_{nc}$       |                           | 0.05                      |                           |                           |
| $\pi_{na}$       |                           | 0.05                      |                           |                           |
Table 8: Simulation Results

**DEY(1)**

| N    | Coverage | Bias       | MSE        | Coverage | Bias       | MSE        |
|------|----------|------------|------------|----------|------------|------------|
|      | Freq     | Freq       | Post. Median | Freq     | Freq       | Post. Median |
| 5000 | 92%      | -3.41E+02  | 6.24E+02   | 98%      | 4.62E+07   | 1.15E+06   |
| 10000| 93%      | 6.08E+01   | 4.44E+02   | 96%      | 2.64E+07   | 6.49E+05   |
| 50000| 95%      | 1.47E+02   | 8.61E+01   | 97%      | 4.42E+06   | 7.48E+04   |

**DEY(0)**

| N    | Coverage | Bias       | MSE        | Coverage | Bias       | MSE        |
|------|----------|------------|------------|----------|------------|------------|
|      | Freq     | Freq       | Post. Median | Freq     | Freq       | Post. Median |
| 5000 | 96%      | 1.42E+02   | 2.00E+02   | 98%      | 9.13E+06   | 1.27E+06   |
| 10000| 96%      | -1.31E+02  | 9.12E+01   | 98%      | 3.50E+06   | 3.49E+05   |
| 50000| 94%      | -3.05E+01  | 9.97E+01   | 97%      | 9.06E+05   | 6.38E+05   |

**DED(1)**

| N    | Coverage | Bias       | MSE        | Coverage | Bias       | MSE        |
|------|----------|------------|------------|----------|------------|------------|
|      | Freq     | Freq       | Post. Median | Freq     | Freq       | Post. Median |
| 5000 | 96%      | -1.58E-03  | 1.19E-02   | 91%      | 4.80E-04   | 5.48E-03   |
| 10000| 93%      | -7.21E-03  | 8.21E-03   | 92%      | 2.80E-03   | 3.26E-03   |
| 50000| 96%      | -1.34E-04  | 4.52E-04   | 94%      | 4.74E-05   | 4.30E-04   |

**DED(0)**

| N    | Coverage | Bias       | MSE        | Coverage | Bias       | MSE        |
|------|----------|------------|------------|----------|------------|------------|
|      | Freq     | Freq       | Post. Median | Freq     | Freq       | Post. Median |
| 5000 | 94%      | -4.17E+04  | -6.73E-03  | 92%      | 3.20E-04   | 3.27E-03   |
| 10000| 94%      | -3.31E-03  | -4.39E-03  | 92%      | 1.74E-04   | 1.93E-04   |
| 50000| 94%      | -2.79E-05  | -7.50E-05  | 91%      | 3.27E-05   | 2.60E-05   |
Table 9: Simulation Results

CADE(0, a_0)

| N   | Coverage | Bias    | MSE    |
|-----|----------|---------|--------|
|     | Freq     | Well-specified | Misspecified | Freq | Well-specified | Misspecified | Freq | Well-specified | Misspecified |
| 5000| 95%      | 93%     | 98%    | 2.75 | -4.86 | -11.78 | 2016 | 2115 | 2326          |
| 10000| 96%     | 92%     | 98%    | 0.16 | -3.44 | -13.79 | 1132 | 1141 | 1369          |

CADE(1, a_1)

| N   | Coverage | Bias    | MSE    |
|-----|----------|---------|--------|
|     | Freq     | Well-specified | Misspecified | Freq | Well-specified | Misspecified | Freq | Well-specified | Misspecified |
| 5000| 96%      | 94%     | 98%    | -1.68| 7.30  | 6.56  | 3578 | 3466 | 3529          |
| 10000| 97%     | 95%     | 98%    | 4.43 | 9.31  | 10.80 | 1705 | 1589 | 1652          |

DEY(0)

| N   | Coverage | Bias    | MSE    |
|-----|----------|---------|--------|
|     | Freq     | Well-specified | Misspecified | Freq | Well-specified | Misspecified | Freq | Well-specified | Misspecified |
| 5000| 97%      | 93%     | 98%    | 0.76 | -1.60 | -9.92 | 267  | 406  | 464           |
| 10000| 95%     | 91%     | 96%    | 0.49 | -2.46 | -8.73 | 390  | 231  | 318           |

DEY(1)

| N   | Coverage | Bias    | MSE    |
|-----|----------|---------|--------|
|     | Freq     | Well-specified | Misspecified | Freq | Well-specified | Misspecified | Freq | Well-specified | Misspecified |
| 5000| 94%      | 94%     | 98%    | -1.37| 1.85  | -2.46 | 657  | 634  | 842           |
| 10000| 97%     | 96%     | 97%    | 0.76 | 4.46  | 6.09  | 267  | 261  | 419           |

52
Table 10: Parameters for additional simulation studies.

|                  | $Z_{ij} = 0$, $A_j = a_0$ | $Z_{ij} = 1$, $A_j = a_0$ | $Z_{ij} = 0$, $A_j = a_1$ | $Z_{ij} = 1$, $A_j = a_1$ |
|------------------|--------------------------|--------------------------|--------------------------|--------------------------|
| $\alpha_{cc}$    | 10                       | 12                       | 10                       | 12                       |
| $\alpha_{aa}$    | 13                       | -                        | 13                       | -                        |
| $\alpha_{nn}$    | 8                        | -                        | 8                        | -                        |
| $\alpha_{ca}$    | 10                       | 11                       | 12                       | -                        |
| $\alpha_{nc}$    | 9                        | -                        | 10                       | 13                       |
| $\alpha_{na}$    | 8.5                      | -                        | 13                       | -                        |
| $\theta_{cc}$    | 100                      | 125                      | 100                      | 125                      |
| $\theta_{aa}$    | 100                      | -                        | 100                      | -                        |
| $\theta_{nn}$    | 90                       | -                        | 90                       | -                        |
| $\theta_{ca}$    | 100                      | 100                      | 83                       | -                        |
| $\theta_{nc}$    | 90                       | -                        | 100                      | 125                      |
| $\theta_{na}$    | 100                      | -                        | 125                      | -                        |
| $p_{cc}$         | 0.1                      | 0.2                      | 0.1                      | 0.2                      |
| $p_{aa}$         | 0.05                     | -                        | 0.05                     | -                        |
| $p_{nn}$         | 0.05                     | -                        | 0.05                     | -                        |
| $p_{ca}$         | 0.1                      | 0.2                      | 0.1                      | -                        |
| $p_{nc}$         | 0.03                     | -                        | 0.1                      | 0.15                     |
| $p_{na}$         | 0.04                     | -                        | 0.06                     | -                        |
| $\pi_{cc}$       |                           |                           | 0.3                      |                           |
| $\pi_{aa}$       |                           |                           | 0.2                      |                           |
| $\pi_{nn}$       |                           |                           | 0.17                     |                           |
| $\pi_{ca}$       |                           |                           | 0.13                     |                           |
| $\pi_{nc}$       |                           |                           | 0.1                      |                           |
| $\pi_{na}$       |                           |                           | 0.1                      |                           |