

Identification of Local Average Treatment Effects with Imperfect Compliance and Interference

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Abstract

This study addresses the identification and estimation of causal effects in scenarios where units interact and imperfect compliance is present. In cases where treatment take-up is endogenous due to imperfect compliance, the standard solution is to use the treatment assignments as instruments for the treatment take-up to identify the local average treatment effects (LATE). The key assumption for identifying LATE is monotonicity in potential treatments. This paper extends this approach to situations involving the interaction of two units by introducing a weak concept of monotonicity that is a generalization of the restrictions on the potential treatment, such as monotonicity and one-sided noncompliance. Under the weak monotonicity, this study proposes a general identification result in this setting, provided that additional exclusion restrictions for the compliance patterns exist. This identification can be applied to situations where traditional assumptions may not be fully satisfied. A two-stage estimator for the identified parameters is introduced, with its properties evaluated through simulation studies. The proposed method is illustrated by real-world data from a randomized experiment that provided access to a savings account.

1 Introduction

This study discusses a method for the identification and estimation of causal effects in cases where the units interact with each other and there is imperfect compliance. The causal effects of a program have been of great interest in various economics studies. Traditionally, it has been assumed that each unit behaves independently in response to the program. Specifically, a unit’s outcome is potentially determined solely by its own treatment, an assumption often referred to as the Stable Unit Treatment Value Assumption (SUTVA). However, this assumption is restrictive in many economic contexts as economic agents often naturally interact with one another. Consequently, recent studies have focused on investigating methodologies for identifying and estimating causal effects in situations where the interactions among units violate SUTVA.

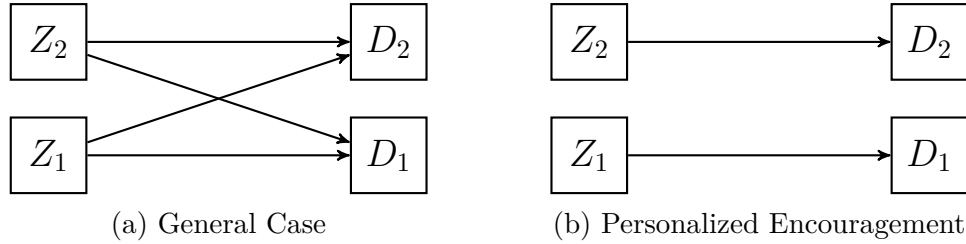
The average treatment effect (ATE) is one of the most popular causal parameters, typically defined as the difference in potential outcomes between two distinct situations: when a unit is treated and when it is not. Since only one treatment status can be observed for any unit at a time, one of these situations is necessarily counterfactual. This poses a fundamental challenge for identification in causal inference. When a program is designed to randomly assign treatment to units, and all units take the treatment when they are assigned to the treatment group, the distribution of counterfactual potential outcomes can be recovered from the observed outcome distribution of treated or untreated units. However, if there is imperfect compliance, where the treatment assignment and the actual take-up of treatment do not coincide, then the treatment is not always exogenous, and we need to consider the pattern of noncompliance.

The issue of imperfect compliance introduces various compliance patterns for units, and understanding these patterns is essential to define and identify certain causal effects. For instance, in the classical case where units are independent, each unit is classified into one of four compliance types: the ‘always taker’, ‘never taker’, ‘complier’, and ‘defier’, as discussed by [Imbens and Angrist \(1994\)](#). These types are not directly observed as they are determined by the realization of potential treatment, making one of them counterfactual for the same reason as potential outcomes. Let $D(1)$ and $D(0)$ represent the potential treatment take-ups for treated and untreated units, respectively. Notably, since ‘always takers’ and ‘never takers’ do not alter their treatment take-up in response

to treatment assignments (i.e., $D(1) = D(0)$), only identifiable effects will arise from ‘compliers’ or ‘defiers’. One key strategy for identification is to exclude ‘defiers’, thereby focusing on the causal effects of ‘compliers’. The resulting causal effect is the Local Average Treatment Effect (LATE) on compliers. The exclusion of ‘defiers’ is equivalently stated by the monotonicity assumption: $D(1) \geq D(0)$ with probability 1.

In scenarios where units interact, these compliance patterns become more complex. Consider two units that may choose to take the treatment based on their own treatment status and that of the other unit. This situation is depicted in panel (a) of Figure 1. Let Z_i be a binary random variable denoting individual i ’s treatment assignment status. Then, we have four possible treatment assignments: $(Z_1, Z_2) \in (1, 1), (1, 0), (0, 1), (0, 0)$. Define $D_i(z_i, z_j)$ as the potential treatment take-up status for unit i , given treatment assignments $(Z_i, Z_j) = (z_i, z_j)$, where $j = 3 - i$. In this setting, there are 16 (2^4) possible compliance types for each unit, and hence 256 (2^8) possible joint compliance configurations of two units, as pointed out in [Kormos, Lieli, and Huber \(2023\)](#). This is significantly more complex compared to the four compliance types in the classical case. Thus, to define and identify some meaningful causal effects, some or many compliance types need to be excluded, as in the strategy used in classical cases.

Figure 1: Interactions in treatment take-up decision



To address this issue, studies in this literature have employed several restrictions on potential treatment take-up. First, the simplest restriction is to assume no interaction in the treatment take-up decision. Using the notations for a two-unit case, this assumption can be formalized as $D_i(z_i, 1) = D_i(z_i, 0)$ with probability 1, indicating that a unit’s potential treatment depends solely on their own treatment assignment. This restriction is called personalized encouragement as in [Kang and Imbens \(2016\)](#), and the situation is depicted in panel (b) of Figure 1. This assumption allows us to categorize four

compliance types as the classical case, without considering joint compliance patterns.

Second, a frequently employed restriction is the “one-sided noncompliance” assumption. This assumption means that only treated units have an opportunity to take the treatment or not. Again, by using the same notation, it can be expressed as $D_i(0, 0) = D_i(0, 1) = 0$ with probability 1¹ for both units, indicating that the unit is not able to take the treatment if their own treatment assignment. This assumption sometimes holds by design of the experiments such as a program that randomly assigns non-transferable vouchers for treatment. However, this assumption may not apply in scenarios where, for example, either spouse in a married couple can take the treatment if at least one has the voucher.

Third, as traditionally understood, the concept of monotonicity plays a crucial role in this literature. When personalized encouragement is assumed, the classical monotonicity can be applied: $D_i(1, \cdot) \geq D_i(0, \cdot)$ with probability 1, which effectively excludes defiers. The fundamentality of monotonicity lies in the almost sure ordering of potential treatments. Therefore, in the case of two units, the natural extension of monotonicity is to impose an almost sure total ordering on the set $\{D_i(1, 1), D_i(1, 0), D_i(0, 1), D_i(0, 0) : i = 1, 2\}$. For example, we can use the following monotonicity:

$$D_i(1, 1) \geq D_i(1, 0) \geq D_i(0, 1) \geq D_i(0, 0), \quad (1)$$

with probability 1 for each unit. This monotonicity is assumed in [Vazquez-Bare \(2022\)](#), and I refer to it as total monotonicity in this paper. This restriction effectively excludes certain compliance types, as there are only 5 compliance types under total monotonicity, which further reduces to 3 when combined with one-sided noncompliance. However, while classical monotonicity is interpreted as excluding defiers, this type of ordering might be difficult to interpret. Overall, this is a strong assumption because such an ordering needs to hold almost surely, restricting the distributions of all potential treatments. For example, when the probabilities of both events $D(1, 1) \geq D(1, 0)$ and $D(1, 1) < D(1, 0)$ are 0.5, total monotonicity fails because we don’t have almost sure ordering on $D(1, 1)$ and $D(1, 0)$.

While these assumptions facilitate the identification of causal effects, they are not

¹Note that the first argument is the own treatment assignments.

only challenging to verify, as they impose restrictions on potential treatments that are not always observable, but they would also be difficult to justify in certain situations. This study explores the identification of causal effects beyond these assumptions, particularly in situations where neither one-sided noncompliance nor total monotonicity are fully satisfied.

The main idea is to generalize the aforementioned assumptions into a weaker concept of monotonicity. We observe that the essence of those assumptions is all about imposing some almost sure ordering on potential treatments. Based on this observation, I introduce a general concept of monotonicity that assumes only almost sure partial ordering on the set of potential treatments, instead of an almost sure total ordering. For example, this approach might only assume that $D_i(0, 1) \geq D_i(0, 0)$ with probability 1, leaving distributions of $D_i(1, 1)$ and $D_i(1, 0)$ unrestricted. More formally, *for some* two different treatment assignments $\mathbf{z} = (z_1, z_2)$ and $\mathbf{z}' = (z'_1, z'_2)$ in $\{0, 1\}$, it assumes $D_i(z_i, z_j) \geq D_i(z'_i, z'_j)$ with probability 1, where $j = 3 - i$ is the other unit's index. As I will show, personalized encouragement, one-sided noncompliance, and total monotonicity are all explained by this concept of monotonicity. Also, there are situations where this version of monotonicity is satisfied, but all the aforementioned assumptions are violated. Therefore, the framework proposed in this study can be viewed as a generalization of existing approaches. This monotonicity concept will be formally introduced in Section 2.2.

If such two different treatment assignments \mathbf{z}, \mathbf{z}' exist, then I call \mathbf{z} and \mathbf{z}' form a *monotone pair*. Then, the compliance types, and hence the causal parameters of interest are defined for each monotone pair. Particularly, I define the unit i as a complier if the monotone relationship holds with strict inequality, i.e., $D_i(z_i, z_j) = 1$ and $D_i(z'_i, z'_j) = 0$ with probability 1. If both potential treatments are 0 or 1, then I define the unit as a never-taker or always-taker, respectively, with respect to the given monotone pair. Therefore, we only consider three compliance types for each unit given a monotone pair.² The parameters of interest consist of direct and indirect local average treatment effects on compliers corresponding to the given monotone pair.

As with the classical local average treatment effect (LATE) estimand, the identi-

²We also need to consider joint compliance patterns. However, it is enough to consider one specific joint compliance pattern in this setting, as shown in Section 3

fication argument begins with observing the intention-to-treatment effects (ITT) on outcomes. The ITT of the outcome is represented as a linear combination of parameters of interest, with the weights being the distribution of compliance types as shown in equation (8). The random variation of treatment assignment is used as instruments for the endogenous treatment take-up, as in the classical case. However, in the general case, we need more instruments to generate exogenous variation in the distribution of compliance types. This entails additional exclusion restrictions that exclusively determine compliance type but does not directly affect the outcome. The main result in this study is a general identification result under the existence of such additional exclusion restrictions, given a monotone pair. I then discuss some interesting special cases, including the classical LATE estimand proposed by [Imbens and Angrist \(1994\)](#), and the direct and indirect LATEs under one-sided noncompliance and total monotonicity proposed by [Vazquez-Bare \(2022\)](#). Moreover, the general result can be applied in situations where both one-sided noncompliance and total monotonicity are violated.

I propose a two-stage estimation procedure to consistently and efficiently estimate the identified parameters. Monte Carlo simulation studies demonstrate that the estimation procedure works well in various designs. Additionally, I apply the estimation method to the data from [Dupas, Keats, and Robinson \(2019\)](#), which conducted a randomized experiment in Kenya. The experiment is well-suited to illustrate the proposed method as both one-sided noncompliance and total monotonicity are not fully satisfied.

In summary, the contributions of this study are twofold. Firstly, I propose a general framework to analyze causal treatment effects under interference and imperfect compliance by generalizing various underlying restrictions such as personalized encouragement, total monotonicity, and one-sided noncompliance to a weaker concept of monotonicity. Secondly, I derive a general identification result when more additional exclusion restrictions are available.

The structure of this paper is as follows: Section [1.1](#) introduces the related studies. Section [2](#) set up the model and notation, and define the parameters of interest. Section [3](#) addresses the identification of these parameters. Section [4](#) proposes a two-stage estimation procedure, and derive its asymptotic properties. Section [5](#) evaluates the proposed estimator through Monte Carlo simulations. Section [6](#) provides an empirical

illustration using a dataset from experiments conducted by [Dupas, Keats, and Robinson \(2019\)](#). Finally, Section 7 concludes the paper.

1.1 Related Literature

This study contributes to the literature on causal inference with interference between units, as an attempt to generalize Local Average Treatment Effects (LATE) ([Imbens and Angrist \(1994\)](#), [Angrist, Imbens, and Rubin \(1996\)](#), and [Imbens and Rubin \(1997\)](#)). As discussed above, compared to scenarios where units behave independently, the complexity of compliance patterns escalates exponentially when interactions between more than one unit are considered.

Studies in this literature avoid the complexity of compliance patterns by imposing restrictions on potential treatments. Some assume no interaction in treatment take-up decisions, referred to as personalized encouragement ([Kang and Imbens \(2016\)](#), [Hoshino and Yanagi \(2023\)](#)), treatment-exclusive restriction ([Blackwell \(2017\)](#)), or individualized offer response ([DiTraglia et al. \(2023\)](#)). One-sided noncompliance is also commonly assumed as it is relatively straightforward to examine by the experimental design ([Vazquez-Bare \(2022\)](#), [Kang and Imbens \(2016\)](#), [Blackwell \(2017\)](#), [DiTraglia et al. \(2023\)](#)). Additionally, most studies in this literature rely on some version of monotonicity regarding potential treatments. For examples, [Vazquez-Bare \(2022\)](#) and [Hoshino and Yanagi \(2023\)](#) assume an almost sure total ordering on the set of all potential treatments in two units, and many units, respectively to identify local average treatment effects.

This study considers interactions between two units, and the baseline settings follows [Vazquez-Bare \(2022\)](#). However, the monotonicity and one-sided noncompliance assumptions, which are key identifying assumptions, are notably relaxed in this study by introducing a weaker concept of monotonicity. Consequently, the proposed identification method can be applied to more general scenarios where both assumptions are violated. Particularly, the findings can be viewed as a direct generalization of [Vazquez-Bare \(2022\)](#), as its main identification result can be explained by a special case of the general identification proposed in this study.

2 Model

2.1 The Basic Setup

Consider a population consisting of G independent groups. Within each group, there are two units denoted by $i = 1, 2$. For each unit i in group g , there are two binary random variables Z_{ig} , and D_{ig} . The variable Z_{ig} takes the value 1 if unit i in group g is assigned to the treatment group, while D_{ig} takes the value 1 if that unit actually takes up the treatment. The ideal situation is when the intended treatment (Z_{ig}) is the same as the actual treatment take-up (D_{ig}). In such a scenario, some causal effects could be identified by the random variation of the treatment. In many cases, however, even when treatment is randomized through the design of the experiment, the take-up decision may not be controlled because of imperfect compliance, i.e., $Z_{ig} \neq D_{ig}$ with positive probability. This study focus on those situations when the treatment is randomly assigned, while the take-up of the treatment is endogenous because of imperfect compliance.

To simplify the notation, I suppress the group index subscript if unnecessary. For each $i \in \{1, 2\}$, let $j = 3 - i$ represent the other unit's index. Let $\mathbf{Z}_i = (Z_i, Z_j)$, where the first component corresponds to unit i 's own treatment assignment, and the second component corresponds to the other unit's treatment assignment. Similarly, let $\mathbf{D}_i = (D_i, D_j)$. For any treatment assignment status $\mathbf{z}_i = (z_i, z_j) \in \{0, 1\}^2$, the potential treatment take-up for unit i is denoted as $D_i(\mathbf{z}_i)$. Furthermore, for each treatment assignment status $\mathbf{z}_i = (z_i, z_j) \in \{0, 1\}^2$ and treatment take-up status $\mathbf{d}_i = (d_i, d_j) \in \{0, 1\}^2$, the corresponding potential outcome is given by $Y_i(\mathbf{d}_i, \mathbf{z}_i)$. Let $\mathbf{X}_i = (X_i, X_j)$ represent a vector of covariates including both unit-specific characteristics and group characteristics. To begin with, I assume the followings by following the literature.

Assumption 1.

1. (*Exclusion Restriction I*) For each individual $i \in \{1, 2\}$ and $\mathbf{d} \in \{0, 1\}^2$, we have

$$Y_i(\mathbf{d}, \mathbf{z}) = Y_i(\mathbf{d}, \mathbf{z}'), \quad \forall \mathbf{z}, \mathbf{z}' \in \{0, 1\}^2.$$

2. (Conditional Independence) Treatment assignments (Z_1, Z_2) are independent of potential outcomes and potential treatment take-ups, conditional on \mathbf{X} , i.e.,

$$\left\{ (Y_i(\mathbf{d}_i, \mathbf{z}_i), D_i(\mathbf{z}_i)) : (\mathbf{d}_i, \mathbf{z}_i) \in \{0, 1\}^4, i \in \{1, 2\} \right\} \perp (Z_1, Z_2) | \mathbf{X}.$$

3. (Overlap) $\Pr(\mathbf{Z}_i = \mathbf{z} | \mathbf{X}) > 0$ with probability 1, for all $\mathbf{z} \in \{0, 1\}^2$ and $i \in \{1, 2\}$.

As often assumed in the literature, the first two parts of Assumption 1 ensure that the treatment \mathbf{Z}_i is (conditionally) randomly assigned, and therefore plays a role of a valid instrument for the endogenous treatment take-up \mathbf{D}_i . In particular, according to the first part of Assumption 1, the potential outcome can be expressed solely as a function of the treatment take-up by abusing the notation:

$$Y_i(\mathbf{d}) = Y_i(\mathbf{d}, \mathbf{z}_i), \quad \forall \mathbf{d} \in \{0, 1\}^2.$$

And the third part of Assumption 1 is the standard overlap assumption that guarantees the existence of corresponding conditional expectations.

Given the setting, I explicitly examine two types of interactions between units. The first interaction occurs during the treatment take-up decision, where each unit's potential treatment depends on both their own and others' treatment assignment ($D_i = D_i(Z_i, Z_j)$). The second interaction pertains to the determination of potential outcomes, as each individual's potential outcome is determined by the treatment take-up status of both individuals ($Y_i = Y_i(D_i, D_j)$). This situation is depicted in Figure 2. In the following subsection, we will discuss how each of these interactions complicates the problem.

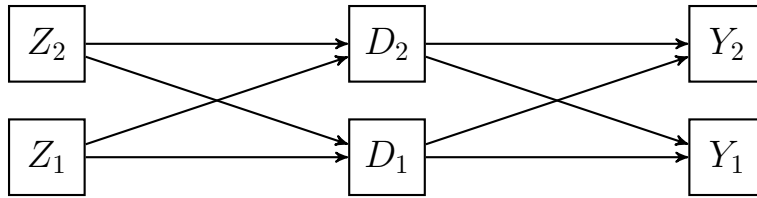


Figure 2: Two Layers of Interactions

2.2 Monotonicity and Compliance Types

Even if the treatment is entirely randomly assigned, the imperfect compliance leads to endogeneity of the treatment, thus making it more challenging to identify the causal effects. Therefore, understanding these (non-)compliance patterns is essential in the analysis.

The compliance pattern or compliance type of each unit is determined by the distribution of their potential treatment. To illustrate, consider the case of a single individual ($N = 1$) first. In this scenario, there are two potential treatment take-up statuses: potential treatment when the unit is treated ($D(1)$), and untreated ($D(0)$). Since each treatment status is binary, there are 4 (2^2) possible compliance types for each unit. The classification and interpretation of each type is summarized in Table 1.

Table 1: Compliance Types without Spillovers on the Take-up Decision

Type	$D(1), D(1, 1) = D(1, 0)$	$D(0), D(0, 1) = D(0, 0)$
Always taker (AT)	1	1
Complier (C)	1	0
Defier (D)	0	1
Never taker (NT)	0	0

note. The assumption $D_i(z_i, z_j) = D_i(z_i, z'_j)$ for all $z_j, z'_j \in \{0, 1\}$ is referred to as personalized encouragement ([Kang and Imbens \(2016\)](#)), treatment exclusion restriction ([Blackwell \(2017\)](#)), or individualized offer response ([DiTraglia et al. \(2023\)](#)).

Classical approach to identify causal effect is to exclude some compliance types by imposing restrictions on the distribution of potential treatments. [Imbens and Angrist \(1994\)](#) introduced the monotonicity assumption, which is stated as:

$$\Pr(D(1) \geq D(0)) = 1.$$

This excludes the possibility of units being defiers (units with $D(1) < D(0) = 1$), and it allows the identification of the local average treatment effect among compliers (units with $1 = D(1) > D(0)$). Another restriction in this setting is one-sided noncompliance (OSN) that imposes that only units in the treatment group can determine whether to

take it up or not, but the units in control group are not able to take the treatment, i.e., $D(0) = 0$ with probability 1. Thus, OSN assumption excludes not only defiers but also always takers (units with $D(1) = D(0) = 1$). Although verifying these sorts of restrictions will not be straightforward since we don't observe all potential treatment statuses, OSN situation will be evident in some situation by the design of the experiment, e.g., providing non-transferable voucher for the treatment take-up.

Next, consider a more general case when there are two units in each group ($N = 2$), which is the primary situation of interest in this study. If treatment take-up responses are determined by the own and the other unit's treatment assignments, there are 4 potential treatment statuses: $D(1, 1)$, $D(1, 0)$, $D(0, 1)$, and $D(0, 0)$. And since each potential treatment has binary values, we have 16 (2^4) possible compliance types for each unit. Furthermore, considering two units, there are a total of 256 (16^2) possible joint compliance types for both units. This number grows exponentially with the number of units, which makes problems more challenging when we consider many units. However, since not all scenarios are of interest, some compliance types would be excluded by imposing appropriate restrictions on potential treatment distribution as the classical cases.

The simplest restriction is to assume that there are no interactions in the treatment take-up decision, as illustrated in Figure 3. This has been often employed in the literature (e.g., personalized encouragement (Kang and Imbens (2016)), treatment exclusion restriction (Blackwell (2017)), or individualized offer response (DiTraglia et al. (2023))) and results in each individual's marginal compliance type being the same as Table 1, with four possible types. However, this assumption could be violated in many situations such as when both individuals can take the treatment whenever at least one of them is eligible.

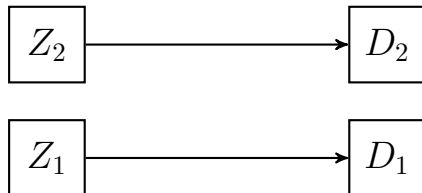


Figure 3: Personalized Encouragement

Another approach is to extend monotonicity to cases with two units. For example, one can define a almost sure total ordering on the set $\mathcal{D} := \{(D_i(z_i, z_j), D_j(z_j, z_i)) : (z_i, z_j) \in \{0, 1\}^2\}$. For example, [Vazquez-Bare \(2022\)](#) assumes the following ordering:

$$D_i(1, 1) \geq D_i(1, 0) \geq D_i(0, 1) \geq D_i(0, 0), \text{ with probability 1,} \quad (2)$$

for all $i \in \{1, 2\}$. I refer this type of monotonicity as ‘total monotonicity’ to distinguish the monotonicity concept employed in this study. Under the total monotonicity, 9 out of 16 compliance types are excluded for each unit. The interpretation of those 5 compliance type according the ‘total monotonicity’ (2) is summarized in Table 2. However, compared to the classical monotonicity that is equivalent to excluding defiers, the total monotonicity is much stronger, and sometimes difficult to interpret. For instance, consider the following situation: for $i \in \{1, 2\}$,

$$\Pr(D_i(0, 0) = 0) = 1. \quad (3)$$

This means that if both units are in the control group, then they cannot take the treatment. This assumption also implies that $D_i(0, 1) \geq 0$ and $D_i(1, 0) \geq 0$ with probability 1, indicating units can take the treatment if at least one unit is treated. In this regard, I refer to this assumption as *Weak One-sided Noncompliance* (WOSN). However, in this case, the distribution of $D_i(1, 1)$, $D_i(1, 0)$, $D_i(0, 1)$ remains unrestricted, hence there is no almost sure ordering on these potential treatments. Therefore, WOSN is an example of violating total monotonicity.

As pointed out in the single-unit case, the one-sided non-compliance (OSN) assumption can be applicable in particular experimental designs, where it is extended by $D(0, 1) = D(0, 0) = 0$ with probability 1.³ This assumption also significantly reduces the number of possible compliance types, and when the OSN assumption and total monotonicity are combined, each unit has three possible compliance types as summarized in Table 2.⁴ However, because of the interaction in the treatment take-up decision, OSN can be violated in general, such as in the WOSN case (3).

³Note that the first argument is the own treatment status.

⁴Following the classification in [Vazquez-Bare \(2022\)](#), they are *complier* ($D(1, 0) > D(0, 1)$), *group complier* ($D(1, 1) > D(1, 0)$), and *never taker* ($D(1, 1) = 0$).

Table 2: Compliance Types with Monotonicity and OSN

Type	$D(1, 1)$	$D(1, 0)$	$D(0, 1)$	$D(0, 0)$
Always taker (AT)	1	1	1	1
Social complier (SC)	1	1	1	0
Complier (C)	1	1	0	0
Cross defier (CD)	0	1	0	0
Group complier (GC)	1	0	0	0
Never taker (NT)	0	0	0	0

note. Assuming one-sided noncompliance (OSN), we can classify each individual as complier (or joint complier, $D(1, 0) > D(0, 1)$), cross defier ($D(1, 1) < D(1, 0)$), group complier (or self complier, $D(1, 1) > D(1, 0)$), and never-taker, as employed in [Hoshino and Yanagi \(2023\)](#). Also, assuming total monotonicity condition $D_i(1, 1) \geq D_i(1, 0) \geq D_i(0, 1) \geq D_i(0, 0)$ results in compliance types of always-taker ($D(0, 0) = 1$), social complier ($D(0, 1) > D(0, 0)$), complier ($D(1, 0) > D(0, 1)$), group complier ($D(1, 1) > D(1, 0)$), and never-taker ($D(1, 1) = 0$), as examined in [Vazquez-Bare \(2022\)](#). Under the combination of two assumptions, only complier, group complier, and never-taker are possible.

While such restrictions allow for the identification of particular causal effects, it's important to note that these restrictions are not universally applicable, as the situation (3) does not satisfy any of them. One of the key distinctions of this study from existing literature is that such cases are still of interest. To this end, I focus on monotonicity that holds for specific treatment assignment scenarios, but not for all possible scenarios. The monotonicity assumption applied in this study is as follows.

Assumption 2 (Monotonicity). *There exist $\mathbf{z} = (z_1, z_2)$, and $\mathbf{z}' = (z'_1, z'_2)$ in $\{0, 1\}^2$ with $\mathbf{z} \neq \mathbf{z}'$ such that*

$$\Pr(r_i(D_i(\mathbf{z}_i) - D_i(\mathbf{z}'_i)) \geq 0 | \mathbf{X}) = 1, \quad i \in \{1, 2\},$$

with probability 1, for some $r_1, r_2 \in \{-1, 1\}$, where $\mathbf{z}_i = (z_i, z_{3-i})$ for $i \in \{1, 2\}$. If such pairs exist, then let $\mathbf{m} = (\mathbf{z}, \mathbf{z}', \mathbf{r}) = ((z_1, z_2), (z'_1, z'_2), (r_1, r_2))$ be a monotone pair of treatment assignments, and let \mathcal{M} be the collection of all monotone pairs. Furthermore, once a monotone pair $\mathbf{m} = (\mathbf{z}, \mathbf{z}', \mathbf{r})$ is given, then define a unit i as \mathbf{m} -always taker if $D_i(\mathbf{z}_i) = D_i(\mathbf{z}'_i) = 1$, \mathbf{m} -never taker if $D_i(\mathbf{z}_i) = D_i(\mathbf{z}'_i) = 0$, and \mathbf{m} -complier if

$r_i(D_i(\mathbf{z}_i) - D_i(\mathbf{z}'_i)) > 0$, conditional on \mathbf{X} with probability 1, respectively.

Recall that total monotonicity imposes an almost sure total ordering on the set $\mathcal{D} := \{(D_i(z_i, z_j), D_j(z_j, z_i)) : (z_i, z_j) \in \{0, 1\}^2\}$. Instead, Assumption 2 imposes an almost sure *partial* ordering on the set \mathcal{D} . If no restriction is imposed on the distribution of potential treatment, then the collection \mathcal{M} is empty. However, because such cases are not of interest in this study, I assume \mathcal{M} is non-empty, hence there is at least one monotone pair of treatment assignments.

If we impose $D_i(\mathbf{z}_i) = D_i(\mathbf{z}'_i)$, then we have two monotone pairs $\mathbf{m}_1 = (\mathbf{z}, \mathbf{z}', \mathbf{r})$ and $\mathbf{m}_2 = (\mathbf{z}', \mathbf{z}, \mathbf{r})$, for any \mathbf{r} . The direction \mathbf{r} will mostly be $(1, 1)$ or $(-1, -1)$, but for the assignment $\mathbf{z} = (1, 0)$ and $\mathbf{z}' = (0, 1)$, it could be $(1, -1)$ or $(-1, 1)$. That is, if we impose $D_i(1, 0) \geq D_i(0, 1)$ for $i = 1, 2$, we can write a monotone pair as $\mathbf{z} = (1, 0)$, $\mathbf{z}' = (0, 1)$, and the direction is flipped for unit 2 since $\mathbf{z}_2 = (0, 1)$ and $\mathbf{z}'_2 = (1, 0)$.

This concept of monotonicity is quite general, in the sense that all three aforementioned restrictions (personalized encouragement, one-sided noncompliance, and total monotonicity) can be explained as special cases of Assumption 2. In other words, we can always specify an appropriate set of monotone pairs for each of those restrictions. In this regard, Assumption 2 can be thought of as a general restriction on potential treatment or a general monotonicity assumption.

Table 3 describes the monotone pairs induced by each of aforementioned assumptions in the literature. Under total monotonicity, or personalized encouragement, all possible pairs $(\mathbf{z}, \mathbf{z}') \in \{0, 1\}^4$ can be formed with monotone pairs for some direction $\mathbf{r} = (r_1, r_2)$. However, because one-sided noncompliance does not impose any ordering on $D(1, 1), D(1, 0)$, some pairs cannot be a monotone pair in Panel (c). Thus, the collection \mathcal{M} induced by one-sided noncompliance is smaller than that of total monotonicity, or personalized encouragement. Also, there are only 2 monotone pairs under weak one-sided noncompliance. And I define unit i as complier (C) if $D_i(1, 0) > D_i(0, 0)$, and social complier (SC) if $D_i(0, 1) > D_i(0, 0)$.

The interpretation of \mathbf{m} -compliance type, especially \mathbf{m} -complier, is context-dependent and is determined by the specific values of \mathbf{m} . Also, once a monotone pair $\mathbf{m} = (\mathbf{z}, \mathbf{z}', \mathbf{r})$ is given, then the interpretation of \mathbf{m} -compliance type might differ between two units unless $\mathbf{z}_1 = \mathbf{z}_2$. In addition, as demonstrated in Table 3, multiple monotone pairs may

Table 3: Monotone pairs

(a) Personalized Encouragement + Monotonicity

z	(1, 1)	(1, 1)	(1, 1)	(1, 0)	(1, 0)	(0, 1)
z'	(1, 0)	(0, 1)	(0, 0)	(0, 1)	(0, 0)	(0, 0)
(r_1, r_2)	(1,1), (-1,1)	(1,1), (1,-1)	(1,1)	(1,-1)	(1,1) (1,-1)	(1,1) (1,-1)
K_1		C	C	C	C	
K_2	C		C	C		C

(b) Total Monotonicity

z	(1, 1)	(1, 1)	(1, 1)	(1, 0)	(1, 0)	(0, 1)
z'	(1, 0)	(0, 1)	(0, 0)	(0, 1)	(0, 0)	(0, 0)
(r_1, r_2)	(1,1)	(1,1)	(1,1)	(1,-1)	(1,1)	(1,1)
K_1	GC	GC, C	GC, C, SC	C	C, SC	SC
K_2	GC,C	GC	GC, C, SC	C	SC	C, SC

(c) One-Sided Noncompliance

z	(1, 1)	(1, 1)	(1, 1)	(1, 0)	(1, 0)	(0, 1)
z'	(1, 0)	(0, 1)	(0, 0)	(0, 1)	(0, 0)	(0, 0)
(r_1, r_2)			(1,1)	(1,-1)	(1,1), (1,-1)	(1,1), (1,-1)
K_1			GC, or C	C, or CD	C, or CD	
K_2			GC, or C	C, or CD		C, or CD

(d) Weak One-Sided Noncompliance

z	(1, 1)	(1, 1)	(1, 1)	(1, 0)	(1, 0)	(0, 1)
z'	(1, 0)	(0, 1)	(0, 0)	(0, 1)	(0, 0)	(0, 0)
(r_1, r_2)					(1,1)	(1,1)
K_1					C	SC
K_2					SC	C

note. Panel (a), (b), and (c) show the monotone pairs $\mathbf{m} = (z, z', \mathbf{r})$ under personalized encouragement and monotonicity on $D_i(1) \geq D_i(0)$, Total monotonicity $D(1, 1) \geq D(1, 0) \geq D(0, 1) \geq D(0, 0)$, one-sided noncompliance $D(0, 1) = D(0, 0) = 0$, and weak one-sided noncompliance $D(0, 0) = 0$. K_i denotes the interpretation of \mathbf{m} -complier for each unit, with the definitions in Tables 1 and 2.

exist under certain assumptions. When there are more than two monotone pairs, it becomes possible to further divide the \mathbf{m} -compliance into finer compliance types. These observations are described in Example 1.

Remark 1 (Compliance Types under Total Monotonicity). Assume total monotonicity in this example. Consider a monotone pair $\mathbf{m}_1 = ((1, 1), (1, 0), (1, 1))$ of the first column in Panel (b) of Table 3. For this monotone pair, unit 1 is an \mathbf{m} -complier when $D(1, 1) > D(1, 0)$ with probability 1 (conditional on \mathbf{X}). This type is classified as a group complier (GC) according to the classification in Table 2 since the individual takes the treatment only when both individuals are treated. On the other hand, unit 2 is an \mathbf{m} -complier when $D(1, 1) > D(0, 1)$, which implies group complier (GC), or complier ($D(1, 0) > D(0, 1)$; C). Additionally, let $\mathbf{m}_2 = ((1, 1), (0, 1), (1, 1))$ and $\mathbf{m}_4 = ((1, 0), (0, 1), (1, -1))$ be the monotone pairs in the second and fourth columns in Panel (b) of Table 3. Then, individual 2 of \mathbf{m}_4 -complier is a complier (C), and \mathbf{m}_2 -complier is a group complier (GC). Thus, \mathbf{m}_1 -complier for individual 2 can be further divided into \mathbf{m}_2 -complier and \mathbf{m}_4 -complier.

2.3 Potential Outcomes and Parameters of Interest

The potential outcome is defined based on the potential treatment take-up status of both individuals to explicitly account for interference in determining potential outcomes, thereby violating SUTVA. Suppose the observed outcome is given by $Y_i = Y_i(D_i, D_j)$. Then, it can be written as:

$$\begin{aligned} Y_i &= \sum_{\mathbf{d} \in \{0,1\}^2} \mathbb{1}\{\mathbf{D}_i = \mathbf{d}\} Y_i(\mathbf{d}) \\ &= Y_i(0, 0) + \Delta_i Y_i(0) D_i + \Delta_j Y_i(0) D_j + [\Delta_i Y_i(1) - \Delta_i Y_i(0)] D_i D_j^5 \end{aligned} \quad (4)$$

where $\Delta_i Y_i(d) = Y_i(1, d) - Y_i(0, d)$, and $\Delta_j Y_i(d) = Y_i(d, 1) - Y_i(d, 0)$, for $d \in \{0, 1\}$. The $\Delta_i Y_i(d)$ is interpreted as the direct effect on individual i 's outcome, in the sense that it represents the causal effect on the outcome resulting from changes in own treatment when the partner's treatment take-up is fixed at $d \in 0, 1$. Similarly, $\Delta_j Y_i(d)$ is

⁵Note that the last term can be written in terms of Δ_j since $\Delta_i Y_i(1) - \Delta_i Y_i(0) = \Delta_j Y_i(1) - \Delta_j Y_i(0)$.

interpreted as the indirect effect on individual i 's outcome when own treatment status is fixed at d .

The conventional approach involves using treatment assignment as the instrument for endogenous treatment take-up to identify some local average treatment effect. The same strategy is used in this study, and the parameters of interest are the local direct ($\Delta_i Y_i(d)$) and indirect ($\Delta_j Y_i(d)$) effects. To define the parameters of interest, suppose we have a monotone pair $\mathbf{m} = (\mathbf{z}, \mathbf{z}', \mathbf{r})$, and denote $\mathbf{z}_i = (z_i, z_{3-i})$ and $\mathbf{z}'_i = (z'_i, z'_{3-i})$ for each $i \in 1, 2$ as before. Next, let \mathbf{T} be any subset of the exogenous variables \mathbf{X} , and define

$$\begin{aligned} K_i^{\mathbf{m}} &:= r_i(D_i(\mathbf{z}_i) - D_i(\mathbf{z}'_i)), \\ P_i^{\mathbf{m}}(\mathbf{T}) &:= \Pr(K_i^{\mathbf{m}} = 1 | \mathbf{T}) = E[K_i^{\mathbf{m}} | \mathbf{T}]. \end{aligned} \tag{5}$$

From the monotonicity, $K_i^{\mathbf{m}}$ is a binary random variable that takes a value of 1 if unit i is an \mathbf{m} -complier. Therefore, $P_i^{\mathbf{m}}(\mathbf{T})$ represents the distribution of unit i being a \mathbf{m} -complier, conditional on \mathbf{T} . In particular, denote $P_i^{\mathbf{m}}$ as the corresponding unconditional distributions. Then, the parameters of interest in this study are defined as follows.

Definition 1 (Parameters of Interest). For each $i \in \{1, 2\}$, $j = 3 - i$, and $d \in \{0, 1\}$,

$$\begin{aligned} \delta_i^{\mathbf{m}}(d) &:= E[Y_i(1, d) - Y_i(0, d) | K_i^{\mathbf{m}} = 1] = E[\Delta_i Y_i(d) | K_i^{\mathbf{m}} = 1], \\ \theta_i^{\mathbf{m}}(d) &:= E[Y_i(d, 1) - Y_i(d, 0) | K_j^{\mathbf{m}} = 1] = E[\Delta_j Y_i(d) | K_j^{\mathbf{m}} = 1]. \end{aligned} \tag{6}$$

$\delta_i^{\mathbf{m}}(d)$ represents the average *direct* effect, which is the average change in the outcome due to an individual's own treatment take-up when the other's take-up status is fixed at d . Additionally, it is a local average within a subpopulation where individual i is an \mathbf{m} -complier. Similarly, $\theta_i^{\mathbf{m}}(d)$ represents the average *indirect* treatment effect, capturing the average changes from the other's treatment take-up when the own treatment status is fixed at d . This is also a local average within a subpopulation where individual j is an \mathbf{m} -complier. Because the interpretations of \mathbf{m} -compliers are determined by an empirical context, the interpretations of these parameters are also context-specific.

3 Identification

This section discusses the identification of causal parameters defined in (6). It builds on Assumptions 1 and 2, as well as a given monotone pair. The argument starts with the observation that the intention-to-treatment (ITT) effects on outcomes are expressed by a weighted average of the parameters, where the weights consist of the distributions of compliers defined in (5). The first observation is that these weights are identified. Subsequently, if we have additional exclusion restrictions for the endogenous treatment, then the parameters are identified, which is the main result of this study. The identification strategy not only encompasses prior findings in the literature as special cases but also extends to scenarios where neither total monotonicity nor one-sided noncompliance are fully satisfied.

3.1 Intention-to-Treatment Effects on Outcomes

In what follows, the intention-to-treatment effects (ITT) are represented as a weighted average of parameters defined in (6). The following Lemma directly follows from equation (4) and Assumption 1.

Lemma 1. *Suppose Assumptions 1, 2 hold and we have a monotone pair $\mathbf{m} = (\mathbf{z}, \mathbf{z}', \mathbf{r})$. Then, the conditional intention-to-treatment effects (ITT) on outcome of unit $i \in \{1, 2\}$ is given by*

$$\begin{aligned} ITT_i^{\mathbf{m}}(\mathbf{X}) &:= E[Y_i | \mathbf{Z}_i = \mathbf{z}_i, \mathbf{X}] - E[Y_i | \mathbf{Z}_i = \mathbf{z}'_i, \mathbf{X}] \\ &= r_i E[K_i^{\mathbf{m}} \Delta_i Y_i(0) | \mathbf{X}] + r_j E[K_j^{\mathbf{m}} \Delta_j Y_i(0) | \mathbf{X}] \\ &\quad + r_i r_j E[K_{ij}^{\mathbf{m}} (\Delta_i Y_i(1) - \Delta_i Y_i(0)) | \mathbf{X}], \end{aligned} \tag{7}$$

where $j = 3 - i$ and $K_{ij}^{\mathbf{m}} := r_i r_j [D_i(\mathbf{z}_i) D_j(\mathbf{z}_j) - D_i(\mathbf{z}'_i) D_j(\mathbf{z}'_j)]$.

By integrating over the distribution of \mathbf{X} and applying law of iterative expectation

on the equation (7), we obtain the following expression:

$$E[ITT_i^{\mathbf{m}}(\mathbf{X})] = r_i \delta_i^{\mathbf{m}}(0) P_i^{\mathbf{m}} + r_j \theta_i^{\mathbf{m}}(0) P_j^{\mathbf{m}} + r_i r_j \zeta_i^{\mathbf{m}} P_{ij}^{\mathbf{m}}, \quad (8)$$

where $P_{ij}^{\mathbf{m}} = \Pr(K_{ij}^{\mathbf{m}} = 1)$, and $\zeta_i^{\mathbf{m}} = E[\Delta_i Y_i(1) - \Delta_i Y_i(0) | K_{ij}^{\mathbf{m}} = 1]$. The first two terms in (8) are related to the local averages of the direct and indirect effects on \mathbf{m} -compliers, and the marginal distributions of being \mathbf{m} -compliers for each unit. The last term consists of the coefficient $\zeta_i^{\mathbf{m}}$ for the distribution $P_{ij}^{\mathbf{m}}$, which is related to the joint compliance patterns between two units. The coefficient $\zeta_i^{\mathbf{m}}$ is not of interest at this moment, as the interpretation of the local average $\zeta_i^{\mathbf{m}}$ is not straightforward. This is because $K_{ij}^{\mathbf{m}} = 1$ if both units are \mathbf{m} -compliers, or one unit is a \mathbf{m} -complier and the other unit is a \mathbf{m} -always taker.

By construction of the monotone pairs, $K_i^{\mathbf{m}}$ is a binary random variable that indicates whether unit i is a \mathbf{m} -complier. This structure allows us to recover the distribution $P_i^{\mathbf{m}}$ of $K_i^{\mathbf{m}}$ from the observed treatment take-up statuses. Proposition 1 shows that the distributions $P_i^{\mathbf{m}}$ and $P_{ij}^{\mathbf{m}}$ are identified.

Proposition 1 (Distribution of \mathbf{m} -Compliers). *Suppose Assumptions 1, 2 hold and we have a monotone pair $\mathbf{m} = (\mathbf{z}, \mathbf{z}', \mathbf{r})$. Let \mathbf{T} be a subset of the set of exogenous variables \mathbf{X} . Then, the following conditional distributions are identified:*

$$\begin{aligned} P_i^{\mathbf{m}}(\mathbf{T}) &:= \Pr(K_i^{\mathbf{m}} = 1 | \mathbf{W}) = r_i E[\omega^{\mathbf{m}} D_i | \mathbf{T}], \\ P_{ij}^{\mathbf{m}}(\mathbf{T}) &:= \Pr(K_{ij}^{\mathbf{m}} = 1 | \mathbf{W}) = r_i r_j E[\omega^{\mathbf{m}} D_i D_j | \mathbf{T}], \end{aligned} \quad (9)$$

where

$$\omega^{\mathbf{m}} = \frac{\mathbb{1}\{\mathbf{Z}_i = \mathbf{z}_i\}}{\Pr(\mathbf{Z}_i = \mathbf{z}_i | \mathbf{X})} - \frac{\mathbb{1}\{\mathbf{Z}_i = \mathbf{z}'_i\}}{\Pr(\mathbf{Z}_i = \mathbf{z}'_i | \mathbf{X})}. \quad (10)$$

In particular, the unconditional distributions $P_i^{\mathbf{m}} = \Pr(K_i^{\mathbf{m}} = 1)$, $P_{ij}^{\mathbf{m}} = \Pr(K_{ij}^{\mathbf{m}} = 1)$ are identified by $r_i E[\omega^{\mathbf{m}} D_i]$, $r_i r_j E[\omega^{\mathbf{m}} D_i D_j]$, respectively.⁶⁷

⁶The weight $\omega^{\mathbf{m}}$ does not depend on individual index, since $\mathbb{1}\{\mathbf{Z}_i = \mathbf{z}_i\} = \mathbb{1}\{\mathbf{Z}_j = \mathbf{z}_j\}$.

⁷Note that for any subset \mathbf{W} of \mathbf{X} , $P_i^{\mathbf{m}}(\mathbf{W}) = E[P_i^{\mathbf{m}}(\mathbf{X}) | \mathbf{W}]$, and $P_{ij}^{\mathbf{m}}(\mathbf{W}) = E[P_{ij}^{\mathbf{m}}(\mathbf{X}) | \mathbf{W}]$.

3.2 General Result with Additional Instrument

The equation (8) can be thought of as a single equation with three unknowns: $\delta_i^m(0)$, $\theta_i^m(0)$, and ζ_i^m . The main idea of the identification strategy is that if we have exogenous variations on P_i^m , P_j^m , and P_{ij}^m in equation (8), then the parameters $\delta_i^m(0)$, $\theta_i^m(0)$, and ζ_i^m can be identified by the coefficients in some linear system induced by equations (8).

To this end, I introduce an additional exclusion restriction for the endogenous treatment. Before stating the required conditions formally, consider a simple example first. Suppose the exogenous variables \mathbf{X} include some discrete random variable T that only takes values in T_1, T_2, T_3 . Assume that T is correlated with the potential treatment take-up, and therefore correlated to the K_i^m, K_j^m and K_{ij}^m . Also assume that T is independent of potential outcomes, once the distributions of potential treatment take-up ($D_i(\cdot), D_j(\cdot)$) are given. Then, by integrating equation (7) over the conditional distribution of \mathbf{X} given T , we obtain the following three equations:

$$E[ITT_i^m(\mathbf{X})|T = T_\ell] = r_i\delta_i^m(0)P_i^m(T_\ell) + r_j\theta_i^m(0)P_j^m(T_\ell) + r_ir_j\zeta_i^mP_{ij}^m(T_\ell), \quad \ell = 1, 2, 3,$$

In this example, the parameters are recovered by

$$\begin{pmatrix} \delta_i^m(0) \\ \theta_i^m(0) \\ \zeta_i^m \end{pmatrix} = \begin{pmatrix} r_iP_i^m(T_1) & r_jP_j^m(T_1) & r_ir_jP_{ij}^m(T_1) \\ r_iP_i^m(T_2) & r_jP_j^m(T_2) & r_ir_jP_{ij}^m(T_2) \\ r_iP_i^m(T_3) & r_jP_j^m(T_3) & r_ir_jP_{ij}^m(T_3) \end{pmatrix}^{-1} \begin{pmatrix} E[ITT_i^m(\mathbf{X})|T = T_1] \\ E[ITT_i^m(\mathbf{X})|T = T_2] \\ E[ITT_i^m(\mathbf{X})|T = T_3] \end{pmatrix},$$

provided that the matrix including $P_i^m(T), P_{ij}^m(T)$ is invertible. Here, the exogenous variable T serves as an additional exclusion restriction for the endogenous treatment, meaning it is related to the potential treatment but does not directly affect the potential outcomes. The next assumption formally states the required conditions for such additional exclusion restrictions in terms of appropriate mean independence.

Assumption 3 (Exclusion Restriction II). *The exogenous variable $\mathbf{X} = (X_1, X_2)$ is divided by two parts: $X_i = (W_i, T_i)$, $i \in \{1, 2\}$. For the vector of exogenous variables*

$\mathbf{T} = (T_1, T_2)$, assume

$$\begin{aligned} E[\Delta_i Y_i(0) | K_i^{\mathbf{m}} = 1, \mathbf{T}] &= E[\Delta_i Y_i(0) | K_i^{\mathbf{m}} = 1], \\ E[\Delta_j Y_i(0) | K_j^{\mathbf{m}} = 1, \mathbf{T}] &= E[\Delta_j Y_i(0) | K_j^{\mathbf{m}} = 1], \\ E[\Delta_i Y_i(1) - \Delta_i Y_i(0) | K_{ij}^{\mathbf{m}} = 1, \mathbf{T}] &= E[\Delta_i Y_i(1) - \Delta_i Y_i(0) | K_{ij}^{\mathbf{m}} = 1]. \end{aligned}$$

Subsequently, Lemma 2 states the observation in the above simple example.

Lemma 2. Suppose Assumptions 1-3 hold and we have a monotone pair $\mathbf{m} = (\mathbf{z}, \mathbf{z}', \mathbf{r}) = ((z_1, z_2), (z'_1, z'_2), (r_1, r_2)) \in \mathcal{M}$. Then, for each $i \in \{1, 2\}$ and $j = 3 - i$,

$$E[ITT_i^{\mathbf{m}}(\mathbf{X}) | \mathbf{T}] = \tilde{\mathbf{P}}_i^{\mathbf{m}}(\mathbf{T})' \boldsymbol{\beta}_i^{\mathbf{m}}, \quad (11)$$

where $\boldsymbol{\beta}_i^{\mathbf{m}} = (\delta_i^{\mathbf{m}}(0), \theta_i^{\mathbf{m}}(0), \zeta^{\mathbf{m}})'$, and $\tilde{\mathbf{P}}_i^{\mathbf{m}}(\mathbf{T}) = (r_i P_i^{\mathbf{m}}(\mathbf{T}), r_j P_j^{\mathbf{m}}(\mathbf{T}), r_i r_j P_{ij}^{\mathbf{m}}(\mathbf{T}))'$.

By stacking equation (11) for two units, we have the following conditional mean equation.

$$ITT(\mathbf{T}) := E \begin{bmatrix} ITT_1^{\mathbf{m}}(\mathbf{X}) \\ ITT_2^{\mathbf{m}}(\mathbf{X}) \end{bmatrix} \bigg| \mathbf{T} = \begin{pmatrix} \tilde{\mathbf{P}}_1^{\mathbf{m}}(\mathbf{T}) & 0 \\ 0 & \tilde{\mathbf{P}}_2^{\mathbf{m}}(\mathbf{T}) \end{pmatrix} \begin{pmatrix} \boldsymbol{\beta}_1^{\mathbf{m}} \\ \boldsymbol{\beta}_2^{\mathbf{m}} \end{pmatrix} := \tilde{\mathbf{P}}^{\mathbf{m}}(\mathbf{T})' \boldsymbol{\beta}^{\mathbf{m}},$$

where $\tilde{\mathbf{P}}$ is the block diagonal matrix of $\tilde{\mathbf{P}}_1^{\mathbf{m}}(\mathbf{T})$ and $\tilde{\mathbf{P}}_2^{\mathbf{m}}(\mathbf{T})$ and $\boldsymbol{\beta} = (\boldsymbol{\beta}_1^{\mathbf{m}}, \boldsymbol{\beta}_2^{\mathbf{m}})'$. Note that the ITTs can be written as $E[ITT_i^{\mathbf{m}}(\mathbf{X}) | \mathbf{T}] = E[\omega^{\mathbf{m}} Y_i | \mathbf{T}]$ by using the weight $\omega^{\mathbf{m}}$ defined in (10), and $\tilde{\mathbf{P}}^{\mathbf{m}}(\mathbf{T}) = E[\omega^{\mathbf{m}} \tilde{\mathbf{D}} | \mathbf{T}]$ by Proposition 1, where $\tilde{\mathbf{D}}$ is the block diagonal matrix of $\tilde{\mathbf{D}}_1$ and $\tilde{\mathbf{D}}_2$, and $\tilde{\mathbf{D}}_i = (D_i, D_j, D_i D_j)'$. Therefore, we have the following conditional moment restriction from Lemma 2:

$$E[g^{\mathbf{m}}(\mathbf{V}, \boldsymbol{\beta}^{\mathbf{m}}) | \mathbf{T}] := E[\omega^{\mathbf{m}} (\mathbf{Y} - \tilde{\mathbf{D}}' \boldsymbol{\beta}^{\mathbf{m}}) | \mathbf{T}] = 0, \quad (12)$$

where $\mathbf{Y} = (Y_1, Y_2)'$, and $\mathbf{V} = (\mathbf{Y}, \mathbf{D}, \mathbf{Z}, \mathbf{X})$. From the conditional moment restriction, IV estimand identifies the parameter vector $\boldsymbol{\beta}^{\mathbf{m}}$. Assumption 4 is a sufficient condition to identification in this setting,

Assumption 4 (Identification). *There exists a matrix $\mathbf{R}(\mathbf{T}) \in \mathbb{R}^{6 \times 2}$ of functions of \mathbf{T} such that $E[\mathbf{R}(\mathbf{T})\tilde{\mathbf{P}}'(\mathbf{T})]$ is nonsingular.*

Because the rank of $\mathbf{R}(\mathbf{T})\tilde{\mathbf{P}}'(\mathbf{T})$ is at most 2 for a fixed \mathbf{T} , \mathbf{T} need to have at least 3 distinct values with positive probability. This rank condition for the exogenous variable \mathbf{T} is intuitive as the observations in the above simple example. Proposition 2 shows the IV estimand is identified correspondingly.

Proposition 2 (Identification). *Suppose Assumptions 1-4 hold. Then, the parameter β^m is identified by*

$$\begin{aligned}\beta^m &= E[\mathbf{R}(\mathbf{T})\omega^m \tilde{\mathbf{D}}']^{-1} E[\mathbf{R}(\mathbf{T})\omega^m \mathbf{Y}]. \\ &= E[\mathbf{R}(\mathbf{T})\tilde{\mathbf{P}}'(\mathbf{T})]^{-1} E[\mathbf{R}(\mathbf{T})ITT(\mathbf{T})].\end{aligned}$$

3.3 Special Cases

This subsection discusses about two special cases where P_i^m , P_j^m , and P_{ij}^m are linearly dependent, hence Assumption 4 is violated.

3.3.1 Identification Under Cross-Monotonicity

The first special case is that the case when we don't need to consider the joint compliance patterns, but also need to ignore the last interaction term $\zeta_i^m P_{ij}^m(\mathbf{T})$ in (11). The following lemma states a property of this special cases.

Lemma 3. *Let \mathbf{T} be any subset of the set exogenous variable \mathbf{X} , and $\mathbf{z} = (z_1, z_2) \in \{0, 1\}^2$ be given. Denote $\mathbf{z}_i = (z_i, z_{3-i})$ for unit $i = \{1, 2\}$. Then, for each unit $i \in \{1, 2\}$ and $j = 3 - i$, $\Pr(D_i(\mathbf{z}_i) \geq D_j(\mathbf{z}_j)|\mathbf{T}) = 1$ if and only if $\Pr(D_j(\mathbf{z}_j) = D_i(\mathbf{z}_i)D_j(\mathbf{z}_j)|\mathbf{T}) = 1$.*

I call the condition $\Pr(D_i(\mathbf{z}_i) \geq D_j(\mathbf{z}_j)|\mathbf{T}) = 1$ as a *cross monotonicity* between $D_i(\mathbf{z}_i)$ and $D_j(\mathbf{z}_j)$ at the assignment \mathbf{z} . Therefore, if we have a monotone pair $\mathbf{m} =$

$(\mathbf{z}, \mathbf{z}', \mathbf{r})$ with $\mathbf{r} = (1, 1)$, and if cross monotonicity holds at both \mathbf{z} and \mathbf{z}' , then we have

$$K_{ij}^m = [D_i(\mathbf{z}_i)D_j(\mathbf{z}_j) - D_i(\mathbf{z}'_i)D_j(\mathbf{z}'_j)] = [D_j(\mathbf{z}_j) - D_j(\mathbf{z}'_j)] = K_j^m,$$

conditional on \mathbf{T} with probability 1. The coefficient ζ_i is now $\theta_i(1) - \theta_i(0)$. In this case, we can cancel the interaction term in equation (11) as follows:

$$E[ITT_i^m(\mathbf{X})|\mathbf{T}] = \delta_i^m(0)P_i^m(\mathbf{T}) + \theta_i^m(1)P_j^m(\mathbf{T}).$$

Proposition 3 summarizes this special case.

Proposition 3. *Suppose Assumptions 1-3 hold and we have a monotone pair $\mathbf{m} = (\mathbf{z}, \mathbf{z}', \mathbf{r})$. Furthermore, assume $\Pr(D_i(\mathbf{z}_i) \geq D_j(\mathbf{z}_j)|\mathbf{T}) = \Pr(D_i(\mathbf{z}'_i) \geq D_j(\mathbf{z}'_j)|\mathbf{T}) = 1$ for $i \in \{1, 2\}$ and $j = 3 - i$. Then, equation (11) is written as*

$$E[ITT_i^m(\mathbf{X})|\mathbf{T}] = \check{\mathbf{P}}_i^m(\mathbf{T})' \check{\boldsymbol{\beta}}_i^m, \quad i \in \{1, 2\}, \quad (13)$$

where $\check{\boldsymbol{\beta}}_i^m = (\delta_i^m(0), \theta_i^m(1))'$, and $\check{\mathbf{P}}_i^m(\mathbf{T}) = (r_i P_i^m(\mathbf{T}), r_j P_j^m(\mathbf{T}))'$. Also, if there exists a matrix $\check{\mathbf{R}}(\mathbf{T}) \in \mathbb{R}^{4 \times 2}$ of functions of \mathbf{T} such that $E[\check{\mathbf{R}}(\mathbf{T})\check{\mathbf{P}}_i^m(\mathbf{T})']$ is nonsingular, the parameter is identified by

$$\check{\boldsymbol{\beta}}^m = \begin{pmatrix} \check{\boldsymbol{\beta}}_1^m \\ \check{\boldsymbol{\beta}}_2^m \end{pmatrix} = E[\check{\mathbf{R}}(\mathbf{T})\omega^m \check{\mathbf{D}}']^{-1} E[\check{\mathbf{R}}(\mathbf{T})\omega^m \mathbf{Y}],$$

where $\check{\mathbf{D}}$ is the block diagonal matrix of $(D_1, D_2)'$ and $(D_2, D_1)'$.

There is an interesting situation in that the cross monotonicity is satisfied.

Remark 2 (Weak one-sided noncompliance with joint take-up). Suppose the weak one-sided noncompliance (WOSN) in (3):

$$\Pr(D_i(0, 0) = 0|\mathbf{X}) = 1 \quad \forall i \in \{1, 2\}.$$

Additionally, suppose in this situation, units can jointly take the treatment if one unit

takes the treatment. Then, we have a cross-monotonicity $D_i(1,0) \geq D_j(0,1)$, since $D_j(0,1)$ is 1 if and only if both units jointly take the treatment. Hence, we have two monotone pairs $\mathbf{m}_1 = ((1,0), (0,0), (1,1))$ and $\mathbf{m}_2 = ((0,1), (0,0), (1,1))$ from the weak one-sided noncompliance, and the cross monotonicity holds for each treatment assignments in $\mathbf{m}_1, \mathbf{m}_2$. Thus, we can apply Proposition 3. \square

3.3.2 Identification without Assumption 3

The second special case is when two out of three terms in equation (11) disappear. Suppose we have a monotone pair $\mathbf{m} = (\mathbf{z}, \mathbf{z}', \mathbf{r})$. If unit j is almost surely a \mathbf{m} -never taker conditional on \mathbf{T} , then $D_j(\mathbf{z}_j) = D_j(\mathbf{z}'_j) = 0$. This implies that $K_j^{\mathbf{m}} = K_{ij}^{\mathbf{m}} = 0$ conditional on \mathbf{T} with probability 1, and hence we have $P_j^{\mathbf{m}}(\mathbf{T}) = P_{ij}^{\mathbf{m}}(\mathbf{T}) = 0$ in equation (11). In this special case, we don't need Assumption 3 because the last two terms in (7), or (8) are zero, so we only have one unknown in one equation. The opposite case occurs when unit j is almost surely a \mathbf{m} -always taker, which results in $P_j^{\mathbf{m}}(\mathbf{T}) = 0$, $P_{ij}^{\mathbf{m}}(\mathbf{T}) = P_i^{\mathbf{m}}(\mathbf{T})$, and $\zeta_i^{\mathbf{m}} = \delta_i^{\mathbf{m}}(1) - \delta_i^{\mathbf{m}}(0)$. Proposition 4 summarizes the identification in these cases.

Proposition 4. *Suppose Assumptions 1-2 hold, and $P_i^{\mathbf{m}} > 0$ for $i \in \{1, 2\}$. Then, for a given monotone pair $\mathbf{m} = (\mathbf{z}, \mathbf{z}', \mathbf{r})$, we have the following result.*

1. *If $j = 3 - i \in \{1, 2\}$ is \mathbf{m} -never taker, conditional on \mathbf{X} with probability 1, i.e., $\Pr(D_j(\mathbf{z}_j) = D_j(\mathbf{z}'_j) = 0 | \mathbf{X}) = 1$, then*

$$\delta_i^{\mathbf{m}}(0) = r_i \frac{ITT_i^{\mathbf{m}}}{P_i^{\mathbf{m}}}, \quad \theta_j^{\mathbf{m}}(0) = r_i \frac{ITT_j^{\mathbf{m}}}{P_i^{\mathbf{m}}}.$$

2. *If $j = 3 - i \in \{1, 2\}$ is \mathbf{m} -always taker, conditional on \mathbf{X} with probability 1, i.e., $\Pr(D_j(\mathbf{z}_j) = D_j(\mathbf{z}'_j) = 1 | \mathbf{X}) = 1$, then*

$$\delta_i^{\mathbf{m}}(1) = r_i \frac{ITT_i^{\mathbf{m}}}{P_i^{\mathbf{m}}}, \quad \theta_j^{\mathbf{m}}(1) = r_i \frac{ITT_j^{\mathbf{m}}}{P_i^{\mathbf{m}}}.$$

Remark 3 (One-sided noncompliance). This result generalizes the identification result demonstrated in [Vazquez-Bare \(2022\)](#) since assuming one-sided noncompliance implies that one unit is almost surely a \mathbf{m} -never taker. In the one-sided noncompliance situation, we have a monotone pair $\mathbf{m} = ((1, 0), (0, 0), (1, 1))$ where unit 2 is a \mathbf{m} -never taker with a probability of 1 (since $D_2(0, 1) = D_2(0, 0) = 0$ with probability 1). The same argument can apply to unit 1 for the monotone pair $\mathbf{m} = ((0, 1), (0, 0), (1, 1))$.

Remark 4 (Personalized encouragement and classical monotonicity). This result can also be considered a generalization of the identification of local average treatment effect proposed in [Imbens and Angrist \(1994\)](#). In situations where units do not interact, i.e., SUTVA is satisfied, and personalized encouragement is also satisfied. Referring to Table 3, under personalized encouragement, we have a monotone pair $\mathbf{m} = ((1, 1), (0, 1), (1, 1))$, where unit 2 is a \mathbf{m} -always taker (as well as a never taker). Additionally, since there is no interaction in the outcome determination, we have $\delta^{\mathbf{m}}(1) = \delta^{\mathbf{m}}(0)$. The same argument can apply to unit 1 for the monotone pair $\mathbf{m} = ((1, 1), (1, 0), (1, 1))$.

4 Estimation

In this section, I propose a two-stage estimation procedure for estimating direct and indirect local average treatment effects discussed in previous sections. Let $\mathbf{V}_g = (\mathbf{Y}_g, \mathbf{Z}_g, \mathbf{D}_g, \mathbf{X}_g)$ be observed data for $g = 1, \dots, G$ and \mathbf{m} be a monotone pair satisfying Assumption 2. The conditional moment restriction (12) derived by Lemma 2 implies the following unconditional moment:⁸⁹

$$E[\mathbf{R}(\mathbf{T}_g)g^{\mathbf{m}}(\mathbf{V}_g, \boldsymbol{\beta})] = 0,$$

⁸The same argument can be used to derive estimator for the special cases in Proposition 3. And [Vazquez-Bare \(2022\)](#) proposed IV estimation in the situation of 4.

⁹If there are multiple monotone pairs, then we can stack all conditional moment conditions and derive the estimator similarly for efficiency gain.

where $\mathbf{R}(\mathbf{T}_g)$ is a 6×2 matrix of functions of \mathbf{T}_g , $g^{\mathbf{m}}(\mathbf{V}_g, \boldsymbol{\beta}) = \omega^{\mathbf{m}}(\mathbf{Y}_g - \tilde{\mathbf{D}}_g' \boldsymbol{\beta}^{\mathbf{m}})$, and

$$\mathbf{Y}_g = \begin{pmatrix} Y_{1g} \\ Y_{2g} \end{pmatrix}, \quad \tilde{\mathbf{D}}_g = \begin{pmatrix} \tilde{\mathbf{D}}_{1g} & 0 \\ 0 & \tilde{\mathbf{D}}_{1g} \end{pmatrix}, \quad \boldsymbol{\beta}^{\mathbf{m}} = \begin{pmatrix} \beta_1^{\mathbf{m}} \\ \beta_2^{\mathbf{m}} \end{pmatrix}, \quad \beta_i^{\mathbf{m}} = \begin{pmatrix} \delta_i^{\mathbf{m}}(0) \\ \theta_i^{\mathbf{m}}(0) \\ \zeta_i^{\mathbf{m}} \end{pmatrix}, i \in \{1, 2\}.$$

For simplicity, consider one monotone pair is given and omit the \mathbf{m} superscript below.¹⁰ The optimal matrix of instruments corresponding to the moment condition is given by

$$\begin{aligned} \mathbf{R}(\mathbf{T}_g) &= -\tilde{\mathbf{P}}'(\mathbf{T}_g) \mathbf{S}^{-1}(\mathbf{T}_g), \\ \tilde{\mathbf{P}}(\mathbf{T}_g) &= E \left[\frac{\partial g(\mathbf{V}_g, \beta_0)}{\partial \beta'} \middle| \mathbf{T}_g \right] = -E[\omega \tilde{\mathbf{D}}_g | \mathbf{T}_g], \\ \mathbf{S}(\mathbf{T}_g) &= E[g(\mathbf{V}_g, \beta_0) g(\mathbf{V}_g, \beta_0)' | \mathbf{T}_g] = E[\omega^2 \boldsymbol{\varepsilon}_g \boldsymbol{\varepsilon}_g' | \mathbf{T}_g], \end{aligned}$$

where $\boldsymbol{\varepsilon}_g = (\mathbf{Y}_g - \tilde{\mathbf{D}}_g \boldsymbol{\beta})$.

To implement efficient IV estimator, we need two-stages to estimate $\boldsymbol{\beta}$. In the first stage, the weight ω , and the instrument $\mathbf{R}(\mathbf{T}_g)$ are estimated, and then $\boldsymbol{\beta}$ is estimated in the second stage by estimated weight and instruments. I propose a parametric first stage. First, suppose the propensity score are estimated by the following parametric model with the parameter $\gamma \in \Gamma \subset \mathbb{R}^{k_\gamma}$.

$$\Pr(\mathbf{Z}_g = \mathbf{z} | \mathbf{X}_g; \gamma) = q(\mathbf{z}, \mathbf{X}_g, \gamma).$$

Then, define the weight ω corresponding to the parameter γ as

$$\omega_g(\gamma) = \frac{\mathbb{1}\{\mathbf{Z}_g = \mathbf{z}\}}{q(\mathbf{z}, \mathbf{X}_g, \gamma)} - \frac{\mathbb{1}\{\mathbf{Z}_g = \mathbf{z}'\}}{q(\mathbf{z}', \mathbf{X}_g, \gamma)},$$

and $\omega_g := \omega_g(\gamma_0)$ for the true value γ_0 of γ . Next, for some consistent estimator $\hat{\gamma}$ of γ ,

¹⁰This can be thought as estimate for each monotone pair separately if we have multiple monotone pairs.

and some feasible instrument $\tilde{R}(\mathbf{T}_g)$, the first-stage IV estimator of β is given by

$$\tilde{\beta} = \left[\frac{1}{G} \sum_{g=1}^G \tilde{R}(\mathbf{T}_g) \omega_g(\hat{\gamma}) \tilde{\mathbf{D}}_g \right]^{-1} \frac{1}{G} \sum_{g=1}^G \tilde{R}(\mathbf{T}_g) \omega_g(\hat{\gamma}) \mathbf{Y}_g.$$

The optimal instrument is estimated by the following parametric model with the parameter $\phi \in \Phi \subset \mathbb{R}^{k_\phi}$.¹¹

$$\begin{aligned} \mathbf{S}(\mathbf{T}_g, \phi) &:= E[\omega_g(\gamma)^2 (\mathbf{Y}_g - \tilde{\mathbf{D}}_g \beta)(\mathbf{Y}_g - \tilde{\mathbf{D}}_g \beta)' | \mathbf{T}_g; \phi], \\ \mathbf{P}(\mathbf{T}_g, \phi) &:= E[\omega_g(\gamma) \tilde{\mathbf{D}}_g | \mathbf{T}_g; \phi], \\ \mathbf{R}(\mathbf{T}_g, \phi) &:= \mathbf{P}'(\mathbf{T}_g, \phi) \mathbf{S}(\mathbf{T}_g, \phi)^{-1}. \end{aligned}$$

And $\mathbf{S}(\mathbf{T}_g) = \mathbf{S}(\mathbf{T}_g, \phi_0)$, $\mathbf{P}(\mathbf{T}_g) = \mathbf{P}(\mathbf{T}_g, \phi_0)$ for the true value ϕ_0 of ϕ . In the second-stage, the parameter β is estimated by

$$\hat{\beta} = \left[\frac{1}{G} \sum_{g=1}^G \mathbf{R}(\mathbf{T}_g, \hat{\phi}) \omega_g(\hat{\gamma}) \tilde{\mathbf{D}}_g \right]^{-1} \frac{1}{G} \sum_{g=1}^G \mathbf{R}(\mathbf{T}_g, \hat{\phi}) \omega_g(\hat{\gamma}) \mathbf{Y}_g.$$

As usual generated instrument, the first-stage estimation of instrument does not affect to the asymptotic variance of the second-stage estimation of β . However, the asymptotic variance need to be adjusted to account for the estimation error of the first-stage estimator of weight, the estimation of $\Pr(\mathbf{Z}_g = \mathbf{z} | \mathbf{X}_g)$. Assumption 5 lists the required regularity conditions for consistency and asymptotic normality of the estimator $\hat{\beta}$, and Proposition 5 states the asymptotic properties of the proposed estimator.

Assumption 5 (Regularity Conditions).

1. $\{\mathbf{V}_g : 1 \leq g \leq G\}$ are independently and identically distributed.
2. $\beta \in B \subset \mathbb{R}^k$, B is compact. And $\beta_0 \in B$, $\gamma_0 \in \Gamma$, and $\phi_0 \in \Phi$ are interior points.
3. $\mathbf{S}(\mathbf{T}_g)$ is positive definite and $E[\mathbf{P}'(\mathbf{T}_g) \mathbf{S}(\mathbf{T}_g) \mathbf{P}(\mathbf{T}_g)]$ is nonsingular.
4. $E[\|\mathbf{Y}_g\|^4] < \infty$.

¹¹The parameter ϕ includes the parameters in the conditional expectations for optimal instrument as well as first-stage estimator $\hat{\gamma}$, $\tilde{\beta}$.

5. (i) There exists q_0 such that $0 < q_0 \leq \inf_{\gamma \in \mathcal{N}_\gamma} q(\mathbf{z}, \mathbf{X}_g, \gamma)$ with probability 1, for all $\mathbf{z} \in \{0, 1\}^2$, for some compact neighborhood \mathcal{N}_γ of γ_0 (ii) q is continuously differentiable in γ with probability 1, and (iii) $E \left[\sup_{\gamma \in \mathcal{N}_\gamma} \left\| \frac{\partial q(\mathbf{z}, \mathbf{X}_g, \gamma)}{\partial \gamma'} \right\|^2 \right] < \infty$ for all $\mathbf{z} \in \{0, 1\}^2$.
6. (i) There exists λ_0 such that $0 < \lambda_0 \leq \inf_{\phi \in \mathcal{N}_\phi} \lambda_{\min} \mathbf{S}(\mathbf{T}_g, \phi)$ with probability 1, for some compact neighborhood \mathcal{N}_ϕ of ϕ_0 , (ii) $\mathbf{S}(\mathbf{T}_g, \phi)$, $\mathbf{P}(\mathbf{T}_g, \phi)$ are continuously differentiable in ϕ with probability 1, and (iii) $E \left[\sup_{\phi \in \mathcal{N}_\phi} \left\| \frac{\partial \mathbf{S}(\mathbf{T}_g, \phi)}{\partial \phi'} \right\|^2 \right] < \infty$, $E \left[\sup_{\phi \in \mathcal{N}_\phi} \left\| \frac{\partial \mathbf{P}(\mathbf{T}_g, \phi)}{\partial \phi'} \right\|^2 \right] < \infty$.
7. For the first-stage estimators,

$$\begin{aligned} \sqrt{G}(\hat{\gamma} - \gamma_0) &= \frac{1}{\sqrt{G}} \sum_{g=1}^G \psi_\gamma(\mathbf{V}_g, \gamma_0) + o_p(1), \quad E[\|\psi_\gamma(\mathbf{V}_g, \gamma_0)\psi_\gamma(\mathbf{V}_g, \gamma_0)'\|] < \infty, \\ \sqrt{G}(\hat{\phi} - \phi_0) &= \frac{1}{\sqrt{G}} \sum_{g=1}^G \psi_\phi(\mathbf{V}_g, \phi_0) + o_p(1), \quad E[\|\psi_\phi(\mathbf{V}_g, \phi_0)\psi_\phi(\mathbf{V}_g, \phi_0)'\|] < \infty. \end{aligned}$$

Proposition 5. Under Assumptions 1-4 and 5, the two-stage estimator $\hat{\beta}$ is a consistent estimator for β_0 , and

$$\sqrt{G}(\hat{\beta} - \beta_0) \xrightarrow{d} N(0, E[\psi_\beta(\mathbf{V}_g, \beta_0)\psi_\beta(\mathbf{V}_g, \beta_0)']),$$

where

$$\begin{aligned} \psi_\beta(\mathbf{V}_g, \beta_0) &= \mathbf{A}^{-1} \left[\mathbf{R}(\mathbf{T}_g) \omega_g(\mathbf{Y}_g - \tilde{\mathbf{D}}_g \beta_0) + \mathbf{B} \psi_\gamma(\mathbf{V}_g, \gamma_0) \right], \\ \mathbf{A} &= E \left[\mathbf{P}'(\mathbf{T}_g) \mathbf{S}^{-1}(\mathbf{T}_g) \mathbf{P}(\mathbf{T}_g) \right], \\ \mathbf{B} &= E \left[\mathbf{R}(\mathbf{T}_g) (\mathbf{Y}_g - \mathbf{D}_g \beta_0) \frac{\partial \omega_g(\gamma_0)}{\partial \gamma'} \right]. \end{aligned}$$

Also, the standard error can be estimated based on the following consistent estimator of

asymptotic variance:

$$\hat{\mathbf{V}} = \hat{\mathbf{A}}^{-1} \frac{1}{G} \sum_{g=1}^G \left[\mathbf{R}(\mathbf{T}_g, \hat{\phi}) \omega_g(\hat{\gamma}) (\mathbf{Y}_g - \tilde{\mathbf{D}}_g \hat{\beta}) + \hat{\mathbf{B}} \hat{\psi}_{\gamma}(\mathbf{V}_g, \hat{\gamma}) \right],$$

where $\hat{\psi}_{\gamma}$ is the empirical influence function of ψ_{γ} , and

$$\begin{aligned} \hat{\mathbf{A}} &= \frac{1}{G} \sum_{g=1}^G \mathbf{R}(\mathbf{T}_g, \hat{\phi}) \omega_g(\hat{\gamma}) \tilde{\mathbf{D}}_g, \\ \hat{\mathbf{B}} &= \frac{1}{G} \sum_{g=1}^G \mathbf{R}(\mathbf{T}_g, \hat{\phi}) (\mathbf{Y}_g - \tilde{\mathbf{D}}_g \hat{\beta}) \frac{\partial \omega_g(\hat{\gamma})}{\partial \gamma'}. \end{aligned}$$

5 Simulation

This section examines how the estimation procedure proposed in Section 4 works through simulations. As illustrated in Figure 2, two units interact with each other both in treatment take-up and potential outcomes. I set up different designs based on various restrictions on potential treatment used in the literature, including total monotonicity, one-sided noncompliance, and personalized encouragement. Additionally, I consider a design where none of the aforementioned assumptions are satisfied (weak one-sided noncompliance). As shown in Table 3, the concept of monotonicity in Assumption 2 can be applied to all of those restrictions. When one-sided noncompliance or personalized encouragement are assumed, the parameters are estimated using the same estimation procedure as outlined in Section 4 according to the identification in Proposition 4. To begin, I introduce the data generating process for generating fake datasets.

5.1 Data Generating Process and Designs

First, exogenous variables $X_i = (T_i, W_i)$ are generated by uniform and standard normal distribution, respectively.

$$T_i \sim \text{Uniform}(0, 1), \quad W_i \sim N(0, 1), \quad i \in \{1, 2\}.$$

5.1.1 Treatment Assignments and Take-up

The treatment assignment and potential treatment take-up statuses are generated by the following single index model:

$$Z_i = \mathbb{1}\{\eta_i \leq \gamma_{i1} + W_i\gamma_{i2} + W_j\gamma_{i3} + T_i\gamma_{i4} + T_j\gamma_{i5}\},$$

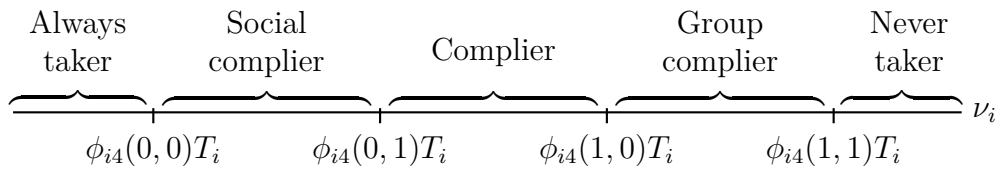
$$D_i(z_i, z_j) = \mathbb{1}\{\nu_i \leq \phi_{i1}(z_i, z_j) + \phi_{i2}(z_i, z_j)W_i + \phi_{i3}(z_i, z_j)W_j + \phi_{i4}(z_i, z_j)T_i\},$$

where η_i, ν_i are generated from standard normal distributions. For the coefficients γ , I set $\gamma_{i0} = 1$ and $\gamma_{ik} = 0.1$ for $k = 2, 3, 4, 5$, for both units $i = 1, 2$. The coefficients ϕ determine the distribution of potential treatment, and I set up four designs according to the value of ϕ .

Design 1 (TM)

Design 1 represents a situation where total monotonicity holds. If $\phi_{ik}(\mathbf{z})$ for $k = 1, 2, 3$ are fixed for all $\mathbf{z} \in \{0, 1\}^2$, then monotonicity follows from the monotonicity on $\phi_{i4}(\mathbf{z})$. In particular, if $\phi_{i4}(1, 1) \geq \phi_{i4}(1, 0) \geq \phi_{i4}(0, 1) \geq \phi_{i4}(0, 0)$ for both units, then the DGP satisfies total monotonicity. In this case, following the classification of compliance types in [Vazquez-Bare \(2022\)](#), unit i 's compliance types are classified by the realization of ν , as illustrated in [Figure 4](#).

Figure 4: Five Compliance Types in Total Monotonicity



Design 2 (TM+OSN)

Design 2 represents a situation where both total monotonicity and one-sided noncompliance hold. The one-sided noncompliance assumption can be implied by generating $D_i(0, 1) = D_i(0, 0) = 0$ with probability 1. Numerically, this case can be generated by setting a large negative number as the constant term $\phi_{i1}(\mathbf{z})$ for all $\mathbf{z} \in 0, 1^2$.

Design 3 (PE)

Design 3 represents a situation where personalized encouragement holds. If $\phi_{ik}(z_i, 0) = \phi_{ik}(z_i, 1)$, then the DGP satisfies the personalized encouragement design since potential treatment does not depend on the other's treatment assignment. Further, classical monotonicity in this case can be implied by setting $\phi_{i4}(1, \cdot) \geq \phi_{i4}(0, \cdot)$ for both units $i = 1, 2$.

Design 4 (WOSN)

Design 4 represents a situation where neither total monotonicity, one-sided noncompliance, nor personalized encouragement are satisfied. To generate this design, $\phi_{ik}(\mathbf{z})$ for $k = 1, 2, 3$ need to be neither fixed nor satisfying certain monotonicity for the almost sure ordering of potential treatments to be not always guaranteed. Also, $D_i(0, 1)$ is generated to be 1 with positive probability, so that one-sided noncompliance does not hold. The only restriction on potential treatments in this design is $D_i(0, 0) = D_j(0, 0) = 0$ with probability 1 to guarantee the existence of a monotone pair. The resulting design actually represents weak one-sided noncompliance.

Table 4 shows the coefficient values for each design.

5.1.2 Outcomes

The common monotone pairs for all 4 designs are $\mathbf{m}_1 = ((1, 0), (0, 0), (1, 1))$ and $\mathbf{m}_2 = ((0, 1), (0, 0), (1, 1))$. I focus on the monotone pair \mathbf{m}_1 in this simulation. To consider heterogeneities in potential outcomes with respect to \mathbf{m}_1 -compliance type, the potential

Table 4: Coefficients for each design

Design	(z_i, z_j)	Unit 1				Unit 2			
		ϕ_{11}	ϕ_{12}	ϕ_{13}	ϕ_{14}	ϕ_{21}	ϕ_{22}	ϕ_{23}	ϕ_{24}
1 (TM)	(1, 1)	-1	0.1	0.1	7	-1.5	0.2	0.2	8
	(1, 0)	-1	0.1	0.1	4	-1.5	0.2	0.2	4
	(0, 1)	-1	0.1	0.1	1	-1.5	0.2	0.2	2
	(0, 0)	-1	0.1	0.1	0	-1.5	0.2	0.2	0
2 (TM+OSN)	(1, 1)	-1	0.1	0.1	7	-1.5	0.2	0.2	8
	(1, 0)	-1	0.1	0.1	4	-1.5	0.2	0.2	4
	(0, 1)	-1000	0.1	0.1	1	-1000	0.2	0.2	2
	(0, 0)	-1000	0.1	0.1	0	-1000	0.2	0.2	0
3 (PE)	(1, 1)	-1	0.1	0.1	7	-1.5	0.2	0.2	8
	(1, 0)	-1	0.1	0.1	7	-1.5	0.2	0.2	8
	(0, 1)	-1	0.1	0.1	1	-1.5	0.2	0.2	2
	(0, 0)	-1	0.1	0.1	1	-1.5	0.2	0.2	2
4 (WOSN)	(1, 1)	-1	0.2	0.1	7	-1.5	0.4	0.2	8
	(1, 0)	-1	0.1	0.2	4	-1.5	0.2	0.4	4
	(0, 1)	-1	0.2	0.2	1	-1.5	0.4	0.4	2
	(0, 0)	-1000	0.1	0.1	0	-1000	0.2	0.2	0

notes. The potential treatment take-up is generated by

$$D_i(z_i, z_j) = \mathbb{1}\{\nu_i \leq \phi_{i1}(z_i, z_j) + \phi_{i2}(z_i, z_j)W_i + \phi_{i3}(z_i, z_j)W_j + \phi_{i4}(z_i, z_j)T_i\},$$

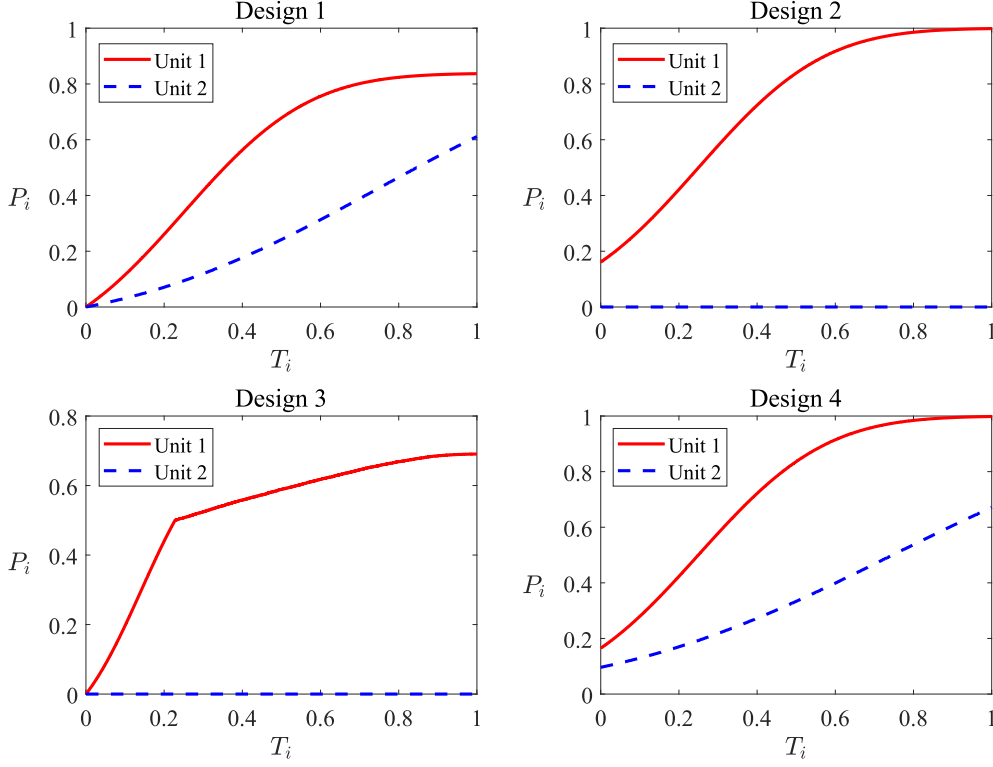
for $i \in 1, 2$ and $j = 3 - i$. Design 1 satisfies total monotonicity. Design 2 satisfies total monotonicity and one-sided noncompliance. Design 3 satisfies personalized encouragement. Design 4 satisfies weak one-sided noncompliance.

outcome is generated by

$$Y_i(d_i, d_j) = Y_i^D(d_i) + Y_i^I(d_j) + W_i + 0.5W_j,$$

where $Y_i^p(d)$ is generated by a normal distribution with mean $K_i^{m_1} \bar{Y}_i^p(d)$ and variance 1, for $p \in D, I$, $i \in 1, 2$, $d \in 0, 1$. Note that this DGP represents that the potential outcome is additively separable with respect to treatment take-up for two units. This additive separability implies that $\Delta_i Y_i(1) = \Delta_i Y_i(0)$, and hence the direct and indirect effects do not depend on the other unit's treatment take-up status. Therefore, the last

Figure 5: Distribution of \mathbf{m}_1 -compliers



notes.

term in equation (4).¹²

It follows that for designs 1 and 4, the moment condition for each unit is given by

$$E[\omega^{m_1} Y_i | \mathbf{T}] = \delta_i^{m_1}(0) P_i^{m_1}(\mathbf{T}) + \theta_i^{m_1}(0) P_j^{m_1}(\mathbf{T}), \quad i = 1, 2.$$

Because unit 2 is \mathbf{m}_1 -never taker under design 2 and \mathbf{m}_1 -always taker under design 3¹³, the moment condition for each unit is

$$E[\omega^{m_1} Y_1 | \mathbf{T}] = \delta_1^{m_1}(0) P_1^{m_1}(\mathbf{T}) \quad E[\omega^{m_1} Y_2 | \mathbf{T}] = \theta_2^{m_1}(0) P_1^{m_1}(\mathbf{T}), \quad (14)$$

¹²This restriction is not necessary for the simulation. See table (n) in the Appendix for the result from a more realistic design of potential outcomes.

¹³In design 2, one-sided noncompliance holds, i.e., $D_2(0, 1) = D_2(0, 0) = 0$ with probability 1. In design 3, personalized encouragement holds, i.e., $D_2(0, 1) = D_2(0, 0)$ with probability 1.

or, the identification of Proposition 4 can be applied. Therefore, the estimable parameters are $\delta_1^{m_1}(0)$ and $\theta_2^{m_1}(0)$ for designs 2 and 3.

5.1.3 Simulation Results

Table 5 presents the simulation results for Design 4. The first two columns use actual propensity scores (corresponding to the true γ s), while the last two columns estimate propensity scores in the first stage. Probit and Linear refer to estimating the optimal instrument ($P_i^{m_1}(\mathbf{T})$) using the probit model and linear probability model, respectively. MSE, MAE, and Cov. Rate represent mean squared error, mean absolute error, and (minimum) 95% coverage rate, respectively, calculated as follows:

$$\begin{aligned} \text{MSE} &= \frac{1}{B} \sum_{b=1}^B \|\hat{\beta}_b - \beta_0\|^2, \\ \text{MAE} &= \frac{1}{4B} \sum_{b=1}^B \sum_{k=1}^4 |\hat{\beta}_{kb} - \beta_{0k}|, \\ \text{Cov. Rate}_k &= \sum_{b=1}^B \mathbb{1} \left\{ \hat{\beta}_{kb} - c\hat{se}(\hat{\beta}_{kb}) \leq \beta_{0k} \leq \hat{\beta}_{kb} + c\hat{se}(\hat{\beta}_{kb}) \right\}, \end{aligned} \tag{15}$$

where $\hat{\beta}_b = (\hat{\beta}_{1b}, \dots, \hat{\beta}_{4b})$ be the estimate in b -th replication, $\beta_0 = (\beta_{01}, \dots, \beta_{04})$ be the true value of parameters, B is the number of replications, and $c = 1.96$. From the design, the actual parameter values are $\delta_1(0) = 20$, $\theta_1(0) = 10$, $\delta_2(0) = 30$, $\theta_2(0) = 15$.

Design 4 is a case where both total monotonicity and one-sided noncompliance do not hold. In this case¹⁴, the mean squared error (MSE) of the estimators decreases in proportion to G^{-1} for all four methods, which verifies Proposition 5. Looking at the coverage rate, inference based on plug-in standard error seems to be valid. It is more efficiently estimated when propensity scores are estimated rather than using actual propensity scores, as the MSE is smaller in the last two columns than in the first two, which aligns with previous findings in the literature (e.g., Hahn (1998), Hirano, Imbens, and Ridder (2003)). Because the estimation of the instrument does not affect the limiting distribution, there would not be a difference between probit and linear models for estimating $P_i^m(\mathbf{T})$, and actually, the results for both methods are very

¹⁴See Tables 14-16 in the appendix for results of Designs 1-3.

similar. However, as the nonlinear model seems slightly more efficient, I report the result of the estimator with first-stage propensity score estimation and a probit model for estimating instruments in all subsequent simulation analyses. Table 6 shows the mean, median, MSE, and coverage rate for each parameter in Design 4.¹⁵ Both the mean and median converge to the actual values.

Table 7 displays the results for four designs, illustrating that the proposed identification and estimators perform well under various restrictions on potential treatment. Designs 2 and 3 represent cases where both total monotonicity (TM) and one-sided noncompliance (OSN) hold. Particularly, Design 3 also satisfies personalized encouragement (PE), where there is no interaction in the treatment take-up decision. In these cases, for the monotone pair \mathbf{m}_1 , the direct effects for unit 1 and indirect effects for unit 2 are identified and estimated using the proposed estimation process corresponding to the moment condition (14). However, in these cases, the estimators corresponding to Proposition 4 are preferred as they do not require additional exclusion restrictions, which will be equivalent to the IV estimator proposed by Vazquez-Bare (2022) (VB estimator hereafter) for the monotone pair \mathbf{m}_1 . On the other hand, for Designs 1 and 4, where total monotonicity or one-sided noncompliance is violated, the VB estimator is no longer valid.

Table 8 shows the biases of two estimators. The estimator denoted by R is the proposed estimator corresponding to the identification in Proposition 2. The estimator denoted by VB is the VB estimator, which is the IV estimator corresponding to the special case identification in Proposition 4. While the R estimator is consistent for all four designs defined above, the VB estimator is consistent only under one-sided noncompliance and total monotonicity. Designs 1 and 4 show the bias of the VB estimator when the assumptions are violated. In Design 1, the bias arises from the violation of one-sided noncompliance, while in Design 4, it reflects bias from the violation of both total monotonicity and one-sided noncompliance. For Designs 2 and 3, both estimators R and VB behave quite similarly, and the biases seem to converge to zero.

¹⁵See Tables 17-18 in the appendix for results of Designs 1-3.

Table 5: Simulation of Design 4

	G	Use true ω		Estimate ω	
		Linear	Probit	Linear	Probit
MSE	2,500	130.61	127.21	93.126	91.105
	5,000	65.611	62.609	45.81	44.147
	10,000	31.503	30.267	22.491	21.978
	20,000	16.401	15.685	11.316	11.01
MAE	2,500	16.825	16.535	14.227	14.017
	5,000	12.025	11.696	9.987	9.783
	10,000	8.325	8.131	7.023	6.917
	20,000	6.019	5.87	4.991	4.907
Cov. Rate	2,500	0.951	0.947	0.95	0.945
	5,000	0.946	0.944	0.946	0.945
	10,000	0.951	0.946	0.949	0.946
	20,000	0.947	0.945	0.948	0.947

notes. This table presents simulation results for $B = 10,000$ replications. The sample sizes are set to $G = 2,500, 5,000, 10,000$ and $20,000$. MSE, MAE, and Cov. Rate denote the mean squared error, mean absolute error, and the average 95% coverage rate computed by formulas (15). The first two columns (“Use true ω ”) use the true propensity score $\Pr(\mathbf{Z}_i = \mathbf{z}|\mathbf{X})$ for the weight ω , while the last two columns (“Estimate ω ”) estimate the propensity score (i.e., estimating γ s) and hence ω .

Table 6: Simulation of Design 4 for each parameter

	G	Design 4			
		$\delta_1(0)$	$\theta_1(0)$	$\delta_2(0)$	$\theta_2(0)$
Mean	2,500	19.825	10.209	30.199	14.806
	5,000	19.925	10.142	30.142	14.927
	10,000	19.941	10.08	30.098	14.929
	20,000	19.97	10.035	30.036	14.971
Median	2,500	19.865	10.271	30.503	14.762
	5,000	19.946	10.176	30.286	14.883
	10,000	19.96	10.074	30.17	14.927
	20,000	19.97	10.081	30.081	14.966
MSE	2,500	5.933	24.216	50.697	10.258
	5,000	2.919	11.841	24.409	4.979
	10,000	1.431	5.895	12.213	2.44
	20,000	0.733	2.94	6.087	1.25
Cov. Rate	2,500	0.953	0.952	0.945	0.953
	5,000	0.948	0.948	0.945	0.947
	10,000	0.951	0.949	0.946	0.951
	20,000	0.951	0.947	0.947	0.948

notes. This table presents simulation results for $B = 10,000$ replications. The sample sizes are set to $G = 2,500, 5,000, 10,000$ and $20,000$. Mean and median are computed over B replications. The means squared error (MSE) for each parameter is computed by

$$\text{MSE}_k = \frac{1}{B} \sum_{b=1}^B (\hat{\beta}_{kb} - \beta_{0k})^2,$$

and the average 95% coverage rate (Cov. Rate) is computed by formula (15). From the design, the actual parameter values are $\delta_1(0) = 20$, $\theta_1(0) = 10$, $\delta_2(0) = 30$, $\theta_2(0) = 15$.

Table 7: Simulation of Designs 1-4

	G	Design 1	Design 2	Design 3	Design 4
MSE	2,500	89.407	2.143	7.009	91.105
	5,000	42.93	1.062	3.494	44.147
	10,000	20.859	0.531	1.844	21.978
	20,000	10.495	0.261	0.884	11.01
MAE	2,500	13.924	1.623	2.951	14.017
	5,000	9.769	1.151	2.093	9.783
	10,000	6.79	0.816	1.512	6.917
	20,000	4.822	0.571	1.049	4.907
Cov. Rate	2,500	0.944	0.944	0.953	0.945
	5,000	0.947	0.946	0.953	0.945
	10,000	0.946	0.948	0.947	0.946
	20,000	0.946	0.95	0.951	0.947

notes. This table presents simulation results for $B = 10,000$ replications. The sample sizes are set to $G = 2,500, 5,000, 10,000$ and $20,000$. MSE, MAE, and Cov. Rate denote the mean squared error, mean absolute error, and the average 95% coverage rate computed by formulas (15).

Table 8: Simulation of designs and bias when (TM), (OSN) are violated

	G	Design 1		Design 4		Design 2		Design 3	
		$\delta_1(0)$	$\theta_2(0)$	$\delta_1(0)$	$\theta_2(0)$	$\delta_1(0)$	$\theta_2(0)$	$\delta_1(0)$	$\theta_2(0)$
R	2,500	-0.014	-0.003	-0.175	-0.194	-0.013	-0.009	-0.016	-0.012
	5,000	-0.016	-0.015	-0.075	-0.073	0.005	0.004	-0.007	-0.004
	10,000	-0.008	-0.009	-0.059	-0.071	-0.009	-0.006	-0.021	-0.015
	20,000	-0.004	-0.003	-0.03	-0.029	-0.002	-0.002	-0.008	-0.006
VB	2,500	4.674	13.998	4.753	14.368	-0.027	-0.021	0.041	0.03
	5,000	4.647	13.954	4.805	14.429	-0.004	-0.003	0.025	0.02
	10,000	4.661	13.974	4.79	14.404	-0.011	-0.008	-0.007	-0.005
	20,000	4.648	13.952	4.794	14.41	-0.004	-0.003	0	-0.001

notes. This table presents simulation results for $B = 10,000$ replications. The sample sizes are set to $G = 2,500, 5,000, 10,000$ and $20,000$. R denotes the proposed estimator using the additional exclusion restriction \mathbf{T} , and VB denotes the IV estimator proposed by [Vazquez-Bare \(2022\)](#), with inverse propensity score weighting to account for the conditioning covariates. Each table reports biases computed by:

$$\text{bias}_k = \frac{1}{B} \sum_{b=1}^B \hat{\beta}_{kb} - \beta_{0k}.$$

To compare estimates for the two estimators, only direct effects for unit 1 ($\delta_1(0)$) and indirect effects for unit 2 ($\theta_2(0)$) are reported. From the design, the actual parameter values are $\delta_1(0) = 20$ and $\theta_2(0) = 15$. Designs 2 and 3 satisfy both total monotonicity (TM) and one-sided noncompliance (OSN). Design 1 only satisfies (TM). Neither condition is satisfied in Design 4.

6 Empirical Illustration

In this section, I demonstrate a simple empirical analysis using the proposed estimation method. The data used here are from the study conducted by [Dupas, Keats, and Robinson \(2019\)](#), which involved a randomized experiment in the rural area of Kenya’s Busia District in the Western province from 2009 to 2012.

In the sampled areas, banks are primarily located in major towns, limiting access to banking services for individuals. Additionally, opening a savings account incurred costs, leading the majority of individuals to not have savings accounts at the start of the experiment, with most people keeping cash at home. The experiment aimed to assess the impact of providing a savings account on their economic behaviors. The main findings in [Dupas, Keats, and Robinson \(2019\)](#) indicate that access to savings accounts makes households less dependent on others in their financial network. The sample consists of 885 households, including 399 female-headed and 486 dual-headed households. In this section, only dual-headed households are used to consider the interactions between spouses.

In the experiment, the treatment involves providing a non-transferable voucher for opening a free savings account. These vouchers were distributed randomly, conditional on each region around market centers (3 markets) and occupation. Specifically, randomization occurred at the individual level, resulting in 17% of households receiving no vouchers, 33% receiving vouchers for both individuals, 26% for females only, and 24% for males only. Individuals who received the vouchers could open an account for free and had the option to open a joint account with their spouse. This means that one-sided noncompliance is not satisfied in this experiment because individual can open a joint account when their spouse has the voucher. The redemption rate for the vouchers was 69%, with 5% being opened for joint accounts.¹⁶

Let individual 1 and 2 denote the female and male household head, respectively. Table 9 shows the relationship between treatment assignment (\mathbf{Z}) and take-up (\mathbf{D}) in round 3¹⁷. Treatment take-up is defined as 1 if the individual opened a savings

¹⁶If we define individuals who satisfy $D(0, 1) > D(0, 0)$ as social compliers, the probability that their spouse is a social complier is around 5%

¹⁷Round 1 is the pre-treatment period, and in round 2, most individuals had not yet opened an account. Therefore, I assume round 3 as the post-treatment period.

account by redeeming the voucher, and 0 if they didn't¹⁸. Then, by construction of the treatment take-up, this experiment satisfies weak one-sided noncompliance, as if both spouses do not have the voucher, then neither of them can open the account, as seen in Table 9.

Table 9: Treatment Assignments and Take-up

Female	Male	# of HH	Female		Male	
			Do not take	Take	Do not take	Take
Treatment	Treatment	106	39	67	35	71
Treatment	Control	77	28	49	74	3
Control	Treatment	70	69	1	22	48
Control	Control	48	48	0	48	0
Total		301	184	117	179	122

notes. Treatment assignments and take-up at round 3.

Because weak one-sided noncompliance holds, we have two monotone pairs $\mathbf{m}_1 = ((1, 0), (0, 0), (1, 1))$ and $\mathbf{m}_2 = ((0, 1), (0, 0), (1, 1))$ ¹⁹. Similar to the classification of compliance types in Table 2, define each individual as a *complier* if $D_i(1, 0) \geq D(0, 0)$ with probability 1, and a *social complier* if $D_i(0, 1) \geq D_i(0, 0)$ with probability 1, conditionally. Then, if the female is a \mathbf{m}_1 -complier, it means she is a complier, and if the male is a \mathbf{m}_1 -complier, then he is a social complier. It is the opposite in the case of \mathbf{m}_2 . Therefore, the \mathbf{m}_1 -direct effect for the female represents the direct local average treatment effect when she is a complier, and the \mathbf{m}_1 -indirect effect represents the indirect local average effect when her spouse is a social complier. The similar interpretation applies to \mathbf{m}_2 . Table 10 shows the mean of estimated probability of being each \mathbf{m} -compliance types.

¹⁸Individuals who did not receive vouchers could still open an account, so the experiment does not enforce $D(0, 0) = 0$. However, when $Z_1 = Z_2 = 0$, the take-up for both females and males is very low, with 3 and 8, respectively. Also, there is a cost associated with opening and maintaining an account, and initially, there were very few account holders at the start of the experiment.

¹⁹i.e., it assumes $D(1, 0) \geq D(0, 0)$ and $D(0, 1) \geq D(0, 0)$ for both individuals conditionally with probability 1.

Table 10: Distribution of Compliance Types

	\mathbf{m}_1		\mathbf{m}_2	
	Complier	Never-Taker	Complier	Never-Taker
Female	0.58	0.42	0.02	0.98
Male	0.03	0.97	0.71	0.29

notes. This table computes the probability of unit i being a \mathbf{m} -complier and \mathbf{m} -never taker, i.e., $P_i = \Pr(K_i^{\mathbf{m}} = 1)$. Because the experiment satisfies weak one-sided noncompliance, $D_1(0, 0) = D_2(0, 0) = 0$ with probability 1, and hence there are no \mathbf{m} -always takers for both monotone pairs \mathbf{m}_1 and \mathbf{m}_2 .

For individual-level outcomes, the authors found that providing the vouchers has a significant positive intention-to-treatment effect on making deposits and withdrawals. Hence, I use these two extensive margin responses as outcome variables in this application. The authors also pointed out that the values of animals and durable goods significantly determine treatment take-up by using regression analysis. Table 11 replicates the regressions for treatment take-ups and individual-level outcomes. It also shows that the values of animals and durable goods (“value” in Table 11) are significant determinants of opening a savings account. Additionally, I found that participation in a qualitative survey (“sq_s” in Table 11), conducted after round 1, and the housing index (“h-index” in Table 11), which indicates if the walls are cement or the roofs are iron or the floors are cement, also determine treatment take-up but do not affect the outcomes of interest. These variables are used as exclusion restrictions for treatment take-up (and hence the exclusion restriction of the distribution of \mathbf{m} -compliers).

Tables 12 and 13 present the estimation of direct and indirect local average treatment effects corresponding to each monotone pair, \mathbf{m}_1 and \mathbf{m}_2 . The columns labeled “VB” use the VB estimator, which is the IV estimator corresponding to the identification in Proposition 4 of the first special case. For the monotone pair \mathbf{m}_1 , the direct effect for unit 1 and the indirect effect for unit 2 are estimable, while it is opposite for the monotone pair \mathbf{m}_2 . The column labeled “R” uses the IV estimator proposed in Section 4 using additional exclusion restrictions as listed above. Age, education, market center, and mobile money usage for both males and females are used as covariates.

The identification in Proposition 4 is valid under a one-sided noncompliance situa-

Table 11: Determinants of Treatment Take-up

Variable	D_1	D_2	D_1D_2	Deposit ₁	Deposit ₂	Withdrawal ₁	Withdrawal ₂
sqs ₁	-0.052 (0.031)	0.068* (0.032)	0.025 (0.026)	-0.002 (0.017)	0.005 (0.019)	-0.003 (0.007)	-0.007 (0.012)
sqs ₂	0.035 (0.031)	0.15*** (0.032)	0.057* (0.025)	-0.008 (0.017)	0.006 (0.019)	0.013 (0.007)	0.01 (0.012)
sqs ₁ × sqs ₂	0.102* (0.044)	-0.253*** (0.046)	-0.055 (0.036)	0.023 (0.024)	0.008 (0.027)	-0.007 (0.01)	0.002 (0.017)
h-index	-0.212 (0.045)	-0.103* (0.047)	-0.123*** (0.037)	0.009 (0.025)	0.044 (0.028)	-0.007 (0.01)	0.024 (0.017)
value	0.02 (0.012)	0.034** (0.013)	0.029** (0.01)	0.009 (0.007)	0.008 (0.008)	0.002 (0.003)	0.004 (0.005)
N	1787	1787	1787	1787	1787	1787	1787

notes. Control variables consist of age, education, regions, indicators of members in ROSCA, indicators of using mobile money, household size, round-fixed effects, and the dependent variable at the baseline period (last 4 columns only) for both the female and male head at the baseline period. *sqs* is the indicator of participation in the qualitative survey conducted after round 1. *h-index* indicates the type of residence. *value* represents the value of animals and durable goods. Standard errors are reported in parentheses. *, **, *** denote the significance levels at 10%, 5%, and 1%, respectively.

tion. Therefore, the VB estimation would be biased as one-sided noncompliance does not hold in the experiment. However, as only about 5% opened the joint account, the extent of violation of one-sided noncompliance is small. This results in both estimations being similar in Tables 12 and 13. However, VB estimation actually estimates some weighted average of other parameters.²⁰

For the comparison of \mathbf{m}_1 , the effects for females on both variables primarily stem from direct effects, and the effects are 9% and 6% for making deposits and making withdrawals, respectively. For \mathbf{m}_2 -effects, making deposits of male heads is significantly estimated with a 12% increase from the treatment, while none of the effects are significant in making withdrawals. For \mathbf{m}_1 , the indirect effect of male heads making deposits is negative. This can be interpreted as if their wife opens an account, the male head reduces their deposits. One possible scenario to explain this is that the husbands transfer bank deposits and withdrawals to their wife's bank once their wife opens a free

²⁰See Appendix A.4 for details.

account, because of the free maintenance fee.

Table 12: Make Deposit

Unit	Effects	m_1		m_2	
		R	VB	R	VB
Female	Direct	0.09** (0.04)	0.11*** (0.04)	-0.42 (0.33)	
	Indirect	0.34 (0.47)		0.04 (0.03)	0.03 (0.03)
Male	Direct	1.49** (0.75)		0.12*** (0.05)	0.13*** (0.05)
	Indirect	-0.09* (0.05)	0.01 (0.04)	-0.09 (0.53)	

notes. The dependent variable is 1 if the individual made at least one deposit. Control variables consist of age, education, regions, indicators of members in ROSCA, and indicators of using mobile money for both the female and male head at the baseline period. “Direct” denotes the local average treatment effects $E[Y_i(1,0) - Y_i(0,0)|K_i^m = 1]$, and “Indirect” denotes $E[Y_i(0,1) - Y_i(0,0)|K_j^m = 1]$. Plug-in clustered standard errors are reported in parentheses. *, **, *** denote the significance levels at 10%, 5%, and 1%, respectively.

Table 13: Make Withdrawal

Unit	Effects	m_1		m_2	
		R	VB	R	VB
Female	Direct	0.06** (0.03)	0.05* (0.02)	0.27 (0.23)	
	Indirect	-0.32 (0.21)		0.003 (0)	0.01 (0.01)
Male	Direct	0.33 (0.4)		0.04 (0.02)	0.04 (0.03)
	Indirect	-0.003 (0.04)	0.003 (0.03)	0.1 (0.54)	

notes. The dependent variable is 1 if the individual made at least one withdrawal. Control variables consist of age, education, regions, indicators of members in ROSCA, and indicators of using mobile money for both the female and male head at the baseline period. “Direct” denotes the local average treatment effects $E[Y_i(1,0) - Y_i(0,0)|K_i^m = 1]$, and “Indirect” denotes $E[Y_i(0,1) - Y_i(0,0)|K_j^m = 1]$. Plug-in clustered standard errors are reported in parentheses. *, **, *** denote the significance levels at 10%, 5%, and 1%, respectively.

7 Conclusion

This study analyzed the identification and estimation of causal effects in situations where units interact with imperfect compliance. In such scenarios, the conventional approach is to restrict the distribution of potential treatment, such as by imposing some form of monotonicity or assuming one-sided noncompliance. I assumed a weak concept of monotonicity that imposes only an almost sure partial ordering on the set of all potential treatments, instead of imposing a total ordering (referred to as total monotonicity in this article) on the set. It allows us to consider the other restrictions used in the literature within a single framework. Furthermore, I showed that if there exist additional exclusion restrictions, the direct and indirect local average treatment effects are identified without assuming one-sided noncompliance and total monotonicity. This additional instrument needs to determine the compliance types exclusively but does not directly affect the potential outcomes. I proposed a two-stage estimation procedure for the identified parameters and verified the performance using Monte Carlo simulations. The bias of the existing estimator when the underlying assumptions are violated was also assessed in the simulation analysis. Additionally, I used a real-world dataset from a randomized experiment by [Dupas, Keats, and Robinson \(2019\)](#) to illustrate the proposed method.

References

- Angrist, Joshua D, Guido W Imbens, and Donald B Rubin (1996). “Identification of causal effects using instrumental variables”. In: *Journal of the American statistical Association* 91.434, pp. 444–455.
- Blackwell, Matthew (2017). “Instrumental variable methods for conditional effects and causal interaction in voter mobilization experiments”. In: *Journal of the American Statistical Association* 112.518, pp. 590–599.
- DiTraglia, Francis J, Camilo García-Jimeno, Rossa O’Keeffe-O’Donovan, and Alejandro Sánchez-Becerra (2023). “Identifying causal effects in experiments with spillovers and non-compliance”. In: *Journal of Econometrics*.
- Dupas, Pascaline, Anthony Keats, and Jonathan Robinson (2019). “The effect of savings accounts on interpersonal financial relationships: Evidence from a field experiment in rural Kenya”. In: *The Economic Journal* 129.617, pp. 273–310.
- Hahn, Jinyong (1998). “On the role of the propensity score in efficient semiparametric estimation of average treatment effects”. In: *Econometrica*, pp. 315–331.
- Hirano, Keisuke, Guido W Imbens, and Geert Ridder (2003). “Efficient estimation of average treatment effects using the estimated propensity score”. In: *Econometrica* 71.4, pp. 1161–1189.
- Hoshino, Tadao and Takahide Yanagi (2023). “Causal inference with noncompliance and unknown interference”. In: *Journal of the American Statistical Association*, pp. 1–12.
- Imbens, Guido and Joshua Angrist (1994). “Identification and Estimation of Local Average Treatment Effects”. In: *Econometrica* 62.2, pp. 467–475.
- Imbens, Guido W and Donald B Rubin (1997). “Estimating outcome distributions for compliers in instrumental variables models”. In: *The Review of Economic Studies* 64.4, pp. 555–574.
- Kang, Hyunseung and Guido Imbens (2016). “Peer encouragement designs in causal inference with partial interference and identification of local average network effects”. In: *arXiv preprint arXiv:1609.04464*.
- Kormos, Mate, Robert P Lieli, and Martin Huber (2023). “Treatment Effect Analysis for Pairs with Endogenous Treatment Takeup”. In: *arXiv preprint arXiv:2301.04876*.

Vazquez-Bare, Gonzalo (2022). “Identification and estimation of spillover effects in randomized experiments”. In: *Journal of Econometrics*.

A Appendix

A.1 Proofs

Proof of Proposition 1. Let $\mathbf{m} = (\mathbf{z}, \mathbf{z}', \mathbf{r})$ be a monotone pair. Then, $K_i^{\mathbf{m}} = r_i(D_i(\mathbf{z}_i) - D_i(\mathbf{z}'_i)) \in \{0, 1\}$, and hence

$$\begin{aligned} P_i^{\mathbf{m}}(\mathbf{X}) &= E[K_i^{\mathbf{m}} | \mathbf{X}] \\ &= r_i E[D_i(\mathbf{z}_i) | \mathbf{X}] - r_i E[D_i(\mathbf{z}'_i) | \mathbf{X}] \\ &= r_i E[D_i | \mathbf{Z}_i = \mathbf{z}_i, \mathbf{X}] - r_i E[D_i | \mathbf{Z}_i = \mathbf{z}'_i, \mathbf{X}], \end{aligned}$$

since potential treatment take-up indicators are independent of \mathbf{Z}_i conditional on \mathbf{X} by Assumption 1. In addition, we have the expression (9) with the weight (10) from the fact that

$$E[\mathbb{1}\{\mathbf{Z}_i = \mathbf{z}\} D_i | \mathbf{X}] = E[D_i | \mathbf{X}] \Pr(\mathbf{Z}_i = \mathbf{z} | \mathbf{X}),$$

for any $\mathbf{z} \in \{0, 1\}$. □

Proof of Lemma 1. From equation (4), we have

$$Y_i = Y_i(0, 0) + \Delta_i Y_i(0) D_i + \Delta_j Y_i(0) D_j + (\Delta_i Y_i(1) - \Delta_i Y_i(0)) D_i D_j.$$

Then, Assumption 1 implies that for any $\mathbf{z} = (z_1, z_2) \in \{0, 1\}$ and $\mathbf{z}_i = (z_i, z_{3-i})$ for $i \in \{1, 2\}$, we have

$$\begin{aligned} E[Y_i | \mathbf{Z}_i = \mathbf{z}_i, \mathbf{X}] &= E[Y_i(0, 0) | \mathbf{Z}_i = \mathbf{z}_i, \mathbf{X}] \\ &\quad + E[\Delta_i Y_i(0) D_i | \mathbf{Z}_i = \mathbf{z}_i, \mathbf{X}] + E[\Delta_j Y_i(0) D_j | \mathbf{Z}_j = \mathbf{z}_j, \mathbf{X}] \\ &\quad + E[(\Delta_i Y_i(1) - \Delta_i Y_i(0)) D_i D_j | \mathbf{Z}_i = \mathbf{z}_i, \mathbf{X}] \\ &= E[Y_i(0, 0) | \mathbf{X}] \\ &\quad + E[\Delta_i Y_i(0) D_i(\mathbf{z}_i) | \mathbf{X}] + E[\Delta_j Y_i(0) D_j(\mathbf{z}_j) | \mathbf{X}] \\ &\quad + E[(\Delta_i Y_i(1) - \Delta_i Y_i(0)) D_i(\mathbf{z}_i) D_j(\mathbf{z}_j) | \mathbf{X}]. \end{aligned}$$

Next, let $\mathbf{m} = (\mathbf{z}, \mathbf{z}', \mathbf{r})$ be a monotone pair. Because $r_i \in \{-1, 1\}$, $r_i^2 = 1$. Therefore,

$$\begin{aligned} E[Y_i | \mathbf{Z}_i = \mathbf{z}_i, \mathbf{X}] - E[Y_i | \mathbf{Z}_i = \mathbf{z}'_i, \mathbf{X}] &= E[\Delta_i Y_i(0) (D_i(\mathbf{z}_i) - D_i(\mathbf{z}'_i)) | \mathbf{X}] + E[\Delta_j Y_i(0) (D_j(\mathbf{z}_j) - D_j(\mathbf{z}'_j)) | \mathbf{X}] \\ &\quad + E[(\Delta_i Y_i(1) - \Delta_i Y_i(0)) (D_i(\mathbf{z}_i) D_j(\mathbf{z}_j) - D_i(\mathbf{z}'_i) D_j(\mathbf{z}'_j)) | \mathbf{X}]. \\ &= r_i E[\Delta_i Y_i(0) r_i (D_i(\mathbf{z}_i) - D_i(\mathbf{z}'_i)) | \mathbf{X}] + r_j E[\Delta_j Y_i(0) r_j (D_j(\mathbf{z}_j) - D_j(\mathbf{z}'_j)) | \mathbf{X}] \\ &\quad + r_i r_j E[(\Delta_i Y_i(1) - \Delta_i Y_i(0)) (r_i D_i(\mathbf{z}_i) r_j D_j(\mathbf{z}_j) - r_i D_i(\mathbf{z}'_i) r_j D_j(\mathbf{z}'_j)) | \mathbf{X}]. \end{aligned}$$

Thus, we have equation (7) with $K_i^{\mathbf{m}} = r_i (D_i(\mathbf{z}_i) - D_i(\mathbf{z}'_i))$ and $K_{ij}^{\mathbf{m}} = r_i D_i(\mathbf{z}_i) r_j D_j(\mathbf{z}_j) - r_i D_i(\mathbf{z}'_i) r_j D_j(\mathbf{z}'_j)$. \square

Proof of Proposition 4. Note that $K_{ij}^m = 1$ if and only if

- i, j are ***m***-compliers, or
- i is ***m***-complier, and j is ***m***-always taker.

Therefore, if one individual is ***m***-never taker

Let $\mathbf{m} = (\mathbf{z}, \mathbf{z}', \mathbf{r})$ be a monotone pair. Suppose $j \in \{1, 2\}$ is ***m***-never taker conditional on \mathbf{X} , i.e., $D_j(\mathbf{z}_j) = D_j(\mathbf{z}'_j) = 0$, conditional on X with probability 1. Then, $K_{ij}^m = K_j^m = 0$ conditional on \mathbf{X} with probability 1. Thus, equation (7) becomes

$$\begin{aligned} E[ITT_i^m(\mathbf{X})] &= r_i E[E[K_i^m \Delta_i Y_i(0) | \mathbf{X}]] \\ &= r_i E[K_i^m \Delta_i Y_i(0)] \\ &= r_i E[\Delta_i Y_i(0)] \Pr(K_i^m = 1). \end{aligned}$$

Next, suppose $j \in \{1, 2\}$ is ***m***-always taker conditional on \mathbf{X} , i.e., $D_j(\mathbf{z}_j) = D_j(\mathbf{z}'_j) = 1$, conditional on X with probability 1. Then, $K_{ij}^m = K_i^m$, and $K_j^m = 0$ conditional on \mathbf{X} with probability 1. Thus, by taking $r_j = 1$, equation (7) becomes

$$\begin{aligned} E[ITT_i^m(\mathbf{X})] &= E[r_i E[K_i^m \Delta_i Y_i(0) | \mathbf{X}] + r_i E[K_i^m (\Delta_i Y_i(1) - \Delta_i Y_i(0)) | \mathbf{X}]] \\ &= r_i E[K_i^m \Delta_i Y_i(1)] \\ &= r_i E[\Delta_i Y_i(1)] \Pr(K_i^m = 1). \end{aligned}$$

By the same argument for θ , we have the results, provided that $P_i^m > 0$ for $i \in \{1, 2\}$. \square

Proof of Proposition 2. The first part of Assumption ?? implies

$$E \left[|K_{ij}^m(\Delta_i Y_i(1) - \Delta_i Y_i(0))| | \mathbf{X} \right] \leq E [|\Delta_i Y_i(1) - \Delta_i Y_i(0)| | \mathbf{X}] = 0,$$

with probability 1. By Lemma 1, we have

$$ITT_i^m(\mathbf{X}) = r_i E[K_i^m \Delta_i Y_i(0) | \mathbf{X}] + r_j E[K_j^m \Delta_j Y_i(0) | \mathbf{X}],$$

and by law of iterative expectation and the second part of Assumption ??,

$$\begin{aligned} E[ITT_i^m(\mathbf{X}) | \mathbf{T}] &= r_i E[K_i^m \Delta_i Y_i(0) | \mathbf{T}] + r_j E[K_j^m \Delta_j Y_i(0) | \mathbf{T}], \\ &= r_i E[\Delta_i Y_i(0) | K_i^m = 1, \mathbf{T}] \Pr(K_i^m | \mathbf{T}) + r_j E[\Delta_j Y_i(0) | K_j^m = 1, \mathbf{T}] \Pr(K_j^m | \mathbf{T}), \\ &= r_i E[\Delta_i Y_i(0) | K_i^m = 1] \Pr(K_i^m | \mathbf{T}) + r_j E[\Delta_j Y_j(0) | K_i^m = 1] \Pr(K_j^m | \mathbf{T}). \end{aligned}$$

□

Proof of Lemma 3. Let \mathbf{W} be a subset of the set of exogenous variables \mathbf{X} , $i \in \{1, 2\}$, and $j = 3 - i$. Note that because potential treatment statuses are binary random variables, $D_i(\mathbf{z}_i) \leq 1$ with probability 1. Therefore,

$$D_i(\mathbf{z}_i)D_j(\mathbf{z}_j) \leq D_j(\mathbf{z}_j), \quad (16)$$

with probability 1. Suppose $D_i(\mathbf{z}_i) \geq D_j(\mathbf{z}_j)$ with probability 1. Then,

$$D_i(\mathbf{z}_i)D_j(\mathbf{z}_j) \geq D_j(\mathbf{z}_j), \quad (17)$$

with probability 1, and the inequality holds with equality by (16). Conversely, suppose (17) holds with probability 1, and suppose $D_i(\mathbf{z}_i) < D_j(\mathbf{z}_j)$ with positive probability. In other words, the event $E = \{D_i(\mathbf{z}_i) = 0, D_j(\mathbf{z}_j) = 1\}$ occurs with positive probability. This contradicts to (17), since the event E implies $1 = D_j(\mathbf{z}_j) \neq D_i(\mathbf{z}_i)D_j(\mathbf{z}_j) = 0$, hence the event $D_j(\mathbf{z}_j) \neq D_i(\mathbf{z}_i)D_j(\mathbf{z}_j)$ occurs with positive probability. The desired result is from applying the above argument conditional on \mathbf{W} . \square

Lemma 4. Let \mathbf{V}_g be a random vector whose support is \mathcal{V} and $\ell : \mathcal{V} \times \Phi \rightarrow \mathbb{R}^M$ be a vector of real valued functions that is integrable with respect to the distribution of \mathbf{V}_g at each point $\phi \in \Phi \subset \mathbb{R}^K$.

$$L_G(\phi) = \frac{1}{G} \sum_{g=1}^G \ell(\mathbf{V}_g, \phi), \quad L(\phi) = E[\ell(\mathbf{V}_g, \phi)].$$

Suppose

- (a) $\{\mathbf{V}_g\}$ is independently and identically distributed.
- (b) $\hat{\phi} \xrightarrow{p} \phi_0$, ϕ_0 is a interior point in Φ .
- (c) $\ell(\mathbf{v}, \phi)$ is continuous at ϕ_0 for all $\mathbf{v} \in \mathcal{V}$.
- (d) For some compact neighborhood \mathcal{N} of ϕ_0 , $E \left[\sup_{\phi \in \mathcal{N}} \|\ell(\mathbf{V}_g, \phi)\| \right] < \infty$.

Then, $L(\phi)$ is continuous at ϕ_0 and $L_G(\hat{\phi}) \xrightarrow{p} L(\phi_0)$.

Proof. Let $\{\phi_n\} \rightarrow \phi_0$ and a neighborhood \mathcal{N} of ϕ_0 satisfying (d) be given. Then,

$$\|\ell(v, \phi_n)\| \leq \sup_{\phi \in \mathcal{N}} \|\ell(v, \phi)\| =: g(v),$$

for all but finite number of n , and $g(v)$ is integrable by (d). Thus, by dominated convergence theorem, we have $\{E[\ell(\mathbf{V}_g, \phi_n)]\} \rightarrow E[\ell(\mathbf{V}_g, \phi_0)]$, which implies continuity of $L(\phi)$ at ϕ_0 . Also, we have $L_G(\phi) \xrightarrow{p} L(\phi)$ uniformly on \mathcal{N} from (a), (d) and the uniform weak law of large number. It follows that for any $\varepsilon > 0$,

$$\begin{aligned} & \Pr \left(\|L_G(\hat{\phi}) - L(\phi_0)\| > \varepsilon \right) \\ & \leq \Pr \left(\|L_G(\hat{\phi}) - L(\hat{\phi})\| > \frac{\varepsilon}{2}, \hat{\phi} \in \mathcal{N} \right) + \Pr \left(\hat{\phi} \notin \mathcal{N} \right) + \Pr \left(\|L(\hat{\phi}) - L(\phi_0)\| > \frac{\varepsilon}{2} \right) \\ & = \Pr \left(\sup_{\phi \in \mathcal{N}} \|L_G(\phi) - L(\phi)\| > \frac{\varepsilon}{2} \right) + \Pr \left(\hat{\phi} \notin \mathcal{N} \right) + \Pr \left(\|L(\hat{\phi}) - L(\phi_0)\| > \frac{\varepsilon}{2} \right). \end{aligned}$$

All three terms converge to zero since uniform convergence of L_G to L over \mathcal{N} , consistency of $\hat{\phi}$, and continuous mapping theorem, respectively. \square

Proof of Proposition 5. Let $\mathbf{m} = (\mathbf{z}, \mathbf{z}', \mathbf{r})$ be a monotone pair. Recall that

$$\begin{aligned}\tilde{\mathbf{D}}_g &= \begin{pmatrix} D_{1g} & D_{2g} & 0 & 0 \\ 0 & 0 & D_{2g} & D_{1g} \end{pmatrix}, \\ \mathbf{P}(\mathbf{T}_g, \phi) &= \begin{pmatrix} P_1(\mathbf{T}_g, \phi) & P_2(\mathbf{T}_g, \phi) & 0 & 0 \\ 0 & 0 & P_2(\mathbf{T}_g, \phi) & P_1(\mathbf{T}_g, \phi) \end{pmatrix},\end{aligned}$$

Because all (random) vectors (matrices) are finite dimensional, every norm should be equivalent. Let $\|\cdot\|$ denote Frobenius norm. Then,

$$\begin{aligned}\|\mathbf{P}(\mathbf{T}_g, \phi)\| &= \sqrt{2(P_1^2(\mathbf{T}_g, \phi) + P_2^2(\mathbf{T}_g, \phi))} \leq 2, \\ \|\mathbf{D}\| &= \sqrt{2(D_{1g} + D_{2g})} \leq 2,\end{aligned}$$

For the compact neighborhood \mathcal{N}_γ satisfying Assumption 5-??,

$$\sup_{\gamma \in \mathcal{N}_\gamma} |\omega(\mathbf{Z}_g, \mathbf{X}_g, \gamma)| = \sup_{\gamma \in \mathcal{N}_\gamma} \left| \frac{\mathbb{1}\{\mathbf{Z}_g = \mathbf{z}\}}{q(\mathbf{z}, \mathbf{X}_g, \gamma)} - \frac{\mathbb{1}\{\mathbf{Z}_g = \mathbf{z}'\}}{q(\mathbf{z}', \mathbf{X}_g, \gamma)} \right| \leq \frac{2}{q_0}.$$

Next, let $\lambda_{\max}(A), \lambda_{\min}(A)$ be maximum, minimum eigenvalue of a square matrix A , respectively. Then, for the compact neighborhood \mathcal{N}_ϕ satisfying Assumption 5-??,

$$\begin{aligned}\sup_{\phi \in \mathcal{N}_\phi} \|\mathbf{S}^{-1}(\mathbf{T}_g, \phi)\| &\leq \sqrt{2} \sup_{\phi \in \mathcal{N}_\phi} \|\mathbf{S}^{-1}(\mathbf{T}_g, \phi)\|_2 \\ &\leq \sqrt{2} \sup_{\phi \in \mathcal{N}_\phi} \lambda_{\max}(\mathbf{S}^{-1}(\mathbf{T}_g, \phi)), \\ &= \sqrt{2} \sup_{\phi \in \mathcal{N}_\phi} \lambda_{\min}(\mathbf{S}(\mathbf{T}_g, \phi))^{-1}, \\ &= \sqrt{2} \left(\inf_{\phi \in \mathcal{N}_\phi} \lambda_{\min}(\mathbf{S}(\mathbf{T}_g, \phi)) \right)^{-1} \leq \frac{\sqrt{2}}{\lambda_0},\end{aligned}$$

with probability 1, where $\|\cdot\|_2$ is the spectral norm, the maximum eigenvalue of the

matrix. Thus, for a compact neighborhood $\mathcal{N} = \mathcal{N}_\gamma \times \mathcal{N}_\phi$ of (ϕ_0, γ_0) , we have

$$\begin{aligned}
& \sup_{(\phi, \gamma) \in \mathcal{N}} \|R(\mathbf{T}_g, \phi) \omega(\mathbf{Z}_g, \mathbf{X}_g, \gamma) \mathbf{D}_g\| \\
&= \sup_{(\phi, \gamma) \in \mathcal{N}} \left\| \mathbf{P}(\mathbf{T}_g, \phi)' \mathbf{S}^{-1}(\mathbf{V}_g, \phi) \omega(\mathbf{Z}_g, \mathbf{X}_g, \gamma) \tilde{\mathbf{D}}_g \right\| \\
&\leq \sup_{\phi \in \mathcal{N}_\phi} \|\mathbf{P}(\mathbf{T}_g, \phi)\| \sup_{\phi \in \mathcal{N}_\phi} \left\| \mathbf{S}^{-1}(\mathbf{V}_g, \phi) \right\| \sup_{\gamma \in \mathcal{N}_\gamma} |\omega(\mathbf{Z}_g, \mathbf{X}_g, \gamma)| \|\tilde{\mathbf{D}}_g\| \\
&\leq \frac{8\sqrt{2}}{\lambda_0 q_0} < \infty, \\
& \sup_{(\phi, \gamma) \in \mathcal{N}} \|R(\mathbf{T}_g, \phi) \omega(\mathbf{Z}_g, \mathbf{X}_g, \gamma) \mathbf{Y}_g\| \\
&= \sup_{(\phi, \gamma) \in \mathcal{N}} \left\| \mathbf{P}(\mathbf{T}_g, \phi)' \mathbf{S}^{-1}(\mathbf{V}_g, \phi) \omega(\mathbf{Z}_g, \mathbf{X}_g, \gamma) \mathbf{Y}_g \right\| \\
&\leq \sup_{\phi \in \mathcal{N}_\phi} \|\mathbf{P}(\mathbf{T}_g, \phi)\| \sup_{\phi \in \mathcal{N}_\phi} \left\| \mathbf{S}^{-1}(\mathbf{V}_g, \phi) \right\| \sup_{\gamma \in \mathcal{N}_\gamma} |\omega(\mathbf{Z}_g, \mathbf{X}_g, \gamma)| \|\mathbf{Y}_g\| \\
&\leq \frac{4\sqrt{2}}{\lambda_0 q_0} \|\mathbf{Y}_g\|
\end{aligned}$$

with probability 1. Thus, we have

$$\begin{aligned}
& E \left[\sup_{(\phi, \gamma) \in \mathcal{N}} \left\| R(\mathbf{T}_g, \phi) \omega(\mathbf{Z}_g, \mathbf{X}_g, \gamma) \tilde{\mathbf{D}}_g \right\| \right] \leq \frac{8\sqrt{2}}{\lambda_0 q_0} < \infty, \\
& E \left[\sup_{(\phi, \gamma) \in \mathcal{N}} \left\| R(\mathbf{T}_g, \phi) \omega(\mathbf{Z}_g, \mathbf{X}_g, \gamma) \mathbf{Y}_g \right\| \right] \leq \frac{4\sqrt{2}}{\lambda_0 q_0} E[\|\mathbf{Y}_g\|] < \infty.
\end{aligned}$$

Thus, by Lemma 4, we have

$$\begin{aligned}
& \frac{1}{G} \sum_{g=1}^G R(\mathbf{T}_g, \hat{\phi}) \omega(\mathbf{Z}_g, \mathbf{X}_g, \hat{\gamma}) \tilde{\mathbf{D}}_g \xrightarrow{p} E[R(\mathbf{T}_g) \omega(\mathbf{Z}_g, \mathbf{X}_g) \tilde{\mathbf{D}}_g] = E[\mathbf{P}(\mathbf{T}_g)' \mathbf{S}^{-1}(\mathbf{T}_g) \mathbf{P}(\mathbf{T}_g)], \\
& \frac{1}{G} \sum_{g=1}^G R(\mathbf{T}_g, \hat{\phi}) \omega(\mathbf{Z}_g, \mathbf{X}_g, \hat{\gamma}) \mathbf{Y}_g \xrightarrow{p} E[R(\mathbf{T}_g) \omega(\mathbf{Z}_g, \mathbf{X}_g) \mathbf{Y}_g] = E[\mathbf{P}(\mathbf{T}_g)' \mathbf{S}^{-1}(\mathbf{T}_g) \mathbf{P}(\mathbf{T}_g)] \beta_0.
\end{aligned}$$

The consistency follows from the Slutsky's theorem.

Since true values of parameters are assumed to be interior points, we have

$$\begin{aligned}
0 &= \frac{1}{\sqrt{G}} \sum_{g=1}^G \mathbf{R}(\mathbf{T}_g, \hat{\phi}) \omega(\mathbf{Z}_g, \mathbf{X}_g, \hat{\gamma}) (\mathbf{Y}_g - \tilde{\mathbf{D}}_g \hat{\beta}) \\
&= \frac{1}{\sqrt{G}} \sum_{g=1}^G \mathbf{R}(\mathbf{T}_g) \omega(\mathbf{Z}_g, \mathbf{X}_g) (\mathbf{Y}_g - \tilde{\mathbf{D}}_g \beta_0) \\
&\quad + \sum_{j=1}^{k_\phi} \frac{1}{G} \sum_{g=1}^G \frac{\partial \mathbf{R}(\mathbf{T}_g, \bar{\phi})}{\partial \phi_j} \omega(\mathbf{Z}_g, \mathbf{X}_g, \bar{\gamma}) (\mathbf{Y}_g - \mathbf{D}_g \bar{\beta}) \sqrt{G} (\hat{\phi}_j - \phi_{j0}) \\
&\quad + \frac{1}{G} \sum_{g=1}^G \mathbf{R}(\mathbf{T}_g, \bar{\phi}) (\mathbf{Y}_g - \mathbf{D}_g \bar{\beta}) \frac{\partial \omega(\mathbf{Z}_g, \mathbf{X}_g, \bar{\gamma})}{\partial \gamma'} \sqrt{G} (\hat{\gamma} - \gamma_0) \\
&\quad - \frac{1}{G} \sum_{g=1}^G \mathbf{R}(\mathbf{T}_g, \bar{\phi}) \omega(\mathbf{Z}_g, \mathbf{X}_g, \bar{\gamma}) \mathbf{D}_g \sqrt{G} (\hat{\beta} - \beta_0).
\end{aligned}$$

Let \mathcal{N}_β be a compact neighborhood of β_0 , and $\sup_{\beta \in \mathcal{N}_\beta} \|\beta\| = B_0 < \infty$. Then,

$$\sup_{\beta \in \mathcal{N}_\beta} \|\mathbf{Y}_g - \tilde{\mathbf{D}}_g \beta\| \leq \|\mathbf{Y}_g\| + \|\tilde{\mathbf{D}}\| \sup_{\beta \in \mathcal{N}_\beta} \|\beta\| \leq \|\mathbf{Y}_g\| + 2B_0.$$

Recall that

$$\sup_{\phi \in \mathcal{N}_\phi} \|\mathbf{S}^{-1}(\mathbf{T}_g, \phi)\| \leq \frac{\sqrt{2}}{\lambda_0}, \quad \sup_{\phi \in \mathcal{N}_\phi} \|\mathbf{P}(\mathbf{T}_g, \phi)\| \leq 2.$$

Thus,

$$\begin{aligned}
&E \left[\sup_{\phi \in \mathcal{N}_\phi} \left\| \frac{\partial \mathbf{R}(\mathbf{T}_g, \phi)}{\partial \phi_j} \right\| \right] \\
&= E \left[\sup_{\phi \in \mathcal{N}_\phi} \left\| \frac{\partial \mathbf{P}(\mathbf{T}_g, \phi)}{\partial \phi_j} \mathbf{S}^{-1}(\mathbf{T}_g, \phi) + \mathbf{P}(\mathbf{T}_g, \phi) \frac{\partial \mathbf{S}^{-1}(\mathbf{T}_g, \phi)}{\partial \phi_j} \right\| \right] \\
&= E \left[\sup_{\phi \in \mathcal{N}_\phi} \left\| \frac{\partial \mathbf{P}(\mathbf{T}_g, \phi)}{\partial \phi_j} \mathbf{S}^{-1}(\mathbf{T}_g, \phi) - \mathbf{P}(\mathbf{T}_g, \phi) \mathbf{S}^{-1}(\mathbf{T}_g, \phi) \frac{\partial \mathbf{S}(\mathbf{T}_g, \phi)}{\partial \phi_j} \mathbf{S}^{-1}(\mathbf{T}_g, \phi) \right\| \right] \\
&\leq \frac{\sqrt{2}}{\lambda_0} E \left[\sup_{\phi \in \mathcal{N}_\phi} \left\| \frac{\partial \mathbf{P}(\mathbf{T}_g, \phi)}{\partial \phi_j} \right\| \right] + \frac{4}{\lambda_0^2} E \left[\sup_{\phi \in \mathcal{N}_\phi} \left\| \frac{\partial \mathbf{S}(\mathbf{T}_g, \phi)}{\partial \phi_j} \right\| \right] < \infty
\end{aligned}$$

Similarly, for the compact neighborhood \mathcal{N}_γ of γ_0 ,

$$\begin{aligned}
& E \left[\sup_{\gamma \in \mathcal{N}_\phi} \left\| \frac{\partial \omega(\mathbf{T}_g, \gamma)}{\partial \gamma'} \right\| \right] \\
&= E \left[\sup_{\phi \in \mathcal{N}_\phi} \left\| -\frac{\mathbb{1}\{\mathbf{Z}_g = \mathbf{z}\}}{q^2(\mathbf{z}, \mathbf{X}_g, \gamma)} \frac{\partial q(\mathbf{z}, \mathbf{X}_g, \gamma)}{\partial \gamma} + \frac{\mathbb{1}\{\mathbf{Z}_g = \mathbf{z}'\}}{q^2(\mathbf{z}', \mathbf{X}_g, \gamma)} \frac{\partial q(\mathbf{z}', \mathbf{X}_g, \gamma)}{\partial \gamma} \right\| \right] \\
&\leq E \left[\frac{1}{q_0^2} \sup_{\phi \in \mathcal{N}_\phi} \left\| \frac{\partial q(\mathbf{z}, \mathbf{X}_g, \gamma)}{\partial \gamma'} \right\| + \frac{1}{q_0^2} \sup_{\phi \in \mathcal{N}_\phi} \left\| \frac{\partial q(\mathbf{z}', \mathbf{X}_g, \gamma)}{\partial \gamma'} \right\| \right] \\
&\leq \frac{2}{q_0^2} \max \left\{ E \left[\sup_{\phi \in \mathcal{N}_\phi} \left\| \frac{\partial q(\mathbf{z}, \mathbf{X}_g, \gamma)}{\partial \gamma'} \right\| \right], E \left[\sup_{\phi \in \mathcal{N}_\phi} \left\| \frac{\partial q(\mathbf{z}', \mathbf{X}_g, \gamma)}{\partial \gamma'} \right\| \right] \right\} < \infty
\end{aligned}$$

Therefore, we have a compact neighborhood $\mathcal{M} = \mathcal{N}_\phi \times \mathcal{N}_\gamma \times \mathcal{N}_\beta$ in that

$$\begin{aligned}
& E \left[\sup_{(\phi, \gamma, \beta) \in \mathcal{M}} \left\| \frac{\partial \mathbf{R}(\mathbf{T}_g, \phi)}{\partial \phi_j} \omega(\mathbf{Z}_g, \mathbf{X}_g, \gamma) (\mathbf{Y}_g - \mathbf{D}_g \beta) \right\| \right] \\
&\leq E \left[\sup_{\phi \in \mathcal{N}_\phi} \left\| \frac{\partial \mathbf{R}(\mathbf{T}_g, \phi)}{\partial \phi_j} \right\|^2 \right]^{\frac{1}{2}} E \left[\sup_{\gamma \in \mathcal{N}_\gamma} \|\omega(\mathbf{Z}_g, \mathbf{X}_g, \gamma)\|^4 \right]^{\frac{1}{4}} E \left[\sup_{\beta \in \mathcal{N}_\beta} \|\mathbf{Y}_g - \mathbf{D}_g \beta\|^4 \right]^{\frac{1}{4}} \\
&\leq \left\{ \frac{2}{\lambda_0^2} E \left[\sup_{\phi \in \mathcal{N}_\phi} \left\| \frac{\partial \mathbf{P}(\mathbf{T}_g, \phi)}{\partial \phi_j} \right\|^2 \right] + \frac{16}{\lambda_0^2} E \left[\sup_{\phi \in \mathcal{N}_\phi} \left\| \frac{\partial \mathbf{S}(\mathbf{T}_g, \phi)}{\partial \phi_j} \right\|^2 \right] \right. \\
&\quad \left. + \frac{8\sqrt{2}}{\lambda_0^3} E \left[\sup_{\phi \in \mathcal{N}_\phi} \left\| \frac{\partial \mathbf{P}(\mathbf{T}_g, \phi)}{\partial \phi_j} \right\|^2 \right] E \left[\sup_{\phi \in \mathcal{N}_\phi} \left\| \frac{\partial \mathbf{S}(\mathbf{T}_g, \phi)}{\partial \phi_j} \right\|^2 \right] \right\}^{\frac{1}{2}} \frac{2}{q_0} E[(\|\mathbf{Y}_g\| + 2B_0)^4]^{\frac{1}{4}} < \infty
\end{aligned}$$

And similarly,

$$\begin{aligned}
& E \left[\sup_{(\phi, \gamma, \beta) \in \mathcal{M}} \left\| \mathbf{R}(\mathbf{T}_g, \phi) (\mathbf{Y}_g - \mathbf{D}_g \beta) \frac{\partial \omega(\mathbf{Z}_g, \mathbf{X}_g, \gamma)}{\partial \gamma'} \right\| \right] \\
&\leq E \left[\sup_{\phi \in \mathcal{N}_\phi} \|\mathbf{R}(\mathbf{T}_g, \phi)\|^4 \right]^{\frac{1}{4}} E \left[\sup_{\beta \in \mathcal{N}_\beta} \|\mathbf{Y}_g - \tilde{\mathbf{D}}_g \beta\|^4 \right]^{\frac{1}{4}} E \left[\sup_{\gamma \in \mathcal{N}_\gamma} \left\| \frac{\partial \omega(\mathbf{Z}_g, \mathbf{X}_g, \gamma)}{\partial \gamma'} \right\|^2 \right]^{\frac{1}{2}} \\
&\leq \frac{2\sqrt{2}}{\lambda_0} E[(\|\mathbf{Y}_g\| + 2B_0)^4]^{\frac{1}{4}} \frac{2}{q_0^2} \max \left\{ E \left[\sup_{\phi \in \mathcal{N}_\phi} \left\| \frac{\partial q(\mathbf{z}, \mathbf{X}_g, \gamma)}{\partial \gamma'} \right\|^2 \right]^{\frac{1}{2}}, E \left[\sup_{\phi \in \mathcal{N}_\phi} \left\| \frac{\partial q(\mathbf{z}', \mathbf{X}_g, \gamma)}{\partial \gamma'} \right\|^2 \right]^{\frac{1}{2}} \right\} \\
&< \infty
\end{aligned}$$

Again, by consistency of estimators and corresponding continuities of $\mathbf{R}, \mathbf{S}, \mathbf{P}, q$, we

have

$$\begin{aligned}
0 &= \frac{1}{\sqrt{G}} \sum_{g=1}^G \mathbf{R}(\mathbf{T}_g) \omega(\mathbf{Z}_g, \mathbf{X}_g) (\mathbf{Y}_g - \tilde{\mathbf{D}}_g \beta_0) \\
&+ \sum_{j=1}^{k_\phi} E \left[\frac{\partial \mathbf{R}(\mathbf{T}_g, \phi_0)}{\partial \phi_j} \omega(\mathbf{Z}_g, \mathbf{X}_g) (\mathbf{Y}_g - \mathbf{D}_g \beta) \right] \sqrt{G} (\hat{\phi}_j - \phi_{j0}) \\
&+ E \left[\mathbf{R}(\mathbf{T}_g) (\mathbf{Y}_g - \mathbf{D}_g \beta_0) \frac{\partial \omega(\mathbf{Z}_g, \mathbf{X}_g, \gamma_0)}{\partial \gamma'} \right] \sqrt{G} (\hat{\gamma} - \gamma_0) \\
&- E \left[\mathbf{R}(\mathbf{T}_g) \omega(\mathbf{Z}_g, \mathbf{X}_g) \tilde{\mathbf{D}}_g \right] \sqrt{G} (\hat{\beta} - \beta_0) + o_p(1)
\end{aligned}$$

The second term is zero because

$$E \left[\frac{\partial \mathbf{R}(\mathbf{T}_g, \phi_0)}{\partial \phi_j} \omega(\mathbf{Z}_g, \mathbf{X}_g) (\mathbf{Y}_g - \mathbf{D}_g \beta) \right] = E \left[\frac{\partial \mathbf{R}(\mathbf{T}_g, \phi_0)}{\partial \phi_j} \underbrace{E [\omega(\mathbf{Z}_g, \mathbf{X}_g) (\mathbf{Y}_g - \mathbf{D}_g \beta) | \mathbf{T}_g]}_{=0} \right],$$

by conditional moment. Note that we have

$$\sqrt{G} (\hat{\gamma} - \gamma_0) = \frac{1}{\sqrt{G}} \psi_\gamma(\mathbf{V}_g, \gamma_0) + o_p(1).$$

Define $V_\gamma = E[\psi_\gamma(\mathbf{V}_g, \gamma_0) \psi_\gamma(\mathbf{V}_g, \gamma_0)']$. Then, by rearranging,

$$\sqrt{G} (\hat{\beta} - \beta_0) = \frac{1}{\sqrt{G}} \sum_{g=1}^G \phi_\beta(\mathbf{V}_g, \beta_0, \gamma_0) + o_p(1),$$

where

$$\begin{aligned}
\phi_\beta(\mathbf{V}_g, \beta_0, \gamma_0) &= \mathbf{A}^{-1} [\psi_\beta(\mathbf{V}_g, \beta_0) + \mathbf{B} \psi_\gamma(\mathbf{V}_g, \gamma_0)] \\
\psi_\beta(\mathbf{V}_g, \beta_0) &= \mathbf{R}(\mathbf{T}_g) \omega(\mathbf{Z}_g, \mathbf{X}_g) (\mathbf{Y}_g - \tilde{\mathbf{D}}_g \beta_0) \\
\mathbf{A} &= E \left[\mathbf{P}'(\mathbf{T}_g) \mathbf{S}^{-1}(\mathbf{T}_g) \mathbf{P}(\mathbf{T}_g) \right], \\
\mathbf{B} &= E \left[\mathbf{R}(\mathbf{T}_g) (\mathbf{Y}_g - \mathbf{D}_g \beta_0) \frac{\partial \omega(\mathbf{Z}_g, \mathbf{X}_g, \gamma_0)}{\partial \gamma'} \right].
\end{aligned}$$

Since $E[\psi_\beta(\mathbf{V}_g, \beta_0)\psi_\beta(\mathbf{V}_g, \beta_0)'] = \mathbf{A}$, we have

$$\begin{aligned} E[\phi_\beta(\mathbf{V}_g, \beta_0, \gamma_0)\phi_\beta(\mathbf{V}_g, \beta_0, \gamma_0)'] &= \mathbf{A}^{-1}[\mathbf{A} + \mathbf{C}'\mathbf{B} + \mathbf{B}'\mathbf{C} + \mathbf{B}\mathbf{V}_\gamma\mathbf{B}]\mathbf{A}^{-1} \\ &= \mathbf{A}^{-1} + \mathbf{A}^{-1}[\mathbf{C}'\mathbf{B} + \mathbf{B}'\mathbf{C} + \mathbf{B}\mathbf{V}_\gamma\mathbf{B}]\mathbf{A}^{-1}, \end{aligned}$$

where $\mathbf{C} = E[\psi_\gamma(\mathbf{V}_g, \gamma_0)\psi_\beta(\mathbf{V}_g, \beta_0)]$.

□

A.2 Additional Tables

A.2.1 Simulation

Table 14: Simulation of Design 1

	G	Use true ω		Estimate ω	
		Probit	Linear	Probit	Linear
MSE	2,500	94.885	101.99	82.737	89.407
	5,000	45.53	48.673	40.047	42.93
	10,000	22.709	24.052	19.635	20.859
	20,000	11.347	12.068	9.845	10.495
MAE	2,500	14.409	14.851	13.476	13.924
	5,000	10.085	10.399	9.46	9.769
	10,000	7.113	7.29	6.624	6.79
	20,000	5.029	5.169	4.683	4.822
Cov. Rate	2,500	0.954	0.946	0.953	0.944
	5,000	0.951	0.951	0.952	0.947
	10,000	0.95	0.95	0.948	0.946
	20,000	0.95	0.946	0.95	0.946

notes. This table presents simulation results for $B = 10,000$ replications. The sample sizes are set to $G = 2,500, 5,000, 10,000$ and $20,000$. MSE, MAE, and Cov. Rate denote the mean squared error, mean absolute error, and the average 95% coverage rate computed by formulas (15). The first two columns (“Use true ω ”) use the true propensity score $\Pr(\mathbf{Z}_i = \mathbf{z}|\mathbf{X})$ for the weight ω , while the last two columns (“Estimate ω ”) estimate the propensity score (i.e., estimating γ s) and hence ω .

Table 15: Simulation of Design 2

	G	Use true ω		Estimate ω	
		Probit	Linear	Probit	Linear
MSE	2,500	3.577	3.49	2.159	2.143
	5,000	1.788	1.747	1.074	1.062
	10,000	0.881	0.863	0.536	0.531
	20,000	0.443	0.435	0.262	0.261
MAE	2,500	2.107	2.079	1.633	1.623
	5,000	1.489	1.474	1.157	1.151
	10,000	1.054	1.044	0.82	0.816
	20,000	0.744	0.737	0.572	0.571
Cov. Rate	2,500	0.945	0.945	0.945	0.944
	5,000	0.944	0.942	0.947	0.946
	10,000	0.954	0.951	0.948	0.948
	20,000	0.948	0.948	0.952	0.95

notes. This table presents simulation results for $B = 10,000$ replications. The sample sizes are set to $G = 2,500, 5,000, 10,000$ and $20,000$. MSE, MAE, and Cov. Rate denote the mean squared error, mean absolute error, and the average 95% coverage rate computed by formulas (15). The first two columns (“Use true ω ”) use the true propensity score $\Pr(\mathbf{Z}_i = \mathbf{z}|\mathbf{X})$ for the weight ω , while the last two columns (“Estimate ω ”) estimate the propensity score (i.e., estimating γ s) and hence ω .

Table 16: Simulation of Design 3

	G	Use true ω		Estimate ω	
		Probit	Linear	Probit	Linear
MSE	2,500	7.102	7.099	7.011	7.009
	5,000	3.557	3.556	3.495	3.494
	10,000	1.874	1.873	1.845	1.844
	20,000	0.905	0.904	0.885	0.884
MAE	2,500	2.97	2.969	2.952	2.951
	5,000	2.115	2.114	2.094	2.093
	10,000	1.525	1.525	1.512	1.512
	20,000	1.058	1.058	1.049	1.049
Cov. Rate	2,500	0.955	0.956	0.953	0.953
	5,000	0.955	0.955	0.952	0.953
	10,000	0.948	0.949	0.947	0.947
	20,000	0.95	0.951	0.951	0.951

notes. This table presents simulation results for $B = 10,000$ replications. The sample sizes are set to $G = 2,500, 5,000, 10,000$ and $20,000$. MSE, MAE, and Cov. Rate denote the mean squared error, mean absolute error, and the average 95% coverage rate computed by formulas (15). The first two columns (“Use true ω ”) use the true propensity score $\Pr(\mathbf{Z}_i = \mathbf{z}|\mathbf{X})$ for the weight ω , while the last two columns (“Estimate ω ”) estimate the propensity score (i.e., estimating γ s) and hence ω .

Table 17: Simulation of Design 1 for each parameter

	G	Design 1			
		$\delta_1(0)$	$\theta_1(0)$	$\delta_2(0)$	$\theta_2(0)$
Mean	2,500	19.986	9.997	29.96	14.997
	5,000	19.984	9.998	29.992	14.985
	10,000	19.992	10.021	30.04	14.991
	20,000	19.996	9.99	29.989	14.997
Median	2,500	19.96	10.16	30.065	15.043
	5,000	19.962	10.026	30.064	15.019
	10,000	20.005	10.047	30.072	15.008
	20,000	20.001	10.029	30.007	15.005
MSE	2,500	7.008	26.121	47.314	8.964
	5,000	3.384	12.378	22.773	4.396
	10,000	1.65	5.961	11.066	2.182
	20,000	0.815	3.009	5.604	1.067
Cov. Rate	2,500	0.952	0.946	0.944	0.952
	5,000	0.951	0.947	0.948	0.953
	10,000	0.953	0.95	0.951	0.946
	20,000	0.952	0.948	0.946	0.953

notes. This table presents simulation results for $B = 10,000$ replications. The sample sizes are set to $G = 2,500, 5,000, 10,000$ and $20,000$. Mean and median are computed over B replications. The means squared error (MSE) for each parameter is computed by

$$\text{MSE}_k = \frac{1}{B} \sum_{b=1}^B (\hat{\beta}_{kb} - \beta_{0k})^2,$$

and the average 95% coverage rate (Cov. Rate) is computed by formula (15). From the design, the actual parameter values are $\delta_1(0) = 20$, $\theta_1(0) = 10$, $\delta_2(0) = 30$, $\theta_2(0) = 15$.

Table 18: Simulation of Design 2 and 3 for each parameter

		Design 2			
	G	$\delta_1(0)$	$\theta_1(0)$	$\delta_2(0)$	$\theta_2(0)$
Mean	2,500	19.987	14.991	19.984	14.988
	5,000	20.005	15.004	19.993	14.996
	10,000	19.991	14.994	19.979	14.985
	20,000	19.998	14.998	19.992	14.994
Median	2,500	20.013	15.01	19.977	14.983
	5,000	20.012	15.011	19.971	14.977
	10,000	19.999	14.998	19.966	14.979
	20,000	19.995	14.996	19.985	14.989
MSE	2,500	1.363	0.78	4.483	2.526
	5,000	0.677	0.385	2.233	1.261
	10,000	0.337	0.193	1.18	0.664
	20,000	0.166	0.095	0.566	0.319
Cov. Rate	2,500	0.945	0.944	0.953	0.953
	5,000	0.946	0.948	0.953	0.953
	10,000	0.949	0.948	0.948	0.947
	20,000	0.95	0.95	0.951	0.951

notes. This table presents simulation results for $B = 10,000$ replications. The sample sizes are set to $G = 2,500, 5,000, 10,000$ and $20,000$. Mean and median are computed over B replications. The means squared error (MSE) for each parameter is computed by

$$\text{MSE}_k = \frac{1}{B} \sum_{b=1}^B (\hat{\beta}_{kb} - \beta_{0k})^2,$$

and the average 95% coverage rate (Cov. Rate) is computed by formula (15). From the design, the actual parameter values are $\delta_1(0) = 20$, $\theta_1(0) = 10$, $\delta_2(0) = 30$, $\theta_2(0) = 15$.

A.2.2 Other Outcome for simulation

This design is under total monotonicity. Let $K_i \in AT, SC, C, GC, NT$ be a discrete random variable denoting unit i 's compliance type under total monotonicity as defined in Table 2. Then, as derived in A.4, we can define direct and indirect local average treatment effects for each compliance type. For the heterogeneity of effects for compliance types, potential outcomes are generated from the following linear structural equation:

$$Y_i(d_1, d_2) = \alpha_i^D(d_1) + \alpha_i^I(d_2) + \rho \alpha_i^D(d_1) \alpha_i^I(d_2) + \beta_i^y Y_j(d_2, d_1) + \beta_{1i}^w W_i + \beta_{2i}^w W_j. \quad (18)$$

Here, $\alpha^D(d)$, $\alpha^I(d)$ are random components generated by

$$\begin{aligned} \alpha_i(d) &= \sum_{k \in \{A, S, C, G, N\}} \mathbb{1}\{K_i = k\} \alpha_i^k(d), \quad \alpha_i^k(1) \sim N(\bar{\alpha}_i^k, 1), \quad \alpha_i^k(0) \sim N(0, 1), \\ \phi_i(d) &= \sum_{k \in \{A, S, C, G, N\}} \mathbb{1}\{K_j = k\} \phi_i^k(d), \quad \phi_i^k(1) \sim N(\bar{\phi}_i^k, 1), \quad \phi_i^k(0) \sim N(0, 1), \end{aligned}$$

for $i \in \{1, 2\}$, $j = 3 - i$, and $(d_1, d_2) \in \{0, 1\}^2$. This reflects that direct effects depends on the own compliance type, while indirect effects depend on the other's compliance type. The third term in (18) is an interaction term, which is a last term in 11. The interaction term vanishes when $\rho = 0$. In the fourth term, β_i^y reflects the endogenous peer effects. Thus (18) imposes for potential outcome to satisfy Assumption 1 and 3. The above structural equation implies the following reduced form outcome:

$$\begin{aligned} Y_i(d_1, d_2) &= \underbrace{\frac{\alpha_i(d_1) + \beta_i \phi_j(d_1)}{1 - \beta_1 \beta_2}}_{:= \tilde{\alpha}_i(d_1)} + \underbrace{\frac{\phi_i(d_2) + \beta_i \alpha_j(d_2)}{1 - \beta_1 \beta_2}}_{:= \tilde{\phi}_i(d_2)} + \rho \underbrace{\frac{\alpha_i(d_1) \phi_i(d_2) + \beta_i \alpha_j(d_2) \phi_j(d_1)}{1 - \beta_1 \beta_2}}_{:= \tilde{\xi}(d_1, d_2)} \\ &\quad + \underbrace{\frac{(\beta_{1i}^w + \beta_i \beta_{2j}^w) W_i + (\beta_{2i}^w + \beta_i \beta_{1j}^w) W_j}{1 - \beta_1 \beta_2}}_{:= \beta_i' W_i}. \end{aligned}$$

Therefore, the observed outcome is generated as follows:

$$\begin{aligned}
Y_i &= Y_i(0, 0) + D_i(Y_i(1, 0) - Y_i(0, 0)) + D_j(Y_i(0, 1) - Y_i(0, 0)) \\
&\quad + D_i D_j(Y_i(1, 1) - Y_i(1, 0) - Y_i(0, 1) + Y_i(0, 0)) \\
&= (\tilde{\alpha}_i(0) + \tilde{\phi}_i(0)) + (\tilde{\alpha}_i(1) - \tilde{\alpha}_i(0))D_i + (\tilde{\phi}_i(1) - \tilde{\phi}_i(0))D_j \\
&\quad + (\tilde{\xi}_i(1, 1) - \tilde{\xi}_i(1, 0) - \tilde{\xi}_i(0, 1) - \tilde{\xi}_i(0, 0))D_j + \beta'_i \mathbf{W}_i.
\end{aligned}$$

The actual parameter values are given as follows.²¹

Table 19: Parameter Values

Individual (i)	1	2
\mathbf{m}_1 -Direct Effect ($\delta_i^{\mathbf{m}_1}(0)$)	64	49.14
\mathbf{m}_1 -Indirect ($\theta_i^{\mathbf{m}_1}(0)$)	36.57	32
\mathbf{m}_1 -Interaction ($\zeta_i^{\mathbf{m}_1}$)	10.24	8.96

Table 20 show the simulation results for this design. The number of repetitions is 1000, and mean squared error (MSE), mean absolute error (MAE), and the average 95% coverage rate (Cov. Rate) are calculated.

Overall, looking at the MSE, all methods show little difference, but use the first-stage estimator for ω appears slightly more efficient. Furthermore, in computing the instrument, the linear probability model appears to be more efficient than the correctly specified probit model. This is partly because estimation becomes unstable when the conditional probability estimated by the probit model approaches 0. As expected from Proposition 5, the inference based on the plug in estimator for the asymptotic variance has asymptotically correct size.

²¹The rest of parameters are set as follows. First, the parameters in potential outcome are $(\beta_1^y, \beta_2^y, \beta_{1i}^w, \beta_{2i}^w) = (0.5, 0.25, 1, 0.5)$, $i \in \{1, 2\}$. Second, the parameters in the potential treatment generation are $\phi_1^d = (-1.5, 6, 2, 9, 1)'$, $\phi_2^d = (-1.5, 8, 2, 9, 0.5)'$. Third, the true means of random components in potential outcome are $\bar{\alpha}_1(1) = [48, 48, 48, 96, 120]$, $\bar{\alpha}_2(1) = [40, 40, 40, 80, 100]$, $\bar{\phi}_1(1) = [12, 12, 12, 16, 20]$, and $\bar{\phi}_2(1) = [16, 16, 16, 32, 40]$, where the ordering is AT, SC, C, GC, NT. Lastly, $\bar{\alpha}_1(0) = \bar{\alpha}_2(0) = \bar{\phi}_1(0) = \bar{\phi}_2(0) = 0$.

Table 20: Monte Carlo Simulation: Design 2

G		Infeasible	Use true ω		Estimate ω	
			Probit	Linear	Probit	Linear
2500	MSE	13.789	14.293	12.629	13.781	12.271
	MAE	0.027	0.029	0.024	0.031	0.027
	Cov. Rate	0.943	0.944	0.961	0.945	0.961
5000	MSE	6.619	6.506	6.12	6.406	5.989
	MAE	0.027	0.027	0.022	0.021	0.015
	Cov. Rate	0.944	0.948	0.959	0.948	0.96
10000	MSE	3.171	3.131	3.003	3.037	2.89
	MAE	0.009	0.011	0.009	0.007	0.005
	Cov. Rate	0.942	0.946	0.951	0.946	0.952
20000	MSE	1.491	1.482	1.363	1.461	1.339
	MAE	0.004	0.005	0.006	0.003	0.004
	Cov. Rate	0.95	0.951	0.961	0.95	0.959

notes. This table shows simulation results for $B = 1,000$ replications. The sample sizes are set by $G = 2500, 5000, 10000, 20000$. MSE, MAE, Cov. Rate denotes the mean squared error, mean absolute error, and the average 95% coverage rate computed by ??.

A.3 Empirical Result by using Nonlinear First-Stage

Table 21: Make Deposit

Unit	Effects	m_1		m_1	
		R	VB	R	VB
Female	Direct	0.09** (0.04)	0.11*** (0.04)	-0.06 (0.06)	
	Indirect	0.45 (0.32)		0.03 (0.03)	0.03 (0.03)
Male	Direct	1.38** (0.54)		0.12*** (0.04)	0.13*** (0.05)
	Indirect	-0.08* (0.05)	0.01 (0.04)	-0.07 (0.34)	

notes. The dependent variable is 1 if the individual made at least one deposit. Control variables consist of age, education, regions, indicators of members in ROSCA, and indicators of using mobile money for both the female and male head at the baseline period. “Direct” denotes the local average treatment effects $E[Y_i(1,0) - Y_i(0,0)|K_i^m = 1]$, and “Indirect” denotes $E[Y_i(0,1) - Y_i(0,0)|K_j^m = 1]$. Plug-in clustered standard errors are reported in parentheses. *, **, *** denote the significance levels at 10%, 5%, and 1%, respectively. The instruments are estimated by using probit model in the first stage.

Table 22: Make Withdrawal

Unit	Effects	m_1		m_1	
		R	VB	R	VB
Female	Direct	0.06** (0.03)	0.05* (0.02)	0.27 (0.4)	
	Indirect	-0.33** (0.13)		<0.001 (0.004)	0.01 (0.01)
Male	Direct	0.17 (0.28)		0.02 (0.03)	0.04 (0.03)
	Indirect	0.003 (0.03)	0.003 (0.03)	0.73 (0.57)	

notes. The dependent variable is 1 if the individual made at least one withdrawal. Control variables consist of age, education, regions, indicators of members in ROSCA, and indicators of using mobile money for both the female and male head at the baseline period. “Direct” denotes the local average treatment effects $E[Y_i(1,0) - Y_i(0,0)|K_i^m = 1]$, and “Indirect” denotes $E[Y_i(0,1) - Y_i(0,0)|K_j^m = 1]$. Plug-in clustered standard errors are reported in parentheses. *, **, *** denote the significance levels at 10%, 5%, and 1%, respectively. The instruments are estimated by using probit model in the first stage.

A.4 Finer Classification of Compliance Types

As pointed out in example 1, we can divide compliance types into finer types if there exists multiple monotone pairs. Again, recall a monotone pair $\mathbf{m}_1 = ((0, 1), (0, 0), (1, 1))$, $\mathbf{m}_2 = ((1, 0), (0, 0), (1, 1))$ and $\mathbf{m}_3 = ((1, 0), (0, 1), (1, -1))$. Then, as summarized in 1, for individual 1, the \mathbf{m}_2 -complier is either \mathbf{m}_1 -complier (social complier) or \mathbf{m}_3 -complier (complier). Since the events for individual 1 to be a \mathbf{m}_1 -complier, and \mathbf{m}_3 -complier are disjoint, we have

$$P_1^{m_2}(\mathbf{T}) = \Pr(K_1^{m_2} = 1|\mathbf{T}) = \Pr(K_1^{m_1} = 1|\mathbf{T}) + \Pr(K_1^{m_3} = 1|\mathbf{T}) = P_1^{m_1}(\mathbf{T}) + P_1^{m_3}(\mathbf{T}).$$

Also, we have

$$\begin{aligned} \delta_1^{m_2} P_1^{m_2}(\mathbf{T}) &= E[\Delta_1 Y_1(0) | K_1^{m_2} = 1, \mathbf{T}] \Pr(K_1^{m_2} = 1 | \mathbf{T}) \\ &= E[\Delta_1 Y_1(0) | K_1^{m_1} = 1] \Pr(K_1^{m_1} = 1 | K_1^{m_2} = 1, \mathbf{T}) \Pr(K_1^{m_2} = 1 | \mathbf{T}) \\ &\quad + E[\Delta_1 Y_1(0) | K_1^{m_3} = 1] \Pr(K_1^{m_3} = 1 | K_1^{m_2} = 1, \mathbf{T}) \Pr(K_1^{m_2} = 1 | \mathbf{T}) \\ &= E[\Delta_1 Y_1(0) | K_1^{m_1} = 1] \Pr(K_1^{m_1} = 1 | \mathbf{T}) \\ &\quad + E[\Delta_1 Y_1(0) | K_1^{m_3} = 1] \Pr(K_1^{m_3} = 1 | \mathbf{T}) \\ &= \delta_1^{m_1} P_1^{m_1}(\mathbf{T}) + \delta_1^{m_3} P_1^{m_3}(\mathbf{T}). \end{aligned}$$

So we have the following relationship between parameters from different monotone pairs:

$$\delta_1^{m_2} = \delta_1^{m_1} \left(\frac{P_1^{m_1}(\mathbf{T})}{P_1^{m_1}(\mathbf{T}) + P_1^{m_3}(\mathbf{T})} \right) + \delta_1^{m_3} \left(\frac{P_1^{m_3}(\mathbf{T})}{P_1^{m_1}(\mathbf{T}) + P_1^{m_3}(\mathbf{T})} \right).$$

Or, we can write

$$\begin{aligned} E[ITT_1^{m_2}(\mathbf{X})|\mathbf{T}] &= \delta_1^{m_2} P_1^{m_2}(\mathbf{T}) + \theta_1^{m_2} P_2^{m_2}(\mathbf{T}) \\ &= \delta_1^{m_1} P_1^{m_1}(\mathbf{T}) + \delta_1^{m_3} P_1^{m_3}(\mathbf{T}) + \theta_1^{m_2} P_2^{m_2}(\mathbf{T}). \end{aligned}$$

Thus, we can also identify the parameters in a finer compliance class directly. \square