Causal Mechanism with Interference

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Abstract

This study examines identification and estimation in the presence of social interactions. Potential outcome depends on both an individual's own treatment and the exposures generated by others' treatment statuses and the underlying network structure. When the network itself is impacted by treatment, the individual's treatment can also alter these exposures. In such cases, the treatment effect can be decomposed into direct and indirect components. The direct effect represents the causal impact on the outcome when exposures are fixed, while the indirect effect captures the influence of changes in the exposure distribution due to the individual's own treatment. As a result, observed exposures can be viewed as mediators of the individual's treatment effect. I employ a causal mediation framework to identify and decompose these effects and propose an estimation method. The performance of the estimator is then evaluated through Monte Carlo simulations, followed by an empirical application examining the impact of coeducational high schools on academic performance.

Keywords: Causal inference; Network change; Mediation Effects

1 Introduction

The identification and estimation of causal effects of a program or policy are of significant interest in economic analysis. Rubin's causal model is a widely used approach for addressing causal effects (e.g., Rubin (1974), Imbens and Rubin (2010)). A key assumption in this framework is the Stable Unit Treatment Value Assumption (SUTVA), which assumes that each individual's potential outcomes are solely determined by their own treatment status. However, as highlighted by Kline and Tamer (2020), social interactions offer an additional mechanism through which a program may influence outcomes. In such cases, potential outcomes can depend on both an individual's own treatment status and the treatment status of others within their network.

Consider a society composed of N individuals where each individual interacts with others within their neighborhood. The potential outcome for an individual i can be expressed as $Y_i(d_i, \boldsymbol{d}_{-i}) = m(d_i, \boldsymbol{d}_{-i}, \varepsilon_i)$, with some response function $m(\cdot)$, where the first argument $d \in \{0, 1\}$ represents the individual's own treatment assignment, the second argument $\boldsymbol{d}_{-i} \in \{0, 1\}^{N-1}$ is a vector representing the treatment assignments of the all individuals in the society other than i, and the last argument ε_i is an individual error term. This model violates SUTVA if $Y_i(d_i, \boldsymbol{d}_{-i}) \neq Y_i(d_i, \boldsymbol{d}'_{-i})$ for some $\boldsymbol{d}_{-i} \neq \boldsymbol{d}'_{-i}$. Additionally, note that each individual has 2^N potential outcomes based on the possible number of treatment statuses. Therefore, the number of potential outcomes expands exponentially in the number of individuals. This introduces challenges in defining and identifying the treatment effects.

However, the number of *effective* treatment is likely to be much smaller 2^N. For example, the potential outcome may depend on the individual's own treatment status and the number of treated friends. If each individual has $M < N$ friends, there are $2M$ possible treatment situations, which is considerably fewer than 2^N. Here, the number of treated friends is often called an exposure. In general, if there exists a function $\psi : \{0, 1\}^{N-1} \to \Psi \subset \mathbb{R}^K$ for some K, such that $\psi(\boldsymbol{d}_{-i}) = \psi(\boldsymbol{d}'_{-i})$ implies $Y_i(d_i, \boldsymbol{d}_{-i}) = Y_i(d_i, \boldsymbol{d}'_{-i})$ with probability 1, then ψ is called an exposure map, or treatment rule in the literature.

In this setting, causal effects can be defined as follows. First, the treatment effect is the impact of changes in an individual's own treatment status on the outcome when exposures are fixed. Second, spillover or exposure effects refer to the impact of changes in exposures on the outcome when the individual's own treatment status remains fixed.

Recent studies emphasize the importance of spillover or exposure effects in program evaluations, as economic agents typically interact with others. The exposure is usually determined by the treatment vector of others, $\boldsymbol{d}_{-i}$, and the underlying network structure, such as the number of treated friends. If the network is fixed or exogenously given, the exposure effect can be interpreted as the causal effect of changes in exposure, such as the effect of adding one more treated friend. However, empirical evidence shows that the underlying network may also be influenced by the program (e.g., Dupas, Keats, and Robinson (2019), Comola and Prina (2021), Banerjee et al. (2024)).

When an individual's treatment status affects the distribution of exposures, we can define the causal impact of their own treatment on the exposure, which means that part of the exposure effect must also be interpreted as the treatment effect. To analyze the causal impact of treatment on exposure, let D_i be the individual i's treatment indicator, and $\psi_i(0), \psi_i(1)$ be potential exposures of individual i when untreated and treated, respectively. The observed exposure for individual i is given by $\psi_i = (1 - D_i)\psi_i(0) + D_i\psi_i(1)$. Then, the causal effect on the outcome (i.e., the treatment effect) can be decomposed as:

$$Y_i(1, \psi_i(1)) - Y_i(0, \psi_i(0)) = Y_i(1, \psi_i(0)) - Y_i(0, \psi_i(0)) + Y_i(1, \psi_i(1)) - Y_i(1, \psi_i(0)).$$

The first term on the right-hand side represents the direct treatment effect, which captures the impact of treatment when exposure remains unchanged. The second term represents the indirect treatment effect, reflecting the effect of changes in exposure resulting from the individual's own treatment status. For example, if the exposure is defined as the number of treated friends, the direct treatment effect measures the casual effect of the treatment when the distributions of the number of treated friends are fixed. In contrast, the indirect treatment effect captures the difference between potential outcomes when only the distribution of the number of treated friends changes due to the own treatment.

The primary contribution of this paper is the separate identification and estimation of direct and indirect treatment effects using a causal mediation model. By decomposing treatment effects, we can better understand the mechanisms through which a policy/program influence outcomes. If the dataset contains complete information on the network adjacency matrix, detailed exposure maps can be constructed. However, this framework is also applicable in situations where full network information is unavailable, as the model only requires data on observed outcomes, treatment status, and some exposures, making it highly advantageous in

many of scenarios.

To identify the decomposition, I use a mediation model where exposure serves as a mediator for the treatment effects on potential outcomes. I propose corresponding frequency estimators and derive their asymptotic properties. Since exposures are influenced by others' treatments and the underlying network structure, they are not independent across individuals. Consequently, the asymptotic theory is based on a boundedness assumption in the data dependency graph, which is compatible with either a sparse single large network or a network consisting of multiple independent groups.

The outline of this paper is as follows. Section 2, the settings and model used in this paper are introduced. Section 3 discusses identification and estimation. Section 4 shows results from Monte Carlo studies to verify the performance of estimators. Section 5 is an empirical application to illustrate the proposed method. Section 6 concludes.

Related Literature

The Rubin causal model (e.g., Rubin (1974), Imbens and Rubin (2010)), based on the potential outcome framework, is widely used in economic analysis for identifying and estimating treatment effects. This model assumes SUTVA, which excludes interactions between individuals affecting their potential outcomes. In this context, Manski (2013) refers to SUTVA as the individualistic treatment response (ITR). However, in the presence of social interactions, an individual's treatment response may depend on the entire treatment vector within their society. Manski (2013) identifies the distribution of potential outcomes under the constant treatment response (CTR) assumption. Let $Y_i(\boldsymbol{d})$ represent the potential outcome for individual i when they face the treatment vector $\boldsymbol{d}$. The CTR assumption states that there exists a function c_i such that $c_i(\boldsymbol{d}) = c_i(\boldsymbol{d}')$ implies $Y_i(\boldsymbol{d}) = Y_i(\boldsymbol{d}')$. Manski refers to the image of this function as the set of effective treatments. The function c_i is called the exposure map or treatment rule in the literature. The CTR assumption generalizes the ITR or SUTVA, as these are special cases where $c_i(\boldsymbol{d}) = d_i$, and c_i is the identity function for all i.

Because analyzing the general unrestricted model, where $c_i(\boldsymbol{d}) = \boldsymbol{d}$ is challenging due to the dimensionality of the potential outcome space, the concept of effective treatment is commonly used in the literature. Studies often assume the effective treatment is finite-dimensional, reducing the complexity of both the treatment and the number of potential out-

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comes. Forastiere, Airoldi, and Mealli (2021) propose the Stable Unit Treatment Value on Neighborhood Assumption (SUTNVA), where potential outcomes depend only on the treatment vector of the individual's neighborhood. This is a natural extension of SUTVA to settings involving neighborhood interactions. Aronow and Samii (2017), Vazquez-Bare (2023) assume the neighborhood size is fixed, meaning each individual has a finite number of potential outcomes.

As such studies, once we fix the dimension of effective treatment, or the domain of potential outcomes, then identifying treatment effect parameters are similar to those of multiple treatment model. Corresponding independence assumptions or ignorabilities imply the identification. As related works, Leung (2020) focus on the number of treated neighbors and the number of neighbors (i.e., degree) as the exposures. The model is therefore a linear-in-means model without endogenous peer effect. The author identifies the average and quantile treatment, exposure effects, and derive their asymptotic normality by using a new version of asymptotic theories. Vazquez-Bare (2023) derive general identification argument of this type of model and propose some estimators. The author assume there are independent groups in data, and the interaction arises within group. By controling the size of group and the number of group, he show asymptotic normality of the frequency estimator.

One important problem is the possibility of misspecification of the exposure map. If the exposure map is incorrect, then the identification and corresponding estimators would be misleading. In this respect, Vazquez-Bare (2023) also shows that if the true exposure map is coarser then the exposure map used in the estimator, then the average potential outcomes are identified as the usualy frequcney estimands, and some weighted average of those frequency estimands otherwise. Intuitively, the less coarsity of true exposure map means the information contained in the exposure map is more rich than that from the true exposure map. Leung (2022) assumes *approximately neighborhood interaction (ANI)* assumption to identify the treatment parameteres when the exposure map is possibly misspecified. This assumption is that the potential outcome distributions are primarily determined by the neighbors within some close distance. However, I assume we have correctly specified exposure map in this study, to focus on the dependency between exposure and the treatment assignments.

Most studies in this literature assume the underlying social network is fixed or independent of treatment assignment. These assumptions exclude the possibility that a policy can change the underlying random graphs of social networks. Some studies consider this possi-

bility. Comola and Prina (2021) suggests a two-period model in that treatments are assigned in the first period, and the network can change in the second period. The outcome in each period follows the linear in means model as in Bramoullé, Djebbari, and Fortin (2009), so they use the similar identifying assumption that there is no intransitive triad in networks of both periods. To apply this model, researchers need to have full information about network adjacency matrices in both periods. However, It would sometimes be hard to get such descent information.

If potential outcomes are functions of the own treatment status and the exposures, the exposures are determined by the underlying social networks. In this study, I assume that the distributions of exposures are influenced by their own treatment status. That is, the own treatment status could change the functional form of the exposure map, or change the random graph of social networks. In both cases, exposure can be thought of as a mediator of own treatment on potential outcomes. In the literature on mediation models, the main purpose is to figure out the mechanism of how treatment influences potential outcomes by decomposing the total treatment effect into direct and indirect effects. Suppose $M(d)$ is the potential mediator when own treatment status is given by $d \in \{0, 1\}$. Then, the observed mediator is $M = dM(1) + (1-d)M(0)$. The direct treatment effect is the effect of the own treatment status when the mediator is fixed, and the indirect treatment effect is the effect of the own treatment only through changes in the mediator. If both treatment assignment and the distributions of mediators are independent of potential outcomes, then it is straightforward to identify distributions of direct and indirect treatment effects separately. Huber (2014) suggests sequential ignorability assumptions, which are weaker conditions than the full independence to identify the direct and indirect effects, and I follow these assumptions and identification arguments in this study.

Even if the treatments are randomly assigned, the data of outcomes and exposures could be dependent due to social interactions. This dependency makes it tricky to derive asymptotic properties. In the case of the sum of independent random variables, Esseen's method (Esseen (1945)) is convenient to approximate normal distribution. Vazquez-Bare (2023) uses the Berry-Esseen bound to derive the asymptotic normality by assuming exposures are independent across groups. However, it is difficult to apply Esseens' method to dependent data. Instead, Stein's method (Stein (1972)) is widely used to deal with dependent data. As an example, Chen and Shao (2004) provides a version of the central limit theorem with a bounded

maximum degree of the dependency graph. Leung (2020) derived conditions on the moment of dependency graph instead of directly applying Stein's method, but in this study, I assume boundedness of the maximum degree of dependency graph and use Stein's method.

2   Model

2.1   Setting and the exposure map

Suppose we have a random sample of N individuals. Let $Y_i \in \mathbb{R}$ represent the outcome and $D_i \in \{0, 1\}$ be the binary treatment indicator for individual i. For a given vector of treatment assignments for all individuals $\boldsymbol{d} \in \{0, 1\}^N$, let $\boldsymbol{d}_{-i}$ represent $(N-1)$-treatment vector of individuals except i. The potential outcome for individual i can be written as a function of the entire treatment vector: $Y_i(\boldsymbol{d}) = Y_i(d_i, \boldsymbol{d}_{-i})$, for $\boldsymbol{d} \in \{0, 1\}^N$.

Following Vazquez-Bare (2023), assume there exists a known exposure map ψ that maps $\boldsymbol{d}_{-i} \in \{0, 1\}^{N-1}$ to $\psi(\boldsymbol{d}_{-i}) \in \Psi \subset \mathbb{R}^K$ and satisfies $Y_i(d_i, \boldsymbol{d}_{-i}) = Y_i(d_i, \boldsymbol{d}'_{-i})$ for all $\boldsymbol{d}_{-i} \in \{0, 1\}^{N-1}$, whenever $\psi(\boldsymbol{d}_{-i}) = \psi(\boldsymbol{d}'_{-i})$, with $K < N$. The exposure map is a rule that relates the treatment vector of neighbors to the potential outcome. In this study, the exposure map is assumed to be correctly specified.[1,2] Furthermore, abusing the notation, write $Y_i(d, s)$ as potential outcome for individual i when the treatment is $D_i = d$, and exposure is $\psi(\boldsymbol{D}_{-i}) = s$.

In this setting, the treatment effect is defined as the effect of a change in d_i on the outcome, while the spillover or exposure effect is defined as the effect of a change in $\psi(\boldsymbol{d}_{-i})$ on the outcome. For example, Leung (2022) uses $\psi(\boldsymbol{d}_{-i}) = \mathbb{1}\left\{\sum_j A_{ij} d_j > 0\right\}$, is an indicator of whether individuals i and j are friends. In this case, the exposure effect represents the difference in potential outcomes between an individual with at least one treated friend and one with no treated friends.

In contrast to previous studies in the literature, I assume that the distribution of exposure $\psi(\boldsymbol{d}_{-i})$ can vary depending on an individual's own treatment status. The exposure may contain information about the underlying social network, allowing individual i's treatment

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[1] Aronow and Samii (2017) discuss about when the exposure map is misspecified. Leung (2022) proposed a solution when the exposure map is misspecified by using the concept of *approximated neighborhood inference*.

[2] Constructing a model using an exposure map is convenient when only limited information of the network structure is available in data instead of the full information of the adjacency matrix.

status to influence network formation or their links with others. Using a slight abuse of notation, let $\psi(d_i, \boldsymbol{d}_{-i})$ represent the potential exposure of individual i, when their treatment status is $D_i = d_i$, and define $\psi_i(d) := \psi(d, \boldsymbol{D}_{-i})$, where $\boldsymbol{D}_{-i}$ is the vector of treatment indicator of all individuals except i. The observed exposure can be written as $\psi_i = \psi_i(D_i) = D_i\psi_i(1) + (1 - D_i)\psi_i(0)$.

If the distributions of $\psi_i(0)$ and $\psi_i(1)$ are identical, or if the treatment does not affect exposure distributions[3], the model simplifies to a potential outcomes model with multiple treatments. In this scenario, as in Vazquez-Bare (2023) and Leung (2020), identification follows from standard independence assumptions.

However, when an individual's treatment influences the exposure distribution, the exposure effect may combine multiple factors, with part of the exposure effect arising from changes in the individual's own treatment status. The goal of this model is to disentangle the treatment effect into two components: the direct treatment effect, which is the effect when exposure is fixed, and the indirect effect, which captures the impact of changes in the exposure distribution resulting from the individual's own treatment.

This model can be viewed as a generalization of existing methods. When $\psi_i(d) = c$ for some constant c for $d = 0, 1$, then the model satisfies SUTVA. When $\psi_i(1) \sim \psi_i(0)$, then the model is equivalent to a treatment effect model with social interactions and a correctly specified exposure map, as studied by Vazquez-Bare (2023). When $\psi(\boldsymbol{d}_{-i})$ is defined as the number of treated friends, the model becomes a linear-in-means model without endogenous peer effects. For instance, using the exposure map from Leung (2020), and Leung (2022), the potential exposure can be defined as:

$$\psi_i(d) = \left(\sum_j A_{ij}(d, \boldsymbol{D}_{-i}) D_j, \sum_j A_{ij}(d, \boldsymbol{D}_{-i}) \right), \quad \text{or,} \quad \psi_i(d) = \mathbb{1}\left\{ \sum_j A_{ij}(d, \boldsymbol{D}_{-i}) D_j > 0 \right\},$$

where $A_{ij}(d_i, \boldsymbol{d}_{-i})$ is a potential indicator of whether individuals i and j are friends, as a function of the treatment vector to represent how the underlying network is affected by the treatment.

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[3]This occurs when exposure is determined solely by the underlying network structure, such as when the network is fixed or independent of treatment.

2.2   Parameters of interest

The observed outcome can be written as:

$$Y_i = D_i Y_i(1, \psi_i(1)) + (1 - D_i) Y_i(0, \psi_i(0)) = \sum_{d \in \{0,1\}} \sum_{s \in \Psi} \mathbb{1}\{D_i = d, \psi_i = s\} Y_i(d, s) \qquad (1)$$

The data consists of $\{(Y_i, D_i, \psi_i) : 1 \leq i \leq N\}$. Thus, we can observe either $Y_i(1, \psi_i(1))$ or $Y_i(0, \psi_i(0))$ but never both. Moreover, the potential outcomes $Y_i(d, \psi_i(d'))$ for $d \neq d'$ are never observed.

It is important to note that the treatment D_i affects the outcome both directly and indirectly through the exposure $\psi_i = \psi_i(D_i)$. As a result, the exposure ψ_i can be considered a mediator. Therefore, following the literature on causal mediation effects (e.g., Huber (2014), Huber (2019)), the overall treatment effect can be decomposed as follows.

The *average overall treatment effect (ATE)* is defined as the mean difference between potential outcomes when the individual's own treatment is exogenously changed:

$$\Delta \equiv E[Y_i(1, \psi_i(1)) - Y_i(0, \psi_i(0))].$$

The *average direct treatment effect (ADTE)* is defined as the average difference between potential outcomes when the individual's own treatment is exogenously changed, but the mediator (network structure) is fixed at its potential distribution for a given $d \in \{0, 1\}$:

$$\theta(d) \equiv E[Y_i(1, \psi_i(d)) - Y_i(0, \psi_i(d))].$$

Similarly, *the average indirect treatment effect (AITE)* is defined as the average difference between potential outcomes when the distribution of the mediator is exogenously changed, while the individual's own treatment status is fixed at $d \in \{0, 1\}$:

$$\delta(d) \equiv E[Y_i(d, \psi_i(1)) - Y_i(d, \psi_i(0))].$$

Therefore, by construction, the ATE can be decomposed into the sum of the direct treatment effect (ADTE) and the indirect treatment effect (AITE): Therefore, by construction, the ATE is decomposed by the sum of DTE and ITE: $\Delta = \theta(0) + \delta(1) = \theta(1) + \delta(0)$.

Spillover effects, or exposure effects, refer to the impact on potential outcomes when the exposure level is exogenously changed as usual definition in the literature. Let s and s' represent two different values of $\psi_i(d)$. The *exposure effect* is then defined as: $\tau(d, s, s') \equiv E[Y_i(d, s) - Y_i(d, s')]$.

3 Identification and Estimation

3.1 Identification

In this section, we discuss the identification results, beginning with the following identifying assumptions:

Assumption 1. $\{Y_i(1, s), Y_i(0, s), \psi_i(1), \psi_i(0) : s \in \Psi\}$ *are independent of* D_i.

Assumption 2. $\{Y_i(1, s), Y_i(0, s) : s \in \Psi\}$ *are independent of* ψ_i *conditional on* D_i.

These assumptions are the sequential independence assumptions discussed Huber (2014). Note that because the randomness of $\psi_i(d)$ is by other's treatment $\boldsymbol{D}_{-i}$, if the treatment is randomly assigned, then Assumption 1 is satisfied. Next, once $D_i = d$ is given, $\psi_i = \psi_i(d)$, and hence Assumption 2 implies that potential outcomes are independent of the distribution of potential exposures, conditional on the treatment. Assumption 2 may fail if a common factor determines both potential outcomes and exposures. This occurs, for example, when there is a random variable X_i with a nontrivial distribution such that $Y_i(\boldsymbol{d}, \boldsymbol{d}_{-i}) = m(d_i, \boldsymbol{d}_{-i}, X_i, \varepsilon_i)$ and $\psi_i = \psi(D_i, \boldsymbol{D}_{-i}, X_i)$.[4]

Assumption 3. *For each* $d \in \{0, 1\}$ *and* $s \in \Psi$, $P(d, s) \equiv P(D_i = d, \psi_i = s) \in (0, 1)$.

Assumption 3 is the overlap assumption that ensures the existence of appropriate conditional moments. The following Lemma 1 states that the distributions of interest are identified.

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[4]In this case, if we observe X_i, then Assumptions 1 and 2 can be stated additionally conditional on X_i.

Lemma 1 (Identification of distributions). *Under Assumptions 1, and 2,*

$$G^{d,s}(y) \equiv \Pr(Y_i(d,s) \le y) = \Pr(Y_i \le y | D_i = d, \psi_i = s),$$

$$F^{d,d'}(y) \equiv \Pr(Y_i(d, \psi_i(d')) \le y) = \sum_{s \in \Psi} \Pr(Y_i \le y | D_i = d, \psi_i = s) \Pr(\psi_i = s | D_i = d'),$$

$$F^d(y) \equiv F^{d,d}(y) = \Pr(Y_i(d, \psi_i(d)) \le y) = \Pr(Y_i \le y | D_i = d).$$

Note that the distributions of $Y_i(d,s)$ and $Y_i(d, \psi_i(d))$ are identified in the usual way from the independence assumptions. Identification of $Y(d, \psi_i(d'))$ for $d \ne d'$ requires the support of $\psi_i(1)$ and $\psi_i(0)$ are the same. If $\text{Supp}(\psi_i(1)) \subsetneq \text{Supp}(\psi_i(0))$, then only $F^{0,1}(y)$ is identified, not $F^{1,0}(y)$. Using Lemma 1, we can derive the following result.

Proposition 1 (Identification of Average Effects). *Under Assumptions 1, 2,*

$$\theta(0) = \sum_{s \in \Psi} E[Y_i | D_i = 1, \psi_i = s] \Pr(\psi_i = s | D_i = 0) - E[Y_i | D_i = 0],$$

$$\delta(1) = E[Y_i | D_i = 1] - \sum_{s \in \Psi} E[Y_i | D_i = 1, \psi_i = s] \Pr(\psi_i = s | D_i = 0),$$

$$\Delta = \delta(1) + \theta(0) = E[Y_i | D_i = 1] - E[Y_i | D_i = 0].$$

$\theta(1)$ and $\delta(0)$ are identified similarly with $\Delta = \theta(1) + \delta(0)$. For each $d \in \{0,1\}$ and for $s, s' \in \Psi$, the exposure effects are identified as $\tau(d, s, s') = E[Y_i | D_i = d, \psi_i = s'] - E[Y_i | D_i = d, \psi_i = s]$.

3.2 Estimation and Inference

Based on the identification results in Proposition 1, we can construct estimators for the averages of potential outcomes and treatment parameters. For notational simplicity, let $\mathbb{1}_i(d,s) = \mathbb{1}\{D_i = d, \psi_i = s\}$, and $\mathbb{1}_i(d) = \mathbb{1}\{D_i = d\}$. Define $N(d,s) \equiv \sum_{i=1}^{N} \mathbb{1}_i(d,s)$, and $N(d) \equiv \sum_{i=1}^{N} \mathbb{1}_i(d) = \sum_{s \in \Psi} N(d,s)$ be the potential number of individuals with $D_i = d, \psi_i = s$, and $D_i = d$, respectively. Next, define $\nu(d,s) := E[Y(d,s)] = E[Y_i | D_i = d, \psi_i = s]$ for $d \in \{0,1\}, s \in \Psi$, $\mu(d,d') := E[Y(d, \psi_i(d'))]$ for $d, d' \in \{0,1\}$, and $\mu(d) := \mu(d,d)$ as the average potential outcomes. These average outcomes can be estimated by the following frequency

estimators:

$$\hat{v}(d,s) = \frac{1}{N(d,s)} \sum_{i=1}^{N} \mathbb{1}_i(d,s) Y_i,$$

$$\hat{\mu}(d) = \frac{1}{N(d)} \sum_{i=1}^{N} \mathbb{1}\{D_i = d\} Y_i,$$

$$\hat{\mu}(d,d') = \frac{1}{N(d')} \sum_{j=1}^{N} \hat{v}(d,\psi_j) \mathbb{1}\{D_j = d'\}$$

$$= \frac{1}{N(d')} \sum_{s \in \Psi} \sum_{j=1}^{N} \hat{v}(d,s) \mathbb{1}_j(d',s) = \sum_{s \in \Psi} \hat{v}(d,s) \frac{N(d',s)}{N(d')}.$$

Here, $v(d,s)$ is the sample average of the observed outcomes in the subsample where $D_i = d$, and $\psi_i = s$. Note that these estimators are undefined if the corresponding cells are empty. Similarly, $\hat{\mu}(d)$ is the sample average of outcomes on the subsample with $D_i = d$. The estimator $\hat{\mu}(d,d')$ is the weighted average of the average potential outcomes $Y(d,s)$, where the weights are the sample analog of $\Pr(\psi_i = s | D_i = d')$. Using these estimators, the overall, direct, and indirect treatment effects can be estimated as follows: $\hat{\Delta} = \hat{\mu}(1) - \hat{\mu}(0)$, $\hat{\Delta} = \hat{\mu}(1) - \hat{\mu}(0)$, $\hat{\delta}(d) = \hat{\mu}(d,1) - \hat{\mu}(d,0)$, and $\hat{\tau}(d,s,s') = \hat{v}(d,s) - \hat{v}(d,s')$.

Next, we derive the consistency and asymptotic normality of estimators for average outcomes. As mentioned earlier, Y_i may exhibit dependence across individuals. However, because the treatment is randomly assigned, the potential outcomes can be independently and identically distributed. Therefore, we assume the following:

Assumption 4. *For $d \in \{0,1\}$ and $s \in \Psi$, (i) $\{Y_i(d,s)\}_i$ are i.i.d.; (ii) $E[Y_i(d,s)^6] < \infty$.*

To apply normal approximation in a dependent data, we need to restrict about the dependency. Specifically, for each individual i, the number of dependent individual grows slower than the number of individuals N. The primary condition is stated in Assumption 5:

Assumption 5. *Let $C_i = (D_i, \psi_i)$, and g_{ij} represent an indicator of whether for two different individuals i and j, C_i is independent of C_j, i.e., $g_{ij} = \mathbb{1}\{i = j, \text{or } C_i \not\perp C_j\}$. Then, $\sum_{j=1}^{N} g_{ij} = O(N^\delta)$ for some $0 < \delta < 1$.*

Then, the estimators for average potential outcome is asymptotically normal and the plug-in standard error is asymptotically valid. Proposition 2 summarizes the result.

Proposition 2. *Under Assumptions 1-5, for each $d \in \{0, 1\}$ and $s \in \Psi$,*

$$\hat{V}(d, s)^{-1/2} \sqrt{N} (\hat{v}(d, s) - v(d, s)) \xrightarrow{d} N(0, 1),$$

$$\hat{V}_\mu(d, d')^{-1/2} \sqrt{N} (\hat{\mu}(d, d') - \mu(d, d')) \xrightarrow{d} N(0, 1),$$

where $\hat{V}(d, s)$, $\hat{V}_\mu(d, d')$ are estimator for

$$V(d, s) = \frac{\text{Var}(Y_i(d, s))}{P(d, s)},$$

$$V_\mu(d, d') = \sum_{s \in \Psi} \frac{P(d', s)^2}{P(d')^2} V(d, s) + \sum_{s \neq s' \in \Psi} \frac{P(d', s)}{P(d')} \frac{P(d', s')}{P(d')} E[Y(d, s)] E[Y(d, s')],$$

respectively, by replacing $\text{Var}(Y(d, s))$ as $N(d, s)^{-1} \sum_{i=1}^{N} \mathbb{1}_i(d, s) [Y_i - \hat{v}(d, s)]^2$.

4 Simulation

This section evaluate the finite sample performance and the asymptotic result derived in Section 3 using Monte Carlo simulations. Specifically, the mean squared errors and the coverage rates of the estimators provide simulation evidence for asymptotic normality. The data for the simulation consists of N units, where each unit has a binary treatment D_i, drawn from a Bernoulli experiment with probability q. The exposure map for each unit is defined as:

$$\psi_i(d) = (\psi_{i,1}(d), \psi_{i,2}(d)) = (M_i(d), N - M_i(d)),$$

where $M_i(d)$ represents the number of treated neighbors when $D_i = d$. Here, $M_i(d)$ is drawn from a truncated normal distribution on $[0, M]$ with mean $M p(d)$ and variance σ^2, where $p(d) = p_1^d p_0^{1-d}$. Potential outcomes are generated by $Y_i(d, s) = \theta_1 + \theta_2 d + \theta_3 \psi_{i,1}(d) + \theta_4 \psi_{i,2}(d) + \varepsilon_i$, where $\varepsilon_i \sim N(0, 1)$. Therefore, this DGP represents a linear-in-sums model without endogenous peer effect. Parameters are set by $\theta = (1, 2, 3, 4)', M = 10, \sigma = 5, p_1 = 0.26298, p_2 = 0.73701, q = 0.5$. The choice of θ, p_1, p_2 makes the true ATE as 6, DTE as 2, and ITE as 4. $\sigma = 5$ is for overlapping assumption. Table 1, and Table 2 show mean squared

errors of each estimator, which are defined as $\frac{1}{S}\sum_{s=1}^{S}(\hat{\theta}_s - \theta^{\star})^2$, where $\hat{\theta}_s$ is the estimate in sth replication, $\theta^{\star}$ is the true parameter value, and the number of replication is $S = 10,000$. This shows that the MSE, hence both bias and variance, of each estimator converges to zero with the theoretical rate of $N^{-1/2}$. Moreover, it presents that the error is small in a relatively small sample size. Next, Table 3, and Table 4 show the coverage rates of each estimator, which are defined as $C(\hat{\theta}, \theta) = \frac{1}{S}\sum_{s=1}^{S}\mathbb{1}\{\theta \in CI(\hat{\theta}_s)\}$, where $CI(\hat{\theta}_s) = [\hat{\theta}_s - 1.96\text{se}(\hat{\theta}), \hat{\theta}_s + 1.96\text{se}(\hat{\theta})]$ is the 95% confidence interval.

Table 1: Mean Squared Errors of Average Potential Outcomes

Design	N	$\mu(0)$	$\mu(0,1)$	$\mu(1,0)$	$\mu(1,1)$
1	500	0.032	0.0325	0.0439	0.0447
	1,000	0.0164	0.0165	0.0176	0.017
	5,000	0.0033	0.0032	0.0033	0.0034
	10,000	0.0016	0.0016	0.0017	0.0017

Table 2: Mean Squared Errors of Treatment Effects

Design	N	Δ	$\theta(1)$	$\theta(0)$	$\delta(1)$	$\delta(0)$
1	500	0.0648	0.0189	0.0201	0.0686	0.0701
	1,000	0.0329	0.0051	0.0047	0.0297	0.0299
	5,000	0.0064	0.0009	0.0009	0.0058	0.0058
	10,000	0.0033	0.0005	0.0005	0.003	0.003

Notes. MSEs are computed by 10,000 simulations. $MSE = \frac{1}{S}\sum_{s=1}^{S}(\hat{\theta}_s - \theta)^2$, where θ is the true value of parameters from the design.

Table 3: 95% Coverage Rates of Average Potential Outcomes

Design	N	$\mu(0)$	$\mu(0,1)$	$\mu(1,0)$	$\mu(1,1)$
1	500	0.9692	0.9813	0.9788	0.9721
	1,000	0.9759	0.986	0.9845	0.9744
	5,000	0.9773	0.9843	0.987	0.9765
	10,000	0.9774	0.9841	0.9847	0.9754

Table 4: 95% Coverage Rates of Treatment Effects

Design	N	Δ	$\theta(1)$	$\theta(0)$	$\delta(1)$	$\delta(0)$
1	500	0.9944	0.9959	0.9958	0.9971	0.9976
	1,000	0.9987	0.9999	0.9999	0.9997	0.9998
	5,000	0.9983	1	1	0.9998	0.9996
	10,000	0.9978	1	1	0.9993	0.9994

Notes. Coverage probabilities are computed by 10,000 replications. For the treatment parameters, the confidence intervals are computed by ignoring the asymptotic covariances of average potential outcomes, resulting in a conservative coverage.

5 Empirical Application

This section presents a simple empirical analysis to demonstrate how the decomposition proposed in Section 3 can be applied to real data. To estimate the treatment effects and decompose them, we require data that consists of a random experiment, outcome, and exposure map. In Korea, high school assignments are nearly random when students graduate from middle school. I utilize this random assignment to estimate the impact of attending coeducational high schools on academic performance.

The data used in this application comes from the Korean Education and Employment Panel II (KEEP II), collected by the Korean Research Institute for Vocational Education and Training (KRIVET). The population consists of second-year high school students in 2016. The initial sample includes 10,558 students from 416 schools.

When students graduate from middle school, they select the type of high school they wish to apply to. After making their choice, high school assignments are nearly random within each type and region, based on the student's residential address. This study leverages the exogenous variation from the random assignment of high schools.

There are five types of high schools in Korea. General high schools are the most common, and most students attending these schools aim to enter a university after graduation. Engineering high schools prepare students for immediate employment upon graduation. Special, Science, and Foreign Language high schools require entrance exams, so students attending these schools are not randomly assigned. In this application, I focus solely on general high

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schools.

I set two outcomes to assess students' academic performance. The first outcome is the student's relative grade within their school. In each high school, students are ranked across nine grade levels, with grade 1 representing the top 4% and grade 9 representing the bottom 4%.[5] The second outcome is an indicator of whether the student attends a university in Seoul. Since many of the top-ranked universities are located in Seoul, this outcome is intended to capture students' academic achievements.[6]

Table 5 shows the distribution of outcome based on school types and gender. The average relative grade for female students is 3.92 in all-female schools and 3.81 in coeducational schools. For male students, the average relative grade is 4.11 in all-male schools and 4.31 in coeducational schools. The percentage of female students entering universities in Seoul is 18.01% in all-female schools and 16.18% in coeducational schools, while for male students, it is 11.35% in all-male schools and 11.08% in coeducational schools. Thus female students outperform male students in both academic outcomes in the sample.

Table 5: Distribution of outcomes over types of high schools

Type	Gender	N	Y_1		Y_2	
			Mean	SD	Mean	SD
Single	Male	736	4.11	1.55	11.35	31.74
Single	Female	1,053	3.92	1.52	18.01	38.45
Both	Male	1,092	4.31	1.65	11.08	31.4
Both	Female	1,262	3.81	1.47	16.18	36.84
	Total	4208	4.02	1.55	14.44	35.16

The distribution of exposure is likely to differ between students attending single-gender schools and those attending coeducational schools. For instance, a female student assigned to a single-gender school will primarily interact with other female students, while in a coeducational school, there would be more opportunities to form friendships with male students. This pattern is evident in Table 6. The percentage of students reporting that they have only same-gender friends is 43% in all-male schools, 48% in all-female schools, and 27.9% in

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[5]The raw scores from the first wave of data are available.

[6]As nearly all students from general high schools attend university after graduation, simply attending university may not accurately reflect performance. Additionally, the precise ranking of each university is available in the data.

coeducational schools. The difference in this distribution appears to be significant between single-gender and coeducational schools.

Table 6: Distribution of friendships over types of high schools

	No friends (%)	Only same gender friends (%)	Both (%)
Male school	3.16	43.57	53.28
Female school	4.51	48.33	47.17
Combined	4.42	27.98	67.59
Total	4.22	34.88	60.9

This suggests that the distribution of same-gender and opposite-gender friends varies significantly depending on an individual's treatment status. Based on the findings in Table 6, I defined the following exposure map:

$$
\psi_i(d) = \begin{cases} 1 & \text{if } i \text{ has no friends when } D_i = d, \\ 2 & \text{if } i \text{ has only friends with same gender when } D_i = d, , d \in \{0,1\}. \\ 3 & \text{if } i \text{ has friends with both genders when } D_i = d, \end{cases}
$$

After cleaning the data by removing non-responses and errors, the final dataset includes 216 schools: 40 all-male schools, 53 all-female schools, and 123 coeducational schools, with a total of 4,208 students (1,850 male and 2,358 female).

Table 7 presents the estimated treatment effects. Y_1 represents relative grades, and Y_2 is the indicator of whether a student entered a university located in Seoul. For Y_1, there appear to be no indirect average treatment effects, but for Y_2, most direct and indirect effects are statistically significant.

Table 7: Estimation of Direct and Indirect Treatment effects

	Y_1			Y_2		
	Total	Male	Female	Total	Male	Female
ATE	0.08**	0.25**	-0.12**	-1.46**	-0.27**	-1.83**
$\delta(1)$	0.05**	0.24**	-0.16**	-1.27**	-1.05**	-1.33**
$\delta(0)$	0.11**	0.27**	-0.1**	-1.44**	-0.23**	-2.04**
$\theta(1)$	-0.03	-0.02	-0.02	-0.02	-0.04**	0.21
$\theta(0)$	0.03	0.01	0.05	-0.19**	0.78**	-0.51**

In this application, the exposure map consists of the number of same-gender and opposite-gender friends. The distribution of this exposure map is likely influenced by the underlying friendship networks and the gender of the students. However, Assumption 2 does not hold in this case, as potential outcomes and exposures would still be correlated, even after conditioning on treatment assignment, if potential outcomes are also influenced by gender. This represents a limitation of the current model, highlighting the need to incorporate covariates into the framework.

6 Conclusion

In this study, I proposed a method to decompose the treatment effect into direct and indirect effects using a potential outcomes framework in the context of social interactions, with treatments randomly assigned. An individual's potential outcome is influenced by their neighbors' treatment status through a correctly specified exposure map. Additionally, the underlying social network, which determines each individual's neighborhood, is assumed to be affected by the treatment as well, leading to different exposure distributions under different treatment statuses. Under the sequential ignorability assumption from mediation model literature, the distributions of potential counterfactual outcomes are identified, and corresponding frequency estimators are proposed. The consistency and asymptotic normality of these estimators is derived.

A key contribution of this study is the identification and estimation of treatment effects in the presence of social interactions, separating them into direct and indirect effects. An advantage of this model is that it does not require detailed knowledge of network formation

or the exact adjacency matrix representing network structures. Identifying indirect effects requires variation in the distribution of exposure values across different treatment statuses.

The proposed model can be extended to incorporate covariates. As noted in Section 5, Assumption 2 may be violated if there is a common factor influencing both potential outcomes and exposure. If this common factor is observable, the identification strategy can be adjusted to account for conditional moments, and a new estimation procedure will be required. Additionally, the exposure map must be carefully defined, as identification depends on overlapping exposure values.

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Appendix

A   Proofs

Proof of Lemma 1. Notice that Assumption 1 and Assumption 3 implies

$$\Pr(\psi_i(d) = s) = \Pr(\psi_i(d) = s | D_i = d) = \Pr(\psi_i = s | D_i = d) > 0.$$

Also, Assumption 1 implies $\Pr(Y_i(d, s) \leq y) = \Pr(Y_i(d, s) \leq y | D_i = d)$. It follows that

$$
\begin{aligned}
G^{d,s}(y) &\equiv \Pr(Y_i(d, s) \leq y) \\
&= \frac{\Pr(Y_i(d, s) \leq y | D_i = d) \Pr(\psi_i = s | D_i = d)}{\Pr(\psi_i = s | D_i = d)} \\
&= \frac{\Pr(Y_i(d, s) \leq y, \psi_i = s | D_i = d)}{\Pr(\psi_i = s | D_i = d)} && \text{by Assumption 2} \\
&= \Pr(Y_i(d, s) \leq y | \psi_i = s, D_i = d) \\
&= \Pr(Y_i \leq y | \psi_i = s, D_i = d). && \text{by (1)}
\end{aligned}
$$

The distributions of potential outcomes $Y_i(d, \psi_i(d'))$ are identifed as

$$
\begin{aligned}
F^{d,d'}(y) &\equiv \Pr(Y_i(d, \psi_i(d')) \leq y) \\
&= \sum_{s \in \Psi} \Pr(Y_i(d, s) \leq y | \psi_i(d') = s) \Pr(\psi_i(d') = s) && \text{by L.I.E.} \\
&= \sum_{s \in \Psi} \Pr(Y_i(d, s) \leq y | \psi_i = s, D_i = d) \Pr(\psi_i = s | D_i = d') && \text{by Assumption 1} \quad (3) \\
&= \sum_{s \in \Psi} \Pr(Y_i \leq y | D_i = d, \psi_i = s) \Pr(\psi_i = s | D_i = d') && \text{by (1)} \\
&= \sum_{s \in \Psi} G^{d,s}(y) \Pr(\psi_i = s | D_i = d').
\end{aligned}
$$

Therefore,

$$
\begin{aligned}
F^d(y) = F^{d,d}(y) &\equiv \Pr(Y_i(d, \psi_i(d)) \leq y) \\
&= \sum_{s \in \Psi} \Pr(Y_i \leq y | D_i = d, \psi_i = s) \Pr(\psi_i = s | D_i = d) \\
&= \Pr(Y_i \leq y | D_i = d). && \text{by L.I.E.}
\end{aligned}
$$

([3](#)) is because

$$\Pr(Y_i(d,s) \le y | \psi_i(d') = s) = \frac{\Pr(Y_i(d,s) \le y, \psi_i(d') = s | D_i = d')}{\Pr(\psi_i(d') = s | D_i = d')} \qquad \text{by Assumption 1}$$

$$= \frac{\Pr(Y_i(d,s) \le y, \psi_i = s | D_i = d')}{\Pr(\psi_i = s | D_i = d')}$$

$$= \frac{\Pr(Y_i(d,s) \le y | D_i = d')\Pr(\psi_i = s | D_i = d')}{\Pr(\psi_i = s | D_i = d')} \qquad \text{by Assumption 2}$$

$$= \Pr(Y_i(d,s) \le y | D_i = d')$$

$$= \Pr(Y_i(d,s) \le y | D_i = d) \qquad \text{by Assumption 1}$$

$$= \Pr(Y_i(d,s) \le y | D_i = d)\frac{\Pr(\psi_i = s | D_i = d)}{\Pr(\psi_i = s | D_i = d)}$$

$$= \frac{\Pr(Y_i(d,s) \le y, \psi_i = s | D_i = d)}{\Pr(\psi_i = s | D_i = d)}$$

$$= \Pr(Y_i(d,s) \le y | \psi_i = s, D_i = d).$$

$\square$

Proof of Proposition 1. By Lemma 1, expectations are identified as follows

$$E[Y_i(d,s)] = \int_{\mathbb{R}} y \, dG^{d,s}(y) = E[Y_i | \psi_i = s, D_i = d],$$

$$E[Y_i(d,\psi_i(d'))] = \int_{\mathbb{R}} y \, dF^{d,d'}(y) = \sum_{s \in \Psi} E[Y_i | D_i = d, \psi_i = s]\Pr(\psi_i = s | D_i = d'),$$

$$E[Y_i(d,\psi_i(d))] = \int_{\mathbb{R}} y \, dF^d(y) = E[Y_i | D_i = d].$$

$\square$

For the asymptotic result, I use the following Lemma that is an application of Stein's bound (Stein, 1972).

Lemma 2. *Let $\{X_i\}_{i=1}^N$ be a random variables with $E(X_i) = 0$ and $E(|X_i|^3) < \infty$. Let $G = (g_{ij}) \in \{0,1\}^{N \times N}$ be a dependency graph for $\{X_i\}$, that is if for all disjoint interval $I_1, I_2 \subset \{1,...,N\}$, we have $\{X_k : k \in I_1\} \perp \{X_\ell : \ell \in I_2\}$ whenever $G_{ij} = 0$ for all $i \in I_1$ and $j \in I_2$. Define $D_N = \max_{1 \le i \le N} \sum_{j=1}^N g_{ij} = \max_{1 \le i \le N} |N_i|$, the maximum degree of the dependency graph, where $N_i = \{j : g_{ij} = 1\}$. Next, define $\sigma_N^2 = \mathrm{Var}\left(\sum_{i=1}^N X_i\right)$ and $Z_N = \frac{1}{\sigma_N} \sum_{i=1}^N X_i$. Let F_N be distribution function for Z_N, and Φ be the distribution function of the standard normal distribution. Then, $d_W(F_N, \Phi) \le \frac{7D_N^2}{\sigma_N^3} \sum_{i=1}^N E|X_i|^3$.*

Proof of Proposition 2. Define $P(c) = \Pr(C_i = c) = \Pr(D_i = d, \psi_i = s)$, $P(c) = \Pr(C_i = c) = \Pr(D_i = d, \psi_i = s)$, $\hat{P}(c) = \frac{1}{N} \sum_{i=1}^{N} \mathbb{1}\{C_i = c\}$, $m(c) = E[Y_i(c)] = E[Y_i(d, s)]$, and $\hat{m}(c) = \frac{1}{N} \sum_{i=1}^{n} \mathbb{1}\{C_i = c\}Y_i = \frac{1}{N} \sum_{i=1}^{n} \mathbb{1}\{C_i = c\}Y_i(c)$. Let $X_i = \frac{V_i}{\sqrt{N}}$, $V_i = \mathbb{1}_i(d, s)Y_i(d, s) - P(d, s)E[Y(d, s)]$, $\sigma_N^2(d, s) = \text{Var}\left(\sum_{i=1}^{N} X_i\right)$, and $Z_N(d, s) = \frac{1}{\sigma_N^2(d,s)} \sum_{i=1}^{N} X_i$. Then, $E(X_i) = 0$. Assume $E|X_i|^3 < \infty$. Then,

$$\sigma_N^2(d, s) = \frac{1}{N} \sum_{i=1}^{N} \text{Var}(V_i) + \frac{1}{N} \sum_{i \neq j} \text{Cov}(V_i, V_j)$$

$$\leq \max \text{Var}(V_i) + \frac{1}{N} \sum_{i=1}^{N} \sum_{j \in N_i} \text{Cov}(V_i, V_j)$$

$$= \max \text{Var}(V_i) + D_N \max \text{Cov}(V_i, V_j),$$

and $Z_N = \sum_{i=1}^{N} X_i$. Then, we have $D = O(N^\delta)$ by assumption 5. Thus, $E[X_i] = \frac{1}{\sqrt{N}} \sum_{i=1}^{N} E[V_i] = 0$, and

$$\sigma^2 = \text{Var}(Z_N) = \sum_{i=1}^{N} \text{Var}(X_i) + \sum_{i \neq j} \text{Cov}(X_i, X_j)$$

$$= \text{Var}(V_i) + \frac{1}{N} \sum_{i \neq j} \text{Cov}(V_i, V_j)$$

$$= \text{Var}(V_i) + O(N^\delta) = O(N^\delta).$$

Also, by Lemma 2,

$$\sup_f |Ef(Z_N/\sigma) - Ef(Z)| \leq \frac{7D^2}{\sigma^3} \sum_{i=1}^{N} E|X_i|^3 \leq 7O(N^{2\delta})O(N^{-\frac{3}{2}\delta})O(N^{-\frac{3}{2}})N = 7O(N^{\frac{\delta}{2} - \frac{1}{2}}) \to 0$$

Let $\text{Var}(V_i) = \sigma_m^2$. Then, $\left|\frac{\sigma^2}{N} - \frac{\sigma_m^2}{N}\right| \to 0$. Therefore,

$$\left|\frac{Z_N}{\sigma} - \frac{Z_N}{\sigma_m}\right| = \left|\frac{\frac{1}{N} \sum_{i=1}^{N} V_i}{\sigma/\sqrt{N}} - \frac{\frac{1}{N} \sum_{i=1}^{N} V_i}{\sigma_m/\sqrt{N}}\right| = \left|\frac{1}{N} \sum_{i=1}^{N} V_i\right|\left|\frac{1}{\sigma/\sqrt{N}} - \frac{1}{\sigma_m/\sqrt{N}}\right| \to 0$$

Hence, for any 1-Lipschitz function f, we have $\left|Ef\left(\frac{Z_N}{\sigma}\right) - Ef\left(\frac{Z_N}{\sigma_m}\right)\right| \to 0$. By triangle inequality,

$$\frac{1}{\sigma_m} \sqrt{N}(\hat{m}(d, s) - m(d, s)) = \frac{1}{\sqrt{N}\sigma_m} \sum_{i=1}^{N} (\mathbb{1}_i(d, s)Y_i(d, s) - P(d, s)E[Y(d, s)]) \xrightarrow{d} N(0, 1),$$

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where $\sigma_m^2 = \text{Var}(V_i) = \text{Var}(\mathbb{1}_i(d,s)Y_i(d,s)) = P(d,s)E[Y_i(d,s)^2] - P(d,s)^2 E[Y_i(d,s)]^2$. Next, by the same argument for $V_i = \mathbb{1}_i(d,s) - P(d,s)$, we have

$$\frac{1}{\sigma_p}\sqrt{N}\left(\hat{P}(d,s) - P(d,s)\right) = \frac{1}{\sqrt{N}\sigma_p}\sum_{i=1}^{N}(\mathbb{1}_i(d,s) - P(d,s)) \xrightarrow{d} N(0,1),$$

where $\sigma_p^2 = \text{Var}(V_i) = \text{Var}(\mathbb{1}_i(d,s)) = P(d,s)(1 - P(d,s))$. Let $\boldsymbol{a} = (a_1, a_2) \in \mathbb{R}^2$. Then,

$$a_1\sqrt{N}\left(\hat{m}(d,s) - m(d,s)\right) + a_2\sqrt{N}\left(\hat{P}(d,s) - P(d,s)\right) = \frac{1}{\sqrt{N}}\sum_{i=1}^{N}a_1 V_{1i} + a_2 V_{2i},$$

where $V_{1i} = \mathbb{1}_i(d,s)Y_i(d,s) - P(d,s)E[Y(d,s)]$, $V_{2i} = \mathbb{1}_i(d,s) - P(d,s)$. Also note that $E[a_1 V_{1i} + a_2 V_{2i}] = 0$ and $E[|a_1 V_{1i} + a_2 V_{2i}|^3] < \infty$ and

$$\begin{aligned}
\sigma_{mp} &\equiv E[a_1 a_2 V_{1i} V_{2i}] \\
&= a_1 a_2 \text{Cov}(\mathbb{1}_i(d,s)Y_i(d,s), \mathbb{1}_i(d,s)) \\
&= a_1 a_2 P(d,s)E[Y(d,s)] - P(d,s)^2 E[Y(d,s)] \\
&= a_1 a_2 P(d,s)(1 - P(d,s))E[Y(d,s)].
\end{aligned}$$

Therefore, by Cramer-Wold device, we have

$$\sqrt{N}\begin{pmatrix} \hat{m}(d,s) - m(d,s) \\ \hat{P}(d,s) - P(d,s) \end{pmatrix} \to N(0, \boldsymbol{V}),$$

where

$$\begin{aligned}
\boldsymbol{V} &= \begin{pmatrix} \sigma_m^2 & \sigma_{mp} \\ \sigma_{mp} & \sigma_P^2 \end{pmatrix} \\
&= \begin{pmatrix} P(d,s)E[Y_i(d,s)^2] - P(d,s)^2 E[Y_i(d,s)]^2 & P(d,s)(1 - P(d,s))E[Y(d,s)] \\ P(d,s)(1 - P(d,s))E[Y(d,s)] & P(d,s)(1 - P(d,s)) \end{pmatrix}.
\end{aligned}$$

By MVT,

$$\begin{aligned}
\sqrt{N}(\hat{v}(d,s) - v(d,s)) &= \sqrt{N}\left(\frac{\hat{m}(d,s)}{\hat{P}(d,s)} - \frac{m(d,s)}{P(d,s)}\right) \\
&= \frac{1}{\tilde{P}(d,s)}\sqrt{N}\left(\hat{m}(d,s) - m(d,s)\right) - \frac{\tilde{m}(d,s)}{\tilde{P}(d,s)^2}\sqrt{N}\left(\hat{P}(d,s) - P(d,s)\right) \longrightarrow N(0, \Sigma),
\end{aligned}$$

where $\Sigma = \frac{\text{Var}(Y_i(d,s))}{P(d,s)}$. Next, consider $\hat{\mu}(d, d')$. Let $\Psi = (s_1, ..., s_K)$, and define

$$\hat{\boldsymbol{B}} = \begin{pmatrix} \frac{N(d',s_1)}{N(d')} & \frac{N}{N(d,s_1)} \\ & \vdots \\ \frac{N(d',s_K)}{N(d')} & \frac{N}{N(d,s_K)} \end{pmatrix}.$$

Then, $\hat{B} \to B$, where $B_k = \frac{\Pr(\psi_i = s_k | D_i = d')}{\Pr(\psi_i = s_k, D_i = d)}$. By the similar argument of using lemma and Cramer-Wold device, we have

$$\sqrt{N} \begin{pmatrix} \hat{m}(d, s_1) - m(d, s_1) \\ \vdots \\ \hat{m}(d, s_K) - m(d, s_K) \end{pmatrix} \xrightarrow{d} N(0, V_m),$$

where $(V_m)_{kk} = \sigma_m(d, s_k)^2 = P(d, s_k) E[Y(d, s_k)^2] - P(d, s_k)^2 E[Y(d, s_k)]^2$ and $(V_{k\ell}) = -P(d, s_k) P(d, s_\ell) E[Y(d, s_k)] E[Y(d, s_\ell)]$. Therefore,

$$\sqrt{N}(\hat{\mu}(d, d') - \mu(d, d')) = \hat{B} \sqrt{N} \begin{pmatrix} \hat{m}(d, s_1) - m(d, s_1) \\ \vdots \\ \hat{m}(d, s_K) - m(d, s_K) \end{pmatrix} \hat{B} \xrightarrow{d} N\left(0, V_\mu(d, d')\right),$$

where

$$
\begin{aligned}
V_\mu(d, d') &= B V_m B' \\
&= \sum_{s \in \Psi} \Pr(\psi_i = s | D_i = d')^2 \frac{\sigma(d, s)^2}{\Pr(D_i = d, \psi_i = s)} \\
&\quad + \sum_{s \neq s' \in \Psi} \Pr(\psi_i = s | D_i = d') \Pr(\psi_i = s' | D_i = d') E[Y(d, s)] E[Y(d, s')]
\end{aligned}
$$

Lastly, note that $\hat{\sigma}^2(d, s) = \frac{1}{N(d, s)} \sum_{i=1}^N \mathbb{1}_i(d, s) Y_i^2 - \hat{v}(d, s)^2$. Let $\hat{P}(d, s) = \frac{1}{N} \sum_{i=1}^N \mathbb{1}_i(d, s)$. Then, in the proof of proposition 2, we have $\hat{P}(d, s) \xrightarrow{p} P(d, s)$. Next, define $\hat{L}(d, s) = \frac{1}{N(d, s)} \sum_{i=1}^N \mathbb{1}_i(d, s) Y_i^2$. Observe that $\hat{\sigma}^2(d, s) = \frac{\hat{L}(d, s)}{\hat{P}(d, s)} - \hat{v}(d, s)^2$, and by the same argument of $\hat{m}(d, s)$ in the proof of proposition 2, we have $|\hat{L}(d, s) - L(d, s)| = O_p\left(N^{-1/2}\right)$. Therefore, by Slutsky's theorem and continuous mapping theorem, we have

$$\hat{\sigma}^2(d, s) = \frac{\hat{L}(d, s)}{\hat{P}(d, s)} - \hat{v}(d, s)^2 \xrightarrow{p} \frac{L(d, s)}{P(d, s)} - v(d, s)^2 = \sigma^2(d, s).$$

$\square$