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Causal Mechanism with Interference

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Abstract

This study investigates the identification and estimation of causal effects when individuals interact with each other. In this context, an individual's potential outcome is influenced by the treatment statuses of all individuals. Specifically, the treatment of others affects outcomes through "exposures," such as the number of treated friends, which generally depend on the underlying network structure. When the network is also impacted by the treatment, an individual's treatment affects the outcome both directly and indirectly—by altering the distribution of exposures. Since the exposure mediates the effect of the treatment on the outcome, it can be considered a mediator. This study decomposes these direct and indirect effects by applying a causal mediation analysis framework. The required observable variables are outcomes, treatment statuses, and exposures for each individual. The variation in exposures based on different treatment statuses is essential to identify these components separately. A simple nonparametric estimation procedure is proposed, and its performance is assessed using Monte Carlo simulations. This approach is then applied to an empirical setting to examine the impact of attending coeducational high schools on academic performance.

Keywords: Causal inference; Network change; Mediation Effects

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1 Introduction

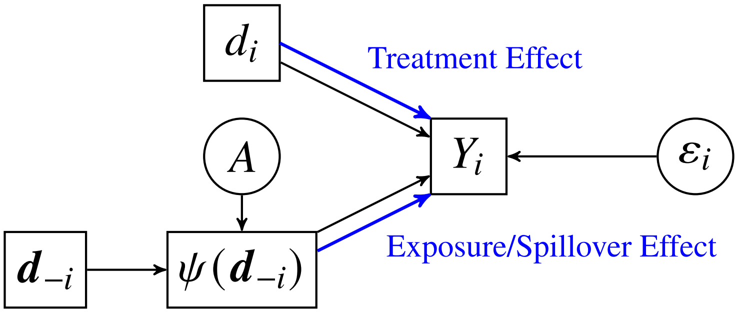
The identification and estimation of causal effects of a program or policy are of significant interest in economic analysis. Rubin's causal model (e.g., Rubin (1974), Imbens and Rubin (2010)) is commonly used to estimate causal effects. A key assumption in this framework is the *Stable Unit Treatment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

Consider a society of N individuals, each interacting with others in their neighborhood. The potential outcome for 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 $d_i \in \{0, 1\}$ represents the individual's own treatment assignment, $\boldsymbol{d}_{-i} \in \{0, 1\}^{N-1}$ represents the treatment assignments of the all other individuals, and ε_i is an error term. This model violates SUTVA if $Y_i(d_i, \boldsymbol{d}_{-i}) \neq Y_i(d_i, \boldsymbol{d}'_{-i})$ for some $\boldsymbol{d}_{-i} \neq \boldsymbol{d}'_{-i}$. This dependence on others' treatments results in each individual having 2^N potential outcomes. This leads to significant challenges in identifying treatment effects as the number of possible outcomes grows exponentially with the number of individuals.

However, the number of *effective* treatment is likely to be much smaller than 2^N. For example, the potential outcome might only 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 scenarios, which is considerably fewer than 2^N. Here, the number of treated friends can be thought of as 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 the treatment rule.

In this setting, causal effects can be defined in two ways. First, the treatment effect refers to the impact of an individual's own treatment status on the outcome, assuming exposures are fixed. Second, spillover or exposure effects refer to the impact of changes in exposures on the outcome, while keeping the individual's treatment status constant. This scenario is illustrated in Figure 1.

Figure 1: Direct and indirect effects



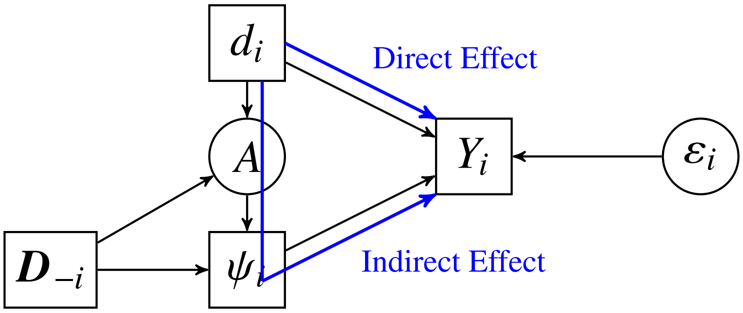
Recent studies emphasize the importance of spillover or exposure effects in program evaluations, as economic agents typically interact with others. As shown in Figure 1, the exposure map $\psi_i(\boldsymbol{d}_{-i})$ generally depends on not only the others' treatment $\boldsymbol{d}_{-i}$, but also the underlying network structure, represented by the $N \times N$ adjacency matrix $\boldsymbol{A}$. To incorporate the network, the exposure map can be redefined as $\psi_i : \{0,1\}^{N-1} \times \mathcal{A} \to \Psi$, where $\mathcal{A}$ represents the space of all possible networks with N nodes. For example, the exposure consists of the number of treated friends. The spillover effect then refers to the causal effect of changes in others' treatments via changes in exposure, while holding the network fixed or independent of the treatments.

However, empirical evidence suggests that the underlying network may also be affected by the treatment or the program (e.g., Dupas, Keats, and Robinson (2019), Comola and Prina (2021), Banerjee et al. (2024)). When the network structure is influenced by the treatment, the exposure depends indirectly on each individual's own treatment, as the potential exposure is now written as $\psi(\boldsymbol{d}_{-i}, \boldsymbol{A}(d_i, \boldsymbol{d}_{-i}))$. Let D_i and $\boldsymbol{D}_{-i}$ represent the treatment indicator for individual i and the treatment indicator vector for others, respectively. With a slight abuse of notation, we can define the potential exposure for individual i as $\psi_i(d_i) = \psi(\boldsymbol{D}_{-i}, \boldsymbol{A}(d_i, \boldsymbol{D}_{-i}))$. The causal effect on the outcome can then 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 effect*, which captures the impact of treatment when exposure remains unchanged. The second term represents the *indirect 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 effect measures the casual effect of the treatment when the number of treated friends are fixed. In contrast, the indirect effect captures the difference between potential outcomes when only the number of treated friends changes due to the own treatment. This situation is described in Figure 2.

Figure 2: Direct and indirect effects



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 statuses, and some exposures, making it highly advantageous in many of empirical situations.

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 outcomes. 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 that of 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) focuses 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 asymptotic theories considering the dependence. Vazquez-Bare (2023) derive general identification argument of this type of model and propose some estimators, where its asymptotic theory is derived based on the independence between groups.

One important issue is the potential misspecification of the exposure map. If the exposure map is incorrect, both identification and the corresponding estimators may be misleading. Vazquez-Bare (2023) shows that if the true exposure map is coarser than the one used in the estimator, the average potential outcomes are identified as usual frequency estimands, while a weighted average of these estimands is identified otherwise. Intuitively, a less coarse true exposure map contains richer information than a misspecified one. Leung (2022) uses the *approximately neighborhood interaction (ANI)* assumption to identify treatment parameters when the exposure map may be misspecified, assuming that potential outcomes are largely determined by neighbors within a close range. In this study, however, I assume the exposure map is correctly specified to focus on the relationship between exposure and treatment assignments.

Most studies in this literature assume that the underlying network is fixed or independent of treatment assignment, excluding the possibility that a policy may alter the structure of the underlying network. However, some studies consider this possibility. Comola and Prina (2021) propose a two-period model in which treatments are assigned after the first period, and the network can change in the second period. The outcome in each period follows the linear-in-means model (e.g., Bramoullé, Djebbari, and Fortin (2009)) 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, and the authors estimate the parameters using a similar instrumental variable strategy proposed by Bramoullé, Djebbari, and Fortin (2009). Applying this model requires complete information about the network adjacency matrices in both periods, which can often be difficult to obtain.

Potential outcome is expressed by a function of both an individual's own treatment status and exposures, and the exposures are generally determined by the other's treatment statuses and the underlying network structure. Therefore, if the underlying network is influenced by the all individuals' treatment, then the distribution of exposures are also influenced by the treatment statuses of all individuals. Looking at the own treatment status, it affect the outcome directly, as well as indirectly via exposures, i.e., exposures mediates the own treatment.

In the literature on mediation models, the goal is to understand how treatment influences potential outcomes by decomposing the total effect into direct and indirect components. 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 effect is the effect of treatment when the mediator is fixed, while the indirect effect measures how treatment affects the outcome through changes in the mediator. If both treatment assignment and mediator distributions are independent of potential outcomes, identifying direct and indirect effects becomes straightforward. Huber (2014, 2019) suggests sequential ignorability assumptions, which are less stringent than full independence. This study follows those assumptions for identification in this study.

Even when treatments are randomly assigned, outcomes and exposures may be dependent due to social interactions, complicating the derivation of asymptotic properties. In the case of independent random variables, Esseen's method (Esseen (1945)) is convenient to approximate normal distribution. For instance, Vazquez-Bare (2023) uses the Berry-Esseen bound to derive the asymptotic normality by assuming exposures are independent across groups. However, applying Esseen's method to dependent data is challenging. Instead, Stein's method (Stein (1972)) is commonly used for dependent data. For example, Chen and Shao (2004) provides a version of the central limit theorem with a bounded maximum degree of the dependency graph. Leung (2020) derives conditions for the moment of the dependency graph rather than directly using Stein's method. In this study, I assume boundedness of the maximum degree in the dependency graph and apply Stein's method.

2 Model

2.1 Setting and the exposure map

Consider a society consisting of N individuals, and let $D_i \in \{0,1\}$ be the binary treatment indicator for individual i. For any N-vector $\boldsymbol{V} = (V_1, \ldots, V_N)$, and for each i, let $\boldsymbol{V}_{-i}$ be the $(N-1)$-vector that excludes V_i from $\boldsymbol{V}$. Thus, the treatment statuses of all individuals in the society can be represented by the N-vector $\boldsymbol{D} = (D_1, \ldots, D_N)$, which is divided by $(D_i, \boldsymbol{D}_{-i})$, where D_i is individual i's treatment status, and $\boldsymbol{D}_{-i}$ is the treatment status of all other individuals. Each individual interacts with others through an underlying network structure. Let A_{ij} be an indicator of whether individuals i and j are friends, where $A_{ii} = 0$ for all i. The network can be represented by an $N \times N$ adjacency matrix $\boldsymbol{A}$ with $[\boldsymbol{A}]_{ij} = A_{ij}$.

Now, consider a potential treatment assignment $\boldsymbol{d} = (d_1, ..., d_N) \in \{0,1\}^N$ for all individuals, and again, denote $(d_i, \boldsymbol{d}_{-i})$ as individual i's treatment, and all other individual's treatment vector, respectively. The potential outcome for each individual needs to be defined by a function of the entire treatment vector $\boldsymbol{d}$ in general. However, I assume the other's treatment vector $\boldsymbol{d}_{-i}$ affect the potential outcome via an exposure map. Specifically, suppose there exists a known function $\psi : \{0,1\}^{N-1} \times \mathcal{A} \rightarrow \Psi$, where $\mathcal{A}$ is the space of networks of N nodes, and $\Psi \subset \mathbb{R}^K$ with $K < N$. This function satisfies the condition that the potential outcome for individual i is the same for any two treatment vectors $\boldsymbol{d}$ and $\boldsymbol{d}'$ as long as $d_i = d'_i$, and $\psi(\boldsymbol{d}_{-i}, \boldsymbol{A}) = \psi(\boldsymbol{d}'_{-i}, \boldsymbol{A})$.

This function, ψ, is an exposure map that provides a rule that links the other's treatment to an individual's potential outcome. In this study, I assume the exposure map is correctly specified.[1,2] Thus, the potential outcome for individual i is well defined by their own treatment status d_i and their exposure $s_i = \psi(\boldsymbol{d}_{-i}, \boldsymbol{A})$, i.e., we denote the potential outcome for individual i as $Y_i(d_i, s_i) = Y_i(d_i, \psi(\boldsymbol{d}_{-i}, \boldsymbol{A}))$. For example, Leung (2020) shows that if the network is anonymous and individuals interact with their neighbors within 1 network distance, then the potential outcome is determined by an individual's own treatment status d_i, the number of neighbors $\sum_j A_{ij}$, and the number of treated neighbors $\sum_j A_{ij} D_j$. In this case, the exposure map is given by $\psi(\boldsymbol{d}_{-i}, \boldsymbol{A}) = (\sum_j A_{ij}, \sum_j A_{ij} d_j)$.

The treatment effect is defined as the effect of changing an individual's own treatment status on the outcome, i.e., $Y_i(1, s) - Y_i(0, s)$, for a given exposure level s. The spillover or exposure effect is defined as the effect of a change in the exposure on the outcome, i.e., $Y_i(d, s') - Y_i(d, s)$, for some d, s', s. For example, Leung (2022) uses the exposure map $\psi(\boldsymbol{d}_{-i}, \boldsymbol{A}) = \mathbb{1}\left\{\sum_j A_{ij} d_j > 0\right\}$. 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. The treatment effect is interpreted as the usual causal effect of an individual's own treatment, while the exposure effect reflects the causal effect of changes in the treatment status of others, provided that the underlying network remains fixed.

Recent empirical studies suggest that the network can also be influenced by the program. For instance, Comola and Prina (2021) use experimental data from Nepal and find that provid-

[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.

ing savings accounts to households leads to changes in their network connections. Similarly, Dupas, Keats, and Robinson (2019) use experimental data from Kenya, where households were given free savings accounts. They observe that these households became less dependent on distant family members and more supportive of neighbors and friends within their village. Given this evidence, I assume that the network can also be altered by the treatment, in contrast to previous studies in the literature that assume a fixed or exogenous network.

To account for changes in the network due to the treatment, let $\boldsymbol{A}(\boldsymbol{d}) = \boldsymbol{A}(d_i, \boldsymbol{d}_{-i})$ represent the potential network based on the treatment assignment $\boldsymbol{d} \in \{0,1\}^N$ of all individuals. The exposure for individual i is then given by $\psi(\boldsymbol{d}_{-i}, \boldsymbol{A}(d_i, \boldsymbol{d}_{-i}))$. As a result, changes in exposure now reflect both the treatment status of others and changes in individual i's own treatment status. This suggests that the previously defined exposure effect not only captures the impact of others' treatment status but also includes changes in the network due to individual i's treatment. Consequently, the latter part of the exposure effect needs to be interpreted as an *indirect* treatment effect via changing the exposure level. This situation is described in Figure 2.

To focus on this additional treatment effect, by an abuse of notation, define $\psi_i(d) := \psi(\boldsymbol{D}_{-i}, A(d, \boldsymbol{D}_{-i}))$ for $d \in \{0,1\}$ as the potential exposure for individual i. The observed exposure for individual i is given by $\psi_i = \psi_i(D_i)$. Similarly, abusing notation again, we can define the potential outcome as $Y_i(d,d') := Y_i(d, \psi_i(d'))$, for $d, d' \in \{0,1\}$. The observed outcome is then $Y_i = Y_i(D_i, D_i)$, and therefore $Y_i(1,1)$ or $Y_i(0,0)$ is observed in the sample, while $Y_i(d,d')$ for $d \neq d'$ is never observed. can be viewed as a mediation model, as studied in Huber (2014, 2019), where the exposure $\psi_i(d)$ plays the role of a mediator.

The different distributions of $\psi_i(1)$ and $\psi_i(0)$ are important for the identification. However, if the distributions of $\psi_i(0)$ and $\psi_i(1)$ are identical,[3], the model simplifies to a potential outcomes framework with multiple treatments. In such a case, as discussed in Vazquez-Bare (2023) and Leung (2020), identification of treatment and exposure effects follows from standard independence assumptions. Furthermore, if the exposure map is a constant function, the model reduces to the classical causal model with SUTVA. Therefore, the framework is a generalization of existing methods.

[3] Or, if network is unaffected by the treatment.

2.2 Parameters of interest

This section defines the key parameters of interest in this paper. The potential outcome for an individual i is denoted as $Y_i(d, \psi_i(d))$. 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 ATE captures the average causal effect of the individual's own treatment, including both the direct effect of the treatment and the indirect effect through exposure. As discussed earlier, exposure can be thought of as a mediator of the treatment. Thus, drawing from the literature on causal mediation effects (e.g., Huber (2014, 2019)), the total treatment effect can be decomposed into direct and indirect effects as follows.

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

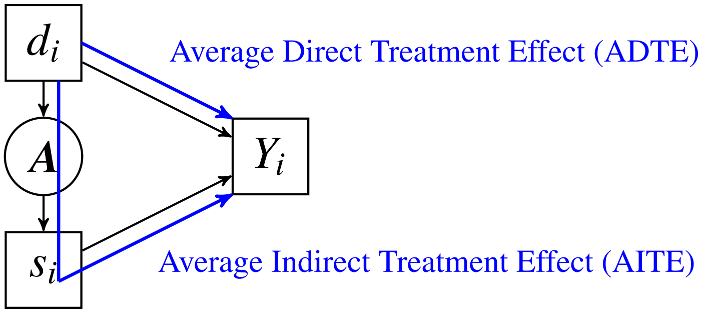
$$\delta(d) \equiv E[Y_i(d, \psi_i(1)) - Y_i(d, \psi_i(0))].$$

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

For a more detailed interpretation, consider the potential outcome is determined by a response function h, such that $Y_i(d, s) = h(d, s, \varepsilon_i)$, where $\varepsilon_i \sim F_{\varepsilon_i}$ is the individual-specific error that is independent of both the treatment and the underlying network. Moreover, let $F_{\boldsymbol{D}_{-i}}(\boldsymbol{d}_{-i}) := \Pr(\boldsymbol{D}_{-i} = \boldsymbol{d}_{-i})$ represent the distribution of other's treatment statuses, and for $d \in \{0, 1\}$, let $F_{\boldsymbol{A},d}(\boldsymbol{a}|\boldsymbol{d}_{-i}) := \Pr(\boldsymbol{A}(d, \boldsymbol{D}_{-i}) = \boldsymbol{a}|\boldsymbol{D}_{-i} = \boldsymbol{d}_{-i})$ denote the distribution of potential network links.

As described in Figure 2, the potential outcome $Y_i(d, \psi_i(d))$ is determined by the distributions $(F_{\varepsilon_i}(\cdot), F_{\boldsymbol{D}_{-i}}(\cdot), F_{\boldsymbol{A},1}(\cdot|\cdot), F_{\boldsymbol{A},0}(\cdot|\cdot))$, and therefore, ADTE and AITE measure the impact of one's own treatment, after integrating out those distributions. It can be simply described in Figure 3.

Figure 3: Average direct and indirect effects


d_i
$\boldsymbol{A}$
s_i
Y_i
Average Direct Treatment Effect (ADTE)
Average Indirect Treatment Effect (AITE)

Specifically, the ADTE can be written by:

$$\begin{aligned}&E[Y_i(\mathbf{1}, d) - Y_i(\mathbf{0}, d)]\\&= \int \left[\int \{h(\mathbf{1}, \psi(\boldsymbol{d}_{-i}, \boldsymbol{a}), e) - h(\mathbf{0}, \psi(\boldsymbol{d}_{-i}, \boldsymbol{a}), e)\} \underline{dF_{\boldsymbol{A},d}(\boldsymbol{a}|\boldsymbol{d}_{-i})} \right] dF_{\boldsymbol{D}_{-i}}(\boldsymbol{d}_{-i}) dF_{\varepsilon_i}(e).\end{aligned}$$

The difference between the inner expectations reflects the variation in potential outcomes due to changes in treatment status, while the network distribution remains fixed at $F_{\boldsymbol{A},d}(\boldsymbol{a}|\boldsymbol{d}_{-i})$. Similarly, the AITE is given by:

$$\begin{aligned}&E[Y_i(d, \mathbf{1}) - Y_i(d, \mathbf{0})]\\&= \int \left[\int h(d, \psi(\boldsymbol{d}_{-i}, \boldsymbol{a}), e) d \left(\underline{F_{\boldsymbol{A},\mathbf{1}}(\boldsymbol{a}|\boldsymbol{d}_{-i})} - \underline{F_{\boldsymbol{A},\mathbf{0}}(\boldsymbol{a}|\boldsymbol{d}_{-i})} \right) \right] dF_{\boldsymbol{D}_{-i}}(\boldsymbol{d}_{-i}) dF_{\varepsilon_i}(e).\end{aligned}$$

Here, the difference captures the change in the distribution of the network, from $F_{\boldsymbol{A},0}(\cdot|\boldsymbol{d}_{-i})$ to $F_{\boldsymbol{A},1}(\cdot|\boldsymbol{d}_{-i})$. Therefore, the AITE can be interpreted as the average causal effect of changes in the network on the potential outcome.

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 of the parameters ATE, AITE, and ADTE as defined in Section 2.2. To begin, assume we observe a random sample of $\{(Y_i, D_i, \psi_i) : 1 \leq i \leq N\}$. The observed outcome can be expressed as:

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

As discussed earlier, either $Y_i(1, \psi_i(1))$ or $Y_i(0, \psi_i(0))$ is observed, but both $Y_i(1, \psi_i(0))$ and $Y_i(0, \psi_i(1))$ are never observed. However, if the treatment is exogenous, we can identify the distributions of the potential exposures $\psi_i(1), \psi_i(0)$. This allows us to identify the average counterfactual outcomes by integrating $Y_i(d, \psi_i(d'))$ over the distribution of $\psi_i(d')$. The following are the identifying assumptions based on Huber (2014) for identifying causal mediation effects.

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 referred to as *sequential independence*. Note that Assumption 1 states that the potential outcome and potential exposure are independent of the treatment. The distribution of the potential outcome, given the individual's own treatment and exposure, is denoted by (d, s) and comes from the distribution of the unobserved individual error term ε_i. The potential exposure $\psi_i(d)$ of individual i's arises from the distribution of others' treatment $\boldsymbol{D}_{-i}$ and the potential network $\boldsymbol{A}(d, \boldsymbol{D}_{-i})$. Therefore, if the treatment is randomly assigned or exogenously given, i.e., $(\boldsymbol{Y}_i(d,s), \boldsymbol{A}(\boldsymbol{d})') \perp \boldsymbol{D}$ for all (d, s) and $\boldsymbol{d}'$, Assumption 1 is satisfied.

Assumption 2 requires independence between the potential outcome and potential exposure. The randomness of $Y_i(d,s)$ is determined by the distribution of individual error ε_i, while $\psi_i(d)$ is determined by others' treatment vectors and the potential network. Therefore, if the treatment is randomly assigned, this assumption implies independence between ε_i and the potential network $A(d, \boldsymbol{D}_{-i})$.[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.

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

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

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

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 $d \neq d'$ 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,*

$$\begin{aligned} \theta(0) &= \sum_{s \in \Psi} E[Y_i | D_i = 1, \psi_i = s] \Pr(\psi_i = s | D_i = 0) - E[Y_i | D_i = 0], \\ \delta(1) &= E[Y_i | D_i = 1] - \sum_{s \in \Psi} E[Y_i | D_i = 1, \psi_i = s] \Pr(\psi_i = s | D_i = 0), \\ \Delta &= \delta(1) + \theta(0) = E[Y_i | D_i = 1] - E[Y_i | D_i = 0]. \end{aligned}$$

$\theta(1)$ 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 $\tau(d,s,s') = E[Y_i | D_i = d, \psi_i = s'] - E[Y_i | D_i = d, \psi_i = s]$.

[4] 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 $\psi_i = \psi(D_i, \boldsymbol{D}_{-i}, X_i)$. If we observe X_i, then Assumptions 1 and 2 can be stated additionally conditional on X_i.

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:

$$
\begin{aligned}
\hat{\nu}(d,s) &= \frac{1}{N(d,s)} \sum_{i=1}^{N} \mathbb{1}_i(d,s) Y_i, \\
\hat{\mu}(d) &= \frac{1}{N(d)} \sum_{i=1}^{N} \mathbb{1}\{D_i = d\} Y_i, \\
\hat{\mu}(d,d') &= \frac{1}{N(d')} \sum_{j=1}^{N} \hat{\nu}(d,\psi_j) \mathbb{1}\{D_j = d'\} \\
&= \frac{1}{N(d')} \sum_{s\in\Psi} \sum_{j=1}^{N} \hat{\nu}(d,s) \mathbb{1}_j(d',s) = \sum_{s\in\Psi} \hat{\nu}(d,s) \frac{N(d',s)}{N(d')}.
\end{aligned}
$$

Here, $\nu(d,s)$ 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 $\hat{\tau}(d,s,s') = \hat{\nu}(d,s) - \hat{\nu}(d,s')$.

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., $g_{ij} = \mathbb{1}\{i = j, or\ C_i \not\perp C_j\}$. 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

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

respectively, by replacing $\mathrm{Var}(Y(d,s))$ 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 $Mp(d)$ 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 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.

[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.

Table 5: Distribution of outcomes over types of high schools

			Y_1		Y_2	
Type	Gender	N	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 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:

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

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^{**}

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

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

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

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

□

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

$$\begin{aligned}
E[Y_i(d,s)] &= \int_{\mathbb{R}} y dG^{d,s}(y) = E[Y_i | \psi_i = s, D_i = d], \\
E[Y_i(d, \psi_i(d'))] &= \int_{\mathbb{R}} y dF^{d,d'}(y) = \sum_{s \in \Psi} E\left[Y_i | D_i = d, \psi_i = s\right] \Pr(\psi_i = s | D_i = d'), \\
E[Y_i(d, \psi_i(d))] &= \int_{\mathbb{R}} y dF^{d}(y) = E\left[Y_i | D_i = d\right].
\end{aligned}$$

□

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 $I_1, I_2 \subset \{1, \ldots, N\}$, 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 $D_N = \max_{1 \leq i \leq N} \sum_{j=1}^N g_{ij} = \max_{1 \leq i \leq N} |N_i|$, 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, $d_W(F_N, \Phi) \leq \frac{7D_N^2}{\sigma_N^3} \sum_{i=1}^N E|X_i|^3$.*

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 $X_i = \frac{V_i}{\sqrt{N}}$, $V_i = \mathbb{1}_i(d,s)Y_i(d,s) - P(d,s)E[Y(d,s)]$, $\sigma_N^2(d,s) = \mathrm{Var}\left(\sum_{i=1}^{N} X_i\right)$, 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,

$$
\begin{aligned}
\sigma_N^2(d,s) &= \frac{1}{N}\sum_{i=1}^{N}\mathrm{Var}(V_i) + \frac{1}{N}\sum_{i \neq j}\mathrm{Cov}(V_i, V_j) \\
&\le \max \mathrm{Var}(V_i) + \frac{1}{N}\sum_{i=1}^{N}\sum_{j \in N_i}\mathrm{Cov}(V_i, V_j) \\
&= \max \mathrm{Var}(V_i) + D_N \max \mathrm{Cov}(V_i, V_j),
\end{aligned}
$$

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

$$
\begin{aligned}
\sigma^2 = \mathrm{Var}(Z_N) &= \sum_{i=1}^{N}\mathrm{Var}(X_i) + \sum_{i \neq j}\mathrm{Cov}(X_i, X_j) \\
&= \mathrm{Var}(V_i) + \frac{1}{N}\sum_{i \neq j}\mathrm{Cov}(V_i, V_j) \\
&= \mathrm{Var}(V_i) + O(N^{\delta}) = O(N^{\delta}).
\end{aligned}
$$

Also, by Lemma 2,

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

Let $\mathrm{Var}(V_i) = \sigma_m^2$. 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,

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

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

$$a_1\sqrt{N}(\hat{m}(d,s) - m(d,s)) + a_2\sqrt{N}\left(\hat{P}(d,s) - P(d,s)\right) = \frac{1}{\sqrt{N}}\sum_{i=1}^{N} a_1V_{1i} + a_2V_{2i},$$

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 $E[a_1V_{1i} + a_2V_{2i}] = 0$ and $E[|a_1V_{1i} + a_2V_{2i}|^3] < \infty$ and

$$\begin{aligned}\sigma_{mp} &\equiv E[a_1a_2V_{1i}V_{2i}]\\ &= a_1a_2\,\mathrm{Cov}(\mathbb{1}_i(d,s)Y_i(d,s), \mathbb{1}_i(d,s))\\ &= a_1a_2P(d,s)E[Y(d,s)] - P(d,s)^2E[Y(d,s)]\\ &= a_1a_2P(d,s)(1 - P(d,s))E[Y(d,s)].\end{aligned}$$

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

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

By MVT,

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

where $\Sigma = \frac{\mathrm{Var}(Y_i(d,s))}{P(d,s)}$. 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, $\hat{\boldsymbol{B}} \to \boldsymbol{B}$, 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

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

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

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

where

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

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

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

□