

Optimal Treatment Assignment Rules on Networked Populations

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Abstract

I study the problem of optimally distributing treatments among individuals on a network in the presence of spillovers in the effect of treatment across linked individuals. I consider the problem of a planner who needs to distribute of a limited number of preventative treatments (e.g., vaccines) for a deadly infectious disease among individuals in a target village in order to maximize population welfare. Since the planner does not know the extent of spillovers or the heterogeneity in treatment effects, she uses data coming from an experiment conducted in a separate pilot village. By placing restrictions on how others' treatments affect one's outcome on the contact network, I derive theoretical limits on how the data from the experiment could be used to best allocate the treatments when the planner observes the contact network structure in both the target and pilot village. For this purpose, I extend the empirical welfare maximization (EWM) procedure to derive an optimal statistical treatment rule. Under restrictions on the shape of the contact network, I provide finite sample bounds for the uniform regret (a measure of the effectiveness of a treatment rule). The main takeaway is that the uniform regret associated with EWM, extended to account for spillovers, converges to 0 at the parametric rate as the size of the pilot experiment grows. I also show that no statistical treatment rule admits a faster rate of convergence for the uniform regret, suggesting that the EWM procedure is rate-optimal.

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1 Introduction

According to a 2019 situation report by *Doctors without Borders*,¹ during the recent Ebola outbreak in the Democratic Republic of Congo, the WHO, faced with the possibility of shortage of vaccines, rationed its limited supply.² The report argues that this ad-hoc rationing of vaccines prolonged the adversity resulting from the outbreak. Recognizing the need to effectively distribute limited treatments, especially during an epidemic, this paper provides a method to estimate an optimal rationing rule in the presence of heterogeneous treatment effects and spillovers. Optimality here is defined as maximizing a population level notion of welfare, e.g., the expected number of uninfected members of the populations one year after the distribution of treatments.

The presence of heterogeneous treatment effects, whereby individuals respond differently to the same treatment, has important implications for the optimal allocation of a limited number of treatments. As an example, a treatment may be highly effective among elderly males, while being ineffective among young females. A policy-maker could leverage this fact to efficiently distribute the treatments within the population. Arguably, at least equally as important is the case where there are *spillovers* in the treatment effect. Suppose the treatment is for a deadly infectious disease; examples include a vaccine for Ebola, an insecticide treated bed net for malaria, and pre-exposure prophylaxis (PrEP) for HIV. Here, a treatment not only directly benefits those treated, but also subsequently restricts the spread of the disease to the untreated. This is what economists call a spillover in the treatment effect. These spillovers are often of large enough magnitude to warrant careful consideration by the policy-maker, particularly in the context of infectious diseases. Allowing for the spillover effect amounts to dropping the Stable Unit Treatment Value Assumption (SUTVA, Rubin (1980)) commonly maintained in the literature, according to which the outcome of one individual depends only on the treatment assigned to that individual and *not* on the treatments assigned to others. This assumption is violated for the case of preventative treatments for an infectious disease, a problem of great current interest, as well as in many other applications (e.g., textbook provision among peer networks in schools, or advertising/marketing on influence networks, conditional cash transfer program to encourage school attendance in Latin America). Especially when supplies are limited, policy-makers or public health officials can

¹Doctors without Borders. (2019, September 23). WHO rationing Ebola vaccines as outbreak still not under control in Democratic Republic of Congo. shorturl.at/jpBGN

²“WHO is restricting the availability of the vaccine in the field and the eligibility criteria and their application for reasons that are unclear ... We think that upping the pace of vaccination is necessary and feasible. At least 2,000-2,500 people could be vaccinated each day, instead of 500-1,000 as is currently the case.” - Dr. Isabelle Defourny, director of operations, *Doctors without Borders*.

maximize welfare by strategically distributing treatments to simultaneously account for the heterogeneity in treatments as well as the spillovers to the untreated. The main contribution of this paper is to establish optimality of a certain class of treatment assignment rules in the presence of heterogeneity and spillovers in treatment effects.

I model the problem as one of contagion on a network. I posit that individuals (or households) are arranged on a contact network where links between individuals represent interactions that facilitate a spillover in treatment effects. In the context of infectious diseases, links represent interactions through which the disease may be transmitted. Identifying treatment effects in the presence of spillovers on networks is infeasible without restrictions on either the types of networks or the types of spillovers permitted or both. Early work in this domain places no restrictions on spillovers but requires data from many independent networks (see Aronow, Samii (2017) and references contained therein).³ Leung (2020), on the other hand, places restrictions on the types of spillovers to enable identifying treatment effects under weaker restrictions on the network. In this paper, I make assumptions on both: the types of networks and the types of spillover. I impose restrictions such that the network has a bounded degree distribution (i.e., the maximum number of individuals to which the disease may be transmitted by a particular individual is small) and is highly clustered (i.e., two individuals that are linked to the same individual are likely to be linked to each other). While these assumptions restrict the generality of the model, they nonetheless allow for a wide range of applications while keeping the problem tractable. A notable similarity between Ebola, malaria, and HIV is that their respective contact networks can be assumed to have a bounded degree distribution.

I consider two separate models of spillovers in treatment effects. The first - local spillovers and exchangeability - assumes that the outcome of an individual depends not only on the treatment assigned to that individual but also on the number or share of its neighbors on the contact network that are assigned the treatment. Neither the identity of the treated neighbors, nor the treatment status among those not directly linked to the individual, affect its outcome. This model of spillovers is popular within economics and is often used in the peer effects literature. The second - a Susceptible-Infected-Recovered (SIR) based model of disease propagation - adapts to the network context the SIR model (Hethcote (1994)) from the epidemiology literature (see Ball, Britton, Leung, Sirl (2019) for references). In a

³ Many independent networks can also be thought of as a large single network with many independent groups of nodes such that there are links between nodes within a group but no links across groups (i.e., components). Thus, the assumption made on the network is that it can be divided in several independent components.

discrete time version of the compartmental model of disease propagation, members of the population (e.g., inhabitants of a village) are divided into three categories. *Susceptible* members of the population who are not yet infected but can become infected upon coming in contact with infected members. *Infected* members who are currently infected and can pass on this infection to those they are linked with on the contact network. *Recovered or Deceased* members who were infected but now are either recovered or deceased and cannot transmit the disease anymore. The probability that an individual transitions to the infected status in the next period depends on whether or not the individual is treated and the fraction of the individual's neighbors that are currently infected.

Throughout the paper, I use two examples to highlight the differences between the two models. To illustrate the local spillovers and exchangeability model of spillovers, I consider the case of malaria. Mosquitoes transmit malarial parasites from infected individuals to uninfected individuals. Mosquitoes also live in lush vegetation with stagnant water and are known to be poor flyers, not flying beyond 100 meters (see Cowman, Healer, Marapana, Marsh (2016) and references therein). Consequently, households can be considered to be linked on the contact network if they are geographically close to one another. This is because if members of one household have malaria, the mosquitoes can spread the malarial parasites to neighboring households. Presently, anti-malarial bed nets are the most cost effective preventative treatment for malaria and the spillovers associated with their use are well characterized by the local spillovers and exchangeability model.

Ebola, on the other hand, is transmitted among humans through close physical contact with infected individuals or objects that are used by them. Additionally, those infected show symptoms early on and are often incapacitated by the resulting symptoms. Consequently, Ebola typically spreads among family, friends, caretakers of infected individuals and workers in funeral and burial services. In particular, nurses and doctors at health care facilities are especially vulnerable (Chowell, Nishiura (2014)). Therefore, in the context of Ebola, individuals are assumed to share an edge on the contact network if they are family, or close friends, or health care workers, or funeral home workers assigned to the individual. An important distinguishing feature here is that the probability of an individual getting infected depends on its fraction of infected neighbors. Contrary to model 1, the spillovers to a unit in this framework operate through the evolution of the infection status of its those it is linked to on the contact network. While assigning a treatment to a linked unit does reduce the likelihood of that linked unit getting infected, it does not change the probability with which the unit gets infected conditional on the linked unit getting infected. Since communicable diseases

such as Ebola require person to person transmission, as opposed to malaria, I believe that the SIR based model is more realistic here.

Working within these frameworks, the main contribution of this paper is to construct a statistically valid estimator for the optimal treatment assignment rule in each settings. A treatment assignment rule specifies the treatment assignment to each individual in the network given the network topology as well as covariates of individuals (or households). The optimal treatment assignment rule maximizes some notion of welfare subject to constraints. For example, a planner might want to assign 20 vaccines to maximize the number of non-infected individuals in a village with 100 individuals. The planner faces an additional statistical challenge: it needs to estimate the heterogeneous treatment effects as well as the spillover effects. To do so, the planner has access to data from a pilot study conducted in a different village where the treatments are randomly assigned on an observed network. I assume that the pilot village has the same joint distribution of outcomes, covariates and links on the contact network as the target village.

I extend the empirical welfare maximization procedure, based on constructing an empirical analogue of the welfare function (in the above example, the estimated number of non-infected individuals) to account for spillovers. The optimal rule is then estimated by maximizing this empirical welfare function. I provide a finite sample bound on the uniform regret associated with the proposed method, i.e., the difference between welfare implied by the proposed rule and the highest welfare that can be attained in a non-arbitrarily complex class of treatment rules. I am able to demonstrate that under the previously discussed restrictions on the contact network, the uniform regret converges to 0 at the \sqrt{n} rate. This is the same rate of converges as attained in the absence of spillovers (see e.g., Kitagawa, Tetenov (2018)). I also provide a lower bound for the uniform regret in the local spillovers and exchangeability case. This demonstrates the optimality⁴ of the rate of convergence obtained by the proposed empirical welfare maximization procedure. A similar lower bound for the SIR based model of disease propagation is research in progress.

Methodologically, I build on the results in Kitagawa, Tetenov (2018) and Manski (2013). Specifically, I extend the empirical welfare maximization approach in Kitagawa, Tetenov (2018) to allow for spillovers. This entails extending their proof strategy to account for the dependence in individuals' outcomes induced by the effect of one individual's treatment on

⁴ No statistical treatment rule has a faster rate of convergence for uniform regret than the \sqrt{n} rate attained by empirical welfare maximization.

its linked individuals. A necessary ingredient in attaining the empirical welfare is the estimation of treatment effects. As discussed earlier, this is not feasible without added structure on either the types of networks or the types of spillovers permitted or both. The assumptions on the network I make are weaker than the many independent networks assumption in Aronow, Samii (2017) but stronger than the assumptions in Leung (2020). In keeping with the trade-off, I consider a broader class of models of spillovers than that in Leung (2020). However, this class of models is smaller than the unrestricted class of spillover models considered in Aronow, Samii (2017). In the language of Neyman-Rubin causal model, while each particular individual has a binary treatment status (either assigned a treatment or not), each individual has more than two potential outcomes, i.e., one for each possible allocation of treatments to itself and to others in the population. Owing to the numerous ways in which the treatments of others in the network might affect an individual, such problems are intractable without further structure. Manski (2013) lists a set of useful restrictions on the spillover effects that facilitate a parsimonious representation of the set of potential outcomes. This representation, defined as effective treatments, allows for identification and estimation of potential outcomes when treatments are experimentally assigned.

A second important contribution of this paper is that it provides results in the single growing network as well as the many networks asymptotic frameworks. Research on estimating treatment effects and spillovers on networks has largely studied settings where many networks are observed, with only a few recent examples of studies on a single large network. The many networks paradigm requires that the pilot study contain information on a large number of components. For example, a pilot study conducted over many villages with no links across villages. Recognizing that this is often very expensive, the single growing network paradigm posits that the experiment is conducted over one village instead.

This paper is structured as follows. Section 2 contextualizes its contributions within the broader literature. Section 3 sets up the problem and defines concepts in statistical decision theory. Assumptions on the network topology are detailed in section 4. Section 5 presents two separate models of spillovers in treatment effects. Section 6 lists restrictions on the data collected from the experiment and proposes an estimator for the welfare function. Section 7 sets up the empirical welfare maximization protocol and section 8 establishes the main results. I provide a numerical example in section 9. Finally, sections 10 and 11 discuss the broader applicability of the concepts introduced in this paper. All proofs are reported in the appendix.

2 Literature Review

This paper falls at the intersection of numerous literatures.

The first one is the literature on optimal statistical treatment rules. Within economics, the study of optimal treatment rules is not new, at least in settings without the spillover in treatment effects. The literature on statistical decision theory originated from Wald (1950) and Savage (1951). The premise of this literature is that data is used for solving a decision problem rather than for inference. This literature has substantially developed since then with seminal contributions coming from both the statistics and the computer science literature. See Chernoff, Yahav (1977) and Berger (2013) for useful references. Within economics, Chamberlain (2000) formulates the popular economic problem of decision making under uncertainty as a problem in statistical decision theory. In particular, he suggests Bayes procedures based on parametric models to construct a predictive distributions. Meanwhile, in a sequence of papers, Manski (1996, 2000, 2004) considers the decision problem of assigning treatments under unknown heterogeneity in treatment effects. Manski (2004) proposes minimax regret as a criterion for evaluating treatment rules. As characterizing the exact minimax regret optimal treatment rules can be challenging, he instead derives a finite sample bound on the uniform regret associated with the Conditional Empirical Success rule. Hirano, Porter (2009) instead circumvent these challenges by using approximations based on the limit of a sequence of Le Cam experiments. Stoye (2009) uses a game between the decision maker and an adversarial nature to derive the exact finite sample minimax regret optimal treatment rules for the same problems considered in Manski (2004). He finds that when the covariate space is large, minimax regret optimality prescribes no-data rules without restrictions on the outcome distribution conditional on covariates. This finding implies that the minimax regret need not converge to zero for a class of treatment rules that may be arbitrarily complex. To overcome this technical challenge, this papers follows the more recent literature by restricting the class of treatment rules to have finite VC dimension. In particular, I build on Kitagawa and Tetenov who use Empirical Welfare Maximization and demonstrates that the uniform regret associated with this treatment rule converges to 0 as the size of the experiment increases. My contribution is to allow for spillovers in treatments. More recently, Athey, Wager (2020) generalize the approach in Kitagawa, Tetenov (2018) to settings with observational data. While they do not provide finite sample results, they use the doubly-robust estimator and retain the \sqrt{n} rate of convergence for uniform regret. An alternate generalization of Kitagawa, Tetenov (2018) is Mbakop, Tabord-Meehan (2021) which allows for a data driven selection of a class of treatment rules from a sequence of

classes of treatment rules with VC dimensions $v_n \rightarrow \infty$ using a regularization method.

Presently, the only paper in this literature to allow for spillovers is Viviano (2019). The first important distinction between Viviano’s paper and mine is the scale at which the treatments are assigned. While Viviano (2019) suggests an individualistic assignment of treatments, I consider a population level assignment of treatments. This distinction has two important consequences: first, the framework I present is readily able to accommodate the case where the planner has to ration a finite number of treatments. Second, the framework presented here is better able to accommodate heterogeneity in the local network topology across different parts of the same network. This is because with a population level assignment of treatments, nodes with identical covariates and local network topologies are not forced to receive the same treatment in my framework. It is worth mentioning that these benefits come at the cost of increased computation complexity. Another distinction is that while both papers consider the local spillovers and exchangeability model, I also consider the SIR based model of disease propagation.

A second literature that my paper relates to is that on identification and inference of spillover effects. Manski (2013) discusses identification of treatment effects and spillover effects under a variety of assumptions. The early work on estimation and inference focuses on the case where data contains many independent networks (see Hudgens, Halloran (2008); Toulis, Kao (2013); Basse, Airoidi (2015)). Vazquez-Bare (2020) identifies spillover effects in an unrestricted model of spillovers and instead restricts the networks to be completely connected clusters. More recent work on estimation and inference focuses on data coming from one single network (see Liu, Hudgens (2014); Ogburn, Sofrygin, Diaz, van der Laan (2017); Aronow, Samii (2017)). Leung (2020) derives consistent estimators and standard errors for treatment and spillover effects under the restrictions of local spillovers and exchangeability. There is a trade-off between the restrictions on network topologies and restrictions on spillover effects in ensuring identification. Kojevnikov, Marmer, Song (2019) and Leung, Moon (2019) characterize aspects of this trade-off.

A related literature, initiated by Ballester, Calvó-Armengol, Zenou (2006), identifies key players in a network. They do so by studying games on networks with linear-quadratic utilities. They demonstrate conditions under which the key player (the one whose removal leads to the greatest change in aggregate activity) is the player with the highest Katz-Bonacich centrality. Subsequently, an active literature estimating these models using spatial autoregressive models has emerged (see, e.g., Lee (2007); Bramoullé, Djebbari, Fortin (2009)).

There are two key distinctions between these papers and the my work here. Firstly, while the key player literature models effort, I model disease propagation (contrary to effort) is not endogenous; however (similarly to effort) is jointly determined across individuals. Secondly, while this literature identifies the key player to remove from the network, I am interested in finding the optimal subgroup to treat when treatments themselves induce heterogenous treatment effects and spillover effects. Another critical difference is that treatments such as vaccines are scarce.

My paper relates also to the network epidemiological literature. Scholarly work in epidemiology recognized the benefits of considering a ‘network approach’ in studying the spread of STDs (see Klovdahl (1985) for an example involving AIDS). Social connections play a vital role in facilitating the spread of the HIV virus, which spreads from an infected individual to a susceptible individual through intimate physical contact (for example exchange of body fluids). Recognizing the role of such social connections in facilitating the spread of diseases, network epidemiologists have suggested ‘targeted immunization protocols’ to minimize the probability of an epidemic. This research has mainly focused on two situations. The first one amounts to showing that for general scale-free networks, even random immunization of unrealistically high density of individuals does not lead to global immunity. For example, Pastor, Satorras, Vespignani (2002) show that vaccinations based on connectivity hierarchy (targeting more connected nodes) sharply lower vulnerability to epidemics. The second amounts to comparing immunization protocols under local information. For example, Holme (2004) demonstrates the benefits of targeting nodes sequentially where one treats the most connected neighbors of individuals already treated in successive iterations. It is worth noting that much of this literature explores treatments that make the treated completely immune to the disease (analogous to node removal). To the contrary, I explore more general notions of treatment. Additionally, in my setting, the planner needs to first estimate treatment effects and spillovers.

Finally, my paper relates to the literature on influence maximization. This literature posits a model of disease propagation and analytically detects nodes with the highest influence on the propagation of disease in the network. As discussed above, in this literature often heterogeneity in treatment response is suppressed in favor of analytical tractability. Examples of this include Domingos, Richardson (2001), Kempe et al. (2003) , Banerjee et al. (2014) and Jackson, Storms (2019).

Apart from these, I use technical results from the empirical process theory literature as

well as the spatial dependence literature. These will be highlighted in-text.

3 Setup

3.1 Decision Problem

There are $J < \infty$ units (either individuals or households) in a *target* population. The planner needs to assign a limited number of treatments within this target population. Each unit $1 \leq j \leq J$ may either be assigned the treatment, recorded as $T_j = 1$; or not, recorded as $T_j = 0$.⁵ The population-level assignment of treatments is simply the $J \times 1$ vector of unit-level treatment assignments, \mathbf{T} . Suppose the planner observes a vector of pre-treatment unit-level covariates X_j supported on some set \mathcal{X} , for each unit $1 \leq j \leq J$. The population-level covariate matrix \mathbf{X} collects each individual unit-level covariate vector. Each unit is endowed with a stochastic treatment response function $Y_j(\cdot) : \{0, 1\}^J \rightarrow [-M, +M]$.⁶ This definition of the treatment response function nests two critical details. First, by allowing the treatment response to depend on the population-level assignment of treatments \mathbf{T} , I drop SUTVA. Second, I assume that the unit-level treatment response function (or outcome) has bounded support. Consequently, the set of unit-level potential outcomes without any additional restrictions on the nature of the spillovers is:

$$\mathcal{Y} \equiv \{Y_j(\mathbf{T}) : \mathbf{T} \in \{0, 1\}^J\} \quad (1)$$

I model the spillovers using a contact network. In the two distinct models of spillovers that I consider, I place restrictions (introduced later in section 5) on the nature of spillovers along edges of this network. The planner observes the *adjacency matrix* associated with this contact network, denoted \mathbf{A} . An adjacency matrix is a $J \times J$ matrix where the ij^{th} element \mathbf{A}_{ij} takes value 1 if units i and j are linked on the contact network; and \mathbf{A}_{ij} takes value 0 if units i and j are not linked. I assume that the adjacency matrix is undirected⁷ and unweighted.⁸ Consequently, $\mathbf{A} \in \mathcal{A}_J$ with $|\mathcal{A}_J| = \frac{J \times (J-1)}{2}$, the set of all pairs of $J \times J$ adjacency matrices that are undirected and unweighted.⁹ An implicit assumption I make is that the assignment of treatment does not induce any change in the contact network. Further, this paper also assumes that the networks are static and do not evolve over time.

⁵ This can easily be generalized to the case with many treatments.

⁶ Much of the previous literature in optimal treatment assignment assumes $Y_j(\mathbf{T}) = Y(T_j)$.

⁷ If unit i is linked to unit j on the network, then unit j is also linked with unit i . Formally, $\mathbf{A}_{ij} = \mathbf{A}_{ji}$.

⁸ A pair of units are either linked or not; there are no stronger or weaker links. Formally, $\mathbf{A}_{ij} \in \{0, 1\}$.

⁹ Following convention, I assume there are no self links. Formally, $\mathbf{A}_{jj} = 0$.

The planner must allocate a population-level treatment vector \mathbf{T} from a *feasible set of treatments* so as to maximize some notion of *welfare*. This feasible set of treatments depends on the number of treatments available $B \leq J$ and is defined as:¹⁰

$$\mathcal{T} \equiv \{\mathbf{T} \in \{0, 1\}^J : \sum_{j=1}^J T_j \leq B\} \quad (2)$$

This can be formalized as choosing a treatment rule $\phi : \mathcal{X}^J \times \mathcal{A}_J \rightarrow \mathcal{T}$ which assigns to any realization of the population-level covariate matrix \mathbf{X} and adjacency matrix \mathbf{A} , a population-level treatment assignment \mathbf{T} . In this paper, I assume that the welfare function is the average household level outcomes implied by the treatment rule. This is compatible with the objective of a utilitarian social planner maximizing expected outcomes. Hence, the planner solves:

$$W(\phi; \mathbf{P}) = \max_{\phi} \mathbb{E}_{\mathbf{P}} \left[\frac{1}{J} \sum_{j=1}^J \left[\sum_{\mathbf{T} \in \{0,1\}^J} Y_j(\mathbf{T}) 1\{\phi(\mathbf{X}, \mathbf{A}) = \mathbf{T}\} \right] \right] \quad (3)$$

The expectation is taken with respect to \mathbf{P} where $\{(Y_j(\cdot))_{j=1}^J, \mathbf{X}, \mathbf{A}\} \sim \mathbf{P}$. I refer to this as the idealized problem since this requires the planner to know the distribution \mathbf{P} .

Running example - malaria: The planner faces an outbreak of a deadly infectious disease, malaria, in a target village. In this simplified example, the village consists of two households ($J = 2$). Malaria is known to be transmitted primarily through mosquitoes, which carry malarial parasites from infected to uninfected individuals. Mosquitoes prefer to make their nest in lush vegetation with stagnant water. In the example, I assume that the planner observes the distance from household to vegetation as the covariate $X_j \sim U(0, 100)$ and IID across houses. Since mosquitoes are said to be poor flyers, not flying beyond a hundred meters, links on the contact network can be thought to be determined by geographical proximity. For example, the 2 houses are said to be linked if and only if the distance between the two houses is less than a hundred meters. I provide a visual illustration of this below.

Presently, the most cost-effective preventative treatment for malaria is the anti-malarial bed net. The planner has 1 bed net to distribute between the 2 houses. The heterogeneity in treatment effects arises because a house that is closer to vegetation has a higher probability of being infected with malaria. Therefore, houses that are closer to vegetation have a higher

¹⁰ There are $Q = \sum_{b=0}^B \binom{J}{b}$ treatments in \mathcal{T} indexed by $1 \leq q \leq Q$.

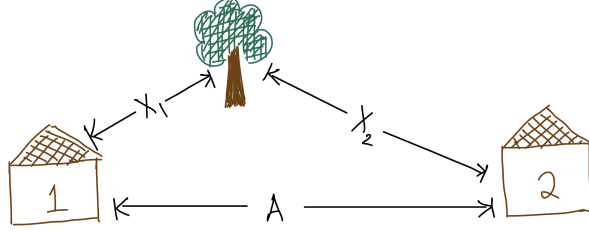


Figure 1: Target Village in Running Example

treatment response to bed nets. Moreover, when the houses are close to each other, i.e. $\mathbf{A}_{12} = 1$, and household 1 receives the bed net, mosquitoes can no longer transmit malarial parasites from infected member or carriers in household 1 to uninfected members in household 2. Therefore, household 2 also benefits when the planner assigns the bed net to household 1. In this example, the outcome $Y_j(\cdot)$ is a composite measure of health outcome of household j . The set of potential outcomes for house i are $\{Y_i(1, 1), Y_i(1, 0), Y_i(0, 1), Y_i(0, 0)\}$ and the feasible set of village-level treatment assignments is $\mathcal{T} = \{(1, 0), (0, 1), (0, 0)\}$. Consequently, the planners problem is:

$$\sup_{\phi: (X_1, X_2), \mathbf{A}_{12} \mapsto \mathbf{T}} \mathbb{E}_{\mathbf{P}} \left[\frac{1}{2} \left[\sum_{\mathbf{T} \in \{0,1\}^2} (Y_1(\mathbf{T}) + Y_2(\mathbf{T})) \cdot \mathbf{1}\{\phi((X_1, X_2), \mathbf{A}_{12}) = \mathbf{T}\} \right] \right] \quad (4)$$

Consider the following example for the treatment response function that reflects the above intuition. First note that in the event the two houses are far from each other, the outcome in house 1 depends only on whether or not it is assigned the bed net and its distance from vegetation. For any $T \in \{0, 1\}$, and $X_1 \in \mathcal{X}$:

$$Y_1(0, T) | (\mathbf{A}_{12} = 0) = \begin{cases} 0, & \text{with probability } 1 - \theta \\ 1, & \text{with probability } \theta \end{cases} \quad (5)$$

$$Y_1(1, T) | (X_1, \mathbf{A}_{12} = 0) = \begin{cases} 0, & \text{with probability } 1 - 2\theta \left(\frac{100 - X_1}{100} \right) \\ 1, & \text{with probability } 2\theta \left(\frac{100 - X_1}{100} \right) \end{cases} \quad (6)$$

When the houses are close to each other, spillovers play an important role as evidenced

by the following treatment response functions:

$$Y_1(0, 0) | (\mathbf{A}_{12} = 1) = \begin{cases} 0, & \text{with probability } 1 - \theta \\ 1, & \text{with probability } \theta \end{cases} \quad (7)$$

$$Y_1(0, 1) | (X_1, \mathbf{A}_{12} = 1) = \begin{cases} 0, & \text{with probability } 1 - \frac{4}{3}\theta \left(\frac{100 - X_1}{100} \right) \\ 1, & \text{with probability } \frac{4}{3}\theta \left(\frac{100 - X_1}{100} \right) \end{cases} \quad (8)$$

$$Y_1(1, 0) | (X_1, \mathbf{A}_{12} = 1) = \begin{cases} 0, & \text{with probability } 1 - 2\theta \left(\frac{100 - X_1}{100} \right) \\ 1, & \text{with probability } 2\theta \left(\frac{100 - X_1}{100} \right) \end{cases} \quad (9)$$

In this example, I will assume that the treatment response function is symmetric for house 2, i.e.,

$$Y_1(T, T') | (X_1 = X) \stackrel{d}{=} Y_2(T', T) | (X_2 = X) \quad (10)$$

Given this true data generating process (DGP), \mathbf{P} , I plot the optimal assignment of treatments in the covariate space $(X_1, X_2) \in [0, 100]^2$ separately for the case where the 2 houses are far from each other ($\mathbf{A}_{12} = 0$) and close to each other ($\mathbf{A}_{12} = 1$).

From the top panel, notice first that if the two houses are far from each other and over 50 meters from vegetation, it is optimal to treat neither household. One way to rationalize this is to consider the treatments often have side effects associated with them. In the context of insecticide treated bed nets that are treated, they are highly flammable. So, when the two houses are far from vegetation and each other, the risk of malaria is outweighed by the risk of the bed net catching on fire. The rest of the top panel shows that if at least one of the two houses is less than 50 meters away from vegetation, it is optimal to treat the house closer to vegetation. The main difference in the bottom panel is that the area where treating neither house is optimal. When the 2 houses are close to each other, mosquitoes can transmit malarial parasites across houses. Hence, the treatment effect on the treated house and the spillover on the untreated house exceed the risk of the bed net catching fire when the houses are between 50 and 67 meters from vegetation.

3.2 Statistical Treatment Rules

The crux of the planner's challenge is that she does *not* know the DGP \mathbf{P} , but is able to estimate the required features of \mathbf{P} from some data. In particular, I assume that the planner has

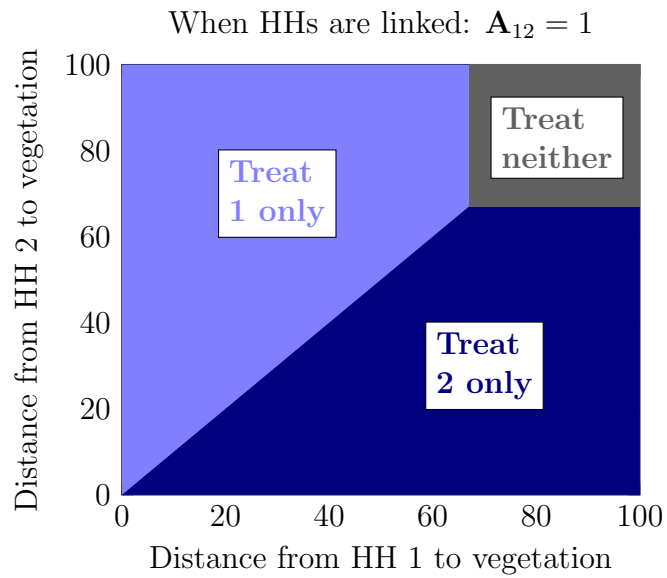
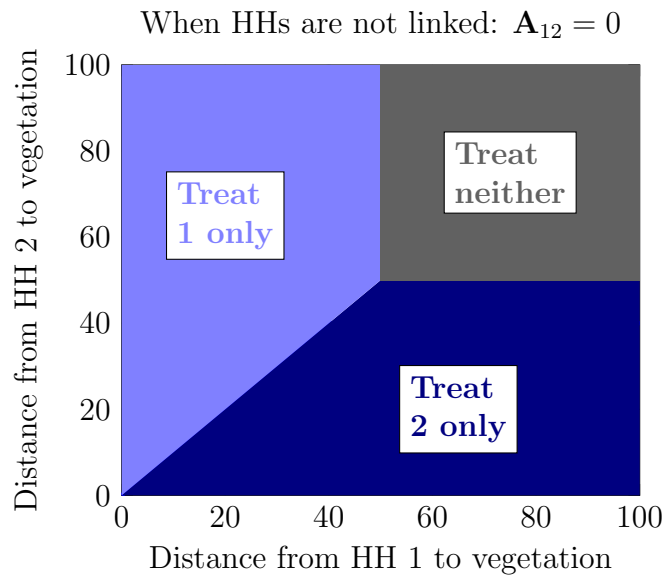


Figure 2: The optimal treatment rule under known DGP

access to data from an experiment conducted on a separate population. This separate population is termed the *pilot* population and contains n units. The associated population-level covariate matrix is denoted by $\tilde{\mathbf{X}} \in \mathcal{X}^n$ and the adjacency matrix is denoted by $\tilde{\mathbf{A}} \in \mathcal{A}_n$.¹¹ She also observes the $n \times 1$ vector of experimentally assigned treatment status across all units $\tilde{\mathbf{T}}$. Finally, she observes unit-level outcomes for each unit $\tilde{Y}_j, 1 \leq j \leq J$.¹² $\tilde{\mathbf{Y}}$ denotes the $n \times 1$ vector of unit-level outcomes. She uses this data $(\tilde{\mathbf{X}}, \tilde{\mathbf{A}}, \tilde{\mathbf{T}}, \tilde{\mathbf{Y}})$ to construct a *statistical treatment rule*.

Definition 1. *Statistical Treatment Rule.*

A statistical treatment rule is a mapping estimated from data that assigns a treatment vector $\mathbf{T} \in \mathcal{T}$, to each condition $(\mathbf{X}, \mathbf{A}) \in \mathcal{X}^J \times \mathcal{A}_J$.

$$Z(\cdot, \cdot | \tilde{\mathbf{X}}, \tilde{\mathbf{A}}, \tilde{\mathbf{T}}, \tilde{\mathbf{Y}}) : \mathcal{X}^J \times \mathcal{A}_J \rightarrow \mathcal{T} \quad (11)$$

For notational convenience, I will index the rules by the *decision sets* they generate. Define $Q \equiv \sum_{q=0}^B \binom{J}{q}$. For notational convenience, I suppress Z 's dependence on $(\tilde{\mathbf{X}}, \tilde{\mathbf{A}}, \tilde{\mathbf{T}}, \tilde{\mathbf{Y}})$.

Definition 2. *Decision Sets.*

G_1, \dots, G_Q is a *partition* of $\mathcal{X}^J \times \mathcal{A}_J$ such that:

$$\forall q, \{(\mathbf{X}, \mathbf{A}) \in \mathcal{X}^J \times \mathcal{A}_J : Z(\mathbf{A}, \mathbf{X}) = \mathbf{T}_q\} = G_q \quad (12)$$

Let \mathcal{G}_{Full} denote the set of all partitions of $\mathcal{X}^J \times \mathcal{A}_J$.

Definition 3. *Welfare Function for a Statistical Treatment Rule.*

For any \mathbf{P} and $\hat{G}_1, \dots, \hat{G}_Q$ that form a partition of $\mathcal{X}^J \times \mathcal{A}_J$ that may depend on the realization of data in the pilot population $(\tilde{\mathbf{X}}, \tilde{\mathbf{A}}, \tilde{\mathbf{T}}, \tilde{\mathbf{Y}})$, define:

$$W(\hat{G}_1, \dots, \hat{G}_Q; \mathbf{P}) = \mathbb{E}_{\mathbf{P}} \mathbb{E}_{\tilde{\mathbf{X}}, \tilde{\mathbf{A}}, \tilde{\mathbf{T}}, \tilde{\mathbf{Y}}} \left[\sum_{\mathbf{T} \in \{0,1\}^J} \left[\frac{1}{J} \sum_{j=1}^J Y_j(\mathbf{T}) 1\{Z(\mathbf{X}, \mathbf{A} | \tilde{\mathbf{X}}, \tilde{\mathbf{A}}, \tilde{\mathbf{T}}, \tilde{\mathbf{Y}}) = \mathbf{T}\} \right] \right] \quad (13)$$

Running example - malaria: In this example, the pilot village contains n households. The distance between house i and vegetation is recorded as \tilde{X}_i . The adjacency matrix of the

¹¹ Implicit in this statement is the assumption that the planner observes the full network in the pilot population.

¹² Realization of the stochastic treatment response function $\tilde{Y}_j(\cdot) : \{0, 1\}^n \rightarrow [-M, +M]$ evaluated at $\tilde{\mathbf{T}}$.

contact network in the pilot village is recorded in $\tilde{\mathbf{A}}$. The experimentally assigned treatment to house i is \tilde{T}_i . Finally, the composite measure of health outcome in house i is \tilde{Y}_i . The contact network in the pilot village may be very different from that in the target village. See Figure 2 for an example.

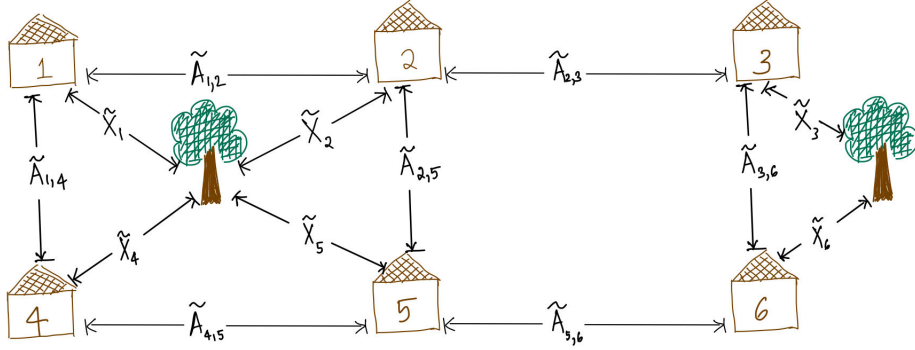


Figure 3: Target Village in Running Example

Here, I present an example of a statistical treatment rule in this context:

$$Z(\mathbf{X}, \mathbf{A} | \tilde{\mathbf{X}}, \tilde{\mathbf{A}}, \tilde{\mathbf{T}}, \tilde{\mathbf{Y}}) = \begin{cases} (1, 0), & \text{if } \mathbf{X} \hat{\alpha}_{\tilde{\mathbf{X}}, \tilde{\mathbf{A}}, \tilde{\mathbf{T}}, \tilde{\mathbf{Y}}} \geq 50 \\ (0, 1), & \text{if } \mathbf{X} \hat{\alpha}_{\tilde{\mathbf{X}}, \tilde{\mathbf{A}}, \tilde{\mathbf{T}}, \tilde{\mathbf{Y}}} < 50 \end{cases} \quad (14)$$

while $\hat{\alpha}_{\tilde{\mathbf{X}}, \tilde{\mathbf{A}}, \tilde{\mathbf{T}}, \tilde{\mathbf{Y}}}$ can be any statistic of the data $\tilde{\mathbf{X}}, \tilde{\mathbf{A}}, \tilde{\mathbf{T}}, \tilde{\mathbf{Y}}$, here I present this simple example:

$$\hat{\alpha}_{\text{data}} = \left(\frac{\sum_{i=1}^n \tilde{Y}_i (1 - \tilde{T}_i)}{n - \sum_{i=1}^n \tilde{T}_i}, \frac{\sum_{i=1}^n \tilde{Y}_i \tilde{T}_i}{\sum_{i=1}^n \tilde{T}_i} \right) \quad (15)$$

The $\hat{\alpha}_{\tilde{\mathbf{X}}, \tilde{\mathbf{A}}, \tilde{\mathbf{T}}, \tilde{\mathbf{Y}}}$ is a vector where the first element is the average outcome among houses in the pilot population who did not receive the treatment, and the second element is the average outcome among house that do receive the bed net. Suppose the planner receives some realization of data in the pilot village such that:

- HHs with bed nets are uninfected: $\tilde{T}_i = 1 \Rightarrow \tilde{Y}_i = 1$
- HHs without bed nets are infected: $\tilde{T}_i = 0 \Rightarrow \tilde{Y}_i = 0$

$$\Rightarrow \hat{\alpha}_{\tilde{\mathbf{X}}, \tilde{\mathbf{A}}, \tilde{\mathbf{T}}, \tilde{\mathbf{Y}}} = (0, 1)$$

Under this realization, the implied treatment decision is as depicted in the figure 4.

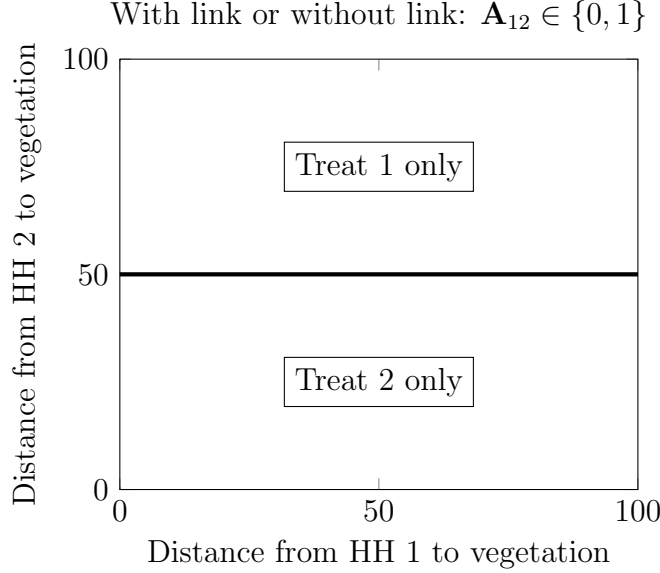


Figure 4: Decision sets implied by (14) when $\hat{\alpha} = (0, 1)$

In this paper, I also assume that the same stationary data generating process governs both, the target population, and the pilot population. This assumption allows the planner to use data from pilot population to the assign treatments in the target population.

Assumption 1. *Relating the DGP across target and pilot.*

- (a) Stationarity: For all $1 \leq j_1, \dots, j_k \leq J$ and for all $1 \leq i_1, \dots, i_k \leq n$, for any k ,

$$\left(Y_{j_p}(\cdot), X_{j_p}, \mathbf{A}(j_1, j_p), \dots, \mathbf{A}(j_k, j_p) \right)_{p=1}^k \sim \left(\tilde{Y}_{i_p}(\cdot), \tilde{X}_{i_p}, \tilde{\mathbf{A}}(j_1, j_p), \dots, \tilde{\mathbf{A}}(j_k, j_p) \right)_{p=1}^k \quad (16)$$

where $\mathbf{A}(i, j)$ represent the i, j th entry of the adjacency matrix.

- (b) Independence:

$$\left\{ \left(Y_j(\cdot) \right)_{j=1}^J, \mathbf{X}, \mathbf{A} \right\} \perp \left\{ \left(\tilde{Y}_i(\cdot) \right)_{i=1}^n, \tilde{\mathbf{X}}, \tilde{\mathbf{A}} \right\} \quad (17)$$

3.2.1 Assumptions on Class of Treatment Rules

Stoye (2009) demonstrates that when $\mathcal{X}^J \times \mathcal{A}_J$ is large, minimax regret optimality prescribes no-data rules without restrictions on the outcome distribution conditional on covariates. Essentially, this negative result stems from the fact that one can always find a probability

distribution such that the empirical welfare does not converge uniformly to the welfare function. This is because each term in expression (4) may individually converge, but the supremum may fail to do so, for a class of treatment rules that may be arbitrarily complex. To overcome this technical challenge, this paper follows the more recent literature by restricting the complexity of the class of treatment rules.

Here, following Kitagawa, Tetenov (2018), the notion of complexity used is that of *VC dimension*. This definition is presented in terms of the *decision sets* below:

Definition 4. *VC dimension (Vapnik, Chervonenkis (1971)).*

Suppose \mathcal{G} is a collection of subsets of $\mathcal{X}^J \times \mathcal{A}_J$. We say \mathcal{G} shatters a (finite) collection of point $S \equiv \{x_1, \dots, x_m\}$ in $\mathcal{X}^J \times \mathcal{A}_J$ if

$$|\{S \cap G : G \in \mathcal{G}\}| = 2^m$$

i.e. all 2^m combinations are recoverable. The *VC dimension* of \mathcal{G} is the cardinality of the largest collection of points that are shattered by \mathcal{G} .

The key insight exploited by Kitagawa, Tetenov (2018) in their work on estimating optimal treatment rules under SUTVA is to restrict the class of treatment rules to have finite VC dimension v . My paper builds on this assumption and modifies it to accommodate the multiplicity of population-level treatment assignments.

Assumption 2. *Partition with elements having finite VC dimension.*

A partition is denoted by $\mathbf{G} = \{G_1, \dots, G_Q\}$. \mathcal{G} denotes the collection partitions and is assumed to be:

$$\mathcal{G} \equiv \left\{ (G_1, \dots, G_Q) \in \mathcal{G}_{full} \text{ such that } \forall 1 \leq q \leq Q, G_q \in \mathbb{G} \right\} \quad (18)$$

where \mathbb{G} has finite VC dimension v and is countable.¹³ Denote the set of statistical treatment rules implied by these decision sets as \mathcal{Z} .

Consider the following example.

Example of class of statistical treatment rules. *Linear combination rules on pre-specified network statistics.*

¹³ This is done to ensure measurability and may be replaced with a more general condition such as the Image Admissible Suslin condition (Talagrand (1987)).

This example makes two simplifying assumptions:

1. The treatment rules depend on the network through L pre-specified network statistics, $\Psi_1(\cdot), \dots, \Psi_L(\cdot)$, where $\Psi_i : \mathcal{A}_J \rightarrow \mathbb{R}^J$. E.g., Degree (the number of links on the contact network), triadic closures (share of mutual friends that are linked themselves), Katz-Bonacich centrality,¹⁴ etc. for each unit in the population.

$$Z(\mathbf{X}, \mathbf{A}) = Z(\mathbf{X}, \Psi_1(\mathbf{A}), \dots, \Psi_L(\mathbf{A}))$$

Suppose further that $\mathcal{X} = \mathbb{R}^K$.

2. Linear combination rules can be expressed as a linear combination of linear scores, i.e.
 - (a) First construct a unit-level score:

$$S_j(\theta, \lambda) = \theta' X_j + \lambda' \Psi(\mathbf{A})_j$$

where $\Psi(\mathbf{A})_j$ is the j -th row of $(\Psi_1(\mathbf{A}), \dots, \Psi_L(\mathbf{A}))$.

- (b) Combine the unit-level scores to make a population level score:

$$Z(\mathbf{X}, \mathbf{A}) = \begin{cases} \mathbf{T}_1, & \text{if } \alpha_1 S_1(\theta, \lambda) > \max_{q \neq 1} [\alpha_q S_q(\theta, \lambda)] \\ \dots & \\ \mathbf{T}_Q, & \text{if } \alpha_Q S_Q(\theta, \lambda) > \max_{q \neq 1} [\alpha_q S_q(\theta, \lambda)] \end{cases}$$

In this example, \mathbb{G} is defined as:

$$\mathbb{G} \equiv \left\{ \mathbf{X}, \mathbf{A} \in \mathcal{X}^J \times \mathcal{A}_J : \alpha_q S_q(\theta, \lambda) > \max_{l \neq q} [\alpha_l S_l(\theta, \lambda)] \text{ such that } \alpha_1, \dots, \alpha_Q \in \mathbb{R}, \theta \in \mathbb{R}^K, \lambda \in \mathbb{R}^L \right\}$$

Since these linear rules yield decision sets that are themselves intersections of half space, we find that any partition G_1, \dots, G_Q constructed from the above linear rules, has finite VC dimension due to the following proposition. The proof is omitted for brevity.

Proposition 1. *Blumer et al. (1989).*

The set of all intersections of Q half spaces in \mathbb{R}^{K+L} has VC dimension, v , finite. Moreover,

$$v = O(Q(K + L) \log Q)$$

¹⁴ For a unit i in a population with contact network adjacency matrix \mathbf{A} : $c_i^{KB}(\mathbf{A}; \delta) = \sum_l \delta^l \sum_j \mathbf{A}_{ij}^l$. Where \mathbf{A}^l is the l -th power of \mathbf{A} .

For any series y_n and x_n , $y_n = O(x_n)$ implies there exists $C < \infty$ such that $\limsup_{n \rightarrow \infty} \frac{y_n}{x_n} \leq C$.

3.3 Evaluating Performance of Statistical Treatment Rules

To evaluate the performance of statistical treatment rules, I define the regret associated with any rule $Z \in \mathcal{Z}$.

Definition 5. *Regret.*

For any $Z \in \mathcal{Z}$ and any distribution \mathbf{P} ,

$$R(Z; \mathbf{P}) = \mathbb{E}_{\tilde{\mathbf{X}}, \tilde{\mathbf{A}}, \tilde{\mathbf{T}}, \tilde{\mathbf{Y}}} \left[\max_{Z \in \mathcal{Z}} W(Z(\cdot | \tilde{\mathbf{X}}, \tilde{\mathbf{A}}, \tilde{\mathbf{T}}, \tilde{\mathbf{Y}}); \mathbf{P}) - W(Z(\cdot | \tilde{\mathbf{X}}, \tilde{\mathbf{A}}, \tilde{\mathbf{T}}, \tilde{\mathbf{Y}}); \mathbf{P}) \right] \quad (19)$$

The regret associated with treatment rule Z given a distribution \mathbf{P} is a measure of the misallocation of treatments in the target population weighed by the penalty associated to the misallocation resulting from the rule Z when compared to the optimal treatment rule the planner would use if she knew the distribution \mathbf{P} .

Definition 6. *Uniform Regret in \mathcal{P}_o .*

For any set of probability distributions \mathcal{P}_o and any $Z \in \mathcal{Z}$,

$$R_{\mathcal{P}_o}(Z) = \sup_{P \in \mathcal{P}_o} R(Z; P) \quad (20)$$

In subsequent sections, for two different models of spillover effects, I define the associated \mathcal{P}_o .

Running example - malaria: Recall that the planner needs to assign 1 bed net between 2 houses in the target village (see Fig. 1). Since she doesn't know the distribution \mathbf{P} governing the treatment response function, she uses data from an experiment conducted in a pilot village (see Fig. 2). Suppose she uses the statistical treatment rule, Z , defined in equations (14) and (15) and realizes data such that $\hat{\alpha}_{\tilde{\mathbf{X}}, \tilde{\mathbf{A}}, \tilde{\mathbf{T}}, \tilde{\mathbf{Y}}} = (0, 1)$. In the figures below, I plot the decision rule implied by rule Z (blue line) and super-impose on to it the optimal treatment assignment the planner would use if she knew the true DGP \mathbf{P} (blue line). The dotted red region corresponds to the covariates on which the statistical treatment rule disagrees with the optimal treatment rule, hence the misallocation error.

The area of the dotted region corresponds to the misallocation error weighted by the penalty associated with that particular misallocation. The dotted area in figure 5 is a result

Data realization: $\hat{a}_{\tilde{\mathbf{X}}, \tilde{\mathbf{A}}, \tilde{\mathbf{T}}, \tilde{\mathbf{Y}}} = (0, 1)$

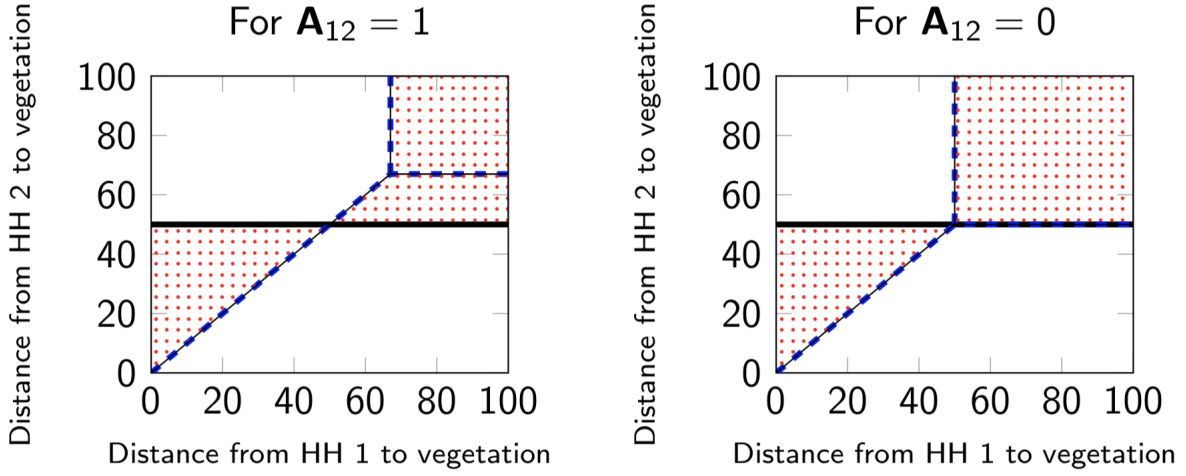


Figure 5:

of the particular realization of data $(\tilde{\mathbf{X}}, \tilde{\mathbf{A}}, \tilde{\mathbf{T}}, \tilde{\mathbf{Y}})$. The definition of regret above aggregates across the different realizations of data that the planner might receive.

4 Latent Space Representation of the Network

In modeling spillovers, I start by assuming the units of the population are arranged on a contact network. In this section, I clarify the assumptions required on this contact network. The main bite of these assumptions will be in controlling the amount of spillover induced correlation between units of the population. To see why this is a challenge, consider the simple network in the figure 6. Here, units i and j_1 maybe correlated due to the link be-

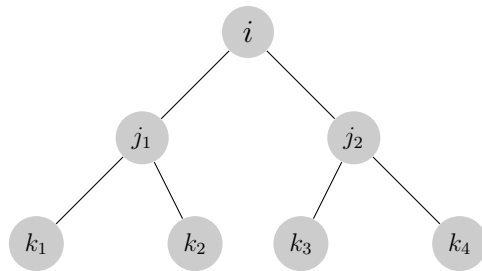


Figure 6: Correlation in Networks

tween them. Likewise, units j_1 and k_1 are also correlated. Consequently, units i and k_1 are

also correlated. Since units i and k_1 are further away on the network than units i and j_1 , one might expect the correlation between i and j_1 to be larger than that between i and k_1 . However, there are more units that are separated from i by 2 links (k_1, k_2, k_3, k_4) compared to units separated from i by 1 link (j_1, j_2). When using data coming from a single network, the planner needs to be careful to account for these spillovers.

For any unit i in the population, the correlation of i 's outcome with other units in the network can be decomposed into two parts:

1. the number of units that are separated from unit i by l links for $l = 1, 2, 3, \dots$,
2. the correlation between unit i and a unit separated by l links for $l = 1, 2, 3, \dots$.

This section details the assumptions along the first component while the next section discusses two classes of models for the second component. Here, I assume that the contact network can be represented as a random graph embedded on some latent lattice. Analogous assumptions are made in the spatial econometrics literature (see Conley (1992) and Lee (2007) for examples). Latent space models are an increasingly popular way in modeling network data (see Hoff, Raftery, Handcock (2002) and McCormick, Zheng (2015) for references). Kojevnikov, Marmer, Song (2020) discusses some of the limitations to modeling network data with latent space models.

Two important testable implications of the restriction I make here are that the resulting network will have a bounded degree distribution and a high clustering coefficient. A bounded degree distribution implies that the maximum number of links a unit has does not increase as the size of the total number of units in the population increases. A contact network between households in a village where links depend on geographical proximity trivially satisfies this. A counter-example is the contact network for influenza. Since individuals with flu are contagious before they exhibit symptoms and only minimum contact is required to transmit the disease, the maximum number of links for a unit is increasing in the size of the population. To see this, consider that a unit in a population like New York city using the public transit system can potentially spread the flu to a large fraction of the population. The second important implication of latent space models is the high clustering coefficient which suggests that a high proportion of any two links of a unit are themselves linked. Again, used to the triangle inequality associated with distance metrics, contact networks defined based on geographical proximity trivially satisfies this condition. A common counter example is the contact network for HIV-AIDS among heterosexual individuals. I formalize the exact

assumptions below.¹⁵

I assume that the units in either the target population or the pilot population are arranged on some underlying latent space. This space is referred to as an *Index Set*.

Definition 7. *Index set and potential nodes.*

Assume that there exists a d -dimensional integer lattice, \mathbb{Z}^d . The *potential nodes* are the set of all points within this set. Each potential node $\mathbf{k} \in \mathbb{Z}^d$ is endowed with a value of a unit-level covariate vector $X(\mathbf{k}) \in \mathcal{X}$.

A population of size m as observed by the planner will be a random draw of a set of m potential nodes from \mathbb{Z}^d .

Assumption 3. *Units are nodes located in the index set.*

The units within any population of size m are located on a stochastic subset of the potential nodes. Formally, the set of units is a realization of the random set \mathcal{D}_m which takes values in the index set. Formally,

$$\mathcal{D}_m \in \{\mathcal{D} : \mathcal{D} \subset \mathbb{Z}_m, |\mathcal{D}| = m\} \quad \text{w.p. } 1 \quad (21)$$

Suppose (Ω, \mathcal{S}) is a measurable space. Then, the formal measurability requirement for $\mathcal{D}_m : \Omega \rightarrow \{\mathcal{D} : \mathcal{D} \subset \mathbb{Z}_m, |\mathcal{D}| = m\}$ to be a set-valued random variable is stated as:

$$\forall D \subset \mathbb{Z}_m, \text{ such that } D \text{ is open, } \mathcal{D}_m^{-1}(D) \equiv \{\omega \in \Omega : \mathcal{D}_m(\omega) \cap D \neq \emptyset\} \in \mathcal{S} \quad (22)$$

I assume there exists a known capacity functional,¹⁶ μ_m , for \mathcal{D}_m , for any compact subset of \mathbb{Z}^d , K ,

$$\mu_m(K) = \mathbf{P}(\mathcal{D}_m \cap K \neq \emptyset) \quad (23)$$

The assumption that μ_m is known is important because the planner receives data containing a single draw of the network. Consequently, unless I specify a full network formation model (as opposed to the latent space representation here), the planner will not be able to

¹⁵ While a latent space representation does not rule out networks that result from a network formation game, the compatibility would need to be checked on a case by case basis. In the event of multiple pairwise stable equilibria in a network formation game, the latent space representation mandates a pre-specified selection mechanism.

¹⁶ See Molchanov, Molinari (2018) for application of random set theory in econometrics.

estimate the joint distribution of (\mathbf{X}, \mathbf{A}) .

Also note that the realization of \mathcal{D}_m is not observed by the planner. Instead, she observes an arbitrary identifiers for each unit, i.e., in a network of size m , the planner labels units as $\{1, \dots, m\}$. A consequence of assumption 3 is that there exists a unique mapping between the arbitrary labels and elements of the index set.

Definition 8. *Location mapping function for network of size n .*

$loc_m : \{1, \dots, m\} \mapsto \mathbb{Z}^d$ maps each unit i to a potential node on the index set when unit i is in network of size m . This location function (or m -dimensional vector) is itself a random variable, since it depends on \mathcal{D}_m .

I will now define a metric on the index set which will then be used to define links.

Definition 9. *Metric on the index set.*

$\forall \mathbf{k}, \mathbf{l} \in \mathbb{Z}^d$,

$$\rho(\mathbf{k}, \mathbf{l}) = \max_{1 \leq b \leq d} |k_b - l_b|$$

where k_b and l_b corresponds to the b -th component of \mathbf{k} and \mathbf{l} respectively.

Under the above definitions, I can define (a stochastic) distance between units i and j in a network of size n :

$$\rho_{ij}^m = \rho(loc_m(i), loc_m(j)) = \max_{1 \leq b \leq d} |(loc_m(i))_b - (loc_m(j))_b| \quad (24)$$

This paper assumes that units share an edge on the contact network if and only if the corresponding potential nodes to which they are mapped are close enough on the latent space. This is formalized below.

Assumption 4. *Edge formation on contact network.*

For a network of size m , assume that units i and j are connected iff $\rho_{ij}^m \leq \bar{\rho}$ for some $\bar{\rho} > 1$.

$$\mathbf{A}_{ij} = 1\{\rho_{ij}^m \leq \bar{\rho}\} \quad w.p. 1$$

The asymptotic framework considers a sequence of networks indexed by the number of nodes they contain, m . In particular, an observation of a population of size m corresponds to observing the entire set of nodes and links for that population. The idea is that as m

increases, the planner observes the entire set of nodes and links for a larger population. Contrast this to the case of snowball sampling where the planner observes m units within a large (possibly infinite) population. In snowball sampling, an increase in n corresponds to observation of a larger number of nodes and their links on the same population. This is formalized in assumption 5.

Assumption 5. *Super-population Sampling Frame.*

For any network of size m , \mathcal{D}_m is the complete population within that network.

The following lemma presents a key implication of the latent space representation - the bounded degree distribution.

Lemma 1. *Bounded degree distribution*

Under assumptions 3,4 and 5, for any population of arbitrary size n , the maximum degree of any node in the contact network is bounded above by:

$$(1 + 2\bar{\rho})^d - 1$$

I now make a strong assumption of stationarity to ensure that populations of different sizes are not distributed differently.

Assumption 6. *Strong stationarity.*

For any $m \leq n$, define the conditional random variable $\mathcal{D}_m(n) = \mathcal{D}_m | \{\mathcal{D}_m \subset \mathcal{D}_n\} \sim \mu_{m|n}$. I assume that with probability one, $\mu_{m|n}$, which is the distribution of $\mathcal{D}_m(n)$ is equal to μ_m .

Denote the distribution of (\mathbf{X}, \mathbf{A}) in a population of size m implied by the above latent space representation as $F(\cdot, \cdot; m)$. This will be used in defining the welfare function in section 7.

4.1 Alternate Framework: Increasing Number of Networks Asymptotics

As an alternate framework, I also consider the case where the planner conducts the experiment across many different pilot populations that are independent of one another. I present this case to demonstrate the flexibility afforded to the planner when she has such rich data. In particular, no additional assumptions on spillover effects are required. In this asymptotic framework, the planner observes the adjacency matrix, covariate vector, experimental

treatment assignment and outcomes for N networks $(\tilde{\mathbf{A}}^{(i)}, \tilde{\mathbf{X}}^{(i)}, \tilde{\mathbf{T}}^{(i)}, \tilde{\mathbf{Y}}^{(i)})_{i=1}^N$. For notational simplicity, I will assume that each pilot population has J nodes, the same size as the target population.

Assumption 7. *No cross population spillovers.*

There are no links between nodes of different villages. In addition, the outcomes in one population is independent of treatment assignments in the other populations.

Assumption 8. *IID populations.*

$(\tilde{\mathbf{A}}^{(i)}, \tilde{\mathbf{X}}^{(i)}, \tilde{\mathbf{T}}^{(i)}, \tilde{\mathbf{Y}}^{(i)})_{i=1}^N$ is IID across i .

5 Models of Spillover in Treatment Effects

5.1 Key Concepts

I adapt the definition of effective treatments suggested by Manski (2013). I begin up setting up the relevant notation in this subsection and adapt them to separate models of spillovers in the following subsections.

Definition 10. *Effective treatment.*

Define $r_j(\cdot; \mathbf{A}) : \{0, 1\}^J \rightarrow \mathcal{R}_j(\mathbf{A})$ to be a function that maps a population-level treatment assignment \mathbf{T} , given the contact network \mathbf{A} , into an implied effective treatment for agent j . The set $\mathcal{R}_j(\mathbf{A})$ is the set of possible effective treatments for unit j given the network. Effective treatments satisfy the property that for any units j and k , and for any $(\mathbf{T}, \mathbf{A}), (\mathbf{T}', \mathbf{A}') \in \{0, 1\}^J \times \mathcal{A}_J$ such that $r_j(\mathbf{T}; \mathbf{A}) = r_k(\mathbf{T}'; \mathbf{A}')$, it holds that $Y_j(\mathbf{T})|\mathbf{A} \stackrel{d}{=} Y_k(\mathbf{T}')|\mathbf{A}'$.

Example 1: *With SUTVA.*

Under the common assumption of SUTVA, the set of effective treatments are $\mathcal{R}_j(\mathbf{A}) = \mathcal{R} \equiv \{0, 1\}$ and the effective treatment mapping is $r_j(\mathbf{T}; \mathbf{A}) = T_j$.

Example 2: *Without any restriction on spillovers.*

For a population with J individuals, the assignment of treatments across all other units could affect outcome of unit j . Consequently, there exists no $r_j(\cdot; \mathbf{A})$ that satisfies definition 9.

Definition 11. *Homogenized effective treatment.*

Define $c_j(\cdot; \mathbf{A}, \mathbf{X}) : \{0, 1\}^J \rightarrow \mathcal{C}_j(\mathbf{A}, \mathbf{X})$ a function that maps a population-level treatment assignment \mathbf{T} , given the contact network \mathbf{A} and covariate vector \mathbf{X} , into an implied homogenized effective treatment for agent j . The set $\mathcal{C}_j(\mathbf{A}, \mathbf{X})$ is the set of possible effective treatments for unit j given the network characteristics. Homogenized effective treatments satisfy the property that for any unites j and k , and for any $(\mathbf{T}, \mathbf{A}, \mathbf{X}), (\mathbf{T}', \mathbf{A}', \mathbf{X}') \in \{0, 1\}^J \times \mathcal{A}_J \times \mathcal{X}^J$ such that $c_j(\mathbf{T}; \mathbf{A}, \mathbf{X}) = c_k(\mathbf{T}'; \mathbf{A}', \mathbf{X}')$, it holds that $Y_j(\mathbf{T})|\mathbf{A}, \mathbf{X} \stackrel{d}{=} Y_k(\mathbf{T}')|\mathbf{A}', \mathbf{X}'$.

5.2 Model 1: Local Spillovers and Exchangeability

Assumption 9. *Local spillover and exchangeability*

The outcome of unit j depends only on the treatment assigned to itself as well as the number of treatments assigned among units it is linked with on the contact network. Then,

$$\forall j, \mathcal{R}_j(\mathbf{A}) = \{0, 1\} \times \left\{ 0, \dots, \sum_i \mathbf{A}_{ij} \right\}^2$$

and

$$\forall j, r_j(\mathbf{T}; \mathbf{A}) = \left(T_j, \sum_i \mathbf{A}_{ij} T_i, \sum_i \mathbf{A}_{ij} \right)$$

This particular assumption has received a lot of attention within the economics literature, owing to its tractability. Consider the following example that economically motivates this assumption. Suppose the planner wants to distribute textbooks in a classroom in an attempt to raise test scores. The planner is aware of study groups that form within these classes and has access to this data in the form of the adjacency matrix \mathbf{A} of the social network. Further, the planner also observes the matrix of covariates for all students in the classroom \mathbf{X} . Here, the above assumption implies that the outcome (test score) of unit j only depends on whether or not node j receives a textbook, and the number/share of study partners of unit j that receive the textbook. Thus, this assumption implies that the identity of the study partners that receive the textbook are inconsequential to unit j 's outcome.

This next assumption restricts how the covariates of other units affects the outcome of a given unit. Here, I assume that unit j 's outcome are independent of the covariates of all other units (\mathbf{X}_{-j}) conditional on its own covariate and effective treatment. This can easily be weakened to allow unit j 's outcome to depend on the average covariates among units with which it is linked. Doing so makes the notation more cumbersome without adding any anything to the exposition, so I refrain from presenting it here. This assumption does

rule out some interesting behavior. In the above textbook example, suppose one covariate measures kindness (willingness to share textbook if assigned one) of the student. Then, it is possible that student j 's test score would depend on the number of its friends that receive the textbook and are kind. Such a mechanism is ruled out by the assumption below.

Assumption 10. *No contextual heterogeneity in treatment effects*

For any j , $\mathbf{T} \in \mathcal{T}$, and for any $\mathbf{A}, \mathbf{X} \in \mathcal{A}_J \times \mathcal{X}^J$, assume that

$$Y_j(\mathbf{T}) \perp\!\!\!\perp \mathbf{X}, \mathbf{A} \mid X_j, r_j(\mathbf{T}; \mathbf{A}) \quad (25)$$

In addition, I make one final assumption on the dependence across units of the population. In particular, I assume that for any two units i and j that are not directly linked (i.e., $\mathbf{A}_{ij} = 0$), their treatment response function and covariates are independent conditional on the treatment response function and covariates of mutually shared links. This is formalized in assumption 11.

Assumption 11. *Dependence structure within a population*

For all i, j that are not linked ($\mathbf{A}_{ij} = 0$)

$$(Y_i(\cdot), X_i) \perp\!\!\!\perp (Y_j(\cdot), X_j) \mid \{(Y_k(\cdot), X_k) : \forall k \text{ with } \mathbf{A}_{ik}\mathbf{A}_{kj} = 1\} \quad (26)$$

In the context of the textbook example, this rules out a population-level (i.e., classroom level) fixed effect, among other things. Thus, features like teacher quality which might effect the test scores of all units within the population cannot belong to the set of covariates as they will be correlated across all units. This restriction will be vital for the identification of the mean treatment responses when the planner observes data from a single pilot population.

Running example - malaria: Returning to the example of assigning bed nets for malaria, consider the following treatment response function for house i :

$$Y_i(\mathbf{T}) \mid \mathbf{A}, \mathbf{X} = T_i + X_i \cdot ((1 - T_i)s_i(\mathbf{T}; \mathbf{A})(1 - \beta)) + \epsilon_i \quad (27)$$

where $s_i(\mathbf{T}; \mathbf{A})$ represents the share of unit i 's links in contact network \mathbf{A} that receive the treatment, i.e.,

$$s_i(\mathbf{T}; \mathbf{A}) = \begin{cases} 1, & \text{if } \sum_{j \neq i} \mathbf{A}_{ij} = 0 \\ \frac{\sum_{j \neq i} A_{ij} T^j}{\sum_{j \neq i} \mathbf{A}_{ij}}, & \text{else} \end{cases} \quad (28)$$

Assume ϵ_i is an idiosyncratic error that is independent of $\mathbf{A}, \mathbf{T}, \mathbf{X}$. This model suggests that house i 's outcome is guaranteed to be 1 if it receives the bed net. If the house does not receive the bed net, its outcome is increasing in both: its distance from vegetation, and the fraction of the neighboring houses using the bed net. Assumption 11 states that the outcomes, covariates and links of two houses that are not neighbors conditional on the outcomes, covariates and links of mutual neighbors, are independent.

In this example, it is trivial to establish that, $r_j(\mathbf{T}; \mathbf{A})$ is the effective treatment to house j . This is because, for any $(\mathbf{T}, \mathbf{A}), (\mathbf{T}', \mathbf{A}') \in \{0, 1\}^J \times \mathcal{A}_J$ such that $r_j(\mathbf{T}; \mathbf{A}) = r_j(\mathbf{T}'; \mathbf{A}')$ implies $T_j = T'_j$ and $s_j(\mathbf{T}; \mathbf{A}) = s_j(\mathbf{T}'; \mathbf{A}')$. Consequently, $Y_j(\mathbf{T})|\mathbf{A} \stackrel{d}{=} Y_j(\mathbf{T}')|\mathbf{A}'$.

5.3 Model 2: SIR Based Model of Disease Propagation

I model the propagation of an infectious disease as a variant of the SIR model which has been studied extensively in the epidemiology literature (for example, see Rusu (2015)). I consider a discrete time formulation of the model where at each time period, units of the population are divided into three categories. *Susceptible* units of the population are not yet infected but can become infected upon coming in contact with infected units. *Infected* units are currently infected and can pass on this infection to those they are linked with on the contact network. *Recovered or Deceased* units are those that were infected but now have either recovered or deceased and cannot transmit the disease anymore. The probability that a unit transitions to the infected status in the next period depends on whether or not the unit is treated and the fraction of the unit's links that are presently infected.

There have been several modifications to this specification, including SIS (Susceptible-Infected-Susceptible), where an infected unit returns to being susceptible once it recovers. This is a more reasonable specification to model flu and flu-like diseases. In the context of HIV-AIDS or Ebola, I find the SIR based model to be more suitable. I consider preventative treatments such as drugs that limit the spread of the disease. Specifically, the treatment is assumed to reduce the probability that a unit gets infected on coming in contact with other infected units. Examples include the Ervebo vaccine for Ebola and the Pre-Exposure Prophylaxis (PrEP) drug for HIV. These treatments are not perfectly effective and their effectiveness depends on the characteristics of the unit being treated.

In this setting I assume that a unit's outcome depends on the category (S-I-R-D) to which the unit belongs κ periods after the assignment of the treatment. Here, κ is assumed

to be fixed, finite and known by the planner. One such example for the unit-level outcome is whether or not the unit remains *susceptible* 10 periods after the assignment of the treatment. Consequently, welfare maximization with this outcome corresponds to maximizing the number of units that do not get infected within the 10 periods, or equivalently minimizing the number of infections within the 10 periods. The restriction that κ is finite implies that the planner cannot use any infinite horizon objective function. In particular, it rules out the problem of minimizing the total number of infections within the population over an infinite horizon. While such problems may indeed be of interest, I would like to highlight that especially in the context of vaccinating against infectious diseases, such infinite horizon problems are often unrealistic for two reasons. First, the dynamic nature of vaccine and treatment development means that their efficacy improve over time. So, the planner will usually need to assign the existing treatment in order to minimize the number of infections until a more effective vaccine or treatment is available. Second, long-lived endemic diseases often mutate over time rendering previously known vaccines ineffective. This naturally limits the window over which the treatments assigned by the planner has any effect.

Additionally, the choice of κ will have implications on the extent of the spillovers in this model. To see this, consider two units, a and b , that are separated by at least $\kappa + 1$ links on the contact network. Suppose that a is initially infected but b is not. Note that there is no way for b to acquire an infection from an infectious path originating at a within κ periods. This is because, in each period, the infection can only be transmitted to the direct links of the currently infected units.

Let $t \in \{0, 1, \dots, \kappa\}$ denote the time periods. Here period will be defined with respect to the length of the infection. Suppose $s_j^{(t)} \in \{S, I, R, D\}$ denotes the state of the j^{th} unit at time t . $\mathbf{s}^{(t)}$ is the $J \times 1$ vector of states of all units within the population at time t . It will be convenient to denote by $I_j^t = 1\{s_j^{(t)} = I\}$. I assume that the planner observes the initial infection state of each node I_j^0 as part of the pre-treatment covariate for the unit.¹⁷ I will abuse notation slightly to write X_j as the vector of pre-treatment covariates excluding the initial infection status. Details of the S-I-R-D model are now presented. For any unit j :

$$\mathbf{P}(s_j^{(t+1)} = D \mid \mathbf{s}^{(t)}, s_j^{(t)} = I, T_i) = \theta; \quad \mathbf{P}(s_j^{(t+1)} = R \mid \mathbf{s}^{(t)}, s_j^{(t)} = I, T_i) = 1 - \theta \quad (29)$$

¹⁷ At $t = 0$ no unit is deceased or recovered category.

This states that the mortality rate of the disease is θ . Further, for any $L \in \{R, D\}$

$$\mathbf{P}(s_j^{(t+1)} = L | \mathbf{s}^{(t)}, s_j^{(t)} = L, T_i) = 1 \quad (30)$$

implying that *Recovered* and *Deceased* are absorbing states. Now, to consider the transmission of the infection,

$$\mathbf{P}(s_j^{(t+1)} = I | \mathbf{s}^{(t)}, s_j^{(t)} = S, T_j, X_j) \equiv \zeta_j^{(t)} = \begin{cases} \frac{\sum_k \mathbf{A}_{jk} 1_{\{s_k^{(t)}=I\}}}{\sum_k \mathbf{A}_{jk}} \cdot [p(X_j) - T_j q(X_j)], & \text{if } \sum_k \mathbf{A}_{jk} \neq 0 \\ 0, & \text{if } \sum_k \mathbf{A}_{jk} = 0 \end{cases} \quad (31)$$

with the restriction that $0 \leq q(x) \leq p(x)$ for all x . $p(\cdot)$ corresponds to the susceptibility of the unit which is the probability that a unit all of whose links are infected gets infected; $q(\cdot)$ corresponds to the treatment response, which is the reduction in the probability with which a treated unit all of whose links are infected gets infected. This structure can be conveniently annotated as follows.

Assumption 12. *SIRD of disease propagation.*

The probability that unit j transition to state $s_j^{(t+1)}$ at time $t + 1$ given the population-level state vector at t , $\mathbf{s}^{(t)}$ is given by:

$$\mathbf{P}(s_j^{(t+1)} | \mathbf{s}^{(t)}, T_i, \mathbf{A}, \mathbf{X}) = \begin{array}{c} S \\ I \\ R \\ D \end{array} \left\| \begin{array}{cccc} S & I & R & D \\ 1 - \zeta_j^{(t)} & \zeta_j^{(t)} & 0 & 0 \\ 0 & 0 & 1 - \theta & \theta \\ 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & 1 \end{array} \right\|$$

The dependence of these transition probabilities of node j on the states of nodes other than j means that the evolution of states of each individual node can be thought of as a non-homogeneous Markov chain.

Assumption 13. *Independent state transitions.*

The evolution of states over time is independent across units.

$$\forall \mathbf{s}^{(t+1)} \in \{S, I, R, D\}^J, \mathbf{P}(\mathbf{s}^{(t+1)} | \mathbf{s}^{(t)}, \mathbf{T}, \mathbf{A}, \mathbf{X}) = \prod_{j \leq J} \mathbf{P}(s_j^{(t+1)} | \mathbf{s}^{(t)}, \mathbf{T}, \mathbf{A}, \mathbf{X})$$

Note here that this assumption does not restrict *susceptible* units who share the exact same neighbors from having the similar probabilities of transitioning into the *infected* state. However, the event that one of the units transitions into *infected* state at time t is independent of the transition of the other unit at time t .

Next, I focus on defining the homogenized effective treatments implied by this model. Contrary to model 1, the spillovers in this framework operate through the evolution of the states at the unit-level. Consider an illustration using the figure 7 below.



Figure 7: Spillovers Propagate through Evolutions of States

In the illustration, unit 1 is initially infected while units 2 and 3 are initially susceptible. Suppose that the unit-level outcome is whether or not the unit remains *susceptible* 2 periods after the assignment of the treatment. The outcome of unit 3 depends not only on whether or not it receives the vaccine but also on the whether unit 2 is vaccinated. This is because, when unit 2 is vaccinated, it has a lower probability of getting infected in the first period. Bear in mind that this treatment assigned to unit 2 does not change the probability that node 3 gets infected in period 2 conditional on node 2 getting infected in period 1. In summary, while assigning a treatment to unit 2 does reduce the likelihood of the unit 2 getting infected, it does not change the probability with which the unit 3 gets infected conditional on unit 2 getting infected.

To get a sense of the homogenized effective treatment implied by this model, it is useful to consider a more complex illustration, see the figure 8.



Figure 8: Homogenized Effective Treatments

In this illustration, units 4 and 5 are initially infected while units 1, 2 and 3 are initially susceptible. Suppose again that the unit-level outcome is whether or not the unit remains *susceptible* 2 periods after the assignment of the treatment. The outcome to unit 1 depends on the number of paths of length 2 or less that originating from an infected unit and ending in unit 1. These paths are weighted by the probability that the infection transmits along

them. This is formalized below. I start by defining paths on the network.

Definition 12. $g(i, d; \mathbf{A})$ - *paths*.

Define $g(i, d; \mathbf{A})$ on network \mathbf{A} to be the set of paths in to i with path length d .

$$\mathbf{g}(i, d; \mathbf{A}) = \{(k_1, \dots, k_d) : \mathbf{A}_{ik_1} = \mathbf{A}_{k_1k_2} = \dots = \mathbf{A}_{k_{d-1}k_d} = 1\}$$

The lemma below formally spells out the homogenized effective treatment implied by this model.

Lemma 2. *Homogenized effective treatments under SIRD.*

Under assumptions 12 and 13, the effective treatments can be characterized as follows:

$$c_i(\mathbf{T}; \mathbf{A}, \mathbf{I}^0, \mathbf{X}) = \left(F_i, (F_{k_1} : k_1 \in g(i, 1; \mathbf{A})), ((F_{k_1}, F_{k_2}) : (k_1, k_2) \in g(i, 2; \mathbf{A})), \dots, \right. \\ \left. ((F_{k_1}, \dots, F_{k_{\kappa-1}}) : (k_1, \dots, k_{\kappa-1}) \in g(i, \kappa - 1; \mathbf{A})) \right)$$

where, $\forall j$,

$$F_j = \left(\sum_k \mathbf{A}_{jk}, \sum_k \mathbf{A}_{jk} I_k^0, X_j, T_j, I_j^0, 1\{S_j^{(0)} = D\} \right) \quad (32)$$

The remainder of this subsection places restrictions on the dependence across units within the population, analogous to assumption 11 in the previous model. In order to do so, I first define neighborhoods around units in the population.

Definition 13. k degree neighborhood of unit i : \mathcal{N}_i^k .

A node j is said to be a k degree neighbor of node i if they are connected by a path on length k .

$$\text{i.e. } \exists i_1, \dots, i_{k-1} : \mathbf{A}_{ii_1} = 1, \mathbf{A}_{i_{k-1}j} = 1 \text{ and } \mathbf{A}_{i_l i_{l+1}} = 1, \forall l = 1, \dots, k-2$$

The set of all k degree neighbors of node i is the k degree neighborhood of node i , \mathcal{N}_i^k .

Assumption 14. *Dependence structure within a population*

For all i, j that are not linked ($\mathbf{A}_{ij} = 0$)

$$(Y_i(\cdot), X_i, I_i^0) \perp\!\!\!\perp (Y_j(\cdot), X_j, I_j^0) \mid \{(Y_k(\cdot), X_k, I_k^0) : \forall k \in \mathcal{N}_i^\kappa \cap \mathcal{N}_j^\kappa\} \quad (33)$$

This assumption states that the treatment response functions and covariates of any unit

j is uncorrelated with units that are not within its κ neighborhood, conditional on the units in the κ neighborhoods of both i and j . This assumption rules out processes where the correlation between two nodes is decreasing in the distance between them but is always non-zero.

6 Experiment

6.1 Assumptions

The first assumption of unconfoundedness (also referred to as selection on observables) requires that the treatment assigned in the pilot population are independent of the treatment response function conditional on observables. While this assumption holds in an experimental study with a randomized treatment assignment, the analysis can be applied to the data from observational studies so long as the unconfoundedness is credible.

Assumption 15. *Unconfounded assignment of treatments in pilot population.*

For each unit $1 \leq i \leq n$ in the pilot population

$$\tilde{Y}_i(\cdot) \perp\!\!\!\perp \tilde{\mathbf{T}} \mid \tilde{\mathbf{A}}, \tilde{\mathbf{X}} \quad (34)$$

The next assumption ensures that the mean treatment responses are comparable across the target population and the pilot population.

Assumption 16. *Equality in mean treatment responses across target and pilot populations.*

For each unit $1 \leq i \leq n$ in the pilot population and $1 \leq j \leq J$ in the target population, if one of the two conditions hold,

- (a) $r_i(\tilde{\mathbf{T}}; \tilde{\mathbf{A}}) = r_j(\mathbf{T}; \mathbf{A})$
- (b) $c_i(\tilde{\mathbf{T}}; \tilde{\mathbf{A}}, \tilde{\mathbf{X}}) = c_j(\mathbf{T}; \mathbf{A}, \mathbf{X})$

then,

$$\mathbb{E}[Y_j(\mathbf{T}) | \mathbf{A}, \mathbf{X}] = \mathbb{E}[Y_i(\tilde{\mathbf{T}}) | \tilde{\mathbf{A}}, \tilde{\mathbf{X}}] \quad (35)$$

The next assumption states that the decision sets in \mathcal{G} (the class of allowed decision sets with finite VC dimension from to which the members of the partitions belong) are not

measure 0. This assumption ensures that the cell-mean estimators presented below are well-defined.

Assumption 17. *Non-trivial decision sets.*

The joint distribution $F(\cdot; \cdot)$ of (\mathbf{X}, \mathbf{A}) satisfies:

$$\forall G \in \mathcal{G}, G \neq \emptyset, \quad F((\mathbf{A}, \mathbf{X}) \in G; J) > 0$$

6.1.1 Strict Overlap for Model 1

While the strict overlap assumption is a mainstay in the treatment effect literature, I have modified it here to account for spillovers in treatment effects. A randomized control trial trivially satisfies this assumption under the bounded degree distribution implied by assumptions 3, 4 and 5. In particular, the standard strict overlap assumption is applied to effective treatments implied by model 1. For any $G \in \mathcal{G}$, and population-level treatment vector \mathbf{T} , the collection of effective treatments to unit j are $D_j^r(\mathbf{T}; G) \equiv \{r_j(\mathbf{T}|\mathbf{A}) : (\mathbf{A}, \mathbf{X}) \in G\}$ has a positive probability of occurring in the pilot population. Recall that $r_j(\mathbf{T}|\mathbf{A})$ is the effective treatment to unit j implied by population-level treatment vector \mathbf{T} given contact network \mathbf{A} .

Assumption 18. *Strict overlap on effective treatments*

There exists a $\bar{\gamma} > 0$ such that:

$$\mathbf{P}_{\tilde{\mathbf{T}}}(r_i(\tilde{\mathbf{T}}; \tilde{\mathbf{A}}) \in D_j^r(\mathbf{T}; G) | \tilde{\mathbf{A}}, \tilde{\mathbf{X}}) \geq \bar{\gamma} \quad (36)$$

where $D_j^r(\mathbf{T}; G) \equiv \{r_j(\mathbf{T}|\mathbf{A}) : (\mathbf{A}, \mathbf{X}) \in G\}$, for any effective treatment $r_j(\mathbf{T}; \mathbf{A}) \in \mathcal{R}_j(\mathbf{A})$, for every $1 \leq i \leq n$ and $1 \leq j \leq J$ and $\forall \tilde{\mathbf{X}}, \tilde{\mathbf{A}} \in \mathcal{X}^n \times \mathcal{A}_n$. Further, I assume that the propensity score is known to the planner.

The assumption that the propensity score is known is vital to the finite sample bound on regret that I attain. It is feasible in an RCT since these probabilities are given by the experimental design. This is analogous to the assumption made in Kitagawa, Tetenov (2018). They also provide a rate of convergence that is slower than the parametric $1/\sqrt{n}$ rate. This rate is improved to the parametric rate by Athey, Wager (2020) using the doubly-robust estimator.

6.1.2 Strict Overlap for Model 2

The strict overlap assumption when applied to homogenized effective treatments implied by model 2, requires further adaptation. For any $G \in \mathbb{G}$, and population-level treatment vector \mathbf{T} , the collection of homogenized effective treatments to unit j are $D_j^c(\mathbf{T}; G) \equiv \{c_j(\mathbf{T}; \mathbf{A}, \mathbf{X}) : (\mathbf{A}, \mathbf{X}) \in G\}$ has a positive probability of occurring in the pilot population. Recall that $c_j(\mathbf{T}; \mathbf{A}, \mathbf{X})$ is the homogenized effective treatment to unit j implied by population-level treatment vector \mathbf{T} , given contact network \mathbf{A} and covariate vector \mathbf{X} .

Assumption 19. *Strict overlap on homogenized effective treatments*

For any $\tilde{\mathbf{A}}, \tilde{\mathbf{X}}$, there exists a $\bar{\gamma} \in (0, 0.5)$ such that:

$$\mathbf{P}_{\tilde{\mathbf{T}}}(c_i(\tilde{\mathbf{T}}; \tilde{\mathbf{A}}, \tilde{\mathbf{X}}) \in D_j^c(\mathbf{T}, G) | \tilde{\mathbf{X}}, \tilde{\mathbf{A}}) \geq \bar{\gamma} \quad (37)$$

where $D_j^c(\mathbf{T}, G) \equiv \{c_j(\mathbf{T}; \mathbf{A}, \mathbf{X}) : (\mathbf{A}, \mathbf{X}) \in G\}$, for any $\mathbf{T} \in \mathcal{T}$ and for any $G \in \mathbb{G}$. Further, I assume that the propensity score is known to the planner.

6.2 Defining Set of Relevant Probability Measures \mathcal{P}^o

Recall that uniform regret (definition 6) is defined with respect to a class of distributions. Here, I specify class of distributions for each model of spillover.

Definition 14. *\mathcal{P}^o under Model 1.*

Define \mathcal{P}_1^o to be the set of all probability that satisfy assumptions 1-6, 9-11, 15-18.

Definition 15. *\mathcal{P}^o under Model 2.*

Define \mathcal{P}_2^o to be the set of all probability that satisfy assumptions 1-6, 12-17 and 19.

6.3 Alternate Framework: Increasing Number of Networks Asymptotics

Recall that I consider an alternate framework in which the planner has access to data from many independent pilot populations. In section 7, I discuss how such data will allow the planner to remain agnostic in modeling spillovers. Here, I present the assumptions about how the treatments are distributed within and across the numerous pilot populations.

Assumption 20. *Unconfounded under many pilot populations.*

For each unit $1 \leq i \leq n$ in each pilot population $1 \leq k \leq N$

$$\tilde{Y}_i^{(k)}(\cdot) \perp\!\!\!\perp \tilde{\mathbf{T}}^{(k)} \mid \tilde{\mathbf{A}}^{(k)}, \tilde{\mathbf{X}}^{(k)} \quad (38)$$

Assumption 21. *Strict overlap under many pilot populations.*

For each unit $1 \leq k \leq N$, there exists $\bar{\gamma} > 0$ such that:

$$\mathbf{P}_{\tilde{\mathbf{T}}}(\tilde{\mathbf{T}}^{(k)} \mid \mathbf{A}^{(k)}, \mathbf{X}^{(k)}) \geq \bar{\gamma} \quad (39)$$

where $\tilde{\mathbf{T}}^{(k)} \in \{0, 1\}^J$ and for any $\mathbf{X}^{(k)}, \mathbf{A}^{(k)} \in \mathcal{X}^J \times \mathcal{A}_J$.

Definition 15. *\mathcal{P}^o under many pilot populations.*

Define \mathcal{P}_3^o to be the set of all probability that satisfy assumptions 1,2, 7, 8, 20 and 21.

7 Empirical Welfare Maximization

Empirical Welfare Maximization (EWM) replaces the expected welfare maximization problem:

$$W_{\mathcal{G}}^*(\mathbf{P}) = \max_{G_1, \dots, G_Q \in \mathcal{G}} \sum_{q=1}^Q F((\mathbf{A}, \mathbf{X}) \in G_q; J) \cdot \left(\frac{1}{J} \sum_{j=1}^J \mathbb{E}_{\mathbf{P}} [Y_j(\mathbf{T}_q) \mid (\mathbf{A}, \mathbf{X}) \in G_q] \right) \quad (40)$$

with the empirical analogue constructed using data from the pilot population, i.e., this replaces the expectation with respect to an unknown distribution with its empirical analogue.

$$W(\hat{\mathbf{G}}_{EWM}; \mathbf{P}) = \max_{G_1, \dots, G_Q \in \mathcal{G}} \sum_{q=1}^Q F((\mathbf{A}, \mathbf{X}) \in G_q; J) \cdot \left(\frac{1}{J} \sum_{j=1}^J \hat{\mathbb{E}} [Y_j(\mathbf{T}_q) \mid (\mathbf{A}, \mathbf{X}) \in G_q] \right) \quad (41)$$

7.1 Cell Mean Estimator

An important component in estimating the welfare function is the estimation of the mean treatment response function, i.e., for any $\mathbf{T} \in \mathcal{T}$, $G \in \mathcal{G}$ and $1 \leq j \leq J$,

$$\mathbb{E}_{\mathbf{P}} \left[Y_j(\mathbf{T}) \mid (\mathbf{X}, \mathbf{A}) \in G \right] \quad (42)$$

To see the intuition behind the cell mean estimator, consider the running example of malaria.

Running example - malaria: Recall that the planner would like to assign one bed net between two linked households in the target population. In particular, suppose the planner would like to estimate the mean treatment response to household 1 when household 1 is assigned the bed net. For the time being, assume away covariates. The planner may use data

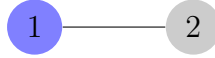


Figure 9: Estimand in the target population

from pilot population to estimate this mean treatment response. Suppose the realization of the data is such that houses A, C and E are assigned the bed net while houses B, D and F are not. Suppose the effect treatment to unit j is defined as the unit j 's treatment status

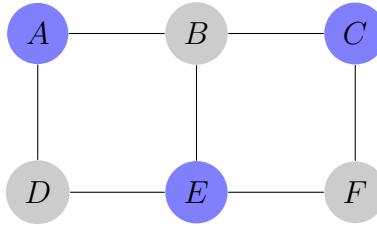


Figure 10: Data in the pilot population

and the share of its links that receive a treatment. Notice that households A, C and E in the pilot population share the same effective treatment as household 1 in the target population. Then, due to unconfounded distribution of treatments in the target population, the mean treatment response to household 1 can be estimated using the average of the outcomes to households A, C and E in the pilot population.

The next section formalizes this intuition for the 2 models of spillovers and also accounts for covariates.

7.1.1 Model 1: Local Spillovers and Exchangeability

Definition 16. *Cell-mean estimator under model 1.*

$$\hat{\mathbb{E}}_{\mathbf{P}}[Y_j(\mathbf{T}) | (\mathbf{X}, \mathbf{A}) \in G] = \frac{1}{n} \sum_{i=1}^n \frac{\tilde{Y}_i 1\{r_i(\tilde{\mathbf{D}}; \tilde{\mathbf{A}}) \in D_j^r(\mathbf{T}; G)\} 1\{\tilde{X}_i \in G_{|X_j}\}}{\mathbf{P}_{\tilde{\mathbf{T}}}(r_i(\tilde{\mathbf{D}}; \tilde{\mathbf{A}}) \in D_j^r(\mathbf{T}; G), \tilde{X}_i \in G_{|X_j} | \tilde{\mathbf{A}}, \tilde{\mathbf{X}})} \quad (43)$$

where

$$D_j^r(\mathbf{T}; G) \equiv \{r_i(\mathbf{T}; \mathbf{A}) : (\mathbf{X}, \mathbf{A}) \in G\},$$

$$G_{|X_j} \equiv \{\mathbf{X}_j : (\mathbf{X}, \mathbf{A}) \in G\}.$$

Lemma 3. *Unbiasedness of cell-mean estimator*

For any $\mathbf{P} \in \mathcal{P}_1^o$, the above estimator is unbiased.

7.1.2 Model 2: SIR Based Model of Disease Propagation

Definition 17. *Cell-mean estimator under model 1.*

$$\hat{\mathbb{E}}_{\mathbf{P}} [Y_j(\mathbf{T}) | (\mathbf{X}, \mathbf{A}) \in G] = \frac{1}{n} \sum_{i=1}^n \frac{\tilde{Y}_i 1\{c_i(\tilde{\mathbf{D}}; \tilde{\mathbf{A}}, \tilde{\mathbf{X}}) \in D_j^c(\mathbf{T}; G)\}}{\mathbf{P}_{\tilde{\mathbf{T}}}(c_i(\tilde{\mathbf{T}}; \tilde{\mathbf{A}}, \tilde{\mathbf{X}}) \in D_j^c(\mathbf{T}; G) | \tilde{\mathbf{X}}, \tilde{\mathbf{A}})} \quad (44)$$

where $D_j^c(\mathbf{T}, G) \equiv \{c_j(\mathbf{T}; \mathbf{A}, \mathbf{X}) : (\mathbf{A}, \mathbf{X}) \in G\}$.

Lemma 4. *Unbiasedness of cell-mean estimator*

For any $\mathbf{P} \in \mathcal{P}_2^o$, the above estimator is unbiased.

The unbiasedness of cell-mean estimator implies that the empirical welfare for any treatment rule is an unbiased estimator of the welfare. Consequently, the finite sample bound on the uniform regret is amounts to proving a concentration inequality of an empirical process indexed by the treatment rule. This is formalized in the lemma below.

Lemma 5. *Uniform regret as an empirical process.*

For any $l \in \{1, 2\}$,

$$\sup_{\mathbf{P} \in \mathcal{P}_l^o} \mathbb{E}_{\tilde{\mathbf{X}}, \tilde{\mathbf{A}}, \tilde{\mathbf{T}}, \tilde{\mathbf{Y}} \sim \mathbf{P}} \left[W_{\mathcal{G}}^*(\mathbf{P}) - W(\hat{\mathbf{G}}_{EWM}; \mathbf{P}) \right] \leq 2 \sum_{q=1}^Q \sup_{\mathbf{P} \in \mathcal{P}_l^o} \mathbb{E}_{\tilde{\mathbf{X}}, \tilde{\mathbf{A}}, \tilde{\mathbf{T}}, \tilde{\mathbf{Y}} \sim \mathbf{P}} \left[\sup_{G \in \mathcal{G}} |W_n^q(G) - W^q(G)| \right] \quad (45)$$

7.2 Alternate Framework: Increasing Number of Networks Asymptotics

Empirical Welfare Maximization (EWM) replaces the expected welfare maximization problem:

$$W_{\mathcal{G}}^*(\mathbf{P}) = \max_{G_1, \dots, G_Q \in \mathcal{G}} \sum_{q=1}^Q \mathbb{E} \left[\frac{1}{J} \sum_{j=1}^J Y_j(\mathbf{T}_q) \cdot \mathbf{1}\{(\mathbf{A}, \mathbf{X}) \in G_q\} \right] \quad (46)$$

with the empirical analogue constructed using data from the pilot population, i.e., this replaces the expectation with respect to an unknown distribution with its empirical analogue.

$$W(\hat{\mathbf{G}}_{EWM}; \mathbf{P}) = \max_{G_1, \dots, G_Q \in \mathcal{G}} \sum_{q=1}^Q \hat{\mathbb{E}} \left[\frac{1}{J} \sum_{j=1}^J Y_j(\mathbf{T}_q) \right] \cdot \mathbf{1}\{(\mathbf{A}, \mathbf{X}) \in G_q\} \quad (47)$$

In this case the cell -mean estimator is defined as:

$$\hat{\mathbb{E}} \left[\frac{1}{J} \sum_{j=1}^J Y_j(\mathbf{T}_q) \cdot \mathbf{1}\{(\mathbf{A}, \mathbf{X}) \in G_q\} \right] = \frac{1}{N} \sum_{k=1}^N \frac{\sum_{q=1}^Q \frac{1}{J} \sum_{j=1}^J Y_j^{(k)} \cdot \mathbf{1}\{\tilde{\mathbf{T}}^{(k)} = \mathbf{T}_q\} \cdot \mathbf{1}\{(\tilde{\mathbf{A}}^{(k)}, \tilde{\mathbf{X}}^{(k)}) \in G_q\}}{\mathbf{P}(\tilde{\mathbf{T}}^{(k)} = \mathbf{T}_q | \tilde{\mathbf{A}}^{(k)}, \tilde{\mathbf{X}}^{(k)})} \quad (48)$$

$$\equiv \frac{1}{N} \sum_{k=1}^N f(\tilde{\mathbf{X}}^{(k)}, \tilde{\mathbf{A}}^{(k)}, \tilde{\mathbf{T}}^{(k)}, \tilde{\mathbf{Y}}^{(k)}, \mathbf{T}_q, G_q) \quad (49)$$

Unbiasedness of the estimator is trivial here.

Lemma 6. Uniform regret as an empirical process.

$$\mathbb{E}_{\tilde{\mathbf{X}}, \tilde{\mathbf{A}}, \tilde{\mathbf{T}}, \tilde{\mathbf{Y}} \sim \mathbf{P}} \left[\sup_{G \in \mathcal{G}} |W_N^q(G) - W^q(G)| \right] \leq \mathbb{E}_{\tilde{\mathbf{X}}, \tilde{\mathbf{A}}, \tilde{\mathbf{T}}, \tilde{\mathbf{Y}} \sim \mathbf{P}} \left[\sup_{f \in \mathcal{F}} |\mathbb{E}_N(f) - \mathbb{E}(f)| \right] \quad (50)$$

8 Results

I start by proving two concentration inequalities that separately handle the case of the two different asymptotic frameworks. These lemma bound an empirical process indexed by class of functions with finite VC dimension. A very crude intuition for the logic in attained by considering that a concentration inequality shows that the average of a large number of draws of a random variable concentrates around the expected value of the random variable. In my case, the draws need not be IID, hence I make assumptions on the correlation and

network representation. Moreover, instead of a random variable, I have an empirical process and will show that this concentration applies uniformly over the domain of the empirical process which requires the VC dimension assumption and assumptions on unconfoundedness and strict overlap. Lemma 7 deals with the single pilot population framework while lemma 8 is for the alternative framework with many network populations.

Lemma 7. Suppose \mathcal{D}_n satisfies assumptions 3-6, $Z_{1:n}$ satisfies assumptions 1, 2 and either 9-11 and 15-18 or 12-17 and 19. Let \mathcal{F} be a class of uniformly bounded functions with $\|f\|_\infty \leq \bar{F}$ for all $f \in \mathcal{F}$. Further, assume that \mathcal{F} is countable and has finite VC dimension. Then, there exists a universal constant C_1 such that

$$\mathbb{E}_{\mathcal{D}_n} \left[\mathbb{E}_{Z_{1:n}|\mathcal{D}_n} \left[\sup_{f \in \mathcal{F}} \left| \frac{1}{n} \sum_{i=1}^n f(Z_i) - \mathbb{E}f \right| \right] \right] \leq 2C_1 [2^d(1 + 2\kappa\bar{\rho})^d] 3/2\bar{F} \sqrt{\frac{v}{n}}$$

Lemma 8. Suppose $\mathbf{Z}_{1:N}$ satisfies assumptions 1, 7, 8, 20 and 21. Let \mathcal{F} be a class of uniformly bounded functions with $\|f\|_\infty \leq \bar{F}$ for all $f \in \mathcal{F}$. Further, assume that \mathcal{F} is countable and has finite VC dimension. Then, there exists a universal constant C'_2 such that:

$$\mathbb{E}_{\mathbf{Z}_{1:N}} \left[\sup_{f \in \mathcal{F}} \left| \mathbb{E}_N f - \mathbb{E}f \right| \right] \leq 2 \left[C'_2 \bar{F} \sqrt{\frac{v}{N}} \right]$$

The first theorem provides an upper bound for local spillovers and exchangeability model with single pilot population.

Theorem 1. *Finite sample bound for Empirical Welfare Maximization.*

$$\sup_{\mathbf{P} \in \mathcal{P}_1^o} \mathbb{E}_{\tilde{\mathbf{X}}, \tilde{\mathbf{A}}, \tilde{\mathbf{T}}, \tilde{\mathbf{Y}} \sim \mathbf{P}} \left[W_{\mathcal{G}}^* - W(\hat{\mathbf{G}}_{EWM}) \right] \leq 4QC_1 [2^d(1 + 2\kappa\bar{\rho})^d] 3/2 \frac{M}{2\bar{\gamma}} \sqrt{\frac{v}{n}}$$

The second theorem proved an upper bound for SIR based model of spillovers with single pilot population.

Theorem 2. *Finite sample bound for Empirical Welfare Maximization.*

$$\sup_{\mathbf{P} \in \mathcal{P}_2^o} \mathbb{E}_{\tilde{\mathbf{X}}, \tilde{\mathbf{A}}, \tilde{\mathbf{T}}, \tilde{\mathbf{Y}} \sim \mathbf{P}} \left[W_{\mathcal{G}}^* - W(\hat{\mathbf{G}}_{EWM}) \right] \leq 4QC_1 [2^d(1 + 2\kappa\bar{\rho})^d] 3/2 \frac{1}{\bar{\gamma}} \sqrt{\frac{v}{n}}$$

The third theorem provides an upper bound under increasing number of networks asymptotic framework.

Theorem 3. *Finite sample bound for Empirical Welfare Maximization.*

$$\sup_{\mathbf{P} \in \mathcal{P}_3^o} \mathbb{E}_{\tilde{\mathbf{X}}^{(1:N)}, \tilde{\mathbf{A}}^{(1:N)}, \tilde{\mathbf{T}}^{(1:N)}, \tilde{\mathbf{Y}}^{(1:N)} \sim \mathbf{P}} \left[W_{\mathcal{G}}^* - W(\hat{\mathbf{G}}_{EWM}) \right] \leq 4QC_1 \frac{M}{2\bar{\gamma}} \sqrt{\frac{v}{N}}$$

The fourth theorem provides a lower bound for the increasing number of networks asymptotic framework.

Theorem 4: For any statistical treatment rule $\hat{\mathcal{Z}}$, it holds that

$$\sup_{\mathbf{P} \in \mathcal{P}_3^o} \mathbb{E}_{Data \sim (\mathbf{P}, F)} \left[W_{\mathcal{G}}^* - W(\hat{\mathbf{G}}_{EWM}) \right] \geq \frac{1}{2} \sqrt{\frac{v}{N}} e^{-2\sqrt{2}}$$

as long as $J^2 N \geq 16v$.

The fifth theorem provides a lower bound for local spillovers and exchangeability model with single pilot population.

Theorem 5*: For any statistical treatment rule $\hat{\mathcal{Z}}$, it holds that

$$\sup_{\mathbf{P} \in \mathcal{P}_1^o} \mathbb{E}_{\tilde{\mathbf{X}}, \tilde{\mathbf{A}}, \tilde{\mathbf{T}}, \tilde{\mathbf{Y}} \sim \mathbf{P}} \left[W_{\mathcal{G}}^* - W(\hat{\mathbf{G}}_{EWM}) \right] \geq C_3(\mathcal{P}_1^o) \sqrt{\frac{1}{N}}$$

* The proof for this is coming soon.

9 Illustration in the Context of the Running Example

In this section, I provide a numerical illustration in the context of the running example for malaria that I set up earlier in the paper. To recap, the planner faces an outbreak of a deadly infectious disease, malaria in a target village consisting of two households ($J = 2$). Here, I assume that the planner observes the distance from each household to vegetation as the covariate $X_j \in \{0, 100\}$. Since mosquitoes are said to be poor flyers, not flying beyond a hundred meters, links on the contact network can be thought to be determined by geographical proximity. For example, the 2 houses are said to be linked (I'll simplify the notation here to $\mathbf{A} = 1$) if and only if the distance between the two houses is less than a hundred meters. The planner has 1 bed net to distribute between the 2 houses. The outcome $Y_j(\cdot)$ can be any composite measure of health outcome of household j . The set of potential outcomes for house j are $\{Y_j(1, 1), Y_j(1, 0), Y_j(0, 1), Y_j(0, 0)\}$ and the feasible set of village-level treatment

assignments is $\mathcal{T} = \{(1, 0), (0, 1), (0, 0)\}$ and hence $Q = 3$.

I assume that $\mathbf{P}(X_j = 0) = \mathbf{P}(X_j = 100) = 1/2$ IID across $1 \leq j \leq 2$. The linking probability is assumed to be $\mathbf{P}(\mathbf{A}_{ij} = 1) = 1/2$ IID across links ij ¹⁸ and independent across \mathbf{X} . These two distribution assumptions jointly determine $F(\cdot, \cdot; J)$ in this context. Formally, I can define the μ_J compatible with assumption 3:

$$F(\mathbf{A}, \mathbf{X}; J) = \frac{1}{8}, \forall \mathbf{A}, \mathbf{X} \in \{0, 1\} \times \{0, 100\}^2 \quad (51)$$

In this example, I assume the following treatment response function consistent with assumptions 9, 10 and 11 in the local spillovers and exchangeability model. Recall the earlier example:

$$\mathbb{E}[Y_i(\mathbf{T}) | \mathbf{A}, \mathbf{X}] = T_i + \frac{X_i}{100} \cdot ((1 - T_i)s_i(\mathbf{T}, \mathbf{A})(1 - \beta))$$

where

$$s_i(\mathbf{T}, \mathbf{A}) = \begin{cases} 1, & \text{if } \sum_{j \neq i} \mathbf{A}_{ij} = 0 \\ \frac{\sum_{j \neq i} \mathbf{A}_{ij} T^j}{\sum_{j \neq i} \mathbf{A}_{ij}}, & \text{else} \end{cases}$$

Here, I build on this assumption as follows:

$$Y_i(\mathbf{T}) | (T_i, s_i(\mathbf{T}, \mathbf{A}), \mathbf{X}) = \begin{cases} 0 \text{ w.p.1} & \text{if } T_i = s_i(\mathbf{T}, \mathbf{A}) = 0 \\ T_i + \frac{X_i}{100} \cdot ((1 - T_i)s_i(\mathbf{T}, \mathbf{A})(1 - \beta)) + \epsilon_i, & \text{else} \end{cases} \quad (52)$$

where

$$\epsilon_i \sim U(-1, +1), \text{ IID across } i \text{ and independent of } \mathbf{A}, \mathbf{X} \quad (53)$$

The planner has access to an experiment conducted on a pilot village containing $n = 6$ households. The distance between house i and vegetation is recorded as \tilde{X}_i . The adjacency matrix of the contact network in the pilot village is recorded in $\tilde{\mathbf{A}}$. The experimentally assigned treatment to house i is \tilde{T}_i . Finally, the composite measure of health outcome in house i is \tilde{Y}_i . I maintain the assumptions that the same stationary data generating process

¹⁸ In the target village there happens to be only 1 pair on nodes.

governs both, the target population, and the pilot population (assumptions 1 and 6). Suppose that the treatment assignment on the pilot population satisfies assumptions 15, 16 and 17. In particular, $\mathbf{P}(\tilde{T}_i = 1) = \mathbf{P}(\tilde{T}_i = 0) = 1/2$ and is independent of $\tilde{\mathbf{A}}, \tilde{\mathbf{X}}$. For the exercise, I fix the data for the pilot population as below. The network structure is represented by figure 3.

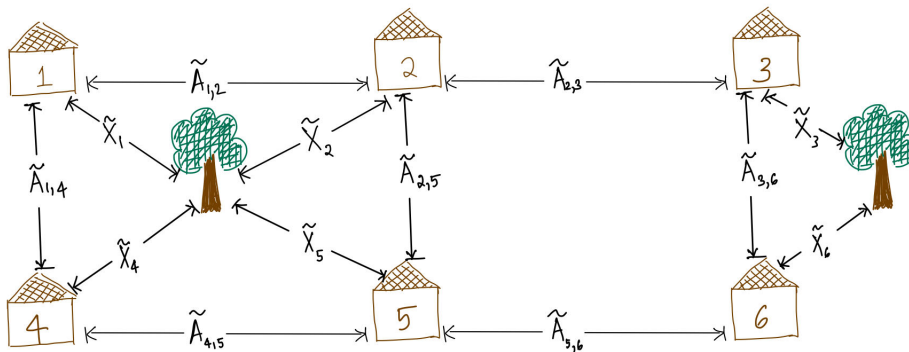


Figure 11: Target Village in Running Example

The observations in the pilot population are provided in table 1. The uncertainty for the planner comes from the fact that she does not observe the true β ¹⁹ and the realizations of ϵ_i .

Variable	1	2	3	4	5	6
X_i	100	0	100	100	0	100
T_i	1	0	1	0	1	0
Y_i	1.03	0.89	1.53	0.06	0.44	0.87

Table 1: Observations in Pilot Population

Following definition 3, I characterize the treatment rules by their implied decision sets G_1, G_2, G_3 which form a partition of the set of all $(\mathbf{A}, \mathbf{X}) \in \{0, 1\} \times \{0, 100\}^2$. Since this set is finite, any partition of decision sets trivially has finite VC dimension and hence satisfies assumption 2. So, let \mathcal{G} denote the set of all partitions. For any $G_1, G_2, G_3 \in \mathcal{G}$, define the empirical welfare function as:

¹⁹ The numerical exercise uses $\beta = 0.5$.

$$\begin{aligned}
W_n(G_1, G_2, G_3) = & \sum_{\mathbf{T} \in \{(0,0), (1,0), (1,0)\}} \frac{1}{2 \cdot 8} \sum_{\mathbf{A} \in \{0,1\}} \sum_{\mathbf{X} \in \{0,100\}^2} F(\mathbf{A}, \mathbf{X}; J) \sum_{j \in \{1,2\}} \frac{1}{n} \\
& \sum_{i=1}^n \frac{\tilde{Y}_i \mathbf{1}\{\tilde{T}_i = T_j, s_i(\tilde{\mathbf{T}}, \tilde{\mathbf{A}}) = s_j(\mathbf{T}, \mathbf{A}), \tilde{X}_i = X_j\}}{\mathbf{P}_{\tilde{\mathbf{T}}}\{\tilde{T}_i = T_j, s_i(\tilde{\mathbf{T}}, \tilde{\mathbf{A}}) = s_j(\mathbf{T}, \mathbf{A}), \tilde{X}_i = X_j | \tilde{\mathbf{A}}, \tilde{\mathbf{X}}\}}
\end{aligned} \tag{54}$$

The empirical welfare maximizing treatment rule is:

$$\hat{Z}(\mathbf{A}, \mathbf{X} | \tilde{\mathbf{Y}}, \tilde{\mathbf{X}}, \tilde{\mathbf{A}}, \tilde{\mathbf{T}}) = \begin{cases} (0, 0) & \text{if } (\mathbf{A}, \mathbf{X}) \in \{(0, 0, 0), (0, 0, 100), (0, 100, 0), (0, 100, 100)\} \\ (1, 0) & \text{if } (\mathbf{A}, \mathbf{X}) \in \{(1, 0, 0), (1, 0, 100), (1, 100, 0), (1, 100, 100)\} \end{cases} \tag{55}$$

If the planner knew the distribution (β) , the optimal treatment rule is:

$$Z^*(\mathbf{A}, \mathbf{X} | \beta = 0.5) = \begin{cases} (0, 0) & \text{if } (\mathbf{A}, \mathbf{X}) \in \{(0, 0, 0)\} \\ (0, 1) & \text{if } (\mathbf{A}, \mathbf{X}) \in \{(1, 100, 0), (1, 100, 100)\} \\ (1, 0) & \text{else} \end{cases} \tag{56}$$

10 Applicability to COVID 19

While a vaccine for COVID-19 is yet to be approved, it is becoming increasingly clear that ramping up supply will be challenging, particularly in its early days. Consequently, governments across the globe will need to ration the limited stock of vaccines. This rationing may be done according to one of several objective functions. One example is maximizing the level of activity subject to a tolerable number of infections. In this section, I discuss some adaptations to the presented framework that will be necessary before the results may be transferred to the study of COVID-19.

The first key distinction involves the contact network. This paper assumes that the degree distribution of the contact network is bounded. Depending on the compliance to social distancing and the use of masks in public places, this may or may not be a suitable assumption for COVID-19. Consider first the extreme case where nobody follows social distancing or the use of masks in public settings. Since the disease is believed to be transmitted primarily through droplets that are expelled by an infected individual coughing, sneezing or talking,²⁰ the implied contact network could be very dense. Consider the example of an

²⁰ Center for Disease Control FAQs on Coronavirus Disease 2019 (COVID-19)

asymptomatic but infected individual passing through a crowded area such a public transit or football game.²¹ The degree of such an individual on the contact network can be very large. In general, I do not expect the contact network to satisfy the bounded degree assumption in this setting. At the other extreme, consider now a situation where all members of society are perfectly adhering to social distancing requirements and the appropriate use of masks. In such an environment, the bounded degree assumption is likely to hold. In a realistic model of COVID-19, individuals' decisions to comply with social distancing norms would need to be endogenized, adding to its complexity.

A second distinction lies in the appropriate model of disease transmission on the contact network. While the models presented here might be a good fit, much remains to be known about the transmission of COVID-19 as of date. Finally, the objective functions considered by the planner also needs special attention. In this paper, I consider the population objective functions to be the aggregate outcome across members of the population. This would be consistent with the welfare maximization by a utilitarian social planner who only cares about the survival of the members of the population. In the COVID-19 case, governments may have a host of other objective functions which might include, at least in part, maximizing economic activity. Extending the results presented here to such welfare functions would require additional work.²²

11 Conclusion

This paper is primarily concerned with establishing theoretical properties of the Empirical Welfare Maximization procedure when extended to accommodate for spillovers in treatments. While the results have been established with deadly communicable diseases such as Ebola, HIV, and malaria in mind, the tools developed herein have wider applicability. In this section, I suggest some broader applications.

11.1 Saving Amazon Rainforests

In the past three decades, deforestation has taken a dramatic toll on the Amazon rainforests. As a consequence, in 2008, the Brazilian government released a Priority List of 36 municipalities with high levels of deforestation. These municipalities were subject to rigorous

²¹ Robinson, J. (2020), The Soccer Match that Kicked Off Italy's Coronavirus Disaster, Wall Street Journal, 1 April. Available at: <https://www.wsj.com/articles/the-soccer-match-that-kicked-off-italys-coronavirus-disaster-11585752012>

²² See Kitagawa, Tetenov (2019b) for equality minded welfare functions.

monitoring and stricter penalty to deforestation.

Stricter monitoring in one municipality is believed to discourage illegal deforestation in neighboring municipalities too. Thus, municipalities of the Amazon rainforest can be thought of as vertices of a network. Two municipalities are said to be linked by an edge if they are geographic neighbors. Assunção, McMillan, Murphy, Souza-Rodrigues (2019) estimate the treatment effect and the spillovers associated with this policy. They then use these estimates to compute the optimal set of municipalities to target in the Priority List. The results in this paper can be extended to establish theoretical guarantees for Empirical Welfare Maximization in optimally targeting municipalities.

11.2 Marketing

In a different paper²³, the authors explore an example from marketing. MS Office is a licensed software which offers a suite of document, spreadsheet, and presentation editors. It also allows users to share documents and work collaboratively between themselves. MS Office recently launched Office Lite, a web based service with limited functionality. The introduction of Office Lite has two opposing effects on the overall purchase of MS Office licenses. On the one hand, the introduction of a free limited-feature product cannibalized the existing product. On the other hand, offering a free version allows for a larger collaborator network. This might induce positive externalities on users causing them to upgrade to the full feature version of MS Office.

The paper quantifies this trade-off and suggests a profit maximizing roll out of MS Office Lite to selected parts of the collaborator network. The results of this paper, when combined with a model of product choice and network formation can help inform theoretical properties of the Empirical Welfare Maximization procedure in optimal marketing on the collaborator network.

²³ Ananth, Molinari, Peng (2020)

Appendix

Proof of Lemma 1

Proof: To prove the claim above, the above lemma is restated in terms of the notation established. Fix any $\mathbf{l} \in \mathbb{Z}^d$, where $\forall \mathbf{l} \neq \mathbf{k} \in \mathbb{Z}^d, \rho(\mathbf{l}, \mathbf{k}) \geq 1$. Then, given assumption #, the above lemma states that

$$|\{\mathbf{k} \in \mathcal{D}_n : \mathbf{k} \neq \mathbf{l}, \rho(\mathbf{k}, \mathbf{l}) \leq \bar{\rho}\}| \leq (1 + 2\bar{\rho})^d - 1 \text{ with probability 1.}$$

Define a lattice L around $\mathbf{l} \in \mathbb{Z}^d$ as follows.

$$L = \{\mathbf{k} \in \mathbb{Z}^d : \forall 1 \leq b \leq d, k_b \in \{l_b, l_b + 1, l_b - 1, \dots, l_b + \bar{\rho}, l_b - \bar{\rho}\}\}$$

Under assumptions made,

$$\{\mathbf{k} \in \mathcal{D}_n : \mathbf{k} \neq \mathbf{l}, \rho(\mathbf{k}, \mathbf{l}) \leq \bar{\rho}\} \subseteq L \setminus \{\mathbf{l}\}$$

Thus,

$$|\{\mathbf{k} \in \mathcal{D}_n : \mathbf{k} \neq \mathbf{l}, \rho(\mathbf{k}, \mathbf{l}) \leq \bar{\rho}\}| \leq |L| - 1 = (1 + 2\bar{\rho}^*)^d - 1 \text{ with probability 1. } \blacksquare$$

Proof of Lemma 2

■ **Proof:** The proof demonstrates that $c_i(\mathbf{T}; \mathbf{A}, \mathbf{I}^0, \mathbf{X})$ completely characterizes the distribution of $Y_i(\mathbf{T}) | \mathbf{A}, \mathbf{I}^0, \mathbf{X}$ given parameters $(\theta, p(\cdot), q(\cdot))$. In the event, $\sum_k \mathbf{A}_{ik} > 0$:

$$\begin{aligned} \mathbf{P}(s_i^{(1)} = a | \mathbf{T}, \mathbf{A}, \mathbf{I}^0, \mathbf{X}) &= 1\{a = D\} \left[(1 - \theta)I_i^0 + 1\{s_i^{(0)} = D\} \right] \\ &\quad + 1\{a = I\} \left[\theta I_i^0 + \frac{\sum_k \mathbf{A}_{ik} I_k^0}{\sum_k \mathbf{A}_{ik}} (p(X_i) - T_i q(X_i)) 1\{s_i^{(0)} = S\} \right] \\ &\quad + 1\{a = S\} \left[\left(1 - \frac{\sum_k \mathbf{A}_{ik} I_k^0}{\sum_k \mathbf{A}_{ik}} (p(X_i) - T_i q(X_i)) \right) 1\{s_i^{(0)} = S\} \right] \end{aligned}$$

When, $\sum_k \mathbf{A}_{ik} = 0$:

$$\begin{aligned} \mathbf{P}(s_i^{(1)} = a | \mathbf{T}, \mathbf{A}, \mathbf{I}^0, \mathbf{X}) &= 1\{a = D\} \left[(1 - \theta)I_i^0 + 1\{s_i^{(0)} = D\} \right] \\ &\quad + 1\{a = I\} \left[\theta I_i^0 \right] + 1\{a = S\} \left[1\{s_i^{(0)} = S\} \right] \end{aligned}$$

Given parameters, $\mathbf{P}(s_i^{(1)} = a | \mathbf{T}, \mathbf{A}, \mathbf{I}^0, \mathbf{X})$ is completely characterized by

$$\left(\sum_k \mathbf{A}_{jk}, \sum_k \mathbf{A}_{jk} I_k^0, X_j, T_j, I_j^0, 1\{s_j^{(0)} = D\} \right)$$

For κ period transmissions:

$$\begin{aligned} \mathbf{P}\left(s_j^{(\kappa)} = b \mid \mathbf{T}, \mathbf{A}, \mathbf{I}^0, \mathbf{X}\right) &= \sum_{\mathbf{a}^{(\kappa-1)}} \mathbf{P}\left(s_j^{(\kappa)} = b \mid \mathbf{T}, \mathbf{A}, \mathbf{X}, \mathbf{s}^{(\kappa-1)} = \mathbf{a}^{(\kappa-1)}\right) \times \\ &\quad \sum_{\mathbf{a}^{(\kappa-2)}} \mathbf{P}\left(\mathbf{s}^{(\kappa-1)} = \mathbf{a}^{(\kappa-1)} \mid \mathbf{T}, \mathbf{A}, \mathbf{X}, \mathbf{s}^{(\kappa-2)} = \mathbf{a}^{(\kappa-2)}\right) \times \\ &\quad \dots \\ &\quad \sum_{\mathbf{a}^{(1)}} \mathbf{P}\left(\mathbf{s}^{(2)} = \mathbf{a}^{(2)} \mid \mathbf{T}, \mathbf{A}, \mathbf{X}, \mathbf{s}^{(1)} = \mathbf{a}^{(1)}\right) \times \mathbf{P}\left(\mathbf{s}^{(1)} = \mathbf{a}^{(1)} \mid \mathbf{T}, \mathbf{A}, \mathbf{I}^0, \mathbf{X}\right) \end{aligned}$$

where $b \in \{S, I, D\}$ and $\mathbf{a}^{(t)} \in \{S, I, D\}^J$ for all $1 \leq t \leq \kappa$. Assumption 1 implies that for any node, each one step transmission is only governed by the infection states of immediate neighbors (and not the rest of the network). Thus, the above equality can be written as:

$$\begin{aligned} \mathbf{P}\left(s_j^{(\kappa)} = b \mid \mathbf{T}, \mathbf{A}, \mathbf{I}^0, \mathbf{X}\right) &= \sum_{\mathbf{a}_{\mathcal{N}_i^1}^{(\kappa-1)}} \mathbf{P}\left(s_j^{(\kappa)} = b \mid \mathbf{T}, \mathbf{A}, \mathbf{X}, \mathbf{s}_{\mathcal{N}_i^1}^{(\kappa-1)} = \mathbf{a}_{\mathcal{N}_i^1}^{(\kappa-1)}\right) \times \\ &\quad \sum_{\mathbf{a}_{\mathcal{N}_i^2}^{(\kappa-2)}} \mathbf{P}\left(\mathbf{s}_{\mathcal{N}_i^1}^{(\kappa-1)} = \mathbf{a}_{\mathcal{N}_i^1}^{(\kappa-1)} \mid \mathbf{T}, \mathbf{A}, \mathbf{X}, \mathbf{s}_{\mathcal{N}_i^2}^{(\kappa-2)} = \mathbf{a}_{\mathcal{N}_i^2}^{(\kappa-2)}\right) \times \\ &\quad \dots \\ &\quad \sum_{\mathbf{a}_{\mathcal{N}_i^{\kappa-1}}^{(1)}} \mathbf{P}\left(\mathbf{s}_{\mathcal{N}_i^{\kappa-2}}^{(2)} = \mathbf{a}_{\mathcal{N}_i^{\kappa-2}}^{(2)} \mid \mathbf{T}, \mathbf{A}, \mathbf{X}, \mathbf{s}_{\mathcal{N}_i^{\kappa-1}}^{(1)} = \mathbf{a}_{\mathcal{N}_i^{\kappa-1}}^{(1)}\right) \times \\ &\quad \mathbf{P}\left(\mathbf{s}_{\mathcal{N}_i^{\kappa-1}}^{(1)} = \mathbf{a}_{\mathcal{N}_i^{\kappa-1}}^{(1)} \mid \mathbf{T}, \mathbf{A}, \mathbf{I}^0, \mathbf{X}\right) \end{aligned}$$

where $\mathbf{a}_{\mathcal{N}_i^k}^{(t)} = (\mathbf{a}_j^{(t)} : j \in \mathcal{N}_i^k)$ and similarly $\mathbf{s}_{\mathcal{N}_i^k}^{(t)} = (\mathbf{s}_j^{(t)} : j \in \mathcal{N}_i^k)$. Further, assumption 2 allows characterizing the joint distribution of each one step transmission as a product of marginals.

$$\mathbf{P}\left(\mathbf{s}_{\mathcal{N}_i^k}^{(t)} = \mathbf{a}_{\mathcal{N}_i^k}^{(t)} \mid \mathbf{T}, \mathbf{A}, \mathbf{X}, \mathbf{s}_{\mathcal{N}_i^{k+1}}^{(t-1)} = \mathbf{a}_{\mathcal{N}_i^{k+1}}^{(t-1)}\right) = \prod_{j \in \mathcal{N}_i^k} \mathbf{P}\left(\mathbf{s}_j^{(t)} = \mathbf{a}_j^{(t)} \mid \mathbf{T}, \mathbf{A}, \mathbf{X}, \mathbf{s}_{\mathcal{N}_i^{k+1}}^{(t-1)} = \mathbf{a}_{\mathcal{N}_i^{k+1}}^{(t-1)}\right)$$

Thus,

$$c_i(\mathbf{T}; \mathbf{A}, \mathbf{I}^0, \mathbf{X}) = \left(F_i, (F_{k_1} : k_1 \in g(i, 1; \mathbf{A})), ((F_{k_1}, F_{k_2}) : (k_1, k_2) \in g(i, 2; \mathbf{A})), \dots, \right. \\ \left. ((F_{k_1}, \dots, F_{k_{\kappa-1}}) : (k_1, \dots, k_{\kappa-1}) \in g(i, \kappa - 1; \mathbf{A})) \right) \blacksquare$$

Proof of Lemma 3

Proof: Define the following useful shorthands:

In the notation of this shorthand, this estimator can be expressed as:

$$\hat{\theta} = \hat{\mathbb{E}}[Y_j(\mathbf{T}|\mathbf{A}) | (\mathbf{A}, \mathbf{X}) \in G] = \sum_{i=1}^n \frac{\tilde{Y}_i 1\{r_i \in D_j^r\} 1\{G|_{X_j}\}}{\mathbf{P}_{\sim}(\{r_i \in D_j^r\}, \{G|_{X_j}\})}$$

Now, taking expectations with respect to data $(\tilde{\mathbf{Y}}, \tilde{\mathbf{D}}, \tilde{\mathbf{X}}, \tilde{\mathbf{A}})$,

$$\mathbb{E}_{\sim}(\hat{\theta}) = \sum_{i=1}^n \frac{1}{\mathbf{P}_{\sim}(\{r_i \in D_j^r\}, \{G|_{X_j}\})} \mathbb{E}_{\sim}(\tilde{Y}_i 1\{r_i \in D_j^r\} 1\{G|_{X_j}\}) \\ = \sum_{i=1}^n \frac{1}{\mathbf{P}_{\sim}(\{r_i \in D_j^r\}, \{G|_{X_j}\})} \mathbb{E}_{\tilde{\mathbf{D}}, \tilde{\mathbf{X}}, \tilde{\mathbf{A}}} (1\{r_i \in D_j^r\} 1\{G|_{X_j}\} \mathbb{E}_{\tilde{\mathbf{Y}}}(\tilde{Y}_i | \tilde{\mathbf{D}}, \tilde{\mathbf{X}}, \tilde{\mathbf{A}}))$$

The second equality follows from the law of iterated expectations. Unconfoundedness implies that $\mathbb{E}_{\tilde{\mathbf{Y}}}(\tilde{Y}_i | \tilde{\mathbf{D}}, \tilde{\mathbf{X}}, \tilde{\mathbf{A}}) = \mathbb{E}_{\tilde{\mathbf{Y}}}(\tilde{Y}_i | \tilde{\mathbf{X}}, \tilde{\mathbf{A}})$. Now, by definition of effective treatments,

$$1\{r_i \in D_j^r\} 1\{G|_{X_j}\} \mathbb{E}_{\tilde{\mathbf{Y}}}(\tilde{Y}_i | \tilde{\mathbf{X}}, \tilde{\mathbf{A}}) = 1\{r_i \in D_j^r\} 1\{G|_{X_j}\} \mathbb{E}(Y_j(\mathbf{T}|\mathbf{A}) | (\mathbf{A}, \mathbf{X}) \in G)$$

Substituting this into the above equation:

$$\mathbb{E}_{\sim}(\hat{\theta}) = \mathbb{E}(Y_j(\mathbf{T}|\mathbf{A}) | (\mathbf{A}, \mathbf{X}) \in G) \sum_{i=1}^n \frac{\mathbb{E}_{\tilde{\mathbf{D}}, \tilde{\mathbf{X}}, \tilde{\mathbf{A}}}(1\{r_i \in D_j^r\} 1\{G|_{X_j}\})}{\mathbf{P}_{\sim}(\{r_i \in D_j^r\}, \{G|_{X_j}\})} \\ = \mathbb{E}(Y_j(\mathbf{T}|\mathbf{A}) | (\mathbf{A}, \mathbf{X}) \in G) \blacksquare$$

Proof of Lemma 4

Proof: Define $\{\mathbf{c} \in D_j^c\} \equiv (\{c_i(\tilde{\mathbf{D}}; \tilde{\mathbf{A}}, \tilde{\mathbf{X}}) \in D_j^c(\mathbf{T}, G)\})_{i=1, \dots, n}$.

$$\begin{aligned}
& \mathbb{E}_{\sim} \left[\hat{\mathbb{E}}[Y_j(\mathbf{T}|\mathbf{A}) | (\mathbf{A}, \mathbf{X}) \in G] \right] \\
&= \mathbb{E}_{\tilde{\mathbf{A}}, \tilde{\mathbf{X}}} \left\{ \mathbb{E}_{\tilde{Y}_i, \tilde{\mathbf{D}}} \left[\sum_{i=1}^n \frac{\tilde{Y}_i \mathbf{1}\{c_i(\tilde{\mathbf{D}}; \tilde{\mathbf{A}}, \tilde{\mathbf{X}}) \in D_j^c(\mathbf{T}, G)\}}{\mathbf{P}[c_i(\tilde{\mathbf{D}}; \tilde{\mathbf{A}}, \tilde{\mathbf{X}}) \in D_j^c(\mathbf{T}, G)]} \middle| \{\mathbf{c} \in D_j^c\} \right] \right\} \\
&= \mathbb{E}_{\tilde{\mathbf{A}}, \tilde{\mathbf{X}}} \left\{ \mathbb{E}_{\tilde{\mathbf{D}}} \left[\sum_{i=1}^n \frac{\mathbb{E}_{\tilde{Y}_i}[\tilde{Y}_i | \mathbf{1}\{\mathbf{c} \in D_j^c\}] \mathbf{1}\{c_i(\tilde{\mathbf{D}}; \tilde{\mathbf{A}}, \tilde{\mathbf{X}}) \in D_j^c(\mathbf{T}, G)\}}{\mathbf{P}[c_i(\tilde{\mathbf{D}}; \tilde{\mathbf{A}}, \tilde{\mathbf{X}}) \in D_j^c(\mathbf{T}, G)]} \middle| \{\mathbf{c} \in D_j^c\} \right] \right\} \\
&= \mathbb{E}_{\sim} \left[\sum_{i=1}^n \frac{\mathbb{E}[Y_j(\mathbf{T}|\mathbf{A}) | (\mathbf{A}, \mathbf{X}) \in G] \mathbf{1}\{c_i(\tilde{\mathbf{D}}; \tilde{\mathbf{A}}, \tilde{\mathbf{X}}) \in D_j^c(\mathbf{T}, G)\}}{\mathbf{P}[c_i(\tilde{\mathbf{D}}; \tilde{\mathbf{A}}, \tilde{\mathbf{X}}) \in D_j^c(\mathbf{T}, G)]} \right] \\
&= \mathbb{E}[Y_j(\mathbf{T}|\mathbf{A}) | (\mathbf{A}, \mathbf{X}) \in G] \sum_{i=1}^n \frac{\mathbb{E}_{\sim}[\mathbf{1}\{c_i(\tilde{\mathbf{D}}; \tilde{\mathbf{A}}, \tilde{\mathbf{X}}) \in D_j^c(\mathbf{T}, G)\}]}{\mathbf{P}[c_i(\tilde{\mathbf{D}}; \tilde{\mathbf{A}}, \tilde{\mathbf{X}}) \in D_j^c(\mathbf{T}, G)]} \\
&= \mathbb{E}[Y_j(\mathbf{T}|\mathbf{A}) | (\mathbf{A}, \mathbf{X}) \in G]
\end{aligned}$$

The first equality follows from law of iterated expectations, the second equality follows from the unconfoundedness, the third follows from the definition of Homogenized effective treatments. ■

Proof of Lemma 5

Proof: Observe that:

$$W_{\mathcal{G}}^* - W(\hat{\mathbf{G}}_{EWM}) \geq 0$$

and that $\forall \mathbf{G} = \{G_1, \dots, G_Q\} \in \mathcal{G}$, define, where

$$W_n(\mathbf{G}) = \sum_{q=1}^Q F((\mathbf{A}, \mathbf{X}) \in G_q) \cdot \left(\frac{1}{J} \sum_{j=1}^J \hat{\mathbb{E}}[Y_j(\mathbf{T}_q|\mathbf{A}) | (\mathbf{A}, \mathbf{X}) \in G_q] \right)$$

Then, write:

$$\begin{aligned}
& W(\mathbf{G}) - W(\hat{\mathbf{G}}_{EWM}) \\
&= W(\mathbf{G}) - W_n(\hat{\mathbf{G}}_{EWM}) - W(\hat{\mathbf{G}}_{EWM}) + W_n(\hat{\mathbf{G}}_{EWM}) \\
&\leq W(\mathbf{G}) - W_n(\mathbf{G}) + \sup_{\mathbf{G} \in \mathcal{G}} |W_n(\mathbf{G}) - W(\mathbf{G})| \quad (\text{Since } W_n(\hat{\mathbf{G}}_{EWM}) \geq W_n(\mathbf{G})) \\
&\leq 2 \sup_{\mathbf{G} \in \mathcal{G}} |W_n(\mathbf{G}) - W(\mathbf{G})|
\end{aligned}$$

By application of triangle inequality,

$$\sup_{\mathbf{G} \in \mathcal{G}} |W_n(\mathbf{G}) - W(\mathbf{G})| \leq \sum_{q=1}^Q \sup_{G \in \mathcal{G}} |W_n^q(G) - W^q(G)|$$

where

$$W^q(G) = F((\mathbf{A}, \mathbf{X}) \in G) \frac{1}{J} \sum_{j=1}^J \mathbb{E}[Y_j(\mathbf{T}_q | \mathbf{A}) | (\mathbf{A}, \mathbf{X}) \in G]$$

and

$$W_n^q(G) = F((\mathbf{A}, \mathbf{X}) \in G) \frac{1}{J} \sum_{j=1}^J \hat{\mathbb{E}}[Y_j(\mathbf{T}_q | \mathbf{A}) | (\mathbf{A}, \mathbf{X}) \in G]$$

Thus, one can bound the uniform regret associated with the *empirical welfare maximization* rule as follows:

$$\sup_{\mathbf{P} \in \mathcal{P}} \mathbb{E}_{Data \sim (\mathbf{P}, F)} \left[W_{\mathcal{G}}^* - W(\hat{\mathbf{G}}_{EWM}) \right] \leq 2 \sum_{q=1}^Q \sup_{\mathbf{P} \in \mathcal{P}} \mathbb{E}_{Data \sim (\mathbf{P}, F)} \left[\sup_{G \in \mathcal{G}} |W_n^q(G) - W^q(G)| \right] \blacksquare$$

Proof of Lemma 6

Proof: Observe that:

$$W_{\mathcal{G}}^* - W(\hat{\mathbf{G}}_{EWM}) \geq 0$$

and that $\forall \mathbf{G} = \{G_1, \dots, G_Q\} \in \mathcal{G}$, define, where

$$W_N(\mathbf{G}) = \frac{1}{N} \sum_{i=1}^N \frac{\sum_{q=1}^Q \mathcal{Y}_{(i)} \cdot \mathbf{1}\{\tilde{\mathbf{D}}_{(i)} = \mathbf{T}_q\} \cdot \mathbf{1}\{(\tilde{\mathbf{A}}_{(i)}, \tilde{b}X_{(i)}) \in G_q\}}{\mathbf{P}(\tilde{\mathbf{D}}_{(i)} = \mathbf{T}_q | \tilde{\mathbf{A}}_{(i)}, \tilde{\mathbf{X}}_{(i)})}$$

Then, write:

$$\begin{aligned} & W(\mathbf{G}) - W(\hat{\mathbf{G}}_{EWM}) \\ &= W(\mathbf{G}) - W_N(\hat{\mathbf{G}}_{EWM}) - W(\hat{\mathbf{G}}_{EWM}) + W_N(\hat{\mathbf{G}}_{EWM}) \\ &\leq W(\mathbf{G}) - W_N(\mathbf{G}) + \sup_{\mathbf{G} \in \mathcal{G}} |W_N(\mathbf{G}) - W(\mathbf{G})| \quad (\text{Since } W_N(\hat{\mathbf{G}}_{EWM}) \geq W_N(\mathbf{G})) \\ &\leq 2 \sup_{\mathbf{G} \in \mathcal{G}} |W_N(\mathbf{G}) - W(\mathbf{G})| \end{aligned}$$

By application of triangle inequality,

$$\sup_{\mathbf{G} \in \mathbb{G}} |W_N(\mathbf{G}) - W(\mathbf{G})| \leq \sum_{q=1}^Q \sup_{G \in \mathbb{G}} |W_N^q(G) - W^q(G)|$$

where

$$W^q(G) = \mathbb{E} \left[\frac{1}{J} \sum_{j=1}^J Y_j(\mathbf{T}_q | \mathbf{A}) \cdot \mathbf{1}\{(\mathbf{A}, \mathbf{X}) \in G_q\} \right]$$

and

$$W_N^q(G) = \frac{1}{N} \sum_{i=1}^N \frac{\mathcal{Y}_{(i)} \cdot \mathbf{1}\{\tilde{\mathbf{D}}_{(i)} = \mathbf{T}_q\} \cdot \mathbf{1}\{(\tilde{\mathbf{A}}_{(i)}, \tilde{\mathbf{X}}_{(i)}) \in G_q\}}{\mathbf{P}(\tilde{\mathbf{D}}_{(i)} = \mathbf{T}_q | \tilde{\mathbf{A}}_{(i)}, \tilde{\mathbf{X}}_{(i)})}$$

Thus, one can bound the uniform regret associated with the *empirical welfare maximization* rule as follows:

$$\sup_{\mathbf{P} \in \mathcal{P}} \mathbb{E}_{Data \sim (\mathbf{P}, F)} \left[W_{\mathcal{G}}^* - W(\hat{\mathbf{G}}_{EWM}) \right] \leq 2 \sum_{q=1}^Q \sup_{\mathbf{P} \in \mathcal{P}} \mathbb{E}_{Data \sim (\mathbf{P}, F)} \left[\sup_{G \in \mathbb{G}} |W_N^q(G) - W^q(G)| \right]$$

I now provide a bound for $\mathbb{E}_{Data \sim (\mathbf{P}, F)} \left[\sup_{G \in \mathbb{G}} |W_N^q(G) - W^q(G)| \right]$ that is independent of \mathbf{P} and hence applies uniformly over \mathcal{P} . To that end, define $\mathbf{Z}_i \equiv (\tilde{\mathbf{Y}}_i, \tilde{\mathbf{D}}_i, \tilde{\mathbf{A}}_i, \tilde{\mathbf{X}}_i)$ and

$$W_N^q(G) = \frac{1}{n} \sum_{i=1}^n f(\mathbf{Z}_i; G)$$

Then, from the unbiasedness results of the cell-mean estimator:

$$\mathbb{E}_n(f(\cdot; G)) \equiv \frac{1}{n} \sum_{i=1}^n f(U_i; G) = W_N^q(G) \text{ and } \mathbb{E}_{Data \sim (\mathbf{P}, F)}(f(\cdot; G)) = \mathbb{E}_{Data \sim (\mathbf{P}, F)}[f(U_i; G)] = W^q(G)$$

Denote by $\mathcal{F} \equiv \{f(\cdot; G) : G \in \mathbb{G}\}$ the collection of all such functions generated by decision sets. By Lemma A.1 of Kitagawa-Tetenov (2018), \mathcal{F} is a VC-subgraph class of functions with VC dimension less than or equal to v , when \mathbb{G} has VC dimension v . Thus, one can write

$$\mathbb{E}_{Data \sim (\mathbf{P}, F)} \left[\sup_{G \in \mathbb{G}} |W_N^q(G) - W^q(G)| \right] \leq \mathbb{E}_{Data \sim (\mathbf{P}, F)} \left[\sup_{f \in \mathcal{F}} |\mathbb{E}_n(f) - \mathbb{E}(f)| \right] \quad \blacksquare$$

Proof of Lemma 7

Proof: The quantity of interest is:

$$\mathbb{E}_{\mathcal{D}_n} \left[\mathbb{E}_{Z_{1:n}|\mathcal{D}_n} \left[\sup_{f \in \mathcal{F}} \left| \mathbb{E}_n f - \mathbb{E} f \right| \right] \right]$$

Using the metric defined on the integer lattice, the following correlation structure is assumed:

$$\mathbf{k} \notin B_\rho(\mathbf{1}, \kappa) \Rightarrow \text{corr}(Z(\mathbf{k}), Z(\mathbf{1})) = 0$$

where $B_\rho(\mathbf{1}, \kappa) = \{\mathbf{k} : \rho(\mathbf{k}, \mathbf{1}) \leq \kappa \bar{\rho}\}$.

Now, I decompose the integer lattice into 2^d categories and use these to then construct Bernstein hypercubes.

For any directional vector $\mathbf{b} \in \{0, 1\}^d$, define:

$$\mathcal{C}_{\mathbf{b}} \equiv \{B_\rho(\mathbf{k}, \kappa) : \text{where } \forall 1 \leq i \leq d, k_i = (2a_i + b_i)\kappa\bar{\rho} \text{ where } \mathbf{a} \in \mathbb{Z}^d\}$$

Now, the idea is to define a Bernstein hypercube with the following feature:

1. Each Bernstein hypercubes contains exactly one elements from each category.

Thus, I define, $\forall \mathbf{m} \in \mathbb{Z}^d$,

$$B_{\mathbf{m}} \equiv \bigcup_{\mathbf{b} \in \{0,1\}^d} \{ \mathbf{l} \in B_\rho(\mathbf{k}, \kappa) : \text{where } \forall 1 \leq i \leq d, k_i = (2a_i + b_i)\kappa\bar{\rho} \}$$

Now, notice that for any fixed \mathcal{D}_n , there exists at most finite number of Bernstein hypercubes which are non-empty. Consequently, these non-empty elements can be numbered: $B(1), B(2), \dots, B(\mu_n)$, where $\mu_n = |\{\mathbf{m} : B_{\mathbf{m}}(\mathcal{D}_n) \neq \emptyset\}|$. Note that by the network formation assumption, for any $1 \leq i \leq \mu_n$, $a_n^i \equiv |B(i)| \leq (1 + 2\kappa\bar{\rho})^d 2^d$. Thus, by definition $n = \sum_{j=1}^{\mu_n} a_n^j$.

Define, $\bar{a}_n = \frac{\sum_{j=1}^{\mu_n} a_n^j}{\mu_n}$, the average density of a Bernstein hypercube. Note here that is stochastic since it depends on \mathcal{D}_n . With this set up, I can make progress on the object of interest.

$$\begin{aligned}
& \mathbb{E}_{\mathcal{D}_n} \left[\mathbb{E}_{Z_{1:n}|\mathcal{D}_n} \left[\sup_{f \in \mathcal{F}} \left| \frac{1}{n} \sum_{i=1}^n f(Z_i) - \mathbb{E}f \right| \right] \right] \\
&= \mathbb{E}_{\mathcal{D}_n} \left[\mathbb{E}_{Z_{1:n}|\mathcal{D}_n} \left[\sup_{f \in \mathcal{F}} \left| \frac{1}{\mu_n} \sum_{j=1}^{\mu_n} \frac{1}{\bar{a}_n} \sum_{\mathbf{b} \in \{0,1\}^d} \left(\sum_{\mathbf{l} \in D_n \cap C_{\mathbf{b}} \cap B(j)} f(Z(\mathbf{l})) \right) - \mathbb{E}f \right| \right] \right]
\end{aligned}$$

By triangle inequality,

$$\leq \mathbb{E}_{\mathcal{D}_n} \left[\mathbb{E}_{Z_{1:n}|\mathcal{D}_n} \left[\sup_{f \in \mathcal{F}} \sum_{\mathbf{b} \in \{0,1\}^d} \left| \frac{1}{\mu_n} \sum_{j=1}^{\mu_n} \frac{1}{\bar{a}_n} \left(\sum_{\mathbf{l} \in D_n \cap C_{\mathbf{b}} \cap B(j)} f(Z(\mathbf{l})) \right) - \mathbb{E}f \right| \right] \right]$$

By splitting the sup and using linearity of \mathbb{E} ,

$$\leq \sum_{\mathbf{b} \in \{0,1\}^d} \mathbb{E}_{\mathcal{D}_n} \left[\mathbb{E}_{Z_{1:n}|\mathcal{D}_n} \left[\sup_{f \in \mathcal{F}} \left| \frac{1}{\mu_n} \sum_{j=1}^{\mu_n} \frac{1}{\bar{a}_n} \left(\sum_{\mathbf{l} \in D_n \cap C_{\mathbf{b}} \cap B(j)} f(Z(\mathbf{l})) \right) - \mathbb{E}f \right| \right] \right]$$

By the strong stationarity imposed in assumption 4, this can be equivalently written as

$$= 2^d \mathbb{E}_{\mathcal{D}_n} \left[\mathbb{E}_{Z_{1:n}|\mathcal{D}_n} \left[\sup_{f \in \mathcal{F}} \left| \frac{1}{\mu_n} \sum_{j=1}^{\mu_n} \frac{1}{\bar{a}_n} \left(\sum_{\mathbf{l} \in D_n \cap C_{\mathbf{b}} \cap B(j)} f(Z(\mathbf{l})) \right) - \mathbb{E}f \right| \right] \right]$$

Notice that for any $\mathbf{b} \in \{0,1\}^d$, elements in $D_n \cap C_{\mathbf{b}} \cap B(j)$ and $D_n \cap C_{\mathbf{b}} \cap B(k)$ are uncorrelated for $j \neq k$. Now, I start the process of symmetrization. In order to do so, create an independent and identically distributed copy $Z'_{1:n}$ of $Z_{1:n}$.

$$\begin{aligned}
&= 2^d \mathbb{E}_{\mathcal{D}_n} \left[\mathbb{E}_{Z_{1:n}|\mathcal{D}_n} \left[\sup_{f \in \mathcal{F}} \left| \frac{1}{\mu_n} \sum_{j=1}^{\mu_n} \frac{1}{\bar{a}_n} \left(\sum_{\mathbf{l} \in D_n \cap C_{\mathbf{b}} \cap B(j)} f(Z(\mathbf{l})) \right) \right. \right. \\
&\quad \left. \left. - \mathbb{E}_{Z'_{1:n}|\mathcal{D}_n} \frac{1}{\mu_n} \sum_{j=1}^{\mu_n} \frac{1}{\bar{a}_n} \left(\sum_{\mathbf{l} \in D_n \cap C_{\mathbf{b}} \cap B(j)} f(Z'(\mathbf{l})) \right) \right| \right] \right] \\
&\leq 2^d \mathbb{E}_{\mathcal{D}_n} \left[\mathbb{E}_{Z_{1:n}, Z'_{1:n}|\mathcal{D}_n} \left[\sup_{f \in \mathcal{F}} \left| \frac{1}{\mu_n} \sum_{j=1}^{\mu_n} \frac{1}{\bar{a}_n} \left(\sum_{\mathbf{l} \in D_n \cap C_{\mathbf{b}} \cap B(j)} f(Z(\mathbf{l})) \right) \right. \right. \right. \\
&\quad \left. \left. - \frac{1}{\mu_n} \sum_{j=1}^{\mu_n} \frac{1}{\bar{a}_n} \left(\sum_{\mathbf{l} \in D_n \cap C_{\mathbf{b}} \cap B(j)} f(Z'(\mathbf{l})) \right) \right| \right] \right]
\end{aligned}$$

The inequality follows from the application of Jensen's inequality. Now, define a Rademacher

sequence $\sigma_{1:\mu_n}$ that is independent of both $Z'_{1:n}$ and $Z_{1:n}$. Then, by triangle inequality

$$\leq 2 \cdot 2^d \mathbb{E}_{\mathcal{D}_n} \left[\frac{1}{\mu_n} \mathbb{E}_{Z_{1:n}, \sigma_{1:\mu_n} | \mathcal{D}_n} \left[\sup_{f \in \mathcal{F}} \left| \sum_{j=1}^{\mu_n} \frac{\sigma_j}{\bar{a}_n} \sum_{\mathbf{l} \in D_n \cap C_{\mathbf{b}} \cap B(j)} f(Z(\mathbf{l})) \right| \right] \right]$$

This is known as the Rademacher complexity bound. Fix any \mathbf{b} , \mathcal{D}_n and $Z_{1:n}$, then define a μ_n dimensional vector

$$\mathbf{f} \equiv \left(\frac{1}{\bar{a}_n} \sum_{\mathbf{l} \in D_n \cap C_{\mathbf{b}} \cap B(j)} f(Z(\mathbf{l})) \right)_{j=1}^{\mu_n} \equiv \left(f_{C_n^j} \right)_{j=1}^{\mu_n}$$

Notice that for any $1 \leq j \leq \mu_n$, $f_{C_n^j} \leq (1 + 2\kappa\bar{\rho})^d \bar{F}$.

Thus, define: $\mathbb{F}_n \equiv \{ \mathbf{f} : \|\mathbf{f}\|_\infty \leq (1 + 2\kappa\bar{\rho})^d \bar{F} \} \subset \mathbb{R}^{\mu_n}$. Now, define the following Euclidean norm to \mathbb{F}_n :

$$d_n(\mathbf{f}, \mathbf{g}) \equiv \left[\frac{1}{\mu_n} \sum_{j=1}^{\mu_n} \left(f_{C_n^j} - g_{C_n^j} \right)^2 \right]^{1/2}$$

Also define the Rademacher complexity maximizing element,

$$\mathbf{f}_n^* \equiv (f_{n,j}^*)_{j=1}^{\mu_n} \in \arg \max_{\mathbf{f} \in \mathbb{F}_n} \left| \sum_{j=1}^{\mu_n} \sigma_j f_{C_n^j} \right|$$

Next, I define a sequence of minimal covers of \mathbb{F}_n . In order to do so, start by defining

$$\mathbf{f}_n^{(0)} \equiv (0, \dots, 0)$$

Then, for any $k \geq 1$, denote a minimal cover of radius $2^{-k}(2\kappa\bar{\rho} + 1)^d \bar{F}$ by M_k^n . Since \mathbb{F}_n is totally bounded, there exists a \bar{K} such that $M_{\bar{K}}^n = \mathbb{F}_n$. Now, within each minimal cover, define the Rademacher complexity maximizing element as

$$\forall 1 \leq k \leq \bar{K}, \mathbf{f}_n^{(k)} \in \arg \max_{\mathbf{f} \in M_k^n} \left| \sum_{j=1}^{\mu_n} \sigma_j f_{C_n^j} \right|$$

A straightforward application of triangle inequality yields:

$$d_n(\mathbf{f}_n^{(k)}, \mathbf{f}_n^{(k-1)}) \leq 3 \cdot 2^{-k} (1 + 2\kappa\bar{\rho})^d \bar{F}$$

Consequently, using the telescopic sum representation:

$$\begin{aligned}
& \mathbb{E}_{\sigma_{1:\mu_n}} \left| \sum_{j=1}^{\mu_n} \sigma_j f_{C_n^j}^* \right| \\
& \leq \sum_{k=1}^{\bar{K}} \mathbb{E}_{\sigma_{1:\mu_n}} \left| \sum_{j=1}^{\mu_n} \sigma_j \left(f_{C_n^j}^{(k)} - f_{C_n^j}^{(k-1)} \right) \right| \\
& \leq \sum_{k=1}^{\bar{K}} \mathbb{E}_{\sigma_{1:\mu_n}} \max_{\mathbf{f} \in M_k^n, \mathbf{g} \in M_{k-1}^n : d_n(\mathbf{f}, \mathbf{g}) \leq 2^{-k}(1+2\kappa\bar{\rho})^d \bar{F}} \left| \sum_{j=1}^{\mu_n} \sigma_j \left(f_{C_n^j} - g_{C_n^j} \right) \right|
\end{aligned}$$

Now, I'd like to use the maximal inequality to provide a bound for the above. In order to do, I first verify that an exponential bound holds. For this, I use Hoeffding's inequality:

$$\begin{aligned}
\mathbb{E}_{\sigma_{1:\mu_n}} \left(e^{s \sum_{j=1}^{\mu_n} \sigma_j (f_{C_n^j} - g_{C_n^j})} \right) &= \prod_{j=1}^{\mu_n} \mathbb{E}_{\sigma_j} \left(e^{s \sigma_j (f_{C_n^j} - g_{C_n^j})} \right) \\
&\leq \prod_{j=1}^{\mu_n} e^{s^2 (f_{C_n^j} - g_{C_n^j})^2 / 2} \\
&= e^{s^2 \mu_n d_n(\mathbf{f}, \mathbf{g})^2 / 2} \\
&\leq e^{s^2 \mu_n (3 \cdot 2^{-k} (1+2\kappa\bar{\rho})^d \bar{F})^2 / 2}
\end{aligned}$$

This exponential bound allows us to write the maximal inequality bound:

$$\begin{aligned}
& \mathbb{E}_{\sigma_{1:\mu_n}} \max_{\mathbf{f} \in M_k^n, \mathbf{g} \in M_{k-1}^n : d_n(\mathbf{f}, \mathbf{g}) \leq 2^{-k}(1+2\kappa\bar{\rho})^d \bar{F}} \left| \sum_{j=1}^{\mu_n} \sigma_j \left(f_{C_n^j} - g_{C_n^j} \right) \right| \\
& \leq 3 \cdot 2^{-k} (1 + 2\kappa\bar{\rho})^d \bar{F} \sqrt{\mu_n} \sqrt{2 \ln(2|M_k^n|^2)} \\
& = 6 \cdot 2^{-k} (1 + 2\kappa\bar{\rho})^d \bar{F} \sqrt{\mu_n} \sqrt{\ln(\sqrt{2}N(2^{-k}(2\kappa\bar{\rho} + 1)^d \bar{F}, \mathbb{F}_n, d_n))}
\end{aligned}$$

where $N(2^{-k}(2\kappa\bar{\rho}+1)^d \bar{F}, \mathbb{F}_n, d_n)$ is the covering number. This allows us to provide an entropy bound on the Rademacher complexity:

$$\begin{aligned}
& \mathbb{E}_{\sigma_{1:\mu_n}} \left| \sum_{j=1}^{\mu_n} \sigma_j f_{C_n^j}^* \right| \\
& \leq \sum_{k=1}^{\bar{K}} 6 \cdot 2^{-k} (1 + 2\kappa\bar{\rho})^{d\bar{F}} \sqrt{\mu_n} \sqrt{\ln(\sqrt{2N}(2^{-k}(2\kappa\bar{\rho} + 1)^{d\bar{F}}, \mathbb{F}_n, d_n))} \\
& \leq 12(1 + 2\kappa\bar{\rho})^{d\bar{F}} \sqrt{\mu_n} \int_0^1 \sqrt{\ln(\sqrt{2N}(\epsilon(2\kappa\bar{\rho} + 1)^{d\bar{F}}, \mathbb{F}_n, d_n))} d\epsilon
\end{aligned}$$

Theorem 2.6.7 from van der Vaart, Wellner (1996) provides a bound for the covering number in terms of the VC dimension.

$$N(\epsilon(2\kappa\bar{\rho} + 1)^{d\bar{F}}, \mathbb{F}_n, d_n) \leq C_3(v + 1)(16e)^{v+1} \left(\frac{1}{\epsilon}\right)^{2v}$$

where C_3 is a universal constant. Then,

$$\mathbb{E}_{\sigma_{1:\mu_n}} \left| \sum_{j=1}^{\mu_n} \sigma_j f_{C_n^j}^* \right| \leq C_2(1 + 2\kappa\bar{\rho})^{d\bar{F}} \sqrt{\mu_n v}$$

Consequently, the main object of interest can be bounded:

$$\begin{aligned}
& \mathbb{E}_{\mathcal{D}_n} \left[\mathbb{E}_{Z_{1:n}|\mathcal{D}_n} \left[\sup_{f \in \mathcal{F}} \left| \frac{1}{n} \sum_{i=1}^n f(Z_i) - \mathbb{E}f \right| \right] \right] \\
& \leq 2^{d+1} \mathbb{E}_{\mathcal{D}_n} \left[C_2(1 + 2\kappa\bar{\rho})^{d\bar{F}} \sqrt{\frac{v}{\mu_n}} \right]
\end{aligned}$$

Now, writing $\mu_n = \frac{n}{\bar{a}_n}$ and subsequently using Jensen's inequality

$$\begin{aligned}
& \mathbb{E}_{\mathcal{D}_n} \left[\mathbb{E}_{Z_{1:n}|\mathcal{D}_n} \left[\sup_{f \in \mathcal{F}} \left| \frac{1}{n} \sum_{i=1}^n f(Z_i) - \mathbb{E}f \right| \right] \right] \\
& \leq C_2(1 + 2\kappa\bar{\rho})^{d\bar{F}} \sqrt{\frac{v}{n}} \sqrt{\mathbb{E}_{\mathcal{D}_n} \bar{a}_n} \\
& \leq 2C_1 [2^d(1 + 2\kappa\bar{\rho})^d] 3/2\bar{F} \sqrt{\frac{v}{n}}
\end{aligned}$$

The last inequality follows from the fact that $a_n^j \leq 2^d(1 + 2\kappa\bar{\rho})^d$ for all j with probability

1. ■

Proof of Lemma 8

Proof: The quantity of interest is:

$$\mathbb{E}_{\mathbf{Z}_{1:N}} \left[\sup_{f \in \mathcal{F}} \left| \mathbb{E}_N f - \mathbb{E} f \right| \right]$$

The steps involved in this proof mirror Lemma A.4 in Kitagawa, Tetenov (2018). The proof is included here for completeness.

In order to symmetrize the process, I create an independent and identically distributed copy $\mathbf{Z}'_{1:N}$ of $\mathbf{Z}_{1:N}$.

$$\mathbb{E}_{\mathbf{Z}_{1:N}} \left[\sup_{f \in \mathcal{F}} \left| \mathbb{E}_N f - \mathbb{E} f \right| \right] = \mathbb{E}_{\mathbf{Z}_{1:N}} \left[\sup_{f \in \mathcal{F}} \left| \frac{1}{N} \sum_{i=1}^N f(\mathbf{Z}_i) - \mathbb{E}_{\mathbf{Z}'_{1:N}} \frac{1}{N} \sum_{i=1}^N f(\mathbf{Z}'_i) \right| \right]$$

An application on Jensen's inequality yields that:

$$\begin{aligned} \mathbb{E}_{\mathbf{Z}_{1:N}} \left[\sup_{f \in \mathcal{F}} \left| \mathbb{E}_N f - \mathbb{E} f \right| \right] &\leq \mathbb{E}_{\mathbf{Z}_{1:N}, \mathbf{Z}'_{1:N}} \left[\sup_{f \in \mathcal{F}} \left| \frac{1}{N} \sum_{i=1}^N f(\mathbf{Z}_i) - \frac{1}{N} \sum_{i=1}^N f(\mathbf{Z}'_i) \right| \right] \\ &= \mathbb{E}_{\mathbf{Z}_{1:N}, \mathbf{Z}'_{1:N}} \left[\frac{1}{N} \sup_{f \in \mathcal{F}} \left| \sum_{i=1}^N \left(f(\mathbf{Z}_i) - f(\mathbf{Z}'_i) \right) \right| \right] \end{aligned}$$

Now, define a Rademacher sequence $\sigma_{1:N}$ that is independent of both $\mathbf{Z}'_{1:n}$ and $\mathbf{Z}_{1:n}$. Then, $\sigma_i(f(\mathbf{Z}_i) - f(\mathbf{Z}'_i)) \sim (f(\mathbf{Z}_i) - f(\mathbf{Z}'_i))$, and so

$$\begin{aligned} \mathbb{E}_{\mathbf{Z}_{1:N}} \left[\sup_{f \in \mathcal{F}} \left| \mathbb{E}_N f - \mathbb{E} f \right| \right] &\leq \mathbb{E}_{\mathbf{Z}_{1:N}, \mathbf{Z}'_{1:N}, \sigma_{1:N}} \left[\frac{1}{N} \sup_{f \in \mathcal{F}} \left| \sum_{i=1}^N \sigma_i \left(f(\mathbf{Z}_i) - f(\mathbf{Z}'_i) \right) \right| \right] \\ &\leq \mathbb{E}_{\mathbf{Z}_{1:N}, \mathbf{Z}'_{1:N}, \sigma_{1:N}} \left[\frac{1}{N} \sup_{f \in \mathcal{F}} \left| \sum_{i=1}^N \sigma_i f(\mathbf{Z}_i) \right| + \left| \sum_{i=1}^N \sigma_i f(\mathbf{Z}'_i) \right| \right] \\ &\leq \frac{2}{N} \mathbb{E}_{\mathbf{Z}_{1:N}, \sigma_{1:N}} \left[\sup_{f \in \mathcal{F}} \left| \sum_{i=1}^N \sigma_i f(\mathbf{Z}_i) \right| \right] \end{aligned}$$

This is known as the Rademacher complexity bound. Fix any $\mathbf{Z}_{1:N}$, then define a N dimensional vector

$$\mathbf{f} \equiv \left(f(\mathbf{Z}_i) \right)_{i=1}^N$$

By definition, for any $1 \leq i \leq N$, $f_i \leq \bar{F}$.

Thus, define: $\mathbb{F} \equiv \{\mathbf{f} : \|\mathbf{f}\|_\infty \leq \bar{F}\} \subset \mathbb{R}^{\mu_n}$. Now, introduce the following Euclidean norm to \mathbb{F} :

$$d(\mathbf{f}, \mathbf{g}) \equiv \left[\frac{1}{N} \sum_{i=1}^N (f_i - g_i)^2 \right]^{1/2}$$

Also define the Rademacher complexity maximizing element,

$$\mathbf{f}^* \equiv (f_i^*)_{i=1}^N \in \arg \max_{\mathbf{f} \in \mathbb{F}} \left| \sum_{i=1}^N \sigma_i f_i \right|$$

Next, I define a sequence of minimal covers of \mathbb{F}_n . In order to do so, start by defining

$$\mathbf{f}^{(0)} \equiv (0, \dots, 0)$$

Then, for any $k \geq 1$, denote a minimal cover of radius $2^{-k}\bar{F}$ by M_k . Since \mathbb{F} is totally bounded, there exists a \bar{K} such that $M_{\bar{K}} = \mathbb{F}$. Now, within each minimal cover, define the Rademacher complexity maximizing element as

$$\forall 1 \leq k \leq \bar{K}, \mathbf{f}^{(k)} \in \arg \max_{\mathbf{f} \in M_k} \left| \sum_{i=1}^N \sigma_i f_i \right|$$

A straightforward application of triangle inequality yields:

$$d(\mathbf{f}^{(k)}, \mathbf{f}^{(k-1)}) \leq 3 \cdot 2^{-k}\bar{F}$$

Consequently, using the telescopic sum representation:

$$\begin{aligned} & \mathbb{E}_{\sigma_{1:N}} \left| \sum_{i=1}^N \sigma_i f_i^* \right| \\ & \leq \sum_{k=1}^{\bar{K}} \mathbb{E}_{\sigma_{1:N}} \left| \sum_{i=1}^N \sigma_i (f_i^{(k)} - f_i^{(k-1)}) \right| \\ & \leq \sum_{k=1}^{\bar{K}} \mathbb{E}_{\sigma_{1:N}} \max_{\mathbf{f} \in M_k, \mathbf{g} \in M_{k-1}: d(\mathbf{f}, \mathbf{g}) \leq 2^{-k}\bar{F}} \left| \sum_{i=1}^N \sigma_i (f_i - g_i) \right| \end{aligned}$$

Now, I'd like to use the maximal inequality to provide a bound for the above. In order to do, I first verify that an exponential bound holds. For this, I use Hoeffding's inequality:

$$\begin{aligned}
\mathbb{E}_{\sigma_{1:N}} \left(e^{s \sum_{i=1}^N \sigma_i (f_i - g_i)} \right) &= \prod_{i=1}^N \mathbb{E}_{\sigma_i} \left(e^{s \sigma_i (f_i - g_i)} \right) \\
&\leq \prod_{i=1}^N e^{s^2 (f_i - g_i)^2 / 2} \\
&= e^{s^2 N d(\mathbf{f}, \mathbf{g})^2 / 2} \\
&\leq e^{s^2 N \cdot (3 \cdot 2^{-k} \bar{F})^2 / 2}
\end{aligned}$$

This exponential bound allows us to write the maximal inequality bound:

$$\begin{aligned}
&\mathbb{E}_{\sigma_{1:N}} \max_{\mathbf{f} \in M_k, \mathbf{g} \in M_{k-1}: d(\mathbf{f}, \mathbf{g}) \leq 2^{-k} \bar{F}} \left| \sum_{i=1}^N \sigma_i (f_i - g_i) \right| \\
&\leq 3 \cdot 2^{-k} \bar{F} \sqrt{N} \sqrt{2 \ln(2|M_k|^2)} \\
&= 6 \cdot 2^{-k} \bar{F} \sqrt{N} \sqrt{\ln(\sqrt{2} N (2^{-k} \bar{F}, \mathbb{F}, d))}
\end{aligned}$$

where $N(2^{-k} \bar{F}, \mathbb{F}, d)$ is the covering number. This allows us to provide an entropy bound on the Rademacher complexity:

$$\begin{aligned}
&\mathbb{E}_{\sigma_{1:N}} \left| \sum_{i=1}^N \sigma_i f_i^* \right| \\
&\leq 6 \cdot 2^{-k} \bar{F} \sqrt{N} \sqrt{\ln(\sqrt{2} N (2^{-k} \bar{F}, \mathbb{F}, d))} \\
&\leq 12 \bar{F} \sqrt{N} \int_0^1 \sqrt{\ln(\sqrt{2} N (\epsilon \bar{F}, \mathbb{F}, d))} d\epsilon
\end{aligned}$$

Theorem 2.6.7 Van Der Vaart, Wellner (1996) provides a bound for the covering number in terms of the VC dimension.

$$N(\epsilon \bar{F}, \mathbb{F}, d) \leq C_3' (v+1) (16e)^{v+1} \left(\frac{1}{\epsilon} \right)^{2v}$$

where C_3 is a universal constant. Then,

$$\mathbb{E}_{\sigma_{1:N}} \left| \sum_{i=1}^N \sigma_i f_i^* \right| \leq C'_2 \bar{F} \sqrt{Nv}$$

Consequently, the main object of interest can be bounded:

$$\mathbb{E}_{\mathbf{Z}_{1:N}} \left[\sup_{f \in \mathcal{F}} \left| \mathbb{E}_N f - \mathbb{E} f \right| \right] \leq 2 \left[C'_2 \bar{F} \sqrt{\frac{v}{N}} \right] \quad \blacksquare$$

Proof of Theorem 1

Proof: I now provide a bound for $\mathbb{E}_{Data \sim (\mathbf{P}, F)} \left[\sup_{G \in \mathbb{G}} |W_n^q(G) - W^q(G)| \right]$ that is independent of \mathbf{P} and hence applies uniformly over \mathcal{P} . To that end, define $Z_i \equiv \tilde{Y}_i, r_i(\tilde{\mathbf{D}}; \tilde{\mathbf{A}}), \tilde{X}_i$ and

$$f(Z_i; G) \equiv \frac{\tilde{Y}_i 1\{r_i(\tilde{\mathbf{D}}; \tilde{\mathbf{A}}) \in D_j^r(\mathbf{T}, G), \tilde{X}_i \in G|_{X_j}\}}{\mathbf{P}_{\sim}[r_i(\tilde{\mathbf{D}}; \tilde{\mathbf{A}}) \in D_j^r(\mathbf{T}, G), \tilde{X}_i \in G|_{X_j}]}$$

Then, from the unbiasedness results of the cell-mean estimator:

$$\mathbb{E}_n(f(\cdot; G)) \equiv \frac{1}{n} \sum_{i=1}^n f(U_i; G) = W_n^q(G) \text{ and } \mathbb{E}_{Data \sim (\mathbf{P}, F)}(f(\cdot; G)) = \mathbb{E}_{Data \sim (\mathbf{P}, F)}[f(U_i; G)] = W^q(G)$$

Denote by $\mathcal{F} \equiv \{f(\cdot; G) : G \in \mathbb{G}\}$ the collection of all such functions generated by decision sets. By Lemma A.1 of Kitagawa-Tetenov (2018), \mathcal{F} is a VC-subgraph class of functions with VC dimension less than or equal to v , when \mathbb{G} has VC dimension v . Thus, one can write

$$\mathbb{E}_{Data \sim (\mathbf{P}, F)} \left[\sup_{G \in \mathbb{G}} |W_n^q(G) - W^q(G)| \right] \leq \mathbb{E}_{Data \sim (\mathbf{P}, F)} \left[\sup_{f \in \mathcal{F}} |\mathbb{E}_n(f) - \mathbb{E}(f)| \right]$$

Using lemma 9, with $\bar{F} = \frac{M}{2\gamma}$, I get the following bound:

$$\sup_{\mathbf{P} \in \mathcal{P}} \mathbb{E}_{Data \sim (\mathbf{P}, F)} \left[W_G^* - W(\hat{\mathbf{G}}_{EWM}) \right] \leq 4QC_1 [2^d (1 + 2\kappa\bar{\rho})^d] 3/2 \frac{M}{2\gamma} \sqrt{\frac{v}{n}} \quad \blacksquare$$

Proof of Theorem 2

Proof: I now provide a bound for $\mathbb{E}_{Data \sim (\mathbf{P}, F)} \left[\sup_{G \in \mathbb{G}} |W_n^q(G) - W^q(G)| \right]$ that is independent of \mathbf{P} and hence applies uniformly over \mathcal{P} . To that end, define $Z_i \equiv \tilde{Y}_i, c_i(\tilde{\mathbf{D}}; \tilde{\mathbf{A}}, \tilde{\mathbf{X}})$ and

$$f(Z_i; G) \equiv \frac{\tilde{Y}_i 1\{c_i(\tilde{\mathbf{D}}; \tilde{\mathbf{A}}, \tilde{\mathbf{X}}) \in D_j^c(\mathbf{T}, G)\}}{\mathbf{P}_{\sim}[c_i(\tilde{\mathbf{D}}; \tilde{\mathbf{A}}, \tilde{\mathbf{X}}) \in D_j^c(\mathbf{T}, G)]}$$

Then, from the unbiasedness results of the cell-mean estimator:

$$\mathbb{E}_n(f(\cdot; G)) \equiv \frac{1}{n} \sum_{i=1}^n f(U_i; G) = W_n^q(G) \text{ and } \mathbb{E}_{Data \sim (\mathbf{P}, F)}(f(\cdot; G)) = \mathbb{E}_{Data \sim (\mathbf{P}, F)}[f(U_i; G)] = W^q(G)$$

Denote by $\mathcal{F} \equiv \{f(\cdot; G) : G \in \mathbb{G}\}$ the collection of all such functions generated by decision sets. By Lemma A.1 of Kitagawa-Tetenov (2018), \mathcal{F} is a VC-subgraph class of functions with VC dimension less than or equal to v , when \mathbb{G} has VC dimension v . Thus, one can write

$$\mathbb{E}_{Data \sim (\mathbf{P}, F)} \left[\sup_{G \in \mathbb{G}} |W_n^q(G) - W^q(G)| \right] \leq \mathbb{E}_{Data \sim (\mathbf{P}, F)} \left[\sup_{f \in \mathcal{F}} |\mathbb{E}_n(f) - \mathbb{E}(f)| \right]$$

Using lemma 9, with $\bar{F} = \frac{1}{1\bar{\gamma}}$, I get the following bound:

$$\sup_{\mathbf{P} \in \mathcal{P}} \mathbb{E}_{Data \sim (\mathbf{P}, F)} \left[W_{\mathcal{G}}^* - W(\hat{\mathbf{G}}_{EWM}) \right] \leq 4QC_1 [2^d(1 + 2\kappa\bar{\rho})^d] 3/2 \frac{1}{1\bar{\gamma}} \sqrt{\frac{v}{n}} \quad \blacksquare$$

Proof of Theorem 3

Proof: I now provide a bound for $\mathbb{E}_{Data \sim (\mathbf{P}, F)} \left[\sup_{G \in \mathbb{G}} |W_n^q(G) - W^q(G)| \right]$ that is independent of \mathbf{P} and hence applies uniformly over \mathcal{P} . To that end, define $Z_i \equiv \tilde{Y}_i, c_i(\tilde{\mathbf{D}}; \tilde{\mathbf{A}}, \tilde{\mathbf{X}})$ and

$$f(Z_i; G) \equiv \frac{\tilde{Y}_i 1\{c_i(\tilde{\mathbf{D}}; \tilde{\mathbf{A}}, \tilde{\mathbf{X}}) \in D_j^c(\mathbf{T}, G)\}}{\mathbf{P}_{\sim}[c_i(\tilde{\mathbf{D}}; \tilde{\mathbf{A}}, \tilde{\mathbf{X}}) \in D_j^c(\mathbf{T}, G)]}$$

Then, from the unbiasedness results of the cell-mean estimator:

$$\mathbb{E}_n(f(\cdot; G)) \equiv \frac{1}{n} \sum_{i=1}^n f(U_i; G) = W_n^q(G) \text{ and } \mathbb{E}_{Data \sim (\mathbf{P}, F)}(f(\cdot; G)) = \mathbb{E}_{Data \sim (\mathbf{P}, F)}[f(U_i; G)] = W^q(G)$$

Denote by $\mathcal{F} \equiv \{f(\cdot; G) : G \in \mathbb{G}\}$ the collection of all such functions generated by decision sets. By Lemma A.1 of Kitagawa-Tetenov (2018), \mathcal{F} is a VC-subgraph class of

functions with VC dimension less than or equal to v , when \mathbb{G} has VC dimension v . Thus, one can write

$$\mathbb{E}_{Data \sim (\mathbf{P}, F)} \left[\sup_{G \in \mathbb{G}} |W_n^q(G) - W^q(G)| \right] \leq \mathbb{E}_{Data \sim (\mathbf{P}, F)} \left[\sup_{f \in \mathcal{F}} |\mathbb{E}_n(f) - \mathbb{E}(f)| \right]$$

Using lemma 9, with $\bar{F} = \frac{1}{1\gamma}$, I get the following bound:

$$\sup_{\mathbf{P} \in \mathcal{P}} \mathbb{E}_{Data \sim (\mathbf{P}, F)} \left[W_{\mathcal{G}}^* - W(\hat{\mathbf{G}}_{EWM}) \right] \leq 4QC_1 \frac{\max\{1, M\}}{2\bar{\gamma}} \sqrt{\frac{v}{N}} \quad \blacksquare$$

Proof of Theorem 4

Proof: Recall that

$$\mathcal{Z}(\cdot | \text{Data}) : \mathcal{S} \rightarrow \{0, 1\}^J$$

Thus, the treatment assignment rules are characterized by the 2^J -way partition on \mathcal{S} , $G^{1:Q} \equiv G^1, \dots, G^Q$ where $Q = 2^J$ and each $G^q \in \mathcal{G}$ for all $1 \leq q \leq Q$. Thus, the village level welfare can be written as:

$$W(G^{1:Q}) = \sum_{j=1}^J \mathbb{E} \left[\sum_{q=1}^Q Y_j(\mathbf{T}_q | \mathbf{A}) 1\{(\mathbf{X}, \mathbf{A}) \in G^q\} \right]$$

where $\mathbf{X} = (X_1, \dots, X_J)$ and $\{Y_j(\mathbf{T} | \mathbf{A}) : \mathbf{T} \in \{0, 1\}^J, \mathbf{A} \in \mathcal{A}\}$ is the exhaustive list of potential outcomes. Under the assumptions of local spillovers and exchangeability, the set of effective treatments $(r_j(\mathbf{T}; \mathbf{A}) = (T_j, \sum_k \mathbf{A}_{jk} T_k))$ are reduced to:

$$\mathcal{R} \equiv \{(t, s) : t \in \{0, 1\}, s \in \mathbb{N}\}$$

As a result, the set of potential outcomes now reduces to:

$$\{Y(r) : r \in \mathcal{R}\}$$

11.2.1 Step 1: construct \mathcal{P}^*

Since \mathcal{G} is assumed to have finite VC dimension, there exists $\{u_1, \dots, u_v\} \subset \mathcal{S}$ that is shattered by \mathcal{G} .

1. $\forall \mathbf{P} \in \mathcal{P}^*$, (\mathbf{X}, \mathbf{A}) has uniform support over $\{u_1, \dots, u_v\}$.
2. $\forall \mathbf{P} \in \mathcal{P}^*$, $D_j \perp \{Y(r) : r \in \mathcal{R}\}, \mathbf{X}, \mathbf{A}$ and D_j are IID across j with $\mathbf{P}(D_j = 1) = \mathbf{P}(D_j = 0) = 1/2$.

3. In this version, we characterize the outcome distributions to be without any spillovers.

- $Y(0, s) = 0$ w.p.1 for all $s \in \mathbb{N}$.
- Elements of \mathcal{P}^* are characterized by bit vector $\mathbf{b} \in \{0, 1\}^v$. For any $s \in \mathbb{N}$,

$$(Y(1, s) | (\mathbf{X}, \mathbf{A}) = u_l) = \begin{cases} +1/2, & \text{w.p. } 1/2 + \gamma b_l - \gamma(1 - b_l) \\ -1/2, & \text{w.p. } 1/2 - \gamma b_l + \gamma(1 - b_l) \end{cases}$$

4. Y_j are IID across j .

In this iteration, I also consider the many village framework where $\mathbf{Z}_{1:N}$ should be interpreted as data from N IID villages with J individuals each.

11.2.2 Step 2: characterizing optimal rule given \mathbf{b}

Under the allowed distributions in \mathcal{P}^* , the representation of the welfare function can be simplified further:

$$\begin{aligned} W(G^{1:Q}) &= \sum_{q=1}^Q \sum_{j=1}^J \mathbb{E}_{\mathbf{b}} [Y_j(\mathbf{T}_q(j), 0) \cdot 1\{(\mathbf{X}, \mathbf{A}) \in G_q\}] \\ &= \sum_{q=1}^Q \sum_{j=1}^J \frac{1}{v} \sum_{l=1}^v [\mathbf{T}_q(j) \cdot (\gamma b_l - \gamma(1 - b_l)) \cdot 1\{u_l \in G_q\}] \\ &= \sum_{q=1}^Q \frac{1}{v} \sum_{l=1}^v [(\gamma b_l - \gamma(1 - b_l)) \cdot 1\{u_l \in G_q\}] \cdot \left[\sum_{j=1}^J \mathbf{T}_q(j) \right] \end{aligned}$$

Thus, if $b_l = 1$, it is optimal to treat everyone, i.e. $\mathbf{T} = \mathbf{1}$. While, when $b_l = 0$, treating no body is optimal, i.e. $\mathbf{T} = \mathbf{0}$. Suppose G^0 corresponds to the set which receives $\mathbf{T} = \mathbf{0}$ and G^Q is the set which receives $\mathbf{T} = \mathbf{1}$. Then, I can write:

$$\begin{aligned} G_{\mathbf{b}}^{*1} &= \{u_l : b_l = 1, 1 \leq l \leq v\} \\ G_{\mathbf{b}}^{*Q} &= \{u_l : b_l = 0, 1 \leq l \leq v\} \\ \forall 1 < q < Q, G_{\mathbf{b}}^{*q} &= \emptyset \end{aligned}$$

Now, I can compute the welfare under the optimal assignment noting that by construction, this partition is feasible within \mathcal{G} :

$$W_{\mathbf{b}}^* = \frac{1}{v} \sum_{l=1}^v J\gamma b_l = \frac{J\gamma}{v} \sum_{l=1}^v b_l$$

11.2.3 Step 3: Bayes Risk Minimization

Start with noting that $\hat{G}^{1:Q} = (\hat{G}^1, \dots, \hat{G}^Q)$. I can write:

$$W_{\mathbf{b}}^* - W_{\mathbf{b}}(\hat{G}^{1:Q}) = \sum_{q=1}^Q \frac{1}{v} \sum_{l=1}^v \sum_{j=1}^J \mathbf{T}_q(j) [(\gamma b_l - \gamma(1 - b_l)) (1\{u_l \in G_{\mathbf{b}}^{*q}\} - 1\{u_l \in \hat{G}^q\})]$$

The Bayes Risk Minimization problem is:

$$\begin{aligned} \mathbb{E}_{\mathbf{b}} \mathbb{E}_{Z_{1:n}} [W_{\mathbf{b}}^* - W_{\mathbf{b}}(\hat{G}^{1:Q})] &= \sum_{q=2}^Q \gamma \left(\sum_{j=1}^J \mathbf{T}_q(j) \right) \mathbb{E}_{\mathbf{b}} \mathbb{E}_{Z_{1:N}} [\mathbf{P}(G^{*1} \cap \hat{G}^q)] \\ &\quad + \sum_{q=1}^{Q-1} \gamma \left(J - \sum_{j=1}^J \mathbf{T}_q(j) \right) \mathbb{E}_{\mathbf{b}} \mathbb{E}_{Z_{1:N}} [\mathbf{P}(G^{*Q} \cap \hat{G}^q)] \end{aligned}$$

For any u_l observe the loss associated with:

$$\begin{aligned} u_l \in \hat{G}^1 &\mapsto J\gamma\pi(b_l = 1|Z_{1:N}) \\ u_l \in \hat{G}^Q &\mapsto J\gamma(1 - \pi(b_l = 1|Z_{1:N})) \\ u_l \in \hat{G}^q &\mapsto \gamma \left(J - \sum_{j=1}^J \mathbf{T}_q(j) \right) \pi(b_l = 1|Z_{1:N}) + \gamma \sum_{j=1}^J \mathbf{T}_q(j) (1 - \pi(b_l = 1|Z_{1:N})) \end{aligned}$$

There are two cases to consider here:

1. When $\pi(b_l = 1|Z_{1:N}) \leq 1/2$:

First of all, it is easy to see:

$$J\gamma\pi(b_l = 1|Z_{1:N}) \leq J\gamma(1 - \pi(b_l = 1|Z_{1:N}))$$

Next,

$$\begin{aligned}
& \gamma \left(J - \sum_{j=1}^J \mathbf{T}_q(j) \right) \pi(b_l = 1 | \mathbf{Z}_{1:N}) + \gamma \sum_{j=1}^J \mathbf{T}_q(j) (1 - \pi(b_l = 1 | \mathbf{Z}_{1:N})) \\
& \geq \gamma \left(J - \sum_{j=1}^J \mathbf{T}_q(j) \right) \pi(b_l = 1 | \mathbf{Z}_{1:N}) + \gamma \sum_{j=1}^J \mathbf{T}_q(j) \pi(b_l = 1 | \mathbf{Z}_{1:N}) \\
& = J\gamma \pi(b_l = 1 | \mathbf{Z}_{1:N})
\end{aligned}$$

Thus, $u_l \in \hat{G}^1$ is the optimal assignment.

2. When $\pi(b_l = 1 | \mathbf{Z}_{1:N}) > 1/2$:

First of all, it is easy to see:

$$J\gamma(1 - \pi(b_l = 1 | \mathbf{Z}_{1:N})) < J\gamma\pi(b_l = 1 | \mathbf{Z}_{1:N})$$

Next,

$$\begin{aligned}
& \gamma \left(J - \sum_{j=1}^J \mathbf{T}_q(j) \right) \pi(b_l = 1 | \mathbf{Z}_{1:N}) + \gamma \sum_{j=1}^J \mathbf{T}_q(j) (1 - \pi(b_l = 1 | \mathbf{Z}_{1:N})) \\
& < \gamma \left(J - \sum_{j=1}^J \mathbf{T}_q(j) \right) (1 - \pi(b_l = 1 | \mathbf{Z}_{1:N})) + \gamma \sum_{j=1}^J \mathbf{T}_q(j) (1 - \pi(b_l = 1 | \mathbf{Z}_{1:N})) \\
& = J\gamma(1 - \pi(b_l = 1 | \mathbf{Z}_{1:N}))
\end{aligned}$$

Thus, $u_l \in \hat{G}^Q$ is the optimal assignment.

Now, I can move on to computing the Minimized Bayes Risk (MBR):

$$MBR = J\gamma \mathbb{E}_{\mathbf{b}} \mathbb{E}_{\mathbf{Z}_{1:N}} \left[\min \{ \pi(b_l = 1 | \mathbf{Z}_{1:N}), 1 - \pi(b_l = 1 | \mathbf{Z}_{1:N}) \} \right]$$

I evaluate $\pi(b_l = 1 | \mathbf{Z}_{1:N})$ in a setting where the data comes from n villages of size J each.

$$\pi(b_l = 1 | \mathbf{Z}_{1:N}) = \frac{(1/2 + \gamma)^{\bar{k}_l^+} (1/2 - \gamma)^{\bar{k}_l^-}}{(1/2 + \gamma)^{\bar{k}_l^+} (1/2 - \gamma)^{\bar{k}_l^-} (1/2 - \gamma)^{\bar{k}_l^+} (1/2 + \gamma)^{\bar{k}_l^-}}$$

where

$$\begin{aligned}\bar{k}_l^+ &= \sum_{i=1}^N 1\{(\mathbf{X}^{(i)}, \mathbf{A}^{(i)}) = u_l\} \cdot |\{j : Y_j^{(i)} D_j^{(i)} = +1/2\}| \\ \bar{k}_l^- &= \sum_{i=1}^N 1\{(\mathbf{X}^{(i)}, \mathbf{A}^{(i)}) = u_l\} \cdot |\{j : Y_j^{(i)} D_j^{(i)} = -1/2\}| \end{aligned}$$

Now, I follow similar steps to that in KT to obtain a lower bound for the MBR.

$$MBR = \frac{J\gamma}{v} \sum_{l=1}^v \mathbb{E} \left[\frac{1}{1 + a^{|\bar{k}_l^+ - \bar{k}_l^-|}} \right]$$

where $a = \frac{1+2\gamma}{1-2\gamma} > 1$ and $|\bar{k}_l^+ - \bar{k}_l^-| = \left| \sum_{i: (\mathbf{X}^{(i)}, \mathbf{A}^{(i)}) = u_l} \sum_{j=1}^J 2Y_j^{(i)} D_j^{(i)} \right|$. Since $a > 1$ then $1 + a^{|x|} > 2$ for any x . So,

$$MBR \geq \frac{J\gamma}{2v} \sum_{l=1}^v \mathbb{E} \left[\frac{1}{a^{|\bar{k}_l^+ - \bar{k}_l^-|}} \right]$$

Using Jensen's inequality with $f(x) = 1/a^x$, for any $x \geq 0$. Here, f is convex, so

$$MBR \geq \frac{J\gamma}{2v} \sum_{l=1}^v a^{-\mathbb{E}|\bar{k}_l^+ - \bar{k}_l^-|}$$

I now evaluate $\mathbb{E}|\bar{k}_l^+ - \bar{k}_l^-|$.

$$\begin{aligned}\mathbb{E}|\bar{k}_l^+ - \bar{k}_l^-| &= \mathbb{E} \left| \sum_{i: (\mathbf{X}^{(i)}, \mathbf{A}^{(i)}) = u_l} \sum_{j=1}^J 2Y_j^{(i)} D_j^{(i)} \right| \\ &\leq \sum_{j=1}^J \mathbb{E} \left| \sum_{i: (\mathbf{X}^{(i)}, \mathbf{A}^{(i)}) = u_l} 2Y_j^{(i)} D_j^{(i)} \right| \end{aligned}$$

The inequality follows from triangular inequality. Now, using that for any i, j , $\mathbf{P}(D_j^{(i)} = 1) = \mathbf{P}(D_j^{(i)} = 0) = 1/2$ and is IID across i, j ,

$$\begin{aligned}
\mathbb{E} \left| \sum_{i: (\mathbf{X}^{(i)}, \mathbf{A}^{(i)}) = u_l} 2Y_j^{(i)} D_j^{(i)} \right| &= \sum_{k=0}^n \binom{N}{k} \left(\frac{1}{2v} \right)^k \left(1 - \frac{1}{2v} \right)^{N-k} \mathbb{E} |Bin(k, 1/2) - k/2| \\
&\leq \sum_{k=0}^n \binom{N}{k} \left(\frac{1}{2v} \right)^k \left(1 - \frac{1}{2v} \right)^{N-k} \mathbb{E} \sqrt{k/4} \\
&\leq \mathbb{E} \sqrt{\frac{Bin(N, 1/2)}{4}} \\
&\leq \sqrt{\frac{\mathbb{E}[Bin(N, 1/2)]}{4}} \\
&= \sqrt{\frac{N}{8v}}
\end{aligned}$$

The last inequality follows by Jensen's inequality. Thus,

$$\mathbb{E} |\bar{k}_l^+ - \bar{k}_l^-| \leq J \sqrt{\frac{N}{8v}}$$

Then:

$$MBR \geq \frac{J\gamma}{2} a^{-J\sqrt{\frac{N}{8v}}}$$

Using that $1 + x \leq e^x$, $\forall x$:

$$MBR \geq \frac{J\gamma}{2} e^{-J\sqrt{\frac{N}{8v}} \frac{4\gamma}{1-2\gamma}}$$

Set $\gamma = \frac{1}{J} \sqrt{\frac{v}{N}}$:

$$\begin{aligned}
MBR &\geq \frac{1}{2} \sqrt{\frac{v}{N}} e^{-\frac{4}{1-2\gamma} \frac{1}{2\sqrt{2}}} \\
&= \frac{1}{2} \sqrt{\frac{v}{N}} e^{-\frac{\sqrt{2}}{1-2\gamma}}
\end{aligned}$$

If $1 - 2\gamma \geq 1/2 \equiv N \geq \frac{16v}{j^2}$, then

$$MBR \geq \frac{1}{2} \sqrt{\frac{v}{N}} e^{-2\sqrt{2}}$$

This bound is identical to Kitagawa, Tetenov. ■

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