Spillover Effects in Experimental Data

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Abstract

We present current methods for estimating treatment effects and spillover effects under “interference”, a term which covers a broad class of situations in which a unit’s outcome depends not only on treatments received by that unit, but also on treatments received by other units. To the extent that units react to each other, interact, or otherwise transmit effects of treatments, valid inference requires that we account for such interference, which is a departure from the traditional assumption that units’ outcomes are affected only by their own treatment assignment. Interference and associated spillovers may be a nuisance or they may be of substantive interest to the researcher. In this chapter, we focus on interference in the context of randomized experiments. We review methods for when interference happens in a general network setting. We then consider the special case where interference is contained within a hierarchical structure. Finally, we discuss the relationship between interference and contagion. We use the interference R package and simulated data to illustrate key points. We consider efficient designs that allow for estimation of the treatment and spillover effects and discuss recent empirical studies that try to capture such effects.
1 Introduction

We present current methods for identifying causal effects under “interference”, a term which covers a very broad class of situations in which a unit’s outcomes depend not only on treatments received by that unit, but also on treatments received by other units (Cox [1958]). This includes effects that spill over from one unit to others. For example, in an agricultural experiment, fertilizer applied to one plot of land may literally spill over into other plots assigned to different treatments, therefore affecting their yields. Interference may arise from interactions between units, such as through social influence processes. For example, in the context of an election, exposing a voter to a persuasive appeal may affect what that voter says to their friends, which in turn may affect the friends’ outcomes.

Interference represents a departure from the traditional assumption wherein the potential outcomes that would be observed for a unit in either the treatment or control condition, depend only on that unit’s, and not the overall, treatment assignment. This traditional assumption is implied by what Rubin (1990) refers to as the “stable unit treatment value assumption” (SUTVA).

Figure 1 displays channels through which interference might occur. The black elements in Figure 1 show a directed acyclic graph that captures potential spillover effects onto unit 2 from a treatment assigned to unit 1. We assume an experiment where unit 1’s treatment, $Z_1$, is randomly assigned. Then, the effect of this treatment, captured by the black arrows flowing from $Z_1$, could be to alter unit 1’s own outcome, $Y_1$, as well as unit 2’s outcome, $Y_2$. This could happen via a pathway in which $Y_1$ mediates the effect of $Z_1$ on $Y_2$. Such outcome-mediated effects are known as contagion effects, which we briefly discuss toward the end of this chapter. Or it could be that $Z_1$ affects $Y_2$ through channels that do not go through $Y_1$. Spillover includes the sum total of the effects from $Z_1$ to $Y_2$. The gray elements in Figure 1 show that spillovers could be running from $Z_2$ to $Y_1$ as well. Finally, the variable $U$ captures other variables that might induce dependency between $Y_1$ and $Y_2$. It is important to recognize that these sources of outcome dependence or clustering are wholly distinct from spillover. However, such confounders undermine the ability to isolate contagion effects from other spillover mechanisms, a point to which we return below.

In this chapter, we assume that the researcher is interested in estimating spillover effects. We focus on randomized experiments for which we have some understanding of the structure through which spillover effects occur. In the first section, we review cases where the interference network is known completely, but then can take almost arbitrary form. In the second section, we review cases where we know only that interference is fully contained within the boundaries of strata that partition the population, but then the interference network within these strata is unknown. The final section makes some points regarding the attempt to distinguish contagion effects from other forms of spillover. We do not discuss work that examine causal
effects in situations where the interference network is fully hidden as in Sävje, Aronow and Hudgens (2017). Moreover, our emphasis is on large-N estimation of spillover effects, and so we omit discussion of methods that work off the experimental randomization to develop exact tests for interference effects (Rosenbaum, 2007 [Aronow] 2012 [Bowers, Fredrickson and Panagopoulos 2013] Athey, Eckles and Imbens 2018). These methods either restrict themselves to testing for interference, or require strong assumptions (for example, a finite-dimensional model of causal effects) to attain interval estimates.

2 Three Motivating Examples

We begin with three examples that allow us to illustrate key points. The first example is a study by Paluck, Shepherd and Aronow (2016), who study the effects of an antibullying program in New Jersey schools. The authors began by measuring the schools' social networks. They did this by asking students to report which other students they chose to spend time with in previous weeks. The experiment then randomly assigned schools to the antibullying program, and within schools, randomly selected students from an eligible subpopulation to actively participate in the program. Assuming the network measures are accurate, the experiment identifies spillover effects onto students who themselves do not participate in the program but have peers who do. The ability to get at such spillover effects depends on the accuracy of the network measure and the ways that one specifies potential exposure to spillovers on the basis of this network. If the researcher assumes that only peers of program recipients can be affected by the anticonflict intervention, but in fact peers of peers can be affected as well, then inferences about the program’s direct and spillover effects may be biased. The section below on arbitrary interference networks discuss this issue along with associated sensitivity analyses.
As a second example, consider Figure 2, which shows the results of an experimental study in Kenya by Haushofer and Shapiro (2018) on the long-term (after 3 years) effects of unconditional cash transfers. The outcome here is monthly household consumption. In this study, villages were randomly selected to be treated, and then within these villages, a half of the households were randomly selected to receive about 400 USD in cash. As Figure 2 shows, average monthly consumption in treatment villages, pooling recipient and non-recipient households, is $211 (green horizontal line), which is similar to the average in control villages ($217). But the inter-village comparison masks variation within treated villages. Recipient households consume $235 per month on average, while non-recipient neighbors in treatment villages consume $188 per month on average. Given that the treatment was assigned in a manner that randomized both across villages and within villages, these three types of households (recipients, neighbors, and control villages) are ex ante exchangeable, in which case the experiment yields an unbiased estimate of a negative within-village spillover effect.

This example illustrates a few additional nuances. First, when spillovers are present, one needs to think about effects in terms of overall assignment patterns. Neither the comparison between directly treated households and control village households nor directly treated households and untreated households in treated villages gets at “the” effect of cash transfers. Effects depend on exactly whom and at what rate potentially interacting households are treated. This particular experiment gives evidence on outcomes for treated households and untreated neighboring households when the within-village treatment rate is 50%. Were the quantity of policy interest to be how outcomes change going from 0% to 100% treatment rates, for example, it is not clear that the experiment could directly speak to that. Second, households presumably
differ not only in how they would respond after receiving the transfer per se, but also in how they would respond given the precise set of other households that receive the transfer. Suppose the village included two business partners, and business production exhibited increasing returns to input capital. Then, outcomes would presumably differ if the households of both partners were treated as compared to only one partner being treated. Outcomes among the untreated neighbors in treatment villages depend not only on the treatment saturation rate (50%), but also the precise configuration of treated and untreated households. This raises the question of how to interpret effects such as those presented in Figure 2—what counterfactual comparisons are being characterized, exactly? The section below on partial interference addresses these issues.

A third example is the experiment by Nickerson (2008) on potential contagion in voter turnout. Households with two registered voters were first randomly assigned to one of three conditions: a get-out-the-vote (GOTV) doorstep appeal, a doorstep appeal to promote recycling, or a control condition in which nothing was done to the household. The recycling appeal was meant to serve as a “placebo” treatment to account for the fact that only some households, and particularly individuals with specific characteristics would open the door to receive an appeal. The experiment thus yielded data on subjects who opened the door and were thus direct recipients of either the GOTV or recycling appeals, their housemates who were not there at the door, and then the full set of control households. Comparing voter turnout among housemates of those who directly received the GOTV appeal to housemates of those who received the recycling appeal, one can estimate whether the GOTV treatment spilled over from the direct recipient to the housemate. Insofar as there is an effect, one may wonder if the mechanism at work is contagion—that is, it is the voting intention of the direct recipient that went on to affect the voting intention of the housemate—or some other mechanism. This distinction between mechanisms would have implications for theories about norms that support voting behavior. In the section below on contagion, we return to this experiment and review assumptions needed to isolate contagion effects.

3 Formal Setting

We now present a formal framework for defining causal effects under interference. Suppose an experimenter intervenes on a finite population $U$ of units indexed by $i = 1, \ldots, N$. Let us suppose further that the intervention is defined by a treatment assignment vector $z = (z_1, \ldots, z_N)'$, where $z_i \in \{1, 0\}$ specifies the possible treatment values that unit $i$ receives. Let $\Omega$ be the set of treatment assignment vectors $z$ with $|\Omega| = 2^N$. An experimental design is a plan for randomly selecting a particular value of $z$ from the $2^N$ different possibilities with predetermined probability $p_z$—for example, Bernoulli assignment (i.e., coin flips) or completely randomized assignment strategy. Therefore, $\Omega = \{z : p_z > 0\}$ and the realized treatment
assignment $\mathbf{Z} = (Z_1, \ldots, Z_N)'$ is a random vector with support $\Omega$ and $\Pr(\mathbf{Z} = \mathbf{z}) = p_\mathbf{z}$. For example, with a population of size $N = 10$, and an experimental design that randomly assigns without replacement a proportion $p = 0.2$ to treatment condition $z_i = 1$ with uniform probability, there are $\binom{N}{pN} = 45$ possible treatment assignments ($|\Omega| = 45$) and the realized treatment assignment $\mathbf{Z}$ has $p_\mathbf{z} = \frac{1}{45}$. The experimental design characterizes precisely the probability distribution of the assigned treatments. In experiments, this is determined by the researcher and is therefore known.

To analyze the effect of different treatment assignments, we compare the different outcomes they produce. These potential outcomes are defined for each unit $i$ as the elements in the image of a function that maps assignment vectors to a real valued outcomes, $y_i : \Omega \rightarrow \mathbb{R}$. Particularly, $y_i(\mathbf{z})$ is the response of unit $i$ to assignment $\mathbf{z}$. For convenience, let $\mathbf{z}_{-i} = (z_i, \ldots, z_{i-1}, z_{i+1}, \ldots, z_N)'$ denote the $(N - 1)$-element vector that removes the $i$th element from $\mathbf{z}$. Then, the potential outcome $y_i(\mathbf{z})$ can equivalently be expressed as $y_i(z_i; \mathbf{z}_{-i})$. Continuing with the example of the cash transfer program above, this quantity would be the potential consumption of household $i$ given its assignment as a transfer recipient or non-recipient ($z_i$) and the treatment assignment of all other households ($\mathbf{z}_{-i}$), including those inside and outside household $i$’s village.

Traditional analyses of experiments, and other chapters in this volume, assume no interference, in which case the potential outcome $y_i(\mathbf{z})$ is restricted to be affected only by $i$’s own treatment. That is, with no interference, for any two treatment assignments $\mathbf{z}$ and $\mathbf{z}'$, for which $z_i$ remains unchanged, we have $y_i(z_i; \mathbf{z}_{-i}) = y_i(z_i; \mathbf{z}_{-i}')$ for all $i \in \{1, \ldots, N\}$. When interference is present, there exist some units $i \in U$ for which $y_i(z_i; \mathbf{z}_{-i}) \neq y_i(z_i; \mathbf{z}_{-i}')$, that is fixing the treatment of $i$ while changing other units’ treatment results in changes of $i$’s outcome.

Let $Y_i$ denote the observed outcome of unit $i$, where the observed outcome is related to the potential outcomes as $Y_i = y_i(\mathbf{Z}) = y_i(Z_i; \mathbf{Z}_{-i})$, where $\mathbf{Z}_{-i}$ denotes the vector $\mathbf{Z}$ net of its $i$th element. In the case of no interference, $Y_i = y_i(Z_i)$. Therefore, when interference is present, we need to account for others’ treatment assignments as well.

## 4 Arbitrary But Known Interference Networks

This section reviews estimation methods in a setting where interference occurs over a network of arbitrary structure, but this structure is known. The analysis follows Aronow and Samii (2017). We represent a unit’s set of interfering units in terms of network ties. Then, depending on the network structure and the treated units’ network characteristics, different treatment assignments may result in different and arbitrary, but known, patterns of interference. For example, assuming that interference happens through direct ties between units, treating any one unit in a fully-connected network generates a pattern in which the treatment of that
one unit interferes with the treatment of every other unit in the network. In a regular lattice, the treatment of any one treated unit interferes only with the treatment of that unit’s four nearest neighbors, and in an irregular network, treatment assignments that treat units with many direct ties generate more interference, than assignments that treat units with just a few ties.

As in the anticonflict social network experiment of Paluck, Shepherd and Aronow (2016), these methods require the researcher to measure the network or to have comprehensive information about connections between experimental units, and to define precise causal effects which reflect the possible types of treatment exposures that might be induced in the experiment, which in turn requires to make specific assumptions about the extent of interference. The goal is to estimate exposure-specific causal effects—for the anticonflict program, for example, we might estimate effects on students for whom at least one peer is a direct program participant, or for whom exactly two peers are participants, etc. Knowing the treatment assignment distribution allows one to account for potential sources of confounding that arise from heterogeneity across units in their likelihood of falling into different exposure conditions (for example, heterogeneity in terms of students’ number of connections with other students). The sections below explain.

4.1 Exposure Mapping

To determine each unit’s treatment exposure under a given treatment assignment, Aronow and Samii (2017) define an exposure mapping that maps the set of assignment vectors and unit-specific traits to an exposure value: \( f : \Omega \times \Theta \rightarrow \Delta \), where \( \theta_i \in \Theta \) quantifies relevant traits of unit \( i \) such as the number of direct ties to other units in the network and, possibly, weights assigned to each of these ties. The set \( \Delta \) contains all of the possible treatment-induced exposures that may be generated in the experiment, and its cardinality depends on the nature of interference. For example, with no interference and a binary treatment the exposure mapping ignores unit specific traits \( f(z, \theta_i) = z_i \), producing two possible exposure values for each unit: no exposure (or control condition, \( z_i = 0 \)) and direct exposure (or treatment condition, \( z_i = 1 \)), in which case \( \Delta = \{0, 1\} \).

Now, consider interference that occurs through direct peer connections. Then, \( \theta_i \) is a column vector equal to the transpose of unit \( i \)’s row in a network adjacency matrix (which captures \( i \)’s direct connections to other units), and the exposure mapping \( f(z, \theta_i) \) can be simply defined to capture direct exposure to treatment—or the effect of being assigned to treatment—and indirect exposure—or the effect of being exposed to treatment of peers. An example of such an exposure mapping (and by no means the only possibility) is the following, whereby indirect exposure occurs when at least one peer is treated:

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1Note that the meaning of “direct” and “indirect” in the interference setting is different than in the mediation setting reviewed in Glynn’s chapter in this volume.
For this particular case $\Delta = \{d_{11}, d_{10}, d_{01}, d_{00}\}$. This characterization of exposures is “reduced form” in that it does not distinguish between the mechanisms through which spillover effects occur.

Specification of the exposure mapping requires substantive consideration of the data generating process. Manski (2013) discusses subtleties that arise in specifying exposure mappings. For example, the author shows how models of simultaneous endogenous choice (due to homophily or common external shocks) can produce restrictions on the potential outcomes $y_i(d)$, and therefore imply that potential outcomes may vary in ways that an otherwise intuitive exposure mapping may fail to capture.

Because units occupy different positions in the interference network, their probabilities of being in one or another exposure condition vary, even if treatment is randomly assigned. Insofar as network position also affects outcomes, then such differences in exposure probabilities need to be taken into account when estimating exposure-specific causal effects. Otherwise, the analysis would be confounded. We show here that when the random assignment mechanism is known, then these exposure probabilities are also known. This allows one to condition on the exposure probabilities directly. To see this, define the exposure that unit $i$ receives as $D_i = f(Z_i, \theta_i)$, a random variable with support $\Delta_i \subseteq \Delta$ and for which $\Pr(D_i = d) = \pi_i(d)$. For each unit $i$ there is a vector, $\pi_i = (\pi_i(d_{11}), \pi_i(d_{10}), \pi_i(d_{01}), \pi_i(d_{00}))'$. We observe the unit-specific traits ($\theta_i$) necessary to define exposures for any treatment assignment vector, and the probability of each possible treatment assignment vector ($p_z$) is known. This allows us to compute $\pi_i(d_k)$ as the expected proportion of treatment assignments which induce exposure $d_k$ for unit $i$. When the set of possible treatment assignment vectors $\Omega$ is small, this can be computed exactly. When $\Omega$ is large, one can approximate the $\pi_i(d_k)$ values with arbitrary precision by taking a large number of random draws from $\Omega$. Aronow and Samii (2017) discuss considerations for how many draws are needed so as to keep biases small. This Monte Carlo method may in some cases require a prohibitive number of draws (for example, if $|\Delta|$ is large), but for some specific designs and exposure mappings it may be possible to compute the $\pi_i(d_k)$ values via a dynamic program, as in Ugander et al. (2013).

The following toy example illustrates how to compute the exposure received by each unit and the
generalized probability of exposure using the `interference` package for R [Zonszein, Aronow and Samii, 2019]. Suppose we have a set of \( N = 10 \) units, we randomly assign (without replacement) a proportion \( p = 0.2 \) to treatment condition \( z_i = 1 \) with uniform probability. In this case, the realized treatment assignment \( Z' \) shows that units 6 and 9 are directly treated.

```r
N <- 10
p <- 0.2
Z <- make_tr_vec_permutation(N, p, R = 1, seed = 56)
Z
```

```
[1,] 0 0 0 0 0 1 0 0 1 0
[2,] 1 0 1 1 0 0 0 0 0 1
[3,] 1 1 0 1 1 1 0 0 1 0
[4,] 0 1 1 0 1 1 1 0 0 0
[5,] 1 0 1 1 0 1 0 1 0 0
[6,] 0 0 1 1 1 0 0 0 0 0
[7,] 0 0 0 1 0 0 0 1 1 0
[8,] 0 0 0 0 1 0 1 0 1 1
[9,] 0 0 1 0 0 0 1 1 0 1
[10,] 0 1 0 0 0 0 1 1 0
```

Now let’s suppose that units are connected according to a draw from a Watts-Strogatz model—a random graph generation model that produces networks with “small world” properties: high clustering in network interconnections and short average path lengths that connect any two arbitrary nodes (units). We assume an undirected network, that is, if \( i \) has a direct connection with \( j \), then \( j \) has one with \( i \), and that on average each unit is directly connected to four other units. A visualization of such a network is given in Figure 3 and its adjacency matrix is:

```r
adj_matrix <- make_adj_matrix(N, model = 'small_world', seed = 492)
adj_matrix
```

```
[1,] 0 1 1 0 1 0 0 0 0 0
[2,] 1 0 1 1 0 0 0 0 0 1
[3,] 1 1 0 1 1 1 0 0 1 0
[4,] 0 1 1 0 1 1 1 0 0 0
[5,] 1 0 1 1 0 1 0 1 0 0
[6,] 0 0 1 1 1 0 0 0 0 0
[7,] 0 0 0 1 0 0 0 1 1 0
[8,] 0 0 0 0 1 0 1 0 1 1
[9,] 0 0 1 0 0 0 1 1 0 1
[10,] 0 1 0 0 0 0 1 1 0
```
Figure 3: Example of interference network with ten units. Each edge (link) represents a possible channel through which spillover effects might transmit.

For the purposes of our example, the adjacency matrix captures $\Theta$, while each row defines $\theta'_i$. The reason is that in our example, exposures are defined strictly through the combination of the treatment assignment and the individual rows of the adjacency matrix. In principle, exposure mappings could take other factors into account, such as covariates that are not related to the adjacency matrix or other properties of the adjacency matrix besides a unit’s row. Returning to the example, from $\theta'_6$ we know that unit 6 has edges to each of units 3, 4 and 5. Using the adjacency matrix (adj_matrix) and Z (Z) as arguments in the exposure mapping function defined above, we obtain the received exposure for every unit. The argument hop = 1 describes a data generating process in which indirect exposure happens through the existence of any direct peer receiving treatment:

```R
D <- make_exposure_map_AS(adj_matrix, Z, hop = 1)
```

\[
\begin{array}{cccc}
\text{dir_ind1} & \text{isol_dir} & \text{ind1} & \text{no} \\
[1,] & 0 & 0 & 0 & 1 \\
[2,] & 0 & 0 & 0 & 1 \\
[3,] & 0 & 0 & 1 & 0 \\
[4,] & 0 & 0 & 1 & 0 \\
[5,] & 0 & 0 & 1 & 0 \\
[6,] & 0 & 1 & 0 & 0 \\
[7,] & 0 & 0 & 1 & 0 \\
[8,] & 0 & 0 & 1 & 0 \\
[9,] & 0 & 1 & 0 & 0 \\
[10,] & 0 & 0 & 1 & 0 \\
\end{array}
\]
We can see that the received exposure for units 3, 4 and 5 is $d_{01} \equiv \text{Indirect Exposure}$, given their direct connection to unit 6, who is directly treated. Likewise for units 7, 8 and 10 who are degree-one neighbors of unit 9. We can also see that there are no units in exposure condition $d_{11}$, because the two directly treated units (6 and 9) are not connected to each other.

Now, to obtain the generalized probability of exposure of each unit we need the exposure mapping function and its arguments: the adjacency matrix and the set of all possible treatment assignments. When $|\Omega|$ is large we can approximate $\Omega$ producing random replicate $z$'s. In this case we could easily compute $\Omega$ because there are only 45 possible treatment assignment profiles, but for expository purposes, we work with 30 random draws from $\Omega$ without replacement (setting the arguments $R = 30$ and  allow_repetitions = FALSE).

```r
omega <- make_tr_vec_permutation(
    N, p,
    R = 30, seed = 420, allow_repetitions = FALSE
)
prob_exposure <- make_exposure_prob(
    omega,
    adj_matrix,
    make_exposure_map_AS,
    list(hop = 1)
)
make_prob_exposure_cond(prob_exposure)

[,1]      [,2]      [,3]      [,4]      [,5]      [,6]
dir_ind1  0.09677419 0.12903226 0.1612903 0.09677419 0.12903226 0.09677419
isol_dir  0.19354839 0.09677419 0.1290323 0.12903226 0.06451613 0.12903226
ind1      0.4193548 0.61290323 0.6774194 0.6129032 0.4516129 0.4193548
no        0.38709677 0.25806452 0.1290323 0.16129032 0.22580645 0.41935484

[,7]      [,8]      [,9]      [,10]
dir_ind1  0.1290323 0.12903226 0.1290323 0.1290323
isol_dir  0.2258065 0.09677419 0.1612903 0.1290323
ind1      0.4193548 0.64516129 0.6129032 0.4516129
no        0.3225806 0.22580645 0.1935484 0.3870968

The columns capture $\pi_i$, the expected proportion of treatment assignments which result in each exposure.
We now formally define spillover effects as contrasts between averages of individual potential outcomes across different exposures. To estimate exposure-specific average potential outcomes, the exposure mapping has to fully characterize interference. This condition implies that $K$ treatment exposures give rise to at most $K$ distinct potential outcomes for each unit $i$ in the population. We write the potential outcomes as $(y_i(d_1), \ldots, y_i(d_K))$, where $y_i(d_k) = y_i(z)$ for all units, $k \in \{1, \ldots, K\}$, and $z \in \Omega$ such that $f(z, \theta_i) = d_k$.

Then, observed outcomes must relate back to the potential outcomes: $Y_i = \sum_{k=1}^K I(D_i = d_k)y_i(d_k)$. The average potential outcome at any exposure level $k$ is then $\mu(d_k) = \frac{1}{N} \sum_{i=1}^N y_i(d_k)$, and the average causal effect of being in exposure condition $d_k$ as opposed to exposure condition $d_l$ is

$$\tau(d_k, d_l) = \frac{1}{N} \sum_{i=1}^N y_i(d_k) - \frac{1}{N} \sum_{i=1}^N y_i(d_l) = \mu(d_k) - \mu(d_l).$$

To estimate $\sum_{i=1}^N y_i(d_k) = y^T(d_k)$ we have to take into account that we observe $y_i(d_k)$ only for those with $D_i = d_k$, and that the probability of observing $y_i(d_k)$ is not equal across units. Using the exposure mapping from the example above, the probability of observing $y_i(d_{10}) = y_i$(Isolated Direct Exposure) is smaller for those with more direct connections to other units in the network. But as we saw above, by design, we can calculate the probability of the exposure conditions for each individual. Then, assuming that all units have nonzero probabilities of being subject to each of the K exposures, the $y^T(d_k)$ can be estimated without bias with the Horvitz-Thompson inverse probability estimator:

$$\bar{y}^T_{HT}(d_k) = \sum_{i=1}^N I(D_i = d_k) \frac{Y_i}{\pi_i(d_k)}.$$

In cases where $|\Omega|$ is high, and therefore we use sampling from $\Omega$ to obtain estimates $\hat{\pi}_i(\cdot)$, those estimates are used in place of the true $\pi_i(\cdot)$ values. A Horvitz-Thompson estimator of the average unit-level causal

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2If $\pi_i(d_k) = 0$ for some units, then estimation of average potential outcomes $\mu(d_k)$ must be restricted to the subset of units for which $\pi_i(d_k) > 0$. Interpretation of contrasts of average potential outcomes as causal effects would require doing so for units such that $\pi_i(d) > 0$ for all $d \in \Delta' \subset \Delta$; for example, when estimating $\tau(d_k, d_l)$ one would need to restrict analysis to units with both $\pi_i(d_k) > 0$ and $\pi_i(d_l) > 0$. 

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effect of exposure $k$ versus $l$, $\tau(d_k, d_l)$, is therefore

$$\hat{\tau}_{HT}(d_k, d_l) = \hat{\mu}_{HT}(d_k) - \hat{\mu}_{HT}(d_l) = \frac{1}{N} \left[ \hat{y}_{HT}(d_k) - \hat{y}_{HT}(d_l) \right].$$

Continuing with the previous example, we show how to compute this estimator. We do so using simulated potential outcomes that exhibit effect heterogeneity and that vary in units’ network degree, in which case naive estimates that do not account for probabilities of exposure would be biased. Specifically, we generate a variable with random values from an absolute standard normal distribution which is correlated with the unit’s first and second order degree—the number of peers and peers of peers, respectively (for which we use the arguments `adj_matrix` and `make_corr_out` in the function below). This variable determines the potential outcome under the $d_{00}$ condition. To build heterogeneous effects into the analysis, we assume what Rosenbaum (1999) refers to as “dilated effects” such that $y_i(d_{11}) = 2 \times y_i(d_{00})$, $y_i(d_{10}) = 1.5 \times y_i(d_{00})$, $y_i(d_{01}) = 1.25 \times y_i(d_{00})$. (The multipliers of $y_i(d_{00})$ can be changed by passing a vector with 3 numbers to `multipliers` in the function below).

```r
potential_outcomes <- make_dilated_out(
  adj_matrix, make_corr_out, seed = 1101,
  multipliers = NULL, hop = 1
)
```

From the potential outcomes and received exposures ($D$), we get the observed outcomes.

```r
observed_outcomes <- rowSums(D*t(potential_outcomes))
```

Next, we compute $\hat{\tau}_{HT}(d_{10}, d_{00})$, which isolates the effect of direct exposure in the absence of any interaction with indirect exposure, as well as $\hat{\tau}_{HT}(d_{01}, d_{00})$, which isolates the effect of indirect exposure in the absence of any interaction with direct exposure. In this case, we cannot compute $\hat{\tau}_{HT}(d_{11}, d_{00})$—the interactive effect of direct and indirect exposure—because in this small scale example no unit received exposure $d_{11}$.

```r
yT_HT <- estimates(D, observed_outcomes, prob_exposure, hop = 1)$yT_ht
```

The object `yT_HT` is a named numeric vector which contains the values of $\hat{y}_{HT}(d_k)$ for $d_k = d_{11}, d_{10}, d_{01}, d_{00}$ in that order. Therefore, in order to compute $\hat{\tau}_{HT}$, we can take the difference of each of these values with the value of exposure condition $d_{00}$ (No Exposure) and then divide by the number of units $N$. 

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\[ \text{tau} \_HT \leftarrow \left( \frac{1}{N} \right) \ast (y_T \_HT - y_T \_HT[^{\text{'no'}}]) \ast \left[ \text{names}(y_T \_HT) != 'no' \right] \]

In fact, the `estimates` function already computes \( \hat{\tau}_{HT} \) directly:

```r
estimates(D, observed_outcomes, prob_exposure, hop=1)$tau_ht
```

dir_ind1 isol_dir ind1

|       |        |        |
|-------|--------|--------|
| NA    | 41.61043 | 39.51643 |

The estimator \( \hat{\tau}_{HT}(d_k, d_l) \) is unbiased when it is estimated with \( \pi_i(d_k) \) rather than \( \hat{\pi}_i(d_k) \). As mentioned above, when estimated with the latter, the estimator is not unbiased, but the bias becomes negligible with a sufficiently large number of replicates \( R \). We implement variance estimators for \( \text{Var} \left[ \hat{y}_{HT}(d_k) \right] \) and \( \text{Var} \left[ \hat{\tau}_{HT}(d_k, d_l) \right] \) as derived in Equation 11 of Aronow and Samii (2017). These are conservative approximations to the exact variances that are guaranteed to have non-negative bias relative to the variance of the randomization distribution of the estimators. Because unbiased estimators for \( \text{Var} \left[ \hat{y}_{HT}(d_k) \right] \) are only identified when the joint exposure probabilities of every pair of units is positive, it is necessary to add a correction term to \( \text{Var} \left[ \hat{y}_{HT}(d_k) \right] \). Second, because the \( \text{Cov} \left[ \hat{y}_{HT}(d_k), \hat{y}_{HT}(d_l) \right] \) is always unidentified, we use an approximation. Both of these corrections, contribute to the non-negative bias of the variance estimator. We also implement the constant effects variance estimator derived in Aronow (2013). This estimator operates under the assumption that exposure effects do not vary across subjects, and therefore \( y_i(d_k) = y_i(d_l) + \tau(d_k, d_l) \) for every unit \( i \). Then, one can estimate the variance by either plugging the estimated \( \tau(d_k, d_l) \) values into the expression of the variance or using them to reconstruct the full schedule of potential outcomes and then simulating new treatment assignments to approximate the distribution of effect estimates. In the following applications, we take the maximum between the constant effects variance estimator and the conservative variance estimator developed in Aronow and Samii (2017). Confidence intervals are based on a large-N normal approximation: \[ \hat{\tau}_{HT}(d_k, d_l) \pm z_{(1-\alpha)/2} \sqrt{\text{Var} \left[ \hat{\tau}_{HT}(d_k, d_l) \right]} \]

Asymptotic convergence of these estimators, and therefore the reliability of normal approximations for inference, depend on whether outcome dependence across units is limited. In particular, consistency of \( \hat{\tau}_{HT}(d_k, d_l) \) follows from limits on the amount of pairwise dependency in exposure conditions induced by both the design and the exposure mapping as the sample size increases. Going back to the antibullying program
Table 1: Comparing Horvitz-Thompson to Hajek Estimator, Using Approximate Exposure Probabilities

| Estimator          | Estimand          | True-Value | Average-Value | Bias   | SD     | RMSE  | MeanSE |
|--------------------|-------------------|------------|---------------|--------|--------|-------|--------|
| Horvitz-Thompson   | $\tau(d_{11}, d_{00})$ | 58.12      | 57.33         | -0.78  | 45.32  | 45.32 | 45.94  |
| Horvitz-Thompson   | $\tau(d_{10}, d_{00})$ | 29.06      | 29.07         | 0.01   | 26.60  | 26.60 | 33.11  |
| Horvitz-Thompson   | $\tau(d_{01}, d_{00})$ | 14.53      | 14.66         | 0.13   | 10.45  | 10.45 | 10.33  |
| Hajek              | $\tau(d_{11}, d_{00})$ | 58.12      | 59.41         | 1.29   | 36.66  | 36.68 | 34.07  |
| Hajek              | $\tau(d_{10}, d_{00})$ | 29.06      | 28.92         | -0.14  | 21.55  | 21.55 | 25.09  |
| Hajek              | $\tau(d_{01}, d_{00})$ | 14.53      | 14.85         | 0.32   | 8.38   | 8.39  | 8.41   |

Note: Horvitz–Thompson estimator with maximum between conservative variance estimator and constant effects variance estimator. Hajek estimator with linearized variance estimator. True-Value = Value of estimand. Average-Value = Value of estimator. SD = Empirical standard deviation from simulation. RMSE = Root-mean-square error. MeanSE = mean standard error estimate. Estimators use approximate exposure probabilities calculated by drawing 10000 treatment assignments without replacement.

An alternative to the Horvitz-Thompson estimator is the Hajek estimator, which improves efficiency with a small cost in terms of finite sample bias. This estimator is a ratio approximation of the Horvitz-Thompson:

$$\hat{\mu}_H(d_k) = \frac{\sum_{i=1}^{N} I(D_i = d_k) \frac{Y_i}{\pi_i(d_k)}}{\sum_{i=1}^{N} I(D_i = d_k) \frac{1}{\pi_i(d_k)}}.$$

In the Horvitz-Thompson estimator $\hat{\mu}_{HT}(d_k)$ is high variance because some randomizations yield units with extremely high values of the weights $1/\pi_i(d_k)$. The Hajek refinement allows the denominator of the estimator to vary according to the sum of the weights $1/\pi_i(d_k)$, therefore shrinking the magnitude of the estimator when its value is large, and increasing the magnitude of the estimator when its value is small.

We extend the example developed above to consider a more realistic sample size. In what follows here and in the following sections, we use a set of $N = 400$ units and we randomly assign without replacement a proportion $p = 0.1$ to treatment condition $z_i = 1$ with uniform probability. As in the previous example, the network is modeled as small-world with each unit directly connected on average to four other units, and the potential outcomes follow the “dilated effects” scenario. We set the number of replications to $R = 10000$ to compute exposure probabilities, and run 3000 simulated replications of the whole experiment.

The result of the simulation shown in Table 1 illustrates that the Hajek estimator is more efficient than the Horvitz-Thompson estimator. The empirical standard deviation from simulation is smaller for the Hajek estimator, and the decrease in variance is with little cost in bias as indicated by a smaller root-mean-square error. Moreover, the variance estimator is consistent, given that the mean of the standard error estimate approaches the empirical standard deviation from simulation.
4.3 Misspecified exposure mappings

We now consider the implications of misspecified exposure mappings. Proposition 8.1 in Aronow and Samii (2017) shows what happens when an exposure condition $D_i = d_k$ specified by the experimenter is actually consistent with multiple potential outcomes for unit $i$, in which case the exposure mapping is too coarse. Here, we examine these issues in the context of our running example. We consider the case where one assumes that interference happens only through direct peer connections (first-degree interference), but in fact units are exposed to treatment of their direct peers and to treatment of peers of their direct peers (it is second-degree). We also consider what happens when the experimenter ignores interference, but in fact it is first- or second-degree.

Let us begin with an exposure mapping as above with four exposures for first-degree interference ($d_{11}$, $d_{10}$, $d_{01}$, $d_{00}$), and then another exposure mapping with eight exposures based on second-order interference ($d_{111}$, $d_{110}$, $d_{101}$, $d_{100}$, $d_{011}$, $d_{010}$, $d_{001}$, $d_{000}$), and finally a no-interference exposure mapping with only two exposures ($d_1$, $d_0$). Then, suppose two possible true data generating processes, with one exhibiting only first-degree interference and the other exhibiting second-degree interference. The exposure mapping is misspecified when the type of interference assumed by the experimenter does not match the true data generating process. This gives rise to six scenarios, two of which have correct exposure mapping specifications and the rest being cases of misspecification.

To define a coherent notion of bias under misspecification, one needs to define quantities of interest in terms of treatment regimes. In our case, we consider the contrast between average outcomes under 100% treatment saturation versus 0% saturation:

$$\tau(1, 0) = \frac{1}{N} \sum_{i=1}^{N} y_i(1) - y_i(0),$$

where the 1 and 0 are meant to denote that 100% or 0% of units are assigned to treatment, respectively.

When the true data generating process involves no interference, then $\tau(1, 0)$ is equivalent to the usual average treatment effect (ATE). Under interference, this is not the case. When the true data generating process involves only the first-degree spillover as per our running example, then $\tau(1, 0) = \tau(d_{11}, d_{00})$. With second-degree spillover, $\tau(1, 0) = \tau(d_{111}, d_{000})$. Misspecification will result in working with inappropriate contrasts to estimate $\tau(1, 0)$. For example, suppose the true data generating process is first-degree, but one assumes no interference. Then, one would mistakenly take the potential outcomes $y_i(d_{11})$ to be equivalent to $y_i(d_{10})$, and use a mixture of such outcomes in estimating the desired average of $y_i(1)$ outcomes. To see this, consider estimating the population mean when everyone is treated, $\mu(1)$, using a Horvitz-Thompson
estimator (which we use here for simplicity, results for the Hajek estimator would be along the same lines).

Then, if we assume no interference, we would compute

\[ \hat{\mu}_{HT, None}(1) = \frac{1}{N} \sum_{i=1}^{N} I(Z_i = 1) \frac{Y_i}{\pi_i} \]

\[ = \frac{1}{N} \sum_{i=1}^{N} \left( I(D_i = d_{11}) \frac{y_i(d_{11})}{\pi_i} + I(D_i = d_{10}) \frac{y_i(d_{10})}{\pi_i} \right), \]

where \( \pi_i = Pr[Z_i = 1] \), while the unbiased estimator would be,

\[ \hat{\mu}_{HT}(1) = \hat{\mu}_{HT}(d_{11}) = \frac{1}{N} \sum_{i=1}^{N} I(D_i = d_{11}) \frac{Y_i}{\pi_i(d_{11})} \]

\[ = \frac{1}{N} \sum_{i=1}^{N} I(D_i = d_{11}) \frac{y_i(d_{11})}{\pi_i(d_{11})}, \]

where \( \pi_i(d_{11}) = Pr[Z_i I(Z_i^\theta_i > 0) = 1] \).

Therefore, the misspecified \( \hat{\mu}_{HT, None}(1) \) is biased for \( \hat{\mu}_{HT}(1) \) insofar as \( y_i(d_{11}) \neq y_i(d_{10}) \) for some \( i \).

When the assumed exposure mapping considers higher-degree interference than the true data-generating process, the resulting estimator can be unbiased, but with a cost in variance. The reason is that this misspecified estimator incorporates only a fraction of the available units to construct the potential outcome average.

Table 2 and Figure 4 illustrate how estimates vary over these different forms of misspecification in our simulated data. For the sake of completeness, we also present results for another simulation where the spillover effects are negative (essentially, choosing dilated effects multipliers when simulating outcome data such that the multipliers for \( d_{11} \) and \( d_{01} \) are smaller than those of \( d_{10} \) and \( d_{00} \), respectively). Looking at Figure 4 from left to right we plot the distribution of point estimates for \( \hat{\tau}(d_1, d_0) \) (assuming no interference), \( \hat{\tau}_{H}(d_{11}, d_{00}) \) (assuming first-degree interference), and \( \hat{\tau}_{H}(d_{111}, d_{000}) \) (assuming second-degree interference). Then, we vary whether the true data generating process exhibits first- or second-degree interference. In all cases, the target of inference is \( \tau(1, 0) \). Under first-degree interference, \( \tau(1, 0) = \tau(d_{11}, d_{00}) \), and under second-degree interference \( \tau(1, 0) = \tau(d_{111}, d_{000}) \). These variations in data-generating processes are shown going up and down Figure 4 for the positive spillovers case in the top panel and the negative spillovers case in the bottom panel. True values of the target quantities are shown with the dashed lines, and the distributions of estimators over 3000 simulation runs are shown with the histograms. We see that estimators are centered around the true quantities when the exposure mapping incorporates equal- or higher-degree interference than the true data generating process. Because the estimator incorporates only a fraction of the available units to construct the potential outcome average, the variance is higher when the exposure mapping
### Table 2: Misspecifying Exposure Conditions

| Spillover | Estimand | Estimator | True-Value | Bias  | SD    | RMSE  |
|-----------|----------|-----------|------------|-------|-------|-------|
| positive  | $\tau(d_{11}, d_{00})$ | $\hat{\tau}_H(d_{11}, d_{00})$ | 58.12 | -23.33 | 19.06 | 30.12 |
| positive  | $\tau(d_{11}, d_{00})$ | $\hat{\tau}_H(d_{111}, d_{000})$ | 58.12 | 1.29 | 36.66 | 36.68 |
| positive  | $\tau(d_{11}, d_{00})$ | $\hat{\tau}_H(d_{11}, d_{00})$ | 58.12 | -32.21 | 21.42 | 38.68 |
| negative | $\tau(d_{11}, d_{00})$ | $\hat{\tau}_H(d_{111}, d_{000})$ | 14.53 | 14.87 | 14.73 | 20.93 |
| negative | $\tau(d_{11}, d_{00})$ | $\hat{\tau}_H(d_{111}, d_{000})$ | 14.53 | 0.85 | 23.36 | 23.37 |
| negative | $\tau(d_{11}, d_{00})$ | $\hat{\tau}_H(d_{111}, d_{000})$ | 7.26 | 23.92 | 13.65 | 27.54 |
| negative | $\tau(d_{11}, d_{00})$ | $\hat{\tau}_H(d_{111}, d_{000})$ | 7.26 | 6.42 | 21.27 | 22.21 |
| negative | $\tau(d_{111}, d_{000})$ | $\hat{\tau}_H(d_{111}, d_{000})$ | 7.26 | 2.58 | 28.05 | 28.16 |

**Note:**
Hajek estimator with linearized variance estimator. True-Value = Value of estimand. SD = Empirical standard deviation from simulation. RMSE = Root-mean-square error. Estimators use approximate exposure probabilities computed by drawing 10000 treatment assignments without replacement from the set of possible treatment profiles.

considers higher-degree interference than the true data generating process. To the contrary, the estimators are biased when the exposure mapping considers a lower-degree interference than the true data generating process. In this case, as we explained above, potential outcomes under different exposure conditions are taken to be equivalent, and therefore, averaged together by the estimator when constructing the potential outcome average. With positive spillover the estimators’ bias is negative (top panel), whereas the bias is positive with a case of negative spillover (bottom panel). For monotonic interference of the kinds considered here, bias is reduced more by considering exposure mappings with more refined patterns of interference (for example, no interference vs. first-degree interference), as shown in Theorem 2.3 of [Eckles, Karrer and Ugander (2017)](Reference). Table 2 presents the evaluation metrics for each of the six scenarios depicted in Figure 4: bias, standard deviation of simulated estimates and root-mean-square error.

Note that this example uses a relatively small set of units ($N = 400$), and the unbiased estimators (in this case, $\hat{\tau}_H(d_{11}, d_{00}), \hat{\tau}_H(d_{111}, d_{000})$) work with smaller subsets of the data than the coarser, biased estimators (here, $\hat{\tau}_H(d_{1}, d_{0})$). This is apparent if one looks at the standard deviations (SD) in Table 2. As such, with these sample sizes, the root-mean-square error is swamped by estimation variance relative to bias. As $N$ gets larger, this would change: the SD would get smaller, but the bias would remain.
Figure 4: From left to right the plot shows the distribution of the point estimates for 3000 simulations when the exposure mapping ignores interference, assumes first-degree, and second-degree interference, respectively. The top panel is for a case of positive spillover effects, and the bottom panel is for a case of negative spillover effects. In each panel, the upper-row DGP is first-degree interference and the lower-row DGP is second-degree interference. The dashed vertical line represents the true value of the target quantity, which is the average effect of going from no one treated to everyone treated.
Figure 5: The plot shows the distribution of point estimates for 3000 simulations given different proportions of missing ties for a case of positive spillover. The dashed vertical line represents the true value of the target quantity.

### 4.4 Misspecified network ties

Another type of misspecification is when the network measured by the experimenter is different than the actual interference network. In this case, the bias of the estimator will increase with the proportion of mis-measured ties. For example, with positive spillover and an elicited network with missing ties, one would mistakenly take a mixture of the potential outcomes $y_i(d_{01})$ and $y_i(d_{00})$ in estimating the average of $y_i(d_{00})$, leading to an overestimate and thereby contributing negative bias for any estimate of an effect relative to $\mu(d_{00})$.

Figure 5 shows more general consequences of working with network estimates based on a network that is randomly missing varying proportions of ties in the interference network. The data generating process is the first-degree interference set-up described above and the figure shows results from 3000 simulation draws, plotting distributions of point estimates for $\hat{\tau}_{H}(d_{01}, d_{00})$, $\hat{\tau}_{H}(d_{10}, d_{00})$, and $\hat{\tau}_{H}(d_{11}, d_{00})$. The distribution of point estimates is centered around the target quantity (the dashed line) when the proportion of unmeasured ties is zero, indicating unbiasedness. However, as the proportion of unmeasured ties increases the distribution’s shift to the left or right, depending on the net effect of the biases for the estimated potential outcome means.
4.5 Sensitivity analysis for misspecification

Egami (2017) proposes a sensitivity analysis for misspecification of the exposure mapping due to unobserved networks. The sensitivity analysis considers a situation where spillover effects occur on an unobserved “offline” network when the experimenter only observes an “online” network. The case captures situations where one uses, for example, online social network data like a Twitter follower network to specify the interference network, but that interference can also occur via offline ties like a network of high school classmates that is not captured by the online network. The analysis could apply to any situation where the measured network fails to capture all relevant ties in the true interference network. The sensitivity analysis focuses on estimating the *average network-specific spillover effect* (ANSE), which is the average causal effect of changing the treatment status of neighbors in the online network (for example, status of Twitter ties), without changing the treatment status of neighbors in the offline network (high school classmates). The analysis also requires the *stratified interference* assumption (to which we return below), which assumes that potential outcomes of unit $i$ are affected by $i$’s own treatment assignment and only the treated proportion of online and offline neighbors; the precise set of treated neighbors does not matter. Under this assumption, Egami (2017) develops parametric and nonparametric sensitivity analysis methods. The parametric method in addition assumes that the unobserved network spillover effect is linear and additive, and helps to derive simple formal conditions under which unobserved networks would explain away the estimated ANSE. The nonparametric method assumes instead that outcomes are just non-negative, and is used to bound the ANSE.

4.6 Efficient designs to estimate effects under interference

We now consider implications of the preceding analysis for designing experiments that estimate exposure-specific effects efficiently. Let us first consider why such designs are needed. If the goal is to estimate the average difference in unit potential outcomes under 100% versus 0% treatment saturation, defined as $\tau(1, 0)$ above, naive designs can perform poorly. Ugander et al. (2013) show that unit-level designs need not even yield asymptotically consistent estimators under first-degree interference if degree is also increasing as the sample size does. The problem is that very few units end up with either all or no first-degree neighbors treated.

Analogously to designs for the partial interference setting considered below, Ugander et al. (2013) propose cluster-randomized designs. In this *graph cluster randomization* the network (or graph) is partitioned into a set of clusters, such that units closer to each other in the network are assigned to the same cluster, and then treatment randomization is performed at the cluster level. Estimation then employs the inverse-probability weighted methods described above. When $\tau(1, 0)$ is the estimand, graph cluster randomization can lead to
exponentially (in sample size) lower estimator variance as compared to unit-level random assignment. This is because under graph cluster randomization, connected units are assigned to the same treatment condition more often than would happen with unit level assignment, increasing the expected number of units who are exposed to one of the full neighborhood exposure conditions. On the other hand, assigning units by cluster can contribute to increases in variance, especially insofar as it means that—due to homophily—units with similar outcomes will tend to be assigned to one or another exposure condition together. Ugander et al. (2013) analyze an intuitive graph clustering method, $\epsilon$-net clustering, where clusters are formed by finding a set of units such that all units in the set are at least $\epsilon$ hops of each other, and every unit outside the set is within $\epsilon - 1$ hops of a unit in the set. With $\epsilon = 3$ graph cluster randomization has some desirable asymptotic properties even as average degree is growing with the sample size. In practice, experimenters can use many other methods for graph partitioning or community detection, but these are more difficult to study analytically; Saveski et al. (2017) includes empirical comparison of such methods.

We compare the Horvitz-Thompson and Hajek estimators and their variance for the causal estimand of full neighborhood exposure, $\tau(d_1, d_0)$, for the case in which units are assigned to treatment via unit-level randomization as opposed to 3-net clustering randomization. In both cases, our simulation assumes that treatment assignment has a Bernoulli(1/2) distribution, there are 400 units connected by a small world network with average degree 4, and units respond to treatment following a dilated effects scenario in which the outcomes have a positive lower bound and are correlated with the unit’s first and second order degree. To compute the exposure probabilities, we set the number of replications to $R = 10000$. We run 3000 simulated replications of the experiment. The results of the simulation are presented in Table 3. They show that graph cluster randomization leads to substantially lower estimator variability with no cost in bias as indicated by a smaller root-mean-square error.

On the other hand, if the exposure model is misspecified, neither design yields unbiased or consistent estimators; however, under some models in which interference is monotonic, graph cluster randomization can nonetheless reduce bias at the cost of variance (Eckles, Karrer and Ugander, 2017).

The above discussion considers contrasting 100% versus 0% treatment saturation, for which graph cluster randomization—and other designs that produce network autocorrelation in treatment—can be advantageous. Other estimands may motivate quite different experimental designs. For example, if the goal is to estimate interference effects (e.g., $\tau(d_{01}, d_{00})$), graph clustered randomization with a perfect partitioning of a network into its $k$ connected components may make estimation impossible, since all units will have the same treatment as their neighbors.
Table 3: Comparing Unit to Cluster Randomization

| Randomization | Estimator         | Estimand       | True-Value | Average-Value | Bias   | SD      | RMSE  |
|---------------|-------------------|----------------|------------|---------------|--------|---------|-------|
| Unit          | Horvitz-Thompson  | $\tau(d_1, d_0)$ | 58.12      | 55.70         | -2.42  | 139.65  | 139.65|
| Cluster       | Horvitz-Thompson  | $\tau(d_1, d_0)$ | 58.12      | 57.52         | -0.60  | 61.31   | 61.30 |
| Unit          | Hajek             | $\tau(d_1, d_0)$ | 58.12      | 48.27         | -9.85  | 52.89   | 53.79 |
| Cluster       | Hajek             | $\tau(d_1, d_0)$ | 58.12      | 55.01         | -3.10  | 28.89   | 29.05 |

Note: Unit = unit-level randomization. Cluster = 3-net clustering. Horvitz–Thompson estimator with maximum between conservative variance estimator and constant effects variance estimator. Hajek estimator with linearized variance estimator. True-Value = Value of estimand. Average-Value = Value of estimator. SD = Empirical standard deviation from simulation. RMSE = Root-mean-square error. Estimators use approximate exposure probabilities computed by drawing 10000 treatment assignments without replacement.

4.7 Empirical Studies

Bond et al. (2012) analyze the influence of peers within a large scale voter-mobilization network experiment delivering messages to 61 million Facebook users during the day of the 2010 U.S. Congressional Election. Users were randomly assigned to a social message (98% of users), an informational message (1%) or a control condition (1%). The two treatment conditions encouraged users to vote, provided a polling-place link, allow them to express they had voted by clicking an “I voted” button and showed a counter with the number of users who had previously reported voting. In addition, the social message presented pictures of friends who had reported voting. The polling-place link was used as a measure of desire to seek information about the election and the “I voted” button as self-reported voting. Additionally, the turnout of about 10% of the users was validated with public voting records. The estimated results suggest positive direct effects of the social message on information seeking, self-reported voting and validated voting, whereas the informational message did not affect turnout. Results suggest positive spillover effects on “close friends” with whom they interact frequently on Facebook and who are likely to have real-world, face-to-face relationships, but no effects on distant friends. The way in which Bond et al. (2012) analyzed spillover effects did not properly account for direct effects, a problem discussed by Taylor and Eckles (2017) when considering sharp null hypothesis tests on indirect effects.

A follow up study was conducted by Jones et al. (2017) during the 2012 Presidential Election. In this study, the experimental conditions were adjusted to better understand the mechanisms that were likely to drive effects and the analysis of spillover effects corrected the problems with Bond et al. (2012). Instead of treating the large majority of users with the social message condition and assigning the rest to an informational or control conditions, this time a 2x2 design varied whether or not individuals saw a post at the top of their News Feeds that encouraged turnout in a similar way than the social message (the banner condition), and whether or not users saw individual posts within their News Feed regarding friends’ voting if at least one of
their friends in the banner condition had clicked on the “I voted” button (the feed condition). In the 2010 experiment, users in the banner condition also saw these messages within their feeds, not allowing to separate the encouragement from the social effect. Findings suggest that users directly exposed to both the banner and feed conditions were significantly more likely to have voted than those in the control condition. Spillover effects happened through those encouraged to vote with the banner condition, whereas the feed treatment did not spill over to close friends. However, this time there were other differences in the messaging due to the context of the 2x2 design. For example, messages in the feed condition did not contain the button to self-report voting or the link to find the polling place. These features would also need to be randomized to learn more about the differences between indirect effects across the banner and feed conditions.

Coppock, Guess and Ternovski (2016) explore direct and spillover effects of a mobilization campaign on Twitter on informal political participation, particularly signing an online petition and sharing the petition link. Followers of an environmental nonprofit advocacy organization were randomly assigned to receive a private (2/3 of followers) or a public message (1/3), and those receiving the private message were either primed to have an identity of high commitment (organizer) or of low commitment (follower) in equal shares. A second manipulation encouraged a random subset of petition signers to share the petition with their own followers (who are also followers of the environmental organization and who mostly follow one petition signer) as a way to measure spillover effects among those who actually respond directly to treatment. The design of this second manipulation reduces the number of exposure conditions that would otherwise be prohibitively large when the researcher is interested in measuring indirect effects for every possible number of treated users who are followed (from 0 to 601, according to the largest units’ degree in this network), while accounting for exposure probabilities, and without parameterizing the response to exposure in the exposure mapping. Results show that direct messages boost petition signatures and tweet behavior, and that priming the follower identity is more effective than the organizer identity. Regarding indirect effects there is evidence that signing the petition was influenced by others’ treatment.

Recent empirical studies exploiting offline networks include Green et al. (2016) who analyze spatial spillover effects in a series of field experiments testing the impact of lawn signs on vote outcomes by planting them in randomly selected voting precincts. In this case, to account for indirect effects the experimental design ensured that two neighboring precincts would not be assigned to direct treatment at the same time. Baicker (2005) exploits exogenous shocks to state medical spending to explore if spending decisions spill over to neighboring states, while Isen (2014) leverages a discontinuity from local referendum results to assess if fiscal decisions of one jurisdiction, particularly taxing and spending, influence the fiscal decisions of its neighbors, and Rogowski and Sinclair (2012) use the House office lottery (in which newly elected members select their office spaces in a randomly chosen order) as an instrumental variable to estimate the impact of
legislative networks on roll call behavior and cosponsorship decisions. The landmark study by Sacerdote (2001) uses natural random assignment of college roommates to measure spillover effects on educational performance outcomes.

Other studies have focused on strategies for targeting interventions in networks (i.e., seeding) so as to capitalize on heterogeneous spillovers. While there is a large theoretical and algorithmic literature on this problem (influence maximization), triggered by Kempe, Kleinberg and Tardos (2003) who provide a set of algorithms to maximize behavior diffusion when the researcher has knowledge of the network, there are only a few randomized experiments. These typically rely on imposing partial interference assumptions (see below) so that outcomes of different, villages or schools, for example, can be treated as independent observations. Some studies used random or haphazard assignment of treatment to analyze what types of units produce the largest spillovers. For example, the previously mentioned antibullying program study by Paluck, Shepherd and Aronow (2016), measured the network structure of 56 schools in New Jersey to analyze peer diffusion effects of randomly selected seed groups of students encouraged to take a stance against conflict at school, finding that students with more direct connections are the most effective at influencing social norms and behavior among their direct peers and at the school-level. Similarly, Banerjee et al. (2013) study the impact of the (non-randomized) choice of targeted individuals in the diffusion of participation in a new micro-finance loan program in India that invited leaders to an informational meeting and asked them to spread information about the loans. The authors develop a model of word-of-mouth diffusion and apply it to network data of 43 villages, which was collected by surveying households before the start of the program. The model distinguishes between information passing (learning about the program from neighbors) and endorsement (being influenced by neighbors’ adoption of the program—what we refer to in this chapter as contagion). This allows one to tease out the likelihood of information passing through participants as opposed to non-participants, and the marginal endorsement effect conditional on being informed. These estimates are used to propose measures of individuals’ effectiveness as seeding points. A smaller number of studies use experiments designed to compare seeding strategies. For example, Kim et al. (2015) compare the effectiveness of three seeding strategies: randomly selected individuals, individuals with the highest number of direct connections, and random friends from a nominated set of friends of random individuals (one-hop targeting), on take-up rates of a public health program in rural municipalities in Honduras; relying on strong parametric and independence assumptions, they find that, for one of two behaviors, one-hop targeting performs best. A similar strategy is examined by Banerjee et al. (2019), but with “ambitious” questions that ask respondents to select someone who would be good at spreading information; the results provide suggestive evidence that this strategy may increase spread over random seeding and seeding using village leaders. Other field experiments have been conducted for diffusion of agricultural knowledge and technology (Beaman and Dillon 2018, Beaman et al. 2018).
Chin, Eckles and Ugander (2018) present estimators and optimal experimental designs for studying seeding strategies that make use of at-most partial network information, as do strategies studied by Kim et al. (2015) and Banerjee et al. (2019).

5 Partial Interference and Marginal Causal Effects

The analysis in the preceding section is quite general in that it does little to restrict the network over which interference can occur. The only restrictions was a “local interference” assumption needed for asymptotic results to hold. The drawback, however, was that one needed to have a specification of the interference networks that was either complete or that overcompensated for interference in the true data generating process. In this section, we review the approach of Hudgens and Halloran (2008), who work under the assumption that the interference network per se is unknown, however one can assume that interference is limited to occurring within well-defined and non-overlapping groups, for example, villages or households. The assumption that interference does not cross group boundaries is known as “partial interference” (Sobel, 2006). If the interference network is not known, then we cannot map each assignment vector \( z \) to an exposure, in which case we cannot estimate exposure-specific effects. Rather, Hudgens and Halloran (2008) define more agnostic “marginal causal effects” that average over sets of assignment profiles that could, in some unspecified way, generate spillover effects. This will be made more precise below. The analysis depends on a two-stage hierarchical design, where groups are first randomly assigned to a level of treatment saturation, and then units within groups are randomly assigned to treatment with probability equal to their group saturation rate.

5.1 Marginal Causal Effects

In the most general case, each treatment assignment \( z \in \Omega \) generates a distinct potential outcome for unit \( i \). Under partial interference, the potential outcome for unit \( i \) depends only on the treatment assignments for units in \( i \)'s group \( g \). For example, suppose that we have six units, \( i = 1, 2, 3, 4, 5, 6 \), split up into two groups such that group A contains units 1, 2, and 3, and group B contains units 4, 5, and 6. Assuming that unit 2 in group A was assigned to treatment, partial interference would imply that unit 1’s potential outcomes would be the same for all assignment vectors in the set

\[
\{(0, 1, 0, z_{i,B}) : z_{i,B} \in \Omega_B \} = \{(0, 1, 0, 0, 0, 0), (0, 1, 0, 1, 0, 0), (0, 1, 0, 0, 1, 0), (0, 1, 0, 0, 0, 1),
(0, 1, 0, 1, 1, 0), (0, 1, 0, 1, 0, 1), (0, 1, 0, 0, 1, 1), (0, 1, 0, 1, 1, 1)\},
\]
where $\Omega_B$ is the set of possible assignments for group B. At the same time, it may very well be that assigning unit 3 to treatment instead of unit 2 would have different implications for unit 1’s potential outcomes—i.e., it may be that $y_{1,A}(0,1,0,z_{i,B}) \neq y_{1,A}(0,0,1,z_{i,B})$. Moreover, it would be safe to assume that either of these might differ from unit 1’s outcome if no one in group A were assigned to treatment, $y_{1,A}(0,0,0,z_{i,B})$, or if both 2 and 3 were assigned to treatment $y_{1,A}(0,1,1,z_{i,B})$. Now, suppose three fair coin flips were used to determine whether unit 1, unit 2, or unit 3 should be assigned to treatment. Then, there is a 50-50 chance that unit 1 would be assigned to control. Conditional on unit 1 being assigned to control, the expected value of unit 1’s outcome would be the average over the four potential outcomes $y_{1,A}$ enumerated above. This expected value is unit 1’s marginal (i.e., average) potential outcome given that unit 1 is not treated but under a regime that assigns units in 1’s group to treatment with 50-50 probability. Hudgens and Halloran (2008)’s analysis defines marginal causal effects as contrasts between such marginal potential outcomes. More generally, we refer to the individual’s marginal potential outcome as $y_{ig}(z;\psi)$ when $i$ is assigned to treatment value $z$ and other treatment assignments are determined by an assignment regime characterized by the parameter $\psi$, which describes the degree of treatment saturation. In the simple example in the preceding paragraph, we have $\psi = 0.50$ to describe the regime where each unit is assigned to treatment using a Bernoulli draw with $p = 0.50$, in which case the expected saturation is 50%. Under complete random assignment, which fixes the number of treated and control units, $\psi$ could index the share of units assigned to treatment. Hudgens and Halloran (2008) consider four types of marginal causal effects: direct, indirect, total, and overall effects. The direct effect for a particular unit corresponds to the difference between the unit’s potential outcomes when its treatment assignment changes while the group treatment assignment is kept fixed to a given saturation. Then, the group average direct causal effect, under treatment saturation $\psi$ and group size $n_g$, can be defined as $\tau^D_g(\psi) = \frac{1}{n_g} \sum_{i=1}^{n_g} y_{ig}(1;\psi) - \frac{1}{n_g} \sum_{i=1}^{n_g} y_{ig}(0;\psi)$, and the population average direct causal effect $\tau^D(\psi)$ (or simply, the average direct causal effect) is the average of $\tau^D_g(\psi)$ across groups. The indirect effect describes the effect on a unit of the treatment received by others in the group, and is obtained from differences across saturation values $\psi$ and $\phi$. Thus, the group average indirect causal effect is $\tau^I_g(\psi,\phi) = \frac{1}{n_g} \sum_{i=1}^{n_g} y_{ig}(0;\psi) - \frac{1}{n_g} \sum_{i=1}^{n_g} y_{ig}(0;\phi)$, while the average indirect causal effect averages across groups. The total causal effect combines the direct and indirect effects to capture the effect of being directly treated and exposed to the treatment by others in the group. Thus, the group average total causal effect is $\tau^{To}_g(\psi,\phi) = \frac{1}{n_g} \sum_{i=1}^{n_g} y_{ig}(1;\psi) - \frac{1}{n_g} \sum_{i=1}^{n_g} y_{ig}(0;\phi)$, and the average total causal effect takes the average of $\tau^{To}_g(\psi,\phi)$ across groups. Under no-interference, the indirect causal effect is zero and the total causal effect equals the direct causal effect. Finally, the overall causal effect corresponds to the group’s response to different treatment saturation levels. The group average overall causal effect can be written
as \( \tau^O_g(\psi, \phi) = \frac{1}{n_g} \sum_{i=1}^{n_g} y_{ig}(\psi) - \frac{1}{n_g} \sum_{i=1}^{n_g} y_{ig}(\phi) \), where \( y_{ig}(\psi) \) is the average potential outcome for unit \( i \) in group \( g \) under all possible assignments given saturation \( \psi \). We average the \( \tau^O_g(\psi, \phi) \) across groups to obtain the average overall causal effect.

Under Bernoulli random assignment, these causal quantities are well defined and have a clean \textit{ceteris paribus} interpretation. Under any other design, the causal interpretation is not always clean. For example, under complete random assignment the individual-level direct effect for unit \( i \) would contrast outcomes with different numbers of group members other than \( i \) assigned to treatment (i.e., \( \psi(n_g - 1) \) when \( i \) is assigned to treatment and \( \psi(n_g) \) when \( i \) is assigned to control). This point is discussed by Sävje, Aronow and Hudgens (2017).

Let the observed outcome be \( Y_{ig} = y_{ig}(Z_{ig}) \) under the group treatment assignment \( Z_{ig} \) and suppose treatment saturation is \( \psi \). Given Bernoulli assignment with probability \( \psi \), an unbiased estimator for \( \frac{1}{n} \sum_{i=1}^{n} y_{ig}(z; \psi) \), with \( z = \{0, 1\} \), is

\[
\hat{g}_g(z; \psi) = \frac{\sum_{i=1}^{n} Y_{ig}(Z_{ig} = z)}{\sum_{i=1}^{n} I(Z_{ig} = z)},
\]

and for the population average potential outcome, it is

\[
\hat{g}(z; \psi) = \frac{\sum_{g=1}^{G} \hat{g}_g(z; \psi) I(S_g = \psi)}{\sum_{g=1}^{G} I(S_g = \psi)},
\]

where \( S_g \) is the saturation level of group \( g \) (conditionally on the denominators of these estimators being nonzero). Therefore, an unbiased estimator for the average direct effect is \( \hat{\tau}^D(\psi) = \hat{g}(1; \psi) - \hat{g}(0; \psi) \). Unbiased estimators for the other causal estimands of interest are defined analogously: \( \hat{\tau}^I(\psi, \phi) = \hat{g}(0; \psi) - \hat{g}(0; \phi) \), \( \hat{\tau}^O(\psi, \phi) = \hat{g}(\psi) - \hat{g}(\phi) \), where \( \hat{g}(\psi) \) is the average across groups assigned to saturation \( \psi \) of the average observed outcomes of units in the group under treatment assignment \( Z_{ig} \).

For inference, Hudgens and Halloran (2008) derive variance estimators under an assumption called stratified interference. This refers to the situation where potential outcomes for unit \( i \) in group \( g \) do not vary on the basis of which other units in group \( g \) are assigned to treatment, only in the number or share of other units assigned to treatment. This assumption reduces the problem statistically to the usual stratified setting without interference. When stratified interference holds, their proposed variance estimators are unbiased if unit causal effects are additive (e.g., \( y_{ig}(1, \psi) = y_{ig}(0, \psi) + d \), where \( d \) is constant), and otherwise positively biased. Liu and Hudgens (2014a) discuss conditions for asymptotic normality. Tchetgen and VanderWeele (2012) extend Hudgens and Halloran (2008)’s results, providing conservative variance estimators, a framework for finite sample inference with binary outcomes, and extensions to observational studies. Liu and Hudgens (2014a) discuss conditions for asymptotic normality. Tchetgen and VanderWeele (2012) extend Hudgens and Halloran (2008)’s results, providing conservative variance estimators, a framework for finite sample inference with binary outcomes, and extensions to observational studies.
(2014b) develop asymptotic results for two-stage designs.

Both Sinclair, McConnell and Green (2012) and Baird et al. (2017) discuss statistical power for hierarchical designs. Baird et al. (2017) offer thorough consideration of the optimal choice (in terms of statistical power) of saturation levels ($\psi$ and $\phi$) and a distribution of these levels over groups for estimating direct, indirect, total, and overall effects. Their methods assume the population is partitioned into equal-sized non overlapping groups, that partial interference and stratified interference hold, and a linear-in-means outcome model. The optimal set of saturations and shares of groups assigned to each saturation depends on the correlation of potential outcomes within groups.

We show with a toy example how to compute these estimators and their variance. Suppose we have 18 units equally divided in 6 groups. In the first stage half of the groups are assigned to saturation $\psi$ and the other half to $\phi$ with equal probability. In the second stage, using complete random assignment two-thirds of units in groups with saturation $\psi$ are assigned to the treatment condition and one-third to the control condition, and one-third of units in groups with saturation $\phi$ are assigned to treatment, while two-thirds to control. For the purposes of the simulation, we will assume stratified interference and compute potential outcomes under a dilated effects scenario such that $y_{ig}(1, \psi) = 2 \times y_{ig}(0, \phi)$, $y_{ig}(1, \phi) = 1.5 \times y_{ig}(0, \phi)$, and $y_{ig}(0, \psi) = 1.25 \times y_{ig}(0, \phi)$, where $y_{ig}(0, \phi)$ is obtained with a random draw from an absolute standard normal distribution.

We display the structure of the post-treatment data that the experimenter has to have to compute the estimators. (We show the data of six units only.) In this case, the realized saturation for group 1 is $\psi$ (the group is assigned to the treatment condition in the first stage as indicated in column “group_tr” with value 1, and 2/3 of units within the group are treated in the second stage as shown in column “indiv_tr”) and for group 4 is $\phi$ (the group is assigned to the control condition in the first stage, and therefore 1/3 of units are treated in the second stage):

| group | group_tr | indiv_tr | obs_outcome |
|-------|----------|----------|-------------|
| 1     | 1        | 1        | 0.9269359   |
| 2     | 1        | 1        | 0.2788864   |
| 3     | 1        | 1        | 0.9606388   |
| 10    | 4        | 0        | 0.9062178   |
| 11    | 4        | 0        | 1.0599419   |
| 12    | 4        | 0        | 0.6051009   |

post_tr_data[c(1:3,10:12),]

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The estimators and their variance are computed under the stratified interference assumption with the following function from the `interference` package:

```r
estimates <- estimates_hierarchical(post_tr_data)
ciausal_effects <- unlist(estimates[[1]])
ciausal_effects <- unlist(estimates[[2]])
```

| direct_psi_hat | direct_phi_hat | indirect_hat | total_hat | overall_hat |
|---------------|---------------|-------------|-----------|-------------|
| 1.0009851     | 0.2318532     | 0.2195894   | 1.2205745 | 0.8096284   |

| var_direct_psi_hat | var_direct_phi_hat | var_indirect_hat |
|--------------------|--------------------|------------------|
| 0.71255260         | 0.04854851         | 0.28000088       |

| var_total_hat | var_overall_hat |
|--------------|-----------------|
| 0.88936810   | 0.39005445      |

### 5.2 Misspecifying partial interference

What happens when the experimenter assumes partial interference but in fact there is interference not only within groups, but also across groups? We show that when partial interference does not hold, the proposed estimators for the direct, indirect, total and overall causal effects are biased.

We assume a scenario in which groups belong to tracts and interference happens within groups and across groups within tracts, but not across tracts. Each tract is composed of two groups. The sample size is 450 units, equally divided into six groups. As before, units are assigned to treatment under a two-stage randomized experiment, with treatment saturation set to \( \psi = 2/3 \) and baseline saturation to \( \phi = 1/3 \). We assume stratified interference at the level of tracts to compute unit potential outcomes. We continue to use a dilated effects scenario, but in this case it is the proportion of treated units in the tract that matters, as opposed to the proportion of treated units in the group.

Causal estimands are in this case defined as differences between unit potential outcomes, conditional on a tract saturation. For example, the population average direct causal effect under treatment intensity \( \psi \), tract size \( n_{tr} \), and \( Tr \) tracts is now defined as

\[
\tau_D(\psi\psi) = \frac{1}{Tr} \sum_{tr=1}^{Tr} \left[ \frac{1}{n_{tr}} \sum_{i=1}^{n_{tr}} y_{itr}(1; \psi\psi) - \frac{1}{n_{tr}} \sum_{i=1}^{n_{tr}} y_{itr}(0; \psi\psi) \right].
\]

The notation \( \tau_D(\psi\psi) \) implies that the realized saturation for unit \( i \)'s group and the other group in \( i \)'s tract is \( \psi \). (In this example, 4/6 of the units in tract \( t_r \) are assigned to the treatment condition, given that groups and
tracts are of equal size.) However, when the interference dependency structure is misspecified, particularly, when partial interference does not hold, $\hat{\tau}_D(\psi)$ is a biased estimator for $\tau_D(\psi\psi)$, given that it includes the observed outcomes of units in groups with realized saturation $\psi$, in tracts where other groups are assigned saturation $\phi$. In other words, the estimator for the population average potential outcome under saturation $\psi$ is

$$\hat{y}(z; \psi) = \frac{\sum_{g=1}^G \hat{y}_g(z; \psi\psi)I(S_g = \psi\psi)}{\sum_{g=1}^G I(S_g = \psi\psi)} + \frac{\sum_{g=1}^G \hat{y}_g(z; \psi\phi)I(S_g = \psi\phi)}{\sum_{g=1}^G I(S_g = \psi\phi)}.$$

The average outcome across groups includes a mixture of the average of observed outcomes for units with treatment $z$ in a group with realized saturation $\psi$ in a tract in which the realized saturation for the other group is $\psi$, and the average of observed outcomes for units with treatment $z$ in a group with realized saturation $\psi$ in a tract in which the realized saturation for the other group is $\phi$.

In Figure 6 we compare the estimators of the direct, indirect, total and overall causal effects when the partial interference is misspecified (top row) on the basis of the data generating process defined in the preceding paragraphs, and when it is correctly specified (bottom row). The estimators are unbiased when partial interference holds. However, the estimators are biased when partial interference does not hold, but the experimenter assumes that it does (top row).

### 5.3 Empirical Studies

Sinclair, McConnell and Green (2012) assess peer effects within a large scale voter-mobilization hierarchical experiment (of about 70,000 individuals) conducted in Chicago during a special election in 2009, using as treatment social-pressure mailings which are sent shortly before the election and disclose whether a member of the household has voted in prior elections. The goal is to estimate total causal effects of mailings and indirect causal effects across and within households. In the first stage of randomization, neighborhoods (which are about equal-sized with at most 15 households) are assigned to one of four different saturations: 100% of households in the neighborhood are treated, 50%, only one, or zero. In the second stage, households are randomly assigned to treatment according to the neighborhood saturation, and exactly one individual within each household is randomly selected to receive the social-pressure message. Therefore, in one-person households, that person has probability one of being treated, and in two-person and three-person households, each person has a one-half and one-third probability of receiving the treatment, respectively. The key assumption is partial interference at the level of neighborhoods. The authors find positive total causal effects of receiving the message on turnout and some evidence of within-household effects but no evidence of interference across households.

Nickerson (2008) runs a two-stage placebo-controlled experiment where in the first stage, households
Figure 6: The plot shows the distribution of point estimates for 3000 simulations with partial interference specified at the level of groups only. In the upper-row the data generating process is with interference within and across groups (general interference) and in the bottom-row within groups only (partial interference). The dashed vertical line represents the value of the estimand.
with two registered voters are assigned to either a 50% saturation, a 0% placebo, or a 0% pure control saturation. In households assigned to the 50% saturation, residents who answer the door receive a face-to-face get-out-the-vote message (GOTV condition), whereas in households assigned to the 0% placebo, residents who answer get a recycling pitch (recycling condition). The experiment was conducted during the 2002 Congressional primaries in Denver and Minneapolis. Households were contacted the weekend before the election. This design identifies the average direct effect of mobilization with a difference-in-means estimator between observed voter turnout among reachable voters (those who answer the door) across the GOTV and recycling conditions. Likewise, the average indirect effect is the difference-in-means between observed voter turnout among unreachable residents (those who did not answer the door) across the GOTV condition and recycling conditions. The author finds a statistically significant increase in turnout of unreachable residents in GOTV households of about 6 percentage points, and of 9.8 percentage points among reachable residents, suggesting that when one voter opens the door to a canvasser, 60% of the effect from the get-out-the-vote appeal is transmitted to the other household member.

With the same design, Bond (2018) analyzes the data of a face-to-face canvassing experiment by Broockman and Kalla (2016) encouraging active perspective taking intended to reduce transphobia. The effects of the perspective taking exercise spill over to household residents who do not answer the door, reducing their anti-transgender prejudice. Other empirical studies using a hierarchical design include Duflo and Saez (2003), who analyze spillover effects in individuals’ choice of retirement plan, Miguel and Kremer (2004) that study the effects of a deworming medical treatment on health and school participation of untreated children in (partially) treated schools and of children in neighboring control schools. Similarly, Angelucci and De Giorgi (2009) analyze indirect effects of the cash transfer program Progresa on consumption. In this case, only the first stage is randomized assigning municipalities to treatment or control, while in the second stage a subset of individuals are offered treatment based on their income. This design identifies direct and indirect effects when there is partial interference, but cannot identify whether these effects vary with intensity of treatment because there is no exogenous variation in treatment saturation. (This is also true of Nickerson (2008)’s and Duflo and Saez (2003)’s design in which the group-level saturations are fixed at 50%.) A similar case is presented by Sur et al. (2009), who study the effectiveness of a typhoid vaccine with a design that randomly assigns geographic clusters to receive the vaccine or a placebo vaccine, but individuals self-selected into treatment in the second stage. Likewise, the design by Wilke, Green and Cooper (2019) to study the effectiveness of education-entertainment on attitudes towards violence against women, teacher absenteeism, and abortion stigma, first assigned villages to treatments and in the second stage individuals self-selected into treatment. Crépon et al. (2013) implement a two-stage hierarchical design in the context of a job placement assistance program in France in which cities are assigned to either one of four positive saturations or a control
condition, and then job seekers are randomly assigned to treatment according to their city saturation. The program has positive direct effects, but negative spillover effects: the likelihood of finding a stable job for untreated job seekers in positive saturation cities is smaller than for untreated job seekers in control areas. 

Bhatti et al. (2017) combine a hierarchical design with network data on family ties to assess spillover effects of a GOTV experiment that mobilized young voters with text messages during municipal and European Parliament elections in Denmark, and Giné and Mansuri (2018) assess direct and indirect effects of a voter awareness campaign on female turnout and candidate choice in Pakistan by assigning first geographical clusters within villages to either one of two treatments or to control (with unequal probabilities because the number of clusters varied by village) and then randomly targeting a subset of households in treatment clusters. Basse and Feller (2018) evaluate an intervention to reduce student absenteeism in Philadelphia with a two-stage randomization design in which households with multiple students were first assigned to treatment or control, then exactly one student in treatment households was randomly selected for the student’s parents to receive student-specific information. The authors address the practical problem presented to researchers when household size varies (or when the number of geographical clusters across villages varies as in Giné and Mansuri (2018)): whether to assign equal weight to individuals or to households, depending on what is relevant from a policy perspective. The authors propose unbiased estimators for individual and household weighted estimands.

6 Contagion

By analogy to biological contagion, (social) contagion refers to processes through which the outcomes of one unit causally affect the outcomes of another unit. Such processes were illustrated above in Figure 1 as the path \( Y_1 \rightarrow Y_2 \). In these narrow terms, contagion is distinct from interference. That said, contagion can be a mechanism through which interference occurs, and one that may call into question assuming a particular exposure mapping that limits the extent of interference (Manski, 2013; Eckles, Karrer and Ugander, 2017). Assessing whether spillover effects are due to contagion amounts to conducting a mediation analysis, where the mediators are treated units’ outcomes (VanderWeele, Tchetgen and Halloran, 2012; Ogburn and VanderWeele, 2017). This was displayed in Figure 1 as the path \( Z_1 \rightarrow Y_1 \rightarrow Y_2 \), where \( Y_1 \) is the mediator in the contagion process. To evaluate whether spillover effects are due to contagion, conditions must hold so as to identify both spillover effects (as discussed in this chapter) as well as mediation effects. Identifying mediation effects requires that other types of conditional independence hold, such as sequential ignorability (Imai, Keele and Yamamoto, 2010; Pearl, 2014). These are strong assumptions about the data generating process and typically cannot be induced directly by an experimental design (Imai, Tingley and Yamamoto).
Imai and Jiang (2019) re-analyze the two-stage placebo-controlled get-out-the-vote (GOTV) experiment by Nickerson (2008). Their goal is to analyze whether canvassing increases the turnout of the voter who does not answer via effects on the vote intention of the reachable voter (contagion), or via other channels such as conversations within the household (non-contagion spillover). They start with a decomposition of the indirect effect into the sum of a contagion effect—canvassing influences the turnout of the untreated voter of a contacted household by changing the vote intention of the treated voter (approximated by turnout)—and effects due to other mechanisms. With reference to the setting of Nickerson (2008), this decomposition is based on the following: there is no spillover across households, reachable voters form a vote intention immediately after being contacted, where we denote potential vote intention as $y^*_{i1}(z_i) \in \{0, 1\}$, and the turnout of all voters and all households is observed. Let $y_{i1}(z_i)$ and $y_{i2}(z_i, y^*_{i1}(z_i))$ represent the potential voting outcome of reachable and unreachable voters in complier household $i$, respectively. Causal quantities of interest are defined only for compliers (that is, residents of households where someone answers the door). As such, the analysis is limited to households in which someone answered the door either in the canvassing or the placebo (recycling) conditions.

The indirect effect of the GOTV campaign is thus defined as the difference in the potential outcomes of unreachable voters when their household is assigned to the GOTV condition ($z_i = 1$) as opposed to a control condition ($z_i = 0$). The average indirect effect is obtained by taking the average difference across complier households and can be expressed (in terms of a finite population of $N_c$ complier households) as

$$\theta = \frac{1}{N_c} \sum_{i=1}^{N_c} y_{i2}(1, y^*_{i1}(1)) - y_{i2}(0, y^*_{i1}(0)).$$

Then, $\theta$ is decomposed into the sum of the contagion effect and the effect of other mechanisms by considering the vote intention of the treated voter $y^*_{i1}$ as the mediator. Imai and Jiang (2019) define an “average contagion effect” as follows (again, written in terms of a finite population of $N_c$ complier households):

$$\gamma(z) = \frac{1}{N_c} \sum_{i=1}^{N_c} y_{i2}(z, y^*_{i1}(1)) - y_{i2}(z, y^*_{i1}(0)).$$

Note that it is possible for $y^*_{i1}(1) = y^*_{i1}(0)$, in which case there would be no contagion effect for household $i$. The quantity $\gamma(z)$ thus aggregates over cases where there could or could not be contagion effects, and then for the former, over the magnitude of any contagion effect. It is therefore analogous to what is known as a “natural” effect, rather than a “controlled” effect, in the mediation literature (Imai, Keele and Yamamoto).
2010). Other, non-contagion mechanisms are captured by

\[ \eta(z) = \frac{1}{N_c} \sum_{i=1}^{N_c} y_{1z}(1, y^*_{i1}(z)) - y_{1z}(0, y^*_{i1}(z)). \]

The indirect effect can be decomposed as \( \theta = \gamma(1) + \eta(0) = \gamma(0) + \eta(1) \).

Identification of this decomposition requires a sequential ignorability assumption from mediation analysis. Such assumption implies that conditional on treatment status and pre-treatment covariates, the vote intention of the treated voter is independent of the potential outcome of the unreachable voter. This assumption would be violated in the presence of unobserved confounders (such as political efficacy) that affect both the vote intention of the reachable voter and turnout of the unreachable voter. It is important to note that this assumption is stronger than the usual ignorability condition necessary for observational studies because it requires so-called “cross-world independence assumptions”—i.e., assumptions about potential outcomes that can never be revealed by experimentation. Under this key assumption, Imai and Jiang (2019) estimate that indirect effects can be largely explained by contagion, even for households whose treated voter is a Democrat and unreachable voter is a Republican. Because this mediation analysis involves such strong assumptions, the authors reasonably conduct a sensitivity analysis, examining how robust these conclusions are to violations of the sequential ignorability assumption.

Other empirical studies exploring contagion effects include Forastiere, Mealli and VanderWeele (2016), who analyze contagion of an encouragement program on households’ use of bed nets in Zambia. Relying on semi-parametric outcome models, Ferrali et al. (2018) study the effects of an encouragement campaign on the adoption of a new political communication technology in Uganda, and Vásquez-Cortés (2018) leverages exogenous shocks to analyze contagion effects of criminal behavior among ex-combatants in Colombia. One way analysis of contagion can avoid the strong sequential ignorability assumptions is to instead assume complete mediation (i.e., an exclusion restriction of instrumental variables estimation) such that all spillover effects are due to contagion via a particular outcome; for example, Eckles, Kizilcec and Bakshy (2016) conduct an experiment in which they posit that treatment of an individual’s peers only affects them via specific directed behaviors.

7 Conclusion

Standard methods for analyzing experiments assume no interference, which assumes that a unit’s own treatment status is all that one needs to know to characterize its outcome. In many settings, including many of the empirical examples discussed in this chapter, such an assumption is unwarranted. Such spillovers
may merely represent a nuisance for estimating quantities of interest. In such cases, experimenters may want to introduce adjustments to their designs so as to minimize the potential for exposure to other units. Alternatively, experimenters could try working at a higher level of aggregation at which interference is less likely to be a concern. On the other hand, researchers may have a substantive interest in estimating spillover effects. This chapter presumed such an interest and proposed methods for doing so.

We reviewed two analytical frameworks for estimating spillover effects in experiments. In the first, the structure of interference is known but can be of almost arbitrary form. In the second, the interference structure is mostly unknown except that the experimenter can be confident that interference is fully contained within non-overlapping groups. We demonstrated how one can work under either framework to estimate spillover effects using the interference R package. We also illustrate the implications of specifying the nature and extent of interference.

Our review of empirical studies demonstrates the relevance of spillover effects to various social phenomena, such as voting, petitioning, student behavior, norms against violence, prejudice, economic decisions, and subjective well-being. These studies also offer examples of designs that operationalize the analytical frameworks. We hope that the analytical foundation and examples provided can help experimenters to push both the methodological and empirical frontiers in our understanding of spillover effects.

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