Average Direct and Indirect Causal Effects

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Abstract

We propose a definition for the average indirect effect of a binary treatment in the potential outcomes model for causal inference under cross-unit interference. Our definition is analogous to the standard definition of the average direct effect, and can be expressed without needing to compare outcomes across multiple randomized experiments. We show that the proposed indirect effect satisfies a decomposition theorem whereby, in a Bernoulli trial, the sum of the average direct and indirect effects always corresponds to the effect of a policy intervention that infinitesimally increases treatment probabilities. We also consider a number of parametric models for interference, and find that our (non-parametric) indirect effect remains a natural estimand when re-expressed in the context of these models.

1 Introduction

The classical way of analyzing randomized trials, following Neyman [1923] and Rubin [1974], is centered around the average treatment effect as defined using potential outcomes. Given a sample of \(i = 1, \ldots, n\) units used to study the effect of a binary treatment \(W_i \in \{0, 1\}\), we posit potential outcomes \(Y_i(0), Y_i(1) \in \mathbb{R}\) corresponding to the outcome we would have measured had we assigned the \(i\)-th unit to control or treatment respectively, i.e., we observe \(Y_i = Y_i(W_i)\). We then proceed by arguing that the (sample) average treatment effect

\[
\tau_{ATE} = \frac{1}{n} \sum_{i=1}^{n} (Y_i(1) - Y_i(0))
\]

admits a simple unbiased estimator under random assignment of treatment.

One limitation of this classical approach is that it rules out interference, and instead bakes in an assumption that the observed outcome for any given unit does not depend on the treatments assigned to other units, i.e., \(Y_i\) is not affected by \(W_j\) for any \(j \neq i\) [Halloran and Struchiner, 1995]. However, in a wide variety of applied settings, such interference effects not only exist, but are often of considerable scientific interest [Bakshy et al., 2012, Bond et al., 2012, Cai et al., 2015, Miguel and Kremer, 2004, Rogers and Feller, 2018, Sacerdote, 2001]. For example, in an education setting, it may be of interest to understand how a didactic innovation affects not only certain targeted students, but also their peers. This has led to a recent surge of interest in methods for studying randomized trials under interference [Aronow and Samii, 2017, Eckles et al., 2017, Hudgens and Halloran, 2008, Leung, 2020, Li and Wager, 2020, Manski, 2013, Sävje et al., 2021, Tchetgen Tchetgen and VanderWeele, 2012].
A major difficulty in working under interference is that we no longer have a single “obvious” average effect parameter to target as in (1). In the general setting, each unit now has $2^n$ potential outcomes corresponding to every possible treatment combination assigned to the $n$ units, and these can be used to formulate effectively innumerable possible treatment effects that can arise from different assignment patterns. As discussed further in Section 3 below, the existing literature has mostly side-stepped this issue by framing the estimand in terms of specific policy interventions. However, this paradigm does not provide researchers with simple, non-parametric and agnostic average causal estimands that can be studied without spelling out a specific a policy intervention of interest.

In this paper, we study a pair of averaging causal estimands, the average direct effect and the average indirect effect, that are valid under interference yet, unlike existing targets, can be defined and estimated using a single experiment and do not need to be defined in terms of hypothetical policy interventions. Qualitatively, the average direct effect measures the extent to which, in a given experiment and on average, the outcome $Y_i$ of a unit affect by its own treatment $W_i$; meanwhile, the average indirect effect measures the responsiveness of $Y_i$ to treatments $W_j$ given to other units $j \neq i$.

The average direct effect we consider is standard and has recently been discussed by a number of authors including VanderWeele and Tchetgen Tchetgen [2011] and Sävje, Aronow, and Hudgens [2021]. Our definition of the average indirect effect is to the best of our knowledge new, and is the main contribution of this paper. We follow this definition with a number of results to validate it. In particular, we prove a universal decomposition theorem whereby the sum of the average direct and indirect effects can always be interpreted as the total effect of an intuitive policy intervention. We also interpret these estimands in the context of a number of parametric models for interference considered by practitioners.

2 Treatment Effects under Interference

We study different experimental designs using the potential outcomes model. The main difference between our setting and the standard Neyman-Rubin model is that potential outcomes for the $i$-th unit may also depend on the intervention given to the $j$-th unit with $j \neq i$. For a further discussion of potential outcomes under interference, see Aronow and Samii [2017] and Hudgens and Halloran [2008].

Assumption 1. For units $i = 1, \ldots, n$, there are potential outcomes $Y_i(w) \in \mathbb{R}$, $w \in \{0, 1\}^n$ such that, given a treatment vector $W \in \{0, 1\}^n$, we observe outcomes $Y_i = Y_i(W)$.

In this context, we define the average direct and indirect effects as follows. For convenience, we use short-hand $Y_i(w_j = x; W_{-j})$ to denote the potential outcome we would observe for the $i$-th unit if we were to assign the $j$-th unit to treatment status $x \in \{0, 1\}$, and otherwise maintained all but the $j$-th unit at their realized treatments $W_{-j} \in \{0, 1\}^{n-1}$.

Definition 1. Under Assumption 1, the average direct effect of a binary treatment is

$$
\tau_{ADE} = \frac{1}{n} \sum_{i=1}^{n} \mathbb{E} [Y_i(w_i = 1; W_{-i}) - Y_i(w_i = 0; W_{-i})].
$$

(2)

Definition 2. Under Assumption 1, the average indirect effect of a binary treatment is

$$
\tau_{AIE} = \frac{1}{n} \sum_{i=1}^{n} \sum_{j \neq i} \mathbb{E} [Y_j(w_i = 1; W_{-i}) - Y_j(w_i = 0; W_{-i})].
$$

(3)
The definition of the direct effect $\tau_{\text{ADE}}$ is standard. It is follows from averaging $Y_i(w_i = 1; W_{-i}) - Y_i(w_i = 0; W_{-i})$ referred to as the direct causal effect by Halloran and Struchiner [1995]. Sävje et al. [2021] provide a recent in-depth discussion on this estimand. This estimand measures the average effect of an intervention $W_i$ on the unit being intervened on—while marginalizing over the rest of the treatment assignments. In a study without interference, $\tau_{\text{ADE}}$ matches the usual average treatment effect (1).

Meanwhile, our definition of the indirect effect is an immediate formal generalization of $\tau_{\text{ADE}}$ to cross-unit treatment effects. It measures the average effect of an intervention $W_i$ on all units except the one being intervened on, again marginalizing over the rest of the process. More precisely, the term $\mathbb{E}[Y_j(w_i = 1; W_{-i}) - Y_j(w_i = 0; W_{-i})]$ is the effect of changing unit $i$’s treatment on the outcome of unit $j$. Thus the sum, $\sum_{j \neq i} \mathbb{E}[Y_j(w_i = 1; W_{-i}) - Y_j(w_i = 0; W_{-i})]$, would correspond to the “total” effect of unit $i$’s treatment on all other units. Then the defined average indirect effect $\tau_{\text{AIE}}$ corresponds to the average of the effects of units’ treatments on other units.

The definition of $\tau_{\text{AIE}}$ formally mirrors that of $\tau_{\text{ADE}}$, and in the no-interference case we clearly have $\tau_{\text{AIE}} = 0$ (as we would want). As a first step towards validating the definition of $\tau_{\text{AIE}}$ under non-trivial interference, we consider the average overall effect induced by adding $\tau_{\text{ADE}}$ and $\tau_{\text{AIE}}$, which aggregates the marginalized effect of all treatments on all outcomes. We then prove that, in a Bernoulli design, this matches the policy effect of infinitesimally increasing each unit’s treatment probability. We use the term Bernoulli design to refer to an experiment where there is a (deterministic) vector $\pi \in (0, 1)^n$ such that the treatments $W_i$ are generated as $W_i \sim \text{Bernoulli}(\pi_i)$ for all $i = 1, \ldots, n$, independently of each other and of the potential outcomes $\{Y_i(w)\}$. For a Bernoulli design with treatment probabilities $\pi \in [0, 1]^n$, we write $\mathbb{E}_\pi[\cdot]$ for expectations over the random treatment assignment, and write $\tau_{\text{ADE}}(\pi)$, $\tau_{\text{AIE}}(\pi)$ and $\tau_{\text{AOE}}(\pi)$ for the corresponding direct, indirect and overall effects.

**Definition 3.** Under Assumption 1, the average overall effect of a binary treatment is

$$\tau_{\text{AOE}} = \tau_{\text{ADE}} + \tau_{\text{AIE}} = \frac{1}{n} \sum_{i=1}^{n} \sum_{j=1}^{n} \mathbb{E}[Y_j(w_i = 1; W_{-i}) - Y_j(w_i = 0; W_{-i})]. \quad (4)$$

**Definition 4.** Under Assumption 1 and in a Bernoulli design, the infinitesimal effect of raising treatment probabilities is

$$\tau_{\text{INF}}(\pi) = 1 \cdot \nabla_{\pi} \mathbb{E}_\pi \left[ \frac{1}{n} \sum_{i=1}^{n} Y_i \right] = \sum_{k=1}^{n} \frac{\partial}{\partial \pi_k} \mathbb{E}_\pi \left[ \frac{1}{n} \sum_{i=1}^{n} Y_i \right]. \quad (5)$$

**Theorem 1.** Under Assumption 1 and in a Bernoulli design, $\tau_{\text{AOE}}(\pi) = \tau_{\text{INF}}(\pi)$.

By connecting our abstract notions of direct, indirect and overall effects to the effect of a concrete policy intervention, Theorem 1 provides an alternative lens on our definition of the indirect effect. Suppose, for example, that a researcher knew they wanted to study nudge interventions (the total effect of which is $\tau_{\text{INF}}(\pi)$), and was also committed to the standard definition of the average direct effect given in Definition 1. Then, it would be natural to define an indirect effect as $\tau_{\text{INF}}(\pi) - \tau_{\text{ADE}}(\pi)$, i.e., to characterize as “indirect effect” any effect of the nudge intervention that is not captured by the direct effect; this is, for example, the approach (implicitly) taken in Heckman et al. [1998]. From this perspective, Theorem 1 could be seen as showing that these two possible definitions of the indirect effect in fact match, i.e., that $\tau_{\text{AIE}}(\pi) = \tau_{\text{INF}}(\pi) - \tau_{\text{ADE}}(\pi)$. We emphasize that Theorem 1 is a direct
consequence of Bernoulli randomization, and holds conditionally on any realization of the potential outcomes \{Y_i(w)\}.

We refer to \(\tau_{\text{INF}}(\pi)\) as a policy effect because, in the ideal situation when one has access to observed outcomes \(Y_i\) for different randomization probabilities \(\pi\), \(\tau_{\text{INF}}(\pi)\) is a quantity that could be measured by averaging observed outcomes \(Y_i\) for different \(\pi\). In the special case where treatment assignment probabilities are a constant, i.e., there is a \(\pi_0 \in (0, 1)\) such that \(\pi_i = \pi_0\) for all \(i = 1, \ldots, n\), \(\tau_{\text{INF}}(\pi)\) takes on a particularly simple form,

\[
\tau_{\text{INF}}(\pi) = \frac{d}{d\pi_0} \mathbb{E}_{\pi_0} \left[ n^{-1} \sum_{i=1}^n Y_i \right].
\]

Infinitesimal policy effects as defined above are not (currently) widely used in the potential outcomes based literature on causal inference under interference, but they are prevalent in social sciences due to their ease of interpretation and desirable analytic properties; see, e.g., Chetty [2009], Carneiro et al. [2010], and references therein. Wager and Xu [2021] discuss welfare implications for a social planner who uses analogous infinitesimal policy effects to optimize a system via gradient-based methods.

3 Alternative Definitions and Related Work

There are also a number of other average causal effect estimands that have recently been discussed in the literature. In the case of direct effects, the main alternative to Definition 1 is the following proposal from Hudgens and Halloran [2008] that relies on conditional expectations:

\[
\tau_{\text{HH}, \text{DE}} = \frac{1}{n} \sum_{i=1}^n \left( \mathbb{E} \left[ Y_i \mid W_i = 1 \right] - \mathbb{E} \left[ Y_i \mid W_i = 0 \right] \right).
\]

In a Bernoulli design, \(\tau_{\text{HH}, \text{DE}} = \tau_{\text{ADE}}\). However, in other designs, e.g., completely randomized designs or stratified designs, these two estimands do not match. As discussed in VanderWeele and Tchetgen Tchetgen [2011] and Sävje et al. [2021], a major drawback of the definition \(\tau_{\text{HH}, \text{DE}}\) is that it conflates the effect of setting \(w_i = x\) on the \(i\)-th unit’s outcome, and the effect of setting \(w_i = x\) on the distribution of \(W_{-i}\). In particular, in completely randomized experiments, it’s possible to have \(\tau_{\text{HH}, \text{DE}} \neq 0\) even when \(Y_i(w_i = 1, w_{-i}) = Y_i(w_i = 0, w_{-i})\) for all units and all possible treatment assignments. In contrast, \(\tau_{\text{ADE}}\) as defined in Definition 1 has a robust causal interpretation as a direct effect.

Meanwhile, as discussed in the introduction, most available notions of indirect effects rely on explicit comparisons between two overall treatment assignment strategies. For example, Hudgens and Halloran [2008] and VanderWeele and Tchetgen Tchetgen [2011] propose a number of indirect effect estimands that, in the case of comparing two Bernoulli trials with randomization probabilities \(\pi\) and \(\pi'\), reduce to

\[
\tau_{\text{IE}}(\pi, \pi') = \frac{1}{n} \sum_{i=1}^n \left( \mathbb{E}_\pi \left[ Y_i(w_i = 0; W_{-i}) \right] - \mathbb{E}_{\pi'} \left[ Y_i(w_i = 0; W_{-i}) \right] \right).
\]

In the case of non-Bernoulli trials, there are a number of subtleties analogous to the ones noted above; see VanderWeele and Tchetgen Tchetgen [2011] for an in-depth discussion. From a practical perspective, \(\tau_{\text{IE}}(\pi, \pi')\) is an interesting quantity to consider if we can actually run many independent experiments that test different overall treatment. However, unlike \(\tau_{\text{ADE}}\), they do not enable a researcher to describe indirect effects in a single randomized study.

Another popular approach to capturing indirect effects is via the exposure mapping approach developed in Aronow and Samii [2017]. The main idea is to assume existence of
functions $h_i : \{0, 1\}^n \to \{1, \ldots, K\}$ such that potential outcomes $Y_i(w)$ only depend on $w$ via the compressed representation $h_i(w)$, i.e., $Y_i(w) = Y_i(w')$ whenever $h_i(w) = h_i(w')$; see also Karwa and Airoldi [2018], Leung [2020] and Sävje [2021] for further discussions and extensions. One can then consider estimators of averages of potential outcome types and define treatment effects in terms of their contrasts:

$$
\mu(k) = \frac{1}{n} \sum_{i=1}^{n} \mathbb{E}[Y_i \mid h_i(W_i) = k], \quad \tau(k, k') = \mu(k') - \mu(k), \quad 1 \leq k \neq k' \leq K.
$$

Definitions of this type are again conceptually attractive and sometimes enable us to very clearly express the answer to a natural policy question; see, e.g., Basse et al. [2019]. However, they again require the analyst to consider specific policy interventions to be able to even talk about indirect effects, and can also be unwieldy to use as the number of possible exposure types $K$ gets large.

Closest to the definition of $\tau_{AIE}$ is the average marginalized response of Aronow et al. [2020]. They consider a setting where treatments are assigned to points in a geographic space, and seek to estimate the average effect of treatment at an intervention point outcomes at points that are a distance $d$ away:

$$
\tau_{AMR}(d; \pi) = \frac{1}{n} \sum_{i=1}^{n} \sum_{j \in S_i(d)} \frac{\mathbb{E}_{\pi}[Y_j(w_i = 1; W_{-i}) - Y_j(w_i = 0; W_{-i})]}{|S_i(d)|}, \quad S_i(d) = \{j : \Delta(i, j) = d\},
$$

where $\Delta(i, j)$ measures the distance between points $i$ and $j$. This “circle average” bears resemblance to Definition 2 in the sense that both of them are marginalized over variation in $W_{-i}$ holding the treatment $W_i$ fixed. However, one key difference is the normalization factor $|S_i(d)|^{-1}$ used in $\tau_{AMR}$. Adding similar normalization to $\tau_{AIE}$ would invalidate Theorem 1.

### 4 Models for Interference

Our discussion so far has focused on an abstract specification where direct and indirect effects are defined via various (marginalized) contrasts between potential outcomes. Much of the existing applied work on causal inference under interference, however, has focused on simpler parametric specifications that, e.g., connect outcomes to treatments via a linear model. The purpose of this section is to examine our abstract, non-parametric definition of the indirect effect given in Definition 2, and to confirm that it still corresponds to an estimand one would want to interpret as an indirect effect once we restrict our attention to simpler parametric models. Below, we do so in 3 examples. Munro et al. [2021] provide an extended study of $\tau_{ADE}$ and $\tau_{AIE}$ in a marketplace model where interference arises via equilibrium price formation. The claimed expressions for $\tau_{ADE}$ and $\tau_{AIE}$ are derived in Appendix B.

**Example 1.** In studying the spillover effects of insurance training sessions on insurance purchase, Cai et al. [2015] work in terms of a network model: There is an edge matrix $E_{ij} \in \{0, 1\}$, such that $W_i$ can only affect $Y_i$ if the corresponding units are connected by an edge ($E_{ij} = 1$). They then consider a linear-in-means model parametrized in terms of this network. For our purpose, we focus on a simple variant of the model of Cai et al. [2015] considered in Leung [2020], where only the effects of ego’s treatment and the proportion of
treated neighbors are considered as covariates. This results in a linear model induced by the structural equation \(^1\)

\[
Y_i = \beta_1 + \beta_2 W_i + \beta_3 \frac{\sum_{j \neq i} E_{ij} W_j}{\sum_{j \neq i} E_{ij}} + \varepsilon_i, \quad \mathbb{E} [\varepsilon_i | W] = 0. \tag{6}
\]

In words, the probability of insurance purchase is modeled as a linear function of whether the farmer attends the insurance training sessions, and the proportion of friends who attend the session. Under this model, it can be shown that \(\tau_{ADE} = \beta_2\), and \(\tau_{AIE} = \beta_3\), under Assumption 1, i.e., the estimands from Definitions 1 and 2 map exactly to the parameters in the model (6) regardless of the experimental design.

**Example 2.** The model in Example 1 assumes the the \(i\)-th unit responds in the same way to treatment assigned to any of its neighbors. However, this restriction may be implausible in many areas; for example, in social networks, there is evidence that some ties are stronger than others, and that peer effects are larger along strong ties [Bakshy et al., 2012]. A natural generalization of Example 1 that allows for variable strength ties is to consider a saturated structural linear model

\[
Y_i = \alpha_i + \beta_i W_i + \sum_{j \neq i} \nu_{ij} W_j + \varepsilon_i, \quad \mathbb{E} [\varepsilon_i | W] = 0, \tag{7}
\]

which allows for both unit-specific direct and indirect effects. Here the individual parameters in this model are not identifiable; however, under (7), \(\tau_{ADE} = \frac{1}{n} \sum_{i=1}^n \beta_i\), and \(\tau_{AIE} = \frac{1}{n} \sum_{i=1}^n \sum_{j \neq i} \nu_{ij}\), i.e., our estimands can be understood as averages of the unit-level parameters (again regardless of the design).

**Example 3.** In studying the effect of persuasion campaigns or other types of messaging, it is common to assume that people respond most strongly if they get a communication directly addressed to them, but may also respond if a member of their neighborhood (or their household) gets a communication. This assumption can be formalized in terms of the following exposure mapping model: Each unit has 4 potential outcomes called “treated & exposed”, “treated”, “exposed” and “none”, such that

\[
Y_i = \begin{cases} 
Y_i(\text{treated & exposed}) & \text{if } W_i = 1 \text{ and } i \text{ has a treated neighbor}, \\
Y_i(\text{treated}) & \text{if } W_i = 1 \text{ but } i \text{ has no treated neighbors}, \\
Y_i(\text{exposed}) & \text{if } W_i = 0 \text{ but } i \text{ has a treated neighbor}, \\
Y_i(\text{none}) & \text{if } W_i = 0 \text{ and } i \text{ has no treated neighbors}.
\end{cases} \tag{8}
\]

Models of this type are considered by Sinclair et al. [2012] for studying voter mobilization, and by Basse and Feller [2018] and Basse et al. [2019] for studying anti-absenteeism interventions. Natural treatment effect parameters to consider following (3) include the average self-treatment and spillover effects

\[
\tau_{\text{SELF},1} = \frac{1}{n} \sum_{i=1}^n (Y_i(\text{treated & exposed}) - Y_i(\text{exposed})), \quad \tau_{\text{SELF},0} = \frac{1}{n} \sum_{i=1}^n (Y_i(\text{treated}) - Y_i(\text{none})),
\]

\[
\tau_{\text{SPILL},1} = \frac{1}{n} \sum_{i=1}^n (Y_i(\text{treated & exposed}) - Y_i(\text{treated})), \quad \tau_{\text{SPILL},0} = \frac{1}{n} \sum_{i=1}^n (Y_i(\text{exposed}) - Y_i(\text{none})).
\]

\(^1\)The relation (6) should be taken as a structural model, meaning that we can generate potential outcomes \(Y_i(w)\) in the sense of Assumption 1 by plugging candidate assignment vectors \(w\) into (6), i.e., \(Y_i(w) = \beta_1 + \beta_2 w_i + \beta_3 \frac{\sum_{j \neq i} E_{ij} w_j}{\sum_{j \neq i} E_{ij}} + \varepsilon_i\), for all \(i = 1, \ldots, n\) and \(w \in \{0, 1\}^n\).
Unlike the previous two examples, the connection between $\tau_{ADE}$ and $\tau_{AIE}$ to $\tau_{SELF}$ and $\tau_{SPILL}$ differs substantially across experimental designs, especially when the design introduces correlation between the units. For the purpose of illustration, we study it in a multi-stage completely randomized design considered in previous works [Sinclair et al., 2012, Basse and Feller, 2018, Basse et al., 2019]. In particular, we focus on the case where there are in total $n/m$ clusters of size $m$. On the first stage, $\rho \cdot n/m$ clusters are assigned to treatment, and $(1-\rho) \cdot n/m$ clusters are assigned to control; on the second stage, a single unit in each treated cluster is randomly chosen to be treated, and all the other units are assigned to control. We can then calculate the marginal distribution of $W_i$ and obtain

$$
\tau_{ADE} = \left( \rho - \frac{\rho}{m} \right) \tau_{SELF,1} + \left( 1 - \rho + \frac{\rho}{m} \right) \tau_{SELF,0},
$$

$$
\tau_{AIE} = (m - 1) \left( \frac{\rho}{m} \tau_{SPILL,1} + \left( 1 - \rho + \frac{\rho}{m} \right) \tau_{SPILL,0} \right).
$$

Therefore, our estimands can be regarded as weighted averages of the treatment effect parameters. Moreover, when the cluster size $m$ is large, $\tau_{ADE}$ is approximately the average of $\tau_{SELF,1}$ and $\tau_{SELF,0}$, weighted by the assignment probability during the first stage, while $\tau_{AIE}$ is approximately $\tau_{SPILL,0}$ times the probability of being assigned to the control group during the first stage, and the factor $m - 1$ simply accounts for the fact that, any treatment will spread spillover effects to $m - 1$ neighbors.

5 Discussion

There are many treatment effect estimation problems in which interference is present and needs to be accounted for, and in which indirect effects are of considerable scientific interest. In the existing literature, indirect effects are usually defined by either comparing outcomes across experiments with different randomization policies (Section 3), or by writing down a parametric model for interference (Section 4). In this paper, we contribute by proposing an alternative average indirect effect metric that can be non-parametrically defined using a single Bernoulli-randomized trial, thus providing practitioners with a starting point for investigating indirect effects that does not require reasoning in terms of additional data sources they do not currently have access to (or in terms of more elaborate models). In Appendix A, we further show that both $\tau_{ADE}$ and $\tau_{AIE}$ always admit unbiased estimators in Bernoulli experiments.

One challenge in defining average effect estimators in the presence of interference, however, is that our estimands will in general depend on the choice of Bernoulli-randomization probabilities $\pi$. In Figure 1, we illustrate this phenomenon by plotting $\tau_{ADE}(\pi)$ and $\tau_{AIE}(\pi)$ as a function of $\pi$ in the following three structural models (while assuming constant treat-
Figure 1: Illustration of $\tau_{ADE}$, $\tau_{AIE}$, $\tau_{INF}$ and $E_{\pi}[Y_i]$: The purple curve corresponds to the expected potential outcome $E_{\pi}[Y_i]$. The slope of the line segments on the purple curve (derivative of $E_{\pi}[Y_i]$) is the same as the value of the blue curve ($\tau_{INF}$). Theorem 1 establishes that $\tau_{INF} = \tau_{ADE} + \tau_{AIE}$. In the plots, the blue curve ($\tau_{INF}$) corresponds to the sum of the red curve ($\tau_{ADE}$) and the green curve ($\tau_{AIE}$). We consider the three settings listed in (10) where, in all cases, we assume constant treatment assignment probabilities $\pi_i = \pi_0$ and take the number of neighbors to be $\sum_{i \neq j} E_{ij} = 100$.

where in each case $E[\varepsilon_i | W] = 0$. Here, qualitatively, Setting 1 resembles the one considered by Cai et al. [2015] and Leung [2020] as discussed in Example 1 from Section 4. Setting 2 exhibits a type of "herd immunity" where units are more sensitive to treatment when most of their neighbors are untreated, while Setting 3 has complicated non-linear interference effects. We then see that, in Setting 1, $\tau_{ADE}(\pi)$ and $\tau_{AIE}(\pi)$ do not vary with $\pi$, but in Settings 2 and 3 they do—and may even change signs.

This potential dependence of $\tau_{ADE}(\pi)$ and $\tau_{AIE}(\pi)$ on $\pi$ is something that any practitioner using these estimands needs to be aware of. However, we believe such dependence to be largely unavoidable when seeking to define non-parametric estimands under the generality considered here. For example, when estimating indirect effects of immunization in a
population where roughly 30% of units have been immunized, definitions of the type developed here could be used to support non-parametric analysis of indirect effects. Now, one should recognize that any such effects would be “local” to the current overall immunization rate at 30%, and would likely differ from indirect effects we would measure at a 50% overall immunization rate. However, it seems unlikely that one could use data from a population with 30% immunization rate to non-parametrically estimate average outcomes we might observe at a 50%; rather, to do so, one would need to either posit a much more elaborate model for how infections spread or collect different data.

Finally, we note that \( \tau_{AIE}(\pi) \) as given in Definition 2 involves normalization by the sample size \( n \), not by the total number of summands \( n(n-1) \), and one can ask whether this scaling is the most natural one in practical settings. In Section 4 we found that, in a number of popular models, \( \tau_{AIE}(\pi) \) coincides with interesting and interpretable quantities and converges as the number of units \( n \) goes to infinity. In other models, however, our \( 1/n \) scaling may not be the best choice. For example, if we have a data-generating distribution with \( Y_i(w) = (\sum_{j=1}^{n} w_j - n\pi) / \sqrt{n\pi(1-\pi)} \), the observed outcomes \( Y_i \) will have a standard normal marginal distribution, but \( \tau_{AIE} = \sqrt{n} \) diverges. One should also note, however, that in this example \( 1/n \sum_{i=1}^{n} Y_i \) does not concentrate.

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A Unbiased Estimation

The main focus of this paper has been on establishing definitions of average treatment effect metrics under interference, and on verifying their interpretability under various modeling assumptions. But in order for these definitions to be useful in applied statistical work, we of course also need these metrics to be readily identifiable and estimable under flexible conditions. A comprehensive discussion of treatment effect estimation under interference—including distributional results and efficiency theory—is beyond the scope of this paper. However, as one result in this direction, we note here that unbiased estimates of the average direct and indirect effects are always available in Bernoulli-randomized experiments via the Horvitz-Thompson construction.

As above, the case of the average direct effect is already well understood in the literature. The Horvitz-Thompson estimator for $\tau_{ADE}(\pi)$ is [Såvje et al., 2021]

$$\hat{\tau}_{ADE}(\pi) = \frac{1}{n} \sum_{i=1}^{n} \left( \frac{W_i Y_i}{\pi_i} - \frac{(1-W_i)Y_i}{1-\pi_i} \right). \quad (11)$$

Furthermore, as is implicitly established in the technical appendix of Såvje et al. [2021], this estimator is unbiased in Bernoulli-randomized experiments, i.e., $E[\hat{\tau}_{ADE}(\pi)] = \tau_{ADE}(\pi)$.

Next, in discussing estimators for $\tau_{AIE}(\pi)$, we will work under a network interference model whereby the analyst has access to an interference graph $E_{ij} \in \{0, 1\}$ and knows that the $j$-th unit’s potential outcomes are unaffected by the treatment given to the $i$-th unit whenever $E_{ij} = 0$, i.e.,

$$Y_j(w_i = 0; w_{-i}) = Y_j(w_i = 1; w_{-i}) \text{ whenever } E_{ij} = 0, \quad (12)$$

for all $i, j = 1, \ldots, n$ and $w_{-i} \in 0, 1^{n-1}$. This type of assumption is not required in principle; and in particular, the condition (12) is vacuous if $E_{ij} = 1$ for all pairs $(i, j)$, i.e., if we assume that any treatment can affect any outcome. However, assumptions of this type are ubiquitous in practice (see, e.g., Examples 1 and 3 considered in Section 4), and when we have access to a sparse interference graph they can considerably improve the precision with which we can estimate indirect effects.

Given this setup, the Horvitz-Thompson estimator for $\tau_{AIE}(\pi)$ is

$$\hat{\tau}_{AIE}(\pi) = \frac{1}{n} \sum_{i=1}^{n} \sum_{\{j \neq i: E_{ij} = 1\}} \left( \frac{W_i Y_j}{\pi_i} - \frac{(1-W_i)Y_j}{1-\pi_i} \right). \quad (13)$$

Formally, this estimator looks like $\hat{\tau}_{ADE}(\pi)$; except now we are measuring associations between the $i$-th unit’s treatment and the $j$-th unit’s outcome, for $i \neq j$. And, as in the case of the direct effect, this estimator in unbiased in Bernoulli-randomized experiments.

Theorem 2. Under Assumption 1 and in a Bernoulli trial, the Horvitz-Thompson estimator (13) is unbiased for the average indirect effect, $E[\hat{\tau}_{AIE}(\pi)] = \tau_{AIE}(\pi)$.

Finally, we again emphasize that although results on unbiased estimation are helpful, they do not provide a complete picture of what good estimators for $\tau_{ADE}(\pi)$ and $\tau_{AIE}(\pi)$ should look like, and what kinds of guarantees we should expect. The direct effect estimator (11) is further studied by Såvje et al. [2021], who provide bounds on its mean-squared error under a moderately sparse network interference model as in (12); roughly speaking, they...
B Proofs

B.1 Proof of Theorem 1

We start with a slightly more formal form of the infinitesimal policy effect:

\[
\tau_{\text{INF}}(\pi) = \sum_{k=1}^{n} \frac{\partial}{\partial \pi_k} \left\{ \frac{1}{n} \sum_{i=1}^{n} E_{\pi'}[Y_i] \right\}_{\pi'=\pi},
\]

i.e., we take derivative with respect to \( \pi' \) and evaluate at \( \pi \). For index \( i \), we can rewrite \( E[Y_i] \) in terms of the potential outcomes:

\[
E_{\pi'}[Y_i] = \sum_{w_{-k} w_k \in \{0, 1\}} Y_i(w_k; w_{-k}) P_{\pi'_{-k}}[W_{-k} = w_{-k}] P_{\pi_k}[W_k = w_k]
\]

\[
= \sum_{w_{-k}} Y_i(w_k; w_{-k}) P_{\pi'_{-k}}[W_{-k} = w_{-k}] (w_k \pi'_k + (1 - w_k)(1 - \pi'_k)).
\]

The dependency of this term on \( \pi'_k \) is clear in this form. Taking a derivative with respect to \( \pi'_k \) and evaluating at \( \pi_k \) yields

\[
\frac{\partial}{\partial \pi_k} E_{\pi'}[Y_i] \big|_{\pi_k=\pi} = \sum_{w_{-k}} (Y_i(w_k = 1; w_{-k}) - Y_i(w_k = 0; w_{-k})) P_{\pi_{-k}}[W_{-k} = w_{-k}]
\]

\[
= E_{\pi} [Y_i(w_k = 1; W_{-k}) - Y_i(w_k = 0; W_{-k})].
\]

If we sum over the index \( i \) and \( k \) and multiply the term by \( 1/n \), we get

\[
\sum_{k=1}^{n} \frac{\partial}{\partial \pi_k} \left\{ \frac{1}{n} \sum_{i=1}^{n} E_{\pi'}[Y_i] \right\}_{\pi_k=\pi} = \frac{1}{n} \sum_{k=1}^{n} \sum_{i=1}^{n} E_{\pi} [Y_i(w_k = 1; W_{-k}) - Y_i(w_k = 0; W_{-k})].
\]

This can be further decomposed into two terms: summation over \( i = k \) and \( i \neq k \).

\[
\sum_{k=1}^{n} \frac{\partial}{\partial \pi_k} \left\{ \frac{1}{n} \sum_{i=1}^{n} E_{\pi'}[Y_i] \right\}_{\pi_k=\pi} = \frac{1}{n} \sum_{k=1}^{n} E_{\pi} [Y_k(w_k = 1; W_{-k}) - Y_k(w_k = 0; W_{-k})] +
\]

\[
\frac{1}{n} \sum_{k=1}^{n} \sum_{i \neq k} E_{\pi} [Y_i(w_k = 1; W_{-k}) - Y_i(w_k = 0; W_{-k})].
\]
The first term matches the definition of $\tau_{ADE}(\pi)$ and the second term matches the definition of $\tau_{AIE}(\pi)$. Thus

$$\tau_{INF}(\pi) = \sum_{k=1}^{n} \frac{\partial}{\partial \pi_k} \left\{ \frac{1}{n} \sum_{i=1}^{n} \mathbb{E}_{\pi} [Y_i] \right\}_{\pi_k=\pi} = \tau_{ADE}(\pi) + \tau_{AIE}(\pi).$$

\section*{B.2 Proof of Theorem 2}

For each pair of indices $i, j$, we can write $\mathbb{E}_{\pi} \left[ \frac{W_i Y_j}{\pi_i} - \frac{(1-W_i)Y_j}{1-\pi_i} \right]$ as

$$\mathbb{E}_{\pi} \left[ \frac{W_i Y_i}{\pi_i} \right] - \mathbb{E}_{\pi} \left[ \frac{(1-W_i)Y_i}{1-\pi_i} \right] = \mathbb{E}_{\pi} \left[ \frac{W_i Y_j(w_i = 1; W_{-i})}{\pi_i} \right] - \mathbb{E}_{\pi} \left[ \frac{(1-W_i)Y_j(w_i = 0; W_{-i})}{1-\pi_i} \right]
= \frac{W_i}{\pi_i} \mathbb{E}_{\pi} [Y_j(w_i = 1; W_{-i})] - \frac{1-W_i}{1-\pi_i} \mathbb{E}_{\pi} [Y_j(w_i = 0; W_{-i})]
= \mathbb{E}_{\pi} [Y_j(w_i = 1; W_{-i}) - Y_j(w_i = 0; W_{-i})].$$

Thus summing over $i$ and $j$, we get

$$\mathbb{E}_{\pi} [\hat{\tau}_{AIE}(\pi)] = \frac{1}{n} \sum_{i=1}^{n} \sum_{\{j \neq i, E_{ij}=1\}} \left( \mathbb{E}_{\pi} \left[ \frac{W_i Y_j}{\pi_i} - \frac{(1-W_i)Y_j}{1-\pi_i} \right] \right)
= \frac{1}{n} \sum_{i=1}^{n} \sum_{\{j \neq i, E_{ij}=1\}} \mathbb{E}_{\pi} [Y_j(w_i = 1; W_{-i}) - Y_j(w_i = 0; W_{-i})].$$

When there is no edge connecting $i$ and $j$, the value of $Y_j$ does not depend on $w_i$, hence $Y_j(w_i = 1; W_{-i}) = Y_j(w_i = 0; W_{-i})$. Hence we can rewrite the above term:

$$\mathbb{E}_{\pi} [\hat{\tau}_{AIE}(\pi)] = \frac{1}{n} \sum_{i=1}^{n} \sum_{j \neq i} \mathbb{E}_{\pi} [Y_j(w_i = 1; W_{-i}) - Y_j(w_i = 0; W_{-i})] = \tau_{AIE}(\pi).$$

\section*{B.3 Derivations for Example 1}

It’s clear from (6) that $Y_i(w_i = 1; W_{-i}) - Y_i(w_i = 0; W_{-i}) = \beta_2$ and $Y_j(w_i = 1; W_{-i}) - Y_j(w_i = 0; W_{-i}) = \beta_3 E_{ij} / \sum_{j \neq i} E_{ij}$. Hence

$$\tau_{ADE} = \frac{1}{n} \sum_{i=1}^{n} \mathbb{E}_{\pi} [Y_i(w_i = 1; W_{-i}) - Y_i(w_i = 0; W_{-i})] = \beta_2,$$

$$\tau_{AIE} = \frac{1}{n} \sum_{i=1}^{n} \sum_{j \neq i} \mathbb{E}_{\pi} [Y_j(w_i = 1; W_{-i}) - Y_j(w_i = 0; W_{-i})] = \frac{1}{n} \sum_{i=1}^{n} \sum_{j \neq i} \left( \beta_3 E_{ij} / \sum_{j \neq i} E_{ij} \right) = \beta_3.$$
B.4 Derivations for Example 2

Model (7) implies that
\[ Y_i(w_i = 1; W_{-i}) - Y_i(w_i = 0; W_{-i}) = \beta_i \] and
\[ Y_j(w_i = 1; W_{-i}) - Y_j(w_i = 0; W_{-i}) = \nu_{ij}. \]

Hence
\[ \tau_{ADE} = \frac{1}{n} \sum_{i=1}^{n} \beta_i, \]
\[ \tau_{AIE} = \frac{1}{n} \sum_{i=1}^{n} \sum_{j \neq i} E[ Y_j(w_i = 1; W_{-i}) - Y_j(w_i = 0; W_{-i})] = \frac{1}{n} \sum_{i=1}^{n} \sum_{j \neq i} \nu_{ij}. \]

B.5 Derivations for Example 3

In this clustered setup, the treatment assignment of units outside of the cluster has no effect on the unit’s observed outcome. With a slight abuse of notation, we use \( W_{-i} \) to denote the treatment vector of all the units in the same cluster as unit \( i \), except the unit itself. To start with, we find the distribution of \( W_{-i} \) marginalized over \( W_i \) under this design, where
\[ P(W_{-i} = w) = \begin{cases} 1 - \rho + \frac{\rho}{m}, & \text{if } w = (0, \ldots, 0) \\ \rho, & \text{if } w \in \{e_1, \ldots, e_{m-1}\} \\ 0, & \text{otherwise,} \end{cases} \]
where \( e_j \in \mathbb{R}^{m-1} \) denotes the vector with a 1 in the \( j \)th coordinate and 0’s elsewhere. It then follows directly from the definitions that
\[ \tau_{ADE}(\pi) = \frac{1}{n} \sum_{i=1}^{n} E[ Y_i(w_i = 1; W_{-i}) - Y_i(w_i = 0; W_{-i})] \]
\[ = \frac{1}{n} \sum_{i=1}^{n} \left( \left( \rho - \frac{\rho}{m} \right) Y_i(\text{treated & exposed}) + \left( 1 - \rho + \frac{\rho}{m} \right) Y_i(\text{treated}) \right) - \left( \rho - \frac{\rho}{m} \right) Y_i(\text{exposed}) - \left( 1 - \rho + \frac{\rho}{m} \right) Y_i(\text{none}) \]
\[ = \left( \rho - \frac{\rho}{m} \right) \tau_{SELF,1} + \left( 1 - \rho + \frac{\rho}{m} \right) \tau_{SELF,0}, \]
\[ \tau_{AIE}(\pi) = \frac{1}{n} \sum_{i=1}^{n} \sum_{j \neq i} E[ Y_j(w_i = 1; W_{-i}) - Y_j(w_i = 0; W_{-i})] \]
\[ = \frac{1}{n} \sum_{i=1}^{n} (m-1) \left( \frac{\rho}{m} Y_j(\text{treated & exposed}) + \left( 1 - \rho \right) Y_j(\text{exposed}) \right) - \frac{\rho}{m} Y_j(\text{treated}) - \frac{\rho}{m} Y_j(\text{exposed}) - \left( 1 - \rho + \frac{\rho}{m} \right) Y_j(\text{none}) \]
\[ = (m-1) \left( \frac{\rho}{m} \tau_{\text{SPILL,1}} + \left( 1 - \rho + \frac{\rho}{m} \right) \tau_{\text{SPILL,0}} \right). \]