

TODO

STAT 548 Qualifying Paper

Kenny Chiu

December 26, 2021

**Abstract.** TODO

## 1 Introduction

## 2 Notation

Throughout this report, we closely follow the notation used by Forastiere et al. (2021). Let  $G = (\mathcal{N}, \mathcal{E})$  be an undirected network where  $\mathcal{N}$  is a set of  $N$  units (nodes) and  $\mathcal{E}$  is a set of edges  $(i, j)$ . A partition  $(i, \mathcal{N}_i, \mathcal{N}_{-i})$  of  $\mathcal{N}$  describes unit  $i$ 's neighbourhood  $\mathcal{N}_i$  (the set of  $N_i$  units connected to unit  $i$ ) and the set  $\mathcal{N}_{-i}$  of all other units that are not  $i$  and are not in  $\mathcal{N}_i$ . Let  $Z_i \in \{0, 1\}$  be the treatment assigned to unit  $i$  and  $Y_i \in \mathcal{Y}$  the observed outcome of unit  $i$ . Denote the treatment and outcome vector for the population  $\mathcal{N}$  as  $\mathbf{Z}$  and  $\mathbf{Y}$ , respectively, and the corresponding vectors for partition  $(i, \mathcal{N}_i, \mathcal{N}_{-i})$  as  $(Z_i, \mathbf{Z}_{\mathcal{N}_i}, \mathbf{Z}_{\mathcal{N}_{-i}})$  and  $(Y_i, \mathbf{Y}_{\mathcal{N}_i}, \mathbf{Y}_{\mathcal{N}_{-i}})$ . Let  $G_i = g_i(\mathbf{Z}_{\mathcal{N}_i}) \in \mathcal{G}_i$  be some known and well-specified summary  $g_i$  of the treatments in unit  $i$ 's neighbourhood, and denote the vector of neighbourhood treatments for the population as  $\mathbf{G}$ . Depending on the size of a unit's neighbourhood, the space  $\mathcal{G}_i$  may differ between units. Let  $V_g = \{i \in \mathcal{N} : g \in \mathcal{G}_i\}$  denote the subset containing  $v_g$  units that have  $g$  as a possible value for the neighbourhood treatment. Let  $\mathbf{X}_i \in \mathcal{X}$  be a vector of covariates for unit  $i$  that partitions into individual-level characteristics  $\mathbf{X}_i^{\text{ind}} \in \mathcal{X}^{\text{ind}}$  and neighbourhood-level/aggregated individual-level characteristics  $\mathbf{X}_i^{\text{neigh}} \in \mathcal{X}^{\text{neigh}}$ .

## 3 Proposed methodology in context of the literature

TODO In this section, we explain the method proposed by Forastiere et al. (2021) and discuss its relevance in the context of the related literature. We also discuss its advantages over other methods used in similar contexts.

### 3.1 Setting, objective and method

Forastiere et al. (2021) examine the problem of performing causal inference of treatment effects from observational network data under interference. The setting is challenging for causal inference because

1. the assignment mechanism of treatments is unknown with observational data and so estimated effects may be non-causal in the presence of unmeasured confounders, and
2. naive inference methods that ignore interference may produce biased estimates.

In the randomized study literature, these issues can generally be dealt with by designing the study in such a way that the influence of confounders is minimized and that inference methods that account for interference can be used (e.g., Saveski et al., 2017; Doudchenko et al., 2020; Jagadeesan et al., 2020; Imai et al., 2021). In the observational setting, these considerations need to be addressed by the inference method in the analysis phase. Forastiere et al. consider a setting involving a binary treatment (e.g., intervention and control) and where the interference on a unit is limited to that of its immediate neighbouring units. They propose a method to estimate the causal treatment and spillover (interference) effects that yield unbiased estimates under certain assumptions.

Under the potential outcome framework, the general procedure for estimating effects is to match units into sets based on covariate values, compute the effect contrast within each matched set, and estimate the effect by the (weighted) average of the contrasts across sets. However, matching may be difficult when the space of possible covariate values is large or if there are many covariates. Forastiere et al. (2021) address this problem in their proposed method by instead matching on a defined joint propensity score  $\psi(z; g; x)$  that factorizes into a neighbourhood propensity score  $\lambda(g; z; \mathbf{x}^g)$  (probability of being exposed to neighbourhood treatment  $g$  given individual treatment  $z$  and relevant covariates  $\mathbf{x}^g$ ) and an individual propensity score  $\phi(z; \mathbf{x}^z)$  (probability of being assigned treatment  $z$  given relevant covariates  $\mathbf{x}^z$ ). Note that  $\mathbf{X}^g$  and  $\mathbf{X}^z$  do not necessarily correspond to  $\mathbf{X}^{\text{neigh}}$  and  $\mathbf{X}^{\text{ind}}$ , respectively, and as they may not be disjoint. The steps for their propensity-based method are as follows:

1. **Subclassify units.**

- (a) Fit a logistic regression model on the individual treatments  $Z_i$  given covariates  $\mathbf{X}_i^z$ , and use the model to predict the individual propensity scores  $\phi(1; \mathbf{X}_i^z)$ .
- (b) Partition the units into  $J$  subclasses  $B_j$ ,  $j \in \{1, \dots, J\}$ , based on similar estimated individual propensity scores  $\hat{\phi}(1; \mathbf{X}_i^z)$  and such that each subclass is approximately balanced in the number of treated and untreated units.

2. **Estimate potential outcomes.** Let  $B_j^g = V_g \cap B_j$ . For each subclass  $B_j$ :

- (a) Fit some regression model on the neighbourhood treatments  $G_i$  given the individual treatments  $Z_i$  and covariates  $\mathbf{X}_i^g$ , and use the model to predict the neighbourhood propensity scores  $\lambda(g; z; \mathbf{X}_i^g)$ .
- (b) Fit some regression model on the potential outcomes  $Y_i(z, g)$  given the estimated neighbourhood propensity scores  $\hat{\lambda}(g; z; \mathbf{X}_i^g)$ .
- (c) Estimate the dose-response function by averaging over the estimated potential outcomes for a particular level of the joint treatment, i.e.,

$$\hat{\mu}_j(z, g; V_g) = \frac{\sum_{i \in B_j^g} \hat{Y}_i(z, g)}{|B_j^g|}.$$

3. **Estimate the average dose-response function (ADRF)**  $\mu(z, g; V_g) = \mathbb{E}[Y_i(z, g) | i \in V_g]$  for a particular level of the joint treatment by taking the weighted average of the estimated dose-response functions over the subclasses, i.e.,

$$\hat{\mu}(z, g; V_g) = \sum_{j=1}^J \hat{\mu}_j(z, g; V_g) \left( \frac{|B_j^g|}{v_g} \right).$$

4. **Estimate** the treatment effects  $\tau(g)$ , overall treatment effect  $\tau$ , spillover effects  $\delta(g; z)$ , and overall spillover effects  $\Delta(z)$  by

$$\begin{aligned} \hat{\tau}(g) &= \hat{\mu}(1, g; V_g) - \hat{\mu}(0, g; V_g), & \hat{\tau} &= \sum_{g \in \mathcal{G}} \hat{\tau}(g) \mathbb{P}(G_i = g), \\ \hat{\delta}(g; z) &= \hat{\mu}(z, g; V_g) - \hat{\mu}(z, 0; V_g), & \hat{\Delta}(z) &= \sum_{g \in \mathcal{G}} \hat{\delta}(g; z) \mathbb{P}(G_i = g). \end{aligned}$$

Forastiere et al. (2021) show that their estimators for the treatment and spillover effects are unbiased under three assumptions, the first two of which form the Stable Unit Treatment on Neighbourhood Value Assumption (SUTNVA, a generalization of SUTVA that relaxes the no interference assumption to allow interference of immediate neighbours) and the third being an unconfoundedness assumption that says the treatment assignment mechanism is conditionally independent of the outcomes for the given set of covariates.

## 3.2 Comparison to the literature

The literature that examines the similar problem of causal inference in observational data under general forms of interference is still relatively new. We summarize the common approaches in this literature and discuss how the work by Forastiere et al. (2021) fits in. We also briefly highlight other work in the related literature.

### 3.2.1 Other approaches under the same context

As noted by Forastiere et al., the majority of the works dealing with the same context involve either inverse probability-weighted (IPW) estimators (Liu et al., 2016) or targeted maximum likelihood estimators (TMLE) (van der Laan, 2014; Ogburn et al., 2017; Sofrygin & van der Laan, 2017) for the causal treatment effect. The main advantage of the method proposed by Forastiere et al. (2021) over these two approaches is the weaker assumptions that it requires.

IPW estimators are weighted averages of the outcomes where the weights are defined with respect to a hypothetical allocation strategy (an assumed distribution over the neighbouring treatments) and a known or correctly modelled generalized propensity score. The Bernoulli allocation strategy (Tchetgen & VanderWeele, 2012) is commonly used, which assumes that each unit in the neighbourhood is treated independently with probability  $\alpha$  and that a unit’s assignment is independent of its neighbours’ assignment. This assumption rules out homophily—the tendency for units with similar characteristics to form ties—which is generally a strong and unrealistic assumption to make (Shalizi & Thomas, 2011). In contrast, the estimators proposed by Forastiere et al. (2021) only use the observed neighbourhood treatments, and therefore no assumptions that explicitly rule out homophily are made (though the issue may still manifest as an unmeasured confounder if the unconfoundedness assumption does not hold for the given set of covariates). Both the IPW estimators and the estimators proposed by Forastiere et al. (2021) rely on being able to correctly model the joint propensity score.

TMLEs are obtained by maximizing the likelihood of the outcomes defined on a structural equation model. Similar to the IPW estimators, TMLE typically involves a randomization assumption (van der Laan, 2014) on the model where the conditional joint distribution of the treatment assignments factorizes into independent conditionals given the covariates for all units, and similarly for the conditional joint distribution of the outcomes given the covariates and the treatment assignments. In comparison, the unconfoundedness assumption in the method proposed by Forastiere et al. (2021) makes a weaker assumption where the outcome and treatment assignment of each unit is conditionally independent given only the covariates of that unit. The significance of these assumptions again circle back to the argument of disregarding homophily and/or other venues of confounding. It is notable that extensions of TMLE that allow for limited forms of homophily were later introduced (Ogburn et al., 2017).

More recently, approaches that can be described as extensions of Forastiere et al. (2021)’s method have been proposed. Jackson et al. (2020) proposed estimators based on propensity score matching but which also explicitly account for homophily by modeling neighbourhood treatment assignments as an incomplete information game. Sánchez-Becerra (2021) questioned the justification of the unconfoundedness assumption with respect to the constructed propensity score (which may be high-dimensional and challenging to estimate accurately), and proposed a two-step method where a defined network propensity score is first estimated and then used as inverse weights to match units.

### 3.2.2 Other contexts

We briefly highlight other works in the literature that consider a slightly different setting of the causal inference problem in observational data under interference. The difference in settings makes it difficult to directly compare the proposed approaches to that of Forastiere et al. (2021).

A large body of the literature examine the inference problem under the assumption of partial interference where units are partitioned into groups with no spillover effects between groups. The focus on partial interference settings seems to be primarily due to momentum of earlier works (e.g., Sobel, 2006; Hudgens & Halloran, 2008) that looked at causal inference in randomized studies with interference, in which group-randomization tends to be more practical. Examples of recent work that assume partial interference include the work by Liu et al. (2019), Barkley et al. (2020), and Qu et al. (2021). IPW estimators are commonly used in the partial interference setting as they were originally introduced for grouped observational data (Tchetgen & VanderWeele, 2012).

A small number of works consider more specific and niche contexts. For example, Toulis et al. (2018) explore the problem of treatment entanglement (where treatment assignments are assumed to satisfy certain restrictions) and proposes a propensity score-based estimator. Zigler and Papadogeorgou (2021) focus on the problem of bipartite causal inference with interference (where the treatment is applied to one unit and the outcome is measured on another) and proposes a IPW estimator.

## 4 Potential bias of naive estimator when unconfoundedness holds

**TODO**In this section, we describe a simple example that illustrates the relevance of Theorem 2.A, Corollary 2 and Corollary 3 in the paper by Forastiere et al. (2021). Specifically, we show how given a simple network under certain assumptions, an unbiased naive estimator for the treatment effect that assumes SUTVA may be biased depending on the exact treatment assignment mechanism.

Consider some undirected network  $G = (\mathcal{N}, \mathcal{E})$  where every unit is paired (has an edge) with exactly one other unit. For simplicity, we index a unit by  $i \in \mathbb{N}$  for the pair and  $j \in \{0, 1\}$  for the unit within a pair. Denote  $Z_{ij} \in \{0, 1\}$  as the treatment assignment of unit  $j$  in pair  $i$ . Let the neighbourhood treatment  $G_{ij}$  be whether a unit's paired counterpart is treated. Therefore,  $G_{ij} = \{0, 1\}$  for all units in the network and  $V_g = \mathcal{N}$  for all  $g \in \{0, 1\}$ . For convenience of notation, we drop the dependence on  $V_g$  where applicable. Let  $\mathbf{X}_{ij} = X_{ij} \in \{0, 1\}$  be some covariate available for each unit, and assume that the covariates of the units in the network are generated independently with  $\mathbb{P}(X_{ij} = 1) = \mathbb{P}(X_{ij} = 0) = 0.5$ . Other details, such as the space of outcomes  $\mathcal{Y}$  and the size of  $\mathcal{N}$ , are assumed but left unspecified due to being irrelevant for the discussion.

**TODO:** make assignment probabilistic (Imbens & Rubin, 2015). We examine the network under two settings. In the first setting, suppose that the covariate  $X_{ij}$  exactly determines the treatment status of a unit, i.e.,  $Z_{ij} = X_{ij}$ . Because  $X_{ij}$  are generated independently, it follows that  $Z_{ij}$  and  $G_{ij}$  are conditionally independent given  $X_{ij}$  in this setting (they are independent unconditionally regardless of  $X_{ij}$ ). In the second setting, suppose that the treatment assignment mechanism follows

$$Z_{ij} = \begin{cases} 1 & \text{if } X_{i1} = X_{i2} \\ 0 & \text{otherwise} \end{cases}.$$

This corresponds to a homophily situation where the units in a pair are treated only if they share the same value of the covariate. Thus,  $Z_{ij}$  and  $G_{ij}$  are not conditionally independent given  $X_{ij}$  in this setting as a unit can only have values  $(Z_{ij} = 1, G_{ij} = 1)$  or  $(Z_{ij} = 0, G_{ij} = 0)$ . In both settings, we further assume that Assumption 1 (no multiple versions of treatment), Assumption 2 (neighbourhood interference), and Assumption 3 (unconfoundedness) of Forastiere et al. (2021) hold. Hence, the assumptions of Corollary 1 are satisfied in the first setting, and the assumptions of Corollary 2 are satisfied in the second setting.

## 4.1 Setting 1: conditionally independent individual and neighbourhood treatments

In our first setting, the individual treatment  $Z_{ij}$  and neighbourhood treatment  $G_{ij}$  are conditionally independent given  $X_{ij}$ . **TODO**example? Then by Corollary 1, an estimator that is unbiased under SUTVA will still be unbiased under the assumptions of our setting.

The treatment effects  $\tau(g)$  and overall treatment effect  $\tau$  in our example are given by

$$\begin{aligned}\tau(0) &= \mu(1, 0) - \mu(0, 0) , \\ \tau(1) &= \mu(1, 1) - \mu(0, 1) , \\ \tau &= \tau(0)\mathbb{P}(G_{ij} = 0) + \tau(1)\mathbb{P}(G_{ij} = 1) \\ &= 0.5(\mu(1, 0) + \mu(1, 1) - \mu(0, 0) - \mu(0, 1)) .\end{aligned}$$

The naive estimator  $\tau_X^{\text{obs}}$  that assumes SUTVA estimates the quantity

$$\begin{aligned}\tau_X^{\text{obs}} &= (\mathbb{E}[Y_{ij}|Z_{ij} = 1, X_{ij} = 0] - \mathbb{E}[Y_{ij}|Z_{ij} = 0, X_{ij} = 0])\mathbb{P}(X_{ij} = 0) \\ &\quad + (\mathbb{E}[Y_{ij}|Z_{ij} = 1, X_{ij} = 1] - \mathbb{E}[Y_{ij}|Z_{ij} = 0, X_{ij} = 1])\mathbb{P}(X_{ij} = 1) \\ &= \mathbb{E}[Y_{ij}|Z_{ij} = 1] - \mathbb{E}[Y_{ij}|Z_{ij} = 0] \\ &= (\mathbb{E}[Y_{ij}|Z_{ij} = 1, G_{ij} = 0] - \mathbb{E}[Y_{ij}|Z_{ij} = 0, G_{ij} = 0])\mathbb{P}(G_{ij} = 0) \\ &\quad + (\mathbb{E}[Y_{ij}|Z_{ij} = 1, G_{ij} = 1] - \mathbb{E}[Y_{ij}|Z_{ij} = 0, G_{ij} = 1])\mathbb{P}(G_{ij} = 1) \\ &= 0.5(\mu(1, 0) + \mu(1, 1) - \mu(0, 0) - \mu(0, 1))\end{aligned}$$

where the third equality as  $X_{i1} \perp\!\!\!\perp X_{i2}$  (and so  $Z_{ij} \perp\!\!\!\perp G_{ij}$ ) and the fourth equality from Assumptions 1 and 2. Note that the conditional expectations where The second equality follows under the “super-population” perspective (Forastiere et al., 2021) where expectations should be interpreted as the exp **TODO**(impossible event?) The above derivations suggest that  $\tau_X^{\text{obs}} - \tau = 0$ , and therefore an unbiased estimator of the naive effect estimator is also unbiased for the treatment effect in this setting.

## 4.2 Setting 2: correlated individual and neighbourhood treatments

- Setting 2:

$$\begin{aligned}\tau(1) &= \mu(1, 1) \\ \tau(0) &= -\mu(0, 0) \\ \tau &= 0.5(\mu(1, 1) - \mu(0, 0))\end{aligned}$$

$$\begin{aligned}\tau_{X^*}^{\text{obs}} &= 0.5(\mathbb{E}[Y|Z = 1, X = 0] - \mathbb{E}[Y|Z = 0, X = 0] + \mathbb{E}[Y|Z = 1, X = 1] - \mathbb{E}[Y|Z = 0, X = 1]) \\ &= \mathbb{E}[Y|Z = 1] - \mathbb{E}[Y|Z = 0] \\ &= \mathbb{E}[Y|Z = 1, G = 1]P(G = 1|Z = 1) + \mathbb{E}[Y|Z = 1, G = 0]P(G = 0|Z = 1) - \mathbb{E}[Y|Z = 0, G = 1]P(G = 1|Z = 0) - \\ &\quad - \mathbb{E}[Y|Z = 0, G = 0]P(G = 0|Z = 0) \\ &= \mathbb{E}[Y|Z = 1, G = 1] - \mathbb{E}[Y|Z = 0, G = 0]\end{aligned}$$

$$\begin{aligned}\tau_{X^*}^{\text{obs}} &= (\mathbb{E}[Y(1, 1)|X = 1]P(G = 1|Z = 1, X = 1) - \mathbb{E}[Y(0, 1)|X = 1]P(G = 1|Z = 0, X = 1))P(X = 1) \\ &\quad + (\mathbb{E}[Y(1, 0)|X = 1]P(G = 0|Z = 1, X = 1) - \mathbb{E}[Y(0, 0)|X = 1]P(G = 0|Z = 0, X = 1))P(X = 1) \\ &\quad + (\mathbb{E}[Y(1, 1)|X = 0]P(G = 1|Z = 1, X = 0) - \mathbb{E}[Y(0, 1)|X = 0]P(G = 1|Z = 0, X = 0))P(X = 0) \\ &\quad + (\mathbb{E}[Y(1, 0)|X = 0]P(G = 0|Z = 1, X = 0) - \mathbb{E}[Y(0, 0)|X = 0]P(G = 0|Z = 0, X = 0))P(X = 0) \\ &= 0.5\mathbb{E}[Y(1, 1)|X = 1] - 0.5\mathbb{E}[Y(0, 0)|X = 1] + 0.5\mathbb{E}[Y(1, 1)|X = 0] - 0.5\mathbb{E}[Y(0, 0)|X = 0] \\ &= \mathbb{E}[Y(1, 1)] - \mathbb{E}[Y(0, 0)]\end{aligned}$$

$$\begin{aligned}
& \tau_{X^*}^{\text{obs}} - \tau \\
&= (\mathbb{E}[Y|Z=1, G=1, X=1] - \mathbb{E}[Y|Z=1, G=0, X=1])(P(G=1|Z=1, X=1) - P(G=1|X=1))P(X=1) \\
&- (\mathbb{E}[Y|Z=0, G=1, X=1] - \mathbb{E}[Y|Z=0, G=0, X=1])(P(G=1|Z=0, X=1) - P(G=1|X=1))P(X=1) \\
&+ (\mathbb{E}[Y|Z=1, G=1, X=0] - \mathbb{E}[Y|Z=1, G=0, X=0])(P(G=1|Z=1, X=0) - P(G=1|X=0))P(X=0) \\
&- (\mathbb{E}[Y|Z=0, G=1, X=0] - \mathbb{E}[Y|Z=0, G=0, X=0])(P(G=1|Z=0, X=0) - P(G=1|X=0))P(X=0) \\
&= 0.25\mathbb{E}[Y|Z=1, G=1, X=1] - 0.25\mathbb{E}[Y|Z=0, G=0, X=1] + 0.25\mathbb{E}[Y|Z=1, G=1, X=0] - 0.25\mathbb{E}[Y|Z=0, G=0, X=0] \\
&= 0.5\mathbb{E}[Y|Z=1, G=1] - 0.5\mathbb{E}[Y|Z=0, G=0]
\end{aligned}$$

## 5 Reproducing results of simulation study

**TODO**In this section, we aim to (partially) reproduce the results of the simulation study conducted by Forastiere et al. (2021). Note that we work with only the subset of the data that they use that is publicly accessible. The subset contains data for 6504 students (**TODO**: schools?) as opposed to the 16,410 students from 29 schools considered in their study.

## 6 Extending the simulation study

**TODO**In this section, we consider a small extension to the simulation study.

## 7 Critical appraisal and concluding remarks

**TODO**We conclude this report with a critical appraisal of the method proposed by Forastiere et al. (2021).

**TODO**contributions, limitations

**TODO**unconfoundedness assumption? Sánchez-Becerra (2021)

## References

- Barkley, B. G., Hudgens, M. G., Clemens, J. D., Ali, M., & Emch, M. E. (2020). Causal inference from observational studies with clustered interference, with application to a cholera vaccine study. *The Annals of Applied Statistics*, 14(3), 1432–1448.
- Doudchenko, N., Zhang, M., Drynkin, E., Airolidi, E. M., Mirrokni, V., & Pouget-Abadie, J. (2020). Causal inference with bipartite designs. *Available at SSRN 3757188*.
- Forastiere, L., Airolidi, E. M., & Mealli, F. (2021). Identification and estimation of treatment and interference effects in observational studies on networks. *Journal of the American Statistical Association*, 116(534), 901–918. <https://doi.org/10.1080/01621459.2020.1768100>
- Hudgens, M. G., & Halloran, M. E. (2008). Toward causal inference with interference. *Journal of the American Statistical Association*, 103(482), 832–842.
- Imai, K., Jiang, Z., & Malani, A. (2021). Causal inference with interference and noncompliance in two-stage randomized experiments. *Journal of the American Statistical Association*, 116(534), 632–644. <https://doi.org/10.1080/01621459.2020.1775612>
- Imbens, G. W., & Rubin, D. B. (2015). *Causal inference in statistics, social, and biomedical sciences*. Cambridge University Press.
- Jackson, M. O., Lin, Z., & Yu, N. N. (2020). Adjusting for peer-influence in propensity scoring when estimating treatment effects. *Available at SSRN 3522256*.
- Jagadeesan, R., Pillai, N. S., & Volfovsky, A. (2020). Designs for estimating the treatment effect in networks with interference. *The Annals of Statistics*, 48(2), 679–712.
- Liu, L., Hudgens, M. G., & Becker-Dreps, S. (2016). On inverse probability-weighted estimators in the presence of interference. *Biometrika*, 103(4), 829–842. <https://doi.org/10.1093/biomet/asw047>
- Liu, L., Hudgens, M. G., Saul, B., Clemens, J. D., Ali, M., & Emch, M. E. (2019). Doubly robust estimation in observational studies with partial interference. *Stat*, 8(1), e214.
- Ogburn, E. L., Sofrygin, O., Diaz, I., & Van der Laan, M. J. (2017). Causal inference for social network data. *arXiv preprint arXiv:1705.08527*.
- Qu, Z., Xiong, R., Liu, J., & Imbens, G. (2021). Efficient treatment effect estimation in observational studies under heterogeneous partial interference. *arXiv preprint arXiv:2107.12420*.
- Sánchez-Becerra, A. (2021). Spillovers, homophily, and selection into treatment: The network propensity score. [https://economics.sas.upenn.edu/system/files/2021-03/AlejandroSanchez\\_JMP\\_March2021\\_0.pdf](https://economics.sas.upenn.edu/system/files/2021-03/AlejandroSanchez_JMP_March2021_0.pdf)
- Saveski, M., Pouget-Abadie, J., Saint-Jacques, G., Duan, W., Ghosh, S., Xu, Y., & Airolidi, E. M. (2017). Detecting network effects: Randomizing over randomized experiments. In *Proceedings of the 23rd acm sigkdd international conference on knowledge discovery and data mining*.
- Shalizi, C. R., & Thomas, A. C. (2011). Homophily and contagion are generically confounded in observational social network studies. *Sociological methods & research*, 40(2), 211–239.
- Sobel, M. E. (2006). What do randomized studies of housing mobility demonstrate? causal inference in the face of interference. *Journal of the American Statistical Association*, 101(476), 1398–1407.
- Sofrygin, O., & van der Laan, M. J. (2017). Semi-parametric estimation and inference for the mean outcome of the single time-point intervention in a causally connected population. *Journal of Causal Inference*, 5(1).
- Tchetgen, E. J. T., & VanderWeele, T. J. (2012). On causal inference in the presence of interference. *Statistical Methods in Medical Research*, 21(1), 55–75.

- Toulis, P., Volfovsky, A., & Airoldi, E. M. (2018). Propensity score methodology in the presence of network entanglement between treatments. *arXiv preprint arXiv:1801.07310*.
- van der Laan, M. J. (2014). Causal inference for a population of causally connected units. *Journal of Causal Inference*, 2(1), 13–74.
- Zigler, C. M., & Papadogeorgou, G. (2021). Bipartite causal inference with interference. *Statistical science: a review journal of the Institute of Mathematical Statistics*, 36(1), 109.