Estimating causal effects when treatments are entangled by network dynamics

Panos Toulis
Booth School of Business
University of Chicago
ptoulis@chicagobooth.edu

Alexander Volfovsky
Department of Statistics
Duke University
alexander.volfovsky@duke.edu

Edoardo M. Airoldi
Fox School of Business
Temple University
airoldi@temple.edu

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Abstract

In many observational studies, the true treatment assignment mechanism is not individualistic, allowing the probability of treatment of a unit to depend on quantities beyond the unit's individual covariates. In such settings, unit treatments may be entangled in complex ways. Here, we consider a particular instance of this problem where the entangled treatment is a function of the social network dynamics among units, which often arises in practice. For instance, when studying the effects of new connections on a social media platform, users form new connections with other users or entire groups; so the treatment on a unit depends on the evolution of the network over time. We encounter a similar situation in many economic studies, e.g., when studying the effects of bilateral trade partnerships on countries' economic growth. The challenge in these settings is that treatment to individual units depend on a network that evolves in a way that is endogenous and cannot be manipulated experimentally. In this paper, we show that classical propensity score methods that ignore entanglement may lead to a large bias. We then offer a solution that involves modeling network dynamics, and calculating individualistic propensity scores by marginalizing over the network evolution. Under an appropriate ignorability assumption, this leads to unbiased estimates of the treatment effect of interest. We develop theoretical insights for the proposed methods, and illustrate via simulation studies based on real-world network data.

1 Introduction

In causal inference, the goal is usually to evaluate the effects of treatments applied individually on units. However, when units form networks the treatment is frequently applied to pairs or groups of connected units, and is therefore not individualistic (Imbens and Rubin 2015). Settings with such entangled individual treatments pervade many fields. For instance, professional connections affect labor market outcomes (Montgomery 1991, 1992; Podolny and Baron 1997; Calvo-Armengol and Jackson 2004), or knowledge diffusion and innovation (Dahl 2002; Kim and Marschke 2005; Agrawal et al. 2006; Granovetter 2005; Topa 2001); centrality in political networks affect coalition development (Keller 2014); and online friendships have value in marketing (Ellison et al. 2007; Hanna et al. 2017; Hobbs et al. 2016; Manchanda et al. 2015), and affect how peer influence propagates (Aral et al. 2009).

In these settings, treatment entanglement presents new methodological challenges that have not been addressed in the literature despite increased interest in evaluating treatment effects on networks. Traditionally,

the concern about treatments on networks is "interference" (Cox 1958; Rubin 1974). Under interference a unit's outcome may depend on other units' treatments. In recent years, a rich literature has emerged to deal with interference in statistics (Rosenbaum 2007; Hudgens and Halloran 2008; Toulis and Kao 2013; Aronow and Samii 2013; Ogburn et al. 2014; Bowers et al. 2013; Basse and Airoldi 2015; Karwa and Airoldi 2016; Choi 2016; Sussman and Airoldi 2017; Eckles et al. 2017; Jagadeesan et al. 2020; Basse et al. 2019b,a), or econometrics (Graham 2008; Vazquez-Bare 2017; Manski 1993; Bramoullé et al. 2009; Manski 2013; Angrist 2014), with a split focus on design and identification, respectively. However, treatment entanglement may still exist under no interference, and so the two problems are separate.

To illustrate the problem of entanglement, Figure 1 depicts six users in a hypothetical online professional network. The units form an emtpy network G^- at time t^-, and the network evolves endogeneously to G^+ at t^+. Suppose that the individual treatment of unit i, denoted by Z_i, is the number of new professional connections i makes from G^- to G^+. Thus, Z_i is a function of the change from G^- to G^+. Outcomes $Y_i \in \mathbb{R}$ are measured for each i at t^+, and may represent, say, whether i moved to a higher-income job. Our goal is here to estimate the causal effect of Z on Y, i.e., the effect of professional networking on worker mobility. Due to endogeneity, estimating this causal effect may be confounded with units' covariates. For example, in Figure 1 it would be tempting to associate making new connections to improved wages, but being more sociable confounds both making more new professional connections and having better professional outcomes.

One popular method to mitigate such endogeneity bias is the propensity score method (Rosenbaum and Rubin 1983, 1984; Heckman 1990). When treatment is binary, the idea is to model the propensity score function, $p(Z_i = 1|X_i)$, and then compare outcomes of units with similar propensities. For instance, to estimate peer effects in a mobile app network, Aral et al. (2009) first estimate the propensity score model (defined as having at least k friends adopt the app), and then compare adoption rates between treated and control units with similar propensities. The classical methodology tacitly assumes that the treatment is applied individually on each unit i. When treatments are entangled, however, individual propensities may be biased because the individual treatments depend on the network evolution from G^- to G^+, which is a population quantity and cannot be individually modeled. In the mobile app example, when some unit adopts the mobile app, all of the unit's friends are treated and so their treatments are entangled.

In this paper, we extend the classical propensity score methodology to settings with treatment entanglement. The idea is to model the propensity score of unit i taking into account information from every other unit j that could connect to i during the evolution from G^- to G^+. We propose a method that relies on an appropriate network evolution model, $p(G^+ \mid G^-, X)$, and produces improved estimates of propensity scores by averaging over likely network evolution trajectories. We show that this approach rectifies the classical propensity score method under a certain condition of network ignorability, and illustrate through several examples.

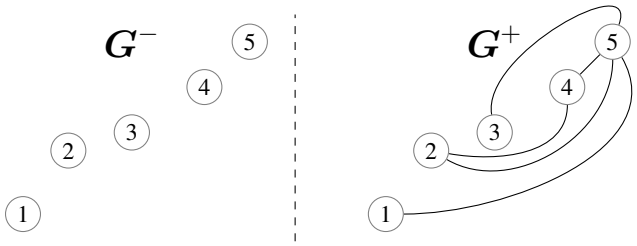
2 Preliminaries

There are N experimental units, indexed by i. The units form a pre-treament network G^- which evolves to the post-treatment network G^+. Let $N_i(G)$ denote the neighborhood of unit i in $G \in \{G^-, G^+\}$, and $d_i(G) = |N_i(G)|$ the unit's degree, i.e., the number of immediate contacts unit i has in G. The treatment of unit i is denoted by Z_i, and is a function of G^- and G^+:

$$Z_i = f_i(G^-, G^+).$$

This definition aims to capture settings where individual treatment is a function of a change in the network. Particular examples of f_i are as follows:

1. $f_i(G^-, G^+) = d_i(G^+) - d_i(G^-)$, the change in neighborhood size of unit i;



user	X_i	Z_i	Y_i
1	-5	1	0
2	-1	2	0
3	0	1	1
4	3	2	1
5	10	4	0

Figure 1: *Left:* The networks before (G^-) and after (G^+) the presumed intervention. *Right:* Observed data: X_i is the covariate value for worker i; Z_i is the treatment of i, i.e., the number of new connections that unit i made in G^+; Y_i denotes the outcome of unit i, where $Y_i = 1$ if unit i increased income after the treatment period, and $Y_i = 0$ otherwise.

2. $f_i(G^-, G^+) = \mathbb{I}\{d_i(G^+) > d_i(G^-)\}$, whether the neighborhood of i grew;

3. $f_i(G^-, G^+) = 1/\sum_{j \in V(G^+)} \text{dist}(i,j) - 1/\sum_{j \in V(G^-)} \text{dist}(i,j)$, where $\text{dist}(i,j)$ is a measure of distance between nodes i and j in a network, and the measure captures, say, a change in individual closeness centrality.

The actual definition of treatment depends on the application, but it has to be a deterministic function of G^-, G^+. For concreteness, in this paper we adopt Specification 1, such that

$$Z_i = f_i(G^-, G^+) = d_i(G^+) - d_i(G^-), \tag{1}$$

which is representative of several real-world applications (see Section 2.1). In this specification, there is treatment entanglement because individual degrees are co-dependent; for instance, $\sum_i d_i(G)$ being an even number is one such co-dependence. The vector $Z = (Z_i : i = 1, \ldots, N)$ is the population treatment assignment vector. The potential outcome for unit i under treatment Z is denoted by $Y_i(Z) \in \mathbb{R}$. Each unit i has covariate $X_i \in \mathcal{X}$, where $\mathcal{X} \subseteq \mathbb{R}^p$, with p fixed.

Broadly speaking, treatment entanglement exists when the treatment assignment of a unit is not individualistic, that is, there exists no function that models the treatment assignment probability for every unit based only on the unit's individual covariates (Imbens and Rubin 2015, Chapter 3). Relaxing the restriction of individualistic treatment is the key point of departure of this paper from the classical framework of causal inference. Specifically, in this paper we consider the setting where entanglement is a function of network dynamics that cannot be manipulated experimentally, and is thus endogenous.

The above definitions imply a multilevel treatment. We therefore need to extend the stable unit treatment assumption (SUTVA) (Rubin 1980), also known as "individualistic treatment response" in econometrics (Manski 2013). Specifically, we assume that the value of Z_i only affects the outcome of i, and that there are no hidden versions of the treatment. We provide the formal generalization of SUTVA below.

Assumption 1 (Multilevel treatment SUTVA)**.** *For every population assignment vectors Z, Z', $Y_i(Z) = Y_i(Z')$ if $Z_i = Z'_i$ for every unit i.*

With a slight abuse of notation, Assumption 1 allows us to write $Y_i(Z)$ as $Y_i(Z_i)$. Since Z_i takes only integer values by definition in Equation (1), let $\mathbb{Y}_i = \{Y_i(-N), \ldots, Y_i(N)\}$ denote all possible potential outcomes for unit i. The observed outcome for i is denoted by $Y_i \in \mathbb{Y}_i$.

Regarding the causal estimand, we suggest the following generalization of the classical average treatment effect (ATE):

$$\tau_m = E\left(Y_i(m) - Y_i(m-1)\right), \tag{2}$$

where the expectation is either over the sample or over an infinite population. Under entanglement the estimand, τ_m, captures the incremental causal effect from adding (or losing) m new connections in the treatment period to adding (or removing) $m-1$ new connections. With only two levels of treatment, τ_1 is the classical ATE.

2.1 Examples

Here, we discuss some examples from the literature that exhibit treatment entanglement. We use these examples to illustrate the setup, and also motivate the technical challenges.

Example 1 (Aral et al. 2009)**.** *This problem was outlined in Section 1. We consider here the cross-sectional snapshot of the problem for simplicity. The treatment for some unit i is binary, and indicates whether more than, say, one friend of i adopted a product in a pre-specified period. Thus, G^- and G^+ are graphs that denote exposures to product adoptions (we assume G^- is empty for simplicity). The treatment can be defined as:*

$$Z_i = \mathbb{I}\{d_i(G^+) - d_i(G^-) \geq 1\}. \tag{3}$$

Individual treatments are therefore entangled. For instance, two units that share a common neighbor that adopts the product are both exposed to the treatment together.

Example 2 (Banerjee et al. 2013)**.** *In this problem, a microfinance program is introduced in some parts of a networked population, and then the information about this opportunity is diffused through the network. The goal is to understand peer effects on information diffusion. In this context, some unit i is treated if i is informed about the program from a friend. In this case, G^-, G^+ denote the (directed) interactions between units. This interaction network overlaps with the social network but the two networks need not be identical. The definition of treatment, Z_i, is the same as in Equation (3), given the new definitions of G^-, G^+ as interaction networks.*

Example 3 (Keller 2014, 2015)**.** *In these problems the question is how different notions of network centrality (such as betweenness and closeness) affect coalition formation. Considering individual centrality as a treatment leads to entanglement because the centralities of nearby units are correlated. While these papers do not do a formal causal analysis, the goal remains the same as in the other two examples. That is, to understand the effect of an entangled network measure on an individual level outcome.*

3 Challenges under treatment entanglement

Since we are in an observational setting, we require a model for the propensity of an individual to select into treatment. Standard methods, such as those described in Section 2.1, model the selection probability as:

$$e(l, X_i) = P(Z_i = l | X_i, G^-),\ l \in \{-N, \ldots, N\}, \tag{4}$$

and then compare outcomes of treated-control units with similar propensity scores. Conditional on similar propensity scores, the treatment is as if randomly assigned under an appropriate ignorability assumption, which leads to valid causal estimates (Rosenbaum and Rubin 1983).

However, this standard approach ignores treatment entanglement. The subtle issue here is that the probability in Equation (4) implicitly conditions on G^+, since Z_i is a function of both G^- and G^+ by Equation (1). Hence, the classical propensity score methodology is actually modeling $P(Z_i = l | X_i, G^+, G^-)$, and not $P(Z_i = l | X_i, G^-)$ as desired. Conditioning on the network and then estimating the propensity scores is incorrect because in the presence of entanglement the treatment is a function of the network.

One appropriate way to compute the propensity scores in Equation (4) is to marginalize over the post-treatment network, accounting for uncertainty in G^+. This relies on a statement about the ignorability of treatment, which we formalize as follows.

Assumption 2 (Ignorability Under Entanglement)**.** *Let $\mathbb{Y} = (\mathbb{Y}_1, \ldots, \mathbb{Y}_N)$, where $\mathbb{Y}_i$ is the set of all possible potential outcomes of unit i. Then, G^+ is conditionally independent of $\mathbb{Y}$ given pre-treatment information $\boldsymbol{X} = (X_1, \ldots, X_N) \in \mathcal{X}^N$ and G^-; that is,*

$$G^+ \perp\!\!\!\perp \mathbb{Y} \mid \boldsymbol{X}, G^-.$$

This assumption is an extension of the standard ignorability assumption of treatment in settings with no entanglement (Rosenbaum and Rubin 1983, Section 1.3). Under this assumption we can correctly calculate the propensity scores, and get unbiased estimates of τ_m, according to the following theorem.

Theorem 1. *Suppose that Assumption 3 holds, and let $E_m(x) = \{e(m-1, x), e(m, x)\}$. The propensity scores are calculated as*

$$e(l, X_i) = p(Z_i = l | \boldsymbol{X}, G^-) = \int_{f_i(G^-, G^+) = l} p(G^+ | G^-, \boldsymbol{X}) d\mu(G^+), \tag{5}$$

where μ is a Lebesgue measure on G^+, and $p(G^+ \mid G^-, \boldsymbol{X})$ is the network evolution model. If the network model is correctly specified, and

$$0 < e(m-1, x), e(m, x) < 1,$$

for all $m \in \{-N+1, \ldots, N\}$, and $x \in \mathcal{X}$, then

$$E\{Y \mid Z = m, E_m(X)\} - E\{Y \mid Z = m-1, E_m(X)\} = E\{Y(m) - Y(m-1) \mid E_m(X)\}.$$

The proof of Theorem 1 is in the Supplement. Intuitively, the key result is that we can use the standard propensity score methodology as usual provided that we have computed the correct propensity scores in Equation (9). In the literature there is variety of ways these propensity scores can be used, including matching, subclassification or inverse weighting (Imbens and Rubin 2015; Rosenbaum 2002). While in this paper we suggest subclassification for additional robustness, choosing the appropriate method is separate from the problem of entanglement.

Remark 1. *Standard propensity score-based methods need to be adjusted to accommodate that treatment is generally multilevel under entanglement. These problems have been partially addressed by Hirano and Imbens (2004); Lee et al. (2015); Lopez et al. (2017). Theorem 1 contributes to this literature, showing that to estimate τ_m we can use the classical methodology through the two-dimensional propensity score, $E_m(x)$.*

Remark 2. *The choice of network model, $p(G^+ | \boldsymbol{X}, G^-)$ is crucial but, ultimately, application-specific. Possible choices include simple rewiring models (Dietz and Hadeler 1988), Temporal Exponential Random Graph Models (a generalization of the ERGM framework, Hanneke and Xing (2007); Hanneke et al. (2010)), and dynamic latent space models (Sarkar and Moore 2006; Sewell and Chen 2015; Durante and Dunson 2014). When it is reasonable to assume that G^- and $G^+ \setminus G^-$ are conditionally independent, one can appeal to the generalizability of (non-)dynamic latent space models (Hoff et al. 2002): one can model G^- conditional on unit and dyadic covariates and use the fitted model to compute the probability of edges in $G^+ \setminus G^-$.*

4 Concrete methodology

Theorem 1 implies that modeling the change in the network and then marginalizing over the treatment definition should allow for proper causal estimation when treatments are entangled. This suggests the following methodology:

1. Calculate the treatment assignments, $Z_i = d_i(G^+) - d_i(G^-)$.
2. Let $G^+|G^-, \boldsymbol{X}$ be modeled via $p(\cdot|\theta, G^-, \boldsymbol{X})$. Obtain estimate $\hat{\theta}$ of the model parameters.
3. Use $\hat{\theta}$ to sample $G^+_{(b)}$, for $b = 1, \ldots, B$, conditional on observed G^-.
4. Use samples $\{G^+_{(b)}\}$ to compute $\hat{e}(l, X_i)$ using the empirical frequencies:

$$\hat{e}_{i,l} = \hat{e}(l, X_i) = \frac{1}{B} \sum_{b=1}^{B} \mathbb{I}\{f_i(G^-, G^+_{(b)}) = l\}.$$

5. Subclassify units according to pairs $(\hat{e}_{i,m-1}, \hat{e}_{i,m})$. That is, units should be grouped together if both pair values are similar.
6. Obtain estimates of τ_m within classes, and combine estimates across classes into $\hat{\tau}_m$.

Several steps in the approach can in fact be tuned by the substantive scientist to better accommodate a particular problem. This tuning should revolve around quantifying the uncertainty in $p(G^+ \mid \boldsymbol{X}, G^-)$. In the method proposed above we followed a parametric bootstrap approach. In the simulations of Section 5 we present an additional approach, where a Bayesian model generates posterior predictive estimates of the propensity scores.

Both of these approaches rely on parametric models, but they could be replaced by non-parametric network models (Wolfe and Olhede 2013; Airoldi et al. 2013; Borgs and Chayes 2017). Moreover, specific econometric or game-theoretic considerations may be taken into account while modeling network evolution (Galeotti et al. 2006; Jackson 2010; Chandrasekhar and Lewis 2011; Graham 2015). Lastly, a clustering technique needs to be selected in Step 5 because we are employing a subclassification-based estimator. In Section 5, for example, we choose k-means clustering, but several other options are available (Friedman et al. 2001). A comparative study of different clustering techniques in terms of bias/variance would be interesting but we leave this for future work.

5 Numerical Examples

5.1 Small multiplicative covariates simulation

Consider again the example in Figure 1. There are five units, each having a one-dimensional covariate $X_i \in \mathbb{R}$. The pre-treatment network G^- has no edges (as might be expected in a product adoption study) and the post-treatment network $G^+ = (g^+_{ij})$ has a probability distribution such that the connection g^+_{ij} between two units i and j is an independent Bernoulli:

$$P(g^+_{ij} = 1|G^-, \boldsymbol{X}) \propto \exp(X_i X_j + 1.0). \tag{6}$$

As before, $e(l, X_i)$ denotes the probability that unit i makes l new connections in total. Our goal is to use the data shown in Figure 1 to estimate $\tau_2 = E(Y_i(2) - Y_i(1))$, i.e., the causal effect of making two new connections relative to making just one.

unit (i)	propensity for $Z_i = \ldots$ 0	1	2	3	4	...
1	0.00	0.27	0.73	0.00	0.00	...
2	0.00	0.24	0.67	0.09	0.00	...
3	0.01	0.06	0.23	0.42	0.28	...
4	0.00	0.24	0.68	0.09	0.00	...
5	0.00	0.27	0.73	0.00	0.00	...

unit (i)	propensity score for $Z_i = \ldots$ 0	1	2	3	4	...
1	0.37	0.37	0.18	0.06	0.02	...
2	0.24	0.34	0.25	0.12	0.04	...
3	0.21	0.33	0.26	0.13	0.05	...
4	0.13	0.26	0.27	0.19	0.10	...
5	0.02	0.08	0.15	0.20	0.20	...

Table 1: Propensity scores from two different models. The left panel is based the methodology described in Section 4 using true model (6). The right panel is based on the misspecified Poisson regression in Eq. (7). Units within dashed lines are subclassified together as having similar propensities to receive $Z_i = 1$ and $Z_i = 2$. The misspecified model leads to incorrect subclassification and, consequently, bias in causal inference.

Our proposed method in Section 4 requires conditioning on the propensity scores for making one or two connections. We compare two models for the propensity scores. The first method relies on the true model in Equation (6). The second method follows the classical propensity score approach that ignores the network structure, and instead fits a Poisson regression model. Based on the data of Figure 1 the fitted model is given by:

$$P(Z_i = l|X_i) \propto \text{Pois}(\lambda_i),\ \log \lambda_i = 0.45 + 0.09X_i, \tag{7}$$

where "Pois" denotes the Poisson density, and the estimates are rounded to two decimal points. Table 1 contains the estimated propensity scores from the two aforementioned models, and outlines the resulting subclassification based on these estimates. Unsurprisingly, the subclassifications lead to different estimates of the causal effect: $\hat{\tau}_2 = 0.5$ using the true model (6), and $\hat{\tau}_2 = 0$ using the classical model (7). In absolute value the bias is 0.5, which is substantial because the range of estimands is [-1, 1] as outcomes are binary.

The explanation for such bias is straightforward. The graph G^+ in the data of Figure 1 is an atypical sample from its true distribution implied by Equation (6). Specifically, unit 3 has only one connection in G^+. However, from the left panel of Table 1 we get $E(Z_3) \approx 2.9$. As mentioned earlier, the classical methodology conditions on G^+, and thus underestimates the propensity scores for unit 3. Additionally, the atypically large number of new connections for unit 5 ($Z_5 = 4$) influences the Poisson model substantially, leading to the association of higher covariate X values with a higher number of connections. This contributes to underestimating the propensity scores of unit 3. This underestimation eventually leads to wrong subclassification and biased estimates of the causal effect.

5.2 Large simulation studies

The simulations in this section first consider the setting of deterministic entanglement of this paper, and then a generalization to probabilistic entanglement for illustration. As before, G^- has no edges and for G^+ the probability of an edge is given by

$$P(g^+_{ij} = 1) = \text{expit}(a_i/2 + a_j/2 + bX_{ij}),$$

where a_i can be thought of as an unobserved measure of individual popularity (or sociability), while $X_{ij} \sim N(0, 1)$ is some dyadic covariate. We consider the case of a symmetric covariate and network: $X_{ij} = X_{ji}$ and $g^+_{ij} = g^+_{ji}$. We study two definitions of treatment:

(1) making at least 1 new friend; that is, $Z_i = \mathbb{I}\{\sum_j g^+_{ij} > 0\}$, and

(2) making more than 10 new friends; that is, $Z_i = \mathbb{I}\{\sum_j g_{ij}^+ > 10\}$.

In the first simulation we let $a_i \overset{iid}{\sim} N(-5, \sigma^2)$ while in the second we let $a_i \overset{iid}{\sim} N(-2, \sigma^2)$. In both settings, as $\sigma^2 \to 0$, the probability of an edge approaches expit$(a + bX_{ij})$, for an appropriate constant a. Finally, when we consider probabilistic entanglement, we also consider "having at least 1 new friend" but we do not enforce $X_{ij} = X_{ji}$ or $g_{ij} = g_{ji}$ and let $a_i \overset{iid}{\sim} N(-5, \sigma^2)$.

To evaluate results we follow the procedure in (DuGoff et al. 2014) and simulate outcome data based on pre-treatment covariate information: $Y_i(0) = 25a_i + \epsilon_i$, and $Y_i(1) = Y_i(0) + 10$, where $\epsilon_i \overset{iid}{\sim} N(0, \sigma^2)$. The observed values are $Y_i = Z_i Y_i(1) + (1 - Z_i)Y_i(0)$. In Table 2 we report the root mean squared error based on 5000 networks with $N = 100$ nodes. Similar to Section 5.1, we report these values when subclassifying on the true propensities (labeled "True") and a misspecified model (labeled "Wrong") that estimates the propensity score via a logistic regression with $\sum_j X_{ij}$ as covariate for unit i. Additionally, when studying Treatment 2 we also demonstrate the substantial improvement over the marginal model by fitting a full network model (labeled "RE" for "random effects"), as proposed in Section 4. In fact we use an asymmetric and misspecified model to demonstrate that modeling the full network behavior is superior to only modeling the margin. The results of the simulation demonstrate that misspecification via a marginal model can lead to very poor estimation of the ATE.

Simulation 1: Making at least one new friend. We only consider the true propensity score and the misspecified marginal logistic model. The results are presented in the leftmost subtable of Table 2. As expected, when the a_i are very different (σ^2 is large), the misspecified model is unable to adapt to the true propensity model, and so the RMSE is very large. As $\sigma^2 \to 0$ the discrepancy between the two approaches reduces. When $\sigma = 0$ the a_i are identical, and so both the misspecified and true model subclassify in exactly the same way since the X_{ij} are independent and identically distributed. In this case, a simple linear model will do just fine is subclassifying units, and hence in estimating the causal effect. However, as σ increases the heterogeneity in the network grows leading to worse subclassification and more biased estimation of the causal effect when the network is ignored.

Simulation 2: Multiple new friends. In this simulation we include a network model where the probability of an edge is a function of X_{ij}, and a random node effect (we note that this model is still misspecified). The results are presented in the middle subtable of Table 2 of the simulation do not differ from the previous ones. However, we see that fitting even a misspecified network model provides substantial improvement (more than two-fold for higher variance a_i) to the estimation of treatment effects when the treatments are entangled compared to the classical linear propensity score model.

Simulation 3: Probabilistic entanglement. Here, we consider a setting where edges are directed, and so it is possible for person i to connect to person j (and hence i be treated) while person j does not connect to person i (and so j remains untreated). The model implies that g_{ij}^+ is correlated with g_{ji}^+, and so it is again reasonable that the performance of a marginal misspecified model will be poor. Here, we report RMSE based on the true propensities and the misspecified model over 5000 simulated networks. We see behavior that is very similar to the symmetric version of this simulation. When σ^2 is large, for example, the true propensities perform substantially better than the misspecified model, but when $\sigma^2 \to 0$, the models become indistinguishable.

6 Discussion

In this paper, we presented a method to mitigate the problems of the classical propensity score methodology when there is treatment entanglement, while leaving the rest of the propensity score methodology

	RMSE	
σ	True	Wrong
2.0	3.41	57.05
1.0	1.43	17.71
0.5	0.94	5.26
.25	0.77	1.88
.125	0.67	0.92
.0625	0.57	0.60

One new friend

	RMSE		
σ	True	RE	Wrong
2.0	6.6	39.9	79.1
1.0	2.5	25.6	35.1
0.5	1.3	11.9	13.3
.25	1.0	4.1	4.3
.125	0.8	1.5	1.5
0.0625	0.7	0.8	0.8

Ten new friends

	RMSE	
σ	True	Wrong
2.0	3.46	57.01
1.0	1.44	17.76
0.5	0.93	5.32
.25	0.79	1.92
.125	0.68	0.93
.0625	0.57	0.61

Probabilistic one new friend

Table 2: RMSE for three different simulations. Each RMSE is computed for $N = 100$ units over 5000 simulated networks. The left and middle subtables are based on symmetric networks generated by expit$(a_i/2 + a_j/2 + X_{ij})$ with $g^+_{ij} = g^+_{ji}$ while the rightmost subtable represents probabilistic entanglement in an asymmetric network where g^+_{ij} and g^+_{ji} are simply correlated.

unchanged. Our work, however, leaves several open problems.

First, it would be interesting to know theoretically the extent of bias due to misspecification of the network evolution model that defines treatment. In Section 5.2 we showed empirically that even misspecified network models can reduce bias compared to standard propensity scores. What kind of theoretical conditions guarantee such reduction is open for future work. It would also be interesting to investigate whether sensitivity analysis is possible in this setting. Second, as discussed in Section 4, it would be interesting to know how to properly select the network evolution model in step 2 of our main method, and the extent to which such selection can be data-adaptive. This may result in additional robustness. Third, subclassification on a multilevel propensity score as in Theorem 1 is never exact, and we did not address the resulting bias.

Finally, while we focused on treatment that was defined between two individuals in a network, in practice it is common to observe treatment on larger subgraphs. Examples of this abound in education and in business transactions, where whole subsets of a school or a sector are observed to receive a treatment while being connected to other subsets. As the fundamental building block for such treatment assignment is the underlying network, we believe our methodology is general enough to encompass it. In particular, after changing the definition of the treatment assignment, the procedure outlined in Section 4 may be applied directly.

7 Conclusion

In this paper we studied the problem of treatment entanglement in observational studies on networks. The statistical methodology presented in this paper is novel, extending the use of propensity scores from individual specific treatment to dyadic or pairs treatment. We showed that ignoring this element of the treatment can lead to biased estimates of causal effects. To mitigate this bias we proposed a methodology to calculate the propensity scores by marginalizing over likely network evolution trajectories. The bias completely goes away under an appropriate network ignorability assumption. In practice, the bias can be mitigated compared to standard approaches even when the network model is misspecified.

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Supplementary material

Supplementary material available at *Biometrika* online includes the proof of Theorem 1.

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Appendix

A Proof of Theorem 1

Recall that our analysis defines

$$e(m, x) = p(Z_i = m \mid X_i = x), \tag{8}$$

and $E_m(x) = (e(m-1, x), e(m, x))$, and makes the following assumption.

Assumption 3 (Ignorability Under Entanglement)**.** *Let $\mathbb{Y} = (\mathbb{Y}_1, \ldots, \mathbb{Y}_N)$, where $\mathbb{Y}_i$ is the set of all possible potential outcomes of unit i. Then, G^+ is conditionally independent of $\mathbb{Y}$ given pre-treatment information $\boldsymbol{X} = (X_1, \ldots, X_N) \in \mathcal{X}^N$ and G^-; that is,*

$$G^+ \perp\!\!\!\perp \mathbb{Y} \mid \boldsymbol{X}, G^-.$$

Theorem 2. *Suppose that Assumption 3 holds, and let $E_m(x) = \{e(m-1, x), e(m, x)\}$. The propensity scores are calculated as*

$$e(l, X_i) = p(Z_i = l | \boldsymbol{X}, G^-) = \int_{f_i(G^-, G^+) = l} p(G^+ | G^-, \boldsymbol{X}) d\mu(G^+), \tag{9}$$

where μ is a Lebesgue measure on G^+, and $p(G^+ \mid G^-, \boldsymbol{X})$ is the network evolution model. If the network model is correctly specified, and

$$0 < e(m-1, x), e(m, x) < 1,$$

for all $m \in \{-N+1, \ldots, N\}$, and $x \in \mathcal{X}$, then

$$E\{Y \mid Z = m, E_m(X)\} - E\{Y \mid Z = m-1, E_m(X)\} = E\{Y(m) - Y(m-1) \mid E_m(X)\}.$$

Proof. Throughout this proof we implicitly condition on G^- to ease notation. Our first goal is to show that Z is strongly ignorable conditionally on $E_m(X)$. This requires two arguments. First, we show that Z is conditionally independent of $\mathbb{Y}$ given the pair of individual propensity scores $E_m(X)$, which is analogous to Theorem 3 in (Rosenbaum and Rubin 1983). Specifically,

$$\begin{aligned}
p\{Z_i = m | \mathbb{Y}, E_m(X_i), \boldsymbol{X}\} &= p(Z_i = m | \mathbb{Y}, \boldsymbol{X}) && \text{because } \boldsymbol{X} \text{ is coarser than } E_m(X_i) \\
&= p\{d_i(G^+) - d_i(G^-) = m \mid \mathbb{Y}, \boldsymbol{X}\} && \text{by definition of } Z_i \\
&= p\{d_i(G^+) - d_i(G^-) = m \mid \boldsymbol{X}\} && \text{by Assumption 3} \\
&= e(m, X_i). && \text{by Definition (8)}
\end{aligned} \tag{10}$$

Similarly, we obtain

$$p\{Z_i = m-1 | \mathbb{Y}, E_m(X_i), \boldsymbol{X}\} = e(m-1, X_i). \tag{11}$$

The second argument is to show conditional independence for outcomes, that is,

$$\begin{aligned}
p\{\mathbb{Y}_i \mid Z_i = m, E_m(X_i)\} &\propto p\{Z_i = m | \mathbb{Y}_i, E_m(X_i)\} p\{\mathbb{Y}_i | E_m(X_i)\} \\
&= E\left[p\{Z_i = m | \mathbb{Y}_i, E_m(X_i), \boldsymbol{X}\} \mid \mathbb{Y}_i, E_m(X_i)\right] p\{\mathbb{Y}_i | E_m(X_i)\} \\
&= E\left[e(m, X_i) \mid \mathbb{Y}_i, E_m(X_i)\right] p\{\mathbb{Y}_i | E_m(X_i)\} \quad \text{from Equation (10)} \\
&\propto p\{\mathbb{Y}_i | E_m(X_i)\}.
\end{aligned}$$

Thus, we obtain

$$p\{\mathbb{Y}_i \mid Z_i = m, E_m(X_i)\} = p\{\mathbb{Y}_i | E_m(X_i)\}. \tag{12}$$

Similarly, we obtain

$$p\{\mathbb{Y}_i \mid Z_i = m-1, E_m(X_i)\} = p\{\mathbb{Y}_i | E_m(X_i)\}. \tag{13}$$

From the result in Equations (12) and (13) it immediately follows that

$$\begin{aligned} E\{Y_i \mid Z_i = m,\ E_m(X_i)\} &= E\{Y_i(m) \mid E_m(X_i)\} \\ E\{Y_i \mid Z_i = m-1,\ E_m(X_i)\} &= E\{Y_i(m-1) \mid E_m(X_i)\}, \end{aligned} \tag{14}$$

and so

$$E\{Y_i \mid Z_i = m,\ E_m(X_i)\} - E\{Y_i \mid Z_i = m-1,\ E_m(X_i)\} = E\{Y_i(m) - Y_i(m-1) \mid E_m(X_i)\}.$$

□