Peer effects in the linear-in-means model may be inestimable even when identified

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Joint Statistical Meetings 2025

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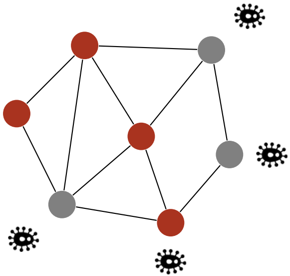
Understanding social influence is fundamental in a highly connected society



Direct effect: if I get vaccinated, I am less likely to get sick

Contagion: if my friends get sick, I am more likely to get sick

Interference: if my friends get vaccinated, I am less likely to get sick

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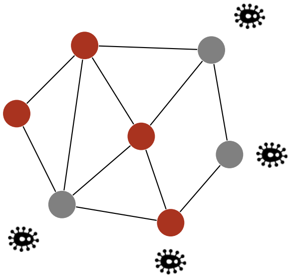

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Interference: if my friends get vaccinated, I am less likely to get sick

* Can be defined counterfactually (Vazquez-Bare, 2023), but we do not consider counterfactual inference in this talk.

This talk is about the linear-in-means model

Very popular tool for estimating social influence

Used in education, crime, health, social policy, etc [1]

I discovered an issue with this model

[1] Sacerdote (2001); Epple and Romano (2011); Soetevent and Kooreman (2007); Trogdon et al. (2008); Duflo and Saez (2003); Bertrand et al. (2000); Glaeser et al. (1996); Patacchini and Zenou (2012); Carrell et al. (2013), etc

Linear-in-means models are a canonical tool to estimate social influence

Outcome	(sick?)	$Y_i \in \{0,1\}$	Base rate	$\alpha \in \mathbb{R}$

$$\underbrace{Y_i}_{\text{sick?}} = \alpha$$

Linear-in-means models are a canonical tool to estimate social influence

Outcome	(sick?)	Y_i	$\in \{0,1\}$
Node degree	(num friends)	d_i	$\in \{0,1,2,\dots\}$
Edge $i \sim j$	(friends?)	A_{ij}	$\in \{0,1\}$

Base rate	α	$\in \mathbb{R}$
Contagion	β	$\in (-1,1)$

$$\underbrace{Y_i}_{\text{sick?}} = \alpha + \beta \underbrace{\frac{1}{d_i} \sum_{j\,:\,A_{ij}=1} Y_j}_{\substack{\text{fraction}\\ \text{sick}\\ \text{friends}}}$$

<u>Linear-in-means</u> models are a canonical tool to estimate social influence

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Edge $i \sim j$	(friends?)	A_{ij}	$\in \{0, 1\}$
Treatment	(vaccinated?)	T_i	$\in \{0, 1\}$

Base rate	α	$\in \mathbb{R}$
Contagion	β	$\in (-1, 1)$
Direct effect	γ	$\in \mathbb{R}$

$$\underbrace{Y_i}_{\text{sick?}} = \alpha + \beta \underbrace{\frac{1}{d_i} \sum_{j : A_{ij} = 1} Y_j}_{\substack{\text{fraction} \\ \text{sick} \\ \text{friends}}} + \gamma \underbrace{T_i}_{\text{vaccinated?}}$$

Linear-in-means models are a canonical tool to estimate social influence

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Linear-in-means models are a canonical tool to estimate social influence

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Node degree	(num friends)	$d_i \in \{0, 1, 2, \dots\}$		Contagion	β	$\in (-1, 1)$
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Letting $G = D^{-1}A$ be the row-normalized adjacency matrix, can write in matrix-vector form:

$$Y = \alpha 1_n + \beta G Y + T\gamma + GT\delta + \varepsilon$$

Identification in the linear-in-means model can be subtle

Review of Economic Studies (1993) **60**, 531–542 0034-6527/93/00270531$02.00

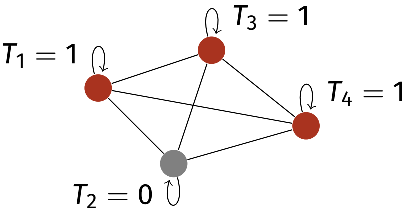
Identification of Endogenous Social Effects: The Reflection Problem

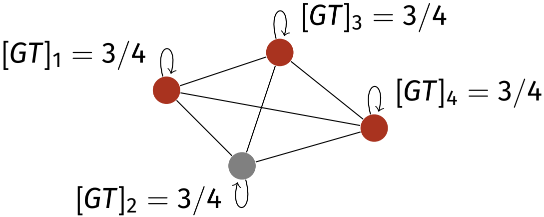
CHARLES F. MANSKI
University of Wisconsin-Madison

First version received December 1991; *final version accepted December* 1992 (*Eds.*)

This paper examines the reflection problem that arises when a researcher observing the distribution of behaviour in a population tries to infer whether the average behaviour in some group influences the behaviour of the individuals that comprise the group. It is found that inference is not possible unless the researcher has prior information specifying the composition of reference groups. If this information is available, the prospects for inference depend critically on the population relationship between the variables defining reference groups and those directly affecting outcomes. Inference is difficult to impossible if these variables are functionally dependent or are statistically independent. The prospects are better if the variables defining reference groups and those directly affecting outcomes are moderately related in the population.

Linear-in-means models are famously susceptible to perfect collinearity





$$
\begin{bmatrix} Y_1 \\ Y_2 \\ Y_3 \\ Y_4 \end{bmatrix} = \begin{array}{c} \begin{array}{cccc} 1_n & GY & T & GT \end{array} \\ \begin{bmatrix} 1 & GY_1 & 1 & 3/4 \\ 1 & GY_2 & 0 & 3/4 \\ 1 & GY_3 & 1 & 3/4 \\ 1 & GY_4 & 1 & 3/4 \end{bmatrix} \end{array} \begin{bmatrix} \alpha \\ \beta \\ \gamma \\ \delta \end{bmatrix} + \begin{bmatrix} \varepsilon_1 \\ \varepsilon_2 \\ \varepsilon_3 \\ \varepsilon_4 \end{bmatrix}
$$

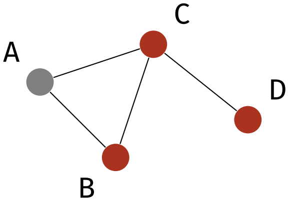
Can’t distinguish base rate α from interference δ due to collinearity

It's widely believed that this "reflection problem" is rarely a problem in practice

Proposition (Bramoullé et al. 2009)

Suppose $\gamma\beta + \delta \neq 0$. If I, G and G^2 are linearly independent, i.e., that $aI + bG + cG^2 = 0$ requires $a = b = c = 0$, then α, β, γ and δ are identified.

There is no perfect collinearity and peer influence is identified when there are open triangles ("intransitivity") in the network



Open: $B \leftrightarrow C \leftrightarrow D \not\leftrightarrow B$

Closed: $A \leftrightarrow B \leftrightarrow C \leftrightarrow A$

Standard wisdom is that collinearity is **not a problem** because most networks have open triangles

We came up with a new estimator for the linear-in-means model

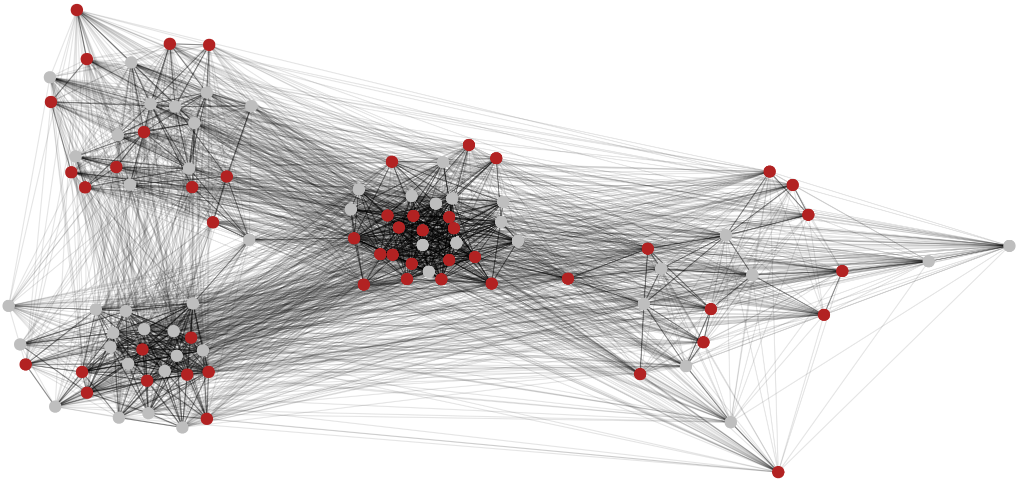
Setting: Treatment random and independent of network. $T_i \overset{\text{iid}}{\sim} \text{Bern}(0.5)$

$$Y = \alpha 1_n + \beta G Y + T\gamma + G T \delta + \varepsilon$$

We started to run a simulation study to confirm that our estimator worked[2]...

[2] Generate Y via the reduced-form specification $Y = (I - \beta G)^{-1}(\alpha 1_n + \gamma T + \delta G T + \varepsilon)$

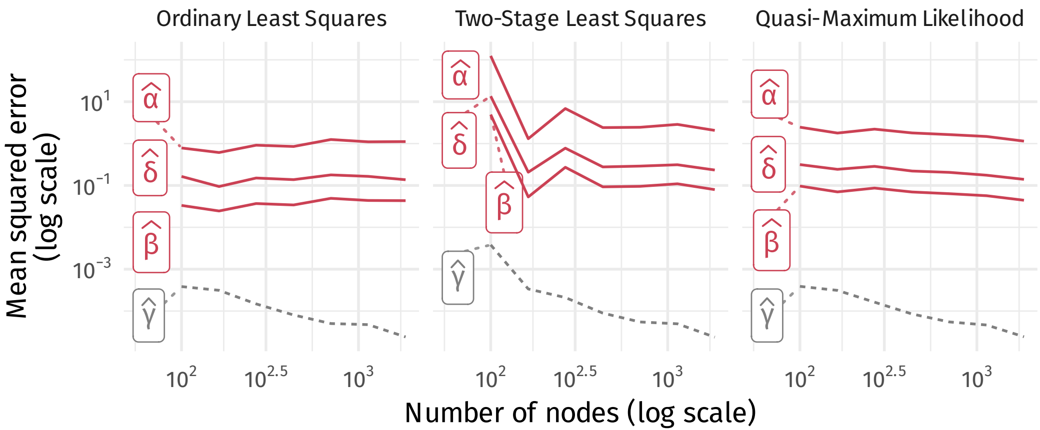
In our simulations, the network had many open triangles...



Treatments are assigned by coin flip and 45% of triangles are open

...but we couldn't estimate peer effects!

It wasn't just us, none of the standard estimators worked!



The issue: the interference column converges to a constant in large samples

$$\underbrace{[GT]_i}_{\substack{\text{fraction} \\ \text{vaccinated} \\ \text{friends}}}$$

The issue: the interference column converges to a constant in large samples

$$\underbrace{[GT]_i}_{\substack{\text{fraction}\\\text{vaccinated}\\\text{friends}}} = \underbrace{\frac{1}{d_i} \sum_{j \,:\, A_{ij}=1} T_j}_{\substack{\text{average of } d_i\\\text{i.i.d. coin flips}}}$$

The issue: the interference column converges to a constant in large samples

When the network grows ($n \to \infty$),

$$\lim_{n\to\infty} \underbrace{[GT]_i}_{\substack{\text{fraction}\\ \text{vaccinated}\\ \text{friends}}} = \lim_{n\to\infty} \underbrace{\frac{1}{d_i} \sum_{j\,:\,A_{ij}=1} T_j}_{\substack{\text{average of } d_i\\ \text{i.i.d. coin flips}}}$$

The issue: the interference column converges to a constant in large samples

When the network grows ($n \to \infty$), if everyone makes more friends ($d_i \to \infty$)

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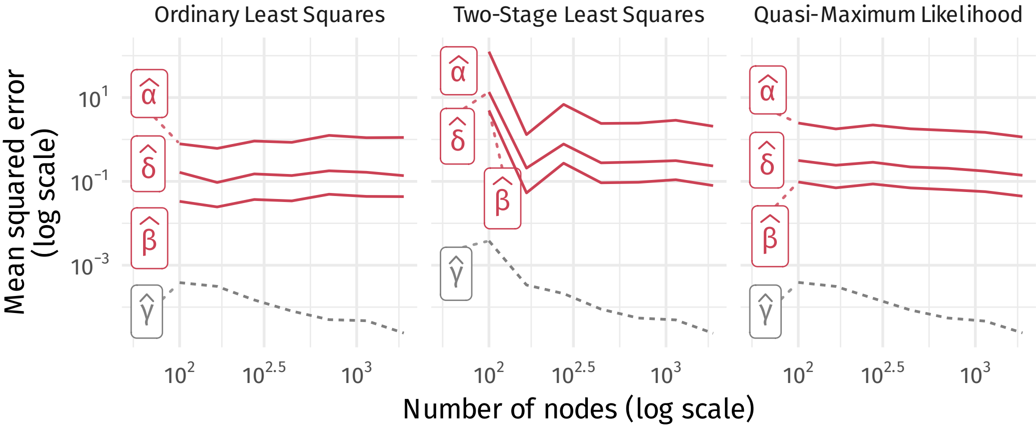
<u>For every single node $i = 1, ..., n$</u>

Base rates and interence are collinear in large samples

$$\begin{bmatrix} Y_1 \\ Y_2 \\ \vdots \\ Y_n \end{bmatrix} = \underbrace{\begin{array}{cccc} 1_n & GY & T & GT \\ \left[\begin{array}{cccc} 1 & GY_1 & 1 & 1/2 \\ 1 & GY_2 & 0 & 1/2 \\ \vdots & \vdots & \vdots & \vdots \\ 1 & GY_n & 1 & 1/2 \end{array}\right] \end{array}}_{\text{as } n \to \infty} \begin{bmatrix} \alpha \\ \beta \\ \gamma \\ \delta \end{bmatrix} + \begin{bmatrix} \varepsilon_1 \\ \varepsilon_2 \\ \vdots \\ \varepsilon_n \end{bmatrix}$$

Sometimes can't distinguish between base rate α and interference δ

In simulations, we couldn't estimate β either



Outcomes are generated by diffusing the treatment over the network

Why is β also affected?

$$Y = \alpha 1_n + \beta GY + \gamma T + \delta GT + \varepsilon$$

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$$Y - \beta G Y = \alpha 1_n + \gamma T + \delta G T + \varepsilon$$

Outcomes are generated by diffusing the treatment over the network

Why is β also affected?

$$
\begin{aligned}
Y &= \alpha 1_n + \beta G Y + \gamma T + \delta G T + \varepsilon \\
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Y &= (I - \beta G)^{-1}(\alpha 1_n + \gamma T + \delta G T + \varepsilon)
\end{aligned}
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Outcomes are generated by diffusing the treatment over the network

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Y &= (I - \beta G)^{-1}(\alpha 1_n + \gamma T + \delta G T + \varepsilon) \\
&\overset{*}{=} \underbrace{\sum_{k=0}^{\infty} \beta^k G^k}_{\text{repeated neighborhood averaging}} \ (\alpha 1_n + \gamma T + \delta G T + \varepsilon)
\end{aligned}
$$

* Must have $|\beta| < 1$, so effect of averaging decays with repetition

The contagion column converges to a constant in large samples

$$GY = \frac{\alpha}{1-\beta}1_n + \underbrace{\gamma GT}_{\substack{\text{neighborhood} \\ \text{average } \to \gamma/2}} + \underbrace{(\gamma\beta+\delta)\sum_{k=0}^{\infty}\beta^k G^{k+2} T}_{\substack{\text{repeated} \\ \text{neighborhood} \\ \text{averages} \\ \text{of } T\text{ *}}} + \underbrace{\sum_{k=0}^{\infty}\beta^k G^{k+1}\varepsilon}_{\substack{\text{repeated} \\ \text{neighborhood} \\ \text{averages} \\ \text{of } \varepsilon \to 0}}$$

Each term in the sum converges to a constant

$$GY \to \eta$$

* Neighborhood average of a constant is that same constant

Base rates, interference and contagion are collinear in large samples

$$\begin{bmatrix} Y_1 \\ Y_2 \\ \vdots \\ Y_n \end{bmatrix} = \underbrace{\begin{array}{c} \begin{array}{cccc} 1_n & GY & T & GT \end{array} \\ \begin{bmatrix} 1 & \eta & 1 & 1/2 \\ 1 & \eta & 0 & 1/2 \\ \vdots & \vdots & \vdots & \vdots \\ 1 & \eta & 1 & 1/2 \end{bmatrix} \end{array}}_{\text{as } n \to \infty} \begin{bmatrix} \alpha \\ \beta \\ \gamma \\ \delta \end{bmatrix} + \begin{bmatrix} \varepsilon_1 \\ \varepsilon_2 \\ \vdots \\ \varepsilon_n \end{bmatrix}$$

Sometimes can't distinguish between base rate α, interference δ and contagion β

Peer effects are asymptotically collinear under very general circumstances

Assumption

1. *$T_1, T_2, \ldots, T_n$ are independent with shared mean $\tau \in \mathbb{R}$, and T is independent of A.*
2. *$\{T_i - \tau : i \in [n]\}$ are independent subgamma random variables.*
3. *$\varepsilon_1, \varepsilon_2, \ldots, \varepsilon_n$ are independent subgamma random variables.*
4. *The minimum degree grows strictly faster than $\log n$, such that*

$$\lim_{n \to \infty} \frac{\min_{i \in [n]} d_i}{\log n} = \infty.$$

Recall:

$$Y = \alpha 1_n + \beta G Y + T\gamma + G T \delta + \varepsilon$$

These assumptions are distributionally agnostic

Definition (Boucheron et al. 2013)

Let Z be a mean-zero random variable with cumulant generating function $\psi_Z(t) = \log \mathbb{E}\left[e^{tZ}\right]$. Z is *subgamma* with parameters $\nu \geq 0$ and $b \geq 0$ if

$$\psi_Z(t) \leq \frac{t^2 \nu}{2(1 - bt)} \quad \text{and} \quad \psi_{-Z}(t) \leq \frac{t^2 \nu}{2(1 - bt)} \quad \text{for all} \quad t < 1/b.$$

We then write that Z is (ν, b)-subgamma.

<u>Examples</u>: Bernoulli, Poisson, Exponential, Gamma, Gaussian, sub-Gaussian, squared sub-Gaussians, bounded distributions, etc

The interference and contagion columns converge uniformly to constants

Lemma

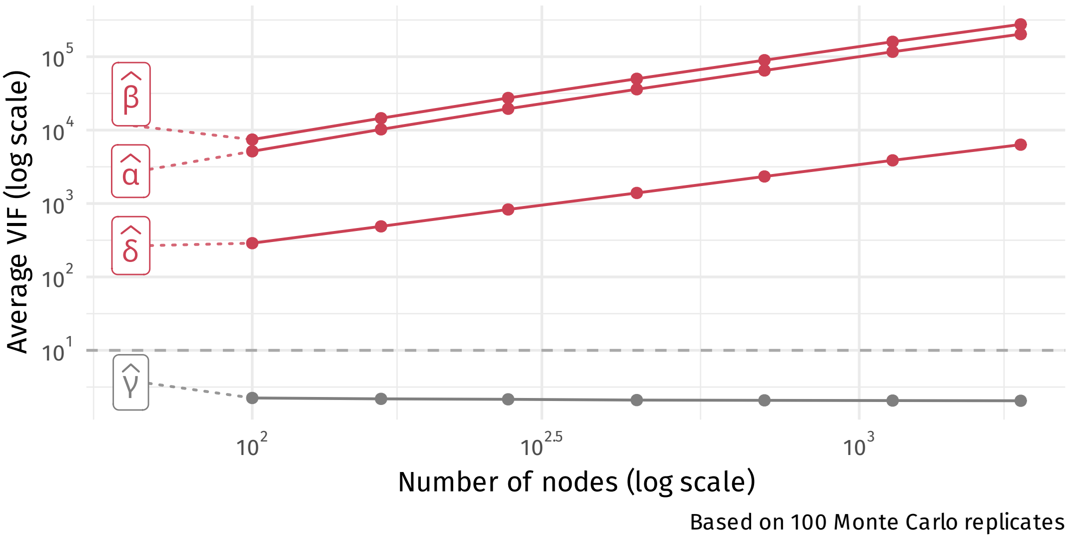
Under the previous assumptions,

$$\max_{i\in[n]} \left|[GT]_i - \tau\right| = o(1) \ \ \textit{almost surely}$$

and there exists $\eta \in \mathbb{R}$ *such that*

$$\max_{i\in[n]} \left|[GY]_i - \eta\right| = o(1) \ \ \textit{almost surely.}$$

Collinearity shows up quickly in finite samples



Asymptotic collinearity can lead to inconsistency

Theorem (Hayes and Levin 2024)

Let $(\hat{\alpha}, \hat{\beta}, \hat{\gamma}, \hat{\delta})$ be the vector of ordinary least squares estimates of $(\alpha, \beta, \gamma, \delta)$. Recall that $G = D^{-1}A$ is the row-normalized adjacency matrix. Suppose that the degrees of the network are such that $\|G\|_F^2 = o(n)$. Then if $\beta = 0$,

$$\min\{|\hat{\alpha} - \alpha|, |\hat{\beta} - \beta|\} = \Omega_P(1)$$

and

$$|\hat{\delta} - \delta| = \Omega_P\left(\frac{1}{\|G\|_F}\right). \tag{1}$$

If $\beta \neq 0$,

$$\min\{|\hat{\alpha} - \alpha|, |\hat{\beta} - \beta|\} = \Omega_P\left(\frac{1}{\|G\|_F}\right).$$

Under the stronger assumption $\|G\|_F^2 = o(\sqrt{n})$, eq. (1) holds for all β.

Asymptotic collinearity can lead to inconsistency

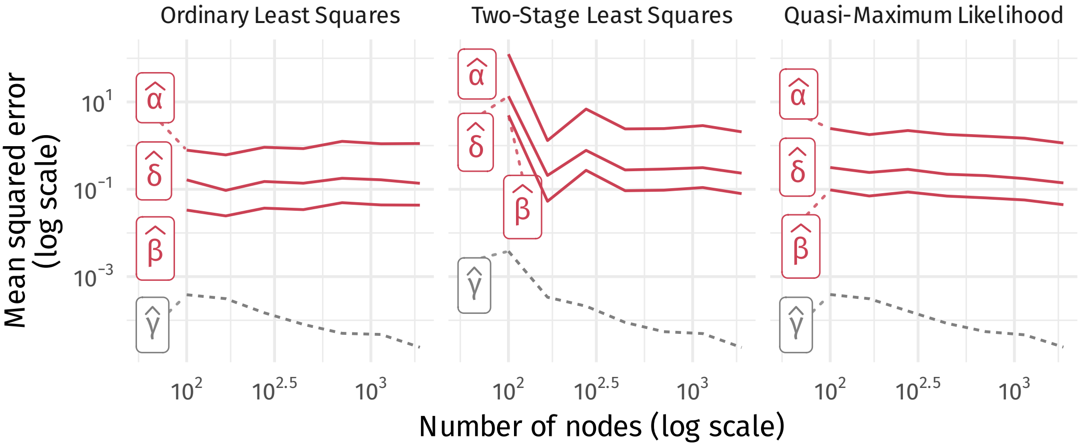
Theorem (Intuitive)

When minimum degree grows, ordinary least squares estimates of α, β *and* δ *are either inconsistent, or at best consistent at* $\sqrt{n/d_{\text{min}}}$ *rates, where* $d_{\text{min}} = \min_{i \in [n]} d_i$.

This is because the signal-to-noise ratio depends on the minimum degree[3].

[3] Our lower bound matches the upper bound on estimation error in Lee (2004).

Asymptotic collinearity affects other estimators as well



Details: weighted and directed networks

Weighted networks: If $A \in \mathbb{R}_{\geq 0}^{n \times n}$ is a positive, weighted network with (ν, b)-subgamma edges A_{ij}, we require that

$$\max_{i \in [n]} \frac{1}{d_i^2} \sum_{j=1}^{n} A_{ij}^2 = o\left(\frac{1}{\nu \log^2 n}\right) \quad \text{and} \quad \max_{j \in [n]} \frac{A_{ij}}{d_i} = o\left(\frac{1}{b \log n}\right).$$

Roughly: no one edge can be too important for a given node

Directed networks: extension possible, but slightly more involved

Isolated nodes: can allow a vanishing fraction of nodes to be isolated

Standard wisdom states that collinearity isn't a problem in linear-in-means in networks with open triangles.

Takeaway 1

When nodal covariates are independent of the network, peer effects may not be estimable, due to collinearity, even when there are many open triangles.

Takeaway 2

Direct effects are estimable even when there is asymptotic collinearity.

The interference literature is well-aware of issues with Bernoulli designs

The Conflict Graph Design: Estimating Causal Effects under Arbitrary Neighborhood Interference

Vardis Kandiros[1], Charilaos Pipis[1], Constantinos Daskalakis[1], and Christopher Harshaw[2]

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[2]*Columbia University*

November 19, 2024

Abstract

A fundamental problem in network experiments is selecting an appropriate experimental design in order to precisely estimate a given causal effect of interest. In fact, optimal rates of estimation remain unknown for essentially all causal effects in network experiments. In this work, we propose a general approach for constructing experiment designs under network interference with the goal of precisely estimating a pre-specified causal effect. A central aspect of our approach is the notion of a *conflict graph*, which captures the fundamental unobservability associated with the casual effect and the underlying network. We refer to our experimental design as the Conflict Graph Design. In order to estimate effects, we propose a modified Horvitz–Thompson estimator. We show that its variance under the Conflict Graph Design is bounded as $\mathcal{O}(\lambda(\mathcal{H})/n)$, where $\lambda(\mathcal{H})$ is the largest eigenvalue of the adjacency matrix of the conflict graph. These rates depend on both the underlying network and the particular causal effect under investigation. Not only does this yield the best known rates of estimation for several well-studied causal effects (e.g. the global and direct effects) but it also provides new methods for effects which have received less attention from the perspective of experiment design (e.g. spill-over effects). Our results corroborate two implicitly understood points in the literature: (1) that in order to increase precision, experiment designs should be tailored to specific causal effects of interest and (2) that "more local" effects are easier to estimate than "more global" effects. In addition to point estimation, we construct conservative variance estimators which facilitate the construction of asymptotically valid confidence intervals for the casual effect of interest.

Graph Cluster Randomization: Network Exposure to Multiple Universes

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ABSTRACT

A/B testing is a standard approach for evaluating the effect of online experiments; the goal is to estimate the 'average treatment effect' of a new feature or condition by exposing a sample of the overall population to it. A drawback with A/B testing is that it is poorly suited for experiments involving social interference, when the treatment of individuals spills over to neighboring individuals along an underlying social network. In this work, we propose a novel methodology using graph clustering to analyze average treatment effects under social interference. To begin, we characterize graph-theoretic conditions under which individuals can be considered to be 'network exposed' to an experiment. We then show how graph cluster randomization admits an efficient exact algorithm to compute the probabilities for each vertex being network exposed under several of these exposure conditions. Using these probabilities as inverse weights, a Horvitz-Thompson estimator can then provide an effect estimate that is unbiased, provided that the exposure model has been properly specified.

Given an estimator that is unbiased, we focus on minimizing the variance. First, we develop simple sufficient conditions for the variance of the estimator to be asymptotically small in n, the size of the graph. However, for general randomization schemes, this variance can be *lower bounded* by an *exponential* function of the degrees of a graph. In contrast, we show that if a graph satisfies a *restricted-growth condition* on the growth rate of neighborhoods, then there exists a natural clustering algorithm, based on vertex neighborhoods, for which the variance of the estimator can be *upper bounded* by a *linear* function of the degrees. Thus we show that proper cluster randomization can lead to exponentially lower estimator variance when experimentally measuring average treatment effects under interference.

1. INTRODUCTION

Social products and services – from fax machines and cell phones to online social networks – inherently exhibit 'network effects' with regard to their value to users. The value of these products to a user is inherently non-local, since it typically grows as members of the user's social neighborhood use the product as well. Yet randomized experiments (or 'A/B tests'), the standard machinery of testing frameworks including the Rubin causal model [14], critically assume what is known as the 'stable unit treatment value assumption' (SUTVA), that each individual's response is affected only by their own treatment and not by the treatment of any other individual. Addressing this tension between the formalism of A/B testing and the non-local effects of network interaction has emerged as a key open question in the analysis of on-line behavior and the design of network experiments [6].

Under ordinary randomized trials where the stable unit treatment value assumption is a reasonable approximation — for example when a search engine A/B tests the effect of their color scheme upon the visitation time of their users — the population is divided into two groups: those in the 'treatment' group who see the new color scheme A and those in the control group who see the default color scheme B. Assuming there are negligible interference effects between users, each individual in the treated group responds just as he or she would if the entire population were treated, and each individual in the control group responds just as he or she would if the entire population were in control. In this manner, we can imagine that we are observing results from samples of two distinct 'parallel universes' at the same time — 'Universe A' in which color scheme A is used for everyone, and 'Universe B' in which color scheme B is used for everyone — and we can make inferences about the properties of user behavior in each of these universes.

Causal folks are interested in treatments that depend on position in network

Estimating Causal Peer Influence in Homophilous Social Networks by Inferring Latent Locations

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ABSTRACT

Social influence cannot be identified from purely observational data on social networks, because such influence is generically confounded with latent homophily, that is, with a node's network partners being informative about the node's attributes and therefore its behavior. If the network grows according to either a latent community (stochastic block) model, or a continuous latent space model, then latent homophilous attributes can be consistently estimated from the global pattern of social ties. We show that, for common versions of those two network models, these estimates are so informative that controlling for estimated attributes allows for asymptotically unbiased and consistent estimation of social-influence effects in linear models. In particular, the bias shrinks at a rate that directly reflects how much information the network provides about the latent attributes. These are the first results on the consistent nonexperimental estimation of social-influence effects in the presence of latent homophily, and we discuss the prospects for generalizing them.

ARTICLE HISTORY

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KEYWORDS

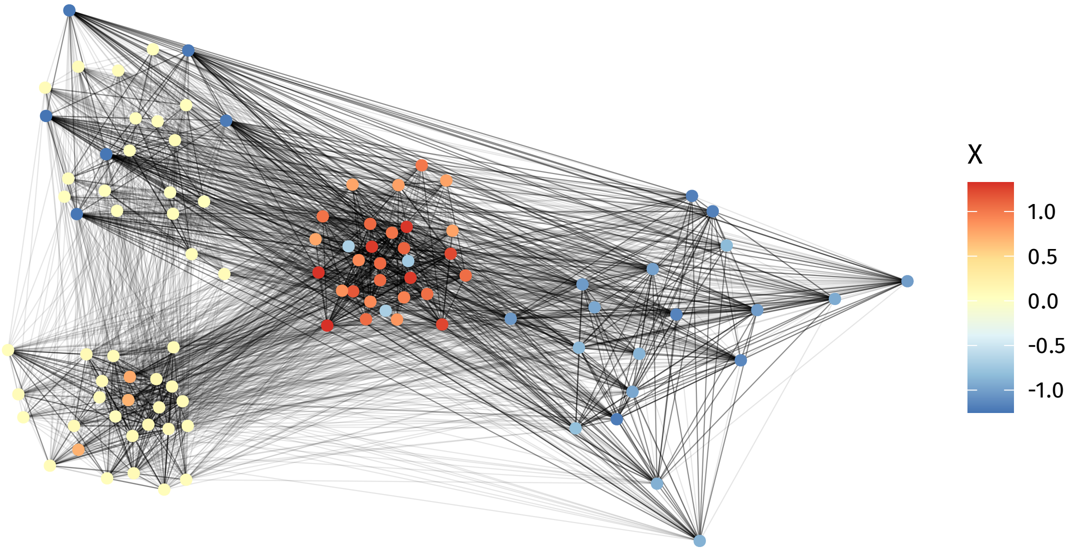
Causal Inference; Homophily; Social Networks; Peer Influence

Dependence between treatment and network might resolve collinearity issues

$$\underbrace{[GT]_i}_{\substack{\text{fraction}\\\text{vaccinated}\\\text{friends}}} = \underbrace{\frac{1}{d_i} \sum_{j\,:\,A_{ij}=1} T_j}_{\text{average of dependent treatments}}$$

GT might not converge, or might converge to non-constant value

We considered models where treatment depended on position in network



We considered models where treatment depended on position in network

Stochastic blockmodels are an intuitive way to induce dependence

Block indicators Z_i
Popularity parameters θ_i
Mixing matrix $B \in [0,1]^{d \times d}$

$$\mathbb{P}[Z,\theta]A_{ij} = 1 = \theta_i Z_i B Z_j^T \theta_j$$

Linear-in-means models on blockmodels are of independent interest!

Causal Network Influence with Latent Homophily and Measurement Error: An Application to Therapeutic Community

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Abstract

The Spatial or Network Autoregressive model (SAR, NAM) is popular for modeling the influence network connected neighbors exert on the outcome of individuals. However, many authors have noted that the *causal* network influence or contagion cannot be identified from observational data due to the presence of homophily. We propose a latent homophily-adjusted spatial autoregressive model for networked responses to identify the causal contagion and contextual effects. The latent homophily is estimated from the spectral embedding of the network's adjacency matrix. Separately, we develop maximum likelihood estimators for the parameters of the SAR model correcting for measurement error when covariates are measured with error. We show that the bias corrected MLE are consistent and derive its asymptotic limiting distribution. We propose to estimate network influence using the bias corrected MLE in a SAR model with the estimated latent homophily added as a covariate. Our simulations show that the methods perform well in finite sample. We apply our methodology to a data-set of female criminal offenders in a therapeutic community (TC) for substance abuse and criminal behavior. We provide causal estimates of network influence on graduation from TC and re-incarceration after accounting for latent homophily.

We prove a partial asymptotic collinearity result for these models

Theorem (Hayes and Levin 2024)

Suppose that A is sampled from a degree-corrected stochastic blockmodel. Define $X_i = \theta_i Z_i$. Let

$$Y = \alpha 1_n + \beta G Y + X\gamma + G X \delta + \varepsilon$$

for $\alpha, \beta \in \mathbb{R}$ and $\gamma, \delta \in \mathbb{R}^d$. Suppose that X has $k \geq 2d$ distinct rows. Then, under some conditions,

$$W_n = \begin{bmatrix} 1_n & GY & X & GX \end{bmatrix}$$

converges uniformly to a limit object with rank $2d$ out of $2d + 2$. If any two entries of $(\alpha, \beta, \delta_1, \ldots, \delta_d)$ are set to zero in the data generating process, the limit object of W_n is a matrix with full rank.

We prove a partial asymptotic collinearity result for these models

Key condition to avoid collinearity: sufficient degree heterogeneity such that X and $D^{-1}X$ are linearly independent

General low-rank networks: if $\mathbb{E}[A_{ij} \mid X] = X_i^T X_j$, a similar result holds, broadly generalizing the partial identification result

We performed a simulation study to confirm the theoretical results

- Bernoulli: Treatment random and independent of network. $T_i \stackrel{\text{iid}}{\sim} \text{Bern}(0.5)$

$$Y = \alpha 1_n + \beta GY + T\gamma + GT\delta + \varepsilon,$$

  with $\alpha = 3, \beta = 0.2, \gamma = 4, \delta = 2$ and $\varepsilon \stackrel{\text{iid}}{\sim} \mathcal{N}(0, \sigma^2)$ with $\sigma = 0.1$.

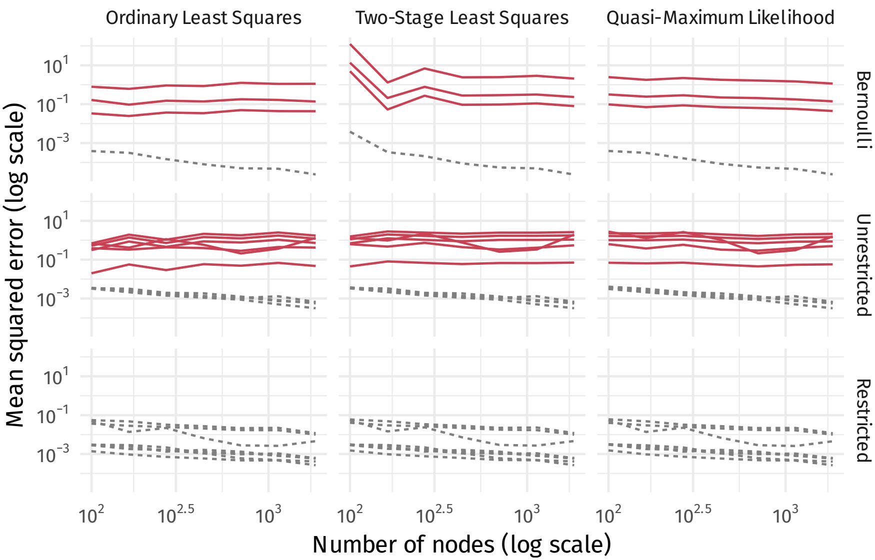
- Unrestricted model: Treatment random and dependent on network. Define $X_i = \theta_i Z_i \in \mathbb{R}^4$

$$Y = \alpha 1_n + \beta GY + X\gamma + GX\delta + \varepsilon,$$

  where $\alpha = 3, \beta = 0.2$ and $\varepsilon \stackrel{\text{iid}}{\sim} \mathcal{N}(0, \sigma^2)$ with $\sigma = 0.1$. Since $X_i \in \mathbb{R}^4$, $\gamma, \delta \in \mathbb{R}^4$ and we fix $\delta = (2, 2, 2, 2)$ and $\gamma = (1.5, 2.5, 3.5, 4.5)$.

- Restricted model: The unrestricted model, but $\delta = (0, 0, 2, 2)$, so there's no asymptotic collinearity.

Dependence prevented asymptotic collinearity and estimation challenges



Takeaway 3

Explicitly modelling dependence between nodal covariates and network structure can aid identifiability and resolve asymptotic collinearity issues.

Takeaway 4

Treatments dependent on network must be considered on a case-by-case basis, considering both the treatment and the network model.

Thank you! Questions?

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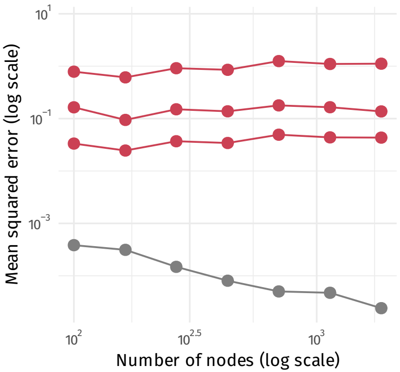

Pre-print

Alex Hayes and Keith Levin. "Peer Effects in the Linear-in-Means Model May Be Inestimable Even When Identified." arXiv, October 14, 2024. `http://arxiv.org/abs/2410.10772`.

A formal definition for identifiability

Definition (Maclaren and Nicholson 2020)

A model $\mathcal{M} = \{P_\theta : \theta \in \Theta\}$ is a collection of probability measures P_θ, indexed by a set Θ. A parameter $q(\theta)$ is *identifiable* if and only if $q(\theta_1) \neq q(\theta_2)$ implies $P_{\theta_1} \neq P_{\theta_2}$.

Several equivalent conditions for identifiability in linear models

In linear models, where $Y_i = X_i\theta + \varepsilon_i$ and $\varepsilon_i \sim \mathcal{N}(0, \sigma^2)$, the following are equivalent (Lewbel, 2019):

1. θ is identified
2. X is full-rank (i.e., there is no perfect collinearity)
3. the covariance matrix $X^T X/n$ is full-rank
4. the log-likelihood

$$-\frac{n}{2}\log(2\pi\sigma^2) - \frac{1}{2\sigma^2}\sum_{i=1}^{n}(y_i - x_i\theta)^2$$

has a unique maximizer.

A linear model that is identified, asymptotically collinear, and inestimable

Suppose that all data points except for the first data point are exactly equal:

$$\begin{bmatrix} Y_1 \\ Y_2 \\ Y_3 \\ \vdots \\ Y_n \end{bmatrix} = \begin{bmatrix} 1 & 2 \\ 1 & 1 \\ 1 & 1 \\ \vdots & \vdots \\ 1 & 1 \end{bmatrix} \begin{bmatrix} \alpha \\ \beta \end{bmatrix} + \begin{bmatrix} \varepsilon_1 \\ \varepsilon_2 \\ \varepsilon_3 \\ \vdots \\ \varepsilon_n \end{bmatrix}$$

Then α and β are identified but cannot be estimated

Estimators

- OLS: `lm`$(y \sim Gy + T + GT)$
- TSLS: `ivreg`$(y \sim Gy + T + GT \mid \underbrace{T + GT + G^2T}_{\text{instruments}})$

Definition (Random Dot Product Graph, Young and Scheinerman 2007)

Let F be a distribution on $\mathbb{R}^d$ such that $0 \leq x^T y$ for all $x, y \in \operatorname{supp} F$ and the convex cone of $\operatorname{supp} F$ is d-dimensional. Draw $X_1, X_2, \ldots, X_n \stackrel{\text{iid}}{\sim} F$, and collect these in the rows of $X \in \mathbb{R}^{n \times d}$ for ease of notation. Conditional on these n vectors, which we call *latent positions*, generate edges by drawing the edges $\{A_{ij} : 1 \leq i < j \leq n\}$ as independent (ν, b)-subgamma random variables with $\mathbb{E}[A_{ij} \mid X] = \rho X_i^T X_j$, where $\rho \in [0, 1]$. Then we say that A is distributed according to an n-vertex random dot product graph with latent position distribution F, (ν, b)-subgamma edges and sparsity factor ρ. We write $(A, X) \sim \mathrm{RDPG}(F, n)$, with the subgamma and sparsity parameters made clear from the context.

Proposition

Let $\mu = \mathbb{E}[X] \in \mathbb{R}^d$ and suppose that $Y_1, Y_2, \ldots, Y_d, Z_1, Z_2, \ldots, Z_d \in \mathbb{R}^d$ are rows of $X \in \mathbb{R}^{n \times d}$ such that $Y_1, Y_2, \ldots, Y_d$ are linearly independent and $Z_1, Z_2, \ldots, Z_d$ are linearly independent.

$$H_Y = \operatorname{diag}\left(Y_1^T \mu, Y_2^T \mu, \ldots, Y_d^T \mu\right) \quad \textit{and} \quad H_Z = \operatorname{diag}\left(Z_1^T \mu, Z_2^T \mu, \ldots, Z_d^T \mu\right).$$

Provided that $Z^{-1} H_Z^{-1} Z - Y^{-1} H_Y^{-1} Y \in \mathbb{R}^{d \times d}$ is invertible, then the matrix

$$M = \begin{bmatrix} X & H^{-1} X \end{bmatrix} \in \mathbb{R}^{n \times 2d}$$

has rank $2d$.

Morally: need degree heterogeneity so that X and $D^{-1}X$ are linearly independent

Technical conditions for partial identification result

- $\rho = \omega\left(\frac{\log^2 n}{\sqrt{n}}\right)$ and $\frac{\nu + b^2}{\rho} = \Theta(1)$
- $\min_{i \in [n]} \left| X_i^T \, \mathbb{E}[X_1] \right| = \omega\left(\frac{\log^2 n}{\sqrt{n}\rho}\right)$ almost surely.
- $\max_{i \in [n]} \|X_i\| = o(\sqrt{n})$ almost surely.
- $\mathbb{E}\|X_1\|^2 < \infty$.

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