

Estimating network-mediated causal effects via spectral embeddings

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This is joint work

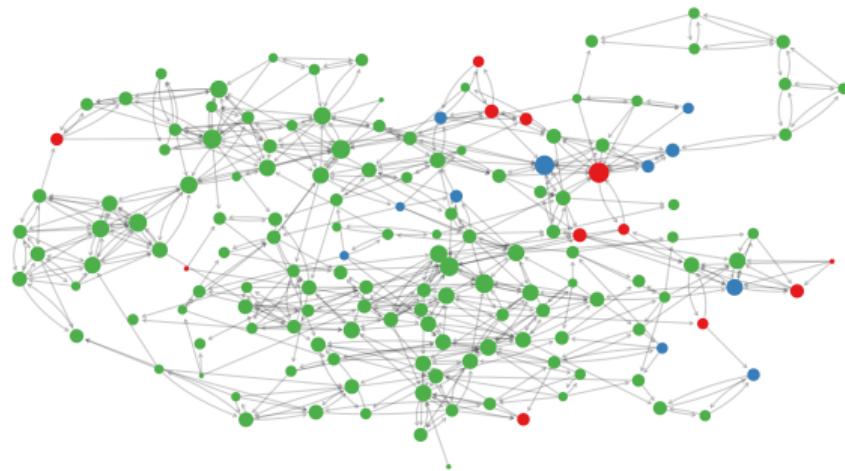


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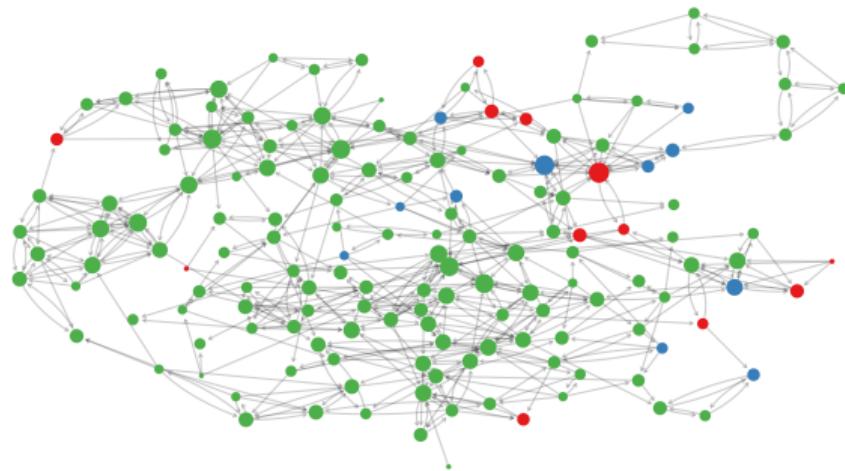
Motivation: understand causes of teenage smoking ([Michell and West, 1996](#))



Tobacco use ● Never ● Occasional ● Regular

- 129 middle-schoolers in Glasgow
- **Social network** based on self-reported friendships
- **Auxiliary data:** spending money, leisure activities
- **Demographics:** sex, age
- **Behavior:** alcohol, cannabis and tobacco use

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Question: how does sex influence tobacco use?

Smoking is a sexually differentiated behavior and also a social behavior



Recorded sex • Female • Male

Direct effect of sex on smoking

- Social and cultural expectations could lead to more or less smoking

Indirect effect of sex on smoking

- Students to be friends with other students of same sex...
- ...friends induce one another to smoke

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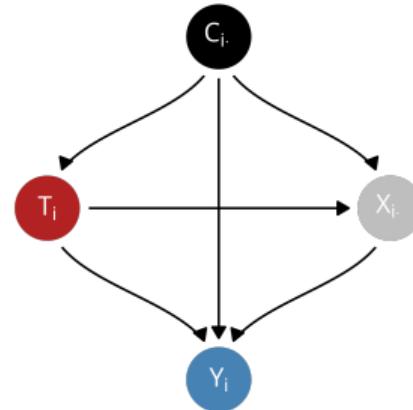
Indirect effect of sex on smoking

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- ...friends induce one another to smoke

Should anti-smoking interventions target the direct or the social mechanism?

Defining causal effects is straightforward when there is no social network

Treatment $T_i \in \{0, 1\}$
Outcome $Y_i \in \mathbb{R}$
Mediators $X_{i \cdot} \in \mathbb{R}^{1 \times d}$
Confounders $C_{i \cdot} \in \mathbb{R}^{1 \times p}$



Definition (Average treatment effect)

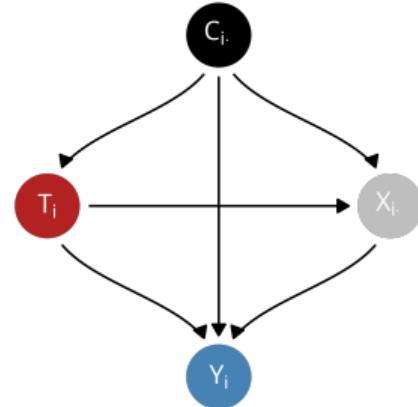
$$\Psi_{ate} = \mathbb{E}[Y(1) - Y(0)]$$

$Y(t)$ is the counterfactual outcome under treatment value $t \in \{0, 1\}$.

Decomposing causal effects is straightforward when there is no social network

Two distinct mechanisms:

1. Direct effect along $T_i \rightarrow Y_i$ path
2. Indirect effect along $T_i \rightarrow X_i \rightarrow Y_i$ path



Definition (Natural direct and indirect effects)

$$\begin{aligned}\Psi_{ate} &= \Psi_{nde} + \Psi_{nie} \\ &= \mathbb{E}[Y(1, M(0)) - Y(0, M(0))] + \mathbb{E}[Y(1, M(1)) - Y(1, M(0))]\end{aligned}$$

$Y(t, m)$ is the counterfactual outcome under treatment t and mediator m

We want to decompose node-level effects into direct and social components



Network $A \in \mathbb{R}^{n \times n}$

For each node i :

- Treatment $T_i \in \{0, 1\}$
- Outcome $Y_i \in \mathbb{R}$
- Confounders $C_{i \cdot} \in \mathbb{R}^p$

The network A is noisy and complex but often exhibits high levels of homophily

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The network A is noisy and complex but often exhibits high levels of homophily

Assume each node belongs to a social group X_i and behaviors vary with social group

Stochastic blockmodels are the canonical model for social groups



$X_{i \cdot} \in \{0, 1\}^d$ one-hot indicator of social group

$B \in [0, 1]^{d \times d}$ between-group friend probabilities

Friendships depend on social group and B

$$\mathbb{P}(A_{ij} = 1 \mid X) = X_{i \cdot} B X_{j \cdot}^T$$

Social groups X are latent (i.e., unobserved)

Nodes colored by social group

We have very rich and general models for latent social groups



Nodes colored by social group

- Degree-correction
- Mixed-membership social groups
- Overlapping social groups
- Group-specific popularity parameters

Definition (Random dot product graph)

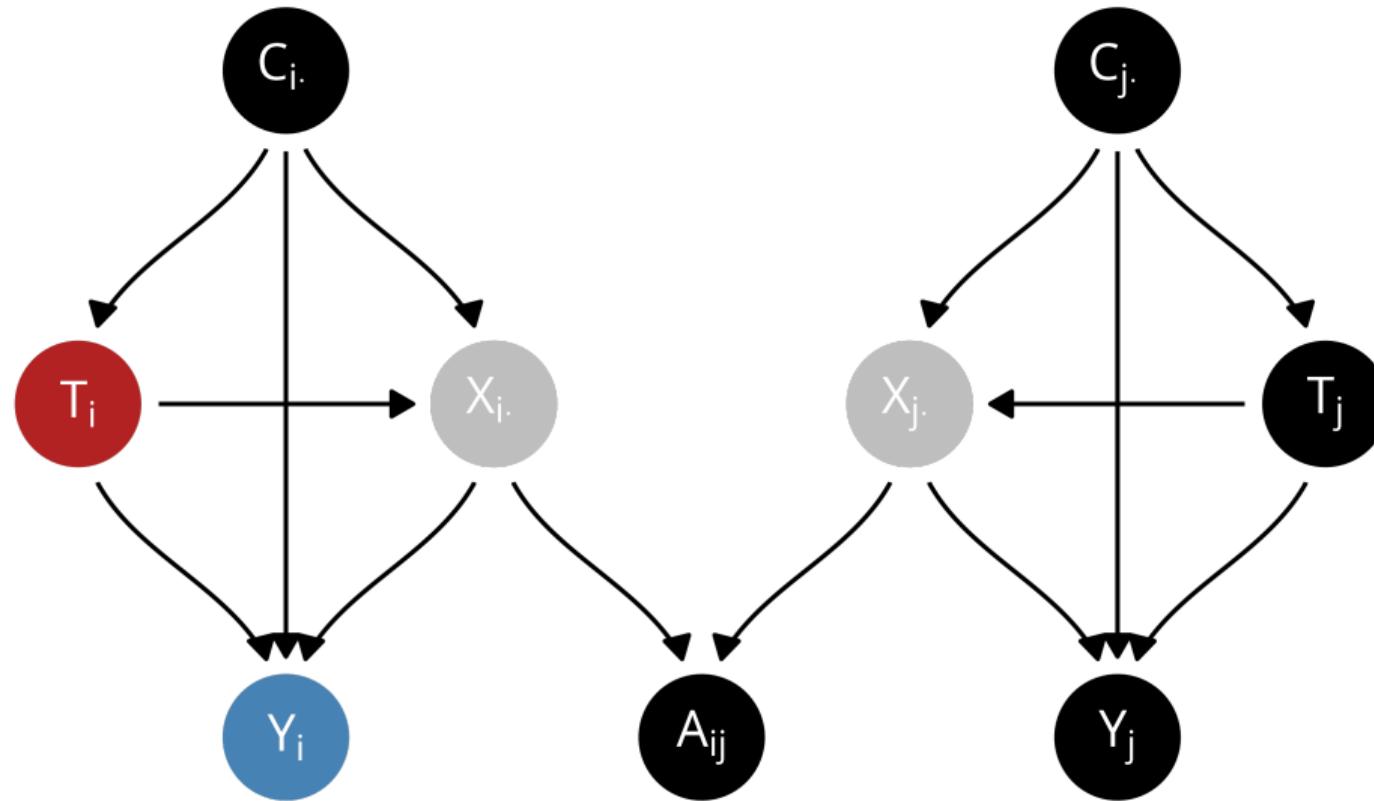
A symmetric, weighted adjacency matrix

$$A = XX^T + E$$

$X_1, \dots, X_n \in \mathbb{R}^{1 \times d}$ i.i.d. latent positions

E independent sub-gamma noise

Causal effects on a network decompose into a social and non-social component



Direct and indirect effects are identified under counterfactual assumptions

The random variables $(Y_i, Y_i(t, x), X_{i\cdot}, X_{i\cdot}(t), C_{i\cdot}, T_i)$ are independent over $i \in [n]$ and obey the following three properties.

1. Consistency:

if $T_i = t$, then $X_{i\cdot}(t) = X_{i\cdot}$ with probability 1, and

if $T_i = t$ and $X_{i\cdot} = x$, then $Y_i(t, x) = Y_i$ with probability 1

2. Sequential ignorability:

$$\{Y_i(t^*, x), X_{i\cdot}(t)\} \perp\!\!\!\perp T_i \mid C_{i\cdot} \quad \text{and} \quad \{Y_i(t^*, x)\} \perp\!\!\!\perp X_{i\cdot} \mid T_i = t, C_{i\cdot}$$

3. Positivity:

$$\mathbb{P}(x \mid T_i, C_{i\cdot}) > 0 \text{ for each } x \in \text{supp}(X_{i\cdot})$$

$$\mathbb{P}(t \mid C_{i\cdot}) > 0 \text{ for each } t \in \text{supp}(T_i)$$

We want to use a regression estimator for direct and indirect effects

Assumption

$$\mathbb{E}[Y_i | T_i, C_{i\cdot}, X_{i\cdot}] = \beta_0 + T_i \beta_t + C_{i\cdot} \beta_c + X_{i\cdot} \beta_x \quad \text{outcome model}$$

$$\mathbb{E}[X_{i\cdot} | T_i, C_{i\cdot}] = \theta_0 + T_i \theta_t + C_{i\cdot} \Theta_c \quad \text{mediator model}$$

Fact: semi-parametric identification of natural mediated effects

Under previous assumption, when natural direct and indirect effect are non-parametrically identified, we have

$$\Psi_{nde} = \beta_t$$

$$\Psi_{nie} = \theta_t \beta_x$$

Problem: We don't see the latent social groups X , can't fit regressions

Latent positions X can be estimated via principal components analysis

Definition (ASE)

Given a network A , the d -dimensional adjacency spectral embedding of A is

$$\hat{X} = \widehat{U}\widehat{S}^{1/2}$$

where $\widehat{U}\widehat{S}\widehat{U}^T$ is the rank- d truncated singular value decomposition of A .

Lemma

Under a suitable network model, there is a $d \times d$ orthogonal matrix Q such that

$$\max_{i \in [n]} \left\| \widehat{X}_{i \cdot} - X_{i \cdot} Q \right\| = o_p(1).$$

Estimated latent positions can plug directly into ordinary least squares

Let $\hat{D} = \begin{bmatrix} 1 & T & C & \hat{X} \end{bmatrix} \in \mathbb{R}^{n \times (2+p+d)}$ and $L = \begin{bmatrix} 1 & T & C \end{bmatrix} \in \mathbb{R}^{n \times (p+2)}$.

$$\begin{bmatrix} \hat{\beta}_0 \\ \hat{\beta}_t \\ \hat{\beta}_c \\ \hat{\beta}_x \end{bmatrix} = (\hat{D}^T \hat{D})^{-1} \hat{D}^T Y \quad \text{and} \quad \hat{\Theta} = (L^T L)^{-1} L^T \hat{X}.$$

$$\hat{\Psi}_{\text{nde}} = \hat{\beta}_t \quad \text{and} \quad \hat{\Psi}_{\text{nie}} = \hat{\theta}_t \hat{\beta}_x$$

Ordinary least squares regression estimates are asymptotically normal

Theorem

Under a suitably well-behaved network model and some moments conditions on regression errors, there is an unknown orthogonal matrix Q such that

$$\sqrt{n} \widehat{\Sigma}_{\beta}^{-1/2} \begin{pmatrix} \widehat{\beta}_w - \beta_w \\ Q \widehat{\beta}_x - \beta_x \end{pmatrix} \rightarrow \mathcal{N}(0, I_d), \text{ and}$$

$$\sqrt{n} \widehat{\Sigma}_{\text{vec}(\Theta)}^{-1/2} \left(\text{vec}(\widehat{\Theta} Q^T) - \text{vec}(\Theta) \right) \rightarrow \mathcal{N}(0, I_{pd}).$$

where $\widehat{\Sigma}_{\text{vec}(\Theta)}^{-1/2}$ and $\widehat{\Sigma}_{\beta}^{-1/2}$ are the typical heteroscedastic robust covariance estimators, with \widehat{X} plugged in for X .

Note: theory requires consistent estimate of latent dimension d

Aside: our regression results are substantially more general than similar work

- Network can be weighted, rather than binary
- No parametric assumptions on edge noise
- No parametric assumptions on regression errors
- Regression errors can be heteroscedastic
- Can model latent positions as outcomes

Principle components + ordinary least squares is a very general, distributionally agnostic tool for network regression

Folklore: similar results hold for asymmetric A , rectangular A , bipartite A , graph Laplacian embeddings rather than adjacency matrix embeddings, general regression M -estimators other than ordinary least squares

Causal estimators are asymptotically normal

Theorem

Under the same statistical assumptions as before, plus counterfactual assumptions required for causal identification,

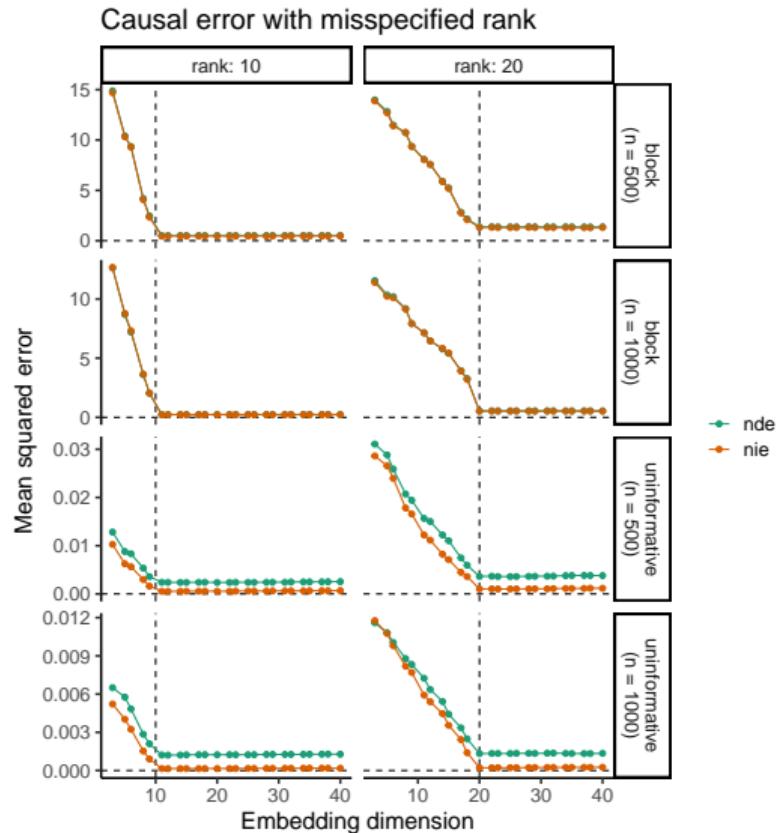
$$\begin{aligned}\sqrt{n \hat{\sigma}_{\text{nde}}^2} (\hat{\Psi}_{\text{nde}} - \Psi_{\text{nde}}) &\rightarrow \mathcal{N}(0, 1), \text{ and} \\ \sqrt{n \hat{\sigma}_{\text{nie}}^2} (\hat{\Psi}_{\text{nie}} - \Psi_{\text{nie}}) &\rightarrow \mathcal{N}(0, 1).\end{aligned}$$

where $\hat{\sigma}_{\text{nde}}^2$ and $\hat{\sigma}_{\text{nie}}^2$ are rather unfriendly variance estimators derived via the delta method and the previous theorem.

A useful cancellation

$$\hat{\Psi}_{\text{nie}} = \hat{\theta}_t \hat{\beta}_x \rightarrow \theta_t Q^T Q \beta_x = \theta_t \beta_x = \Psi_{\text{nie}}$$

Simulations show that overestimating the embedding dimension is okay



Application to Glasgow data

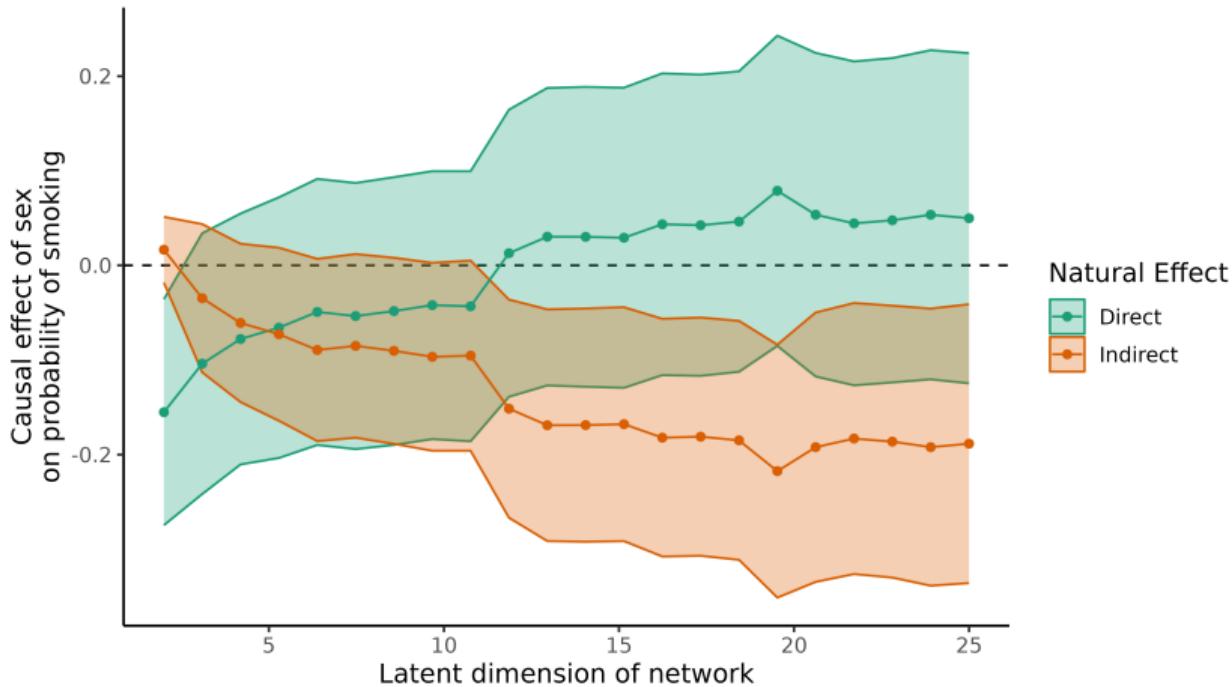
We fit the following regressions

$$\text{smoking} \sim \text{sex} + \text{age} + \text{church} + X\hat{a}t$$

$$X\hat{a}t \sim \text{sex} + \text{age} + \text{church}.$$

smoking a zero-one indicator

Application to Glasgow data



- No direct effect of sex on smoking
- Indirect social effect leads girls to smoke more
- Interventions should try to disrupt social mechanisms

Takeaway 1: network regression is straightforward and easy

Use ordinary least squares and principal components analysis to fit models

$$\mathbb{E}[Y_i | T_i, C_{i\cdot}, X_{i\cdot}] = \beta_0 + T_i \beta_t + C_{i\cdot} \beta_c + X_{i\cdot} \beta_x$$

$$\mathbb{E}[X_{i\cdot} | T_i, C_{i\cdot}] = \theta_0 + T_i \theta_t + C_{i\cdot} \Theta_c$$

No distributional assumptions needed!

Takeaway 2: network embeddings might not be confounders

Current practice: estimate \hat{X} , blindly throw into regression to control for
“confounding”

This will lead to overcontrol bias!

Takeaway 2: network embeddings might not be confounders

When latent positions are confounders:

$$\mathbb{E}[Y_i | T_i, C_{i\cdot}, X_{i\cdot}] = \beta_0 + T_i \underbrace{\beta_t}_{\text{average treatment effect}} + C_{i\cdot} \beta_c + X_{i\cdot} \beta_x$$

When latent positions are mediators:

$$\mathbb{E}[Y_i | T_i, C_{i\cdot}, X_{i\cdot}] = \beta_0 + T_i \underbrace{\beta_t}_{\text{natural direct effect}} + C_{i\cdot} \beta_c + X_{i\cdot} \underbrace{\beta_x}_{\text{effect of } X \text{ on } Y}$$

$$\mathbb{E}[X_{i\cdot} | T_i, C_{i\cdot}] = \theta_0 + T_i \underbrace{\theta_t}_{\text{effect of } T \text{ on } X} + C_{i\cdot} \Theta_c$$

Thank you! Questions?

Future work:

- Varimax rotation for improved interpretability
- Peer effects

Pre-print

Alex Hayes, Mark M. Fredrickson, and Keith Levin. "Estimating Network-Mediated Causal Effects via Spectral Embeddings." arXiv, April 14, 2023. <https://arxiv.org/abs/2212.12041>.

Contact info & slides: <https://www.alexphayes.com>

Appendix

Can we include peer effects in the outcome regression model?

$$Y_i = \beta_0 + T_i\beta_t + C_i.\beta_c + X_i.\beta_x + \underbrace{\beta_{gt} \sum_{j \neq i} \frac{A_{ij}T_j}{\sum_i A_{ij}}}_{\text{interference}} + \underbrace{\beta_{gy} \sum_{j \neq i} \frac{A_{ij}Y_j}{\sum_i A_{ij}}}_{\text{"contagion"}} + \varepsilon_i$$

- Identifying peer effects in these models is very nuanced
 - Not one but two forthcoming manuscripts about this
- When peer effects are identified, ordinary least squares sometimes works
- When peer effects are unidentified, typically due to aliasing with β_x or β_0
 - Aliasing might occur only in the asymptotic limit
 - Might already be accounting for peer effects via aliasing

Intervention has downstream impacts on network structure

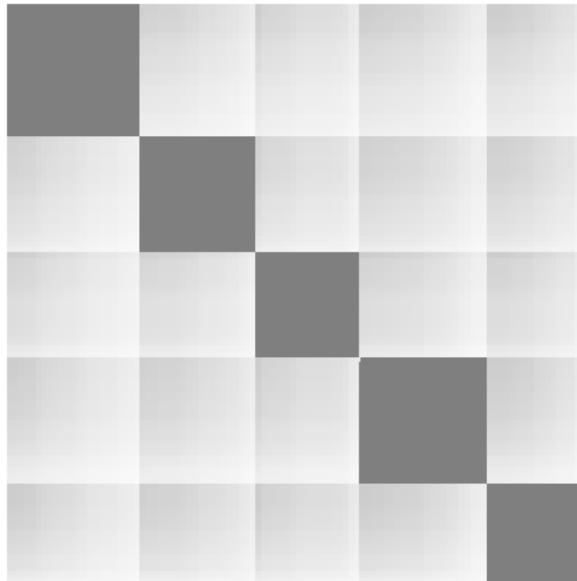


Figure 1: $\mathbb{E}[A | X]$ prior to intervention.

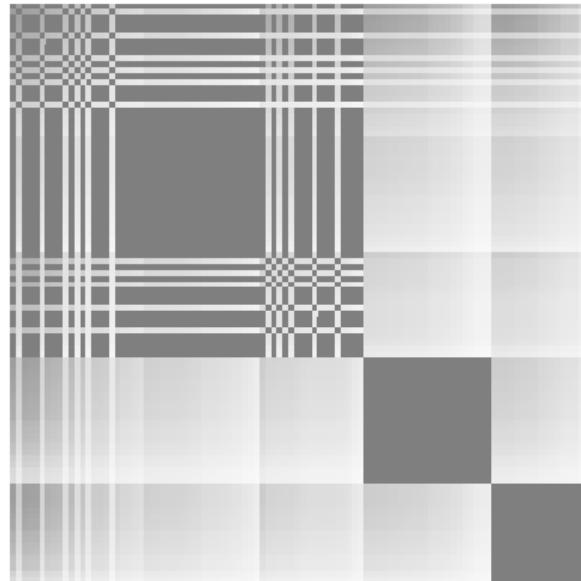


Figure 2: $\mathbb{E}[A | X]$ after intervention.

Intervention has downstream impacts on network structure

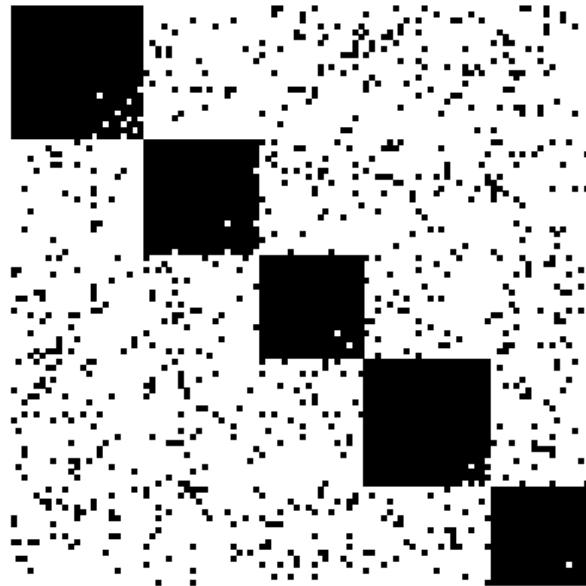


Figure 3: Realized network before intervention

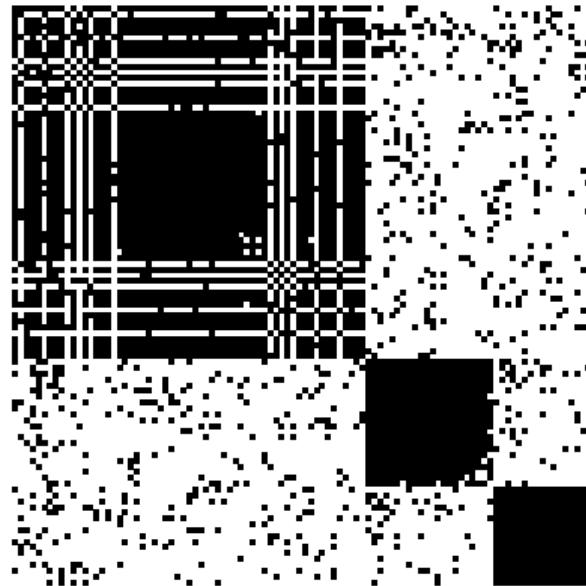


Figure 4: Realized network after intervention

Interpreting interventions on social group membership

Treatment T_i is allowed to cause:

- Changes in popularity within a group
- Movement to a new friend group
- Becoming a member of a new friend group while remaining in current friend group
- Friendships becoming more or less likely between distinct friend groups
- Combinations of the above

References

Michell, L. and P. West (1996). Peer pressure to smoke: The meaning depends on the method. Health Education Research 11(1), 39–49.