

Estimating network-mediated causal effects via spectral embeddings

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Setting



Network $A \in \{0, 1\}^{n \times n}$

Nodal covariates $\mathbf{W}_1, \dots, \mathbf{W}_n \in \mathbb{R}^p$

Nodal outcomes $Y_1, \dots, Y_n \in \mathbb{R}$

Regression incorporating network principle components

Regression with rank- k truncated eigendecomposition of A

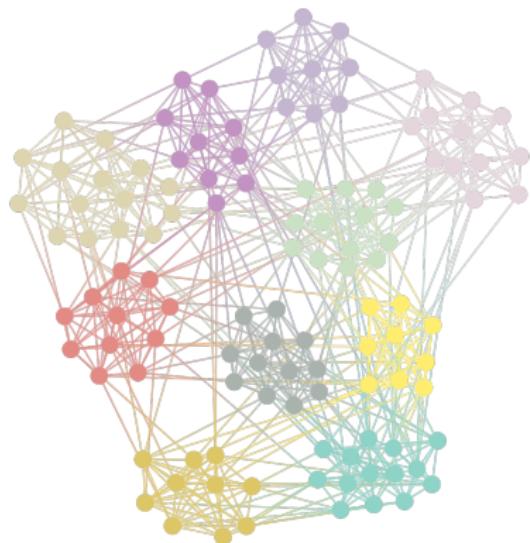
$$\mathbb{E}[A | \mathbf{X}] = \mathbf{X}\mathbf{X}^T = \mathbf{U}\mathbf{S}\mathbf{U}^T$$

$$\mathbb{E}[Y_i | \mathbf{W}_i, \mathbf{X}_i] = \beta_0 + \mathbf{W}_i\beta_w + \mathbf{X}_i\beta_x$$

where $\mathbf{X} = \mathbf{U}\mathbf{S}^{1/2} \in \mathbb{R}^{n \times k}$ is the adjacency spectral embedding,
i.e. principle components, of the network

β_x has a nice interpretation under stochastic blockmodels

Network model: stochastic blockmodel (SBM)



Degree-corrected SBM

k “blocks” or communities

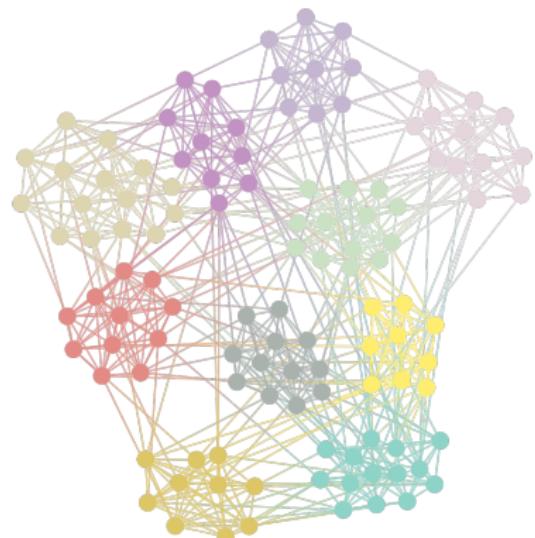
$B \in [0, 1]^{k \times k}$ inter-block edge probabilities

$z(i) \in \{1, \dots, k\}$ node i 's block

$\gamma_i \in [0, 1]$ node i 's popularity

$$\mathbb{P}[A_{ij} = 1 \mid z, \gamma] = \gamma_i \cdot B_{z(i), z(j)} \cdot \gamma_j$$

Network model: broad generalization of the SBM



Random dot product graphs

$$A_{ij} \mid \mathbf{X} \sim \text{Bernoulli}\left(\mathbf{x}_i^T \mathbf{x}_j\right)$$

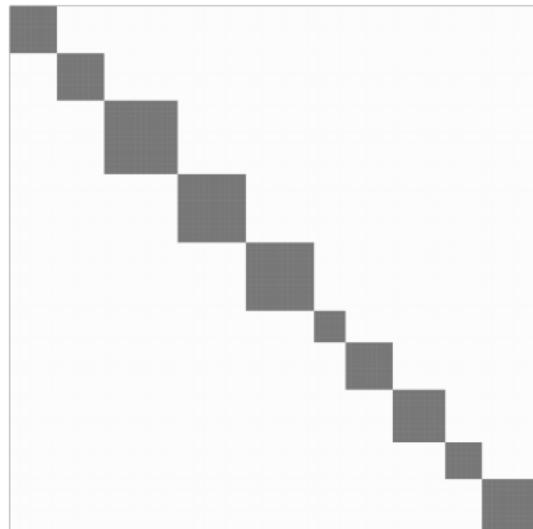
$$\mathbf{X}_i \sim F$$

F a k -dimensional inner-product distribution

$$(A, \mathbf{X}) \sim \text{RDPG}(F, n)$$

Random dot product graphs have low rank structure

Population adjacency $\mathbb{E}[A | \mathbf{X}]$



Sample adjacency A



Stochastic block model with rank $k = 10$

Interpreting network regression

Regression with rank- k truncated eigendecomposition of A

$$\mathbb{E}[A \mid \mathbf{X}] = \mathbf{X}\mathbf{X}^T = \mathbf{U}\mathbf{S}\mathbf{U}^T$$

$$\mathbb{E}[Y_i \mid \mathbf{W}_i, \mathbf{X}_i] = \beta_0 + \mathbf{W}_i\beta_w + \mathbf{X}_i\beta_x$$

β_x roughly “effect of being in block $z(i)$ with popularity γ_i ”, but only identifiable up to rotation

Regression incorporating network principle components

This has been done in [Le and Li \(2021\)](#)!

$$\mathbb{E}[Y_i | \mathbf{W}_i, A] = \mathbf{W}_i \beta_w + \xi + \alpha$$

Let S_k be truncated eigenspace of $\mathbb{E}[A | \mathbf{X}]$ and define

$\mathcal{R} = \text{col}(\mathbf{W}) \cap S_k$. Require $\xi \in \mathcal{R}$, $\mathbf{W}_i \beta_w \perp \mathcal{R}$ and $\alpha \perp \mathcal{R}$

$$\mathbb{E}[Y_i | \mathbf{W}_i, A] = \underbrace{\mathbf{W}_i \beta_w}_{\text{covariate effect}} + \underbrace{\mathbf{W}_i \theta}_{\text{shared effect}} + \underbrace{\alpha}_{\text{network effect}}$$

AddHealth data from Le and Li (2021)

- Data on 2,152 high school students (nodes)
- 7,986 self-reported friendships (edges)
- Outcome: measure of mental health
- Covariates: race, sex, grade in school

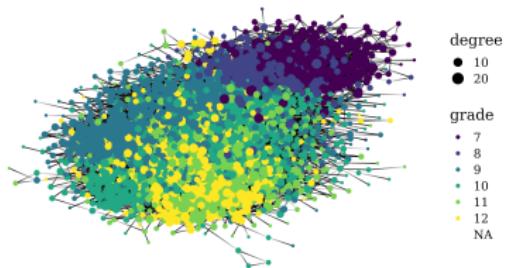


Figure 1: Grade based homophily in a high school social network.

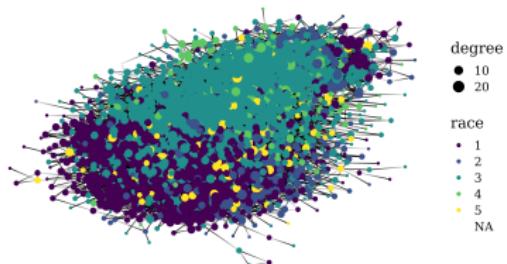


Figure 2: Race based homophily in a high school social network.

Results when applied to AddHealth data

- k estimated to be 9
- $\dim(\mathcal{R})$ estimated to be zero, no network-outcome confounding
- β : significant sex and grade effects conditional on network
- β : weak effect of race conditional on network, especially when compared to OLS estimates

Results when applied to AddHealth data: a mystery

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Why does controlling for latent position in network make the race effect go away?

My hypothesis: race causes group membership, which in turn causes mental health outcomes, i.e. network position mediates rather than confounds race \leftrightarrow mental health relationship

Causal network regression



Network $A \in \{0, 1\}^{n \times n}$

Nodal covariates $\mathbf{W}_1, \dots, \mathbf{W}_n \in \mathbb{R}^p$

Nodal outcomes $Y_1, \dots, Y_n \in \mathbb{R}$

Partition $\mathbf{W}_i = (T_i, \mathbf{C}_i)$

Treatment $T_i \in \{0, 1\}$

Controls $\mathbf{C}_i \in \mathbb{R}^p$

No interference or contagion!

Mediation in network-linked data for low-rank network models

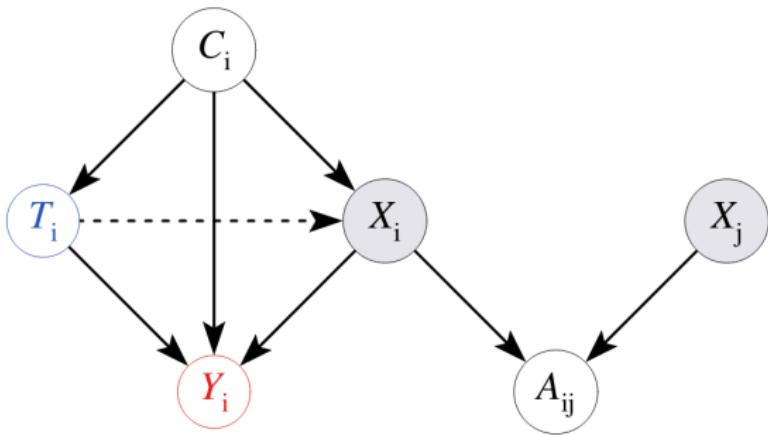


Figure 3: Directed acyclic graph (DAG) relating for node i in a reduced rank network regression model. Portions of the DAG corresponding to nodes $j \neq i$ are omitted. X_i and X_j are not observed.

Causal estimands for mediated effects

There are a couple causal effects of interest

$$\Psi_{\text{ate}}(t, t^*) = \mathbb{E}[Y_t - Y_{t^*}] = \Psi_{\text{nde}}(t, t^*) + \Psi_{\text{nie}}(t, t^*),$$

$$\Psi_{\text{cde}}(t, t^*, \mathbf{x}) = \mathbb{E}[Y_{t \mathbf{x}} - Y_{t^* \mathbf{x}}],$$

$$\Psi_{\text{nde}}(t, t^*) = \mathbb{E}[Y_t \mathbf{x}_{t^*} - Y_{t^*} \mathbf{x}_{t^*}], \text{ and}$$

$$\Psi_{\text{nie}}(t, t^*) = \mathbb{E}[Y_t \mathbf{x}_t - Y_t \mathbf{x}_{t^*}].$$

Primarily interested in $\Psi_{\text{nde}}(t, t^*)$ and $\Psi_{\text{nie}}(t, t^*)$, natural direct and natural indirect effects.

Non-parametric causal identification

Proposition (non-parametric identification, Imai et al. (2010))

Under our DAG, $\Psi_{nde}(t, t^*)$ and $\Psi_{nie}(t, t^*)$ are identified, as

$$\mathbb{E}[Y_t \mathbf{x}_{t^*}] = \iint \mathbb{E}[y \mid T = t, \mathbf{c}, \mathbf{x}] p(\mathbf{x} \mid \mathbf{c}, T = t^*) p(\mathbf{c}) d\mathbf{x} d\mathbf{c}.$$

i.e. under assumptions in DAG our counterfactual estimand is strictly a function of observable functionals

Semi-parametric regression assumptions

Further, if

$$\underbrace{\mathbb{E}[Y | T, \mathbf{C}, \mathbf{X}]}_{\mathbb{R}} = \underbrace{\beta_0}_{\mathbb{R}} + \underbrace{t}_{\{0,1\}} \underbrace{\beta_t}_{\mathbb{R}} + \underbrace{\mathbf{c}}_{\mathbb{R}^{1 \times p}} \underbrace{\beta_c}_{\mathbb{R}^p} + \underbrace{\mathbf{x}}_{\mathbb{R}^{1 \times k}} \underbrace{\beta_x}_{\mathbb{R}^k}$$
$$\underbrace{\mathbb{E}[\mathbf{X} | T, \mathbf{C}]}_{\mathbb{R}^{1 \times k}} = \underbrace{\boldsymbol{\theta}_0}_{\mathbb{R}^{1 \times k}} + \underbrace{t}_{\{0,1\}} \underbrace{\boldsymbol{\theta}_t}_{\mathbb{R}^{1 \times k}} + \underbrace{\mathbf{c}}_{\mathbb{R}^{1 \times p}} \underbrace{\boldsymbol{\Theta}_c}_{\mathbb{R}^{p \times k}}$$

Then:

$$\Psi_{\text{cde}}(t, t^*, \mathbf{x}) = \Psi_{\text{nde}}(t, t^*) = (t - t^*) \beta_t$$

$$\Psi_{\text{nie}}(t, t^*) = (t - t^*) \boldsymbol{\theta}_t \beta_x.$$

What this means for network regressions

Suppose the latent positions \mathbf{X} are mediators. Then:

- β_t is the natural direct effect of T on Y , not the average treatment effect
- If we compute a separate regression, we can compute the natural indirect effect of T on Y (i.e. race causes group memberships causes mental health outcomes)
- These two effects add up to the average treatment effect
- Ordinary least squares ignoring the network structure can be used to estimate the average treatment effect

A causal re-interpretation of Le and Li (2021)'s results

Recall results (new causal interpretations in cyan)

- k estimated to be 9
- $\dim(\mathcal{R})$ estimated to be zero, no network outcome confounding race, grade and sex do not perfectly cause membership in any friend group
- β : significant sex and grade direct effects
- β : weak direct effect of race estimated by network regression, strong total effect of race estimated by ordinary least squares
⇒ large indirect effect of race

Constructing purpose-built causal estimators

Recall that we assume

$$\mathbb{E}[Y \mid T, \mathbf{C}, \mathbf{X}] = \beta_0 + t \beta_t + \mathbf{c} \beta_c + \mathbf{x} \beta_x$$

$$\mathbb{E}[\mathbf{X} \mid T, \mathbf{C}] = \boldsymbol{\theta}_0 + t \boldsymbol{\theta}_t + \mathbf{c} \boldsymbol{\Theta}_c$$

and we want to estimate

$$\Psi_{\text{cde}}(t, t^*, \mathbf{x}) = \Psi_{\text{nde}}(t, t^*) = (t - t^*) \beta_t$$

$$\Psi_{\text{nie}}(t, t^*) = (t - t^*) \boldsymbol{\theta}_t \beta_x.$$

Standard to fit two regressions and multiply coefficients to estimate indirect effect ([VanderWeele and Vansteelandt, 2014](#))

Adjacency spectral embedding

Lemma (Lyzinski et al. (2015), Lemma 5)

Suppose that $(A, \mathbf{X}) \sim \text{RDPG}(F, n)$. Then, letting $\widehat{\mathbf{X}}_i \in \mathbb{R}^d$ denote the i -th row of $\widehat{\mathbf{X}}$, there exists a universal constant C and a sequence of orthogonal matrices $Q_n \in \mathbb{R}^{k \times k}$ such that eventually,

$$\max_{i \in [n]} \|Q_n \widehat{\mathbf{X}}_i - \mathbf{X}_i\| \leq \frac{C \log n}{\sqrt{n}}.$$

This occurs even if A is observed with sub-gamma noise (Levin et al., 2022).

Our estimator: plug ASE into ordinary least squares

Use least squares (with robust standard errors) to estimate the regression coefficients, then plug into causal estimators

$$\mathbf{W} = \begin{bmatrix} 1 & T & \mathbf{C} \end{bmatrix} \in \mathbb{R}^{n \times (1+1+p)}.$$

For estimates $\hat{\mathbf{X}}$ of \mathbf{X} , possibly equal to \mathbf{X} itself, we estimate θ_0 , θ_t , and Θ_c

$$\begin{bmatrix} \hat{\theta}_0(\hat{\mathbf{X}}) \\ \hat{\theta}_t(\hat{\mathbf{X}}) \\ \hat{\Theta}_c(\hat{\mathbf{X}}) \end{bmatrix} = (\mathbf{W}^T \mathbf{W})^{-1} \mathbf{W}^T \hat{\mathbf{X}}.$$

Our idea: plug ASE into ordinary least squares

$$\mathbf{w}(\hat{\mathbf{X}}) = \begin{bmatrix} 1 & \boldsymbol{\tau} & \mathbf{C} & \hat{\mathbf{X}} \end{bmatrix} \in \mathbb{R}^{n \times (1+1+p+k)}$$

Then

$$\begin{bmatrix} \hat{\beta}_0(\hat{\mathbf{X}}) \\ \hat{\beta}_t(\hat{\mathbf{X}}) \\ \hat{\beta}_c(\hat{\mathbf{X}}) \\ \hat{\beta}_x(\hat{\mathbf{X}}) \end{bmatrix} = \left[\mathbf{w}(\hat{\mathbf{X}})^T \mathbf{w}(\hat{\mathbf{X}}) \right]^{-1} \mathbf{w}(\hat{\mathbf{X}})^T \mathbf{Y}.$$

Our idea: plug ASE into ordinary least squares

Plug estimates into standard product-of-coefficients estimator

$$\begin{aligned}\hat{\Psi}_{\text{cde}}(\hat{\mathbf{X}}) &= \hat{\Psi}_{\text{nde}}(\hat{\mathbf{X}}) = (t - t^*) \hat{\beta}_t(\hat{\mathbf{X}}) \\ \hat{\Psi}_{\text{nie}}(\hat{\mathbf{X}}) &= (t - t^*) \hat{\theta}_t(\hat{\mathbf{X}}) \hat{\beta}_x(\hat{\mathbf{X}})\end{aligned}$$

Theoretical results

Theorem (informal)

Asymptotically, regression coefficients using $\hat{\mathbf{X}}$ and \mathbf{X} converge to the same distribution under generic low-rank models for A with i.i.d. sub-gamma noise

Corollary (informal)

Regression coefficients based on $\hat{\mathbf{X}}$ are asymptotically normal and converge at \sqrt{n} -rates.

Corollary (informal)

$\hat{\Psi}_{cde}(\hat{\mathbf{X}})$ and $\hat{\Psi}_{nie}(\hat{\mathbf{X}})$ are asymptotically normal and converge at \sqrt{n} -rates.

Rotational unidentifiability of mediator coefficients

There exists some unknown orthogonal rotation Q such that

$$\sqrt{n} \begin{pmatrix} \widehat{\boldsymbol{\theta}}_0(\widehat{\mathbf{X}}) Q^T - \boldsymbol{\theta}_0 \\ \widehat{\boldsymbol{\theta}}_t(\widehat{\mathbf{X}}) Q^T - \boldsymbol{\theta}_t \\ \widehat{\boldsymbol{\Theta}}_c(\widehat{\mathbf{X}}) Q^T - \boldsymbol{\Theta}_c \end{pmatrix} \rightarrow \text{Normal}(0, \Sigma_{\theta})$$

Rotational unidentifiability of outcome coefficients

There exists some unknown orthogonal rotation Q (same as in last slide) such that

$$\sqrt{n} \begin{pmatrix} \hat{\beta}_0(\hat{\mathbf{X}}) - \beta_0 \\ \hat{\beta}_t(\hat{\mathbf{X}}) - \beta_t \\ \hat{\beta}_c(\hat{\mathbf{X}}) - \beta_c \\ Q \hat{\beta}_x(\hat{\mathbf{X}}) - \beta_x \end{pmatrix} \rightarrow \text{Normal}(0, \Sigma_{\beta})$$

Rotational unidentifiability of β_x and θ_t cancel each other out in causal estimator for $\Psi_{\text{nie}}(t, t^*)$!

Re-analyzing the AddHealth data with our estimators

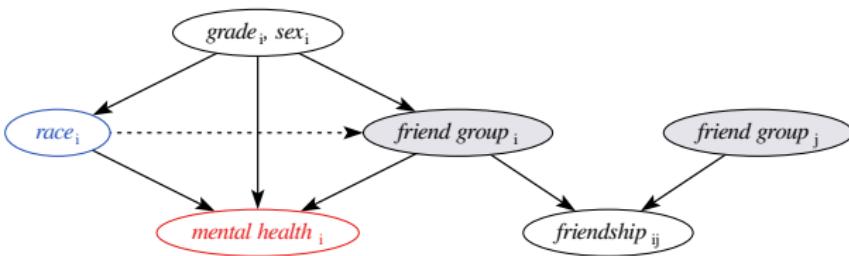
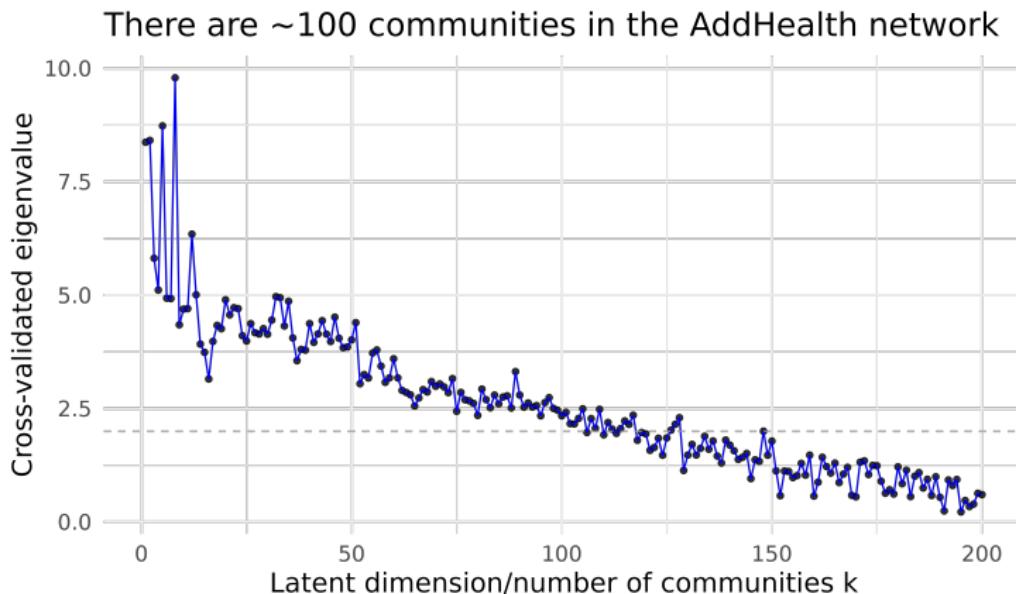


Figure 4: Assumptions about the causal structure of the AddHealth data

Estimation: need to choose dimension k of spectral embedding

Choosing the rank of the network

- Use cross-validated eigenvalues by [Chen et al. \(2021\)](#)
- Check sensitivity of results to choice of k



Mediated causal effects in the AddHealth data

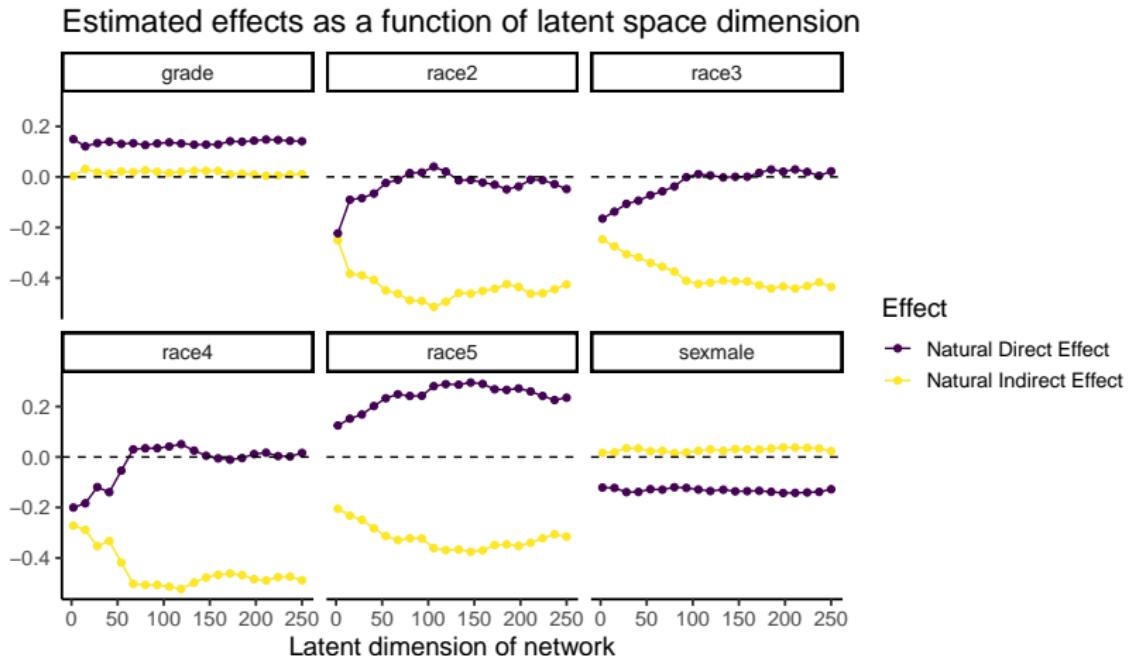


Figure 5: Point estimates of natural direct and indirect effects in the AddHealth dataset as a function of varying embedding dimension k .

Thank you! Questions?

Interventions on a network

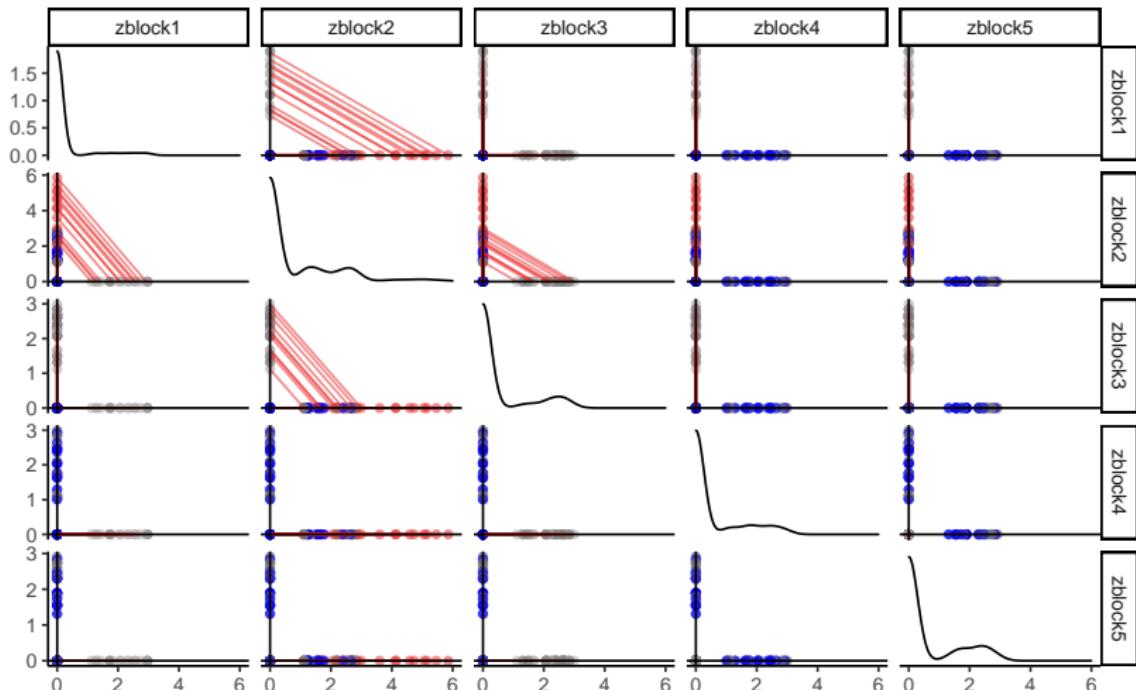


Figure 6: Canonical intervention when \mathbf{C} is highly informative.

Interventions on a network

$$\mathbb{E}[\mathbf{X} \mid T, \mathbf{C}] = \underbrace{\theta_0}_{\mathbb{R}^{1 \times k}} + \underbrace{t}_{\{0,1\}} \underbrace{\theta_t}_{\mathbb{R}^{1 \times k}} + \underbrace{\mathbf{c}}_{\mathbb{R}^{1 \times p}} \underbrace{\Theta_c}_{\mathbb{R}^{p \times k}}, + \underbrace{t}_{\{0,1\}} \underbrace{\mathbf{c}}_{\mathbb{R}^{1 \times p}} \underbrace{\Theta_{tc}}_{\mathbb{R}^{p \times k}}$$

In Figure 6, \mathbf{C} are latent parameters for a DC-SBM and
 $\theta_0 = \vec{0}, \theta_t = \vec{0}, \Theta_c = I_k$ and

$$\Theta_{tc} = \begin{bmatrix} -1 & 2 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 1 & -1 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix}$$

Overcontrol bias from causal misspecification

When \mathbf{X} is a mediator

$$(t - t^*) \beta_t = \Psi_{nde}(t, t^*) = \Psi_{ate}(t, t^*) - \Psi_{nie}(t, t^*)$$

When \mathbf{X} is a confounder

$$(t - t^*) \beta_t = \Psi_{ate}(t, t^*)$$

Abstract

The last several years have seen a renewed and concerted effort to incorporate network data into standard tools for regression analysis, and to make network-linked data legible to practicing scientists. Thus far, this literature has primarily developed tools to infer associative relationships between nodal covariates and network structure. In contrast, we augment a statistical model for network regression with counterfactual assumptions and show how causal effects on a network can be partitioned into a direct effect that is uninfluenced by the network, and an indirect effect that is induced by homophily. The method is a conceptually straightforward integration of random dot product models for networks into the well-known product-of-coefficients mediation estimator.

References

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