Lecture 21: Interference and Spillover Effects

POL-GA 1251
Quantitative Political Analysis II
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Motivation

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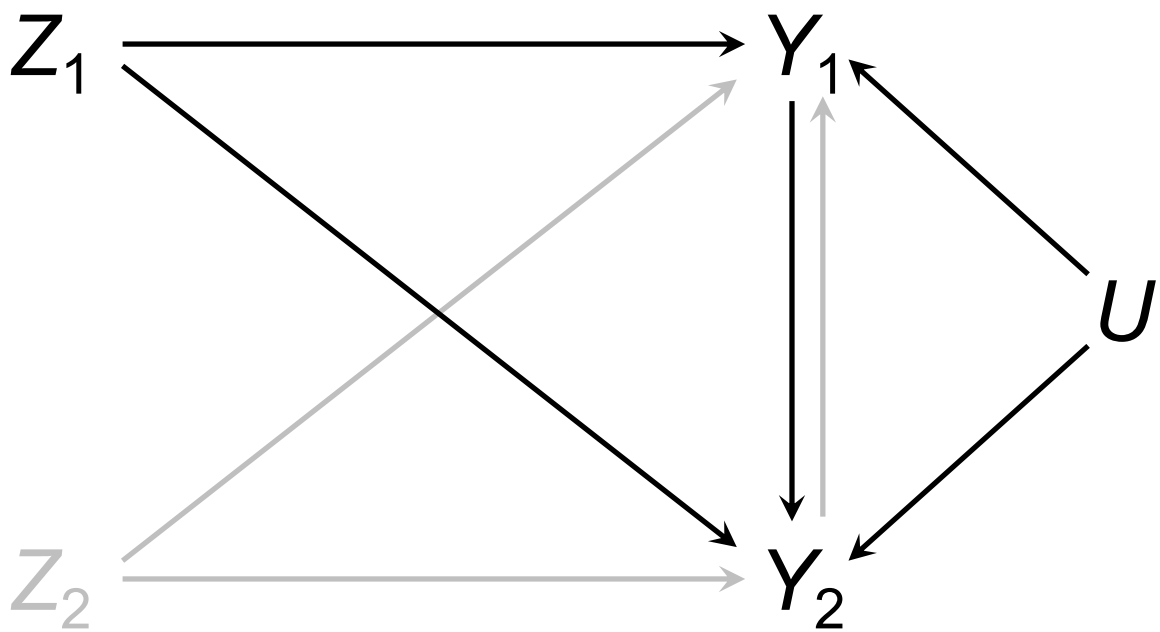
- Most crucially, this rules out "interference," through which your potential outcomes might depend on *others'* treatment assignment.



Examples

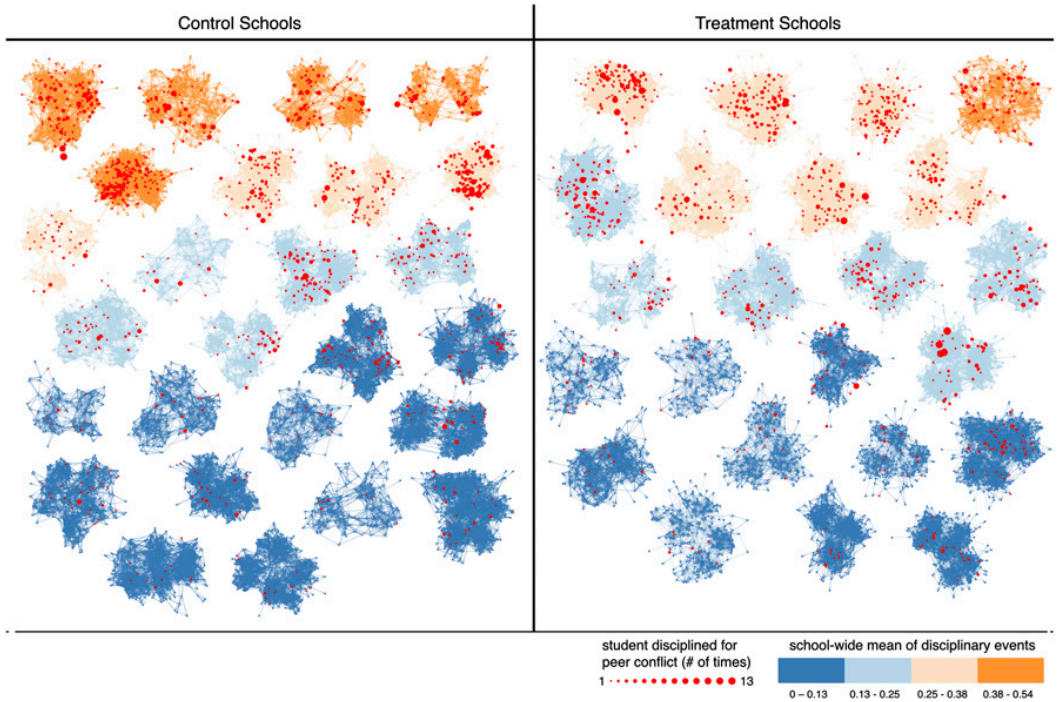
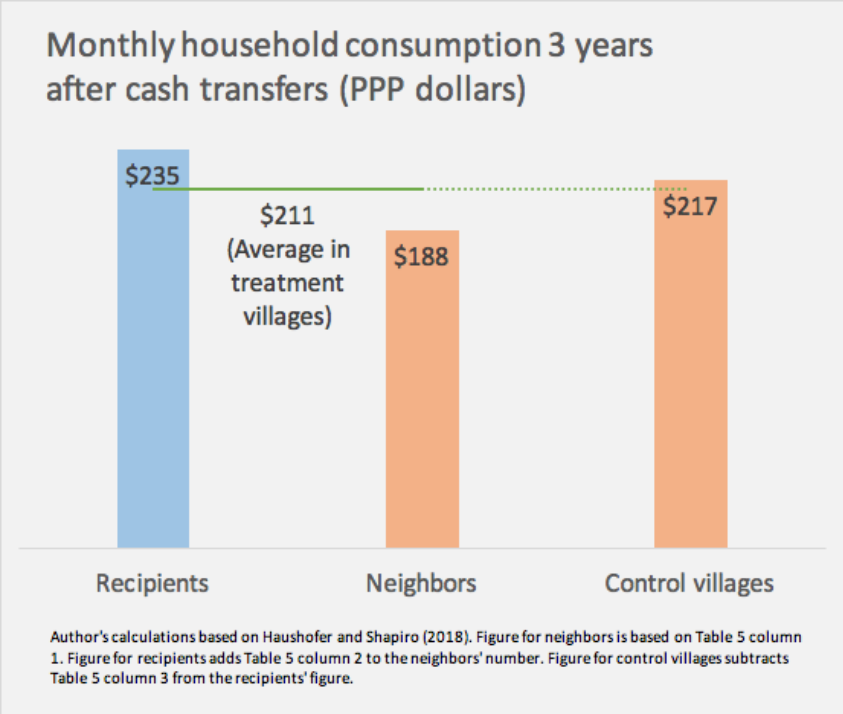


Fig. 1. Overall school climate results: distribution of disciplinary events throughout school networks, comparing treatment, and control schools. Visualization of the effect of treatment on disciplinary reports of peer conflict among the 49 schools that provided administrative data (26 in control, 23 in treatment). Color coding reveals the average number of times each student in the school was disciplined for peer conflict, from dark blue (little conflict) to dark orange (many disciplinary events; higher concentration of dark oranges among control schools). Student nodes are colored red when the student was disciplined for conflict, and their node is scaled to the number of times they were disciplined during the year.

- Aronow et al. (2016) randomly "seeded" schools with kids trained in anti-bullying.
- How to measure effects on peers?
- Need to measure peer networks and specify channels of indirect exposure. What if there is error in these?

Examples



- Haushofer & Shapiro (2018) randomly assigned households within randomly selected villages to receive cash transfers.
- Allows for between-village and within-village analysis.
- So is there "an" effect of transfers? Or multiple?
- How do effects depend on *how many* are treated?
- How do effects depend on *whom* is treated? Consider two business partners in a village...

Examples

TABLE 1. Possible Outcomes under placebo protocol

		Probability of Event Occurring	Voting Rate of Answerer	Voting Rate of Person Who Did Not Answer Door
GOTV	Door Answered	π	$\mu_1 + T$	$\mu_2 + S$
	No Answer	$1 - \pi$	N.A.[a]	μ_3
Recycling	Door Answered	π	μ_1	μ_2
	No Answer	$1 - \pi$	N.A.	μ_3

[a] N.A. = Not applicable.

- Nickerson (2008) used a placebo-controlled design to estimate within-household spillovers.
- To what extent are such spillovers due to *contagion*?

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- Define $\mathbf{z}_{-i}$ as assignments to all units other than i.
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- Experiment randomly assigns $\mathbf{Z}$. Define $\mathbf{Z}_{-i}$ as above.
- Observed outcome is $Y_i = y_i(\mathbf{Z}) = y_i(Z_i, \mathbf{Z}_{-i})$.

Analytical approaches

Three approaches to analyzing spillover effects:

1. Arbitrary spillover effects, when you can specify the network structure for these spillovers (Aronow & Samii 2017).
2. When spillover structure is unknown but you know it is contained within groups ("partial interference"; Hudgens & Halloran 2008) or spatially localized (Aronow, Samii, & Wang 2020).
3. When studying contagion as a mechanism for spillover effects (Manski 2013; Ogburn & Vanderweele 2017; Imai & Jiang 2019).

See Aronow, Eckles, Samii, & Zonszein (2019) for a review.

Arbitrary but known spillover networks

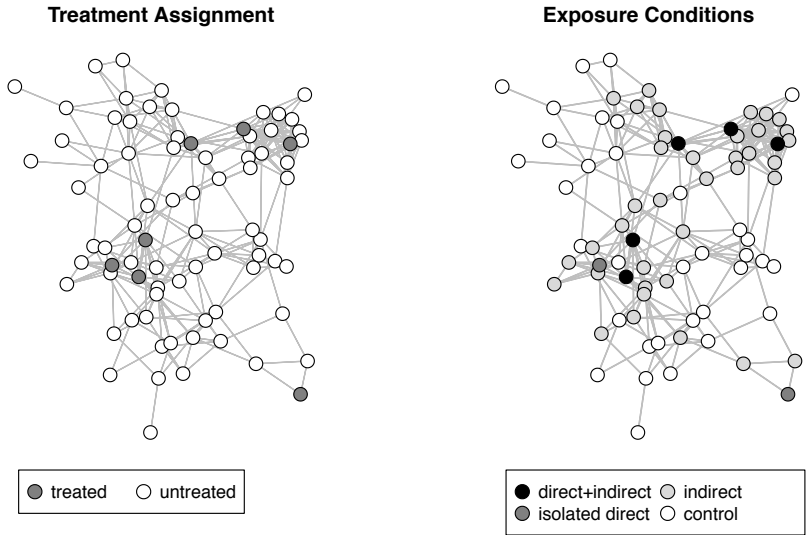

FIG 1. *Illustration of a treatment assignment (left) and then treatment-induced exposures (right) for one of the school classes in the study. Each dot is a student, and each line represents an undirected friendship tie.*

- "Exposure mapping": $f(\mathbf{z}, \theta_i) = D_i$. Based on substantive judgment about interference network.
- Example: let θ_i be row i in adjacency matrix, and

$$f(\mathbf{z}, \theta_i) = \begin{cases} d_{11}(\text{Direct + Indirect Exposure}): & z_i \mathbf{I}(\mathbf{z}'\theta_i > 0) = 1, \\ d_{10}(\text{Isolated Direct Exposure}): & z_i \mathbf{I}(\mathbf{z}'\theta_i = 0) = 1, \\ d_{01}(\text{Indirect Exposure}): & (1 - z_i)\mathbf{I}(\mathbf{z}'\theta_i > 0) = 1, \\ d_{00}(\text{No Exposure}): & (1 - z_i)\mathbf{I}(\mathbf{z}'\theta_i = 0) = 1 \end{cases}$$

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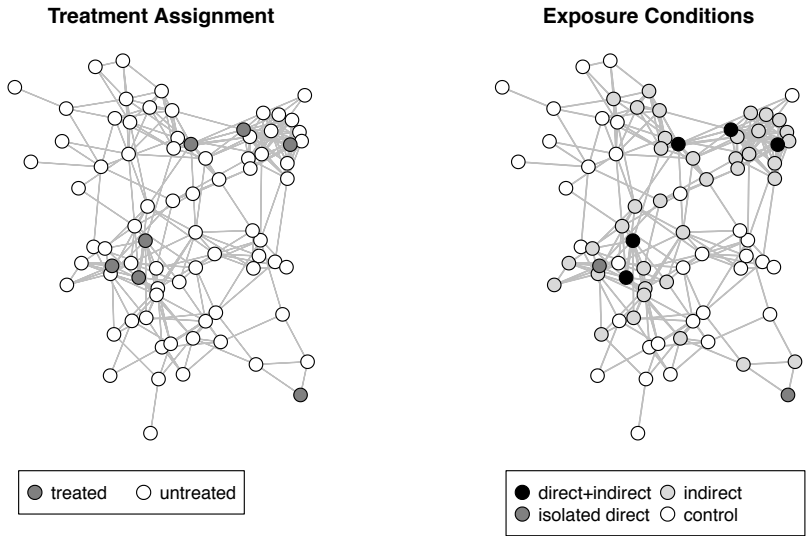

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- However, θ_i may be high-dimensional. So what to do?

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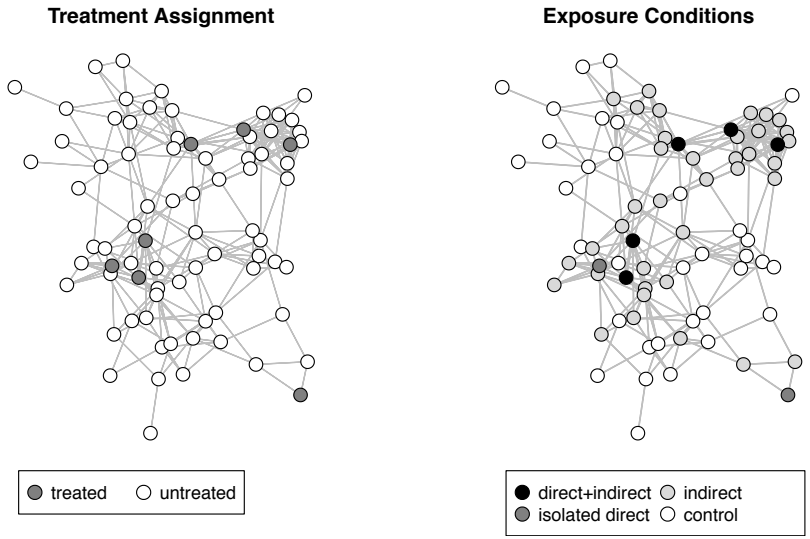

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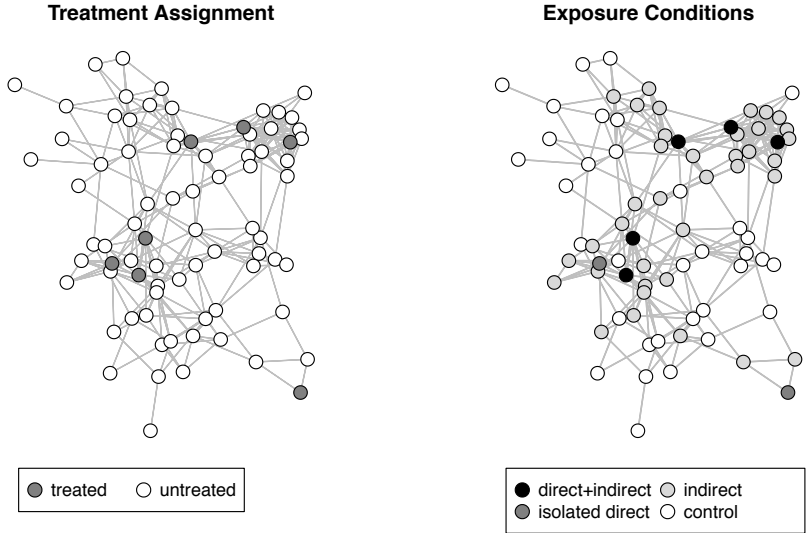
- $\mathbf{Z}$ randomly assigned, but D_i depends on θ_i, so need adjustment.
- However, θ_i may be high-dimensional. So what to do?
- By $\mathbf{Z}$ random assignment, you can calculate $\pi_i(d) = \Pr(D_i = d)$ by simulating alternative randomizations.
- Adjusting for $\pi_i(d)$ is sufficient to identify effect of D_i.

Arbitrary but known spillover networks



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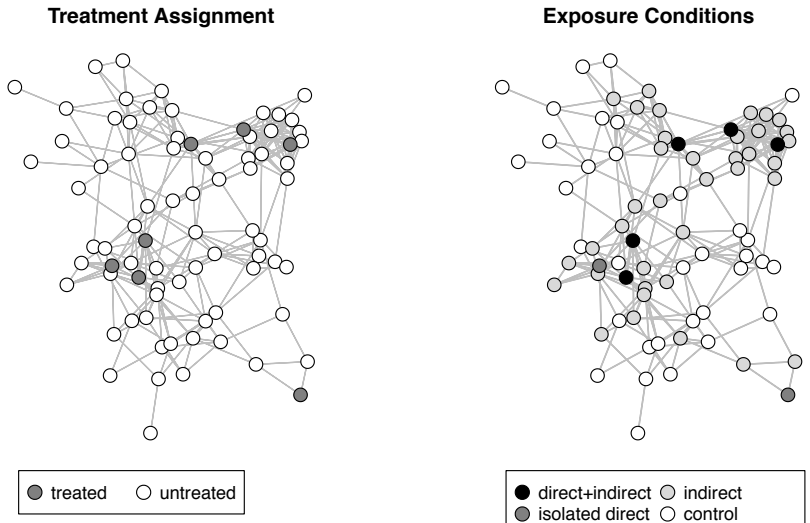
- Define effect of exposure d_k vs d_l:

$$\tau(d_k, d_l) = \frac{1}{N}\sum_{i=1}^{N} y_i(d_k) - \frac{1}{N}\sum_{i=1}^{N} y_i(d_l) = \mu(d_k) - \mu(d_l).$$

- IPW estimator: $\widehat{\mu}_H(d_k) = \dfrac{\sum_{i=1}^{N} \mathbf{I}(D_i{=}d_k)\frac{Y_i}{\pi_i(d_k)}}{\sum_{i=1}^{N} \mathbf{I}(D_i{=}d_k)\frac{1}{\pi_i(d_k)}}$, similar for d_l.
- Inference has to account for complex clustering patterns.

Arbitrary but known spillover networks



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- Implementation through the `interference` R package (Zonszein, Samii, & Aronow, 2019).

For this approach to work

- Need to specify exposure mapping properly (one hop, two hop, more?).
- Need to measure associated interference network properly.

Partial interference and marginal causal effects

Hudgens & Halloran (2008):

- Interference networks are unknown, except
- It is known that spillover is contained within clearly demarcated groups.
- “Partial interference.”
- Propose a way to identify and estimate “margainal” direct and indirect effects.

Partial interference and marginal causal effects

- Suppose $U = \{1,2,3,4,5,6\}$, split into group A $= \{1,2,3\}$ and B $= \{4,5,6\}$.
- Think about unit 1's potential outcome when assigned $z_{1,A} = 0$.

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- Suppose $z_{2,A} = 1$ and $z_{3,A} = 0$. By partial interference, potential outcome for unit 1 is same for assignments in the set,

$$\begin{aligned}\{(0,1,0,\mathbf{z}_{i,B}) : \mathbf{z}_{i,B} \in \Omega_B\} = \{&(0,1,0,0,0,0),(0,1,0,1,0,0),\\ &(0,1,0,0,1,0),(0,1,0,0,0,1),\\ &(0,1,0,1,1,0),(0,1,0,1,0,1),\\ &(0,1,0,0,1,1),(0,1,0,1,1,1)\},\end{aligned}$$

- But it may be that $y_{1,A}(0,1,0,\mathbf{z}_{i,B}) \neq y_{1,A}(0,0,1,\mathbf{z}_{i,B})$, and these each probably differ from $y_{1,A}(0,0,0,\mathbf{z}_{i,B})$ and $y_{1,A}(0,1,1,\mathbf{z}_{i,B})$.

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- So even if we fix $z_{1,A} = 0$, unit 1's potential outcome can vary based on assigned profiles in group A (but not B, if interference is partial).

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- A unit is treated vs control with 50-50 chance.
- Conditional on $z_{1,A} = 0$, it is equally likely that group A treatment assignments are $(0,1,1), (0,0,1), (0,1,1)$, or $(0,0,0)$.

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- Then, the *expected value* of unit 1's outcome given $z_{1,A} = 0$ is the average of unit 1's potential outcomes given group A is assigned as $(0,1,1), (0,0,1), (0,1,1)$, or $(0,0,0)$.
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- This is unit 1's *marginal* potential outcome when $z_{1,A} = 0$.
- A *marginal causal effect* is a contrast between such marginal potential outcomes.

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- Hudgens & Halloran consider two-stage hierarchical randomization:
- First groups are randomly assigned to a level of *treatment saturation*,
- Then, within groups, units are randomly assigned to treatment with probability equal to the group saturation rate.
- The design allows for estimating “total”, “direct”, and “indirect” effects defined in terms of marginal causal effects.

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- We can then average across *groups* to get the sample or population version of these effects.
- Within-group randomization identifies the *direct* effects.
- Between group randomization identifies the *indirect effects*.
- Their combination identify the *total* and *overall* effects.
- Implementation: `interference` R package.

Spatial interference and marginal causal effects

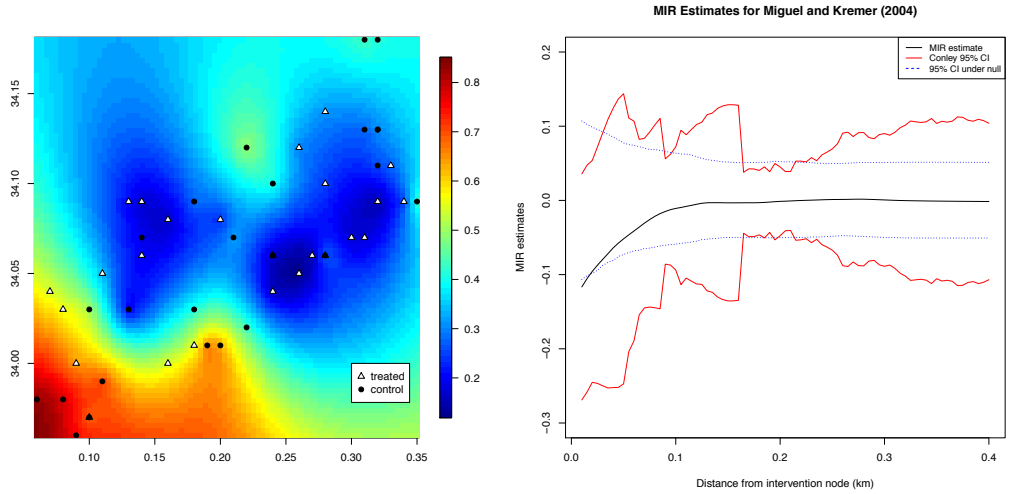
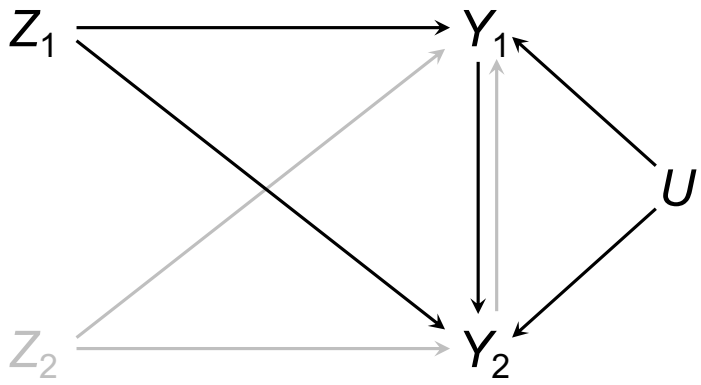


Figure 8: The plot on the left demonstrates both the treatment status and interpolated outcome values from kriging in the experiment. White triangles are schools under control and black circle are treated schools. The color on the map indicates the infection rate. The plot on the right presents results using our methods. The black curve represents the MIR estimate. The red curves are 95% confidence intervals constructed from spatial HAC standard errors. The blue lines are the 95% confidence intervals under sharp null.

- Aronow, Samii, & Wang (2020) analyze *spatial* interference and associated marginal causal effects.
- "Average marginalized response" (AMR): consider outcome at distance d from intervention point. How does it change when treatment is assigned at intervention point, marginalizing over effects from other intervention points?

Contagion



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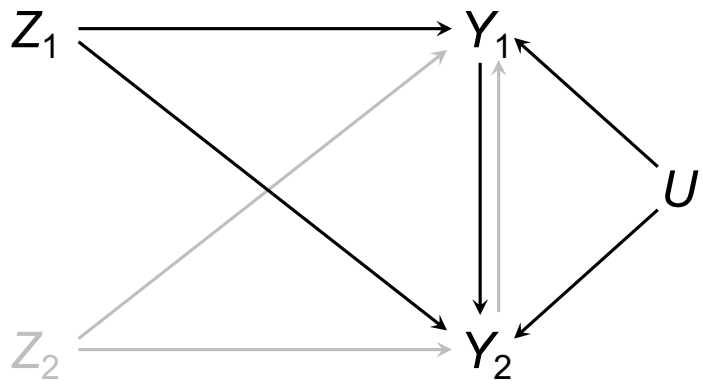

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- When one unit's *outcome* affects another's outcome, this is called *contagion*. (Also, "peer effects," "social effects," "social interactions," etc.)

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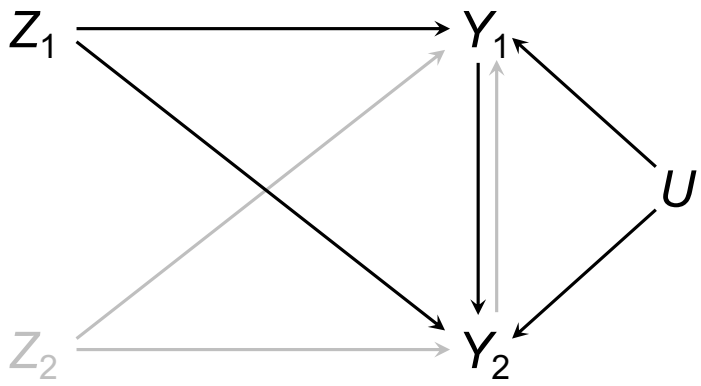
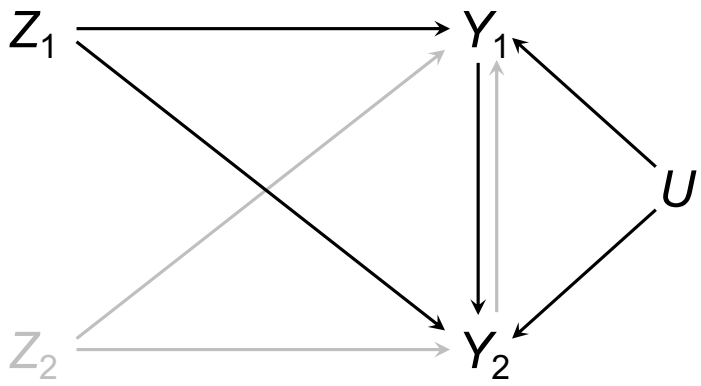


- So far we have treated spillover effects as "reduced form".
- When one unit's *outcome* affects another's outcome, this is called *contagion*. (Also, "peer effects," "social effects," "social interactions," etc.)
- Contagion can be a *mechanism* through which spillover effects occur.
- As such, analyzing contagion is equivalent to doing a *mediation analysis* of spillover effects.

Contagion



- So far we have treated spillover effects as "reduced form".
- When one unit's *outcome* affects another's outcome, this is called *contagion*. (Also, "peer effects," "social effects," "social interactions," etc.)
- Contagion can be a *mechanism* through which spillover effects occur.
- As such, analyzing contagion is equivalent to doing a *mediation analysis* of spillover effects.
- To identify contagion effects, you need identification for *both* the spillover effects *and* the mediation effect.

Contagion

TABLE 1. Possible Outcomes under placebo protocol

		Probability of Event Occurring	Voting Rate of Answerer	Voting Rate of Person Who Did Not Answer Door
GOTV	Door Answered	π	$\mu_1 + T$	$\mu_2 + S$
	No Answer	$1 - \pi$	N.A.[a]	μ_3
Recycling	Door Answered	π	μ_1	μ_2
	No Answer	$1 - \pi$	N.A.	μ_3

[a] N.A. = Not applicable.

Following Imai & Jiang (2019), for those who didn't answer door:

- Avg. indirect effect: $\theta = \frac{1}{N_c} \sum_{i=1}^{N_c} y_{i2}(1, y_{i1}^*(1)) - y_{i2}(0, y_{i1}^*(0))$.
- Avg. contagion: $\gamma(z) = \frac{1}{N_c} \sum_{i=1}^{N_c} y_{i2}(z, y_{i1}^*(1)) - y_{i2}(z, y_{i1}^*(0))$.
- Avg. non-contagion: $\eta(z) = \frac{1}{N_c} \sum_{i=1}^{N_c} y_{i2}(1, y_{i1}^*(z)) - y_{i2}(0, y_{i1}^*(z))$.
- $\theta = \gamma(1) + \eta(0) = \gamma(0) + \eta(1)$.
- Identification requires sequential ignorability.
- Cannot verify, so sensitivity analysis is key

Contagion

TABLE 1. Possible Outcomes under placebo protocol

		Probability of Event Occurring	Voting Rate of Answerer	Voting Rate of Person Who Did Not Answer Door
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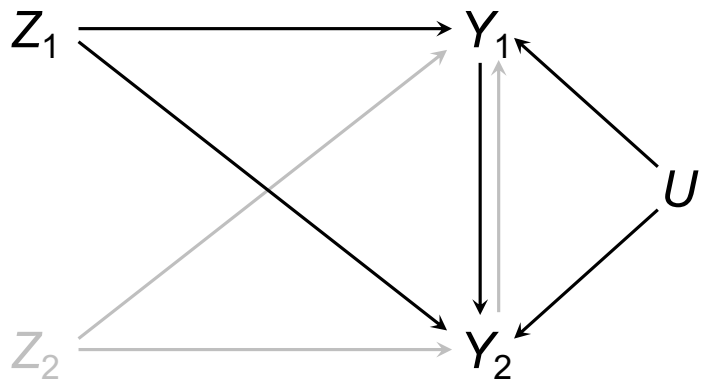
[a] N.A. = Not applicable.

Other approaches to contagion rely on linear-in-means regression specifications, motivated by, e.g., linear complementarity models.

- Manski (1993) is classic analysis, discussing "reflection problem" in trying to assess whether average behavior in a group affects behavior of group members or not.
- See Kline & Tamer (2020) for a current review.
- Jackson (2008) textbook covers strategic complementarity models.

Discussion



- At seminars one often hears "what about SUTVA violations?"
- You don't have to wave your hands helplessly!
- Today we discussed *estimating* spillover effects. See Aronow, Eckles, Samii, Zonszein (2019) for a review.
- Also note: estimators for ATE target marginal causal effects under SUTVA violations of unspecified form (Savje et al. 2019).
- So SUTVA violations do not necessarily undermine our ability to do robust causal inference, although they introduce some complexities.