**CISER** Causal Inference Workshop

#### Session 4: Interference and Spillover Effects

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# SUTVA Assumption

The Stable Unit Treatment Value Assumption (SUTVA) is a fundamental assumption in causal inference.

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SUTVA is violated when the potential outcomes of one unit depend on the treatment assignment of other units.

Such phoneomena are often referred to as interference or spillover effects.

- Social networks
- Transportation networks
- Field experiments

- Let  $\mathbf{Z} = (Z_1, \ldots, Z_N) \in \{0, 1\}^N$  be the treatment assignment vector for all N units.
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- Let Y<sub>i</sub>(Z) be the potential outcome for unit i given treatment assignment vector Z.
- SUTVA assumes that for all i,

$$Y_i(\boldsymbol{Z}) = Y_i(\boldsymbol{Z}')$$
 whenever  $Z_i = Z'_i$ 

The consequence is we can simply write  $Y_i(Z_i)$  instead of  $Y_i(\mathbf{Z})$ .

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The consequence is we can simply write  $Y_i(Z_i)$  instead of  $Y_i(\mathbf{Z})$ .

▶ If SUTVA is violated, we cannot write  $Y_i(Z)$  as  $Y_i(Z_i)$  because  $Y_i$  depends on the treatment assignment of other units.

#### Interference

To represent the dependence of  $Y_i$  on  $Z_{i'}$  for  $i' \neq i$ , we consider a directed graph  $\mathfrak{G}$ .

• Vertices:  $V = \{1, \dots, N\}$ , representing the units.

• Edges: 
$$E = \{(i, i') : Y_i \text{ depends on } Z_{i'}\}$$

The indegree neighbor of unit i is defined as

$$\mathcal{N}_i = \{i' : (i', i) \in E\}.$$

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$$\mathcal{N}_1 = \{4\}$$
  
 $\mathcal{N}_2 = \{1\}$   
 $\mathcal{N}_3 = \{1, 2\}$   
 $\mathcal{N}_4 = \{3\}$ 

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## SUTNVA Assumption

When the interference is present, we assume the **Stable Unit Treatment on Neighborhood Value Assumption** (SUTNVA):

1. For each *i*, for any two treatment assignments  $\mathbf{Z} = (Z_i, Z_{\mathcal{N}_i}, Z_{\mathcal{N}_{-i}})$  and  $\mathbf{Z}' = (Z'_i, Z'_{\mathcal{N}_i}, Z'_{\mathcal{N}_{-i}})$ , we have

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2. For each i,

$$Y_i^{obs} = Y_i(\boldsymbol{Z})$$

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2. For each i,

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Under SUTNVA, we can write the potential outcome as

 $Y_i( Z_i , Z_{\mathcal{N}_i})$ 

direct treatment interference

For demonstration purpose, we consider a simpler case the the interference graph  ${\cal G}$  is undirected.

**Inteference Graph** 



#### Neighbors

$$\mathcal{N}_1 = \{2, 3, 4\}$$
$$\mathcal{N}_2 = \{1, 3\}$$
$$\mathcal{N}_3 = \{1, 2, 4\}$$
$$\mathcal{N}_4 = \{1, 3\}$$

#### **Potential Outcomes**

$$Y_1^{obs} = Y_1(Z_1, Z_2, Z_3, Z_4)$$
  

$$Y_2^{obs} = Y_2(Z_2, Z_1, Z_3)$$
  

$$Y_3^{obs} = Y_3(Z_3, Z_1, Z_2, Z_4)$$
  

$$Y_4^{obs} = Y_4(Z_4, Z_1, Z_3)$$

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The exposure mapping is a function  $g_i : \{0, 1\}^{N_i} \to \mathcal{G}_i$  for all *i*, such that SUTNVA holds for  $G_i = g_i(Z_{\mathcal{N}_i})$ :

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We can write the potential outcomes under interference as

$$Y_i(Z_i, G_i)$$
 with  $G_i = g_i(Z_{\mathcal{N}_i}).$ 

Common choices of exposure mapping:

number of treated neighbors:

$$G_i = \sum_{i' \in \mathcal{N}_i} Z_{i'}$$

proportion of treated neighbors:

$$G_i = N_i^{-1} \sum_{i' \in \mathcal{N}_i} Z_{i'},$$

where  $N_i = |\mathcal{N}_i|$  is the number of neighbors of unit *i*.

heterogeneous interference from neighbors:

$$G_i = \sum_{i' \in \mathcal{N}_i} w_{ii'} Z_{i'},$$

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where  $w_{ii'}$  is usually determined by the distance between their covariates.

trivial exposure mapping:

$$G_i = \mathbf{Z}_{\mathcal{N}_i}$$

Consequences of misspecification of exposure mapping:

Aronow & Samii (2017), Estimating Average Causal Effects Under General Interference. AOAS





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#### Entanglement between Treatment and Interference

Consider the assignment mechanisum:

 $P(\boldsymbol{Z}, \boldsymbol{G} \mid \boldsymbol{X}, \mathbb{Y}, \mathfrak{G})$ 

•  $\mathbf{Z} = (Z_1, \ldots, Z_N)$  is the treatment assignment vector.

- $G = (G_1, \ldots, G_N)$  is the interference exposure vector.
- $X = (X_1, \ldots, X_N)$  is the covariate vector.
- $\mathbb{Y} = \{Y_i(z,g), i = 1, \dots, N : z \in \{0,1\}, g \in \mathcal{G}_i\}$  is all potential outcomes.

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The biggest problem in the inference framework is that G is a deterministic function of Z given all the conditions.

The unconfoundedness condition now becomes:

 $Z_i, G_i \perp Y_i(z,g) \mid X_i$ 

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► Any randomization on the treatment that is independent of X<sub>i</sub> satisfies the unconfoundedness condition.

## Causal Effect

Unit-level direct treatment effect:

$$\tau_i^{(d)}(g) = Y_i(1,g) - Y_i(0,g)$$

Unit-level indirect/spillover treatment effect:

$$\tau_i^{(i)}(g, g'; z) = Y_i(z, g) - Y_i(z, g')$$

Unit-level total treatment effect: (often the most interesting one)

$$\tau_i^{(t)} = Y_i(1,\overline{g}) - Y_i(0,\underline{g})$$

where

$$\overline{g} = g_i(\mathbf{1}), \quad \underline{g} = g_i(\mathbf{0}).$$

The population average treatment effects are defined as the average of the unit-level treatment effects.

#### Average Dose Response Function

We define the following populational average potential outcomes:

$$\mu(z,g) = E[Y_i(z,g) \mid i \in V_g], \quad \forall z \in \{0,1\}, g \in \mathcal{G},$$

where  $V_g = \{i : g \in \mathcal{G}_i\}$  is the set of units with possible exposure g and  $\mathcal{G} = \bigcup_{i=1}^N \mathcal{G}_i$  is the set of all possible exposures.

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It is also called the average dose response function (ADRF).

The population average treatment effects are defined as the contrast of ADRFs.

Now consider the observational study problem.

- ▶ Observed, fixed interference graph 𝔅.
- Observed confounders X.
- Observed treatment assignment Z.
- $\blacktriangleright$  Observed interference exposure G usually computed from Z and  $\mathfrak{G}$ .

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Goal: estimate the casual effects, as well as  $\mu(z,g)$ .

The joint propensity score of (z,g) for unit i is

$$\psi(z;g;x) = P(Z_i = z, G_i = g \mid X_i = x)$$

Assumptions used here:

Unconfoundedness.

• The probability depends on its own covariates  $X_i$  only.

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- Unconfoundedness.
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The joint propensity score works as a balancing score:

$$Y_i(z,g) \perp Z_i, G_i \mid \psi(z;g;X_i) \quad \forall z,g$$

The propensity score can be expanded as

$$\psi(z;g;x) = P(G_i = g \mid Z_i = z, X_i^z = x^z) P(Z_i = z \mid X_i^g = x^g)$$

where  $X_i^g$  and  $X_i^z$  are the covariates that are used to predict  $Z_i$  and  $G_i$ , respectively.

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> The first term is the **neighborhood propensity score**:

$$P(G_i = g \mid Z_i = z, X_i^z = x^z) = \lambda(g; z; x^g)$$

> The second term is the **individual propensity score**:

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They jointly satisfy the unconfoundedness condition:

$$Z_i, G_i \perp Y_i(z,g) \mid \lambda(g;z;X_i^g), \phi(z;X_i^z) \quad \forall z,g$$

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- 1. Stratification on the individual propensity score  $\phi(z; X_i^z)$ :
  - 1.1 Fit  $\phi(1; X_i^z)$  using logistic regression.
  - 1.2 Divide the units into J strata,  $B_1, \ldots, B_J$ , based on the estimated propensity score.

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- 2. Estimation within each stratum:
  - 2.1 Fit  $\lambda(g;z;X^g_i)$  using logistic regression.
  - 2.2 Fit a parametric model  $Y_i(z,g) \sim Z_i + G_i + \hat{\lambda}_i$ .
  - 2.3 For the pair (z,g), for each eligible unit, make a prediction of  $Y_i(z,g)$  using the fitted model.
  - 2.4 The estimator is

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3. The final estimator is

$$\hat{\mu}(z,g) = \sum_{j=1}^{J} \hat{\mu}_j(z,g) \pi_j^g$$

where  $\pi_j^g = |B_j^g|/|B_j|$  is the proportion of units in stratum j with exposure g.

More details in Forastiere, Airoldi, & Mealli (2020). Identication and estimation of treatment and interference effects in observational studies on networks. JASA.

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Why parametric model in the second step: for example,

- A network of N = 1000 units.
- Each unit has  $|\mathcal{N}_i| = 4$  neighbors.
- ▶  $N_t = 500$  units are randomly assigned to treatment.
- We want to estimate  $\mu(1,2)$  for exposure mapping of number of treated neighbors.

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$$\approx 1000 \times \frac{1}{2} \times \binom{4}{2} \times \frac{1}{2^4} \approx 188 \ll 1000.$$

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Use parametric model to "impute" the potential outcomes for other units.

# **Empirical Matching**

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A natural way is to find two subsets of the samples:

$$\mathcal{A} = \{i : Z_i = z, G_i = g\}, \quad \mathcal{B} = \{i : Z_i = z', G_i = g'\}.$$

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- Drawback 1: Small sample size.
- Drawback 2: Correlation.

## Experimental Design

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For the further demonstrations, we consider the following exposure mapping:

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The proportion of treated neighbors.

The total treatment effect becomes

$$\tau^{(t)} = \mu(1,1) - \mu(0,0).$$

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#### An ego-cluster consists of a unit (ego center) and all its neighbors (alters).

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#### In an ego-centric design.

- Find maximal disjoint ego-clusters  $C_1, \ldots, C_K$  from the interference graph.
- Randomly assign half of the ego-clusters to treatment and half to control.
- The total treatment effect is estimated by the difference-in-means estimator on the ego-center's outcomes.

# Ego-Centric Design



Saint-Jacques, Varshney, Simpson, & Xu (2019). Using Ego-Clusters to Measure Network Effects at LinkedIn.

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Advntages:

- Easy control of the interference.
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- ► Easy control of the interference.
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Disadvantages:

- Limited sample size.
- Require sparse interference graph.

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- Find a maximal independent set I from the interference graph. Call the rest of the vertices the auxiliary set A.
- We focus on the units in  $\mathcal{I}$ . Their interference exposure is  $G_{\mathcal{I}} = \Gamma Z_A$ , where  $\Gamma$  is the (normalized) adjacency matrix between  $\mathcal{I}$  and  $\mathcal{A}$ .
- $\triangleright$   $Z_A$  is chosen to maximize the variance of the interference exposure:

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Units in *I* are assigned according to the interference exposure:

$$Z_i = \begin{cases} 1 & \text{if } G_i \geq 0.5 \\ 0 & \text{otherwise} \end{cases}$$

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The total treatment effect is estimated by the difference-in-means estimator on the units in *I*.



Cai, Zhang, & Airoldi (2025). Independent-Set Design of Experiments for Estimating Treatment and Spillover Effects under Network Interference. ICLR.

Advantages:

- Independent control on the treatment and interference.
- Large independent set size (compared to ego-centeric design) with high probability.

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Disadvantages:

- Computation of the maximal independent set is NP-hard.
- Could have bias.
- Require sparse interference graph.

#### In a randomized saturation design,

- Units are divided into K clusters.
- A set of proportions π = {π<sub>1</sub>,..., π<sub>K</sub>} is randomly assigned to the clusters.
   W.L.O.G., assume cluster k is assigned π<sub>k</sub>.
- For the  $N_k$  units in cluster k,  $\pi_k N_k$  units are randomly assigned to treatment and the rest are assigned to control.

▶ The causal effects are estimated by the difference-in-means.





Population: A collection of J clusters of units.

#### Two-step Randomization:

- 1. Randomly generate a *proportion vector*  $\boldsymbol{\pi} = [\pi_1, \dots, \pi_J]$  from  $\boldsymbol{\Pi}$ .
- 2. Randomly assign  $n_j = \lfloor \pi_j N_j \rfloor$  units in cluster j to treatment.

Example: A realization of treatment assignment generated by a randomized saturation design where the realized proportion vector is  $\boldsymbol{\pi} = \begin{bmatrix} \frac{2}{3}, \frac{1}{2}, \frac{1}{6}, \frac{5}{6} \end{bmatrix}$ .

Itreated units

 $\bigcirc$ : control units

Advantages:

- Easy to implement.
- Rough control of the interference.

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- Rough control of the interference.

Disadvantages:

- Bias from inter-cluster interference.
- Require partial interference assumption (in oppose to our local interference assumption).

 $G_i =$ proportion of treated unit in its cluster

- Proposed by Hudgens and Halloran (2008). Toward causal inference with interference. JASA.
- Theoretical properties: Jiang, Imai & Malani (2022). Statistical inference and power analysis for direct and spillover effects in two-stage randomized experiments. Biometrics.

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- Common clustering strategies:
  - Community detection algorithm. (tons of reference here)
  - Randomly assign units to clusters. Ugandar & Yin (2020). Randomized Graph Cluster Randomization.
  - Sample disjoint clusters from the population.
- Critization on poor clustering structures:

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In practice, to estimate the total treatment effect, the proportion vector has K/2 1's and K/2 0's.

# Thank you for joining the workshop!

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