Model-assisted design of experiments in the presence of network correlated outcomes (G.W. Basse & E.M. Airoldi, 2018+)

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2018-10-24

Introduction

Interference: When people have friends

- Observational units are connected – so far, we've been dealing with causal analyses *in a vacuum*.
- Sometimes, it's reasonable to assume that units do not affect one another; often, it's not.
- A central assumption in causal models, necessary for identification results, is the Stable Unit Treatment Value Assumption (Rubin 1978) & (Rubin 1980).
- *Interference* is often defined through the loosening of this assumption (Hudgens and Halloran 2008).

Networks: Are you (still) on facebook too?

- In a population of causally connected units, several types of network structures may arise, each posing unique challenges for statistics.
- Broadly, the central statistical challenge is *"how to account for the presence of connections, or network data, observed pre-intervention, possibly with uncertainty, and often missing"* Basse and Airoldi (2018).

Networks: Two perspectives

- Two main problem settings have been discussed in the causal inference literature
    1. *Network interference*: When the potential outcomes of a given unit are a function of its assigned treatment and that of others.
    2. *Network-correlated outcomes*: When the potential outcomes of units in a network are related through their baseline covariates.
- The first problem has been the subject of much attention in the literature, so Basse and Airoldi (2018) focus on resolving issues in the second setting.

Discussion – a preview

- Question 1:
- Question 2:
- Question 3:

G.W. Basse and E.M. Airoldi, 2018+, *Biometrika*

Goals and Motivation

- *The problem*: "how to assign treatment in a randomized experiment, when the correlation among the outcomes is informed by a network available at the design stage."
- Identify and estimate the causal effect of interference in the presence of confounding induced by correlated outcomes.
- How can information about a network be used to inform randomization strategies for estimating causal effects?

Approach

- Use *model-assisted restricted randomization strategies*, leveraging a static network known pre-intervention.
- Restricted randomization has a long history in experimental design – Basse and Airoldi (2018) build off of this, using strategies that balance covariates properly.

Approach

- Posit a working model for the potential outcomes, conditional on the network known pre-intervention.
- Restrict the set of allowed randomization strategies such that the estimator of interest achieves low MSE.
- In turn, focus on MSE suggests new notions of balance in network-based randomization (related to network degree statistics).

Findings

- Proposed approach maintains design unbiasedness of the difference-in-means estimator, even when the working model is misspecified (i.e., robustness).
- When the working model is correct, inference is improved through higher precision of the estimator of interest.

Notation

- N observational units, indexed $i = 1, \ldots, N$.
- Binary treatment Z, where $Z_i = 1$ denotes assignment to treatment arm.
- Real-valued outcome Y_i, with potential outcomes $Y_i(Z_i)$:
  - $Y_i(1)$ for $Z_i = 1$ and
  - $Y_i(0)$ for $Z_i = 0$.

Assumptions

- *Stable Unit Treatment Value Assumption* (Rubin 1974) & (Rubin 1978).
  - i.e., $Y_i(Z) = Y_i(Z_i)$
  - explicitly disallows network interference
- Finite population setting: recall that potential outcomes $Y(Z)$ are unknown but constant quantities, given Z.

Assumptions

- *Randomized experiment*: only source of variation is the allocation of treatment to units (controlled by experimenter).
- Treatment allocated based on distribution on the space of all binary vectors of length N, i.e., randomization distribution (Imbens and Rubin 2015).

Parameter of interest: ATE

- For illustration, focus on ATE as the inferential target.
- With the notation previously given, the ATE is defined as

$$\tau^* = \frac{1}{N}\sum_{i=1}^{N}\{Y_i(1) - Y_i(0)\}$$

- Focus also on the difference-in-means estimator for the ATE:

$$\hat{\tau}(Y \mid Z) = \frac{\sum_{i=1}^{N} Z_i Y_i}{\sum_{i=1}^{N} Z_i} - \frac{\sum_{i=1}^{N}(1 - Z_i) Y_i}{\sum_{i=1}^{N}(1 - Z_i)}$$

An undirected network

- Let the network be an undirected graph $\mathcal{G}$ over N units, where
  - $\mathcal{G}$ is simply an $N \times N$ binary adjacency matrix A, where all diagonal entries are unary (i.e., $A_{ii} = 1$), and
  - the neighborhood of unit i be the index set $\mathcal{N}_i = \{j : A_{ij} = 1\}$.
- The proposed methodology requires that a network be known at the design stage (pre-specified).

A simplified model

- For illustrative purposes, assume the *normal-sum model*:

$$X_j \sim_{iid} N(\mu, \sigma^2)$$
$$Y_i(0) \mid X \sim_{ind} N(\sum_{j \in \mathcal{N}_i} X_j, \gamma^2)$$
$$Y_i(1) = Y_i(0) + \tau$$

- Observations in the same group are taken to have originated from a Normal distribution with the same mean.
- "The network induces correlation among the outcomes that are assigned to control because the mean of each $Y_i(0)$ is given by the sum of the covariate values X_j of units j in a neighborhood of i" (Basse and Airoldi 2018).

A simplified model

- Constant treatment effect model: τ is the difference between the potential outcomes $\{Y_i(0), Y_i(1)\}$.
- *Intuition*: in the absence of network connections and treatment $Z_i = 0$:
  - $Y_i(0)$ is a measure of an intrinsic property of the observational unit (e.g., time spent on social media), as determined by covariates X.
  - Network connections alter the natural value $Y_i(0)$ that would occur, through the induced network structure.
  - The intervention $\text{do}(Z_i = 1)$ induces a causal effect τ such that $Y_i(1) = Y_i(0) + \tau$.
- The *normal-sum* model is just a starting point...

Optimal treatment allocation

- To ascertain an optimal treatment allocation strategy, need a notion of error to define optimality.
- Basse and Airoldi (2018) propose the *conditional MSE*:
  1. fix a treatment allocation vector Z, then
  2. for the *normal-sum model*, $\text{MSE}(\hat{\tau} \mid Z) \equiv \mathbb{E}\{(\hat{\tau} - \tau^*)^2 \mid Z\}$
- Now, an optimal treatment allocation $Z^* \in \mathcal{Z}$ is one that minimizes the conditional MSE.

Where are the networks?

- A decomposition of the conditional MSE is informative of network statistics:

$$\text{MSE}(\hat{\tau} \mid Z) = \mu^2\{\delta_N(Z)\}^2 + \gamma^2\omega(Z)^T\omega(Z) + \sigma^2\omega(Z)^T A^T A\omega(Z)$$

- Each of the terms in the MSE decomposition is informative
  - Bias^2: $\mu^2\{\delta_N(Z)\}^2$
  - *Network-agnostic* variance component: $\gamma^2\omega(Z)^T\omega(Z)$
  - *Network-aware* variance component: $\sigma^2\omega(Z)^T A^T A\omega(Z)$
- Model-assisted restriction randomization strategies seek to minimize the conditional MSE, but tradeoffs occur.

Bias

- The bias term admits the decomposition

$$\mu \cdot \delta_{\mathcal{N}} = \mu \cdot \left(\frac{1}{N_1} \sum_{(i:Z_i=1)} |\mathcal{N}_i| - \frac{1}{N_0} \sum_{(i:Z_i=0)} |\mathcal{N}_i| \right)$$

- The bias is proportional to the *average degree* of each of the experimental arms (treatment and control groups).
- This is the difference in the average neighborhood sizes of the treated and untreated units – i.e., balance!
- Desirable treatment allocation vectors ($Z \in \mathcal{Z}^b$) will minimize this difference in neighborhood sizes.

Network-agnostic variance term

- The first part of the variance term may be decomposed

$$\gamma^2 \omega^T \omega = \gamma^2 \left(\frac{1}{N_1} + \frac{1}{N_0} \right)$$

- Similar to bias term, minimized when $N_1 = N_0$.
- Thus, this term penalizes a difference in the size of treatment and control units, and is satisfied through *balance*.
- This is similar to prior work in balanced randomizations outside of the context of network-correlated outcomes.

Network-aware variance term

- The second part of the variance term may be written

$$\begin{aligned}\sigma^2 \cdot \omega^T A^T A \omega = & \frac{\sigma^2}{N_1^2} \cdot \sum_{i,j:Z_i=Z_j=1} |\mathcal{N}_i \cap \mathcal{N}_j| \\ & + \frac{\sigma^2}{N_0^2} \cdot \sum_{i,j:Z_i=Z_j=0} |\mathcal{N}_i \cap \mathcal{N}_j| \\ & - \frac{2\sigma^2}{N_1 \cdot N_0} \cdot \sum_{i,j:Z_i=1 \text{and} Z_j=0} |\mathcal{N}_i \cap \mathcal{N}_j|\end{aligned}$$

- Minimize contribution of this term to the MSE by
  1. assigning units with shared neighbors to different groups, and
  2. avoiding assigning treatment or control to clusters of densely connected units.

Classical randomization

- **Q:** What's a randomization strategy?
- **A:** Probability distributions on the set of binary vectors $\mathcal{Z}$
- Let $Z_i \sim Bern(p)$ for $p \in (0, 1)$. A *Bernoulli randomization strategy* is $Z = (Z_1, \ldots, Z_n) \in \mathcal{Z}$.

Classical randomization

- *Completely randomized strategy*: restrict to $Z \in \mathcal{Z}$ such that $\sum_{i-1}^{n} Z_i = N_1$, where
  - N_1 is the size of the treatment group and N_0 is the size of the control group, so $N_0 + N_1 = N$
  - *Balanced* if $N_0 = N_1 = \frac{1}{2} \cdot N$
- Such randomization strategies restrict to a set of desirable treatment allocation vectors (e.g., eliminating or minimizing covariate imbalance).

Restricted randomization

- **Q:** What's a restricted randomization strategy?
- **A:** Probability distributions on the set of binary vectors $\mathcal{Z}$ implied by discarding allocation vectors $Z \in \mathcal{Z}$ according to a user-defined set of rules.
- To proceed, let $\mathcal{Z} \equiv \{0, 1\}^N$, the set of all possible treatment allocation vectors on N units

Restricted randomization

- Basse and Airoldi (2018) propose **4** model-based trestricted randomization strategies:
    1. balanced restricted randomization strategies
    2. unbiased restricted randomization strategies
    3. optimal restricted randomization strategies
    4. unconstrained/optimal restricted randomization strategies

$\mathcal{Z}^b$: balanced restricted randomization

- Such a strategy restricts to treatment allocation vectors in

$$\mathcal{Z}^b \equiv \{Z \in \mathcal{Z} : N_1 - N_0 = 0\}$$

- Aim to minimize the contribution of the total variance to the conditional MSE.
- Defines *balanced restricted randomization design*: $Z \in \mathcal{Z}^b$

$\mathcal{Z}^u$: unbiased restricted randomization

- Such a strategy restricts to treatment allocation vectors in

$$\mathcal{Z}^u \equiv \left\{ Z \in \mathcal{Z} : \frac{1}{N_1} \sum_{i:Z_i=1} |\mathcal{N}_i| - \frac{1}{N_0} \sum_{i:Z_i=0} |\mathcal{N}_i| = 0 \right\}$$

- Aim to minimize the contribution of the bias term to the conditional MSE.
- Defines *balanced/unbiased restricted randomization design*: $Z \in \mathcal{Z}^b \cap \mathcal{Z}^u$

$\mathcal{Z}^o$: optimal restricted randomization

- Such a strategy restricts to treatment allocation vectors in

$$\mathcal{Z}^o \equiv \{Z \in \mathcal{Z} : \text{MSE}(\hat{\tau} \mid Z) \leq q_\alpha^{\text{MSE}}\},$$

  where q_α^{MSE} is the α^{th}-quantile of the distribution of the conditional MSE.

- Defines *balanced/unbiased/optimal restricted randomization design*: $Z \in \mathcal{Z}^b \cap \mathcal{Z}^u \cap \mathcal{Z}^o$
  - n.b., $\mathcal{Z}^b \cap \mathcal{Z}^u \cap \mathcal{Z}^o$ contains at least two elements Z if $\mathcal{Z}^b \cap \mathcal{Z}^u \neq \emptyset$ (Basse and Airoldi 2018).

$\mathcal{Z}^o$: optimal restricted randomization

- Control the conditional MSE through three effects:
    1. Minimize average number of shared neighbors among pairs of treated units;
    2. minimize average number of shared neighbors among pairs of untreated units; and
    3. maximize average number of shared neighbors among pairs of units, one of which is treated and the other untreated.

$\mathcal{Z}^{\text{min}}$: unconstrained/optimal restricted randomization

- Such a strategy restricts to treatment allocation vectors in

$$\mathcal{Z}^{\text{min}} \equiv \{Z \in \mathcal{Z} : \text{argmin}\text{MSE}(\hat{\tau} \mid Z)\}$$

- Trades off small increases in bias for significant reductions in variance.
- Basse and Airoldi (2018) do *not* focus on this – similarly, we'll restrict our attention to $\mathcal{Z}^b$, $\mathcal{Z}^u$, and $\mathcal{Z}^o$.

Model-based optimal treatment allocation strategies

- *Idea*: use an estimator of the ATE implied by the model, then select a set of treatment allocation strategies based on this new estimator.
  - i.e., abandon the difference-in-means estimator
- The *optimal maximum likelihood design* is defined as

$$\mathcal{Z}^{\text{min}} \equiv \{Z \in \mathcal{Z} : \text{argmin} MSE(\hat{\tau}_{\text{MLE}} \mid Z)\}$$

- Now, the strategy is married to the chosen model (e.g., normal-sum).
  - If we're clever about how we restrict our randomizations, we can still maintain unbiasedness (i.e., $Z \in \mathcal{Z}^b \cap \mathcal{Z}^{\text{min}}$).

Restricted randomization and re-randomization

- What was all this about *re-randomization* again? Rejection sampling! (Let's let the computer do the work for us.)
- To use re-randomization for restricted randomization designs, based on whether certain criteria are satisfied

$$\begin{aligned}\phi^b(Z) =& \mathbb{I}\{\sum_i^N Z_i = \sum_i^N (1 - Z_i)\} \\ \phi^u(Z) =& \mathbb{I}\{\mu \cdot \delta_{\mathcal{N}}(Z) = 0\} \\ \phi^o(Z) =& \mathbb{I}\{\mathsf{MSE}(\hat{\tau} \mid Z) \leq q_\alpha^{\mathsf{MSE}}\}\end{aligned}$$

- Draw treatment allocation vector Z from original design, accept if $\phi(Z) = 1$ or discard if $\phi(Z) = 0$.

Issues in inference

- Neymanian intervals – restricted randomization makes these challenging as asymptotic theory incompatible with networks:
    1. number of covariates must be fixed, but this applies to neighbors in a network setting, and
    2. constraints must be a function of difference in group means and variance-covariance of that vector (proven not to hold).
- Bootstrap intervals? Difficult to implement due to potential complexities in correlation structure.
- What's left? Fisher intervals!

Fisher intervals and inference

- Obtained by inverting a sequence of Fisher exact tests.
- Accomplished through re-randomization, where the proposed restricted randomization distributions are treated as the permutation distributions.
- Under balance (i.e., $Z \in \mathcal{Z}^b$), the sharp null hypothesis

$$H_{\tau^*} : Y_i(1) = Y_i(0) + \tau^* \forall i,$$

may be inverted to generate a confidence interval wrt a sequence of τ^*.

  - See B.1 in the appendix of Basse and Airoldi (2018) for details on the construction.

Main result

- *Design unbiasedness*: An estimator $\hat{\tau}$ is unbiased wrt a distribution on $\mathcal{Z}$ if $\mathbb{E}_{\mathcal{Z}}(\hat{\tau} - \tau) = 0$
- The difference-in-means estimator $\hat{\tau}$ is an unbiased estimator of the ATE wrt
  1. uniform distribution on $\mathcal{Z}^b$, defining the *balanced* design;
  2. uniform distribution on $\mathcal{Z}^b \cap \mathcal{Z}^u$, defining the *balanced/unbiased* design;
  3. uniform distribution on $\mathcal{Z}^b \cap \mathcal{Z}^o$, defining the *balanced/optimal* design; and
  4. uniform distribution on $\mathcal{Z}^b \cap \mathcal{Z}^u \cap \mathcal{Z}^o$, defining the *balanced/unbiased/optimal* design.

Main result

- As a consequence of design unbiasedness (as well as of the increasingly nested supports), variance of $\hat{\tau}$ may be compared across designs:

$$\mathbb{E}\{\mathsf{Var}_{\mathcal{Z}^b \cap \mathcal{Z}^o}(\hat{\tau} \mid Y)\} \leq \mathbb{E}\{\mathsf{Var}_{\mathcal{Z}^b}(\hat{\tau} \mid Y)\}$$

- Similar inequalities exist for any pair of nested designs as long as $\mathcal{Z}^b$ is included in the support of designs.
- The arguments rely on *symmetry* provided by the inclusion of $\mathcal{Z}^b$ – in fact, we have the following Lemma: *For* $Z \in \mathcal{Z}^b$, *we have* $\hat{\tau}(1 - Z) = 2\tau - \hat{\tau}(Z)$.

Towards general network models

- The *normal-sum model* we discussed is just a simple case of a much broader family of models

$$Y_i(0) \mid X \sim^{ind} N(g[\{X_j\}_{j \in \mathcal{N}_i}], \gamma^2)$$

- Need a few regularity conditions on g to ensure that the quantity $\mathbb{E}(g[\{X_j\}_{j \in \mathcal{N}_i}] \mid \{X_j\}_{j \in \mathcal{S}})$ is well behaved for any subset of nodes $\mathcal{S} \subset \mathcal{N}_i$.
  - Positivity
  - Symmetry
  - Monotonicity

Examples of general network models

- **How well does this notion of network models generalize?**
- Let $g[\{X_j\}_{j \in \mathcal{N}_i}] = \sum_{j \in \mathcal{N}_i} X_j$.
  - *Intuition*: Mean for each group is the sum of their covariates.
  - This is just the *normal-sum* model again.
- Let $g[\{X_j\}_{j \in \mathcal{N}_i}] = \mathbb{I}\left(\sum_{j \in \mathcal{N}_i} X_j > c\right)$.
  - *Intuition*: Mean for each group is an indicator of whether the sum of their covariates is greater than a cutoff of interest c.
- Let $g[\{X_j\}_{j \in \mathcal{N}_i}] = \max(\{X_j\}_{j \in \mathcal{N}_i})$.
  - *Intuition*: Mean for each group is the max over their covariates.

Lessons for *good designs*

- From this extended discourse on good designs, a few new rules come to light.
- Let's ask – so, what exactly should a *good design* do?
    1. Decrease number of shared neighbors in treatment groups.
    2. Increase number of shared units between treatment groups.
    3. Balance the size of the groups and the distribution of the sizes of neighborhoods.

Alright...I've talked enough

Discussion – as promised

- Question 1:
- Question 2:
- Question 3:

References

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