Spatial statistics and causal inference:
Spatial confounding and interference in air pollution research

Georgia Papadogeorgou

UNC Environmental Epidemiology seminar - Feb 8, 2019
Spatial data and causal inference in air pollution research

- Variables are expected to have spatial structure
  - Exposure, outcome, covariates

Spatial data and causal inference in air pollution research

- Variables are expected to have spatial structure
  - Exposure, outcome, covariates
- Questions of interest are often causal
  - What is the effect of a specific intervention on polluting sources?
Spatial data and causal inference in air pollution research

- Variables are expected to have spatial structure
  - Exposure, outcome, covariates

- Questions of interest are often causal
  - What is the effect of a specific intervention on polluting sources?

- Integration of spatial data and causal inference
  - Spatial correlation of confounding variables
  - Interference, spillover effects

NO\textsubscript{x} emission control technologies

- Regulations such as the Clear Air Act enforce stricter rules on emissions
- Power plants follow different compliance strategies
- We focus on the installation of NO\textsubscript{x} emission reduction control technologies

---

NO\textsubscript{x}: Nitric oxide and nitrogen dioxides, precursors of ozone, reacting with other compounds in the presence of sunlight to create ozone
NO$_x$ emission control technologies

- Regulations such as the Clear Air Act enforce stricter rules on emissions
- Power plants follow different compliance strategies
- We focus on the installation of NO$_x$ emission reduction control technologies
- Selective Catalytic Reduction (SCR) and Selective Non-Catalytic Reduction (SNCR) are the most effective in reducing NO$_x$

---

NO$_x$: Nitric oxide and nitrogen dioxides, precursors of ozone, reacting with other compounds in the presence of sunlight to create ozone
NO$_x$ emission control technologies

- Regulations such as the Clear Air Act enforce stricter rules on emissions
- Power plants follow different compliance strategies
- We focus on the installation of NO$_x$ emission reduction control technologies
- Selective Catalytic Reduction (SCR) and Selective Non-Catalytic Reduction (SNCR) are the most effective in reducing NO$_x$
- Are SCR/SNCR more effective than alternative strategies in reducing ambient ozone concentrations?

---

NO$_x$: Nitric oxide and nitrogen dioxides, precursors of ozone, reacting with other compounds in the presence of sunlight to create ozone.
Data

- Coal and natural gas power plants during June-August 2004
- $A = 1$ if at least half of facility heat input is used by units with installed SCR/SNCR technologies, $A = 0$ otherwise
- 152 treated facilities, 321 controls
- $Y$: NO$_x$ emissions / 4$^{th}$ maximum ambient ozone concentration
- Covariates: Power plant characteristics, demographics, weather

Publicly available data sources: Air Markets Program Data, 2000 Census, EPA monitoring sites
Statistical challenges

- **Unmeasured spatial confounding**
  - Volatile organic compounds and sunlight is necessary for the creation of ozone
  - They may confound the relationship of NO\textsubscript{x} control strategies and ambient ozone
  - Weather and atmospheric covariate information varies spatially
Statistical challenges

- Unmeasured spatial confounding
  - Volatile organic compounds and sunlight is necessary for the creation of ozone
  - They may confound the relationship of NO$_x$ control strategies and ambient ozone
  - Weather and atmospheric covariate information varies spatially

- Interference
  - Pollution travels with the wind
  - “Upwind” pollution sources can affect ambient concentrations in the area surrounding power plants at long distances
  - Discussed in vaccine trials, herd immunity, spillover effects
Adjusting for Unmeasured Spatial Confounders
Notation

- For unit $i$
  - Treatment $A_i \in \{0, 1\}$
  - Potential outcomes $Y_i(1), Y_i(0)$ (SUTVA)
  - Covariates $L_i = (L_{i1}, L_{i2}, \ldots, L_{ip})$
Notation

- For unit $i$
  - Treatment $A_i \in \{0, 1\}$
  - Potential outcomes $Y_i(1), Y_i(0)$ (SUTVA)
  - Covariates $L_i = (L_{i1}, L_{i2}, \ldots, L_{ip})$

- Average Treatment Effect on the Treated:

$$ATT = E[Y(1) - Y(0)|A = 1]$$
Notation

For unit $i$
- Treatment $A_i \in \{0, 1\}$
- Potential outcomes $Y_i(1), Y_i(0)$ (SUTVA)
- Covariates $L_i = (L_{i1}, L_{i2}, \ldots, L_{ip})$

Average Treatment Effect on the Treated:

$$ATT = E[Y(1) - Y(0) | A = 1]$$

Positivity: $P(A = 1 | L) \in (0, 1)$

Ignorability: $Y(1), Y(0) \perp A | L$
Notation

- For unit $i$
  - Treatment $A_i \in \{0, 1\}$
  - Potential outcomes $Y_i(1), Y_i(0)$ (SUTVA)
  - Covariates $L_i = (L_{i1}, L_{i2}, \ldots, L_{ip})$

- Average Treatment Effect on the Treated:

  $$ATT = E[Y(1) - Y(0)|A = 1]$$

- Positivity: $P(A = 1|L) \in (0, 1)$

- Ignorability: $Y(1), Y(0) \perp\!\!\!\!\!\!\perp A|L$

- Propensity score matching
  - PS model $P(A = 1|L)$
  - Match treated units to controls with similar PS estimates
Unmeasured spatial confounding

- Confounders $L = (X, U)$
  - $X$ are observed, $U$ are unobserved

- If $U$ varies spatially, can we adjust for it?
  - If a matched pair is sufficiently close, the treated and control units will have similar values of $U$

Rosenbaum and Rubin [1983]
Unmeasured spatial confounding

- Confounders $L = (X, U)$
  - $X$ are observed, $U$ are unobserved

- If $U$ varies spatially, can we adjust for it?
  - If a matched pair is sufficiently close, the treated and control units will have similar values of $U$

- Observed variables $X$:
  - Use the propensity score to adjust for the observed confounders
  - $P(A_i = 1|X_i) = f(X_i) = \expit(X_i^T \beta)$
Unmeasured spatial confounding

- Confounders $L = (X, U)$
  - $X$ are observed, $U$ are unobserved

- If $U$ varies spatially, can we adjust for it?
  - If a matched pair is sufficiently close, the treated and control units will have similar values of $U$

- Observed variables $X$:
  - Use the propensity score to adjust for the observed confounders
  - $P(A_i = 1|X_i) = f(X_i) = \expit(X_i^T\beta)$

- Navigate the tradeoff between:
  1. Making matches as similar as possible with respect to $X$
  2. Small distance of matched pairs to capture similarity in $U$

Rosenbaum and Rubin [1983]
Distance Adjusted Propensity Score Matching

For a treated unit $i$ and a control unit $j$ define

$$DAPS_{ij} = w|PS_i - PS_j| + (1 - w) \cdot Dist_{ij}, \quad w \in [0, 1]$$

where $PS$ propensity score estimates, and $Dist$ spatial proximity.

- $w$: relative importance of the observed and unobserved confounders
  - High values of $w$ - most matching weight on observed covariates
  - Low values of $w$ - most matching weight on spatial proximity
Matches

- Average distance of matched pairs
  - Naïve: 1066 miles
  - DAPSm: 141 miles
Results

- Reduction by 205 tons of NO$_x$ emissions (95% CI: 4 – 406)
- \(-0.27\) (95% CI: \(-2.1\) to \(1.56\)) parts per billion in ambient ozone

---

- The national ambient air quality standard for ozone is 70 parts per billion.
- Keele et al. [2015]
Conclusions

- We propose a method to reduce bias from spatial unmeasured confounding
- SCR/SNCR control technologies lead to
  - Reductions in NO$_x$ emissions
  - Their effect on ozone is not significant

Additional information in the paper:
- How to pick the tuning parameter $w$
- Robustness to the choice of $w$ as an indication of no unmeasured spatial confounding
- Comparison with other methods for incorporating spatial information

Papadogeorgou, Choirat, and Zigler [2018a]
Relaxing the no interference assumption
Interference

- Previously, we assumed that each unit had $Y(0), Y(1)$
  - *Your* outcome has nothing to do with *my* treatment
- Treatment effects with “interference”
  - *Your* outcome may depend on your and *my* treatment
  - Potential outcomes $Y(0, 0, \ldots, 0), Y(0, 0, \ldots, 0, 1)$, etc
Interference

- Previously, we assumed that each unit had $Y(0), Y(1)$
  - *Your* outcome has nothing to do with *my* treatment
- Treatment effects with “interference”
  - *Your* outcome may depend on *your* and *my* treatment
  - Potential outcomes $Y(0, 0, \ldots, 0), Y(0, 0, \ldots, 0, 1)$, etc
- Vaccine trials, infectious diseases
Interference

- Previously, we assumed that each unit had $Y(0), Y(1)$
  - Your outcome has nothing to do with my treatment
- Treatment effects with “interference”
  - Your outcome may depend on your and my treatment
  - Potential outcomes $Y(0, 0, \ldots, 0), Y(0, 0, \ldots, 0, 1)$, etc
- Vaccine trials, infectious diseases
- Ambient pollution concentrations are affected by multiple sources
  - Pollution emitted “locally”
  - Pollution that is transported from nearby sources
Partial interference

- Partial interference: Partition of units in interference clusters
  - A unit’s outcome can depend on the treatment level of units in their cluster
  - Does not depend on treatment of units in other clusters
Defining estimands in the presence of interference

- Causal inference with interference was introduced in the context of two-stage randomized trials [Hudgens and Halloran, 2008]
- Extensions to observation studies consider estimands for two-stage randomized design (Tchetgen Tchetgen and VanderWeele [2012], Perez-Heydrich et al. [2015])
Defining estimands in the presence of interference

- Causal inference with interference was introduced in the context of two-stage randomized trials [Hudgens and Halloran, 2008]
- Extensions to observation studies consider estimands for two-stage randomized design (Tchetgen Tchetgen and VanderWeele [2012], Perez-Heydrich et al. [2015])
- Such estimands represent
  - What would we observe if treatment was assigned randomly to units with probability $\alpha$?
Causal inference with interference was introduced in the context of two-stage randomized trials [Hudgens and Halloran, 2008]

Extensions to observation studies consider estimands for two-stage randomized design (Tchetgen Tchetgen and VanderWeele [2012], Perez-Heydrich et al. [2015])

Such estimands represent:
- What would we observe if treatment was assigned randomly to units with probability $\alpha$?

Are these estimands interpretable?
- Covariates can be predictors of treatment allocation [Barkley et al., 2017]
- Dependence between units
Let $P_{\alpha,L}$ be the **counterfactual treatment allocation**

- How treatment is assigned in a hypothesized world
- $\alpha$ represents the cluster-average propensity of treatment
Counterfactual treatment allocation under realistic interventions

- Let $P_{\alpha, L}$ be the **counterfactual treatment allocation**
  - How treatment is assigned in a hypothesized world
  - $\alpha$ represents the cluster-average propensity of treatment
- How would treatment arise in cluster $i$ if
  - The cluster-average propensity of treatment was set to $\alpha$?
  - Individual treatment adoption depended on a covariate $L$ with log-odds $\delta_L$?
Notation

- Clusters $i \in \{1, 2, \ldots, N\}$ with $n_i$ units
- For unit $j$ in cluster $i$
  - Treatment $A_{ij} \in \{0, 1\}$
  - Potential outcomes $Y_{ij}(\cdot) = \{Y_{ij}(a_i), a_i \in \{0, 1\}^{n_i}\}$,
  - Unit covariates $L_{ij} = (L_{ij1}, L_{ij2}, \ldots, L_{ijp})$
Notation

- Clusters $i \in \{1, 2, \ldots, N\}$ with $n_i$ units
- For unit $j$ in cluster $i$
  - Treatment $A_{ij} \in \{0, 1\}$
  - Potential outcomes $Y_{ij}(\cdot) = \{Y_{ij}(a_i), a_i \in \{0, 1\}^{n_i}\}$
  - Unit covariates $L_{ij} = (L_{ij1}, L_{ij2}, \ldots, L_{ijp})$
- For cluster $i$
  - Cluster treatment $A_i = (A_{i1}, A_{i2}, \ldots, A_{in_i})$
  - Cluster treatment excluding unit $j$: $A_{i, -j}$
  - Cluster potential outcomes $Y_i(\cdot)$
  - Cluster covariates $L_i$
Covariate dependent counterfactual treatment allocation

How would treatment arise in cluster $i$ if

- The cluster-average propensity of treatment was set to $\alpha$?
- Individual treatment adoption depended on a covariate $L$ with log-odds $\delta_L$?
Covariate dependent counterfactual treatment allocation

How would treatment arise in cluster $i$ if

- The cluster-average propensity of treatment was set to $\alpha$?
- Individual treatment adoption depended on a covariate $L$ with log-odds $\delta_L$?

Define

$$\text{logit} P_{\alpha,L}(A_{ij} = 1|L_{ij}; \delta_L) = \xi_i + L_{ij}\delta_L$$

where

$$\frac{1}{n_i} \sum_{j=1}^{n_i} P_{\alpha,L}(A_{ij} = 1|L_{ij}; \xi_i, \delta_L) = \alpha$$
Average potential outcome

- Individual average potential outcome

\[ \overline{Y}_{ij}(a; \alpha) = \sum_{s} Y(A_{ij} = a, A_{i,-j} = s) P_{\alpha,L}(A_{i,-j} = s | A_{ij} = a) \]

Average all possible treatment allocations where

- Observation \( ij \) gets treatment \( a \)
- Cluster-level treatment probability is \( \alpha \)
Average potential outcome

- **Individual average potential outcome**

\[
\overline{Y}_{ij}(a; \alpha) = \sum_{s} Y(A_{ij} = a, A_{i,-j} = s) P_{\alpha,L}(A_{i,-j} = s | A_{ij} = a)
\]

- **Average all possible treatment allocations where**
  - Observation \(ij\) gets treatment \(a\)
  - Cluster-level treatment probability is \(\alpha\)

- **Group average potential outcome**

\[
\overline{Y}_i(a; \alpha) = \frac{1}{n_i} \sum_{j=1}^{n_i} \overline{Y}_{ij}(a; \alpha)
\]

- **Population average potential outcome**

\[
\overline{Y}(a; \alpha) = E_{G_0} [\overline{Y}_i(a; \alpha)],
\]

for super-population of clusters \(G_0\).
Average potential outcome

- Individual average potential outcome

\[ \bar{Y}_{ij}(a; \alpha) = \sum_s Y(A_{ij} = a, A_{i,-j} = s) P_{\alpha,L}(A_{i,-j} = s | A_{ij} = a) \]

Average all possible treatment allocations where
- Observation \( ij \) gets treatment \( a \)
- Cluster-level treatment probability is \( \alpha \)

- Group average potential outcome \( \bar{Y}_i(a; \alpha) = \frac{1}{n_i} \sum_{j=1}^{n_i} \bar{Y}_{ij}(a; \alpha) \)

- Population average potential outcome \( \bar{Y}(a; \alpha) = E_{G_0}[\bar{Y}_i(a; \alpha)] \), for super-population of clusters \( G_0 \)

- Direct effect for fixed cluster-average treatment propensity

\[ DE(\alpha) = \bar{Y}(1, \alpha) - \bar{Y}(0, \alpha) \]

- Indirect effect between two fixed cluster-average treatment propensity

\[ IE(\alpha_1, \alpha_2) = \bar{Y}(0, \alpha_1) - \bar{Y}(0, \alpha_2) \]
Group and population potential outcome estimators

Assumptions

- **Positivity**: \( P(A_i = a_i | L_i) > 0 \), for all \( a_i \in \{0, 1\}^{n_i} \)
- **Ignorability**: \( Y_i(\cdot) \perp A_i | L_i \)
Group and population potential outcome estimators

Assumptions

- **Positivity**: \( P(A_i = a_i | L_i) > 0 \), for all \( a_i \in \{0, 1\}^{n_i} \)
- **Ignorability**: \( Y_i(\cdot) \perp A_i | L_i \)

Define

\[
\hat{Y}_i(a, \alpha) = \sum_{j=1}^{n_i} \frac{P_{\alpha, L}(A_i, -j | A_{ij} = a, L_i; \delta)}{f_{A|L,i}(A_i | L_i; \gamma) n_i} I(A_{ij} = a) Y_{ij}
\]

and

\[
\hat{Y}(a; \alpha) = \frac{1}{N} \sum_{i=1}^{N} \hat{Y}_i(a, \alpha)
\]

where \( f_{A|L,i}(A_i | L_i; \gamma) \) is the propensity score of the observed treatment vector
Theoretical and practical results

- All results assume that positivity and ignorability hold
- $\hat{Y}_i(a, \alpha)$ is unbiased for $\bar{Y}_i(a, \alpha)$ (for known propensity score)
- $\hat{Y}(a; \alpha)$ is consistent (for correctly-specified estimated propensity score)
- Asymptotic results are derived for increasing number of clusters
- Asymptotic or bootstrap CIs are acquired
- Performance was checked in an extensive simulation study
- Coverage of bootstrap CIs was better than asymptotic CIs for a small number of clusters
Propensity score and counterfactual treatment allocation

- Propensity score of \textit{observed} treatment

$$\text{logit} P(A_{ij} = 1|L_{ij}) = \delta_0 + b_i + L_{ij}^T \delta, \ b_i \sim N(0, \sigma_b^2)$$

Cluster propensity score:

$$f_{A|L,i}(A_i|L_i; \gamma) = \int \prod_{j=1}^{n_i} P(A_{ij}|L_{ij}, \delta, b_i) f(b_i|\sigma_b^2) db_i$$
Propensity score and counterfactual treatment allocation

- Propensity score of observed treatment

\[
\text{logit} P(A_{ij} = 1|L_{ij}) = \delta_0 + b_i + L_{ij}^T \delta, \ b_i \sim N(0, \sigma_b^2)
\]

Cluster propensity score:

\[
f_{A|L,i}(A_i|L_i; \gamma) = \int \prod_{j=1}^{n_i} P(A_{ij}|L_{ij}, \delta, b_i) f(b_i|\sigma_b^2) db_i
\]

- Use the observed treatment allocation to inform \(P_{\alpha,L}\)

\[
\text{logit} P_{\alpha,L}(A_{ij} = 1|L_{ij}; \delta) = \xi_i^\alpha + L_{ij}^T \delta, \ \text{where}
\]

\[
\frac{1}{n_i} \sum_{j=1}^{n_i} P_{\alpha,L}(A_{ij} = 1|L_{ij}; \delta) = \alpha
\]
Direct and indirect effect of SCR on ambient ozone

\[ DE(\alpha) = \bar{Y}(1, \alpha) - \bar{Y}(0, \alpha) \]
\[ IE(\alpha_1, \alpha_2) = \bar{Y}(0, \alpha_1) - \bar{Y}(0, \alpha_2) \]

Ozone is measured in parts per million
The national ozone air quality standard of 0.07 parts per million.
Concluding remarks

- Estimands for realistic public health interventions
  - Cluster-average propensity of treatment
  - Distribution of cluster-average propensity of treatment
- Proposed consistent estimators and derived asymptotic variances
- SCR/SNCR technologies are more effective in decreasing ozone against alternatives
  - In the surrounding area
  - In the surrounding area of other power plants

Papadogeorgou, Mealli, and Zigler [2018b]
References


