Topics in spatial causal inference

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Adjusting for unmeasured spatial confounders

- Environmental and epidemiological data are often observational and spatially correlated
- A fundamental task is to estimate the effect of a treatment variable on a response variable
- The key assumption that there are no missing confounders is generally impossible to verify
- However, it may be possible to remove the effects of unmeasured confounders that are smooth spatial functions
- We examine several methods to adjust for missing spatial confounding variables

Motivating Example

- Wu et al (2020) ¹ found that an increase of 1 μg/m³ in ambient fine particulate matter (PM_{2.5}) is associated with a 15% increase in the COVID-19 mortality rate.
- The response variable Y is the cumulative COVID-19 mortality counts through May 12, 2020 for US counties.
- The exposure variable X is county-level average exposure to PM_{2.5} for 2000-2016

¹Wu, Nethery, Sabath, Braun, Dominici (2020). Air pollution and COVID-19 mortality in the United States: Strengths and limitations of an ecological regression analysis, *Science Advances*.

Introduction

Exposure variable, PM_{2.5} concentration



Outcome variable, log mortality rate



Log(deaths/population)



Other applications

• Using an app \rightarrow reduced air pollution exposure?

► Fishing regulations → ecological diversity?

Standard spatial linear regression model

- ▶ The response at location $\mathbf{s} \in \mathcal{R}^2$ is $Y(\mathbf{s})$
- ► The treatment variable of interest is *A*(**s**)
- The model is $Y(\mathbf{s}) = \beta_0 + A(\mathbf{s})\beta_A + \theta(\mathbf{s}) + \varepsilon(\mathbf{s})$
- Our goal is to estimate β_A
- The residuals have two components: θ(s) is spatially correlated and ε(s) ^{iid} Normal(0, τ²)

Standard spatial linear regression model

- The spatial term θ(s) is a Gaussian process with E{θ(s)} = 0 and Var{θ(s)} = σ²
- The correlation decays with distance

$$\mathsf{Cor}\{\theta(\mathbf{s}_i), \theta(\mathbf{s}_j)\} = \rho(d_{ij})$$

where d_{ij} is the distance between \mathbf{s}_1 and \mathbf{s}_j

For example, the exponential correlation is

$$\rho(d) = \exp(-d/\phi)$$

Potential confounding variables

Variable X(s) is a confounder if it is correlated with A(s) and Y(s)

► It is a **spatial confounder** if has spatial correlation

It is an missing spatial confounder it is unknown or unobserved

Missing confounders and residual correlation

Say the truth is $Y(\mathbf{s}) = \beta_0 + A(\mathbf{s})\beta_A + X(\mathbf{s})\beta_X + \varepsilon(\mathbf{s})$

- ▶ If X is observed, we can use non-spatial regression
- This would be fine even if X and A are correlated
- If X is unobserved, it would contribute to spatial component of the error term X(s) + ε(s)
- We might fit the spatial model

$$Y(\mathbf{s}) = \beta_0 + A(\mathbf{s})\beta_A + \theta(\mathbf{s}) + \varepsilon(\mathbf{s})$$

and hope that θ accounts for *X*

Simulation with a missing spatial confounder

- ▶ We generate *X*(**s**) and *A*(**s**) as spatial processes
- The correlation between X and A ranges from 0 to 1
- The data are generated as

$$Y(\mathbf{s}) = A(\mathbf{s})\beta_A + X(\mathbf{s})\beta_X + \varepsilon(\mathbf{s})$$

where
$$\beta_A = \beta_X = 1$$
 and $\varepsilon(\mathbf{s}) \stackrel{iid}{\sim} \text{Normal}(0, 0.1^2)$

We fit non-spatial and spatial regression models that exclude X Introduction

Coverage and root mean squared error for β_A



Spatial regression works well when Cor{X(s), A(s)} = 0
However, even a small correlation causes huge problems

Possible remedies for a missing spatial confounder¹

1. Scientific knowledge

2. Propensity scores

3. Matching

4. Mean adjustments

¹ Reich, Yang, Guan, Giffin, Miller, Rappold. (2020). A review of spatial causal inference methods for environmental and epidemiological applications. https://arxiv.org/abs/2007.02714

Adding known confounders

Wu et al include several potential confounder variables:

Climate variables such as temperature

- SES variables including income and race
- Access to ICU beds

• Call the known confounders $W_1(\mathbf{s}), ..., W_p(\mathbf{s})$

The fitted model is

$$Y(\mathbf{s}) = \beta_0 + \sum_{j=1}^{p} W_j(\mathbf{s})\alpha_j + A(\mathbf{s})\beta_A + \theta(\mathbf{s}) + \varepsilon(\mathbf{s})$$

A logical extension to environmental application is to replace the W_i(s) with numerical model output

EXAMPLE: Adding mathematical/process models

- Fine particular matter (PM_{2.5}) is a criteria pollutant monitored by the EPA to protect human health
- In the US, emissions of PM_{2.5} from most sources are steadily declining except for forest fire smoke
- How much PM_{2.5} and health burden is causally-attributed to wildfires?
- This is difficult to answer directly because only total PM_{2.5} (background + fire) can be measured
- We² combine numerical models (CMAQ), observational data and causal inference

²Larsen et al. A spatial causal analysis of wildland fire-contributed PM2.5 using numerical model output. *Annals of Applied Statistics.*

CMAQ: The Community Multiscale Air Quality Modeling System



* plume rise, biogenic, lightning generated NO, sea salt, windblown dust, bidirectional exchange of ammonia

(1) Scientific knowledge

Annual average CMAQ ($12km \times 12km$)



• [1.85,3.07] • (3.07,4.4] • (4.4,6.79] • (6.79,30.4]

CMAQ run without fires



• [1.16,2.21] • (2.21,3.74] • (3.74,5.93] • (5.93,29.3]

Difference between the runs (as % of total)





- (23.5,91.8]
- (12,23.5]
- (7.43,12]
- [0.84,7.43]

EPA Monitoring Stations (background + fire)



PM_{2.5} (μg/m³)

- (11.5,16.8]
- (9.95,11.5]
- (7.76,9.95]
- [3.69,7.76]

PM_{2.5} is measured every 3-6 days; this is the 2008-2012 average

Time series plot for one site in Northern CA



Data sources and potential outcomes notation

- Monitor data at location **s** and day *t*: $Y_t(s)$
- CMAQ run without fires: $\hat{\theta}_t(\mathbf{s})$
- CMAQ run with fires: $\hat{\theta}_t(\mathbf{s}) + \hat{\delta}_t(\mathbf{s})$
- ► The "treatment" is the regime
 - A = 0: world without wildland fires
 - A = 1: current world with wildland fires
- $Y_t(\mathbf{s}, 0)$ and $Y_t(\mathbf{s}, 1)$ are the potential PM_{2.5} outcomes
- Model: $Y_t(\mathbf{s}, \mathbf{0}) = \theta_t(\mathbf{s})$ and $Y_t(\mathbf{s}, \mathbf{1}) = \theta_t(\mathbf{s}) + \delta_t(\mathbf{s})$
- θ_t and δ_t are stochastic processes

Potential outcomes framework

The causal effect is

$$\Delta(\mathbf{s}) = \mathsf{E}[Y_t(\mathbf{s},1) - Y_t(\mathbf{s},0)]$$

where the average is over the distribution of fires over the entire spatial domain

• Challenge: we never observe data under A = 0

- To address this we use
 - CMAQ output
 - Causal assumptions

Assumptions

We assume there exist

- $C_t(\mathbf{s}) \in \{0, 1\}$ where **s** is affected by smoke iff $C_t(\mathbf{s}) = 1$
- Bias-correction functions B₀ and B₁

so that the following assumptions hold:

(A1) Consistency:
$$Y_t(\mathbf{s}) = Y_t[\mathbf{s}, C_t(\mathbf{s})]$$

(A2) No unmeasured confounders given model output:

 $\theta_t(\mathbf{s}) = B_0[\hat{\theta}_t(\mathbf{s})] + e_{1t}(\mathbf{s}) \text{ and } \delta_t(\mathbf{s}) = B_1[\hat{\delta}_t(\mathbf{s})] + e_{2t}(\mathbf{s}),$

where $\mathbf{e}_t(\mathbf{s}) = [\mathbf{e}_{1t}, \mathbf{e}_{2t}]$ is independent of A and C

Assumptions

Are these assumptions reasonable?

(A1) assumes that we have some observations we are sure are not affected by fire smoke ... probably OK?

(A2) assumes that the CMAQ modelers have included the important drivers of fine particulate matter maybe OK? Have we accounted for all feedback loops?

Causal interpretation

- Theory: We prove that under these assumptions the estimate effect has a causal interpretation
- We specify parametric models for the bias correction functions B₀ and B₁ and the spatial process e_t(s)
- Theory: We show that all parameters, including the correlation between counterfactuals, are identifiable
- This serves as a basis for using a Bayesian approach to estimating Δ(s) and quantify its uncertainty

Data and estimates for one site in CA



Causal estimate, $\Delta(\mathbf{s})$, posterior mean



(1) Scientific knowledge

Cumulative health burden by county



EXAMPLE: Propensity scores

- Smoke Sense is a cell phone app design by the US EPA to educate and engage citizens
- The app provides users current and forecasted air quality and alerts of fires
- Users record their smoke observations, health symptoms and preventative actions

Users also play educations games, earn badges, etc.

The smoke Sense app



Smoke Sense data and notation

- Response: Y_{it} is the number of self-reported symptoms (asthma attack, chest pain, etc.) by user *i* in week *t*
- Treatment: A_{it} is a binary indicator that user i took preventive measures (stay indoors, use mask, etc.)
- Covariates: baseline variables (age, gender, etc.) and time-varying variables (smoke exposure, visibility, etc.)
- We use n = 1076 users from 2019 and the number of observations ranges from 1-15
- Our objective is determine if using the app leads to a reduction in symptoms

Challenges

App-based platforms provide unprecedented opportunities to reach users, but pose challenges:

- Self-selection: Protective behaviors were left to the user and may depend on their characteristics
- Informative missingness: Participants are more likely to self-report when they experience smoke or symptoms
- Spatial variation: The causal effect of treatment may vary over the study's large and diverse domain

Potential outcomes (PO)

- For simplicity, we³ drop the user subscript (*i*) and assume regular observation times t ∈ {1, 2, ...}
- ► History is denoted by overbar, so \bar{A}_m and \bar{X}_m are the set of values before time *m*
- ▶ POs are denoted by \bar{a}_m is the set of treatments before time *m* and a_m is the set of treatment taken after time *m* < *t*
- ▶ The PO at time *t* is $Y_t(\bar{a}_m, a_m)$
- Our goal is to estimate the causal effect which is the mean difference between Y_t(ā_m, a_m) and Y_t(ā_m, 0)
- We do this within the structured nested mean model of Robins (1992)

³Wu et al, https://arxiv.org/abs/2005.12017

Structured nested mean model

• The causal effect γ as a function of parameters θ is

$$\mathsf{E}\left\{Y_t(\bar{a}_m, a_m) - Y_t(\bar{a}_m, 0) | \bar{a}_m, \bar{x}_m\right\} = \gamma_{tm}(\bar{a}_m, \bar{x}_m; \theta) = \gamma_{tm}(\theta)$$

An example with covariate and lagged treatment effects is

$$\gamma_{tm}(\boldsymbol{\theta}) = \boldsymbol{x}_m \boldsymbol{\beta} + \psi \exp\left\{-\frac{(t-m-\mu)^2}{2\sigma^2}\right\} \boldsymbol{a}_m$$

and $\boldsymbol{\theta} = \{\boldsymbol{\beta}, \psi, \mu, \sigma\}$

• The treatment at time $m = t - \mu$ has the most impact

• The magnitude of treatment at lag μ is ψ

Estimation

Sequential randomization assumption: treatment at time m is a function only of the propensity score

$$e_m = \operatorname{Prob}(A_m = 1 | \bar{A}_{m-1}, \bar{X}_m, \bar{Y}_{m-1})$$

► To estimate the lag *m* effect, we must remove effects for time *l* ∈ {*m*, ..., *t* − 1}

• Let
$$R_{tm}(\theta) = Y_t - \sum_{l=m}^{t-1} \gamma_{tl}(\theta)$$
 and $\mu_{tm}(\theta) = \mathsf{E}(R_{tm})$

The weighted (by w_{tm}) estimating equation is

$$G(\theta) = \sum_{t=1}^{n_t} \sum_{m=1}^{t-1} w_{tm} \{ R_{tm}(\theta) - \mu_{tm}(\theta) \} (A_t - e_t)$$

Spatially-varying effects

- We extend this structured nested mean model to allow for spatially-varying θ
- This could lead to tailoring the app to individual environments
- We use geographically weighted (*w_{tm}*)local polynomial estimation
- The local effect at s* is taken to be polynomial in s near s*
- Observations are kernel weighted by distance ||s s*||
- Double robustness: We prove consistency if either the potential-outcome or mean models are correct

Smoke Sense – Local estimate of $\psi(\mathbf{s})$

Wild CIs of ψ with bias correction



Ψ

EXAMPLE: Spatial matching

Commercial fishing is highly regulated for conservation

- There is variation in the type and degree of regulation
- Using data from global Gill et al ⁴ we compare two broad classes of regulations: no take (NT) and multi-use (MU)
- Our objective is to compare the relative effectiveness of these two policies

⁴Gill et al. (2017). Capacity shortfalls hinder the performance of marine protected areas globally. *Nature*, **543**, 665–669

Data and notation

▶ Response: Y_i is the log biomass at site i = 1, ..., n = 9987

• Treatment: $A_i = 1$ for NT and $A_i = 0$ for MU

Covariates: X_i are known covariates

 Dependence structure: the *n* observations are nested in 215 Marine Protection Areas (MPAs) (3) Matching

Response (*Y*) by treatment (*A*)



Covariates (X)

	Site-level	MPA-level
Continuous	Latitude (degree) Longitude (degree) Depth (m) Wave exposure (kW/m) Distance to shoreline (km) Distance to market (km) Coastal population (million/100km ²) Sample date (year) Minimum sea surface temperature (°C) Chlorophyll-A (mg/m ³) Reef area with 15km (km ²) MPA age (years)	MPA size (km²)
Categorical	Habitat type (16) Marine ecoregion (56) Sampling protocol (6)	Country (43)

Dependence structure



Challenges

The treatment variables are not randomly assigned

The allocation of treatments is likely influenced by biomass

Sites are nested within MPA, giving (spatial) dependence

Potential outcomes (POs) and assumptions

• $Y_i(a)$ is the PO under treatment $a \in \{0, 1\}$

▶ The PO model for a site in MPA *r* is

$$Y_i(a) = \mu_a(\mathbf{X}_i) + \alpha_r + \varepsilon_i$$

where α_r is an MPA random effect

- Assumption: the POs for each unit are not influenced by the treatment assigned to other units
- Assumption: conditional independence between treatment and the POs when accounting for the covariates

Matching estimator

- We propose a matching estimator to account for spatial dependence and missing confounders
- Each site is matched by space and covariates with M observations with the opposite treatment
- ► The missing PO (i.e., $Y_i(0)$ if $A_i = 1$) are imputed using the mean of these *M* observations
- The naive matching estimator for the average treatment effect (ATE) is

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

The average treatment effect on the treated (ATT) is similar

Extensions

- The naive estimator is biased if µ_a(X) varies by a
- For fit several regression models including random forests to remove this bias
- Deriving the standard error is challenging because of MPA effects and sites are members of multiple pairs
- We develop a wild bootstrap for this purpose
- Theory: We prove that our estimator is consistent and asymptotically normal

Results

Table: Summary of the average treatment effect (ATE) and the average treatment effect on the treated (ATT) with estimated standard errors in parentheses when comparing the multi-use (MU) policy and no-taken (NT) policy in MPAs where MU is considered as treatment group; Response is log (Fish Biomass).

	ATE		ATT	
	Point Estimate	95% CI	Point Estimate	95% CI
Matching on MPA-level covariates				
Sieve Method	-0.49 (0.31)	(-1.10, 0.13)	-0.67 (0.50)	(-1.64, 0.30)
Smooth Spline	-0.27 (0.25)	(-0.77, 0.23)	-0.19 (0.38)	(-0.93, 0.56)
Regression Forest	-0.57 (0.35)	(-1.26, 0.12)	-0.82 (0.49)	(-1.77, 0.13)
Matching on all covariates				
Sieve Method	-0.41 (0.17)	(-0.76, -0.07)	-0.58 (0.26)	(-1.10, -0.06)
Smooth Spline	-0.34 (0.30)	(-0.93, 0.26)	-0.41 (0.41)	(-1.22, 0.40)
Regression Forest	-0.70 (0.32)	(-1.32, -0.07)	-1.03 (0.53)	(-2.06, 0.00)

EXAMPLE: Adjusting for unmeasured spatial confounders

- The key assumption that there are no missing confounders is generally impossible to verify
- However, it may be possible to remove the effects of unmeasured confounders that are smooth spatial functions
- We propose a method to adjust for missing spatial confounding variables using spectral methods.
- ► Motivating example Wu et al (2020) ⁵ found that an increase of 1 µg/m³ in PM_{2.5} is associated with a 15% increase in the COVID-19 mortality rate.

⁵Wu, Nethery, Sabath, Braun, Dominici (2020). Air pollution and COVID-19 mortality in the United States: Strengths and limitations of an ecological regression analysis, *Science Advances*.

Exposure variable, PM_{2.5} concentration



The exposure variable X is county-level average exposure to $PM_{2.5}$ for 2000-2016

Outcome variable, log mortality rate



The response variable *Y* is the cumulative COVID-19 mortality counts through May 12, 2020 for US counties.

Our approach

- We assume that local information is more informative than global information
- If you could design the experiment you would assign different A(s) to neighbors, right?
- We propose a joint model for A and the unobserved spatial confounder, X⁶
- We derive the optimal confounder adjustment under the joint model
- We then discuss assumptions needed to identify the parameters in the counfounder adjustment

⁶See Schnell and Papadogeorgou (2019) for areal data, Dupont, Wood and Augustin (2020) for splines and Stokes and Purdon (2017) for time series.

Unmeasured confounders

- X(s) is unknown or not measured
- Obviously, additional assumptions are required to estimate its correlation with A(s)
- We assume $[A(\mathbf{s}), X(\mathbf{s})]$ follows a bivariate spatial GP
- Then X can be written $X(\mathbf{s}) = \widehat{X}(\mathbf{s}) + \delta(\mathbf{s})$ where

$$\widehat{X}(\mathbf{s}) = \mathsf{E}\{X(\mathbf{s})|\mathbf{A}\}$$

δ(s) is a Gaussian process that is independent of A

Unmeasured confounders

The observation equation then becomes

$$Y(\mathbf{s}) = \alpha + A(\mathbf{s})\beta_A + \widehat{X}(\mathbf{s})\beta_X + \delta(\mathbf{s}) + \epsilon(\mathbf{s})$$

• $\hat{X}(\mathbf{s})$ is the required confounder adjustment

- The spatial term $\delta(\mathbf{s})$ is independent of **A**
- Therefore, if we know \hat{X} we eliminate spatial confounding



We model A and X using spectral methods

- If A and X are stationary they have spectral representations
- The spectral representations are

$$X(\mathbf{s}) = \int \exp(i\omega^{T}\mathbf{s})x(\omega)d\omega$$
$$A(\mathbf{s}) = \int \exp(i\omega^{T}\mathbf{s})a(\omega)d\omega$$

 $\blacktriangleright \omega \in \mathcal{R}^2$ is a frequency

▶ $x(\omega)$ and $a(\omega)$ are mean zero and independent across ω

Spectral decomposition in 1D



Full signal (black): X(s) = ∫ exp(iω^Ts)x(ω)dω
Low-frequencies (red): X̃(s) = ∫_{||ω||<T} exp(iω^Ts)x(ω)dω

Spectral decomposition in 1D



Full signal (black): X(s) = ∫ exp(iω^Ts)x(ω)dω
High-frequencies (red): X̃(s) = ∫_{||ω||>T} exp(iω^Ts)x(ω)dω

We model X and A using spectral methods

Dependence is measured by the coherence¹:

$$\alpha(\boldsymbol{\omega}) = \operatorname{Cor}\{\boldsymbol{x}(\boldsymbol{\omega}), \boldsymbol{a}(\boldsymbol{\omega})\}$$

which we allow to vary by frequency

However, we assume that

$$\lim_{||\boldsymbol{\omega}|| \to \infty} \alpha(\boldsymbol{\omega}) = \mathbf{0}$$

and prove that we can identify the treatment effect

- This allows for confounding of large-scale features (small ω) but assumes independence at small scales (large ω)
- This is a similar motivation for matching spatial neighbors

¹ Kleiber (2017) has a good review

Model in the spatial domain

The model in the spatial domain is

$$Y(\mathbf{s}) = \beta_0 + A(\mathbf{s})\beta_x + \widehat{X}(\mathbf{s})\beta_X + \delta(\mathbf{s}) + \epsilon(\mathbf{s})$$

The confounder adjustment is

$$\widehat{X}(\mathbf{s}) = \int K(\mathbf{u} - \mathbf{s}) A(\mathbf{u}) \mathrm{d}\mathbf{u},$$

where $K(\mathbf{u} - \mathbf{s})$ is the inverse Fourier transform of $\alpha(\boldsymbol{\omega})$

- X is a smoothed version of A that is included to adjust for spatial confounding
- The methods can be fit using standard MCMC/ML methods

Plot of the covariate A (left) versus $\hat{\mathbf{X}}$ (right)

 $v_{XZ} = 1v_X$

$$v_{XZ} = 3v_X$$



COVID mortality/PM



- Spatial causal inference is an important and rapidly-evolving area
- We have covered a wide variety of methods, and this is just the surface
- For a more complete review of spatial causal inference, see Reich, et al. (2020). A review of spatial causal inference methods for environmental and epidemiological applications. *International Statistical Review*
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