Causal inference for ex-post evaluation of transport interventions

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Focus on **statistical modelling** approaches for **causal inference**
Why is ‘causality’ relevant?
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★ Fuel taxation to reduce transport emissions
★ Congestion charging to reduce traffic volumes
★ Road cameras to reduce speeds / mitigate accidents
★ Infrastructure investment to boost the productivity of the economy
★ Public transport upgrades to improve network performance
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Research challenge: use observed data linking interventions with outcomes to quantify cause-effect relationships
Causal inference models in statistics
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In practice, the observed data typically do not fulfill these criteria: we have incomplete data and confounding.
Statistical solutions for causal inference
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1. *Impacts of urban road network capacity expansions in the US*
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1. *Impacts of urban road network capacity expansions in the US*
2. *Regional economic impacts of High Speed Rail investments in Spain*
Case study 1: impacts of urban road capacity expansions

TTI urban mobility data on road traffic conditions for 101 US cities (1982-2007)
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**Contribution:** a statistical approach for ex-post evaluation

- provides a unified framework to assess relative effects of capacity expansions on demand, network performance, and productivity
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**Results:** quantify changes in ‘responses’ (i.e demand, performance, productivity) caused by treatments (i.e amount of capacity expansion) net of confounding effects
Ex-post evaluation via causal ATE estimation
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**Set-up:** observed data $z_i = (y_i, d_i, x_i)$, $i = 1, ..., n$, where $y_i$ is a response, $d_i$ is treatment (dose), and $x_i$ is a vector of covariates.
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Target of inference: we want to estimate Average Treatment Effects (ATEs)

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\tau(d^*) = \mathbb{E}[Y(d^*)] - \mathbb{E}[Y(0)]
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for all doses \( d^* \in D \subseteq \mathbb{R} \) of interest.
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This is done by calculating **generalised propensity scores**
Generalised Propensity Score (GPS) adjustment
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The GPS measures the conditional probability of assignment to treatment given confounders

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i. Estimate the GPSs using a flexible model: $\hat{\pi}(d|X; \hat{\alpha})$

ii. Adjust for confounding via a mean response model: $\mathbb{E}[Y|D, \hat{\pi}(d|X; \hat{\alpha}); \beta]$
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iii. Use $\hat{\beta}$ to calculate expected response at dose $d^*$

$$\hat{\mu}(d^*) = \mathbb{E}[Y(d^*)] = \mathbb{E}_X \left[ \mathbb{E}(Y | d^*, \hat{\pi}(d^* | X ; \hat{\alpha}); \hat{\beta}) \right],$$

and repeat for all doses of interest.
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iv. Calculate ATEs: \( \hat{\tau}(d^*) = \hat{\mu}(d^*) - \hat{\mu}(0) \), using (block) bootstrap for variance estimation
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- GPS can be used to form a number of ATE estimators via weighting, matching, or regression (combine for doubly robust)
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- GPS can be used to form a number of ATE estimators via weighting, matching, or regression (combine for doubly robust)
- A **longitudinal mixed model extension** of the GPS can address *unobserved effects* and *bi-directional causality* between response and treatment by subsuming these effects within the GPS
Methodological contribution of the paper

ATE estimates are unbiased if the estimated GPS consistently estimates the true GPS

A necessary condition is that $X$ is sufficient to represent confounding

We show that with longitudinal data the GPS can be estimated via a mixed model approach to address

- **Unmeasured confounding:** condition on unit level random effects, or correlated random effects, to adjust for unobserved time-invariant confounding: $\hat{\pi}(d^* | x_{it}, u_i; \hat{\alpha})$

- **Reverse causality:** condition on lagged values of the response $y_{it-p}$, or the response history $\mathcal{H}^y_{i,t-1}$, to allow for endogeneity from reverse causation: $\hat{\pi}(d^* | x_{it}, u_i, \mathcal{H}^y_{i,t-1}; \hat{\alpha})$

- **Dynamic assignment:** include lagged values of the treatment $d_{i,t-p}$, or treatment history $\mathcal{H}^d_{i,t-1}$, to represent the dynamic nature of assignment: $\hat{\pi}(d^* | x_{it}, u_i, \mathcal{H}^y_{i,t-1}, \mathcal{H}^d_{i,t-1}; \hat{\alpha})$
Algorithm for ATE estimation via mixed GPS model

1. Use a flexible mixed model (i.e. GAMM) to estimate $f_{D|X}(d|x, u; \alpha)$

2. Use $\hat{\alpha}$, with the appropriate density function, to calculate the GPSs: $\hat{\pi}(d^*|x, u; \hat{\alpha})$, for all $d^*$ of interest

3. Ensure common support by selecting only units which have a reasonable probability of being treated across the range of dose

4. Estimate $\mathbb{E}(Y|D, \hat{\pi}(d|x, u; \hat{\alpha}))$ using a penalised spline model

5. Average over predicted values from 4., evaluated at at dose $d^*$, to obtain a point estimate of the expected response at $d^*$: $\hat{\mu}(d^*)$

6. Repeat for all dose of interest, form the dose-response curve, and estimate ATEs:

   $$\hat{\tau}(d^*) = \hat{\mu}(d^*) - \hat{\mu}(0)$$

7. Use a single (block) bootstrap re-sampling scheme over 1. to 6. to obtain standard errors
Urban longitudinal data (TTI and MSA)

- **Responses:** annual proportional change in traffic volume (vmt), network performance (delay per vmt), and productivity (average wage)

- **Treatment:** annual proportional change in network lane miles (freeway and arterial)

- **Pre-treatment covariates (confounders):**
  - Lagged responses: to capture reverse causality
  - Congestion & traffic volume: measured by delay and vmt
  - Network scale & mix: network length, mix of freeway / arterial
  - Traffic mix: volume on freeway / arterial
  - Mode characteristics: public transport patronage, state fuel price
  - Economy: productivity, income and economic structure
  - Employment and population distribution and growth

- **Unobserved confounders:** physical characteristics, geographical features, aspects of road network design, activity/travel behaviour patterns
  - Random city-level effects specified in longitudinal mixed models

- **Models:** Normal GAMMs for all sub models
Results: traffic volumes (vmt)

![Graph showing traffic volumes (vmt) vs. capacity expansion (%). The x-axis represents capacity expansion (%) ranging from 1 to 5, and the y-axis represents average treatment effect (%) ranging from -2 to 6. The graph shows a positive correlation between capacity expansion and average treatment effect.]
Results: traffic volumes (vmt)

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- evidence of induced demand over the range of dose having adjusted for confounding
- ATE > proportional to treatment for doses \( \leq 2 \)
- on average 10% increase in lane miles \( \rightarrow 9\% \)
  increase in vmt net of ‘natural growth’ (estimated 1.4% p.a.)
Results: traffic volumes (vmt)

- Evidence of induced demand over the range of dose having adjusted for confounding
- ATE > proportional to treatment for doses ≤ 2
- On average 10% increase in lane miles → 9% increase in vmt net of ‘natural growth’ (estimated 1.4% p.a.)
- Capacity expansions in the range considered have not in general reduced traffic density (vol. / cap.)
Results: network performance (delay per vmt)
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* capacity expansions have not ameliorated urban congestion
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* average road user has not experienced reduced delay
Results: network performance (delay per vmt)

รายละเอียด:

- ความเร็วของเครือข่าย (delay per vmt)

- ค่าอัตราการขยายความสามารถ (Capacity Expansion (%))
- อัตราผลประโยชน์เฉลี่ย (Average Treatment Effect (%))

★ ความต่อเนื่องของการขยายความสามารถไม่ได้แกวการจราจรในเมือง
★ ผู้ใช้ทางถนนทั่วไปไม่ได้รับความปลอดภัยในการจราจร
★ ต้นทุนรวมของความต่ำต้านการจราจรเพิ่มขึ้น

![Graph showing network performance results](image-url)
Results: network performance (delay per vmt)

- capacity expansions have not ameliorated urban congestion
- average road user has not experienced reduced delay
- aggregate cost of congestion has increased
- no statistically significant effects on delay per vmt
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- This is the case even for large capacity expansions
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- capacity expansions have not ameliorated urban congestion
- average road user has not experienced reduced delay
- aggregate cost of congestion has increased
- no statistically significant effects on delay per vmt
- this is the case even for large capacity expansions
- due to natural growth congestion worsens further (approx. 3% p.a.)
Results: productivity (average MSA wage)
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- But no significant ATEs having isolated a viable sample and adjusted for confounding.
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- ‘Naive’ regressions of productivity on treatment do indicate a positive association
- But no significant ATEs having isolated a viable sample and adjusted for confounding
- No fall in interaction costs and apparently no scale effects
Case study 1: conclusions
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Model indicates that urban road network expansions have induced demand but have not ameliorated congestion or raised productivity

Results do not imply that there are no economic benefits from road capacity expansions per se:
Case study 1: conclusions

Causal mixed model GPS approach provides a highly flexible framework for ex-post evaluation of transport interventions.

Model indicates that urban road network expansions have induced demand but have not ameliorated congestion or raised productivity.

Results do not imply that there are no economic benefits from road capacity expansions per se:

- results specific to marginal changes on mature congested urban networks.
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To improve urban road network performance and raise productivity a combination of efficient pricing with investment in both roads and mass transit may be more effective
The problem of confounding

The relationship between capacity and productivity is **confounded** by a set of city characteristics which

- Are important for productivity
- Influence the level of capacity expansion received