Quantifying the effects of urban road capacity expansions on network performance and productivity via a mixed model generalised propensity score estimator

Dan Graham
Professor of Statistical Modelling

Centre for Transport Studies
d.j.graham@imperial.ac.uk
Introduction

**Background:** existing research has quantified effects of road capacity changes on

- travel demand (e.g. ‘induced’ demand),
- network performance (e.g. speeds, travel times, flow, density etc)
- productivity (e.g. GVA, wages, TFP, growth etc)

But largely distinct perspectives using different approaches and data

Worth considering these effects together as there could be interesting relations between them

Do **productivity effects** from transport investments arise via

- Improvements in network performance
- Increased scale of ‘activity’ (i.e. agglomeration effect)
Introduction

**Objective:** develop a common framework to study *relative* effects of capacity expansions on demand, network performance, and productivity

**Method:** develop a causal inference approach for *average treatment effect* (ATE) estimation with longitudinal data

- Where *treatments* are ‘*doses*’ of urban road network capacity expansion
- *Assignment* is *non-random*, and the probability of receiving a given dose varies systematically with city characteristics (*confounding*)
- Quantify relationship between expected ‘*response*’ (i.e demand, performance, productivity) and dose, net of confounding effects
Causal inference for continuous ATE estimation

Typical set-up: we observe data \( z_i = (y_i, d_i, x_i), i = 1, ..., n \), where \( y_i \) is a response, \( d_i \) is treatment (dose), and \( x_i \) a vector of covariates.

Target of inference: we seek to estimate ATEs

\[
\tau(d^*) = \mathbb{E}[Y(d^*)] - \mathbb{E}[Y(0)]
\]

for all doses \( d^* \in D \subseteq \mathbb{R} \) of interest. But we have confounding

\[
f_Z(z) = f_{Y|D,X}(y|d,x)f_{D|X}(d|x)f_X(x)
\]

Implies that \( Y(d) \not\perp D = d \), so comparison of mean outcomes across different treatment strata will not in general reveal a causal effect.

But we do have conditional independence between \( Y \) and \( D \) given \( X \)

\[
Y(d) \perp D = d|X = x \quad \text{for all} \quad d \in D \subseteq \mathbb{R},
\]

so we can estimate ATEs by somehow adjusting for \( X \). We do so via \textit{generalised propensity scores} for longitudinal data.
Generalised Propensity Score (GPS) adjustment

The GPS is a scalar that measures the conditional probability of assignment to treatment given confounding characteristics.

We can define the GPS for any dose \( d^* \) (observed or unobserved):

\[
\pi(d^* | X; \alpha) = \Pr(D = d^* | X = x)
\]

Literature shows that conditional independence holds via the GPS:

\[
Y(d^*) \perp \perp D = d^* | \pi(d^* | X; \alpha) \quad \text{for all} \quad d^* \in D
\]

providing a means of adjusting for confounding across doses of interest.

We use a 4 step **semiparametric regression** approach:

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

ii. Adjust for confounding via a mean response model: \( \mathbb{E}[Y | D, \hat{\pi}(d | X; \hat{\alpha}); \beta] \)

iii. Use \( \hat{\beta} \) to calculate \( \hat{\mu}(d^*) = \mathbb{E}[Y(d^*)] = \mathbb{E}_X \left[ \mathbb{E}(Y | d^*, \hat{\pi}(d^* | X; \hat{\alpha}); \hat{\beta} \right], \) and repeat for all doses of interest.

iv. Calculate **ATEs**: \( \hat{\tau}(d^*) = \hat{\mu}(d^*) - \hat{\mu}(0) \), using (block) bootstrap for variance estimation.
Advantages of the causal GPS approach

- Clearly defined ‘causal’ framework based on measurable manipulation of a treatment
- Circumvents need for a comprehensive theoretical model, though theory informs selection of confounders
- Approach can estimate ATEs across multiple doses rather than a single point estimate
- Modelling with a scalar PS, rather than high-dimensional $X$, allows use of flexible forms (i.e. GAMs and high-order polynomial)
- 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 accommodate measured confounding, unmeasured time-invariant confounding, and bi-directionality between response and treatment
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 $y_{i,t-p}$, 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 demand (vmt), network performance (delay per vmt), and productivity (average wage)
- **Treatment**: annual proportional change in network lane miles
- **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 (unknown) confounders**: zone / area / region characteristics, road network design, activity/travel behaviour.
  - Random city-level effects specified in longitudinal mixed models
- **Models**: Normal GAMMs for all sub models
Results: demand (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)

- Capacity expansions have not ameliorated urban congestion.
- Average road user has not experienced change in delay from capacity expansions.
- No statistically significant effects on delay per vmt.
- This is the case even for large capacity expansions.
- Due to natural growth, congestion has worsened (approx. 3% p.a.).
Results: productivity (average MSA wage)

- Urban road network expansions have not induced higher productivity.
- ‘Naïve’ regressions of productivity on treatment do indicate a positive association.
- But no significant ATEs having isolated a viable sample and adjusted for confounding.
- No change in transaction costs and apparently no scale effects.
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
★ increased mobility with aggregate volume / capacity ratios constant
★ network generalised costs do not improve and total urban delay rises
★ the scale (increased traffic) effect does not appear to influence productivity (either +ve or -ve)

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