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

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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

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

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

$$f_Z(z) = f_{Y|D,X}(y|d,x) f_{D|X}(d|x) f_X(x)$$ ▸ example

Implies that $Y(d) \not\perp\!\!\!\perp D = d$, 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

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

so we can estimate ATEs by somehow adjusting for X. We do so via *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

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

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: $\widehat{\pi}(d|X;\widehat{\alpha})$

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

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

iv. Calculate **ATEs**: $\widehat{\tau}(d^*) = \widehat{\mu}(d^*) - \widehat{\mu}(0)$, 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: $\widehat{\pi}(d^*|x_{it}, u_i; \widehat{\alpha})$
- ⋆ **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: $\widehat{\pi}(d^*|x_{it}, u_i, \mathcal{H}^y_{i,t-1}; \widehat{\alpha})$
- ⋆ **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: $\widehat{\pi}(d^*|x_{it}, u_i, \mathcal{H}^y_{i,t-1}, \mathcal{H}^d_{i,t-1}; \widehat{\alpha})$

Algorithm for ATE estimation via mixed GPS model

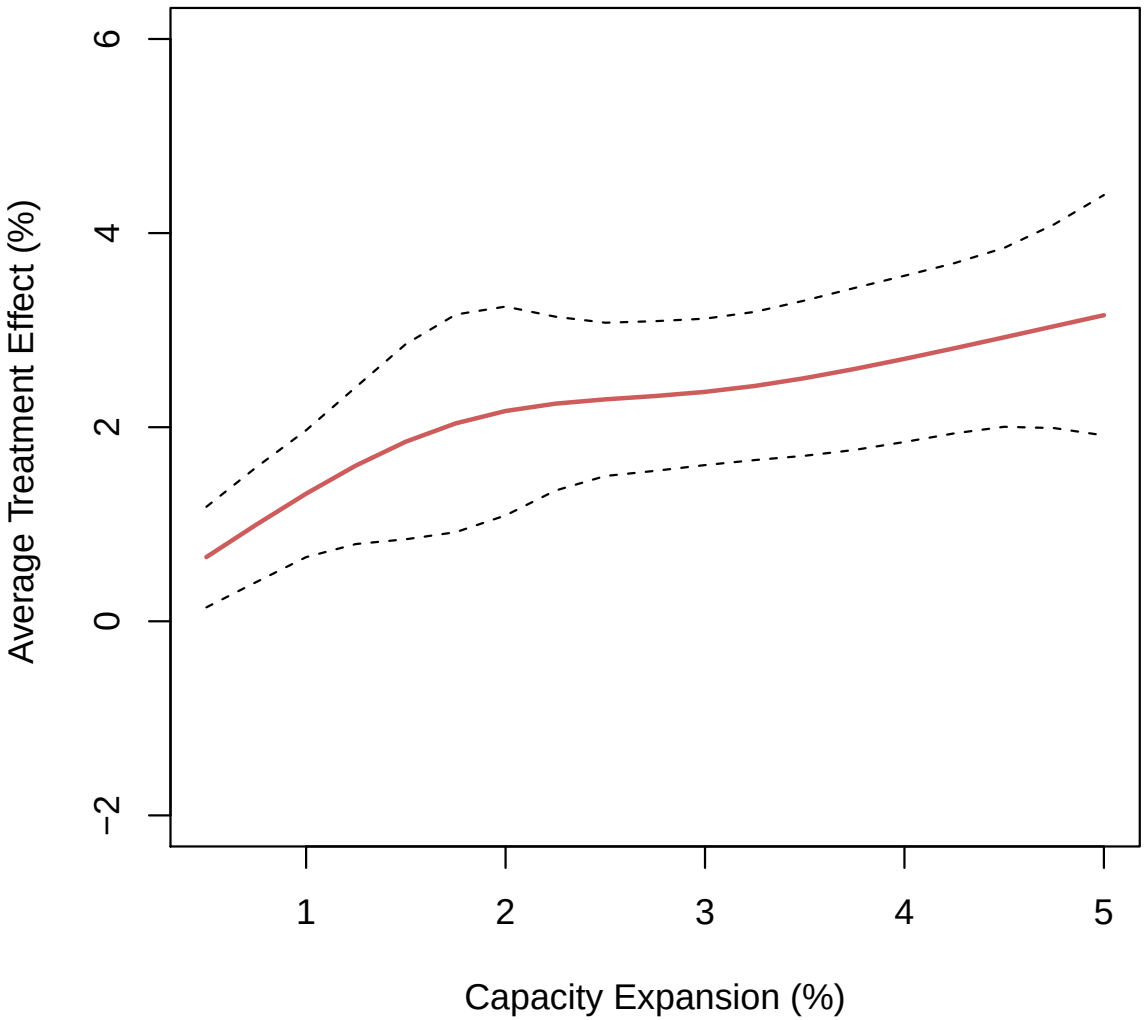
1. Use a flexible mixed model (i.e. GAMM) to estimate $f_{D|X}(d|x,u;\alpha)$
2. Use $\widehat{\alpha}$, with the appropriate density function, to calculate the GPSs: $\widehat{\pi}(d^*|x,u;\widehat{\alpha})$, for all d^* of interest
3. Ensure common support by selecting only units which have a reasonable probability of being treated across the range of dose
4. Estimate $\mathbb{E}(Y|D,\widehat{\pi}(d|x,u;\widehat{\alpha}))$ 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^*: $\widehat{\mu}(d^*)$
6. Repeat for all dose of interest, form the dose-response curve, and estimate ATEs:
$$\widehat{\tau}(d^*) = \widehat{\mu}(d^*) - \widehat{\mu}(0)$$
7. Use a single (block) bootstrap re-sampling scheme over 1. to 6. to obtain standard errors

Data: TTI urban mobility data, 101 US cities, (1982-2007)

Urban longitudinal data (TTI and MSA)

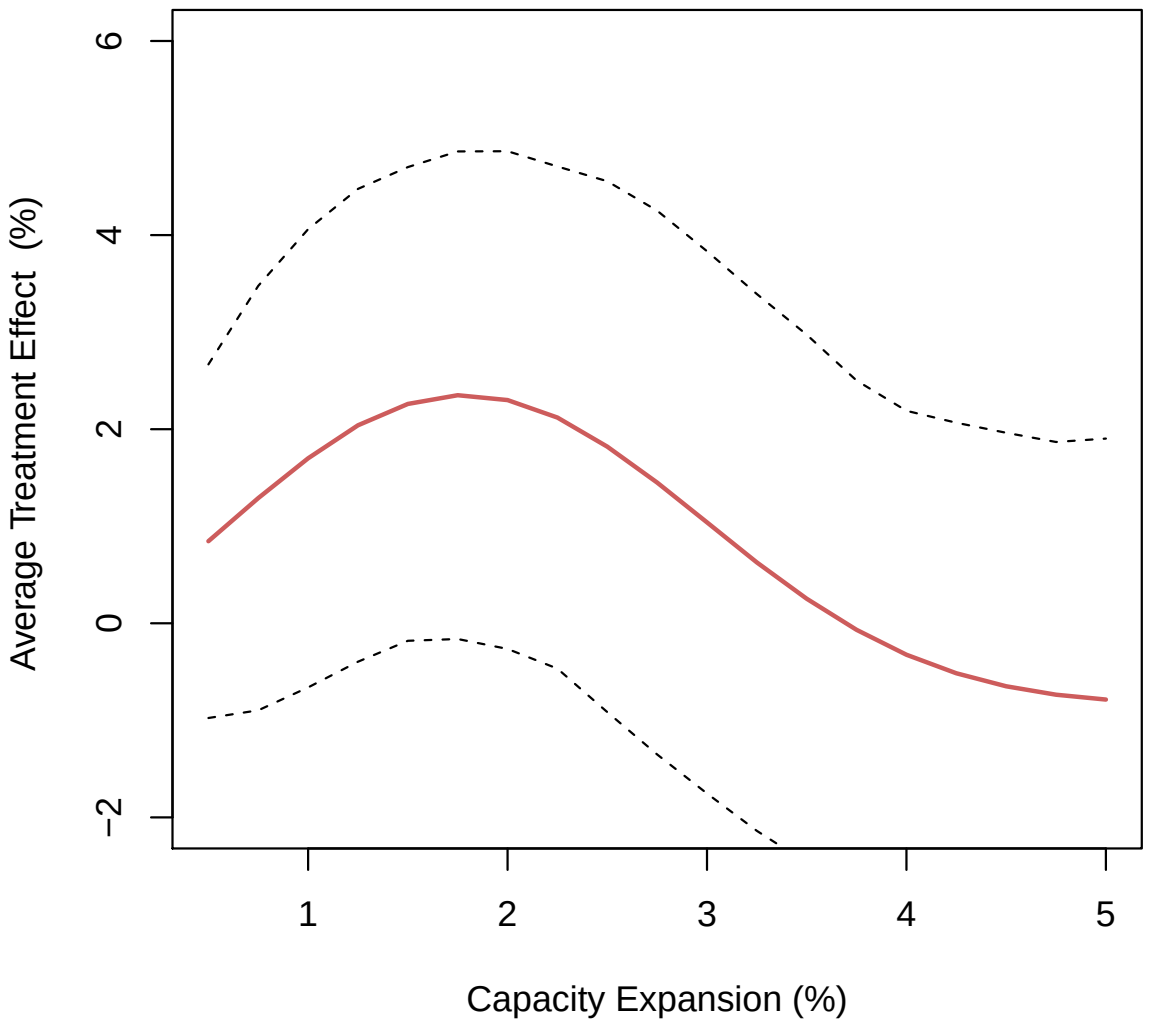
- **Responses:** annual proportional change in demand (vmt), network performance (delay per vmt), and productivity (average wage)
- **Treatment:** annual proportional change in network lane miles
- **Pre-treatment covariates (confounders):**
  - ⋆ *Lagged responses:* to capture reverse causality
  - ⋆ *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)



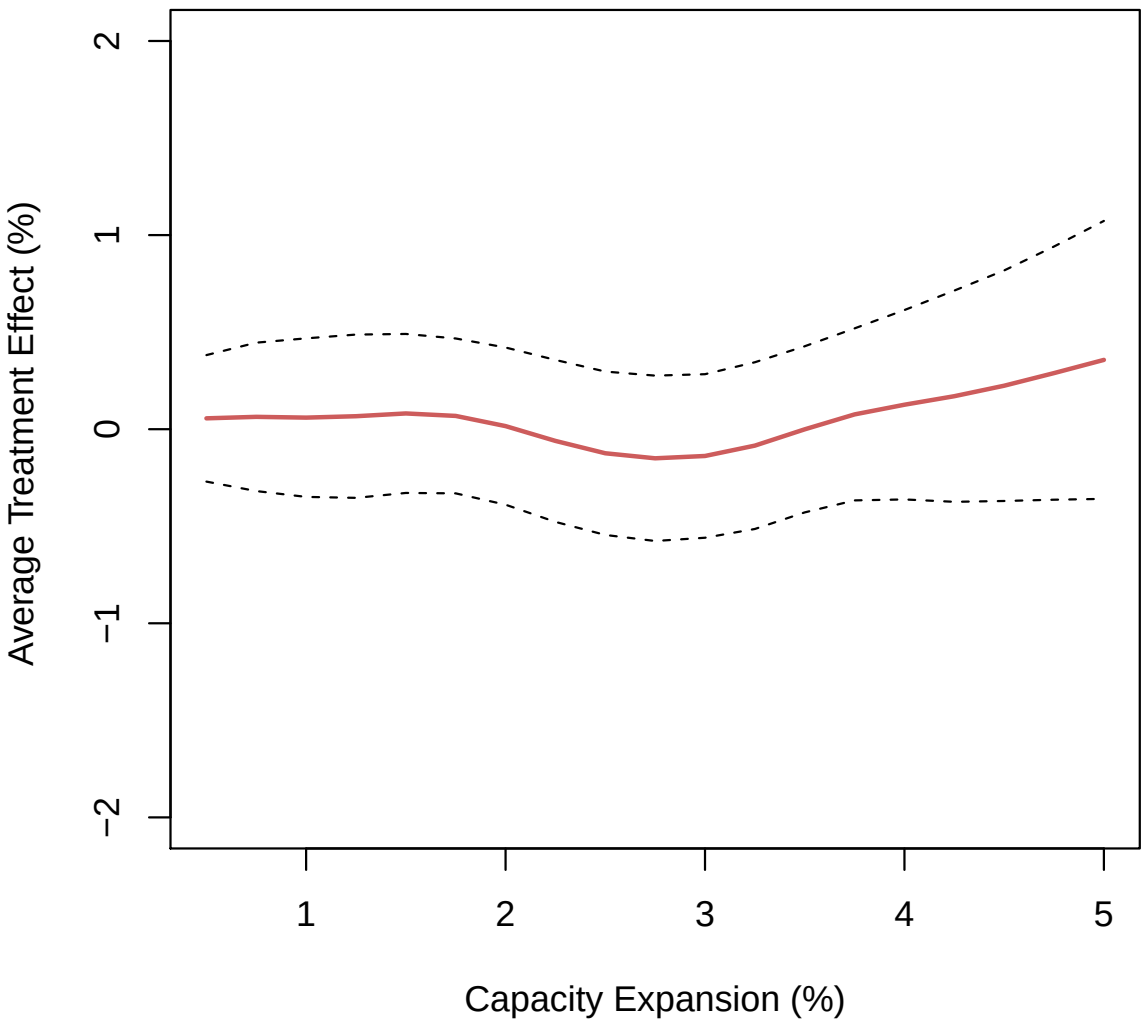
- ⋆ evidence of induced demand over the range of dose having adjusted for confounding
- ⋆ ATE $>$ proportional to treatment for doses ≤ 2
- ⋆ on average 10% increase in lane miles $\rightarrow$ 9% increase in vmt net of 'natural growth' (estimated 1.4% p.a.)
- ⋆ capacity expansions in the range considered 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
- ⋆ 'naive' regressions of productivity on treatment do indicate a +ve 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

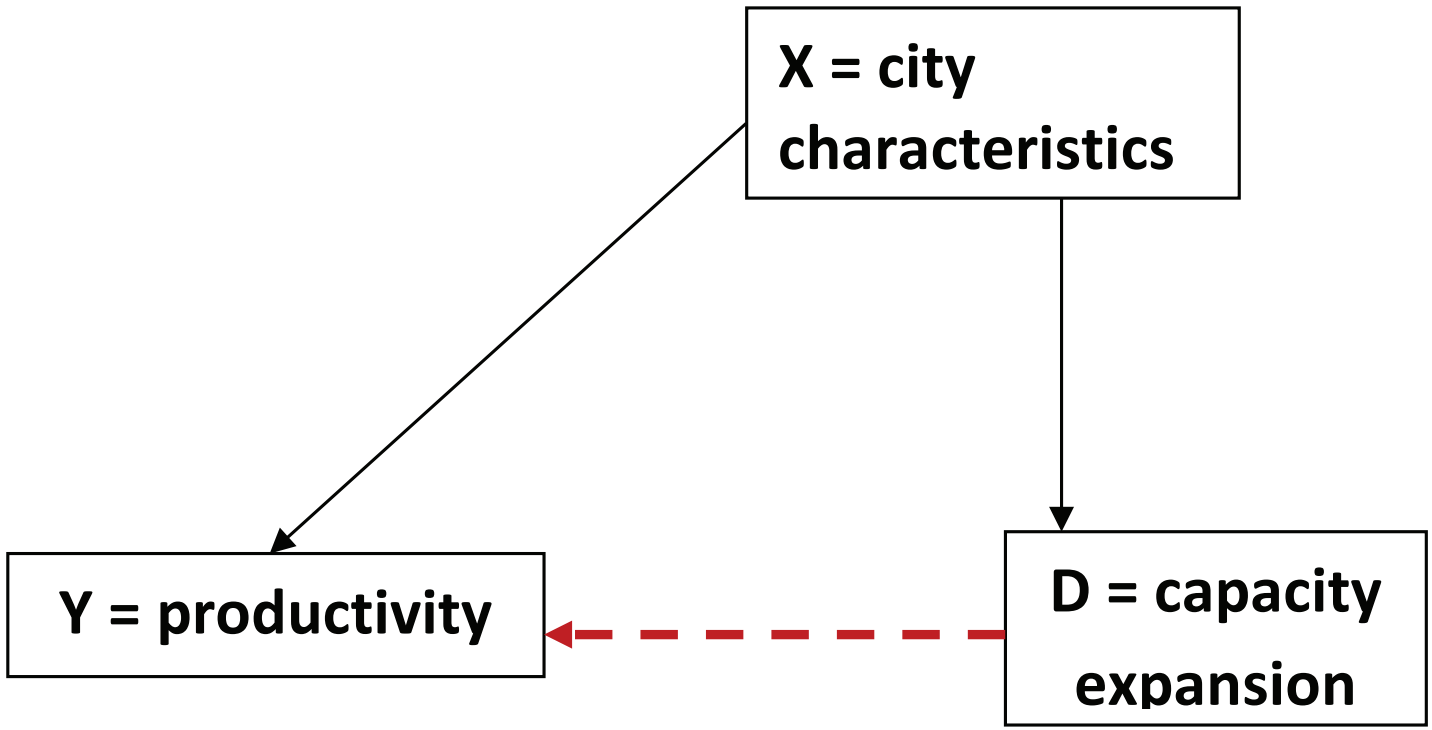
Model indicates that urban road network expansions have induced demand but have not ameliorated congestion or raised productivity

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

return