Quantifying the impact of road network capacity expansion on congestion and productivity via a mixed model generalised propensity score estimator

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1 Introduction

There is an extensive body of literature on congestion and its assumed relationship with network capacity. The nature of this relationship is a contentious issue amongst transport engineers as some propose capacity expansion as a remedy for traffic congestion while others argue that capacity expansions themselves generate additional traffic. The latter viewpoint is based on the theory of 'induced demand', which argues that capacity expansions reduce travel times on the network, at least initially, causing the impedance (or cost) of trips to fall, which in turn cause the level of demand to rise. Impacts on network congestion and flow will in turn affect the cost and scale of economic transactions with potential implications productivity and growth. In this paper, we estimate the impact of capacity expansions on congestion and productivity for US cities using a propensity score (PS) based treatment effects approach. The data available for analysis take the form of a panel for 125 US cities over a 25 year period.

2 Methods

The typical set up for propensity score models under the potential outcomes framework is one in which the data available for estimation take the form of a random vector, $z_i =$

(y_i, d_i, x_i), $i = 1, ..., n$, where for the i-th unit of observation y_i denotes a response, d_i the treatment (or exposure) received, and x_i a vector of pre-treatment covariates. In the absence of experimental data we cannot assume that the treatment (or exposure) is assigned randomly, and consequently, simple comparisons of mean responses across different treatment groups will not in general reveal a 'causal' effect due to potential for confounding. If, however, the vector of covariates x_i can be used to ensure conditional independence of potential outcomes and treatment assignment, then consistent 'causal' estimates of treatment effects can be obtained in a variety of ways.

The conditional independence, or unconfoundedness, assumption is key and in the case of binary treatments amounts to:

$$(Y_i(0), Y_i(1)) \perp\!\!\!\perp D_i | X_i, \tag{1}$$

where $Y(1)$ and $Y(0)$ indicate potential outcomes under treated or control status respectively.

The assumption of 'unconfoundedness' can be restated using a scalar known as the propensity score, which measures the conditional probability of assignment to the treatment given the covariates,

$$e(x) = Pr(D_i = 1 | X_i = x).$$

If unconfoundedness given X_i holds, and if the propensity score effectively balances the distribution of the observed covariates within strata of of the sample that have the same propensity score such that $X_i \perp\!\!\!\perp D_i | e(X_i)$, then $(Y_i(0), Y_i(1)) \perp\!\!\!\perp D_i | e(X_i)$.

The development of a number of useful nonparametric estimators for binary treatments, based for instance on matching, stratification and weighting, has relied on the propensity score as a minimal sufficient reduction of the potentially high dimensional covariate vector X_i. More recently, propensity score methods have allowed the potential outcomes framework to be extended to multi-valued and continuous treatments, in which a treatment $D = d$ can take values in k categories $\mathcal{D} \equiv (d_0, d_1, ..., d_k)$ or in some bounded interval in $\mathbb{R}$ [2, 1]. The relevant question here is what is the mean response to a given dose.

3 Methodological contribution

This paper presents a mixed model propensity score (PS) approach for quantification of dose-response relationships. The problem of interest in one in which estimates of the average treatment effect (ATE) of a continuous exposure are required at various doses, but unobserved confounding is present. The paper shows that a mixed model PS approach can be useful in adjusting for unobserved heterogeneity, potentially leading to improved quantification of dose-response relationships. However, since the predicted random effects cannot distinguish between unobserved effects that arise from confounding or non-confounding characteristics, the approach involves more extensive conditioning than is strictly necessary for causal comparison. The paper shows that while this can adversely affect the efficiency of ATE estimation, consistent estimates of the ATE can still be obtained. Moreover, we also show that the PS approach provides a practical means of ensuring that overlap exists in support of the covariate distributions which is a necessary pre-condition for establishment of causal effects.

4 Results

Our analysis investigates how congestion and productivity (the responses) reacts to changes in the size of the road network (the dose), but recognises that the causal treatment effect is obscured due to confounding and reverse causality. The results show that network capacity expansions do not tend to eliminate, or even reduce, road traffic congestion. Due to induced demand, the delay experienced by the road user remains much the same over an extensive range of dose and total delay at the metropolitan level increases. However, by allowing a greater volume of trips to take place, productivity benefits can arise from capacity expansions through scale effects not dissimilar to those of agglomeration.

References

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