External Effects of Diesel Trucks Circulating Inside the São Paulo Megacity

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Abstract The medical literature documents adverse health effects of acute exposure to diesel exhaust, yet quasi-experimental evidence of a policy intervention sustained over months at the scale of a metropolis is lacking. Exploiting the inauguration of a beltway that removed 20,000 cargo trucks passing daily through inner-city roads in São Paulo, we examine the spatially differentiated impacts on the megacity's traffic, air quality and public health. We combine rich panel data on road congestion, ambient NOx concentrations (as a signature of diesel exhaust), and hospital admissions and deaths. The policy reduced congestion, pollution, and hospitalizations, with effects attenuating at increasing distances from a key inner-city corridor used by the transit trucks prior to the beltway opening. The change in congestion was transient, as gasoline-ethanol passenger cars responded by filling the space the diesel trucks left behind. Effects on air and health persisted thanks to the compositional change in road users. We use 2SLS regression, taking policy-induced variation in NOx to instrument for measured pollution, to quantify about one annual hospitalization for every 10 to 20 trucks – and one annual death for every 100 to 200 trucks – using inner-city roads. Policymakers in megacities where humans and diesel vehicles reside and transit in close proximity may learn from São Paulo's experience.

Keywords: Diesel exhaust; diesel particulate matter; truck emissions; air pollutants/adverse effects; public health; transport policy; fundamental law of road congestion; megacities

JEL codes: H23, I18, Q51, Q53, R11, R41

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1. Introduction.

Road vehicles are a major source of localized air pollution in the world's megacities, right by where people circulate and live (Molina and Molina, 2004). Whereas a sizable literature examines the public health impacts of urban traffic flows, there is much less research on the health impact of the *fleet composition* associated with these aggregate traffic quantities, heavy-duty *diesel* vehicles in particular (Beatty and Shimshack, 2011; Brunekreef et al., 1997). Due to data limitations, the literature typically lumps vehicle emissions – whether from diesel or gasoline engines, heavy versus light duty, aging or new – together as a single category of environmental bads.[1] Moreover, to the best of our knowledge, there is no observational study of a fleet-composition intervention on public health, air quality and road congestion at the scale of a real-world megacity.[2] A recent cross-disciplinary review calls for more quasi-experimental evidence, based on identifiable and credibly exogenous policy shifts, to complement observational studies that document statistical associations among human health outcomes and airborne pollutants (Dominici et al., 2014). In the context above, one such policy would shift the diesel-gasoline urban transportation fuel mix while holding the usage of urban roads fixed. Cities in rich Europe, where the penetration of diesel-fueled vehicles is high relative to North America, as well as in the

[1] Small and Kazimi (1995) compute the external harm per vehicle-mile for the 1992 Los Angeles fleet as 3.3 cents for a gasoline car and 52.7 cents for a heavy-duty diesel truck (1992 US dollars, only health damage was estimated). Auffhammer (2017) writes: "Big trucks are largely powered by diesels… The externalities from these trucks are likely significant in terms of pollution, congestion and accidents. But I am aware of next to no papers in the economics literature which have attempted to quantify these externalities."

[2] Some work has measured the impact of traffic composition on airborne pollutant concentrations. Kinney et al. (2000) and Lena et al. (2002) associate black carbon concentrations with concurrent traffic counts for diesel trucks and buses along New York city streets. Wolff (2014) studies the effect on particle pollution (PM10) in some German cities that implemented "low-emission zones" restricting the circulation of vehicles according to their age (Euro 1 to 4) and fuel (diesel versus gasoline).

developing world, such as Delhi and Calcutta, are considering whether to restrict the use of diesel (Economist, 2016; Mitral, 2016; Sharman, 2015).[3]

Seeking to fill this gap, this study exploits a rare large-scale quasi-experiment in the subtropical megacity of São Paulo, of population 20 million. The metropolis lies on busy commercial routes that connect the Atlantic port of Santos, Latin America's largest and 100 km away, to the wider state of São Paulo, which alone accounts for one-third of Brazil's Gross Domestic Product. Until the recent opening of a beltway along mostly undeveloped land at a 25-km radius from the city center, each day about 20,000 heavy vehicles transiting between points other than São Paulo city (including Santos port) had to pass through congested inner-city roads. Described by the state government as "the most important infrastructure project for the state," the beltway's southern section, inaugurated in April 2010, immediately enabled the passing commercial vehicles to bypass the densely populated city, in particular, a key inner-city corridor, Avenida dos Bandeirantes, en route to and from the port. Alleviating traffic congestion was the policy's stated goal: "The project will prevent these (19,000) heavy-duty vehicles, as well as passenger cars, from transiting through the city, causing severe traffic congestion" (São Paulo State Government, 2008). Indeed, Appendix Figure A.1 confirms that about 20,000 heavy vehicles (3 axles or more) paid tolls per day in 2011 along the beltway's southern section. On top of this, in the immediate wake of the beltway, the São Paulo city government tightened restrictions on trucks circulating in the inner city. Since 2008, also with the goal of relieving congestion, restrictions had applied to a proportion of the truck fleet, certain roads and times of the day (Mayor's Office, 2008). Over the third quarter of 2010, these restrictions became more stringent,

[3] Economist (2017) writes: "NOx emissions cause the premature deaths of an estimated 72,000 Europeans a year… This week the city of Oslo used new powers to ban diesel cars temporarily in order to improve air quality. Paris, Madrid and Athens are set to ban diesels altogether by 2025." Singapore's latest budget selectively introduces a volume-based duty on diesel to reduce its use (Lam, 2017).

for example, along Av. dos Bandeirantes, during weekday daytime hours, and for larger trucks in particular (Diário Oficial da Cidade, 2010).[4]

We document three unintended consequences of road congestion policies to abate truck flows inside the São Paulo metropolis – which we hereafter refer to as "truck abatement policy" or simply "policy." First, examining over 5,100 traffic-monitored road segments across the city, we find that the truck policy did instantly relieve road congestion, by about 20% emanating from the key inner-city corridor used previously by the passing trucks, but this effect on traffic volume was short-lived, as passenger cars substituted into the scarce inner-city road space that the trucks left behind. The behavioral response by households in a city with gridlocked roads was such that within a couple of years the traffic relief brought about by the policy was largely undone. Our study provides a clean and vivid demonstration of the "fundamental law of road congestion."[5]

Second, the policy achieved abatement in diesel pollution, as proxied by ambient NOx (nitrogen oxides, $NO + NO_2$) concentrations (Anenberg et al., 2017), again emanating from the key inner-city corridor. This second effect on air was not an original policy motivation – it is in this sense that this effect was "unintended." Importantly, the drop in NOx levels survived the return of traffic volumes to pre-policy levels, as gasoline-ethanol passenger cars increased their share of road space. Our finding that pollution levels remained lower even after traffic congestion rebounded demonstrates the critical influence of fleet composition beyond fleet size.

The policy's third unintended consequence was a long-lasting reduction in public health damage, also due to the compositional change in traffic, away from heavy-duty diesel vehicles

[4] Restrictions on the circulation of light vehicles, based on registration plate and similar to those in Beijing, Bogotá and Mexico City (Davis, 2008; Eskeland and Feyzioglu, 1997; Gallego et al., 2013; Viard and Fu, 2015; Zhang et al., 2017), have been in place since the late 1990s.

[5] We provide references on this phenomenon below. Appendix A.1 (online) provides a simple model of this mechanism. Relatedly, Gallego et al. (2013) document the fast response by households to transport policy shocks on car use.

toward gasoline-ethanol light vehicles. With a study period between 2008 and 2013, and 3.3 million individual public hospital records aggregated up to the level of month by patient's residential zip code by age group, the health effect we examine is for changes in exposure sustained over months, not from daily variation in diesel exhaust.[6] Consistent with the policy's effect on air, we find that cardiovascular and respiratory hospitalizations abated in increasing proximity to the key inner-city corridor, and particularly in the vulnerable age groups. Estimates amount to 886 (standard error, s.e., of 141) less public hospital admissions (down 8% from total) and 116 (s.e. 37) less in-hospital deaths (down 9%) per year per one million residents per 10 ppb (parts per billion) abatement in NOx equivalent units of diesel pollution. We quantify one annual hospitalization for every 11 to 23 diesel trucks – and one annual death for every 86 to 172 trucks – using roads with high human exposure. We test for the potentially confounding "sorting" hypothesis, by which wealthier, more educated and healthier households would have moved in response to the policy, by shortening the sample period further, as well as by including admissions that are unlikely to be triggered by pollution as additional controls in our health equations (wealthier households are presumably healthier across the disease range).

Our goal is not to provide benefit-cost analysis of alternative policies to abate diesel pollution. In particular, our intent is not to evaluate the net benefits associated with the beltway and tightened truck circulation, or how a beltway on undeveloped land today might affect urban sprawl and economic growth (and thus emissions and human exposure) in the future, questions we leave for subsequent research.[7] What we do is *use* these interventions, irrespective of whether they

[6] At the same time, we do not examine the effect of chronic exposure to diesel exhaust, i.e., sustained over years and decades (Chen et al., 2015; Garshick et al., 2004; USEPA, 2002). We subsequently provide references to the medical literature on health impacts from acute exposure to diesel combustion, and do not repeat them here.
[7] For example, logistics or retail operations might locate along the beltway (Souza, 2009). Anecdotal evidence suggests that land development was not immediate, perhaps in part due to the distance (a 25-km radius) from the city center, or land use restrictions. For example, by February 2011, ten months after inauguration, no fuel retailer had located, or been allowed to locate, along the beltway's southern section (Transporta Brasil, 2011).

are sound, to evaluate the health damage – today – caused by human exposure to diesel exhaust. Our contribution is to quantify external effects of an actual, not hypothetical, diesel truck fleet observed transiting in the densely urbanized areas of a real-world megacity, under existing operating/maintenance conditions, in close proximity to human populations. By sharing the São Paulo experience, we hope to alert policymakers in urban areas with similar levels of immediate exposure, in developing and rich nations alike – such as Europe, Singapore and India.[8]

2. Data and reduced-form econometric models of environmental outcome variables.

2.1 Summary statistics.

We combine spatial data on the different outcomes of interest, namely traffic volume, exposure to diesel pollution, and hospital admissions, along with data on meteorology and fuel prices, variables that may influence traffic, pollution and/or health. The traffic, air and hospital data are not only of interest in their own right but, importantly, brought together they tell a consistent, credible story of how different urban road users (firms, households) responded to the policy, and what their behavioral response reveals about the external damage from the use of diesel. The study period is November 1, 2008 to May 31, 2013. Table 1 provides summary statistics for our datasets.

We proxy for traffic volume using hourly observations on road congestion at the road segment level. The traffic authority monitors a grid of main roads comprising 5,133 segments across the city, with a total extension of 406 km (Figure 1(3)). Thus, the unit of observation measures 80 m on average. For each road segment by hour pair, we observe "congestion" or "no congestion," per the traffic authority's definition. There were 1,070 regular weekdays (i.e.,

[8] For example, Kumar et al. (2011) estimated that heavy-duty diesel vehicles contributed 65% of nanoparticle number emissions in Delhi in 2010, but only 4% of vehicle kilometers traveled. In a source contribution study in rich Singapore, Engling et al. (2014) find that diesel exhaust contributes a dominant 62% to suspended particulate matter on typical days.

workdays excluding public holidays and a vacation fortnight around New Year) in the study period; on these weekdays' afternoon commute hours, between 5pm and 8pm, the extent of congestion throughout the city averages 83 km. Further reflecting the state of gridlock, the proportion of the afternoon commute that a road segment exhibits congestion (from 0 to 1, if congested continuously from 5pm to 8pm) averages 0.15 across 5.5 million road segment by weekday pairs.

As a signature of diesel exhaust (Pérez-Martínez et al., 2015; Pérez-Martínez et al., 2014), we observe hourly ambient NOx concentrations at each of 11 monitoring sites across the São Paulo metropolis, all located inside of the beltway and mostly in the inner city (Figure 1(2), sites marked with circles). The 24-hour mean concentration averages 64 ppb across 10,364 site by weekday pairs and 56 ppb if all days including weekends are considered.[9] We also observe hourly NOx concentrations at monitoring sites located in three cities 50-100 km away from the metropolis (Figure 1(1)). NOx levels at these sites outside the metropolis are comparable to levels in the metropolis excluding sites that are close to large city roads.

We note that routine monitoring of 2.5-μm-diameter particulate matter (PM2.5) began only in January 2011, nine months after the beltway opened, and at a single site in the metropolis (CETESB, 2008-2013, several years). "Submicron" particles, with diameter smaller than 1 μm including gas-like "nanoparticles" with diameter measured in nanometers [10] that recent toxicological research suggests are highly damaging to health (Choi et al., 2010; Donaldson et al., 2005; Nel, 2005; Smita et al., 2012), are not officially monitored. Similarly, the authorities do not

[9] 56 ppb is about 105 $\mu g/m^3$, a level that can double at roadside monitors, and lies between 1998 means for New York at 79 $\mu g/m^3$ and Mexico City at 130 $\mu g/m^3$ (Molina and Molina, 2004, Table 2). Based on remote sensing, Beirle et al. (2003, Fig. 1) reports hotspots for global tropospheric NO_2 in São Paulo, Mexico City, Johannesburg, Jakarta, East Asia, Los Angeles, Eastern US, Western Europe and selected Middle Eastern cities.
[10] 1 nm = 10^{-3} μm = 10^{-9} m. A micron is 1 μm or 1000 nm.

monitor black carbon, a species of particulate matter that is understood to be more toxic compared to inorganic compounds such as nitrates, sulfates and crustal material (Lelieveld et al., 2015; Lippmann et al., 2013). For the later part of the study period, Figure 2 reports the tight correlation between NOx, PM2.5, submicron particle and black carbon levels in São Paulo measured by the authorities or researchers concurrently in time and space.[11] The scatterplots are consistent with diesel combustion being a major if not dominant source across these pollutants, including ultrafine and carbonaceous particulates, and underscore the role of routinely monitored NOx as a marker of diesel exhaust. We provide technical references on diesel exhaust and diesel particulate matter (DE and DPM) below.

We obtained admissions at all public hospitals in the metropolis, comprising 3.3 million individual records over the study period. We observe the patient's dates of admission and discharge (including death), her condition on admission (per the International Classification of Disease, ICD-10), the admitting hospital, and the patient's residential zip code and age. Importantly, we use the *patient's* 3-digit zip code, not the hospital's, as a proxy for the site of main exposure to diesel pollution (Figure 1(2), zip codes marked with balloons). To compute incidence, we obtained resident population over time by age group by district (a subset of a zip code; see Appendix A.2). Across the 55 month by 43 zip code (i.e., 2,365) pairs in the sample, mean cardiovascular and respiratory admissions rates are each about 50 per month per 100,000 residents. Admissions due to these conditions that ultimately result in death are an order of magnitude lower (not shown in Table 1 for brevity). We do not observe mortality outside of public hospitals but note that in São Paulo the majority of deaths occur in (public) hospitals (Bravo et al., 2016). Mean monthly

[11] We obtained hourly number concentrations for submicron particles (PM 7-100 nm and PM 7-800 nm, i.e., PM 0.007-0.1 and PM 0.007-0.8), and mass concentrations for black carbon, sampled during field campaigns beginning October 2010 and lasting about one year at a site near to NOx monitoring site 31 in Figure 1 (Salvo et al., 2017).

admissions rates due to trauma, excluding those related to traffic accidents, are 7 per 100,000 residents. Monthly admissions due to all causes are about 400 per 100,000 residents (again not shown), so admissions that are recorded as cardiorespiratory (cardiovascular + respiratory) account for about one-quarter of the total.

2.2 Reduced-form econometric models.

To let the data speak, and prior to developing a structural model of the policy affecting public health through its impact on pollution, we first present reduced-form evidence. We use Ordinary Least Squares (OLS) regression to estimate the following reduced-form model separately for each localized outcome variable, denoted by y_{lt}, namely traffic congestion, diesel pollution and hospital admissions, where *l* and *t* index location and time, respectively:

$$y_{lt} = \alpha_1 I(\text{truck})_t + \alpha_2 d_l + \alpha_3 I(\text{truck})_t d_l + x_{lt}{}'\alpha_{4l} + \phi_l + \delta_t + \varepsilon_{lt} \qquad [1, \textbf{reduced form}]$$

The traffic outcome is the proportion (frequency) of afternoon hours between 5pm and 8pm that a road segment *l* was congested on day *t*. The diesel pollution outcome is the 24-hour mean NOx concentration at a monitoring site *l* on day *t*. Hospital outcomes are, separately, cardiovascular and respiratory admissions rates for the resident population in a zip code *l* in month *t*.

We allow each outcome variable to shift on March 30, 2010, when the beltway opened (and just before inner-city truck circulation was restricted further), indicated by the dummy variable $I(\text{truck})_t$. Put simply, the dummy is set to one for all time periods *t* on or after this date, and zero otherwise. To capture the spatially differentiated policy effects, emanating from the key inner-city corridor – Av. dos Bandeirantes – that prior to the beltway was heavily used by trucks, we interact the time dummy $I(\text{truck})_t$ with location *l*'s distance (in logarithms) to the nearest point on this key inner-city corridor, d_l. The main coefficients of interest to be estimated are thus α_1 and

α_3. The core Av. dos Bandeirantes, labeled "original truck route," is shown in Figure 1(2). Figure 3 shows how levels of traffic congestion in the city systematically fall as one moves away from this key inner-city corridor, an aspect that the policy sought to address.[12]

To account for spatial heterogeneity, we specify location fixed effects, ϕ_l, i.e., separate intercepts by road corridor in the traffic model, by NOx monitoring site in the diesel pollution model, or by residential zip code in the hospital admissions model. In the traffic model, there are 178 road corridors comprising the 5,133 traffic-monitored road segments, such that one road corridor consists of about 30 road segments.[13] In the traffic and pollution models, for which observations are at the day level, day-of-week and week-of-year fixed effects, δ_t, account for systematic weekly and annual cycles. In the hospital admissions model, with more aggregated temporal variation at the monthly level, we use month-of-year fixed effects instead. Except in the traffic model with its numerous locations, for added flexibility we interact seasonal controls (e.g., day-of-week, week-of-year) with location fixed effects – for simplicity, notation in [1] does not reflect this. We further include in δ_t a linear time trend, common across locations, to capture any long-run changes over the 2008 to 2013 sample period.[14] Vector x_{lt} includes extensive controls to account for other potential determinants of traffic, air quality and health outcomes, namely meteorology, the occurrence of atmospheric thermal inversions within 500 m of the ground, and consumer prices for fuels used in the heavy-vehicle (diesel) and light-vehicle (gasoline, ethanol)

[12] For clarity, Figure 3 does not report a policy impact. The figure shows how central Av. dos Bandeirantes is to mobility in São Paulo (a level). Policy impacts across outcomes of interest, including localized short-run effects on road congestion, are illustrated, for example, in Figure 5.

[13] An alternative to specifying 178 corridor fixed effects would be to include 5,133 road segment fixed effects, in which case the latter would subsume the term $\alpha_2 d_l$, since distance varies within corridor but not within segment, the spatial unit of observation. Results are similar.

[14] Appendix Table A.3 specifies year fixed effects. Several time series for the wider economy (omitted for brevity) all paint a picture of economic stability over the 2008-2013 period. These include the size of the economically active population, the mean real wage, public bus ridership and city airport activity in the São Paulo metropolis, as well as industrial activity and wholesale diesel quantities (including the highway market) for São Paulo state.

fleets (see Table 1). The table of results that we present after spelling out our hypotheses summarizes the covariates in each reduced-form model. The idiosyncratic disturbance term in each reduced-form equation is denoted by ε_{lt}.

2.3 Hypotheses.

Reduced-form model [1] estimates the environmental effects of abating truck flows emanating from the key original truck route, and allows the effects at locations to differ based on their distance from this route. We hypothesize that the policy relieved road congestion and, unintentionally, reduced ambient NOx levels (among other unobserved contaminants associated with diesel exhaust) and hospital visits due to cardiovascular and respiratory disease, i.e., $\alpha_1 < 0$ for all three environmental externalities. Moreover, we expect the environmental impacts of abating diesel truck flows to attenuate as we move away from the original truck route, i.e., $\alpha_3 > 0$.

With trucks being induced out of the inner city's gridlocked roads, we further expect that the road space they left behind was gradually filled by light vehicles, consistent with the law of road congestion (Downs, 1962; Duranton and Turner, 2011; Hsu and Zhang, 2014; Vickrey, 1969). Figure 4(1) indeed shows a large drop in recorded congestion (deseasoned) along the key inner-city corridor when the policy was introduced, and that the change in congestion was short-lived. To formally test for this phenomenon, on applying model [1] to traffic congestion we additionally specify a time trend that begins only on the date the beltway opened, and interact this "trend after policy implementation" with d_l, road segment l's (log) distance from the original truck route.[15] We hypothesize that the truck policy's effect on inner-city traffic volume – which was the original policy motivation – to be undone in the medium run as light vehicles selected into newly available

[15] In the alternative specification of Appendix Table A.3, we use, besides a full set of year fixed effects, year fixed effects beginning 2011 × ln(distance) from the original truck route.

road space. (For brevity, only Table 2, but not expression [1], reflect these additional terms.) In contrast, the spatial effects of the policy on diesel pollution and hospitalizations were long lasting, as the policy did change traffic *composition* even if it did not meet the goal of relieving congestion – see Figures 4(2) and 4(3) and the analysis that follows.

2.4 Unintended policy consequences.

Table 2 reports estimates of model [1] applied to traffic volume (column 1), diesel pollution (column 2) and hospitalizations (columns 3 and 4) across different locations in the metropolis as the truck abatement policy was implemented. All of our hypotheses are borne out in the data.[16] First, road congestion, ambient NOx levels and cardiovascular and respiratory hospitalizations *all* fell (with significance at the 1% level) as truck flows in the inner city abated. Second, the reduction in *all* of these environmental outcomes was less pronounced (again, with significance at the 1% level) the more distant the site of exposure – road segment, air monitor *and* residential zip code – from the key original truck route. Third, as Figure 4(1) suggests, we find evidence of reversion in the temporary traffic relief that the abatement of truck flows brought about.[17]

To help interpret Table 2 estimates, Table 3 fits the different models to varying distances from the key inner-city corridor (50 m, 1, 2, 5, 10 km from Av. dos Bandeirantes) at varying points in time (April 2009, 2010, 2012). The proportion of the afternoon commute that a road segment suffered congestion right by the original truck route (50 m) one year prior to policy implementation, in April 2009, was 0.48 (s.e. 0.01) – see panel (1). As fitted by the model,

[16] See Appendix Table A.3 for variations on the reduced-form analysis presented in Table 2.
[17] São Paulo's roads are most congested during the afternoon rush, between 5 pm and 8 pm, thus our focus on these hours (see, for example, Table 2 in Salvo and Wang (2017)). Applying Table 2, specification 1 to the proportion of congestion time during the earlier daytime window from 7am to 4pm, with data also at the road segment by day level, a similar policy impact in space and over time is observed, though it is less pronounced (while equally statistically significant – see Appendix Table A.3, column 1).

congestion at this location fell sharply by 20% right after policy implementation, in April 2010, to 0.38 (s.e. 0.01). Only two years after policy implementation, however, the effect on congestion at this location from removing trucks was 50% undone, with the predicted congestion frequency rising back to 0.43 (s.e. 0.01). In the face of widespread gridlock on the city's roads and repressed demand for travel among city dwellers, passenger cars increased their share of road space (for a simple theoretical economic model of this mechanism, see Appendix A.1). The policy's mild effect on traffic volume only a few years from implementation was its first unintended consequence.

As Table 3, panel (1) further reports, the model's estimate that the policy hardly affected traffic congestion at locations distant from the key inner-city corridor is reassuring. For example, 5 km from Av. dos Bandeirantes congestion frequency was 0.29 in April 2009, slightly down to 0.26 in April 2010, and back up to 0.28 in April 2011. Figure 5(1) provides stark visualization of the immediate policy effect, i.e., in April 2010, estimated by the reduced-form traffic model. To prepare the scatterplot, we use a more flexible variant than in Table 2: instead of constraining the effect to vary in proportion to log distance, we interact the intervention indicator $I(\text{truck})_t$ with 5,133 segment fixed effects and estimate 5,133 coefficients, plotting these against distance.

The second unintended consequence of the policy to induce trucks off the megacity's roads was to abate diesel pollution, particularly right by the densely populated inner-city corridor (Table 2, column 2). The reduced-form model applied to NOx levels predicts that 50 m from the key-inner city corridor, a 24-hour mean of 148 ppb (s.e. 3 ppb) in April 2009, a year before policy implementation, dropped by 25% to 111 ppb (s.e. 4 ppb) on the month that immediately followed the beltway opening, with little variation thereafter (Table 3, panel (2)). For example, two years after implementation, NOx levels 50 m from the core corridor remained at 109 ppb. Again, the predicted abatement in ambient NOx is decreasing in the distance from this core road. For example,

the fitted model suggests that 5 km from Av. dos Bandeirantes, NOx levels did not fall upon policy implementation (116 ppb in April 2010 versus 113 ppb a year earlier).

Emissions inventories published by the environmental authority estimate that heavy-duty (diesel) vehicles account for over three-quarters of vehicular NOx emissions in the metropolis (CETESB, 2008-2013, several years). The passenger car and motorcycle fleets run almost entirely on mixtures of gasoline and ethanol (Salvo and Geiger, 2014). Power generation in southeastern Brazil is mostly hydroelectric, and mild winters imply minimal residential heating (Negri, 2010).[18] Together these features imply that ambient NOx levels in the metropolis serve more broadly as a marker for diesel exhaust, which contains or forms many toxins beyond NOx that are not routinely monitored by the environmental authority, including submicron particles and black carbon shown in Figure 2 (Brito et al., 2013; Burtscher, 2005; Gentner et al., 2012; Kittelson et al., 2004; Liu et al., 2012; Rollins et al., 2012; Vara-Vela et al., 2016; Zhu et al., 2002). In Appendix A.3, we use approximate emission factors and other assumptions to show that diverting 20,000 trucks to the beltway, and replacing each inner-city truck-km by twice as many light-vehicle-km, may have abated the equivalent of 17% and 18%, respectively, of NOx and PM2.5 emissions in the metropolis (against a 2% increase in aggregate CO emissions).[19] Even at the aggregate metropolis level, this was a large-scale intervention, shifting the urban transportation fuel mix and associated pollutants away from diesel.[20]

Reduced-form model estimates of the spatially differentiated removal of trucks on health damage parallel those for diesel pollution. Such joint evidence is important since the dependent

[18] Regarding potential shifts in averting behavior that could make our estimated health effects conservative, we note here that São Paulo homes are typically naturally ventilated, and that the use of face masks is rare.
[19] Anenberg et al. (2017) cites studies to support the statement that around the world "current diesel vehicles emit far more NOx under real-world operating conditions than during laboratory certification testing" (p.467).
[20] For perspective, some other studies in the environmental economics literature exploit interventions of this magnitude, e.g., a 35% drop in NOx emissions due to a cap-and-trade policy in northeastern US (Deschenes et al., 2017); a 20% drop in SO_2 pollution due to the closure of a refinery in Mexico City (Hanna and Oliva, 2015).

variables – hospitalization rates and ambient NOx levels – come from distinct data sources, respectively the health and environmental authorities. The last two columns of Table 2 present estimates for cardiovascular and respiratory hospital admissions for patients of all ages. Hospitalizations for patients living in zip codes near to the key inner-city corridor dropped upon policy implementation and this drop attenuates as we move away from the original truck route. Findings are similar if we condition on vulnerable age groups, such as cardiovascular disease in the elderly subpopulation or respiratory disease among children aged 0-4 years. Table 3 predictions, based on a less-flexible reduced-form model without zip code fixed effects – only for the purpose of identifying a distance coefficient to describe the variation – suggest that monthly hospitalization rates for residences 1 km from the key inner-city corridor dropped by 4 (cardiovascular) and 3 (respiratory) per 100,000, but did not fall 10 km away. Instead, 10 km away, they rose by 3 and 1 per 100,000, respectively.[21] Estimates are similar in Figures 5(3) and 5(4) when we use a more flexible model with the policy indicator $I(\text{truck})_t$ interacted with a full set of zip code fixed effects (rather than an interaction with log distance as in Table 2).

Data plotted in Figure 4(3) shows that monthly cardiovascular rates per 100,000 residents in the 10 zip codes closest versus the 10 zip codes furthest from the core Av. dos Bandeirantes were higher prior to policy implementation (e.g., due to high exposure to diesel pollution) *but similar after the intervention*. Figure 1(2) shows that the 10 zip codes furthest from the core road tend to lie to the northeast of the city, in contrast to the beltway, southwest of it and even further away. Importantly, all the patterns reported up to here come directly from the data; the structural model we turn to next additionally allows us to quantify the public health impact of an actual fleet of diesel-fueled trucks crossing densely populated neighborhoods of a megacity.

[21] Figure 1(2) shows 10 km is still well inside of the beltway. Recall that the beltway lies on mostly undeveloped land at a 25-km radius from the center.

3. Estimating the effect of diesel pollution on health in a real-world megacity.

3.1 Structural econometric model.

The reduced-form analysis separately and significantly linked spatial environmental outcomes of interest – road congestion, ambient NOx and hospitalizations – to the intervention. The empirical strategy we now propose maps month-by-zip code level diesel pollution directly to hospitalizations, by way of a dose-response function – label this the structural health equation.[22] One approach is to fit the health equation by OLS regression. An alternative approach, and our preferred approach, is to fit the health equation using Two-Stage Least Squares (2SLS) regression. The 2SLS estimator requires the use of an instrumental variable that is correlated with the measured dose variable (pollution exposure) and that does not drive the response variable (hospitalizations) directly. The use of 2SLS alleviates concern with regard to possible unobserved determinants of hospitalizations that correlate with diesel pollution – but not with the instrument – that under OLS generate "omitted variable bias," or concern with measurement error in pollution exposure – measurement error that is uncorrelated with the instrument. Measurement error in the measured dose can lead to "attenuation bias," i.e., an OLS estimate of the dose-response slope that is biased toward zero. 2SLS is standard in environmental economics research into the health response to air pollution (Moretti and Neidell, 2011; Schlenker and Walker, 2015). Policy interventions typically function as instruments, to the extent that they affect health outcomes only indirectly through their effect on emissions (Isen et al., 2016).

Our 2SLS estimates use policy-induced variation in NOx as an instrument for pollution variation that is included in the health equation. One takeaway from the reduced-form analysis is

[22] We note that we identify the particular change induced by the natural experiment, not a continuous dosage.

heightened confidence that localized exposure to diesel pollution shifted with the truck policy, $I(\text{truck})_t$. The identifying assumption is that, in our sample, the policy to abate truck flows affected health in the metropolis only by shifting local air pollutant composition, away from diesel exhaust and to gasoline-ethanol emissions.[23] We report not only 2SLS but also OLS estimates of the health effect of exposure to diesel pollution; to preview our results, we obtain larger magnitudes under 2SLS (which tends to be the case in the cited literature).

The health equation is:

$$health_{lt} = \beta_{1l} diesel_{lt} + x_{lt}'\beta_2 + \mu_l + \xi_t + \epsilon_{lt} \qquad [2, \textbf{structural health}]$$

Health outcomes of interest are hospitalizations, including admissions that terminate in death, by age group and by condition – cardiovascular, respiratory and, as a placebo, non-traffic related trauma. Similar to the reduced form, an observation is a residential zip code *l* by month *t* pair (43 zips in the metropolis, 55 months). We proxy for exposure to diesel exhaust, $diesel_{lt}$, using monthly mean ambient NOx at the closest monitor – but not to exceed 5 km of a zip code's district centroid or, for robustness, 10 km (Bravo et al., 2016). Covariates (μ_l, ξ_t, x_{lt}) control for potentially confounding spatial and temporal variation including residential zip code fixed effects, seasonality (month-of-year by zip code fixed effects), month-of-sample fixed effects (more

[23] A threat to instrument validity would be the possibility that the localized truck abatement policies might have boosted economic activity in the surrounding areas. For example, workers in the inner city would now spend less time commuting and thus accept lower wages, benefiting local business; higher business income would then somehow spill over to the local population, helping explain improved health. However, we note from origin-destination surveys reported in Office of Metropolitan Transport (2013) that most workers live far from their workplace, suggesting that local rent sharing is of lesser concern. Median travel time on public transport is over 2 hours/day, and the distribution of travel time was invariant between 2007 and 2012, on either side of the 2010 policy. Over this period, Office of Metropolitan Transport (2013) further reports that growth in both the number of jobs as well as trips by point of origin have been somewhat *less* pronounced in the center of the metropolis (defined as 16% of its total land area) than around the center (84% of the area). Jobs in an even tighter central area that is crossed by the Av. dos Bandeirantes and is home to 3.5 million people (24% of the population in the 43 zip codes listed in Appendix Table A.1) grew at *exactly* the same rate as in the metropolis, +1.5% per year from 2007 to 2012. Moreover, the assumption that the policy intervention benefited inner-city business has been questioned by industry representatives (Martins, 2012). We verify that our inference is robust to relaxing the exclusion restriction, i.e., for a range of direct policy effects on health, likely due in part to the strength of our instruments (Conley et al., 2012).

flexible than a time trend), and meteorology (subsumed by month-of-sample, if not interacted with zip code). In a robustness test, we introduce local road congestion as an additional control in the health equation (since congestion can be an additional channel through which hospitalizations are affected, e.g., Peters et al. (2004)). The econometric error is ϵ_{lt}.[24]

To generate the instrument $\widehat{diesel}_{lt}$, we predict variation in diesel (NOx) pollution that with confidence we attribute to the truck policy, by fitting a model that is spatially more flexible than the reduced-form pollution model [1]:

$$diesel_{lt} = \alpha_1 I(\text{truck})_t + \alpha_{2l,l\in metro} I(\text{truck})_t + x_{lt}{}'\alpha_{3l} + \phi_l + \delta_t + \varepsilon_{lt}$$

[3, **diesel pollution instrument**]

As in the reduced form, an observation is a NOx monitoring site *l* by day *t* pair, except that the sample now includes all sites in cities 50-100 km away from the São Paulo metropolis, in addition to the 11 sites in the metropolis (Figure 1). Model [3] is a "difference in difference" estimator, the "after" versus "before" policy – i.e., $I(\text{truck})_t$ equal to 1 versus 0 – variation in localized NOx in the metropolis (site $l \in metro$) corrected for any common time-varying NOx determinants outside the metropolis ($l \notin metro$) that are omitted. Supporting this assumption, Figure 4(2) shows deseasoned ambient NOx at these "control sites" and at the site in which "treatment" was most severe.[25] As we show in a robustness test, our findings are qualitatively similar if we base our policy instrument only on variation within the metropolis instead.

As we predict $\widehat{diesel}_{lt}$ at the site by day level, we average these fitted values across days within each month; again $\widehat{diesel}_{lt}$ by monitoring site is assigned to zip code of residence based on

[24] Our linear form for the concentration-response is supported by the extant health literature, at least over the range of particle concentrations that São Paulo's population is exposed to (Di et al., 2017; Mills et al., 2015; Pope et al., 2015; WHO, 2006).

[25] Inspection of Figure 4 suggests that the policy impact may have occurred over a few months, rather than instantly when the beltway opened. We caution that Figure 4 describes data, without accounting for meteorology.

proximity, not to exceed a distance threshold (5 km in the base case). Model [2] is then estimated by 2SLS, with $\widehat{diesel}_{lt}$ instrumenting for $diesel_{lt}$. Through model [3] we restrict variation in our diesel exhaust measure in health equation [2] to that explained by the policy, flexibly by location (β_{1l}). To emphasize, our empirical design, including the use of policy to instrument for spatially differentiated changes in pollution, follows other examples in the environmental economics literature (Chay and Greenstone, 2005; Isen et al., 2016; Schlenker and Walker, 2015).[26]

3.2 Effect of truck policy on diesel pollution, by monitor.

Table 4 reports estimates of pollution model [3] across different specifications, to assess sensitivity. In column 1, 24-hour NOx levels at a site only 20 m from the original truck route (site 8) were, on average, 59 ppb (s.e. 3 ppb) lower after the policy was implemented compared to before, relative to average changes over time at sites 50-100 km away (Figure 4(2)). The mean policy effect at four sites 2-6 km from the original truck route is estimated to be an order of magnitude lower, at -6 ppb (s.e. 1 ppb; Table 1 sample mean of 56 ppb). For brevity, Table 4 reports mean effects across sites grouped according to distance from the original truck route, but Figure 5(2) and Appendix Table A.4 show the individual effect estimated for each of the 11 sites in the metropolis, $\alpha_{2l,l\in metro}$.

Individual effects at NOx monitors are quite informative. The three largest reductions occurred at precisely the three sites with the highest levels of ambient NOx from surrounding traffic: besides site 8 (down 59 s.e. 3 ppb), these are sites 10 and 17, with estimated reductions of

[26] To be clear, the estimation routine implements a first stage in which monthly zip code-specific measured NOx levels are projected on fitted NOx (fitted from [3] and averaged across days within a month) and non-pollution covariates included in health equation [2]. Isen et al. (2016), for example, similarly instrument for pollution using fitted pollution imputed from a policy intervention.

17 (s.e. 2) and 12 (s.e. 2) ppb, respectively.[27] Site 17 is located en route to a heavily congested exit to a key highway (Castello Branco), on the inside of the beltway, which trucks bound from and to Santos port were able to bypass after April 2010 (Figure 1), explaining the 12 ppb NOx abatement associated with the policy. For perspective, such reductions, with point estimates of 59, 17 and 12 ppb, are large compared to pre-policy average NOx levels of 146, 75 and 106 ppb at sites 8, 10 and 17, respectively (proportionate changes of -40%, -23% and -11%). Further, we average the immediate change in congestion frequency – road segment level estimates shown in Figure 5(1) – over all road segments within 1 km of each NOx monitor, and compare this with the estimated change in NOx. The (transient) reduction in congestion brought about by the policy is strongly associated with the sustained change in diesel pollution, with a correlation coefficient of 0.91.[28]

Estimated policy effects on localized diesel pollution are robust across specifications. In column 2, we replace month-of-sample fixed effects by a less-flexible quadratic trend and now control for fuel prices (month-of-sample previously accounted for fuel prices). In column 3, we allow the effects of meteorology to vary by site – notice that precision grows. In column 4, we shorten the sample to three years (2009 to 2011) – notice that estimated magnitudes shrink somewhat, as does precision. In column 5, dependent and independent variables are means over daytime hours, 7am to 8pm, when traffic flows are higher, rather than 24-hour (0am to 11pm) means – estimated policy effects grow. We also confirm that our findings are robust to alternatively specifying the daily maximum NOx level, with a sample mean of 227 ppb compared to 110 ppb for the daily mean (not reported in Table 4 for brevity, but see Appendix Table A.3, column 2). Another robustness test we perform is to control for both citywide road congestion and congestion

[27] A plot similar to site 8's Figure 4(2) shows a drop in (deseasoned) NOx across these sites 10 and 17, of over 10 ppb in April 2010 (not shown for brevity).
[28] Seven NOx monitors have road segments monitored for congestion within a 1 km radius (Figures 1(2) and 1(3)). If we consider an alternative radius of 0.5 km from each NOx monitor, the correlation coefficient is 0.92 (N=5).

within a 2 km radius of a site, interacting such controls with site fixed effects for flexibility (again not shown).

Table 4 further indicates that ambient NOx at sites 50-100 km from the São Paulo metropolis were about 6 ppb higher after the policy implementation date compared to before (see the row labeled "Policy implemented," showing α_1 estimates across alternative specifications). In column 6, we exclude sites outside the metropolis from the estimation sample and instead use sites 10-20 km from the original truck route as controls.

3.3 Effects of diesel pollution on morbidity and mortality.

Table 5 reports the mean estimated coefficient of interest in health equation [2], β_{1l}, across the 25 zip codes for which NOx is monitored at a distance of 5 km or less.[29] By age group and health outcome, we report both: (1) 2SLS estimates – where measured NOx ($diesel_{lt}$) is instrumented with the component of NOx variation that is explained by the policy change ($\widehat{diesel}_{lt}$, fitted per specification 1, Table 4); and (ii) OLS estimates – where $diesel_{lt}$ serves as its own instrument.

Coefficients on diesel exposure, by zip code, estimated by 2SLS tend to be 2 to 4 times those estimated by OLS. 2SLS estimates indicate that a *10 ppb abatement* in the diesel exhaust marker generates monthly morbidity benefits per one million residents of: (i) *42 fewer cardiovascular admissions* (s.e. 7; sample mean of 531); and (ii) *32 fewer respiratory admissions* (s.e. 7; sample mean of 432). Conditional on specific age group-disease pairs, monthly admissions rates fall by as much as 208 (age 65+, cardiovascular, s.e. 59) and 229 (age 0-4, respiratory, s.e.

[29] See the table notes and Appendix A.4 for details on how we assign monitor-level NOx to zip codes, and the robustness tests below for similar estimates using different assignment rules (e.g., 34 zip codes for which NOx is monitored within 10 km).

64) per one million residents in the age group. Estimated benefits of diesel control on the lower respiratory disease subcategory are also significant (e.g., pneumonia, not reported).

We also find a significant effect on mortality: a 10 ppb drop in NOx equivalent units of diesel pollution causes *10 fewer monthly in-hospital deaths* per one million residents across age groups and cardiorespiratory conditions (s.e. 3, sample mean of 102). Moreover, estimated impacts of diesel exposure on the placebo – non-traffic related trauma – are not significantly different from zero.

Evaluated at the 2SLS point estimate reported in Table 5, the implied elasticity for all-age cardiorespiratory deaths with respect to diesel pollution is (+10 / 102) / (10 / 56) ≈ 0.55, evaluated at the sample means reported in Tables 5 and 1 for the dependent variable and key regressor, respectively. Similarly, the elasticity among persons aged 65+ years is (+67 / 749) / (10 / 56) ≈ 0.50. These elasticities are three times those implied by a recent study in the US of all-cause mortality among Medicare seniors and different subgroups[30] from long-term (i.e., annual average) exposure to PM2.5 (Di et al., 2017). Mean PM2.5 in the US sample (11 $\mu g/m^3$) is about half the level in São Paulo (20 $\mu g/m^3$). In contrast, Chen et al. (2013) obtain higher elasticities of ~0.9 for cardiorespiratory mortality responses to coal combustion due to a sustained winter heating policy in China.[31] Differences in the chemical and physical composition of emissions likely explain at least in part the different findings across studies; Lelieveld et al. (2015), for example, assumes "carbonaceous PM2.5 is five times more toxic than inorganic particles" (p.367).

Examining traffic-induced pollution variation – overall vehicle exhaust, not specific to diesel or gasoline, and week to week – Knittel et al. (2016) find "a 1 unit decrease in PM10 saves

[30] For perspective, blacks exhibit the highest PM2.5-mortality elasticity, with a point estimate of 0.24.
[31] Averaging across estimates for three alternative 2SLS specifications for the (log) cardiorespiratory mortality rate (Chen et al., 2013, Table 3), +0.20 / (100 / 453.2) ≈ 0.91 (+100 $\mu g/m^3$ of total suspended particulates, with sample mean of 453 $\mu g/m^3$). OLS estimates imply a similar elasticity.

roughly 10 lives per 100,000 live births, an elasticity of approximately 1" (p.350). Currie and Walker (2011) find that a localized intervention that reportedly lowered road congestion and (overall) vehicle emissions reduced the incidence of low weight and prematurity at birth by about 10 percent.[32] A rare study examining an (assumed yet credible) shift in diesel exposure from a school bus retrofit program in Washington state finds "that adopter districts experienced 23 percent fewer children's bronchitis and asthma cases… relative to a control group… (and) 37 percent fewer children's pneumonia cases" (Beatty and Shimshack, 2011, p.997).

OLS estimates, while smaller in magnitude, also indicate statistically significant public health benefits from abating diesel trucks. In what follows, we provide sensitivity analysis showing that the preferred 2SLS estimates are robust to alternative modeling choices. These include the assignment of monitor-level diesel pollution to patients' residential zip code, constructing the instrumental variable according to alternative Table 4 specifications such as dropping NOx monitors 50-100 km away, and adding local traffic volume controls in the health equation.

A key assumption in our analysis is that shifts in local demographic composition in response to the policy – say wealthier, more educated and healthier households moving to zip codes near the key inner-city corridor – were not significant within our relatively short study period. We tried to obtain district-level demographic data to test the assumption, but the available data are collected once every ten years (the last Census occurring the year the beltway opened). Our relatively short sample of 4.5 years, including the before and after intervention periods, suggests that sorting is unlikely to be confounding our estimates. The robustness test reported below that shortens the sample period even further, to three years, is consistent with this. In two

[32] Currie and Walker (2011) lack traffic and pollution data across toll plazas, but they state (see their references) "a crude estimate is that E-ZPass reduced NO_2 emissions from traffic by about 6.8 percent… " (p.70), which would imply elasticities above 1. The study does not discuss the fuel mix (diesel versus gasoline in trucks and cars).

recent studies that used a design based on spatially differentiated pollution abatement over time, sample periods of similar length were specified, of five and six years. The studies did not detect short-run demographic shifts or sorting in response to localized abatement policy – namely the introduction of electronic collection at US highway toll plazas, and the closure of a refinery within Mexico City (Currie and Walker, 2011; Hanna and Oliva, 2015).[33] Moreover, it is plausible that the elderly are less likely to shift residence in response to the policy,[34] as well as less likely to commute[35] and suffer from traffic congestion other than through the pollution it produces (Peters et al., 2004). Thus, the significant estimates we obtain for the elderly subpopulation provide further reassurance that we are picking up a direct health response from exposure to air contaminants.

As further evidence against the sorting hypothesis, a robustness test we report includes non-traffic related trauma admissions as a *control* in the cardiorespiratory health equations. Presumably, were they to be moving near to the inner-city corridor in response to the policy, wealthier and healthier households would require less medical attention on account not only of cardiorespiratory symptoms but also symptoms that are less likely triggered by pollution, such as trauma. As an alternative, we include the *three-quarters of all* admissions that are not cardiorespiratory or trauma related as controls in the cardiorespiratory equations: wealthier and healthier households would require less medical attention across the disease range. This test is likely to err on the side of caution, as other symptoms caused by pollution, or cardiorespiratory symptoms that were misdiagnosed or misclassified, would be included in this control. Our

[33] Office of Metropolitan Transport (2013, Figure 5) shows almost identical distributions for travel time in the São Paulo metropolis, whether on public or private transport, on comparing two years (2007, 2012) on either side of the policy intervention (2010). At least from this angle, horizon and at this level of aggregation, we do not see spatial shifts in the population of residences relative to jobs (about 45% of total daily trips), schools (32%), etc.
[34] Anderson (2016, footnote 7) reports that the median individual aged over 65 in a Los Angeles cross-section has lived at the current location for 25 years.
[35] Office of Metropolitan Transport (2013, Figure 19) reports an inverted-U relationship between commuter age and commuting frequency, with adults aged 30 to 39 traveling (trips/day) more than twice as often as persons aged 60+ years, and, similarly, compared to children under 4 years.

statistically significant estimates of the effect of diesel pollution on hospital admission and death rates survive. We show estimates for these and other tests next.

3.4 Robustness tests.

Table 6 reports estimates for alternative specifications of the health model [2]. Across all specifications, estimated effects of diesel pollution on hospitalizations and in-hospital death rates are of similar magnitude to those in Table 5.

In the order presented in the rows of Table 6: to assign diesel pollution to zip code of residence, we specify the closest NOx monitor within a 10 km, rather than a 5 km, distance of each zip code's district centroid. As another alternative, we specify the mean concentration across all NOx monitors within 5 km (alternatively, 10 km) of a district centroid, rather than the concentration at the closest NOx monitor, and accordingly modify the instrument (NOx variation fitted by the policy variation).

To generate the instrument $\widehat{diesel}_{lt}$, we alternatively use the specifications reported in columns 3 and 6 of Table 4, respectively allowing the effects of meteorology to vary by site,[36] and excluding sites outside the metropolis from the estimation sample and instead using sites 10-20 km from the original truck route as controls. In forming the instrument, we alternatively specify the policy implementation date as May 31, rather than March 30, 2010.

[36] Additional tests reported in Table 6 include interactions of the policy indicator $I(\text{truck})_t$ with wind conditions (speed and direction, per Table 1) in generating the pollution instrument; we do this either by restricting interactions to have common effects across sites in the metropolis, or allow these to vary by site. Thus, exogenous site-specific policy effects on diesel exposure shift with observed wind. We note that winds speeds in the metropolis are about one half those in Chicago and Los Angeles (Anderson, 2016; Herrnstadt and Muehlegger, 2015), the predominant wind direction is southeasterly and northwesterly, and wind patterns have been stable over the sample period.

As an alternative to our preferred November 2008 to May 2013 study period, we shorten the sample to January 2009 to December 2011. Instrument $\widehat{diesel}_{lt}$ is then constructed according to specification 4, Table 4, which also uses the shorter sample.

We specify additional controls in the structural health equation. We include local road congestion (summed across road segments within 2 km of the NOx monitor closest to each district, as explained, and during daytime hours, 7am to 8pm, when traffic flows are higher) interacted with zip code fixed effects for flexibility.[37] As a test of the sorting hypothesis, as discussed above, we include traffic-unrelated trauma admissions, and as an "acid test" we include all admissions other than cardiorespiratory and traffic-unrelated trauma (the mean value in the sample is 305 monthly admissions per 100,000 residents, to be compared to 53 for cardiovascular and 43 for respiratory). We include mean distance between the patient and the admitting hospital to capture, for example, patients seeking better hospitals further from home in response to the temporary traffic relief.

An alternative to specifying month-of-sample fixed effects in the health equation is to use a less-flexible quadratic trend. Instrument $\widehat{diesel}_{lt}$ is then constructed according to specification 2, Table 4, with month-of-sample fixed effects – which subsume fuel prices – replaced by a quadratic time trend and fuel prices. We show estimates for a health equation that includes a quadratic trend but not fuel prices (in which case fuel prices provide instruments in addition to the policy change), and estimates for a specification with both a quadratic trend and fuel prices.

We weigh the 2SLS regression specifying as weights the square root of the zip code population in the given month by age group.

[37] We implement this robustness test in two ways, instrumenting for measured NOx with the component of NOx variation that is explained by: (i) the policy change (NOx fitted per specification 1, Table 4) and, on top of this, (ii) local and citywide road congestion.

As an alternative to reporting the arithmetic mean for β_{1l} across all zip codes l, in the last row within each disease category/death of Table 6 we report a population-weighted average for β_{1l} across all zip codes l.[38]

Finally, Appendix Table A.5, panel A considers the logarithm of the admissions or death rate, by age group-disease pair, as the dependent variable. Effects are consistent with (or in some cases slightly higher) than those in Table 5. Point estimates for respiratory are higher in the vulnerable age groups. Panel B considers the (within-month average of) daily maximum NOx, rather than daily mean NOx, as an alternative measure of the severity of diesel exposure, modifying the instrument accordingly (policy-induced maximum NOx variation). We repeated the analysis, now further stratifying by gender, and find some evidence that respiratory admissions respond to diesel exhaust more for boys than for girls aged 0-4 years, but is similar by age 5-19 years (Clougherty, 2010).[39]

3.5 A multi-pollutant model.

The interpretation we offer for our adopted single-pollutant approach is that of NOx as a wider "indicator" (Dominici et al., 2010) for variation in atmospheric toxins both (i) emitted by diesel combustion – e.g., particles of varying chemical and physical composition such as black carbon and PM2.5 to ultrafine – as well as (ii) formed from or that react with these tailpipe emissions. As

[38] We also estimated a quadratic version of health equation [2], i.e., with exposure terms $\beta_{11l}diesel_{lt} + \beta_{12l}diesel_{lt}^2$, adding $\widehat{diesel}_{lt}^2$ to the vector of instruments (fitted NOx and the square of fitted NOx interacted with zip code fixed effects). The evidence suggests that the linear model is not overly restrictive. Point estimates indicate slight, and statistically insignificant, concavity, or supra-linearity, around the mean NOx in the sample, 56 ppb.

[39] Estimates are available on request. While São Paulo's child population is almost evenly distributed across genders (and slightly more male), by middle age women begin to outlive men, reaching a ratio of 65% to 35% among those aged 75+ years. We find that the response of cardiorespiratory deaths to diesel pollution is significant among elderly women, and for men it is imprecisely estimated, perhaps due in part to the decline of men in that subpopulation.

noted, the bulk of primary and secondary pollutants associated with diesel exhaust, numbering in the hundreds or thousands of compounds, are unmonitored.

One co-pollutant that is monitored, and is depleted by vehicular NO in proximity to roads, is ground-level ozone. Indeed, plots of deseasoned ozone measured at sites 5, 27 and 31, the three sites nearest to the original truck route,[40] show an increase over the fall of 2010, after the beltway opened. However, rising ozone was observed elsewhere in the metropolis and in cities 50-100 km away. This was due in large part to unseasonal rising temperatures that contribute to atmospheric ozone production, illustrating why correcting for meteorology is key (plots omitted for brevity).

Taking the single-pollutant 2SLS model in Tables 4 and 5 as our point of departure, we estimate a multi-pollutant NOx-ozone health model in Tables 7 and 8 (and Table 4 above). Table 7 reports estimates for an ozone pollution model similar to Table 4, but where the dependent variable is the daily ozone concentration either averaged over 24 hours or the maximum 8-hour average.[41] We now require a second exclusion restriction, available by virtue of price-induced shifts in the gasoline-ethanol mix used in the light-vehicle fleet (Salvo and Geiger, 2014). This additional source of identifying variation is temporal, as fluctuations in the price of ethanol relative to gasoline were similar across São Paulo state, so our chosen specification controls for long-run ozone drifts using a quadratic trend as in Table 4, column 2 for NOx. Month-of-sample fixed effects would subsume most of the variation in fuel prices (Table 4, column 1). As seen in Table 7, ozone levels fall in the ethanol-to-gasoline price ratio.[42] Consistent with NO's depletion of

[40] We deseason ozone as in Figure 4(2) for NOx at site 8, which does not monitor ozone.
[41] São Paulo state's 8-hour ozone standard is 140 μg/m^3, or about 71 ppb, comparable to the US EPA's 70 ppb (Salvo and Wang, 2017).
[42] Salvo and Huse (2013) find that the ethanol share among consumers equipped with "flexible fuel" gasoline-ethanol vehicles falls roughly linearly in the ethanol-to-gasoline price ratio. Salvo and Geiger (2014) document lower ozone levels – and unchanged traffic congestion, speeds and public transport use – in the metropolis in response to higher relative ethanol prices that induce shifts to gasoline in the light-vehicle fuel mix.

ozone, when the truck policy was introduced maximum 8-hour ozone rose by 4 μg/m^3 (2 ppb) at the sites closest to the key inner-city corridor, against a mean of 65 μg/m^3 for the metropolis.

Table 8 reports estimates when we include 24-hour or maximum 8-hour $ozone_{lt}$, in addition to $diesel_{lt}$ (i.e., 24-hour NOx), in health equation [2], now with a quadratic trend instead of Table 5's month-of-sample fixed effects to correct for long-run unobserved health determinants. We include NOx and ozone variation induced by the truck policy and ethanol-gasoline prices (fitted NOx and ozone using corresponding specifications in Tables 4 and 7) in the vector of excluded instruments.[43] While point estimates on the NOx coefficients (means over 21 or 33 zip codes[44]) are larger or smaller depending on whether we proxy for multi-pollutant exposure using the closest monitor up to 5 or 10 km from a zip code's district centroid, results are consistent in magnitude and significance with those reported in the single pollutant analysis of Table 5. While the epidemiological/economics literature (Arceo et al., 2016; Gouveia and Fletcher, 2000) finds collinearity in multi-pollutant analysis of health challenging, the key feature of our pollution data is the large drop in NOx emanating from the inner city coinciding with the truck policy.[45]

4. Concluding remarks.

We find that abating diesel truck traffic in the urban core of the São Paulo megacity resulted in sustained reductions in ambient NOx – in its "indicator role" for diesel exhaust known to contain

[43] The instruments are different but the chemistry that underlies the NOx and ozone variation generated by the instruments is the same, highlighting why estimating separate health coefficients can be challenging. Moreover, ethanol-gasoline price variation is only monthly. Recent research also suggests that ozone variation induced by shifts in the gasoline-ethanol mix may inversely correlate with variation in ultrafine particles (Salvo et al., 2017).
[44] NOx and ozone are both monitored within 5 km (resp., 10 km) for 21 (resp., 33) zip codes.
[45] Dominici et al. (2010) discuss the "vast…challenges" the multi-pollutant approach faces and "caution (that) the results of any regression model become highly unstable when incorporating two or more pollutants that are highly correlated" (p.188). Appendix Figure A.2 shows negative correlation between NOx and ozone.

many toxic substances not routinely monitored[46] – of magnitude 5-50 ppb, depending on proximity of roadside exposure, with quantifiable morbidity and mortality benefits. The research focus is not whether traffic pollution harms health, but to identify the types of traffic that cause large damage. Summing cardiovascular and respiratory conditions, we estimate reductions of 886 (s.e. 141) public hospital admissions and 116 (s.e. 37) in-hospital deaths, or 8% and 9% of total, per year per one million residents per 10 ppb abatement in NOx equivalent units of diesel pollution (e.g., Table 5, (4.2 + 3.2) × 12 months/year × 1 million / 100,000). Considering: (i) a 1 to 2 million inner-city population that benefited from a 10 ppb-equivalent diesel pollution abatement, and (ii) a 20,000 abatement in trucks passing daily, we arrive at a back-of-the-envelope *annual impact of one hospitalization for every 11 to 23 diesel trucks, and one death for every 86 to 172 diesel trucks, driving daily through the inner city* (e.g., 20,000 / (886 × 2) to 20,000 / 886).

Studies in toxicology and epidemiology have established causal links between exposure to diesel exhaust and human health damage (Baulig et al., 2003; Castranova et al., 2001; Gauderman et al., 2005; Jalava et al., 2010; Kilburn, 2000; Krivoshto et al., 2008; Marks et al., 2010; McCreanor et al., 2007; Ohtoshi et al., 1998; Patel et al., 2011). Yet, to our knowledge, this is the first observational study to combine evidence at the scale of a gridlocked megacity, exploiting a large, abrupt and identifiable policy-induced shift in the composition of a real-world circulating fleet. The evidence is based on spatially and temporally resolved observations of hospitalizations, ambient diesel pollution, traffic volume, meteorology, and other controls. With measurements from different sources, the joint distribution of variables is compelling. Upon implementation, the beltway-access cum tightened-truck-circulation policy abated road congestion, NOx levels, and

[46] To support this statement, we not only cite the environmental literature; we show the tight correlation between NOx and particles in urban air, specifically in the currently unmonitored and unregulated health-relevant ultrafine size range and carbonaceous composition. To our knowledge, ours is the first economics study to report such data.

cardiorespiratory hospitalizations, particularly in the road segments, air monitoring sites and residential zip codes that were most exposed to the passing truck traffic. Offering a uniquely clean demonstration of the fundamental law of road congestion (Duranton and Turner, 2011), the traffic volume relief brought about by the intervention – its stated motivation – was temporary. However, the resulting shift in road user composition unintentionally improved air quality and public health, enabling us to quantify the health effect of urban exposure to diesel exhaust. More broadly, other world megacities might stand to gain similarly by curbing the circulation of aging heavy-duty diesel fleets at times and locations of high human exposure, including investing in fleet renovation.

Appendix.

A.1 A simple theoretical model: Adding composition effects to the Law of Road Congestion.

To help fix ideas, we lay out a simple conceptual framework. Consider an open-access resource such as urban (inner-city) road space. A user's cost to access this resource (including time spent commuting), denoted $t(q)$, is common across users and is increasing and convex in the aggregate number of users, $q \geq 0$ (Small and Verhoef, 2007). The nominal ("boilerplate") capacity of the resource is of measure k. We thus specify $t(q) > 0$, $t'(q) \geq 0$ and $t''(q) \geq 0$, with $\lim_{q \to k} t(q) \to \infty$. On the demand side, there are two groups of users, l and h, of mass ("market size") m_l and m_h, each user type with heterogeneous preferences over using the resource distributed according to the cumulative density functions F_l and F_h, with domain $\mathbb{R}^+$ (Figure A.3 (a)-(c)). These functions, along with user mass, describe both aggregate demand for the open-access resource as well as the composition of demand. Thus, for example, the number of users of type l with value up to v is given by $m_l F_l(v)$. In our setting, l and h represent light- and heavy-duty vehicles, or low- and high-diesel-exhaust vehicles, respectively. Given our setting, we make the following assumption:

Assumption 1. (Megacity) $m_l + m_h \gg k$

User i's utility from accessing the resource is thus given by $u_i = v_i - t(q)$, with the second term capturing the congestion externality. Normalize the utility from the outside option, u_0, to be equal across user types and users, at 0, i.e., $u_{l0} = u_{h0} = 0$. User i's selection into road space is then given by:

$$\max(0, u_i(q))$$

In equilibrium, the marginal users of each type, with values (v_l, v_h), are defined implicitly (assuming interior solutions) by the system:

$$v_l - t\left(m_l\big(1 - F_l(v_l)\big) + m_h\big(1 - F_h(v_h)\big)\right) = 0$$

$$v_h - t\left(m_l\big(1 - F_l(v_l)\big) + m_h\big(1 - F_h(v_h)\big)\right) = 0$$

where the aggregate number of users, q^*, is $m_l\big(1 - F_l(v_l)\big) + m_h\big(1 - F_h(v_h)\big)$.

We now briefly consider how the composition of road users changes under different shocks. As a summary measure, let s_h denote the type-*h* user share among all users, i.e.,

$$s_h = \frac{m_h\big(1 - F_h(v_h)\big)}{m_l\big(1 - F_l(v_l)\big) + m_h\big(1 - F_h(v_h)\big)}$$

Shock 1. (Opening of a beltway around the urban area) Suppose $du_{h0} > 0$. The "immediate" effect of raising the value of an alternative to urban road space for type-*h* users is that the mass of type-*h* users falls by $m_h\big(F_h(v_h + du_{h0}) - F_h(v_h)\big)$—see shift (i) in Figure A.3 (d)-(f). As urban road space becomes available, and the cost of accessing the resource drops, marginal users of both types select into road space—see shift (ii), Figure A.3 (d)-(f). As illustrated, the higher the density around $F_l(v_l)$ (i.e., the probability density function $\mathrm{d}F_l(v)/dv$ evaluated at v_l) relative to the density at $F_h(v_h + du_{h0})$, the more will the type-*h* share fall after the beltway opens, $ds_h/du_{h0} < 0$. Stated differently, the steeper is $F_l(v_l)$ relative to $F_h(v_h + du_{h0})$—Δu_{h0} is large, say, as in the figure—the more will the type-*h* share fall. The aggregate number of users falls by a measure, $dq^*/du_{h0} < 0$, that depends also on the curvature of $t(q)$.

Shock 2. (Investment in light rail) Suppose $du_{l0} > 0$. This shift in the value of the outside option to type-*l* users is analogous to Shock 1. The ensuing increase in the type-*h* share, $ds_h/du_{l0} > 0$, depends on the density around $F_h(v_h)$ relative to the density at $F_l(v_l + du_{l0})$. Also, $dq^*/du_{l0} < 0$.

Shock 3. (Exogenous demand growth) Consider a proportionate shift in population such that $dm_l/m_l = dm_h/m_h > 0$, with no change to the preference distributions F_l and F_h. It is clear that

this shock will raise the aggregate number of users, q^*, while not necessarily changing the composition of demand—see shift (iii), Figure A.3 (g)-(i). It is intuitive that a combination of shock 1 (or shock 2) followed by population growth may actually result in an increased aggregate number of users.

A.2 Data.

Localized diesel pollution. Hourly ambient NOx mass concentrations at all monitoring sites in the São Paulo metropolis (*N*=11) and in cities 50-100 km away (*N*=4), with measurements over the November 1, 2008 to May 31, 2013 study period, were obtained from the São Paulo State Environmental Protection Agency (CETESB). Across the 1,673 day by 15 site (i.e., 25,095) pairs in the sample, and considering that at most 23 hourly measures are available in a day (with NOx instrument calibration occurring in the hour preceding 1am), data availability is 88% across day-hour-site triples, ranging between 65% and 96% across the different sites. Hourly NOx data availability at the site only 20 m from the original truck route (site 8) is 95%.

For each day by site pair, we compute a 24-hour mean NOx concentration when at least 12 hourly measures are available; otherwise, the 24-hour mean concentration is marked as missing. Following this procedure, we computed 24-hour means for 89% of the 25,095 day-site pairs in the sample, ranging between 66% and 96% across the different sites. For the site only 20 m from the original truck route, we computed 24-hour means for 1,600 of the 1,673 days over the study period (i.e., 96%).

The fact that the São Paulo state EPA maintains, at considerable cost, multiple NOx monitors across the metropolis (Figure 1(2)) indicates the importance of local emissions sources, such as localized truck traffic, and non-uniform mixing across space (CETESB 2011). Indeed,

regressing day-site level 24-hour NOx concentration means in our sample on day-of-week and week-of-year fixed effects, to capture seasonality, yields an R^2 of 14%; importantly, adding site fixed effects to the regression raises R^2 to 56%. Similarly, a large-scale multi-area study in Europe estimated that 70% of NOx variation is attributable to variation from neighborhood to neighborhood (Cyrys et al., 2012).

For use in the multi-pollutant model, we collected hourly ambient ozone mass concentrations at all 12 monitoring sites in the São Paulo metropolis. Seven of these sites double as NOx monitors. The three sites with the highest NOx levels – and those with the largest policy-induced NOx abatement, namely, sites 8, 10 and 17 (Section 3.2) – do not monitor ozone, likely due to the heavy influence of passing traffic (NO emitted from the tailpipe "titrates," or depletes, ozone). The four NOx monitoring sites in cities 50-100 km from the metropolis also monitor ozone. As with NOx, for each day by site pair, we compute a 24-hour mean ozone concentration (as well as a maximum 8-hour mean) when at least 12 hourly measures are available. Ozone measurements are missing even less than for NOx.

Meteorology and thermal inversion. Meteorological conditions were obtained from the São Paulo State Environmental Protection Agency (CETESB, all parameters but precipitation and thermal inversion), the Institute for Meteorology (INMET, precipitation), and the Brazilian Air Force (FAB, thermal inversion). CETESB and INMET data are available by weather station and by hour. We aggregate such data across weather stations inside the São Paulo, following Salvo and Geiger (2014), and into daily means. Thermal inversion data contain records twice a day, at 9am (12:00 UTC) and 9pm (0:00 UTC). Each record informs whether an inversion was detected and, if so, the number of layers of warmer air and the altitude at the top and bottom ("base") of each

layer. We control for the observation of thermal inversion within 500 m from ground level. Table 1 indicates very high data availability across all meteorological controls within the November 1, 2008 to May 31, 2013 study period.

Public hospital admissions and resident population. From the Ministry of Health Information System (DATASUS), we obtained the universe of records for admissions at public hospitals for patients with residential address in the São Paulo metropolitan area. Between November 1, 2008 and May 31, 2013 and across residents of all ages in the 43 zip codes, a total of 1,990 patients were admitted each day on average, including: 251 cardiovascular (ICD-10 codes I00-I99) cases, of which 25 terminated in death; 219 respiratory (J00-J99) of which 25 terminated in death; and 29 non-traffic related trauma (T20-T98). Children aged 0-4 years accounted for 12% of all admissions and 38% of respiratory admissions. Elderly residents (65+ years) accounted for 17% of all, 43% of cardiovascular and 22% of respiratory admissions. Elderly residents accounted for 60% of cardiovascular and respiratory in-hospital deaths.

We obtained resident population by administrative district (all 96 districts in the city of São Paulo, plus 7 adjoining, or "satellite," towns inside the São Paulo metropolis), by age group (5-year intervals) and year (2008 to 2013) from the Brazilian Institute for Geography and Statistics (IBGE). We linearly interpolate the annual data to estimate population by month. Table A.1 lists resident population (in 2010) by district for each of the 43 3-digit residential zip codes in our sample. Since we estimate the structural model using a subset of the 43 zip codes, dropping zip codes for which no district centroid is located within 5 km (or 10 km) of a NOx monitor, Table A.1 also lists the distance from each district's centroid to the closest NOx monitoring site (more below). Table A.2 shows the age distribution across all 43 3-digit residential zip codes over time.

We compute the (straight-line) distance from each hospital to each residential district centroid. We take the minimum of this distance across districts within a zip code (103 districts to 43 zip codes) to proxy for the distance to hospital traveled by an admitted patient with observed residence at a given zip code. Summing across admissions and dividing by the admissions count for each residential zip code by month, the sample mean across 2,365 observations is 5.2 km, with hardly any variation over time (5.2 km in 2009, 2010, 2012 and 5.1 km in 2011).

A.3 Back-of-the-envelope emissions inventory of the truck abatement policy.

We roughly appraise the shift in tailpipe emissions inside the city from removing 19,000 diesel-burning trucks off its roads – to use the government estimate cited in the main text – and replacing these with gasoline-ethanol powered light vehicles.

To do so, we reference the literature on emission factors.[47] We average emission factors in g/km reported in Martins et al. (2006), Sanchez-Ccoyllo et al. (2009), NAEI (2012), Cai et al. (2013), EEA (2014) and Pérez-Martínez et al. (2014). Emission factors can vary widely according to actual driving conditions (e.g., stop-and-go traffic on gridlocked roads) and the fleet's state and age (Beaton et al., 1992), thus our estimate of the abatement in emissions presented here is only indicative and may have a wide margin of error.

Assuming that the 19,000 trucks transited 80 km/day along the city's roads and with an average NOx emission factor of 11.6 g/km, would imply the removal of 6,411 tons of NOx emissions per year. If we assume that each truck-km is replaced, in the medium run, by twice as many light-vehicle-km, each light vehicle with an average NOx emission factor of 0.64 g/km, the

[47] For perspective, emissions standards adopted in 2012 for new heavy-duty diesel vehicles (Phase 7 of PROCONVE) were similar to Euro V, but regulations on new vehicles sold impact the circulating stock only gradually. Earlier plans to tighten regulations in 2009 (Phase 6 of PROCONVE) were not implemented in part due to the unavailability of low sulfur grade (50 ppm) diesel (CETESB, 2008-2013, several years).

net removal would be 5,696 tons of NOx emissions per year. To provide perspective, this would amount to displacing, from the urban area to the beltway, 17% of the aggregate NOx emissions estimated for the São Paulo metropolis by the state's environmental authority (CETESB 2011, with adjustments by Gallardo et al. (2012)). Since the passing trucks concentrated on specific inner-city corridors, such as Av. dos Bandeirantes, generating localized pollution, proportionate reductions in ambient NOx at specific locations should be much higher, as indeed we find (Table 4). Similar calculations suggest that the changes in PM2.5 and CO emissions from replacing the urban road space occupied by each diesel truck by two gasoline-ethanol light vehicles were equivalent to about -18% and +2.3% of aggregate estimated PM2.5 and CO emissions, respectively.

A variation on the above paragraph's calculation is to assume that the space opened up by removing each truck can be filled, in the limit, by three light vehicles. Changes to NOx, PM2.5 and CO emissions would then amount to -16%, -16% and +4.5%, respectively, of aggregate emissions estimated for the metropolis.

Relatedly, Pérez-Martínez et al. (2014) and Pérez-Martínez et al. (2015) show that the ratio of NOx to CO emissions increases in the share of heavy relative to light vehicle traffic (measure ds_h in Appendix A.1). Moreover, in anticipation of a future fine-particle regulatory standard, the São Paulo state EPA began hourly monitoring of ambient PM2.5 concentrations at two sites in the metropolis in 2011, and at another two sites by 2013. Though these measurements post-date the beltway inauguration, PM2.5 levels—once measurements began—correlate positively with NOx. Pairwise correlations between mean daily PM2.5 and NOx range from 0.63 to 0.68 (Figure 2).

A.4 Zip code-level distance to the key inner-city corridor (reduced-form models), and exposure to diesel pollution based on distance to a NOx monitor (structural model).

Following Bravo et al. (2016), we take the coordinates of the centroid of each of the 96 administrative districts in the city of São Paulo, plus 7 adjoining towns inside the São Paulo metropolis, from the Global Administrative Areas database (http://gadm.org/). For each of these 103 "districts" (districts plus adjoining towns), the district centroid is the geographical center of the district, with the exception of Santo André and São Bernardo do Campo, where the large mountain areas to the south are excluded.

For the reduced-form analysis of health outcomes, we calculate the distance from each district centroid to the nearest point on the key inner-city corridor, Av. dos Bandeirantes. We then average the district centroid-key corridor distance across districts within each 3-digit residential zip code. (Aggregating up to the zip code by taking district population as weights yields similar estimates.) Recall that there is a ratio of 103 districts to 43 zip codes (Table A.1) and that we observe a patient's residential address at the 3-digit zip code, not district, level.

To determine exposure to diesel pollution in the structural health equation [2], our preferred specification (Table 5) assigns each district within a zip code to the nearest NOx monitoring site from the district centroid, with a 5 km threshold. We then average NOx across districts within each zip code. To repeat, Table A.1 reports the distance from each district centroid to the nearest NOx monitor. In our preferred specification in the structural analysis, 47 districts in 25 zip codes have the closest NOx monitor within 5 km distance of the district centroid; thus, 25 zip codes are included in the sample. In alternative specifications for the structural analysis (Table 6), we expand the district-to-monitor threshold from 5 to 10 km; the number of zip codes in the sample grows

from 25 to 34. We also test robustness to alternatives where, instead of taking the nearest NOx monitor, we taking the mean across all monitors, still subject to the 5 or 10 km threshold.

A.5 Back-of-the-envelope interpretation of the estimated morbidity and mortality effects of passing trucks.

There is good reason to believe that the approximate morbidity and mortality impacts of the passing trucks that we quantify are conservative. First, we do not have access to admissions and mortality at private hospitals, or mortality at home (Bravo et al., 2016). Second, emergency visits are included in our data insofar as they lead to admissions into hospital; in particular, within-day emergency visits (e.g., in response to an asthma attack which is soon brought under control and the patient goes home) are not covered by our data. Third, we do not quantify the disutility from (willingness-to-pay to avoid) hospitalization, beyond counting hospitalizations and deaths. Fourth, our research design does not capture any long-run effects, such as cancer, from chronic inhalation of diesel exhaust, sustained over years.

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Table 1. Summary statistics for road congestion, NOx concentration (as a proxy for exposure to diesel exhaust), hospital admissions, meteorology, and other control variables. The sample period is November 1, 2008 to May 31, 2013, i.e., 1,673 days or 55 months. An observation is either a day (citywide congestion, meteorology except wind direction, fuel price), a day by monitoring site pair (NOx, wind direction, ozone), a road segment by day (road segment congestion), or a 3-digit residential zip code by month pair (hospital admissions), in the São Paulo metropolis. Citywide congestion is the mean extension during afternoon commute hours (5pm to 8pm). Road segment congestion is the proportion of afternoon commute hours that a road segment exhibits congestion (per traffic authority definition). NOx and meteorology are daily (24-hour) means unless noted otherwise. Sources: ANP, CET, CETESB, DATASUS, FAB, IBGE, INMET.

Variable	N	Mean	Std.Dev.	Min	Max
Road congestion					
Citywide congestion, weekdays only (km)	1,070	83.2	33.9	0.1	226.7
Citywide congestion (km)	1,673	55.8	46.1	0.0	226.7
Road segment congestion, weekday only (a prop.)	5.5m	0.15	0.25	0.00	1.00
Road segment congestion (a proportion)	8.6m	0.10	0.21	0.00	1.00
Exposure to diesel exhaust					
NOx concentration, weekdays only (ppb)	10,364	64.0	49.9	0.4	490.2
NOx concentration (ppb)	16,152	56.0	46.1	0.0	490.2
Hospital admissions rate					
Cardiovascular (per 100,000 residents per month)	2,365	54.2	19.8	10.7	166.4
Respiratory (per 100,000 residents per month)	2,365	46.5	20.8	5.0	130.0
Trauma (per 100,000 residents per month)	2,365	6.6	3.3	0.0	26.0
Meteorology and thermal inversion					
Ground temperature (°C)	1,673	20.8	3.4	10.5	29.6
Relative humidity (%)	1,669	77.3	10.4	29.6	98.0
Solar radiation (7am to 8pm, Wm^{-2})	1,672	299.2	113.7	25.5	648.3
Atmospheric pressure (hPa)	1,673	932.2	4.8	912.1	945.2
Wind speed (ms^{-1})	1,673	1.4	0.5	0.3	2.9
Wind blows from N-E (a prop., all 24 hours=1, none=0)	6,692	0.15	0.17	0.00	1.00
Wind blows from S-E (a prop., all 24 hours=1, none=0)	6,692	0.44	0.35	0.00	1.00
Wind blows from S-W (a prop., all 24 hours=1, none=0)	6,692	0.08	0.16	0.00	1.00
Wind blows from N-W (a prop., all 24 hours=1, none=0)	6,692	0.18	0.22	0.00	1.00
Precipitation (mm/h)	1,673	0.21	0.49	0.00	4.37
Some precipitation on day (yes=1)	1,673	0.45	0.50	0.00	1.00
Thermal inversion at 9am or 9pm (yes=1)	1,673	0.49	0.50	0.00	1.00
Other control variables					
Gasoline price (BRL$/liter, Oct-2008 CPI)	1,673	2.24	0.09	2.06	2.44
Ethanol price (BRL$/liter, Oct-2008 CPI)	1,673	1.44	0.17	1.06	1.93
Diesel price index (Oct-2008 CPI = 1)	1,673	0.86	0.06	0.78	1.01
Ozone concentration, 24-hour mean ($\mu g/m^3$)	18,803	35.2	17.0	0.1	114.2
Ozone concentration, maximum 8-hour mean ($\mu g/m^3$)	18,803	64.7	32.0	0.4	233.4

Table 2. Reduced-form analysis of traffic volume, diesel pollution, and hospitalizations: Effects of the truck abatement policy on different outcome variables across the São Paulo metropolis. An observation is: **(1)** a day by road segment, **(2)** a day by monitoring site pair, or **(3, 4)** a month by 3-digit residential zip code. The sample period is November 2008 to May 2013. All models control for: location fixed effects (road corridor, NOx monitoring site, or zip code); location by seasonality fixed effects (week-of-year and day-of-week, or month-of-year); meteorology and thermal inversion; linear time trend; and road transportation fuel prices. Policy implemented indicates an observation: **(1, 2)** on or after March 30, 2010, or **(3, 4)** on or after April 2010. OLS regressions. Standard errors, in parentheses, are: **(1, 2)** one-way clustered by week-of-sample, or **(3, 4)** robust. Clustering by month-of-sample in **(1, 2)**, or two-way clustering by week-of-sample and segment in **(1)**, yields slightly higher standard errors, but the same significance levels. Allowing for spatial and serial correlation (cutoffs at 5 km and up to 2 months) yields similar precision. ***, **, * denote significance at the 0.01, 0.05 and 0.1 levels, respectively.

Dependent variable	**(1)** **Road segment congestion** (% afternoon commute hours)	**(2)** **Diesel exhaust marker** (24-hour NOx, ppb)	**(3)** **Cardiovascular** (all ages, per 100,000 per month)	**(4)** **Respiratory** (all ages, per 100,000 per month)
Truck policy implemented (yes=1)	**-0.049*** **	**-9.93*** **	**-5.80*** **	**-3.24** **
	(0.004)	**(2.24)**	**(1.76)**	**(1.67)**
ln(distance) from key original truck route	-0.042***	subsumed in site FEs	subsumed in zip FEs	subsumed in zip FEs
	(0.001)			
Truck policy implemented (yes=1)	**0.018*** **	**8.78*** **	**3.24*** **	**1.61*** **
× ln(distance) from orig. truck route	**(0.002)**	**(0.49)**	**(0.59)**	**(0.58)**
Trend after policy implementation	0.066***	-	-	-
	(0.019)	-	-	-
Trend after policy implementation	-0.013***	-	-	-
× ln(distance) from orig. truck route	(0.002)	-	-	-
Road corridor fixed effects (FEs)	Yes (178)	-	-	-
NOx monitoring site FEs	-	Yes (11)	-	-
3-digit residential zip code FEs	-	-	Yes (43)	Yes (43)
Week-of-year FEs	Yes	-	-	-
Week-of-year by location FEs	-	Yes	-	-
Day-of-week FEs	Yes	-	-	-
Day-of-week by location FEs	-	Yes	-	-
Month-of-year by location FEs	-	-	Yes	Yes
Meteorology and thermal inversion	Yes	Yes	Yes	Yes
Time trend over the entire sample period	Yes	Yes	Yes	Yes
Fuel prices (gasoline, ethanol, diesel)	Yes	Yes	Yes	Yes
May-Sep 2009 swine flu epidemic (yes=1)	-	-	Yes	Yes
Number of observations	8,561,844	16,106	2,365	2,365
Number of regressors	259	699	576	576
Number of locations	5,133 segments	11 sites	43 zips	43 zips
R^2	0.296	0.757	0.902	0.912
Mean value of dependent variable	0.102	55.92	54.21	46.52

Table 3. Fitted road congestion, diesel pollution and health outcomes one year before, upon (April 2010), and two years after policy implementation. Point estimates and standard errors, in parentheses, are based on, or more restrictive versions of, the reduced-form models reported in Table 2. For traffic outcome **(1)**, to the sample mean for road segment congestion on the original truck route, we apply the estimated temporal by spatial variation (respectively, pre and post policy, 50 m to 10 km away), according to estimates in the first 5 rows of column 1, Table 2 and the time trend. For diesel pollution outcome **(2)**, to the sample mean for NOx at one site 20 m from the original truck route, we apply variation estimated in a more restrictive model than in Table 2, without monitor fixed effects, in order to identify the coefficient on (log) distance. For hospital outcomes **(3, 4)**, to the sample mean for hospitalizations among residents of three zip codes closest to the original truck route, we apply variation estimated in a more restrictive model than in Table 2, without zip code fixed effects, in order to identify the coefficient on (log) distance. The closest zip code – according to the population-weighted average centroid of a zip code's districts – is 2 km away.

Outcome	Distance from key original truck route	One year before policy implemented (April 2009)	Upon policy implementation (April 2010)	Two years after policy implemented (April 2012)
(1) Road segment congestion (% afternoon commute hours, 5pm to 8pm)	**50 m away**	**0.482 (0.005)**	**0.376 (0.010)**	**0.429 (0.012)**
	1 km away	0.355 (0.003)	0.302 (0.009)	0.331 (0.012)
	2 km away	0.326 (0.003)	0.284 (0.009)	0.308 (0.012)
	5 km away	0.287 (0.004)	0.262 (0.009)	0.278 (0.012)
	10 km away	**0.258 (0.005)**	**0.244 (0.009)**	**0.256 (0.012)**
(2) Diesel exhaust marker (24-hour ambient NOx levels, ppb)	**50 m away**	**148.1 (3.3)**	**111.0 (4.2)**	**109.4 (4.8)**
	1 km away	125.2 (0.4)	114.4 (2.3)	112.8 (3.3)
	2 km away	119.9 (0.9)	115.2 (2.3)	113.6 (3.3)
	5 km away	112.9 (1.8)	116.3 (2.7)	114.6 (3.5)
	10 km away	**107.6 (2.6)**	**117.1 (3.1)**	**115.4 (3.9)**
(3) Cardiovascular admissions (all ages, per 100,000 residents per month)	50 m away	out of sample	out of sample	out of sample
	1 km away	**49.3 (0.2)**	**45.0 (2.1)**	**44.6 (2.2)**
	2 km away	49.2 (1.4)	47.2 (2.3)	46.7 (2.4)
	5 km away	49.0 (3.2)	50.0 (3.5)	49.5 (3.5)
	10 km away	**48.9 (4.5)**	**52.1 (4.7)**	**51.6 (4.7)**
(4) Respiratory admissions (all ages, per 100,000 residents per month)	50 m away	out of sample	out of sample	out of sample
	1 km away	**41.2 (0.2)**	**38.2 (1.9)**	**38.4 (2.0)**
	2 km away	42.9 (1.2)	41.0 (2.0)	41.2 (2.1)
	5 km away	45.1 (2.7)	44.7 (2.9)	44.9 (3.0)
	10 km away	**46.8 (3.8)**	**47.5 (3.9)**	**47.7 (4.0)**

Table 4. The spatial effects of the truck abatement policy on localized diesel pollution, using ambient NOx as a marker. An observation is a day by NOx monitoring site pair. The dependent variable is the mean NOx concentration (in ppb) over 24 hours (all specifications but **(5)**) or between 7am and 8pm (specification **(5)**). Specifications **(1)** to **(5)** are "difference in difference" models, with the second difference corresponding to sites in the metropolis versus sites 50-100 km away. **(6)** excludes sites 50-100 km away. We allow policy effects to vary by site but in the table show means across sites grouped according to distance from the original truck route (Appendix Table A.4 shows individual effects). The sample period is November 2008 to May 2013. We control for: site fixed effects; seasonality (week-of-year and day-of-week) by site fixed effects, month-of-sample fixed effects; and meteorology and thermal inversion. **(2)** replaces month-of-sample fixed effects by a quadratic time trend and fuel prices. **(3)** allows the effects of meteorology to vary by site. **(4)** restricts the sample to 36 months from January 2009 to December 2011. OLS regressions. Standard errors, in parentheses, are one-way clustered by week-of-sample. ***, **, * denote significance at the 0.01, 0.05 and 0.1 levels, respectively.

Model specification	**(1)**	**(2)**	**(3)**	**(4)**	**(5)**	**(6)**
	Base	**Trend, not year-mo.**	**Site x meteor.**	**36 months**	**Daytime 7am-8pm**	**Metro only**
Dependent variable (ppb)	**24h NOx**	**24h NOx**	**24h NOx**	**24h NOx**	**14h NOx**	**24h NOx**
Policy implemented (yes=1) × Site FEs						
One site 20 m from original truck route	**-59.2*** **	**-59.0*** **	**-59.5*** **	**-51.8*** **	**-74.6*** **	**-56.6*** **
(site 8 in Figure 1)	**(3.1)**	**(3.1)**	**(2.6)**	**(3.7)**	**(3.0)**	**(3.2)**
Sites 2-6 km from original truck route	**-6.0*** **	**-5.9*** **	**-6.7*** **	**-4.7*** **	**-6.7*** **	**-3.2*** **
(mean sites 5, 10, 27, 31 in Fig. 1)	**(1.3)**	**(1.3)**	**(1.1)**	**(1.5)**	**(1.1)**	**(1.1)**
Sites 6-10 km from original truck route	-2.0	-2.1*	-2.3**	0.7	-3.4***	0.6
(mean sites 1, 7, 20 in Fig. 1)	(1.2)	(1.2)	(1.1)	(1.5)	(1.0)	(0.9)
Sites 10-20 km from original truck route	-2.7**	-2.9**	-2.9***	0.3	-4.0***	-
(mean sites 17, 22, 29 in Fig. 1)	(1.2)	(1.2)	(1.0)	(1.4)	(1.3)	-
Policy implemented (yes=1)	6.1***	5.3***	7.4***	4.5**	6.8***	4.9*
	(2.1)	(1.5)	(2.2)	(2.3)	(1.9)	(2.8)
Site fixed effects (FEs)	Yes	Yes	Yes	Yes	Yes	Yes
Week-of-year, day-of-week by site FEs	Yes	Yes	Yes	Yes	Yes	Yes
Meteorology and thermal inversion	Yes	Yes	Yes	Yes	Yes	Yes
Month-of-sample FEs	Yes	-	Yes	Yes	Yes	Yes
Quadratic time trend	-	Yes	-	-	-	-
Fuel prices (gasoline, ethanol, diesel)	-	Yes	-	-	-	-
Number of observations	22,168	22,168	22,168	14,674	22,244	16,106
Number of regressors	1,007	958	1,175	986	1,007	756
R^2	0.791	0.786	0.847	0.803	0.805	0.767
Mean value of dep. var. (for site 8)	110.3	110.3	110.3	121.1	113.7	110.3

Table 5. Effects of diesel pollution on hospital admission and death rates (structural analysis of health). An observation is a 3-digit residential zip code by month pair. The dependent variable is the hospitalization rate for a given disease-age group pair (except pairs with very low incidence, e.g., cardiovascular among children aged 0-4 years). For each of 14 disease-age pairs, we estimate health equation [2] separately by 2SLS regression and by OLS regression; we thus report mean β_{1l}, averaged across all zip codes l, for each of 14×2=28 regressions. 2SLS estimates use fitted NOx per the specification reported in Table 4, column 1 as an instrument for measured NOx. We assign each district within a zip code (there is a ratio of 103 districts to 43 zip codes) to the nearest NOx monitor from the district centroid; we then average NOx across districts within each zip code. Across the multiple first-stage equations in the 2SLS implementation, the first-stage F-statistic for the excluded instruments averages 93 (range 6 to 258; endogenous regressors are measured NOx interacted with zip code fixed effects and excluded instruments are fitted NOx interacted with zip code fixed effects). The sample period is November 2008 to May 2013. We control for: zip code fixed effects; seasonality (month-of-year) by zip code fixed effects; and month-of-sample fixed effects (these subsume meteorology and the May to September 2009 swine flu epidemic). Robust standard errors are reported in parentheses. ***, **, * denote significance at the 0.01, 0.05 and 0.1 levels, respectively.

Dependent variable: Hospitalization rate per 100,000 residents per month	**All ages**	**0-4 years**	**5-19 years**	**20-64 years**	**65+ years**
Cardiovascular admissions					
Diesel exhaust (in 10 ppb NOx, mean over zip)	**4.2*** (0.7)**	**-**	**-**	**2.7*** (0.7)**	**20.8*** (5.9)**
… compare 2SLS estimate to OLS estimate:	0.6* (0.4)	-	-	0.3 (0.4)	3.1 (3.0)
Mean of dependent variable	53.1	-	-	42.9	275.6
Respiratory admissions					
Diesel exhaust (in 10 ppb NOx, mean over zip)	**3.2*** (0.7)**	**22.9*** (6.4)**	**3.7*** (1.1)**	**0.9* (0.5)**	**11.5*** (3.9)**
… compare 2SLS estimate to OLS estimate:	1.0*** (0.4)	9.5*** (3.4)	0.6 (0.6)	0.5** (0.2)	0.7 (2.0)
Mean of dependent variable	43.2	251.5	29.9	18.3	120.5
Cardiovascular/respiratory death					
Diesel exhaust (in 10 ppb NOx, mean over zip)	**1.0*** (0.3)**	**-**	**-**	**0.5* (0.3)**	**6.7** (3.0)**
… compare 2SLS estimate to OLS estimate:	0.2 (0.2)	-	-	0.0 (0.1)	1.2 (1.7)
Mean of dependent variable	10.2	-	-	5.4	74.9
Placebo: Trauma admissions (excl. traffic)					
Diesel exhaust (in 10 ppb NOx, mean over zip)	0.1 (0.2)	-	-	0.3 (0.3)	0.8 (1.1)
… compare 2SLS estimate to OLS estimate:	0.0 (0.1)	-	-	0.1 (0.1)	0.5 (0.5)
Mean of dependent variable	6.9	-	-	6.7	14.0
Number of observations	1,343	1,343	1,343	1,343	1,343
Number of zip codes (w/ nearby NOx monitor)	25	25	25	25	25

Table 6. Effects of diesel pollution on hospital admission and death rates: Robustness tests of the structural analysis. The first row within each disease category/death reproduces estimates for our preferred specification (Table 5). Each row presents a different robustness test (with separate 2SLS regressions by age group in the columns). As in Table 5, we report β_{1l}, averaged across all zip codes l, for a 10 ppb increase in NOx equivalent units of diesel pollution. See Table 5 notes. IV denotes instrumental variable. Robust standard errors are reported in parentheses. ***, **, * denote significance at the 0.01, 0.05 and 0.1 levels, respectively.

Dependent variable: Hospitalization rate per 100,000 residents per month	Number of observations	Number of zip codes	All ages	0-4 years	5-19 years	20-64 years	65+ years
Cardiovascular admissions							
Preferred specification (Table 5, 2SLS)	1,343	25	4.2*** (0.7)	-	-	2.7*** (0.7)	20.8*** (5.9)
Closest monitor within 10 km	1,830	34	4.3*** (0.8)	-	-	3.1*** (0.8)	21.6*** (6.2)
Mean of monitors within 5 km	1,343	25	3.7*** (0.7)	-	-	2.4*** (0.7)	18.5*** (5.4)
Mean of monitors within 10 km	1,839	34	3.9*** (0.7)	-	-	3.2*** (0.7)	16.9*** (5.6)
Site-specific meteorology effects (IV)	1,343	25	2.7*** (0.6)	-	-	1.7*** (0.7)	13.4** (5.4)
Include policy-wind interactions (IV)	1,343	25	4.2*** (0.8)	-	-	2.7*** (0.7)	21.1*** (6.0)
Incl. site-specific policy-wind interact. (IV)	1,343	25	2.8*** (0.6)	-	-	1.7** (0.7)	15.2*** (5.3)
Drop control sites 50-100 km away (IV)	1,343	25	4.8*** (1.0)	-	-	3.3*** (1.0)	17.9** (8.1)
Policy implemented May 31, 2010 (IV)	1,343	25	4.2*** (0.7)	-	-	3.1*** (0.7)	19.0*** (5.7)
Shorter period of 36 months	879	25	2.1** (0.9)	-	-	1.0 (0.9)	11.9* (7.0)
Local road congestion as health control	1,343	25	4.1*** (0.7)	-	-	2.8*** (0.7)	21.4*** (5.6)
Repeat w/ IV based on policy & congestion	1,343	25	3.7*** (0.7)	-	-	2.5*** (0.7)	18.8*** (5.2)
Trauma admissions as health control	1,343	25	4.1*** (0.8)	-	-	2.5*** (0.7)	21.0*** (5.9)
All other admissions as health control	1,343	25	2.7*** (0.7)	-	-	1.7** (0.7)	17.8*** (6.0)
Distance from patient to hospital as control	1,343	25	4.0*** (0.7)	-	-	2.5*** (0.7)	20.5*** (5.9)
Dist. from patient to hospital * zip code FE	1,343	25	3.6*** (0.8)	-	-	2.6*** (0.8)	18.1*** (5.8)
Quadratic trend	1,343	25	3.8*** (0.8)	-	-	2.6*** (0.8)	15.5*** (5.7)
Quadratic trend plus fuel prices	1,343	25	4.6*** (0.8)	-	-	3.0*** (0.8)	23.7*** (5.7)
Weighted 2SLS regression (sq.rt.pop.)	1,343	25	3.3*** (0.7)	-	-	2.0*** (0.7)	16.4*** (5.5)
Population-weighted average for β_{1l}	1,343	25	3.3*** (0.8)	-	-	1.8** (0.8)	19.3*** (5.1)
Respiratory admissions							
Preferred specification (Table 5, 2SLS)	1,343	25	3.2*** (0.7)	22.9*** (6.4)	3.7*** (1.1)	0.9* (0.5)	11.5*** (3.9)
Closest monitor within 10 km	1,830	34	3.0*** (0.7)	22.8*** (6.7)	3.3*** (1.0)	0.8* (0.5)	14.7*** (3.9)
Mean of monitors within 5 km	1,343	25	2.4*** (0.6)	19.7*** (5.8)	3.3*** (1.0)	0.5 (0.4)	8.6** (3.6)
Mean of monitors within 10 km	1,839	34	1.4** (0.7)	9.9* (5.9)	1.4 (0.9)	0.6 (0.4)	11.6*** (3.6)
Site-specific meteorology effects (IV)	1,343	25	2.5*** (0.6)	25.2*** (5.8)	1.6* (1.0)	0.7* (0.4)	3.1 (3.6)

Dependent variable: Hospitalization rate per 100,000 residents per month	Number of observations	Number of zip codes	All ages	0-4 years	5-19 years	20-64 years	65+ years
Include policy-wind interactions (IV)	1,343	25	3.2*** (0.7)	23.7*** (6.5)	3.9*** (1.1)	0.9* (0.5)	11.8*** (3.9)
Incl. site-specific policy-wind interact. (IV)	1,343	25	2.6*** (0.6)	23.3*** (5.6)	1.6* (1.0)	0.8** (0.4)	4.1 (3.6)
Drop control sites 50-100 km away (IV)	1,343	25	4.2*** (1.0)	28.3*** (9.3)	6.3*** (1.5)	1.3** (0.6)	11.2** (4.8)
Policy implemented May 31, 2010 (IV)	1,343	25	3.3*** (0.7)	27.1*** (6.8)	4.0*** (1.0)	0.9** (0.5)	12.1*** (3.8)
Shorter period of 36 months	879	25	2.4*** (0.8)	24.0*** (8.1)	0.8 (1.2)	0.3 (0.5)	8.7* (4.9)
Local road congestion as health control	1,343	25	3.4*** (0.7)	24.2*** (6.2)	3.6*** (1.0)	1.1** (0.5)	12.6*** (3.7)
Repeat w/ IV based on policy & congestion	1,343	25	3.3*** (0.6)	23.3*** (5.6)	3.3*** (1.0)	1.2*** (0.4)	11.3*** (3.4)
Trauma admissions as health control	1,343	25	3.0*** (0.7)	22.6*** (6.5)	3.8*** (1.1)	0.8* (0.5)	11.5*** (3.9)
All other admissions as health control	1,343	25	1.9*** (0.7)	13.7** (6.3)	2.0* (1.1)	0.5 (0.5)	10.7*** (3.9)
Distance from patient to hospital as control	1,343	25	2.9*** (0.7)	22.4*** (6.3)	3.5*** (1.1)	0.8* (0.5)	10.8*** (3.7)
Dist. from patient to hospital * zip code FE	1,343	25	2.7*** (0.7)	20.9*** (6.1)	3.0*** (1.1)	0.8 (0.5)	11.4*** (3.8)
Quadratic trend	1,343	25	2.9*** (0.8)	24.2*** (7.3)	1.7 (1.1)	0.8 (0.5)	12.4*** (4.1)
Quadratic trend plus fuel prices	1,343	25	3.8*** (0.7)	30.9*** (6.9)	3.5*** (1.1)	1.1** (0.5)	11.7*** (3.7)
Weighted 2SLS regression (sq.rt.pop.)	1,343	25	2.5*** (0.6)	18.5*** (5.8)	2.5*** (0.9)	0.7* (0.4)	10.1*** (3.6)
Population-weighted average for β_{1l}	1,343	25	2.9*** (0.7)	20.6*** (7.3)	3.4*** (1.2)	1.0** (0.5)	9.9*** (3.4)
Cardiovascular/respiratory death							
Preferred specification (Table 5, 2SLS)	1,343	25	1.0*** (0.3)	-	-	0.5* (0.3)	6.7** (3.0)
Closest monitor within 10 km	1,830	34	1.3*** (0.3)	-	-	0.8*** (0.3)	8.5*** (2.9)
Mean of monitors within 5 km	1,343	25	0.8*** (0.3)	-	-	0.4* (0.3)	5.4* (2.8)
Mean of monitors within 10 km	1,839	34	0.7*** (0.2)	-	-	0.3 (0.2)	5.4** (2.6)
Site-specific meteorology effects (IV)	1,343	25	0.7*** (0.3)	-	-	0.4* (0.2)	3.6 (2.9)
Include policy-wind interactions (IV)	1,343	25	1.0*** (0.3)	-	-	0.5* (0.3)	7.0** (3.1)
Incl. site-specific policy-wind interact. (IV)	1,343	25	0.8*** (0.3)	-	-	0.4 (0.2)	4.6 (2.8)
Drop control sites 50-100 km away (IV)	1,343	25	1.1** (0.4)	-	-	0.6 (0.4)	7.4* (3.9)
Policy implemented May 31, 2010 (IV)	1,343	25	0.7** (0.3)	-	-	0.3 (0.3)	5.1* (2.9)
Shorter period of 36 months	879	25	0.8** (0.4)	-	-	0.5 (0.3)	6.0* (3.5)
Local road congestion as health control	1,343	25	1.0*** (0.3)	-	-	0.4* (0.3)	8.2*** (2.9)
Repeat w/ IV based on policy & congestion	1,343	25	0.9*** (0.3)	-	-	0.4 (0.2)	7.4*** (2.7)
Trauma admissions as health control	1,343	25	0.9*** (0.3)	-	-	0.5* (0.3)	6.6** (3.0)
All other admissions as health control	1,343	25	0.7** (0.3)	-	-	0.4 (0.3)	6.0** (3.0)
Distance from patient to hospital as control	1,343	25	0.9*** (0.3)	-	-	0.5* (0.3)	6.4** (3.0)
Dist. from patient to hospital * zip code FE	1,343	25	0.8** (0.3)	-	-	0.4 (0.3)	5.0 (3.1)
Quadratic trend	1,343	25	1.0*** (0.3)	-	-	0.6** (0.3)	6.6** (3.1)
Quadratic trend plus fuel prices	1,343	25	1.1*** (0.3)	-	-	0.7*** (0.3)	7.6** (3.0)
Weighted 2SLS regression (sq.rt.pop.)	1,343	25	0.6** (0.3)	-	-	0.2 (0.2)	3.9 (2.7)
Population-weighted average for β_{1l}	1,343	25	0.8** (0.3)	-	-	0.4 (0.3)	6.3** (2.7)

Dependent variable: Hospitalization rate per 100,000 residents per month	**Number of observations**	**Number of zip codes**	**All ages**	**0-4 years**	**5-19 years**	**20-64 years**	**65+ years**
Placebo: Trauma admissions (excl. traffic)							
Preferred specification (Table 5, 2SLS)	1,343	25	0.1 (0.2)	-	-	0.3 (0.3)	0.8 (1.1)
Closest monitor within 10 km	1,830	34	-0.1 (0.2)	-	-	0.1 (0.3)	0.2 (1.1)
Mean of monitors within 5 km	1,343	25	0.0 (0.2)	-	-	0.2 (0.3)	1.1 (1.0)
Mean of monitors within 10 km	1,839	34	-0.2 (0.2)	-	-	0.0 (0.2)	-0.5 (0.9)
Site-specific meteorology effects (IV)	1,343	25	-0.2 (0.2)	-	-	0.2 (0.3)	-0.5 (1.0)
Include policy-wind interactions (IV)	1,343	25	0.1 (0.2)	-	-	0.3 (0.3)	0.9 (1.1)
Incl. site-specific policy-wind interact. (IV)	1,343	25	-0.1 (0.2)	-	-	0.3 (0.3)	-0.7 (1.0)
Drop control sites 50-100 km away (IV)	1,343	25	0.2 (0.3)	-	-	0.2 (0.4)	1.1 (1.4)
Policy implemented May 31, 2010 (IV)	1,343	25	0.2 (0.2)	-	-	0.3 (0.3)	0.9 (1.1)
Shorter period of 36 months	879	25	0.0 (0.3)	-	-	-0.1 (0.4)	1.6 (1.3)
Local road congestion as health control	1,343	25	0.1 (0.2)	-	-	0.2 (0.3)	0.6 (1.0)
Repeat w/ IV based on policy & congestion	1,343	25	0.0 (0.2)	-	-	0.1 (0.3)	0.5 (1.0)
Distance from patient to hospital as control	1,343	25	0.1 (0.2)	-	-	0.2 (0.3)	0.9 (1.1)
Dist. from patient to hospital * zip code FE	1,343	25	-0.2 (0.3)	-	-	0.1 (0.3)	0.7 (1.1)
Quadratic trend	1,343	25	0.4 (0.2)	-	-	0.5* (0.3)	1.0 (1.1)
Quadratic trend plus fuel prices	1,343	25	0.2 (0.2)	-	-	0.3 (0.3)	1.4 (1.1)
Weighted 2SLS regression (sq.rt.pop.)	1,343	25	0.2 (0.2)	-	-	0.3 (0.3)	0.9 (0.9)
Population-weighted average for β_{1l}	1,343	25	0.1 (0.2)	-	-	0.4 (0.3)	0.6 (1.0)

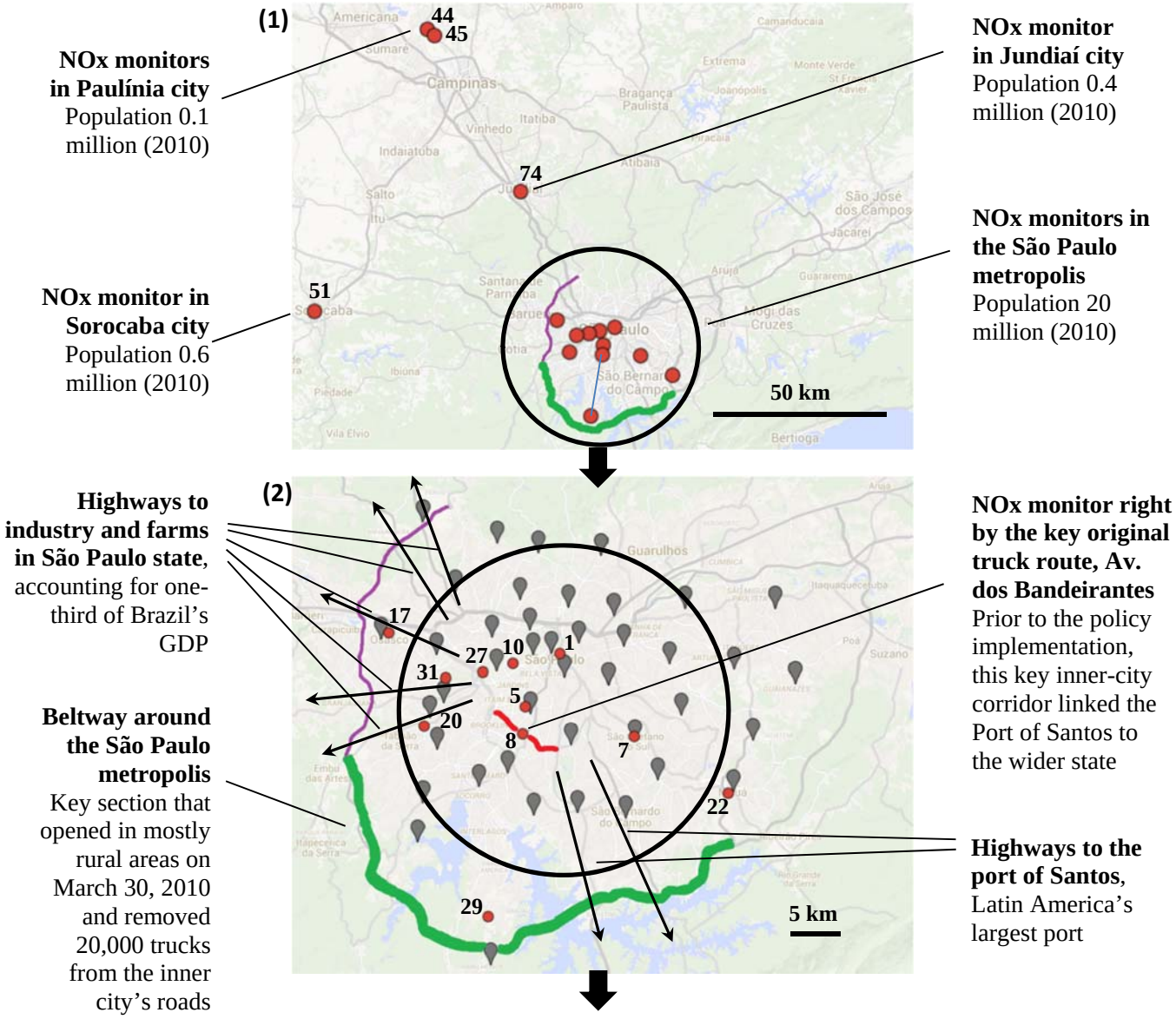
Table 7. The effects of the (spatially differentiated) truck abatement policy and the (spatially invariant) light-vehicle gasoline-ethanol fuel mix on localized ozone pollution. An observation is a day by ozone monitoring site pair. The dependent variable is the daily ozone concentration (in μg/m^3) averaged over 24 hours (specifications **(1)** to **(3)**) or the maximum 8-hour average (specifications **(4)** to **(6)**). Specifications **(1, 2)** and **(4, 5)** are "difference in difference" models, with the second difference corresponding to sites in the metropolis, potentially impacted by the truck policy, versus sites 50-100 km away. **(3)** and **(6)** exclude sites 50-100 km away. We allow the effects of both the truck policy and the light-vehicle gasoline-ethanol mix to vary by site but the table show means across sites grouped according to distance from the original truck route (site-specific coefficients on truck policy) or over all sites (site-specific coefficients on per-liter price ratio for ethanol to gasoline). The sample period is November 2008 to May 2013. We control for: site fixed effects; seasonality (week-of-year and day-of-week) by site fixed effects; meteorology and thermal inversion; and diesel prices. **(1)** and **(4)** include year fixed effects, whereas all other specifications include a quadratic time trend. OLS regressions. Standard errors, in parentheses, are one-way clustered by week-of-sample. ***, **, * denote significance at the 0.01, 0.05 and 0.1 levels, respectively.

Dependent variable (μg/m^3)	**Ozone, daily 24-hour mean**			**Ozone, daily maximum 8-hour mean**		
	(1)	**(2)**	**(3)**	**(4)**	**(5)**	**(6)**
Model specification	**Year FE**	**Quadratic trend**	**Metro only**	**Year FE**	**Quadratic trend**	**Metro only**
Truck policy in metro (yes=1) × Site FEs						
Sites 2-6 km from original truck route	1.6**	1.5**	3.2***	4.3***	4.2***	4.5***
(mean three sites: 5, 27, 31)	(0.7)	(0.7)	(0.8)	(1.4)	(1.4)	(1.2)
Sites 6-10 km from original truck route	-0.0	-0.1	1.7***	2.0	1.9	2.2**
(mean four sites: 15, 7, 1, 3)	(0.8)	(0.8)	(0.6)	(1.5)	(1.5)	(1.0)
Sites 10-20 km from original truck route	-1.5**	-1.7**	-	-0.0	-0.1	-
(mean five sites: 2, 6, 18, 29, 22)	(0.7)	(0.7)	-	(1.3)	(1.3)	-
Policy implemented (yes=1)	2.9*	2.9*	0.6	5.1*	2.6	3.1
	(1.8)	(1.8)	(1.6)	(2.7)	(2.4)	(2.6)
Ethanol-to-gasoline price ratio × Site FEs	-27.2***	-29.5***	-30.9***	-35.8**	-46.2***	-50.0***
(mean 12 sites in metropolis, 4 outside)	(8.7)	(7.6)	(7.7)	(14.2)	(12.3)	(13.0)
Site fixed effects (FEs)	Yes	Yes	Yes	Yes	Yes	Yes
Week-of-year, day-of-week by site FEs	Yes	Yes	Yes	Yes	Yes	Yes
Meteorology and thermal inversion	Yes	Yes	Yes	Yes	Yes	Yes
Diesel price (ethanol-gasoline ratio above)	Yes	Yes	Yes	Yes	Yes	Yes
Year FEs	Yes	-	-	Yes	-	-
Quadratic time trend	-	Yes	Yes	-	Yes	Yes
Number of observations	25,183	25,183	18,753	25,183	25,183	18,753
Number of regressors	1,038	1,035	778	1,038	1,035	778
R^2	0.663	0.660	0.650	0.700	0.699	0.703
Mean value of dep. var.	36.4	36.4	35.2	67.0	67.0	64.8

Table 8. Effects of diesel pollution on hospital admission and death rates: Multi-pollutant model with ozone. An observation is a 3-digit residential zip code by month pair. The first row within each disease category/death reproduces estimates for our preferred single-pollutant specification, using NOx as a marker for diesel exhaust (Table 5). The following rows each represent a different 2SLS regression, where the dependent variable is the all-age hospitalization rate for the given disease category/death, and where ozone exposure is proxied by either the daily 24-hour or maximum 8-hour concentration, as indicated. We instrument for NOx and ozone using fitted NOx and fitted ozone per the specifications reported, respectively, in Table 4(2) and Table 7 (column as indicated), and plotted in Appendix Figure A.2. Similar to Table 5, we report β_{1l}^{NOx} and β_{1l}^{ozone}, the coefficients on $diesel_{lt}$ and $ozone_{lt}$, respectively, averaged across all zip codes l, for increases of 10 ppb in (24-hour) NOx-equivalent units of diesel pollution and 10 µg/m^3 in (24-hour or maximum 8-hour) ozone. We assign each district within a zip code to the nearest NOx monitor and nearest ozone monitor from the district centroid, not to exceed 5 km (or, in the last row, 10 km); we then average NOx and ozone across districts within each zip code. The first-stage F-statistic for the excluded instruments averages 29. The sample period is November 2008 to May 2013. We control for: zip code fixed effects; seasonality (month-of-year) by zip code fixed effects; a quadratic time trend; and meteorology and thermal inversion. Robust standard errors are reported in parentheses. ***, **, * denote significance at the 0.01, 0.05 and 0.1 levels, respectively.

Dependent variable: All-age hospitalization rate per 100,000 residents per month	Fitted pollut. as IV, from	Number of obs.	No. of zip codes	NOx (+10 ppb)	Ozone (+10 µg/m^3)
Cardiovascular admissions					
Preferred single-pollutant model (Table 5)	Table 4(1)	1,343	25	4.2*** (0.7)	-
Include 24-hour ozone	Tab.4(2),7(2)	1,126	21	6.3*** (2.0)	-2.8 (2.1)
Include maximum 8-hour ozone	Tab.4(2),7(5)	1,126	21	6.7*** (1.8)	-1.3 (1.0)
Include max. 8-hour ozone (within 10 km)	Tab.4(2),7(5)	1,771	33	2.8** (1.1)	-1.1 (0.7)
Respiratory admissions					
Preferred single-pollutant model (Table 5)	Table 4(1)	1,343	25	3.2*** (0.7)	-
Include 24-hour ozone	Tab.4(2),7(2)	1,126	21	4.9** (2.0)	-1.7 (1.9)
Include maximum 8-hour ozone	Tab.4(2),7(5)	1,126	21	5.1*** (1.8)	-0.3 (0.9)
Include max. 8-hour ozone (within 10 km)	Tab.4(2),7(5)	1,771	33	3.3*** (1.2)	-0.6 (0.7)
Cardiovascular/respiratory death					
Preferred single-pollutant model (Table 5)	Table 4(1)	1,343	25	1.0*** (0.3)	-
Include 24-hour ozone	Tab.4(2),7(2)	1,126	21	1.9** (0.8)	-0.6 (0.8)
Include maximum 8-hour ozone	Tab.4(2),7(5)	1,126	21	2.0*** (0.7)	-0.4 (0.4)
Include max. 8-hour ozone (within 10 km)	Tab.4(2),7(5)	1,771	33	1.1** (0.4)	-0.2 (0.3)
Placebo: Trauma admissions (excl. traffic)					
Preferred single-pollutant model (Table 5)	Table 4(1)	1,343	25	0.1 (0.2)	-
Include 24-hour ozone	Tab.4(2),7(2)	1,126	21	0.8 (0.6)	-0.5 (0.7)
Include maximum 8-hour ozone	Tab.4(2),7(5)	1,126	21	0.9 (0.6)	-0.2 (0.3)
Include max. 8-hour ozone (within 10 km)	Tab.4(2),7(5)	1,771	33	0.6 (0.4)	-0.2 (0.2)

Figure 1. The beltway around the São Paulo metropolis that removed trucks from urban roads, and locations in the combined datasets. (1, 2) Southeast of São Paulo state and the São Paulo metropolis, respectively. Circles (filled in red, labeled according to the environmental authority's identifier, *N*=15) indicate 15 NOx monitoring sites. Balloons (filled in gray, *N*=43) indicate the 3-digit zip codes (district centroids averaged by population) contained in the residential addresses of patients admitted to hospital. The beltway (new route) and Avenida dos Bandeirantes (key link in the original route) are shown. Arrows indicate 7 (out of ten) major highways connecting farms and industry in the state, via the metropolis, to the port of Santos. **(3)** Road segments monitored for traffic congestion in the city of São Paulo. Circles (*N*=5,133) indicate the midpoints of monitored road segments. Sources: CET, CETESB, DATASUS.



(3)

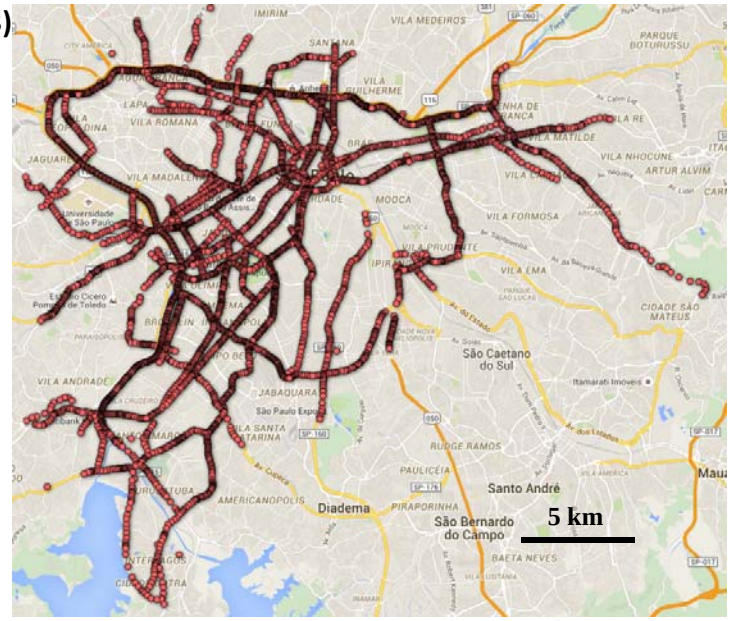

5 km
Santo André
São Bernardo do Campo
Diadema
São Caetano do Sul
MOOCA
VILA FORMOSA
VILA EMA
CIDADE SÃO MATEUS
VILA MADALENA
VILA ROMANA
VILA MATILDE
VILA NHOCUNE
ARTUR ALVIM
JABAQUARA
AMERICANOPOLIS
RUDGE RAMOS
PAULICÉIA
PIRAPORINHA
BAETA NEVES
VILA ANDRADE
VILA MEDEIROS
PARQUE BOTURUSSU
IMIRIM
JAGUARÉ

Figure 2. NOx as a marker for diesel exhaust and diesel particulate matter, including ultrafines and carbonaceous. (1) Daily (24-hour) mean PM2.5 (μg/m^3) against daily mean NOx (ppb) concentrations at the three sites in the São Paulo metropolis that concurrently monitored PM2.5 and NOx after policy implementation (PM2.5 starting in: January 2011 for site 8 in Figure 1, August 2011 for site 31, January 2012 for site 27; through the end of the sample period in May 2013). **(2)** Daily mean submicron 7-800 nm ("PM 0.007-0.8") number concentration (cm^{-3}), **(3)** daily mean ultrafine 7-100 nm ("PM 0.007-0.1") number concentration (cm^{-3}), or **(4)** daily mean black carbon mass concentration (μg/m^3), against NOx at the University of São Paulo (at or very close to site 31 in Figure 1) after policy implementation (October 2010 to September 2011 for submicron and ultrafines, or to July 2012 for black carbon). An observation is a site by day in (**1**) or a day in (**2, 3, 4**). The scatters show de-seasoned concentrations, i.e., residuals fitted after separately regressing each pollutant concentration on a set of month-of-year dummies. Scatters plotting raw concentrations show similar (and even tighter) correlation. Source: CETESB, field campaigns in Salvo et al. (2017).

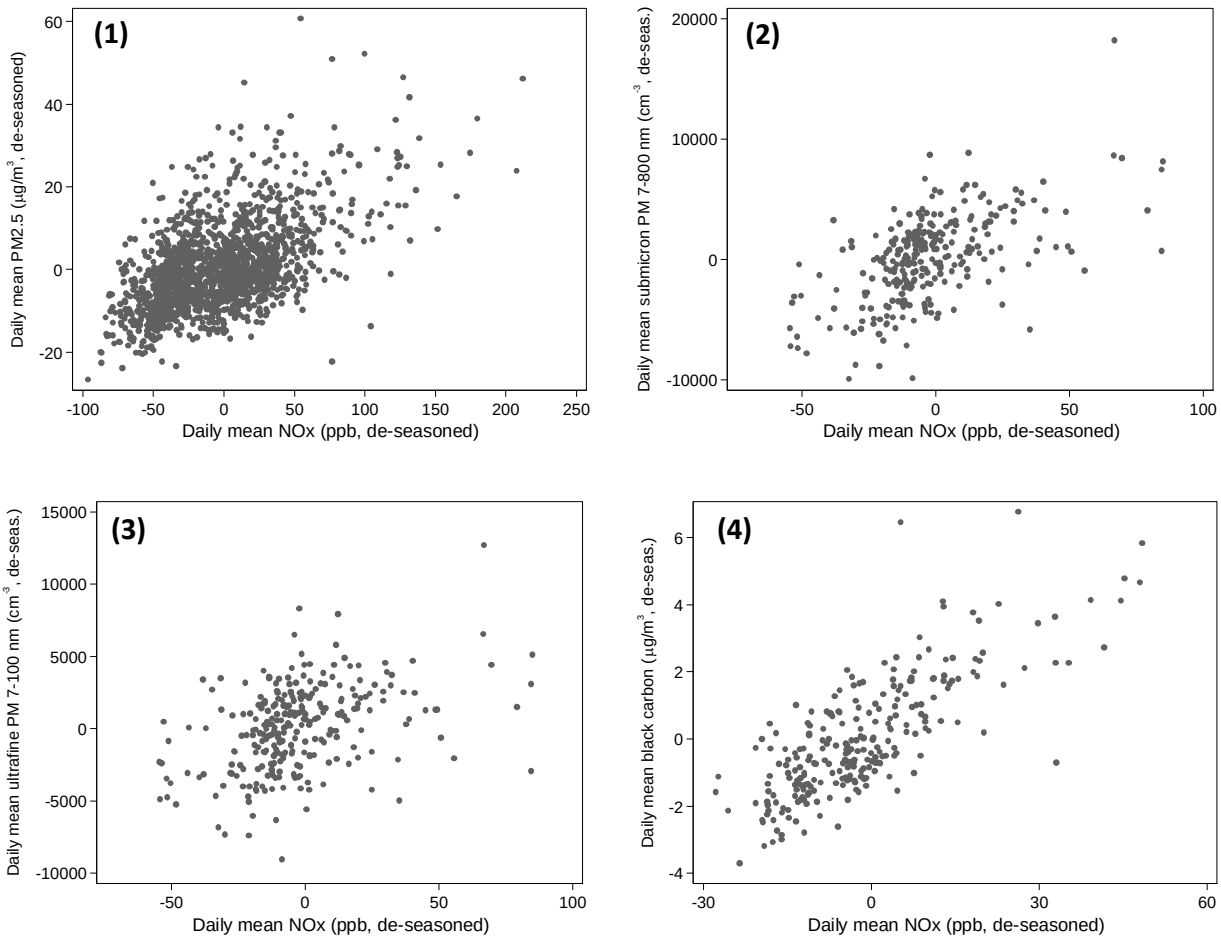


Figure 3. Levels of traffic congestion in the city of São Paulo systematically fall in the distance from the key inner-city corridor, Avenida dos Bandeirantes. Each observation in the scatterplot represents a bin, of length 100 m, at increasing radial distance from the original truck route, i.e., 0-100 m away, 100-200 m away, etc. Plotted is the mean proportion of the weekday afternoon commute (5pm to 8pm) that all road segments in a given distance bin exhibit congestion (per traffic authority definition, as in Table 1). We average segment-level proportions over afternoon commute hours within each weekday, aggregate in space weighting by segment length (within each bin), then average over all weekdays in the November 1, 2008 to May 31, 2013 sample. Source: CET.

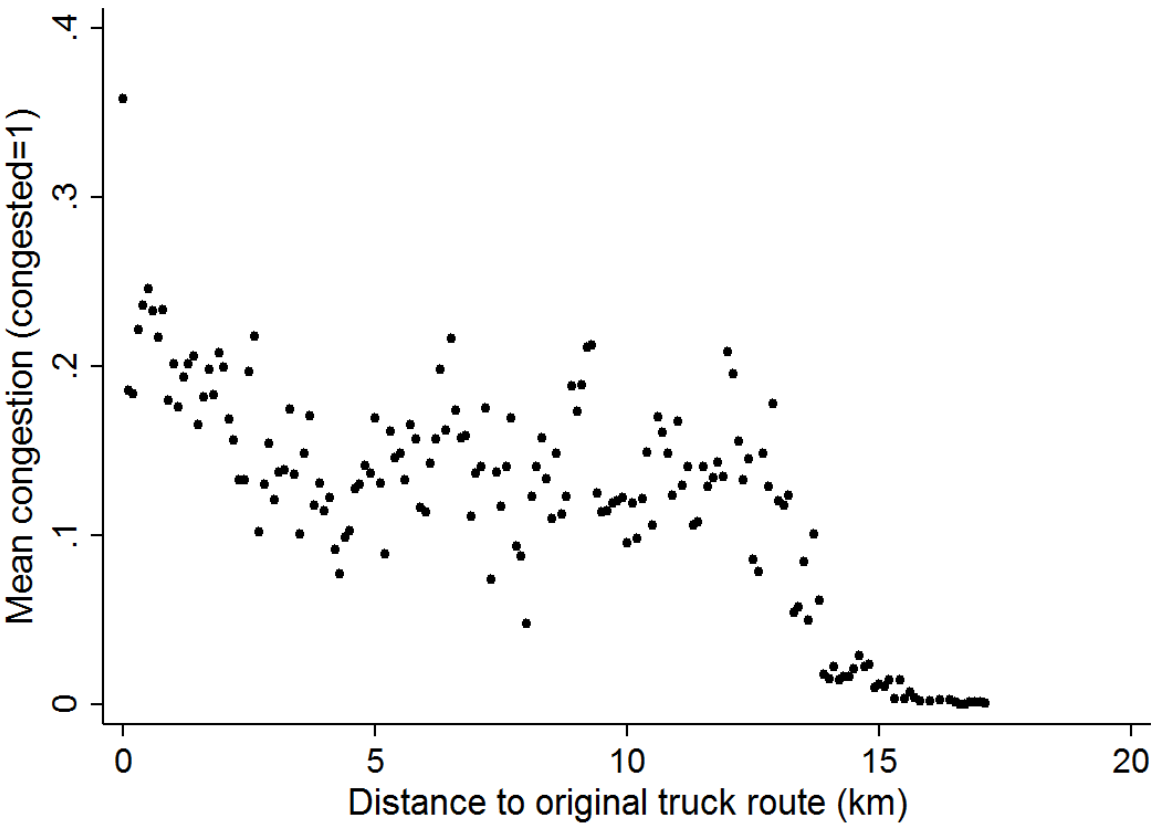


Figure 4. Road congestion, diesel pollution, and hospitalizations near and far and over time. **(1)** Proportions of the city's road segments that are congested: along Av. dos Bandeirantes (black, solid); and between 10 and 20 km from this key inner-city corridor (red, dotted). **(2)** Ambient NOx concentrations: at a site only 20 m from Av. dos Bandeirantes (black solid); and at four sites 50-100 km away from the São Paulo metropolis (red, dotted). **(3)** Cardiovascular admission rates among residents: at the 10 zip codes (25%) that are closer to the Av. dos Bandeirantes (black, solid, left axis); at the 10 zip codes that are furthest from this key inner-city corridor (red, dotted, right axis); and the difference (blue, thick, right axis). In **(1)**, we average segment-level proportions over afternoon commute hours within each weekday, aggregate in space weighting by segment length, deseason (regress on week-of-year and day-of-week), and plot mean residuals by month. In **(2)**, we average monitor-level concentrations over 24 hours within each weekday, deseason, and plot mean residuals by month (and across sites outside the metropolis). In **(3)**, we sum admissions over all days within each quarter and across residents of each group of 10 zip codes, and divide by the resident population. The vertical line indicates the opening of the beltway.

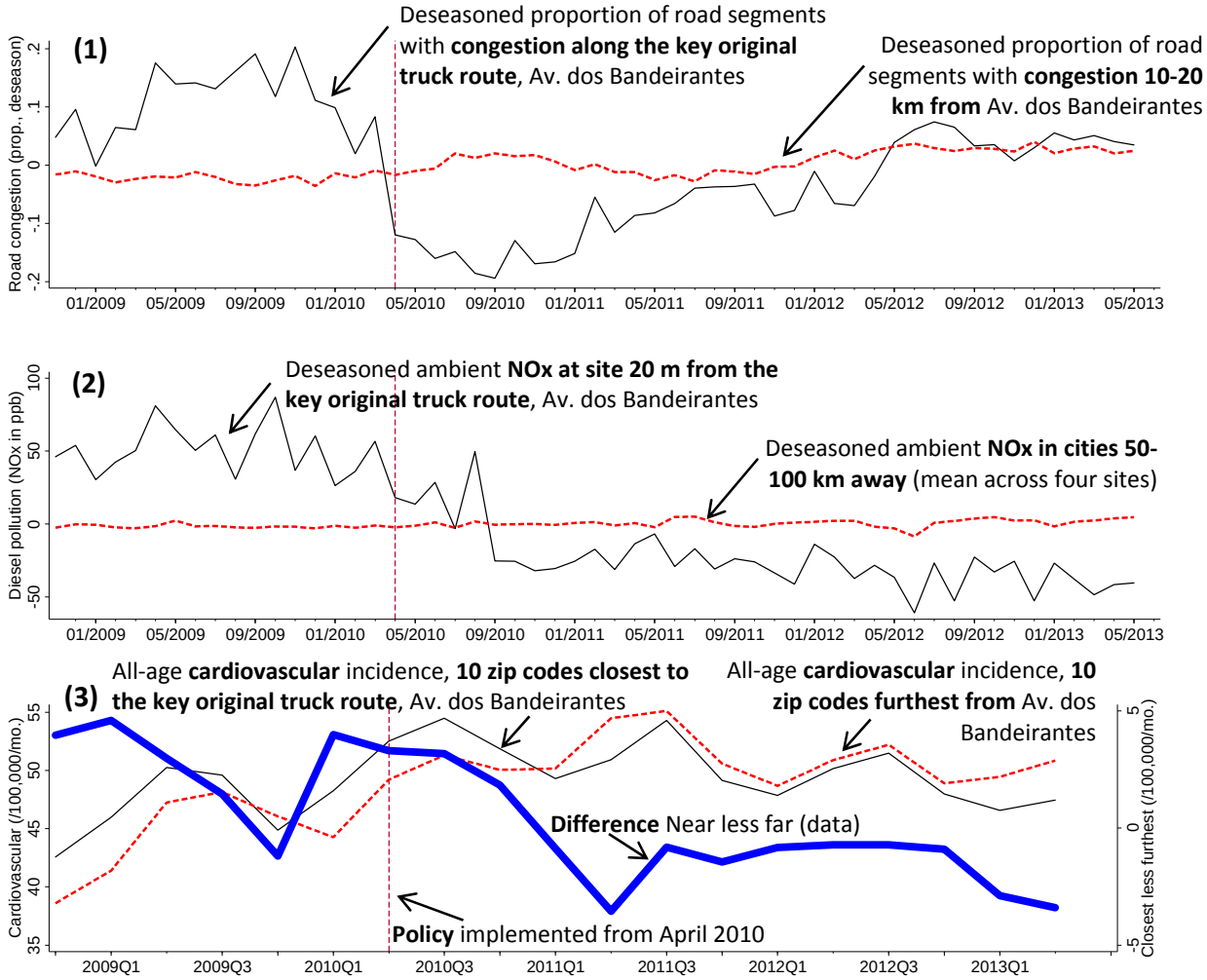
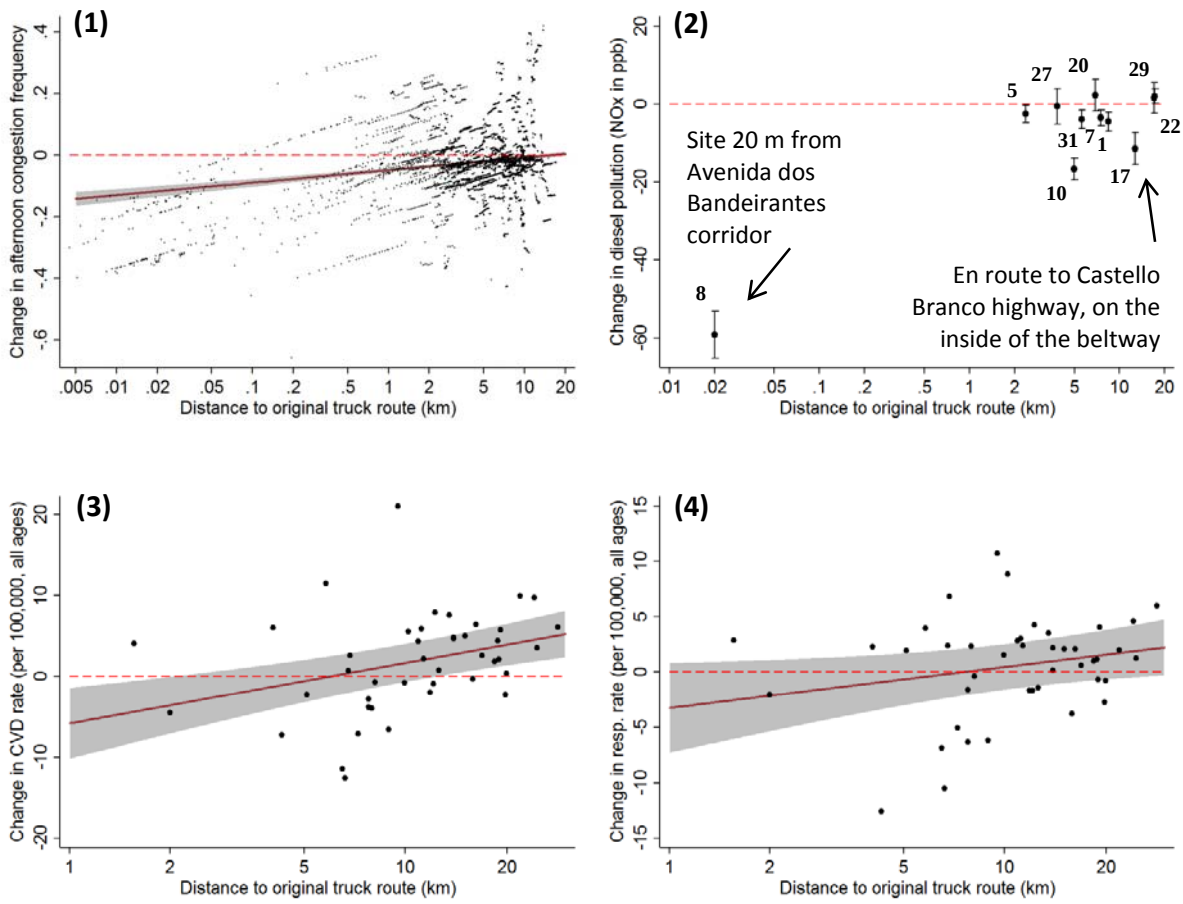


Figure 5. Estimated localized policy effects on road congestion, diesel pollution and cardiovascular and respiratory hospitalizations. An observation in each scatterplot marks a specific location in the São Paulo metropolis, at a distance from the original truck route: **(1)** 5,133 road segments, **(2)** 11 NOx monitoring sites, or **(3, 4)** 43 3-digit residential zip codes. Scatters in **(1)** and **(3, 4)** are estimates based on reduced-form models as in Table 2, except that we interact the policy implementation indicator with a full set of location fixed effects (*N*=5,133 or 43). This is more flexible than interacting policy implementation with (log) distance from the original truck route, as Table 2 reports for ease of exposition. Moreover, the fitted line and 95% confidence intervals overlaid on these three scatters are obtained from the more restrictive models used in Table 3, in which policy implementation is interacted with (log) distance and detailed location fixed effects are excluded in order to identify the coefficient on (log) distance. In **(2)**, 95% CI are from Table 4, specification 1 (Appendix Table A.4 details individual effects, *N*=11).



Appendix Tables and Figures

Table A.1. Three-digit residential zip codes, districts, population, and relevant distances in the São Paulo metropolis. Our sample covers all 96 districts in the city of São Paulo in addition to 7 satellite towns adjoining the city of São Paulo and inside the São Paulo metropolis (namely, Santo André, São Bernardo do Campo, São Caetano do Sul, Diadema, Mauá, Osasco, and Taboão da Serra).

Zip code in the São Paulo metropolis (*N*=43)	**Districts (includes 7 towns adjacent to São Paulo city)** (*N*=103)	**Population in 2010, all ages**	**Distance from district centroid to the nearest point on the original truck route (km)**	**Distance from district centroid to the closest NOx monitoring site (km)**
010xx	República	56,898	7.54	1.40
	Sé	23,620	7.97	0.49
011xx	Barra Funda	14,371	8.37	3.56
	Bom Retiro	33,825	9.47	2.30
012xx/013xx	Bela Vista	69,406	5.77	2.67
	Consolação	57,342	6.09	1.44
	Santa Cecília	83,606	8.02	2.92
014xx/054xx	Alto de Pinheiros	43,128	5.60	1.83
	Jardim Paulista	88,651	4.08	1.53
	Pinheiros	65,345	3.18	1.44
015xx	Cambuci	36,872	7.26	2.45
	Liberdade	69,030	6.41	2.46
020xx	Santana	118,845	12.29	5.01
	Vila Guilherme	54,295	12.90	4.56
021xx	Vila Maria	113,467	13.93	6.18
022xx/023xx	Jaçanã	94,585	18.88	10.57
	Tremembé	196,952	22.33	14.68
	Tucuruvi	98,447	15.89	7.99
	Vila Medeiros	130,005	16.46	8.08
024xx/026xx	Cachoeirinha	143,555	15.86	10.63
	Mandaqui	107,543	15.91	9.56
025xx/027xx	Casa Verde	85,608	10.97	5.72
	Limão	80,245	10.90	6.21
028xx	Brasilândia	264,764	16.21	11.73
029xx/051xx	Freguesia do Ó	142,349	11.97	7.81
	Jaguara	24,902	10.87	4.89
	Pirituba	167,879	13.11	8.43
	São Domingos	84,825	12.60	5.87
030xx	Belém	45,010	10.74	3.43
	Brás	29,229	9.20	1.03
	Pari	17,277	10.64	2.27
031xx	Mooca	75,613	8.22	3.69
	Vila Prudente	104,225	7.08	3.34
	Água Rasa	84,971	9.06	5.97
032xx	São Lucas	142,323	9.50	2.91
033xx/034xx/035xx	Aricanduva	89,664	12.98	6.33

Zip code in the São Paulo metropolis (*N*=43)	**Districts (includes 7 towns adjacent to São Paulo city)** (*N*=103)	**Population in 2010, all ages**	**Distance from district centroid to the nearest point on the original truck route (km)**	**Distance from district centroid to the closest NOx monitoring site (km)**
	Artur Alvim	105,317	17.65	11.30
033xx/034xx/035xx (continued)	Carrão	83,238	12.62	7.69
	Tatuapé	91,563	11.86	6.17
	Vila Formosa	94,792	10.87	5.66
	Vila Matilde	104,931	15.06	9.57
036xx/037xx	Cangaíba	136,628	18.32	12.22
	Penha	127,791	15.56	10.31
038xx	Ermelino Matarazzo	113,556	21.30	15.79
	Ponte Rasa	93,929	18.70	13.07
039xx	Sapopemba	284,503	12.44	4.94
	São Mateus	155,138	15.37	7.81
040xx/041xx/045xx	Itaim Bibi	92,474	0.19	2.74
	Moema	83,261	2.07	0.44
	Saúde	130,671	1.42	2.28
	Vila Mariana	130,427	4.29	2.61
042xx	Ipiranga	106,797	4.92	5.52
	Sacomã	247,681	3.16	4.51
043xx	Cursino	109,029	0.87	4.61
	Jabaquara	223,699	2.24	4.24
044xx/046xx/047xx	Campo Belo	65,760	1.36	1.39
	Campo Grande	100,631	6.40	6.87
	Cidade Ademar	266,479	4.96	6.45
	Pedreira	144,165	8.48	9.06
	Santo Amaro	71,462	4.43	4.81
	Socorro	37,794	9.15	9.27
048xx	Cidade Dutra	196,317	12.05	5.60
	Grajaú	360,538	17.88	3.32
	Marsilac	8,259	34.44	17.70
	Parelheiros	130,913	23.35	6.33
049xx	Jardim Ângela	294,979	15.06	9.83
050xx	Lapa	65,692	8.16	4.35
	Perdizes	111,087	6.33	1.85
052xx	Anhanguera	65,561	20.98	10.73
	Jaraguá	184,451	17.24	10.47
	Perus	80,101	21.46	14.04
053xx	Jaguaré	49,797	8.04	2.70
	Rio Pequeno	118,401	7.27	2.27
	Vila Leopoldina	39,360	8.48	4.11
055xx/056xx	Butantã	54,184	4.43	1.20
	Morumbi	46,839	1.65	3.94
	Raposo Tavares	100,086	9.49	3.34
	Vila Sônia	108,247	4.77	2.26
057xx	Campo Limpo	211,186	8.54	2.99
	Vila Andrade	126,439	5.00	3.60
058xx	Capão Redondo	268,481	12.15	7.00
	Jardim São Luís	267,617	10.17	8.13

Zip code in the São Paulo metropolis (*N*=43)	**Districts (includes 7 towns adjacent to São Paulo city)** (*N*=103)	**Population in 2010, all ages**	**Distance from district centroid to the nearest point on the original truck route (km)**	**Distance from district centroid to the closest NOx monitoring site (km)**
060xx/061xx/062xx	Osasco (town)	666,621	12.28	0.39
067xx	Taboão da Serra (town)	244,095	9.94	3.08
080xx	Jardim Helena	135,075	27.35	20.93
	São Miguel	92,124	23.83	17.51
	Vila Curuçá	149,030	25.42	18.37
	Vila Jacuí	142,365	22.14	16.11
081xx	Itaim Paulista	223,974	28.40	20.08
082xx	Cidade Líder	126,512	15.83	9.05
	Itaquera	204,841	20.56	13.87
	José Bonifácio	123,970	21.14	11.73
	Parque do Carmo	68,222	17.93	10.32
083xx	Iguatemi	127,418	20.68	7.35
	São Rafael	143,821	17.77	4.71
084xx	Cidade Tiradentes	211,309	23.78	11.42
	Guaianazes	103,948	24.05	13.87
	Lajeado	164,451	24.89	16.14
090xx/091xx/092xx	Santo André (town)	676,177	11.84	5.46
093xx	Mauá (town)	416,585	19.00	1.97
095xx	São Caetano do Sul (town)	149,185	6.49	1.29
096xx/097xx/098xx	São Bernardo do Campo (town)	764,922	11.34	10.10
099xx	Diadema (town)	385,838	7.75	10.35

Table A.2. Age distribution across all 43 3-digit residential zip codes over time. These zip codes cover all 96 districts in the city of São Paulo in addition to 7 towns adjoining the city of São Paulo and inside the São Paulo metropolis (namely, Santo André, São Bernardo do Campo, São Caetano do Sul, Diadema, Mauá, Osasco, and Taboão da Serra).

	Population by age group				
Year	0-4 years	5-19 years	20-64 years	65+ years	Total
2008	969,843	3,287,276	9,023,533	1,075,874	14,356,526
2009	947,417	3,250,610	9,143,842	1,108,968	14,450,837
2010	925,392	3,214,358	9,266,335	1,143,321	14,549,406
2011	952,934	3,173,293	9,330,880	1,176,973	14,634,080
2012	946,699	3,237,723	9,352,532	1,151,587	14,688,909
2013	940,464	3,264,488	9,393,761	1,155,189	14,753,901

Table A.3. Variations on the reduced-form analysis of traffic volume, diesel pollution, and hospitalizations reported in Table 2. **(1)** follows Table 2(1) but considers the proportion of congestion time from 7am to 4pm as the dependent variable. **(2)** follows Table 2(2) but considers daily maximum NOx as the dependent variable. **(3)** to **(6)** follow Table 2(1) to (4) but control for long-term changes using year fixed effects. An observation is: **(1, 3)** a day by road segment, **(2, 4)** a day by monitoring site pair, or **(5, 6)** a month by 3-digit residential zip code. Standard errors in parentheses. Other controls as in Table 2 are included but omitted from the table. Other notes to Table 2 apply exactly. ***, **, * denote significance at the 0.01, 0.05 and 0.1 levels, respectively.

Dependent variable	**(1)** **Road segment congestion:** % hours **7am to 4pm**	**(2)** **Diesel exhaust marker:** Daily **maximum** NOx, ppb	**(3)** **Road segment congestion:** % hours **5pm to 8pm**	**(4)** **Diesel exhaust marker:** Daily **mean** NOx, ppb	**(5)** **Cardiovascular admissions:** All-age, per 100,000 per month	**(6)** **Respiratory admissions:** All-age, per 100,000 per month
Truck policy implemented (yes=1)	**-0.028*** **	**-14.18** **	**-0.046*** **	**-13.18*** **	**-9.15*** **	**-4.61** **
	(0.003)	**(6.03)**	**(0.006)**	**(2.93)**	**(2.08)**	**(1.93)**
ln(distance) from key original truck route	-0.015***	subsumed in	-0.042***	subsumed in	subsumed in	subsumed in
	(0.001)	site FEs	(0.001)	site FEs	zip FEs	zip FEs
Truck policy implemented (yes=1)	**0.007*** **	**16.39*** **	**0.018*** **	**8.83*** **	**3.24*** **	**1.61*** **
× ln(distance) from orig. truck route	**(0.001)**	**(0.96)**	**(0.002)**	**(0.50)**	**(0.59)**	**(0.58)**
Trend after policy implementation	0.002	-	-	-	-	-
	(0.012)	-	-	-	-	-
Trend after policy implementation	-0.010***	-	-	-	-	-
× ln(distance) from orig. truck route	(0.002)	-	-	-	-	-
Time trend over the entire sample period	Yes	Yes	No	No	No	No
			2009: -0.003			
			2010: 0.005			
Year FEs	-	-	2011: 0.023**	Yes	Yes	Yes
			2012: 0.047***			
			2013: 0.049***			
2011 × ln(dist.) from orig. truck route	-	-	-0.008*** (0.002)	-	-	-
2012 × ln(dist.) from orig. truck route	-	-	-0.008*** (0.002)	-	-	-
2013 × ln(dist.) from orig. truck route	-	-	-0.011*** (0.002)	-	-	-
Number of observations	8,561,844	16,106	8,561,844	16,106	2,365	2,365
Number of regressors	259	699	264	703	580	580
R^2	0.245	0.694	0.297	0.757	0.903	0.912
Mean value of dependent variable	0.057	131.68	0.102	55.92	54.21	46.52

Table A.4. The spatial effects of the truck abatement policy on localized diesel pollution, using ambient NOx as a marker: Individual effects. This table reports the estimated individual policy effect at each of the 11 NOx monitoring sites in the metropolis. All specifications follow Table 4 exactly (for brevity, Table 4 reports mean effects across sites grouped according to distance from the original truck route, as shown here in the second column). Site 17 is located close to a heavily congested exit to a key highway (Castello Branco), on the inside of the beltway, which trucks bound from and to Santos port were able to bypass after April 2010 (Figure 1). OLS regressions. Standard errors, in parentheses, are one-way clustered by week-of-sample. ***, **, * denote significance at the 0.01, 0.05 and 0.1 levels, respectively.

Model specification & Dependent variable	Distance to original truck route (km)	(1) Base 24h NOx	(2) Trend, not year-mo. 24h NOx	(3) Site x meteor. 24h NOx	(4) 36 months 24h NOx	(5) Daytime 7am-8pm 14h NOx	(6) Metro only 24h NOx
Policy implemented (yes=1) × Site FEs							
Congonhas	0.02	-59.2***	-59.0***	-59.5***	-51.8***	-74.6***	-56.6***
(site 8)		(3.1)	(3.1)	(2.6)	(3.7)	(3.0)	(3.2)
Ibirapuera	2.36	-2.5**	-2.4**	-3.9***	0.7	-2.5***	0.1
(site 5)		(1.1)	(1.1)	(1.0)	(1.4)	(0.8)	(1.0)
Pinheiros	3.85	-0.7	-0.8	0.2	1.1	-1.5	2.0
(site 27)		(2.3)	(2.3)	(2.0)	(2.8)	(1.9)	(2.2)
Cerqueira César	5.00	-16.7***	-16.5***	-15.5***	-15.4***	-19.2***	-13.9***
(site 10)		(1.5)	(1.5)	(1.3)	(1.7)	(1.7)	(1.3)
IPEN-USP	5.59	-3.9***	-3.8***	-7.7***	-5.2***	-3.6***	-1.1
(site 31)		(1.2)	(1.2)	(1.1)	(1.4)	(1.1)	(1.2)
Taboão da Serra	6.90	2.2	2.1	2.2	7.4***	0.7	5.0***
(site 20)		(2.0)	(2.0)	(1.7)	(2.6)	(1.8)	(1.7)
São Caetano do Sul	7.52	-3.6***	-3.9***	-3.9***	-4.1***	-5.0***	-1.0
(site 7)		(1.0)	(1.1)	(1.0)	(1.5)	(0.9)	(1.0)
Parque Dom Pedro II	8.42	-4.6***	-4.6***	-5.3***	-1.4	-5.8***	-2.1*
(site 1)		(1.2)	(1.3)	(1.1)	(1.5)	(1.1)	(1.1)
Osasco	12.67	-11.5***	-11.6***	-11.6***	-3.5*	-13.5***	-
(site 17)		(2.1)	(2.2)	(1.8)	(2.0)	(2.3)	-
Parelheiros	17.01	1.5	0.9	2.4	2.4	-0.2	-
(site 29)		(2.0)	(1.9)	(1.5)	(2.3)	(2.2)	-
Mauá	17.22	2.0**	2.0**	0.4	2.0	1.6**	-
(site 22)		(1.0)	(1.0)	(0.6)	(1.4)	(0.7)	-

Table A.5. Structural analysis of health: (A) Semi-log specification, (B) Daily maximum NOx. The dependent variable is **(A)** the logarithm of the hospitalization rate, or **(B)** the hospitalization rate, for a given disease-age group pair. An observation is a 3-digit residential zip code by month pair (for **(A)**, where the hospitalization rate is strictly positive). Estimates for 14 2SLS regressions by disease-age in each panel. For each regression, we report β_{1l}, averaged across all zip codes l, for a 10 ppb increase in daily **(A)** mean, or **(B)** maximum, NOx equivalent units of diesel pollution (averaged within month). **(B)** uses fitted daily maximum (rather than mean) NOx (averaged within month) to instrument for measured NOx. Other notes to Table 5 apply here exactly. ***, **, * denote significance at the 0.01, 0.05 and 0.1 levels, respectively.

Hospitalization rate per 100,000 residents per month	**All ages**	**0-4 years**	**5-19 years**	**20-64 years**	**65+ years**
(A) Dependent variable: Logarithm of the hospitalization rate, NOx measure: Daily mean (as in Table 5)					
Cardiovascular admissions					
Diesel exhaust (+ 10 ppb NOx)	**.087*** (.016)**	**-**	**-**	**.080*** (.019)**	**.077*** (.020)**
Number of observations (rate > 0)	1,343	-	-	1,343	1,343
Respiratory admissions					
Diesel exhaust (+ 10 ppb NOx)	**.102*** (.018)**	**.130*** (.030)**	**.088** (.038)**	**.088*** (.029)**	**.132*** (.031)**
Number of observations (rate > 0)	1,343	1,339	1,319	1,343	1,337
Cardiovascular/respiratory death					
Diesel exhaust (+ 10 ppb NOx)	**.124*** (.034)**	**-**	**-**	**.093** (.047)**	**.094** (.037)**
Number of observations (rate > 0)	1,338	-	-	1,294	1,328
Placebo: Trauma admissions					
Diesel exhaust (+ 10 ppb NOx)	.040 (.034)	-	-	.047 (.046)	-.039 (.064)
Number of observations (rate > 0)	1,335	-	-	1,318	1,201
(B) Dependent variable: Hospitalization rate (as in Table 5), **NOx measure: Daily maximum**					
Cardiovascular admissions					
Diesel exhaust (+ 10 ppb max NOx)	**1.10*** (0.24)**	**-**	**-**	**0.67*** (0.24)**	**6.47*** (1.98)**
Respiratory admissions					
Diesel exhaust (+ 10 ppb max NOx)	**0.73*** (0.21)**	**6.59*** (2.15)**	**0.86** (0.34)**	**0.08 (0.15)**	**2.63** (1.28)**
Cardiovascular/respiratory death					
Diesel exhaust (+ 10 ppb max NOx)	**0.21** (0.10)**	**-**	**-**	**0.08 (0.09)**	**1.60 (1.05)**
Placebo: Trauma admissions					
Diesel exhaust (+ 10 ppb max NOx)	-0.10 (0.08)	-	-	0.00 (0.10)	0.22 (0.36)
Number of observations)	1,343	1,343	1,343	1,343	1,343
Number of zip codes	25	25	25	25	25

Figure A.1. Daily number of southern beltway users, by vehicle type. Data, based on toll-paying vehicles, are daily means within calendar month since toll operator SPMar's contract began in August 2011. "Heavy commercial vehicles" are commercial vehicles with at least three axles. Source: SPMar.

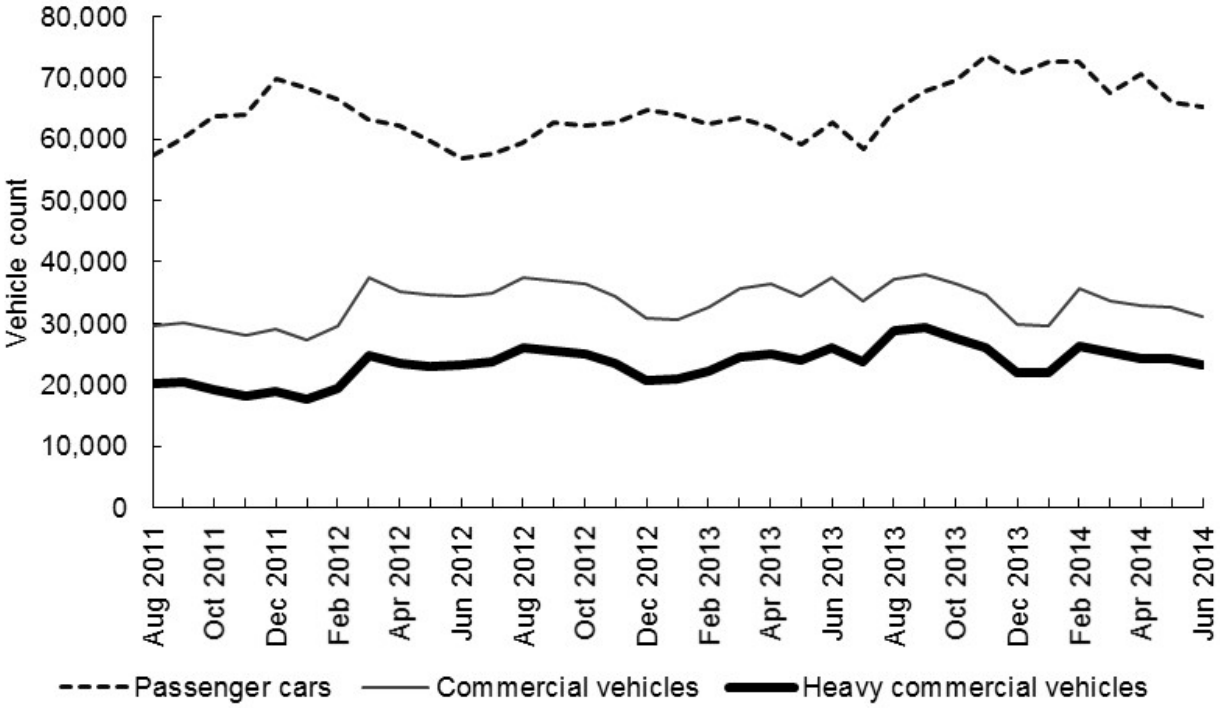


Figure A.2. Ozone versus NOx concentrations in the São Paulo metropolis. (1) Daily (24-hour) mean ozone (μg/m^3), or **(2)** daily maximum 8-hour mean ozone (μg/m^3), against daily mean NOx (ppb). Seven sites in the metropolis concurrently monitor both NOx and ozone. An observation is a site by month-of-sample. We show (site-specific) means over days within month-of-sample. **(3)** Fitted 24-hour ozone (μg/m^3), or **(4)** fitted maximum 8-hour ozone (μg/m^3), against fitted 24-hour NOx (ppb), per the excluded instruments in the 2SLS multi-pollutant health models of Table 8. An observation is a 3-digit residential zip code by month-of-sample. Source: CETESB, Tables 4(2), 7(2) and 7(5).

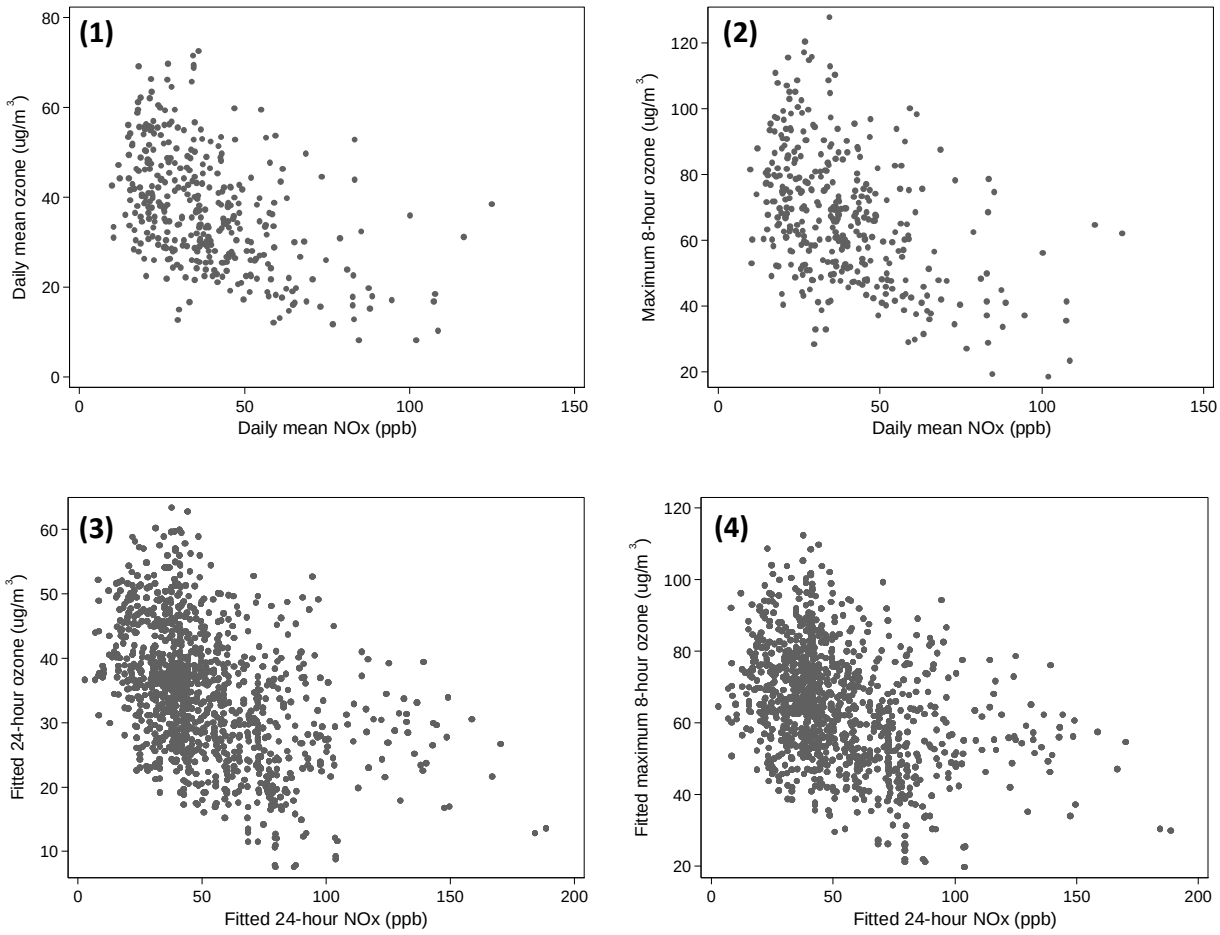
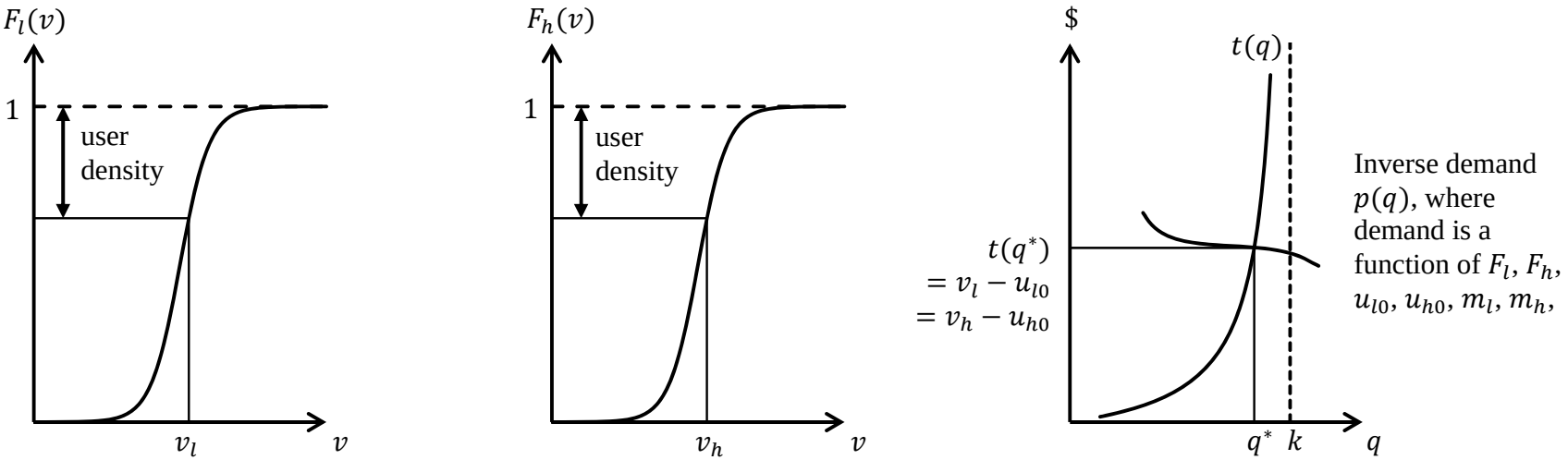


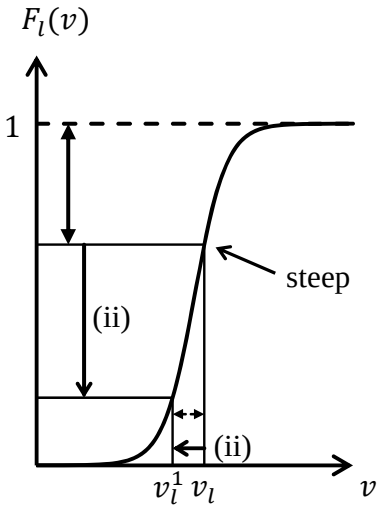
Figure A.3. Adding composition effects to the Law of Road Congestion. The top panels (a)-(c) indicate the marginal user of each type, and the aggregate number of users and individual cost in equilibrium. In the middle panels (d)-(f), the opening of a beltway raises the value of the outside option to urban road space for type-*h* users. In the bottom panels (g)-(i), exogenous demand growth shifts the demand curve to the right.



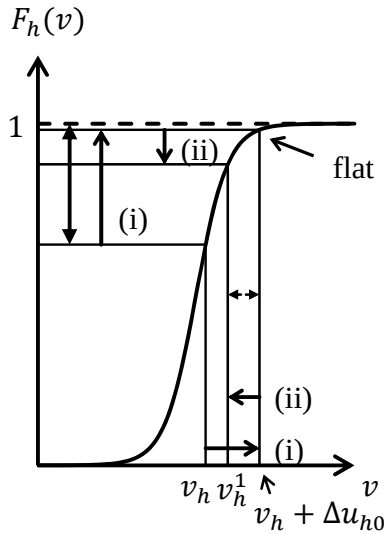
(a) Type-*l* preference distribution

(b) Type-*h* preference distribution

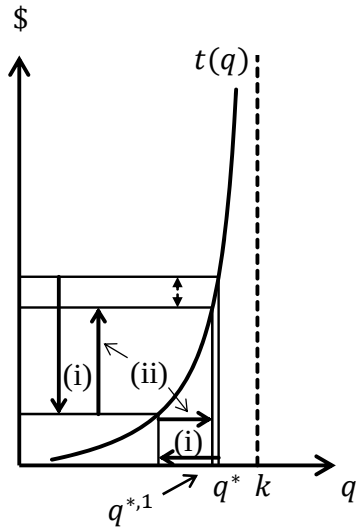
(c) Average (individual) user cost, common across users



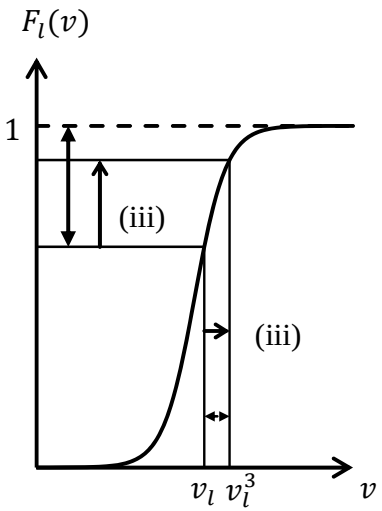
(d) Type-*l* preference distribution



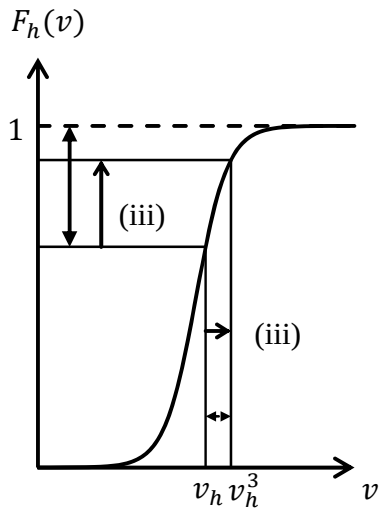
(e) Type-*h* preference distribution



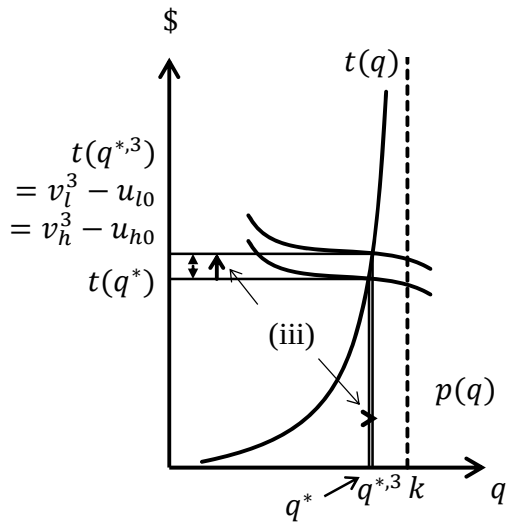
(f) Average (individual) user cost



(g) Type-*l* preference distribution



(h) Type-*h* preference distribution



(i) Average (individual) user cost