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Les effets de court terme sur la santé des perturbations dans les transports publics : pollution de l'air et transmission virale

Résumé

Lorsque les transports publics ne sont pas disponibles, la santé des populations urbaines peut en être affectée. D'abord, la pollution de l'air augmente du fait d'un report vers les véhicules personnels : les pathologies respiratoires peuvent être exacerbées à court terme. Ensuite, des contacts interpersonnels moins nombreux peuvent donner lieu à une moindre propagation virale. Nous mettons en évidence ces deux canaux, par différence de différences, en considérant les grèves dans les transports publics des dix plus grandes aires urbaines françaises sur la période 2010-2015. Les deux jours suivant une grève, les admissions en urgence à l'hôpital pour les grippes, les gastro-entérites et pathologies associées sont moins nombreuses. Malgré l'existence de cet effet de moindre contagion, qui touche aussi des pathologies respiratoires comme la grippe, l'effet néfaste de la pollution de l'air reste substantiel. Un jour de grève, les admissions pour les pathologies aiguës des voies respiratoires supérieures sont plus fréquentes, et le jour suivant, il en est de même pour les anomalies de la respiration. Nos résultats suggèrent que les choix de mode de transport des populations urbaines sont importants puisqu'ils sont responsables d'effets mesurables sur leur santé le jour même et les quelques jours suivants.

Mots-clés : Effet dynamique sur la santé, Grève dans les transports, Pollution de l'air, Contagion, différence de différences

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Short-term health effects of public transport disruptions: air pollution and viral spread channels

Abstract

When public transport supply decreases, urban population health may be strongly affected. First, as ambient air pollution increases, respiratory diseases may be exacerbated during a few days. Second, reduced interpersonal contacts may lead to a slower viral spread, and therefore, after a few incubation days, lower morbidity. We evidence these two channels, using a difference-in-differences strategy, considering public transport strikes in the ten most populated French cities over the 2010-2015 period. On the two days following the strike, we find less emergency hospital admissions for influenza and gastroenteritis. In spite of the existence of this contagion channel, which tends to mitigate the increase of admissions for respiratory diseases, we also evidence a substantial air pollution channel. On the strike day, we find more admissions for acute diseases of the upper respiratory system, while on the following day of the strike, more abnormalities of breathing. Our results suggest that urban population daily transportation choices do matter as they engender dynamic spillovers on health.

Keywords: Dynamic health effects, transport strike, air pollution, contagion, difference-in-differences

Classification JEL : I12, I18, C23, L91, Q53, R41

1 Introduction

For urban populations, public transportation is an essential part of daily life, but the health consequences of a well-functioning public transportation network are not yet fully acknowledged. As it offers an alternative to personal cars, the European commission, among others, "strongly encourages the use of public transport as part of the mix of modes which each person living or working in a city can use".[1] Indeed, when personal vehicle traffic increases, not only commuters are affected, but also the whole population of the area as air pollution increases (Bauernschuster, Hener and Rainer (2017)). Systematic correlations between mortality and outdoor air pollution are accumulating, but exogenous variations of air pollution are difficult to isolate though crucial in terms of causal evaluation. Therefore, public transport strikes have been considered to gauge the effect of traffic on air pollution (Basagaña et al. (2018), da Silva et al. (2012)). By relying on this variation of air pollution, we could indirectly observe and confirm the causal impact of air pollution on health in relatively moderate pollution levels typical of advanced economy urban pollution. But the health consequences of air pollution following the quasi-experiment triggered by a public transport strike is not that simple to analyze for two reasons. First, an additional channel, contagion, may intervene, due to lower interpersonal contact during a strike event. Second, the dynamic dimension matters as the air pollution shock may not be restricted to the very day of the strike. This quasi-experiment sheds light on the dynamic health consequences of the daily choices of urban transportation, when part of the population divert from public transport toward personal cars.

In this paper, we study emergencies admission rates in the ten most populated urban area in France to evidence two mechanisms. First, following a strike event, air pollution increases which leads to detrimental health effects, especially for frail populations (i.e. young children). Though this effect is documented by a huge epidemiological literature, quasi-experimental evidence is still scarce[2]. Times series analysis are abundant in the medical literature but do not rely on exogenous variation of ambient air pollution. This is a problem for instance if there are avoidance behaviours: when a pollution peak occurs and warnings are broadcasted to the population, fragile persons may spend less time outdoor to protect their health. Secondly, important confounding factors are weather conditions. If most studies take this into account by controlling by weather variables in regressions, they

[1]European commission policy statement that can be found at `https://ec.europa.eu/transport/themes/urban/urban_mobility/urban_mobility_actions/public_transport_en`

[2]If some papers address this question there are mostly restricted to the study of mortality (e.g. Currie and Neidell (2005), Arceo, Hanna and Oliva (2016) among others). Recent exceptions addressing morbidity are, on a yearly study Filippini, Masiero and Steinbach (2017) or Schlenker and Walker (2015) at daily frequency around airports.

must specify a functional form that will be crucial in the estimation. To cope with these issues, one can ideally rely on exogenous variation triggered by an unrelated event.[3] Following Bauernschuster, Hener and Rainer (2017), we argue that a public transport strike event triggers exogenous variation in ambient air pollution. Nevertheless, we do not assume that ambient air pollution is the only channel through which respiratory health is affected, nor that the consequences of a strike day are restricted to the day of the event. As interpersonal contacts may decrease in public transports themselves, but also by cancellation of trips to work or to school, viral spread is expected to decelerate. For some pathologies, in particular acute upper respiratory system diseases[4] the two channels under study have opposite effects on health: in this quasi-experimental setting, the delayed negative impact of air pollution is mitigated by less contagion. Our objective is therefore twofold. Firstly, by evidencing an increase in respiratory emergency admissions on the very day of the strike, we show the short-term importance of a well-functioning and widely used day-to-day public transportation network in terms of health. Secondly, we evidence that the limitations of social interactions likely occurring in these days may lead to positive health effects, in the spirit of Adda (2016). Acknowledging this second channel is important in properly interpreting ambient air pollution delayed effects: when we find evidence of detrimental effects on health, it is most probably a lower bound. We here contribute to the small but growing literature linking causally air pollution and morbidity (e.g. Schlenker and Walker (2015), Filippini, Masiero and Steinbach (2017), Jans, Johansson and Nilsson (forthcoming))[5], in particular showing that taking into account the dynamics of the effects over a few days is important: pollution accumulates, has its own dynamics chemistry and the body may not react immediately to a pollution shock. One of the strength of our dataset is to provide a rare perspective on pollution as we monitor the reaction of six pollutants. It is of importance in the dynamic dimension as some pollutants are secondary, i.e. not emitted directly but formed from primary pollutants.

We study separately the impact of a strike on several outcomes of interest, using a difference-in-difference strategy. To this end, we make use of four rich datasets that provide information on hospital emergency admissions, air pollution, traffic and congestion in the ten most populated urban areas in France, over the period 2010-2015. Using 91 one-day strike events staggered over time and cities, we exploit both time and cross-sectional dimensions in order to quantify

[3] For example Moretti and Neidell (2011) use daily variation in boat traffic and Schlenker and Walker (2015) use excess airplane idling to estimate the impact of respectively ozone and carbon monoxide on local residents respiratory diseases.

[4] For example influenza, pneumonia: pathologies that are mostly due to a viral infection but also favored by air pollution (See Ciencewicki and Jaspers (2007))

[5] This literature follows a larger literature linking mortality and air pollution: among others, Currie and Neidell (2005), Arceo, Hanna and Oliva (2016) ..

the impact of a strike over traffic, congestion, air pollution and health. We use a difference-in-difference strategy at the city-date level and first check that cities affected by a strike show an increase in congestion and in traffic relative to other cities. Travel times increase by 7% on average in the day. Around 9 a.m., vehicle flows display a significant and large increase: in the morning peak hours, monitoring stations record an increase by about 20 % of counted vehicles. Air pollution increases gradually: on the day of the strike, we find an increase in carbon monoxide concentration, a pollutant whose traffic-related emissions have been dramatically reduced in recent cars thanks to European norms. Particulate matters increase by 10% the day following the strike, probably due to a gradual accumulation. Finally, ozone is of interest as it is a secondary pollutant formed from traffic-related primary pollutants. As expected from its dynamics, ozone increases later by 5%, on the second day following the strike. This indirectly confirms that the primary pollutants increase before secondary ones. Because pollution dynamics is complex, the consequences of an increase in traffic can last severals days. Secondary pollutants arrive in a second phase and when air does not circulate, an accumulation of pollutants occurs.

Our results show that the upper respiratory system is affected first, with emergency admissions increasing by 30% on the strike day. This upsurge is borne by young children (0-4 year-old). The following day, children from 5 to 14 year-old also show up more than a regular day in emergency rooms for this pathology. Lagged adverse effect is also observed for abnormality of breathing. However, surprisingly, emergencies for respiratory diseases taken as a whole, i.e. adding most notably pathologies with chronic origins and influenza seem to be unaffected. We argue that the delayed effect of ambient air pollution is compensated by a lower incidence of viral pathology such as influenza and pneumonia that is found across several age groups but most firmly for 5-14-year-old, on the two days following the event. This timing is in line with the expected incubation time if contagion is weakened on the strike day. To assert this interpretation, we show a similar result for gastroenteritis, which is subject to contagion with a lower incubation period, but not to air pollution. Despite being in a context where we may underestimate the delayed effect of air pollution, we find a sizeable effect on health, borne primarily by children.

Finally, we conduct a set of robustness checks. First, we conduct a falsification test on top emergency causes that we believe are unrelated to the quasi-experiment of a one day strike: pregnancy and digestive diseases. As could be expected, the strike do not affect other diseases, unrelated to ambient air pollution and not caused by a virus. Second, controlling by date fixed effects allows to account for national level shocks which may be correlated with the event of a strike.[6] However, it may be possible that in a given city, strikers choose a date where large traffic is expected for some reason unknown to the econometrician.[7] In this case, the exogeneity of the variation triggered by the strike is undermined. To check that it is most likely not the case, we use the hotel occupancy rate as a proxy for the happening of city-specific events and verify that it is not different from other days.

The remainder of the paper is organized as follow. Section 2 presents the institutional context surrounding strike events and review the main mechanisms under investigation in the literature. Section 3 describe the data sets, section 4 the econometric strategy and section 5 our results. Section 6 provides robustness checks and Section 7 concludes.

2 Institutional context and mechanisms

Public transports in France are well developed: aside the well equipped Paris area, other regions count 11 metro lines, 54 tramways and 3691 bus lines in 2012 that respectively account for 133, 589 and 50 695 kilometers (Commissariat général au développement durable (2015)). They are an important mode of transportations of urban populations. For instance in the Nantes *métropôle*, 15% of trips from Monday to Friday are done thanks to the collective transport system. We analyze the ten most populated urban area as listed in Table 1, that all rely on a well developed public transport network. Their geographical position and extent is represented in Figure 1

In this paper, we use behavioral variations caused by public transport perturbations in the event of a strike. The International Labour Organisation (ILO) recognizes strike action as a fundamental right of organized labor. In France, it is a constitutional right that has been written in the constitution since 1946. Regarding French terrestrial transports, a 2007 law provided two guidelines on strike right. First, strikers have to declare at least 48 hours in advance whether they are willing to strike. Second, negotiations between the transport firm and trade unions have to start before the strike. This law does not prohibit strike action, but the first guideline limits its potential impact. It makes easier for the transport firm to redeploy the non-strikers to lessen the effect of strike actions.

[6]For example, if strikers choose preferably a day when large departure traffic is expected because of a national bank holiday.

[7]For example, if strikers choose preferably a day when large traffic is expected because of a city-specific event.

Table 1: The ten most populated urban area in France

Urban area	Population (in thousands)		
	All age	0-4	over 70
Paris	12,470	845	1,203
Lyon	2,259	152	249
Marseille - Aix-en-Provence	1,744	103	231
Toulouse	1,312	81	137
Bordeaux	1,195	67	135
Lille	1,182	80	111
Nice	1,006	52	171
Nantes	934	61	97
Strasbourg	777	45	89
Rennes	708	46	70

For Lille and Strasbourg urban areas, only the French part is considered.

Source: 2013 census

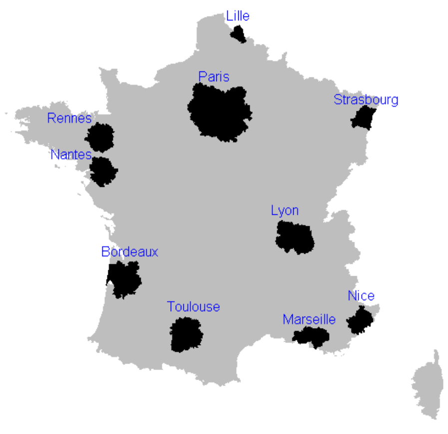

Figure 1: Geographic location of the ten most populated urban areas in France.
Source: Insee, urban areas database, 2010.

2.1 Air pollution and health

A large epidemiology literature has consistently linked air pollution to respiratory and cardiovascular diseases, both at long and short term (Seaton et al. (1995)). The wake-up call could be dated to 1952 when a massive smog episode in London provoked an historical number of deaths.[8] Nevertheless, health effects are not only observed under extraordinary events but as well at pollution levels commonly occurring in developed countries.

Ambient air pollution penetrates into the respiratory system by inhalation. The biological mechanisms are still under active survey, but the highly suspected mechanisms are as follows: air pollution can provoke inflammatory responses at different level of the respiratory system (Ozone, NOx and particulate matters are potent oxidants, Brunekreef and Holgate (2002)). Cells oxidative stress appears as a mediator of the pulmonary inflammatory response. Particulate matter can penetrate into the lower respiratory system. PM2.5 (Particulate matter of less than 2.5 μm) can even penetrate into the gas-exchange region of the lung, irritate and corrode the alveolar wall.[9] Importantly, symptoms may appear several days following an increased exposure level and may persist for several days. In general, studies emphasize distinct effects by age groups. The lung functions of the young child are not completely developed and may be particularly subject to external aggression (Gauderman et al. (2007)). Elderly are subject to reduced lung functions that occur naturally with aging and to increased co-morbidity. We here focus on the short-term effect of ambient air pollution. Long term effects are also of great interest, and represent a huge medical literature, often referred to as "cohort studies". Anderson (2015) and Deryugina et al. (2016) are recent papers addressing this question in the econometric literature.

An important caveat, not addressed here, is that the estimates have to be interpreted cautiously. We can not distinguish what is caused (without pollution, the subject would not have been ill) and what has been exacerbated (without pollution but with another adverse shock, the subject would have been ill as he was in the brink of falling ill). We will only be able to check whether in case of a resurgence of emergency admissions a given day, we observe the converse a few days later (the extreme case where it will only displace admissions that were meant to happen). This hypothesis is referred to the "harvesting hypothesis" when studying mortality.

[8] Bell and Davis (2001) estimate that 12,000 excess deaths occurred due to both the acute and persisting effect of the 1952 London smog.

[9] The alveolar membrane is the gas exchange surface. Carbon dioxide rich blood is pumped from the rest of the body and through the alveolar system released while oxygen is absorbed and afterward directed to the heart.

2.2 Collectivity exposure and viral diseases spread

Using public transport strikes as a quasi-experiment to infer the health responses to increased ambient air pollution might be dampen by a second channel. On a strike-day, we may expect reduced interpersonal contacts for several reasons: reduced frequentation of public transportation, reduced attendance at school, impossibility to go to one's workplace, choice to take a day off and stay at home... In turn, reduced contact may lead to slower viral spread. This effect has long been recognized in the literature (e.g. Thélot and Bourrillon (1996)) and regained interest recently (e.g. Adda (2016)). Moreover, as ambient air pollution has been pointed as making a favorable ground for contagious diseases such as pneumonia, flu or pharyngitis, we may underestimate the delayed impact of air pollution on health if we do not acknowledge the existence of this channel.

The role of children as primary vector of transmission of influenza has been emphasized: because they have the highest risk factors (Viboud et al. (2004)) there is a high benefit of their vaccination (Weycker et al. (2005)). Therefore, schools closure have been pointed-out as non-pharmaceutical intervention to contain pandemic episode and used for instance by several countries in the 2009 flu pandemic (Cauchemez et al. (2014)). The effect of school holidays on flu transmission has been studied by epidemiologists (Cauchemez et al. (2008)) as well as economists (Adda (2016)) who reach similar conclusions: the effect is the most pregnant for children and there are limited side effects on adults (e.g. parents, grandparents). The short incubation period of influenza[10] is an important parameter in our context, as it gauges the time between reduced contagion (on a strike day) and reduced morbidity (on strike day plus the incubation time).

Evidence for public transport strike is more scarce. Thélot and Bourrillon (1996) comment the coincidence of an unusually low bronchiolitis episode in Paris region and a multiple weeks public transport strike. The authors interpret this coincidence by lower attendance of young children to daycare. Adda (2016) studies both school closures and railway transport strikes at a weekly frequency in French regions. In the transport strike case, he founds no effect in acute diarrhea[11] the week of the strike but significant negative effects in flu-like illnesses (measured by visits to general practitioner thanks to the *Réseau Sentinelles* in France). In particular, the strongest reduction is found for children, even though there is some evidence of spillovers on adults.

[10] According to WHO, 2 days on average and in a range of 1 to 4 days.

[11] Whose incubation period is estimated to be short as well: 0-2 days, Adda (2016)

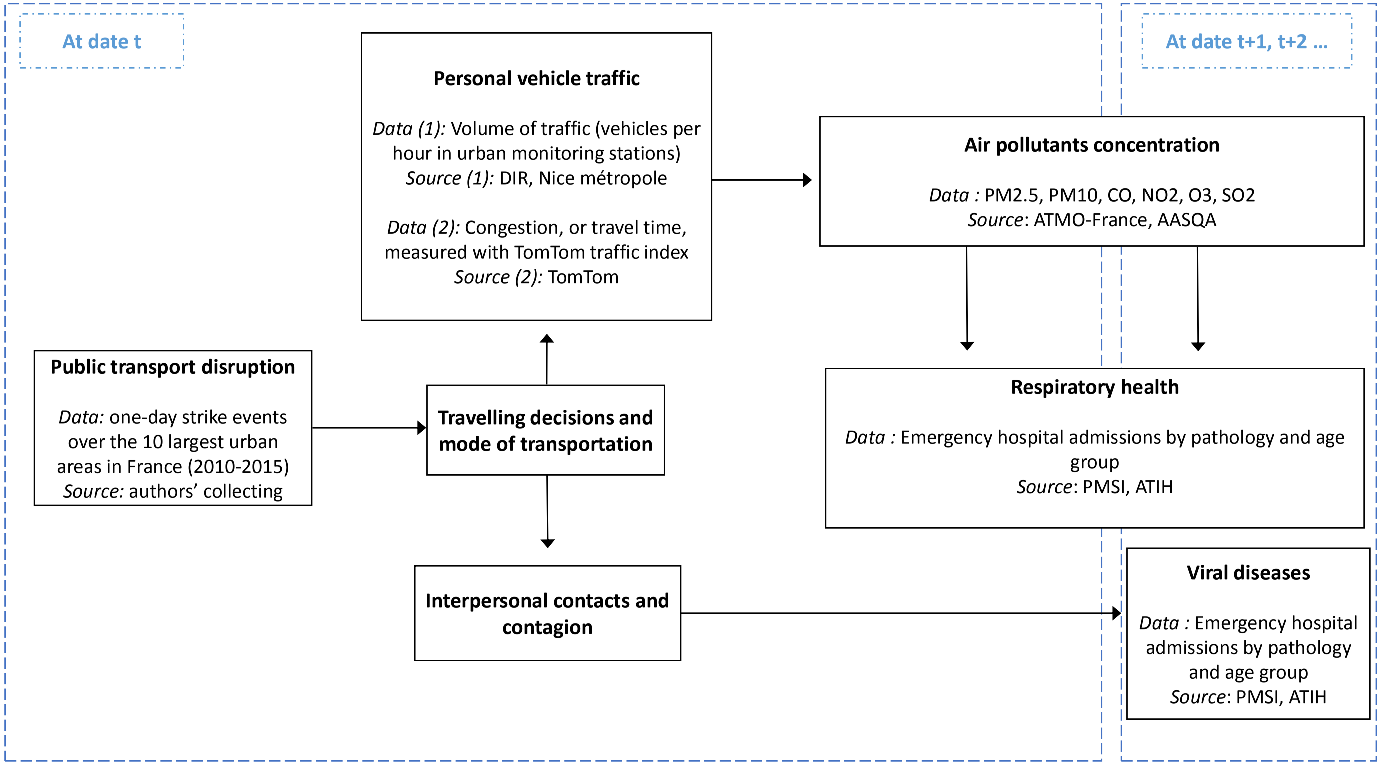

Figure 2: Causal chain and summary of data sources.

3 Data

In this section, we describe the data sources which cover the ten most populated urban areas in France (Table 1) over the 2010-2015 period. Figure 2 represents the causal chain studied in this paper and enumerate the main data sources to monitor the different outcomes. The following section provide more details.

3.1 Strikes

We gathered strike events in public transportation services, collecting ourselves the data following a cautious process: every recorded event has to be reported by two distinct media. For each event, we record where and when it occurs, its length and an estimation of the magnitude of the perturbation, in the sense whether newspaper information refers to a perturbation of at least part of the network or whether there was only a negligible perturbation (although a strike was announced). Because we face an heterogeneous phenomenon, with long strike events (the longest strike in our sample lasts 75 days), we restrict the strike events under consideration to one-day strikes. We also withdraw the minor perturbations as we do not expect the shock to be sufficient and to reflect the absence of public transport for part of the users.[12] To simplify interpretations when considering delayed estimates, when strike episodes are adjacent in a two day window, we exclude the strikes episodes with the two-day window at the city-date level (as we compute delayed effects up to two days).

In the estimation sample, we exclude school holidays when traffic and therefore pollution are not easily captured by our extensive set of fixed effects. Holidays adjacent days are as well excluded as they are characterized by departure and return trips. Our sample is therefore restricted to days within a regular week.

Finally, we are left with 91 one-day strike episodes that occur within an "usual" week and that we use in our estimations. Our baseline sample has 13,500 city-date observations, as long as there is no missing values in the outcome considered (which is the case for congestion and emergency admissions). Our sample is somewhat reduced when considering air pollutants as not all cities record all pollutants at all time, due to physical sensor malfunctions.[13]

[12] i.e. we exclude the city-date observations when there is a longer strike or a minor strike event in all our regressions. Adjacent days of strike episodes that we do not consider here are also excluded in the city-date sample.

[13] Considering a sample where all pollutants are measured at the city-date level entails losing half of the observations and strike events. We nevertheless check that the event of the strike is unrelated to pollutants' missingness.

Table 2 shows the different filters from collected sample to the strike episodes used in our regressions. Figure 3 represents how strikes in the estimation sample are staggered across time and space. The autumn of 2010 was marked by a social movement against pension reforms which raised the non-penalized retirement age for pensions from 65 to 67 and the minimum legal age from age 60 to 62. In the robustness section, we check that excluding 2010 or September do not affect our results.

Table 2: Differences between collected and in-sample strikes: impact of filters (categories from rows (1) to (4) are non-exclusive)

Strike episodes	Collected	209
	In estimation sample	91
Not considered because	(1) Occuring in holidays	34
	(2) Over 1 day duration	34
	(3) Minor perturbation	73
	(4) Another strike within a 2 days windows	38

3.2 Congestion

In the causal chain, we first expect a diversion from public transport to personal cars and therefore, more vehicles on the roads and more congested conditions. Congestion is measured by the Tomtom traffic index[14] which aggregates Tomtom users' individual delays compared to a free flow situation. Tomtom is a worldwide company specialized in navigation device, and provides in particular embedded GPS and navigation assistance in vehicles. We obtained the historic of the daily Tomtom index over 2010-2015 for the ten most populated French urban areas.

The methodology of the index is described in Cohn, Kools and Mieth (2014): starting 2007, GPS speed measurements were collected and mapped to road segments. For each individual road segment r, non-congested (free-flow) travel times $T_{0,r}$ are determined based on the shortest travel times of two years of measurements. The overall congestion level value for a city is the weighted average extra travel time experienced by drivers in that city when compared to the free flow travel times i.e. for r a road segment, $100 \times (< \frac{T_r}{T_{0,r}} > -1)$, where travel time is denoted T and T_0 under a free-flow situation. The average is weighted by the volume of users on each road segment. Thus, the value of the congestion index may be read as a percentage increase in travel time, with a weighting scheme which allows to over-represent larger volume roads compared to quiet roads.

[14]https://www.tomtom.com/trafficindex/

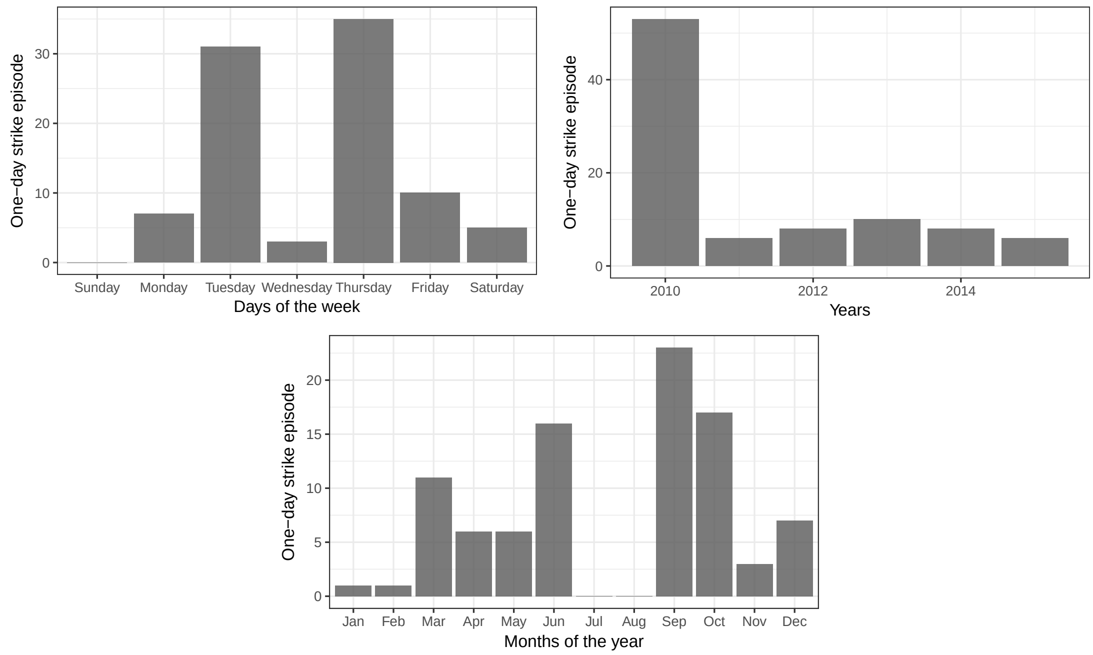

Figure 3: Strike repartition over time and space, among in sample strikes (See Table 2). *Source: Authors' collecting, from media reports*

Table 3 shows the daily congestion level by city: on average over the period, trips in Paris are 33% longer than under free-flow conditions. The least congested city is Rennes. We might be concerned that active Tomtom customers are not numerous enough for the least populated cities, and that it weakens the aggregate Tomtom traffic index.[15] However, the right panel of the Table 3 show a quality proxy for the index: the number of vehicles kilometers used to compute the index, or in other words, the number of users times the traveled kilometers. The first decile in Nice, the least congested urban area on average, is 23 000 kilometers per day which seems rather reasonable (it would correspond for example to about 750 persons traveling 30 kilometers to produce a daily measure).

3.3 Traffic

An additional source is considered to study traffic conditions on strike days, on top of extra travel time: count of vehicles on main roads. Traffic data were gathered from the regional *Directions Interdépartementale des Routes*[16] (DIR), decentralized governmental administrations in charge of managing, maintaining and operating roads. As part of their transportation policy, they collect hourly

[15]Ideally, we would check whether Tomtom customer's travels are representative of urban area's travels, but we have no data to check whether it is the case.

[16]DIR Centre-Est, DIR Ouest, DIR Méditerranée, DIR Est, DIR Atlantique, DIR Nord, DIR Sud-Ouest and DIR Ile-de-France

Table 3: Tomtom traffic index over 2010-2015 in the ten most populated urban area in France

	Daily congestion level				Daily total vehicle kilometres (/1000)			
City	Mean	D1	D9	sd	Mean	D1	D9	sd
Marseille	36.2	19.7	50.1	11.7	49	31	71	15
Paris	32.9	16.4	48.0	12.0	2806	1907	3899	738
Lyon	24.7	11.3	36.0	9.7	303	198	430	87
Nice	24.7	13.2	33.6	7.9	37	23	52	12
Bordeaux	24.3	8.4	37.5	11.2	180	112	258	55
Toulouse	21.7	8.5	35.0	10.4	149	93	214	45
Strasbourg	21.6	7.3	33.6	10.2	59	40	80	15
Nantes	19.4	6.8	30.3	9.1	123	80	173	35
Lille	18.0	5.2	30.5	10.0	214	146	293	55
Rennes	16.3	4.9	26.9	8.9	63	42	88	19

Daily congestion level measures the percentage of increased travel time compared to a free flow situation. Vehicle kilometres is the sum of the users' trips in kilometres a given day in the urban area. *Source: TomTom*

traffic flow (number of vehicles per hour) on their network (state-owned roads) and disclose annual statistics to the public. In general, this network covers correctly the immediate surrounding of the cities and includes ring roads. Except for Nice however, the city center is not covered. Each city data set is provided by a distinct DIR (except for Rennes and Nantes that both depend on the same DIR *Ouest*). One exception is Nice, where the data has been provided by the *Métropôle* (an intercommunal structure, centered on the city of Nice).

Some stations were not constantly operating and we often observed breaks in the times series (possible explanation might be temporary road works on one lane or the dysfunction of a captor). We therefore excluded stations where a break was observed or when a major time period was missing (e.g. more than a year). Maps showing the remaining stations are shown in appendix. In general, monitoring station are well distributed around cities. Then, we applied the following filter: we compute the average daily traffic flow per monitoring station and exclude the observations for that station on days where the daily traffic was under 10% of this value. Table 6 shows summary statistics, and Figure 8 in appendix shows the average hourly traffic pattern.

Table 4: Traffic flow in number of vehicles.

		Hourly traffic flow		
City	Monitors	Mean	D1	D9
Bordeaux	11	1861	219	3700
Lille	12	1175	137	2525
Lyon	13	2109	282	4228
Marseille	9	1098	89	2620
Nantes	20	1164	111	2498
Nice	12	483	28	1141
Paris	9	2183	302	4383
Rennes	15	1120	111	2599
Strasbourg	16	1995	227	4312
Toulouse	11	2167	264	4344

Note: When there are two measures for the two opposite directions at the same geographical location, we consider that there are two monitors. *Source: DIRs, Nice Métropôle*

3.4 Air pollution

Air pollution in urban areas, closely linked to congestion and vehicles on the roads, is the third outcome expected to be modified by the strike.

Air quality is measured by regional associations called AASQA (*associations agréées de surveillance de la qualité de l'air*), which are grouped in a national federation called ATMO France. They operate numerous air quality measurement stations all over France. We consider the stations located in the 10 most populated urban areas. We focus on a rich set of air pollutants: the 6 pollutants that are widely available on an hourly basis are carbon monoxide (CO), particulate matter of less than 2.5 micrometers (PM2.5), particulate matter of less than 10 micrometers (PM10),[17] nitrogen dioxide (NO2), ozone (O3) and sulfur dioxide (SO2). We usually have data for several measurement stations per urban area,[18] which we average at the city and hourly level on a constant set of monitoring stations. For each city and each day (24 observations), we compute three quartiles of air pollutants' concentration.

NO2, O3, CO, particulate matters and SO2 are long recognized to be of critical importance in environmental policies: they cause smog and damage air quality, and anthropogenic emissions are large. The European Commission but also municipalities set threshold values over these pollutants' concentrations used in particular

[17] In French, PM2.5 and PM10 are called *particules fines*

[18] We have at least one measurement station for each pollution in each urban area.

to inform the population in case of pollution peaks. Most are primary pollutants, directly emitted in the atmosphere, except NO2 (quickly formed from NO, a primary pollutant) but most notably O3. Ozone is a secondary pollutant which is formed from a group of precursors pollutants under the action of UV rays. Precursors of ozone are predominantly those emitted during the combustion of fossil fuels, i.e. NOx but also CO. If ozone is not directly emitted by traffic sources, its production is indirectly strongly connected. As for the other pollutants, in 2010, personal vehicle traffic represented 22% of NOx emissions in metropolitan France, 13% of PM10, 11% of PM2.5, 10% of CO emissions but only 0.1% of SO2, which is primarily industrial.[19]

Figure 4 shows how pollution is tightly linked to human week: in light of all the primary pollutants' concentrations, Sunday is the least polluted day, followed by Saturday. PM, CO and NO2 clearly display an accumulation process throughout the week, while SO2 seem rather constant throughout the business week. O3 dynamics is singular for several reasons: the transformation from precursors is a slow process (NO2+O2 $\rightarrow$ O3+NO) which needs particular conditions but also, this reaction is inhibited (or reversed) by NO, which is emitted by traffic as well. As a result, O3 is at its peak after a week accumulating precusors pollutants, when other pollutants are at their lowest levels. This explains why ozone is higher in rural areas rather than in urban areas.[20]

3.5 Hospital admissions

The first and foremost outcome of interest is health, considered here through hospital emergency admissions.

The data is obtained from the ATIH (*Agence Technique de l'Information Hospitalière*) that gathers an administrative and exhaustive database which records all admissions in both public and private hospitals. An admission is systematically recorded in case of an overnight stay and a doctor may decide to admit a patient if care is provided within the day or if patient surveillance is necessary.[21] Its primary use is to compute hospitals' fundings based on their activity. We were provided the daily count of emergency admissions for each final diagnosis and each urban areas. Further, this information breaks down by age range (0-4, 5-9, up to 75-79 plus over 80). More precisely, an emergency admission is an entrance through the hospital emergency unit that led to an admission from patients coming from

[19] Inventories of air pollutants' emissions are realized in France by the CITEPA and transferred to the UE. See https://www.citepa.org/fr/activites/inventaires-des-emissions/secten

[20] For a more detailed presentation on the role of road traffic on ground-level ozone, see Munir, Chen and Ropkins (2012).

[21] Not all persons presenting to emergency units are admitted.

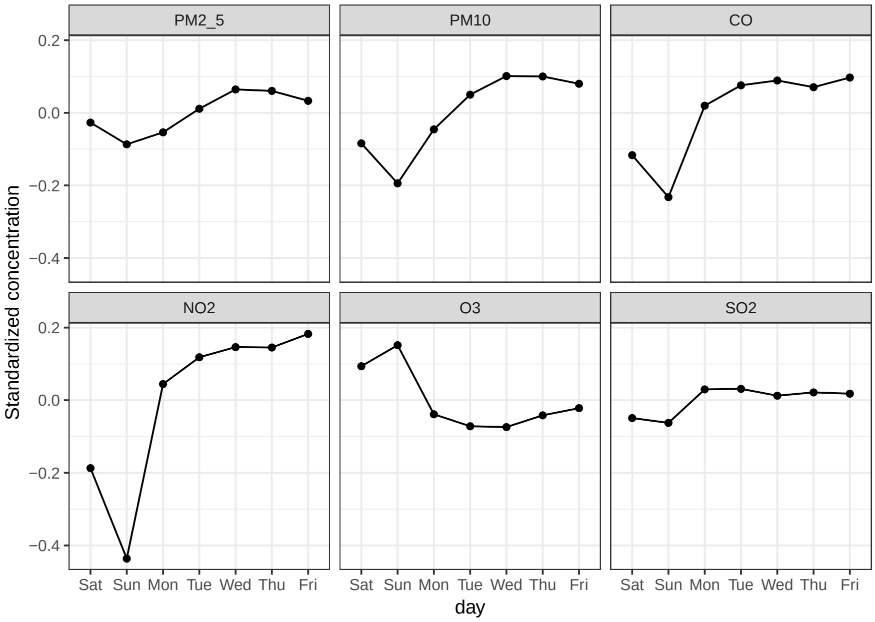

Figure 4: Pollution concentration along the week. *Source: ATMO France*

their residence (i.e. not transferred from another hospital) or from public space.[22] Therefore, elective care, long-term and recurring care are excluded. The diagnosis used here is coded at the end of the patient stay. It accounts for the main diagnosis which gave rise to the highest care resources. When and only when a diagnosis is not reached, the code is relative to the observed symptoms.

We divide the daily count of admissions by the appropriate age range urban area population (Source: Insee, legal population 2013). Our variable of interest is the emergency rate of admission per 100 000 persons. For some diseases and age ranges, admissions are extremely rare. We first restrict our sample to respiratory diseases and symptoms related to the breathing system. To this sample, we add another pathology with viral causes to check on contagious effects: gastroenteritis. We show in Figure 5 the 6 leading causes of emergency admissions. Respiratory diseases ranks first for the youngest children (0-4 year-old). Among these six leading causes of emergency, four might be affected by one-day-strikes (respiratory diseases, cardiovascular diseases, injuries, general symptoms). The last two (pregnancy and digestive admissions) will serve for falsification tests. Figure 5 shows clearly how age is a relevant dimension: more fragile and subjected to comorbidity, elderly people are over represented in emergency admissions. The sharp increase begin around 60. In respiratory diseases and symptoms, children aged from 0 to 4 are over represented. From then on, we define three populations of interest: all,

[22]When due to hospital organization, emergency room is the main entry point, doctors should not use the code "emergency" systematically (only when the individual situation in the views of the patient, his relatives or his general practitioner is an emergency).

0-4 and over 60. For viral contagion, we will also focus on school-aged children from 5 to 14. Table 5 enumerates the pathologies of interest according to the ICD-10 classification.[23].

Table 5: Daily rate of admission per 100 000 persons and ICD-10 classification.

Pathology	ICD10	Rate of admissions per 100 000 persons									
		All		0-4		5-14		15-59		Over 60	
		Mean	sd	Mean	sd	Mean	sd	Mean	sd	Mean	sd
Intestinal infection (e.g. gastroenteritis)	A08-A09	0.24	0.22	2.24	2.87	0.17	0.37	0.07	0.11	0.15	0.25
Diseases of the respiratory system	J00-J99	1.37	0.72	5.47	5.65	0.50	0.70	0.49	0.32	3.12	1.67
- Acute upper respiratory infections	J00-J06	0.08	0.10	0.77	1.24	0.08	0.24	0.03	0.07	0.01	0.08
- Influenza and Pneumonia	J09-J18	0.43	0.28	0.70	1.22	0.11	0.29	0.15	0.16	1.33	0.94
Abnormalities of breathing	R06	0.08	0.10	0.12	0.48	0.01	0.08	0.03	0.07	0.23	0.33
Pregnancy	O00-O99	0.90	0.83	< 0.01	< 0.01	< 0.01	0.04	1.51	1.36	< 0.01	0.01
Diseases of the digestive system	K00-K93	2.79	0.97	1.84	2.53	1.40	1.57	2.18	0.95	5.53	2.41

Sample from 01/01/10 to 31/12/15 in the ten largest urban ares of France. *Source: ATIH*
Chapters of the ICD-10 are in bold (highest level of classification). Other are subcategories. Mean is $< r_{s,a,t,c} >_{s,a}$ for s a pathology, a an age range, t a given date, c a city, where $r_{s,a,t,c} = \frac{n_{s,a,t,c}}{N_{a,c}}$ the ratio of admissions n over population N is our outcome of interest.

3.6 Weather

Weather conditions play a key role in human activity (including the decision to travel and the driving behaviour, to some extent) and air pollution formation. We hence consider a full set of weather conditions in our estimations. Data come from Météo France and are available on an hourly basis for our 10 urban areas. We consider seven weather parameters as control variables: temperature, rainfall, wind speed, wind direction, insolation, humidity and vapor pressure. We also consider a day-level dummy coding for presence of snow. Measurement stations are located at nearby airport[24], except for Paris, where the measurement station is located in a garden in the center of Paris (parc Montsouris).

[23]International Statistical Classification of Diseases and Related Health Problems (ICD)

[24]Specifically for insolation in Lille, we use the measurement station in Lillers, nearby Lille, as this parameter was not available in Lille-Lesquin airport station over the whole studied period.

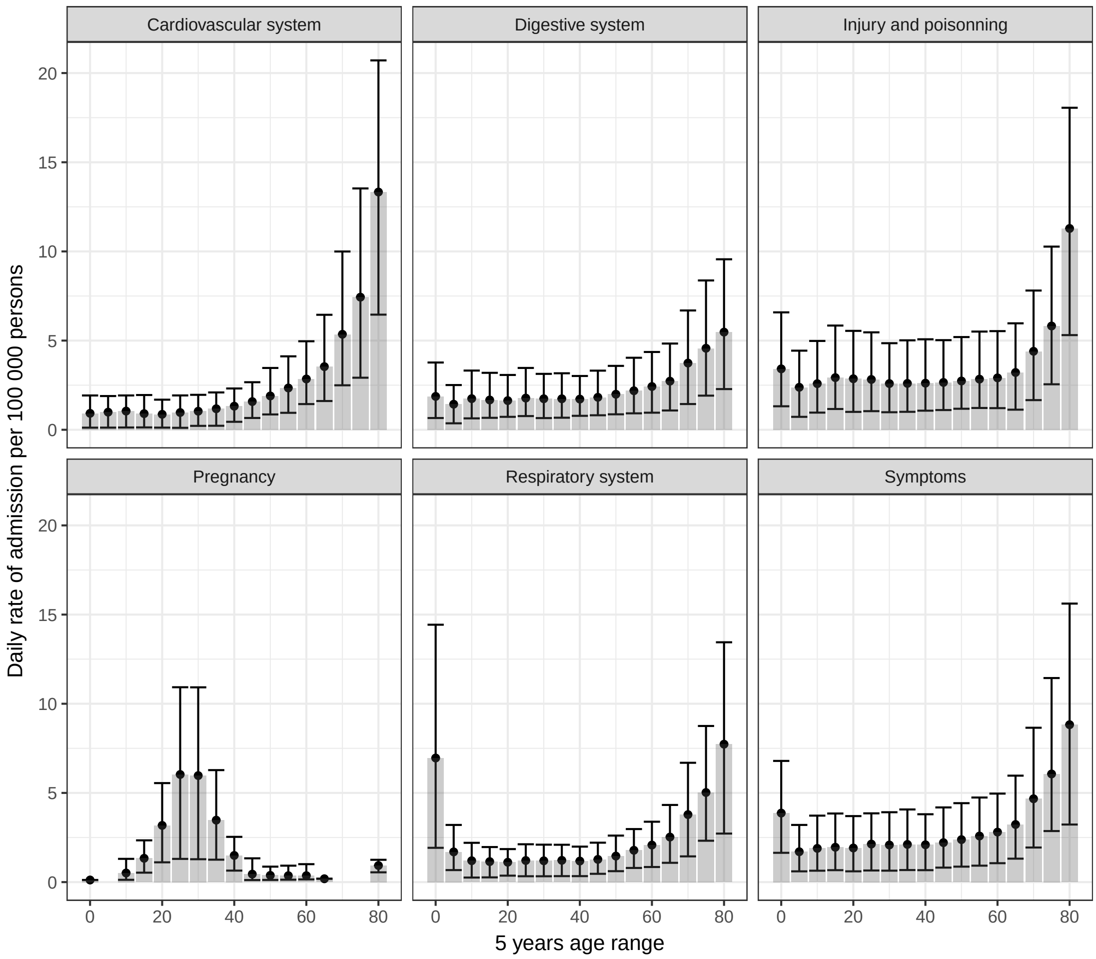

Figure 5: The 6 most important pathologies in terms of average daily rate of admissions: rate of admissions per 100 000 persons of a given 5-year age bracket (e.g. 0-4, 5-9, to over 80). The error bars show the first and last decile of observed daily admissions. Symptoms are emergency admissions where no final diagnosis was reached: the doctors described the symptoms rather than a particular disease. *Source: ATIH*

4 Empirical strategy

We use a difference-in-differences strategy where the control group rotates among urban area for the 91 events used in the estimation. We use the exact same specification for each output variable available on a daily basis : congestion, pollutant concentrations, emergency hospital admissions. For date t, city c, day-of-the-week d, month-of-the-year m, and output variable $y_{c,t}$ the specification writes

$$y_{c,t} = \sum_{k=-1}^{k=2} \alpha_k S_{c,t+k} + \beta X_{c,t} + \gamma_{d,c} + \delta_{c,m} + \nu_t + \epsilon_{c,t} \quad (1)$$

where $S_{c,t}$ is a dummy that equals one when a public transport strike is on going in city c at date t. We also include two leads and one lag of $S_{c,t}$ so as to explore potential anticipation and delayed effects. To implement a difference-in-difference, we include city and date[25] fixed effects. Thus, our identification strategy compares, within a given date, cities where a strike is ongoing with cities where there is not. $X_{c,t}$ are weather controls, including temperature, rainfall, humidity, insulation and vapor pressure (for these five variables, we include the day-level mean in a polynomial of order 2). So as to reflect that air pollutants' concentrations are highly dependent on the wind direction and strength, and that the dispersion and the arrival of pollutants depends on city specific topography, we include a polynomial of order 2 in wind speed interacted with wind direction at four times in the day (midnight, 6a.m., noon, 6p.m.), and interacted with cities dummies. We also include a daily dummy for the presence of snow (at least very relevant for traffic outcomes but not only). Finally, we include a day-of-the-week and month-of-the-year interacted with the city to allow for local patterns. Standard errors are clustered at the city level, following Bertrand, Duflo and Mullainathan (2004).

At the hourly level, we adopt for traffic a very similar equation: we adapt the set of fixed effects by including hour-of-the-day and monitoring station fixed effects (when a station records two opposite directions, there is a fixed effect per direction) on top of date dummies, and day-of-the-week and month-of-the-year interacted with the city fixed effects. The weather covariates are taken hourly.[26] We take into account the variable number of monitoring stations per city by weighting this regression by the inverse of the number of observations in each city.

Finally, depending on air pollutants, concentrations are not always observed, but missingness is unrelated to the event of the strike[27].

[25] We consider each date in the scope from 1 January 2010 to 31 December 2015. To deal with the high number of fixed effects, we use the reghdfe procedure by Correia (2016).

[26] Keeping the weather covariates at their daily mean instead does not change the results.

[27] See Table 14 in appendix

5 Results

We present first our results regarding congestion, traffic and air pollution. Second, we review the results on health.

5.1 Congestion, traffic and air pollution

We present in Table 6 the estimates from Equation 1 regarding extra-travel time incurred by drivers. On a strike day, we observe a congestion level superior by about 7.5% compared to a day without strike. Whereas it might seem small, it is likely that the congestion impact is much higher in morning hours. Indeed, hourly traffic estimates in Figure 6 shows that car flow increase by more than 20% vehicles per monitoring stations but that this effect is very localized in the first commuting hours from 8 to 10 a.m..[28] Between 8 and 9 a.m., there are 300 more vehicles passing by monitoring stations than on a regular day. Even though this effect appears quite strong in morning hours, it is not significant the rest of the day, whereas we might expect a return peak in evening hours. Contrary to congestion which is measured so as to reflect the whole network, traffic data is measured punctually on main axis. In the evening return, some vehicles coming back may divert from the main roads, especially if drivers are learning from the morning difficulties.[29]

As a whole, we observe evidence that traffic is perturbed on a strike day: flow of vehicles passing the main axis increases substantially in the morning peak hours and congestion increases significantly. In what follows, we examine how, with more vehicles and congested conditions, the mix of pollutants is modified in the days following the strike. We focus on the dynamics during several days, as what is suspected to be modified is air pollutants emissions (flows), whereas what we measure is the air pollutants concentration (stocks).

Table 7 shows the results when estimating Equation 1 by air pollutant for the three day-level quartiles.[30] The first quartile of carbon monoxide increases by 30 $\mu g/m^{-3}$ on the strike day which is 0.14 of the standard deviation of CO concentration and represents a 10.1% increase. This is in line with the increase of congestion and traffic on that day, and with the fact that CO car emissions are particularly favored by low speed (Ntziachristos and Samaras (2000)). A fleet composition effect may also be at stake. Drivers are more likely to use an older

[28]See Figure 19 in appendix to compare these estimates with those of the adjacent days.

[29]Another explanation may be related to cancellation of trips that are not work-related and that can be postponed: Figure 8 in appendix shows the asymmetry of traffic flow suggesting that evening trips are not only returns from work.

[30]That is, in linear regression 1, the outcomes are the quartile of air pollutant concentrations over the 24 daily measures.

Table 6: Log daily congestion level around strike day S. Estimates from Equation (1)

	Log Congestion
S-1	0.00477
	(0.0242)
S	0.0663***
	(0.0199)
S+1	0.0309
	(0.0369)
S+2	0.00426
	(0.0302)
Observations	13541
R^2	0.920

Notes: All regressions are run at the date-city level as in Equation (1), and include date fixed effects and city-specific day-of-week, week-of-year fixed effects, and weather controls. Number of 1-day strikes used in estimation sample: 91. Cluster-robust standard errors (robust to the presence of heteroskedasticity) are in parentheses. $*$: p-value < 0.1,$**$: p-value < 0.05,$***$: p-value < 0.01

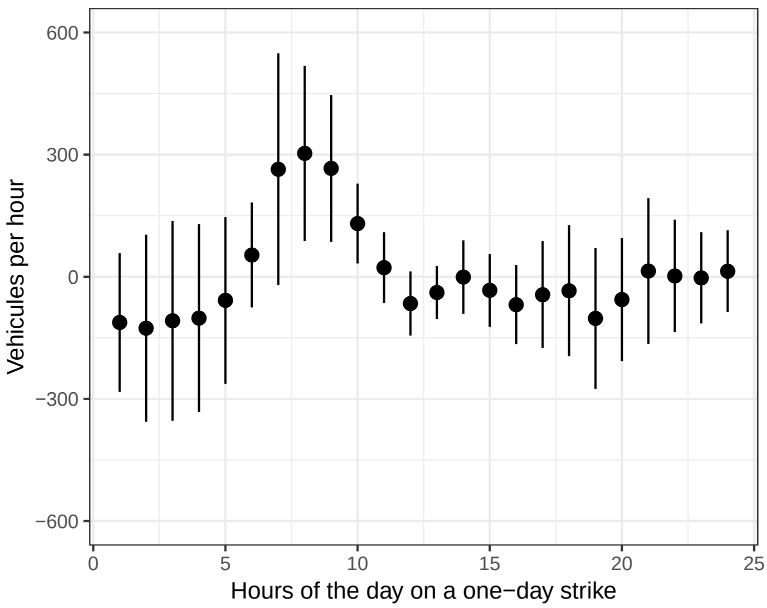

Figure 6: Traffic flow on a one-day strike. Estimates from the hourly regression described in section 3 are reported with their 95% confidence intervals

car that they do not use usually on a strike day. And older cars emit more CO, as European norms have been more and more binding over the last two decades.

Particulate matters increase mainly on the following day of the strike. This result is found for the three quartiles. The three quartile of PM2.5 increase respectively by 10.1%, 12.3% and 12.8%. The first and the third quartile PM10 increase respectively by 7.7% and 8.2%. The delay is rather in line with what Figure 4 suggests on a regular week: particulate matters are subject to an accumulation process, with yesterday's emitted particles being influential on today's concentration. PM2.5 first quartile significantly increases two days after the day, whereas PM10 first quartile does not. This may be explained by the fact that the biggest particulate matters fall more rapidly to the ground, due to their weight.

The daily median of O3 increases two day after the strike. It represents a 4.5% increase. These dynamics are in line with the fact that ozone is a secondary pollutant, formed from precursor pollutants, such as carbon monoxide, non-methane volatile organic compounds and nitrogen oxides, all of them emitted by cars.

Cars emit nitrogen monoxide (NO). Therefore if there are more cars driving at a given speed, the equilibrium $NO2 + O2 \leftrightarrow NO + O3$ tends to shift to the left (a higher NO2 concentration, not observed here). Ozone increases two days after the strike. This indirectly suggests the consumption of the ozone precursors observed here, NO2 and CO, in the reaction producing O3, mitigating their accumulation. The reason why we do not observe an increase in NO2 could also be related to the relation between speed and pollutant emissions. In general, pollutant emissions as a function of speed are U-shaped with a minimum specific to each sources which may range from 40km/h to 80km/h.[31]

Finally, we observe a decrease of the first quartile of SO2 by 36%, emitted mainly by industries, on the day after the strike. We can conjecture that the strike affects plant operations, as not every worker may go to work. This could in turn result in a lower SO2 concentration the following day.

[31]A difference between CO and NO2 is related to how their emissions depend on speed. The emissions of nitrogen oxides per unit of distance are a relatively flat U-shaped function of speed (compared to the other precursor), with a minimum at 40km/h (for example decreasing speed from 80 to 40 km/h leads to a decrease in NO2 emissions, and reducing speed below leads to a relatively flat increase), while CO emissions are strongly favored by low speed and increase strongly and constantly when speed is reduced from 80 to below 5 km/h, see (Ntziachristos and Samaras (2000)). On the speed range below 80km/h, the combined effects of more cars on the road and lower speed is therefore unambiguous for CO, but not for NOx.

Table 7: Air pollution: daily estimates around a one-day strike

	Pollutant concentration ($\mu m/m^-3$)					
	Day first quartile					
	CO	PM2.5	PM10	NO2	O3	SO2
S-1	11.36	0.277	0.328	2.280	-1.546	-0.229
	(21.68)	(0.567)	(0.835)	(1.533)	(1.710)	(0.174)
S	30.37**	0.963	1.216	0.556	0.889	-0.224
	(12.66)	(1.031)	(1.155)	(1.236)	(1.537)	(0.132)
S+1	18.49	1.353**	1.536*	0.216	-0.241	-0.369**
	(18.29)	(0.442)	(0.695)	(1.707)	(1.736)	(0.139)
S+2	-9.072	1.666*	1.219	-0.801	0.531	-0.184
	(15.09)	(0.862)	(0.990)	(0.529)	(1.247)	(0.110)
Mean dep. var.	296.5	12.9	19.9	27.7	35.1	0.5
Observations	10605	12458	11856	12579	13052	11895
R^2	0.753	0.768	0.745	0.850	0.848	0.437
	Day median					
	CO	PM2.5	PM10	NO2	O3	SO2
S-1	6.478	0.834	-0.590	0.758	-1.623	-0.287
	(26.11)	(0.832)	(0.839)	(1.337)	(1.548)	(0.247)
S	15.63	0.685	0.552	-0.380	-0.0839	0.0609
	(20.62)	(1.092)	(1.595)	(1.054)	(1.363)	(0.343)
S+1	35.33	1.977**	1.433	0.625	0.348	-0.216
	(24.21)	(0.708)	(0.782)	(1.076)	(1.598)	(0.251)
S+2	-16.09	1.753	1.807	-0.935	2.759*	-0.269
	(22.14)	(1.288)	(1.799)	(1.000)	(1.449)	(0.180)
Mean dep. var.	417.2	16.1	25.4	38.7	48.6	0.9
Observations	10605	12458	11856	12579	13052	11895
R^2	0.807	0.783	0.771	0.876	0.884	0.462
	Day third quartile					
	CO	PM2.5	PM10	NO2	O3	SO2
S-1	-8.614	-0.0399	-1.973	1.815	1.464	-0.254
	(34.71)	(1.032)	(1.219)	(1.741)	(1.328)	(0.304)
S	18.27	-0.610	-0.582	-2.333	0.221	0.0250
	(23.85)	(1.306)	(2.038)	(1.668)	(1.292)	(0.394)
S+ 1	30.18	2.547**	2.578***	0.0792	1.993	0.00792
	(38.22)	(0.926)	(0.766)	(1.267)	(2.062)	(0.336)
S+2	-18.84	1.508	2.564	0.0470	2.811**	-0.422
	(27.90)	(1.268)	(2.180)	(1.747)	(1.115)	(0.340)
Mean dep. var.	543.7	20	31.2	49.7	62.7	1.5
Observations	10605	12458	11856	12579	13052	11895
R^2	0.816	0.784	0.770	0.869	0.901	0.494

Notes: All regressions are run at the date-city level as in Equation (1), and include date fixed effects and city-specific day-of-week, week-of-year fixed effects, and weather controls. Number of 1-day strikes used in estimation sample respectively by pollutants: 57, 65, 55, 55, 62, 59 (different from 91 due to missing values in the outcomes). Cluster-robust standard errors (robust to the presence of heteroskedasticity) are in parentheses. $*$: p-value < 0.1,$**$: p-value < 0.05,$***$: p-value < 0.01

5.2 Health effects

Congestion is higher on strike days and traffic-related air pollutants are higher than usual from that day up to two days after. We now focus on emergency hospital admissions. Due to more air pollution, respiratory diseases are expected to increase. But some respiratory diseases are also caused by infectious agents (mainly viruses). If contagion decreases due to lower interpersonal contact on the strike days, the net effect on respiratory diseases is unclear one or two days after the strike. We show evidence of the existence of these two channels through which health is affected. Table 8 presents our main results on emergency hospital admissions, for respiratory diseases, and for diseases due to an infectious agent with a short incubation period. Influenza and pneumonia belong to both categories. They are respiratory diseases suspected to be favoured by air pollution and are caused by a infectious agent (a virus for influenza, a virus or a bacteria for pneumonia). Consequently, they can be subject to both an air pollution and a contagion channel with opposite effects. As for gastroenteritis, it is not favored by air pollution and only a contagion channel can be at stake.

Evidence of air pollution impact

Here again, the observed impact is not reduced to the very day of the strike. The first column shows the admissions for abnormalities of breathing, that is persons reporting difficulties in breathing for whom no diagnosis was reached. The day after the strike, there is an increase by about 20% (+0.018 admissions per 100 000 persons in the urban area) of abnormalities of breathing, significant at the 5% level. The second column report admissions for the whole set of diseases of the respiratory system. We observe no significant effects. First, we might want to restrict to pathologies the most likely to be sensitive at short-term to air pollution. When we restrict to acute (as opposed to chronic) diseases that are specific to the upper respiratory system on the third column (i.e. nose, pharynx and larynx who are among other things responsible of the filtering and cleaning of the air we inhale) we evidence an increase by about 38% on the strike day. Second, as argued before, on a strike day people may be less likely to interact and fall ill due to viral causes the following days. As for influenza, the incubation period is very short (from one to four days, with an average at two days) and this channel may lead to underestimate the impact of the air pollution shock triggered by the strike. The fourth column of Table 8 shows that it is indeed the case: on the two days following a strike day, emergency admissions for influenza and pneumonia are lower than usual by respectively 14 and 18%.[32]

For influenza, the contagion channel is of larger magnitude than the air pollution channel. For acute disease of the upper respiratory system, on the very day of the strike, only the the air pollution channel is at work. Afterwards, we may underestimate the delayed air pollution detrimental impact here estimated to be non significant, as pathologies in this category are also subject to infectious agent (e.g. pharyngitis). As far as abnormalities of breathing are concerned, the air pollution channel prevail on the contagion channel, should the second one exist.

On Table 9, we focus on acute respiratory diseases to study the impact by age group.[33] Two facts emerge. First, the detrimental health impact attributable to air pollution is borne by children. Second, this effect is large, although the marginal increase in air pollution is relatively weak and representative of small variation as opposed to an air pollution peak. On a regular day, a city with 100 000 children between 0 and 4 sees 0.8 admissions, with a standard deviation of 1.2. On a day following a strike, the same city sees on average 0.4 admissions on top. For the 5 to 14 years-old, the impact is almost of the same magnitude than the baseline mean: + 0.07 admissions per 100 000 5-to-14-year-old children.

Evidence of viral spread

To investigate further the contagion channel, we focus on a disease caused by an infectious agent, but which is not favoured by air pollution : gastroenteritis. The last columns of Table 8 show that there are 19% less admissions two days after the strike compared to a regular day.

We refine results by age group for the two short incubation period diseases. For influenza and pneumonia, the reaction is faster for young children than for teenagers and old people. For gastroenteritis, only the central age group (15-59) is affected, whereas this category was not affected for influenza and pneumonia. Overall, observed delays are in line with what is medically known about average incubation periods of these diseases, which are between one and two days. Taking both infectious pathologies together, decreases are pervasive across age groups and compatible with incubation periods by viruses. For influenza in particular, school-age children are most significantly affected in line with past literature.

[32] All respiratory diseases (ICD-10 J00-J99) can be split in acute diseases of the upper respiratory system (ICD-10 J00-J06), influenza and pneumonia (ICD-10 J09-J18), and the rest (ICD-10 J20-J99): for the last, not shown here, we provide the estimation in appendix where we find no effects.

[33] Table 16 and 17 complement the results by age groups.

Table 8: Emergency hospital admissions for respiratory diseases and diseases with infectious causes (short incubation period)

	Rate of admission per 100 000 inhabitants				
				Infectious diseases	
	Related to air pollution				Unrelated
		Diseases of the respiratory system			
	Abnormalities of breathing	All pathologies	Acute diseases of the upper respiratory system	Influenza and pneumonia	Gastro-enteritis
IDC10	R06	J00-J99	J00-J06	J09-J18	A08-A09
S-1	0.00438	0.00719	0.0204	0.0134	0.00345
	(0.0142)	(0.0790)	(0.0237)	(0.0289)	(0.0198)
S	0.00972	0.0668	0.0309***	-0.0193	0.0226
	(0.0117)	(0.0614)	(0.00448)	(0.0326)	(0.0195)
S+1	0.0183**	-0.0917	0.0175	-0.0637*	0.00867
	(0.00741)	(0.0641)	(0.0116)	(0.0303)	(0.0204)
S+2	0.00386	-0.0828	0.000126	-0.0802*	-0.0406*
	(0.0129)	(0.0893)	(0.0111)	(0.0367)	(0.0220)
Mean dep. var.	0.08	1.42	0.08	0.44	0.24
Obs.	13547	13547	13547	13547	13547
R^2	0.410	0.775	0.429	0.602	0.657

Notes: All regressions are run at the date-city level as in Equation (1), and include date fixed effects and city-specific day-of-week, week-of-year fixed effects, and weather controls. 91 one-day strikes are used (no missing values in the outcome). Cluster-robust standard errors (robust to the presence of heteroskedasticity) are in parentheses. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table 9: Emergency admissions for acute diseases of the upper respiratory system, by age group

	Rate of admission per 100 000 inhabitants of the corresponding age bracket			
	Acute diseases of the upper respiratory system ICD10 J00-J06			
Age group	0-4	5-14	15-59	Over 60
S-1	0.306	0.00337	0.00227	0.00267
	(0.316)	(0.0341)	(0.0125)	(0.00658)
S	0.386***	0.0205	0.00694	-0.0000905
	(0.104)	(0.0400)	(0.00665)	(0.00662)
S+1	0.189	0.0714**	0.00136	-0.00953
	(0.145)	(0.0278)	(0.00838)	(0.00642)
S+2	0.0859	0.00846	-0.00903	0.00143
	(0.188)	(0.0280)	(0.0146)	(0.0117)
Mean dep. var.	0.82	0.09	0.03	0.01
Observations	13547	13547	13547	13547
R^2	0.422	0.277	0.265	0.235

Notes: All regressions are run at the date-city level as in Equation (1), and include date fixed effects and city-specific day-of-week, week-of-year fixed effects, and weather controls. 91 one-day strikes are used (no missing values in the outcome). Cluster-robust standard errors (robust to the presence of heteroskedasticity) are in parentheses. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table 10: Diseases with viral causes, suspected to be exacerbated by air pollution (influenza) or not (gastroenteritis)

	Rate of admission per 100 000 inhabitants of the corresponding age bracket			
	Influenza and pneumonia ICD10 J09-J18			
	0-4	5-14	15-59	Over 60
S- 1	-0.0191	0.00673	0.00402	0.0454
	(0.0995)	(0.0648)	(0.0225)	(0.0906)
S	0.0485	0.0241	-0.0140	-0.0700
	(0.165)	(0.0745)	(0.0263)	(0.110)
S+1	-0.139*	-0.00835	-0.0178	-0.209
	(0.0663)	(0.0324)	(0.0239)	(0.125)
S+2	-0.210	-0.0835**	-0.0228	-0.203*
	(0.159)	(0.0368)	(0.0171)	(0.105)
Mean dep. var.	0.76	0.12	0.15	1.32
Observations	13547	13547	13547	13547
R^2	0.446	0.297	0.365	0.497
	Gastroenteritis ICD10 A08-A09			
	0-4	5-14	15-59	Over 60
S-1	-0.0180	0.00459	0.0140	-0.0127
	(0.212)	(0.0457)	(0.0189)	(0.0474)
S	0.309	-0.00877	0.0116	-0.000863
	(0.297)	(0.0409)	(0.0217)	(0.0411)
S+1	0.231	-0.00557	-0.0299**	0.0530
	(0.281)	(0.0330)	(0.0113)	(0.0538)
S+2	-0.436	0.0369	-0.0223	-0.0400
	(0.242)	(0.0492)	(0.0169)	(0.0550)
Mean dep. var.	2.41	0.17	0.07	0.15
Observations	13547	13547	13547	13547
R^2	0.670	0.333	0.294	0.273

Notes: All regressions are run at the date-city level as in Equation (1), and include date fixed effects and city-specific day-of-week, week-of-year fixed effects, and weather controls. 91 one-day strikes are used (no missing values in the outcome). Cluster-robust standard errors (robust to the presence of heteroskedasticity) are in parentheses. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

6 Robustness

6.1 Falsification tests with unrelated emergency admissions

As a robustness check, we verify that no effect is found for digestive diseases and pregnancy that are a priori unrelated to the channels we focus on and are among the most frequent admissions in emergency.[34] As expected, we find no significant effects of the strike neither on the same day nor on adjacent days. Another criticism that these falsification tests weaken is the idea that stress caused by a strike event fragilize health and therefore play a role in emergency admissions. The digestive system is sensitive to stress through a number of nervous connections (Bhatia and Tandon (2005)) while we see no impact on emergency admissions for this pathology.

Table 11: Falsification tests with unrelated emergency admissions

	Rate of admission per 100 000 inhabitants of the corresponding age bracket					
	Pregnancy O00-O99	Diseases of the digestive system K00-K93				
	All	All	0-4	5-14	15-59	Over 60
S-1	0.0532	-0.0732	-0.261	-0.220	-0.164	0.260
	(0.0848)	(0.128)	(0.425)	(0.236)	(0.135)	(0.321)
S	0.0581	-0.00767	0.334	0.0457	-0.0558	-0.0304
	(0.0564)	(0.101)	(0.383)	(0.222)	(0.125)	(0.205)
S+1	0.0388	-0.103	-0.180	-0.191	0.0195	-0.258
	(0.0339)	(0.110)	(0.336)	(0.297)	(0.133)	(0.386)
S+2	0.0273	-0.118	-0.205	-0.315	-0.0409	-0.245
	(0.0395)	(0.141)	(0.381)	(0.240)	(0.154)	(0.346)
Mean dep. var.	0.9	2.83	1.87	1.53	2.21	5.57
Observations	13547	13547	13547	13547	13547	13547
R^2	0.894	0.552	0.356	0.347	0.427	0.396

Notes: All regressions are run at the date-city level as in Equation (1), and include date fixed effects and city-specific day-of-week, week-of-year fixed effects, and weather controls. 91 one-day strikes are used (no missing values in the outcome). Cluster-robust standard errors (robust to the presence of heteroskedasticity) are in parentheses. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

[34] We found no significant impact on other leading causes of emergency admissions that have not been chosen for falsification tests as they could be affected (injury and cardiovascular diseases, see Table 15 in appendix).

6.2 Do strikers favor days with particular events?

Our empirical strategy would be endangered if strikes coincide with particular events which are by themselves responsible for a higher traffic. By controlling for date fixed effects, we allow strikes to be correlated to national shocks (bank holidays, world cup games etc..). However, we do not control for strikers targeting dates in their city when a particular event takes place, for instance to cause maximal disruption. If the strike has a large impact on users and beyond, it might increase strikers bargaining power as the transport firm may wish a fast conflict resolution. But it may not be a good strategy as it may influence negatively public opinion. To check indirectly whether strikes coincide with particular events, we verify that hotel occupancy is not particularly high on strikes days (or the converse, that hotel occupancy is affected by the strike). We use the responses at the *Enquête mensuelle sur la fréquentation touristique* which corresponds to the number of rooms available and occupied at each date of the years 2010-2015, that we aggregate at the urban area level to compute an occupancy rate (all occupied rooms over all available rooms). As shown by Table 12, the occupancy rate is not modified on the event of a strike. This suggests that although strikes may not be random, they are most probably not happening concomitantly with mass events conditional on our extensive set of control variables and fixed effects.

Table 12: Hotel occupancy around one-day strikes

	Occupancy rate
S-1	0.0127
	(0.00960)
S	0.0174
	(0.0143)
S+1	0.000448
	(0.0115)
S+2	-0.00637
	(0.0105)
Mean dep. var.	0.63
Observations	13547
R^2	0.923

Notes: All regressions are run at the date-city level as in Equation (1), and include date fixed effects and city-specific day-of-week, week-of-year fixed effects, and weather controls. 91 one-day strikes are used (no missing values in the outcome). Cluster-robust standard errors (robust to the presence of heteroskedasticity) are in parentheses. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

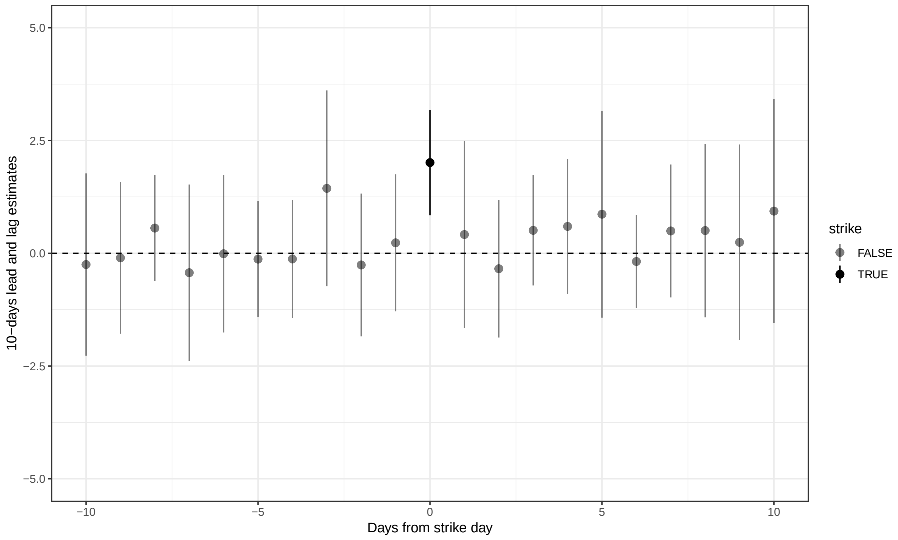

Figure 7: Pre and post trends. Estimates from Equation (1) modified to includes 10 leads and 10 lags of the strike. 91 one-day strikes are used.

6.3 Pre and post trends

In this section, we check on a longer period whether cities are indeed comparable before and after the strike event. This legitimizes a difference-in-difference strategy. Figure 7 shows the daily congestion estimates from the regression (1) on a symmetric window of 10 days. The strike day estimate is the highest coefficient in absolute value and no other coefficient is significant at the 5% level. Figure 19 in appendix shows the hourly estimates from the traffic equation when considering a 5 days window around the strike event. Here as well, the coefficient at 8 a.m. is the highest in absolute value. If some other coefficients are significant at the 5% level, very few are significant both in log and in level (as opposed to the morning peak hours on a strike day) and most are at night time (when traffic is somewhat less stable from one day to the other). We could use a p-value correction for multiple hypothesis testing because we here test 21 and 120 coefficients significances thus false positive are very likely. Because more conservative on detectable effects, it would lead to strengthen how the traffic coefficients on a strike day stand out.[35]

6.4 Sample

Finally, we verify what happens to our estimates when dropping particular periods marked by social movements (the year 2010, September months in the 6 years)

[35]Such a procedure applied to our main results (e.g., dividing p-values by 3 in a Bonferroni manner, because we search for strike impact on 3 days) would lead to consider as insignificant the effects found for the contagion channel. But even from this conservative perspective (control of type I error at the type II error expense), the argument for air pollution channel through congestion, particulate matters and acute respiratory diseases estimates, will remain.

when a high number of strike events occur, or when we drop the largest urban area of the sample (Paris).

Table 13 provides the main estimates when modifying the sample.[36] First, across all samples, estimates for both daily congestion level and emergency admissions for acute respiratory diseases are very similar. Dropping Paris reinforces the contagion channel: in particular the influenza admission drop two days after the strike, which was only significant at the 10% level for the whole population, becomes highly significant (1%). Air pollution effects are however less discernible, with significance for PM2.5 increases the day following the strike dropping at 10%. When dropping 2010, on the converse, air pollution effects are reinforced. CO median concentration which was not affected in the baseline (only the first quartile was) is significantly higher on a strike day happening between 2011 and 2015 than on a regular day of the same period. PM2.5 median concentration is significantly affected not only at strike + 1 day but also at strike + 2 days. Over this period however, emergency admissions for abnormalities of breathing seem unaffected. Finally, dropping all September months does not affect the results, except air pollutions that is somewhat less significant for CO and PM2.5. However, the effect for PM10 and O3 are not modified compared to using the baseline sample (Table 19). As a whole, these robustness exercises suggest that our conclusions are not driven by a particular city or a particular period.

[36] Tables 18,19,20 in appendix show all estimates for air pollutant concentrations.

Table 13: Robustness when dropping the largest urban area (Paris), dropping periods marked by social movement (year 2010, month September) for congestion and health outcomes.

	Congestion	*Pollutants median concentration*		*Emergency admissions per 100 000 inhabitants*					
				All				Children (0-4)	
Without …	Log daily congestion	CO	PM2.5	Abnorm. of breath.	Acute resp. dias.	Influenza	Diarrhea	Acute resp. dias.	Influenza
… Paris	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
S-1	0.399	-10.57	0.413	0.0118	0.0416*	0.0286	-0.00116	0.541	0.0160
	(0.941)	(28.64)	(1.150)	(0.0158)	(0.0217)	(0.0326)	(0.0283)	(0.323)	(0.149)
S	1.917**	15.42	0.976	0.00947	0.0339***	-0.0236	0.0184	0.439***	0.0668
	(0.659)	(29.13)	(1.659)	(0.0141)	(0.00499)	(0.0439)	(0.0261)	(0.117)	(0.234)
S+1	1.163	17.94	2.276*	0.0210*	0.0146	-0.0721	0.0181	0.142	-0.140
	(0.938)	(35.24)	(1.089)	(0.0105)	(0.0122)	(0.0420)	(0.0238)	(0.150)	(0.126)
S+2	-0.148	-22.30	1.651	0.0140	0.00513	-0.117***	-0.0546*	0.178	-0.345**
	(0.817)	(34.33)	(1.896)	(0.0128)	(0.0131)	(0.0275)	(0.0270)	(0.254)	(0.145)
One-day strikes	75	56	45	75	75	75	75	75	75
Obs.	12147	9317	11120	12153	12153	12153	12153	12153	12153
R^2	0.908	0.770	0.773	0.412	0.436	0.598	0.648	0.429	0.451
… 2010	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
S-1	0.393	4.633	0.807	0.00572	0.00876	0.0293	0.00863	0.0367	0.0428
	(0.712)	(31.59)	(0.809)	(0.0179)	(0.0267)	(0.0310)	(0.0185)	(0.203)	(0.163)
S	2.234***	40.39**	1.153	0.0168	0.0428***	-0.0208	0.0294	0.634**	0.0414
	(0.506)	(15.46)	(0.733)	(0.00943)	(0.0130)	(0.0439)	(0.0218)	(0.204)	(0.187)
S+1	0.938	35.40	2.186***	0.0227	0.0391*	-0.0616	0.0218	0.424	-0.150*
	(1.561)	(30.65)	(0.623)	(0.0144)	(0.0198)	(0.0463)	(0.0310)	(0.271)	(0.0752)
S+2	-0.0271	-6.353	2.034**	-0.0101	-0.00503	-0.0753	-0.0533*	0.0323	-0.380
	(0.800)	(27.39)	(0.661)	(0.0120)	(0.0130)	(0.0566)	(0.0266)	(0.192)	(0.309)
One-day strikes	38	23	25	38	38	38	38	38	38
Obs.	11369	8666	10586	11369	11369	11369	11369	11369	11369
R^2	0.923	0.829	0.793	0.466	0.447	0.623	0.672	0.443	0.471
… Sept.	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
S-1	0.0772	5.458	1.410	0.00338	0.0119	0.00142	0.0211	0.142	-0.0315
	(0.686)	(30.51)	(0.814)	(0.0145)	(0.0215)	(0.0293)	(0.0240)	(0.296)	(0.0735)
S	1.824***	15.08	1.002	0.00515	0.0278***	-0.0237	0.0242	0.369***	0.0259
	(0.461)	(21.62)	(1.196)	(0.0132)	(0.00491)	(0.0326)	(0.0237)	(0.0867)	(0.181)
S+1	0.234	44.28	1.756*	0.0171**	0.0144	-0.0685*	0.00327	0.194	-0.161*
	(0.966)	(26.42)	(0.785)	(0.00683)	(0.0103)	(0.0304)	(0.0239)	(0.142)	(0.0788)
S+2	-0.197	-20.45	1.719	0.00456	-0.0104	-0.0841*	-0.0305	-0.0824	-0.176
	(0.658)	(23.33)	(1.275)	(0.0136)	(0.0121)	(0.0375)	(0.0220)	(0.227)	(0.160)
One-day strikes	68	50	45	68	68	68	68	68	68
Obs.	11965	9400	11028	11971	11971	11971	11971	11971	11971
R^2	0.913	0.811	0.792	0.421	0.436	0.609	0.667	0.424	0.458

Notes: All regressions are run at the date-city level as in Equation (1), and include date fixed effects and city-specific day-of-week, week-of-year fixed effects, and weather controls. Cluster-robust standard errors (robust to the presence of heteroskedasticity) are in parentheses. $*$: p-value < 0.1,$**$: p-value < 0.05,$***$: p-value < 0.01

7 Conclusion

In this paper, we exploit the increase in traffic-related air pollution around a one-day public-transport strike event to evidence the detrimental effect of ambient air pollution on health. We scrutinized carefully the causal chain from the one-day strike to emergency admissions and its dynamics, as the effects are not restricted to the strike day. In particular, we evidence the existence of a second channel, contagion, linking the strike event to emergency admissions and conclude that we underestimate the delayed effect of air pollution on health. Using the variations caused by a strike has strong advantages compared to correlations studies, but also its side effects: one limit is that the delayed impact on acute respiratory diseases may be underestimated as evidence of a slower viral spread among children is shown following a strike event. Even though, we find a quite substantial lower bound of the detrimental impact of ambient air pollution on health, borne in priority by children. This study emphasizes that the short-term (within a few days) impact of ambient air pollution on health is sizeable even for moderate shocks and air pollution levels. Indeed, we find evidence of an increase of particulate matters concentration by about 10 to 12%, spread all along the following day of the event. This is comparable with the differential in particulate matters concentration between a Sunday and a Thursday, that is, it is representative of usual, non-remarkable fluctuations. We evidence that the pollution shock caused by a perturbation in public transport and an increase in traffic a given day has delayed effects, due to accumulation but also to the complex chemistry of pollution.

A lot of efforts have been done from a regulatory perspective to limit emissions of new cars and have led to a decreasing trend in main traffic-related pollutants. However, even at modern level of ambient air pollution and for moderate pollution shocks, it is still possible to find negative effects on health in a real-word quasi-experiment. Therefore, by studying a day where public transport are not available, this study indirectly shows the short-term benefit of choosing public transports against polluting alternatives and the importance in terms of dynamic health spill-overs of urban population daily transport choices.

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Appendices

A Figures

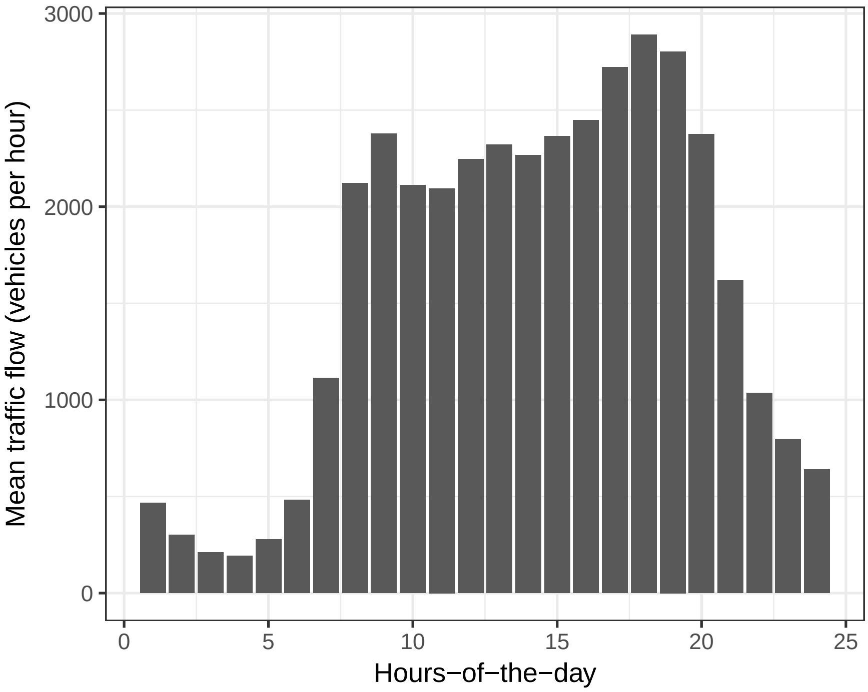


Figure 8: Mean hourly traffic over the estimation sample by hour-of-the-day

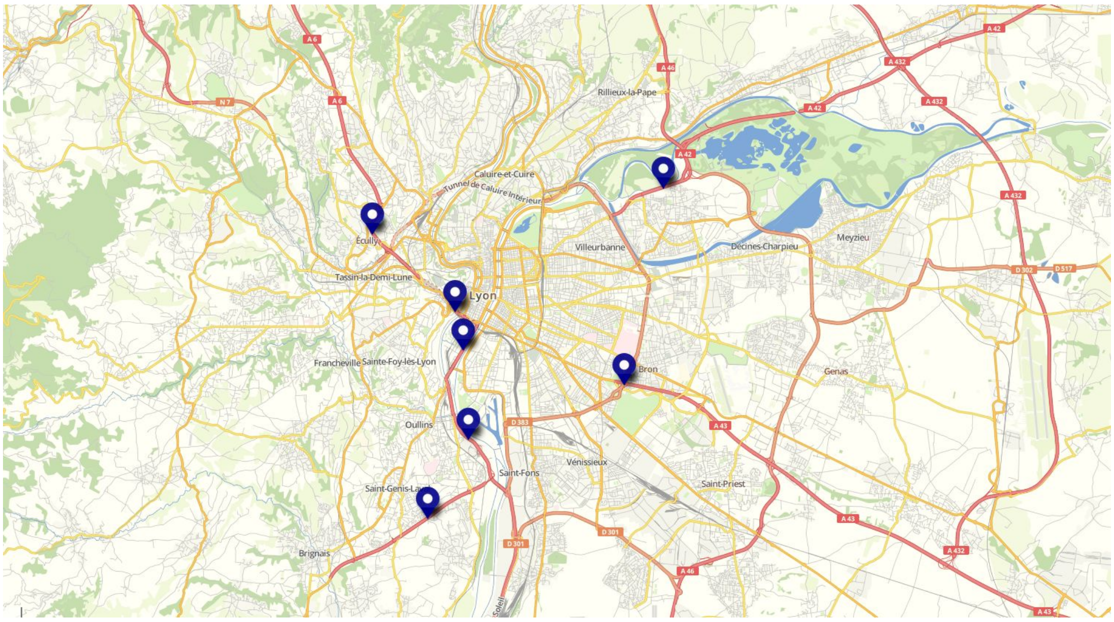


Figure 9: Traffic monitoring stations in Lyon

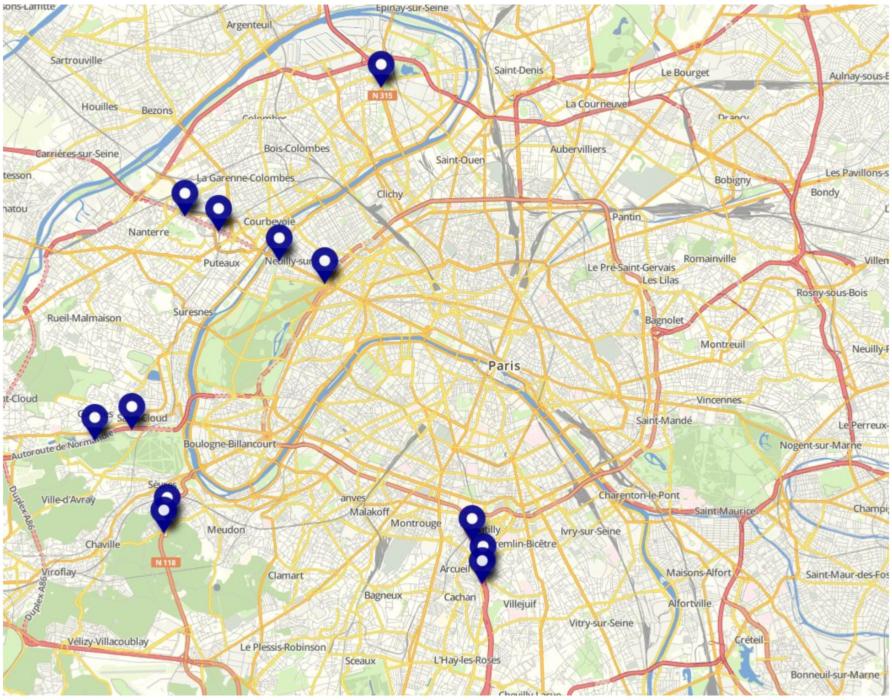


Figure 10: Traffic monitoring stations in Paris

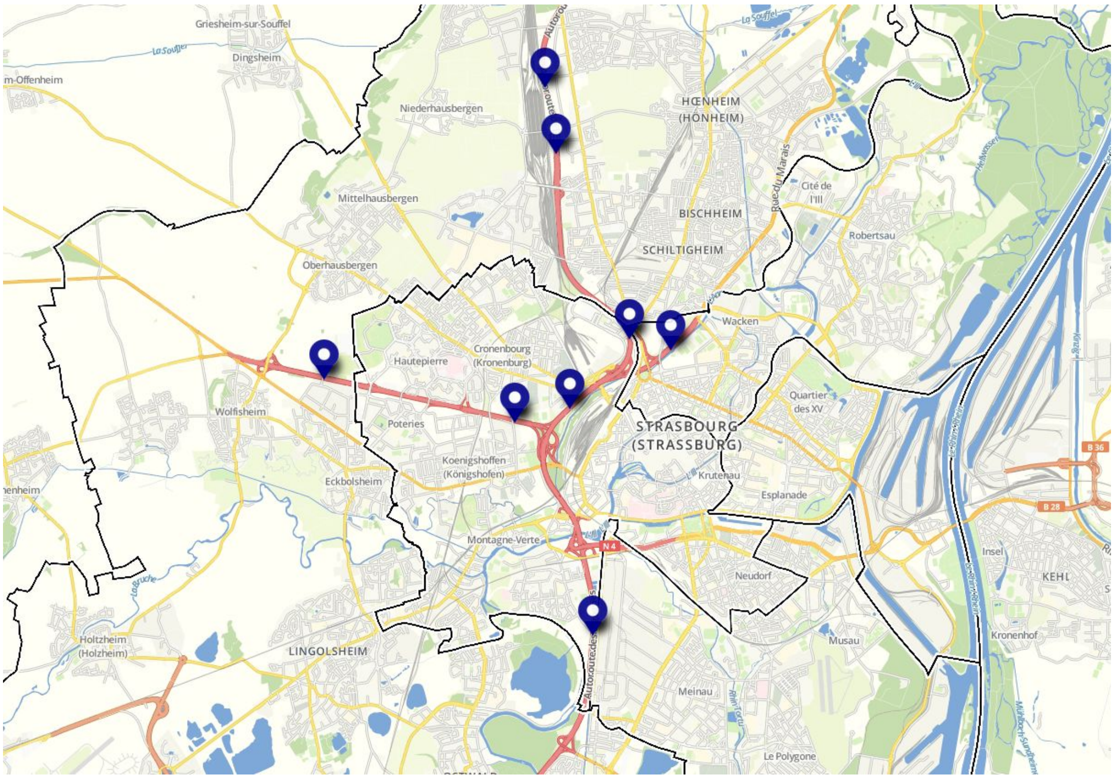


Figure 11: Traffic monitoring stations in Strasbourg

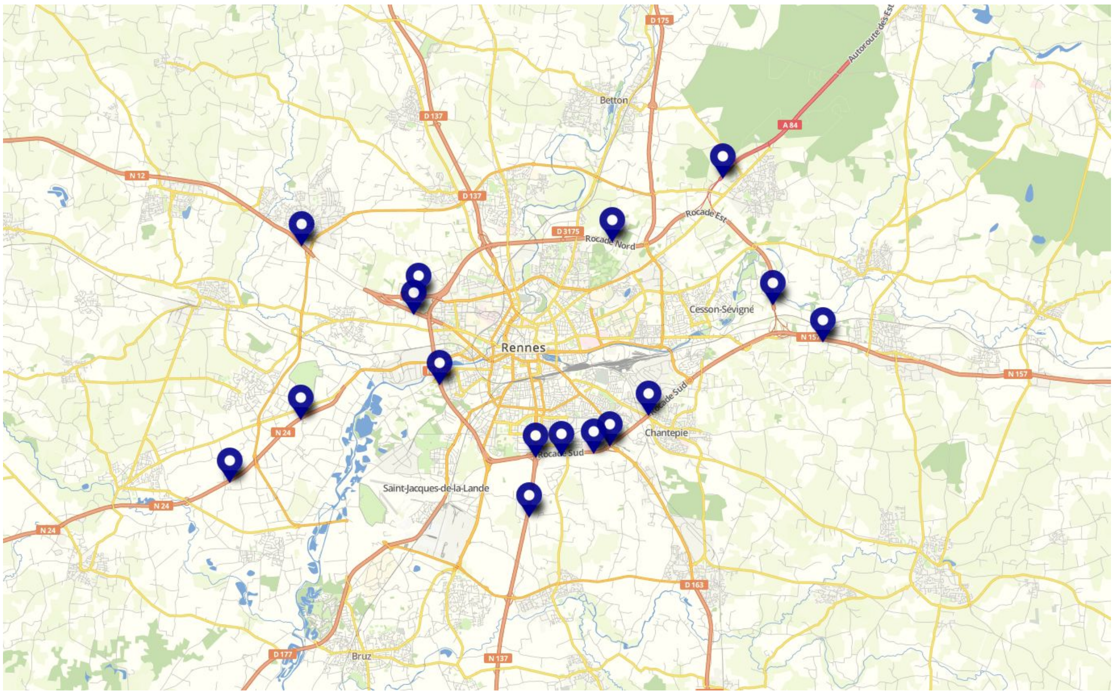


Figure 12: Traffic monitoring stations in Rennes

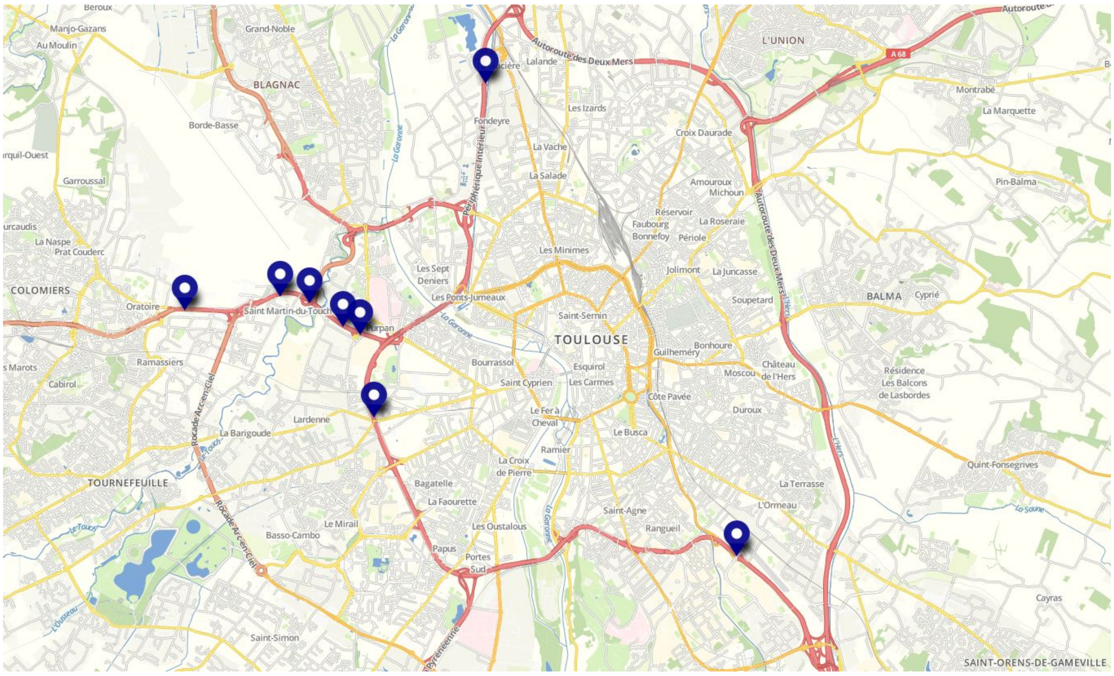


Figure 13: Traffic monitoring stations in Toulouse

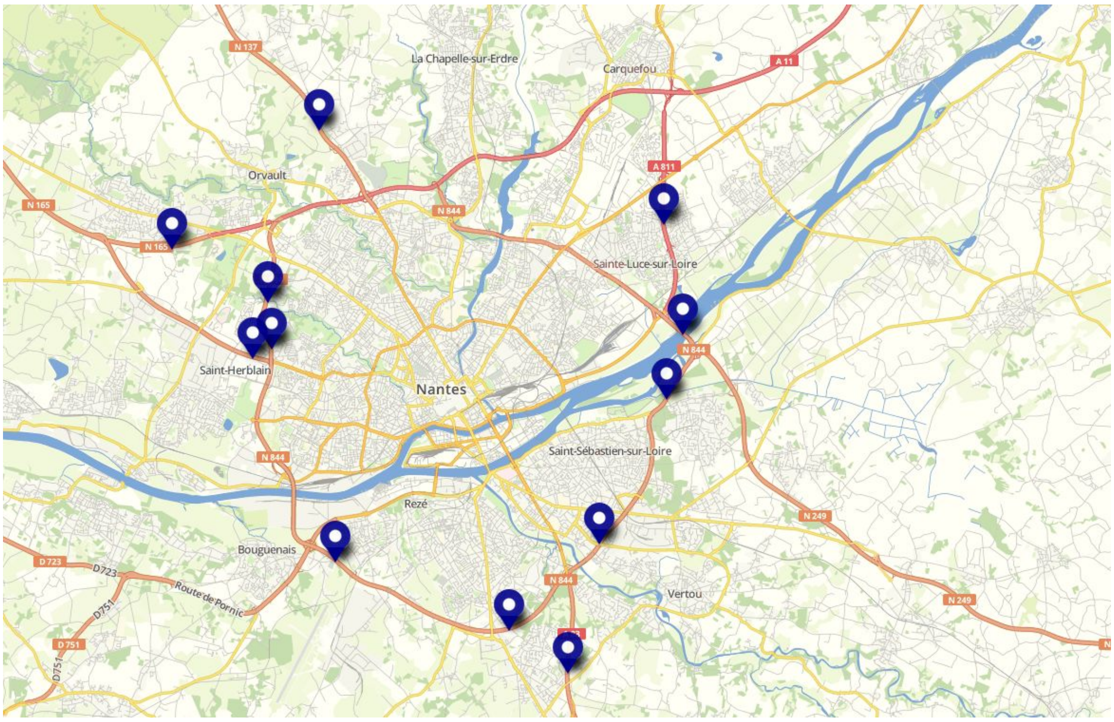


Figure 14: Traffic monitoring stations in Nantes

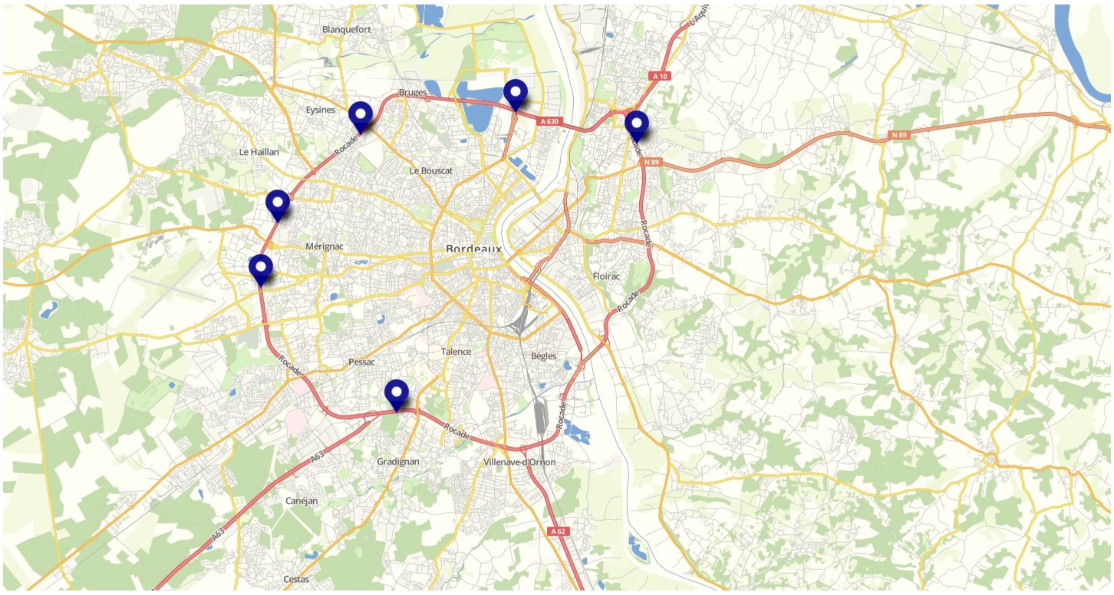


Figure 15: Traffic monitoring stations in Bordeaux

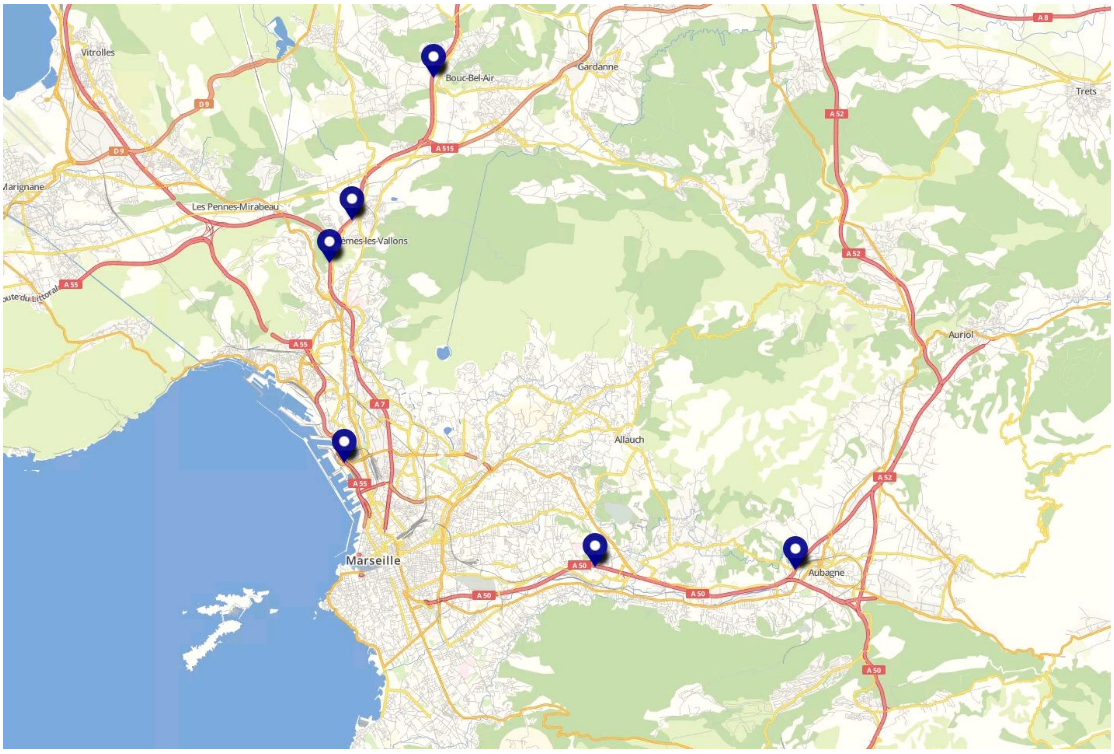


Figure 16: Traffic monitoring stations in Marseille

Figure 17: Traffic monitoring stations in Nice

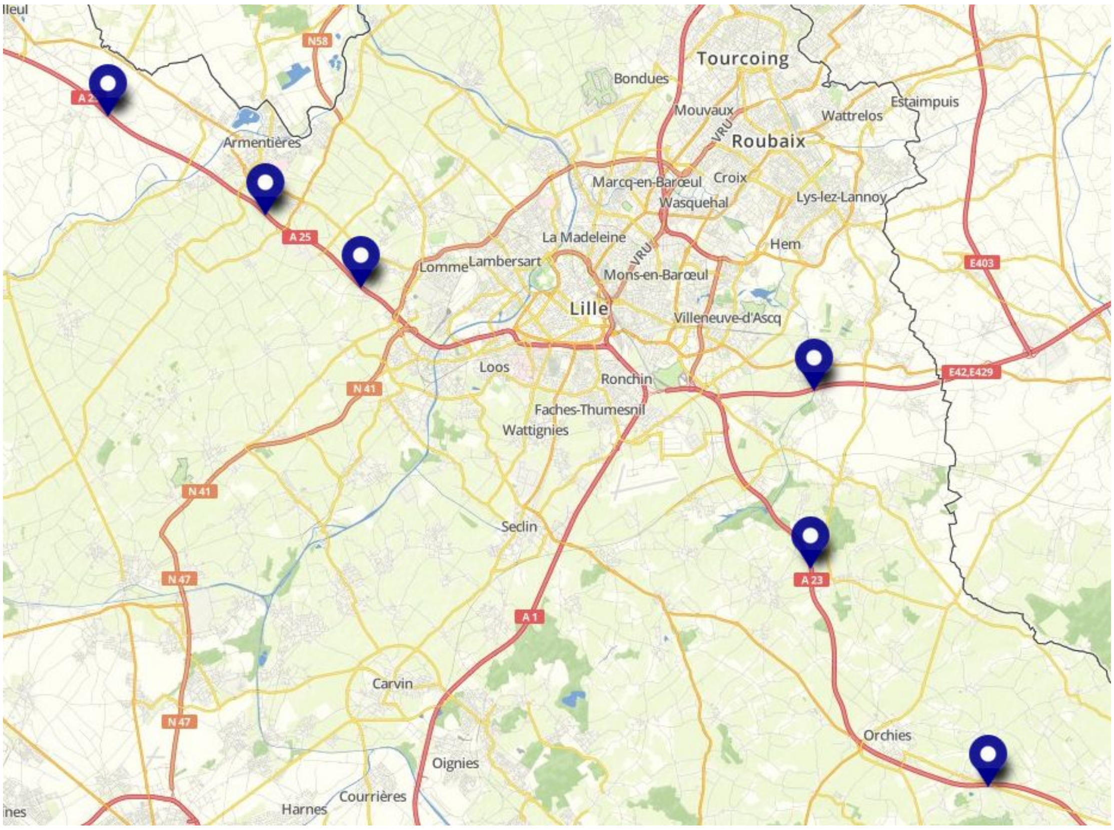
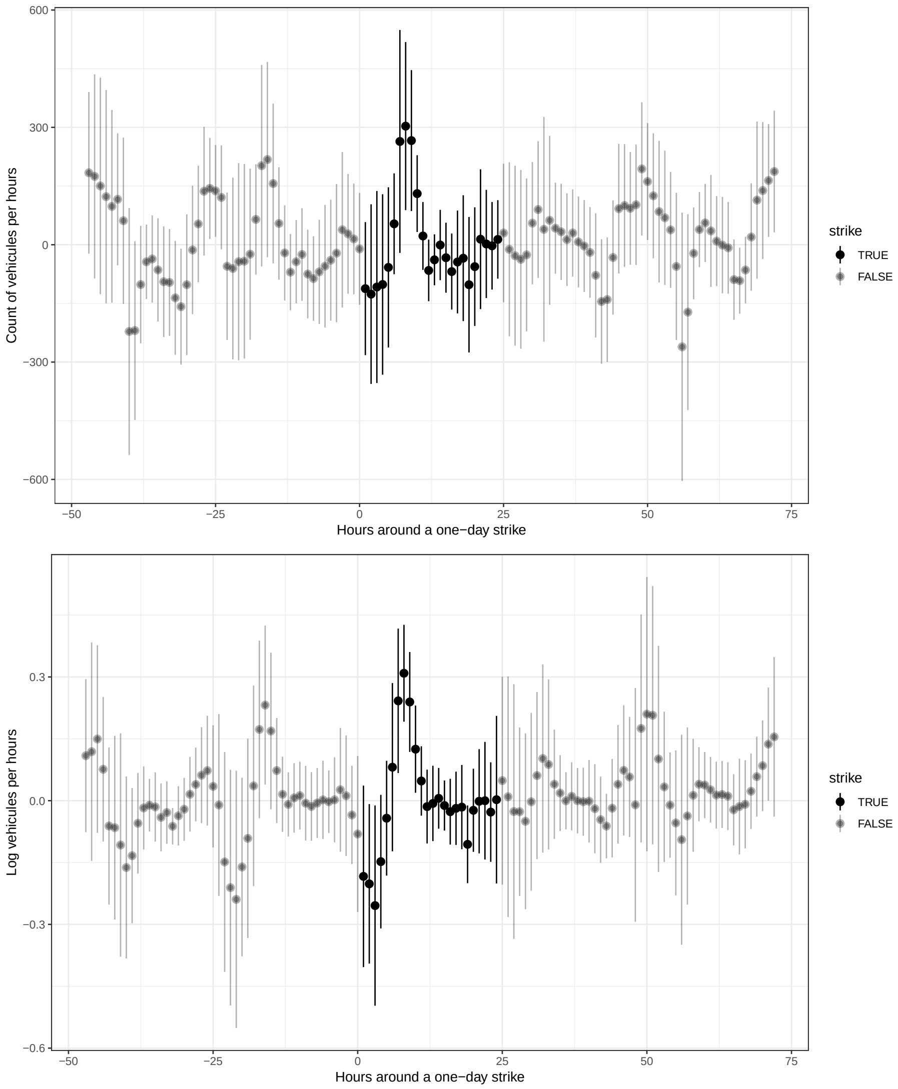


Figure 18: Traffic monitoring stations in Lille

Figure 19: Hourly traffic around the one-day strike. Estimates from the hourly regression described in section 3 are reported at α =5% significance level



B Tables

Table 14: Testing whether missing air pollutants concentrations are related to strikes.

	Dummy =1 if missing					
	PM2.5	PM10	CO	O3	NO2	SO2
S	0.0191	-0.0171	-0.00121	0.0259	-0.0952*	0.0337
	(0.0279)	(0.0501)	(0.0460)	(0.0394)	(0.0477)	(0.0432)
Observations	13547	13547	13547	13547	13547	13547
R^2	0.377	0.549	0.614	0.335	0.371	0.415

Notes: All regressions are run at the date-city level as in Equation (1), and include date fixed effects and city-specific day-of-week, week-of-year fixed effects, and weather controls. Cluster-robust standard errors (robust to the presence of heteroskedasticity) are in parentheses. $*$: p-value < 0.1,$**$: p-value < 0.05,$***$: p-value < 0.01

	Other respiratory diseases ICD10 J20-J99				
	All	0-4	5-14	15-59	Over 60
S-1	-0.0266	-0.673	0.101	0.00479	-0.0305
	(0.0477)	(0.396)	(0.108)	(0.0298)	(0.121)
S	0.0552	0.259	0.0129	0.00381	0.132
	(0.0455)	(0.357)	(0.0773)	(0.0364)	(0.0867)
S+1	-0.0455	-0.246	0.000260	-0.00423	-0.137
	(0.0764)	(0.650)	(0.0628)	(0.0511)	(0.172)
S+2	-0.00265	0.219	-0.00157	-0.0212	-0.0140
	(0.0645)	(0.310)	(0.0768)	(0.0501)	(0.154)
Observations	13547	13547	13547	13547	13547
R^2	0.724	0.728	0.415	0.396	0.537

Notes: All regressions are run at the date-city level as in Equation (1), and include date fixed effects and city-specific day-of-week, week-of-year fixed effects, and weather controls. Cluster-robust standard errors (robust to the presence of heteroskedasticity) are in parentheses. $*$: p-value < 0.1,$**$: p-value < 0.05,$***$: p-value < 0.01

Table 15: Other leading admissions

	Rate of admission per 100 000 inhabitants	
	Cardiovascular diseases ICD10 I00-I99	Injuries ICD10 S00-T14
S-1	-0.0420	0.0414
	(0.0684)	(0.128)
S	-0.0696	0.0814
	(0.0596)	(0.124)
S+1	0.0413	-0.103
	(0.0662)	(0.105)
S+2	0.0531	0.101
	(0.0521)	(0.0912)
Observations	13547	13547
R^2	0.615	0.622

Notes: All regressions are run at the date-city level as in Equation (1), and include date fixed effects and city-specific day-of-week, week-of-year fixed effects, and weather controls. Cluster-robust standard errors (robust to the presence of heteroskedasticity) are in parentheses. $*$: p-value < 0.1,$**$: p-value < 0.05,$***$: p-value < 0.01

Table 16: Emergencies admissions by age group: abnormalities of breathing

	Rate of admission per 100 000 inhabitants of the corresponding age bracket			
	Abnormalities of breathing ICD10 R06			
Age group	0-4	5-14	15-59	Over 60
S-1	0.102	0.00668	-0.000143	-0.0191
	(0.0682)	(0.0122)	(0.00918)	(0.0542)
S	0.0107	-0.0110	0.0107	0.0243
	(0.0416)	(0.00816)	(0.00736)	(0.0493)
S+1	-0.0162	-0.00137	0.0225**	0.0299
	(0.0673)	(0.00742)	(0.00712)	(0.0483)
S+2	0.0209	-0.00764	-0.00180	0.0243
	(0.0449)	(0.00973)	(0.00998)	(0.0337)
Observations	13547	13547	13547	13547
R^2	0.320	0.242	0.308	0.329

Notes: All regressions are run at the date-city level as in Equation (1), and include date fixed effects and city-specific day-of-week, week-of-year fixed effects, and weather controls. Cluster-robust standard errors (robust to the presence of heteroskedasticity) are in parentheses. $*$: p-value < 0.1,$**$: p-value < 0.05,$***$: p-value < 0.01

Table 17: Emergencies admissions by age group: diseases of the respiratory system

	Rate of admission per 100 000 inhabitants of the corresponding age bracket			
	Diseases of respiratory system ICD10 J00-J99			
Age group	0-4	5-14	15-59	Over 60
S-1	-0.385	0.111	0.0111	0.0176
	(0.682)	(0.138)	(0.0447)	(0.124)
S	0.694*	0.0574	-0.00321	0.0615
	(0.364)	(0.153)	(0.0590)	(0.170)
S+1	-0.197	0.0633	-0.0206	-0.355
	(0.527)	(0.0805)	(0.0398)	(0.215)
S+2	0.0952	-0.0766	-0.0530	-0.215
	(0.473)	(0.0789)	(0.0659)	(0.196)
Observations	13547	13547	13547	13547
R^2	0.742	0.434	0.461	0.626

Notes: All regressions are run at the date-city level as in Equation (1), and include date fixed effects and city-specific day-of-week, week-of-year fixed effects, and weather controls. Cluster-robust standard errors (robust to the presence of heteroskedasticity) are in parentheses. $*$: p-value < 0.1,$**$: p-value < 0.05,$***$: p-value < 0.01

Table 18: Robustness when dropping the largest urban area (Paris) for pollutants outcomes.

Without Paris	(1)	(2)	(3)	(4)	(5)	(6)
			Day first quartile			
	CO	PM2.5	PM10	NO2	O3	SO2
S-1	8.630	0.0963	0.0418	2.894	-2.630	-0.124
	(29.55)	(0.786)	(1.180)	(1.755)	(2.079)	(0.136)
S	31.94*	1.623	1.072	1.336	0.807	-0.315
	(17.06)	(1.393)	(1.751)	(1.161)	(1.931)	(0.225)
S+1	13.67	1.468	1.421	2.054*	0.592	-0.440**
	(27.99)	(0.830)	(1.070)	(0.933)	(2.039)	(0.188)
S+2	-4.278	1.665	0.793	-0.658	0.579	-0.186
	(22.45)	(1.252)	(1.481)	(0.686)	(1.875)	(0.156)
Observations	9317	11120	10476	11632	11813	10551
R^2	0.704	0.761	0.740	0.851	0.852	0.418
			Day median			
S-1	-10.57	0.413	-1.208	1.615	-2.179	-0.154
	(28.64)	(1.150)	(0.992)	(1.282)	(2.100)	(0.244)
S	15.42	0.976	-0.507	0.134	0.261	-0.385
	(29.13)	(1.659)	(2.090)	(1.101)	(1.865)	(0.242)
S+1	17.94	2.276*	1.602	1.423	-0.0607	-0.254
	(35.24)	(1.089)	(1.327)	(1.132)	(2.118)	(0.328)
S+2	-22.30	1.651	1.171	-1.052	3.342*	-0.396*
	(34.33)	(1.896)	(2.450)	(1.280)	(1.626)	(0.201)
Observations	8666	10586	10049	10678	11038	9968
R^2	0.829	0.793	0.782	0.881	0.887	0.481
			Day third quartile			
S-1	-37.97	-0.316	-2.951*	1.411	1.355	-0.108
	(34.87)	(1.810)	(1.576)	(1.876)	(1.739)	(0.322)
S	11.29	-0.825	-2.145	-0.819	0.297	-0.484
	(34.81)	(1.918)	(2.313)	(1.380)	(1.984)	(0.286)
S+1	-9.660	2.189	2.406*	0.610	1.564	0.171
	(41.45)	(1.316)	(1.196)	(1.719)	(2.962)	(0.389)
S+2	-23.54	1.227	2.250	0.228	2.152*	-0.653
	(45.86)	(1.936)	(2.977)	(2.122)	(0.988)	(0.422)
Observations	9317	11120	10476	11632	11813	10551
R^2	0.770	0.773	0.770	0.878	0.886	0.439

Notes: All regressions are run at the date-city level as in Equation (1), and include date fixed effects and city-specific day-of-week, week-of-year fixed effects, and weather controls. Cluster-robust standard errors (robust to the presence of heteroskedasticity) are in parentheses. $*$: p-value < 0.1,$**$: p-value < 0.05,$***$: p-value < 0.01

Table 19: Robustness when dropping periods marked by social movement (month September) for pollutants outcomes.

Without September	(1)	(2)	(3)	(4)	(5)	(6)
			Day first quartile			
	CO	PM2.5	PM10	NO2	O3	SO2
S-1	11.09	0.516	0.438	3.173*	-1.106	-0.118
	(24.24)	(0.585)	(0.846)	(1.561)	(1.882)	(0.198)
S	27.03*	1.250	1.578	0.656	1.949	-0.124
	(14.30)	(1.238)	(1.270)	(1.383)	(1.906)	(0.116)
S+1	26.66	1.019*	1.493*	0.519	0.103	-0.352**
	(20.18)	(0.513)	(0.775)	(1.774)	(1.935)	(0.136)
S+2	-10.21	1.641*	1.285	-0.579	0.587	-0.112
	(14.97)	(0.822)	(1.143)	(0.543)	(1.292)	(0.110)
Observations	9400	11028	10518	11118	11528	10519
R^2	0.758	0.776	0.750	0.855	0.857	0.465
			Day median			
S-1	5.458	1.410	-0.298	1.015	-1.110	-0.161
	(30.51)	(0.814)	(0.866)	(1.251)	(1.385)	(0.291)
S	15.08	1.002	1.066	-0.258	0.462	0.219
	(21.62)	(1.196)	(1.647)	(1.222)	(1.804)	(0.341)
S+1	44.28	1.756*	1.231	1.142	0.650	-0.218
	(26.42)	(0.785)	(1.048)	(1.028)	(1.686)	(0.249)
S+2	-20.45	1.719	1.807	-0.857	2.830*	-0.172
	(23.33)	(1.275)	(2.002)	(1.080)	(1.483)	(0.182)
Observations	9400	11028	10518	11118	11528	10519
R^2	0.811	0.792	0.776	0.881	0.894	0.491
			Day third quartile			
SS-1	-23.53	0.609	-1.199	1.936	0.781	-0.101
	(44.88)	(1.025)	(1.120)	(1.162)	(1.540)	(0.354)
S	14.22	-0.354	0.238	-2.364	-0.0149	0.227
	(29.58)	(1.387)	(1.995)	(1.611)	(1.585)	(0.389)
S+1	41.24	2.315*	2.493**	0.802	2.565	0.0478
	(39.48)	(1.036)	(0.886)	(1.241)	(2.590)	(0.323)
S+2	-20.50	1.452	2.814	-0.399	2.892**	-0.312
	(30.28)	(1.223)	(2.509)	(1.830)	(1.053)	(0.279)
Observations	9400	11028	10518	11118	11528	10519
R^2	0.820	0.792	0.776	0.873	0.908	0.530

Notes: All regressions are run at the date-city level as in Equation (1), and include date fixed effects and city-specific day-of-week, week-of-year fixed effects, and weather controls. Cluster-robust standard errors (robust to the presence of heteroskedasticity) are in parentheses. $*$: p-value < 0.1,$**$: p-value < 0.05,$***$: p-value < 0.01

Table 20: Robustness when dropping periods marked by social movement (year 2010) for pollutants outcomes.

Without 2010	(1)	(2)	(3)	(4)	(5)	(6)
			Day first quartile			
	CO	PM2.5	PM10	NO2	O3	SO2
S-1	6.438	0.348	0.813	0.800	-0.646	-0.360*
	(20.79)	(0.404)	(0.696)	(1.463)	(1.245)	(0.169)
S	42.60***	1.045	1.944*	-0.828	0.885	-0.207
	(11.47)	(0.639)	(0.916)	(1.521)	(1.543)	(0.135)
S+1	23.64	1.734**	1.508	-1.158	1.599	-0.193
	(20.67)	(0.549)	(1.144)	(1.667)	(2.730)	(0.176)
S+2	13.61	1.988***	2.362***	-1.894**	1.922	-0.0877
	(25.89)	(0.572)	(0.653)	(0.836)	(2.549)	(0.110)
Observations	8666	10586	10049	10678	11038	9968
R^2	0.778	0.778	0.756	0.855	0.852	0.458
			Day median			
S-1	4.633	0.807	0.163	0.116	0.393	-0.419**
	(31.59)	(0.809)	(0.928)	(1.673)	(1.428)	(0.166)
S	40.39**	1.153	1.210	-0.751	0.658	0.272
	(15.46)	(0.733)	(1.684)	(1.232)	(1.709)	(0.510)
S+1	35.40	2.186***	1.355	-0.532	1.814	-0.0465
	(30.65)	(0.623)	(1.418)	(1.330)	(1.930)	(0.230)
S+2	-6.353	2.034**	2.731***	-1.528**	2.364	-0.158
	(27.39)	(0.661)	(0.817)	(0.646)	(1.635)	(0.148)
Observations	8666	10586	10049	10678	11038	9968
R^2	0.829	0.793	0.782	0.881	0.887	0.481
			Day third quartile			
S-1	2.862	0.136	-0.703	2.166	3.124	-0.484**
	(39.30)	(1.230)	(1.268)	(1.659)	(2.098)	(0.165)
S	34.05	0.656	0.150	-3.062	0.335	0.179
	(19.78)	(0.824)	(2.271)	(2.060)	(1.747)	(0.579)
S+1	34.45	2.602***	1.511	-1.631	4.280*	0.101
	(55.18)	(0.785)	(1.462)	(1.610)	(2.037)	(0.332)
S+2	-6.765	1.561**	4.090**	0.218	3.588**	-0.182
	(30.29)	(0.595)	(1.673)	(1.428)	(1.372)	(0.260)
Observations	8666	10586	10049	10678	11038	9968
R^2	0.837	0.794	0.780	0.874	0.904	0.524

Notes: All regressions are run at the date-city level as in Equation (1), and include date fixed effects and city-specific day-of-week, week-of-year fixed effects, and weather controls. Cluster-robust standard errors (robust to the presence of heteroskedasticity) are in parentheses. $*$: p-value < 0.1,$**$: p-value < 0.05,$***$: p-value < 0.01

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