

REVIEW

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# A review of health effects associated with exposure to jet engine emissions in and around airports

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## Abstract

**Background:** Airport personnel are at risk of occupational exposure to jet engine emissions, which similarly to diesel exhaust emissions include volatile organic compounds and particulate matter consisting of an inorganic carbon core with associated polycyclic aromatic hydrocarbons, and metals. Diesel exhaust is classified as carcinogenic and the particulate fraction has in itself been linked to several adverse health effects including cancer.

**Method:** In this review, we summarize the available scientific literature covering human health effects of exposure to airport emissions, both in occupational settings and for residents living close to airports. We also report the findings from the limited scientific mechanistic studies of jet engine emissions in animal and cell models.

**Results:** Jet engine emissions contain large amounts of nano-sized particles, which are particularly prone to reach the lower airways upon inhalation. Size of particles and emission levels depend on type of aircraft, engine conditions, and fuel type, as well as on operation modes. Exposure to jet engine emissions is reported to be associated with biomarkers of exposure as well as biomarkers of effect among airport personnel, especially in ground-support functions. Proximity to running jet engines or to the airport as such for residential areas is associated with increased exposure and with increased risk of disease, increased hospital admissions and self-reported lung symptoms.

**Conclusion:** We conclude that though the literature is scarce and with low consistency in methods and measured biomarkers, there is evidence that jet engine emissions have physicochemical properties similar to diesel exhaust particles, and that exposure to jet engine emissions is associated with similar adverse health effects as exposure to diesel exhaust particles and other traffic emissions.

**Keywords:** Jet engine emissions, Airports, Occupational exposure, Particulate matter, Polycyclic aromatic hydrocarbons, Biomarkers

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## Background

Exposure to air pollution, including ultrafine particulate matter (UFP), from industry and traffic is associated with adverse health effects [1–4]. Airports are significant high-emission sources and human exposure to these emissions is a growing health concern. Importantly, airport personnel are at risk of occupational exposure to jet engine emissions [5]. More knowledge is needed on exposure risks, adverse health effects, biomarkers and risk management options related to the diverse factors influencing human exposure to airport emissions [6] (Fig. 1).

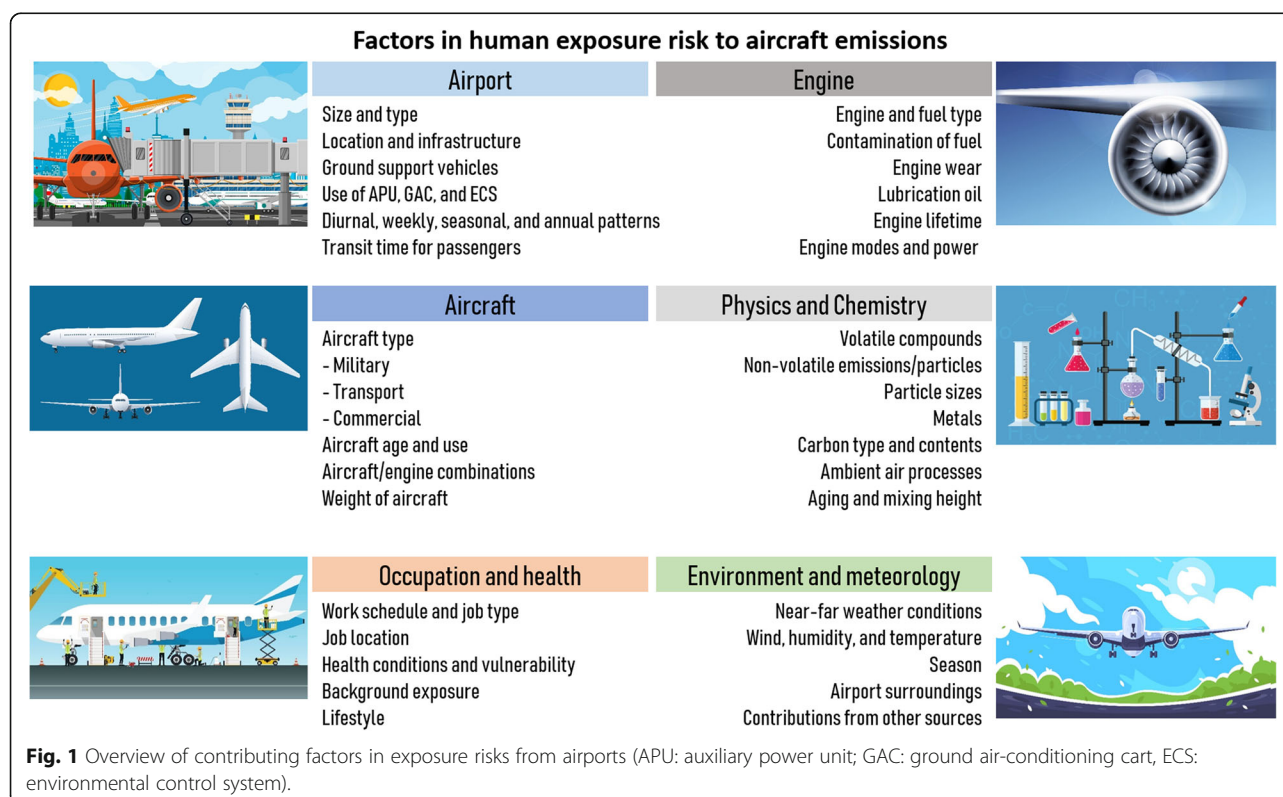
However, data collection seems challenging. Commercial airports are large, complex and diverse work places, where aircraft, ground-support equipment (GSE), and related vehicles all contribute to mixed emissions [7, 8]. In turn, commercial airports as well as military air stations are year-round active high security areas with restricted access, which can reduce the options for external researchers to collect optimal or sufficient measurements. Consensus or formal guidelines for optimal measurement design, instrumentation and analysis methods for the different emission components are lacking, which further complicates comparison of data and risk assessment [5, 9].

With this review, we seek to compile available studies in the open scientific literature on health effects of jet engine emissions in occupational settings and in residential areas around airports, along with mechanistic effects studied in animal and cell models. The studies were selected based on

key papers and systematic searches (search terms, method and selection criteria are disclosed in the Additional file 1). We briefly summarize the characteristics of jet engine emissions and highlight the complexity of this field of research, but detailed research on emissions and physical-chemical studies is beyond the scope of this review.

## Toxicity of jet fuel exposure

The toxicity of (unburned) jet fuel as such has been considered in many studies (reviewed in [10]) since the early 1950's, where the specifications of the hydrocarbon-based jet fuel, JP-4 (jet propellant-4), was published by the US air force. Major toxic effects reported for JP-4 were skin irritation, neurotoxicity, nephrotoxicity, and renal carcinogenicity in rats [11]. Jet fuels are mixtures of gasoline and kerosene with performance additives [10]. In 1994, US Air Force converted to JP-8, developed to be less volatile and less explosive upon crash incidents compared to JP-4. JP-8 (NATO F-34) is equivalent to Jet A-1 fuel used in commercial aircraft. A range of other kerosene-based jet fuels are in use, depending on aircraft type and differing in kerosene ratio and requirements for additives [5]. Measurements of a range of the common aircraft pollutants such as benzene, toluene, and chlorinated compounds in breath samples from exposed personnel on an airbase before and after work tasks showed significant exposure for all subjects, ranging from minor elevations up to > 100 times the values of



the control group for fuel workers [12]. The uptake of JP-8 components both occur via inhalation and dermal contact, and apart from benzene, naphthalene in air and in exhaled breath condensate (EBC) may be useful as a biomarker of exposure to and uptake of JP-8 fuel components in the body [13]. Although most studies report low acute toxicity for both JP-4 and JP-8, JP-8 was reported to show effects such as respiratory tract sensory irritation [11], inflammatory cytokine secretion in exposed alveolar type II epithelial cells and in pulmonary alveolar macrophages [14], increased pulmonary resistance and decreased weight gain in rats upon inhalation exposure for 7 or 28 days [15, 16]. Subchronic 90-days studies with rats with various exposure levels of JP-4 and JP-8 showed little toxicity, apart from male rat hydrocarbon nephropathy [11]. However, JP-8 fuel exposure has been linked to noise-activated ototoxic hearing loss in animal studies [17, 18] and in occupational exposure cases [19, 20], and to immunotoxicity [21, 22].

It is likely that fuel refinements will advance in the future and be an important factor in emission reductions. A newer synthetic jet fuel (Fischer-Tropsch Synthetic Paraffinic Kerosene) under development to replace JP-8 in the future, was evaluated for toxicity in the required range of tests used to develop occupational exposure limits (OELs). The highest exposure level of 2000 mg/m<sup>3</sup> (6 h per day, 5 days a week for 90 days) produced multifocal inflammatory cell infiltrations in rat lungs, whereas no genotoxicity or acute inhalation effects were observed, and the sensory irritation assay indicated that the refined synthetic fuel was less irritating than JP-8 [23]. Evidence of cancer risk is, however, normally evaluated in two-year inhalation studies in rats.

#### Characteristics of jet engine emissions

Like other combustion engines, jet engines produce volatile organic compounds (VOC) such as CO<sub>2</sub>, NO<sub>x</sub>, CO, SO<sub>x</sub> and low molecular weight polycyclic aromatic hydrocarbons (PAH), and particulate matter (PM) with associated PAH, and metals [24]. Incomplete combustion of fossil fuels, including kerosene, results in the formation of carbon-rich (> 60%), aromatic bi-products called char, and condensates, which are known as soot. Char and soot can either be measured as elemental carbon (EC, used in atmospheric sciences) or black carbon (BC, used in soil and sediment sciences) [25]. This terminology originates from their measurement methods (BC is light-absorbing, determined by optical methods and EC is refractory, determined by thermo-optical and oxidizing methods) [26]. BC is often used in physical/chemical aerosol studies of airport- and urban emissions, such as in Costabile et al. [27] and Keuken et al. [28]. However, there is no apparent consistent correlation between BC concentrations and particle number

concentrations across exposure studies at airports, but data is limited as noted by Stacey [9].

In general, emission levels are high, but vary depending on engine conditions and fuel type, as well as on operation modes such as idling, taxi, take-off, climb-out and landing [29].

#### Particulate matter (PM)

PM is divided by size ranges according to the aerodynamic diameter of the particles, where UFP are in the nanoscale of < 100 nm. Several studies have shown that aircraft emissions are dominated or even characterized by high concentrations of very small particles. This was underlined in a recent study by Stacey, Harrison and Pope carried out at Heathrow London in comparison to traffic background [30]. Some report particles in the range of 5–40 nm [31], and others particle diameters of 20 nm as compared to larger particles of > 35 nm measured at surrounding freeways [32]. Campagna et al. studied the contributions of UFP from a military airport to the surrounding area, by sampling on the airport grounds during flight activities, nearby the airport, in an urban area and in a rural area. The smallest primary particles were found within the airport (~ 10 nm) and the largest in the urban area (~ 72 nm). The highest UFP levels inside the airport were measured during taxi and take-off activities ( $4.0 \times 10^6$  particles/cm<sup>3</sup>) [33]. Westerdahl et al. reported very high particle number concentrations at take-off of a single jet aircraft, with a 10 s peak of 4.8 million particles/cm<sup>3</sup> together with elevated NO<sub>x</sub> and BC levels [34].

The small particles are emitted in large numbers and tend to form complex agglomerates in ambient air that can be detected in larger particle size modes [35, 36] (see [5] for elaboration). In a recent study in Montreal-Pierre-Elliott-Trudeau International Airport, the total particle number concentration over all sizes at the airport apron reached  $2.0 \times 10^6$ /cm<sup>3</sup>, which was significantly higher compared to downtown Montreal ( $1 \times 10^4$ /cm<sup>3</sup>). The geometric mean of observed ultrafine particle number density of nanoparticles was  $1 \times 10^5$ /cm<sup>3</sup> at the apron and  $1.1 \times 10^4$ /cm<sup>3</sup> outside the Departure Level entrance [37]. We recently published exposure measurements conducted at a commercial airport and non-commercial airfield, where air concentrations were measured to  $7.7 \times 10^6$  particle/cm<sup>3</sup> or 1086 µg/m<sup>3</sup> of total particles during take-off of one single jet plane [36]. The majority of these particles were below the size detection limit of 10 nm for the instruments [36], which was also shown, and highlighted as a general challenge, by others [38].

The nanostructure of carbon particles are influenced by fuel type and combustion processes. Low thrust settings are associated with the smallest particle sizes. In

one of their studies, Vander Wal et al. characterized the aircraft particles as predominantly organic carbon at low thrust and EC at higher thrust settings [38]. In turn, it was reported that soot reactivity, characterized by an outer amorphous shell, of soot particles from a turbofan test engine was lower in particles from ground idle as compared to particles from climb-out engine mode for two fuel types. Biofuel blending slightly lowered this soot reactivity at ground idle, but had the opposite effect at the higher power condition of climb-out. The authors comment that for soot reactivity, measured by an outer amorphous shell in the study, biofuels may be beneficial in airports where ground idle engine conditions are often in use, but the effect on emissions in climb-out conditions is undetermined [31]. According Moore et al., a 50:50 biofuel blending reduces particle emissions from aircraft with 50–70%, compared to conventional Jet-A fuel [39]. Another study did extensive analyses of emissions from four on-wing commercial aircraft turbo engines (two newer CFM56–7 engines and two CFM56–3 engines), also demonstrating that the type of emissions were significantly dependent on power. PM emission indices ( $\text{g/kg}^{-1}$  fuel) were reported to increase from 0.011 to  $0.205 \text{ g/kg}^{-1}$  fuel with a power increase from idle to 85%. In turn, the data showed that hydrocarbons are mostly emitted at ground idle engine conditions, as opposed to PM emissions being more significant at higher power thrusts, such as take-off and landing. EC fraction of PM also increased with increase in power [40]. Targino et al. measured large EC (BC) concentrations during boarding and disembarking (mean  $3.78 \mu\text{g}/\text{m}^3$ ), at the airport concourse (mean  $3.16 \mu\text{g}/\text{m}^3$ ) and also inside an aircraft on the ground with open doors (mean  $2.78 \mu\text{g}/\text{m}^3$ ) [41].

#### Lubrication oil and organophosphate esters

A recent study found that intact forms of unburned jet engine lubrication oil was a major component of emissions from aircraft [42]. Organophosphate esters (OPEs) are a large group of chemicals with toxic properties used as stabilizing agents in numerous consumer – and industrial products, including in aircraft lubricating oil and hydraulic fluids. Airplane emissions are thought to be an important source of OPEs in the environment. Not only does these chemicals accumulate in ecosystems, but it is also a concern due to the location of airports near populated areas [5]. Li et al. recently studied the concentrations of 20 OPEs in ambient air, soil, pine needles, river water, and outdoor dust samples collected around an airport in Albany, New York, and reported elevated total OPE concentrations in all samples. The spatial distribution of OPEs in air, soil, and pine needles correlated with distance to the airport. The average daily intake of OPEs via air inhalation and outdoor dust ingestion in the

vicinity of the airport was up to  $1.53 \text{ ng/kg bw/day}$  for children and  $0.73 \text{ ng/kg bw/day}$  for adults [43]. Another study examined organophosphates, such as tri-*n*-butyl phosphate, dibutyl phenyl phosphate, triphenyl phosphate and tricresyl phosphate from turbine and hydraulic oils, as well as oil aerosol/vapors and total volatile organic compounds (VOC) in air with potential for occupational exposure for airport ground personnel. The measured exposure levels were mainly below the limit of quantification during work tasks, but provoked exposure situations resulted in significantly higher exposure levels compared to normal conditions, illustrated by oil aerosol up to  $240 \text{ mg}/\text{m}^{-3}$  and tricresyl phosphate concentrations up to  $31 \text{ mg}/\text{m}^{-3}$ . Highest exposure levels were measured during loading from jet engine aircraft [44].

Exposure to toxic compounds via contaminated bleed air (from engine compressors), including OPEs, has been widely studied among cabin crew and pilots, and has been associated with adverse neurological effects and respiratory illness [45, 46].

#### Metals and other elements

Metals which might be specific to airport emissions, either by abundance or type, such as the heavy-metal vanadium [47], could be potential chemical fingerprints. Abegglen et al. applied single particle mass spectrometry to investigate metal content and sources in emissions from different jet engines at various combustion conditions, and Mo, Ca, Na, Fe, Cu, Ba, Cr, Al, Si, Mg, Co, Mn, V, Ni, Pb, Ti and Zr were found to be significant frequently occurring metals. Fuel, lubrication oil, grease and engine wear are potential sources, but several metals were allocated to multiple sources [48].

In the studies of He et al and Shirmohammadi et al, particles were collected at Los Angeles Airport (LAX) and central Los Angeles (LA) and among other analyses, allocated according to elements associated with different sources [49, 50]. S was considered as aviation-related and particle-bound Na was viewed as ocean-related, due to sea salt from the ocean near by LAX. Al, Ca, Ti and K were considered as trace elements for road dust from LAX and central LA. Mn, Fe, Cu, Zn, Ba, Pb, Ni, and Mg were associated with traffic emissions, including fuel and lubricating oil combustions and brake abrasions, engine and tire wear. In LAX particles, S accounted for the largest fraction (49.5%), followed by road dust elements (21.8%) and traffic-related elements (15.9%). In particles from central LA, elements from traffic, road dust, and aviation were represented equally (28.5, 31.5, and 33.4%, respectively) [49, 50]. In a study from Montreal-Pierre-Elliott-Trudeau International Airport, several metals were found to be abundant in the particle fraction, such as Fe, Zn, and Al, and the authors speculate, that



airports in fact may be hotspots for nanoparticles containing emerging contaminants [37]. A recent study investigated the levels of 57 elements at five sampling sites within the vicinity of Eskisehir Hasan Polatkan Airport in Turkey, based on moss bag biomonitoring using *Sphagnum* sp. in combination with chemical analyses of lubrication oil and aviation gasoline fuel used by general aviation, piston-engine, and turboprop aircraft. Moss bag biomonitoring was a useful tool in identification of the elements that accumulated downwind of the airport emissions. Characterization of the metal contents in moss bags and oil and fuel were in agreement, showing that Pb, along with Cd, Cu, Mo, Cr, Ni, Fe, Si, Zn, Na, P, Ca, Mg, and Al were dominating elements in the general aviation aircraft emissions [51].

#### Polycyclic aromatic hydrocarbons/volatile organic compounds

Polycyclic aromatic hydrocarbons (PAH), including several known carcinogens, are also candidates for chemical airport emission tracers. PAH are semi-volatile compounds, in between the gaseous and particulate phases. Lighter-weight PAHs (<4 rings) present almost exclusively in the vapour-phase and PAHs with higher molecular weights (>4 rings) are almost completely particle-bound [5]. It was reported that the apron of the Fiumicino Airport in Rome had higher levels of measured PAH ( $27.2 \mu\text{g}/\text{m}^3$ ) compared to PAH levels in the airport building and terminal [52]. Another study of PAH in airport emissions at the apron reported that the five most abundant species of particle bound-PAHs for all sampling days were naphthalene, phenanthrene, fluoranthene, acenaphthene, and pyrene, with total concentrations between  $0.152 \mu\text{g}/\text{m}^3$  -  $0.189 \mu\text{g}/\text{m}^3$  ( $152.21$ – $188.94 \text{ ng}/\text{m}^3$ ) depending on season. The most abundant fractions of benzo(a)pyrene (BaP) equivalent concentration (BaP<sub>eq</sub>) in different molecular weights were high-weight PAHs (79.29%), followed by medium-weight PAHs (11.57%) and low-weight PAHs (9.14%). The percentages of total BaP<sub>eq</sub> in the very small particles <  $0.032 \mu\text{m}$  were 52.4% (mean concentration  $0.94 \text{ ng}/\text{m}^3$ ) and 70.15% in particles <  $100 \mu\text{m}$  (mean concentration  $1.25 \text{ ng}/\text{m}^3$ ) [53]. Studies of the emissions from a helicopter engine at different thrusts included analysis of 22 PAH compounds, where 97.5% of the total PAH emissions were two- and three-ringed PAHs, with a mean total PAH concentration of  $843 \mu\text{g}/\text{m}^3$  and a maximum of  $1653 \mu\text{g}/\text{m}^3$  during ground idle. This was 1.05–51.7 times higher compared to a heavy-duty diesel engine, a motor vehicle engine, and an F101 aircraft engine. In turn, total level of BaP during one landing and take-off cycle (LTO) ( $2.19 \text{ mg}/\text{LTO}$ ) [54] was higher than the European Commission emission factor of  $1.24 \text{ mg}/\text{LTO}$ , stated in their PAH position paper, where emission

factors are used to calculate the degree to which a source contributes to the total emission of a specific pollutant [55]. The Danish occupational exposure limit for PAH is  $200 \mu\text{g}/\text{m}^3$  [56], and reported PAH concentrations in ambient air across studies were below this level.

Volatile organic compounds (VOC) comprise a diverse group of organic chemicals, with different physicochemical and toxicological properties. Scientific studies of these emission compounds were meticulously reviewed by Masiol et al. [5], and as noted by the authors there is insufficient knowledge in terms of the significance of these compounds for airport exhaust health impacts [5]. Some VOC have known toxicities and other are suspected to have adverse health effects, and among the hydrocarbons found in aircraft exhaust, 14 single or complex compounds are listed as hazardous by the Federal Aviation Administration, which in addition to PAH compounds comprise benzene, styrene, xylene, toluene, acetaldehyde, 1,3-butadiene, n-hexane, acrolein, propionaldehyde, ethylbenzene, formaldehyde, and lead compounds [57]. A recent study assessed 46 VOC in the indoor air of the control tower maintenance room, potentially affecting employees, where a correlation was found between aircraft number and concentrations of light aldehydes/ketones [58].

#### Summary and perspectives

Emission measurement studies are continuously conducted at international airports, such as Amsterdam Airport Schiphol (AMS) [28, 59], Rome Ciampino (CIA) [60], London Heathrow (LHR) [61, 62], Beirut-Rafic Hariri International Airport (RHIA) [63], Hartsfield-Jackson Atlanta International Airport [64], Los Angeles International Airport (LAX) [32, 49, 65], and other large airports in California [66] which besides measurements of the previously mentioned compounds, also often include analyses of emission patterns and weather conditions, and characterizations of particle size- and mass distributions [67]. The data from these emission studies and physicochemical studies of emissions including particle matter (PM), from which we referenced some in the previous sections, were recently reviewed thoroughly [9]. To summarize the previous section, we repeat some selected important points regarding airport-sourced particles that were deducted from the available data by Stacey [9]:

- 1) *Particle numbers near airports are significantly higher than away from airports and jet engines are a significant source of UFP.* This means that urban areas in the vicinity of airports are at risk of increased exposure to UFP in addition to normal daily background and traffic-related emissions, but airport personnel working on the ground are in significant risk of exposure, simply due to proximity.

- 2) *The highest concentrations of UFP are measured downwind of aircraft.* Due to the occupational potential of exposure for airport ground workers there is a growing necessity of further studies of dispersion, size distributions and environmental factors affecting these emissions. Stacey [9] highlights that measurements at longer distances are highly influenced by physical and chemical processes affecting the emissions in the air, including volatile compounds. As such, there is a need for increased standardization of methods and instruments to facilitate valid comparisons between studies within this field, as has been established in general for environmental particulate matter (PM) measurements.
- 3) *Aircraft emissions are dominated by very small particles of < 20 nm.* This may be a way to separate these from other emission sources, such as road traffic, where the main particle fraction are of larger sizes. Smaller particle size means higher specific surface area. Smaller particles deposit in the deep end of the lung during inhalation and the total surface area of the deposited nanoparticles has been suggested to be predictive of toxicological potential in the lung [68].
- 4) *The majority of non-volatile airport emission particles are carbonaceous (consisting of elemental and organic carbon compounds).* The emissions from aircraft consists of high numbers of soot particles with associated PAHs and metals, and thus, their physico-chemical composition is similar to diesel exhaust particles [36].

Diesel exhaust is classified as carcinogenic to humans by IARC [69], and cause lung cancer, systemic inflammation, and inflammatory responses in the airways [70]. Animal studies have shown that the particulate fraction of diesel exhaust is mutagenic and carcinogenic [71], whereas filtered diesel exhaust does not cause cancer [72]. Exposure to standard reference diesel particle SRM1650b and carbon black (CB) induce pulmonary acute phase response, neutrophil influx, and genotoxicity in mouse models [73–78]. Genotoxicity has been observed even at very low doses of CB [79]. In a meta-analysis of exposure to diesel exhaust and lung cancer occurrence in three occupational studies, the identified dose-response relationship showed that occupational exposure to 1  $\mu\text{g EC}/\text{m}^3$  during a 45 year work life would cause 17 excess lung cancers per 10,000 exposed using the EC content of diesel exhaust as metric [80]. Another recent analysis of 14 case-control studies estimated exposure to diesel exhaust particles using job-exposure matrices. In this study, occupational exposure to 1  $\mu\text{g EC}/\text{m}^3$  during a 45 year work life would cause 4 excess lung cancers per 10,000 exposed using the EC content of diesel exhaust as metric [81].

Carcinogenic substances are evaluated and listed by the International Agency of Research in Cancer (IARC) under WHO according to accumulated scientific findings in cellular, animal and human studies. Group 1 entails substances with sufficient evidence of carcinogenicity in humans and group 2 includes substances that IARC has classified as probably (2A) or possibly (2B) carcinogenic to humans [82]. As almost all current aviation fuel/jet fuels are extracted from the middle distillates of crude oil (kerosene fraction), which is between the fractions for gasoline and diesel [5] (whose combustion emissions are classified as group 2B and group 1 carcinogens, respectively [69]), there is cause for concern in terms of the potential carcinogenicity of exposure to jet fuel combustion products.

### Exposure studies

Reported exposure levels for PAH, BC and UPF in the studies below are presented in Table 1.

### Occupational exposure

Childers et al. (2000): An extensive study of PAH concentrations at an airbase was carried out, using real-time monitors and air samplers on different locations and in different flight-related and ground-support activities. Airborne and particle-bound PAH were measured in a break room, downwind from an aircraft (C-130H) during engine tests, in a maintenance hangar, in an aircraft (C-130H) cargo bay during cargo-drop training and during engine running on/off loading and backup exercises, and downwind from aerospace ground equipment (diesel-powered electrical generator and a diesel-powered heater). Measurements were carried out with three different monitors. Total PAH concentrations followed a general trend of downwind from two diesel aerospace ground equipment units > engine on/off-loading exercise > engine tests > maintenance hangar during taxi and takeoff > background measurements in the maintenance hangar. Reported mean total PAH concentrations in integrated air samples (vapor phase) were 0.6011  $\mu\text{g}/\text{m}^3$  (hangar background), 1.0254  $\mu\text{g}/\text{m}^3$  (hangar taxiing), 2.8027  $\mu\text{g}/\text{m}^3$  (engine test), 6.7953  $\mu\text{g}/\text{m}^3$  (engine running on/off) and 9.8111  $\mu\text{g}/\text{m}^3$  (aerospace ground equipment). Dominating PAH in all exposure scenario was naphthalene, the alkyl-substituted naphthalenes, and other PAHs in the vapor phase. Particle-bound PAHs, such as fluoranthene, pyrene, and benzo[a]pyrene were also found. During flight-related exercises, PAH concentrations were 10–15 higher than in ambient air, and it was found that PAH contents fluctuated rapidly from < 0.02 to > 4  $\mu\text{g}/\text{m}^3$  during flight-related activities [83].

Iavicoli et al. (2006): In this study, occupational exposure risk to PAH and biphenyl was evaluated in an Italian airport during winter. Concentration and purification of 12 samples of 25 PAH by gas chromatography-ion trap

**Table 1** Overview of reported levels of occupational exposures of PAH, BC, and particles in airports. *Mean levels are presented if reported. For detailed data, see references*

Description	Reported mean levels Ambient air	Reported mean levels Personal monitors	Reference
<b>PAH</b>			
Total mean PAH concentrations in integrated air samples at an airbase on different locations and in different flight-related and ground-support activities	601.1 ng/m <sup>3</sup> (hangar background) 1025.4 ng/m <sup>3</sup> (hangar taxiing) 2802.7 ng/m <sup>3</sup> (engine test) 6795.3 ng/m <sup>3</sup> (engine running on/off) 9811.1 ng/m <sup>3</sup> (diesel-fueled aerospace ground equipment) <i>During flight-related exercises, PAH concentrations were 10–15 times higher than in ambient air</i>	NA	Childers et al. (2000) [83]
PAH compounds of highest levels measured for 24 h in three different locations	130–13,050 ng/m <sup>3</sup> (naphthalene) 64–28,500 ng/m <sup>3</sup> (2-methylnaphthalene) 24–35,300 ng/m <sup>3</sup> (1-methylnaphthalene) 24–1610 ng/m <sup>3</sup> (biphenyl) 54.2 ng/m <sup>3</sup> (fluoranthene) 8.6 ng/m <sup>3</sup> (benzo[a]pyrene)	NA	Iavicoli et al. (2006) [84]
Total mean of 23 PAH (vapor and particle-bound) measured during 24 h of 5 work days at the airport apron, airport building and terminal/office area	27.703 µg/m <sup>3</sup> (apron) 17.275 µg/m <sup>3</sup> (airport building) 9.494 µg/m <sup>3</sup> (terminal departure area) <i>Highest levels in the airport apron particularly for 1 and 2-methylnaphthalene and acenaphthene</i>	NA	Cavallo et al. (2006) [52]
Total mean particle-bound PAH measured in the vicinity of LAX to assess the spread of airport emissions in up – and downwind ambient air to the immediate neighborhood	18.2 ng/m <sup>3</sup> (upwind from the airport) 24.6 ng/m <sup>3</sup> (downwind from the airport) 50.1 ng/m <sup>3</sup> (at the taxiway) 60.1 ng/m <sup>3</sup> (terminal region) <i>Particle-bound PAH mean levels measured on two freeways were 47.0 ng/m<sup>3</sup> and 169.4 ng/m<sup>3</sup></i>	NA	Westerdahl et al. (2008) [34]
<b>Black carbon</b>			
Mean black carbon concentrations measured at different micro-environments of airports and in commercial flights	3.78 µg/m <sup>3</sup> (during boarding/disembarking) 3.16 µg/m <sup>3</sup> (airport concourse) 2.78 µg/m <sup>3</sup> (inside aircraft with open doors) 0.81 µg/m <sup>3</sup> (inside aircraft on the ground with closed doors)	NA	Targino et al. (2017) [41]
BC levels measured in the vicinity of LAX to assess the spread of airport emissions in up – and downwind ambient air to the immediate neighborhood	0.3 µg/cm <sup>3</sup> (upwind from the airport) 0.7 µg/cm <sup>3</sup> (downwind from the airport) 1.8 µg/cm <sup>3</sup> (at the taxiway) 3.8 µg/cm <sup>3</sup> (terminal region)	NA	Westerdahl et al. (2008) [34]
Contributions of airport activities to measured BC levels at Amsterdam Schiphol were measured for 32 sampling days over 6 months	Mean BC: 0.6 mg/m <sup>3</sup>	NA	Pirhadi et al. (2020) [85]
<b>Particles</b>			
UFP and size distributions measured in the vicinity of LAX to assess the spread of airport emissions in up – and downwind ambient air to the immediate neighborhood	Average UFP counts of $5 \times 10^4$ particles/cm <sup>3</sup> (500 m downwind of the airport), which were significantly influenced by aircraft operations where peaks were observed Maximum UFP measured was $4.8 \times 10^6$ particles/m <sup>3</sup> downwind from a jet aircraft taking off Particle size: 90 nm (upwind from airport) 10–15 nm (downwind from airport)	NA	Westerdahl et al. (2008) [34]
Total mean concentration of 10 daily UFP samples with personal monitors placed with crew chief and hangar operator	$6.5 \times 10^3$ particles/cm <sup>3</sup> (downwind site)	$2.5 \times 10^4$ particles/cm <sup>3</sup> (crew chief) $1.7 \times 10^4$ particles/cm <sup>3</sup> (hangar operator) <i>Median number concentrations for 2 months measurement period</i>	Buonanno et al. (2012) [86]
Geometric means of personal exposure to particle number concentration carried out in five different occupational groups	NA	$37 \times 10^3$ UFP/cm <sup>3</sup> (baggage handlers) $5 \times 10^3$ UFP/cm <sup>3</sup> (landside security) $12\text{--}20 \times 10^3$ UFP/cm <sup>3</sup> (catering drivers, cleaning staff and airside security)	Møller et al. (2014) [87]
Particle and metal exposure in ambient air and in airport workers using exhaled breath condensates	$1.0 \times 10^4\text{--}2.1 \times 10^7$ particles/cm <sup>3</sup> (apron workers) $10^3\text{--}10^4$ (office staff) <i>Airport workers were exposed to significantly smaller particles (mean geometric size: 17.7 nm) compared</i>	Particulate content was found in exhaled breath condensates, but no difference was found between the two study groups	Marie-Desvergne et al. (2016) [88]

**Table 1** Overview of reported levels of occupational exposures of PAH, BC, and particles in airports. Mean levels are presented if reported. For detailed data, see references (Continued)

Description	Reported mean levels Ambient air	Reported mean levels Personal monitors	Reference
	<i>to office workers (mean geometric size: 23.7 nm).</i>		
Number concentrations and size distributions inside the cabin of an aircraft waiting for take-off compared to outdoor	10–40 × 10 <sup>3</sup> particles/cm <sup>3</sup> <i>A 40 min wait 100 m downwind of the runway was calculated to be equal to 4 h exposure in a clean urban background environment away from the airport</i>	NA	Ren et al. (2018) <sub>a</sub> [89]
Potential exposure to passengers and indoor airport staff investigated by PM <sub>2.5</sub> concentrations in the terminal building at three seasons	Arrival hall: 337 µg/m <sup>3</sup> (Winter) 105 µg/m <sup>3</sup> (Spring) 167 µg/m <sup>3</sup> (Summer) Departure hall: 385 µg/m <sup>3</sup> (Winter) 130 µg/m <sup>3</sup> (Spring) 170 µg/m <sup>3</sup> (Summer) Ambient airport air: 400 µg/m <sup>3</sup> (Winter) 156 µg/m <sup>3</sup> (Spring) 216 µg/m <sup>3</sup> (Summer) <i>1.9–5.9 times higher particles number concentrations in the terminal buildings than measured in a normal urban environment</i> <i>Total UFP exposure during an entire average waiting period (including in the terminal building and airliner cabin) of a passenger was estimated to be equivalent to 11 h of exposure to normal urban emissions</i>	NA	Ren et al. (2018) <sub>b</sub> [90]
UFP monitoring at several sampling sites in the vicinity of Lisbon Airport for 19 non-consecutive days	Downwind average particle number concentration range: 3.3 × 10 <sup>4</sup> cm <sup>3</sup> to 5.9 × 10 <sup>4</sup> particles per cm <sup>3</sup> Measured range of peaks: 2.3 × 10 <sup>5</sup> particles per cm <sup>3</sup> to 3.4 × 10 <sup>5</sup> particles per cm <sup>3</sup>	NA	Lopes et al. (2019) [91]
Maximal measurements at a commercial airport and exposure assessment at a non-commercial airfield	10 <sup>6</sup> –10 <sup>8</sup> particles/cm <sup>3</sup> (main combustion events of plane leaving and arriving) 1086 µg/m <sup>3</sup> (single peak event of plane leaving) <i>10.7% was predicted to deposit in the alveolar lung regions</i>	<i>Personal exposure levels were similar to air concentrations</i>	Bendtsen et al. (2019) [36]
Maximal UFP number concentration of UFP exposures investigated for 33 male employees working in an airport taxiway	9.59 × 10 <sup>6</sup> (during support tasks in taxiing and taking off of the aircraft)	2.44 × 10 <sup>3</sup> particles/cm <sup>3</sup> <i>Median UFP number concentration</i>	Marcias et al. (2019) [92]
Contributions of airport activities to measured particle number concentrations (PNCs) at Amsterdam Schiphol were measured for 32 sampling days over 6 months	Mean total PNC: 35,308 particles/cm <sup>3</sup> <i>Aircraft departures and aircraft arrivals contributed to 46.1 and 26.7% of PNC, respectively. Ground support equipment and local road traffic accounted for 6.5% of PNC and were characterized by diameters of 60–80 nm. Traffic from surrounding freeways was characterized by particles of 30–40 nm and contributed to 18% of PNC</i> Mean PM <sub>2.5</sub> : 7.4 mg/m <sup>3</sup> Particle size range: 10–20 nm	NA	Pirhadi et al. (2020) [85]

mass spectrometry sampled for 24 h in three different locations of the airport showed general low levels, with highest levels of naphthalene (0.13–13.05 µg/m<sup>3</sup>), 2-methylnaphthalene (0.064–28.5 µg/m<sup>3</sup>), 1-methylnaphthalene (0.024–35.3 µg/m<sup>3</sup>), and biphenyl (0.024–1.610 µg/m<sup>3</sup>). Measured levels of the carcinogens benzo[b + j + k]fluoranthene and benzo[a]pyrene were 0.0542 µg/m<sup>3</sup> and 0.0086 µg/m<sup>3</sup> respectively [84].

Buonanno et al. (2012): Occupational exposure and particle number distributions were studied at an aviation base on a downwind site, close to the airstrip and by 10

daily UFP samples with personal monitors placed with a crew chief (assists the pilots during ground activities) and a hangar operator (aircraft maintenance). Particle number distribution averaged a total concentration of 6.5 × 10<sup>3</sup> particles/cm<sup>3</sup> at the downwind site. Short-term peaks during the working day mainly related to takeoff, landing and pre-flight operations of jet engines were measured in the proximity of the airstrip. Personal exposure concentrations were higher than stationary monitoring measurements. Personal exposure of workers were at a median number concentration of 2.5 × 10<sup>4</sup>



particles/cm<sup>3</sup> for the crew chief and  $1.7 \times 10^4$  particles/cm<sup>3</sup> for the hangar operator during the 2 months measurement period. The crew chief experienced the highest exposures, with maximum values at approximately  $8 \times 10^4$  particles/cm<sup>3</sup> [86].

Møller et al. (2014): Personal exposure monitoring of particle number concentration was carried out in five different occupational groups, namely baggage handlers, catering drivers, cleaning staff, airside security and land-side security in CPH, for 8 days distributed over 2 weeks. The study reported significant differences among the occupational groups. Highest exposures were found in baggage handlers (geometric mean:  $37 \times 10^3$  UFP/cm<sup>3</sup>), which was 7 times higher in average compared to land-side security which are indoor employees (geometric mean:  $5 \times 10^3$  UFP/cm<sup>3</sup>). In between highest and lowest exposure groups, were catering drivers, cleaning staff and airside security with similar exposure levels (geometric mean:  $12\text{--}20 \times 10^3$  UFP/cm<sup>3</sup>) [87].

Targino et al. (2017): Black carbon (BC) particle concentrations were measured within different micro-environments of 12 airports and on 41 non-smoking commercial flights. Great variability was seen depending on environment measured. 70% of personal exposure during a journey occurred in the airport concourses and during transit to/from the aircraft. 18% was contributed to the waiting time onboard an aircraft with open doors waiting for loading. Largest BC exposure were found during boarding and disembarking (mean BC =  $3.78 \mu\text{g}/\text{cm}^3$ ; 25th, 50th, 75th percentiles: 1.29, 2.15, 4.68), at the airport concourse (mean BC =  $3.16 \mu\text{g}/\text{cm}^3$ ; 25th, 50th, 75th percentiles: 1.20, 2.15, 4.0) and inside parked aircraft with open doors (mean BC =  $2.78 \mu\text{g}/\text{cm}^3$ ; 25th, 50th, 75th percentiles: 0.35, 0.72, 2.33). BC levels were low in the aircraft on the ground with closed doors (mean BC =  $0.81 \mu\text{g}/\text{cm}^3$ ; 25th, 50th, 75th percentiles: 0.2, 0.35, 0.72, respectively). Lowest concentration was found during flights in the air [41].

Ren et al. (2018)<sup>a</sup>: The number concentrations and size distributions inside the cabin of an aircraft waiting for take-off were investigated and analyzed in comparison to outdoor UFP and the use of the ground air-conditioning cart (GAC) and environmental control system (ECS), which are used to provide conditioned air between boarding and doors closing to prepare for take-off. The study showed that environmental particle number concentration varied significantly, ranging from 10 to  $40 \times 10^3$  particles/cm<sup>3</sup> depending on wind, and take-off and landing activities. When the GAC was on, the indoor particle numbers followed those outdoors, with the ECS providing protection factors for crew and passengers from 1 to 73% for 15–100 nm particles, and from 30 to 47% for 100–600 nm particles. A 40 min wait 100 m downwind of the runway was calculated to be equal to 4

h exposure in a clean urban background environment away from the airport [89].

Ren et al. (2018)<sup>b</sup>: In this study, the potential exposure to passengers as well as indoor airport staff was investigated by measurements in the terminal building of Tianjin Airport in Beijing of CO<sub>2</sub>, PM<sub>2.5</sub>, and UFP concentration and particle size distribution during three seasons. The effects on the indoor air quality of airliner-generated particles penetrating from the outdoor environment through open doors and by heating, ventilation and air-conditioning systems was studied.

PM<sub>2.5</sub> concentrations in the terminal building varied during the seasons of winter, spring and summer with 337–105–167  $\mu\text{g}/\text{m}^3$  in the arrival hall, 385–130–170  $\mu\text{g}/\text{m}^3$  in the departure hall, and 400–156–216  $\mu\text{g}/\text{m}^3$  in ambient airport air, respectively. These were significant higher levels compared to Chinese standard and WHO annual mean value of 10  $\mu\text{g}/\text{m}^3$  during all the tested seasons. The indoor environment was significantly affected by the outdoor air levels (Spearman:  $p < 0.01$ ). Particle number concentration in the terminal building displayed two size distribution, with one mode at 30 nm and a mode at 100 nm, which was significantly different from the size distribution measured in a normal urban environment, which had one peak at 100 nm. The study reports particle number concentrations of 1.9–5.9 times higher in the terminal buildings than the concentrations measured in a normal urban environment by different size bins. Measured total UFP exposure during an entire average waiting period (including in the terminal building and airliner cabin) of a passenger was estimated to be equivalent to 11 h of exposure to normal urban emissions [90].

Bendtsen et al. (2019): In this study, the occupational exposure levels to particles was evaluated by measurements at a non-commercial airfield and particles were collected and characterized at a non-commercial airfield and from the apron of a commercial airport.

Electron microscopy showed that the aerosol at the non-commercial airfield appeared to be mainly aggregates of soot, whereas the aerosol at the apron of the commercial airport appeared much more complex dominated by agglomerated soot particles, salt crystals and pollen. At the commercial airport, particles were mainly below 300 nm in diameter and distributed in two modes with geometric mean diameters of < 20 nm and approximately 140 nm. At the non-commercial airfield, two full cycles of a normal workflow of plane leaving, plane arriving and refueling by were recorded in a jet shelter using stationary and portable devices including in the breathing zone of personnel. Average particle number concentration for a full workflow cycle of 170 min were  $1.22 \times 10^6$  particles/cm<sup>3</sup>. For take-off and landing of one jet plane, average particle number concentrations and mass were 7.7 particles/cm<sup>3</sup> and 1086  $\mu\text{g}/\text{m}^3$  and 2.67

particles/cm<sup>3</sup> and 410 µg/m<sup>3</sup>, respectively. During the main combustion events of plane leaving and arriving, the instruments reached their upper detection limits of 10<sup>6</sup> particles/cm<sup>3</sup> (DiSCmini, which measures particle number concentration, mean particle size and lung-deposited surface area) and 10<sup>8</sup> particles/cm<sup>3</sup> (ELPI, which monitors real-time particle levels), including in the breathing zone monitor of the personnel. Prevalent particle sizes suggested that the jet engine combustion particles were < 10 nm in aerodynamic diameter [36].

Mokalled et al. (2019): In this study, 48 volatile organic compounds (VOC) from approximately 100 commercial aircraft during real operations of different engine modes at Beirut Rafic Hariri International Airport were assessed to identify specific markers, together with measurements of Jet A-1 kerosene fuel vapors and gasoline exhaust.

Heavy alkanes (C8-C14, mainly n-nonane and n-decane) contributed to 51–64% of the total mass of heavy VOCs emitted by aircraft. Heavy aldehydes (nonanal and decanal) was reported as potential tracers for aircraft emissions due to their exclusive presence in aircraft-related emissions in combination with their absence from gasoline exhaust emissions. Total concentration of heavy alkanes in the ambient air was 47% of the total mass of heavy VOCs measured. No aircraft tracer was identified among the light VOCs (≤ C7). VOC compositions in jet exhaust varied with combustion power, and it was shown that light VOC emissions decrease as the engine power increases. Auxiliary power unit (APU) emissions were identified to be of the same order of magnitude as main engine emissions [93].

Marcias et al. (2019): In this study, occupational exposure to ultrafine particles and noise was investigated for 33 male employees working in an airport taxiway in a smaller Italian airport. Job categories represented were aircraft ground equipment personnel, firefighting officer, flight security agent, and aviation fuel administration staff. Both stationary sampling (ELPI) and personal particle measurements were included. The morphology and chemical composition was determined by EM and EDS, and showed small soot particles in aggregates with sodium, potassium, magnesium, calcium, aluminium, carbon, nitrogen, silicon, oxygen, fluorine, chlorine and sulphur. The maximal UFP number concentration ( $9.59 \times 10^6$  particles/cm<sup>3</sup>) on stationary equipment was measured during support tasks in taxiing and taking off of the aircraft. Median UFP number concentration measured with personal monitors on the 33 operators was  $2.44 \times 10^3$  particles/cm<sup>3</sup> and a maximum of  $13 \times 10^3$  particles/cm<sup>3</sup>. Average size range was 35–103 nm. A significant difference in mean size and distributions was found between job tasks, where flight security officers were exposed to particles with lower mean sizes as compared to aircraft ground equipment operators [92].

### Residential exposure

Westerdahl et al. (2008): Air measurements were carried out in the vicinity of LAX to assess the spread of airport emissions in downwind ambient air to the immediate neighborhood. Ultrafine particle numbers (UFP), size distributions, particle size, black carbon (BC), nitrogen oxides (NO<sub>x</sub>), and particle-bound PAH were measured. The lowest levels of pollutants were measured upwind of the airport, where UFP ranged from 580 to 3800 particles/cm<sup>3</sup>, black carbon from 0.2 to 0.6 µg/m<sup>3</sup>, and particle-bound PAH from 18 to 36 ng/m<sup>3</sup>. In contrast, at 500 m downwind of the airport, average UFP counts of 50,000 particles/cm<sup>3</sup> were observed, which were significantly influenced by aircraft operations where peaks were observed. Black carbon, particle-bound PAH, and NO<sub>x</sub> were also elevated, although not in the same extent, and the authors observed that BC, particle numbers, and NO<sub>x</sub> levels varied together in similar patterns indicating they were associated with similar sources. Black carbon concentrations varied across the measurement sites, with a mean of 0.3 µg/cm<sup>3</sup> upwind from the airport, 0.7 µg/cm<sup>3</sup> downwind from the airport, 1.8 µg/cm<sup>3</sup> at the taxiway, and 3.8 µg/cm<sup>3</sup> in the terminal region. Mean PM-PAH levels were 18.2, 24.6, 50.1 and 60.1 ng/m<sup>3</sup> at the measurement sites, respectively. PM-PAH mean levels measured on two freeways were 47.0 ng/m<sup>3</sup> and 169.4 ng/m<sup>3</sup>. The maximum UFP measured was  $4.8 \times 10^6$  particles/m<sup>3</sup> downwind from a jet aircraft taking off. NO<sub>x</sub> levels before the take-off were around 8 ppb and increased to 1045 ppb, mostly due to NO. Black carbon rose from approximately 800 to 9550 ng/m<sup>3</sup>, and PM-PAH values increased from 37 to 124 ng/m<sup>3</sup>. Significant variations were observed in particle sizes, where upwind measurements were dominated by particles of 90 nm, and downwind particles were of 10–15 nm in size. The author noted that UFP levels from aircraft were measured to persist up to 900 m from the runways, indicating potential risks for the nearby communities [34].

Lopes et al. (2019): In this study, data is presented from UFP monitoring at several sampling sites in the vicinity of Lisbon Airport in 2017 and 2018, for 19 non-consecutive days. Measurements included sites further away from the airport, under the landing/take-off path. Correlation analysis between air traffic activity and UFP concentrations was conducted and show the occurrence of high UFP concentrations in the airport vicinity. The particle counts increased 18–26 fold at locations near the airport, downwind, and 4-fold at locations up to 1 km from the airport. Results show that particle number increased with the number of flights and decreased with the distance to the airport [91].

Pirhadi et al. (2020): In this study, the contributions of airport activities to particle number concentrations (PNCs) at Amsterdam Schiphol was quantified by use of

the positive matrix factorization (PMF) source apportionment model. Various pollutants were measured, including NO<sub>x</sub> and CO, black carbon, PM<sub>2.5</sub> mass, and the number of arrivals and departures were measured for 32 sampling days over 6 months. Airport activities accounted for 79.3% of PNCs divided in aircraft departures, aircraft arrivals, and ground service equipment (GSE) (with contributions of local road traffic, mostly from airport parking areas). Aircraft departures and aircraft arrivals contributed to 46.1 and 26.7% of PNCs, respectively, and were characterized by particle diameters < 20 nm. GSE and local road traffic accounted for 6.5% of the PNCs and were characterized by diameters of around 60–80 nm. Traffic from surrounding freeways was characterized by particles of 30–40 nm and contributed to 18% of PNCs. In comparison, the urban background emissions dominated the mass concentrations with 58.2%, but had the least contribution to PNCs with 2.7% [85].

### Summary of exposure studies

Occupational exposure to increased levels of nanosized particles [36, 85–90, 92], increased levels of PAH including known human carcinogens [52, 83, 84], and black carbon [41] were reported in the literature. Levels of exposure reported in these studies are summarized in Table 1. One study reported that personnel monitors measured higher levels compared to stationary equipment [87], and it was shown that ground support equipment, such as diesel-powered electrical generators and heaters [83] and auxiliary power units [93] contribute significantly to emissions.

Three important main factors were identified which significantly influenced occupational exposure: *proximity to emission sources*, where levels were generally higher in close proximity and down-wind to aircraft, *fluctuations in emission levels*, characterized by exposure peak events such as landing- or take-off, and *job type*, where outdoor ground-affiliated work types are at highest risk of exposure. As such, airport personnel can likely be grouped in low (office staff/landside jobs with indoor work, far away from emission sources), medium (catering/cleaning/landside security staff with intermittent outdoor work) and high (baggage handlers/aircraft mechanics/ crew chief) exposure groups.

The majority of studies on the contribution of airport emissions to air pollution in the surrounding environment are physical/chemical studies of particle numbers, mass and related air pollutants, which are reviewed elsewhere as previously described.

More studies reported increased risk of exposure correlating with decreased distance to airports [94–96] and time spent downwind from an airport [97], hence a significant factor for potential health effects for neighboring

residential areas based on these studies is *distance to airports*, which relating to wind and atmospheric conditions is an important determinant for pollution levels.

### Health effects

Here we present studies in which direct health effects have been assessed in humans, including in biomonitoring and epidemiological studies, and biological mechanisms-of-action assessed in animal or cell studies. Our main focus is particle exposure, however, studies focusing more on VOC/PAH are also presented.

### Occupational studies

Møller et al. (2017 and 2019): A prospective, occupational cohort study in CPH, encompassing 69,175 men in unskilled positions as baggage handlers or in other outdoor work used register information of socioeconomic, demographic and health data together with a job-exposure matrix was based on GPS measurements within the airport, detailed information on tasks from 1990 to 2012, exposure to air pollution at home, and lifestyle details. Occupational exposure groups were categorized according to work time at the apron, “apron-years” (non-exposed, 0.1–2.9, 3.0–6.9 and ≥ 7 years). The reference group comprised different low-exposure occupational groups [98]. A follow-up study was conducted on an exposed group of 6515 male airport workers at 24–35 years of age in unskilled positions with a reference group of 61,617 men from greater Copenhagen area in unskilled jobs. Exposure was assessed by recordings of time spent on the airport apron and diagnoses of ischemic heart disease and cerebrovascular disease was obtained from the National Patient Register. No associations between cumulative apron-years and the two disease outcomes were found. On the other hand, since the exposed group had a mean age of 24–35 years, a 22-year follow-up may have been too short to detect cardiovascular effects [99].

Lemasters et al. (1997): In this early study, mixed low-level exposure to fuel and solvent was studied in a repeated measures design with male aircraft workers at a military air station serving as their own controls from pre-exposure to 30 weeks post-exposure. The study group consisted of six aircraft sheet metal workers mainly exposed to solvents, adhesives and sealants, six aircraft painters exposed to solvents and paints, 15 jet fueling operations personnel ( $n = 15$ ) responsible for fuel delivery, fueling and defueling aircraft and repairing fuel systems, and 23 workers in the flight line crew exposed to jet fuel, jet exhaust, solvents and paint, and included ground crew and jet engine mechanics. Expired breath analysis was carried out for different trace compounds, but was found to have low values (< 25 parts per billion). An increase in sister chromatid exchange (SCE)

compared to pre-exposure was found after 30 weeks of exposure for sheet metal workers (mean SCE per cell increased from 6.5 (SD: 0.8, range: 5.5–7.7) to 7.8 (SD: 0.3, range: 7.4–8.2) and painters (mean SCE per cell increased from 5.9 (SD: 0.7, range: 5.0–6.8) to 6.7 (SD: 1.0, range 5.3–7.8)), indicating exposure to genotoxic substances for these subgroups [100].

Tunncliffe et al. (1999): In Birmingham International Airport, occupational exposure to aircraft fuel and jet stream exhaust was evaluated in terms of respiratory symptoms and spirometry in 222 full-time employees according to job title. Data was collected by questionnaire and with on-site measurement of lung function, skin prick tests, and exhaled carbon monoxide concentrations. Occupational exposure was assessed by job title, where baggage handlers, airport hands, marshallers, operational engineers, fitters, and engineering technicians were considered as high exposure groups, security staff, fire fighters, and airfield operations managers as medium exposure group, and low exposure groups consisted of terminal and office workers. Upper and lower respiratory tract symptoms were commonly reported in the questionnaire and 51% had one or more positive allergen skin tests. Cough with phlegm and runny nose were found to be significantly associated with high exposure (adj. OR = 3.5, CI: 1.23–9.74; adj. OR = 2.9, CI: 1.32–6.4, respectively). Upper and lower respiratory symptoms were common among exposed workers, but no significant difference was found in lung function. The authors conclude that it is more likely that these symptoms reflect exposure to exhaust rather than fuel [101].

Yang et al. (2003): The aim of this study was to evaluate self-reported adverse chronic respiratory symptoms and acute irritative symptoms among 106 airport workers in risk of exposure to jet fuel or exhaust (jet fuel handlers, baggage handlers, engineers etc.) compared to 305 terminal or office workers (control group) at Kao-hsiung International Airport (KIA) in Taiwan. The odds ratio analyses were adjusted for possible confounding factors, such as age, marital status, education, duration of employment, smoking status, and previous occupational exposure to dust or fumes. The prevalence of acute irritative symptoms was not significantly different, whereas chronic respiratory symptoms such as cough (adj. OR = 3.41, CI: 1.26–9.28) and dyspnea (adj. OR = 2.34, CI: 1.05–5.18) were significantly more common among airport workers. The study did not report exposure measurements, but the authors conclude that the expected higher exposure of aviation fuel or exhaust in the ground personnel is the likely explanation for the increased incidence of self-reported chronic respiratory health-effects compared to the office personnel [102].

Whelan et al. (2003): Prevalence of respiratory symptoms among female flight attendants along with teachers

was investigated by self-reported questionnaire in comparison to database-derived data on blue collar workers with no known occupational exposures, and it was found that female flight attendants and teachers were significantly more likely to report work related eye (12.4 and 7.4%), nose (15.7 and 8.1%), and throat symptoms (7.5 and 5.7%), and more episodes of wheezing and flu, compared to other female workers (2.9% eye, 2.7% nose, and 1.3% throat symptoms). Female flight attendants were significantly more likely than teachers and controls to report chest illness 3 years in retrospective (flight attendants: 32.9%, teachers: 19.3%, female workers: 7.2%) [103].

Cavallo et al. (2006): In this study, 41 airport employees in jobs with very close proximity to aircraft in service (fitters, airport hands, marshallers, baggage handlers) or in jobs with some proximity to aircraft (security staff, maintenance service personnel, cleaning staff, air field operations managers, runway shuttle drivers) in Leonardo da Vinci airport in Rome were evaluated for exposure to aircraft emissions along with biomarkers of genotoxicity in comparison to a control group of 31 office workers at the same airport. Job tasks in very close proximity to aircraft in service were considered to be high exposure jobs. Urinary PAH metabolites were used as biomarker of endogenous PAH exposure in parallel with PAH analyses of air samples. Exfoliated buccal cells and blood were evaluated for DNA damage, e.g. micronuclei, chromosomal aberrations and sister chromatid exchange (SCE). PAH exposure was measured during 24 h of 5 work days at the airport apron, airport building and terminal/office area from January to February 2005. Total mean of 23 PAHs (particle and vapour) at the apron, airport building and terminal departure area were 27.7, 17.2, and 9.5  $\mu\text{g}/\text{m}^3$ , respectively, with a prevalence of 2–3 ring PAHs with highest levels in the airport apron particularly for 1- and 2-methylnaphthalene and acenaphthene. Urinary PAH metabolite levels were similar for high exposure job groups and controls. The exposed group showed increased SCE (mean number:  $4.61 \pm 0.80$ ) compared to control group ( $3.84 \pm 0.58$ ) and increased levels of chromosomal aberrations and DNA strand breaks in the Comet assay in both buccal cells and lymphocytes, indicating genotoxic exposures [52].

Radican et al. (2008): A follow-up study of 14,455 workers from 1990 to 2000 evaluated the mortality risk from trichloroethylene and other chemical exposures in aircraft maintenance workers. Relative risk (RR) for exposed compared to unexposed workers were calculated, and positive associations with several cancers were observed, but mortality had not changed substantially since 1990, with increased risk of all-cause mortality (RR = 1.04, CI: 0.98–1.09) or death from all cancers (RR = 1.03, CI: 0.91–1.17) [104].



Erdem et al. (2012): A study group consisting of 43 aircraft fuel maintenance staff, fuel specialists, and mechanics occupationally exposed to JP-8 fuel directly or via engines of jet planes were evaluated for the metabolites 1- and 2-naphthol and creatinine in urine as biomarkers of exposure to jet fuel. In turn, sister chromatid exchange (SCE) and micronuclei were evaluated in blood-derived lymphocytes as biomarkers of genotoxic exposure. Urinary markers and SCE were significantly increased in exposed workers (1-naphthol: 99.01  $\mu\text{mol/mol}$  creatinine; 2-naphthol: 77.29  $\mu\text{mol/mol}$  creatinine), by 10-fold as compared to a control group of 38 employees working in the same area without any work-related exposure to JP-8 fuel [105].

Marie-Desvergne et al. (2016): In this study, exposure to airport nanoparticles and metals was evaluated in airport workers by exhaled breath condensate (EBC) as a non-invasive representative of the respiratory system. EBC was collected from 458 airport workers from Marseille Provence Airport and Roissy Charles de Gaulle Airport in Paris, working directly on the apron (exposed) or in the offices (less exposed). In addition, ambient nanoparticle exposure levels were characterized in terms of particle number concentration, size distribution and by electron microscopy.

The study showed that airport workers were exposed to significantly higher particle numbers ( $1.0 \times 10^4$ – $2.1 \times 10^7$  particles/ $\text{cm}^3$ ) compared to office staff ( $10^3$ – $10^4$  range equivalent to background traffic emissions), although office workers were periodically exposed to peaks of  $10^4$ – $10^5$  when the building doors were open. Airport workers were exposed to significantly smaller particles (mean geometric size: 17.7) compared to office workers (mean geometric size: 23.7). EBC was characterized by volume, total protein content, and a multi-elemental analysis was used to.

measure Na, Al, Cd, and Cr. Particles in EBC were analyzed with dynamic light scattering and electron microscopy (SEM-EDS).

A significantly higher concentration of Cd was found in apron worker EBC (mean:  $0.174 \pm 0.326 \mu\text{g/l}$ ) in comparison with office workers (mean:  $0.108 \pm 0.106 \mu\text{g/l}$ ). Particulate content in EBC was confirmed by DLS and SEM-EDS, but no differences were found between the two study groups, and measured EBC particle contents did not correlate with ambient exposure levels [88].

#### Studies on effects of residential exposure to airport emissions

Visser et al. (2005): In this population-based study, it was investigated if the residents living around Amsterdam Schiphol Airport were at higher risk of developing cancer compared to the general Dutch population. The regional cancer registry was used, estimating the cancer incidence from 1988 to 2003 in the

population residing near the airport compared to the national cancer incidence. The exposure was defined by aircraft noise and postal code areas, as historical data on ambient air pollution were unavailable. The study did not include information on lifestyle factors, and therefore, did not control for smoking and other potential confounders. A core zone closest to the airport and a remaining ring zone was studied. Thirteen thousand two hundred seven cancer cases were identified in the study area, and a significant increase in the incidence of hematological cancers (standardized incidence ratio, SIR = 1.12, CI: 1.05–1.19) was found, mainly due to non-Hodgkin lymphoma (SIR = 1.22, 95% CI: 1.12, 1.33) and acute lymphoblastic leukemia (SIR = 1.34, CI: 0.95, 1.83). Respiratory system cancer incidence was significantly decreased (SIR = 0.94, CI: 0.90, 0.99), due to the low rate in males (SIR = 0.89). The study concludes that the overall cancer incidence in the residential areas closest to Amsterdam Schiphol Airport was similar to the national incidence. The increase in the risk of hematological cancers could not be explained by higher levels of ambient air pollution in the area [106].

Lin et al. (2008): In this cross-sectional study, it was assessed whether residents living near commercial airports had increased rates of hospital admissions due to respiratory diseases compared to those living further away. The study included all residents living within 12 miles from the center of each of three airports (Rochester in Rochester, LaGuardia in New York City and MacArthur in Long Island). Hospital admission data were collected by the New York State Department of Health for all residents who were hospitalized for asthma, chronic bronchitis, emphysema, chronic obstructive pulmonary disease and, for children aged 0–4 years, bronchitis and bronchiolitis during 1995–2000. Exposure indicators were distance from the airport and dominant wind patterns from the airports.

The relative risks of hospital admissions due to respiratory conditions for residents living < 5 miles from the airport were 1.47 (CI: 1.41–1.52) for Rochester and 1.38 (CI: 1.37–1.39) for LaGuardia, as compared to those living > 5 miles from the airports. No differences were observed for MacArthur airport. When considering hospital admission rates by distance for 12–1 miles towards the airports, a significant trend of increasing hospital admissions with closer distance to the airport was observed for the Rochester airport. The authors reported a stronger effect for traditionally lower socio-economic groups [94], which may be of more relevance in the US, due to the medical insurance system.

Habre et al. (2018): In this study, 22 non-smoking volunteers with mild to moderate asthma were recruited to do scripted mild walking activity in parks inside or outside a zone of high airport-related ultrafine particle

exposure downwind of LAX. Physiological parameters were measured before and after exposure, and the study was conducted as a cross-over study, such that the participants served as their own controls. Personal exposure to black carbon, PAH, ozone, and PM<sub>2.5</sub> were measured and combined with source appointment analysis and health models. A difference in PM exposure was found between the high (mean particle number concentration of 53,342 particles/cm<sup>3</sup> and mean particle size of 28.7 nm) and the low exposure zone (mean particle number concentration of 19,557 particles/cm<sup>3</sup> and mean particle size of 33.2 nm). It was reported that IL-6 levels in blood were increased after the walk in the high exposure zone compared to the low exposure zone. Airport-related PM was distinguished from roadway traffic emissions by principal component analysis, and increase of airport-related PM was significantly associated with increased IL-6 levels [107].

Amsterdam Schiphol report (2019): Based on three studies with 191 primary school children from residential areas near Schiphol Airport, 21 healthy adults living adjacent to the airport [108], and an in vitro study [109], respectively, this Dutch report (not subjected to peer review) describes the findings of reduced lung function in children and adults following higher short-term exposure to ultrafine particles near Schiphol Airport. On days with high exposure, children suffered more from respiratory complaints and used more medicine. In the adults, short-term reductions in heart function were also found. The authors note that these effects may be larger for individuals already suffering from medical conditions. The authors point out that the effects are results of ultrafine particles from both air and road traffic, and that there are no indications that health effects of air traffic emissions are different from those caused by road traffic [59].

Lammers et al. 2020: This study investigated the health effects of controlled short-term exposure of 21 healthy non-smoking volunteers aged 18–35 years to UFP near Schiphol Airport Amsterdam. The volunteers were exposed 2–5 times to ambient air for 5 h while cycling. Cardiopulmonary outcomes such as spirometry, forced exhaled nitric oxide, electrocardiography and blood pressure were measured before and after exposure, and compared to measured total- and size-specific particle number concentrations (PNC). Average PNC was 53,500 particles/cm<sup>3</sup> (range 10,500–173,200). Increase in exposure to UFP was associated with a decrease in FVC and a prolongation of the corrected QT interval, which were associated with particle sizes < 20 nm (UFP from aviation), but not with particles > 50 nm (UFP from road traffic). Although the effects were relatively small and measured after single exposures of 5 h in young healthy adults [108], such effects could be important in susceptible sub-populations.

#### Animal studies and in vitro studies

Ferry et al. (2011): Immature primary human monocyte-derived dendritic cells (DCs) from healthy donor blood were exposed for 18 h to different doses of experimental jet exhaust particles in absence or presence of *E. coli* lipopolysaccharides (LPS). Antigen-presenting and stimulatory molecules were measured along with tumor necrosis factor (TNF $\alpha$ ) and IL-10. The effects were assessed on immature and mature DCs as well as on cells during the maturation process.

The primary particles collected from the jet exhaust by direct impaction were found to be spherical and carbonaceous primary particles of ~ 10 nm and aggregates up to ~ 93 nm. No toxic effects were observed for doses below of 100  $\mu$ g/mL jet engine particles. Maturation of immature dendritic cells by LPS stimulation induced a significant 500-fold increase in TNF $\alpha$  and 30-fold increase in IL-10. Immature dendritic cells produced low amounts of TNF $\alpha$  (fold change from LPS: 0.006) and IL-10 (fold change from LPS: 0.11), which increased non-significantly upon stimulation with particles (fold change from LPS: TNF $\alpha$ : 0.11, IL-10: 0.19). However, simultaneous exposure to LPS and a high particle dose of 100  $\mu$ g/mL induced a 2-fold increase in TNF $\alpha$  production compared to LPS-maturation ( $p = 3 \times 10^{-5}$ ). Different activation patterns were seen for the expression of HLA DR and CD86, which are dendritic cell maturation markers. It was concluded that jet exhaust particles may act as adjuvants to endotoxin-induced dendritic cell maturation, which may influence potential effects on human health [110].

Shirmohammadi et al. (2018): PM<sub>0.25</sub> collected at the vicinity of Los Angeles Airport (LAX) and from central Los Angeles (LA) close to and downwind from major freeways, from stationary sampling stations used for air quality control, were investigated. The particles were subjected to source allocation analyses of elements and carbon contents (see Introduction), and ROS formation was compared in rat alveolar macrophage cells (NR8383).

ROS activity measured as units of Zymosan equivalents were normalized by total PM<sub>0.25</sub> mass to represent the intrinsic toxicity of the particles, and this mass-normalized ROS activity was similar for LAX (4600.93  $\pm$  1516.98  $\mu$ g Zymosan/mg PM) and central LA (4391.22  $\pm$  1902.54  $\mu$ g Zymosan/mg PM). According to the authors, volume-normalization of the ROS activity can be used as a metric for comparison of inhalation exposures, as an indicator of exposure severity. A slightly higher PM<sub>0.25</sub> mass concentration in central LA meant overall similar volume-normalized ROS activity levels with no significant difference between the observed averages (LAX: 24.75  $\pm$  14.01  $\mu$ g Zymosan/m<sup>3</sup>, central LA: 27.77  $\pm$  20.32  $\mu$ g Zymosan/m<sup>3</sup>). Thus, there were similar levels of ROS activity and similar toxic potential of the PM in the vicinity of LAX and in the vicinity of freeways in central LA [49].

He et al. (2018): PM<sub>0.25</sub> collected at Los Angeles Airport (LAX) and from central Los Angeles (LA) close to and downwind from major freeways (similar collection sites as in [49]) were investigated and compared. Particles were source-allocated by analyzing elements (see Introduction). Particles collected at LAX were primarily associated with aircraft emissions, and particles from central LA with urban traffic, road and dust emissions. The reactive oxygen species (ROS) potential was evaluated intracellularly in human bronchial epithelial cells (16HBE) after 1, 2, and 4 h of exposure, and IL-6, IL-8 and TNF were measured as markers of inflammation.

Exposure of 16HBE cells to 10 µg/mL particles produced significantly elevated ROS levels for both samples compared to unexposed cells. Particles from central LA generated slightly more ROS than LAX samples per mass unit, and both were at negative control level after 20 h recovery. ROS potential in PM from both airport and central LA correlated with some of the measured traffic-related transition metals (Fe and Cu). Particles from LAX induced increased expression of IL-6, IL-8 and TNFα compared to the negative control (1.7, 1.8, and 1.4-fold, respectively), whereas central LA-particles induced slightly lower expressions (1.3, 1.3, and 1.1-fold, respectively). Hence, overall LAX particles had similar inflammatory potency as particles from central LA, showing that airport PM<sub>0.25</sub> contributions to urban emission PM pollution possess similar inflammatory properties [50].

Jonsdottir et al. (2019): In this study, aerosol was collected from the world's most used aircraft turbine (CFM56–7B26, run-in and airworthy) in a test cell at Zurich Airport. The test cell is open to the ambient environment and the aerosol was collected from both standard Jet A-1 fuel and a HEFA fuel blend. The toxicity of the non-volatile PM emissions was studied by direct particle deposition onto air-liquid interface cultures of human bronchial epithelial cells (BEAS-2B).

Cytotoxicity was evaluated by the release of cytosolic LDH from damaged cells, expression of the oxidative stress marker HMOX-1 and inflammatory cytokines IL-6 and IL-8.

Single, short-term (1 h) exposure to PM increased cell membrane damage, lead to oxidative stress and increased pro-inflammatory cytokines in bronchial epithelial cells, depending on fuel type and combustion conditions from which the particles were produced. PM from conventional fuel at ground-idle conditions was most potent, and the authors comment that PM from aircraft turbine exhaust may be a risk to respiratory health, also by making airway epithelia vulnerable to secondary exposure of other air pollution compounds and pathogens [111].

Bendtsen et al. (2019): In this study, the toxicity of particles collected in a commercial and a non-commercial airport were evaluated in vivo by intratracheal instillation in

mice (see section 2.3 for occupational exposure measurements). Adult female C57BL/6 mice were exposed to 6, 18, and 54 µg particles/mouse dispersed in Nanopure water by sonication. The exposure doses were calculated on the basis of worst case scenario: of the maximum exposure level measured at the non-commercial airport of 1086 µg/m<sup>3</sup> at the peak event of plane departure, 9.6% were estimated to deposit in the alveolar lung regions. This was adjusted to the volume of a mouse lung and to 8 h of work, estimating exposure of 4, 12, and 39 days of work, respectively. Control mice were exposed to Nanopure water, and positive controls were carbon black Printex90 nanoparticles and SRM2975 diesel particles. Exposed mice were euthanized on day 1, 28, and 90 post-exposure. Inflammation was measured as inflammatory cell influx in bronchoalveolar lavage fluid as well as by the acute-phase response marker *serum amyloid A (Saa)* in lung (mRNA), liver (mRNA) and blood (protein). Genotoxicity was assessed by the comet assay on lung and liver tissue and cells from the bronchoalveolar lavage fluid. Analysis of the particles by scanning and transmission electron microscopy showed small primary particles and agglomerates of soot, which appeared uniform for non-commercial airport particles (mainly from jet engine emissions) and more heterogeneous for the commercial airport particles (emissions from aircraft, ocean, traffic and background). Pulmonary exposure to particles from both airports induced genotoxicity and dose-dependent acute phase response, and inflammation at same levels as standard diesel exhaust particles and carbon black nanoparticles [36].

He et al. 2020: In this study, UFPs from aviation or road traffic emissions were collected near the major international airport, Amsterdam-Schiphol airport (AMS), along with UFPs from an aircraft turbine engine at low and full thrust. The toxicity of the particles was tested in human bronchial epithelial cells (Calu-3) combined with an air-liquid interface (ALI) system with exposure to UFPs at low doses from 0.09 to 2.07 µg/cm<sup>2</sup>. Cell viability, cytotoxicity and IL-6 and -8 secretion were assessed after 24 h exposure. Cell viability was < 80% for all doses. LDH release as measure of cytotoxicity was observed at the highest exposure dose around 1.5 µg/cm<sup>2</sup> together with increased production of IL-6 and IL-8 compared to control exposure (blank filter extraction or re-suspension solution). It was concluded that airport and road traffic UFP as well as UFP samples from the turbine engine had similar inflammatory properties [109].

### Summary of health effect studies

Increased levels of metabolites in urine as biomarkers of internal exposure to jet fuel [105] were reported in biomonitoring studies of occupational exposure to airport emissions. Exposure to airport emissions was associated with increased levels of biomarkers of genotoxicity, in

terms of increased levels of SCE [52, 100, 105] and DNA strand breaks in the Comet assay [52], which indicates exposure to genotoxic and potential carcinogenic agents in the emissions. In turn, there were occupational studies reporting increased levels of self-reported respiratory complaints [101–103].

We identified a limited number of studies and one report reporting correlations between airport emission levels and health effects of residents in the vicinity of airports: Aircraft emission levels were associated with increased hospitalization for asthma, respiratory, and heart conditions especially in susceptible subgroups such as children below 5 years of age, elderly above 65 years of age [66, 94] and lower socioeconomic groups [97, 112]. A Dutch report on Schiphol similarly reported that school children and adults took more medication and had more respiratory complaint on days with increased exposure to aircraft emissions and concludes that health effects of air traffic emissions are similar to those caused by road traffic [59]. A biomonitoring study showed increased blood levels of the inflammatory marker IL-6 in volunteers with mild to moderate asthma after a walk in a zone with high levels of aircraft emissions [107]. It is well-known that other types of air pollution including diesel exhaust cause morbidity and mortality [113]. Taken together, these results suggest that the exposure to aircraft emissions induce pulmonary and systemic inflammation, which potentially contributes to cancer, asthma, respiratory and coronary heart disease.

Five mechanistic studies on the toxicity of airport particles were identified, one animal study in mice and four cell studies: Airport particles were reported to act as adjuvants in the activation of inflammatory cells or pathways [110] and induce pro-inflammatory cytokines [111]. Airport particles were shown to have similar inflammatory potency and similar ability to induce DNA damage as traffic emission particles [50], such as diesel exhaust particles [36]. In turn, airport particles induced significant levels of the biomarker Saa following intratracheal instillation in mice, associated with risk of cardiovascular disease [36], and they have the potential to generate ROS at similar levels as traffic emission particles [49, 50]. Thus, the conclusions from these in vitro and in vivo studies support the overall concern addressed in previous sections that airport emission particles are capable of inducing toxic responses comparable to the responses observed for other air pollution particles such as diesel exhaust particles.

## Discussion

Although a range of kerosene-based aircraft fuel types are in use, they are overall similar in chemical composition [24, 29]. Kerosene lies between the distilled crude oil fractions of gasoline (gasoline combustion exhaust,

IARC group 2b) and diesel (diesel combustion exhaust, IARC group 1) and the carcinogenic potential of jet fuel combustion products could be anticipated given the reported similarities to diesel exhaust particles. We highlight two important reported characteristics of airport particles:

- The majority of non-volatile airport emission particles are carbonaceous and aircraft engines emit large amounts of nanoparticles, which are dominated by very small particles of < 20 nm, which form aggregates/agglomerates in ambient air
- Particle numbers near airports are significantly higher than away from airports and jet engines are a significant source of UFP in ambient air. The highest concentrations of UFP are measured downwind of aircraft

The reported PAH levels [52, 83, 84] were all below the current Danish occupational exposure limit of 200  $\mu\text{g}/\text{m}^3$ . One study reported BC levels at the apron of 3.78  $\mu\text{g}/\text{m}^3$  and particle levels was overall reported to be between  $\sim 10^3$  and  $10^8$  particles/ $\text{cm}^3$  for exposed airport personnel (Table 1). The new exposure limit for diesel exhaust particles in EU is defined by the elemental carbon (EC) level and is 50  $\mu\text{g}$  EC/ $\text{m}^3$  [114]. The Netherlands recently endorsed an OEL for diesel exhaust particles at 0.01 mg/ $\text{m}^3$  measured as respirable EC. This was based on socioeconomic considerations and the Dutch prohibition risk level (OEL) is at 1.03  $\mu\text{g}$  EC/ $\text{m}^3$  [115], a level corresponding to 4 extra death cases of lung cancer per 1000 exposed, for 40 years of occupational exposure. Thus, the reported BC level [41] are well below the new EU OEL for diesel exhaust as well as the Dutch OEL, but exceed the Dutch prohibition risk level. Recently published data on the dose-response relationship between exposure to diesel exhaust particles and lung cancer in epidemiological studies estimated that occupational exposure to 1  $\mu\text{g}/\text{m}^3$  EC would cause 4 to 17 excess lung cancer cases per 10,000 exposed [80, 81].

The particle exposure levels can be compared to nanoparticle reference values used in The Netherlands, Germany and Finland as a provisional substitute when nano-specific OELs or DNELs for engineered nanoparticles are not available [116]. For low density insoluble nanomaterials such as carbon-based nanoparticles, the reference value is 40,000 particles/ $\text{cm}^3$ . Compared to this reference value for engineered nanoparticles, the reported occupational exposure levels are high for some job groups.

Significant variations in emission levels are observed between airports, depending on factors such as size, type, location, and wind direction. However, the closer to the source of emissions, the higher the exposure. Proximity to



exposure peak events such as landing and take-off is also an important determinant of high exposure. This is evident from the combined literature of occupational exposure measurements and ambient air measurements in residential areas around airports. As such, the highest levels of occupational exposure is found for airport personnel working at the apron, in close proximity to running jet engines. Airport personnel can likely be grouped in low (office staff/landside jobs with indoor work, far away from emission sources), medium (catering/cleaning/landside security staff with intermittent outdoor work) and high (baggage handlers/aircraft mechanics, crew chiefs) exposure groups [52, 86–88, 92, 98, 100–102]. To reduce occupational exposure, emission sources can be moved, the distance to emission sources can be increased, time spent in proximity to emission sources can be reduced and personal protection equipment can be used during peak exposures. Personal exposure may be higher than measured by stationary monitors, and thus, routine monitoring of personal exposure levels could be suggested.

Workplace experts, airport leaders and personnel groups have the necessary intrinsic knowledge and experience to suggest feasible, realistic options for reducing the exposure for specific job functions at individual airports.

The similarity of airport emission particles with diesel exhaust particles and pure carbon nanoparticles, with respect to physico-chemical properties as well as specific toxicological parameters was demonstrated in the animal study from our laboratory [36], and a growing number of studies report similar toxicity and health effects of emissions from airports and traffic. Airport emission particles likely have similar physico-chemical properties as diesel exhaust particles even though the primary particle size of jet engine emissions is somewhat smaller than the primary size of diesel exhaust particles. Diesel exhaust is classified as carcinogenic to humans by IARC [69], cause lung cancer, systemic inflammation, and inflammatory responses in the airways [70].

Aircraft emissions are associated with biomarkers of exposure, biomarkers of disease and health outcomes both for exposed workers [36, 41, 52, 83, 84, 86–90, 92, 100–103, 105] and for the general population living down-wind of airports [59, 66, 94–97, 107, 112]. Occupational exposure to aircraft emissions were associated with:

- Biomarkers of exposure to jet fuel emissions
- Biomarkers of genotoxic exposure
- Self-reported respiratory distress

The reported adverse effects correlate with effects demonstrated in animal studies and in *in vitro* studies, where aircraft emission particles caused inflammation [50, 110, 111], acute phase response [36], reactive

oxygen species [49, 50] and DNA damage [36], which are biomarkers of risk of cancer, cardiovascular disease and respiratory disease. This supports the notion of a causal relationship between exposure to airport emissions and the observed health effects. Although mechanistic studies on airport emissions are scarce, knowledge from other closely related scientific areas still applies, such as particle toxicity, carcinogenicity/toxicity of VOCs and OPEs and epidemiological studies of health effects caused by air pollution [117].

Another relevant concern to raise in this context is the adverse health effects of low-level chronic occupational exposure to these chemicals, which is difficult to study [118]. OPEs have been associated with adverse health effects reported from cabin crew and pilots after occupational exposure to bleed air and fume events during flights, with symptoms of respiratory illness and neurological effects [119]. The dominant OPE used in lubrication oil is tricresyl phosphate (TCP), which are among the highly neurotoxic OPEs [120]. It has been suggested that brain exposure may occur via inhalation of circulating small jet particles associated with OPEs, crossing the blood-brain barrier [121] – neurotoxic effects of OPEs may also be an understudied occupational risk of apron staff.

It has been shown that air pollutants worsen pre-existing diseases, such as allergy or other inflammatory (airway) or cardiovascular conditions [2–4, 122–124]. One example is a study examining the relationship between personal exposure to traffic emissions and acute respiratory health in school children with asthma residing in the Bronx, New York, which have the highest asthma incidence in New York City and state [125]. Personal samples of PM<sub>2.5</sub>, including the EC fraction, were collected 24 h daily for 40 school children with asthma from four schools, with spirometry and symptoms assessed several times daily. The study found increased relative risks of different airway symptoms, such as wheeze (RR = 1.45, CI: 1.03–2.04), shortness of breath (RR = 1.41, CI: 1.01–1.99), with relative risk of total symptoms of 1.30 (CI: 1.04–1.62). Interestingly, the symptoms were associated with increase in average 2-day school site and personal EC levels, but not mass of PM<sub>2.5</sub> [125]. As such, as demonstrated in asthmatic volunteers, residents living near airports, and supported by inflammatory effects shown in available *in vitro* studies, airport UFP and associated pollutants are, in addition to their direct adverse effects, likely to have the ability of worsen pre-existing disease.

## Conclusion

The reported adverse health effects of jet engine emissions are similar to those caused by exposure to diesel exhaust and air pollution. However, given the lack of

consensus on optimal measurement methods, equipment and quality control for near- and far field airport emissions and human risk assessments markers, more studies of exposure and of toxicological mechanisms are necessary.

These drawbacks are summarized efficiently by Lighty et al. in their paper on combustion compounds and health: *“There is a need for better integration of the combustion, air pollution control, atmospheric chemistry, and inhalation health research communities. Epidemiology has demonstrated that susceptible individuals are being harmed by ambient PM. Particle surface area, number of ultrafine particles, bioavailable transition metals, polycyclic aromatic hydrocarbons (PAH), and other particle-bound organic compounds are suspected to be more important than particle mass in determining the effects of air pollution. Time- and size-resolved PM measurements are needed for testing mechanistic toxicological hypotheses, for characterizing the relationship between combustion operating conditions and transient emissions, and for source apportionment studies to develop air quality plans”* [24].

Based on the accumulated knowledge so far, measures to reduce occupational exposure and emission levels at airports should be increased.

## Supplementary Information

The online version contains supplementary material available at <https://doi.org/10.1186/s12940-020-00690-y>.

### Additional file 1.

## Authors' contributions

Conceptualization, Methodology, Data Curation, and Writing – Original Draft and Review and Editing: KMB; Conceptualization and Methodology: UB, Data Curation (Systematic Database Search): EB; Writing – Review and Editing: ATS and UB. The authors read and approved the final manuscript.

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## Availability of data and materials

Data sharing not applicable to this article as no datasets were generated or analyzed during the current study.

## Ethics approval and consent to participate

Not applicable.

## Consent for publication

Not applicable.

## Competing interests

The authors declare that they have no competing interests.

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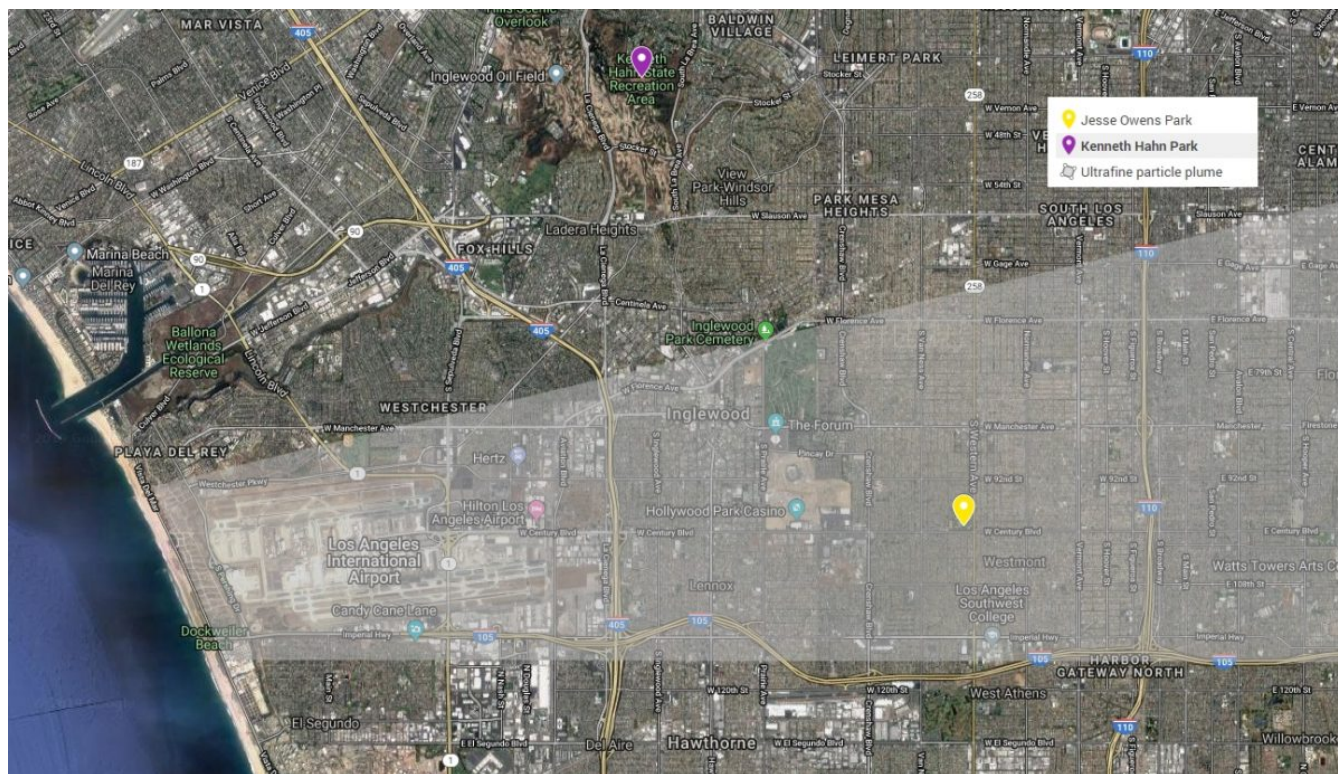


RESEARCH February 14, 2019

# Airport pollution linked to acute health effects among people with asthma in Los Angeles

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The map above shows the two parks where the study took place. The grey shaded area shows the approximate location of the plume of ultrafine particles created by air traffic around LAX, that usually occurs when the winds are blowing steadily from the West.

by Wendy Gutschow

A recent research study by Dr. Rima Habre took a detailed look at the short-term health impacts caused by breathing in ultrafine particulate (UFP) matter that is emitted from aircraft activity at the Los Angeles International Airport (LAX). Several years ago, **USC researchers identified** a clear pattern of UFP emissions from takeoff and landing aircraft activities at LAX. Levels of the dangerous UFPs were found to be 4 to 5 times greater than background levels in downwind communities. “Ultrafine particulate matter is known to contribute to reduced lung function, and airway inflammation in individuals with asthma. We wanted to take a close look at short term effects on health when individuals breathe air that contains UFPs from airplanes,” said Habre. The study participants were made up of adults with doctor diagnosed asthma.



Rima Habre holds an ultrafine particle monitor while a plane flies overhead. Photo courtesy of *Something in the Air* documentary.

Dr. Rima Habre has been with USC for five years. Dr. Habre's expertise lies in air pollution exposure assessment, analyzing patterns of how people get exposed to air pollution across time and space and studying how specific pollutants impact their health.

Recently Dr. Habre's work reached the international stage through a documentary, produced by the Canadian Broadcasting Company, called *Something in the Air*. "The producers were very interested in learning more about our latest work around air pollution and asthma – specifically around the latest technologies we are using to better understand the impacts of small particles on a personal level – in children and adults with asthma, and in pregnant women." Dr. Habre was interviewed about her work around ultrafine particle exposures downwind of major airports and its effects on asthma, as well as her work to understand how children's personal exposure to air pollution predict their risk of experiencing an asthma attack. *Something in the Air* will be released this week in Canada, with an international release to be announced.

### ***Airport-related ultrafines affect health differently than traffic-related ultrafine particles***

Habre and her team designed this study to test the short-term effects of breathing ultrafine particles by asking study participants to walk in a Los Angeles park located within the known higher levels of UFPs emitted from airplanes and near heavily trafficked roads, and another park farther away from the airport and busy roads with lower levels of UFPs.

"In our study, we found that inhaling UFPs led to higher inflammation in the blood in adults with asthma shortly after exposure. However, different inflammation markers responded to aircraft-related versus traffic-related UFPs – both of which are major ultrafine particle sources in dense urban areas. We were able to see these different signals because we managed to overcome the challenge of separating the air pollution mixture into its major sources using sophisticated measurements and modeling techniques," said Habre. The pollutants measured by the study included UFP particle number, particle size, black carbon, carbon dioxide, particle-bound polyaromatic hydrocarbons, and ozone.



The significance of Habre's study is that in such a short time, following regular walking exposure and a higher exposure, they were able to see significant elevation in inflammation systemically, not just in the lungs but in the overall blood circulation. Inflammation is tied to a lot of disease processes; cardiovascular, respiratory, and metabolic. "We don't know specifically what this inflammation will lead to down the line, but we know that inflammation is generally a bad thing, and will complicate or exacerbate existing conditions. Ideally, we would have liked to have been able to monitor people long-term to see if that inflammation persists or if it goes down after a while but we were not able to do that in this specific study, that's a future direction of this research I'd like to look at," said Habre.

When asked what this research means to the overall population, Habre described the current body of research that has found ultrafine particles to be much more toxic than the larger sizes of particulate matter, UFPs are not regulated, and UFPs impact large numbers of people who live in communities surrounding airports.

### ***Ultrafine particulate matter research: future directions***

Dr. Habre also leads environmental exposure assessment efforts in multiple research studies being conducted at USC, including the **MADRES** study of pregnant women and babies and the **LA DREAMERs** study of children's health across the life course, and in partnership with other research groups such as the **Los Angeles PRISMS Center**, a UCLA/USC partnership. Her work in the **Los Angeles PRISMS Center** is taking a deeper dive into the different sources of air pollution asthmatic children encounter in their day to day lives in Los Angeles and how it impacts their health. This study focuses on personal experiences, using Bluetooth-enabled wearable sensors to monitor environmental exposures, location, activity, medications and symptoms, to ultimately generate new information to help predict and prevent asthma attacks. To learn more about the Los Angeles PRISMS Center, watch this video:

<https://www.youtube.com/watch?v=-m72NkwolgU&feature=youtu.be>

As she moves forward with her research on the health effects of ultrafine particulate matter in urban areas, Dr. Habre plans to build on her current work by studying how people with asthma are affected, as well as those who are obese, have diabetes, or cardiovascular issues. "I would like to be able to capture a wider variety of sources of ultrafines in urban areas and also be able to monitor individuals for a longer period of time to really understand what happens next. In this study we saw very quick and acute effects, but do people tend to recover after a day? I think the ultimate goal would be to

really understand if people living in these high exposure locations, for extended periods of time, and breathing this mixture in regularly are at a significantly higher risk or not,” she said.

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For more information on the “Something in the Air” documentary that Dr. Habre’s work is featured in, on the documentary’s website: [www.somethingintheair.ca](http://www.somethingintheair.ca). Once the documentary is released in the United States, USC Environmental Health Centers will publish the release date and viewing information.

Rima Habre, Hui Zhou, Sandrah P. Eckel, Temuulen Enebish, Scott Fruin, Theresa M. Bastain, Edward B. Rappaport, and Frank D Gilliland, 2018. **Short-term effects of airport-associated ultrafine particle exposure on lung function and inflammation in adults with asthma**. Environment International.  
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# AIRPORTS, AIR POLLUTION, AND CONTEMPORANEOUS HEALTH

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## Abstract

We link daily air pollution exposure to measures of contemporaneous health for communities surrounding the 12 largest airports in California. These airports are some of the largest sources of air pollution in the United States, and they experience large changes in daily air pollution emissions depending on the amount of time planes spend idling on the tarmac. Excess airplane idling, measured as residual daily taxi time, is due to network delays originating in the Eastern United States. This idiosyncratic variation in daily airplane taxi time significantly impacts the health of local residents, largely driven by increased levels of carbon monoxide (CO) exposure. We use this variation in daily airport congestion to estimate the population dose-response of health outcomes to daily CO exposure, examining hospitalization rates for asthma, respiratory, and heart related emergency room admissions. A one standard deviation increase in daily pollution levels leads to an additional \$540 thousand in hospitalization costs for respiratory and heart related admissions for the 6 million individuals living within 10km (6.2 miles) of the airports in California. These health effects occur at levels of CO exposure far below existing EPA mandates, and our results suggest there may be sizable morbidity benefits from lowering the existing CO standard.

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The effect of pollution on health remains a highly debated topic. The US Clean Air Act (CAA) requires the Environmental Protection Agency (EPA) to develop and enforce regulations to protect the general public from exposure to airborne contaminants that are known to be hazardous to human health. In January 2011, the EPA decided against lowering the existing CAA carbon monoxide standard due to insufficient evidence that relatively low carbon monoxide levels adversely affect human health. In order to assess the benefits of lowering the standard, accurate estimates are needed that link contemporaneous air pollution exposure to observable health outcomes at levels of pollution currently faced by local populations. However, these estimates are hard to come by as pollution is rarely randomly assigned across individuals, and individuals who live in areas of high pollution may be in worse health for reasons unrelated to pollution. Preferences for clean air may covary with unobservable determinants of health (e.g., exercise) which can lead to various forms of omitted variable bias in regression analysis. Moreover, heterogeneity across individuals in either preference for, or health responses to, ambient air pollution implies that individuals may self-select into locations on the basis of these unobserved differences. In both cases, estimates of the health effects of ambient air pollution may reflect the response of various subpopulations and/or spurious correlations pertaining to omitted variables. While recent research attempts to address the issue of non-random assignment using various econometric tools such as fixed effects or instrumental variables, these studies often focus on infant health over longer periods of time (Chay & Greenstone 2003, Currie & Neidell 2005). Much less is known about short-term, daily effects of ambient air pollution on the health of the more general population, such as the non-elderly, non-child, adult population.<sup>1</sup>

We develop a framework for estimating the contemporaneous effect of air pollution on health using variation in local air pollution driven by airport runway congestion. Airports are one of the largest sources of air pollution in the United States with Los Angeles International Airport (LAX) being the largest source of carbon monoxide in the state of California (Environmental Protection Agency 2005). A large fraction of airport emissions come from airplanes, with the largest aggregate channel of emissions stemming from airplane idling (Transportation Research Board 2008). We show that airport runway congestion, as measured by the total time planes spent taxiing between the gate and the runway, is a significant predictor of local pollution levels. Since local runway congestion may be correlated with other determinants of pollution such as weather, we exploit the fact that California airport congestion is driven by network delays that began in large airports outside of California.<sup>2</sup> A recent article in the New York Times (New York Times January 27, 2012)

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<sup>1</sup>There is a larger literature in epidemiology which focuses on daily responses to air pollution (see e.g. Ito, Thurston & Silverman (2007), Linn et al. (1987), Peel et al. (2005), Schildcrout et al. (2006), Schwartz et al. (1996)). The work in our paper complements the existing epidemiological literature by focusing on issues pertaining to measurement error, avoidance behavior, and self-selection bias in the context of susceptibility to pollution exposure. Each of these issues is critically important to providing unbiased estimates of the causal relationship between pollution and health. The instrumental variables approach in this paper exploits arguably exogenous pollution shocks that are unlikely to be known by local residents, allowing us to simultaneously address issues of measurement error and avoidance behavior. Recent work in economics and environmental health, discussed in more detail below, suggests that short run variation in pollution exposure may be significant predictors of mortality and morbidity (Moretti & Neidell 2011, Knittel, Miller & Sanders 2011).

<sup>2</sup>This relationship is well known within the transportation literature (Welman, William & Hechtman 2010). Opti-

provides a useful motivation:

[Airplane] delays ripple across the country. A third of all delays around the nation each year are caused, in some way, by the New York airports, according to the F.A.A. Or, as Paul McGraw, an operations expert with Airlines for America, the industry trade group, put it, “When New York sneezes, the rest of the national airspace catches a cold.”

Our analysis hence links health outcomes of residents living near California airports to changes in air pollution driven by runway congestion at airports on the East Coast. The identifying variation in California pollution is caused by events several thousand miles away (e.g., weather in Atlanta), which is unlikely to be correlated with determinants of health in California.

The goal of this paper is to identify the ways in which short run, daily variation in air pollution affects population health. In doing so, this paper makes four primary contributions to the existing literature in this area. First, while most existing literature focuses on the health impacts of infants or elderly, we are able to examine the health responses of the entire population. We find that infants as well as the elderly are most sensitive to ambient air pollution. At the same time, a one-unit increase in pollution has much larger aggregate effects for adults aged 20-64, given their large share of the overall population. Studies that focus on infants or the elderly significantly underestimate overall health effects.

The second contribution of this paper is to estimate the contemporaneous effect of multiple pollutants simultaneously. It has traditionally been difficult to decipher which pollutant is responsible for adverse health outcomes since short-term fluctuations among ambient air pollutants are highly correlated. Our solution to this identification problem is to rely on the fact that wind speed and wind direction transport individual pollutants in different ways. By using interactions between taxi time, wind speed, and wind angle from airports, we can pin down the direct effect of each pollutant, while holding the others constant. We use over-identified models to instrument for several pollutants simultaneously, an approach that was simultaneously developed in related work by Knittel, Miller & Sanders (2011). We find that CO is responsible for the majority of the observed increase in hospital admissions, although we cannot rule out that this may be driven by other unobserved pollutants that are correlated with airplane-driven CO emissions. This finding has direct policy implications. The EPA recently decided to maintain the current CO pollution standard, citing a lack of evidence that reducing CO below current ambient levels would improve population health outcomes.

We believe there are two additional features that set this paper apart from existing work in both economics and epidemiology. Our paper is most closely related to the recent work of Moretti & Neidell (2011) and Knittel, Miller & Sanders (2011) who also instrument daily pollution in health regressions with variation in local transportation conditions (i.e. container shipping in Long

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mal airplane scheduling incorporates anticipated ripple effect. For example, Pyrgiotisa, Maloneb & Odoni (2013) use queuing theory to simulate how delays propagate through the system. They quote a study that found a multiplier effect of seven, i.e., each 1-hour delay of a particular airplane leads to a combined 7 hours delay for the airline.

Beach, CA and automobile congestion in Central and Southern CA, respectively). Relative to these papers and the existing literature, we believe this paper is the first to use the network structure of transportation to generate local variation in congestion that is driven by events that occur several thousand miles away. This matters because one of the key drivers of transportation congestion is local weather, and local weather is also likely to affect ambient pollution, violating the identifying assumptions of the model. By way of example, we show that instrumenting local airport congestion with network delays that are not correlated with local weather doubles our point estimates, relative to the baseline case. We also explicitly model the spatial dispersion of air pollution emissions, as it varies with wind speed, wind direction, and distance from the airport. Pollutant transport is very locally heterogeneous, and failing to account for this spatial heterogeneity leads to bias when estimating the population dose-response function.

The fourth contribution of this study is the use of newly available Emergency Discharge Data to better capture the morbidity impacts of air pollution. Previous research has predominantly focused on the effects of pollution on mortality or morbidity as measured in Inpatient Discharge Records. Inpatient Discharge data consist only of observations for patients that stayed overnight in a hospital, and thus exclude a large fraction of respiratory-related emergency room admissions that do not require overnight hospital visits. We show that estimates using the more commonly used Inpatient Discharge data substantially underestimate the morbidity impacts of air pollution, relative to estimates from the combined Emergency Discharge and Inpatient Discharge datasets.<sup>3</sup>

In summary, our approach combines newly available data with arguably exogenous daily changes in air pollution that originate several thousand miles away and are unknown to the local population. The instrumental variables setting allows us to simultaneously address issues pertaining to both avoidance behavior and classical forms of measurement error, each of which lead to significant downward bias in conventional dose-response estimates. The primary estimation framework examines how zip code level emergency room admissions covary with these quasi-experimental increases in air pollution stemming from airports.

We find that a one standard deviation increase in daily pollution explains roughly one third of average daily admissions for asthma problems. It leads to an additional \$540 thousand per day in hospitalization costs for respiratory and heart related admissions of individuals within 10km of one of the 12 largest airports in California. This is likely a significant lower bound of the social costs as the willingness to pay to avoid a sickness might be significantly larger than the medical reimbursement cost (Grossman 1972). Our baseline IV estimates are an order of magnitude larger than uninstrumented fixed effects estimates, highlighting the importance of accounting for measurement error and/or avoidance behavior in conventional estimators. We find no evidence that airport runway congestion affects diagnoses unrelated to air pollution such as bone fractures, stroke, or appendicitis. We also present a variety of evidence in favor of a non-linear dose-response function. As pollution levels increase the marginal effect of a 1 unit increase in pollution increases but at a

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<sup>3</sup>Knittel, Miller & Sanders (2011) focus on infant mortality, while Moretti & Neidell (2011) examine morbidity outcomes, but only for individuals of Emergency Room visits that eventually get admitted to an overnight stay as the authors rely on the Inpatient Discharge data.

decreasing rate. This is consistent with thresholds at which the health effect of air pollution levels off (i.e. the dose-response function is not convex over the levels of pollution we observe), and along the lines of what research in epidemiology has observed (Pope et al. 2009, Pope III et al. 2011).

We present several sensitivity checks of results that do not alter our conclusions. For example, we focus on morning airport congestion in the East since it is possible that California airport delays impact airports on the East Coast, which then feedback to California airports. Due to the difference in time zones, very few flights from California reach East Coast airports before 12pm. Estimates remain similar to our baseline estimates. A distributed lag model finds no evidence for delayed impacts or forward displacement, i.e., that individuals on the brink of an asthma or heart attack may experience an episode that would have otherwise occurred in the next few days anyway. A Poisson model linking sickness counts to pollution levels gives comparable estimates to our baseline linear probability model, which does not account for the truncation of daily sickness rates at zero. Lastly, we find little evidence of treatment effect heterogeneity that would raise concerns pertaining to forms of self-selection bias and/or the external validity of the underlying dose-response estimates.

The findings in this paper suggest that daily variation in ambient air pollution has economically significant health effects at levels below current EPA mandates, at least for the population that comprises our study. We believe this is particularly important due to the fact that in January 2011, the EPA decided against lowering the existing CAA carbon monoxide standard due to insufficient evidence that relatively low carbon monoxide levels adversely affect human health. The maximum hourly CO concentration in our data is 7.5ppm (see Table A2), which is below the ambient air quality standard of 35ppm for any 1-hour reading or 9ppm for any 8-hour average, i.e., air quality levels were always within the limit.<sup>4</sup> Yet, fluctuations in pollution levels significantly below the standard still have sizable health consequences. While a full-fledged benefit-cost analysis would have to balance the cost of reducing CO against the benefits, EPA indicated there were no appreciable benefits from lowering the standard to begin with, which we find not to be the case.

## 1 Background: Airports, Airplanes, and Air Pollution

Regulators have long been aware of the pollution generated by cars, trucks, and public transit. There have been countless legislative policies designed to curtail harmful emissions from these sources (Auffhammer & Kellogg 2011). However, aircraft and airport emissions have only recently become the subject of regulatory scrutiny, although little has been done to reduce or manage emissions generated by airports and air travel. While there has been some effort to curtail the substantial CO<sub>2</sub> emissions generated by aircraft,<sup>5</sup> there has been relatively little effort to control or contain some of the more pernicious air pollutants generated by jet engines. This lack of regulatory scrutiny can be traced back to the way in which pollutants are regulated in the United States

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<sup>4</sup>The same is not true for NO<sub>2</sub>. The maximum 1-hour reading in our data is 136ppb, which is above the 1-hour standard of 100ppb.

<sup>5</sup>The European Union has recently approved greenhouse gas measures, which oblige airlines, regardless of nationality, that land or take off from an airport in the European Union to join the emissions trading system starting on January 1, 2012.



under the Clean Air Act. Current Federal law preempts all federal, state, and local agencies except the Federal Aviation Administration from establishing measures to reduce emissions from aircraft due to potential interstate and international commerce conflicts that might arise from other decentralized regulations.<sup>6</sup>

Aircraft jet engines, like many other mobile sources, produce carbon dioxide ( $\text{CO}_2$ ), nitrogen oxides ( $\text{NO}_x$ ), carbon monoxide ( $\text{CO}$ ), oxides of sulfur ( $\text{SO}_x$ ), unburned or partially combusted hydrocarbons (also known as volatile organic compounds, or VOCs), particulates, and other trace compounds (Federal Aviation Administration 2005). Each of these pollutants is emitted at different rates during various phases of operation, such as idling, taxiing, takeoff, climbing, and landing.  $\text{NO}_x$  emissions are higher during high power operations like takeoff when combustor temperatures are high. On the other hand,  $\text{CO}$  emissions are higher during low power operations like taxiing when combustor temperatures are low and the engine is less efficient (Federal Aviation Administration 2005).<sup>7</sup> Even though the aircraft engine is often idling during taxi-out, the per minute  $\text{CO}$  and  $\text{NO}_x$  emissions factors are higher than at any other stage of a flight (Environmental Protection Agency 1992). Combining this with the long duration of taxi-out times during peak periods of the day, total taxiing over the course of a day can add up to a substantial amount. Consistent with these facts, Los Angeles International airport is estimated to be the largest point source of  $\text{CO}$  emissions in the state of California, the second largest of  $\text{NO}_x$ , the twenty-ninth largest of  $\text{SO}_2$ , and the 2763 and 2782 largest of  $\text{PM}_{10}$  and  $\text{PM}_{2.5}$ , respectively (Environmental Protection Agency 2005).

Airports provide a particularly compelling setting through which to estimate the contemporaneous relationship between air pollution and health. Not only are airports some of the largest polluters of ambient air pollution in the United States but they also have extraordinarily rich data on daily operating activity, detailing for each domestic flight the length of time spent taxiing to and from the gate before takeoff and after landing. This allows for a precise understanding of the aggregate amount of daily runway congestion at airports. Moreover, daily runway congestion at airports exhibits a great degree of residual variation even after controlling for normal scheduling patterns. Much of the variation in runway congestion is driven by network delays propagating from major airport hub delays thousands of miles away. Network delays at distant airports serve as an ideal instrumental variable for local pollution; the effect of a snow storm in Chicago on congestion at LAX should be orthogonal to any other confounding influences of air pollution in the Los Angeles area. In addition, local residents are likely unaware of increases in taxi time and hence cannot engage in self-protective behavior. Lastly, every airport has detailed weather data, allowing researchers to exploit the spatial distribution of airport-generated pollution. We can therefore estimate how areas downwind of an airport on a given day are disproportionately affected by runway

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<sup>6</sup>Currently, the Environmental Protection Agency has an agreement with the FAA to voluntarily regulate ground support equipment at participating airports known as the Voluntary Airport Low Emission (VALE) program (United States Environmental Protection Agency 2004).

<sup>7</sup>As a result, reducing engine power for a given operation like takeoff or climb out generally increases the rate of  $\text{CO}$  emissions and reduces the rate of  $\text{NO}_x$  emissions.

congestion relative to areas upwind. Understanding this spatial variation in pollutant transport improves the efficiency of our estimates, while also providing important tests of the validity of our research design.

## 2 Data

This project uses the most comprehensive data currently available on airport traffic, air pollution, weather, and daily measures of health in California. This data is rich in both temporal and spatial dimension, allowing for fine-grained analysis of how daily airport congestion impacts areas downwind of an airport on a given day. The various datasets and linkages are described in more detail below.

### 2.1 Airport Traffic Data

A useful feature of a study involving airports is the detailed nature of daily flight data. The Bureau of Transportation Statistics (BTS) Airline On-Time Performance Database contains flight-level information by all certified U.S. air carriers that account for at least one percent of domestic passenger revenues. It has a wealth of information on individual flights: flight number, the origin and departure airport, scheduled departure and arrival times, actual departure and arrival times, the time the aircraft left the runway and when it touches down. We construct a daily congestion measure for each of the 12 major airports in California by aggregating the combined taxi time of all airplanes at an airport. This measure consists of (i) the time airplanes spend between leaving the gateway and taking off from the runway and (ii) the time between landing and reaching the gate. An interesting feature of aggregate daily taxi time is the large amount of residual variation remaining after controlling for daily airport scheduling, weather, and holidays. We relate this variation to local measures of pollution and health in our econometric analysis. One caveat of the BTS data is that it only includes information for major domestic airline passenger travel.<sup>8</sup> As long as international flights are not treated differently in the queuing system and are hence colinear to the taxi time of domestic flights, congestion of national flights should be a good proxy for overall congestion.

We limit our analysis to the 12 largest airports in California by passenger count. These airports are in alphabetical order (including airport call sign in brackets): Burbank (BUR), Los Angeles International (LAX), Long Beach (LGB), Oakland International (OAK), Ontario International (ONT), Palm Springs (PSP), San Diego International (SAN), San Francisco International (SFO), San Jose International (SJC), Sacramento International (SMF), Santa Barbara (SBA), and Santa Ana / Orange County (SNA). The locations of these airports are shown as dots in Figure 1. Average flight statistics at each of these airports are reported in Table A1. There is significant variation in

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<sup>8</sup>In January 2005, international departures (both cargo and passenger) accounted for 8.5% of total departures, whereas cargo (both international and domestic) accounted for 5.9% of all United States airport departures (Department of Transportation 2009).

daily ground congestion at airports: the standard deviation of daily taxi time at the largest airport (LAX) is 1852 minutes. Once we account for year, month, weekday and holiday fixed effects as well as local weather, the remaining variation is still 891 minutes. Most of the airports are close to urban areas as they serve the travel needs of these populations. Seven airports in California rank among the top 50 busiest airports in the nation according to passenger enplanement (Federal Aviation Administration 2009).

A potential concern when linking daily airport activity to daily ambient air pollution levels is that runway congestion in California airports may be highest in the late afternoon and evening. This would lead us to erroneously misclassify some of the daily airport effects to the wrong day. Figure A2 plots the distribution of aggregate taxi time within a day. Most ground activity at airports is skewed towards the beginning of the day. We will address the sensitivity of our estimates towards these issues of misclassification or across-day spillovers in subsequent sections.

## 2.2 Pollution Data

We construct daily measures of air pollution surrounding airports using the monitoring network maintained by the California Air Resource Board (CARB). This database combines pollution readings for all pollution monitors administered by CARB, including information on the exact location of the monitor. Data includes both daily and hourly pollution readings. We concentrate on the set of monitors with hourly emission readings for CO, NO<sub>2</sub>, and O<sub>3</sub> in the years 2005-2007.<sup>9</sup> The locations of all CO and NO<sub>2</sub> monitors in relation to airports are shown in Figure 1.

A unique feature of pollution data is the significant number of missing observations in the database. We therefore use the following algorithm when we aggregate the hourly data to daily pollution readings: Our measure of the daily maximum pollution reading is simply the maximum of all hourly pollution readings. The daily mean is the duration-weighted average of all hourly pollution readings. We define the duration as the number of hours until the next reading.<sup>10</sup> We prefer this approach to simply taking the arithmetic average of all hourly readings on a day since hourly pollution data exhibit great temporal dependence. A missing hourly observation is better approximated by the previous non-missing value than the daily average. We also keep track of the number of observations per day. In a sensitivity check (not reported) we rerun the analysis using only monitors with at least 20 or 12 readings per day.<sup>11</sup>

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<sup>9</sup>While data exists for other pollutants in California, we limit our analysis to using CO, NO<sub>2</sub> as they are directly emitted by airplanes and have better coverage than PM10. O<sub>3</sub> forms from VOC and NO<sub>x</sub>. In a sensitivity check we do *not* find that O<sub>3</sub> pollution levels are impacted by airport congestion. Nevertheless, we present sensitivity analyses that include O<sub>3</sub> and PM10 as controls with little effect on our results. While monitor data exists as far back as 1993, portions of our hospital data, described further in this section, exists only from 2005 onwards.

<sup>10</sup>Readings occur on the hour of each day ranging from midnight to 11pm. If readings at the beginning of a day (midnight, 1am, etc.) are missing, we adjust the duration of the first reading from midnight to the second reading. For example, if readings occur on 3am, 5am, and 8am, the 3am reading would be assigned a duration of 5 hours and the 5am reading would be assigned a duration of 3 hours. By the same token, if the last reading of a day is not 11pm, the duration of that last reading is from the time of the reading until midnight.

<sup>11</sup>If a monitor has not a single reading for a day, we approximate it's value in a three step procedure: (i) we derive the cumulative density function (cdf) at each monitor; (ii) take the inverse-distance weighted average of the cdf for a given day at all monitors with non-missing data; (iii) we fill the missing observation with the same percentile of

We create daily zip code pollution measures by taking the average monitor reading of all monitors within 15km of a zip code centroid, weighting by the inverse distance between the monitor and the zip code centroid.<sup>12</sup> Summary statistics are given in Panel A of Table A2. Since we have both the longitude and latitude of all airports and zip code centroids, we are able to derive (i) the distance between the airport and a zip code, and (ii) the angle at which the zip code is located relative to the airport. In order to leverage the spatial features of our data, we normalize the angle between a zip code centroid and an airport to 0 if the zip code is lying to the north of the airport. Degrees are measured in clockwise fashion, e.g., a zip code that is directly east of an airport will have an angle of 90 degrees. The angle between an airport and a zip code allows us to explore the link between airport emissions and pollution downwind of airports using the weather data described next.

### 2.3 Weather Data

We use temperature, precipitation, and wind data in our analysis to both control for the direct effects of weather on health (Deschênes, Greenstone & Guryan 2009) and also to leverage the quasi-experimental features of wind direction and wind speed in distributing airport pollution from airports. Our weather data comes from Schlenker & Roberts (2009), which provides minimum and maximum temperature as well as total precipitation at a daily frequency on a  $2.5 \times 2.5$  mile grid for the entire United States.<sup>13</sup> To assign daily weather observations to an airport or zip code, we use the grid cell in which the zip code centroid is located. Summary statistics for the zip-code level data are given in Panel B of Table A2.

Average wind speed and wind direction come from the National Climatic Data by the National Oceanic and Atmospheric Administration’s (NOAA) hourly weather stations. Most airports have weather stations with hourly readings. We construct wind direction, which is normalized to equal zero if the wind is *blowing* northward and counted in clockwise fashion. If the angles of the zip code and the wind direction are identical, the zip code is hence exactly downwind from the airport. An angle of 180 degrees implies that the zip code is upwind from the airport. The hourly wind speed and wind direction is aggregated to the daily level by calculating the duration-weighted average between readings comparable to the pollution data above. The distribution of wind directions is shown in Figure 2. Airports at the ocean predominantly have winds coming from the direction of the ocean. For example, Santa Barbara, located on the only portion of the California coast that runs east-west has winds blowing northward. Note again that we are measuring the direction in

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the station’s cdf. For example, if surrounding monitors with non-missing data on average have pollution levels that correspond to the 80th percentile of their respective distributions, we fill the missing value of a station with the 80th percentile of it’s own distribution of pollution readings. This procedure gives us a balanced panel.

<sup>12</sup>Inverse distance weighting pollution measures has been used to impute pollution in previous research. See for example, Currie & Neidell (2005).

<sup>13</sup>There is one exception: in a set of regression models where we estimate the effect of airport weather on taxi time we use the closest non-missing daily weather station data from NOAA’s COOP station data set for each airport. This is because Schlenker & Roberts (2009) use a spatial interpolation procedure that might result in artificial correlation between weather data at airports due to the spatial interpolation technique.



which the wind is blowing, not from which it is coming. In our empirical analysis, we use this daily variation in wind speed and wind direction to predict how pollution from airports disproportionately impacts some zip codes more than others on a given day.

## 2.4 Hospital Discharge and Emergency Room Data

Health effects are measured by overnight hospital admission and emergency room visits to any hospital in the state of California. We use the California Emergency Department & Ambulatory Surgery data set for the years 2005-2007.<sup>14</sup> The dataset gives the exact admission date, the zip code of the patient’s residence (as well as the hospital), the age of the patient, as well as the primary and up to 24 secondary diagnosis codes. An important limitation of the Emergency Department data is that any person who visits an ER and is subsequently admitted to an overnight stay drops out of the dataset. This is done to prevent double counting in California’s hospital admissions records, as overnight hospital stays are logged in California’s Inpatient Discharge data. Therefore we also obtained Inpatient Discharge data for all individuals who stayed overnight in a hospital in the years 2005-2007. In our baseline model we focus on the sum of emergency room visits and overnight stays in a zip code-day to avoid non-random attrition in the ER data. Focusing only on emergency room admittance would suffer from selection bias as higher pollution levels (and more severe health outcomes) could result in more overnight stays, yet the emergency room numbers would actually appear smaller.

We count the daily admissions of all people in a zip code who had a diagnosis code pertaining to three respiratory illnesses: asthma, acute respiratory, and all respiratory. Note that each category adds additional sickness counts but includes the previous. For example, asthma attacks are also counted in all respiratory problems. We also count heart related problems, which Peters et al. (2001) have shown to be correlated with pollution. Finally, we include three placebos: stroke, bone fractures, and appendicitis.<sup>15</sup> In our baseline model, we count a patient as suffering from a sickness if either the primary or one of the secondary diagnosis codes lists the illness in question.

We merge the zip code level hospital data with age-specific population counts in each zip code obtained from both the 2000 and 2010 Censuses. We use the weighted average between the 2000 (weight 0.4) and 2010 (weight 0.6) counts, as the midpoint of our data is 2006. We limit our analysis to the 164 zip codes whose centroid lies within 10km of an airport and which have at least 10000 inhabitants.<sup>16</sup> The total population of these 164 zip codes is around 6 million people, or roughly one sixth of the overall population of California at the time. Summary statistics for the zip codes in the study are given in Panel C of Table A2. We use these age-specific population counts to construct daily hospitalization rates for each zip code. Table A3 provides sickness rates per 10

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<sup>14</sup>The Emergency Room data was not collected prior to 2005.

<sup>15</sup>The exact ICD-9 codes are: asthma: [493, 494); acute respiratory: [460,479), [493,495), [500,509), [514,515), [516,520); all respiratory: [460, 520); heart problems: [410, 430); stroke [430, 439); bone fractures [800, 830); appendicitis: [540, 544).

<sup>16</sup>The latter sample restriction excludes 0.8 percent of the total population that lives in a zip code whose centroid is within 10km of an airport but has less than 10000 inhabitants.

million inhabitants for both the entire population as well as population subgroups of those over 65 years of age and under 5 years of age.

## 2.5 External Validity - Populations Close to Airports

Our analysis focuses on areas within 10km of airports. This raises the broader question as to how our estimated results generalize to populations outside of the 10km airport radius. Table A4 investigates this question by examining zip code characteristics from the 2000 Census. We present three comparisons: First, we look at zip codes that are in our sample in columns (1a)-(1c) but divide them into zip codes whose centroids are within [0,5]km and (5,10]km of an airport. Second, we compare zip codes within 10km of an airport versus neighboring zip codes that are between 10 and 20km of an airport in columns (2a)-(2c). Third, we compare zip codes within 10km of an airport to all other zip code in California in columns (3a)-(3c).

For the first two sets of comparisons, few comparison tests are significant, roughly at a rate that should happen due to randomness. In other words, areas [0,5]km from an airport are comparable to areas (5,10]km or (10,20]km.<sup>17</sup> On the other hand, the third set of comparisons shows that areas within 10km are not comparable to the rest of the state of California, which includes more rural areas. Zip codes closer to airports are on average more urban, more populated, wealthier, and have higher housing prices. Therefore, we would caution against interpreting the estimated dose response relationship as representative for the entire population at large. From the standpoint of airport externalities, the population close to airports is the population of interest. Moreover, much of the air pollution regulation in the United States is spatially targeted towards urban areas (i.e. those areas with higher degrees of ambient air pollution), and in that case, these estimates may be more appropriate for regulatory analysis than a dose response function averaged over individuals in both urban and rural locations.

## 3 Empirical Methodology

We are estimating the link between ground level airport congestion, local pollution levels, and contemporaneous hospitalization rates for major airports in the state of California. To begin, we consider the effects of increased levels of airport traffic congestion on local measures of pollution.

### 3.1 Aggregate Daily Taxi Time and Local Pollution Levels

Ambient air pollution is a function of the distance between a point source and the receptor location, as well as many other atmospheric variables including, but not limited to, wind speed, wind direction, humidity, temperature, and precipitation. To model the effects of increases in aggregate

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<sup>17</sup>47% of Californians live in a zip code within 20km of an airport.

airport taxi time on pollution levels, we adopt the following additive linear regression model

$$\textbf{Model 1: } p_{zat} = \alpha_1 T_{at} + \underbrace{\mathbf{W}_{zt}\Phi + weekday_t + month_t + year_t + holiday_t}_{\mathbf{Z}_{zt}\Gamma} + \nu_{za} + e_{zat} \quad (1)$$

where pollution  $p_{zat}$  in zip code  $z$  that is paired with airport  $a$  on day  $t$  is specified as a function of taxi time  $T_{at}$  and a vector of zip-code level controls  $\mathbf{Z}_{zt}$  that include weather controls  $\mathbf{W}_{zt}$ .<sup>18</sup> Our baseline regressions include 17 weather controls: A quadratic in minimum and maximum temperature, precipitation and wind speed (8 terms) as well as 9 terms for wind direction that are included in equation (3) below.<sup>19</sup> To model this relationship formally, we define wind direction by the cosine of the difference between the wind direction and the direction in which the zip code is located. The variable will be equal to 1 in the case that the angle in which the wind is blowing equals the direction in which the zip code is located, and the variable will be equal to zero when they are at a right angle (the difference is 90 degrees). The vector  $\mathbf{W}_{zt}$  includes all possible time-varying interactions between distance, wind speed and angle (up and downwind) to control for pollution formation not directly influenced by taxi time. We also control for temporal variation in pollution by including weekday fixed effects ( $weekday_t$ ), month fixed effects ( $month_t$ ), and year fixed effects ( $year_t$ ) as well holiday fixed effects ( $holiday_t$ ) to limit the influence of airport congestion outliers.<sup>20</sup> In a sensitivity check (available upon request), we instead include day fixed effects, i.e., one for each of the 1095 days, and the results remain robust. Since there may be time-invariant unobserved determinants of pollution for any given zip code, all regressions include zip code fixed effects,  $\nu_{za}$ . The parameter of interest is  $\alpha_1$ , which tells us the effect of a 1000 minute increase in aggregate daily ground congestion on local ambient air pollution levels. Increased airplane taxiing leads to an increase in airplane emissions and presumably increases in ambient air pollution. Hence, we would expect this coefficient to be positive.

We also estimate models similar to equation (1), where we interact taxi time (or instrumented taxi time) with the distance between an airport and the monitor. The idea would be to allow the marginal effect of taxi time to differ based on monitors that were closer relative to further from the airport. This results in the following equation:

$$\textbf{Model 2: } p_{zat} = \alpha_1 T_{at} + \alpha_2 T_{at} d_{za} + \mathbf{Z}_{zt}\Gamma + \nu_{za} + e_{zat} \quad (2)$$

The additional coefficient is  $\alpha_2$ . The effect of taxi time on pollution should fade out with distance, and we would hence expect this coefficient to be negative. The marginal effect of taxi time in model

<sup>18</sup>In principle a zip-code  $z$  could be paired with more than one airport  $a$ . In practice, our baseline model uses zip codes whose centroid is within 10km of an airport. Each zip code is assigned to exactly one airport as none is within 10km of two airports.

<sup>19</sup>Specifically, our weather controls include the terms corresponding to  $\alpha_3, \alpha_4$  and  $\alpha_6 - \alpha_{12}$  in equation (3) without the interaction with taxi time  $T_{at}$ . Results are robust to different functional forms of weather control variables. Additionally, we have estimated models that exclude all weather controls, and the coefficients for our primary pollutant of interest (CO see below) are not significantly affected (although the standard errors increase).

<sup>20</sup>We include fixed effects for New Year, Memorial Day, July 4th, Labor Day, Thanksgiving, and Christmas, as well as the three days preceding and following the holiday.

2 is  $\alpha_1 + \alpha_2 d_{za}$ .

In a third step we also include interactions with wind direction and wind speed. The intuition is that both wind direction and speed transport airport emissions across space. Thus, holding speed constant, areas downwind should be relatively more affected by aggregate daily taxi time relative to areas upwind. To model this relationship formally, we let  $v_{at}$  be the wind speed and  $c_{zat}$  the cosine of the difference between the wind direction and the direction in which the zip code is located, which can differ upwind  $c_{zat} > 0$  and downwind  $c_{zat} < 0$ .<sup>21</sup> Allowing for all possible time-varying interactions we get:<sup>22</sup>

$$\begin{aligned}
\textbf{Model 3: } p_{zat} = & \alpha_1 T_{at} + \alpha_2 T_{at} d_{za} + \alpha_3 T_{at} c_{zat} I_{[c_{zat} > 0]} + \alpha_4 T_{at} c_{zat} I_{[c_{zat} < 0]} \\
& + \alpha_5 T_{at} v_{at} + \alpha_6 T_{at} d_{za} c_{zat} I_{[c_{zat} > 0]} + \alpha_7 T_{at} d_{za} c_{zat} I_{[c_{zat} < 0]} \\
& + \alpha_8 T_{at} d_{za} v_{at} + \alpha_9 T_{at} c_{zat} I_{[c_{zat} > 0]} v_{at} + \alpha_{10} T_{at} c_{zat} I_{[c_{zat} < 0]} v_{at} \\
& + \alpha_{11} T_{at} d_{za} c_{zat} I_{[c_{zat} > 0]} v_{at} + \alpha_{12} T_{at} d_{za} c_{zat} I_{[c_{zat} < 0]} v_{at} \\
& + \mathbf{Z}_{zt} \mathbf{\Gamma} + \nu_{za} + e_{zat}
\end{aligned} \tag{3}$$

The new coefficients are  $\alpha_3$  through  $\alpha_{12}$ . The predicted signs of these coefficients are less intuitive. While higher wind speeds can clear the air they may also carry greater amounts of the pollutant further distances.<sup>23</sup> Moreover, downwind areas should have higher pollution levels relative to those areas upwind, but aircrafts usually start against the wind. To better interpret the combination of all of these interactions, we plot the marginal effects of this particular regression model using contour plots in subsequent sections. These contour plots provide strong visual evidence of the relationship between daily aggregate airport taxi time, wind speed, wind direction, and local pollution levels.

One potential cause for concern in equations (1)-(3) are any omitted transitory determinants of local pollution levels that may also covary with ground congestion. If such omitted variables exist, then least squares estimates of the coefficients on taxi time (e.g.  $\alpha_1$ ) will be biased. This could occur, for example, if weather adversely affected airport activity while also affecting local pollution levels. To address this potential source of bias, we need an instrumental variable that is correlated with changes in ground congestion at an airport but is unrelated to local levels of pollution. A natural instrument comes from delays at major airport hubs outside California, which propagate through the air network as connecting flights are delayed, leading to more ground congestion at airports in California. The basic logic is that instead of smoothing out scheduling over the course of the day, planes now arrive in more distinct blocks of time, leading to more waiting/taxiing by those planes taking off as the runway space is shared. Specifically, we instrument taxi time at each California airport with taxi time at major airports outside of California (Atlanta (ATL), Chicago

<sup>21</sup>The cosine is 0 if the angle is 90degrees, i.e., the separately estimated effect is different upwind and downwind.

<sup>22</sup>The exact dispersion of pollution depends on additional factors like acceleration and height of emissions. Benson (1984) presents a formal model of pollution dispersion around roads that includes many variables we do not observe. The standard in the literature has hence been to estimate reduced-form relationship with wind direction and speed (Batterman, Zhang & Kononowech 2010).

<sup>23</sup>Recall that we are already controlling for overall wind speed in  $\mathbf{W}_{zt}$ , but it has so far not been interacted with taxi time or any other weather measure.



O’Hare (ORD), and New York John F. Kennedy (JFK)), in the following first stage regression:<sup>24</sup>

$$T_{at} = \alpha_{a0} + \sum_{k=1}^3 \sum_{a=1}^{12} \alpha_{ak} T_{kt} I_a + \mathbf{Z}_{at} \boldsymbol{\Theta} + \omega_{at} \quad (4)$$

Figure A1 shows the location of those airports in relation to the California airports. We allow the coefficients  $\alpha_{ak}$  in equation (4) to vary by airport  $a$  by interacting taxi time with an airport indicator  $I_a$ . These interactions allow for heterogeneity in the impact of delays from major airports outside of California  $T_{kt}$  on each of the California airports  $T_{at}$ . This is important as the impact of delays in Atlanta on California airports is likely to differ across airports. Our baseline model utilizes 36 instruments (3 airports outside California interacted with each of the 12 airports in California).<sup>25</sup> We use two-way cluster robust standard errors for inference, clustering on both zip code and day. The two-way cluster robust variance-covariance estimator implicitly adjusts standard errors to properly account for both spatial correlation across zip codes on a given day, which are all due to the same network delays, as well as within-zip code serial correlation in air pollution over time.<sup>26</sup>

The standard conditions for consistent estimation of  $\alpha_1$  in the context of our 2SLS estimator are that  $\alpha_{ak} \neq 0$  in equation (4) and  $\mathbb{E}[T_{kt} \cdot e_{azt} \mid \mathbf{Z}_{zt}, \nu_{za}] = 0$ . Subsequent sections will show that the first condition clearly holds; taxi time at airports on the East Coast leads to large increases in taxi time at California airports. The second condition requires that the error term in the instrumental variable regression be uncorrelated with taxi time at major airports outside of California,  $T_{kt}$ . This condition would be violated if ground congestion in Chicago somehow co-varied with pollution levels in California through reasons unrelated to California airport congestion due to network delays.

While the second condition is not explicitly testable, our data and research design permit several indirect tests. First, we show evidence that taxi time in California is predicted by weather fluctuations at airports inside and outside of California, but the reverse is not true: weather at the major airports in California has no significant effect on taxi time at Eastern airports. Second, we show that network delays propagate East to West rather than West to East. Taxi time in Atlanta is not higher due to increased taxi time in Los Angeles.<sup>27</sup> Further sensitivity checks show that using only taxi time before noon at Eastern Airports or directly instrumenting with observed weather variables at airports in the Eastern United States has little impact on our baseline estimates. In the

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<sup>24</sup>These airports were chosen because they are among the largest airports in the country, they serve different regions, and they are subject to different weather systems. The results are robust to different airport specifications.

<sup>25</sup>Model 2 instruments both  $T_{at}$  and  $T_{at}d_{az}$  with the taxi time outside California  $T_{kt}$  and  $T_{kt}d_{az}$ , and thus uses 72 instruments. Similarly, model 3 instruments all 12 interaction of taxi time  $T_{at}$  at the 12 airports by the taxi time at the three largest airports outside California  $T_{kt}$ , which results in  $12 \times 12 \times 3 = 432$  instruments.

<sup>26</sup>Standard errors clustering on both airport and day tend to be smaller than those using zip code and day. We choose the latter when conducting inference, as they tend to be the more conservative of the two. Results with airport and day clustering are available upon request.

<sup>27</sup>This issue is largely addressed by the difference in time zones between our instrumental variable airports and California. Airplane traffic in the United States generally starts around 6am in the morning and slows down in the evening. Due to the change in time zones, a flight that leaves at LAX in the morning to go to one of the airports does not reach of the three airports outside California before noon. On the other hand, a flight that leaves at 6am on the East Coast will reach California by 9am.

following sections we use the variation in California airport taxi time, and the spatial distribution of emissions from an airport, as a predictor of local air pollution measures in order to better understand contemporaneous relationships between elevated levels of air pollution and hospital admissions.

### 3.2 Aggregate Daily Taxi Time, Local Pollution, and Health

To estimate the pollution-health association in our data we begin by assuming that the relationship between health and ambient air pollution can be summarized by the following linear model:

$$y_{zat} = \beta p_{zat} + \mathbf{Z}_{zt}\boldsymbol{\Pi} + \eta_{za} + \epsilon_{zat} \quad (5)$$

where the dependent variable  $y_{zat}$  is our observable measure of health in zip code  $z$  when paired with airport  $a$  on day  $t$ .<sup>28</sup> The remaining notation is consistent with the previous models,  $\mathbf{Z}_{zt}$  are the same weather and time controls and  $\eta_{za}$  is a zip code fixed effect.

We focus primarily on respiratory related hospital admissions as defined by International Statistical Classification of Diseases and Related Health Problems ICD-9 (Friedman et al. 2001, Seaton et al. 1995). The dependent variable  $y_{zat}$  is the number of admissions to either the emergency room or an overnight hospital stay where either the primary or one of the secondary diagnosis code fell in one of the following admission categories: asthma, acute respiratory, all respiratory, or heart related diagnoses. These daily zip code counts are scaled by zip code population so that the dependent variable represents hospitalization rates per 10 million zip code residents. We also estimate models for diagnoses unrelated to pollution: strokes, bone fractures, and appendicitis. These outcomes are meant to serve as an important test for the internal validity of our research design. Since these health outcomes are unrelated to pollution exposure, they should not be significantly related to changes in pollution.

The coefficient of interest in this model is  $\beta$  which provides an estimate of the effect of a one unit increase in pollution levels on daily hospitalization rates in zip code  $z$  and time  $t$ . Consistent estimation of  $\beta$  requires  $\mathbb{E}[p_{zat} \cdot \epsilon_{zat} \mid \mathbf{Z}_{zt}, \eta_{za}] = 0$ . The inclusion of a zip code fixed effect implicitly controls for any time invariant determinants of local health that also covary with average pollution levels. For example, if relatively disadvantaged households live in more polluted areas and have poorer health for reasons unrelated to air pollution, then the zip code fixed effect will control for this time-invariant unobserved heterogeneity. However, least squares estimation of  $\beta$  will be biased if there are time-varying influences of both health and pollution (e.g., weather), and/or if there is measurement error in  $p_{zat}$ . Since we are proxying for pollution exposure using the average level of pollution in a zip code on a given day, measurement error might be substantial (i.e. people's actual exposure to ambient air pollution might differ significantly from that which is reported by a monitor).

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<sup>28</sup>Our analysis implicitly assumes that we can summarize health responses and behavior at the zip code level and that the effect of interest,  $\beta$ , is stable over time and across airports.

Instrumental variables provide a convenient solution to the bias from omitted variables as well as the bias introduced from measurement error in the independent variable.<sup>29</sup> We use airport ground congestion as an instrumental variable for local pollution levels in the following first stage regression equation:

$$\textbf{First Stage (Model 1): } p_{zat} = \alpha_1 \widehat{T}_{at} + \mathbf{Z}_{zt}\mathbf{\Gamma} + \nu_{za} + e_{zat} \quad (6)$$

The first stage regression, equation (6), estimates the degree to which instrumented airport taxi time  $\widehat{T}_{at}$  predicts local pollution levels in areas surrounding airports.<sup>30</sup> The second stage equation uses the predicted values from the first stage to estimate the impact of local pollution variation on health. We also estimate versions of equation (6) using models that interact  $\widehat{T}_{at}$  with distance, wind speed, and wind direction as in equations (2) and (3), models 2 and 3, respectively.

Aside from the relationship between pollution and health, we also explore “reduced form” relationships between health outcomes and taxi time. These “reduced form” estimates are directly policy relevant; namely, how does aggregate daily taxi time impact the health of nearby residents? Understanding the degree to which variation in airport runway congestion directly impacts health has implications for both managing congestion through either demand pricing mechanisms (e.g., a congestion tax) or a more efficient runway queuing system.

### 3.3 Health Outcomes: Alternative Models

We supplement our baseline health regressions with several alternative models, exploring model specification and model dynamics in more detail. These various regression models are described in more detail below.

#### 3.3.1 Health Outcomes: Non-linearities and Threshold Effects

There is reason to suspect that the relationship between pollution and health outcomes is non-linear in the level of pollution. Do highly polluted days matter more for predicting negative health outcomes than moderately polluted days? We test these hypotheses in two different ways. First, we examine heterogeneity in the dose-response relationship between seasons of the year as pollution levels of CO and NO<sub>2</sub> are higher in the winter months as shown in Figure A3. We interact all variables in all regressions (first and second stage) with a dummy for summer (April-September), thereby allowing the effect to be different for two subsets of the year. Marginal changes at higher

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<sup>29</sup>Instrumental variables only solves the bias from measurement error in the independent variable when the measurement error is classical, namely mean zero and i.i.d. (Griliches & Hausman 1986).

<sup>30</sup>We are using predicted aggregate taxi time  $\widehat{T}_{at}$  as an instrumental variable in these regression models. In standard OLS regression, inference using generated regressors should be corrected for first stage sampling variance (e.g. Murphy & Topel (2002)). When the generated regressor is used as an instrumental variable this is no longer the case. Wooldridge (2002, p. 117) presents a weak set of assumptions for which the standard errors of 2SLS regressions using generated instruments are unbiased. The key assumption turns on strict exogeneity between the error term in the structural model and the covariates used to generate the instrument in the auxiliary regression. See Dahl & Lochner (2012) for a similar approach, using a predicted variable as an instrumental variable in a 2SLS setting. These issues are also discussed tangentially in Wooldridge (1997) and Wooldridge (2003).

baseline levels of pollution (i.e. winter) should be larger than marginal changes at lower levels of baseline pollution (i.e. summer) if the dose-response function was in fact non-linear. There may be other important differences in health outcomes across seasons that could explain these seasonal disparities. For example, pollen levels might be higher in the winter as most precipitation occurs in the winter and hence flowering occurs in early spring. A body that is weakened by the elevated pollen levels might be more (or less) susceptible to pollution shocks. The nonlinearity we are measuring might be an interaction effect with other substances and not directly related to the average pollution level.

Second, we use the over-identified model 3 to instrument higher order polynomials of average daily pollution levels and plot the responding dose-response function. Pollution spreads nonlinearly in wind direction and wind speed, and our overidentified models allow us to identify higher-order polynomials.

### 3.3.2 Health Outcomes: Dynamic Effects and Forward Displacement

By looking at the daily response of health outcomes to contemporaneous pollution shocks, we may be neglecting important dynamic effects of pollution and health. For example, contemporaneous exposure to air pollution may have lagged effects on health, leading people to seek care one or two days after the initial pollution episode. Our contemporaneous regression models might miss these important lagged impacts. Alternatively, health estimates may be driven by various forms of forward displacement. Short-term spikes in pollution might lead individuals on the brink of an asthma or heart attack to experience an episode that would have otherwise occurred in the next few days anyway. Such behavior would overestimate the dose-response function as an increase in hospitalization rates is followed by a decrease once pollution levels subside. We explore the dynamic effects of pollution on health by estimating the following distributed lag model:

$$y_{zat} = \sum_{k=0}^3 \beta_k p_{za(t-k)} + \mathbf{Z}_{zt} \boldsymbol{\Pi} + \eta_{za} + \epsilon_{zat} \quad (7)$$

Instrumented pollution  $p_{zat}$  is again obtained using either model 1, 2, or 3 from previous sections. In the case of forward displacement, the spike in hospital admissions should be followed by a decrease in admissions, and hence  $\sum_{k=0}^3 \beta_k < \beta$ , where the latter  $\beta$  comes from the baseline, contemporaneous regression. In a sensitivity check (available upon request) we include 6 lags and 3 leads.

### 3.3.3 Health Outcomes: Heterogeneity and Self-Selection

Our baseline models rely upon the relatively unattractive assumption that the relationship between pollution and health is the same for everyone in the population. If there is heterogeneity in a person's relative susceptibility to pollution (or in how people respond to adverse health outcomes), then people may sort themselves into locations based on these observed or unobserved differences. This heterogeneity may manifest itself through access to medical care or through biological differences in



the pollution-health relationship among certain segments of the population. Previous research (e.g., Chay & Greenstone (2003)) and results presented in subsequent sections of this paper suggest that health effects differ by observable characteristics of the population. If people sort themselves based on this underlying heterogeneity, then our estimates may identify the average effect of pollution on health for a nonrandom subpopulation in the data (Willis & Rosen 1979, Garen 1984, Wooldridge 1997, Heckman & Vytlacil 1998).

We address these issues in various ways. In a sensitivity check, we limit our estimates to people 65 and older who have guaranteed health insurance in the form of Medicare. Thus, any heterogeneity in hospitalization should no longer be driven by access to health insurance. Another concern is that the severity of the particular health shock determines whether a person will seek emergency care. We therefore also include heart problems as a category, which are severe enough that patients will seek medical help independent of their insurance or financial situation. There may also exist significant heterogeneity based on unobservable characteristics. Previous research suggests that individuals engage in avoidance behavior on days where pollution is predicted to be high (Neidell 2009), which implies there is likely heterogeneity in  $\beta$  as well as correlation between  $\beta$  and  $p_{zat}$ . In a previous version of this paper, we developed a framework to test whether selection on unobserved heterogeneity leads to bias in our estimates (Schlenker & Walker 2011), but did not find this to be the case. The lack of self-selection bias may be in part driven by our research design, where airport-driven pollution is relatively stochastic and unforecastable, making it difficult to select on.

### 3.3.4 Health Outcomes: Poisson Model

Since our dependent variable is measured as hospital visits in a given zip code day (before we convert it to a sickness rate), we also estimate regression models that account for the non-negative and discrete nature of the data. Specifically, we use a conditional (“fixed effects”) quasi-maximum likelihood Poisson model (Hausman, Hall & Griliches 1984, Wooldridge 1999).<sup>31</sup> To account for the endogeneity of pollution exposure, we generalize the standard conditional Poisson model into an instrumental variables setting. To do this, we adopt a control-function approach to the conditional Poisson model (see e.g., Wooldridge (1997) and Wooldridge (2002)), whereby we include the residual ( $\widehat{e_{zat}}$ ) from our first stage regression (i.e., the effect of taxi time on pollution) in our regression equation of interest:

$$\mathbb{E}[s_{zat}|p_{zat}, T_{at}, \mathbf{Z}_{zt}, \eta_{za}] = \eta_{za} \exp(\beta p_{zat} + \gamma_1 \widehat{e_{zat}} + \mathbf{Z}_{zt} \boldsymbol{\Pi}) \quad (8)$$

where  $s_{zat}$  are sickness counts (no longer rates),  $p_{zat}$  is the observed pollution level in a county, and  $\widehat{e_{zat}}$  is the residual from one of the first-stage regression of pollution on taxi time using model 1, 2, or 3. The fixed effect model allows the marginal effect of pollution to differ by zip code. The model

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<sup>31</sup>The Poisson model is generally preferred to alternative count data models, such as the negative binomial model, because the Poisson model is more robust to distributional misspecification provided that the conditional mean is specified correctly (Cameron & Trivedi 1998, Wooldridge 2002).

accounts for the fact that zip codes have different number of residents through the fixed effects  $\eta_{za}$ .

While including the first-stage error purges the estimates of the various selection biases outlined above (Wooldridge 2002, p. 663), the standard errors need to be corrected for the variation coming from the first stage estimation. To account for the first stage sampling error in the  $e_{zat}$ , we bootstrap the regression using a block-bootstrap procedure where we randomly draw the entire history of a zip code with replacement.

## 4 Empirical Results

### 4.1 Aggregate Daily Taxi Time and Local Pollution Levels

We start by examining the effect of airport congestion on pollution levels in surrounding areas. Table A5 gives the first-stage results when taxi time is instrumented using runway congestion at the three major airports outside of California. There is one noteworthy result: For major hubs in California, an increase in taxi time at East Coast airports increases taxi time as delays propagate through the system. On the other hand, the sign reverses for smaller airports: an increase in taxi time at East Coast airports decreases local taxi time. As Pyrgiotisa, Maloneb & Odoni (2013) point out, propagation through the system can have “counter-intuitive results.” If planes bunch up at one hub, the effects on close-by commuter airports can be the opposite as the connectors now arrive more evenly spread, or because flights are canceled.<sup>32</sup>

Table 1 presents regression estimates using the specifications outlined in equation (1), (2), and (3), presented in columns a, b, and c, respectively. Each column represents a different regression, where the dependent variable in the columns (1a)-(1c) is the daily mean CO measured in parts per billion (ppb). Columns (2a)-(2c) report regression estimates for daily mean NO<sub>2</sub>, while columns (3a)-(3c) report estimates for ozone O<sub>3</sub>.<sup>33</sup> Taxi time is reported in thousands so that the coefficients in Table 1 report the marginal effect of a 1000 minute increase in taxi time on local pollution levels. All regressions report robust standard errors, clustering on both zip code and day.<sup>34</sup>

Column (1a) suggests that a 1000 minute increase in taxi time increases ambient CO concentrations in zip codes within 10km of an airport by 45ppb (an 8% increase relative to the mean,

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<sup>32</sup>For example, flights out of Santa Barbara frequently get canceled if Los Angeles is backed up to reduce the queue of incoming airplanes into Los Angeles.

<sup>33</sup>OLS estimates are presented in Table A6.

<sup>34</sup>The heavily over-identified models from equation (3) impose significant computational burdens when estimating IV models containing two-way, cluster-robust standard errors. To circumvent this issue, we report the results from running the first stage and then using the predicted values in the second stage without accounting for the fact that we are using generated regressors in the second stage. Plugging in the predicted regressors is computationally much easier because we don’t cluster all the first-stage regressions, instead we simply recover the point estimates from each regression. Two-way cluster robust routines require estimating three variance-covariance matrices, one corresponding to the first cluster group, one corresponding to the second cluster group, and one corresponding to the two-way expansion of the two groups. Since we have more than a hundred instruments in model 3 (12 variables times 12 airports times 3 east coast airports = 432 first stage regressions), this imposes a significant computational burden. To understand the likely magnitude of this bias, Table A7 reports two sets of standard errors for equations (1) and (2): (i) the IV results; and (ii) running the first stage and using the predicted values in the second stage with two-way clustered errors but no other adjustments. The results suggest that the standard errors from the IV are quite similar to those from manual 2SLS.

or 12% of the day-to-day standard deviation). Since the standard deviation of taxi time at LAX in Table A1 is 1852, a one-standard deviation increase in taxi time leads to 0.23 standard deviation increase in CO pollution of the zip codes around LAX. Column (1b) of Table 1 includes an interaction of taxi time with distance to the airport. The non-interacted taxi time coefficient now reports the effect of airplane idling on pollution levels directly at the airport. The point estimate implies that a one standard deviation increase in taxi time at LAX leads to 0.28 standard deviation increase in CO levels in areas adjacent to LAX. The interaction term shows how this effect decays linearly with distance.

Lastly, column (1c) reports the coefficients from the estimated version of equation (3) that interacts taxi time with wind speed and wind angle from an airport. The F-test for the joint significance of these coefficients is given in the last two rows of the table and shows that they are highly significant. Since individual coefficients are difficult to interpret, we plot the marginal effect of an extra 1000 minutes of taxi time for four wind speeds in the first row of Figure 3. Wind speeds increase from left to right. The color indicates the marginal impact ranging from low (blue) to high (red). If a zip code is directly downwind, it is on the positive x-axis, while areas upwind are on the negative x-axis.<sup>35</sup> Figure 3 makes clear that there is significant spatial heterogeneity in the marginal effect of taxi time, and this heterogeneity depends on distance from an airport, wind speed, and wind direction. As such, equation (3) (i.e. model 3) is best able to capture this heterogeneity.

Columns (2a)-(2c) of Table 1 give estimates pertaining to the effect of taxi time on NO<sub>2</sub> levels. The results are comparable to those from CO, although the linear decrease in distance from the airport is not significant. A one standard deviation increase in taxi time at LAX increases NO<sub>2</sub> concentrations by roughly 1ppb, or 10% of the day-to-day standard deviation. The second row of Figure 3 shows again that downwind areas are much more impacted than upwind areas. Both Table 1 and Figure 3 show that the relative impact of NO<sub>2</sub> is different than CO: the range of marginal impacts for CO in Figure 3 is between -71% and +43% relative to the average impact from column (1a) in Table 1. In contrast, the marginal effect of taxi time on NO<sub>2</sub> varies between -60% and +33% relative to the average effect from column (2a) of Table 1. The spatial pattern is also somewhat different. In subsequent sections, we use these relative differences in pollutant dispersion to jointly estimate the effect of both CO and NO<sub>2</sub>. Recall from Section 1 that CO emissions are higher during low power operation, while NO<sub>2</sub> is higher during high power operation. Larger wind speeds require more thrust during takeoff and hence change the mix of CO and NO<sub>2</sub> emissions.

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<sup>35</sup> Areas downwind are more affected by taxi time than areas upwind. For the very highest wind speeds, the largest marginal impact of taxi time can be found just upwind from the centroid of the airport (although the average marginal impact remains highest downwind). This is possibly due to the fact that airplanes start against the wind and mostly line up in the opposite direction, i.e., the direction in which the wind is blowing. Local wind is highly predictive of congestion. When local wind is strong and the average local taxi time is high and the queue is long, an additional unit of congestion due to network delays will hence “add” an additional plane that is idling upwind from the airport centroid. For example, the four runways of LAX are between 2.7km and 3.7km long, which is significant as we are examining monitors within 10km of the airport centroid.

Finally, columns (3a)-(3c) replicate the same analysis for ozone ( $O_3$ ), a pollutant that is not directly emitted from airplanes.<sup>36</sup> The results in Table 1 suggest that airport taxi time has little significant impact on ozone levels, although some of the interaction terms are significant. In the remainder of the analysis we focus on CO and  $NO_2$ , the two criteria air pollutants for which airplanes are large emitters, while acknowledging that we may be picking up the health effects of other pollutants that are correlated with airplane emissions.

Our baseline pollution estimates presented above come from models in which airport taxi time is instrumented with taxi time at large airports outside of California. We instrument taxi time because delays and runway congestion might be correlated with local weather, which in turn might impact pollution levels. In addition, there is likely measurement error in our taxi time variable as it only includes domestic, commercial flight activity. While we control for weather in our regressions, there might be unobserved weather (or other) variables that jointly impact both pollution and taxi time. Table A6 replicates the baseline IV analysis of Table 1 using local taxi time at California airports, which is not instrumented. The estimated effect is generally half as big for CO and  $NO_2$ . The smaller OLS estimates are consistent with adverse weather (e.g., precipitation) causing both airport delays and at the same time reducing ambient air pollution. Alternatively, these results could be driven by the well known attenuation bias stemming from measurement error in fixed effects models. In the remainder of the paper we rely on instrumented taxi time stemming from network delays.

We use taxi time at three major airports in our baseline regressions: Atlanta (ATL), Chicago (ORD), and New York (JFK). Table A7 presents first-stage F-statistics if we instrument taxi time at California on up to four airports outside of California. Recall that we allow the coefficients to vary by airport, as network congestion will have different absolute effects on California airports. Irrespective of whether we use 1, 2, 3, or 4 airports outside of California, the F-statistic is well above 10. In our baseline model we use three airports that cover weather patterns in three regions of the Eastern United States: Southeast (Atlanta), Midwest (Chicago), and Northeast (New York JFK), and the first-stage F-stat is 50. The fourth large airport outside of California that we include in columns (d) is Dallas/Fort Worth (DFW). While results are not particularly sensitive to including DFW, we exclude it from our baseline specifications as it is significantly closer to California airports and thus may be more endogenous than the other three airports (i.e. Dallas/Fort Worth may be delayed because California airports are delayed).

Reverse causality is less of a concern for the other three airports: A flight that leaves a California airport at 6am will not reach Atlanta, Chicago, or New York until roughly noon due to the change in time zones. Table A8 tests for reverse causality directly by regressing taxi time at an airport on eight weather measures we generally include as controls: a quadratic in minimum and maximum temperature, precipitation, as well as wind speed.<sup>37</sup> The column heading gives the airport at which

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<sup>36</sup>Ozone is formed through a complicated chemical reaction between both nitrogen dioxides and VOC's in the presence of sunlight. As Auffhammer & Kellogg (2011) have shown, increasing VOC in VOC-rich environments can have no effect on ozone or slightly decrease it, while it will increase ozone if VOCs are limited compared to  $NO_x$ . This poses a challenge for the monotonicity assumption behind IV regressions.

<sup>37</sup>Weather measures in our baseline regression also include the direction in which the wind is blowing relative to

the congestion is measured while the row indicates the airport at which the weather variables are measured.<sup>38</sup> The table reports p-values of a hypothesis test pertaining to the joint significance of the weather variables. The diagonal is highly significant as local weather measures impact airport taxi time. While weather at the eastern airports (ATL, ORD, or JFK) sometimes impacts taxi time at the two largest airports in California (LAX and SFO), the reverse is not true. This is consistent with weather at Eastern airports causing local network delays that propagate through the airspace and impact taxi time in California. The reverse direction does not hold. California airports do not affect East Coast airports on the same day. This result is not simply an artifact of there being less weather variation in California, as weather at LAX significantly impacts taxi time at SFO.

We have also run two sensitivity checks to further rule out endogeneity through reverse causality, the results of which are reported at the end of the subsequent section on health effects and shown in Table A13. First, we only utilize the combined taxi time between 5am and noon at the three major Eastern airports to rule out California feedback effects. This reduces the F-stat in model 1 from 50 to 35.5, but the results remain similar to baseline estimates. Second, instead of using taxi time at the three major Eastern airports, we use the eight weather variables at each of these airports. Since this effectively increases the number of instruments by a factor of eight, we no longer estimate model 3 (which had 432 instruments to begin with). The F-statistic for the weather-instrumented regression is 5436. Again, results remain similar to our baseline estimates but the standard errors in the second stage increase. Going forward we instrument using the overall daily taxi time, as it has a higher F-statistic than focusing only on the mornings yet is more tractable than using weather measures, which would result in 3456 instruments in model 3.

We conduct two last robustness checks. First, since the variation in pollution due to delays outside of California should be uncorrelated with weather in California, we have estimated models (not reported) that exclude California weather controls altogether. Reassuringly, our baseline estimates for the most important pollutant (CO, see below) are similar whether we include or exclude California weather controls, but the error terms increase. Second, there may of course be some omitted variable that affects congestion outside of California and health outcomes in California. This hypothesis is not directly testable, but we have estimated models (available upon request) which include taxi time at other CA airports as a control variable in our baseline reduced form regressions, and the results remain very similar.

To put the magnitude of these effects into perspective, it is useful to consider the current ambient air standards in place for CO as regulated by the EPA under the Clean Air Act. The current one hour carbon monoxide standard specifies that pollution may not exceed 35 ppm (or 35000 ppb) more than once per year. California has their own CO standard which is 20ppm. A one standard deviation increase in LAX airplane idling (1852 minutes) translates into an 83 ppb increase ( $44.78 \times 1.852$ ) in carbon monoxide levels for areas within 10km of LAX using estimates

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the direction in which the zip code is located. Since the dependent variable in the current regression is at the airport level and not the zip code level, these variables are not well defined and hence dropped.

<sup>38</sup>If we pair airport taxi time with weather from another airport, we also include the local weather measure as control. The local weather measures are not included in the joint test of significance.



from column (1a) of Table 1. Adding this number to the average daily maximum CO level at zip codes from Panel A of Table A2 (1234 ppb), the estimated increase in pollution concentrations is far below the current EPA standard. Similarly, for NO<sub>2</sub>, the current EPA 1-hour standard is 100 ppb. Using estimates from column (2a) of Table 1, a standard deviation increase in LAX taxi time would lead to a 1ppb increase in NO<sub>2</sub> levels. Evaluated relative to the average daily maximum NO<sub>2</sub> levels of 35.5 ppb, these are again well below the ambient criteria standard. Note that the maximum of the maximum daily NO<sub>2</sub> levels is above the standard as some areas are out of attainment. The remaining sections estimate the social costs of these congestion related increases in ambient air concentrations by focusing on health outcomes of the populations most affected by these emissions.

## 4.2 Effects of Taxi Time on Local Measures of Health

We begin by investigating the “reduced form” health effects of airports, relating aggregate daily taxi time to local measures of health. Namely, how does variation in airport congestion predict local health outcomes? Table 2 presents the results from a regression relating daily measures of airport taxi time to local hospital admissions for the overall population as well as two susceptible subgroups: individuals below 5 years of age and individuals aged 65 and above. The dependent variable is measured as the daily sum of hospital and emergency room visits for persons living in a particular zip code scaled by the population (per 10 million individuals) in that particular zip code. The regressions are weighted by zip code population size, and taxi time is instrumented using taxi time at three major airports in the East. The estimated coefficient on the taxi time variable corresponds to the increased rate of hospitalizations per 10 million individuals in a zip code for an extra 1000 minutes of taxi time. Using various diagnosis codes, we examine the impact of taxi time on asthma, respiratory, and heart related admissions separately. As a falsification exercise, we also estimate the incidence of taxi time on strokes, bone fractures, and appendicitis rates. The reported standard errors are clustered on both zip code and day.

For the overall population (Panel A), all respiratory sickness rates as well as heart problems are significantly impacted by taxi time, while the placebo effects for stroke, bone fractures, and appendicitis are not significantly affected. Results become larger in magnitude for the at-risk age groups. For the population 65 years and above, the incidence of stroke and bone fractures is marginally significant at the 10% level. This may be do to statistical chance or may be explained by the fact that senior citizens may also be more susceptible to sicknesses that covary with one another (e.g., a respiratory problem might make them fall and break a bone). Additionally, Medicare provides doctors implicit incentives to add additional diagnosis codes to receive higher reimbursement rates. Consistent with this explanation, models for which the dependent variable is measured only using the primary diagnosis code, the placebo effects for 65 and older are no longer significant.

## 4.3 Hospital Admissions and Instrumented Pollution Exposure

Results thus far have shown that aggregate airplane taxi time generates variation in pollution levels of nearby communities. We exploit this variation to examine the relationship between pollution and

health explicitly. Table 3 summarizes regression results for various pollutants and illnesses using a variety of traditional econometric specifications. Each entry corresponds to a different regression, where the dependent variable is measured as hospital admission rates, and the independent variable is the daily mean ambient pollution concentration in a particular zip code. As before, regression estimates are weighted by zip code population and standard errors are clustered on both zip code and day.<sup>39</sup>

The first row within each panel presents estimates from a pooled OLS version of equation (5) without any controls  $\mathbf{Z}_{zt}$ , which suggests that increased ambient air concentrations lead to adverse health outcomes for respiratory and heart problems. Since various pollutants are often correlated with one another, these estimates should be interpreted with caution, as the pollutant of interest will proxy for other correlated air pollutants. Each consecutive row adds more controls. The second row uses time controls (year, month, weekday, and holiday fixed effects), and the third row additionally adds weather controls (quadratic in minimum and maximum temperature, precipitation and wind speed as well as controls for wind direction). To control for unobserved, time-invariant determinants of health, the fourth row of each panel in Table 3 reports regression estimates from a model using zip code fixed effects. The model is identified by examining how *within* zip code changes in pollution are related to hospitalization rates of that particular zip code. Again, pollution is often strongly correlated with health, although the estimates in the fourth row are usually smaller than those in the first three. These smaller point estimates are consistent with time-invariant omitted variables introducing bias into the estimates from rows one through three. Alternatively, classical measurement error in the pollution variable may lead to significant attenuation bias in fixed effects models (Griliches & Hausman 1986), and this may be responsible for the smaller point estimates in the last row.

Aside from attenuation bias, fixed effects models may also suffer from biases introduced by any unobserved, time-varying determinants of both pollution and health (e.g., weather). To explore this issue further, Table 4 presents instrumental variable estimates of the pollution-health relationship, using instrumented aggregate airport taxi time as an instrumental variable for daily mean pollution. Table 4 presents results for both the overall population in Panel A as well as children below 5 in Panel B and people aged 65 and above in Panel C.<sup>40</sup> The three rows (labeled model 1-3) use (i) taxi time, (ii) taxi time interacted with distance, and (iii) taxi time interacted with distance, wind speed, and wind direction, respectively. These are the specifications outlined in equation (1), (2), and (3) above.

The estimates in Table 4 are usually an order of magnitude larger than the OLS, fixed-effects estimates from Table 3. To put the magnitudes into perspective: The average asthma sickness rate for the overall population is 339 per 10 million inhabitants (Panel A1 and A2 of Table A3). The

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<sup>39</sup>Unweighted regressions yield similar results and are available upon request.

<sup>40</sup>Results for the two remaining groups: children ages 5-19 and adults ages 19-64 are given in Table A9. Children between 5 and 19 years of age show no sensitivity to pollution shocks. Conversely, the estimated dose-response for adults are roughly comparable to the baseline estimates, which is not surprising since they are the largest share of the overall population.

asthma coefficient for CO (model 3) in Table 4 implies that a one standard deviation increase in CO pollution leads to an additional  $0.194 \times 368 = 71$  asthma attacks per 10 million people,<sup>41</sup> which is 21% of the daily mean.<sup>42</sup> This suggests that fluctuations in air pollution are a major cause of asthma related illnesses. For heart related problems, the relative magnitude is 18% of the daily mean. It is important to note that the estimated CO effect may not necessarily be coming from CO itself but from some other pollutant that’s co-emitted in jet exhaust that we do not observe (e.g. a toxic VOC or particulate matter that’s emitted due to incomplete combustion). In addition to measurement error or avoidance behavior, the fact that variation in CO comes from airplanes may be a further explanation for the discrepancy between OLS and IV estimates. However, the Federal Aviation Administration (2005) suggests that aircraft engines produce the same types of emissions as automobiles, which are the largest single source of carbon monoxide emissions in the United States.

Models 2 and 3 in Table 4 estimate over-identified models instrumenting pollution with both taxi time and taxi time interactions. While estimates in model 2 are similar to those from model 1, estimates from model 3 are generally smaller. The reason for the difference in magnitudes between models 2 and 3 is not entirely clear, but we believe there are two competing explanations. The first explanation stems from the inability of models 1 and 2 to capture the spatial heterogeneity in the effect of taxi time. Recall that model 3 uses distance as well as wind direction and wind speed. Marginal impacts of airport congestion vary greatly across space as shown in Figure 3, much more than in a model that only includes distance. Failing to model this heterogeneity in pollution exposure may lead to inaccurate scaling of the reduced form relationships in our IV/2SLS setting. A competing explanation as to why model 3 estimates differ from models 1 and 2 stems from measurement error in the location of exposure. While we know the exact location of each pollution monitor and hence can correctly model the pollution surface in space, we only know the zip code of a person’s residence and the hospital, not the exact location where they fell ill. As a result, all models will pair sickness counts with incorrect pollution measures if they are not close to the centroid of the zip code when they fell ill, but this might be aggravated by model 3 that explicitly uses the spatial distribution of the pollution surface. Table A10 investigates this latter hypothesis by looking at various subsets of the data. Panel A presents our baseline results, Panel B assigns pollution data based on the zip code of the residence, while Panel C assigns pollution based on the hospital zip code. A few results are noteworthy: first, the estimates using model specification 3 are very close to the estimates using specification 1 and 2 in Panel B1 where we only count sicknesses if both the zip code of the residence and hospital are within 10km of the same airport. On the other hand, model specification 3 diverges in panel B2 where the hospital zip code is outside the 10km radius from airports, perhaps because we measure exposure less accurately (e.g. the person

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<sup>41</sup>Panel A of Table A2 shows that the standard deviation for CO is 368.

<sup>42</sup>This back-of-the-envelope calculation increases the pollution level in each zip code by the average *overall* standard deviation of pollution fluctuations. Moreover, the average sickness rate is not population weighted. In subsequent sections, we increase pollution in each zip code by the zip-code specific standard deviation in pollution fluctuations and calculate the population-weighted average sickness count. The relative impact decreases to 17% of the daily mean under the linear probability model and 19% under a Poisson count model.

might have been at work). In addition, panel B3 shows that there are no significant results where the hospital is within 10km of another airport, suggesting that we are not simply picking up a daily pattern that is common to all airports.<sup>43</sup> As a secondary bit of evidence, model 3 in Panel B of our baseline Table 4 gives comparable point estimates to model 1 and 2 for children under the age of 5, whom are more likely to be at home or in a close-by day care. Due to these competing explanations for the differences across models, we continue to present all model estimates whenever possible, allowing the reader to choose their preferred estimate.

There are two additional explanations for the discrepancies between models 3 and 1 and 2 which we find less salient. First, there is a well-known bias of 2SLS estimators when instruments are weak and when there are many over-identifying restrictions (Bound, Jaeger & Baker 1995). In linear models with iid errors, Stock, Wright & Yogo (2002) propose rule-of-thumb thresholds for F-statistics for the first stage. However, in both the non-iid case (i.e. with clustered standard errors) and in cases with multiple endogenous variables, less is known about the relationship between the F-statistic and the properties of instrumental variables estimates. Baum, Schaffer & Stillman (2003) suggest comparing the test statistic to the Stock, Wright & Yogo (2002) critical values for the Cragg-Donald F statistic with a single instrument. According to this metric, results from Table 1 suggest that model 3 is a strong first-stage predictor of local pollution levels with a F-statistic that is 14 for CO pollution and to a lesser extent for NO<sub>2</sub> pollution (F-stat of 5). The first stage in model 3 is not as strong as in models 1 and 2, and the model is highly over-identified with 12 excluded instruments. Bound, Jaeger & Baker (1995) show how the bias of 2SLS increases in the number of instruments and decreases in the strength of the first stage. The bias of 2SLS in the case of weakly identified or over-identified models is towards the OLS counterpart. Since this is consistent with model 3 estimates in Table 4 being smaller than both model 1 and 2 but still above the OLS estimates, Table A11 estimates models 2 and 3 using Limited Information Maximum Likelihood (LIML), which is median-unbiased for over-identified, constant-effects models (Davidson & MacKinnon 1993). Results remain similar, which suggests that weak instrument attenuation is less of a concern (Angrist & Pischke 2008). Finally, a second alternative explanation for why model 3 gives lower point estimates is that the hourly wind data represent snapshots of the wind speed and direction and include significant measurement error. However, this is somewhat at odds with the fact that we find such significant spatial patterns in the pollution regressions.

Panels B and C of Table 4 present estimates for children and senior citizens. While the dose-response relationships are larger, so are average sickness rates. In relative terms, a one standard deviation increase in CO pollution now causes a 37% increase in asthma cases for children under 5 compared to the average daily mean. On the other hand, a one standard deviation increase in CO pollution causes a 24% increase in heart problems for people 65 and above. The higher *absolute* sensitivity in Panel B and C suggests that there may exist significant heterogeneity in the population response to ambient air pollution exposure. Since the population aged 65 and older has guaranteed access to health insurance through Medicare, they may be more inclined to visit

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<sup>43</sup>If we assign pollution based on the hospital zip code in panels C, results are generally not significant.

the emergency room or hospital relative to the rest of the population, leading to larger estimated effects. On the other hand, the relative magnitude compared to average sickness rates are only slightly larger than for the overall population.

Columns (3)-(5) of each panel includes results for one of three placebos: strokes, bone fractures, and appendicitis. Both strokes and appendicitis are severe enough that people should go to the hospital. None of the results are significant for the overall population in Panel A. Consistent with the reduced form evidence in Table 2, some of the coefficients in Panel C are significant at the 10% level. In Table A12 we replicate the analysis using only the primary diagnosis code. None of the placebo regressions remain significant. Since we are interested in the overall effect of pollution on hospitalization rates, our baseline models continue to count total sickness counts for both primary and secondary diagnoses.

Table A13 further investigates the sensitivity of our IV estimates to different choices of instrumental variables. As a point of comparison, Panel A replicates the baseline results of Table 4 for all ages. Panel B instruments for pollution using only the taxi time between 5am and noon at Eastern airports to rule out endogeneity through reverse causality. The results remain robust to this change. Panel C goes one step further and instruments for taxi time at California airports using only weather measures at the three major airports in the Eastern United States. While the point estimates remain comparable, the standard errors generally increase.<sup>44</sup>

#### 4.3.1 Jointly Estimating the Effect of Ambient Air Pollutants

A common challenge in studies linking health outcomes to pollution measures is that ambient air pollutants are highly correlated. It is therefore difficult to determine empirically which pollutant is the true cause of any observed changes in health. Our research design provides one possible solution to the identification problem. Wind speed and wind direction differentially affect both CO and NO<sub>2</sub> dispersion patterns. Moreover, the rate of CO and NO<sub>2</sub> emissions depend on the thrust produced by the engine, and higher wind speeds require more engine thrust. Wind speed hence impacts both the rate at which pollutants are produced and how they disperse. Table 5 estimates the joint effect of both CO and NO<sub>2</sub> on health using our first stage model with wind speed and wind direction interactions (model 3).

In all specifications for which we have multiple endogenous variables, we report the Angrist & Pischke (2008) conditional F-statistics in the tables and text, although these are somewhat hard to interpret. As mentioned above, there are no rule-of-thumb thresholds for linear models with non-iid errors or for models with multiple endogenous variables. When comparing the conditional F-statistics to the Stock, Wright & Yogo (2002), the F-statistics suggest that the first stage is “weak”. Perhaps more usefully, in all specifications for which we have multiple endogenous variables, we also present two tests that are robust to issues pertaining to weak instruments, the Anderson-Rubin test statistic and the closely related Stock-Wright (2000) S statistic. The null hypothesis tested in both cases is that the coefficients of the endogenous regressors in the structural equation are jointly equal

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<sup>44</sup>We do not estimate model 3 using weather variables as it would include 3456 instruments.



to zero, and, in addition, that the overidentifying restrictions are valid. We use a cluster-robust version of both test statistics that has the correct size even under weak identification (Chernozhukov & Hansen 2008). The tests are equivalent to estimating the reduced form of the equation (with the full set of instruments as regressors) and testing that the coefficients of the excluded instruments are jointly equal to zero. In most specifications, inference based on the Anderson-Rubin and Stock-Wright tests are consistent with inference based on the Wald test of the same null hypothesis. This suggests that we are not drawing spurious inferences based on weak instruments. We also present results using LIML because LIML is approximately median unbiased for overidentified models, and the results are similar between 2SLS and LIML. When 2SLS is subject to weak instrument bias, the 2SLS estimand will diverge significantly from the LIML estimand toward the OLS estimand. Thus, the fact that LIML and 2SLS deliver similar results assuages our concerns pertaining to significant biases associated with weak instruments.<sup>45</sup>

Table 5 shows that the coefficient for CO remains comparable in size to our baseline estimates from Table 4, albeit slightly larger. Conversely, the coefficients on NO<sub>2</sub> switch sign and are mostly negative and insignificant. We have also used the methods proposed by Chernozhukov & Hansen (2008) to build non-spherical confidence regions for the multiple endogenous variables. Within the joint parameter space of CO and NO<sub>2</sub>, the joint confidence region lies in the quadrant where CO is positive and NO<sub>2</sub> is weakly negative.<sup>46</sup>

We interpret these findings as evidence that the returns from regulating CO exceed those from regulating NO<sub>2</sub>, at least for the population that comprises our sample. One possible explanation for our results stems from the work done by Auffhammer & Kellogg (2011). Figure 9 of Auffhammer & Kellogg (2011) shows that the Southern California coastline, the location of most of the zip codes in our study, ozone generation seems VOC limited, i.e., a reduction in VOC reduces ozone. Conversely, regions further inland and in Northern California are NO<sub>2</sub> limited. Reducing NO<sub>2</sub> in areas that are VOC limited has little effect on ozone, and this may be the reason we observe small and insignificant results for NO<sub>2</sub>. In the remainder of the paper, we therefore focus on CO.

#### 4.3.2 Threshold Effects and Non-Linearities in the Pollution-Health Relationship

We explore the functional form of the dose-response function in four separate ways. First, Table A14 estimates the relationship separately for the summer (April-September) and the winter (October-March). Each panel of the table provides the point estimates for the two seasons from a joint regression where all variables and instruments are interacted with seasonal dummies as well as the p-values of a test whether the coefficients are the same. Especially for the case of children under the age of 5, the effect seem to be significantly higher during winter months when average pollution levels are higher.

Recall that CO and NO<sub>2</sub> pollution are higher during the winter months, so a nonlinear dose-response function that has increasing marginal damages of pollution should exhibit larger coeffi-

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<sup>45</sup> A similar diagnostic exercise of this nature can be found on pages p. 213-215 of Angrist & Pischke (2008).

<sup>46</sup> The full set of results, which consist of 2 dimensional plots for each hypothesis test, are available upon request.

cients for the winter months. The coefficient for the winter months is almost always larger than for the summer months for the illnesses that are related to pollution (columns (1a)-(2)). These results are consistent with increasing marginal impacts of pollution. However, there may be other important differences in health outcomes across seasons that could explain these disparities. An obvious candidate for differences between the summer and the winter would be the level of ambient ozone concentrations which tend to be much higher in the summer than the winter. In additional results (Table A15), we control for ozone levels as a potential confound and the results are nearly identical. One possible explanation for why ozone does not impact the baseline regression results stems from measurement error in the excluded ozone regressor and/or avoidance behavior pertaining to ozone.

We have also explored models which estimate the possible non-linear effects of pollution on health outcomes by including higher order polynomials. Models with higher order pollution terms increase the number of endogenous variables in our regressions, and we use the overidentified model 3 to instrument for the higher order terms. Since higher order polynomials can be difficult to interpret, Figure A5 plots the predicted marginal effects of the pollutant on a range of health outcomes as a function of the *level* of the pollutant on the given day. That is, we plot the dose response function, where the y-axis measures the health response and the x-axis measures the level of pollution. Since we are fitting non-linear models, the responsiveness is allowed to vary across the x-axis. The dashed line displays the results from our baseline, linear dose-response model (constant marginal damage). The solid represents results from a quadratic model where the 95% confidence interval is added in grey. The four columns represent the four sicknesses that are related to pollution fluctuations (asthma, acute respiratory, all respiratory, and heart problems, respectively). The predicted marginal effect is plotted over the empirical distribution of daily pollution levels, from the 5th to the 95th percentile.<sup>47</sup> While there is some evidence that respiratory problems (columns 1-3) exhibit increasing marginal damages as pollution levels start to increase, again especially for children under the age of 5, the confidence intervals reflect an inability to reject the null that the damage function is constant over the observed range of CO values.

We have investigated non-linearities in two additional ways that are broadly consistent with the findings above (results available upon request). First, we estimated models whereby we interacted our daily pollution variation of interest with the mean pollution *level* in a zip code. This allows the dose-response curve to vary (linearly) in the level of average pollution levels of a zip code. If this interaction term is zero, this would support the hypothesis that the marginal effect of a one unit increase in emissions is the same regardless of the level of ambient air pollution (i.e. a constant, linear dose-response). If the coefficient on the interaction was significantly positive, then this would support the hypothesis that the marginal effect of ambient air pollution on health outcomes is progressively worse in areas with higher than average pollution levels. A challenge with this particular test is that the average level of ambient air pollution in a zip code can be correlated with many observed and unobserved factors that may contribute to heterogeneity in the dose-response relationship. For example, people in more polluted areas may lack basic preventive

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<sup>47</sup>Figure A4 shows the observed distribution of daily CO levels in our data set.

health services and thus be *more* responsive to marginal changes in air pollution because of their underlying health conditions rather than any non-linearity in the dose-response. Nevertheless, results suggest (available upon request) that CO exhibits an increasing dose-response function. Second, we explored the shape of the dose-response function in an OLS, fixed effects setting. While we think the regression coefficient *magnitudes* may be attenuated by things such as measurement error and/or avoidance type behaviors, the *shape* of the dose-response curve is likely less sensitive to these concerns (unless of course the bias varied with the level of pollution - which might happen through avoidance behavior such as “bad air day” alerts). We use this logic to explore the shape of the dose-response function by fitting OLS, fixed-effect regression models that include polynomials in the daily mean pollution level (i.e. quadratic, cubic, or quartic). We then plot the predicted marginal effects of the pollutant on a range of health outcomes as a function of the *level* of the pollutant on the given day (as in Figure A5). We see that for both asthma and respiratory illness, the predicted marginal effect is increasing in the level of the pollutant. The patterns suggests some sort of “threshold” by which the marginal effect of CO on health outcomes “flattens out”.

While the various results in this section come from different econometric models, the conclusions pertaining to the shape of the dose-response function remain similar across the specifications. The evidence suggests that the marginal effect of pollution is increasing in the level of the pollutant, but at a decreasing rate. The diminishing marginal damages of the dose-response function is also consistent with modern evidence from epidemiology (see e.g. Pope et al. (2009) and Pope III et al. (2011)).

### 4.3.3 Potential Confounding Sources of Variation

While our estimates suggest that CO is primarily responsible for the observed health responses, there may be other sources of unobserved, concomitant variation that may lead to similar relationships. For example, while we estimate the effect of CO and NO<sub>2</sub> in the same model, we do not directly control for other pollutants such as ozone. It seems unlikely that ozone O<sub>3</sub> is causing the observed relationship. As mentioned above, Table A14 estimates the relationship separately for the summer (April-September) and the winter (October-March). Ozone is higher during the summer, while CO and NO<sub>2</sub> are higher during the winter. The observed health effects are larger and more significant during the winter time when ozone is not a big problem. We have also estimated models that directly control for ozone (Table A15), and the results remain similar and a bit more precise than our baseline estimates. The standard errors are also much larger for the summer, especially in the case of acute respiratory problems and overall respiratory problems. This is not surprising, because other pollutants like ozone also impact health outcomes, which will be part of the error term.

One potential omitted variable that we unfortunately cannot measure well is particulate matter, a pollutant which may emerge from combustion emissions and has been shown in the past to increase infant mortality due to respiratory causes (Currie & Neidell 2005). Particulate matter monitors in California are limited in both their spatial and temporal coverage; readings on ambient particulate

monitors are conducted every few days (as opposed to hourly data from other pollutants), and there are far fewer monitors. These limitations do not square well with our research design which relies on high-frequency, daily variation across very localized areas. Nevertheless, we have directly explored the degree to which particulate matter predicts adverse health outcomes for the subsample of days and locations for which we have particulate monitor data. Table A16 presents results using the full set of particulate monitors for PM<sub>2.5</sub>.<sup>48</sup> Table A16 suggests that PM does not have much explanatory power in predicting health outcomes, although the standard errors preclude definitive conclusions.<sup>49</sup> Recall that Los Angeles Airport is not a significant point source of particulate matter. While it is the single-largest point source for CO emissions in the state of California, it only ranks 2763 and 2782 among emissions of PM<sub>10</sub> and PM<sub>2.5</sub>. Even still, we believe that some amount of caution is warranted in interpreting CO as the unique pollutant-related causal channel leading to adverse health outcomes; there may be in fact other unobserved sources of ambient air pollution that covary with CO that may also affect health.

#### 4.3.4 Inpatient versus Outpatient Data

Traditionally, studies have relied on Inpatient data sets to examine health responsiveness to various external factors such as pollution. One limitation of such data is that a person only enters the Inpatient data set if they are admitted for an overnight stay in the hospital. Many ER visits result in a discharge the same day and hence never result in an overnight stay. Starting in 2005, California began collecting Outpatient (Emergency Room) data. Previous published estimates all relied on Inpatient data only. To better understand the differences between these two datasets as well as compare our results to those from the previous literature, we replicate the analysis using sickness counts from only the Inpatient data in Panels A1-C1 in Table A17. By the same token, panels A2-C2 only uses the Outpatient data.<sup>50</sup> Not surprisingly, there is a significant relationship between pollution and heart problems (column 2) in the Inpatient data for patient ages 65 and above (as these conditions usually require an overnight stay), but no or very limited sensitivity of asthma or overall respiratory illnesses (column 1a and 1c) to pollution. Conversely, the Outpatient (ER) data shows a much larger sensitivity of respiratory problems to changes in pollution. These results show the importance of Outpatient (ER) data when studying the morbidity effects of ambient air pollution on health outcomes.

#### 4.3.5 Temporal Displacement and Dynamics

Our baseline regression models examine only the contemporaneous effect of pollution on health. Contemporaneous estimates may lead to underestimates of the total effects of air pollution on health

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<sup>48</sup>Unfortunately, we only observe 2 PM<sub>10</sub> pollution monitors within 15km of an airport (or equivalently 2 zip codes) which makes our research design infeasible due to the importance of distance and wind angle/speed heterogeneity.

<sup>49</sup>All of the estimates in Table A16 come from limited information maximum likelihood estimates as opposed to 2SLS (although results are similar).

<sup>50</sup>Patients that enter the ER and are later admitted for an overnight stay are dropped from the ER data to avoid double counting.

if health effects respond sluggishly to changes in pollution. Conversely, estimates may overstate the hypothesized effect due to temporal displacement: if spikes in daily pollution levels make already sick people go to the hospital one day earlier, contemporaneous models overestimate the true effect associated with permanently higher pollution levels. If temporal displacement is important, the contemporaneous increase in sickness rates should be followed by a decrease in sickness rates in subsequent periods.

We investigate both of these issues by estimating a distributed lag regression model, including three lags in the pollution variable of interest. Table 6 presents the distributed lag results of pollution for the overall population. We present individual coefficients as well as the combined effect (the sum of the four) in the last row of each panel. To preserve space, we only list the results for the sickness categories that are impacted by changing CO pollution levels. Since regulatory policy is concerned with the health effects of a permanent change in pollution, we focus on cumulative effects of the model over the estimated 4 day horizon. The cumulative effect is slightly larger than the comparable baseline results in Table 4. This might be because some individuals delay hospital visits, although the exact dynamics are hard to determine empirically given the lack of significance of the individual coefficients. We have also experimented with different leads/lags (available upon request). For example, in a model with 3 leads and 6 lags, the sum of the six lags and contemporaneous terms are similar in magnitude. The three leads, on the other hand, are not jointly significant.

#### 4.3.6 Count Model

Our baseline health estimates consist of linear probability models, relating the population-scaled hospital admission rates to changes in pollution. To account for the non-negative and discrete nature of the hospital admission data, Table 7 presents estimates from a quasi-maximum likelihood, conditional Poisson IV estimator given in equation (8). In contrast to the baseline linear probability health models, these models are not weighted. In addition, since we use a control function to address issues pertaining to measurement error and omitted variables, we adjust standard errors for the first stage sampling variation using a block-bootstrap sampling procedure, resampling zip codes.<sup>51</sup> Analogous to the linear probability model, we find that respiratory illnesses and heart problems are sensitive to pollution fluctuations, while the three placebos are not (with the usual caveat applying to sickness counts for people aged 65 and above).

The coefficients no longer give marginal impacts and are difficult to interpret. In order to compare the marginal impacts of pollution exposure and congestion across all of our models, Table 8 presents the predicted increase in sickness counts from (i) a one standard deviation increase in taxi time, and (ii) a one standard deviation increase in pollution levels in each zip code. The results are then added for all zip codes that are within 10km of an airport. The table also summarizes population surrounding airports. Various admission categories are given in rows, while the columns

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<sup>51</sup>This is equivalent to clustering by zip code instead of two-way clustering by zip code and day. An unweighted regression of the linear probability model (available upon request) that clusters by zip code gives comparable results.



show the results for each of the 12 airports. The last column gives the combined impact among all 12 airports.

Panels A, B, and C give the predicted increase in hospital admissions using estimates from the baseline linear probability model whereby pollution is instrumented using model 3 (pollution instrumented with taxi time + interactions with distance and wind direction). These results are presented for the overall population (Panel A), children below 5 years (Panel B), and senior citizens 65 and above (Panel C). Panel D gives the results for the overall population using the count model shown in Table 7. Impacts are evaluated at the sample mean for the nonlinear Poisson model. The results from the Poisson model are similar to those from the linear probability model in Panel A. Panel E gives the average daily sickness count in 2005-2007 for the overall population for comparison.

Pollution fluctuations have a large effect on the 6 million people living within 10km of one of the 12 airports: A one standard deviation increase in a zip-codes specific pollution fluctuations increases asthma counts for the overall population by 17% under the linear probability model and 19% under the Poisson count model.<sup>52</sup> Overall, a one standard deviation increase in zip-code specific *daily* pollution levels results in 107 additional admissions for respiratory problems and 49 additional admissions for heart problems, which are 17% and 9% of the daily mean. For respiratory problems, infants only account for roughly one third of the overall impacts. Studies focusing only on the impact on infants therefore would miss a significant portion of the overall impacts. Not surprisingly, the elderly are responsible for the largest share of heart related impacts.

Airport congestion significantly contributes to the overall impacts: a one standard deviation increase in taxi time increases respiratory and heart admissions by roughly 1% of the daily mean. At LAX, the largest airport in California, a one standard deviation increase in taxi time is responsible for roughly one-fourth of the effect of a one-standard deviation increase in pollution. On the other hand, smaller airports (e.g., Santa Barbara or Long Beach) are responsible for a much lower share of the overall pollution impacts.

#### 4.3.7 Economic Cost

In order to monetize the health impacts associated with both pollution exposure, we use the diagnosis-specific reimbursement rates offered to hospitals through Medicare.<sup>53</sup> We view this measure as a lower bound on the total health costs for several reasons: first, our methodology measures limited impacts on both a temporal and spatial scale. By focusing on day-to-day fluctuations, we

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<sup>52</sup>Recall that these estimates are smaller than what we reported under Table 4, where we increased pollution levels in each zip code by the average *overall* standard deviation in pollution levels and took an average baseline sickness rate that was not population weighted.

<sup>53</sup>This information comes from a translation between our hospital diagnosis codes (ICD-9) and Diagnosis Related Group (DRG) codes. We used the crosswalk from the AMA Code Manager Online Elite. Using the set of DRG codes, we calculate the Medicare reimbursement rates using the DRG Payment calculator provided by TRICARE (<http://www.tricare.mil/drgrates/>). In accordance with Medicare reimbursement policy, we adjust the DRG payments using the average wage index in our sample. The average cost for respiratory problems and heart related admissions are US\$ 2702 and 6501, respectively.

do not address the long run, cumulative effect of pollution on health. If these are sizable relative to the contemporaneous effects, the overall cost estimate will be higher. Similarly, our focus has been on individuals living within 10km of an airport. Some of our estimates suggest the marginal impact of taxi time extends beyond the 10km radius, in which case we would be understating the overall effect. Second, we only count people that are sick enough to go to the hospital - anybody who sees their primary care physician or stays home feeling sick will not be counted. Recent work by Hanna & Oliva (2015) finds that pollution decreases labor supply in Mexico City, imposing real economic costs on society not measured in our analysis. Similarly, Deschênes, Greenstone & Shapiro (2012) find that increased levels of ambient NO<sub>2</sub> lead to increased levels of spending on respiratory related prescription medicines, an outcome not measured in our analysis. Third, and most importantly, the marginal willingness to pay to avoid treatment is likely higher than the cost of treatment. For example, severe heart related problems that are not treated within a narrow time frame will likely result in death. The statistical value of life that EPA uses for its benefit-cost analyses is around 6 million dollars, which is 1000 times as large as our medical reimbursement cost for heart-related problems. Individuals might be willing to pay significantly more than medical reimbursement rates to avoid illnesses that, if not adequately treated, have dire consequences. Using the predicted increase in hospital visits under the linear probability model given in Table 8, a one standard deviation increase in pollution levels amounts to about a \$540,000 increase in hospitalization payments related to respiratory and heart related hospital admissions under model 3.<sup>54</sup> Since a one-standard deviation change in pollution is an extrapolation from the fluctuations caused by airport congestion, we also analysis counterfactual where peak exposure levels are capped using the nonlinear models of Figure A5 but find comparable results that are available upon request.<sup>55</sup>

## 5 Conclusions

This study has shown how daily variation in ground level airport congestion due to network delays significantly affects both local pollution levels as well as local measures of health. In doing so, we develop a framework through which to credibly estimate the effects of exogenous shocks to local air pollution on contemporaneous measures of health. Daily local pollution shocks are caused by events that occur several thousand miles away and are arguably exogenous to the local area. We address several longstanding issues pertaining to non-random selection and behavioral responses to pollution. In addition, we show how newly available data on the universe of emergency room provides much cleaner insight as to the sensitivity of populations to ambient pollution levels, relative

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<sup>54</sup>The corresponding number under model 1 is \$920 thousand. These figures are calculated by taking the estimated increase in hospital visits and multiplying it by the average Medicare reimbursement for each of the respective diagnoses.

<sup>55</sup>Specifically, we test the sensitive of our results to assumed linear extrapolation through a counterfactual where all CO levels in 2005-2007 are caped at half the observed mean, i.e., values that exceed half the historic mean are reduced to equal half the historic mean. The implied pollution reduction is evaluated both using our linear baseline model as well as a quadratic or cubic in pollution exposure. The predicted economic benefits are 520 thousand in the linear model and 650 thousand in the two nonlinear models, suggesting that allowing for increasing marginal damages of pollution might give slightly larger damages.

to existing Inpatient Discharge records. Our results suggest that ground operations at airports are responsible for a tremendous amount of local ambient air pollution. Specifically, a one standard deviation change in daily congestion at LAX is responsible for a 0.28 standard deviation increase in levels of CO next to the airport that fades out with distance. The average impact for zip codes within 10km is 0.23 standard deviations.

When connecting these models to measures of health, we find that admissions for respiratory problems and heart disease are strongly related to these pollution changes. A one standard deviation increase in daily zip-code specific pollution levels increases asthma counts by 17% of the baseline average, total respiratory problems by 17%, and heart problems by 9%. Infants and the elderly show a higher sensitivity to pollution fluctuations, and marginal damages of pollution seem to be increasing in pollution for infants. At the same time, adults age 20-64 are also impacted. For respiratory problems, the general adult population accounts for the majority of the total impacts despite the lower sensitivity to fluctuations as they are the largest share of the population. A one standard deviation increase in pollution levels is responsible for 540 thousand dollars in hospitalization costs for the 6 million people living within 10km of one of the 12 airports of our study. This is likely a significant lower bound as the willingness to pay to avoid such illnesses will be higher than the Medicare reimbursement rates.

Examining various mechanisms for the observed pollution-health relationship, we find that CO is primarily responsible for the observed health effects as opposed to NO<sub>2</sub> or O<sub>3</sub>. We find no evidence of forward displacement or delayed impacts of pollution. We also find no evidence that people in areas with larger pollution shocks are less susceptible or less responsive to pollution.

These estimates suggest that relatively small amounts of ambient air pollution can have substantial effects on the incidence of local respiratory illness, at least for the population that comprises our study. While EPA recently decided against lowering the existing carbon monoxide standards due to lack of sufficient evidence of the harmful effects of CO at levels below current EPA mandates, we find significant impacts on morbidity. Recent research suggests that the rates of respiratory illness in the United States are rising dramatically, even as ambient levels of air pollution have continued to fall (Center for Disease Control 2011). Why asthma rates continue to rise is an open question, but the increase in asthma rates is most pronounced amongst African Americans who disproportionately live in densely populated, congested areas. At the same time, traffic congestion in cities has been rising dramatically. Results presented here suggest that at least part of the increased rate of asthma in urban areas can be explained by increased levels of traffic congestion. The exact mechanism remain beyond the scope of the current study, but this remains an interesting area for further research.<sup>56</sup>

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<sup>56</sup>Currently, the highest rates of asthma incidence in the United States are found in Bronx, New York (Garg et al. 2003). This area of northern New York City is bisected by 5 major highways, that rank among the most congested in the United States (Bruner 2009).

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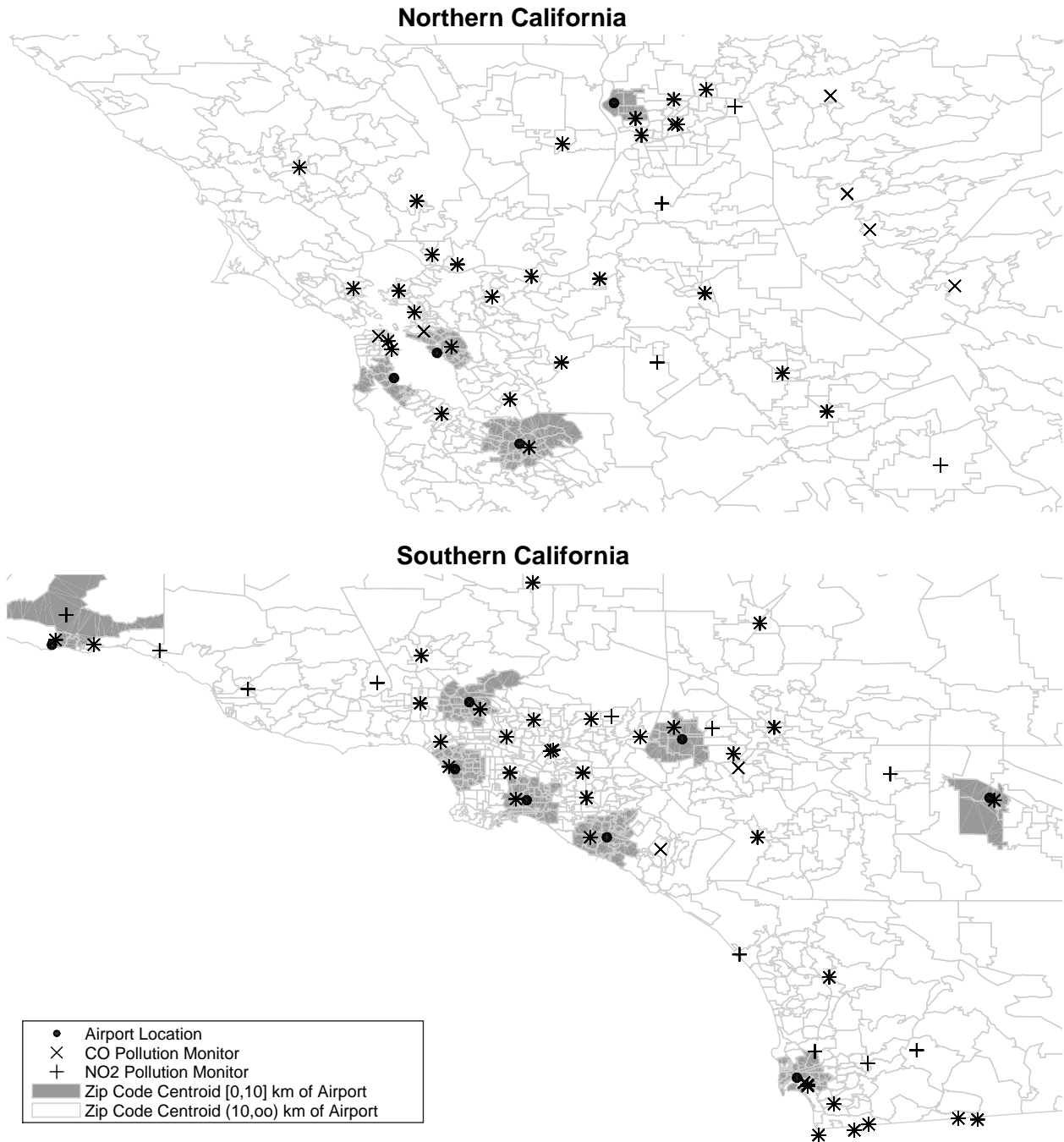
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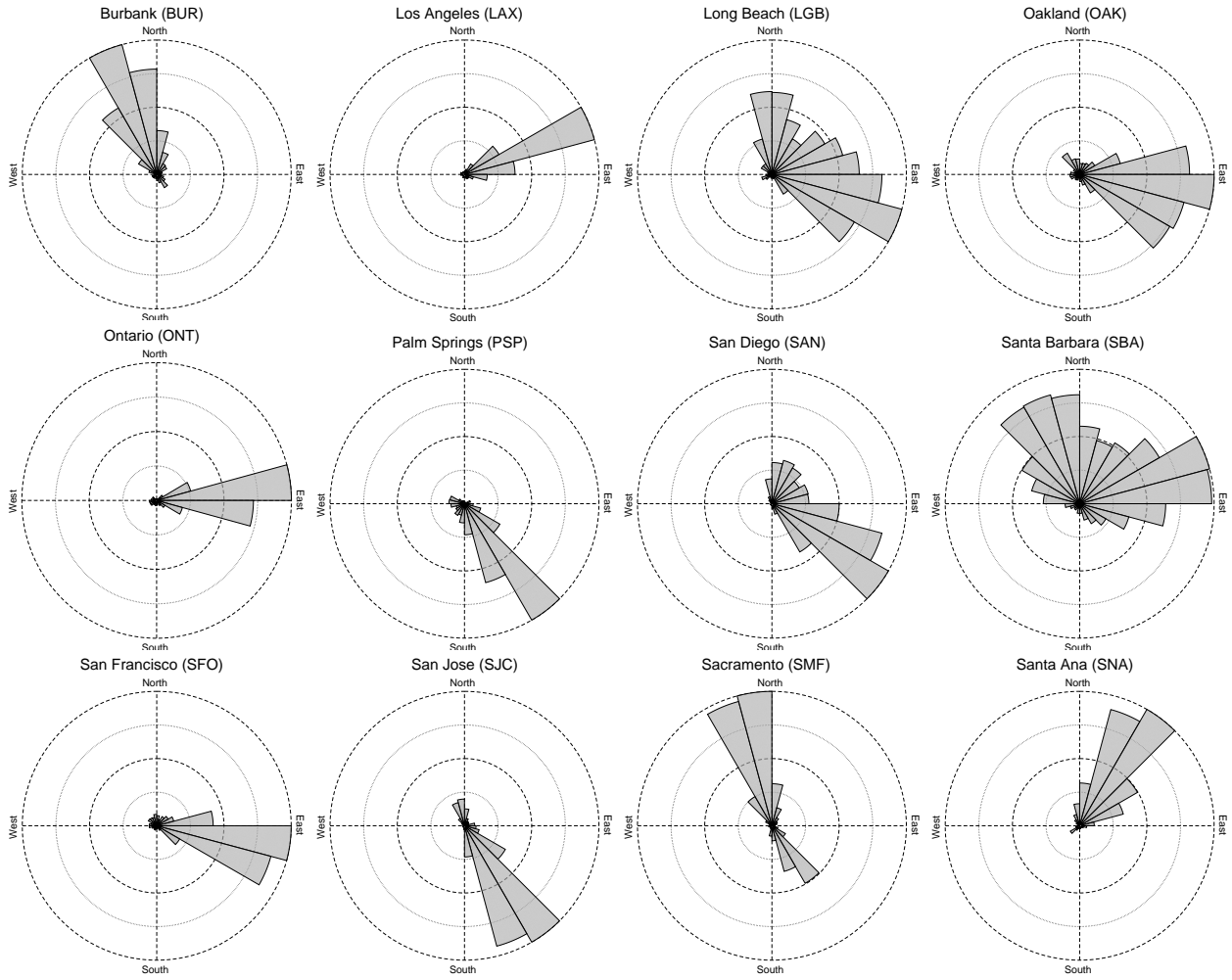
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Figure 1: Location of Airports, Pollution Monitors, and Zip Codes



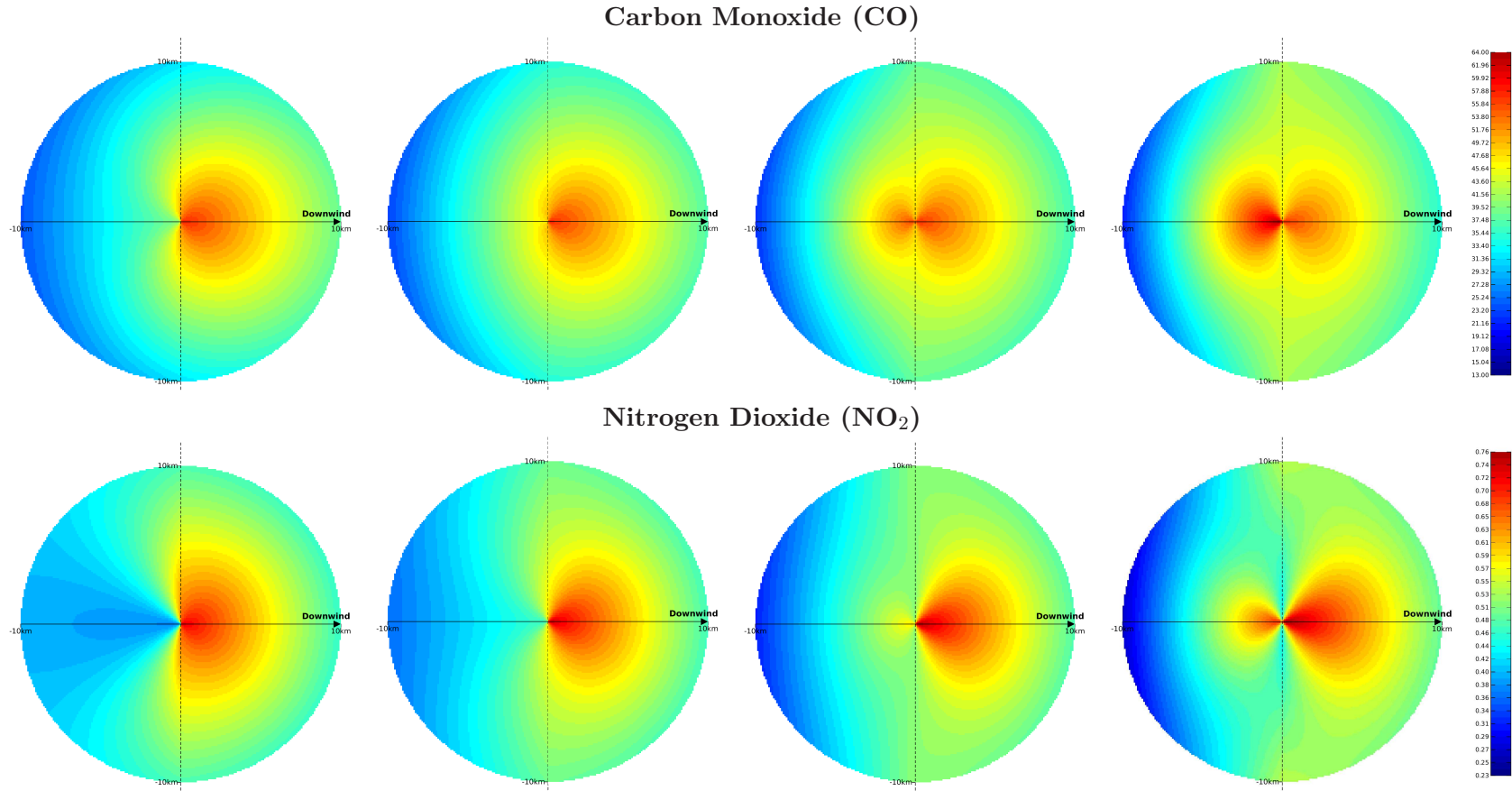
*Notes:* The 12 largest airports in California are shown as dots. The location of CO pollution monitors in the California Air Resource Board (CARB) data base are shown as x, the location of NO<sub>2</sub> monitors as +. Zip code boundaries are shown in grey. They are shaded if the centroid is within 10km (6.2miles) of an airport.

Figure 2: Histogram of Daily Wind Direction At Airports



*Notes:* Histogram of the distribution of daily directions in which the wind is blowing (2005-2007). Plot is normalized to the most frequent category. The four circles indicate the quartile range. Airport locations are shown in Figure 1.

Figure 3: Contour Maps: Marginal Impact of Taxi Time on Pollution Levels



*Notes:* Graphs display the marginal impact of taxi time (ppb per 1000 minute of taxi time, i.e., kmin) on pollution levels across space for different wind speeds. The x-axis shows the direction in which the wind is blowing: positive x-values imply the location is downwind, negative value imply they are upwind. Points on the y-axis are at a right angle to the wind direction. The wind speeds in columns 1-4 are 0.1m/s, 1m/s, 2m/s, and 3m/s corresponding to the 0.1, 10.6, 34.5, and 66.5 percentiles of the distribution of wind speeds in 2005-2007 at the 12 airports in our study (see Figure 1).



Table 1: Pollution Regressed On Instrumented Taxi Time

Variable	CO Pollution			NO <sub>2</sub> Pollution			O <sub>3</sub> Pollution		
	(1a)	(1b)	(1c)	(2a)	(2b)	(2c)	(3a)	(3b)	(3c)
Taxi Time	44.78*** (5.04)	56.26*** (9.48)	52.56*** (10.49)	0.57*** (0.09)	0.67*** (0.15)	0.67*** (0.22)	-0.00 (0.09)	0.08 (0.11)	0.16 (0.20)
Taxi x Distance		-1.62 (1.22)	-2.13 (1.37)		-0.01 (0.02)	-0.02 (0.03)		-0.01 (0.01)	-0.03 (0.02)
Taxi x Angle <sub>u</sub>			13.16* (7.78)			0.31 (0.22)			-0.50*** (0.18)
Taxi x Angle <sub>d</sub>			5.48 (6.97)			0.05 (0.18)			0.05 (0.12)
Taxi x Speed			-2.05 (1.89)			-0.08* (0.04)			0.04 (0.05)
Taxi x Distance x Angle <sub>u</sub>			-0.60 (1.10)			-0.02 (0.03)			0.05** (0.02)
Taxi x Distance x Angle <sub>d</sub>			0.16 (0.89)			-0.01 (0.03)			-0.01 (0.02)
Taxi x Distance x Speed			0.55** (0.25)			0.01* (0.01)			-0.00 (0.01)
Taxi x Angle <sub>d</sub> x Speed			1.70 (2.66)			0.10* (0.05)			-0.07 (0.06)
Taxi x Angle <sub>u</sub> x Speed			-10.41*** (3.74)			-0.19** (0.10)			0.26*** (0.09)
Taxi x Dist. x Angle <sub>u</sub> x Speed			1.50*** (0.50)			0.03** (0.01)			-0.03** (0.01)
Taxi x Dist. x Angle <sub>d</sub> x Speed			-0.63* (0.35)			-0.01 (0.01)			0.01 (0.01)
Observations	179580	179580	179580	179580	179580	179580	179580	179580	179580
Zip Codes	164	164	164	164	164	164	164	164	164
Days	1095	1095	1095	1095	1095	1095	1095	1095	1095
F-stat(joint sig.)	78.48	42.24	14.11	39.67	19.80	4.88	0.00	0.76	1.26
p-value (joint sig.)	1.33e-15	1.66e-15	7.89e-20	2.68e-09	2.00e-08	7.48e-07	.9773	.4705	.2452

*Notes:* Table regresses zip-code level pollution measures on airport congestion (total taxi time in 1000min) in 2005-2007. Taxi time at the local airport is instrumented with the taxi time at three major airports in the Eastern United States. All regressions include weather controls (quadratic in minimum and maximum temperature, precipitation and wind speed as well as controls for wind direction), temporal controls (year, month, weekday, and holiday fixed effects), and zip code fixed effects. Regressions are weighted by the total population in a zip code. Errors are two-way clustered by zip code and day. Significance levels are indicated by \*\*\* 1%, \*\* 5%, \* 10%.

Table 2: Sickness Rates Regressed On Instrumented Taxi Time

	<b>Asthma (1a)</b>	<b>Acute Respiratory (1b)</b>	<b>All Respiratory (1c)</b>	<b>All Heart (2)</b>	<b>Stroke (3)</b>	<b>Bone Fractures (4)</b>	<b>Appen- dicitis (5)</b>
<b>Panel A: All Ages</b>							
Taxi Time	13.84*** (2.72)	24.77*** (7.73)	33.89*** (9.82)	19.35*** (5.24)	2.55 (1.71)	-1.33 (2.87)	0.26 (0.68)
<b>Panel B: Ages Below 5</b>							
Taxi Time	24.46** (11.21)	84.28 (51.35)	116.12* (62.46)	6.63* (3.47)	0.80 (0.94)	2.16 (5.84)	-0.29 (1.38)
<b>Panel C: Age 65 and Above</b>							
Taxi Time	36.89*** (11.39)	63.80*** (16.43)	100.53*** (25.25)	156.54*** (36.98)	22.87* (12.95)	19.13* (9.95)	0.75 (1.21)
Observations	179580	179580	179580	179580	179580	179580	179580
Zip Codes	164	164	164	164	164	164	164
Days	1095	1095	1095	1095	1095	1095	1095

*Notes:* Table regresses zip-code level sickness rates (counts for primary and secondary diagnosis codes per 10 million people) on daily congestion (taxi time in 1000min) that is caused by network delays (taxi time at three major airports in the Eastern United States). All regressions include weather controls (quadratic in minimum and maximum temperature, precipitation and wind speed as well as controls for wind direction), temporal controls (year, month, weekday, and holiday fixed effects), and zip code fixed effects. Regressions are weighted by the total population in a zip code. Errors are two-way clustered by zip code and day. Significance levels are indicated by \*\*\* 1%, \*\* 5%, \* 10%.

Table 3: Sickness Rates Regressed On Pollution

	Asthma (1a)	Acute Respiratory (1b)	All Respiratory (1c)	All Heart (2)	Stroke (3)	Bone Fractures (4)	Appen- dicitis (5)
<b>Panel A: CO Pollution - All Ages</b>							
No Controls	0.070*** (0.017)	0.265*** (0.041)	0.353*** (0.053)	0.035 (0.028)	-0.002 (0.006)	-0.022*** (0.007)	-0.001 (0.001)
Time Controls	0.030 (0.024)	0.058 (0.057)	0.070 (0.075)	-0.022 (0.040)	-0.014* (0.008)	-0.008 (0.010)	0.001 (0.001)
Time + Weather	0.056** (0.028)	0.047 (0.068)	0.071 (0.091)	0.002 (0.052)	-0.005 (0.010)	-0.012 (0.012)	-0.001 (0.001)
Time + Weather + Zip Code FE	0.011 (0.007)	0.049*** (0.018)	0.077*** (0.022)	0.027*** (0.008)	-0.001 (0.003)	-0.007* (0.004)	0.002 (0.001)
<b>Panel B: NO<sub>2</sub> Pollution - All Ages</b>							
No Controls	3.1*** (0.5)	10.7*** (1.3)	14.6*** (1.7)	4.3*** (1.1)	0.6*** (0.2)	-0.3 (0.2)	0.1** (0.0)
Time Controls	1.7** (0.7)	6.0*** (1.5)	7.9*** (2.1)	1.0 (1.4)	-0.1 (0.3)	0.6* (0.3)	0.1** (0.0)
Time + Weather	4.2*** (1.0)	8.3*** (2.6)	11.5*** (3.6)	3.0 (2.4)	0.8* (0.5)	0.7 (0.5)	-0.0 (0.1)
Time + Weather + Zip Code FE	0.1 (0.2)	1.2* (0.6)	2.5*** (0.8)	0.9*** (0.3)	0.1 (0.1)	-0.0 (0.2)	0.1* (0.0)
Observations	179580	179580	179580	179580	179580	179580	179580
Zip Codes	164	164	164	164	164	164	164
Days	1095	1095	1095	1095	1095	1095	1095

*Notes:* Table regresses zip-code level sickness rates (based on primary and secondary diagnosis codes) on daily pollution (ppb) in 2005-2007. Each entry is a separate regression. Columns use sickness rates (counts per 10 million people) for different diseases, while rows use different controls. The first specification (row) in each panel has no controls, while the second adds time controls (year, month, weekday as well as holiday fixed effects), the third adds weather controls (quadratic in minimum and maximum temperature, precipitation and wind speed as well as controls for wind direction), and the fourth adds zip code fixed effects. All regressions are weighted by the total population in a zip code. Errors are two-way clustered by zip code and day. Significance levels are indicated by \*\*\* 1%, \*\* 5%, \* 10%.

Table 4: Sickness Rates Regressed On Instrumented Pollution

	Asthma (1a)	Acute Respiratory (1b)	All Respiratory (1c)	Heart Problems (2)	Stroke (3)	Bone Fractures (4)	Appen- dicitis (5)
<b>Panel A: All Ages</b>							
Model 1: CO	0.311*** (0.065)	0.556*** (0.162)	0.761*** (0.207)	0.434*** (0.134)	0.057 (0.039)	-0.030 (0.063)	0.006 (0.015)
Model 2: CO	0.307*** (0.062)	0.550*** (0.163)	0.755*** (0.210)	0.419*** (0.128)	0.050 (0.038)	-0.030 (0.064)	0.003 (0.015)
Model 3: CO	0.194*** (0.047)	0.396*** (0.125)	0.515*** (0.165)	0.226*** (0.079)	0.020 (0.030)	-0.039 (0.040)	0.002 (0.011)
Model 1: NO <sub>2</sub>	24.5*** (6.2)	43.8*** (16.2)	59.9*** (20.5)	34.2*** (10.5)	4.5 (3.1)	-2.4 (5.2)	0.5 (1.2)
Model 2: NO <sub>2</sub>	24.3*** (6.1)	43.6*** (16.3)	59.8*** (20.8)	33.5*** (10.4)	4.2 (3.1)	-2.4 (5.2)	0.3 (1.2)
Model 3: NO <sub>2</sub>	12.4*** (4.0)	18.9* (11.0)	24.2* (14.2)	17.1** (7.1)	0.7 (2.2)	-1.0 (3.0)	0.3 (0.9)
<b>Panel B: Ages Below 5</b>							
Model 1: CO	0.565** (0.240)	1.948* (1.124)	2.683** (1.353)	0.153* (0.081)	0.018 (0.021)	0.050 (0.136)	-0.007 (0.032)
Model 2: CO	0.579** (0.235)	1.930* (1.111)	2.624* (1.356)	0.127 (0.078)	0.020 (0.023)	0.064 (0.132)	-0.013 (0.034)
Model 3: CO	0.669*** (0.170)	2.166*** (0.796)	2.493** (0.980)	0.075 (0.057)	0.023 (0.015)	-0.012 (0.122)	-0.009 (0.022)
Model 1: NO <sub>2</sub>	42.2** (20.5)	145.3 (95.2)	200.2* (117.3)	11.4* (6.4)	1.4 (1.6)	3.7 (10.0)	-0.5 (2.4)
Model 2: NO <sub>2</sub>	43.2** (20.2)	144.1 (94.3)	195.9* (117.6)	9.5 (6.2)	1.5 (1.7)	4.8 (9.7)	-0.9 (2.5)
Model 3: NO <sub>2</sub>	43.6*** (14.9)	122.2* (67.7)	140.8* (82.4)	4.5 (4.6)	2.9** (1.3)	3.7 (9.3)	0.6 (2.0)
<b>Panel C: Ages 65 and Older</b>							
Model 1: CO	0.849*** (0.312)	1.469*** (0.440)	2.314*** (0.642)	3.604*** (1.001)	0.526* (0.297)	0.440* (0.242)	0.017 (0.028)
Model 2: CO	0.815*** (0.288)	1.413*** (0.422)	2.275*** (0.637)	3.529*** (0.971)	0.502* (0.302)	0.409* (0.241)	0.016 (0.028)
Model 3: CO	0.493** (0.204)	0.696** (0.309)	1.424*** (0.511)	1.937*** (0.620)	0.198 (0.249)	0.187 (0.161)	-0.025 (0.026)
Model 1: NO <sub>2</sub>	66.5*** (22.8)	114.9*** (34.3)	181.1*** (52.3)	282.0*** (76.1)	41.2* (24.2)	34.5* (18.2)	1.4 (2.2)
Model 2: NO <sub>2</sub>	66.5*** (22.8)	115.1*** (34.3)	181.4*** (52.3)	282.2*** (76.2)	41.2* (24.3)	34.5* (18.2)	1.4 (2.2)
Model 3: NO <sub>2</sub>	35.3** (14.2)	38.9 (24.6)	75.8* (41.3)	131.6*** (47.8)	3.6 (16.5)	12.2 (12.0)	-0.8 (1.6)
Observations	179580	179580	179580	179580	179580	179580	179580
Zip Codes	164	164	164	164	164	164	164
Days	1095	1095	1095	1095	1095	1095	1095

*Notes:* Table regresses zip-code level sickness rates (counts for primary and secondary diagnosis codes per 10 million people) on daily instrumented pollution levels (ppb) in 2005-2007. Each entry is a separate regression. Pollution is instrumented on airport congestion (taxi time) that is caused by network delays (taxi time at three major airports in the Eastern United States). Model 1 assumes a uniform impact of congestion on pollution levels at all zip codes surrounding an airport, while model 2 adds an interaction with the distance to the airport, and model 3 furthermore adds interactions with wind direction and speed (columns (a)-(c) in Table 1). All regressions include weather controls (quadratic in minimum and maximum temperature, precipitation and wind speed as well as controls for wind direction), temporal controls (year, month, weekday, and holiday fixed effects), and zip code fixed effects. Regressions are weighted by the total population in a zip code. Errors are two-way clustered by zip code and day. Significance levels are indicated by \*\*\* 1%, \*\* 5%, \* 10%

Table 5: Sickness Rates Regressed On Instrumented Pollution - Joint Estimation

	Asthma (1a)	Acute Respiratory (1b)	All Respiratory (1c)	Heart Problems (2)	Stroke (3)	Bone Fractures (4)	Appen- dicitis (5)
<b>Panel A: All Ages</b>							
Model 3: CO	0.222** (0.106)	0.867*** (0.313)	1.189** (0.476)	0.105 (0.139)	0.054 (0.049)	-0.127* (0.073)	-0.006 (0.017)
Model 3: NO <sub>2</sub>	-2.2 (8.0)	-40.1 (25.6)	-57.4 (38.4)	10.7 (13.4)	-2.9 (3.5)	7.6 (5.6)	0.7 (1.5)
F <sub>(1st stage)</sub> - CO	4.18	4.18	4.18	4.18	4.18	4.18	4.18
F <sub>(1st stage)</sub> - NO <sub>2</sub>	1.57	1.57	1.57	1.57	1.57	1.57	1.57
P <sub>(Anderson-Rubin)</sub>	0.0000	0.0000	0.0000	0.0012	0.0027	0.1418	0.0484
P <sub>(Stock-Wright S)</sub>	0.0413	0.0795	0.1141	0.1232	0.5319	0.4795	0.5710
<b>Panel B: Ages Below 5</b>							
Model 3: CO	0.901 (0.599)	4.685** (2.167)	5.388** (2.483)	0.133 (0.142)	-0.073 (0.051)	-0.385 (0.355)	-0.102 (0.072)
Model 3: NO <sub>2</sub>	-18.8 (46.1)	-206.8 (174.9)	-237.6 (199.0)	-4.8 (11.5)	8.0* (4.1)	30.9 (27.3)	7.7 (6.4)
F <sub>(1st stage)</sub> - CO	3.39	3.39	3.39	3.39	3.39	3.39	3.39
F <sub>(1st stage)</sub> - NO <sub>2</sub>	1.30	1.30	1.30	1.30	1.30	1.30	1.30
P <sub>(Anderson-Rubin)</sub>	0.0000	0.0001	0.0000	0.0185	0.0474	0.0687	0.6100
P <sub>(Stock-Wright S)</sub>	0.0588	0.0886	0.2163	0.4046	0.3567	0.3185	0.5764
<b>Panel C: Age 65 and Above</b>							
Model 3: CO	0.268 (0.323)	0.775* (0.445)	1.726** (0.772)	1.279 (0.821)	0.490 (0.399)	0.147 (0.281)	-0.051 (0.046)
Model 3: NO <sub>2</sub>	20.1 (22.1)	-6.4 (36.3)	-25.8 (62.6)	60.3 (67.7)	-25.5 (25.9)	3.7 (21.4)	2.2 (3.1)
F <sub>(1st stage)</sub> - CO	4.96	4.96	4.96	4.96	4.96	4.96	4.96
F <sub>(1st stage)</sub> - NO <sub>2</sub>	2.09	2.09	2.09	2.09	2.09	2.09	2.09
P <sub>(Anderson-Rubin)</sub>	0.1146	0.0026	0.0035	0.0009	0.0164	0.1595	0.0017
P <sub>(Stock-Wright S)</sub>	0.4530	0.1484	0.2756	0.1831	0.3849	0.2969	0.1264
Observations	179580	179580	179580	179580	179580	179580	179580
Zip Codes	164	164	164	164	164	164	164
Days	1095	1095	1095	1095	1095	1095	1095

*Notes:* Table regresses zip-code level sickness rates (counts for primary and secondary diagnosis codes per 10 million people) on daily instrumented pollution levels (ppb) in 2005-2007. The effect of the two pollutants is jointly estimated for the over-identified model 3 using LIML. Pollution is instrumented on airport congestion (taxi time) that is caused by network delays (taxi time at three major airports in the Eastern United States). All regressions include weather controls (quadratic in minimum and maximum temperature, precipitation and wind speed as well as controls for wind direction), temporal controls (year, month, weekday, and holiday fixed effects), and zip code fixed effects. Regressions are weighted by the total population in a zip code. Errors are two-way clustered by zip code and day. Significance levels are indicated by \*\*\* 1%, \*\* 5%, \* 10%.



Table 6: Sickness Rates of All Ages Regressed On Instrumented CO Pollution - Lagged Pollution

	<b>Asthma</b>	<b>Acute Respiratory</b>	<b>All Respiratory</b>	<b>Heart Problems</b>
<b>Model 1</b>				
Pollution in t-3	0.026 (0.096)	0.220 (0.188)	0.345 (0.250)	0.022 (0.139)
Pollution in t-2	0.130 (0.143)	0.109 (0.246)	0.023 (0.336)	0.003 (0.255)
Pollution in t-1	-0.017 (0.132)	-0.060 (0.251)	-0.001 (0.289)	-0.020 (0.183)
Pollution in t	0.200** (0.101)	0.355 (0.263)	0.485 (0.331)	0.422*** (0.134)
Cum. Effect	0.339*** (0.070)	0.624*** (0.163)	0.853*** (0.210)	0.427*** (0.151)
<b>Model 2</b>				
Pollution in t-3	0.040 (0.094)	0.229 (0.188)	0.353 (0.250)	0.022 (0.138)
Pollution in t-2	0.117 (0.141)	0.098 (0.245)	0.013 (0.331)	-0.002 (0.250)
Pollution in t-1	-0.021 (0.133)	-0.062 (0.253)	-0.004 (0.291)	-0.028 (0.184)
Pollution in t	0.203** (0.099)	0.352 (0.262)	0.485 (0.331)	0.415*** (0.132)
Cum. Effect	0.338*** (0.066)	0.618*** (0.163)	0.847*** (0.214)	0.408*** (0.143)
<b>Model 3</b>				
Pollution in t-3	-0.002 (0.041)	0.126 (0.095)	0.121 (0.124)	0.045 (0.057)
Pollution in t-2	0.079 (0.060)	0.023 (0.116)	0.020 (0.151)	-0.014 (0.087)
Pollution in t-1	-0.059 (0.056)	0.008 (0.154)	0.020 (0.191)	-0.004 (0.111)
Pollution in t	0.177*** (0.067)	0.316 (0.201)	0.420 (0.263)	0.225** (0.100)
Cum. Effect	0.195*** (0.052)	0.473*** (0.115)	0.582*** (0.153)	0.252*** (0.067)
Observations	179088	179088	179088	179088
Zip Codes	164	164	164	164
Days	1092	1092	1092	1092

*Notes:* Table replicates the results of CO pollution on sickness counts for all ages in Table 4 except that three lags of the instrumented pollution levels are included. Each column in each panel presents the coefficients from one regression as well as the cumulative effect (sum of all four coefficients). Significance levels are indicated by \*\*\* 1%, \*\* 5%, \* 10%.

Table 7: Sickness Counts Regressed On Instrumented CO Pollution - Poisson Model

	Asthma (1a)	Acute Respiratory (1b)	All Respiratory (1c)	Heart Problems (2)	Stroke (3)	Bone Fractures (4)	Appen- dicitis (5)
<b>Panel A: All Ages</b>							
Model 1: CO	0.834*** (0.171)	0.596*** (0.109)	0.577*** (0.112)	0.488*** (0.128)	0.270 (0.184)	-0.114 (0.184)	0.325 (0.454)
Model 2: CO	0.846*** (0.172)	0.589*** (0.111)	0.573*** (0.116)	0.482*** (0.128)	0.246 (0.185)	-0.116 (0.188)	0.245 (0.466)
Model 3: CO	0.561*** (0.132)	0.399*** (0.090)	0.378*** (0.087)	0.292*** (0.095)	0.132 (0.195)	-0.150 (0.133)	0.163 (0.325)
<b>Panel B: Ages Below 5</b>							
Model 1: CO	1.202*** (0.387)	0.237 (0.179)	0.303 (0.208)	2.061* (1.148)	3.334 (2.876)	0.187 (0.572)	-0.369 (2.923)
Model 2: CO	1.202*** (0.396)	0.216 (0.179)	0.278 (0.207)	1.891* (1.105)	3.347 (2.799)	0.233 (0.567)	-0.691 (2.963)
Model 3: CO	1.133*** (0.287)	0.261** (0.132)	0.256* (0.143)	1.297 (0.966)	4.238* (2.480)	-0.064 (0.495)	-1.290 (2.643)
<b>Panel C: Ages 65 and Older</b>							
Model 1: CO	1.287*** (0.364)	0.757*** (0.208)	0.610*** (0.173)	0.634*** (0.165)	0.397* (0.219)	0.626** (0.314)	1.190 (1.247)
Model 2: CO	1.264*** (0.341)	0.743*** (0.202)	0.608*** (0.174)	0.630*** (0.166)	0.388* (0.224)	0.589* (0.313)	1.135 (1.291)
Model 3: CO	0.804*** (0.275)	0.413** (0.180)	0.397** (0.154)	0.369*** (0.126)	0.159 (0.223)	0.292 (0.219)	-0.852 (1.185)
Observations	179580	179580	179580	179580	179580	179580	179580
Zip Codes	164	164	164	164	164	164	164
Days	1095	1095	1095	1095	1095	1095	1095

*Notes:* Table replicates the results for regression models of CO in Table 4 except that we use a Poisson count model instead of a linear probability model. Further differences are that the regressions are unweighted and standard errors are obtained from 100 clustered bootstrap draws (drawing entire zip code histories with replacement), which is comparable to clustering by zip code in the baseline regression. Significance levels are indicated by \*\*\* 1%, \*\* 5%, \* 10%.

Table 8: Impact of CO Pollution on Health (Model 3)

	LAX	SFO	SAN	OAK	SJC	SMF	SNA	ONT	BUR	SBA	LGB	PSP	Total
<b>Panel A: Linear Probability Model - All Ages</b>													
Population	812	182	540	448	910	41	822	454	794	59	875	93	6028
One Standard Deviation Increase in Taxi Time													
Asthma	1.20	0.16	0.29	0.15	0.21	0.01	0.26	0.09	0.12	0.00	0.10	0.01	2.60
Acute Respiratory	2.44	0.34	0.59	0.30	0.44	0.02	0.53	0.19	0.25	0.01	0.20	0.02	5.31
All Respiratory	3.18	0.44	0.77	0.39	0.57	0.03	0.68	0.25	0.32	0.01	0.25	0.03	6.91
Heart Disease	1.40	0.19	0.34	0.17	0.25	0.01	0.30	0.11	0.14	0.00	0.11	0.01	3.04
One Standard Deviation Increase in Pollution													
Asthma	4.80	0.52	4.00	1.37	5.96	0.18	5.13	1.98	5.92	0.18	6.45	0.15	36.63
Acute Respiratory	9.82	1.06	8.17	2.80	12.18	0.36	10.49	4.04	12.10	0.36	13.19	0.30	74.87
All Respiratory	12.78	1.38	10.63	3.64	15.85	0.47	13.65	5.26	15.75	0.47	17.17	0.40	97.44
Heart Disease	5.61	0.61	4.67	1.60	6.96	0.21	5.99	2.31	6.92	0.21	7.54	0.17	42.79
<b>Panel B: Linear Probability Model - Ages 5 and Below</b>													
Population	54	11	33	32	68	4	58	35	55	3	65	6	424
One Standard Deviation Increase in Taxi Time													
Asthma	0.27	0.03	0.06	0.03	0.05	0.00	0.06	0.02	0.03	0.00	0.02	0.00	0.59
Acute Respiratory	0.87	0.11	0.19	0.11	0.17	0.01	0.20	0.08	0.09	0.00	0.08	0.01	1.91
All Respiratory	1.00	0.13	0.22	0.13	0.20	0.01	0.23	0.09	0.10	0.00	0.09	0.01	2.20
Heart Disease	0.03	0.00	0.01	0.00	0.01	0.00	0.01	0.00	0.00	0.00	0.00	0.00	0.07
One Standard Deviation Increase in Pollution													
Asthma	1.14	0.11	0.84	0.33	1.54	0.06	1.24	0.53	1.42	0.03	1.68	0.03	8.96
Acute Respiratory	3.69	0.37	2.73	1.08	4.98	0.19	4.03	1.73	4.59	0.09	5.43	0.10	28.99
All Respiratory	4.25	0.42	3.14	1.24	5.74	0.22	4.63	1.99	5.28	0.11	6.25	0.11	33.37
Heart Disease	0.13	0.01	0.09	0.04	0.17	0.01	0.14	0.06	0.16	0.00	0.19	0.00	1.00
<b>Panel C: Linear Probability Model - Ages 65 and Above</b>													
Population	82	26	54	51	88	3	79	34	79	12	89	18	615
One Standard Deviation Increase in Taxi Time													
Asthma	0.30	0.06	0.07	0.04	0.05	0.00	0.06	0.02	0.03	0.00	0.02	0.01	0.67
Acute Respiratory	0.43	0.08	0.10	0.06	0.07	0.00	0.09	0.03	0.04	0.00	0.03	0.01	0.94
All Respiratory	0.87	0.17	0.21	0.12	0.15	0.00	0.18	0.05	0.09	0.01	0.07	0.02	1.93
Heart Disease	1.19	0.23	0.29	0.16	0.20	0.01	0.24	0.07	0.12	0.01	0.10	0.02	2.63
One Standard Deviation Increase in Pollution													
Asthma	1.23	0.19	1.02	0.39	1.46	0.03	1.28	0.38	1.51	0.09	1.69	0.08	9.33
Acute Respiratory	1.73	0.26	1.44	0.55	2.06	0.04	1.80	0.54	2.13	0.13	2.38	0.11	13.15
All Respiratory	3.54	0.54	2.94	1.14	4.21	0.08	3.68	1.10	4.36	0.26	4.87	0.22	26.93
Heart Disease	4.82	0.74	4.00	1.54	5.73	0.11	5.01	1.49	5.93	0.35	6.62	0.29	36.63
<b>Panel D: Poisson Model - All Ages</b>													
One Standard Deviation Increase in Taxi Time													
Asthma	1.41	0.21	0.35	0.24	0.16	0.01	0.16	0.09	0.11	0.00	0.11	0.01	2.87
Acute Respiratory	2.62	0.40	0.61	0.42	0.34	0.02	0.43	0.20	0.26	0.00	0.23	0.03	5.55
All Respiratory	3.44	0.53	0.81	0.54	0.45	0.02	0.55	0.26	0.34	0.01	0.31	0.04	7.30
Heart Disease	1.59	0.28	0.40	0.23	0.22	0.01	0.26	0.11	0.16	0.00	0.14	0.02	3.42
One Standard Deviation Increase in Pollution													
Asthma	6.35	0.67	5.29	2.29	5.02	0.22	3.60	2.00	6.22	0.09	8.54	0.14	40.41
Acute Respiratory	11.47	1.35	8.93	3.98	10.22	0.33	9.09	4.48	13.99	0.19	16.81	0.39	81.24
All Respiratory	15.00	1.76	12.01	5.07	13.26	0.41	11.79	5.91	18.16	0.28	22.70	0.57	106.93
Heart Disease	6.72	0.87	5.85	2.13	6.30	0.16	5.62	2.48	8.62	0.23	10.03	0.30	49.31
<b>Panel E: Baseline Average - All Ages</b>													
Asthma	33.1	7.9	22.3	25.4	24.2	1.6	18.1	14.9	26.0	0.9	36.0	3.0	213.6
Acute Respiratory	87.4	21.7	55.2	63.2	71.8	3.6	66.9	48.0	85.8	3.1	104.3	11.8	623.0
All Respiratory	121.3	30.3	78.2	85.2	98.7	4.7	91.6	67.2	117.9	4.6	149.0	18.0	866.8
Heart Disease	72.8	20.3	50.0	46.4	61.7	2.4	56.9	36.9	73.4	5.0	86.1	12.3	524.2

*Notes:* Table gives population as well as daily hospital admissions for all zip codes that are within 10km (6.2miles) of one of the 12 major California airports. Panels A-D give predicted *changes* in sickness counts, while Panel E gives baseline *averages*. Panels A-C use the linear probability model 1 for CO from Table 4, while panel D uses the Poisson model 1 for CO from Table 7. Panel E gives average daily sickness counts in the data. The first 12 columns give impacts by airport, while the last column gives the total for all 12 airports. Population is in thousand. Predicted changes in hospitalization are for both inpatient as well as outpatient admissions.

REVIEW

Open Access



# A review of health effects associated with exposure to jet engine emissions in and around airports

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## Abstract

**Background:** Airport personnel are at risk of occupational exposure to jet engine emissions, which similarly to diesel exhaust emissions include volatile organic compounds and particulate matter consisting of an inorganic carbon core with associated polycyclic aromatic hydrocarbons, and metals. Diesel exhaust is classified as carcinogenic and the particulate fraction has in itself been linked to several adverse health effects including cancer.

**Method:** In this review, we summarize the available scientific literature covering human health effects of exposure to airport emissions, both in occupational settings and for residents living close to airports. We also report the findings from the limited scientific mechanistic studies of jet engine emissions in animal and cell models.

**Results:** Jet engine emissions contain large amounts of nano-sized particles, which are particularly prone to reach the lower airways upon inhalation. Size of particles and emission levels depend on type of aircraft, engine conditions, and fuel type, as well as on operation modes. Exposure to jet engine emissions is reported to be associated with biomarkers of exposure as well as biomarkers of effect among airport personnel, especially in ground-support functions. Proximity to running jet engines or to the airport as such for residential areas is associated with increased exposure and with increased risk of disease, increased hospital admissions and self-reported lung symptoms.

**Conclusion:** We conclude that though the literature is scarce and with low consistency in methods and measured biomarkers, there is evidence that jet engine emissions have physicochemical properties similar to diesel exhaust particles, and that exposure to jet engine emissions is associated with similar adverse health effects as exposure to diesel exhaust particles and other traffic emissions.

**Keywords:** Jet engine emissions, Airports, Occupational exposure, Particulate matter, Polycyclic aromatic hydrocarbons, Biomarkers

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## Background

Exposure to air pollution, including ultrafine particulate matter (UFP), from industry and traffic is associated with adverse health effects [1–4]. Airports are significant high-emission sources and human exposure to these emissions is a growing health concern. Importantly, airport personnel are at risk of occupational exposure to jet engine emissions [5]. More knowledge is needed on exposure risks, adverse health effects, biomarkers and risk management options related to the diverse factors influencing human exposure to airport emissions [6] (Fig. 1).

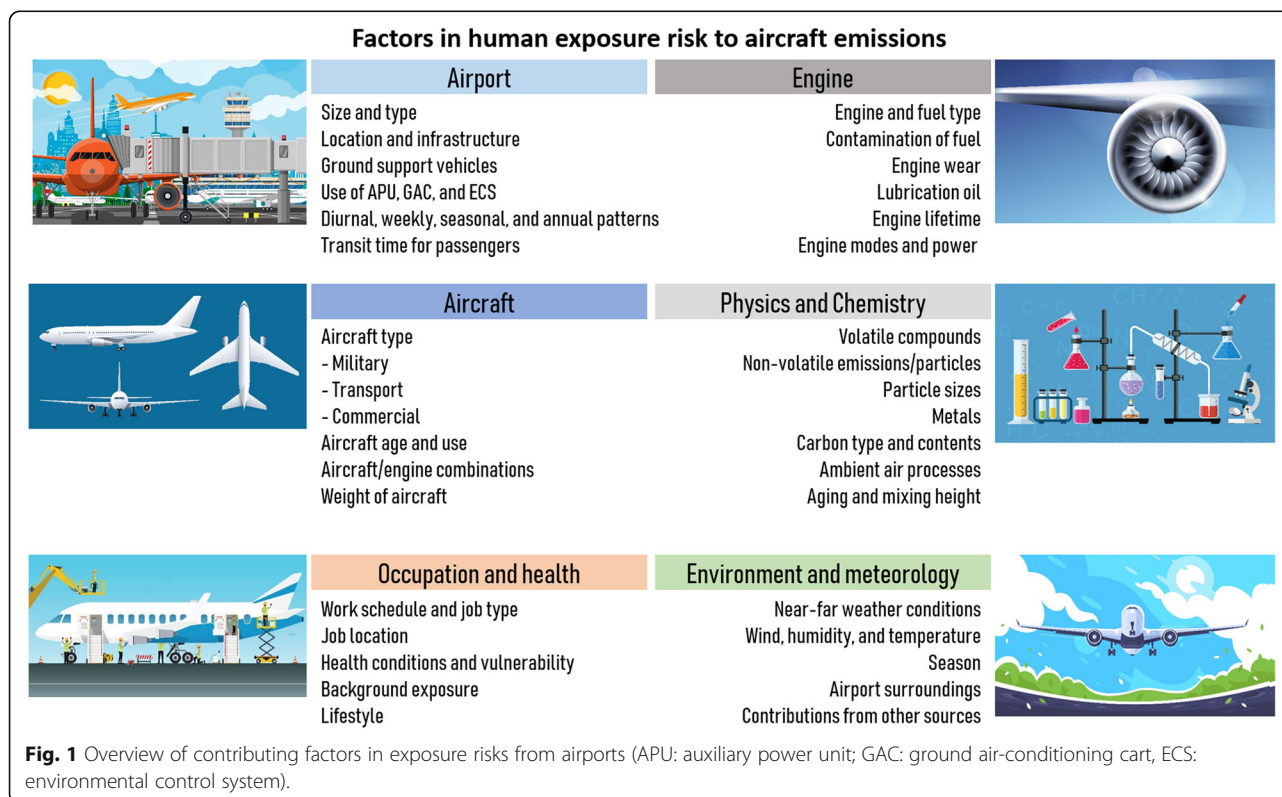
However, data collection seems challenging. Commercial airports are large, complex and diverse work places, where aircraft, ground-support equipment (GSE), and related vehicles all contribute to mixed emissions [7, 8]. In turn, commercial airports as well as military air stations are year-round active high security areas with restricted access, which can reduce the options for external researchers to collect optimal or sufficient measurements. Consensus or formal guidelines for optimal measurement design, instrumentation and analysis methods for the different emission components are lacking, which further complicates comparison of data and risk assessment [5, 9].

With this review, we seek to compile available studies in the open scientific literature on health effects of jet engine emissions in occupational settings and in residential areas around airports, along with mechanistic effects studied in animal and cell models. The studies were selected based on

key papers and systematic searches (search terms, method and selection criteria are disclosed in the Additional file 1). We briefly summarize the characteristics of jet engine emissions and highlight the complexity of this field of research, but detailed research on emissions and physical-chemical studies is beyond the scope of this review.

## Toxicity of jet fuel exposure

The toxicity of (unburned) jet fuel as such has been considered in many studies (reviewed in [10]) since the early 1950's, where the specifications of the hydrocarbon-based jet fuel, JP-4 (jet propellant-4), was published by the US air force. Major toxic effects reported for JP-4 were skin irritation, neurotoxicity, nephrotoxicity, and renal carcinogenicity in rats [11]. Jet fuels are mixtures of gasoline and kerosene with performance additives [10]. In 1994, US Air Force converted to JP-8, developed to be less volatile and less explosive upon crash incidents compared to JP-4. JP-8 (NATO F-34) is equivalent to Jet A-1 fuel used in commercial aircraft. A range of other kerosene-based jet fuels are in use, depending on aircraft type and differing in kerosene ratio and requirements for additives [5]. Measurements of a range of the common aircraft pollutants such as benzene, toluene, and chlorinated compounds in breath samples from exposed personnel on an airbase before and after work tasks showed significant exposure for all subjects, ranging from minor elevations up to > 100 times the values of





the control group for fuel workers [12]. The uptake of JP-8 components both occur via inhalation and dermal contact, and apart from benzene, naphthalene in air and in exhaled breath condensate (EBC) may be useful as a biomarker of exposure to and uptake of JP-8 fuel components in the body [13]. Although most studies report low acute toxicity for both JP-4 and JP-8, JP-8 was reported to show effects such as respiratory tract sensory irritation [11], inflammatory cytokine secretion in exposed alveolar type II epithelial cells and in pulmonary alveolar macrophages [14], increased pulmonary resistance and decreased weight gain in rats upon inhalation exposure for 7 or 28 days [15, 16]. Subchronic 90-days studies with rats with various exposure levels of JP-4 and JP-8 showed little toxicity, apart from male rat hydrocarbon nephropathy [11]. However, JP-8 fuel exposure has been linked to noise-activated ototoxic hearing loss in animal studies [17, 18] and in occupational exposure cases [19, 20], and to immunotoxicity [21, 22].

It is likely that fuel refinements will advance in the future and be an important factor in emission reductions. A newer synthetic jet fuel (Fischer-Tropsch Synthetic Paraffinic Kerosene) under development to replace JP-8 in the future, was evaluated for toxicity in the required range of tests used to develop occupational exposure limits (OELs). The highest exposure level of 2000 mg/m<sup>3</sup> (6 h per day, 5 days a week for 90 days) produced multifocal inflammatory cell infiltrations in rat lungs, whereas no genotoxicity or acute inhalation effects were observed, and the sensory irritation assay indicated that the refined synthetic fuel was less irritating than JP-8 [23]. Evidence of cancer risk is, however, normally evaluated in two-year inhalation studies in rats.

#### Characteristics of jet engine emissions

Like other combustion engines, jet engines produce volatile organic compounds (VOC) such as CO<sub>2</sub>, NO<sub>x</sub>, CO, SO<sub>x</sub> and low molecular weight polycyclic aromatic hydrocarbons (PAH), and particulate matter (PM) with associated PAH, and metals [24]. Incomplete combustion of fossil fuels, including kerosene, results in the formation of carbon-rich (> 60%), aromatic bi-products called char, and condensates, which are known as soot. Char and soot can either be measured as elemental carbon (EC, used in atmospheric sciences) or black carbon (BC, used in soil and sediment sciences) [25]. This terminology originates from their measurement methods (BC is light-absorbing, determined by optical methods and EC is refractory, determined by thermo-optical and oxidizing methods) [26]. BC is often used in physical/chemical aerosol studies of airport- and urban emissions, such as in Costabile et al. [27] and Keuken et al. [28]. However, there is no apparent consistent correlation between BC concentrations and particle number

concentrations across exposure studies at airports, but data is limited as noted by Stacey [9].

In general, emission levels are high, but vary depending on engine conditions and fuel type, as well as on operation modes such as idling, taxi, take-off, climb-out and landing [29].

#### Particulate matter (PM)

PM is divided by size ranges according to the aerodynamic diameter of the particles, where UFP are in the nanoscale of < 100 nm. Several studies have shown that aircraft emissions are dominated or even characterized by high concentrations of very small particles. This was underlined in a recent study by Stacey, Harrison and Pope carried out at Heathrow London in comparison to traffic background [30]. Some report particles in the range of 5–40 nm [31], and others particle diameters of 20 nm as compared to larger particles of > 35 nm measured at surrounding freeways [32]. Campagna et al. studied the contributions of UFP from a military airport to the surrounding area, by sampling on the airport grounds during flight activities, nearby the airport, in an urban area and in a rural area. The smallest primary particles were found within the airport (~ 10 nm) and the largest in the urban area (~ 72 nm). The highest UFP levels inside the airport were measured during taxi and take-off activities ( $4.0 \times 10^6$  particles/cm<sup>3</sup>) [33]. Westerdahl et al. reported very high particle number concentrations at take-off of a single jet aircraft, with a 10 s peak of 4.8 million particles/cm<sup>3</sup> together with elevated NO<sub>x</sub> and BC levels [34].

The small particles are emitted in large numbers and tend to form complex agglomerates in ambient air that can be detected in larger particle size modes [35, 36] (see [5] for elaboration). In a recent study in Montreal-Pierre-Elliott-Trudeau International Airport, the total particle number concentration over all sizes at the airport apron reached  $2.0 \times 10^6$ /cm<sup>3</sup>, which was significantly higher compared to downtown Montreal ( $1 \times 10^4$ /cm<sup>3</sup>). The geometric mean of observed ultrafine particle number density of nanoparticles was  $1 \times 10^5$ /cm<sup>3</sup> at the apron and  $1.1 \times 10^4$ /cm<sup>3</sup> outside the Departure Level entrance [37]. We recently published exposure measurements conducted at a commercial airport and non-commercial airfield, where air concentrations were measured to  $7.7 \times 10^6$  particle/cm<sup>3</sup> or 1086 µg/m<sup>3</sup> of total particles during take-off of one single jet plane [36]. The majority of these particles were below the size detection limit of 10 nm for the instruments [36], which was also shown, and highlighted as a general challenge, by others [38].

The nanostructure of carbon particles are influenced by fuel type and combustion processes. Low thrust settings are associated with the smallest particle sizes. In

one of their studies, Vander Wal et al. characterized the aircraft particles as predominantly organic carbon at low thrust and EC at higher thrust settings [38]. In turn, it was reported that soot reactivity, characterized by an outer amorphous shell, of soot particles from a turbofan test engine was lower in particles from ground idle as compared to particles from climb-out engine mode for two fuel types. Biofuel blending slightly lowered this soot reactivity at ground idle, but had the opposite effect at the higher power condition of climb-out. The authors comment that for soot reactivity, measured by an outer amorphous shell in the study, biofuels may be beneficial in airports where ground idle engine conditions are often in use, but the effect on emissions in climb-out conditions is undetermined [31]. According Moore et al., a 50:50 biofuel blending reduces particle emissions from aircraft with 50–70%, compared to conventional Jet-A fuel [39]. Another study did extensive analyses of emissions from four on-wing commercial aircraft turbo engines (two newer CFM56–7 engines and two CFM56–3 engines), also demonstrating that the type of emissions were significantly dependent on power. PM emission indices ( $\text{g/kg}^{-1}$  fuel) were reported to increase from 0.011 to 0.205  $\text{g/kg}^{-1}$  fuel with a power increase from idle to 85%. In turn, the data showed that hydrocarbons are mostly emitted at ground idle engine conditions, as opposed to PM emissions being more significant at higher power thrusts, such as take-off and landing. EC fraction of PM also increased with increase in power [40]. Targino et al. measured large EC (BC) concentrations during boarding and disembarking (mean 3.78  $\mu\text{g}/\text{m}^3$ ), at the airport concourse (mean 3.16  $\mu\text{g}/\text{m}^3$ ) and also inside an aircraft on the ground with open doors (mean 2.78  $\mu\text{g}/\text{m}^3$ ) [41].

#### Lubrication oil and organophosphate esters

A recent study found that intact forms of unburned jet engine lubrication oil was a major component of emissions from aircraft [42]. Organophosphate esters (OPEs) are a large group of chemicals with toxic properties used as stabilizing agents in numerous consumer – and industrial products, including in aircraft lubricating oil and hydraulic fluids. Airplane emissions are thought to be an important source of OPEs in the environment. Not only does these chemicals accumulate in ecosystems, but it is also a concern due to the location of airports near populated areas [5]. Li et al. recently studied the concentrations of 20 OPEs in ambient air, soil, pine needles, river water, and outdoor dust samples collected around an airport in Albany, New York, and reported elevated total OPE concentrations in all samples. The spatial distribution of OPEs in air, soil, and pine needles correlated with distance to the airport. The average daily intake of OPEs via air inhalation and outdoor dust ingestion in the

vicinity of the airport was up to 1.53 ng/kg bw/day for children and 0.73 ng/kg bw/day for adults [43]. Another study examined organophosphates, such as tri-n-butyl phosphate, dibutyl phenyl phosphate, triphenyl phosphate and tricresyl phosphate from turbine and hydraulic oils, as well as oil aerosol/vapors and total volatile organic compounds (VOC) in air with potential for occupational exposure for airport ground personnel. The measured exposure levels were mainly below the limit of quantification during work tasks, but provoked exposure situations resulted in significantly higher exposure levels compared to normal conditions, illustrated by oil aerosol up to 240  $\text{mg}/\text{m}^{-3}$  and tricresyl phosphate concentrations up to 31  $\text{mg}/\text{m}^{-3}$ . Highest exposure levels were measured during loading from jet engine aircraft [44].

Exposure to toxic compounds via contaminated bleed air (from engine compressors), including OPEs, has been widely studied among cabin crew and pilots, and has been associated with adverse neurological effects and respiratory illness [45, 46].

#### Metals and other elements

Metals which might be specific to airport emissions, either by abundance or type, such as the heavy-metal vanadium [47], could be potential chemical fingerprints. Abegglen et al. applied single particle mass spectrometry to investigate metal content and sources in emissions from different jet engines at various combustion conditions, and Mo, Ca, Na, Fe, Cu, Ba, Cr, Al, Si, Mg, Co, Mn, V, Ni, Pb, Ti and Zr were found to be significant frequently occurring metals. Fuel, lubrication oil, grease and engine wear are potential sources, but several metals were allocated to multiple sources [48].

In the studies of He et al and Shirmohammadi et al, particles were collected at Los Angeles Airport (LAX) and central Los Angeles (LA) and among other analyses, allocated according to elements associated with different sources [49, 50]. S was considered as aviation-related and particle-bound Na was viewed as ocean-related, due to sea salt from the ocean near by LAX. Al, Ca, Ti and K were considered as trace elements for road dust from LAX and central LA. Mn, Fe, Cu, Zn, Ba, Pb, Ni, and Mg were associated with traffic emissions, including fuel and lubricating oil combustions and brake abrasions, engine and tire wear. In LAX particles, S accounted for the largest fraction (49.5%), followed by road dust elements (21.8%) and traffic-related elements (15.9%). In particles from central LA, elements from traffic, road dust, and aviation were represented equally (28.5, 31.5, and 33.4%, respectively) [49, 50]. In a study from Montreal-Pierre-Elliott-Trudeau International Airport, several metals were found to be abundant in the particle fraction, such as Fe, Zn, and Al, and the authors speculate, that

airports in fact may be hotspots for nanoparticles containing emerging contaminants [37]. A recent study investigated the levels of 57 elements at five sampling sites within the vicinity of Eskisehir Hasan Polatkan Airport in Turkey, based on moss bag biomonitoring using *Sphagnum* sp. in combination with chemical analyses of lubrication oil and aviation gasoline fuel used by general aviation, piston-engine, and turboprop aircraft. Moss bag biomonitoring was a useful tool in identification of the elements that accumulated downwind of the airport emissions. Characterization of the metal contents in moss bags and oil and fuel were in agreement, showing that Pb, along with Cd, Cu, Mo, Cr, Ni, Fe, Si, Zn, Na, P, Ca, Mg, and Al were dominating elements in the general aviation aircraft emissions [51].

#### Polycyclic aromatic hydrocarbons/volatile organic compounds

Polycyclic aromatic hydrocarbons (PAH), including several known carcinogens, are also candidates for chemical airport emission tracers. PAH are semi-volatile compounds, in between the gaseous and particulate phases. Lighter-weight PAHs (<4 rings) present almost exclusively in the vapour-phase and PAHs with higher molecular weights (>4 rings) are almost completely particle-bound [5]. It was reported that the apron of the Fiumicino Airport in Rome had higher levels of measured PAH ( $27.2 \mu\text{g}/\text{m}^3$ ) compared to PAH levels in the airport building and terminal [52]. Another study of PAH in airport emissions at the apron reported that the five most abundant species of particle bound-PAHs for all sampling days were naphthalene, phenanthrene, fluoranthene, acenaphthene, and pyrene, with total concentrations between  $0.152 \mu\text{g}/\text{m}^3$  -  $0.189 \mu\text{g}/\text{m}^3$  ( $152.21$ – $188.94 \text{ ng}/\text{m}^3$ ) depending on season. The most abundant fractions of benzo(a)pyrene (BaP) equivalent concentration (BaP<sub>eq</sub>) in different molecular weights were high-weight PAHs (79.29%), followed by medium-weight PAHs (11.57%) and low-weight PAHs (9.14%). The percentages of total BaP<sub>eq</sub> in the very small particles <  $0.032 \mu\text{m}$  were 52.4% (mean concentration  $0.94 \text{ ng}/\text{m}^3$ ) and 70.15% in particles <  $100 \mu\text{m}$  (mean concentration  $1.25 \text{ ng}/\text{m}^3$ ) [53]. Studies of the emissions from a helicopter engine at different thrusts included analysis of 22 PAH compounds, where 97.5% of the total PAH emissions were two- and three-ringed PAHs, with a mean total PAH concentration of  $843 \mu\text{g}/\text{m}^3$  and a maximum of  $1653 \mu\text{g}/\text{m}^3$  during ground idle. This was 1.05–51.7 times higher compared to a heavy-duty diesel engine, a motor vehicle engine, and an F101 aircraft engine. In turn, total level of BaP during one landing and take-off cycle (LTO) ( $2.19 \text{ mg}/\text{LTO}$ ) [54] was higher than the European Commission emission factor of  $1.24 \text{ mg}/\text{LTO}$ , stated in their PAH position paper, where emission

factors are used to calculate the degree to which a source contributes to the total emission of a specific pollutant [55]. The Danish occupational exposure limit for PAH is  $200 \mu\text{g}/\text{m}^3$  [56], and reported PAH concentrations in ambient air across studies were below this level.

Volatile organic compounds (VOC) comprise a diverse group of organic chemicals, with different physicochemical and toxicological properties. Scientific studies of these emission compounds were meticulously reviewed by Masiol et al. [5], and as noted by the authors there is insufficient knowledge in terms of the significance of these compounds for airport exhaust health impacts [5]. Some VOC have known toxicities and other are suspected to have adverse health effects, and among the hydrocarbons found in aircraft exhaust, 14 single or complex compounds are listed as hazardous by the Federal Aviation Administration, which in addition to PAH compounds comprise benzene, styrene, xylene, toluene, acetaldehyde, 1,3-butadiene, n-hexane, acrolein, propionaldehyde, ethylbenzene, formaldehyde, and lead compounds [57]. A recent study assessed 46 VOC in the indoor air of the control tower maintenance room, potentially affecting employees, where a correlation was found between aircraft number and concentrations of light aldehydes/ketones [58].

#### Summary and perspectives

Emission measurement studies are continuously conducted at international airports, such as Amsterdam Airport Schiphol (AMS) [28, 59], Rome Ciampino (CIA) [60], London Heathrow (LHR) [61, 62], Beirut-Rafic Hariri International Airport (RHIA) [63], Hartsfield-Jackson Atlanta International Airport [64], Los Angeles International Airport (LAX) [32, 49, 65], and other large airports in California [66] which besides measurements of the previously mentioned compounds, also often include analyses of emission patterns and weather conditions, and characterizations of particle size- and mass distributions [67]. The data from these emission studies and physicochemical studies of emissions including particle matter (PM), from which we referenced some in the previous sections, were recently reviewed thoroughly [9]. To summarize the previous section, we repeat some selected important points regarding airport-sourced particles that were deducted from the available data by Stacey [9]:

- 1) *Particle numbers near airports are significantly higher than away from airports and jet engines are a significant source of UFP.* This means that urban areas in the vicinity of airports are at risk of increased exposure to UFP in addition to normal daily background and traffic-related emissions, but airport personnel working on the ground are in significant risk of exposure, simply due to proximity.

- 2) *The highest concentrations of UFP are measured downwind of aircraft.* Due to the occupational potential of exposure for airport ground workers there is a growing necessity of further studies of dispersion, size distributions and environmental factors affecting these emissions. Stacey [9] highlights that measurements at longer distances are highly influenced by physical and chemical processes affecting the emissions in the air, including volatile compounds. As such, there is a need for increased standardization of methods and instruments to facilitate valid comparisons between studies within this field, as has been established in general for environmental particulate matter (PM) measurements.
- 3) *Aircraft emissions are dominated by very small particles of < 20 nm.* This may be a way to separate these from other emission sources, such as road traffic, where the main particle fraction are of larger sizes. Smaller particle size means higher specific surface area. Smaller particles deposit in the deep end of the lung during inhalation and the total surface area of the deposited nanoparticles has been suggested to be predictive of toxicological potential in the lung [68].
- 4) *The majority of non-volatile airport emission particles are carbonaceous (consisting of elemental and organic carbon compounds).* The emissions from aircraft consists of high numbers of soot particles with associated PAHs and metals, and thus, their physico-chemical composition is similar to diesel exhaust particles [36].

Diesel exhaust is classified as carcinogenic to humans by IARC [69], and cause lung cancer, systemic inflammation, and inflammatory responses in the airways [70]. Animal studies have shown that the particulate fraction of diesel exhaust is mutagenic and carcinogenic [71], whereas filtered diesel exhaust does not cause cancer [72]. Exposure to standard reference diesel particle SRM1650b and carbon black (CB) induce pulmonary acute phase response, neutrophil influx, and genotoxicity in mouse models [73–78]. Genotoxicity has been observed even at very low doses of CB [79]. In a meta-analysis of exposure to diesel exhaust and lung cancer occurrence in three occupational studies, the identified dose-response relationship showed that occupational exposure to 1  $\mu\text{g EC}/\text{m}^3$  during a 45 year work life would cause 17 excess lung cancers per 10,000 exposed using the EC content of diesel exhaust as metric [80]. Another recent analysis of 14 case-control studies estimated exposure to diesel exhaust particles using job-exposure matrices. In this study, occupational exposure to 1  $\mu\text{g EC}/\text{m}^3$  during a 45 year work life would cause 4 excess lung cancers per 10,000 exposed using the EC content of diesel exhaust as metric [81].

Carcinogenic substances are evaluated and listed by the International Agency of Research in Cancer (IARC) under WHO according to accumulated scientific findings in cellular, animal and human studies. Group 1 entails substances with sufficient evidence of carcinogenicity in humans and group 2 includes substances that IARC has classified as probably (2A) or possibly (2B) carcinogenic to humans [82]. As almost all current aviation fuel/jet fuels are extracted from the middle distillates of crude oil (kerosene fraction), which is between the fractions for gasoline and diesel [5] (whose combustion emissions are classified as group 2B and group 1 carcinogens, respectively [69]), there is cause for concern in terms of the potential carcinogenicity of exposure to jet fuel combustion products.

### Exposure studies

Reported exposure levels for PAH, BC and UPF in the studies below are presented in Table 1.

#### Occupational exposure

Childers et al. (2000): An extensive study of PAH concentrations at an airbase was carried out, using real-time monitors and air samplers on different locations and in different flight-related and ground-support activities. Airborne and particle-bound PAH were measured in a break room, downwind from an aircraft (C-130H) during engine tests, in a maintenance hangar, in an aircraft (C-130H) cargo bay during cargo-drop training and during engine running on/off loading and backup exercises, and downwind from aerospace ground equipment (diesel-powered electrical generator and a diesel-powered heater). Measurements were carried out with three different monitors. Total PAH concentrations followed a general trend of downwind from two diesel aerospace ground equipment units > engine on/off-loading exercise > engine tests > maintenance hangar during taxi and takeoff > background measurements in the maintenance hangar. Reported mean total PAH concentrations in integrated air samples (vapor phase) were 0.6011  $\mu\text{g}/\text{m}^3$  (hangar background), 1.0254  $\mu\text{g}/\text{m}^3$  (hangar taxiing), 2.8027  $\mu\text{g}/\text{m}^3$  (engine test), 6.7953  $\mu\text{g}/\text{m}^3$  (engine running on/off) and 9.8111  $\mu\text{g}/\text{m}^3$  (aerospace ground equipment). Dominating PAH in all exposure scenario was naphthalene, the alkyl-substituted naphthalenes, and other PAHs in the vapor phase. Particle-bound PAHs, such as fluoranthene, pyrene, and benzo[a]pyrene were also found. During flight-related exercises, PAH concentrations were 10–15 higher than in ambient air, and it was found that PAH contents fluctuated rapidly from < 0.02 to > 4  $\mu\text{g}/\text{m}^3$  during flight-related activities [83].

Iavicoli et al. (2006): In this study, occupational exposure risk to PAH and biphenyl was evaluated in an Italian airport during winter. Concentration and purification of 12 samples of 25 PAH by gas chromatography-ion trap



**Table 1** Overview of reported levels of occupational exposures of PAH, BC, and particles in airports. Mean levels are presented if reported. For detailed data, see references

Description	Reported mean levels Ambient air	Reported mean levels Personal monitors	Reference
<b>PAH</b>			
Total mean PAH concentrations in integrated air samples at an airbase on different locations and in different flight-related and ground-support activities	601.1 ng/m <sup>3</sup> (hangar background) 1025.4 ng/m <sup>3</sup> (hangar taxiing) 2802.7 ng/m <sup>3</sup> (engine test) 6795.3 ng/m <sup>3</sup> (engine running on/off) 9811.1 ng/m <sup>3</sup> (diesel-fueled aerospace ground equipment) <i>During flight-related exercises, PAH concentrations were 10–15 times higher than in ambient air</i>	NA	Childers et al. (2000) [83]
PAH compounds of highest levels measured for 24 h in three different locations	130–13,050 ng/m <sup>3</sup> (naphthalene) 64–28,500 ng/m <sup>3</sup> (2-methylnaphthalene) 24–35,300 ng/m <sup>3</sup> (1-methylnaphthalene) 24–1610 ng/m <sup>3</sup> (biphenyl) 54.2 ng/m <sup>3</sup> (fluoranthene) 8.6 ng/m <sup>3</sup> (benzo[a]pyrene)	NA	Iavicoli et al. (2006) [84]
Total mean of 23 PAH (vapor and particle-bound) measured during 24 h of 5 work days at the airport apron, airport building and terminal/office area	27.703 µg/m <sup>3</sup> (apron) 17.275 µg/m <sup>3</sup> (airport building) 9.494 µg/m <sup>3</sup> (terminal departure area) <i>Highest levels in the airport apron particularly for 1 and 2-methylnaphthalene and acenaphthene</i>	NA	Cavallo et al. (2006) [52]
Total mean particle-bound PAH measured in the vicinity of LAX to assess the spread of airport emissions in up – and downwind ambient air to the immediate neighborhood	18.2 ng/m <sup>3</sup> (upwind from the airport) 24.6 ng/m <sup>3</sup> (downwind from the airport) 50.1 ng/m <sup>3</sup> (at the taxiway) 60.1 ng/m <sup>3</sup> (terminal region) <i>Particle-bound PAH mean levels measured on two freeways were 47.0 ng/m<sup>3</sup> and 169.4 ng/m<sup>3</sup></i>	NA	Westerdahl et al. (2008) [34]
<b>Black carbon</b>			
Mean black carbon concentrations measured at different micro-environments of airports and in commercial flights	3.78 µg/m <sup>3</sup> (during boarding/disembarking) 3.16 µg/m <sup>3</sup> (airport concourse) 2.78 µg/m <sup>3</sup> (inside aircraft with open doors) 0.81 µg/m <sup>3</sup> (inside aircraft on the ground with closed doors)	NA	Targino et al. (2017) [41]
BC levels measured in the vicinity of LAX to assess the spread of airport emissions in up – and downwind ambient air to the immediate neighborhood	0.3 µg/cm <sup>3</sup> (upwind from the airport) 0.7 µg/cm <sup>3</sup> (downwind from the airport) 1.8 µg/cm <sup>3</sup> (at the taxiway) 3.8 µg/cm <sup>3</sup> (terminal region)	NA	Westerdahl et al. (2008) [34]
Contributions of airport activities to measured BC levels at Amsterdam Schiphol were measured for 32 sampling days over 6 months	Mean BC: 0.6 mg/m <sup>3</sup>	NA	Pirhadi et al. (2020) [85]
<b>Particles</b>			
UFP and size distributions measured in the vicinity of LAX to assess the spread of airport emissions in up – and downwind ambient air to the immediate neighborhood	Average UFP counts of 5 × 10 <sup>4</sup> particles/cm <sup>3</sup> (500 m downwind of the airport), which were significantly influenced by aircraft operations where peaks were observed Maximum UFP measured was 4.8 × 10 <sup>6</sup> particles/m <sup>3</sup> downwind from a jet aircraft taking off Particle size: 90 nm (upwind from airport) 10–15 nm (downwind from airport)	NA	Westerdahl et al. (2008) [34]
Total mean concentration of 10 daily UFP samples with personal monitors placed with crew chief and hangar operator	6.5 × 10 <sup>3</sup> particles/cm <sup>3</sup> (downwind site)	2.5 × 10 <sup>4</sup> particles/cm <sup>3</sup> (crew chief) 1.7 × 10 <sup>4</sup> particles/cm <sup>3</sup> (hangar operator) <i>Median number concentrations for 2 months measurement period</i>	Buonanno et al. (2012) [86]
Geometric means of personal exposure to particle number concentration carried out in five different occupational groups	NA	37 × 10 <sup>3</sup> UFP/cm <sup>3</sup> (baggage handlers) 5 × 10 <sup>3</sup> UFP/cm <sup>3</sup> (landside security) 12–20 × 10 <sup>3</sup> UFP/cm <sup>3</sup> (catering drivers, cleaning staff and airside security)	Møller et al. (2014) [87]
Particle and metal exposure in ambient air and in airport workers using exhaled breath condensates	1.0 × 10 <sup>4</sup> –2.1 × 10 <sup>7</sup> particles/cm <sup>3</sup> (apron workers) 10 <sup>3</sup> –10 <sup>4</sup> (office staff) <i>Airport workers were exposed to significantly smaller particles (mean geometric size: 17.7 nm) compared</i>	Particulate content was found in exhaled breath condensates, but no difference was found between the two study groups	Marie-Desvergne et al. (2016) [88]



**Table 1** Overview of reported levels of occupational exposures of PAH, BC, and particles in airports. Mean levels are presented if reported. For detailed data, see references (Continued)

Description	Reported mean levels Ambient air	Reported mean levels Personal monitors	Reference
	<i>to office workers (mean geometric size: 23.7 nm).</i>		
Number concentrations and size distributions inside the cabin of an aircraft waiting for take-off compared to outdoor	10–40 × 10 <sup>3</sup> particles/cm <sup>3</sup> <i>A 40 min wait 100 m downwind of the runway was calculated to be equal to 4 h exposure in a clean urban background environment away from the airport</i>	NA	Ren et al. (2018) <sub>a</sub> [89]
Potential exposure to passengers and indoor airport staff investigated by PM <sub>2.5</sub> concentrations in the terminal building at three seasons	Arrival hall: 337 µg/m <sup>3</sup> (Winter) 105 µg/m <sup>3</sup> (Spring) 167 µg/m <sup>3</sup> (Summer) Departure hall: 385 µg/m <sup>3</sup> (Winter) 130 µg/m <sup>3</sup> (Spring) 170 µg/m <sup>3</sup> (Summer) Ambient airport air: 400 µg/m <sup>3</sup> (Winter) 156 µg/m <sup>3</sup> (Spring) 216 µg/m <sup>3</sup> (Summer) <i>1.9–5.9 times higher particles number concentrations in the terminal buildings than measured in a normal urban environment</i> <i>Total UFP exposure during an entire average waiting period (including in the terminal building and airliner cabin) of a passenger was estimated to be equivalent to 11 h of exposure to normal urban emissions</i>	NA	Ren et al. (2018) <sub>b</sub> [90]
UFP monitoring at several sampling sites in the vicinity of Lisbon Airport for 19 non-consecutive days	Downwind average particle number concentration range: 3.3 × 10 <sup>4</sup> cm <sup>3</sup> to 5.9 × 10 <sup>4</sup> particles per cm <sup>3</sup> Measured range of peaks: 2.3 × 10 <sup>5</sup> particles per cm <sup>3</sup> to 3.4 × 10 <sup>5</sup> particles per cm <sup>3</sup>	NA	Lopes et al. (2019) [91]
Maximal measurements at a commercial airport and exposure assessment at a non-commercial airfield	10 <sup>6</sup> –10 <sup>8</sup> particles/cm <sup>3</sup> (main combustion events of plane leaving and arriving) 1086 µg/m <sup>3</sup> (single peak event of plane leaving) <i>10.7% was predicted to deposit in the alveolar lung regions</i>	<i>Personal exposure levels were similar to air concentrations</i>	Bendtsen et al. (2019) [36]
Maximal UFP number concentration of UFP exposures investigated for 33 male employees working in an airport taxiway	9.59 × 10 <sup>6</sup> (during support tasks in taxiing and taking off of the aircraft)	2.44 × 10 <sup>3</sup> particles/cm <sup>3</sup> <i>Median UFP number concentration</i>	Marcias et al. (2019) [92]
Contributions of airport activities to measured particle number concentrations (PNCs) at Amsterdam Schiphol were measured for 32 sampling days over 6 months	Mean total PNC: 35,308 particles/cm <sup>3</sup> <i>Aircraft departures and aircraft arrivals contributed to 46.1 and 26.7% of PNC, respectively. Ground support equipment and local road traffic accounted for 6.5% of PNC and were characterized by diameters of 60–80 nm. Traffic from surrounding freeways was characterized by particles of 30–40 nm and contributed to 18% of PNC</i> Mean PM <sub>2.5</sub> : 7.4 mg/m <sup>3</sup> Particle size range: 10–20 nm	NA	Pirhadi et al. (2020) [85]

mass spectrometry sampled for 24 h in three different locations of the airport showed general low levels, with highest levels of naphthalene (0.13–13.05 µg/m<sup>3</sup>), 2-methylnaphthalene (0.064–28.5 µg/m<sup>3</sup>), 1-methylnaphthalene (0.024–35.3 µg/m<sup>3</sup>), and biphenyl (0.024–1.610 µg/m<sup>3</sup>). Measured levels of the carcinogens benzo[b + j + k]fluoranthene and benzo[a]pyrene were 0.0542 µg/m<sup>3</sup> and 0.0086 µg/m<sup>3</sup> respectively [84].

Buonanno et al. (2012): Occupational exposure and particle number distributions were studied at an aviation base on a downwind site, close to the airstrip and by 10

daily UFP samples with personal monitors placed with a crew chief (assists the pilots during ground activities) and a hangar operator (aircraft maintenance). Particle number distribution averaged a total concentration of 6.5 × 10<sup>3</sup> particles/cm<sup>3</sup> at the downwind site. Short-term peaks during the working day mainly related to takeoff, landing and pre-flight operations of jet engines were measured in the proximity of the airstrip. Personal exposure concentrations were higher than stationary monitoring measurements. Personal exposure of workers were at a median number concentration of 2.5 × 10<sup>4</sup>

particles/cm<sup>3</sup> for the crew chief and  $1.7 \times 10^4$  particles/cm<sup>3</sup> for the hangar operator during the 2 months measurement period. The crew chief experienced the highest exposures, with maximum values at approximately  $8 \times 10^4$  particles/cm<sup>3</sup> [86].

Møller et al. (2014): Personal exposure monitoring of particle number concentration was carried out in five different occupational groups, namely baggage handlers, catering drivers, cleaning staff, airside security and land-side security in CPH, for 8 days distributed over 2 weeks. The study reported significant differences among the occupational groups. Highest exposures were found in baggage handlers (geometric mean:  $37 \times 10^3$  UFP/cm<sup>3</sup>), which was 7 times higher in average compared to land-side security which are indoor employees (geometric mean:  $5 \times 10^3$  UFP/cm<sup>3</sup>). In between highest and lowest exposure groups, were catering drivers, cleaning staff and airside security with similar exposure levels (geometric mean:  $12\text{--}20 \times 10^3$  UFP/cm<sup>3</sup>) [87].

Targino et al. (2017): Black carbon (BC) particle concentrations were measured within different micro-environments of 12 airports and on 41 non-smoking commercial flights. Great variability was seen depending on environment measured. 70% of personal exposure during a journey occurred in the airport concourses and during transit to/from the aircraft. 18% was contributed to the waiting time onboard an aircraft with open doors waiting for loading. Largest BC exposure were found during boarding and disembarking (mean BC =  $3.78 \mu\text{g}/\text{cm}^3$ ; 25th, 50th, 75th percentiles: 1.29, 2.15, 4.68), at the airport concourse (mean BC =  $3.16 \mu\text{g}/\text{cm}^3$ ; 25th, 50th, 75th percentiles: 1.20, 2.15, 4.0) and inside parked aircraft with open doors (mean BC =  $2.78 \mu\text{g}/\text{cm}^3$ ; 25th, 50th, 75th percentiles: 0.35, 0.72, 2.33). BC levels were low in the aircraft on the ground with closed doors (mean BC =  $0.81 \mu\text{g}/\text{cm}^3$ ; 25th, 50th, 75th percentiles: 0.2, 0.35, 0.72, respectively). Lowest concentration was found during flights in the air [41].

Ren et al. (2018)<sup>a</sup>: The number concentrations and size distributions inside the cabin of an aircraft waiting for take-off were investigated and analyzed in comparison to outdoor UFP and the use of the ground air-conditioning cart (GAC) and environmental control system (ECS), which are used to provide conditioned air between boarding and doors closing to prepare for take-off. The study showed that environmental particle number concentration varied significantly, ranging from 10 to  $40 \times 10^3$  particles/cm<sup>3</sup> depending on wind, and take-off and landing activities. When the GAC was on, the indoor particle numbers followed those outdoors, with the ECS providing protection factors for crew and passengers from 1 to 73% for 15–100 nm particles, and from 30 to 47% for 100–600 nm particles. A 40 min wait 100 m downwind of the runway was calculated to be equal to 4

h exposure in a clean urban background environment away from the airport [89].

Ren et al. (2018)<sup>b</sup>: In this study, the potential exposure to passengers as well as indoor airport staff was investigated by measurements in the terminal building of Tianjin Airport in Beijing of CO<sub>2</sub>, PM<sub>2.5</sub>, and UFP concentration and particle size distribution during three seasons. The effects on the indoor air quality of airliner-generated particles penetrating from the outdoor environment through open doors and by heating, ventilation and air-conditioning systems was studied.

PM<sub>2.5</sub> concentrations in the terminal building varied during the seasons of winter, spring and summer with 337–105–167  $\mu\text{g}/\text{m}^3$  in the arrival hall, 385–130–170  $\mu\text{g}/\text{m}^3$  in the departure hall, and 400–156–216  $\mu\text{g}/\text{m}^3$  in ambient airport air, respectively. These were significant higher levels compared to Chinese standard and WHO annual mean value of 10  $\mu\text{g}/\text{m}^3$  during all the tested seasons. The indoor environment was significantly affected by the outdoor air levels (Spearman:  $p < 0.01$ ). Particle number concentration in the terminal building displayed two size distribution, with one mode at 30 nm and a mode at 100 nm, which was significantly different from the size distribution measured in a normal urban environment, which had one peak at 100 nm. The study reports particle number concentrations of 1.9–5.9 times higher in the terminal buildings than the concentrations measured in a normal urban environment by different size bins. Measured total UFP exposure during an entire average waiting period (including in the terminal building and airliner cabin) of a passenger was estimated to be equivalent to 11 h of exposure to normal urban emissions [90].

Bendtsen et al. (2019): In this study, the occupational exposure levels to particles was evaluated by measurements at a non-commercial airfield and particles were collected and characterized at a non-commercial airfield and from the apron of a commercial airport.

Electron microscopy showed that the aerosol at the non-commercial airfield appeared to be mainly aggregates of soot, whereas the aerosol at the apron of the commercial airport appeared much more complex dominated by agglomerated soot particles, salt crystals and pollen. At the commercial airport, particles were mainly below 300 nm in diameter and distributed in two modes with geometric mean diameters of < 20 nm and approximately 140 nm. At the non-commercial airfield, two full cycles of a normal workflow of plane leaving, plane arriving and refueling by were recorded in a jet shelter using stationary and portable devices including in the breathing zone of personnel. Average particle number concentration for a full workflow cycle of 170 min were  $1.22 \times 10^6$  particles/cm<sup>3</sup>. For take-off and landing of one jet plane, average particle number concentrations and mass were 7.7 particles/cm<sup>3</sup> and 1086  $\mu\text{g}/\text{m}^3$  and 2.67

particles/cm<sup>3</sup> and 410 µg/m<sup>3</sup>, respectively. During the main combustion events of plane leaving and arriving, the instruments reached their upper detection limits of 10<sup>6</sup> particles/cm<sup>3</sup> (DiSCmini, which measures particle number concentration, mean particle size and lung-deposited surface area) and 10<sup>8</sup> particles/cm<sup>3</sup> (ELPI, which monitors real-time particle levels), including in the breathing zone monitor of the personnel. Prevalent particle sizes suggested that the jet engine combustion particles were < 10 nm in aerodynamic diameter [36].

Mokalled et al. (2019): In this study, 48 volatile organic compounds (VOC) from approximately 100 commercial aircraft during real operations of different engine modes at Beirut Rafic Hariri International Airport were assessed to identify specific markers, together with measurements of Jet A-1 kerosene fuel vapors and gasoline exhaust.

Heavy alkanes (C8-C14, mainly n-nonane and n-decane) contributed to 51–64% of the total mass of heavy VOCs emitted by aircraft. Heavy aldehydes (nonanal and decanal) was reported as potential tracers for aircraft emissions due to their exclusive presence in aircraft-related emissions in combination with their absence from gasoline exhaust emissions. Total concentration of heavy alkanes in the ambient air was 47% of the total mass of heavy VOCs measured. No aircraft tracer was identified among the light VOCs (≤ C7). VOC compositions in jet exhaust varied with combustion power, and it was shown that light VOC emissions decrease as the engine power increases. Auxiliary power unit (APU) emissions were identified to be of the same order of magnitude as main engine emissions [93].

Marcias et al. (2019): In this study, occupational exposure to ultrafine particles and noise was investigated for 33 male employees working in an airport taxiway in a smaller Italian airport. Job categories represented were aircraft ground equipment personnel, firefighting officer, flight security agent, and aviation fuel administration staff. Both stationary sampling (ELPI) and personal particle measurements were included. The morphology and chemical composition was determined by EM and EDS, and showed small soot particles in aggregates with sodium, potassium, magnesium, calcium, aluminium, carbon, nitrogen, silicon, oxygen, fluorine, chlorine and sulphur. The maximal UFP number concentration ( $9.59 \times 10^6$  particles/cm<sup>3</sup>) on stationary equipment was measured during support tasks in taxiing and taking off of the aircraft. Median UFP number concentration measured with personal monitors on the 33 operators was  $2.44 \times 10^3$  particles/cm<sup>3</sup> and a maximum of  $13 \times 10^3$  particles/cm<sup>3</sup>. Average size range was 35–103 nm. A significant difference in mean size and distributions was found between job tasks, where flight security officers were exposed to particles with lower mean sizes as compared to aircraft ground equipment operators [92].

### Residential exposure

Westerdahl et al. (2008): Air measurements were carried out in the vicinity of LAX to assess the spread of airport emissions in downwind ambient air to the immediate neighborhood. Ultrafine particle numbers (UFP), size distributions, particle size, black carbon (BC), nitrogen oxides (NO<sub>x</sub>), and particle-bound PAH were measured. The lowest levels of pollutants were measured upwind of the airport, where UFP ranged from 580 to 3800 particles/cm<sup>3</sup>, black carbon from 0.2 to 0.6 µg/m<sup>3</sup>, and particle-bound PAH from 18 to 36 ng/m<sup>3</sup>. In contrast, at 500 m downwind of the airport, average UFP counts of 50,000 particles/cm<sup>3</sup> were observed, which were significantly influenced by aircraft operations where peaks were observed. Black carbon, particle-bound PAH, and NO<sub>x</sub> were also elevated, although not in the same extent, and the authors observed that BC, particle numbers, and NO<sub>x</sub> levels varied together in similar patterns indicating they were associated with similar sources. Black carbon concentrations varied across the measurement sites, with a mean of 0.3 µg/cm<sup>3</sup> upwind from the airport, 0.7 µg/cm<sup>3</sup> downwind from the airport, 1.8 µg/cm<sup>3</sup> at the taxiway, and 3.8 µg/cm<sup>3</sup> in the terminal region. Mean PM-PAH levels were 18.2, 24.6, 50.1 and 60.1 ng/m<sup>3</sup> at the measurement sites, respectively. PM-PAH mean levels measured on two freeways were 47.0 ng/m<sup>3</sup> and 169.4 ng/m<sup>3</sup>. The maximum UFP measured was  $4.8 \times 10^6$  particles/m<sup>3</sup> downwind from a jet aircraft taking off. NO<sub>x</sub> levels before the take-off were around 8 ppb and increased to 1045 ppb, mostly due to NO. Black carbon rose from approximately 800 to 9550 ng/m<sup>3</sup>, and PM-PAH values increased from 37 to 124 ng/m<sup>3</sup>. Significant variations were observed in particle sizes, where upwind measurements were dominated by particles of 90 nm, and downwind particles were of 10–15 nm in size. The author noted that UFP levels from aircraft were measured to persist up to 900 m from the runways, indicating potential risks for the nearby communities [34].

Lopes et al. (2019): In this study, data is presented from UFP monitoring at several sampling sites in the vicinity of Lisbon Airport in 2017 and 2018, for 19 non-consecutive days. Measurements included sites further away from the airport, under the landing/take-off path. Correlation analysis between air traffic activity and UFP concentrations was conducted and show the occurrence of high UFP concentrations in the airport vicinity. The particle counts increased 18–26 fold at locations near the airport, downwind, and 4-fold at locations up to 1 km from the airport. Results show that particle number increased with the number of flights and decreased with the distance to the airport [91].

Pirhadi et al. (2020): In this study, the contributions of airport activities to particle number concentrations (PNCs) at Amsterdam Schiphol was quantified by use of

the positive matrix factorization (PMF) source apportionment model. Various pollutants were measured, including NO<sub>x</sub> and CO, black carbon, PM<sub>2.5</sub> mass, and the number of arrivals and departures were measured for 32 sampling days over 6 months. Airport activities accounted for 79.3% of PNCs divided in aircraft departures, aircraft arrivals, and ground service equipment (GSE) (with contributions of local road traffic, mostly from airport parking areas). Aircraft departures and aircraft arrivals contributed to 46.1 and 26.7% of PNCs, respectively, and were characterized by particle diameters < 20 nm. GSE and local road traffic accounted for 6.5% of the PNCs and were characterized by diameters of around 60–80 nm. Traffic from surrounding freeways was characterized by particles of 30–40 nm and contributed to 18% of PNCs. In comparison, the urban background emissions dominated the mass concentrations with 58.2%, but had the least contribution to PNCs with 2.7% [85].

### Summary of exposure studies

Occupational exposure to increased levels of nanosized particles [36, 85–90, 92], increased levels of PAH including known human carcinogens [52, 83, 84], and black carbon [41] were reported in the literature. Levels of exposure reported in these studies are summarized in Table 1. One study reported that personnel monitors measured higher levels compared to stationary equipment [87], and it was shown that ground support equipment, such as diesel-powered electrical generators and heaters [83] and auxiliary power units [93] contribute significantly to emissions.

Three important main factors were identified which significantly influenced occupational exposure: *proximity to emission sources*, where levels were generally higher in close proximity and down-wind to aircraft, *fluctuations in emission levels*, characterized by exposure peak events such as landing- or take-off, and *job type*, where outdoor ground-affiliated work types are at highest risk of exposure. As such, airport personnel can likely be grouped in low (office staff/landside jobs with indoor work, far away from emission sources), medium (catering/cleaning/landside security staff with intermittent outdoor work) and high (baggage handlers/aircraft mechanics/ crew chief) exposure groups.

The majority of studies on the contribution of airport emissions to air pollution in the surrounding environment are physical/chemical studies of particle numbers, mass and related air pollutants, which are reviewed elsewhere as previously described.

More studies reported increased risk of exposure correlating with decreased distance to airports [94–96] and time spent downwind from an airport [97], hence a significant factor for potential health effects for neighboring

residential areas based on these studies is *distance to airports*, which relating to wind and atmospheric conditions is an important determinant for pollution levels.

### Health effects

Here we present studies in which direct health effects have been assessed in humans, including in biomonitoring and epidemiological studies, and biological mechanisms-of-action assessed in animal or cell studies. Our main focus is particle exposure, however, studies focusing more on VOC/PAH are also presented.

### Occupational studies

Møller et al. (2017 and 2019): A prospective, occupational cohort study in CPH, encompassing 69,175 men in unskilled positions as baggage handlers or in other outdoor work used register information of socioeconomic, demographic and health data together with a job-exposure matrix was based on GPS measurements within the airport, detailed information on tasks from 1990 to 2012, exposure to air pollution at home, and lifestyle details. Occupational exposure groups were categorized according to work time at the apron, “apron-years” (non-exposed, 0.1–2.9, 3.0–6.9 and ≥ 7 years). The reference group comprised different low-exposure occupational groups [98]. A follow-up study was conducted on an exposed group of 6515 male airport workers at 24–35 years of age in unskilled positions with a reference group of 61,617 men from greater Copenhagen area in unskilled jobs. Exposure was assessed by recordings of time spent on the airport apron and diagnoses of ischemic heart disease and cerebrovascular disease was obtained from the National Patient Register. No associations between cumulative apron-years and the two disease outcomes were found. On the other hand, since the exposed group had a mean age of 24–35 years, a 22-year follow-up may have been too short to detect cardiovascular effects [99].

Lemasters et al. (1997): In this early study, mixed low-level exposure to fuel and solvent was studied in a repeated measures design with male aircraft workers at a military air station serving as their own controls from pre-exposure to 30 weeks post-exposure. The study group consisted of six aircraft sheet metal workers mainly exposed to solvents, adhesives and sealants, six aircraft painters exposed to solvents and paints, 15 jet fueling operations personnel ( $n = 15$ ) responsible for fuel delivery, fueling and defueling aircraft and repairing fuel systems, and 23 workers in the flight line crew exposed to jet fuel, jet exhaust, solvents and paint, and included ground crew and jet engine mechanics. Expired breath analysis was carried out for different trace compounds, but was found to have low values (< 25 parts per billion). An increase in sister chromatid exchange (SCE)



compared to pre-exposure was found after 30 weeks of exposure for sheet metal workers (mean SCE per cell increased from 6.5 (SD: 0.8, range: 5.5–7.7) to 7.8 (SD: 0.3, range: 7.4–8.2) and painters (mean SCE per cell increased from 5.9 (SD: 0.7, range: 5.0–6.8) to 6.7 (SD: 1.0, range 5.3–7.8)), indicating exposure to genotoxic substances for these subgroups [100].

Tunncliffe et al. (1999): In Birmingham International Airport, occupational exposure to aircraft fuel and jet stream exhaust was evaluated in terms of respiratory symptoms and spirometry in 222 full-time employees according to job title. Data was collected by questionnaire and with on-site measurement of lung function, skin prick tests, and exhaled carbon monoxide concentrations. Occupational exposure was assessed by job title, where baggage handlers, airport hands, marshallers, operational engineers, fitters, and engineering technicians were considered as high exposure groups, security staff, fire fighters, and airfield operations managers as medium exposure group, and low exposure groups consisted of terminal and office workers. Upper and lower respiratory tract symptoms were commonly reported in the questionnaire and 51% had one or more positive allergen skin tests. Cough with phlegm and runny nose were found to be significantly associated with high exposure (adj. OR = 3.5, CI: 1.23–9.74; adj. OR = 2.9, CI: 1.32–6.4, respectively). Upper and lower respiratory symptoms were common among exposed workers, but no significant difference was found in lung function. The authors conclude that it is more likely that these symptoms reflect exposure to exhaust rather than fuel [101].

Yang et al. (2003): The aim of this study was to evaluate self-reported adverse chronic respiratory symptoms and acute irritative symptoms among 106 airport workers in risk of exposure to jet fuel or exhaust (jet fuel handlers, baggage handlers, engineers etc.) compared to 305 terminal or office workers (control group) at Kao-hsiung International Airport (KIA) in Taiwan. The odds ratio analyses were adjusted for possible confounding factors, such as age, marital status, education, duration of employment, smoking status, and previous occupational exposure to dust or fumes. The prevalence of acute irritative symptoms was not significantly different, whereas chronic respiratory symptoms such as cough (adj. OR = 3.41, CI: 1.26–9.28) and dyspnea (adj. OR = 2.34, CI: 1.05–5.18) were significantly more common among airport workers. The study did not report exposure measurements, but the authors conclude that the expected higher exposure of aviation fuel or exhaust in the ground personnel is the likely explanation for the increased incidence of self-reported chronic respiratory health-effects compared to the office personnel [102].

Whelan et al. (2003): Prevalence of respiratory symptoms among female flight attendants along with teachers

was investigated by self-reported questionnaire in comparison to database-derived data on blue collar workers with no known occupational exposures, and it was found that female flight attendants and teachers were significantly more likely to report work related eye (12.4 and 7.4%), nose (15.7 and 8.1%), and throat symptoms (7.5 and 5.7%), and more episodes of wheezing and flu, compared to other female workers (2.9% eye, 2.7% nose, and 1.3% throat symptoms). Female flight attendants were significantly more likely than teachers and controls to report chest illness 3 years in retrospective (flight attendants: 32.9%, teachers: 19.3%, female workers: 7.2%) [103].

Cavallo et al. (2006): In this study, 41 airport employees in jobs with very close proximity to aircraft in service (fitters, airport hands, marshallers, baggage handlers) or in jobs with some proximity to aircraft (security staff, maintenance service personnel, cleaning staff, air field operations managers, runway shuttle drivers) in Leonardo da Vinci airport in Rome were evaluated for exposure to aircraft emissions along with biomarkers of genotoxicity in comparison to a control group of 31 office workers at the same airport. Job tasks in very close proximity to aircraft in service were considered to be high exposure jobs. Urinary PAH metabolites were used as biomarker of endogenous PAH exposure in parallel with PAH analyses of air samples. Exfoliated buccal cells and blood were evaluated for DNA damage, e.g. micronuclei, chromosomal aberrations and sister chromatid exchange (SCE). PAH exposure was measured during 24 h of 5 work days at the airport apron, airport building and terminal/office area from January to February 2005. Total mean of 23 PAHs (particle and vapour) at the apron, airport building and terminal departure area were 27.7, 17.2, and 9.5  $\mu\text{g}/\text{m}^3$ , respectively, with a prevalence of 2–3 ring PAHs with highest levels in the airport apron particularly for 1- and 2-methylnaphthalene and acenaphthene. Urinary PAH metabolite levels were similar for high exposure job groups and controls. The exposed group showed increased SCE (mean number:  $4.61 \pm 0.80$ ) compared to control group ( $3.84 \pm 0.58$ ) and increased levels of chromosomal aberrations and DNA strand breaks in the Comet assay in both buccal cells and lymphocytes, indicating genotoxic exposures [52].

Radican et al. (2008): A follow-up study of 14,455 workers from 1990 to 2000 evaluated the mortality risk from trichloroethylene and other chemical exposures in aircraft maintenance workers. Relative risk (RR) for exposed compared to unexposed workers were calculated, and positive associations with several cancers were observed, but mortality had not changed substantially since 1990, with increased risk of all-cause mortality (RR = 1.04, CI: 0.98–1.09) or death from all cancers (RR = 1.03, CI: 0.91–1.17) [104].



Erdem et al. (2012): A study group consisting of 43 aircraft fuel maintenance staff, fuel specialists, and mechanics occupationally exposed to JP-8 fuel directly or via engines of jet planes were evaluated for the metabolites 1- and 2-naphthol and creatinine in urine as biomarkers of exposure to jet fuel. In turn, sister chromatid exchange (SCE) and micronuclei were evaluated in blood-derived lymphocytes as biomarkers of genotoxic exposure. Urinary markers and SCE were significantly increased in exposed workers (1-naphthol: 99.01  $\mu\text{mol/mol}$  creatinine; 2-naphthol: 77.29  $\mu\text{mol/mol}$  creatinine), by 10-fold as compared to a control group of 38 employees working in the same area without any work-related exposure to JP-8 fuel [105].

Marie-Desvergne et al. (2016): In this study, exposure to airport nanoparticles and metals was evaluated in airport workers by exhaled breath condensate (EBC) as a non-invasive representative of the respiratory system. EBC was collected from 458 airport workers from Marseille Provence Airport and Roissy Charles de Gaulle Airport in Paris, working directly on the apron (exposed) or in the offices (less exposed). In addition, ambient nanoparticle exposure levels were characterized in terms of particle number concentration, size distribution and by electron microscopy.

The study showed that airport workers were exposed to significantly higher particle numbers ( $1.0 \times 10^4$ – $2.1 \times 10^7$  particles/ $\text{cm}^3$ ) compared to office staff ( $10^3$ – $10^4$  range equivalent to background traffic emissions), although office workers were periodically exposed to peaks of  $10^4$ – $10^5$  when the building doors were open. Airport workers were exposed to significantly smaller particles (mean geometric size: 17.7) compared to office workers (mean geometric size: 23.7). EBC was characterized by volume, total protein content, and a multi-elemental analysis was used to.

measure Na, Al, Cd, and Cr. Particles in EBC were analyzed with dynamic light scattering and electron microscopy (SEM-EDS).

A significantly higher concentration of Cd was found in apron worker EBC (mean:  $0.174 \pm 0.326 \mu\text{g/l}$ ) in comparison with office workers (mean:  $0.108 \pm 0.106 \mu\text{g/l}$ ). Particulate content in EBC was confirmed by DLS and SEM-EDS, but no differences were found between the two study groups, and measured EBC particle contents did not correlate with ambient exposure levels [88].

#### Studies on effects of residential exposure to airport emissions

Visser et al. (2005): In this population-based study, it was investigated if the residents living around Amsterdam Schiphol Airport were at higher risk of developing cancer compared to the general Dutch population. The regional cancer registry was used, estimating the cancer incidence from 1988 to 2003 in the

population residing near the airport compared to the national cancer incidence. The exposure was defined by aircraft noise and postal code areas, as historical data on ambient air pollution were unavailable. The study did not include information on lifestyle factors, and therefore, did not control for smoking and other potential confounders. A core zone closest to the airport and a remaining ring zone was studied. Thirteen thousand two hundred seven cancer cases were identified in the study area, and a significant increase in the incidence of hematological cancers (standardized incidence ratio, SIR = 1.12, CI: 1.05–1.19) was found, mainly due to non-Hodgkin lymphoma (SIR = 1.22, 95% CI: 1.12, 1.33) and acute lymphoblastic leukemia (SIR = 1.34, CI: 0.95, 1.83). Respiratory system cancer incidence was significantly decreased (SIR = 0.94, CI: 0.90, 0.99), due to the low rate in males (SIR = 0.89). The study concludes that the overall cancer incidence in the residential areas closest to Amsterdam Schiphol Airport was similar to the national incidence. The increase in the risk of hematological cancers could not be explained by higher levels of ambient air pollution in the area [106].

Lin et al. (2008): In this cross-sectional study, it was assessed whether residents living near commercial airports had increased rates of hospital admissions due to respiratory diseases compared to those living further away. The study included all residents living within 12 miles from the center of each of three airports (Rochester in Rochester, LaGuardia in New York City and MacArthur in Long Island). Hospital admission data were collected by the New York State Department of Health for all residents who were hospitalized for asthma, chronic bronchitis, emphysema, chronic obstructive pulmonary disease and, for children aged 0–4 years, bronchitis and bronchiolitis during 1995–2000. Exposure indicators were distance from the airport and dominant wind patterns from the airports.

The relative risks of hospital admissions due to respiratory conditions for residents living < 5 miles from the airport were 1.47 (CI: 1.41–1.52) for Rochester and 1.38 (CI: 1.37–1.39) for LaGuardia, as compared to those living > 5 miles from the airports. No differences were observed for MacArthur airport. When considering hospital admission rates by distance for 12–1 miles towards the airports, a significant trend of increasing hospital admissions with closer distance to the airport was observed for the Rochester airport. The authors reported a stronger effect for traditionally lower socio-economic groups [94], which may be of more relevance in the US, due to the medical insurance system.

Habre et al. (2018): In this study, 22 non-smoking volunteers with mild to moderate asthma were recruited to do scripted mild walking activity in parks inside or outside a zone of high airport-related ultrafine particle

exposure downwind of LAX. Physiological parameters were measured before and after exposure, and the study was conducted as a cross-over study, such that the participants served as their own controls. Personal exposure to black carbon, PAH, ozone, and PM<sub>2.5</sub> were measured and combined with source appointment analysis and health models. A difference in PM exposure was found between the high (mean particle number concentration of 53,342 particles/cm<sup>3</sup> and mean particle size of 28.7 nm) and the low exposure zone (mean particle number concentration of 19,557 particles/cm<sup>3</sup> and mean particle size of 33.2 nm). It was reported that IL-6 levels in blood were increased after the walk in the high exposure zone compared to the low exposure zone. Airport-related PM was distinguished from roadway traffic emissions by principal component analysis, and increase of airport-related PM was significantly associated with increased IL-6 levels [107].

Amsterdam Schiphol report (2019): Based on three studies with 191 primary school children from residential areas near Schiphol Airport, 21 healthy adults living adjacent to the airport [108], and an in vitro study [109], respectively, this Dutch report (not subjected to peer review) describes the findings of reduced lung function in children and adults following higher short-term exposure to ultrafine particles near Schiphol Airport. On days with high exposure, children suffered more from respiratory complaints and used more medicine. In the adults, short-term reductions in heart function were also found. The authors note that these effects may be larger for individuals already suffering from medical conditions. The authors point out that the effects are results of ultrafine particles from both air and road traffic, and that there are no indications that health effects of air traffic emissions are different from those caused by road traffic [59].

Lammers et al. 2020: This study investigated the health effects of controlled short-term exposure of 21 healthy non-smoking volunteers aged 18–35 years to UFP near Schiphol Airport Amsterdam. The volunteers were exposed 2–5 times to ambient air for 5 h while cycling. Cardiopulmonary outcomes such as spirometry, forced exhaled nitric oxide, electrocardiography and blood pressure were measured before and after exposure, and compared to measured total- and size-specific particle number concentrations (PNC). Average PNC was 53,500 particles/cm<sup>3</sup> (range 10,500–173,200). Increase in exposure to UFP was associated with a decrease in FVC and a prolongation of the corrected QT interval, which were associated with particle sizes < 20 nm (UFP from aviation), but not with particles > 50 nm (UFP from road traffic). Although the effects were relatively small and measured after single exposures of 5 h in young healthy adults [108], such effects could be important in susceptible sub-populations.

#### Animal studies and in vitro studies

Ferry et al. (2011): Immature primary human monocyte-derived dendritic cells (DCs) from healthy donor blood were exposed for 18 h to different doses of experimental jet exhaust particles in absence or presence of *E. coli* lipopolysaccharides (LPS). Antigen-presenting and stimulatory molecules were measured along with tumor necrosis factor (TNF $\alpha$ ) and IL-10. The effects were assessed on immature and mature DCs as well as on cells during the maturation process.

The primary particles collected from the jet exhaust by direct impaction were found to be spherical and carbonaceous primary particles of ~ 10 nm and aggregates up to ~ 93 nm. No toxic effects were observed for doses below of 100  $\mu$ g/mL jet engine particles. Maturation of immature dendritic cells by LPS stimulation induced a significant 500-fold increase in TNF $\alpha$  and 30-fold increase in IL-10. Immature dendritic cells produced low amounts of TNF $\alpha$  (fold change from LPS: 0.006) and IL-10 (fold change from LPS: 0.11), which increased non-significantly upon stimulation with particles (fold change from LPS: TNF $\alpha$ : 0.11, IL-10: 0.19). However, simultaneous exposure to LPS and a high particle dose of 100  $\mu$ g/mL induced a 2-fold increase in TNF $\alpha$  production compared to LPS-maturation ( $p = 3 \times 10^{-5}$ ). Different activation patterns were seen for the expression of HLA DR and CD86, which are dendritic cell maturation markers. It was concluded that jet exhaust particles may act as adjuvants to endotoxin-induced dendritic cell maturation, which may influence potential effects on human health [110].

Shirmohammadi et al. (2018): PM<sub>0.25</sub> collected at the vicinity of Los Angeles Airport (LAX) and from central Los Angeles (LA) close to and downwind from major freeways, from stationary sampling stations used for air quality control, were investigated. The particles were subjected to source allocation analyses of elements and carbon contents (see Introduction), and ROS formation was compared in rat alveolar macrophage cells (NR8383).

ROS activity measured as units of Zymosan equivalents were normalized by total PM<sub>0.25</sub> mass to represent the intrinsic toxicity of the particles, and this mass-normalized ROS activity was similar for LAX (4600.93  $\pm$  1516.98  $\mu$ g Zymosan/mg PM) and central LA (4391.22  $\pm$  1902.54  $\mu$ g Zymosan/mg PM). According to the authors, volume-normalization of the ROS activity can be used as a metric for comparison of inhalation exposures, as an indicator of exposure severity. A slightly higher PM<sub>0.25</sub> mass concentration in central LA meant overall similar volume-normalized ROS activity levels with no significant difference between the observed averages (LAX: 24.75  $\pm$  14.01  $\mu$ g Zymosan/m<sup>3</sup>, central LA: 27.77  $\pm$  20.32  $\mu$ g Zymosan/m<sup>3</sup>). Thus, there were similar levels of ROS activity and similar toxic potential of the PM in the vicinity of LAX and in the vicinity of freeways in central LA [49].

He et al. (2018): PM<sub>0.25</sub> collected at Los Angeles Airport (LAX) and from central Los Angeles (LA) close to and downwind from major freeways (similar collection sites as in [49]) were investigated and compared. Particles were source-allocated by analyzing elements (see Introduction). Particles collected at LAX were primarily associated with aircraft emissions, and particles from central LA with urban traffic, road and dust emissions. The reactive oxygen species (ROS) potential was evaluated intracellularly in human bronchial epithelial cells (16HBE) after 1, 2, and 4 h of exposure, and IL-6, IL-8 and TNF were measured as markers of inflammation.

Exposure of 16HBE cells to 10 µg/mL particles produced significantly elevated ROS levels for both samples compared to unexposed cells. Particles from central LA generated slightly more ROS than LAX samples per mass unit, and both were at negative control level after 20 h recovery. ROS potential in PM from both airport and central LA correlated with some of the measured traffic-related transition metals (Fe and Cu). Particles from LAX induced increased expression of IL-6, IL-8 and TNFα compared to the negative control (1.7, 1.8, and 1.4-fold, respectively), whereas central LA-particles induced slightly lower expressions (1.3, 1.3, and 1.1-fold, respectively). Hence, overall LAX particles had similar inflammatory potency as particles from central LA, showing that airport PM<sub>0.25</sub> contributions to urban emission PM pollution possess similar inflammatory properties [50].

Jonsdottir et al. (2019): In this study, aerosol was collected from the world's most used aircraft turbine (CFM56–7B26, run-in and airworthy) in a test cell at Zurich Airport. The test cell is open to the ambient environment and the aerosol was collected from both standard Jet A-1 fuel and a HEFA fuel blend. The toxicity of the non-volatile PM emissions was studied by direct particle deposition onto air-liquid interface cultures of human bronchial epithelial cells (BEAS-2B).

Cytotoxicity was evaluated by the release of cytosolic LDH from damaged cells, expression of the oxidative stress marker HMOX-1 and inflammatory cytokines IL-6 and IL-8.

Single, short-term (1 h) exposure to PM increased cell membrane damage, lead to oxidative stress and increased pro-inflammatory cytokines in bronchial epithelial cells, depending on fuel type and combustion conditions from which the particles were produced. PM from conventional fuel at ground-idle conditions was most potent, and the authors comment that PM from aircraft turbine exhaust may be a risk to respiratory health, also by making airway epithelia vulnerable to secondary exposure of other air pollution compounds and pathogens [111].

Bendtsen et al. (2019): In this study, the toxicity of particles collected in a commercial and a non-commercial airport were evaluated in vivo by intratracheal instillation in

mice (see section 2.3 for occupational exposure measurements). Adult female C57BL/6 mice were exposed to 6, 18, and 54 µg particles/mouse dispersed in Nanopure water by sonication. The exposure doses were calculated on the basis of worst case scenario: of the maximum exposure level measured at the non-commercial airport of 1086 µg/m<sup>3</sup> at the peak event of plane departure, 9.6% were estimated to deposit in the alveolar lung regions. This was adjusted to the volume of a mouse lung and to 8 h of work, estimating exposure of 4, 12, and 39 days of work, respectively. Control mice were exposed to Nanopure water, and positive controls were carbon black Printex90 nanoparticles and SRM2975 diesel particles. Exposed mice were euthanized on day 1, 28, and 90 post-exposure. Inflammation was measured as inflammatory cell influx in bronchoalveolar lavage fluid as well as by the acute-phase response marker *serum amyloid A (Saa)* in lung (mRNA), liver (mRNA) and blood (protein). Genotoxicity was assessed by the comet assay on lung and liver tissue and cells from the bronchoalveolar lavage fluid. Analysis of the particles by scanning and transmission electron microscopy showed small primary particles and agglomerates of soot, which appeared uniform for non-commercial airport particles (mainly from jet engine emissions) and more heterogeneous for the commercial airport particles (emissions from aircraft, ocean, traffic and background). Pulmonary exposure to particles from both airports induced genotoxicity and dose-dependent acute phase response, and inflammation at same levels as standard diesel exhaust particles and carbon black nanoparticles [36].

He et al. 2020: In this study, UFPs from aviation or road traffic emissions were collected near the major international airport, Amsterdam-Schiphol airport (AMS), along with UFPs from an aircraft turbine engine at low and full thrust. The toxicity of the particles was tested in human bronchial epithelial cells (Calu-3) combined with an air-liquid interface (ALI) system with exposure to UFPs at low doses from 0.09 to 2.07 µg/cm<sup>2</sup>. Cell viability, cytotoxicity and IL-6 and -8 secretion were assessed after 24 h exposure. Cell viability was < 80% for all doses. LDH release as measure of cytotoxicity was observed at the highest exposure dose around 1.5 µg/cm<sup>2</sup> together with increased production of IL-6 and IL-8 compared to control exposure (blank filter extraction or re-suspension solution). It was concluded that airport and road traffic UFP as well as UFP samples from the turbine engine had similar inflammatory properties [109].

### Summary of health effect studies

Increased levels of metabolites in urine as biomarkers of internal exposure to jet fuel [105] were reported in biomonitoring studies of occupational exposure to airport emissions. Exposure to airport emissions was associated with increased levels of biomarkers of genotoxicity, in

terms of increased levels of SCE [52, 100, 105] and DNA strand breaks in the Comet assay [52], which indicates exposure to genotoxic and potential carcinogenic agents in the emissions. In turn, there were occupational studies reporting increased levels of self-reported respiratory complaints [101–103].

We identified a limited number of studies and one report reporting correlations between airport emission levels and health effects of residents in the vicinity of airports: Aircraft emission levels were associated with increased hospitalization for asthma, respiratory, and heart conditions especially in susceptible subgroups such as children below 5 years of age, elderly above 65 years of age [66, 94] and lower socioeconomic groups [97, 112]. A Dutch report on Schiphol similarly reported that school children and adults took more medication and had more respiratory complaint on days with increased exposure to aircraft emissions and concludes that health effects of air traffic emissions are similar to those caused by road traffic [59]. A biomonitoring study showed increased blood levels of the inflammatory marker IL-6 in volunteers with mild to moderate asthma after a walk in a zone with high levels of aircraft emissions [107]. It is well-known that other types of air pollution including diesel exhaust cause morbidity and mortality [113]. Taken together, these results suggest that the exposure to aircraft emissions induce pulmonary and systemic inflammation, which potentially contributes to cancer, asthma, respiratory and coronary heart disease.

Five mechanistic studies on the toxicity of airport particles were identified, one animal study in mice and four cell studies: Airport particles were reported to act as adjuvants in the activation of inflammatory cells or pathways [110] and induce pro-inflammatory cytokines [111]. Airport particles were shown to have similar inflammatory potency and similar ability to induce DNA damage as traffic emission particles [50], such as diesel exhaust particles [36]. In turn, airport particles induced significant levels of the biomarker Saa following intratracheal instillation in mice, associated with risk of cardiovascular disease [36], and they have the potential to generate ROS at similar levels as traffic emission particles [49, 50]. Thus, the conclusions from these in vitro and in vivo studies support the overall concern addressed in previous sections that airport emission particles are capable of inducing toxic responses comparable to the responses observed for other air pollution particles such as diesel exhaust particles.

## Discussion

Although a range of kerosene-based aircraft fuel types are in use, they are overall similar in chemical composition [24, 29]. Kerosene lies between the distilled crude oil fractions of gasoline (gasoline combustion exhaust,

IARC group 2b) and diesel (diesel combustion exhaust, IARC group 1) and the carcinogenic potential of jet fuel combustion products could be anticipated given the reported similarities to diesel exhaust particles. We highlight two important reported characteristics of airport particles:

- The majority of non-volatile airport emission particles are carbonaceous and aircraft engines emit large amounts of nanoparticles, which are dominated by very small particles of < 20 nm, which form aggregates/agglomerates in ambient air
- Particle numbers near airports are significantly higher than away from airports and jet engines are a significant source of UFP in ambient air. The highest concentrations of UFP are measured downwind of aircraft

The reported PAH levels [52, 83, 84] were all below the current Danish occupational exposure limit of 200  $\mu\text{g}/\text{m}^3$ . One study reported BC levels at the apron of 3.78  $\mu\text{g}/\text{m}^3$  and particle levels was overall reported to be between  $\sim 10^3$  and  $10^8$  particles/ $\text{cm}^3$  for exposed airport personnel (Table 1). The new exposure limit for diesel exhaust particles in EU is defined by the elemental carbon (EC) level and is 50  $\mu\text{g}$  EC/ $\text{m}^3$  [114]. The Netherlands recently endorsed an OEL for diesel exhaust particles at 0.01 mg/ $\text{m}^3$  measured as respirable EC. This was based on socioeconomic considerations and the Dutch prohibition risk level (OEL) is at 1.03  $\mu\text{g}$  EC/ $\text{m}^3$  [115], a level corresponding to 4 extra death cases of lung cancer per 1000 exposed, for 40 years of occupational exposure. Thus, the reported BC level [41] are well below the new EU OEL for diesel exhaust as well as the Dutch OEL, but exceed the Dutch prohibition risk level. Recently published data on the dose-response relationship between exposure to diesel exhaust particles and lung cancer in epidemiological studies estimated that occupational exposure to 1  $\mu\text{g}/\text{m}^3$  EC would cause 4 to 17 excess lung cancer cases per 10,000 exposed [80, 81].

The particle exposure levels can be compared to nanoparticle reference values used in The Netherlands, Germany and Finland as a provisional substitute when nano-specific OELs or DNELs for engineered nanoparticles are not available [116]. For low density insoluble nanomaterials such as carbon-based nanoparticles, the reference value is 40,000 particles/ $\text{cm}^3$ . Compared to this reference value for engineered nanoparticles, the reported occupational exposure levels are high for some job groups.

Significant variations in emission levels are observed between airports, depending on factors such as size, type, location, and wind direction. However, the closer to the source of emissions, the higher the exposure. Proximity to



exposure peak events such as landing and take-off is also an important determinant of high exposure. This is evident from the combined literature of occupational exposure measurements and ambient air measurements in residential areas around airports. As such, the highest levels of occupational exposure is found for airport personnel working at the apron, in close proximity to running jet engines. Airport personnel can likely be grouped in low (office staff/landside jobs with indoor work, far away from emission sources), medium (catering/cleaning/landside security staff with intermittent outdoor work) and high (baggage handlers/aircraft mechanics, crew chiefs) exposure groups [52, 86–88, 92, 98, 100–102]. To reduce occupational exposure, emission sources can be moved, the distance to emission sources can be increased, time spent in proximity to emission sources can be reduced and personal protection equipment can be used during peak exposures. Personal exposure may be higher than measured by stationary monitors, and thus, routine monitoring of personal exposure levels could be suggested.

Workplace experts, airport leaders and personnel groups have the necessary intrinsic knowledge and experience to suggest feasible, realistic options for reducing the exposure for specific job functions at individual airports.

The similarity of airport emission particles with diesel exhaust particles and pure carbon nanoparticles, with respect to physico-chemical properties as well as specific toxicological parameters was demonstrated in the animal study from our laboratory [36], and a growing number of studies report similar toxicity and health effects of emissions from airports and traffic. Airport emission particles likely have similar physico-chemical properties as diesel exhaust particles even though the primary particle size of jet engine emissions is somewhat smaller than the primary size of diesel exhaust particles. Diesel exhaust is classified as carcinogenic to humans by IARC [69], cause lung cancer, systemic inflammation, and inflammatory responses in the airways [70].

Aircraft emissions are associated with biomarkers of exposure, biomarkers of disease and health outcomes both for exposed workers [36, 41, 52, 83, 84, 86–90, 92, 100–103, 105] and for the general population living down-wind of airports [59, 66, 94–97, 107, 112]. Occupational exposure to aircraft emissions were associated with:

- Biomarkers of exposure to jet fuel emissions
- Biomarkers of genotoxic exposure
- Self-reported respiratory distress

The reported adverse effects correlate with effects demonstrated in animal studies and in *in vitro* studies, where aircraft emission particles caused inflammation [50, 110, 111], acute phase response [36], reactive

oxygen species [49, 50] and DNA damage [36], which are biomarkers of risk of cancer, cardiovascular disease and respiratory disease. This supports the notion of a causal relationship between exposure to airport emissions and the observed health effects. Although mechanistic studies on airport emissions are scarce, knowledge from other closely related scientific areas still applies, such as particle toxicity, carcinogenicity/toxicity of VOCs and OPEs and epidemiological studies of health effects caused by air pollution [117].

Another relevant concern to raise in this context is the adverse health effects of low-level chronic occupational exposure to these chemicals, which is difficult to study [118]. OPEs have been associated with adverse health effects reported from cabin crew and pilots after occupational exposure to bleed air and fume events during flights, with symptoms of respiratory illness and neurological effects [119]. The dominant OPE used in lubrication oil is tricresyl phosphate (TCP), which are among the highly neurotoxic OPEs [120]. It has been suggested that brain exposure may occur via inhalation of circulating small jet particles associated with OPEs, crossing the blood-brain barrier [121] – neurotoxic effects of OPEs may also be an understudied occupational risk of apron staff.

It has been shown that air pollutants worsen pre-existing diseases, such as allergy or other inflammatory (airway) or cardiovascular conditions [2–4, 122–124]. One example is a study examining the relationship between personal exposure to traffic emissions and acute respiratory health in school children with asthma residing in the Bronx, New York, which have the highest asthma incidence in New York City and state [125]. Personal samples of PM<sub>2.5</sub>, including the EC fraction, were collected 24 h daily for 40 school children with asthma from four schools, with spirometry and symptoms assessed several times daily. The study found increased relative risks of different airway symptoms, such as wheeze (RR = 1.45, CI: 1.03–2.04), shortness of breath (RR = 1.41, CI: 1.01–1.99), with relative risk of total symptoms of 1.30 (CI: 1.04–1.62). Interestingly, the symptoms were associated with increase in average 2-day school site and personal EC levels, but not mass of PM<sub>2.5</sub> [125]. As such, as demonstrated in asthmatic volunteers, residents living near airports, and supported by inflammatory effects shown in available *in vitro* studies, airport UFP and associated pollutants are, in addition to their direct adverse effects, likely to have the ability of worsen pre-existing disease.

## Conclusion

The reported adverse health effects of jet engine emissions are similar to those caused by exposure to diesel exhaust and air pollution. However, given the lack of

consensus on optimal measurement methods, equipment and quality control for near- and far field airport emissions and human risk assessments markers, more studies of exposure and of toxicological mechanisms are necessary.

These drawbacks are summarized efficiently by Lighty et al. in their paper on combustion compounds and health: *“There is a need for better integration of the combustion, air pollution control, atmospheric chemistry, and inhalation health research communities. Epidemiology has demonstrated that susceptible individuals are being harmed by ambient PM. Particle surface area, number of ultrafine particles, bioavailable transition metals, polycyclic aromatic hydrocarbons (PAH), and other particle-bound organic compounds are suspected to be more important than particle mass in determining the effects of air pollution. Time- and size-resolved PM measurements are needed for testing mechanistic toxicological hypotheses, for characterizing the relationship between combustion operating conditions and transient emissions, and for source apportionment studies to develop air quality plans”* [24].

Based on the accumulated knowledge so far, measures to reduce occupational exposure and emission levels at airports should be increased.

## Supplementary Information

The online version contains supplementary material available at <https://doi.org/10.1186/s12940-020-00690-y>.

### Additional file 1.

## Authors' contributions

Conceptualization, Methodology, Data Curation, and Writing – Original Draft and Review and Editing: KMB; Conceptualization and Methodology: UBV, Data Curation (Systematic Database Search): EB; Writing – Review and Editing: ATS and UBV. The authors read and approved the final manuscript.

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## Availability of data and materials

Data sharing not applicable to this article as no datasets were generated or analyzed during the current study.

## Ethics approval and consent to participate

Not applicable.

## Consent for publication

Not applicable.

## Competing interests

The authors declare that they have no competing interests.

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## Health-Related Quality of Life is Impacted by Proximity to an Airport in Noise-Sensitive People

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### Abstract

#### Introduction:

The aim of the study was to determine whether those who are noise sensitive are more adversely affected by airport noise than those who are not noise sensitive.

#### Participants and Methods:

One area was very close to Wellington International Airport and the other was distant from the airport and any other major sources of noise such as motorways and railways. Noise sensitivity was self-rated on a three-point scale as follows: non-noise sensitive, moderately noise sensitive, or highly noise sensitive. Statistical analysis consisted of analyses of variance using the domains of the WHOQOL score with the year, area (airport or the control), and noise sensitivity as covariates.

#### Results:

Noise-sensitive people were found to have a significantly poorer HRQOL than others when they lived near an airport, but not when they lived in the control area. The same effect was present at both of the time points investigated, suggesting that it is a general finding.

#### Discussion:

This finding is consistent with similar studies using the WHOQOL-BREF for investigating noise from road traffic, suggesting consistency in effect across transport noise sources.

**Keywords:** Aircraft, annoyance, noise, questionnaire, sensitivity, WHOQOL

### INTRODUCTION

Aviation noise contains substantial low-frequency components.[1] This is known to induce annoyance that poses acoustic measurement challenges, thus undermining current approaches to noise control and public health research.[2] Consistent with the “mode of transport effect,”[3] aviation noise is evaluated as being more annoying than both road traffic and rail noise.[4] Meta-analyses of multiple European airport studies suggest a prevalence of severe annoyance from aircraft noise at levels between 60 and 65 Ldn or between 17 and 25%.[4] A New Zealand study reported a similar prevalence of 17%.[5] The World Health Organisation (WHO) rates an outdoor noise of 55 Ldn or more as “seriously annoying.”[6]

Recent research conducted in New Zealand suggests that significant noise around people’s dwellings lead to a reduction in the health-related quality of life (HRQOL).[5,7,8] These studies showed that for residents the level of annoyance from either road traffic or air traffic correlated with HRQOL as measured by the World Health Organisation’s Quality of Life instrument (WHOQOL).[9] These findings are consistent with other reports that environmental noise, especially noise from transport, may be detrimental to health.[10,11,12]

Noise sensitivity is a personality trait that predicts noise annoyance.[10,11,12] The key characteristics of noise-sensitive individuals are that they are more likely to attend to sound and evaluate it negatively (e.g., find it threatening or annoying), and they have stronger emotional reactions to noise, and, as a consequence, have greater difficulty habituating to noise.[13] Noise sensitivity has a large impact on noise annoyance ratings, lowering annoyance thresholds by up to 10 dB.[10] A criticism of using noise sensitivity as a measure is that it may really reflect a greater tendency to poor health or vulnerability.[14] Under this hypothesis, people experiencing higher levels of noise sensitivity would be expected to experience worse health, irrespective of their degree of noise exposure.

In a previous study,[8] we used a natural experiment to address this criticism by showing that noise-sensitive people only had poorer self-reported health if they lived in a noisy environment, in this case, near a motorway. If they lived in quieter (but socioeconomically matched) areas, distant from major noise sources such as large roads, airports, trains, or industry, noise-sensitive people did not differ in terms of their self-reported health compared with non-noise-sensitive people.

This study addresses the following two other areas of doubt: that the findings are specific to motorway noise and that the findings are temporary and may not be sustained over time. Both of these are addressed in this study by considering data, based on the methodology used previously,[8] collected in the same two areas (one close to an international airport area and the other in a quiet area), and twice over a 3-year period.

## PARTICIPANTS AND METHODS

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### Participants

Data for this study were collected in Wellington city, New Zealand in 2012 and 2015. Questionnaires were delivered to the mailboxes of residents living within 250 meters of Wellington airport and within the 65 dB Ldn contour (Airport Group) or living in a socioeconomically matched Wellington suburb (Non-airport Group), which was not near the airport or aircraft flight paths or close to any other significant source of noise. Socioeconomic matching was performed using data from the New Zealand Deprivation Index, which assesses socioeconomic status based on car and telephone access, the receipt of means-tested benefits, unemployment, household income, sole parenting, educational qualifications, home ownership, and home living space.[15] Residents over the age of 18 years were invited to participate by completing the questionnaires anonymously and returning them using a postage-paid envelope that we gave them.

The study was approved by the Auckland University of Technology Ethics Committee (AUTEC Reference number 12/256).



Wellington's airport is built close to residential areas [Figure 1]. The number of flights at the airport did not change much between 2012 (90,000) and 2015 (93,000).[16]

## Instrument

The questionnaire was entitled "Wellbeing and Neighbourhood Survey" and was designed to disguise the true intent of the study, with residents invited to participate in research investigating their place of living and their well-being. The survey contained 58 items categorized as HRQOL (26 items), amenity (two items), neighborhood issues (14 items), environmental annoyances (seven items), demographic information (eight items), and noise sensitivity (one item), which were presented in this order.

To measure health, we employed the short form of the WHO's HRQOL (WHOQOL) scale, called the WHOQOL-BREF, which adheres to the WHO's definition of health as "a state of complete physical, mental and social wellbeing and not merely the absence of disease or infirmity." The WHOQOL adopts a multidimensional profile of HRQOL, dividing it into the following four domains: physical health (seven items), psychological well-being (six items), social relationships (three items), and environmental factors (eight items). Two additional items assess the overall quality of life and self-rated health. Each item was scored on a five-point scale, where a low score corresponded to a negative assessment of that aspect of life and a high score corresponded to a positive assessment. Example questions include the following: "Do you have enough energy for everyday life?" (physical), "How often do you have negative feelings such as blue mood, despair, anxiety or depression?" (psychological), "How satisfied are you with the support you get from your friends?" (social), and "How satisfied are you with the conditions of your living space?" (environmental).

Amenity and neighborhood problem items were included primarily to "camouflage" our interest in noise exposures and were not used in this study's analyses. We asked the respondents how much they agreed with the following two statements: "I am satisfied with my neighbourhood/living environment," and "My neighbourhood/living environment makes it difficult for me to relax at home." The neighborhood problem scale consisted of 14 items.

Of the seven items enquiring about annoyance, four asked about air quality, while three asked about annoyance because of aircraft, neighbors, or other sources of noise. The annoyance to noise items were based on recommendations issued by the International Commission on the Biological Effects of Noise[19] and in our own previous research.[7,9,10] Respondents were asked to consider the last 6 months and how annoyed they had been by noise from traffic, neighbors, and "other" sources. They were asked to respond to each item on a five-point scale from 1 (not annoyed at all) to 5 (extremely annoyed).

Noise sensitivity was assessed using a three-point scale, wherein each participant was asked to rate himself or herself as "not noise sensitive," "moderately noise sensitive," or "very noise sensitive." This question was placed near the end of the questionnaire form among the demographic questions.

Demographic information was also collected, which consisted of information on gender, ethnicity, age, the highest level of education completed, current employment status, and whether the respondent was currently ill or had a known medical condition.

## Procedure

Two surveys and a prepaid, return-addressed envelope were deposited into the letterboxes of eligible houses. The participants were asked to complete the surveys independently at a convenient time, to think about their life in the last 2 weeks, and circle the number on the scale that best reflected their answer to each question. After the completion of the survey, the participants were instructed to return the survey/s in the prepaid envelope provided. No incentives to participate were offered.

## Statistical analysis



Five separate analyses of variance were conducted for the overall WHOQOL score and for each of the four WHOQOL domains (physical, psychological, social, and environmental). Year (2012 and 2015), area (airport and non-airport), and noise sensitivity (not, moderate, and very) were modeled as the factors, with WHOQOL scores as the dependent variables. Evidence for a differential effect of noise sensitivity on health for different noise environments would be a significant two-way interaction between noise sensitivity and the area (airport or non-airport). Evidence for a change in this relationship over time would be a significant three-way interaction among the year, the area, and noise sensitivity.

On the basis of preliminary analyses using chi-squared tests, [Table 1](#) shows disparities between the areas in education completed (both years) and current illness (2015). All analyses were conducted controlling statistically for these factors.

## RESULTS

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The two areas were closely matched demographically, except that the members of the Airport Group tended to be less well educated in both 2012 and 2015 than in the Non-airport Group, and the Airport Group was more likely to have current illness or a medical condition in 2015 compared with 2012 [[Table 1](#)]. There was no difference in the noise sensitivity profile of the two groups in either year [[Table 1](#)].

There was a two-way (area by noise sensitivity) interaction ( $F(2, 353) = 4.06, P = 0.018$ ), suggesting that noise sensitivity had a differential effect on WHOQOL score depending on the area of residence [[Figure 2](#)]. This shows that noise sensitivity was not associated with WHOQOL score in people living in the non-airport area, whereas for those living near the airport, greater noise sensitivity was associated with lower WHOQOL scores. There was no three-way (area by year by noise sensitivity) interaction ( $F(2, 342) = 1.16, P = 0.314$ ), suggesting that the effect did not change over time [[Figure 3](#)].

The four WHOQOL domains (physical, psychological, social, and environmental) were analyzed separately and using the same approach as for the overall WHOQOL score. In no case was there a three-way (area by year by noise sensitivity) interaction (all  $P > 0.25$ ), implying that the pattern of the effects of noise sensitivity by area remained the same across the years. There was a two-way (area by noise sensitivity) interaction for physical ( $F(2, 338) = 3.30, P = 0.038$ ) and social ( $F(2, 341) = 3.67, P = 0.027$ ) domains, marginally for psychological ( $F(2, 338) = 2.35, P = 0.097$ ) domain, and no interaction for environmental ( $F(2, 338) = 1.62, P = 0.199$ ) domain. All of the two-way interactions are displayed in [Figure 4](#).

## DISCUSSION

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Several findings reported in this study indicate that noise sensitivity influences the relationship between aviation noise and health. These effects can be explained by three competing hypotheses. Hypothesis 1 is that noise sensitivity is a genuine risk factor and as such mediates the relationship between noise and health.[\[7\]](#) Hypothesis 2 is that noise-sensitive individuals simply have a propensity to report poor health without necessarily experiencing it, and thus personality factors (i.e., negative affect) account for the link between noise and health.[\[17,18\]](#) The third hypothesis is that noise sensitivity may reflect a vulnerability to illness in general, such that sensitivity to noise is merely a symptom of multiple other conditions and, therefore, would be expected to act as a moderator of the noise and health relationship as we have described previously.[\[8\]](#) These hypotheses will now be explored in the light of the main findings.

The main finding was an interaction between the area of dwelling (airport or non-airport) and noise sensitivity grouping [[Figure 2](#)]. This shows that people who are noise sensitive but do not live near noise sources have similar health to people who are not noise sensitive. However, among those dwelling near the airport, greater noise sensitivity was associated with poorer health. This finding is best explained by conceptualizing noise sensitivity as a moderator (Hypothesis 1), a notion that has been supported by previous research performed near a New Zealand airport.[\[5\]](#) If noise sensitivity simply reflected a

tendency to complain due to personality factors such as negative affect (Hypothesis 2), then equivalent mean WHOQOL scores would be expected for the noise-sensitive groups in both the airport and non-airport areas. This would also be the case if the self-report health ratings were driven by vulnerability to health problems (Hypothesis 3). Similarly, earlier findings[8] suggested that noise-sensitive people had poorer self-reported health if they lived near a motorway, but not in quieter locations, and the results of this study suggest that the same applies to aviation noise.

A key finding is that, for the non-airport sample, there are no differences in mean WHOQOL scores across the three noise sensitivity categories. This finding is also mirrored when decomposing the WHOQOL into its four constituent domains [Figure 4]. This finding speaks against the hypothesis that noise sensitivity is simply a marker for other disorders, disabilities, or disease processes, because higher noise sensitivity would be expected to be linked with lower self-reported health. This finding is also inconsistent with the negative affect hypothesis of noise sensitivity, which would predict that self-reported health would decrease as noise sensitivity increased, even in areas with little noise. However, these results can be explained if noise sensitivity is considered a moderator of noise-exposure-related health effects. Pertinently, in the absence of major noise sources, health integrity is equivalent across noise sensitivity categories.

The consistency of the data across time is reasonable, with the results showing no detectable difference in the effect across two periods of observation separated by 3 years. Our findings showing significant differences in HRQOL between those residing in the proximity of a major airport versus those in a matched area support previous research undertaken in Australia.[19] Accounting for important confounding variables and using the short-form health survey (SF-36) as a measure of self-reported health, Black and Black[19] reported that mean physical functioning, general health, vitality, and mental health scores in a group exposed to aviation noise were significantly lower than a matched control group. Such a finding would be expected if aviation noise interfered with human biological processes such as sleep or cardiac function, and such impacts are now generally accepted.[11,12] Our data support the findings of Black and Black and reinforce the case that noise exposure impacts HRQOL consistently over time.

The findings also suggest that noise sensitivity could be the prevailing risk factor for noise-related health effects, and we would argue that any measure of the impact of noise on people should be taken alongside a measure of noise sensitivity because it is the interaction between the exposure (detectable sound) and the trait (noise sensitivity) that gives rise to the health effect seen here. Because the effects observed are interactions, and, therefore, have complexity, apparently simple measures will be misleading. For example, a slight health effect of noise measured over a sample would actually represent a combination of large effects in noise-sensitive people and the lack of effects in non-noise-sensitive people.

We took great care over our socioeconomic status (SES) matching: noise sensitivity may potentially occur with a number of psychological conditions.[e.g., 20,21,22] Individuals with mental illnesses often dwell in low SES or high deprivation neighborhoods, this being the central tenet of the so-called “drift” hypothesis.[23] By demographically matching our airport and non-airport areas, we selected areas that could meaningfully be compared. Our data suggest that noise-sensitive individuals may be putting their health at risk by living in areas containing noise. As in our previous research,[8] the proportion of noise-sensitive and non-noise-sensitive people in each of the areas was very similar. This appears surprising; why would a person who reports himself or herself to be highly sensitive to noise live in a noisy area? We suspect that the cultural norms around noise sensitivity may govern this; because noise is regarded as acceptable and unavoidable by New Zealand society at large, those who are noise sensitive may feel that the annoyance and health effects that they experience are not sufficiently important to raise when making a decision about the choice of dwelling, particularly given the numerous other factors that also need to be considered when making such a decision. Qualitative research investigating this is called for, as are biomedical studies designed to reveal the neuropsychological underpinnings of noise sensitivity.[24]

One limitation of this research, and a possible reason for some of the weaker effects, is that the people in the non-airport area sometimes complain of noise too. The effects may have been clearer had the control area been truly quiet; however, the use of a real-world control with matched socioeconomic status allowed a fair comparison that showed the impact of noise from airplanes over and above the other forms of noise. Another limitation was the difference in the educational and health status of the two groups. Despite careful matching on the basis of the socioeconomic status of each area, the respondents differed somewhat on these demographic indicators. Nonetheless, statistical control could be applied, and findings were, therefore, not contaminated by these sampling differences.

In summary, noise-sensitive people who are exposed to noise from aircraft have poorer self-reported health than non-noise-sensitive people with the same exposure, and noise-sensitive people who are not so exposed. We have replicated our previous findings relating to noise exposure from motorways in a different (airport) setting, and have also demonstrated that the effect was present in the same geographical areas when measured at time points separated by 3 years.

### Financial support and sponsorship

Nil.

### Conflicts of interest

There are no conflicts of interest.

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## Figures and Tables

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**Figure 1**

Wellington International Airport runway and nearby dwellings



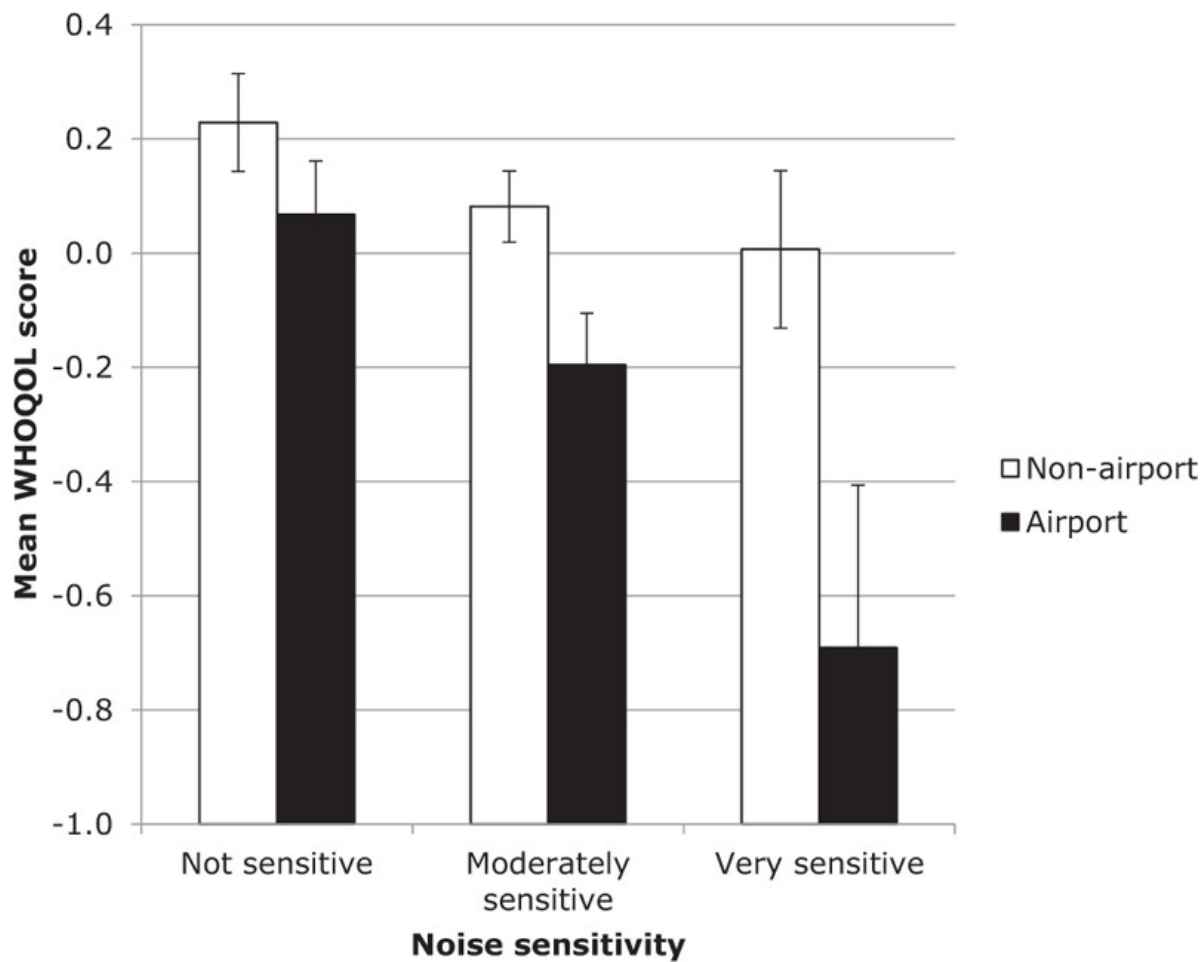
**Table 1**

Group demographics by year

Variables	2012		$\chi^2$ -test sig.	2015		$\chi^2$ -test sig.
	Airport ( <i>n</i> = 87)	Non-airport ( <i>n</i> = 91)		Airport ( <i>n</i> = 86)	Non-airport ( <i>n</i> = 103)	
	<i>N</i> * (%)	<i>N</i> * (%)		<i>N</i> * (%)	<i>N</i> * (%)	
Sex			NS			NS
Male	28 (33)	29 (32)		31 (38)	34 (33)	
Female	58 (67)	62 (68)		51 (62)	67 (69)	
Age groups (years)			NS			NS
18–20	3 (4)	2 (2)		2 (2)	3 (3)	
21–30	7 (8)	8 (9)		5 (6)	6 (6)	
31–40	16 (19)	18 (20)		15 (18)	22 (21)	
41–50	16 (19)	19 (21)		13 (15)	28 (27)	
51–60	14 (16)	20 (22)		22 (26)	19 (18)	
61–70	16 (19)	16 (18)		15 (18)	17 (17)	
70+	14 (16)	7 (8)		13 (15)	8 (8)	
Education (completed)			<i>P</i> < 0.05			<i>P</i> < 0.05
Secondary school	29 (36)	18 (20)		42 (49)	16 (16)	
Technical college	20 (25)	16 (17)		12 (14)	9 (9)	
University degree	32 (40)	57 (63)		31 (37)	77 (76)	
Employment status			NS			NS
Full time	38 (44)	42 (47)		47 (55)	53 (52)	
Part time	20 (23)	23 (26)		6 (7)	18 (18)	
Retired	17 (20)	8 (9)		20 (23)	17 (17)	
Student	5 (6)	8 (9)		2 (2)	5 (5)	

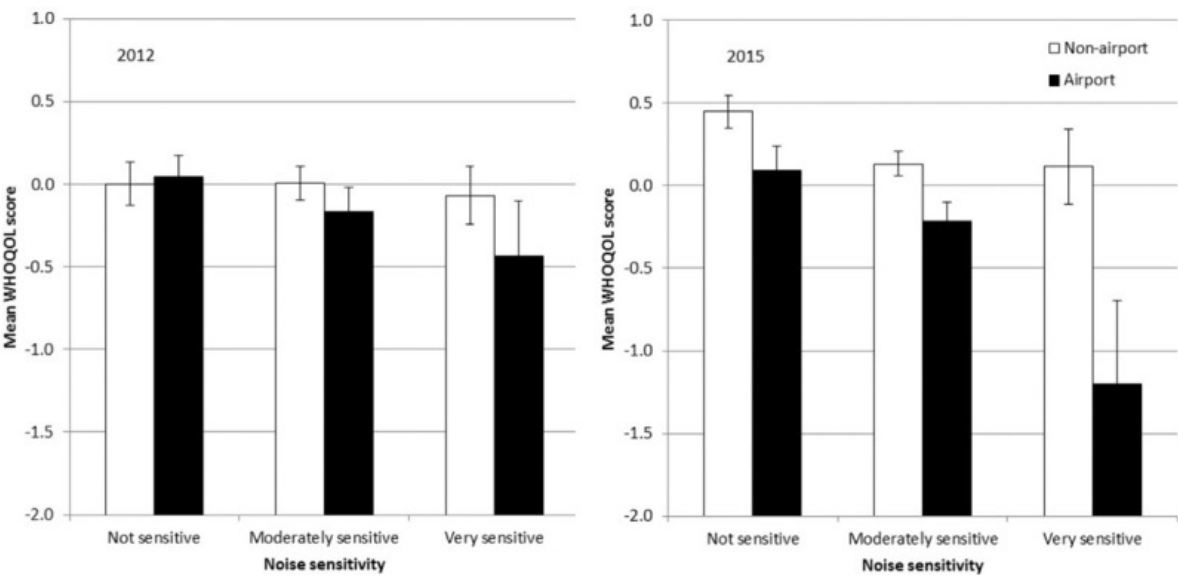
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NS = not statistically significant. Pearson's  $\chi^2$  was used to test disparities (criterion  $\alpha = 0.05$ ). \*Totals may differ due to missing data.

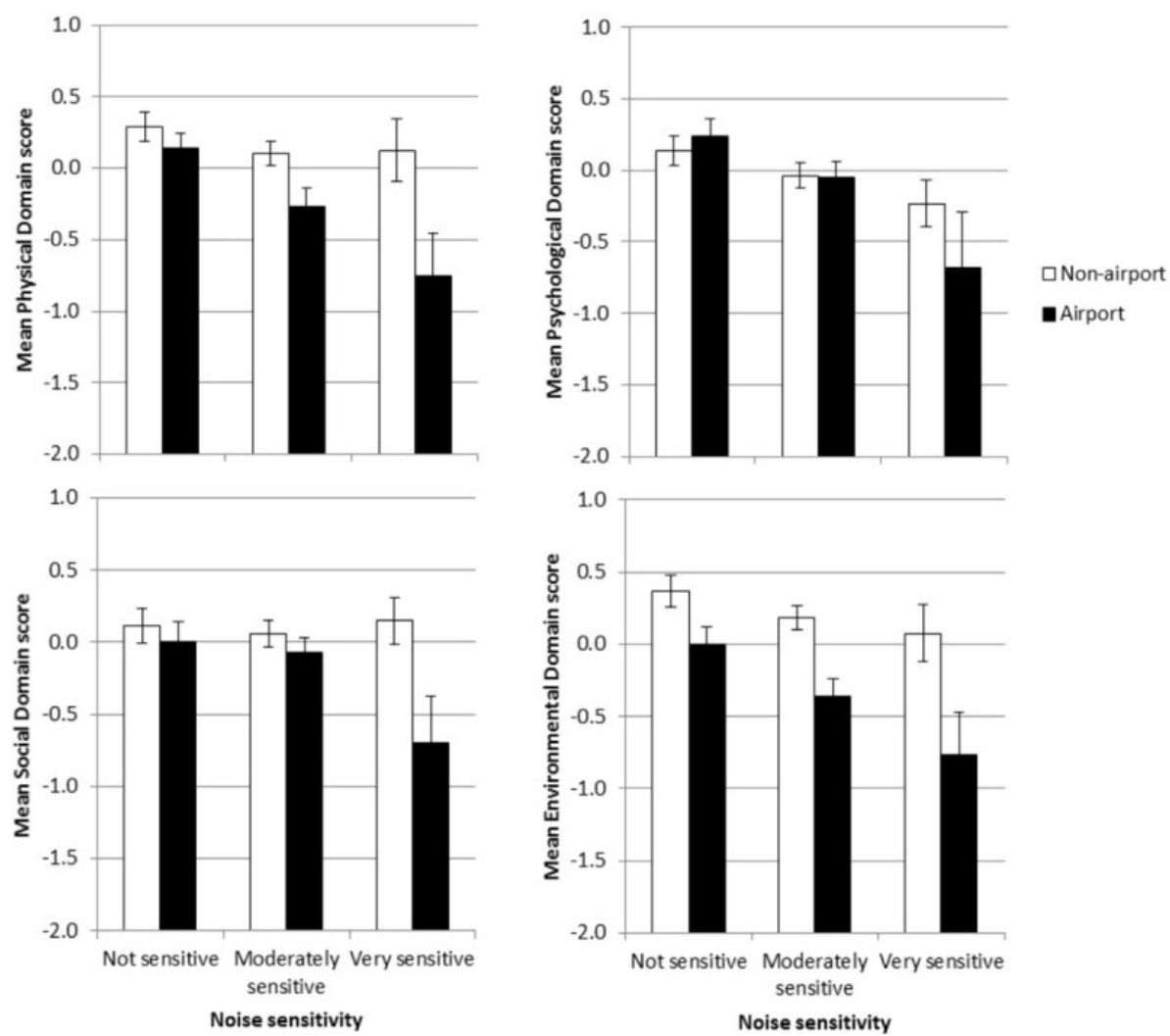
**Figure 2**

Mean WHOQOL score for each noise sensitivity group and in each area of residence across the years 2012 and 2015. Error bars represent one standard error of the mean

Figure 3



Mean WHOQOL score by noise sensitivity and in each area of residence presented separately for each of the 2 years. Error bars represent one standard error of the mean

**Figure 4**

[Open in a separate window](#)

WHOQOL domain (physical, psychological, social, and environmental) score for each noise-sensitivity group and in each area of residence. Error bars represent one standard error of the mean

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