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November 9, 2019

Mr. Shannon Hatcher, Air Pollution Specialist
California Air Resources Board
1001 I Street
P.O. Box 2815
Sacramento, CA 95812

Ms. Kate Gordon, Director
Office of Planning and Research
1400 10th Street
Sacramento, CA 95814

Re: Inglewood Basketball and Entertainment Center Project – Response to GHG Emissions Commitment Letter (Clearing House Tracking No. 2018021056)

Dear Mr. Hatcher and Ms. Gordon:

We are writing on behalf of MSG Forum, LLC in response to the Clippers' "GHG Emissions Commitment Letter" submitted on November 1, 2019.

The Clippers still fail to provide a meaningful local GHG reduction program to address the over 500,000 metric tons of GHGs that their project will generate. The Clippers have steadfastly refused, since their initial application, to do what they promised the legislature and the community: be net zero GHG, achieve 50% of the GHG reduction through local, direct measures, and achieve the substantial health co-benefits for the community.

ARB and others have extensively studied the effects of emissions of GHGs, criteria air pollutants, and toxic air contaminants, and the conclusions are clear.[1] GHG emissions reductions

[1] *See* ARB, Research on Health Effects of Air Pollution, available at https://ww2.arb.ca.gov/research/research-health-effects-air-pollution; *see also* Exhibit 1, ARB Health Website; Exhibit 2, ARB, Estimating the Health Benefits of Reductions in Emissions of PM2.5 or its Precursors; Exhibit 3, UCLA Center for Health Policy Research, Is Disparity in Asthma among Californians due to Higher Pollution Exposures, Greater Vulnerability, or Both?, February 24, 2012, prepared for ARB and the California Environmental Protection Agency; Exhibit 4, ARB, Status Of Research On Potential Mitigation Concepts To Reduce Exposure To Nearby Traffic Pollution, August 23, 2012; Exhibit 5, Suzanne Paulson, Identifying Urban Designs And Traffic Management Strategies For Southern California That Reduce Air Pollution Exposure, prepared for ARB, February 2017; and

provide significant health benefits. Local, direct GHG emissions reductions provide significant local health benefits.

The disproportionate impact of air pollution on Black and Hispanic communities also is well documented. A recent landmark study concluded in part that "in the United States, PM2.5 exposure is disproportionately caused by consumption of goods and services mainly by the non-Hispanic white majority, but disproportionately inhaled by black and Hispanic minorities."[2] These impacts and the benefits of local GHG reductions are why the legislature required 50% of the Clippers' reductions to be real and local. That is the letter of AB 987 and the spirit of AB 987.

The Clippers' methodology has been and remains fundamentally flawed. They still stand by their January and June applications and rely on the "market-shift" theory. MSG has already submitted comments and documentation evidencing that such a theory is wrong and sets a terrible precedent for California. Those submissions are incorporated herein by this reference.

The appropriate and agency-accepted methodology is simple. The Clippers estimate that the project's GHG emissions are 568,185 MT $CO_2e$.[3] It is well accepted that the Clippers can take credit for the 40,902 MT $CO_2e$ from permanently demolishing buildings on the arena site. AB 987 requires the Clippers to offset the difference (**527,283 MT $CO_2e$**) and achieve half of those reductions through local measures (**263,641 MT $CO_2e$**).

It is really that simple. This is math. It should not be political. In fact, it is what the Clippers committed to in the legislative process resulting in AB 987. Anything else is shortchanging the commitment made, increasing emissions on a community already heavily impacted by pollution.

Rather than simply providing ARB a plan that offsets their actual emissions, the Clippers now claim that, even if the existing venues "backfill," the existing event emissions are greater than the estimated future event emissions and the Clippers should get the benefit of that difference. In other words, the Clippers claim their baseline should be the existing emissions from events at other venues less the supposedly lower emissions from *future* events at those venues. As noted below, there is no support for this analysis or for many of the Clippers' other assertions.

ARB should reject any reliance on market-shifted events and the 100% backfill scenario. To do otherwise undermines ARB's efforts to address GHG emissions and health impacts on low-income communities.

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Exhibit 6, Union of Concerned Scientists, Inequitable Exposure to Air Pollution from Vehicles in California, February 2019.

[2] Exhibit 7, PNAS, Inequity in consumption of goods and services adds to racial–ethnic disparities in air pollution exposure, February 4, 2019.

[3] Our letter dated February 1, 2019 detailed why this figure very likely understates the project's overall emissions. The Clippers provide no analysis suggesting that our assessment is wrong.

The Inglewood community will experience an unprecedented cumulative increase in GHGs, toxic air contaminants, and criteria pollutants with this project and the adjacent Hollywood Park development. This "Los Angeles Stadium and Entertainment District," as the proponents have referred to Hollywood Park and the proposed Clippers arena, will introduce millions of new annual car trips to this community. The Hollywood Park development, at which the new stadium is under construction, is not required to provide GHG reductions. That is more than 1.5 million square feet of uses and 2,500 housing units without GHG reductions. Now the City and the Clippers propose to add thousands of more tons of additional air pollutants with no real, meaningful local reduction measures.

And as noted above, ARB has done study after study demonstrating the link between tailpipe emissions and health, particularly of low-income minority communities. The Clippers would do nothing in their "local reduction measures" to offset these GHG emissions or pollutants. Approval of the application will subject the community to health risks the law was written to avoid.

The following summarizes some of the key mistakes and flaws in the Clippers' November 1 submittal that could be identified in the 5 business days provided and which are discussed in more detail below.[4]

- The Clippers' new backfill scenario improperly credits the Clippers with baseline emissions from multiple venues that will continue to exist and operate, based on unsupported assumptions of reduced future emissions.

- As ARB is aware, buying renewable energy credits is <u>not</u> a local, direct reduction as provided for by AB 987 – it does not reduce energy consumption at the stadium or increase local renewable generation.

- The waste diversion measure (by far the largest claimed reduction) may reduce emissions at a landfill more than 30 miles away but is not the local, direct measure envisioned by AB 987.

- The Clippers inappropriately take credit for waste reductions that are already separately required. This double-counting violates AB 987 and belies the Clippers' own description of their "commitment."

- Recent reductions in exports of recycled materials and limitations on remanufacturing infrastructure seriously call into question the Clippers' waste diversion estimate.

[4] We were disappointed that additional time to respond to the Clippers' submission was not provided, given the substantial volume of new information the Clippers submitted.

- The "Smart Parking System" will encourage more patrons to drive to the arena. That is the exact opposite of the goal AB 987 sets forth and what ARB seeks to encourage with its smart growth programs.

- 10 electric city cars and 2 electric buses would never make a dent in the emissions from the nearly 4 million new vehicle trips per year that the Clippers arena will bring to Inglewood.

- Even with these and other errors and data gaps, the Clippers still fall far short of the 50% local reduction requirement.

- The Clippers' methodology is so speculative that even the Clippers admit it would require ARB to review verification reports for the life of the project.

Our request is simple. ARB should treat the Clippers like everyone else. ARB should apply the same methodology to the AB 987 application that it has for all other projects. Do not permit the Clippers to shirk their responsibility to provide local reductions. Do not overcomplicate what is a straightforward requirement.

Contrary to the Clippers' assertions, they are not unfairly being held to a higher standard. First, AB 987 has more stringent requirements than AB 900. That was the deal the Clippers bargained for and touted. Second, the Clippers' methodology is flatly inconsistent with other stadium/arena projects approved under AB 900.

We are also concerned with the Clippers' written representation in their submission to ARB that there is already a "joint agreement" between ARB and the Clippers. We have requested a copy of the "agreement" and have been told by ARB that no such written agreement exists. Any such agreement should be provided to the public for review and comment.

If ARB approves the Clippers' application, ARB's action on this application will be a written determination of compliance with ARB's regulations. It will set a precedent for other projects, from how GHG is to be calculated to what is a local, direct reduction measure. We urge ARB to enforce AB 987 and require the Clippers to follow the broadly applied agency methodology that does not include the theoretical market-shift and backfill theories that the Clippers invented in order to avoid a real local mitigation program and to require the Clippers to provide real local, direct mitigation measures.

The community should not be made to choose between healthy air quality and economic development.

## I. THE CLIPPERS' ACCOUNTING OF BACKFILLED EVENTS IS AS FLAWED AS THEIR "MARKET-SHIFT" THEORY

The Clippers complain about the length of time it has taken to certify its project under AB 987. They have no one to blame but themselves. This is now their third attempt to try to explain their convoluted GHG analysis.

The Clippers' first attempt underreported project emissions by about 120,000 MT CO2e.

The Clippers' second attempt required two "market studies" to try to justify their flawed assumptions. However, those market studies included express disclaimers stating that the information should not be relied upon. We implore you to heed the warnings from the authors of those reports.

The Clippers' third attempt to explain their GHG analysis adds yet more complexity and stacks guesses on top of guesses by predicting attendance at events 30 years into the future at venues over which they have no control. It is so speculative that it requires ARB to review verification reports for the life of the project. And for what? All of these efforts by the Clippers are designed to avoid implementing meaningful "local" measures to reduce GHG emissions and related toxic air emissions that will mitigate the impacts of their project. There is no scientific, legal or economic basis for that refusal. ARB should reject this approach and demand that the Clippers do what every other project is required to do.

As best we can understand it, the basic idea of the Clippers' new "100% backfill scenario," is (1) the baseline includes the "market-shifted" Clippers games and the alleged market-shifted events from Staples Center, the Forum, and the Honda Center and (2) the analysis then assumes that all of those dates will be backfilled with new events and the emissions associated with those events will be added to the emissions the Clippers are required to offset. In other words, the Clippers will not get "credit" for the emissions created by the backfilled events.

The Clippers claim that each year, 175 events will relocate from Staples Center, the Forum, and the Honda Center to their new arena. In its baseline, the Clippers take credit for the 2019 emissions levels associated with these events.

However, for the "additional emissions" for the "100% backfill scenario" the Clippers reduce the emissions associated with these events each year into the future, presumably because it is assumed that the events will emit fewer GHGs as technologies improve. ***But these are the same events.*** So, on one hand, the Clippers claim these events will emit 11,715 MT $CO_2e$ when it is useful to them to have large emissions. On the other hand, the Clippers claim these events will emit between 4,343 and 7,833 MT $CO_2e$ when it is useful to them to have smaller emissions. This creative accounting substantially reduces the amount of GHG emissions the Clippers need to offset.

Imagine two events in 2030. One is at the Clippers' new arena. The Clippers claim that this event has shifted from the Staples Center. The second event is the event that has "backfilled" the Staples Center. Under the Clippers current methodology, the emissions associated with the shifted event are counting towards the Clippers baseline. The "backfilled" event (*i.e.*, the one occurring at Staples Center) is counted towards "additional emissions" (at about 65% of the total the Clippers are taking credit for in the baseline). In fact, the emissions will be the same. However, the Clippers improperly eliminated 35% of the emissions that they need to offset.

The Clippers' analysis arbitrarily eliminates emissions that they need to offset. This effort should be rejected and the Clippers should be required to offset the true emissions under the 100% backfill scenario: 568,185 MT $CO_2e$ - 40,902 MT $CO_2e$ = **527,283 MT $CO_2e$**.

## II. THE CLIPPERS' "LOCAL" REDUCTIONS FALL FAR SHORT OF THE NUMBERS CLAIMED

The Clippers still claim that their TDM program will result in substantial GHG emission reductions. For all the reasons enumerated previously, we disagree. We strongly disagree with the Clippers' assumptions regarding the TDM's program's efficacy and urge ARB to reject the Clippers' conclusions. With their latest submittal, many of the emissions are not offset locally and the calculations largely overstate their emission reduction value.

### A. The Clippers' New Programs Are Not "Local"

The large majority of new local reductions come from two measures – renewable energy credits and waste reduction – neither of which are local reductions as contemplated by AB 987.

#### 1. A Contract to Purchase Renewable Energy Is Not a Local, Direct Reduction

Buying renewable energy credits is a paper transaction that may benefit global climate change but does not reduce energy consumption at the arena or increase local renewable generation. This is why AB 987 only considers "***onsite*** renewable energy generation, including a solar roof on the arena with a minimum peak generation capacity of 500 kilowatts"[5] to be an on-site reduction measure that can count towards the 50% local reduction requirement. The November 1 submittal provides scant details about the program other than that the Clippers will purchase electricity through their utility's "Green Rate" program. This is a renewable energy credit program that allows the buyer (the Clippers) to take "credit" for renewable generation produced somewhere else in California.[6] There is no evidence the solar power comes from the City of Inglewood or any guarantee that the utility will deliver any of the "credited" renewable energy to the site.[7] This is <u>not a local</u> program. This is an offsets purchase. To the extent the Clippers consider this program, it must fall under the offsets bucket and not in the local measures bucket.

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[5] Public Resources Code 21186.6.8(j)(3)(A)(iii) (emphasis added).

[6] See Southern California Edison Green Rate program, 2019 Prospective Product Content Label, available at: https://www.sce.com/sites/default/files/inline-files/SCE_Green_Rate_PPCL%202019%20%283%29.pdf

[7] For example, SCE's 2018 corporate sustainability report indicates that the electric power sector accounted for 16% of California's GHG emissions in 2016. Of this 16%, 6% was from electricity imported from outside California. Therefore, while GHG emissions reductions associated with the purchase of Green Power through SCE's Green Rates program may be considered as offset credits, they should not be considered as reductions from local, direct measures as the GHG emissions reductions may occur outside California.

### 2. The Waste Diversion Method Is Not a Local, Direct Reduction

The waste diversion measure (by far the largest reduction) is also not the type of local reduction called for by AB 987. AB 987 lists the types of reductions that are deemed "local, direct greenhouse emissions reduction measures." (See Pub. Resources Code, § 21168.6.8, subd. (j)(3).) Reducing the amount of waste that goes to landfills that are far away from Inglewood, is inconsistent with AB 987's letter and its spirit. That waste not going to a landfill, does not mean that it is not picked up and transported by polluting commercial trucks. Any co-benefits associated with a reduction in these GHG emissions will not be received in Inglewood. This also must fall under the offsets bucket and not in the local measures bucket.

## B. The Clippers New Programs Will Not Achieve the Claimed GHG Reductions

In addition to inappropriately counting certain measures as "local," there are numerous underlying issues associated with the Clippers' analysis of local reduction measures. Their analysis is replete with errors and data gaps and provides insufficient information to verify the validity of their calculation methodologies and assumptions.

### 1. The Waste Reduction Program's GHG Emission Predicted Reductions Are Overstated

AB 987 requires that the project comply with Public Resource Code Division 30, Chapter 12.9 (commencing with Section 42649.8).[8] This obligation, as well as the many others to divert landfill waste that already exist, certainly falls on the Clippers. Yet, the Clippers claim 100% of the GHG emission reductions for actions that they are already required to take. AB 987 anticipates and precludes this sort of double-counting with respect to design features needed to meet LEED gold certification. (Pub. Resources Code, § 21168.6.8, subd. (j)(3)(A)(A).) In these circumstances, ARB simply cannot allow the Clippers to take double credit for their so-called "Zero Waste Program."

Even if ARB credits any of the proposed waste program's GHG emission reduction, it should discount the Clippers' figures because the figures are overstated. The calculations in the Clippers' analysis suggest that the arena's on-site Waste Reduction and Diversion Program will reduce GHG emissions by 31,587 MT over the life of the project. These emissions reductions are calculated based on an assumed waste reduction / diversion rate of 80%-90%, but the Clippers do not detail what additional actions they will take beyond what AB 987 requires as a matter of law. The GHG emission benefits of the arena's on-site Waste Reduction and Diversion Program are overestimated if the starting calculations already assume existing waste diversion.

Moreover, it is not enough for the Clippers to document its on-site activities (which they have not done). The Clippers must prove that there is adequate infrastructure in the near term and long term to achieve the level of recycling and composting required by their program.

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[8] Among other requirements, the law provides that "On and after January 1, 2019, a business that generates four cubic yards or more of commercial solid waste,…, per week, shall arrange for recycling services specifically for organic waste." (Public Resource Code, § 42649.81(a)(3).)

Historically, California has exported as much as 60% of recyclables to China. China has announced a restriction on imports of 24 types of materials, including recyclables.[9] Thus, recycling facilities, composting facilities, and landfills will be strained by the need to take on an additional volume of recyclables which are not being exported. The Clippers must discuss and account for this limitation in their analysis.

CalRecycle estimates that California needs 44 million tons of remanufacturing infrastructure in-state to sustainably manage recyclables. As of September 2013, the existing remanufacturing infrastructure in California was handling only a little over two million tons and had minimal remaining capacity. Therefore, unless significant investments are made in remanufacturing infrastructure, it will be difficult for California to have sufficient capacity to sustainably manage its own waste.[10]

In short, not only do the Clippers not provide any evidence from an operational standpoint on how they will achieve a high waste reduction rate, the supporting infrastructure necessary to achieve these goals is not even in place. Therefore, ARB cannot accept these GHG reduction figures.

### 2. The "Smart Parking System" Program's GHG Emission Reductions Are Overstated

The Clippers' "Smart Parking System" measure is particularly specious. AB 987 requires the Clippers to reduce the number of vehicle trips. Yet, the Clippers are now touting as a GHG emission reduction measure their effort to make parking less time-consuming for their customers. Streamlining the parking and driving experience will only encourage patrons to drive more. That is the exact opposite of the goal AB 987 establishes and contrary to what ARB seeks to encourage through its smart growth programs.

The Clippers also must provide additional analysis and data supporting assumed garage utilization, average time-savings, and annual occurrence of events to avoid overestimated assumed benefits. No references or documentation have been provided to support these assumptions. We do not see how ARB can verify the submittal's conclusions.

### 3. The Electric Vehicle and Car Charging Program's GHG Emission Reductions Are Overstated

While the Clippers take credit for reductions in emissions from the purchase of a few electric vehicles and buses, they do not take into account the increased electricity demand from these vehicles in their emission calculations. While tailpipe emissions may be reduced, the

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[9] Resource Recycling, *How China fallout is shaking recycling in California*, January 30, 2018, available at: https://resource-recycling.com/recycling/2018/01/30/china-fallout-shaking-recycling-california/#more-8398, (last accessed October 2018).

[10] CalRecycle, AB341 White Paper – Recycling, Reuse, and Remanufacturing, September 17, 2013, available at: http://www.calrecycle.ca.gov/Actions/Document.ashx?id=3432, (last accessed October 2018).

electricity generated currently and for many years going forward is not expected to be 100% renewable.

The table below shows that the electricity emissions may be on the order of fve metric tons per year per bus, or 305 metric tons over 30 years for two buses, and 0.4 metric tons per year per vehicle, or 141 metric tons over 30 years for 10 vehicles. Collectively, this adds 445 MT $CO_2e$ of GHGs or about half of the total reduction the Clippers are attempting to claim from the electric buses and vehicles.

| **GHG Emissions from Electric Buses and Vehicles[11]** | | |
|---|---|---|
| **Electric Buses** | | |
| SCE Electricity Emission Factor | 0.17 | (MT $CO_2e$/MWh) |
| Fuel Economy of Electric Bus | 1.50 | (kWh/mile) |
| Annual Miles Driven per Transit Vehicle | 23,000 | (miles/yr) |
| Annual GHG Emissions per Electric Bus | 5 | (MT $CO_2e$/yr) |
| Missing GHG emissions for Project | 305 | MT $CO_2e$ |
| **Electric Vehicles** | | |
| SCE Electricity Emission Factor | 0.17 | (MT $CO_2e$/MWh) |
| Fuel Economy of Electric Vehicle | 0.25 | (kWh/mile) |
| Annual Miles Driven per Vehicle | 11,000 | (miles/yr) |
| Annual GHG Emissions per Electric Vehicle | 0.47 | (MT $CO_2e$/yr) |
| Missing GHG emissions for Project | 140 | MT $CO_2e$ |

Additionally, as part of Inglewood's Climate Action Plan, Inglewood has already promised to replace fleet vehicles with low- or zero-emission vehicles. The Clippers have not demonstrated that these replacement vehicles offered now are additional to this and other relevant city programs.

Furthermore, the Clippers do not provide the analysis and data required surrounding the assumed lifespan of the EV chargers. Without this data the benefits of these charges likely are overestimated.

### 4. No Evidence That Renewable Natural Gas Can Be Utilized

[11] CO2e weighted intensity factor for SCE accounts for CO2 and CH4 emissions rates consistent with the 50% Renewable Portfolio Standard. Fuel economy of the bus is based on data from Proterra and BYD. VMT is based on project application.

The Clippers' on-site use of renewable energy measures calculates the emissions reductions for renewable natural gas "on the assumption that the proposed Project's use of 100% renewable natural gas will eliminate all anthropogenic $CO_2$ emissions from Project's natural gas usage." However, there is no description of how the renewable natural gas will be obtained, or if sufficient renewable natural gas will be available after project buildout to support 100% of the project's operational natural gas demand. Renewable natural gas is generally provided to the transportation fuel market, and there is zero evidence to suggest there will be sufficient 100% renewable natural gas available for this project.

### C. The Clippers Must Prove GHG Reductions Are Real; Any Post-Approval "Verification Program" Must Meet ARB's Rigorous GHG Reporting Standards

Even employing the flawed analysis outlined above, the Clippers concede that they still fall far short of the 50% local reduction requirement. The Clippers attempt to cover up this shortfall through a verification program that defers the analysis far into the future (long after the project is approved) and outside the public view. Stated differently, the Clippers want to "true up" their emissions, without public scrutiny, after the arena is built. The Clippers provide barely any detail on how this verification will unfold, the level of evidence and analysis that will be required, or who will ultimately approve the analysis.

AB 987 says nothing about a verification program. The Clippers must prove in advance that they will achieve real local reductions. The legislature included a verification and "true up" provision for the TDM program. If one was envisioned for GHGs, the legislature would have included it in the bill.

If the Clippers are allowed to follow some type of verification program, it must be one that has been vetted and approved by ARB through an existing legal mechanism, is third-party verified, and is subject to public review.

Any such verification program needs to provide a clear and substantive description of the annual verification process that should meet the same standards as other ARB annual verification reports (*i.e.*, the mandatory ARB GHG Reporting program[12]). Additional information on all assumed variables used to generate the emissions inventory should be gathered by the Clippers as part of any annual verification process and included in the annual reports to verify the validity of the GHG emission estimates for the project construction and operations. This includes the events at Staples Center and other venues given the Clippers' position regarding taking credit for such events (if ARB approves such position, which it should not). Additional requirements for any verification process must include the following:

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[12] Amendments to the Regulation for the Mandatory Reporting of Greenhouse Gas Emissions Article 2: Mandatory Greenhouse Gas Emissions Reporting Subarticle 1. General Requirements for Greenhouse Gas Reporting § 95100, *available at*: https://ww3.arb.ca.gov/cc/reporting/ghg-rep/regulation/mrr-2018-unofficial-2019-4-3.pdf?_ga=2.63211561.704742149.1573149821-2008260070.1572389583, (last accessed November 2019).

- These reports must be released publicly to allow the opportunity for public review and comment.
- These reports should be verified by an independent third party and this third party should have an accreditation with ARB through the mandatory GHG reporting program.
- The conflict of interest provisions for the independent third party as established by ARB should be followed.
- The methods and approach to measure the actual data should be documented at this time for review. For example,
    - The method to measure all related mobile source information such as the number of trips, trip length (origin), vehicle type (*e.g.*, age, model, fuel type), and idling time
    - The method to track energy usage
    - The method to measure water usage
    - The method to track waste generation and waste diversion
        - Including the fate of waste diverted
    - The construction equipment usage
        - Including type of equipment (*e.g.*, age, model type, load, fuel type)
        - Workers (*e.g.*, number, transportation method, vehicle type, vehicle fuel type, age of vehicle, idling time)
    - The emission factors should be based on actual vehicle age, types, and fuel (*i.e.*, fleet average factors should not be used)
    - The number of events at each venue being assessed has to be rigorously tracked and include:
        - Attendance numbers
        - Specific tracking of transportation methods.

Moreover, the Clippers' "compliance program" contains no consequences for missing the mark. Long after they have obtained the benefits of AB 987 and the arena is open and the community is suffering, if the Clippers' guesses are wrong, then the Clippers propose all they would have to do is meet the standards that AB 987 already establishes – 50% of additional emissions to be offset locally and 50% elsewhere. How is this an incentive? If ARB is really to accept a "trust us" approach, there must be real consequences for not meeting the mark. We recommend, similar to the penalties outlined in AB 987 for failure to reach TDM goals, that when the Clippers fall short of their local GHG emission reductions, they be required to offset an additional amount of emissions locally. Only this way will the Clippers be properly incentivized to meet the letter and spirit of AB 987.

Until more information is provided on the verification framework, it is impossible to comment meaningfully on its validity under AB 987. ***At a bare minimum, any verification program should have to meet the rigor and public reporting requirements of California's GHG reporting rule.***

### D. The Verification Program Appears Designed to Limit the Clippers' Obligations to the Community's Detriment

The complexity the Clippers build into their proposed verification program seems to have one goal – to reduce the amount of GHGs the Clippers are required to offset.

For example, the Clippers define "Actual Events" as events and attendance occurring at the Clippers arena and Staples Center, the Forum, and the Honda Center but "2028 Olympic events and related events shall be subtracted from the total of Actual Events." (Nov. 1 Letter, p. 6.) Why? If Olympic events are held at the Clippers arena, they should ***not*** be excluded from the arena's emissions. The entire point of AB 987's local offset requirements was to benefit the immediate community's overall health. If not for the Clippers' arena, the events would not occur in the community and the community would not be burdened by the health impacts that they create.

Similarly, why are "home games of existing or future sports teams at Existing Venues" excluded from the calculations? (Nov. 1 Letter, p. 6.) As the Oak View Group explained in its report attached to our September 4, 2019 letter, with the Clippers moving out of Staples Center, Staples Center will have more flexibility to fill its arena. Under the Clippers' approach, new Arena Football games or other new athletic events would not be counted towards the emissions the Clippers must offset. Again, why? There is no rational basis for excluding these emissions or for excusing the Clippers from offsetting them.

The discount for renovations to existing arenas is also solely intended to reduce the Clippers' requirements and, thereby, increase the burden on the local Inglewood community. (*See* Nov. 1 Letter, pp. 6-7.) None should be provided.

Lastly, it is unclear why the amount of "Additional GHG Emission Reductions" would be capped. If the Clippers insist on clinging to a "market-shift" theory and if ARB accepts it, which it should not, then there should not be a cap on total emissions to be offset. If it turns out that Staples Center is leased for 325 days a year, which the Clippers' relocation would facilitate, then the Clippers must be required to offset all of these emissions. The Clippers want to have it both ways: all the benefit of the "market-shift" without any of the offset obligations. ARB should not allow this.

### E. We Are Not Asking ARB to Hold the Clippers to a Different Standard

The Clippers have complained that they are being held to a different standard. As we have detailed in prior correspondence, ARB has consistently rejected the "market-shift" methodology the Clippers' continue to pursue doggedly. All MSG, the community, and environmental groups have requested is that ARB ensure that AB 987's mandates are met.

In any event, AB 987 *is more stringent* than AB 900. AB 987's author characterized the bill as "setting a new gold standard for green standards." ARB must ensure that is the case.

With respect to the two other arena and stadium projects that ARB determined were net GHG neutral under AB 900, ARB's approach did not accept a "market-shift" methodology.

The Qualcomm Stadium Reconstruction Project (Case No. 2015061061) proposed demolition of an existing multi-purpose stadium and reconstruction of a new stadium on the same site. ARB correctly credited the GHG emissions associated with the to-be-demolished stadium as part of the project's baseline for GHG emission purposes. Of course, here, none of Staples Center, the Forum, or Honda Center is a part of the project site and none will be demolished. Activities at these three arenas should be excluded from the Clippers' arena's GHG emission baseline.

Similarly, the Event Center and Mixed-Use Development at Mission Bay Blocks (Warriors SF Arena) (Case No. 2014112045) does not support the Clippers' methodology. The Warriors did not argue that a "market shift" would occur and take credit for existing events at arenas at which they do not even play. Here, the Clippers are attempting to take credit for events that are currently at two other venues (Forum and Honda Center) that have nothing to do with the Clippers' new arena.

Further, the Warriors were leaving an arena where they were the sole, long-term tenant. That is not the case with Staples Center. In its decision on the Warriors application, ARB noted: "In the absence of any formal plans for redevelopment of the existing Oracle Arena, ARB assumed Oracle Arena remains as the reference point for the purpose of defining a baseline" and only credited 50% of events. Here, there is no need to "redevelop" Staples Center. Staples Center will continue to be a premier event venue with multiple existing tenants and non-sporting events.

Staples Center is not being demolished, has three other tenants and does not need redevelopment. The impact of the Clippers leaving the Staples Center is much smaller than the impact of the Warriors leaving Oracle. In fact, as detailed in prior submissions by an expert report, Staples Center would likely host more events with the Clippers gone.

This is not unfair to the Clippers. No one is asking ARB to treat the Clippers differently other than the Clippers. Rejection of this application is not a rejection of the project as a whole.

* * * *

We appreciate your attention to this matter. If you have any questions, please do not hesitate to call me at 213-891-7540.

Very truly yours,

s/ Maria Pilar Hoye

Maria Pilar Hoye
of LATHAM & WATKINS LLP

cc: Mary D. Nichols, Chairwoman, California Air Resources Board
Richard Corey, Executive Director, Air Resources Board
Steven Cliff, Executive Office, Air Resources Board

# EXHIBIT 1

# Health

Air pollution poses a significant risk to public health. Exposure to polluted air is linked to a number of health effects. Some of these are: worsened asthma, hospitalizations, and even premature death related to heart and lung disease. Toxic air pollutants emitted from cars, trucks and industry can also cause cancer.

## AIR POLLUTION HEALTH AFFECTS

More than 90% of Californians breathe unhealthful air during some part of the year, harming people's health and the economy.

LEARN MORE

## FEATURED

### People at Risk

Who is at risk, and where?

### Protecting Your Health

How can you protect your health?

### Outdoor Air Quality Standards

What is CARB doing to protect health?

## PROGRAMS

**Air Cleaners & Ozone Generating Products**

**Ambient Air Monitoring - Regulatory**

**Community Health**

**Exposure**

**Indoor Air Quality**

**Outdoor Air Quality Standards**

**People at Risk**

ALL PROGRAMS

## RESOURCES

### Information

Estimating the Health Benefits of Reductions in Emissions of PM2.5 or its Precursors: Short Description

CARB's Methodology for Estimating the

### Fact Sheet

Air Pollution and the Brain

National Ambient Air Quality Standards

California Ambient Air Quality Standards

### Research

Selected References: Children's School Bus Exposure and Mitigation Studies

Research Synthesis #16-01 "Exposure to Fine Particulate Matter

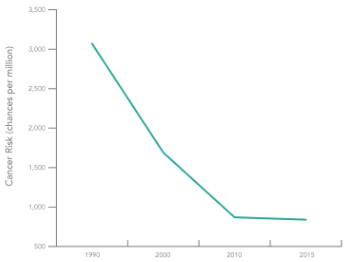
Health Effects of Air Pollution

Understanding the Health of Our Communities

(PM2.5) Increases Health Risks for Californians"

Research Synthesis #17-03 "Reducing Near Roadway Exposure to Air Pollution"

## THE DATA




Health Impacts from Traffic Sources

Combustion-source air pollution & premature death

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California Governor

**Gavin Newsom**

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Secretary for Environmental Protection

**Jared Blumenfeld**

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Chair, California Air Resources Board

**Mary D. Nichols**

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# EXHIBIT 2

# Estimating the Health Benefits of Reductions in Emissions of PM2.5 or its Precursors: Short Description

Reductions in PM2.5 emissions are associated with reductions in the risk of premature deaths, hospitalizations and emergency room visits, especially for sensitive groups

## CATEGORIES

**Topics** Health, Air Pollution, Cars & Trucks, Construction & Earthmoving Equipment, Environmental Justice, Oceangoing Vessels & Harbor Craft, Freight & Goods Movement, Trains & Railyards, Transit, VW Diesel Vehicles,

**Programs** Exposure, Community Air Protection Program , Community Health, Zero-Emission Powertrain Certification, Alternative Diesel Fuels, In-Use Off-Road Diesel-Fueled Fleets Regulation, Study of Neighborhood Air near Petroleum Sources

**Type** Information

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The U.S. EPA's 2010 document, Quantitative Health Risk Assessment for Particulate Matter, has noted that reductions in PM2.5 emissions are associated with reductions in the risk of premature deaths, hospitalizations and emergency room visits. Sensitive groups such as the elderly, and people with chronic heart or lung disease, and children are at particular risk. To estimate these health benefits, staff use CARB's incidence-per-ton (IPT) methodology. It is based on a methodology developed by the U.S. EPA.[1] [2] [3] It is used to estimate benefits of reductions in PM2.5 emitted directly from sources, and PM2.5 formed from precursors by chemical processes in the atmosphere.

The basis of the IPT methodology is that changes in emissions are approximately proportional to changes in health outcomes. IPT factors equal the number of PM-related health effects divided by emissions of PM2.5 or the precursor NOx. The calculation is performed separately for each air basin.

The IPT factor is used to estimate of the reduction in health outcomes achieved by CARB regulations. For future years, the IPT factor is adjusted to account for population growth. CARB's current IPT factors are based on a 2014-2016 baseline scenario.

CARB's Estimating Health Benefits Associated with Reductions in Diesel PM and NOx Emissions provides a detailed explanation of how health impacts are estimated.


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2. Fann N, Baker KR, Fulcher CM. 2012. Characterizing the PM2.5-related health benefits of emission reductions for 17 industrial, area and mobile emission sectors across the U.S. Environ Int. 2012 Nov 15;49:141-51.
3. Fann N, Baker K, Chan E, Eyth A, Macpherson A, Miller E, Snyder J. 2018. Assessing Human Health PM2.5 and Ozone Impacts from U.S. Oil and Natural Gas Sector Emissions in 2025. Environ. Sci. Technol. 52 (15), pp 8095–8103.


## RELATED RESOURCES

**Commercial Harbor Craft Tier 4 Feasibility Report**

**Label Vendors for Off-Road Diesel Vehicles and Large Spark-Ignited (LSI) Equipment**

**Selected References: Children's School Bus Exposure and Mitigation Studies**


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# Estimating Health Benefits Associated with Reductions in PM and NOx Emissions: Detailed Description

## 1. Introduction

CARB estimates premature death and other health effects related to PM2.5 exposure using one of two methods, a health model or the *incidence-per-ton* (IPT) method. In most cases, CARB uses the IPT method to estimate health effects from emissions data. The IPT methodology is a simplified procedure that uses pre-calculated results, obtained by running a mathematical health model on a baseline scenario, to compute estimates of the number of cases of adverse health outcomes. In cases where measured or modeled PM2.5 concentrations are available, CARB staff input them directly into a health model to obtain estimates of health effects.

## 2. Health model

The health model is based on the methodology used by US EPA's BenMAP benefits mapping and analysis software [US EPA BenMAP]. The health model enables automation of repetitive tasks and facilitates the incorporation of California-specific data. The health model uses a multi-step process to estimate health impacts from measured or modeled PM2.5 concentrations. These steps are described below.

The health model estimates the incidence of premature death and other health outcomes at each census tract or modeling grid cell using the equation:

$$\text{Incidence} = [\text{population}]_i \times [\text{baseline incidence}]_i \times [\ 1 - \exp(-\beta \times \text{PM2.5}\ )\ ]$$

where the subscript *i* indexes the age groups. The specific form of this equation is determined by the type of statistical model used by the health studies to model the relationship between PM2.5 exposure and health risk. All the studies selected by CARB use a log-linear relationship, which takes the form shown above. The incidence is summed over age groups to obtain the total incidence for the census tract. The coefficient β is taken from one of the health studies discussed below. The source of PM2.5 comes from monitored or modeled air quality data.

CARB draws upon health studies used by the U.S. EPA for its risk assessments (US EPA 2010). CARB uses a subset of the endpoints used by U.S. EPA, chosen on the basis of their strength and robustness. For premature mortality, CARB uses the cardiopulmonary mortality risk coefficient

for the 1999-2000 time period from Krewski et al., 2009, among the largest studies of its kind, with 360,000 participants. For cardiovascular and respiratory hospitalizations, CARB used Bell et al., 2008, and for emergency room visits for asthma CARB used Ito et al., 2007. The process for selecting these studies was described in detail in CARB's 2010 PM2.5 mortality report (CARB 2010a).

**Estimating exposure from measured PM2.5**

The health model estimates population-weighed exposure to primary and secondary PM2.5 from annual average concentrations measured at monitors located throughout California. The model estimates exposure between monitor locations. This is accomplished using a spatial interpolation method known as inverse distance-squared weighting. Separate exposure estimates are made for PM2.5 emitted directly from diesel sources (primary PM2.5) and from PM2.5 formed from precursor gases (secondary PM2.5).

Estimating Diesel particulate matter concentrations

Annual diesel particulate matter (DPM) concentrations are not measured directly. Rather, they are estimated from annual average NOx concentrations by multiplying them by air basin and year-specific DPM/NOx emission ratios computed from CARB emission inventories.

The methodology and its rationale is described in greater detail in CARB 2010b and Propper et al., 2015. DPM concentrations were estimated at 106 monitors located throughout the state. In order for a measurement to be considered valid, the data were required to be at least 75% complete.

Estimating secondary ammonium nitrate concentrations

In addition to DPM, CARB computes health impacts for secondary ammonium nitrate PM2.5 formed in the atmosphere from NOx. To estimate ammonium nitrate PM2.5 exposure, CARB staff use speciated PM2.5 nitrate ion ($NO_3^-$) concentration data from two sources: the air quality monitoring network maintained by CARB and local air quality districts and the IMPROVE visibility network (IMPROVE Visibility Network).

CARB and air pollution control districts operate a network of PM2.5 monitors around the state, mostly in urban areas (CARB AQMN). PM2.5 samples are collected as 24-hour filter samples, once every 3-6 days. Samples from some monitors are further analyzed to determine the concentration of nitrate ion and other constituents. During 2014-2016, nitrate data were available from 18 urban monitors. Data for these monitors are retrieved from CARB's ADAM air quality database (CARB ADAM).

In addition to the urban monitors, the national IMPROVE visibility network operated 20 PM2.5 nitrate ion monitors in California during 2014-2016, mainly in national parks and other remote locations (IMPROVE Visibility Network). These instruments collected one sample every three days. IMPROVE data were retrieved from the project web site (IMPROVE Visibility Network).

Daily samples were aggregated by monitor to obtain annual averages. In order for an annual average to be considered valid, the data were required to be at least 75% complete. To convert from nitrate ion concentration to ammonium nitrate ($NH_4NO_3$), the annual averages were multiplied by the ratio of the molecular weight of ammonium nitrate to that of the nitrate ion.

Prior to May, 2019 CARB used PM10 nitrate data instead of more accurate PM2.5 nitrate data to estimate ammonium nitrate aerosol concentrations to compute health impacts. This is because speciated PM10 data was available for more locations than speciated PM2.5. However, the number of monitors in the speciated PM10 network has shrunk and is now comparable in size and coverage to the speciated PM2.5 network. Therefore, in May, 2019 CARB began using PM2.5 nitrate data to compute health effects. The PM2.5 nitrate monitors are more accurate because they store the filters in a refrigerated compartment, and less of the sample is lost to volatilization. Consequently, the estimated PM2.5 nitrate concentrations and associated IPT factors for NOx emissions are approximately 50% higher than those used prior to May, 2019.

**Estimating exposure using modeled PM2.5 concentrations**

The health model can also be run with concentrations derived from an air quality model instead of monitored data. Air quality models include dispersion models, which model how pollutants are dispersed by the wind, and photochemical models, which are more elaborate and capture the effects of sunlight, temperature, and chemical reactions on pollutants. Dispersion models are only used for primary pollutants, as they are not capable of modeling formation of secondary pollutants. Air quality models generate gridded results, with grid cells typically in the range of 500-2,000m square.

**Population projections at the census tract level**

The health model uses age-resolved population data at the census tract level. CARB uses data from the 2010 Census (U.S. Census Bureau). These were projected to 2011-2060 using age-resolved county population projections from the California Department of Finance (CDOF).

Age-specific growth factors for each county, for each year, were computed from the CDOF projections by dividing each county population for the target year by the average county

population for the base years 2014-2016. These growth factors were applied to each census tract in every county, for each age group separately. Population was projected for five-year age groups 0-4 through 80-84, and for age 85 and older.

This method of projection reflects growth in overall county population, but does not model changes in population distribution within counties, such as expansion of urban areas into surrounding rural land.

**Estimating baseline incidence**

The health model uses incidence data for cardiopulmonary mortality extracted from the Center of Disease Control (CDC) Wonder database. Incidence data for hospitalizations for cardiovascular and respiratory causes, and emergency room visits for asthma are taken from US EPA BenMAP benefits mapping software (US EPA BenMAP). Baseline incidence rates vary by age bracket. Incidence was estimated separately for five-year age groups 0-4 through 80-84, and for age 85 and older. Mortality incidence data are county-specific. Incidence data for other health outcomes is uniform throughout California.

**Aggregating health outcomes by air basin**

To aggregate results from census tracts to larger geographical subdivisions such as counties or air basins, the health model uses a geospatial technique called areal interpolation. Areal interpolation is a procedure for translating spatial data from one set of geographical subdivisions to another when the boundaries do not overlap. Numerous variants of the technique exist, but for the purpose of this analysis the simplest form, which uses area of polygon intersection, was employed (Goodchild and Lam, 1980, Flowerdew and Green, 1994). The precision of this method depends on the size of the geographical subdivisions and the spatial homogeneity of the quantity being apportioned. In urban areas, where census tracts are small and population is distributed more evenly, areal interpolation to larger subdivisions such as air basins yields relatively precise estimates. In rural areas where the population is distributed unevenly over large census tracts, estimates are less precise.

## 3. Incidence-per-ton methodology

CARB uses the IPT methodology to quantify the health benefits of regulations and programs that reduce PM2.5 and precursor emissions. It is based on an approach developed by the US EPA, as described by Fann et al. (2009, 2012, 2018). The mathematical relationship between changes in emissions and changes in health outcomes is approximately linear. The IPT methodology is based upon this relationship, and makes the following assumptions:

(1) Changes in health outcomes are proportional to changes in PM concentration;
(2) Changes in primary pollutant concentrations are proportional to changes in emissions; and
(3) Changes in secondary pollutant concentrations are approximately proportional to changes in emissions. It should be noted that there may be cases where the relationship between emission of oxides of nitrogen (NOx) and ammonium nitrate aerosol is not linear.

Due to the approximately linear relationship between premature deaths (or other health outcomes) and emission concentrations, the number of premature deaths can be estimated by multiplying emissions by a scaling factor: the *IPT factor*. IPT factors are developed by applying a health model to air pollution concentrations for a baseline period to estimate the number of health outcomes associated with PM2.5 exposure, then dividing by emissions of PM2.5 or a precursor.

Current IPT factors were developed from a baseline scenario using air quality data, incidence data and emission inventories for 2014-2016, and age-stratified population projections for 2010 through 2060. IPT factors were calculated separately for each air basin.

IPT factors are currently available for two types of PM: diesel particulate matter (DPM) primarily from on-road sources, and secondary ammonium nitrate particles formed from NOx. Health effects of primary PM2.5 from sources other than on-road diesel engines are estimated by using IPT factors developed for DPM and multiplied by a relative potency factor, as described below.

In addition to premature mortality from cardiopulmonary causes, CARB currently uses IPT factors to estimate hospitalizations due to cardiovascular and respiratory causes and respiratory emergency room visits including asthma.

Since the total incidence of heath effects is proportional to population, results for future years are adjusted by the ratio of the projected population in the target year to the average population in the base years 2014-2016.

## 4. Relative potency factors for non on-road diesel sources

To quantify the health benefits of reductions in primary PM2.5 from sources other than on-road diesel vehicles, CARB uses IPT factors developed for DPM and multiplies the results by a relative potency factor specific to the source and location of the emissions.

Relative potency may be determined in several ways, including but not limited to

- The ratio of the intake fraction of the source to the intake fraction for DPM. The intake fraction is a measure of the fraction of the emissions from a given source that is inhaled by the receptor population. It is specific to a source and a location; e.g., a particular type of facility in a given air basin.
- Comparison of IPT results with direct estimation results for the same scenario. The ratio of the results obtained by the two methods may then be used to adjust the results obtained by IPT factors in a larger setting. For example, the ratio of results obtained by IPT and the health model for one air basin may be used to adjust results for other air basins.
- General consideration of conditions under which emissions take place are also important. For example, if an on-road vehicle delivers goods from a facility in a remote location to a facility located in an urban area, half of idling emissions may be considered to occur far from receptor populations. Hence an adjustment factor of 0.5 may be appropriate for computing the health benefits of reducing idling emissions.

## 5. Uncertainty in health impact estimates

This methodology is well-established and includes up-to-date information. However, there are uncertainties in the underlying data and assumptions:

- Air quality data is subject to natural variability from meteorological conditions, local activity, etc.
- The assumption that changes in concentrations of pollutants are proportional to changes in emissions of those pollutants or their precursors is an approximation. There may be cases where actual changes in concentrations are higher or lower than predicted.
- The estimation of DPM concentrations and DPM/NOx emission ratios is subject to uncertainty. Emissions are reported at an air basin resolution, and do not capture local variations.
- Inverse distance-squared weighting, the spatial interpolation method is used to estimate concentrations each census tract. Compared with other geospatial estimation methods such as Kriging, inverse distance-squared interpolation has the virtue of simplicity, and does not require selection of parameters. When data are abundant, most simple interpolation techniques give similar results (Jarvis et al.,

2001). All geospatial estimation techniques exhibit greater uncertainty when data points are sparser, and uncertainty increases with distance from the nearest data points.

- Future population estimates are subject to increasing uncertainty as they are projected further into the future. For reasons of computational efficiency, the spatial resolution of population estimates is limited to census tract resolution.
- Observed baseline incidence rates change over time, and are subject to random year-to-year variation and systematic shifts as population characteristics and medical treatments evolve. Sample size requirements necessitate estimating baseline incidence rates at large geographic scales, state or county.
- Relative risks in the concentration response function are estimated with uncertainty and reported as confidence ranges.

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# EXHIBIT 3

# Is Disparity in Asthma among Californians due to Higher Pollution Exposures, Greater Vulnerability, or Both?

Contract No: 07-309

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Revised February 24, 2012

Prepared for the California Air Resources Board and the California Environmental Protection Agency

DISCLAIMER

The statements and conclusions in this Report are those of the contractor and not necessarily those of the California Air Resources Board. The mention of commercial products, their source, or their use in connection with material reported herein is not to be construed as actual or implied endorsement of such products.

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## Abstract

This study addresses the question: Is the disproportionate burden of asthma or asthma-like symptoms among low socioeconomic status individuals related to greater pollutant exposures, greater vulnerabilities, or both? Using Geographic Information System (GIS) software, we linked California Health Interview Survey (CHIS) 2003 respondents' residential addresses to government air monitoring stations for $O_3$, $PM_{10}$, $PM_{2.5}$, and $NO_2$. We calculated annual pollutant averages and days exceeding air quality standards and assessed traffic density and residential distance to roadways. Higher exposures were estimated for low income and racial/ethnic minority respondents with asthma for $NO_2$, $PM_{10}$, and $PM_{2.5}$, but not $O_3$. Among adults with asthma, we observed increases in adverse asthma outcomes, such as daily/weekly symptoms, asthma attacks, daily medication use, and asthma-related work absences and emergency department visits with increasing annual average pollutant concentrations. Among children with asthma, daily asthma medication use and school absences were associated with increased annual average $NO_2$ concentration. Similar positive associations were observed between $O_3$, $PM_{10}$, and $PM_{2.5}$ exceedance days and asthma outcomes, mainly for adults. When adjusting for confounders, associations between pollutants and asthma outcomes persisted. Notably, racial/ethnic minority and low income respondents had greater increases in adverse asthma outcomes for similar increases in $NO_2$ and $PM_{10}$ exposures.

## Executive Summary


### Background

Children, the elderly (Babey, Hastert et al. 2007), racial/ethnic minorities (Meng, Babey et al. 2007), and low-income Californians (Babey, Hastert et al. 2007) suffer disproportionately from asthma burdens and asthma-like symptoms. Linking air pollutant data from ambient monitors and traffic data with California Health Interview Survey (CHIS) 2003 data, this study tested the following hypotheses: 1) Vulnerable sub-populations in California (e.g., racial/ethnic minorities and low-income individuals) with asthma or asthma-like symptoms have higher exposures to air pollution; 2) Individuals with asthma or asthma-like symptoms exposed to higher levels of air pollution are more likely to report adverse health outcomes; 3) Air pollution exposures, low socioeconomic status (SES), and certain vulnerability factors exert independent adverse effects on individuals with asthma or asthma-like symptoms; and 4) Higher pollutant exposures interact with vulnerability factors, resulting in greater air pollution impacts on asthma in vulnerable sub-populations.

### Methods

We conducted a cross-sectional study linking California Health Interview Survey (CHIS) 2003 data to existing air pollutant and traffic data. We selected CHIS 2003 adult respondents ages 18 or older and child respondents ages 0-17 with self-or caregiver-reported lifetime asthma (N=5,620 adults and 1,889 children), then focused on those with current asthma (N=3,587 adults and 1,224 children). Additionally, we selected respondents without an asthma diagnosis who had asthma-like symptoms (N=4,413 adults and 1,109 children). Respondents living at their current addresses or neighborhoods for ≥9 months were included. Using Geographic Information System (GIS) software, we linked respondents' residential addresses to air monitoring stations measuring $O_3$, $PM_{10}$, $PM_{2.5}$, and/or $NO_2$. We calculated annual pollutant averages for the 12-months prior to respondents' interview dates and the number of federal/state exceedance days for pollutant concentrations and assessed traffic density and distance from residence to roadways as proxies for traffic-related air pollution exposure. We performed logistic regression analyses for respondents with asthma or asthma-like symptoms, separately for children and adults. We also conducted pollutant-outcome analyses adjusting for potential confounders related to vulnerability. Interaction terms were used to evaluate increased vulnerability to pollutants among sub-populations. We performed sensitivity analyses on length of residence, employment status, distance from pollutant monitors, and asthma medication use.

### Results

We observed disparities in exposure to air pollutants by income and race/ethnicity among Californians with current asthma. Adults and children with current asthma living below 200% of the federal poverty level (FPL) had higher annual average exposures to $NO_2$, $PM_{10}$, and $PM_{2.5}$ than those living at or above 400% of the FPL. Latino and African American adults and children had higher $PM_{2.5}$ annual averages than whites; Latino and Asian/Pacific Islander children had higher $NO_2$ annual averages than white children. However, white adults and children had higher exposures to $O_3$ than Latinos, African Americans, and Asian/Pacific Islanders. Similar exposure disparities were seen for respondents with asthma-like symptoms.

We observed positive associations between increased annual average pollutant concentrations for $O_3$, $PM_{10}$, and $PM_{2.5}$ and adverse asthma outcomes among adults, such as frequent asthma symptoms (daily/weekly symptoms), asthma attacks or episodes, use of daily

medication to control asthma, work absences, and asthma-related emergency department (ED) visits. Among children, use of daily asthma medication and missing 2 or more days of school/day care were associated with higher exposures to $NO_2$. We also observed positive associations between asthma outcomes and the number of federal or state exceedance days for $O_3$, $PM_{2.5}$ and $PM_{10}$. In adults with asthma-like symptoms, $O_3$, $PM_{10}$, and $PM_{2.5}$ increases were associated with increased odds of asthma-like outcomes, and among children, $O_3$ and $NO_2$ were associated with increased asthma-like outcomes. We detected few associations between traffic density and distance to roadways and asthma or asthma-like outcomes.

When adjusting for vulnerability factors as possible confounders, such as access to care, risk behaviors, asthma severity and indoor triggers, positive associations between criteria pollutants and asthma outcomes persisted, as did positive associations between asthma outcomes and belonging to minority or low income sub-populations. Having heart disease and having adult onset asthma increased odds for visiting the ED and using daily asthma medication among those with current asthma. Notably, positive interactions were observed between criteria pollutant exposure and race/ethnicity. Specifically, African American and Asian/PI/other adults had a greater increase in odds of missing two or more days of work due to asthma compared to white adults with the same increase in annual average $NO_2$. African American adults also had greater increases in odds of experiencing daily/weekly asthma symptoms for the same increase in $NO_2$. Compared to white children, American Indian/Alaska Native and Asian/PI/other children had a greater increase in odds of experiencing daily/weekly asthma symptoms for the same increase in $NO_2$. Latino children had a greater increase in odds of using daily asthma medication for the same increase in $PM_{10}$, and African-American and Asian/PI/other children had greater increases in odds of daily/weekly symptoms than white children for a comparable increase in $PM_{10}$. We also found that children living below 200% of the FPL had a greater increase in odds of ED visit compared to those living at or above 400% of the FPL for the same increase in $NO_2$.

**Conclusions**

In conclusion, we observed disparities in exposure to air pollutants by federal poverty level and race/ethnicity among Californians with current asthma. In general, higher annual average exposures were observed for lower income groups and racial/ethnic minorities for $NO_2$, $PM_{10}$, and $PM_{2.5}$. We observed increases in the odds of having adverse asthma outcomes with increasing annual average pollutant concentrations for $O_3$, $PM_{10}$, and $PM_{2.5}$ among adults and $NO_2$ among children with current asthma. We also observed associations with the number of days exceeding federal or state standards for $O_3$, $PM_{10}$, and $PM_{2.5}$. In respondents with asthma-like symptoms, positive associations were observed between the odds of having asthma-like symptoms and annual air pollutant averages and exceedance measures. When adjusting for potential confounders, pollutant associations for $O_3$, $PM_{10}$, and $PM_{2.5}$ remained. Novel findings include interactions for race/ethnicity and household federal poverty level with annual average pollutant exposures for $NO_2$ and $PM_{10}$, suggesting that racial/ethnic minority and low-income groups have greater increases in adverse asthma outcomes with similar increases in exposures.

These results provide a more comprehensive understanding of the impact of air pollution on Californians suffering from asthma and asthma-like symptoms and indicate that current air quality in California needs to be further improved in order to protect California residents, especially those in vulnerable sub-populations.

# I. INTRODUCTION

## Scope and Purpose

In October 2003, the California Air Resources Board (CARB) developed a Vulnerable Population Research Program that aims to protect all California residents, particularly individuals considered especially at risk, to the adverse effects of air pollution. For the first time, low-income neighborhoods and communities of color were designated as vulnerable sub-populations, in addition to children, the elderly, people with preexisting cardiovascular and/or pulmonary disease, and individuals who spend a large amount of time outdoors. This research was designed to provide much needed information on the effects of long-term air pollution exposure on severe asthma and asthma-like symptoms in vulnerable populations.

According to the estimates from the 2003 California Health Interview Survey (CHIS 2003), 4.5 million Californians suffer from asthma and an additional 3.4 million Californians suffer from asthma-like symptoms (Babey, Meng et al. 2006). Although asthma cannot be cured, most individuals with asthma can become symptom-free by avoiding or controlling environmental triggers and by taking proper medications. However, children, the elderly (Babey, Hastert et al. 2007), racial/ethnic minorities (Meng, Babey et al. 2007), and low-income Californians (Babey, Hastert et al. 2007) suffer disproportionately from asthma and asthma-like symptoms. Previous studies also indicate some sub-populations are more affected by pollutants due to increased susceptibility or higher exposures. For instance, children are especially susceptible to the damaging effects of $O_3$ in part because their lungs are still developing , which makes them more sensitive to pollutant damage (Gilliland, McConnell et al. 1999). Minorities may be more affected due to differential exposure to air pollution and vulnerability (Clark, Brown et al. 1999; Ostro, Lipsett et al. 2001; Mortimer, Neas et al. 2002; Perera, Illman et al. 2002). More studies need to be conducted on other vulnerable populations, such as those with low socioeconomic status (O'Neill, Jerrett et al. 2003).

The overall goal of the proposed project was to examine whether the disproportionate asthma burden among these California sub-populations (e.g., low-income and ethnic minorities) is related to higher exposure to air pollutants, greater vulnerability due to low socioeconomic status (SES) related factors, or both. Here we defined “vulnerability” based on a “triple-jeopardy” theory (Jerrett, Burnett et al. 2001; Levy, Greco et al. 2002). We tested hypotheses, namely: 1) among adults with current asthma, certain sub-populations (e.g., groups with low SES) are exposed to higher levels of air pollution; 2) these individuals already have poorer health due to social determinants, such as poverty, lack of adequate health care, and psychosocial stress; and 3) this combination of higher air pollution exposures and poorer baseline health interacts, resulting in greater air pollution impacts on asthma in these vulnerable groups. No routine asthma surveillance system, such as a registry, exists in California except for mortality statistics and hospital discharge/emergency department (ED) visit data. CHIS data makes it possible, for the first time, to relate exposure to health outcome data for a large number of people with asthma (larger than the National Health Interview Survey (NHIS)). The CHIS sample is representative of California’s non-institutionalized population and asks many standard health questions from the NHIS. Additionally, CHIS provides a unique

opportunity to study the adverse effects of air pollution because it collects information on residential address and duration of residence in the same neighborhood.

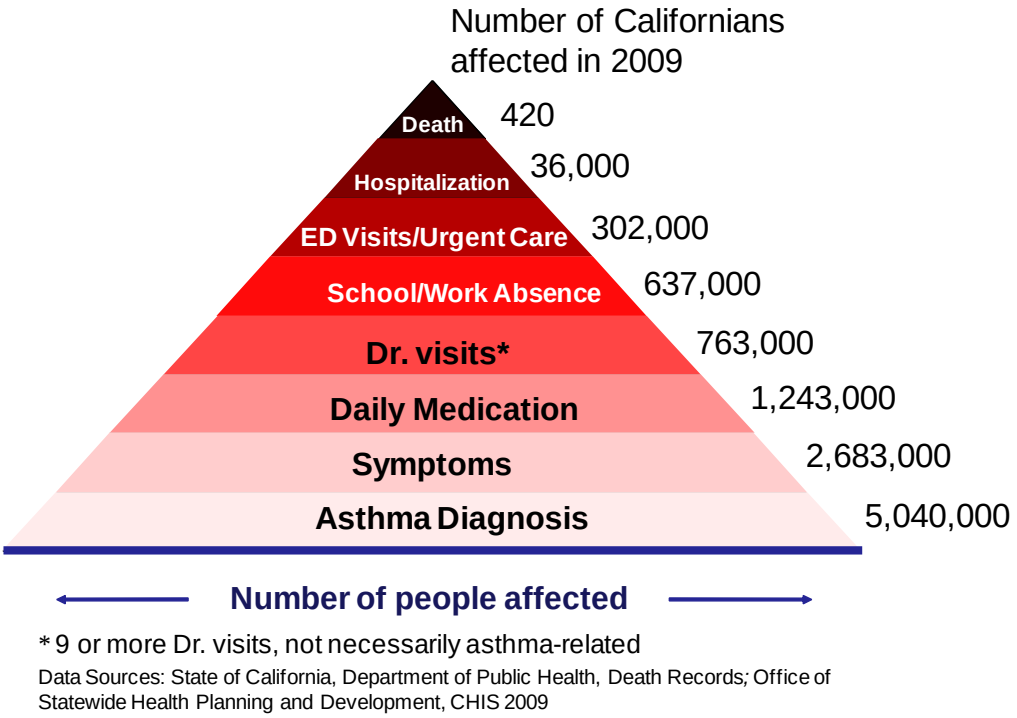
The objectives of this study were to: 1) characterize air pollution exposures by linking geocoded CHIS 2003 respondent residence locations to appropriate air monitoring stations and calculating annual average air pollutant concentrations ($O_3$, $PM_{10,}$ $PM_{2.5}$, and $NO_2$) from the nearest monitoring station (e.g., 10 km) or interpolated pollutant concentrations for a maximum of three monitoring stations within a specified radius (e.g., 50 km), and exceedance frequencies (e.g., number of days or hours above a certain cut-off point); 2) develop GIS-based residential annual average traffic density and distance to major roadways/freeways measures using data from the California Department of Transportation (Caltrans) for each CHIS 2003 respondent; 3) identify sub-populations (e.g., low-income, children, the elderly, rural/urban residents, and ethnic minorities) that have higher exposures to a single pollutant or pollutant mixes, and/or potentially greater vulnerability to these exposures; 4) determine whether the disproportionate burden of asthma or asthma-like symptoms among low SES individuals is associated with greater pollutant exposures, greater vulnerabilities, or both, including evaluating factors that contribute to or modify the impact of air pollution on these sub-populations; and 5) develop a report and disseminate the results to policy makers, public health and environmental agencies, community-based organizations, and the public.

## Previous Studies on Asthma Exacerbations and Pollutant Exposures among Vulnerable Populations

### Pollutant Impacts on Asthma

A wide-ranging spectrum of negative heath effects related to air pollution was recognized by the American Thoracic Society. These effects are ordered in the pyramid below according to their frequency of occurrence within the population of California (Figure 1). Though previous studies have mostly focused on the more extreme outcomes, such as hospitalizations and deaths, these outcomes have impacted a relatively small fraction of the population. When we consider that the ratio of asthma diagnoses to asthma-related deaths is about 10,000 to 1, it is clear that these less severe health effects are equally in need of attention because of the large of number of people they affect. As noted in Figure 1, an estimated 5.08 million people in the state of California live with an asthma diagnosis based on CHIS 2009 data. Of those, 2.7 million were affected by asthma-related symptoms and 1.2 million had to take a daily asthma medication; 637,000 missed school or work due to asthma, and 763,000 visited the doctor 9 or more times for any reason. Emergency department/urgent care visits due to asthma were reported by over 302,000 of those with an asthma diagnosis; 36,000 of those were hospitalized (data from the Office of Statewide Health Planning and Development, California), and 420 died in 2009 (data from the Department of Public Health, California). This study provides a unique and much needed opportunity to assess the spectrum of the impact of air pollution on all people with asthma or asthma-like symptoms in California.

Figure 1. Pyramid of Asthma Burden in California (Adapted from the American Thoracic Society)




Over the past few decades, studies have linked ozone ($O_3$), nitrogen dioxide ($NO_2$), and particulate matter (PM) exposure to negative respiratory health outcomes, including reduced lung function, respiratory inflammation, and lung congestion. In addition to these outcomes, studies connect greater air pollutant exposure to increases in asthma attacks and other asthma-related negative health events, such as ED/hospital visits, medication use, and absences from school. The following is a brief summary of the existing literature.

**Increased Asthma Symptoms and Medication Use:** Exposure to criteria pollutants is associated with increases in asthma symptoms and medication use. A study by Thurston and Lippmann observing asthma outcomes in children with moderate to severe asthma attending summer camp found that the children had 40% more asthma symptoms when $O_3$ levels increased from an average $O_3$ level of 84 ppb to 160 ppb (Thurston, Lippmann et al. 1997). Additionally, elevations in $O_3$ levels have been associated with increases in medication use among children (Gent, Triche et al. 2003; Yang, Holz et al. 2005).

Moreover, in panel studies among children with asthma, increased PM exposure was associated with increases in asthma symptoms (Ward and Ayres 2004). A study based in Southern California found that as exposure to PM increased, children that exhibited the most symptoms at baseline and were not taking asthma medication were most likely to experience increased asthma symptoms (Delfino, Zeiger et al. 1998). Similar associations between PM and asthma medication use have been noted by others (Pope, Dockery et al. 1991; Slaughter, Lumley et al. 2003; Kerkhof, Postma et al. 2010).

Similarly, studies indicated a positive relationship between $NO_2$ exposure and increases in both asthma symptoms (Mortimer, Neas et al. 2002; Delfino, Gong et al. 2003; McConnell, Berhane et al. 2003; Gauderman, Avol et al. 2005; Schildcrout, Sheppard et al. 2006) and

medication use (Gauderman, Avol et al. 2005; Schildcrout, Sheppard et al. 2006). For example, in a study of 208 children in 10 cities in Southern California, children were twice as likely to take asthma medication with increasing $NO_2$ exposure (Gauderman, Avol et al. 2005).

**Increased School Absences:** Increases in criteria pollutant levels coincide with increases in students' absence from school. A study performed in southern California found that a short-term, 20 ppb spike in $O_3$ levels was associated with an 82.9% increase in student absences due to respiratory illness (Gilliland, Berhane et al. 2001). Likewise, increased PM and $NO_2$ levels were associated with increases in school absences among inner city children with asthma from 7 cities across the U.S. (O'Connor, Neas et al. 2008).

**Increased Emergency Department (ED) visits/Hospitalizations:** Increases in exposure to criteria pollutants, such as $O_3$, have been linked to increases in ED visits and hospitalizations due to asthma-related events (Romieu, Meneses et al. 1995; Anderson, Ponce de Leon et al. 1998; Tolbert, Mulholland et al. 2000; Lin, Liu et al. 2008; Moore, Neugebauer et al. 2008; Meng, Rull et al. 2010). White et. al compared the number of ED visits due to respiratory problems to fluctuations in $O_3$ levels and noted a 37% increase in the number of visits to the ED subsequent to $O_3$ level increases (White, Etzel et al. 1994). The direct relationship between $O_3$ and ED visits/hospitalizations has been documented in both directions; as $O_3$ levels decrease, so do the number of asthma-related hospital visits. Following a change in traffic patterns due to the 1996 Summer Olympics and the resulting decrease in $O_3$ exposure, Atlanta children experienced a 42% decline in health care utilization for asthma (Friedman, Powell et al. 2001).

Furthermore, the number of ED visits has been shown to escalate as PM exposure increases. In a study among inner city children in Seattle, an 11% increase in asthma-related ED visits was observed as exposure to $PM_{2.5}$ increased (Norris, YoungPong et al. 1999). An association between daily PM2.5 and ED visits for asthma at lag days 2 and 3 was observed in the greater Tacoma, Washington area. The relative risk for lag day 2 was 1.04 and for lag day 3 was 1.03(Mar, Koenig et al. 2010).

Studies also demonstrated a positive relationship between $NO_2$ and ED visits/hospitalizations (Lin, Chen et al. 2003; Barnett, Williams et al. 2005; Villeneuve, Chen et al. 2007). Among them, a study in Barcelona, Spain, documented increases in ED visits corresponding with $NO_2$ exposure in both winter and summer months (Castellsague, Sunyer et al. 1995).

**Vulnerable Populations**

Air pollution affects people in all groups, spanning all ages, races, and income levels; however, the burden of the air pollution effects is not equally shared. Some sub-populations, such as low income and/or minority groups, children, and the elderly, have been shown to have higher exposures or increased risk for adverse asthma outcomes due to air pollution compared to the rest of the population.

**Children:** Children's physiology and activity patterns leave them more susceptible to the negative effects of air pollutants on their respiratory health (Schwartz 2004; Trasande and Thurston 2005; Bateson and Schwartz 2008). Children's lungs continue developing from birth to adolescence. Since their lungs are still developing, their respiratory extracellular lining fluid (RELF) is not as effective at protecting against the damaging effects of air pollutant penetration as the lining in adult lungs (Gilliland, McConnell et al. 1999). They are more receptive and

responsive to exposures because the surface area of their airways is smaller. Additionally, children often breath through their mouths, instead of their noses, so fewer air pollution particles are filtered out before reaching the lungs (Bateson and Schwartz 2008), compounded by the fact that children simply breathe more than adults. Higher breathing rates among children means they take in more air, and therefore potentially more air pollutants, than adults per unit of body weight (Arcus-Arth and Blaisdell 2007). In addition to physiological susceptibility, children more frequently come in contact with air pollution because they participate in outdoor activities. Children usually engage in over 5 times the amount of outdoor physical activity as adults (Wiley, Robinson et al. 1991; Wiley, Robinson et al. 1991) and do so during high $O_3$ periods, such as during the afternoon or summer.

**Elderly:** Though studies observing the effects of air pollution on asthma in the adult population are relatively rare, some studies have suggested that the elderly may be more susceptible to the effects of air pollutants. This vulnerability may be due to greater lifetime exposure and weaker immune system responses (Sandstrom, Frew et al. 2003), though studies also suggest that comorbidities, especially cardiovascular and respiratory diseases, may also contribute to increases in negative health outcomes related to asthma among the elderly population (Gouveia and Fletcher 2000; Aga, Samoli et al. 2003; Anderson, Atkinson et al. 2003; Sandstrom, Frew et al. 2003; Filleul, Rondeau et al. 2004; Gauderman, Avol et al. 2004; Meng, Wilhelm et al. 2007).

**Populations with Low Socioeconomic Status:** Populations with low SES have been shown to be more affected by pollutants due to their greater vulnerability or higher exposures (Clark, Brown et al. 1999; Ostro, Lipsett et al. 2001; Mortimer, Neas et al. 2002; Perera, Illman et al. 2002) Several studies reported disparities in pollution exposures by SES. For instance in California, census block groups in the lowest quartile of median family income were three times more likely to have high-traffic density than block groups in the highest income quartile (Gunier, Hertz et al. 2003). Children of color were also more likely to live in high traffic areas than white children (Gunier, Hertz et al. 2003). Studies in other states have reported low SES individuals are more likely to be exposed to $O_3$ (Korc 1996) and other pollutants (Neumann, Forman et al. 1998). Additionally, there is evidence that low SES populations are more affected than high SES populations when exposed to the same levels of air pollution. In Toronto, Canada, the risks of asthma-related physician visits for the low socioeconomic group were significantly greater than those for the high socioeconomic group when the two groups had comparable levels of $SO_2$ and $PM_{2.5}$ exposure (Burra, Moineddin et al. 2009). The high prevalence of frequent asthma symptoms among low income Californians has also been shown to be related to both higher traffic-related pollution exposures and increased vulnerability due to differences in overall health status and access to care; therefore, those in poverty appeared to be more strongly affected by heavy traffic near their residences than those above poverty (Meng, Wilhelm et al. 2008).

**Minorities:** Gwynn and Thurston (2001) also examined whether racial minorities are more adversely affected by ambient air pollution than their white counterparts and assessed the contribution of socioeconomic status to observed racial differences in pollution effects. They found attributable risks from air pollution (in terms of excess admissions per day per million persons) were larger for minorities than whites. However, when insurance status was used as an indicator of socioeconomic/health coverage status, higher relative risks were

indicated for the poor/working poor (i.e., those on Medicaid and the uninsured) than for those who were economically better off (i.e., the privately insured), even among non-Hispanic whites (Gwynn and Thurston 2001).

**Study Hypotheses**

The previous studies on asthma-related effects tend to focus on the impact of short-term (days or weeks) pollutant exposures on mortality and hospitalizations (Schwartz, Slater et al. 1993; Anderson, Ponce de Leon et al. 1998; Delfino, Murphy-Moulton et al. 1998; Sunyer, Basagana et al. 2002). However, death and hospitalizations represent just the tip of the iceberg of the overall asthma burden. More studies are needed to examine many outcome measures that affect a much larger population, such as ED visits, medication use, frequency of asthma symptoms, and school/work days missed due to asthma. Also, most of the studies have focused on the air pollution impacts on children; limited numbers of studies are available on the adult population. Previous studies indicate that vulnerable subpopulations, such as low-income and communities of color in California, have higher exposures to air pollution. Studies have also shown that children, the elderly (Babey, Hastert et al. 2007), racial/ethnic minorities (Meng, Babey et al. 2007), and low-income Californians (Babey, Hastert et al. 2007) suffer disproportionately from asthma and asthma-like symptoms. More studies are needed to examine whether the disproportionate asthma burden among these subpopulations is related to higher exposure to air pollutants, greater vulnerability due to low socioeconomic status and associated factors such as compromised health status, poor access to care, and behavioral risk factors, or to a combination of these factors. This study was designed to address the above mentioned gaps in the literature, and specifically to **provide much needed information on the effects of long-term air pollution exposure on asthma symptoms in especially vulnerable sub-populations, such as children, the elderly, racial/ethnic minorities, and low-income Californians.** As mentioned above, we defined "vulnerability" based on a "triple-jeopardy" theory (Jerrett, Burnett et al. 2001; Levy, Greco et al. 2002). Our specific study hypotheses were:

1) Among those with asthma or asthma-like symptoms, vulnerable sub-populations in California (e.g., racial/ethnic minorities and low-income individuals) have higher exposures to air pollution;
2) **Individuals with asthma** exposed to higher levels of air pollution are more likely to report adverse asthma outcomes, such as: asthma attacks or episodes, asthma emergency department (ED) visits, use of daily medication to control asthma, school or work absences, and daily/weekly asthma symptoms. **Individuals with asthma-like symptoms** (defined here as individuals without physician-diagnosed asthma but reported wheezing) and exposed to higher levels of air pollution are more likely to report: wheezing or whistling sound in the chest, attacks of wheezing or whistling, seeking medical care for such symptoms, and work/school days missed due to such symptoms;
3) Air pollution exposures, low socioeconomic status (SES), and certain "vulnerability factors" associated with low SES, exert independent adverse effects on individuals with asthma or asthma-like symptoms. The vulnerability factors examined were: **co-morbidity** (such as diabetes or heart disease); **access to care** (health insurance status, usual source of care); **disease management/asthma severity** (taking daily medication to

control asthma, receiving an asthma management plan); **health behaviors** (being overweight/obese, smoking, walking outdoor, engaging in physical activity); **exposure to indoor triggers** (environmental tobacco smoke and indoor allergens, cockroaches, dogs and cats); and **housing conditions** (single family dwelling or apartment, crowding); and

4) Higher pollutant exposures interact with these vulnerability factors resulting in greater air pollution impacts on asthma in vulnerable sub-populations (racial/ethnic minorities, low-income individuals).

## Background on the California Health Interview Survey (CHIS) 2003

CHIS is a population-based random-digit dial telephone survey of California's population that is conducted every two years. First conducted in 2001, CHIS is the largest health survey ever conducted in any state and one of the largest health surveys in the nation. CHIS is a collaborative project of the UCLA Center for Health Policy Research, the California Department of Health Services, and the Public Health Institute. CHIS collects extensive information for all age groups on health status, health conditions, health-related behaviors, health insurance coverage, access to health care services, and other health and development issues. The goal is to provide health planners, policymakers, state, county and city health agencies, and community organizations with information on the health and health care needs facing California's diverse population.

CHIS provides a representative sample of the state's non-institutionalized population. The CHIS sample is designed to meet two broad objectives: 1) provide local-level estimates for counties with populations of 100,000 or more; and 2) provide statewide estimates for California's overall population and its larger racial/ethnic groups, as well as for several smaller ethnic groups. To address the first objective, the sample was allocated by large counties (those with a population over 100,000) and aggregates of smaller counties (those with a population less than 100,000) with supplemental samples of selected populations and cities. To accomplish the second objective — assuring adequate sample sizes for larger racial/ethnic groups and some smaller ones, CHIS 2001 used two strategies. First, sufficient samples were allocated to the larger urban counties in which the populations of color disproportionately reside to generate adequate samples for major ethnic groups of color. Second, supplemental samples were designed to improve the sample size and precision of the estimates for specific ethnic groups. To capture the rich diversity of the California population, interviews were conducted in six languages: English, Spanish, Chinese (Mandarin and Cantonese dialects), Vietnamese, Korean, and Khmer (Cambodian). These languages were chosen based on research that identified these as the languages that would cover the largest number of Californians who did not speak English or did not speak English well enough to participate in an interview. As a result, CHIS allows us to study disparities in health status among California's most-represented racial and ethnic groups.

CHIS had a multi-stage sample design. First, the state was divided into 41 geographic sampling strata, including 33 single-county strata and 8 groups that included the 25 other counties with small population sizes. Second, within each geographic stratum, households were selected through random-digit dial (RDD), and within each household, an adult (age 18 and over) respondent was randomly selected. In addition, in those households with children (under age 12) or adolescents (ages 12-17) associated with the sampled adult, one child and one

adolescent were randomly sampled, so up to three interviews could have been completed in each sampled household. The sampled adult was interviewed, and the parent or guardian most knowledgeable about the health and care of the sampled child was interviewed. The sampled adolescent responded for him or herself, but only after a parent or guardian gave permission for the interview. Adjustment factors for the selection mechanisms have been incorporated into the data's sample weights.

CHIS collects information on major chronic diseases, such as asthma, heart disease, hypertension, cancer, arthritis and diabetes. Since many chronic diseases have multiple causes and are influenced by many factors, the development and control of these chronic diseases can be very complex. Therefore, it is important to examine the relationship between disease and exposure to hazards after controlling for confounding factors. For example, control of asthma exacerbations may not only relate to reducing exposures to environmental triggers, but also to improving access to timely and quality healthcare. In this regard, CHIS has advantages over many administrative data sources such as vital statistics, hospital discharge data, cancer registry data or claim data. These administrative data sets usually lack detailed information related to socioeconomic status, access to healthcare, and health risk behaviors. However, CHIS 2001 collected many measures for health outcomes, access to care, and socio-demographic information. Beginning with CHIS 2003, CHIS has collected residential address information for respondents. This geographic information allows us to link CHIS respondents' data to the air pollution data collected at fixed monitoring stations, as well as traffic data or other environmental hazard data. This linkage also allows us to assess the health effects of exposure to environmental hazards. These kinds of linkages are usually not possible or meaningful for NHIS and BRFSS since these surveys are not designed to provide information below the state level. Hospital Discharge data does provide patients' zip code information. However, this data source only contains information about people admitted to the hospital and is not a source of information on disease prevalence.

Westat, a private firm that specializes in statistical research and large-scale sample surveys, conducted the CHIS 2003 data collection. The overall response rate for CHIS 2003 is a composite of the screener completion rate (i.e., success in introducing the survey to a household and randomly selecting an adult to be interviewed), and the extended interview completion rate (i.e., success in getting the selected person to complete the full interview). In 2003, the screener completion rate was 55.9 percent, and the rate was higher for those households that could be sent a letter introducing them to the survey in advance. The extended interview completion rate was 60.0 percent for the adult survey. The CHIS response rate is comparable to response rates of other scientific telephone surveys in California, such as the California Behavioral Risk Factor Surveillance System (BRFSS) survey.

In summary, CHIS data provide the first-ever opportunity to provide population-based information examining the association between exposure to air pollution and adverse respiratory health outcomes while also incorporating socioeconomic status, disease management/asthma severity , risk factors such as smoking and obesity, and access to care. Such an effort would usually be very time-consuming and costly. The availability of CHIS data made this type of study possible with relatively modest means in terms of time and resources.

# II. MATERIALS AND METHODS

## Study Design

To investigate the effects of air pollution on those with asthma and asthma-like symptoms in California and to identify potentially vulnerable subgroups, we conducted a cross-sectional study linking California Health Interview Survey (CHIS) 2003 data to existing air pollutant and traffic data. First, we selected CHIS 2003 respondents with current asthma and those not diagnosed with asthma but reported experiencing asthma-like symptoms. We linked these respondents' residential addresses to the nearest government air monitoring station for each of four criteria pollutants ($O_3$, $PM_{10}$, $PM_{2.5}$, and $NO_2$). We then calculated annual pollutant averages for the 12-month period prior to respondents' CHIS interview dates. We also assessed traffic density and distance to roadways as proxies for traffic-related air pollution exposure. We performed univariate and multivariate logistic regression analyses to examine associations between air pollution and asthma outcomes. Interaction terms were used to evaluate increased vulnerability to pollutants among sub-populations.

## Study Population

CHIS 2003 interviews were conducted from August 2003 to February 2004. CHIS 2003 collected information on approximately 54,500 non-institutionalized Californians, including 12,500 children (<18 years of age). Respondents were asked if they had ever been told they have asthma by a doctor and at what age. In addition to asking about asthma outcomes, CHIS respondents never diagnosed with asthma were asked if they experienced any wheezing or whistling sound in their chests in the past 12 months. About 15% (n=1,889) of children (<18 years of age) and 12% (n=5,620) of adults reported a physician diagnosis of asthma at some point in their lives, here defined as "lifetime asthma" (Table 1). Among those with a lifetime asthma diagnosis, 4,811 (3,587 adults and 1,224 children) had "current asthma", defined as reporting that they still have asthma and/or that they had an asthma attack in the year prior to their CHIS interview. An additional 10% of Californians not reporting to ever have been diagnosed with asthma (n=5,522, 4,413 adults and 1,109 children) reported experiencing asthma-like symptoms, i.e., wheezing or whistling in the chest in the past year. We restricted our study population to those who lived in the same home or neighborhood for at least 9 months and some analyses were also limited to the respondents with geocodable home addresses. Three hundred and fifteen respondents with current asthma and 442 respondents with asthma-like symptoms were excluded because they did not live in the same neighborhood for at least 9 months. Residential geocodes were based on address (83.8%), nearest cross-streets (4.2%), or zip code (11.9%). For traffic density and distance to roadway analyses, geocodes based on residential zip code were excluded (n=537). For air pollution analyses, only respondents living within 5 miles of an air monitoring station were included to reduce potential exposure misclassification. However, sensitivity analyses were conducted for 3-, 5-, and 10-mile linkage distances.

Table 1. Number of CHIS 2003 adults and children with asthma or asthma-like symptoms

| | Adults | Children |
|---|---|---|
| **Lifetime Asthma** | 5,620 | 1,889 |
| ≥ 9 months in neighborhood | 5,236 | 1,783 |
| Geocoded by address or nearest cross-street | 4,595 | 1,579 |
| **Current Asthma** | 3,587 | 1,224 |
| ≥ 9 months in neighborhood | 3,343 | 1,153 |
| Geocoded by address or nearest cross-street | 2,941 | 1,018 |
| **Asthma-Like Symptoms** | 4,413 | 1,109 |
| ≥ 9 months in neighborhood | 4,129 | 951 |
| Geocoded by address or nearest cross-street | 3,629 | 833 |

## Measures of Air Pollutant Exposure

### Annual Average Air Pollution Concentrations for $O_3$, $PM_{10}$, $PM_{2.5}$, and $NO_2$

Annual air pollutant averages were calculated for the 12-month period prior to each respondent's CHIS interview date by linking respondents to the nearest government air monitoring station within 20 miles of their residential addresses. All mapping work was performed using ESRI ArcGIS software. In cases where residential address was not available, respondents' residential locations were geocoded based on nearest cross streets, and in some cases residential 5-digit zip codes. Pollutant averages were estimated using air pollution data from the CARB 2008 Air Quality Data DVD (Table 2).

Table 2. Description of pollutant averages calculated for CHIS 2003 respondents

| Pollutant | Exposure measure |
|---|---|
| $O_3$ | 12-month averages of 8 hour daily maximum values (dlygas.dbf, OZMX8ST)) |
| $NO_2$ | 12-month averages of daily (24-hour) averages (calculated using hourly data) |
| $PM_{10}$ & $PM_{2.5}$ | 12-month averages of daily (24-hour) averages (most stations have 24-hour averages; for the few stations that measure hourly, we used the CARB calculated 24-hr averages) |

For $O_3$, annual averages were based on daily 8-hr maximums provided by CARB (variable name OZMX8ST). To ensure sufficient monitoring data were available, we first estimated monthly averages, requiring at least 50% of daily values to be available (15 daily values/month).

For $NO_2$, we estimated daily (24-hr) averages based on hourly measurement data. We required at least 50% of hourly values be available per 24-hr period and at least 50% of hourly values be available during 8am-8pm. If these two criteria were not met, we recorded a missing value for the 24-hr average for that day. Then, we averaged the $NO_2$ 24-hr averages for each month, requiring at least 50% of daily values to be available per month, i.e. 15 daily values/month.

Most PM stations recorded 24-hr averages every 6 and 3 days for $PM_{10}$ and $PM_{2.5}$, respectively. For the few stations with hourly PM data, we used CARB's calculated daily (24-hr) averages. For $PM_{10}$, we required that at least 50% of expected values be available for each monitor frequency, i.e., at least 3 (out of 5) daily values per month for stations that monitored

every 6 days and at least 15 daily values per month for stations that monitored every day. For $PM_{2.5}$, we required at least 5 (out of 10) daily values to be available per month for stations that monitored every 3 days and at least 15 daily values per month for stations that monitored every day.

Some PM stations had collocated (multiple) monitors. In these cases, we checked the recorded data for each monitor to determine whether it met the above sufficiency criteria. If both monitors met the criteria, we averaged all available daily measures from both stations for the given month. If only one monitor met the criteria, then we used data from that monitor.

Finally, annual averages were then estimated based on monthly averages for subjects who had 12 monthly values available. For all pollutants, if data did not meet the sufficiency criteria defined above, we searched to see if there was another monitor measuring that pollutant within 20 miles. If a more distant station had more complete data that met the sufficiency criteria, data from that station was used to calculate exposure averages. We generated variables to record if there was no station available within 20 miles, and which station was used to generate the exposure average. We also recorded the distance to the station used to estimate the average.

We further restricted our study population to individuals residing within a relatively close proximity (5 miles $\approx$ 8 km) to a monitoring station, after sensitivity analyses were conducted for 3-, 5-, and 10-mile linkage distances.

**Exceedances of Federal and State Standards**

Annual exceedances of federal and state standards for $O_3$, $PM_{10}$, $PM_{2.5}$, and $NO_2$ were calculated for the 12-month period prior to each respondent's CHIS interview date, again linking respondents to the nearest government air monitoring station within 20 miles of their residential addresses (Table 3).

Table 3. List of exceedance exposure measures calculated for CHIS 2003 respondents

| Exceedance measure | Description |
|---|---|
| $O_3$ 1-hr (State) | Number of days in 12-months prior to interview date where 1-hour daily ozone max (OZMAX1HR) >0.09 ppm |
| $O_3$ 8-hr (State) | Number of days in 12-months prior to interview date where 8-hour daily ozone max (OZMX8ST) >0.070 ppm |
| $O_3$ 8-hr (Federal) | Number of days in 12-months prior to interview date where 8-hour daily ozone max (OZMX8ST) >0.08 ppm |
| $NO_2$ 1-hr (State) | Number of days in 12-months prior to interview date where 1-hr daily $NO_2$ max (NO2MAX1H) >0.18 ppm |
| $PM_{10}$ 24-hr (State) | Number of days in 12-months prior to interview date where 24-hour average $PM_{10}$ >50 ug/m3 |
| $PM_{10}$ 24-hr (Federal) | Number of days in 12-months prior to interview date where 24-hour average $PM_{10}$ >150 ug/m3 |
| $PM_{2.5}$ 24-hr (federal) | Number of days in 12-months prior to interview date where where 24-hour average $PM_{2.5}$ >35 ug/m3 |

For $NO_2$ and $O_3$, we used 1-hr and 8-hr daily maximum values provided by CARB to estimate number of exceedance days, i.e., days above state and federal standards. For $NO_2$, the number of exceedance days was counted where NO2MAX1H>0.18 ppm for the state 1-hour standard. There is no equivalent federal standard. Similar to the annual average air pollution averages, we required at least 50% of daily values per month to be available to generate a non-missing monthly count value. Because almost 100% of the study population had no days exceeding the $NO_2$ standard, this measure was not used in the analyses.

For $O_3$, the number of exceedance days was counted where OZMAX1HR>0.09 ppm for the state 1-hr standard. There is no federal 1-hr standard. We also counted exceedance days where OZMX8ST>0.070 ppm for the state 8-hr standard and where OZMX8ST>0.08 ppm for the federal 8-hr standard. Again, we required at least 50% of daily values per month be available. The $O_3$ federal 8-hr standard was not used in the regression analyses comparing quartiles because more than 25% had a value of 0, resulting in having no < 25$^{th}$ percentile reference group to use.

For $PM_{10}$, we counted the number of days where 24-hr averages>50 μg/m$^3$ (state 24-hr standard) and 24-hr average>150 μg/m$^3$ (federal 24-hr standard), requiring at least 50% of expected values for each monitor frequency (i.e., at least 3 (out of 5) daily values per month for stations that monitored every 6 days and at least 15 daily values per month for stations that monitored every day.

For $PM_{2.5}$, we counted the number of days where 24-hr averages>35 μg/m$^3$ for the federal 24-hr standard. There is no state 24-hr standard. We required at least 50% of expected values for each monitor frequency (i.e., at least 5 (out of 10) daily values per month for stations that monitored every 3 days and at least 15 daily values per month for stations that monitored every day.

For PM stations with collocated monitors, we checked whether each station met the above sufficiency criteria. If both monitors met the criteria, we averaged available daily measures from both stations for the given month. If only one monitor met the criteria, then we used the data from that monitor.

If data were sufficient, we took the sum of monthly counts to generate final annual exceedance counts for each pollutant. If data did not meet the sufficiency criteria defined above, we searched to see if there was another monitor measuring that pollutant within 20 miles. If a more distant station had more complete data, that station was used to calculate the exceedance value. Again, information was recorded on distance to station and whether the closest or a more distant station was used due to implementation of the sufficiency criteria.

**Interpolated Pollutant Concentrations**

We originally proposed to interpolate air pollution measurement data from monitoring stations assigned to residential locations in rural areas using inverse distance weighting and a maximum of three monitoring stations for each interpolation. However, even expanding the interpolation radius out to 10 miles, only a small percent of rural subjects (9% (n=48), 21% (n=177), 14% (n=120) and 5% (n=37) for $NO_2$, $O_3$, $PM_{10}$ and $PM_{2.5}$, respectively) had more than one monitoring station available to inform such modeling. Since interpolation would not be relevant for ≥80% of the rural subjects, even with a large 10 mile radius, we excluded this exposure modeling method.

## Measures of Traffic Exposure

We generated several measures based on distance to and traffic levels on roadways near CHIS respondent homes as proxies for traffic exhaust exposures. We estimated traffic density within 500, 750, and 1000 feet around each subject's home location using Tele Atlas' Dynamap traffic count data from Spatial Insights Inc., Bethesda, MD. These data were imputed to all road segments in the state based on roadway type. We also calculated the distance from

each home to the nearest interstate highway, state highway, and major road using the Tele Atlas Dynamap 2000 roadway map. All work was completed using ESRI's ArcGIS software.

**State-wide Imputation of Tele Atlas Traffic Data**

We collaborated with Drs. Michael Jerrett and Jason Su at UC Berkeley to use Tele Atlas' Dynamap data (Spatial Insights Inc., Bethesda, MD) to derive a state-wide traffic count map for estimating residential traffic density. We used an imputation method to attribute available measured traffic counts to un-counted road segments in the state. We used Tele Atlas Dynamap 2000 as our roadway map for the imputation because the underlying road network had the most accurate spatial representation when compared to digital orthophotos. The Tele Atlas Dynamap traffic data (in the form of annual average daily traffic or AADT) were combined into a mosaic from individual county files and repeated road segments were removed. Measured traffic counts were available for 2.0% of the road segments in California (56734 out of 2784428 segments) during the period from 1987 to 2005 (Table 4). For the imputation, the median traffic count from measured road segments within a given road category was assigned to un-counted road segments within the same category. The road feature classification codes (FCC) were aggregated into the following seven road categories for the imputation: (1) primary road with limited access (i.e., interstate highway: A1), (2) primary road without limited access (i.e., state highway: A2), (3) secondary and connecting road (i.e., major road: A3), (4) local, neighborhood or rural road (A4), (5) vehicle trail (A5), (6) road ramp (A6), and (7) bicycle, pedestrian trail or drive way (A7).

Table 4. Distribution of traffic volumes for major roadway categories based on Tele Atlas Dynamap traffic data – State of California

| Road category[a] | Traffic volume measurements | | | | | | Tele Atlas data | |
|---|---|---|---|---|---|---|---|---|
| | # roads | Minimum | Maximum | Mean | Median | Std | # roads | % |
| A1 | 6076 | 1300 | 210500 | 56229.93 | 46500 | 40894.38 | 76286 | 7.96 |
| A2 | 3419 | 210 | 76000 | 13154.88 | 11150 | 10406.72 | 44430 | 7.70 |
| A3 | 27242 | 10 | 239000 | 12253 | 10463 | 9342.19 | 442460 | 6.16 |
| A4 | 19824 | 1 | 88680 | 4003.83 | 2317 | 4860.98 | 1965782 | 1.01 |
| A5 | 3 | 564 | 2100 | 1092.67 | 614 | 872.73 | 56049 | 0.01 |
| A6 | 158 | 906 | 210500 | 25983.21 | 14150 | 32918.15 | 106883 | 0.15 |
| A7 | 12 | 95 | 29900 | 6576.42 | 1280 | 10559.1 | 92538 | 0.01 |
| Total: | 56734 | | | | | | 2784428 | 2.04 |

[a]A1: Primary highway with limited access; A2: primary road without limited access; A3: secondary and connecting road; A4: local, neighborhood and rural road; A5: vehicular trail; A6: road access ramp; A7: road as other thoroughfare.

**Residential Traffic Density**

Mapped home locations for CHIS 2003 respondents were then overlaid with the Tele Atlas Dynamap 2000 roadway map containing the imputed traffic count data. We drew 500-, 750-, and 1000-foot buffers around each subject's home location and identified all roadways within these buffers. Similar to Gunier et al.(Gunier, Hertz et al. 2003) and Reynolds et al.(Reynolds, Von Behren et al. 2004), the traffic density value for each subject was estimated by first calculating the Vehicle Meters Traveled (VMT) for each road segment within the buffered area. VMT was estimated by multiplying the AADT value by the corresponding road

segment length. Traffic density was then calculated as the sum of the VMT for all road segments in the buffer divided by the area of the buffer, i.e.,

$$TD = \sum(AADT \times L)/A_B,$$

where TD is traffic density (vehicles x meters/day/meters$^2$), AADT the annual average daily traffic count (vehicles/day), L the length of roadway segment (meters), and $A_B$ the area of the buffer: 500 ft (152.4 m): 72966 m$^2$; 750 ft (228.6 m): 164173 m$^2$; 1000 ft (304.8 m): 291864 m$^2$.

**Distance to Roadways**

Again using the Tele Atlas Dynamap 2000 roadway map, we also calculated distance from mapped home locations to nearest interstate highways, state highways and major roads. Distance to roadway measures do not rely on the availability of traffic data near respondents' residences and, therefore, can be calculated for respondents without using imputation. Also, freeways and highways may be particularly important exposures for those with respiratory problems, since they have more diesel truck traffic and higher traffic volumes than smaller roads. For these analyses, we determined the distance in meters from subjects' homes to the nearest interstate highway, state highway, and major road (see Table 5 for a description of roadway groupings).

Table 5. Tele Atlas roadway groupings for distance to roadway calculations

| Tele Atlas FCC code | Tele Atlas Description | Our grouping |
|---|---|---|
| A10 | Primary interstate highway, major category | Interstate highways |
| A11 | Primary limited access or interstate highway, unseparated | Interstate highways |
| A12 | Primary limited access or interstate highway, unseparated, in | Interstate highways |
| A15 | Primary limited access or interstate highway, separated | Interstate highways |
| A16 | Primary limited access or interstate highway, separated, in | Interstate highways |
| A17 | Primary limited access or interstate highway, separated, | Interstate highways |
| A20 | Primary US and State highways, major category | State highways |
| A21 | Primary US and State highways, unseparated | State highways |
| A22 | Primary US and State highways, unseparated, in tunnel | State highways |
| A25 | Primary US and State highways, separated | State highways |
| A26 | Primary US and State highways, separated, tunnel | State highways |
| A27 | Primary US and State highways, separated, underpassing | State highways |
| A30 | Secondary State and County highways, major category | Major road |
| A31 | Secondary State and County highways, unseparated | Major road |
| A32 | Secondary State and County highways, unseparated, in tunnel | Major road |
| A33 | Secondary State and County highways, unseparated, | Major road |
| A34 | Secondary State and County highways, unseparated, with rail | Major road |
| A35 | Secondary State and County highways, separated | Major road |
| A36 | Secondary State and County highways, separated, in tunnel | Major road |
| A37 | Secondary State and County highways, separated, underpassing | Major road |
| A38 | Secondary State and County highways, separated, with center | Major road |

## Respiratory Health Outcomes in Respondents with Diagnosed and Undiagnosed Asthma

CHIS collected information regarding respiratory health outcomes from respondents with and without a diagnosis of asthma. Respondents with current asthma were asked to report *how often have you had asthma symptoms such as coughing, wheezing, shortness of breath, chest tightness or phlegm (not at all, less than every month, every month, every week, or every day)* and whether or not they experienced the following asthma-related health outcomes in the 12 months prior to their CHIS interview date: ED or urgent care visits, use of daily medication, and missed day care/school or work days. Although respondents were also asked the number of doctor visits for any reason during this period, we omitted this variable as an asthma outcome from our analyses because the question was not specific to doctor visits for asthma. Also, teenagers (12-17 years of age) were not asked if they missed school due to asthma, so this outcome is only available for children ages 0-11 years.

In addition to asking about asthma outcomes among respondents with a lifetime asthma diagnosis, CHIS 2003 contained a series of questions on asthma-like symptoms, i.e. wheezing in respondents never diagnosed with asthma. They were asked about the number of wheezing attacks, the number of times they sought medical attention for the breathing problem, and whether they missed any days of work or school/day care due to these problems in the 12 months prior to interview. Teenagers were not asked about how many attacks of wheezing or whistling they experienced or if they missed any school days due to wheezing. In summary, we examined the following health effect measures reported by respondents as occurring within the 12 months preceding the interview:

*Health effect measures for CHIS 2003 respondents for CHIS 2003 child and adult respondents (except those noted below) with physician-diagnosed asthma:*

The following measure is applied to those with a **lifetime asthma diagnosis** only:

- Asthma episode or attack (dichotomous);

The following measures are applied to those with **current asthma** only:

- Asthma symptoms among those with current asthma: persistent asthma (with daily or weekly symptoms) vs. intermittent asthma (with monthly, less than monthly, or no symptoms);
- Currently taking daily medication to control asthma (dichotomous);
- ED/urgent care clinic visit for asthma, abbreviated to ED visits throughout the report (dichotomous);
- Two or more work days missed due to asthma, adults only (dichotomous); and
- Two or more days of day care or school missed due to asthma, children ages 0-11 only (dichotomous).

*Health effect measures for CHIS 2003 child and adult respondents (except those noted below)* ***with asthma-like symptoms*** *among those without asthma diagnoses:*

- Asthma-like symptoms, wheezing or whistling sound in chest (dichotomous);

- Two or more attacks of wheezing or whistling (dichotomous), excluding teen respondents;
- Sought medical care for such symptoms at least once (dichotomous);
- Two or more work days missed due to such symptoms, adults only (dichotomous); and
- Two or more days of day care or school missed due to such symptoms, children ages 0-11 only (dichotomous).

## Potential Confounders and Vulnerability Characteristics

CHIS is a rich data source; in addition to health outcomes, information was collected on several important potential confounders and vulnerability characteristics for asthma or asthma-like symptoms. Particularly relevant to this study, CHIS 2003 collected information on basic demographics, overall health status, access to health care, asthma disease management, health behaviors, indoor asthma triggers, and housing conditions. For all the adjusted analyses, we included age, sex, race/ethnicity and federal poverty level (FPL) as covariates. We considered the following vulnerability-related risk factors as potential confounders of air pollution health effects estimates:

- **Access to health care**: having health insurance currently, having experienced delays in getting care for any medical reason, having a usual source of care;
- **Overall health status**: co-morbidity such as diabetes or heart disease;
- **Disease management/asthma severity indicators:** year of asthma diagnosis, receiving an asthma management plan, taking daily medication to control asthma;
- **Health behaviors:** being overweight/obese, smoking, and walking for transportation or leisure;
- **Housing conditions:** type of housing, such as single family dwelling or apartment, and crowding;
- **Indoor triggers**: smoking in the home, dog/cat in the home, cockroaches in the home, and
- **Residence**: urban/rural residence, length of residence at current address/neighborhood.

CHIS established if respondents' household income was above or below the FPL based on federal poverty guidelines. For example, 100% of the FPL means an annual household income of $8,980 for a one member household, $12,120 for a two member household, $15,260 for a three member household, and $18,400 for a four member household, while 200% of the FPL means household income was double the relevant amount. We decided to use 200% of the FPL as a cut point since the cost of living in California is higher in general than in most parts of the country due to housing costs.

CHIS used the U.S. Center for Disease Control body mass index (BMI) criteria to define overweight or obese based on self-reported height and weight. For instance, for adult men and women, the categories are underweight $\leq$18.5 BMI, normal weight=18.5–24.9 BMI, overweight=25–29.9 BMI and obese=BMI of 30 or greater.

CHIS assigned respondents to four levels of urbanicity based on definitions developed by the commercial company Claritas: 1) urban, 2) 2$^{nd}$ city, 3) suburban, 4) small town/rural. Using

population density of an area and neighboring areas, Claritas classified mega-cities with density scores of 85-99 (on scale of 0 to 99) as "urban"; cities and big towns with density scores of 40-85 as "2nd cities"; suburbs of urban and 2nd city areas, with density scores of 40-90 as "suburban"; and exurbs and towns with density less than 40 as "town/rural". CHIS classified respondents based on the most prevalent Claritas household type in their residential zip code. Household crowding refers to households with more than one occupant per room (not counting bathrooms) based on the U.S. Census Bureau definition.

## Statistical Methods

Once the data were linked, we conducted analyses to examine whether the disproportionate burden of asthma or asthma-like symptoms among low SES individuals is associated with greater pollutant exposures, greater vulnerabilities or both (Objective 3-4). Under Objective 3, we tested Hypothesis 1: Among those with asthma or asthma-like symptoms, vulnerable sub-populations in California (e.g., racial/ethnic minorities and low-income individuals) have higher exposures to air pollution. We examined distributions of exposures for the four criteria air pollutants and traffic metrics among CHIS 2003 respondents and tested whether exposures varied by sub-populations, characterized by rural and urban residency (rural/town, urban, second city and suburban), age (0-5, 6-11, 12-17, 18-34, 35-64 and ≥65 years), gender, income level (0-199% FPL, 200-399% FPL, and ≥400% FPL), and by racial and ethnic group (white, Latino, African American, Alaskan Native/American Indian, Asian and Pacific Islanders and other minorities). We also examined differences in distributions of health outcomes across these subgroups. We performed t-tests and z-tests for proportions to identify disparities in pollutant exposures and respiratory outcomes within these sub-populations.

To examine whether there were positive associations between air pollution exposure and the respiratory outcomes of interest, and to identify additional factors that might contribute to the variations in association (Objectives 3 and 4), our analysis was comprised of several steps. First, we tested our hypothesis that individuals with asthma exposed to higher levels of air pollution are more likely to report adverse asthma outcomes, such as: asthma attacks or episodes, asthma emergency department (ED) visits, use of daily medication to control asthma, school or work absences, and daily/weekly asthma symptoms. Individuals with asthma-like symptoms and exposed to higher levels of air pollution are more likely to report: wheezing or whistling sound in the chest, attacks of wheezing or whistling (Hypothesis 2). We examined crude associations between individual air pollutants and asthma outcomes using tabular analyses and logistic regression modeling adjusting for age, sex, race/ethnicity and federal poverty level. For regression analyses, annual pollutant averages were included in the model as continuous measures scaled by a fixed number of units depending on the distributions of the pollutant averages and as commonly practiced in the literature. Specifically, we scaled $O_3$ by 10 ppb, $NO_2$ by 10 ppb, $PM_{10}$ by 10 μg/m$^3$, and additionally we scaled $PM_{2.5}$ by 5 μg/m$^3$ based on the distribution after univariate analysis. Categorical variables were used for exceedance days and traffic measures to explore the shape of the exposure-outcome associations and evaluate possible exposure-response relations. To illustrate, we fit the following logistic model for the binary outcome asthma (noted here as A, where A=1 if a respondent reported persistent asthma (daily/weekly symptoms); a similar model would apply if we considered A to be an indicator of asthma-like symptom prevalence):

$$\text{logit}(\Pr(A=1 \mid O_3)) = \beta_0 + \beta_1 (O_3)$$

Here exp ($\beta_1$) represents the odds ratio for asthma corresponding to a 10 ppb change in $O_3$ exposure.

Second, to test if air pollution exposures, low SES status, and certain vulnerability factors associated with low SES exert independent adverse effects on individuals with asthma or asthma-like symptoms (Hypothesis 3), we used multiple logistic regression analyses to quantify associations between air pollution exposures and outcomes after including and excluding suspected confounders, such as insurance status, cigarette smoking, and delays in care. We fit three models for adults: (1) a base model, which includes each pollutant measure individually, plus age, race, federal poverty level, and sex; (2) the base model plus adjustment for major possible confounders related to access to care, health behaviors and overall health status, such as insurance status, overweight or obesity, heart disease, work status, and smoking status; and (3) the base model, including other possible confounders, such as urban vs. rural residence, having a usual source of care, having a delay in care for any medical reason, age of asthma onset, taking a daily asthma medication, having an asthma management plan, the presence of household smoking, having a dog or cat in the home, having cockroaches in the home, housing type, household crowding, having diabetes, and walking for leisure or transportation. For Model 3, we purposely excluded additional factors from Model 2, namely insurance status, overweight or obese, heart disease, work status, and smoking status, since some of them may be highly correlated with variables in Model 2, e.g. having heart disease and diabetes. After the models were selected, covariates that could be reasonably related were tested for possible correlations, and no significant correlations were observed for covariates in the same model. We focused the Model 1-Model 3 analyses on three asthma-related outcomes: ED visits, daily asthma medication use, and 2 or more missed work days due to asthma in relationship to three criteria pollutants ($O_3$, $PM_{10}$, $PM_{2.5}$) for adults and use of daily asthma medication in relationship with $PM_{2.5}$ exposures for children. For children, the base model was the same as the base model for adults. In Model 2, we included the base model, plus adjusted for major possible confounders among children, such as insurance status, the presence of household smoking, having a dog or cat in the home, and having cockroaches in the home; Model 3 included the base model, as well as other possible confounders, such as urban vs. rural residence, having a delay in care for any medical reason, taking a daily asthma medication, having an asthma management plan, housing type, and household crowding.

Third, we tested the hypothesis that higher pollutant exposures interact with these vulnerability factors resulting in greater air pollution impacts on asthma in vulnerable sub-populations, i.e. racial/ethnic minorities, and low-income individuals (Hypothesis 4). We examined interactions between exposure and sub-populations characterized by age, race/ethnicity, income, and urban/rural residency. If an interaction term was statistically significant (based on a p-value $\leq 0.05$), we calculated the interaction odds ratios using the formula:

$$OR(x)=\exp(b_1+b_x)$$

where 1 represents the reference group and x represents the comparison group. We then calculated the standard error (SE) using the formula:

$$SE(x)=\sqrt{(var_1+var_x+2cov_{1x})}$$

and used the standard error to calculate the confidence intervals (CI) for each interaction odds ratio, To calculate the CIs we used the formula:

$$95\%\ CI\ (x)=\exp[b_1+b_x \pm 1.96*SE(x)]$$

If an interaction term was statistically significant (based on a p-value ≤ 0.05), we also conducted stratified analyses, for example, by income level or racial/ethnic group. None of the stratified analyses produced meaningful results (at least one group's confidence intervals crossed the null) due to insufficient sample size (results not reported). As a result, we were unable to estimate population attributable risk (PAR) within the sub-group strata.

In addition to the above mentioned analyses, we also performed several sensitivity analyses. First, we stratified on length of residence in the same home or neighborhood (<3 years versus ≥3 years) to examine whether associations between air pollution exposure and odds of reporting asthma symptoms are greater in long-term residents who have been consistently exposed to higher pollution for a longer period of time. We also compared unemployed with employed adults to examine the influence of potentially greater measurement error in residential exposure measures for employed adults due to additional exposures incurred while commuting or at the workplace. Additionally, we examined changes in air pollution estimates by residential distance to nearest monitoring station (3-, 5- and 10 miles), assuming exposure measures for subjects living closer to a station are less misclassified. Finally, we conducted stratified analyses based on asthma medication use assuming that air pollution may have different effects on those taking medication and those not taking medication.

All analyses incorporated sampling weights that adjust for the unequal probabilities of selection into the CHIS sample. In our adjusted analyses, some study respondents were excluded due to missing data for exposure measures. Final sample sizes for each model are reported in the results tables. Additionally, weighted population estimates were calculated using a weight variable constructed through a complex, iterative process; the weight variable was then applied to the sample data. Separate weights were created for adults, children, and adolescents, which were then used to calculate statewide estimates representative of the entire state population. As a result, CHIS 2003 estimates were consistent with the 2003 California Department of Finance (DOF) Population Projections.

Air pollution, traffic and distance to roadway measures were checked for accuracy and completeness by inspecting the raw data files and univariate statistics for the measures. For many of the descriptive and regression analyses, SAS macros were developed with our statistical staff to expedite the analysis process and reduce the possibility for human error while cutting and pasting results into tables.

# III. RESULTS

## CHIS 2003 Respondents with Current Asthma

### Exposure Distributions for CHIS 2003 Respondents with Current Asthma

Distributions of annual average pollutant exposures, exceedance days, and traffic density are shown in Table 6 in the Appendix. Among adults with current asthma, annual average exposures to $O_3$ ranged from 22.8 to 63.5 ppb, with a mean of 41.6 ppb, while $NO_2$ averages ranged from 1.6 to 36.1 ppb, with a mean of 21.1 ppb. Annual averages ranged from 12.3 μg/m$^3$ to 80.1 μg/m$^3$ for $PM_{10}$ (mean=28.6 μg/m$^3$) and 4.1 μg/m$^3$ to 27.5 μg/m$^3$ for $PM_{2.5}$ (mean=16.0 μg/m$^3$).

Based on the 1-hr state standard, adults with current asthma had a maximum of 122 $O_3$ exceedance days and an average of 22.4 $O_3$ exceedance days in the year prior to CHIS interview; under the 8-hr state standard, there was a maximum of 153 exceedance days and a mean of 31.1 $O_3$ exceedance days. Using the 8-hr federal standard, there was a maximum of 114 exceedance days and an average of 16.8 exceedance days. Based on the $PM_{10}$ federal standard, the maximum number of exceedance days was 4 days (mean=0.1 days); when using the state standard, the maximum was 66 exceedance days (mean=7.2 days). For $PM_{2.5}$, the maximum number of days exceeding the federal standard was 54 days, with an average of 15.5 exceedance days.

The mean traffic density within a 750-foot buffer was 66.0 VMT/day/meters$^2$, with a minimum of 0.09 and a maximum of 583.0.

Among children with current asthma, annual average $O_3$ exposure ranged from 23.0 ppb to 64.2 ppb with a mean of 41.3 ppb (Table 6 in Appendix). Annual $PM_{10}$ averages ranged from 13.0-80.1 μg/m$^3$ (mean=30.0 μg/m$^3$), and $PM_{2.5}$ annual average exposure ranged from 7.4 to 26.2 μg/m$^3$ (mean=16.8 μg/m$^3$). $NO_2$ annual averages varied from 1.6-36.0 ppb, with a mean of 22.0 ppb.

The maximum number of $O_3$ exceedance days was 122 (mean=24.9 days) under the 1-hr state standard, 153 under the 8-hr state standard (mean=33.5 days), and 114 (mean=18.5 days) under the 8-hr federal standard. The maximum number of $PM_{10}$ exceedance days was 4 (mean=0.1 days) under the federal standard and 65 (mean=7.8 days) under the state standard. The maximum number exceeding the federal $PM_{2.5}$ standard was 54, with an average 17.5 exceedance days. Traffic density ranged from 1.1-793.4 VMT/day/meters$^2$, with an average of 70.1 among children with current asthma (Table 6 in Appendix).

Frequencies for distance to roadway measures are shown in Table 7 in the Appendix. Less than 10% of adults with current asthma lived either within 300 meters of a state highway (5.4%) or interstate highway (9.9%). One-fifth (20.5%) of adults with current asthma lived within 50 meters of a major road. Five percent of children with current asthma lived within 300 meters of a state highway, and 12.1% lived within 300 meters of an interstate highway. One-fifth (19.1%) of children with current asthma lived within 50 meters of a major road.

### Correlations among Air Pollution Exposure Estimates

We estimated Pearson correlation coefficients for exposure metrics assigned to CHIS 2003 respondents with current asthma (Table 8 in Appendix). Annual average exposure

estimates for $PM_{10}$ and $PM_{2.5}$ were strongly correlated (r=0.78), as were estimates for $PM_{2.5}$ and $NO_2$ (r=0.72). Moderate positive correlations were observed between $PM_{10}$ and $NO_2$ (r=0.56), as well as $PM_{10}$ and $O_3$ (r=0.49). Annual average exposures were strongly correlated with exceedance measures for $O_3$ (r≈0.8), while $PM_{10}$ annual averages were strongly correlated with exceedances of the state (r=0.81) but not federal (r=0.43) standards. Annual average $PM_{2.5}$ exposures were moderately correlated with exceedances of the federal standard (r=0.69). There were low level correlations between annual average exposures and exceedance measures across pollutants. The traffic density and distance to roadway measures were weakly correlated with the criteria pollutant exposure metrics and with each other.

**Health Outcomes and Characteristics of Adults and Children with Current Asthma**

Table 9 below shows prevalence of asthma outcomes among respondents with current asthma. Among respondents with lifetime asthma, 35.0% of adults experienced an asthma attack in the year prior to their interview. Among adults with current asthma, the prevalence of daily or weekly symptoms was 29.8%. About half of adults with current asthma (47.6%) reported taking daily asthma medication, and 16.4% reported an ED visit within the past year. Additionally, 13.2% of adults with current asthma missed work at least twice.

Thirty-six percent of children with lifetime asthma experienced an asthma attack in the year prior to the interview. Of children with current asthma, 11.7% experienced daily or weekly symptoms, 21.7% had visited the ED, and 37.0% took daily asthma medication for their illness. Close to half of children with current asthma (45.9%) missed at least two school days.

Table 9. Prevalence of asthma outcomes for CHIS 2003 adults and children with current asthma[a]

| | Adults (≥18 years) | | Children (< 18 years) | |
|---|---|---|---|---|
| Outcome | n | % (Weighted) | n | % (Weighted) |
| Asthma Attack[b] | 1,951 | 35.0 | 664 | 36.0 |
| ED visits | 500 | 16.4 | 237 | 21.7 |
| Daily Asthma Medication | 1,653 | 47.6 | 421 | 37.0 |
| Missed At Least 2 Work/School Days Due to Asthma[c] | 347 | 13.2 | 294 | 45.9 |
| Daily/Weekly Asthma Symptoms | 1,058 | 29.8 | 129 | 11.7 |

[a]For CHIS 2003, current asthma is defined as reporting an asthma attack in the previous 12 months or answering yes to the question, "Do you still have asthma?"; only those who lived in the same home or neighborhood for at least 9 months were included.
[b]Respondents ever diagnosed with asthma who lived in the same home or neighborhood for at least 9 months were included.
[c]Data not collected for teen respondents.

Demographic characteristics of respondents with current asthma are shown in Table 10 in the Appendix. Among adults with current asthma, there were more women than men (65.3% vs. 34.7%). The majority of adult respondents with current asthma were between the ages of 35-64 (55.0%); 28.8% were 18-34, and 16.2% were 65 years old or older. The racial/ethnic distribution of the adult population with current asthma was as follows: 60.3% white, 17.3% Latino, 12.0% Asian/other, 8.4% African American, and 2.0% American Indian/Alaska Native. Close to a third (34.3%) of adults with current asthma had received a high school diploma at most, while 43% had attended college or vocational school, and 11.3% had attended graduate school. The remaining 11.4% were under the age of 25, an age group that is often still pursuing

an education, so educational attainment was not assessed for these respondents. Nearly two-fifths (38.2%) of respondents with current asthma were unemployed. Approximately one-third (32.5%) of adults with current asthma lived below 200% of the federal poverty level (FPL), 23.8% lived between 200-399% of the FPL, and the remaining 43.6% lived at or above 400% of the FPL.

While the majority of respondents were insured throughout the previous year, 16.1% of adults with current asthma were either not insured or only insured for part of the year. Similarly, more than 90% of adult respondents with current asthma reported a usual source of care; however, 21.0% reported a delay in needed care for any medical reason in the last 12 months. Only 22.1% of adults with current asthma reported visiting the doctor 0-1 time for any reason during the past year, while 45.4 % visited the doctor 2-5 times and 32.5% visited 6 or more times in the past year. The majority were diagnosed with asthma in adulthood. Nearly half (49.8%) of adults with current asthma were diagnosed between the ages of 18-64, and 5.4% were diagnosed at the age of 65 or older. Approximately a third (33.9%) were diagnosed between the ages of 0 and 11, and 10.9% were diagnosed between the ages of 12-17. Just less than half of adults with current asthma (47.6%) reported taking asthma medication daily, and only 37.6% said they had an asthma management plan.

When asked about current health status, 32.8% of adult respondents with current asthma stated their health status to be either poor or fair. The remaining 67.2% self-reported their health as good, very good, or excellent. Heart disease was reported in 11.3% of adults with current asthma; among them, 31.7% reported congestive heart failure. Based on BMI, 61.7% of adults with current asthma were classified as overweight or obese, as opposed to normal or underweight; 9.5% reported being diabetic, and 1.0% reported being borderline diabetic. While 45.5% of adults stated they currently or previously smoked, only 11.1% said they lived in a household with a current smoker. Nearly three-fourths (71.1%) of respondents reported walking for transportation or leisure. Almost half (47.7%) had dogs or cats in the home, and cockroaches were reported in the home among 12.2% of adults with current asthma.

The residences of most adults with current asthma were classified as urban (39.9%), as compared to 2$^{nd}$ city (27.5%), suburban (19.8%), or town/rural (12.7%). Most adult respondents (79.9%) had lived at their current addresses for 3 or more years. Two-thirds of adults (66.7%) stated they lived in houses, while the remainder described their housing units as apartments, duplexes, or mobile homes, and 16.3% of adults reported household crowding.

Child respondents with current asthma were 58.4% boys and 41.6% girls (Table 10 in Appendix). One-fifth (21.1%) of the children with current asthma were between the ages of 0-5, 37.5% were ages 6-11, and 41.4% were ages 12-17. Children with current asthma were 43.5% white, 26.9% Latino, 12.1% Asian/other, 14.6% African American, and 2.9% American Indian/Alaska Native. Over a third (38.3%) of children with current asthma had parents or guardians who had completed a high school education or less. Half of the parents or guardians of child respondents had completed college or vocational school, and the remaining 11.7% had graduate degrees. More than a third (38.3%) children with current asthma lived in households earning below 200% of the FPL, 30.8% lived between 200-399% of the FPL, and 30.8% lived in households earning 400% or more than the FPL.

The majority of children with current asthma were insured in the year prior to interview, though 4.9% of children were either not insured at all or only insured for part of the year. Most children (90.5%) were reported to have a usual source of care; still, 8.1% of children reportedly had a delay in needed care for any medical reason in the last 12 months. Among children with current asthma, 22.9% visited the doctor 0-1 time for any reason, 60.8% visited 2-5 times, and 16.3% visited 6 or more times in the past year. Most children with current asthma (90.5%) were diagnosed between the ages of 0 and 11. Thirty-seven percent took daily asthma medication, and 40.7% had an asthma management plan.

Most child respondents with current asthma were reported to have good, very good, or excellent health (81.3%), though 18.7% were reported to have poor or fair health. Based on BMI, 36.5% of teens with current asthma were classified as overweight or obese. Only 7.6% of children with current asthma lived in a household with a smoker. Two-fifths (40.9%) had dogs or cats in the home, while 15.4% had cockroaches in the home.

The greatest percentage of children with current asthma lived in urban residences (40.7%) compared to 2$^{nd}$ city (28.0%), suburban (21.0%), or town/rural residences (10.3%). More than three-fourths (76.6%) had lived at their current addresses for 3 or more years. Most children lived in houses (71.0%), while the remainder lived in apartments, duplexes, or mobile homes. One-third of children (33.4%) lived with household crowding.

**Disparities in Asthma Outcomes and Exposure Measures among Sub-Populations**

In this part of the study, we tested Hypothesis 1: Among those with asthma, vulnerable sub-populations in California (e.g., racial/ethnic minorities and low-income individuals) have higher exposures to air pollution. We present differences in estimated criteria pollutant exposures across various sub-populations, characterized by income level, racial and ethnic group, rural and urban residency, sex, and age. We also present results regarding differences in distributions of asthma outcomes across these subgroups.

*Disparities in Annual Average Criteria Pollutant Exposure Measures*

Adult respondents with current asthma and living below 200% of the FPL had higher annual average exposures to all criteria pollutants, except for ozone, than those living at 400% or above the FPL (Figure 2 and Table 11). Specifically, adults living below 200% of the FPL had a mean annual average $NO_2$ level of 22.4 ppb compared to 20.1 ppb among those living at or above 400% of the FPL. Additionally, adults living below 200% of the FPL had mean $PM_{10}$ and $PM_{2.5}$ exposures of 29.9 μg/m$^3$ and 16.7 μg/m$^3$, respectively, compared to 27.8 μg/m$^3$ and 15.0 μg/m$^3$ for those living at 400% or greater than the FPL. Mean annual average exposures to $O_3$ did not vary across poverty levels, however.

Disparities in criteria pollutant exposures were also observed across races/ethnicities (Figure 3). Latino adults had greater mean exposures to $NO_2$, $PM_{10}$ and $PM_{2.5}$ than whites (24.2 ppb vs. 19.6 ppb, 31.3 μg/m$^3$ vs. 27.6 μg/m$^3$ and 17.9 μg/m$^3$ vs. 15.1 μg/m$^3$, respectively). Asian and Pacific Islander adults had greater mean $NO_2$ exposures (22.6 ppb vs. 19.6 ppb), and adult African Americans also had greater mean annual average exposure to $PM_{2.5}$ (16.2 μg/m$^3$ vs. 15.1 μg/m$^3$. However, whites had greater mean exposures to $O_3$ than any of the aforementioned groups (whites: 42.5 ppb; Latinos: 41.1 ppb; Asian and Pacific Islanders: 40.4 ppb; African Americans: 39.3 ppb).

There were also disparities in estimated criteria pollutant exposures across respondents' location of residence. Adults with current asthma living in urban residences had the highest exposure to $NO_2$, with a mean annual average exposure more than twice that of adult respondents living in rural/town residences (24.4 ppb vs. 10.6 ppb). Adults in urban residences had a mean $PM_{2.5}$ exposure of 16.2 μg/m$^3$, versus a mean of 15.1 μg/m$^3$ for adult respondents living in second cities and 10.8 μg/m$^3$ for adult respondents in town/rural residences. However, for all respondents, living farther from urban areas increased their annual exposure to $O_3$. Adult respondents in urban residences had a mean annual average $O_3$ exposure of 38.9 ppb compared to 42.8 ppb for those in second city residences, 44.6 ppb for those in suburban residences, and 47.6 ppb for those in town/rural residences.

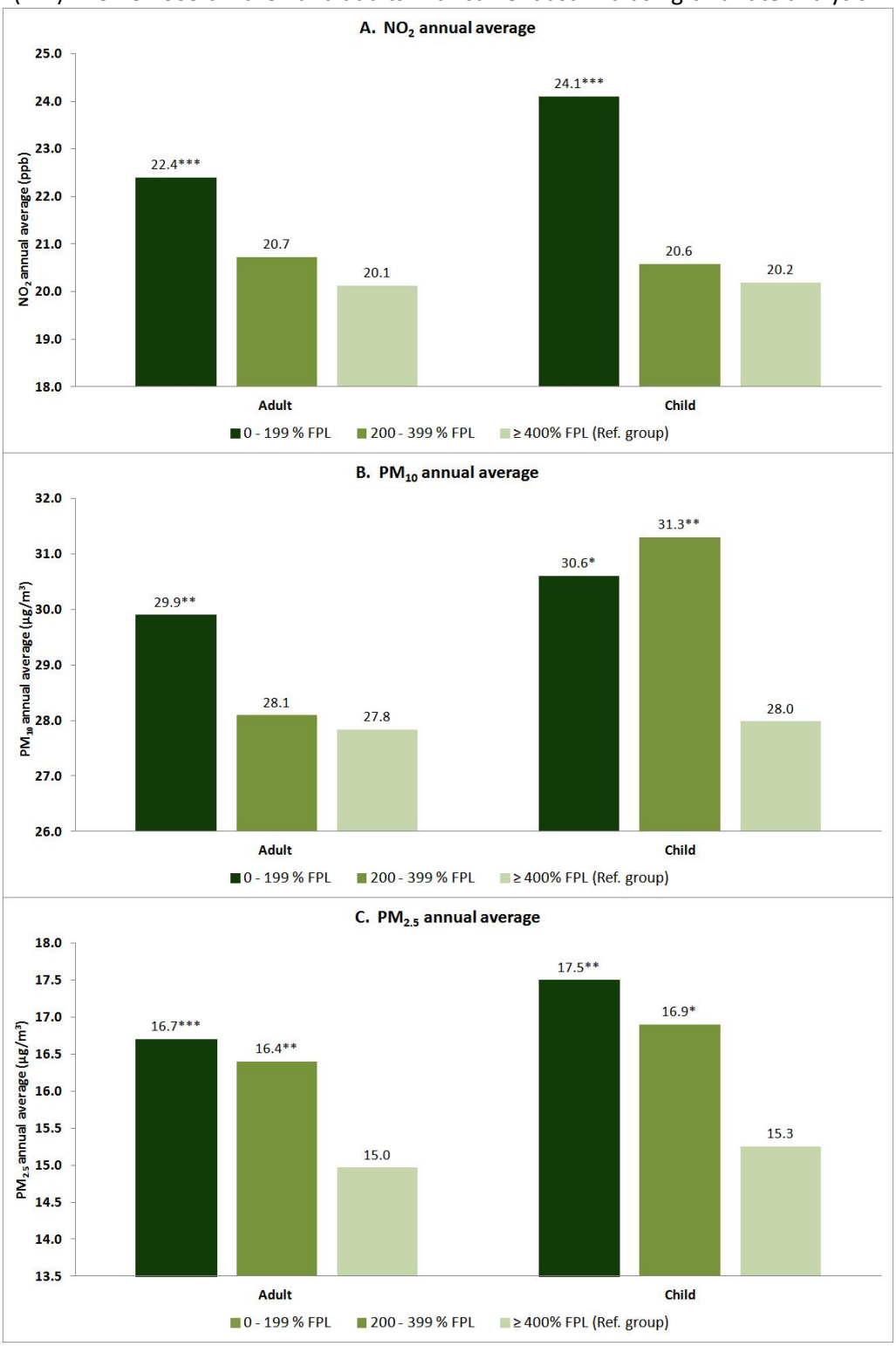
Few significant differences were observed in annual pollutant exposure levels across age groups or for men versus women. Adult females had greater mean $PM_{10}$ exposures at 29.1 μg/m$^3$ compared to 27.7 μg/m$^3$ among males, while adults ages 18-34 had greater mean $NO_2$ exposures than adults 65+ years of age (22.0 ppb vs. 19.9 ppb).

Among children with current asthma, those living between 0-199% of the FPL had a mean annual $NO_2$ exposure of 24.1 ppb versus 20.2 ppb for those living at or above 400% of the FPL (Figure 2 and Table 11). For those living below 200% of the FPL and those living between 200-399% of the FPL, mean annual exposure to $PM_{10}$ was estimated at 30.6 μg/m$^3$ and 31.3 μg/m$^3$, respectively, compared to 28.0 μg/m$^3$ for those living at or above 400% of the FPL. Mean $PM_{2.5}$ exposure was 17.5 μg/m$^3$ for children living at 0-199% of the FPL and 16.9 μg/m$^3$ for children living at 200-399% of the FPL, while children living at or above 400% of the FPL had a mean annual average exposure of 15.3 μg/m$^3$.

Children in minority populations were found to have higher average exposure to most air pollutants compared to their white peers (Figure 3). Latino, African American, and Asian/Pacific Islander/other children had higher mean annual exposures to $NO_2$ at 23.9 ppb, 22.1 ppb, and 23.8 ppb respectively, compared to 19.7 ppb for white children. Latino children had 17.2 μg/m$^3$ of annual average $PM_{2.5}$ exposure, and African American children had 17.8 μg/m$^3$ of annual average $PM_{2.5}$ exposure, compared to 15.7 μg/m$^3$ among white children. However, children of these race/ethnicities had lower $O_3$ exposures compared to white children (Latino: 40.6 ppb; African American: 40.0 ppb; Asian/PI/Other: 38.1 ppb; White: 43.1 ppb).

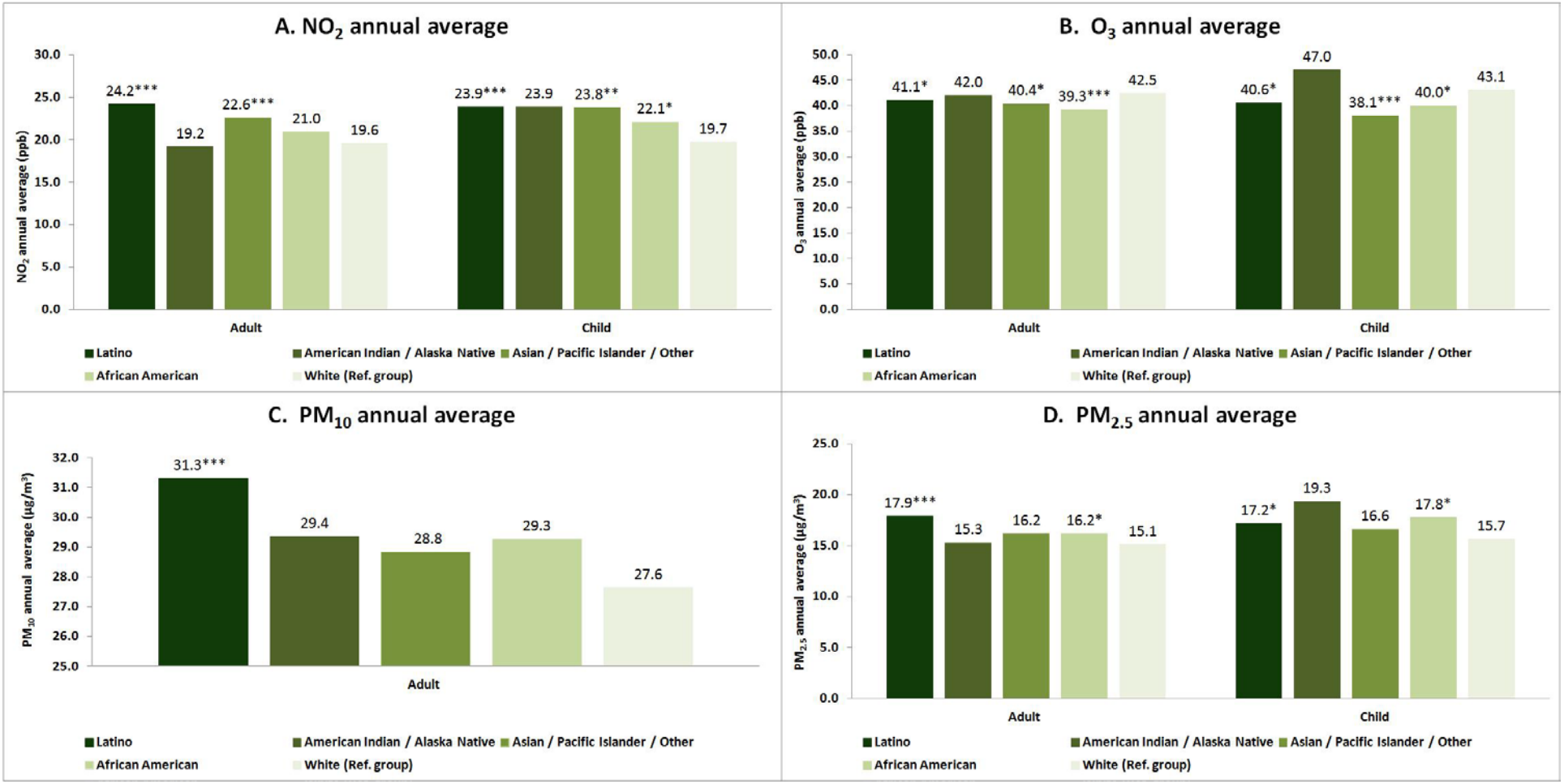
The mean annual $NO_2$ exposure for urban children was greater than mean exposures for children living in all other areas; for example, urban children had mean $NO_2$ averages nearly twice as high as children in town or rural residences (25.1 ppb vs. 13.7 ppb). Those living in non-urban areas had higher average exposure to $O_3$ than their urban-dwelling counterparts (2$^{nd}$ city: 44.3 ppb; Suburban: 45.5 ppb; Town/Rural: 43.5 ppb vs. Urban: 37.8 ppb). Compared to urban children, suburban children had higher $PM_{10}$ exposure (29.3 μg/m$^3$ vs. 33.7 μg/m$^3$), while town/rural children had lower $PM_{2.5}$ exposure than urban children (13.9 μg/m$^3$ vs. 17.0 μg/m$^3$). Among children with current asthma, boys had greater average exposure to $PM_{2.5}$ at 17.4 μg/m$^3$ versus 15.9 μg/m$^3$ for girls.

Figure 2. Disparities in weighted mean annual pollutant concentrations by Federal Poverty Level (FPL) in CHIS 2003 children and adults with current asthma using bivariate analysis[a]




[a]For CHIS 2003, current asthma is defined as reporting an asthma attack in the previous 12 months or answering yes to the question, "Do you still have asthma?"; only respondents who lived within 5 miles of an air monitoring station and lived in the same home or neighborhood for at least 9 months were included.
*p < 0.05, **p < 0.01, ***p < 0.001

Figure 3. Disparities in weighted mean annual pollutant concentrations by race/ethnicity in CHIS 2003 children and adults with current asthma using bivariate analysis[a,b]




[a]For CHIS 2003, current asthma is defined as reporting an asthma attack in the previous 12 months or answering yes to the question, "Do you still have asthma?"; only respondents who lived within 5 miles of an air monitoring station and lived in the same home or neighborhood for at least 9 months were included.
[b]For Figure 3C, results for children with current asthma showed no significant differences between groups and are not shown in the graph.
*$p < 0.05$, **$p < 0.01$, ***$p < 0.001$

Table 11 (Highlights). Disparities in weighted mean annual pollutant concentrations by various demographic characteristics in CHIS 2003 children and adults with current asthma using bivariate analysis[a]

| | | $NO_2$ annual average (ppb) | | $O_3$ annual average (ppb) | | $PM_{10}$ annual average ($\mu g/m^3$) | | $PM_{2.5}$ annual average ($\mu g/m^3$) | |
|---|---|---|---|---|---|---|---|---|---|
| Demographics | | Adult mean | Child mean | Adult mean | Child mean | Adult mean | Child mean | Adult mean | Child mean |
| Household Federal Poverty Level (FPL) | 0 - 199 % FPL | 22.4*** | 24.1*** | 41.1 | 41.0 | 29.9** | 30.6* | 16.7*** | 17.5** |
| | 200 - 399 % FPL | 20.7 | 20.6 | 42.1 | 42.0 | 28.1 | 31.3** | 16.4** | 16.9* |
| | ≥ 400% FPL† | 20.1 | 20.2 | 41.8 | 41.1 | 27.8 | 28.0 | 15.0 | 15.3 |
| Race/ethnicity | Latino | 24.2*** | 23.9*** | 41.1* | 40.6* | 31.3*** | 30.8 | 17.9*** | 17.2* |
| | American Indian / Alaska Native | 19.2 | 23.9 | 42.0 | 47.0 | 29.4 | 32.1 | 15.3 | 19.3 |
| | Asian / Pacific Islander / Other | 22.6*** | 23.8** | 40.4* | 38.1*** | 28.8 | 28.8 | 16.2 | 16.6 |
| | African American | 21.0 | 22.1* | 39.3*** | 40.0* | 29.3 | 31.7 | 16.2* | 17.8* |
| | White† | 19.6 | 19.7 | 42.5 | 43.1 | 27.6 | 29.1 | 15.1 | 15.7 |

[a]For CHIS 2003, current asthma is defined as reporting an asthma attack in the previous 12 months or answering yes to the question, "Do you still have asthma?"; only respondents who lived within 5 miles of an air monitoring station and lived in the same home or neighborhood for at least 9 months were included.
†Reference Group
*p < 0.05, **p < 0.01, ***p < 0.001

*Disparities in Traffic Exposure Measures*

Disparities in traffic density and residential proximity to roadways are shown in Table 12 in the Appendix. African American adults with current asthma had higher mean traffic density within a 750-foot buffer than white adults with current asthma (76.5 VMT/day/meters$^2$ vs. 60.2 VMT/day/meters$^2$). Urban dwelling adults had the highest mean traffic density at 80.6 VMT/day/meters $^2$, compared to 59.4 VMT/day/meters$^2$ among second city dwellers, 59.5 VMT/day/meters $^2$ among suburban dwellers, and 40.6 VMT/day/meters $^2$ among town/rural dwellers.

Adults with current asthma with lower household incomes were more likely to live within 50 meters of a major road than those with higher incomes. Of those living below 200% of the FPL, 26.4% lived near a major road, compared to 23.7% among those living between 200-399% of the FPL, and 17.5% among those living at or above 400% of the FPL. Fewer Alaskan Natives/American Indians lived near an interstate highway compared to whites (3.4% vs. 9.8%). Urban adults with current asthma were more likely to live within 300 meters of an interstate highway than town/rural-dwelling asthmatic adults (10.9% vs. 6.9%); however, those living in towns or rural areas were more than three times as likely to live within 300 meters of a state highway as those living in urban areas (13.3% vs. 4.4%). Twice as many women lived within 300 meters of a state highway as men (6.5% vs. 3.3%).

Asthmatic children living closer to the federal poverty line were more likely to live near all types of roadways and in places with greater traffic density than children living farther from the poverty line. Children living between 0-199% of the FPL had a mean traffic density of 84.6 VMT/day/meters $^2$, and children living between 200-399% of the FPL had a mean traffic density of 70.2 VMT/day/meters $^2$, while children living at or above 400% of the FPL had a mean traffic density of only 52.7 VMT/day/meters$^2$ (Table 12 in Appendix). Both Latino and African American children had higher mean traffic density measures than white children (93.6 VMT/day/meters$^2$ and 89.8 VMT/day/meters$^2$ vs. 53.2 VMT/day/meters$^2$). Urban children had greater mean traffic density than both 2$^{nd}$ city and town/rural children (84.7 VMT/day/meters$^2$ vs. 57.4 VMT/day/meters$^2$ and 46.4 VMT/day/meters$^2$).

Children with asthma living at 0-199% of the FPL were close to three times more likely to live within 300 meters of a state highway compared to children from households earning 400% or more than the FPL (9.5% vs. 2.6%). Children living below 200% of the FPL were also more than twice as likely to live within 300 meters of an interstate highway (16.4% vs. 6.3%), and nearly a quarter (24.7%) lived within 50 meters of a major road compared to only 15.3% of children living at or above 400% of the FPL. Latino children were more than twice as likely as white children to live within 300 meters of an interstate highway (18.1% vs. 8.5%). Urban children were nearly twice as likely to live within 50 meters of a major road as 2nd city and town/rural dwelling children (27.1% vs. 14.2% and 15.6%).

*Disparities in Asthma Outcomes*

Table 13 in the Appendix provides prevalence measures for asthma outcomes among CHIS 2003 respondents with current asthma by various demographic characteristics. Adults living below 200% of the FPL had the highest prevalence for all measured asthma outcomes compared to those living at or above 400% of the FPL. The prevalence of asthma attacks for adults with lifetime asthma living below 200% of the FPL was 40.3% compared to 32.9% for those living at or above 400% of the FPL. The prevalence of asthma-related ED visits in the year prior to the CHIS 2003 interview for adults with current asthma living below 200% of the FPL was nearly twice that of adults with current asthma living at or above 400% of the FPL (24.6% vs. 12.6%). Over half (53.8%) of the adults with current asthma living below 200% of FPL used daily asthma medication as compared to 43.7% living at or above 400% of the FPL. The prevalence of daily/weekly asthma symptoms for adults with current asthma among adults living below 200% of the FPL and adults living at 200-399% of the FPL were greater than the prevalence among those living at or above 400% of the FPL (35.0% and 31.0% vs. 25.2%).

When comparing asthma outcomes in adults with current asthma by race/ethnicity, the percentage of Latino adults who visited the ED was over twice that of white adults (26.8% vs. 12.7%). African American adults reported a higher prevalence of daily asthma medication use at 59.4%, as compared to 48.0% of white adults. African Americans (19.7%), Latinos (21.9%), and Asian/Pacific Islander/others (16.7%) all reported missing at least 2 days of work at a prevalence higher than that of white respondents (8.8%). However, more white respondents (32.7%) reported daily/weekly asthma symptoms than either Latino (22.2%) or Asian/Pacific Islander/other respondents (25.2%).

The prevalence of asthma outcomes among those with current asthma differed by location of residence. Fewer adult respondents who lived in 2nd city residences (11.3%) or town/rural residences (9.6%) reported missing at least 2 days of work versus adults living in urban residences (15.6%). However, a lower percentage of adults living in urban residences reported daily/weekly asthma symptoms than adults living in town/rural residences (27.7% vs. 35.3%).

Among adults with lifetime asthma, more female respondents experienced an asthma attack than male respondents (41.8% vs. 25.9%). Furthermore, a higher percentage of women with current asthma had visited the ED (17.8% vs. 13.76%) and missed at least 2 days of work (14.7% vs. 10.3%), and 31.7% of women with current asthma experienced asthma symptoms daily or weekly compared to 26.1% of men.

Significant differences in prevalence of asthma outcomes were found among the youngest and oldest respondents. More than two-thirds of adults with current asthma in the oldest age group, age 65 or older, took asthma medication daily at 67.3% compared to 48.4% of adults ages 35-64 and 35.2% of adults ages 18-34. More than one-third (38.1%) of adults with current asthma who were 65 or older reported experiencing daily or weekly asthma symptoms compared to 32.3% of those ages 35-64 and only 20.3% of those ages 18-34. However, adults ages 35-64 were more likely than those 65 or older to have visited the ED in the year prior to the interview (18.7% vs. 13.1%).

Among children with lifetime asthma, those living at or above 400% of the FPL had the highest prevalence of asthma attacks at 40.4% compared to 32.1% for those living at less than 200% of the FPL; however, for all other asthma outcomes, children with current asthma living below 200% of the FPL had the highest prevalence (Table 13 in Appendix). Children with current asthma whose household incomes fell below 200% of the FPL or between 200-399% of the FPL had higher prevalence of ED visits at 26.4% and 23.0% respectively, compared to 14.7% prevalence for those living at or above 400% of the FPL . Additionally, 45.7% of children with current asthma living below 200% of the FPL took asthma medication daily as opposed to only 28.7% living at or above 400% of the FPL. Over half (54.3%) of those living below 200% of the FPL were reported to have missed ≥2 days of school versus 33.2% of those living at or above 400% of the FPL.

The prevalence of asthma outcomes also varied by race/ethnicity among child respondents. Although Latino children with lifetime asthma had a lower prevalence of asthma attacks (29.8%) compared to white children (37.9%), more Latino children with current asthma took a daily asthma medication (45.4% vs. 31.5%). Nearly a third (32.3%) of African American children with current asthma reported visiting the ED in the past year compared to only 18.8% of white children with current asthma. Compared to children with current asthma living in town/rural residences, more children with current asthma living in urban residences had visited the ED (25.0% vs. 9.7%), taken daily asthma medication (39.6 % vs. 27.7%), and/or missed at least 2 days of school (45.1% vs. 29.2%).

The prevalence of an asthma attack in the past year was greater among children with lifetime asthma ages 0-5 (55.2%) and 6-11 years old (41.1%) than among children with lifetime asthma ages 12-17 (23.4%). While only 5.8% of children with current asthma in the oldest age group had visited the ED in the past 12 months, 26.7% of children with current asthma ages 6-11 and 44.3% of children with current asthma ages 0-5 had been taken to the ED within the year prior to the interview; yet, the oldest children with current asthma reported the highest prevalence of daily or weekly asthma symptoms at 16.1% in comparison to 8.9% among those 6-11 years old and 7.9% among those ages 0-5.

**Associations between Air Pollution Exposure Metrics and Asthma Health Outcomes**

In this part of the study, we tested Hypothesis 2, that individuals with asthma exposed to higher levels of air pollution are more likely to report adverse asthma outcomes, such as: asthma attacks or episodes, asthma emergency department (ED) visits, use of daily medication to control asthma, school or work absences, and daily/weekly asthma symptoms. We present results of crude associations (Crude Odds Ratio) between individual air pollutants and asthma

outcomes and adjusted associations controlling for age, sex, race/ethnicity and federal poverty level (Adjusted Odds Ratio) using logistic regression modeling.

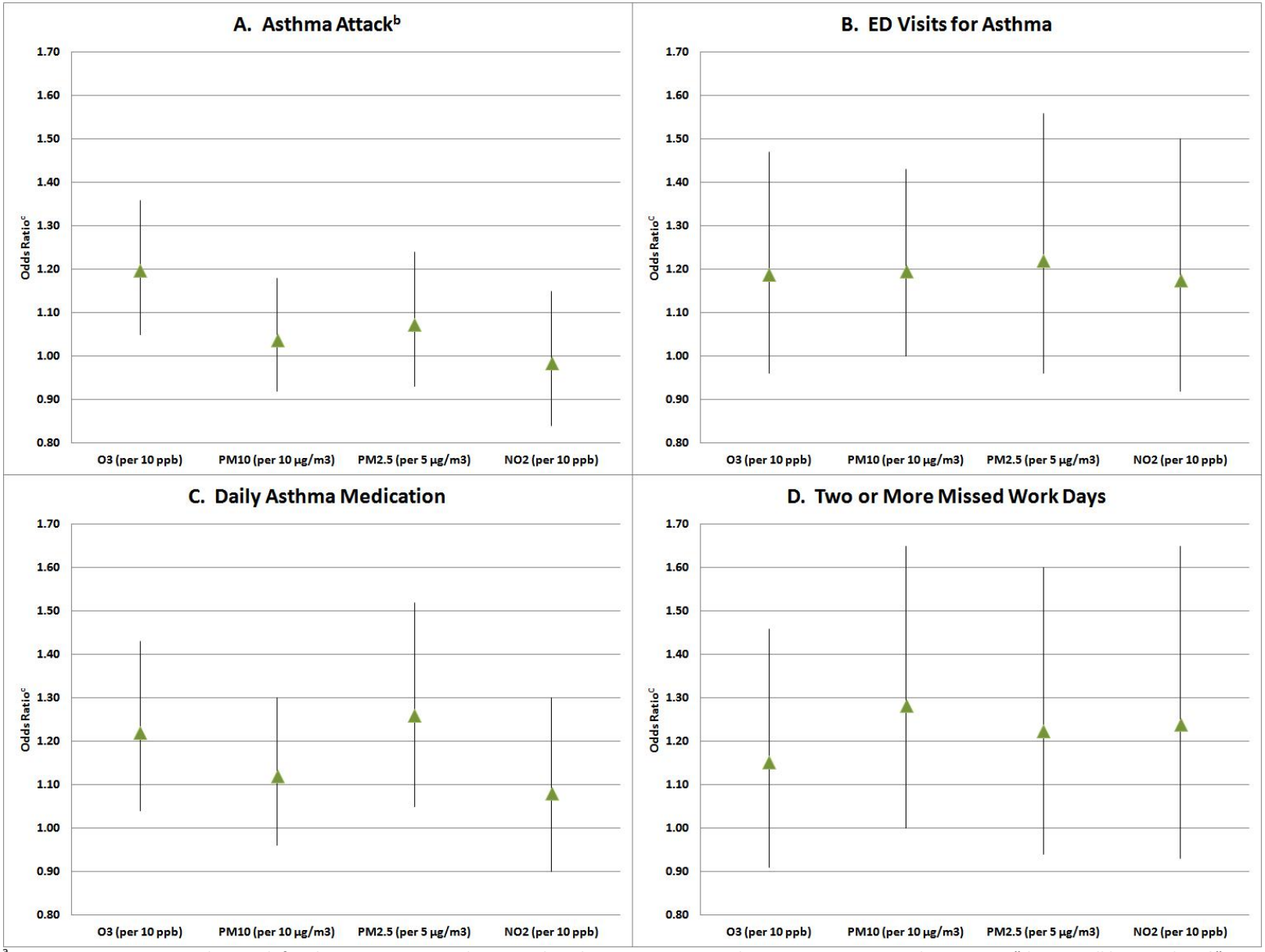
*Associations of 12-month pollutant averages with asthma outcomes*

We observed positive associations between annual average criteria pollutant concentrations and asthma outcomes after controlling for age, race, poverty level and sex (Figure 4 and Table 14). A 10 ppb increase in annual average $O_3$ concentration was associated with 20% higher odds of experiencing an asthma attack in the previous year (95% CI=1.05-1.36) among adults with lifetime asthma. A 10 ppb increase in annual average $O_3$ was also associated with a 22% (95% CI=1.04-1.43) increase in the odds of using daily asthma medication and a suggested 19% (95% CI=0.96-1.47) increase in the odds of visiting the ED within the past year in adults with current asthma.

Among adults with current asthma, a 10 $\mu g/m^3$ increase in annual average $PM_{10}$ concentration was associated with a 20% (95% CI 1.00-1.43) increase in the odds of visiting the ED within the past year and a 28% (95% CI=1.00-1.65) increase in the odds of 2 or more asthma-related absences from work. Associations were also suggested between $PM_{10}$ and an increase in the odds of taking a daily asthma medication (OR=1.12, 95% CI=0.96-1.30). For every 5 $\mu g/m^3$ increase in annual average $PM_{2.5}$ concentration, the odds of taking daily asthma medication increased by 26% (95% CI=1.05-1.52); the odds of visiting the ED increased by 22% (95% CI=0.96-1.56); and the odds of experiencing daily/weekly asthma symptoms increased by 15% (95% CI=0.96-1.39). A 5 $\mu g/m^3$ increase in $PM_{2.5}$ concentration was also associated with a suggested 23% (95% CI=0.94-1.60) increase in the odds of missing ≥2 work days. Additionally, we observed a suggested 24% (95% CI=0.93-1.65) increase in odds of having ≥2 asthma-related work absences and an 18% (OR=1.18, 95% CI=0.92-1.50) increase in odds of visiting the ED per 10 ppb increase in mean annual average $NO_2$.

In general, we observed few associations between annual average concentrations of criteria pollutants and asthma symptoms in children. We estimated a 36% (95% CI=0.99-1.87) increase in the odds of taking a daily asthma medication and a 35% (95% CI=0.94-1.96) increase in missing 2 or more days of school per 10 ppb increase in annual average $NO_2$.

Figure 4. Associations (OR (95% CI)) between 12-month pollutant averages and asthma outcomes in CHIS 2003 adults with current asthma[a]




[a]For CHIS 2003, current asthma is defined as reporting an asthma attack in the previous 12 months or answering yes to the question "do you still have asthma?"; only respondents who lived within 5 miles of an air monitoring station and lived in the same neighborhood for at least 9 months were included.
[b]For respondents ever diagnosed with asthma
[c]Adjusted for age, race, poverty level, and sex

Table 14 (Highlights). Associations (OR (95% CI)) for 12-month pollutant averages and respiratory outcomes in CHIS 2003 adults and children with current asthma[a]

| | Asthma Attack[b] | | | | ED Visit for Asthma | | | | Daily Asthma Medication | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Pollutant | Cases | Non-Cases | Adj. OR[d] | 95% C.I. | Cases | Non-Cases | Adj. OR[d] | 95% C.I. | Cases | Non-Cases | Adj. OR[d] | 95% C.I. |
| Adults | | | | | | | | | | | | |
| $O_3$ (per 10 ppb) | 965 | 1582 | **1.20** | [1.05, 1.36] | 245 | 1372 | 1.19 | [0.96, 1.47] | 815 | 802 | **1.22** | [1.04, 1.43] |
| $PM_{10}$ (per 10 µg/m$^3$) | 770 | 1272 | 1.04 | [0.92, 1.18] | 212 | 1092 | **1.20** | [1.00, 1.43] | 659 | 645 | 1.12 | [0.96, 1.30] |
| $PM_{2.5}$ (per 5 µg/m$^3$) | 592 | 974 | 1.07 | [0.93, 1.24] | 160 | 830 | 1.22 | [0.96, 1.56] | 494 | 496 | **1.26** | [1.05, 1.52] |
| $NO_2$ (per 10 ppb) | 782 | 1313 | 0.99 | [0.84, 1.15] | 200 | 1115 | 1.18 | [0.92, 1.50] | 667 | 648 | 1.08 | [0.90, 1.30] |
| Children | | | | | | | | | | | | |
| $NO_2$ (per 10 ppb) | 282 | 430 | 0.97 | [0.74, 1.27] | 113 | 356 | 1.17 | [0.81, 1.69] | 171 | 298 | 1.36 | [0.99, 1.87] |

[a]For CHIS 2003, current asthma is defined as reporting an asthma attack in the previous 12 months or answering yes to the question, "Do you still have asthma?"; only respondents who lived within 5 miles of an air monitoring station and lived in the same neighborhood for at least 9 months were included.
[b]Respondents ever diagnosed with asthma who lived within 5 miles of an air monitoring station and in the same home or neighborhood for at least 9 months were included.
[c]Data not collected for teen respondents.
[d]Adjusted for age, race, poverty level, and sex.

Table 14 (Highlights). Associations (OR (95% CI)) for 12-month pollutant averages and respiratory outcomes in CHIS 2003 adults and children with current asthma[a] (continued)

| | Missed ≥2 School/Work Days Due to Asthma[c] | | | | Daily/Weekly Asthma Symptoms | | | |
|---|---|---|---|---|---|---|---|---|
| Pollutant | Cases | Non-Cases | Adj. OR[d] | 95% C.I. | Cases | Non-Cases | Adj. OR[d] | 95% C.I. |
| Adults | | | | | | | | |
| $O_3$ (per 10 ppb) | 176 | 1208 | 1.15 | [0.91, 1.46] | 504 | 1113 | 1.03 | [0.87, 1.22] |
| $PM_{10}$ (per 10 µg/m$^3$) | 142 | 977 | **1.28** | [1.00, 1.65] | 415 | 889 | 1.03 | [0.89, 1.20] |
| $PM_{2.5}$ (per 5 µg/m$^3$) | 118 | 743 | 1.23 | [0.94, 1.60] | 316 | 674 | 1.15 | [0.96, 1.39] |
| $NO_2$ (per 10 ppb) | 147 | 985 | 1.24 | [0.93, 1.65] | 410 | 905 | 1.03 | [0.85, 1.25] |
| Children | | | | | | | | |
| $NO_2$ (per 10 ppb) | 132 | 166 | 1.35 | [0.94, 1.96] | 51 | 418 | 1.13 | [0.72, 1.75] |

[a]For CHIS 2003, current asthma is defined as reporting an asthma attack in the previous 12 months or answering yes to the question, "Do you still have asthma?"; only respondents who lived within 5 miles of an air monitoring station and lived in the same neighborhood for at least 9 months were included.
[b]Respondents ever diagnosed with asthma who lived within 5 miles of an air monitoring station and in the same home or neighborhood for at least 9 months were included.
[c]Data not collected for teen respondents.
[d]Adjusted for age, race, poverty level, and sex.

*Associations for annual number of days exceeding air pollution standards and asthma outcomes*

We observed associations between the number of days annually in which criteria pollutant levels exceeded maximum state and/or federal standards and asthma outcomes. Exceedances of state maximum 1-hr $O_3$ standards (i.e. over 36.7 days per year) were associated with a 40% (95% CI=1.03-1.91) increase in the odds of having an asthma attack in the past year among adults with lifetime asthma compared to those with <0.8 exceedance days (Table 15 in Appendix). They are also associated with a 91% (95% CI=1.14-3.18) increase in the odds of visiting the ED in the last 12 months and a 52% (95% CI=1.03-2.24) increase in the odds of taking a daily asthma medication in adults with current asthma. When looking at exceedance days for the $O_3$ 1-hr state standard, there appeared to be a pollutant exposure-response trend for all three outcomes mentioned above.

Based on the $O_3$ 8-hr state standard, adults with lifetime asthma and ≥51.2 exceedance days were 44% more likely to have asthma attacks in the previous year (95% CI=1.07-1.94). Adults with current asthma and ≥51.2 exceedance days were 50% more likely to require a daily asthma medication (95% CI=1.04-2.18) than those with <1.9 exceedance days.

In addition to associations with $O_3$, asthma outcomes were also associated with exceedance days for particulate matter. Adults with current asthma and ≥6.6 days when $PM_{10}$ concentrations exceeded the 24-hr state standard had 77% (95% CI=1.05-2.97) greater odds of visiting the ED in the year prior to the interview. Adults with current asthma and ≥23.9 days when $PM_{2.5}$ concentrations exceeded the 24-hr federal standard were twice (OR=2.01, 95% CI=1.10-3.68) as likely to visit the ED in the past year as adults with <4.8 exceedance days, and there was a suggested exposure-response trend for this outcome. Adults with asthma with ≥23.9 exceedance days were also 66% (95% CI=1.02-2.68) more likely to experience daily or weekly asthma symptoms compared to those with <4.8 $PM_{2.5}$ exceedance days.

Among those with current asthma, children with ≥36.7 days when maximum 1-hr $O_3$ concentrations exceeded the 1-hr state standard were 3 times as likely (OR=3.00, 95% CI=1.20-7.51) to miss 2 or more school days compared to children with <0.8 exceedance days (Table 15 in Appendix). We did not observe associations between any of the other exceedance measures or asthma outcomes in children.

Further associations were suggested between particulate matter exceedance days and asthma outcomes among adults with current asthma. A suggested 70% (95% CI=0.91-3.18) increase in the odds of missing ≥2 work days due to asthma was observed for respondents with ≥6.6 $PM_{10}$ exceedance days compared to their counterparts with <1.6 exceedance days. Also, a suggested 49% (95% CI=0.92-2.39) increase in the odds of needing a daily asthma medication was observed for those with ≥23.9 compared to those with <4.8 $PM_{2.5}$ exceedance days.

*Associations for traffic density/distance to roadway and asthma outcomes*

We observed few consistent associations between traffic density and residential proximity to roadway measures and asthma outcomes (Table 16 in Appendix). An interquartile increase in traffic density within 750 feet of respondent's homes was associated with a 8% (95% CI= 0.97-1.21) increase in odds of reporting asthma ED visits in the past year, but analyses based on quartiles of exposure did not demonstrate a clear exposure-response pattern for this outcome. Traffic density was not associated with any other outcome in adults. Living within 300 m of an interstate highway was associated with a 51% (95% CI=0.91-2.48) suggested increase in

the odds of visiting the ED in the past year, as well as a 34% (95% CI=0.95-1.90) suggested increase in the odds of needing a daily asthma medication.

For children with current asthma, we observed a 58% (95% CI=0.94-2.66) suggested increase in the odds of taking an asthma medication daily for children in the 3rd versus 1st quartile of TD exposure, but a 43% (95% CI=0.83-2.47) for children in the highest TD quartile (Table 16 in Appendix).

**Pollutant and Asthma Outcome Relationship after Adjusting Vulnerability (Confounding) Factors**

In this part of the study, we tested Hypothesis 3: Do air pollution exposures, low SES status, and certain vulnerability factors associated with low SES exert independent adverse effects on individuals with asthma? We fit three models (1) a base model, which includes each pollutant measure, plus socio-demographics, such as age, race, federal poverty level, and sex; (2) the base model plus adjustment for access to care (i.e. health insurance) and major risk factors, such as smoking and being overweight or obese, and heart disease; and (3) the base model plus adjustment for indicators of asthma severity, such as age of asthma onset, taking a daily asthma medication, having an asthma management plan; as well as indoor triggers, such as the presence of household smoking, having a dog or cat in the home, having cockroaches in the home, housing type, and household crowding.

*Associations between ED visits and $O_3$ adjusting for vulnerability factors in adults*

We observed persistent associations between ED visits and $O_3$, controlling for vulnerability factors by using three different models in order to observe variations in the pollutant effect when these factors are taken into account (Table 17). Specifically, for $O_3$, a positive association between increases in $O_3$ and ED visits remained across all models. We observed a suggested 19% (95% CI=0.96-1.47) increase in the odds of an ED visit in the last year per 10 ppb increase in annual average $O_3$ concentration among adults with current asthma, when using our base model after adjusting for socio-demographics (Model 1). The estimated association between annual average $O_3$ and ED visits remained after further adjusting for access to care and risk behaviors under Model 2 (OR= 1.18; 95% CI=0.95-1.47) and after adjusting for asthma severity and indoor triggers in Model 3 (OR=1.15, 95% CI=0.91-1.45), although in Model 3, the estimated odds were slightly lower and less precise.

Furthermore, we observed the increased odds of ED visits across races/ethnicities, age groups, poverty levels, and genders, which persisted across all three models. Based on Model 1, African Americans had 86% (95% CI= 1.04-3.32) greater odds and Latinos had twice greater odds (95% CI=1.27-3.23) of having an ED visit as white respondents. Adults with current asthma ages 35-64 years old had nearly twice (OR=1.91, 95% CI=1.15-3.20) greater odds of having visited the ED in the last year than asthmatic adults 65 years or older, and respondents living below 200% of the FPL had 93% greater odds of an ED visit (95% CI=1.26-2.97) compared to those living at or above 400% of the FPL. Women had higher odds of ED visits than men (OR=1.45, 95% CI=0.97-2.18). Under model 2, Latino and African American respondents, those 35-64 years old, females, and those living below 200% of the FPL continued to have higher odds of ED visits. Under Model 3, the pattern of increased odds of ED visits remained the same in

general, though for African Americans and women the estimated odds of ED visits moved closer toward the null.

In addition to the above associations observed in all models, under Model 2 we observed a 69% (95% CI=1.01-2.84) increase in the odds of having an ED visit in the last year for those who reported having heart disease and a 65% (95% CI=1.08-2.54) increase in odds for those with adult onset of asthma compared to childhood onset. Under Model 3 those not using a daily asthma medication and those without an asthma management plan had lower odds of ED visits (OR=0.35, 95% CI=0.23-0.52 and OR=0.52, 95% CI=0.36-0.76, respectively).

*Associations between ED visits and $PM_{10}$ adjusting for vulnerability factors in adults*

A positive association between ED visits and annual average $PM_{10}$ concentration persisted across all models. We estimated a 20% (95% CI=1.00-1.43) increase in the odds of ED visits in the last 12 months per 10 $\mu g/m^3$ increase in annual average $PM_{10}$ after adjusting for socio-demographics under our base model (Model 1). The association stayed nearly the same after further adjusting for access to care and risk behaviors under Model 2 (OR=1.18, 95% CI=0.95-1.47) and increased slightly (OR=1.25, 95% CI=1.03-1.51) after adjusting for asthma severity and indoor triggers under Model 3.

Positive associations were also observed between ED visits and race/ethnicity, age, and poverty level across all models. Based on Model 1, we estimated increased odds of ED visits for adults 35-64 years versus ≥65 years (OR=1.76, 95% CI=0.99-3.13), Latinos versus whites (OR=2.42, 95% CI=1.46-4.00), and individuals living below 200% of the FPL versus those at ≥400% of the FPL (OR=2.02, 95% CI=1.27-3.20). Under Models 2 and 3, the strength of association between ED visits and being Latino remained approximately the same. Compared to Model 1, the odds of ED visits increased for respondents ages 35-64, but decreased for those living below 200% of the FPL under Models 2 and 3.

We also observed associations between ED visits and several model-specific vulnerability factors. Having heart disease doubled the odds of an ED visit (OR=2.07, 95% CI=1.22-3.52), and adult versus childhood asthma onset increased the odds of an ED visit by 50% (95% CI=0.94-2.39). Those not taking a daily asthma medication (OR=0.30, 95% CI=0.20-0.47) and those without an asthma management plan (OR=0.56, 95% CI=0.37-0.84) had lower odds of visiting the ED.

*Associations between ED visits and $PM_{2.5}$ adjusting for vulnerability factors in adults*

A 5 $\mu g/m^3$ increase in annual average $PM_{2.5}$ increased the odds of visiting the ED in the last 12 months by 22% (95% CI=0.96-1.56) among adults with current asthma after adjusting for socio-demographics per Model 1. The positive association between ED visits and $PM_{2.5}$ remained nearly the same after further adjusting for access to care and risk behaviors under Model 2 (OR=1.21, 95% CI=0.95-1.54) and after adjusting for asthma severity and indoor triggers under Model 3 (OR=1.20, 95% CI=0.92-1.56).

Under Model 1 we observed that Latinos (OR=2.03, 95% CI=1.18-3.49) and African Americans (OR=1.92, 95% CI=0.99-3.71) had twice greater odds of visiting the ED. Adults with asthma living below 200% of the FPL had odds 84% (95% CI=1.11-3.03) greater than those living at 400% of the FPL or above. The positive association between ED visits and being Latino persisted across all models, though increased under Model 2, while the positive association

between ED visits and being African American was present in Model 2, but not Model 3. For respondents living below 200% of the FPL, estimated odds moved toward the null under Models 2 and 3.

Having heart disease increased the odds of ED visits by 2.12 times (95% CI=1.20-3.73). A negative relationship was observed between ED visits and not taking a daily asthma medication (OR=0.29, 95% CI=0.17-0.49), as well as not having an asthma management plan (OR=0.63, 95% CI=0.40-1.00).

*Associations between daily asthma medication and $O_3$ adjusting for vulnerability factors in adults*

For every 10 ppb increase in annual average $O_3$ concentration, we estimated a 22% (95% CI=1.04-1.43) increase in the odds of needing a daily asthma medication among adults with current asthma, according to Model 1 after adjusting for socio-demographics (Table 18). The relationship between $O_3$ and daily asthma medication was similar across all models after further adjusting for access to care and risk behaviors or asthma severity and indoor triggers.

Based on Model 1, being 18-34 years old (OR=0.32, 95% CI=0.21-0.48) or 35-64 (OR=0.55, 95% CI=0.39-0.78) was found to have lower odds of needing a daily asthma medication. In addition, we observed suggested associations between taking daily asthma medication and being African American (OR=1.49, 95% CI=0.92-2.41) and living below 200% of the FPL (OR=1.30, 95% CI=0.94-1.79). As in Model 1, being <65 versus ≥65 years of age was found to have lower odds in general, but African Americans had greater odds of needing a daily asthma medication under Models 2 and 3. The positive association between daily asthma medication and living below 200% of the FPL did not appear under Model 2, but was seen in Model 3.

Model-specific vulnerability factors were also found to have associations with increased odds of needing a daily asthma medication. Having current insurance (OR=1.56, 95% CI=1.02-2.38), having heart disease (OR=1.77, 95% CI=1.16-2.69), and ever smoking (OR=1.41, 95% CI=1.07-1.86) were found to increase respondents' odds of daily asthma medication use under Model 2. Moreover, individuals without a usual source of care (OR=2.28, 95% CI=1.35-3.85) had higher odds of daily asthma medication use under Model 3, while not having an asthma management plan (OR=0.46, 95% CI=0.35-0.61) and having cockroaches in the home (OR=0.58, 95% CI=0.38-0.88) were shown to have a negative association with needing a daily asthma medication.

*Associations between daily asthma medication and $PM_{10}$ adjusting for vulnerability factors in adults*

According to our base model (Model 1), a 10 $\mu g/m^3$ increase in annual average $PM_{10}$ was associated with a 12% (95% CI=0.96-1.30) increase in the odds of daily asthma medication use after adjusting for socio-demographics among adults with current asthma. The positive association between $PM_{10}$ and daily medication use stayed the same under Model 2 (OR=1.12, 95% CI=0.96-1.30) after further adjusting for access to care and risk behaviors and increased slightly after adjusting for asthma severity and indoor triggers under Model 3 (OR=1.23, 95% CI=1.04-1.45).

Under Model 1, African Americans (OR=1.85, 95% CI=1.07-3.19) and American Indian/Alaska Natives (OR=2.46, 95% CI=0.92-6.62) had higher odds of daily asthma medication use. Younger adult respondents had lower odds of needing a daily asthma medication compared to adult respondents ≥65 years (18-34 years: OR=0.32, 95% CI=0.20-0.51; 35-64 years: OR=0.53, 95% CI=0.36-0.79).

The positive association between odds of daily asthma medication use and being African American increased in Models 2 and 3, while estimates for American Indian/Alaska Natives remained the same and precision increased. The lower odds for those between the ages of 18-34 years old remained, but lower odds for those 35-64 years old was only evidenced under Model 2.

Adults with heart disease had 81% (95% CI=1.12-2.91) higher odds, and those who had ever smoked had 31% (95% CI=0.97-1.77) higher odds of needing a daily asthma medication under Model 2. Under Model 3, odds of daily medication use was greater for those with a usual source of care (OR=1.83, 95% CI=1.02-3.28). On the other hand, those without an asthma management plan (OR=0.44, 95% CI=0.32-0.60), those who had experienced a delay in care for any medical reason (OR=0.68, 95% CI=0.47-0.97), and those with the presence of cockroaches in the home (OR=0.54, 95% CI=0.33-0.87) had lower odds of using a daily asthma medication.

*Associations between daily asthma medication and $PM_{2.5}$ adjusting for vulnerability factors in adults*

The odds of daily medication use increased 26% (95% CI=1.05-1.52) per 5 $\mu g/m^3$ increase in annual average $PM_{2.5}$ concentration level after adjusting for socio-demographics based on Model 1. The odds of using a daily asthma medication increased after further adjusting for access to care and risk behaviors under Model 2 (OR=1.30, 1.07-1.57), and increased even further after adjusting for asthma severity and indoor triggers under Model 3 (OR=1.44, 95% CI=1.18-1.76).

Under Model 1, respondents with current asthma between the ages of 18-34 and 35-64 years had lower odds of needing a daily asthma medication than respondents 65 years old or older (OR=0.40, 95% CI=0.24-0.66 and OR=0.56, 95% CI=0.36-0.88, respectively). There also appeared to be a positive association between daily asthma medication use and being African American (OR=1.62, 95% CI=0.92-2.84). Under Models 2 and 3, younger respondents, ages 18-34, had lower odds of using a daily asthma medication than those with current asthma 65 years old or older, but the previous negative associations for ages 35-64 years (OR=0.64, 95% CI=0.42-0.97) only remained under Model 3. Positive associations between increased odds of taking a daily asthma medication and being African American persisted under Models 2 and 3.

In addition to the above associations seen across all models, being currently insured (OR=1.86, 95% CI=1.11-3.11), having heart disease (OR=1.73, 95% CI=1.05-2.84), and ever smoking (OR=1.47, 95% CI=1.04-2.06) all increased the odds of daily asthma medication use under Model 2. Based on Model 3, those with a usual source of care had increased odds (OR=3.07, 95% CI=1.57-5.99), while having an asthma management plan (OR=0.41, 95% CI=0.29-0.59) or cockroaches in the home (OR=0.59, 95% CI=0.36-0.97) had a negative relationship with daily asthma medication use.

*Associations between missed work days and $O_3$ adjusting for vulnerability factors in adults*

According to results from Model 1, we observed a 15% suggested increase (95% CI=0.91-1.46) in the odds of missing ≥2 days of work as $O_3$ annual averages increased by 10 ppb among adults with current asthma after adjusting for socio-demographics (Table 19). Model 2 appeared to also evidence a 15% increase (95% CI=0.91-1.44) after further adjusting for access to care and risk behaviors, and Model 3 appeared to evidence a 13% increase in odds of missing ≥2 days of work due to asthma after adjusting for asthma severity and indoor triggers (95% CI=0.88-1.44), though precision was low.

Based on Model 1, African American, Asian/Pacific Islander/Other race, and Latino adults had two to four times greater odds of missing ≥2 days of work than white adults (OR=3.79, 95% CI=2.02-7.13; OR=2.06, 95% CI=1.07-3.97; OR=3.42, 95% CI=2.06-5.68, respectively). Women had twice greater odds of missing ≥2 days of work as men (OR=2.26, 95% CI=1.39-3.68). Living below 200% of the FPL was shown to have a negative relationship with missing 2 or more work days. For all $O_3$ vulnerability characteristic models and all subsequent pollutant vulnerability characteristic models, the odds ratios for the age variable were not estimated due to small sample size. Associations observed based on results from Model 1 were maintained in Models 2 and 3, and changes in point estimates for these factors were minimal. Beyond the associations evidenced across all models controlling for $O_3$, those without a daily asthma medication (OR=0.44, 95% CI=0.28-0.69) had lower odds of missing ≥2 days of work.

*Associations between missed work days and $PM_{10}$, adjusting for vulnerability factors in adults*

Based on Model 1, we observed a 28% (95% CI=1.00-1.65) increase in the odds of missing ≥2 days of work per 10 $\mu g/m^3$ increase in annual average $PM_{10}$ concentration after adjusting for socio-demographics. Associations between $PM_{10}$ and missing ≥2 days of work were similar after further adjusting for access to care and risk behaviors in Model 2 (OR=1.30, 95% CI=1.01-1.68) or adjusting for asthma severity and indoor triggers in Model 3(OR=1.25, 95% CI=0.96-1.63).

Both African Americans and Latinos had nearly 4 times greater odds of missing ≥2 days of work than their white counterparts (OR=3.60, 95% CI=1.73-7.47 and OR=3.60, 95% CI=2.00-6.47, respectively), and women had twice greater odds than men to miss ≥2 days of work (OR=2.14, 95% CI=1.25-3.65) under Model 1. Positive associations were similarly maintained for African Americans, Latinos, and women under Models 2 and 3. As with all models investigating $O_3$ associations with the adverse asthma outcomes mentioned above, those not taking a daily asthma medication had lower odds of missing ≥2 days of work than those taking a daily asthma medication (OR=0.44, 95% CI=0.26-0.74).

*Associations between missed work days and $PM_{2.5}$ adjusting for vulnerability factors in adults*

We observed a 23% suggested increase in the odds of missing ≥2 days of work (95% CI=0.94-1.60) for every 5 $\mu g/m^3$ increase in $PM_{2.5}$ annual averages after adjusting for socio-demographics under Model 1. The association between missing ≥2 days of work and $PM_{2.5}$ was of similar magnitude after further adjusting for access to care and risk behaviors in Model 2 (OR=1.23, 95% CI=0.94-1.60) and after adjusting for asthma severity and indoor triggers in Model 3 (OR=1.24, 95% CI=0.93-1.66).

African American and Latino respondents had greater odds of missing ≥2 days of work than white respondents (OR=4.69, 95% CI=2.24-9.81 and OR=3.28, 95% CI=1.76-6.10, respectively), as did women compared to men (OR=2.23, 95% CI=1.24-4.03) under Model 1. Respondents living below 200% of the FPL appeared to have lower odds of missing ≥2 days of work than those living at or above 400% of the FPL (OR=0.37, 95% CI=0.21-0.67). The positive associations for African Americans, Latinos, and women and the negative association for those living below 200% of the FPL remained similar in Models 2 and 3. Similar to Models 1-3 investigating $O_3$ and $PM_{10}$ associations, those not taking a daily asthma medication had lower odds of missing ≥2 days of work (OR=0.49, 95% CI=0.28-0.86).

*Associations between daily asthma medication and $PM_{2.5}$ adjusting for vulnerability factors in children*

Based on Model 1, we observed a 20% (95% CI=0.88-1.63) suggested increase in the odds of using a daily asthma medication for every 5 $\mu g/m^3$ increase in annual average $PM_{2.5}$ concentration after adjusting for socio-demographics among children with current asthma. The positive association between daily asthma medication use and $PM_{2.5}$ persisted across all models, though confidence intervals crossed the null after adjusting for insurance status, the presence of household smoking, having a dog or cat in the home, and having cockroaches in the home (Model 2); as well as after adjusting for urban vs. rural residence, having a delay in care, taking a daily asthma medication, having an asthma management plan, housing type, and household crowding in Model 3.

In Model 1, children living below 200% and those living between 200-399% of the FPL had three times greater odds of using a daily asthma medication compared to those living at ≥400% of the FPL (OR=2.64, 95% CI=1.22-5.72; OR=3.00, 95% CI=1.46-6.14). Associations between living below 200% or between 200-399% of the FPL and using a daily asthma medication were similar in Models 2 and 3. In addition to associations seen across models, children without an asthma management plan had lower odds of using a daily asthma medication (OR=0.38, 95% CI=0.19-0.75), according to results from Model 3.

Table 17 (Highlights). Associations between ED visits and criteria pollutants ($O_3$, $PM_{10}$, and $PM_{2.5}$) adjusting for vulnerability characteristics among CHIS adults with current asthma[a]

| | | $O_3$ (per 10 ppb) | | | | | | | | | $PM_{10}$ (per 10 µg/m$^3$) | | | | | | | | | $PM_{2.5}$ (per 5 µg/m$^3$) | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Model 1[b] (245, 1372)[e] | | | Model 2[c] (245, 1365)[e] | | | Model 3[d] (240, 1337)[e] | | | Model 1[b] (212, 1092)[e] | | | Model 2[b] (212, 1085)[e] | | | Model 3[d] (209, 1058)[e] | | | Model 1[b] (160, 830)[e] | | | Model 2[c] (160, 825)[e] | | | Model 3[d] (157, 807)[e] | | |
| **Vulnerability Characteristic** | | OR | 95% C.I. | | OR | 95% C.I. | | OR | 95% C.I. | | OR | 95% C.I. | | OR | 95% C.I. | | OR | 95% C.I. | | OR | 95% C.I. | | OR | 95% C.I. | | OR | 95% C.I. | |
| 12-Month Pollutant Average[f] | | 1.19 | 0.96 | 1.47 | 1.18 | 0.95 | 1.47 | 1.15 | 0.91 | 1.45 | 1.20 | 1.00 | 1.43 | 1.18 | 0.99 | 1.42 | 1.25 | 1.03 | 1.51 | 1.22 | 0.96 | 1.56 | 1.21 | 0.95 | 1.54 | 1.20 | 0.92 | 1.56 |
| Age (Ref. ≥65) | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| | 18 - 34 | 1.09 | 0.61 | 1.93 | 1.49 | 0.78 | 2.85 | 2.05 | 1.00 | 4.20 | 1.00 | 0.53 | 1.88 | 1.44 | 0.72 | 2.90 | 1.91 | 0.88 | 4.16 | 0.94 | 0.45 | 1.93 | 1.66 | 0.76 | 3.63 | 1.31 | 0.57 | 2.99 |
| | 35 - 64 | 1.91 | 1.15 | 3.20 | 2.26 | 1.30 | 3.94 | 2.33 | 1.29 | 4.19 | 1.76 | 0.99 | 3.13 | 2.19 | 1.20 | 4.00 | 2.29 | 1.19 | 4.41 | 1.64 | 0.86 | 3.15 | 2.20 | 1.13 | 4.26 | 1.88 | 0.94 | 3.76 |
| Race (Ref. White) | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| | African American | 1.86 | 1.04 | 3.32 | 1.76 | 0.97 | 3.20 | 1.40 | 0.73 | 2.68 | 1.42 | 0.74 | 2.71 | 1.30 | 0.67 | 2.53 | 1.16 | 0.58 | 2.29 | 1.92 | 0.99 | 3.71 | 1.95 | 0.98 | 3.87 | 1.38 | 0.71 | 2.67 |
| | American Indian / Alaska Native | 1.51 | 0.43 | 5.29 | 1.46 | 0.42 | 5.16 | 0.96 | 0.29 | 3.16 | 1.36 | 0.38 | 4.93 | 1.27 | 0.33 | 4.88 | 0.83 | 0.24 | 2.91 | 0.97 | 0.16 | 5.95 | 0.89 | 0.14 | 5.58 | 0.59 | 0.10 | 3.34 |
| | Asian / Pacific Islander / Other | 1.26 | 0.71 | 2.26 | 1.38 | 0.77 | 2.47 | 1.41 | 0.79 | 2.51 | 0.80 | 0.42 | 1.54 | 0.84 | 0.44 | 1.59 | 0.90 | 0.45 | 1.79 | 0.96 | 0.47 | 1.96 | 1.08 | 0.52 | 2.23 | 0.92 | 0.46 | 1.84 |
| | Latino | 2.03 | 1.27 | 3.23 | 2.08 | 1.30 | 3.35 | 2.23 | 1.32 | 3.78 | 2.42 | 1.46 | 4.00 | 2.46 | 1.47 | 4.12 | 2.40 | 1.37 | 4.22 | 2.03 | 1.18 | 3.49 | 2.38 | 1.35 | 4.22 | 2.05 | 1.09 | 3.88 |
| Poverty (Ref. ≥400% FPL)[g] | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| | 0 - 199% FPL | 1.93 | 1.26 | 2.97 | 1.62 | 1.02 | 2.57 | 1.82 | 1.12 | 2.95 | 2.02 | 1.27 | 3.20 | 1.72 | 1.06 | 2.80 | 1.95 | 1.15 | 3.31 | 1.84 | 1.11 | 3.03 | 1.51 | 0.90 | 2.54 | 1.64 | 0.93 | 2.90 |
| | 200 - 399% FPL | 1.14 | 0.69 | 1.89 | 1.07 | 0.64 | 1.80 | 1.11 | 0.68 | 1.83 | 1.34 | 0.79 | 2.27 | 1.31 | 0.76 | 2.25 | 1.37 | 0.79 | 2.39 | 1.09 | 0.57 | 2.06 | 1.09 | 0.58 | 2.05 | 0.98 | 0.51 | 1.90 |
| Sex | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| | Female vs. Male | 1.45 | 0.97 | 2.18 | 1.46 | 0.97 | 2.21 | 1.37 | 0.89 | 2.10 | 1.28 | 0.83 | 1.97 | 1.23 | 0.80 | 1.88 | 1.25 | 0.80 | 1.96 | 1.45 | 0.89 | 2.37 | 1.50 | 0.91 | 2.45 | 1.51 | 0.89 | 2.55 |
| Heart Disease | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| | Yes vs No | | | | 1.69 | 1.01 | 2.84 | | | | | | | 2.07 | 1.22 | 3.52 | | | | | | | 2.12 | 1.20 | 3.73 | | | |
| Onset of Asthma | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| | Adult vs Child | | | | | | | 1.65 | 1.08 | 2.54 | | | | | | | 1.50 | 0.94 | 2.39 | | | | | | | | | |
| Daily Asthma Medication | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| | No vs Yes | | | | | | | 0.35 | 0.23 | 0.52 | | | | | | | 0.30 | 0.20 | 0.47 | | | | | | | 0.29 | 0.17 | 0.49 |
| Asthma Management Plan | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| | No vs Yes | | | | | | | 0.52 | 0.36 | 0.76 | | | | | | | 0.56 | 0.37 | 0.84 | | | | | | | 0.63 | 0.40 | 1.00 |

[a]For CHIS 2003, current asthma is defined as reporting an asthma attack in the previous 12 months or answering yes to the question, "Do you still have asthma?"; only respondents who lived within 5 miles of an air monitoring station and lived in the same home or neighborhood for at least 9 months were included.
[b]Model 1 controlled for age, race, poverty level, and sex.
[c]Model 2 controlled for the following vulnerability factors: insurance status, obesity, heart disease, smoking status, and work status, in addition to age, race, poverty level, and sex.
[d]Model 3 controlled for the following vulnerability factors: urban/rural, usual source of care, delay in care, onset of asthma, daily asthma medication, asthma management plan, household smoking, dog or cat in home, cockroaches in home, housing type, household crowding, diabetes, and walking, in addition to age, race, poverty level, and sex.
[e](Cases, Non-cases)
[f]Refers to the criteria pollutant noted in the column heading.
[g]FPL = Federal Poverty Level

Table 18 (Highlights). Associations between daily asthma medication and criteria pollutants ($O_3$, $PM_{10}$, and $PM_{2.5}$) adjusting for vulnerability characteristics among CHIS adults with current asthma[a]

| | $O_3$ (per 10 ppb) | | | | | | | | | $PM_{10}$ (per 10 µg/m$^3$) | | | | | | | | | $PM_{2.5}$ (per 5 µg/m$^3$) | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Model 1[b] (815, 802)[e] | | | Model 2[c] (811, 799)[e] | | | Model 3[d] (789, 788)[e] | | | Model 1[b] (659, 645)[e] | | | Model 2[c] (654, 643)[e] | | | Model 3[d] (634, 633)[e] | | | Model 1[b] (494, 496)[e] | | | Model 2[c] (491, 494)[e] | | | Model 3[d] (479, 485)[e] | | |
| **Vulnerability Characteristic** | OR | 95% C.I. | | OR | 95% C.I. | | OR | 95% C.I. | | OR | 95% C.I. | | OR | 95% C.I. | | OR | 95% C.I. | | OR | 95% C.I. | | OR | 95% C.I. | | OR | 95% C.I. | |
| 12-Month Pollutant Average[f] | 1.22 | 1.04 | 1.43 | 1.23 | 1.05 | 1.46 | 1.24 | 1.05 | 1.48 | 1.12 | 0.96 | 1.30 | 1.12 | 0.96 | 1.30 | 1.23 | 1.04 | 1.45 | 1.26 | 1.05 | 1.52 | 1.30 | 1.07 | 1.57 | 1.44 | 1.18 | 1.76 |
| Age (Ref. ≥65) | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| 18 - 34 | 0.32 | 0.21 | 0.48 | 0.52 | 0.33 | 0.83 | 0.30 | 0.19 | 0.48 | 0.32 | 0.20 | 0.51 | 0.51 | 0.30 | 0.87 | 0.33 | 0.19 | 0.55 | 0.40 | 0.24 | 0.66 | 0.80 | 0.44 | 1.44 | 0.41 | 0.23 | 0.73 |
| 35 - 64 | 0.55 | 0.39 | 0.78 | 0.76 | 0.52 | 1.12 | 0.51 | 0.35 | 0.74 | 0.53 | 0.36 | 0.79 | 0.72 | 0.46 | 1.13 | 0.50 | 0.32 | 0.77 | 0.56 | 0.36 | 0.88 | 0.91 | 0.55 | 1.49 | 0.52 | 0.32 | 0.85 |
| Race (Ref. White) | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| African American | 1.49 | 0.92 | 2.41 | 1.54 | 0.95 | 2.49 | 1.52 | 0.92 | 2.51 | 1.85 | 1.07 | 3.19 | 1.98 | 1.15 | 3.41 | 2.16 | 1.22 | 3.84 | 1.62 | 0.92 | 2.84 | 1.87 | 1.05 | 3.32 | 1.76 | 0.97 | 3.18 |
| American Indian / Alaska Native | 1.88 | 0.74 | 4.75 | 1.89 | 0.77 | 4.66 | 1.77 | 0.74 | 4.24 | 2.46 | 0.92 | 6.62 | 2.56 | 0.97 | 6.77 | 2.47 | 1.02 | 5.99 | 1.85 | 0.55 | 6.29 | 1.85 | 0.57 | 6.00 | 1.84 | 0.59 | 5.73 |
| Asian / Pacific Islander / Other | 0.75 | 0.47 | 1.20 | 0.86 | 0.54 | 1.38 | 0.71 | 0.44 | 1.16 | 0.72 | 0.41 | 1.26 | 0.81 | 0.46 | 1.42 | 0.86 | 0.48 | 1.53 | 0.77 | 0.45 | 1.31 | 0.84 | 0.49 | 1.42 | 0.87 | 0.50 | 1.52 |
| Latino | 0.81 | 0.54 | 1.20 | 0.90 | 0.59 | 1.36 | 0.90 | 0.58 | 1.39 | 1.06 | 0.66 | 1.68 | 1.17 | 0.72 | 1.90 | 1.33 | 0.80 | 2.21 | 0.65 | 0.40 | 1.06 | 0.79 | 0.48 | 1.29 | 0.78 | 0.45 | 1.36 |
| Poverty (Ref. ≥400% FPL)[g] | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| 0 - 199% FPL | 1.30 | 0.94 | 1.79 | 1.16 | 0.82 | 1.65 | 1.41 | 0.98 | 2.03 | 1.16 | 0.81 | 1.65 | 1.06 | 0.72 | 1.57 | 1.28 | 0.86 | 1.90 | 1.28 | 0.86 | 1.91 | 1.12 | 0.73 | 1.72 | 1.45 | 0.94 | 2.22 |
| 200 - 399% FPL | 0.96 | 0.68 | 1.36 | 0.96 | 0.68 | 1.36 | 0.96 | 0.68 | 1.36 | 0.92 | 0.62 | 1.35 | 0.94 | 0.63 | 1.38 | 0.93 | 0.62 | 1.38 | 0.85 | 0.55 | 1.30 | 0.87 | 0.56 | 1.34 | 0.89 | 0.57 | 1.37 |
| Sex | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Female vs. Male | 1.12 | 0.85 | 1.48 | 1.11 | 0.84 | 1.48 | 1.09 | 0.82 | 1.47 | 0.95 | 0.69 | 1.30 | 0.98 | 0.71 | 1.35 | 0.94 | 0.67 | 1.31 | 1.06 | 0.75 | 1.51 | 1.07 | 0.75 | 1.53 | 0.98 | 0.68 | 1.41 |
| Currently Insured | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Yes vs No | | | | 1.56 | 1.02 | 2.38 | | | | | | | | | | | | | | | | 1.86 | 1.11 | 3.11 | | | |
| Heart Disease | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Yes vs No | | | | 1.77 | 1.16 | 2.69 | | | | | | | 1.81 | 1.12 | 2.91 | | | | | | | 1.73 | 1.05 | 2.84 | | | |
| Smoker | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Ever vs Never | | | | 1.41 | 1.07 | 1.86 | | | | | | | 1.31 | 0.97 | 1.77 | | | | | | | 1.47 | 1.04 | 2.06 | | | |
| Usual Source of Care | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Yes vs No | | | | | | | 2.28 | 1.35 | 3.85 | | | | | | | 1.83 | 1.02 | 3.28 | | | | | | | 3.07 | 1.57 | 5.99 |
| Delay in Care | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Yes vs No | | | | | | | | | | | | | | | | 0.68 | 0.47 | 0.97 | | | | | | | | | |
| Asthma Management Plan | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| No vs Yes | | | | | | | 0.46 | 0.35 | 0.61 | | | | | | | 0.44 | 0.32 | 0.60 | | | | | | | 0.41 | 0.29 | 0.59 |
| Cockroaches | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Yes vs No | | | | | | | 0.58 | 0.38 | 0.88 | | | | | | | 0.54 | 0.33 | 0.87 | | | | | | | 0.59 | 0.36 | 0.97 |

[a]For CHIS 2003, current asthma is defined as reporting an asthma attack in the previous 12 months or answering yes to the question, "Do you still have asthma?"; only respondents who lived within 5 miles of an air monitoring station and lived in the same home or neighborhood for at least 9 months were included.

[b]Model 1 controlled for age, race, poverty level, and sex.

[c]Model 2 controlled for the following vulnerability factors: insurance status, obesity, heart disease, smoking status, and work status, in addition to age, race, poverty level, and sex.

[d]Model 3 controlled for the following vulnerability factors: urban/rural, usual source of care, delay in care, onset of asthma, asthma management plan, household smoking, dog or cat in home, cockroaches in home, housing type, household crowding, diabetes, and walking, in addition to age, race, poverty level, and sex.

[e](Cases, Non-cases)

[f]Refers to the criteria pollutant noted in the column heading.

[g]FPL = Federal Poverty Level

Table 19 (Highlights). Associations between missing 2 or more days of work and criteria pollutants ($O_3$, $PM_{10}$, and $PM_{2.5}$) adjusting for vulnerability characteristics among CHIS adults with current asthma[a]

| | | $O_3$ (per 10 ppb) | | | | | | | | | $PM_{10}$ (per 10 µg/m$^3$) | | | | | | | | | $PM_{2.5}$ (per 5 µg/m$^3$) | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Model 1[b] (176, 1208)[e] | | | Model 2[c] (176, 1208)[e] | | | Model 3[d] (175, 1185)[e] | | | Model 1[b] (142, 977)[e] | | | Model 2[c] (142, 977)[e] | | | Model 3[d] (141, 957)[e] | | | Model 1[b] (118, 743)[e] | | | Model 2[c] (118, 743)[e] | | | Model 3[d] (118, 727)[e] | | |
| **Vulnerability Characteristic** | | OR | 95% C.I. | | OR | 95% C.I. | | OR | 95% C.I. | | OR | 95% C.I. | | OR | 95% C.I. | | OR | 95% C.I. | | OR | 95% C.I. | | OR | 95% C.I. | | OR | 95% C.I. | |
| 12-Month Pollutant Average[f] | | 1.15 | 0.91 | 1.46 | 1.15 | 0.91 | 1.44 | 1.13 | 0.88 | 1.44 | 1.28 | 1.00 | 1.65 | 1.30 | 1.01 | 1.68 | 1.25 | 0.96 | 1.63 | 1.23 | 0.94 | 1.60 | 1.23 | 0.94 | 1.60 | 1.24 | 0.93 | 1.66 |
| Race (Ref. White) | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| | African American | 3.79 | 2.02 | 7.13 | 3.61 | 1.92 | 6.79 | 3.81 | 1.82 | 7.99 | 3.60 | 1.73 | 7.47 | 3.45 | 1.69 | 7.05 | 3.25 | 1.43 | 7.42 | 4.69 | 2.24 | 9.81 | 4.45 | 2.11 | 9.41 | 5.50 | 2.35 | 12.89 |
| | American Indian / Alaska Native | 1.73 | 0.51 | 5.85 | 1.62 | 0.46 | 5.79 | 1.86 | 0.52 | 6.71 | 1.83 | 0.55 | 6.08 | 1.67 | 0.45 | 6.16 | 1.93 | 0.57 | 6.52 | 1.13 | 0.18 | 7.28 | 1.01 | 0.15 | 6.86 | 1.34 | 0.21 | 8.53 |
| | Asian / Pacific Islander / Other | 2.06 | 1.07 | 3.97 | 1.98 | 1.01 | 3.87 | 2.18 | 1.11 | 4.30 | 1.15 | 0.49 | 2.68 | 1.05 | 0.46 | 2.42 | 1.14 | 0.47 | 2.78 | 1.74 | 0.82 | 3.70 | 1.69 | 0.78 | 3.64 | 1.92 | 0.91 | 4.05 |
| | Latino | 3.42 | 2.06 | 5.68 | 3.30 | 1.99 | 5.49 | 3.86 | 2.25 | 6.64 | 3.60 | 2.00 | 6.47 | 3.51 | 1.94 | 6.36 | 3.82 | 2.03 | 7.18 | 3.28 | 1.76 | 6.10 | 3.31 | 1.76 | 6.23 | 4.21 | 2.12 | 8.33 |
| Poverty (Ref. ≥400% FPL)[g] | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| | 0 - 199% FPL | 0.57 | 0.34 | 0.93 | 0.59 | 0.34 | 1.04 | 0.45 | 0.24 | 0.87 | 0.58 | 0.33 | 1.02 | 0.67 | 0.36 | 1.23 | 0.54 | 0.27 | 1.08 | 0.37 | 0.21 | 0.67 | 0.39 | 0.21 | 0.74 | 0.36 | 0.16 | 0.78 |
| | 200 - 399% FPL | 1.13 | 0.67 | 1.89 | 1.16 | 0.69 | 1.96 | 1.25 | 0.75 | 2.10 | 1.30 | 0.73 | 2.32 | 1.47 | 0.82 | 2.63 | 1.53 | 0.85 | 2.76 | 0.92 | 0.50 | 1.70 | 1.00 | 0.53 | 1.90 | 1.17 | 0.64 | 2.15 |
| Sex | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| | Female vs. Male | 2.26 | 1.39 | 3.68 | 2.14 | 1.31 | 3.51 | 2.05 | 1.24 | 3.40 | 2.14 | 1.25 | 3.65 | 1.99 | 1.16 | 3.40 | 2.13 | 1.20 | 3.80 | 2.23 | 1.24 | 4.03 | 2.11 | 1.15 | 3.88 | 2.18 | 1.18 | 4.03 |
| Daily Asthma Medication | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| | No vs Yes | | | | | | | 0.44 | 0.28 | 0.69 | | | | | | | 0.44 | 0.26 | 0.74 | | | | | | | 0.49 | 0.28 | 0.86 |

[a]For CHIS 2003, current asthma is defined as reporting an asthma attack in the previous 12 months or answering yes to the question, "Do you still have asthma?"; only respondents who lived within 5 miles of an air monitoring station and lived in the same home or neighborhood for at least 9 months were included.
[b]Model 1 controlled for age, race, poverty level, and sex.
[c]Model 2 controlled for the following vulnerability factors: insurance status, obesity, heart disease, smoking status, and work status, in addition to age, race, poverty level, and sex.
[d]Model 3 controlled for the following vulnerability factors: urban/rural, usual source of care, delay in care, onset of asthma, daily asthma medication, asthma management plan, household smoking, dog or cat in home, cockroaches in home, housing type, household crowding, diabetes, and walking, in addition to age, race, poverty level, and sex.
[e](Cases, Non-cases)
[f]Refers to the criteria pollutant noted in the column heading.
[g]FPL = Federal Poverty Level

Table 20 (Highlights). Associations between daily asthma medication and $PM_{2.5}$ adjusting for vulnerability characteristics among CHIS 2003 children with current asthma[a]

| | | $PM_{2.5}$ (per 5 μg/m$^3$) (132, 203)[b] | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | Model 1[c] | | | Model 2[d] | | | Model 3[e] | | |
| **Vulnerability Characteristic** | | OR | 95% C.I. | | OR | 95% C.I. | | OR | 95% C.I. | |
| 12-Month Pollutant Average[f] | | 1.20 | 0.88 | 1.63 | 1.20 | 0.87 | 1.64 | 1.28 | 0.93 | 1.76 |
| Age (Ref. 6-11) | | | | | | | | | | |
| | ≤ 6 | 0.89 | 0.40 | 1.99 | 0.89 | 0.40 | 1.99 | 1.04 | 0.46 | 2.36 |
| | 12 - 17 | 1.07 | 0.56 | 2.07 | 1.07 | 0.55 | 2.09 | 1.51 | 0.74 | 3.12 |
| Race (Ref. White) | | | | | | | | | | |
| | African American | 1.16 | 0.46 | 2.92 | 1.15 | 0.43 | 3.07 | 1.03 | 0.40 | 2.71 |
| | American Indian / Alaska Native | 1.11 | 0.15 | 8.44 | 1.11 | 0.14 | 8.82 | 0.84 | 0.11 | 6.26 |
| | Asian / Pacific Islander / Other | 0.92 | 0.34 | 2.49 | 0.97 | 0.36 | 2.60 | 0.86 | 0.32 | 2.29 |
| | Latino | 1.43 | 0.66 | 3.10 | 1.49 | 0.68 | 3.26 | 1.31 | 0.57 | 3.00 |
| Poverty (Ref. ≥400% FPL)[g] | | | | | | | | | | |
| | 0 - 199% FPL | 2.64 | 1.22 | 5.72 | 2.51 | 1.14 | 5.56 | 3.21 | 1.40 | 7.39 |
| | 200 - 399% FPL | 3.00 | 1.46 | 6.14 | 2.86 | 1.40 | 5.87 | 3.56 | 1.66 | 7.60 |
| Sex | | | | | | | | | | |
| | Female vs. Male | 0.96 | 0.52 | 1.75 | 0.96 | 0.52 | 1.76 | 0.92 | 0.51 | 1.69 |
| Asthma Management Plan | | | | | | | | | | |
| | No vs Yes | | | | | | | 0.38 | 0.19 | 0.75 |

[a]For CHIS 2003, current asthma is defined as reporting an asthma attack in the previous 12 months or answering yes to the question, "Do you still have asthma?"; only respondents who lived within 5 miles of an air monitoring station and lived in the same home or neighborhood for at least 9 months were included.
[b](Cases, Non-cases)
[c]Model 1 controlled for age, race, poverty level, and sex.
[d]Model 2 controlled for the following vulnerability factors: household smoking, dog or cat in home, cockroaches in home, and insurance status, in addition to age, race, poverty level, and sex.
[e]Model 3 controlled for the following vulnerability factors: urban/rural residency, delay in care, asthma management plan, housing type, and household crowding, in addition to age, race, poverty level, and sex.
[f]Refers to the criteria pollutant noted in the column heading.
[g]FPL = Federal Poverty Level

*Interactions between Pollutant Exposures and Vulnerability of Sub-Populations*

In this part of the study, we tested the hypothesis that higher pollutant exposures interact with these vulnerability factors resulting in greater air pollution impacts on asthma in vulnerable sub-populations, i.e. racial/ethnic minorities, and low-income individuals (Hypothesis 4). We tested for interactions between the pollutant exposure and vulnerability from belonging to a low SES sub-population using logistical regression models that included the main effect and interaction terms and held all control variables constant at the reference level. We present the findings of interactions between pollutant exposure and sub-populations, characterized by race/ethnicity and income.

*Interactions of $NO_2$ with poverty*

We examined the potential modifying effects of income measured by poverty level on the relationships between air pollutants and asthma outcomes among respondents with current asthma by adding appropriate interaction terms to our regression models while adjusting for age, race, and sex. For children with current asthma, we found that those living below 200% of the FPL had greater increases in odds of ED visits as $NO_2$ annual average concentrations increased than children living at or above 400% of the FPL (p=0.01) (Figure 5 and Table 21).

*Interactions of $NO_2$ and $PM_{10}$ with race/ethnicity*

Significant interactions between $NO_2$ and race/ethnicity were also observed (Figure 6 and Table 22). Among adults, African Americans and Asians/PIs/Others had greater increases in odds of missing 2 or more work days as annual average $NO_2$ increased (p=0.03 and p<0.01, respectively). African American adults also experienced greater increases in odds of daily/weekly symptoms than white respondents with the same increase in annual average $NO_2$ (p=0.03). American Indian/Alaska Native children (p < 0.001) and Asian/PI/other children (p=0.04) showed greater increases in odds of daily/weekly asthma symptoms than white children with the same increase in annual average $NO_2$.

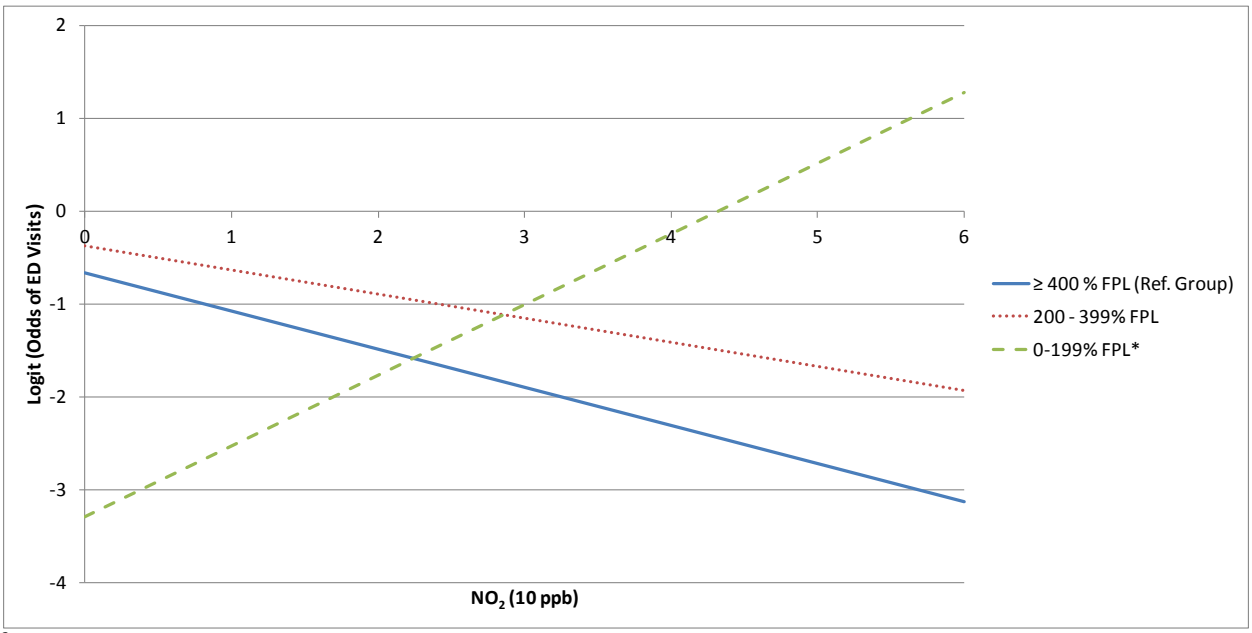
For $PM_{10}$, significant interactions with race/ethnicity were found among children for two outcomes, using daily asthma medication and experiencing daily/weekly asthma symptoms (Figure 7 and Table 23). Latino children had greater increases in odds of daily asthma medication use compared to white children as annual average $PM_{10}$ increased (p=0.05). Both African American children (p=0.03) and Asian/PI/other (p=0.03) children had greater increases in odds of daily/weekly symptoms in comparison to white children with the same level of increase in $PM_{10}$ annual average concentration.

*Estimated odds in vulnerable sub-populations*

Based on the significant interaction models described above, we calculated odds ratios and 95% confidence intervals to quantify the increased influence of criteria pollutants on asthma outcomes by sub-population group. While significant differences in pollutant effect on asthma outcomes by sub-population were observed, there was not enough statistical power to quantify the strength of association by racial/ethnic group or poverty level in most models. Therefore, the confidence intervals for the ORs often crossed the null except for a few

interaction odds. The results of these calculations are noted in Tables 24 and 25 in the Appendix.

Figure 5. Interaction between mean $NO_2$ annual exposure and Federal Poverty Level (FPL) on log odds of ED visits in CHIS 2003 children with current asthma[a,b]




[a]For CHIS 2003, current asthma is defined as reporting an asthma attack in the previous 12 months or answering yes to the question, "Do you still have asthma?"; only respondents who lived within 5 miles of an air monitoring station and lived in the same home or neighborhood for at least 9 months were included.
[b]Adjusted for age, race, poverty level, and sex.
*p < 0.05, **p < 0.01, ***p < 0.001

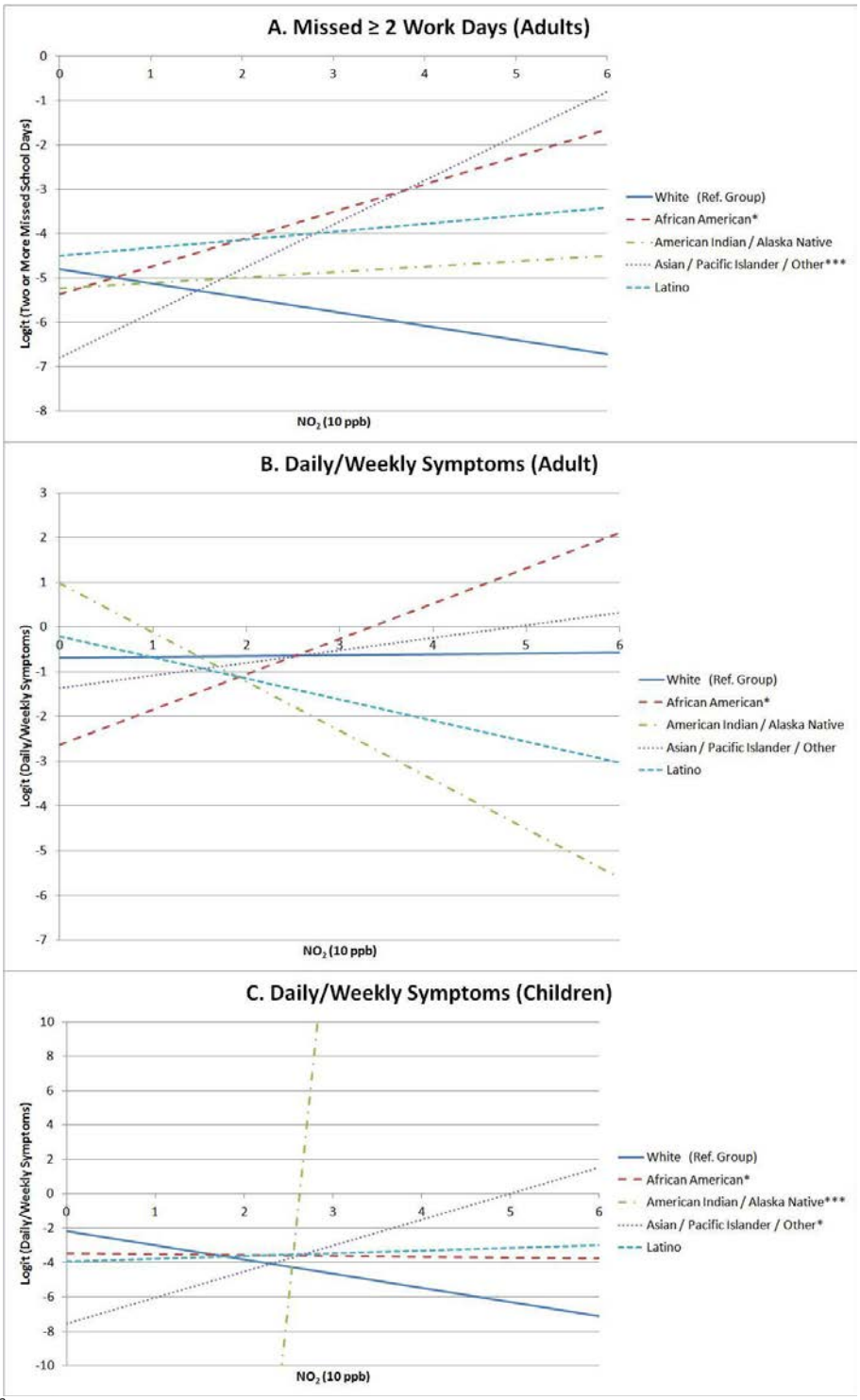
Table 21. Interaction between mean $NO_2$ annual exposure and Federal Poverty Level (FPL) on log odds of ED visits in CHIS 2003 children with current asthma

| | | ED Visits (Children) | | |
|---|---|---|---|---|
| Variables | | Estimate | Std Error | p-value |
| Intercept | | -0.67 | 0.76 | 0.38 |
| $NO_2$ (per 10 ppb)[b] | | -0.41 | 0.36 | 0.25 |
| Age | | | | |
| (Ref. 6 - 11 year | 0-5 | 0.88 | 0.33 | 0.01 |
| | 12-17 | -1.70 | 0.42 | <.0001 |
| Race (Ref. Whites) | | | | |
| | African American | 0.67 | 0.42 | 0.11 |
| | American Indian / Alaska Native | 0.65 | 0.93 | 0.48 |
| | Asian / Pacific Islander / Other | -0.61 | 0.55 | 0.27 |
| | Latino | 0.32 | 0.39 | 0.41 |
| Poverty (Ref. ≥ 400% FPL) | | | | |
| | 0 - 199% FPL | -2.62 | 1.03 | 0.01 |
| | 200 - 399% FPL | 0.30 | 0.98 | 0.76 |
| Sex (Ref. Male) | | | | |
| | Female | 0.08 | 0.31 | 0.79 |
| $NO_2$ * Poverty (0-199% FPL) | | 1.17 | 0.46 | 0.01 |
| $NO_2$ * Poverty (200-399% FPL) | | 0.15 | 0.48 | 0.76 |

[a]For CHIS 2003, current asthma is defined as reporting an asthma attack in the previous 12 months or answering yes to the question "do you still have asthma?"; only respondents who lived within 5 miles of an air monitoring station and lived in the same home or neighborhood for at least 9 months were included.

[b]Represents the pollutant estimate for the reference group of the interaction term, in this case respondents living at ≥ 400% FPL with current asthma.

Figure 6. Interaction between mean $NO_2$ annual exposure and race/ethnicity on log odds of various asthma outcomes in CHIS 2003 children and adults with current asthma[a,b]




[a]For CHIS 2003, current asthma is defined as reporting an asthma attack in the previous 12 months or answering yes to the question, "Do you still have asthma?"; only respondents who lived within 5 miles of an air monitoring station and lived in the same home or neighborhood for at least 9 months were included.

[b]Adjusted for age, race, poverty level, and sex

*p < 0.05, **p < 0.01, ***p < 0.001

Table 22. Interaction between mean $NO_2$ annual exposure and race/ethnicity on log odds of various asthma outcomes in CHIS 2003 children and adults with current asthma[a]

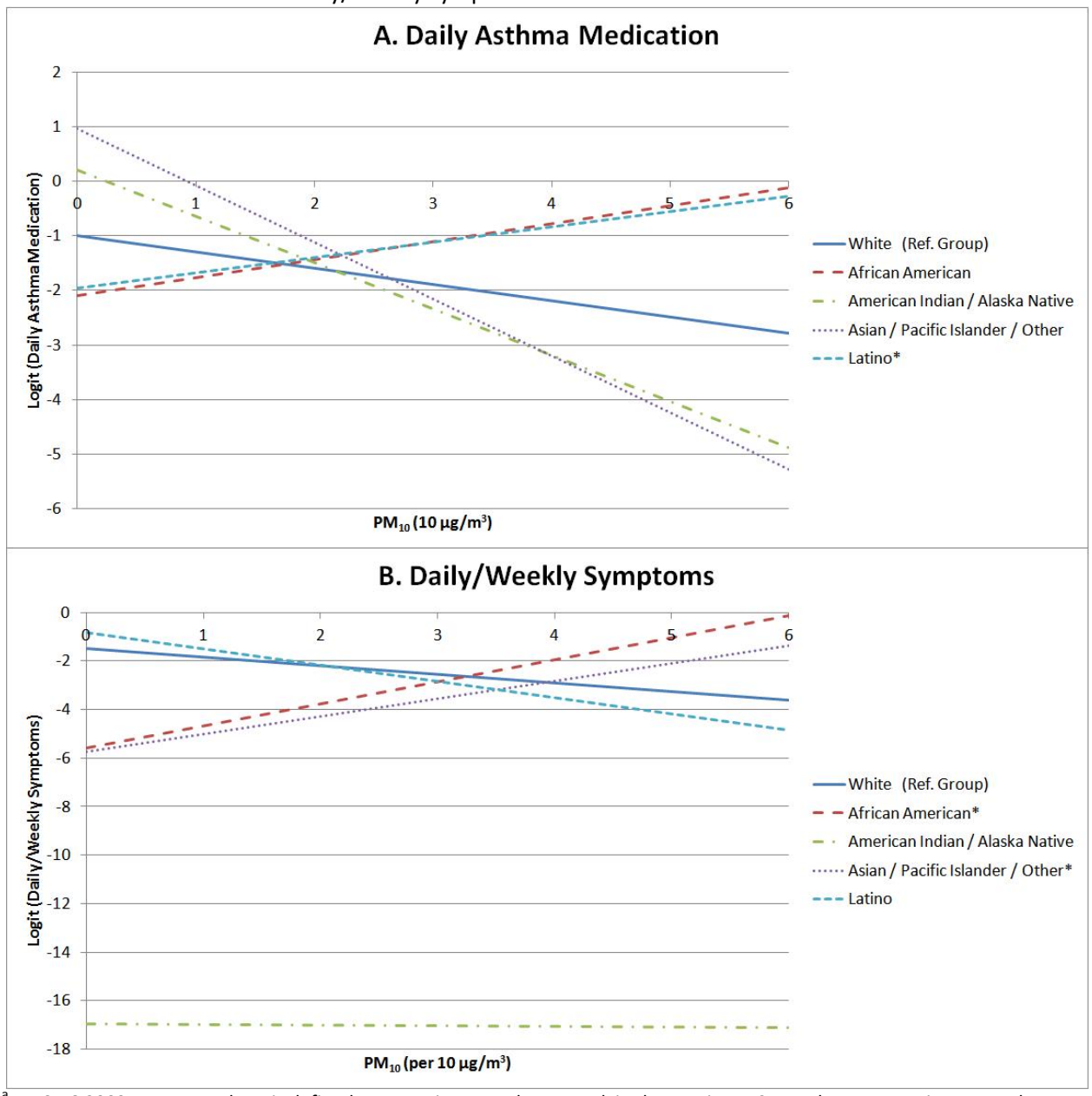
| | | Missed ≥ 2 Work Days (Adults) | | | Daily/Weekly Symptoms (Adults) | | | Daily/Weekly Symptoms (Children) | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Variables | | Estimate | Std Error | p-value | Estimate | Std Error | p-value | Estimate | Std Error | p-value |
| Intercept | | -4.80 | 1.12 | <.0001 | -0.69 | 0.31 | 0.03 | -2.19 | 0.83 | 0.01 |
| $NO_2$ (per 10 ppb)[b] | | -0.32 | 0.22 | 0.15 | 0.02 | 0.12 | 0.85 | -0.82 | 0.38 | 0.03 |
| Age | | | | | | | | | | |
| (Ref. 6 - 11 year old) | 0-5 | | | | | | | 0.06 | 0.59 | 0.92 |
| | 12-17 | | | | | | | 0.70 | 0.47 | 0.14 |
| (Ref. ≥ 65 years old) | 18-34 | 2.49 | 1.05 | 0.02 | -0.85 | 0.24 | 0.00 | | | |
| | 35-64 | 2.70 | 1.03 | 0.01 | -0.18 | 0.19 | 0.34 | | | |
| Race (Ref. Whites) | | | | | | | | | | |
| | African American | -0.57 | 1.01 | 0.57 | -1.95 | 0.86 | 0.02 | -1.27 | 1.69 | 0.45 |
| | American Indian / Alaska Native (AI/AN) | -0.43 | 1.88 | 0.82 | 1.67 | 1.23 | 0.17 | -128 | 6.27 | <.0001 |
| | Asian / Pacific Islander (PI) / Other | -2.00 | 1.07 | 0.06 | -0.68 | 0.75 | 0.37 | -5.37 | 3.19 | 0.09 |
| | Latino | 0.30 | 0.81 | 0.72 | 0.48 | 0.68 | 0.49 | -1.77 | 1.17 | 0.13 |
| Poverty (Ref. ≥ 400% FPL)[c] | | | | | | | | | | |
| | 0 - 199% FPL | -0.68 | 0.28 | 0.02 | 0.49 | 0.19 | 0.01 | 1.21 | 0.54 | 0.03 |
| | 200 - 399% FPL | -0.04 | 0.29 | 0.88 | -0.01 | 0.21 | 0.95 | 1.33 | 0.52 | 0.01 |
| Sex (Ref. Male) | | | | | | | | | | |
| | Female | 0.80 | 0.28 | <0.01 | 0.13 | 0.17 | 0.44 | 0.24 | 0.42 | 0.57 |
| $NO_2$ * Race (African American) | | 0.94 | 0.44 | 0.03 | 0.77 | 0.36 | 0.03 | 0.77 | 0.78 | 0.33 |
| $NO_2$ * Race (AI/AN) | | 0.44 | 0.84 | 0.60 | -1.12 | 0.78 | 0.15 | 50.4 | 2.43 | <.0001 |
| $NO_2$ * Race (Asian/PI/Other) | | 1.32 | 0.43 | 0.00 | 0.26 | 0.32 | 0.42 | 2.33 | 1.13 | 0.04 |
| $NO_2$ * Race (Latino) | | 0.50 | 0.34 | 0.14 | -0.49 | 0.28 | 0.08 | 0.98 | 0.53 | 0.06 |

[a]For CHIS 2003, current asthma is defined as reporting an asthma attack in the previous 12 months or answering yes to the question, "Do you still have asthma?"; only respondents who lived within 5 miles of an air monitoring station and lived in the same home or neighborhood for at least 9 months were included.

[b]Represent the pollutant estimate for the reference group of the interaction term, in this case white respondents with current asthma

[c]FPL = Federal Poverty Level

Figure 7. Interaction between mean $PM_{10}$ annual exposure and race/ethnicity on log odds of daily asthma medication use and daily/weekly symptoms in CHIS 2003 children with current asthma[a,b]




[a]For CHIS 2003, current asthma is defined as reporting an asthma attack in the previous 12 months or answering yes to the question, "Do you still have asthma?"; only respondents who lived within 5 miles of an air monitoring station and lived in the same home or neighborhood for at least 9 months were included.
[b]Adjusted for age, race, poverty level, and sex
*p < 0.05, **p < 0.01, ***p < 0.001

Table 23. Interaction between mean $PM_{10}$ annual exposure and race/ethnicity on log odds of daily asthma medication use and daily/weekly symptoms in CHIS 2003 children with current asthma[a]

| | | Daily Asthma Medication (Children) | | | Daily / Weekly Symptoms (Children) | | |
|---|---|---|---|---|---|---|---|
| Variable | | Estimate | Std Error | p-value | Estimate | Std Error | p-value |
| Intercept | | -0.99 | 0.60 | 0.10 | -1.47 | 0.81 | 0.07 |
| $PM_{10}$ (per 10 $\mu g/m^3$)[b] | | -0.30 | 0.19 | 0.12 | -0.36 | 0.27 | 0.17 |
| Age (Ref. 6-11 years old) | | | | | | | |
| | 0-5 | 0.25 | 0.39 | 0.52 | 0.28 | 0.63 | 0.66 |
| | 12-17 | -0.23 | 0.30 | 0.45 | 0.15 | 0.45 | 0.74 |
| Race (Ref. Whites) | | | | | | | |
| | African American | -1.11 | 1.57 | 0.48 | -4.11 | 2.06 | 0.05 |
| | American Indian / Alaska Native (AI/AN) | 1.20 | 2.16 | 0.58 | -15.50 | 1.38 | <.0001 |
| | Asian / Pacific Islander (PI) / Other | 1.96 | 1.31 | 0.13 | -4.26 | 1.54 | 0.01 |
| | Latino | -0.97 | 0.96 | 0.31 | 0.64 | 1.25 | 0.61 |
| Poverty (Ref. ≥ 400% FPL)[c] | | | | | | | |
| | 0 - 199% FPL | 0.81 | 0.35 | 0.02 | 0.41 | 0.50 | 0.40 |
| | 200 - 399% FPL | 1.05 | 0.33 | 0.00 | 0.79 | 0.43 | 0.07 |
| Sex (Ref. Male) | | | | | | | |
| | Female | 0.57 | 0.28 | 0.04 | -0.06 | 0.43 | 0.90 |
| $PM_{10}$ * Race (African American) | | 0.63 | 0.52 | 0.23 | 1.27 | 0.57 | 0.03 |
| $PM_{10}$ * Race (AI/AN) | | -0.55 | 0.67 | 0.41 | 0.34 | 0.45 | 0.45 |
| $PM_{10}$ * Race (Asian/PI/Other) | | -0.74 | 0.45 | 0.10 | 1.09 | 0.49 | 0.03 |
| $PM_{10}$ * Race (Latino) | | 0.58 | 0.30 | 0.05 | -0.31 | 0.44 | 0.48 |

[a]For CHIS 2003, current asthma is defined as reporting an asthma attack in the previous 12 months or answering yes to the question, "Do you still have asthma?"; only respondents who lived within 5 miles of an air monitoring station and lived in the same home or neighborhood for at least 9 months were included.

[b]Represent the pollutant estimate for the reference group of the interaction term, in this case white respondents with current asthma.

[c]FPL = Federal Poverty Level

## CHIS 2003 Respondents with Asthma-like Symptoms

### Exposure distributions for CHIS 2003 respondents with asthma-like symptoms

Distributions of annual average criteria pollutant exposures, exceedance days, and traffic density for CHIS 2003 respondents with asthma-like symptoms are shown in Table 26 in the Appendix. Among adults, annual average $O_3$ exposure ranged from 22.9-65.7 ppb, with a mean of 41.7 ppb. Annual average exposure to $PM_{10}$ ranged from 7.9- 82.8 µg/m$^3$, with a mean of 30.1 µg/m$^3$, and annual average exposure to $PM_{2.5}$ ranged from 4.1-26.9 µg/m$^3$ with a mean of 16.5 µg/m$^3$. Annual average exposure to $NO_2$ ranged from 1.4-36.1 ppb, with a mean of 22.0 ppb.

On average, adults experienced 23.6 days when maximum 1-hr $O_3$ concentrations exceeded the 1-hr state standard (range of 0-131 days), 32.1 days when the maximum 8-hr $O_3$ concentration exceeded the 8-hr state standard (range 0-160 days) and 17.7 days when the maximum 8-hr $O_3$ concentration exceeded the 8-hr federal standard (range of 0-130 days). Adult 24-hr averages for $PM_{10}$ rarely exceeded the federal standard (mean=0.10 days, range 0-4 days), but averages more frequently exceeded the state standard (mean=8.2 days, range 0-66 days). Adults experienced an average of 16.1 days when $PM_{2.5}$ levels exceeded the federal standard (range of 0-54 days).

Adults with asthma-like symptoms had a mean traffic density of 65.8 VMT/day/meters$^2$, with a minimum of 1.0 and a maximum of 745.4. Among children with asthma-like symptoms, annual average exposures to $O_3$ ranged from 22.9-63.5 ppb, with a mean of 42.1 ppb. Annual average $PM_{10}$ exposure ranged from 12.8-82.8 µg/m$^3$ (mean=29.9 µg/m$^3$), and annual average $PM_{2.5}$ exposure ranged from 6.6-26.3 µg/m$^3$ (mean=16.5 µg/m$^3$). Annual averages for $NO_2$ ranged from 5.0-36.0 ppb, with a mean of 22.3 ppb.

Children with asthma-like symptoms had a mean of 24.6 $O_3$ exceedance days based on the 1-hr state standard (range 0-122 days), a mean of 33.9 exceedance days based on the 8-hr state standard (range of 0-153 days), and a mean of 18.6 exceedance days based on the 8-hr federal standard (range 0-114 days) in the year prior to the CHIS interview. Under the federal $PM_{10}$ standard, children with asthma-like symptoms had a maximum of 4 exceedance days and an average of 0.12 exceedance days; under the state $PM_{10}$ standard, they had a maximum of 64 exceedance days and an average of 7.9 exceedance days. The maximum number of $PM_{2.5}$ exceedance days was 54 days, with a mean of 16.6. Traffic density exposure ranged from 0.5-637.2 VMT/day/meters$^2$, with a mean of 64.1, among children with asthma-like symptoms. Frequencies for distance to roadway measures among adults and children with asthma-like symptoms are shown in Table 27 in the Appendix. Among adults with asthma-like symptoms, 6.1% lived within 300 meters of a state highway, and 10.4% lived within 300 meters of an interstate highway. One-fifth (19.7%) of adults with asthma-like symptoms lived within 50 meters of a major road. Among children with asthma-like symptoms, 5.3% lived within 300 meters of a state highway, and 10.8% lived within 300 meters of an interstate highway. Close to one-fifth (17.8%) lived within 50 meters of a major road.

### Correlations among air pollutant exposure estimates

Table 28 in the Appendix shows Pearson correlation coefficients for exposure metrics among CHIS 2003 respondents with asthma-like symptoms. Annual average exposure estimates

for $PM_{10}$ and $PM_{2.5}$ were strongly positively correlated (r=0.74), as were estimates for $PM_{2.5}$ and $NO_2$ (r=0.71). $PM_{10}$ annual averages were moderately correlated with $O_3$ and $NO_2$ (r=0.48 and r=0.50). State and federal $O_3$ exceedance measures had strong positive correlations with annual average $O_3$ exposure (r≈0.8) and moderate correlations with annual average $PM_{10}$ exposure (r≈0.6). Annual average $PM_{10}$ exposure was highly correlated with $PM_{10}$ state standard exceedances (r=0.83). Exceedances of the $PM_{10}$ state standard were moderately correlated with exceedances of state and federal standards for $O_3$ (r≈0.6). Moderate to weak correlations were observed between annual average exposures and exceedance measures across the other pollutants. Weak correlations were observed between traffic density and distance to roadway measures and the criteria pollutant exposure metrics.

**Health outcomes and characteristics of adults and children with asthma-like symptoms**

Table 29 shows health outcomes among respondents without an asthma diagnosis who reported asthma-like symptoms and had lived in the same home or neighborhood for at least 9 months prior to being interviewed. Among adults without an asthma diagnosis, 11.5% reported symptoms described as wheeze, and nearly two-thirds (62.0%) had experienced two or more wheeze attacks within the past year. Additionally, 16.8% had missed at least two days of work, and 41.3% had sought medical help for wheezing.

Among children without an asthma diagnosis, 9.9% had experienced wheezing, and over half (51.7%) had suffered two or more wheeze attacks. Close to half (45.4%) had missed at least two school days, and almost two-thirds (65.5%) had sought medical help due to wheezing.

Table 29. Prevalence of asthma-like outcomes for CHIS 2003 adults and children with asthma-like symptoms[a]

| | Adults (≥18 years) | | Children (< 18 years) | |
|---|---|---|---|---|
| **Outcome** | n | % (Weighted) | n | % (Weighted) |
| Wheeze[b] | 4,129 | 11.5 | 951 | 9.9 |
| Missed ≥ 2 Work/ School Days[c] | 510 | 16.8 | 313 | 45.4 |
| Wheeze Attacks (2 or more)[c] | 2,313 | 62.0 | 324 | 51.7 |
| Sought Medical Help for Wheezing (1 or more) | 1,497 | 41.3 | 643 | 65.5 |

[a]For CHIS 2003, having asthma-like symptoms is defined as reporting wheezing or whistling in the previous 12 months without an asthma diagnosis; only respondents who lived in the same home or neighborhood for at least 9 months were included.
[b]Respondents without an asthma diagnosis who lived within 5 miles of an air monitoring station and had lived in the same home or neighborhood for at least 9 months were included. Respondents with wheezing symptoms were designated as having asthma-like symptoms.
[c]Data not collected for teen respondents.

Asthma-like symptoms were reported in nearly the same percentage of male adults as female, 50.5% vs. 49.5% respectively (Table 30 in Appendix). More than half of adult respondents with asthma-like symptoms were between 35-64 years old (56.4%), 28.3% were 18-34 years old, and 15.3% were 65 years old or older. The racial/ethnic distribution of the adult population with asthma-like symptoms was as follows: 58.5% white, 23.0% Latino, 9.5% Asian/other, 6.7% African American, and 2.3% American Indian/Alaska Native.

Among adults with asthma-like symptoms, 43.3% had a high school education or less. More than a third (37.8%) had completed college or a vocational school, and 8.6% had a graduate degree. The remaining 10.3% were 25 years old or younger; for these respondents, educational attainment was not assessed. More than one-third (39.0%) of adults with asthma-like symptoms lived below 200% of the FPL, and 24.7% lived between 200-399% of the FPL. Close to one-third (36.4%) of adult respondents with asthma-like symptoms had an income ≥400% of the FPL. Nearly a third (32.5%) of adults with asthma-like symptoms were unemployed.

Approximately one-fourth (24.5%) of adults with asthma-like symptoms were not insured or were only insured for part of the year. Similarly, 16.2% of these adults reported that they did not have a usual source of care, and 25.8% of adults reported a delay in needed care for any medical reason in the last 12 months. Nearly a quarter (24.5%) of adults reported visiting the doctor six or more times for any reason in the last year, and 41.8% of adults visited the doctor between two and five times for any reason in the past year. The remaining third (33.8%) had visited the doctor 0-1 time for any reason in the past year.

When self-reporting health status, 35.8% of adults with asthma-like symptoms reported poor or fair health as compared to 64.2% who reported good, very good, or excellent health. Heart disease was reported in 11.3%, and 41.3% of those reported congestive heart failure. Based on BMI, 62.5% of adults were classified as overweight/obese as opposed to normal or underweight.

More than half (59.3%) of adults were currently smoking or had been smokers in the past, though only 17.1% reported smokers living in the home. More than two-thirds (68.9%) of adult respondents reported walking for transportation or leisure. Dogs and/or cats lived in the home with 43.3% of adult respondents. Cockroaches were reported in the home for 20.3% of adults.

The majority (43.7%) of adult respondents with asthma-like symptoms lived in urban residences. Among those remaining, 26.2% lived in 2$^{nd}$ city locations, 16.3% in the suburbs, and 13.7% in towns or areas designated as rural. Most adults (80.4%) with asthma-like symptoms had lived at their residences for three years or more. Household crowding was reported for 23.5% of adults. The majority (63.1%) of adults with asthma-like symptoms lived in houses, and the remainder lived in apartments, duplexes, or mobile homes.

In children with asthma-like symptoms, slightly more boys reported experiencing wheezing (54.9%) as compared to girls (45.1%). Children ages 0-5 composed 41.2% of child respondents with asthma-like symptoms, 28.5% were ages 6-11, and the remaining 30.3% were ages 12-17. Children with asthma-like symptoms were 42.6% white, 34.3% Latino, 14.7% Asian/other, 7.4% African American, and 1.1% American Indian/Alaska Native.

The majority of children with asthma-like symptoms lived in households in which the adult who had responded on their behalf had either a college education (46.3%) or a high school education or less (42.8%). Eleven percent lived in households in which the adult respondent had completed graduate school. Forty-two percent of children with asthma-like symptoms lived below 200% of the FPL, 23.6 percent lived in households between 200-399% of the FPL, and the remaining 34.0% lived at or above 400% of the FPL.

Though the majority of children with asthma-like symptoms were insured, 10.1% of children were not insured or were only insured for part of the year, 10.6% of children reported

that they did not have a usual source of care, and 12.3% of children reported a delay in needed care for any medical reason in the last 12 months. Fifteen percent of children reported visiting the doctor six or more times for any reason in the last year, and 60.0% of children visited the doctor between two and five times for any reason in the past year. A quarter (25.0%) had visited the doctor one time or less for any reason in the last year.

Among children with asthma-like symptoms, 13.5% self-reported poor or fair health. Among respondents below the age of 18, BMI was assessed for teenage respondents only. For teens with asthma-like symptoms 25.5% were classified as overweight/obese.

The percentage of children with smokers living in the home was 7.1%. Dogs and/or cats within the home were reported for 38.2% of child respondents. A quarter (25.3%) of children with asthma-like symptoms reportedly also had cockroaches in the home.

Most (42.5%) child respondents with asthma-like symptoms lived in urban residences, compared to 25.0% of children who lived in 2$^{nd}$ city locations, 20.3% in the suburbs, and 12.2% in towns or rural areas. Sixty-two percent of children with asthma-like symptoms had lived at their residences for three years or more. Household crowding was reported for more than a third (37.3%) of children with asthma-like symptoms. Most children with asthma-like symptoms lived in houses (68.8%), while the remainder lived in apartments, duplexes or mobile homes.

**Disparities in Asthma-like Symptoms and Exposure Measures among Sub-Populations**

In this part of the study, we tested Hypothesis 1: Among those with asthma-like symptoms, vulnerable sub-populations in California (e.g., racial/ethnic minorities and low-income individuals) have higher exposures to air pollution. We presented differences in estimated criteria pollutant exposures across various sub-populations, characterized by rural and urban residency, age, gender, income level, and by racial and ethnic group. We also presented results regarding differences in distributions of asthma-like symptoms across these subgroups.

*Disparities in Annual Average Criteria Pollutant Exposure Measures*

We observed differences in mean annual average criteria pollutant exposures for CHIS respondents with asthma-like symptoms according to demographic characteristics (Table 31 in Appendix). In comparison to those living at or above 400% of the FPL, adult respondents with asthma-like symptoms living below 200% of the FPL had higher average annual exposure to three of the four criteria pollutants (Figure 8). The mean annual average $NO_2$ level was 23.3 ppb for those living below 200% of the FPL, compared to 21.1 ppb for those living at or above 400% of the FPL. The mean annual average $PM_{10}$ level was 32.4 $\mu g/m^3$ for those living below 200% of the FPL, compared to 29.5 $\mu g/m^3$ for those living at or above 400% of the FPL. A dose-response relationship was observed between poverty level and annual average $PM_{2.5}$ exposure. Individuals living at or above 400% of the FPL had a mean exposure of 15.3 $\mu g/m^3$, which increased to 16.4 $\mu g/m^3$ among those living between 200-399% of the FPL and 17.4 $\mu g/m^3$ for those living at less than 200% of the FPL.

When comparing exposures across ethnicities, we observed that Latino adult respondents had higher mean exposures to $NO_2$ (24.3 ppb vs. 20.5 ppb), $PM_{10}$ (33.7 $\mu g/m^3$ vs. 28.6 $\mu g/m^3$), and $PM_{2.5}$ (18.2 $\mu g/m^3$ vs. 15.7 $\mu g/m^3$) than white adult respondents (Figure 9). Mean annual average $NO_2$ exposures were also higher among African Americans than whites

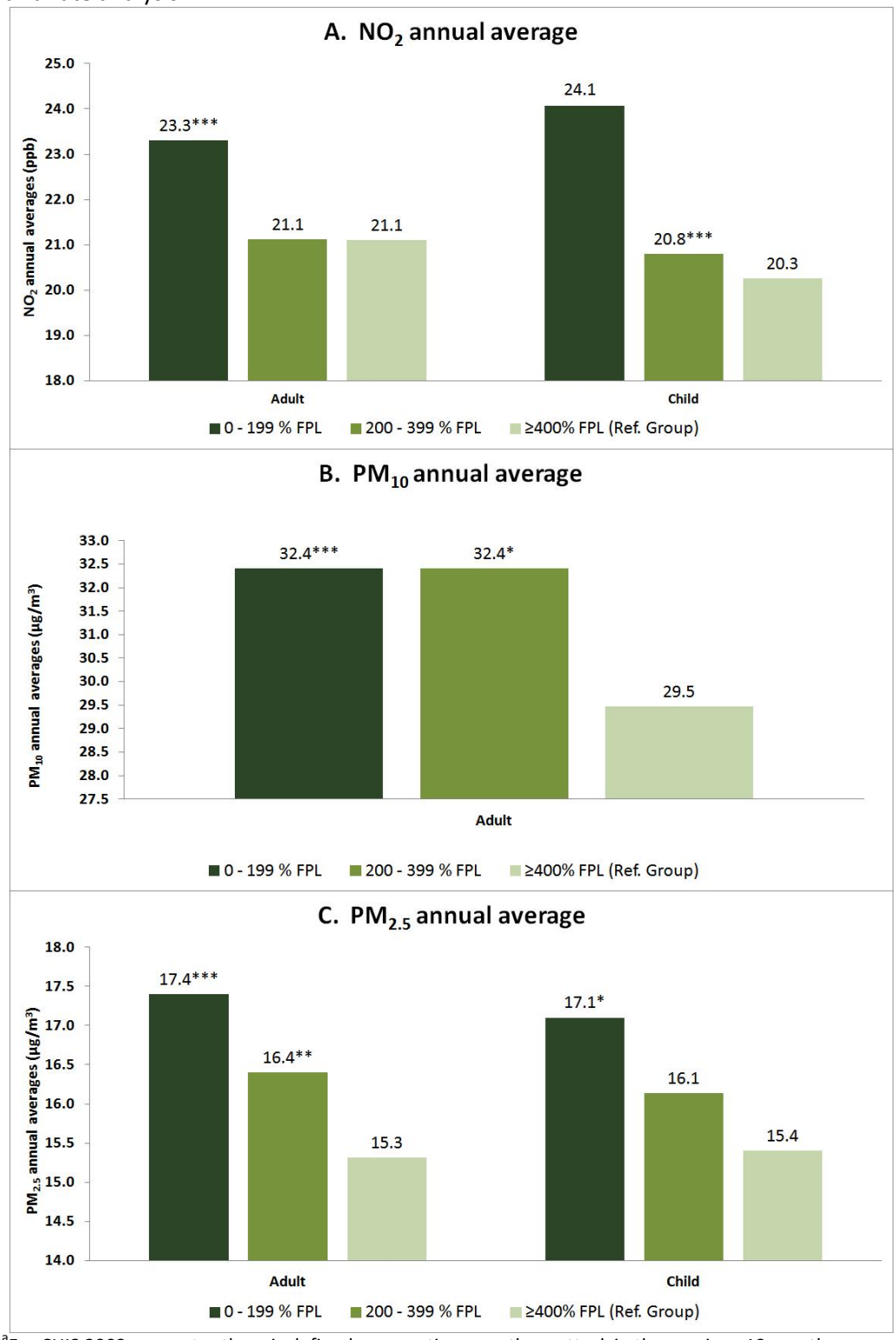
(23.3 ppb vs. 20.5 ppb); however white respondents had higher mean annual $O_3$ concentrations than African Americans (42.4 ppb vs. 38.9 ppb).

Mean annual average $NO_2$ levels were highest among urban dwellers (24.7 ppb), compared to 2nd city (18.4 ppb), suburban (19.8 ppb), and town/rural (11.0 ppb) dwellers. However, as residential distance from city centers increased, so did annual average $O_3$ exposure. Urban dwellers had the lowest annual average $O_3$ exposure at 38.8 ppb, versus 44.2 ppb for 2nd city, 44.7 ppb for suburban, and 48.1 ppb for town/rural dwellers. Urban dwellers also had lower annual average $PM_{10}$ levels than both 2nd city and suburban dwellers (29.2 $\mu g/m^3$ vs. 30.7 $\mu g/m^3$ and 33.0 $\mu g/m^3$, respectively). Adults living in urban residences had higher annual average $PM_{2.5}$ levels (16.6 $\mu g/m^3$) than those living in town/rural residences (10.1 $\mu g/m^3$), but lower levels than those living in suburban residences (17.7 $\mu g/m^3$). Adult females had higher average annual $PM_{10}$ concentrations at 30.7 $\mu g/m^3$, compared to 29.3 $\mu g/m^3$ among males.

Among children with asthma-like symptoms, individuals living between 200-399% of the FPL had slightly higher annual average $NO_2$ levels than children living at or above 400% of the FPL (20.8 ppb vs. 20.3 ppb). Compared to white children, Latino and African American children had higher mean annual average levels of $NO_2$ (19.3 ppb vs. 24.8 ppb and 24.0 ppb, respectively). Latino children also had higher mean annual average concentrations of $PM_{10}$ than white children (32.6 $\mu g/m^3$ vs. 28.6 $\mu g/m^3$).

Children living in urban residences had the highest annual average $NO_2$ levels at 25.6 ppb, compared to 18.0 ppb among 2nd city residents, 19.9 ppb suburban residents, and 18.8 ppb among town/rural residents. Conversely, urban children had the lowest mean annual $O_3$ exposure at 39.0 ppb, as opposed to 44.2 ppb among 2nd city children, 45.3 ppb among suburban children, and 47.7 ppb among town/rural children. Suburban children had higher annual average $PM_{10}$ concentrations than urban children (33.9 $\mu g/m^3$ vs. 29.0 $\mu g/m^3$). Urban children had higher $PM_{2.5}$ annual average concentrations at 16.8 $\mu g/m^3$, compared to 2nd city and town/rural children at 14.6 $\mu g/m^3$ and 9.2 $\mu g/m^3$, respectively.

Figure 8. Disparities in weighted mean annual pollutant concentrations by Federal Poverty Level (FPL)in CHIS 2003 children and adults with asthma-like symptoms using bivariate analysis[a,b]

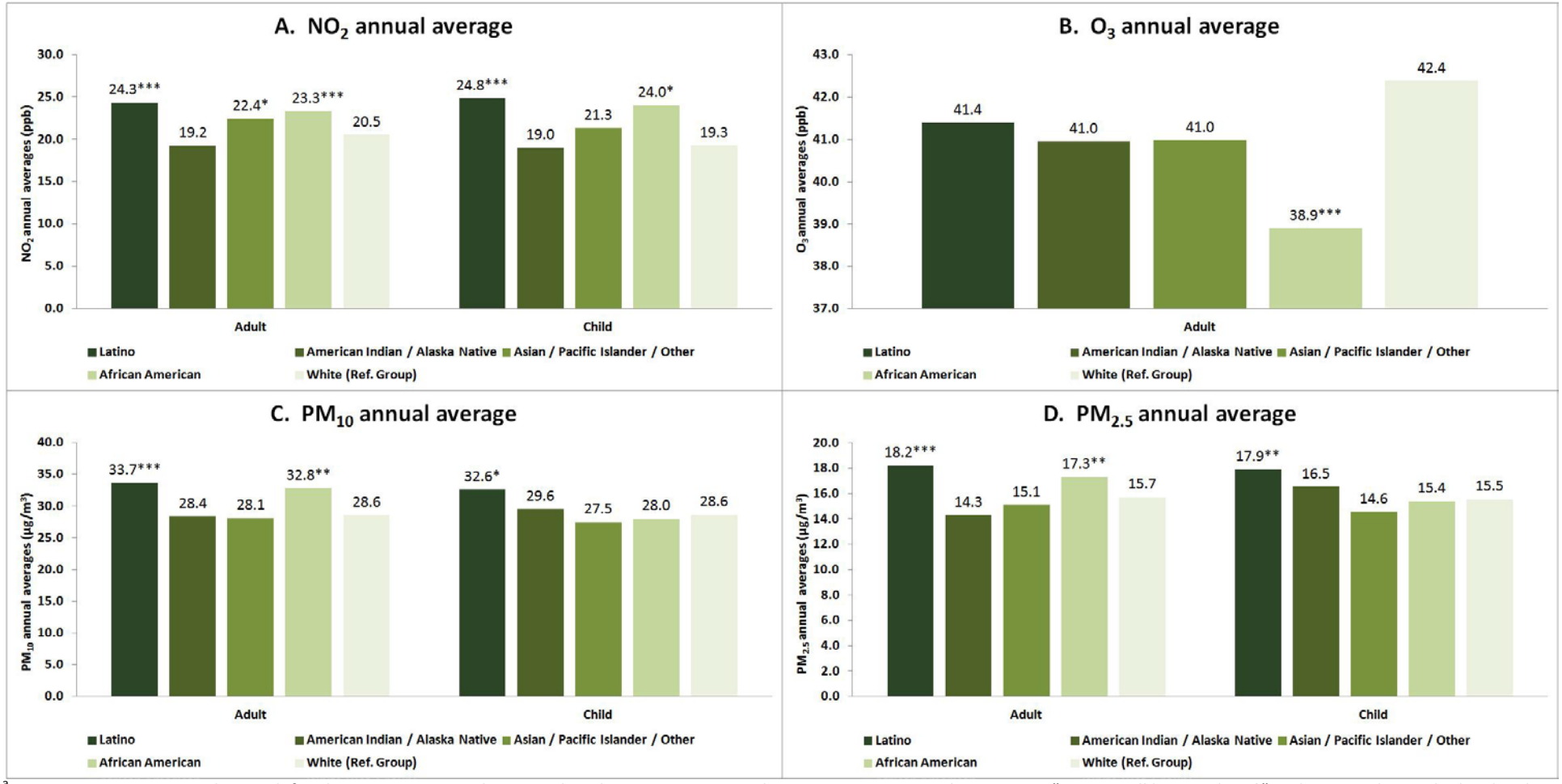



[a]For CHIS 2003, current asthma is defined as reporting an asthma attack in the previous 12 months or answering yes to the question, "Do you still have asthma?"; only respondents who lived within 5 miles of an air monitoring station and lived in the same home or neighborhood for at least 9 months were included.

[b]For Figure 8B, results for children with asthma-like symptoms showed no significant differences between groups and are not shown in the graph.

*$p < 0.05$, **$p < 0.01$, ***$p < 0.001$

Figure 9. Disparities in weighted mean annual pollutant concentrations by race/ethnicity in CHIS 2003 children and adults with asthma-like symptoms using bivariate analysis[a]




[a]For CHIS 2003, current asthma is defined as reporting an asthma attack in the previous 12 months or answering yes to the question, "Do you still have asthma?"; only respondents who lived within 5 miles of an air monitoring station and lived in the same home or neighborhood for at least 9 months were included.

[b]For Figure 9B, results for children with asthma-like symptoms showed no significant differences between groups and were not shown in the graph.

*p < 0.05, **p < 0.01, ***p < 0.001

*Disparities in asthma-like symptoms*

Asthma-like symptoms were not equally reported across all demographic characteristics. Among adults, 9.8% of those living at 400% or greater than the FPL, 11.7% of those living between 200-399% of the FPL, and 13.4% of those living below 200% of the FPL reported wheezing (Table 32 in Appendix).

When comparing asthma-like outcomes across races/ethnicities, Alaskan Natives/American Indians had the highest prevalence of wheeze (26.4%), more than twice the prevalence than that of white adults (13.1%). However, Latino and Asian/Pacific Islander respondents had lower prevalence of wheezing compared to white respondents, at 9.8% and 7.2%, respectively. Nearly a quarter (24.5%) of Latino respondents reported missing ≥2 days of work, compared to 14.3% of whites, and nearly half (46.1%) sought medical help at least once because of breathing problems, compared to 38.5% of whites. Additionally, just over half (51.7%) of Latino respondents reported having 2 or more wheeze attacks, although comparatively more white respondents reported 2 or more wheeze attacks (64.8%). Forty-nine percent of African Americans sought medical help for a breathing problem compared to 38.5% of whites.

A higher percentage of adult respondents living in 2$^{nd}$ city (12.2%) or town/rural (14.4%) residences reported experiencing wheezing as compared to their urban counterparts (11.0%). More female adult respondents reported missing at least 2 days of work (20.3%) and seeking medical help at least once due to breathing problems (50.3%) compared to males (13.6% and 32.8%).

More adults between the ages of 18-34 (18.3%) and 35-64 (17.0%) reported missing at least 2 days of work. Adult respondents in the oldest age bracket (≥65, 48.6%) had a higher prevalence of seeking medical help due to breathing problems than adult respondents in the lowest age bracket (18-34, 35.5%).

Among child respondents with asthma-like symptoms, Asians/Pacific Islanders/Others reported a higher prevalence of ≥2 wheeze attacks in the last year, at 75.5% compared to 48.1% among white children, but they reported a lower prevalence of missing at least 2 days of school due to wheezing (29.6% vs. 45.5%). The prevalence of wheezing was greater amongst boys than girls (10.9% vs. 8.9%). Fourteen percent of children ages 0-5 were reported to experience wheezing compared to 8.4% of children ages 12-17. As children got older, the prevalence of seeking medical help at least once in the last year because of breathing problems decreased. The prevalence of seeking medical attention due to breathing problems was 82.8% in children ages 0-5, 73.6% in children ages 6-11, and 34.2% in children ages 12-17.

**Associations between air pollution exposure metrics and asthma-like symptoms**

In this part of the study, we tested Hypothesis 2 that individuals with asthma-like symptoms and exposed to higher levels of air pollution are more likely to report: wheezing or whistling sound in the chest, attacks of wheezing or whistling. We present results of crude associations (Crude Odds Ratio) between individual air pollutants and asthma-like symptoms using tabular analyses and logistic regression modeling adjusting for age, sex, race/ethnicity and federal poverty level (Adjusted Odds Ratio).

*Associations between 12-month pollutant averages and asthma-like symptoms*

Among adults with asthma-like symptoms, a 10 ppb increase in annual average $O_3$ concentration was associated with a 9% (95% CI=1.01-1.18) increase in odds of reporting wheeze and an 11% increase in odds of reporting 2 or more wheezing attacks (95% CI=0.94-1.30, Table 33). A 10 µg/m$^3$ increase in $PM_{10}$ annual averages was associated with a 9% increase in wheeze (95% CI=1.01-1.18), a 10% increase in odds of reporting ≥2 wheeze attacks (95% CI=0.94-1.29), and a 9% (95% CI=0.94-1.27) increase in seeking medical help in the past year. A 5 µg/m$^3$ increase in $PM_{2.5}$ annual averages was associated with a 7% (95% CI=0.97-1.17) increase in odds of reporting wheeze. The odds of missing ≥2 days of work due to wheezing appeared to decrease with increasing $NO_2$ exposure (OR=0.73, 95% CI=0.56-0.96).

Among children with asthma-like symptoms, a 10 ppb increase in $O_3$ annual averages was associated with a 9% (95% CI=0.92-1.29) suggested increase in odds of wheeze and a 29% (95% CI=0.92-1.81) suggested increase in the likelihood of reporting 2 or more wheeze attacks. A 10 ppb increase in $NO_2$ was suggested to increase the odds of seeking medical help due to breathing problems by 33% (95% CI=0.90-1.98).

Table 33 (Highlights). Associations (OR (95% CI)) for 12-month pollutant averages and asthma-like outcomes in CHIS 2003 adults and children with asthma-like symptoms[a]

| | Wheeze[b] | | | | Missed ≥2 School/Work Days Due to Wheezing[c] | | | | ≥2 Wheeze Attacks[c] | | | | Sought Medical Help for Breathing Problem | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Pollutant | Cases | Non-Cases | OR[d] | 95% C.I. | Cases | Non-Cases | OR[d] | 95% C.I. | Cases | Non-Cases | OR[d] | 95% C.I. | Cases | Non-Cases | OR[d] | 95% C.I. |
| Adults | | | | | | | | | | | | | | | | |
| $O_3$ (per 10 ppb) | 2,044 | 14,824 | **1.09** | [1.01, 1.18] | 258 | 1,296 | 0.91 | [0.74, 1.12] | 1,121 | 657 | 1.11 | [0.94, 1.30] | 739 | 1,039 | 1.06 | [0.91, 1.24] |
| $PM_{10}$ (per 10 µg/m$^3$) | 1,614 | 11,229 | **1.09** | [1.01, 1.18] | 198 | 1,027 | 0.84 | [0.66, 1.06] | 912 | 488 | 1.10 | [0.94, 1.29] | 577 | 823 | 1.09 | [0.94, 1.27] |
| $PM_{2.5}$ (per 5 µg/m$^3$) | 1,253 | 9,331 | 1.07 | [0.97, 1.17] | 162 | 797 | 0.88 | [0.69, 1.12] | 691 | 396 | 1.00 | [0.83, 1.19] | 462 | 625 | 1.06 | [0.89, 1.26] |
| $NO_2$ (per 10 ppb) | 1,687 | 12,534 | 0.93 | [0.86, 1.02] | 222 | 1,061 | 0.73 | [0.56, 0.96] | 905 | 566 | 1.09 | [0.91, 1.30] | 622 | 849 | 0.93 | [0.78, 1.11] |
| Children | | | | | | | | | | | | | | | | |
| $O_3$ (per 10 ppb) | 441 | 4,109 | 1.09 | [0.92, 1.29] | 143 | 167 | 0.96 | [0.67, 1.36] | 157 | 153 | 1.29 | [0.92, 1.81] | 295 | 146 | 0.99 | [0.72, 1.36] |
| $NO_2$ (per 10 ppb) | 364 | 3,504 | 0.94 | [0.79, 1.11] | 113 | 146 | 1.25 | [0.82, 1.90] | 134 | 125 | 1.02 | [0.69, 1.50] | 243 | 121 | 1.33 | [0.90, 1.98] |

[a]For CHIS 2003, having asthma-like symptoms is defined as reporting wheezing or whistling in the previous 12 months without an asthma diagnosis; only respondents who lived within 5 miles of an air monitoring station and lived in the same home or neighborhood for at least 9 months were included.
[b]Respondents without an asthma diagnosis who lived within 5 miles of an air monitoring station and had lived in the same home or neighborhood for at least 9 months were included. Respondents with wheezing symptoms were designated as having asthma-like symptoms.
[c]Data not collected for teen respondents.
[d]Adjusted for age, race, poverty level, and sex.

*Associations for annual days exceeding air pollution standards and asthma-like symptoms*

Adults who experienced ≥51.2 days when maximum 8-hr $O_3$ concentrations exceeded the 8-hr state standard had an estimated 16% (95% CI=0.97-1.38) increase in odds of reporting wheeze in the previous year, compared to adults with <1.9 exceedance days (Table 34 in Appendix). Adults with 14.3-<51.2 days when maximum 8-hr $O_3$ concentrations exceeded the state standard had estimated odds increases of 42% (95% CI=0.99-2.04) for reporting ≥2 wheeze attacks in the previous year compared to adults with <1.9 exceedance days, but those with ≥51.2 exceedance days had only a 30% increase (95% CI=0.91-1.87). Adults who experienced ≥6.6 days with $PM_{10}$ concentrations exceeding the state standard had estimated odds increases for wheeze of 26% (95% CI=1.04-1.53).

Children with asthma-like symptoms experiencing 0.8-<8.7 or ≥36.7 days when maximum 1-hr $O_3$ concentrations exceeded the 1-hr state standard were nearly 3 (OR=2.79, 95% CI=1.10-7.10) or 4 (OR=3.98, 95% CI=1.56-10.11) times more likely, respectively, to have experienced ≥2 wheeze attacks in the last year than their peers with <0.8 exceedance days (Table 34 in Appendix). Children with maximum 8-hr $O_3$ concentrations exceeding the state standard 1.9-<14.3 or ≥51.2 $O_3$ days had a 34% (95% CI=0.92-1.96) or 32% (95% CI=0.91-1.91) suggested increase in odds of experiencing wheezing in comparison to those with <1.9 exceedance days. Children with 14.3-<51.2 days exceeding the 8-hr $O_3$ state standard were also more than twice as likely to miss 2 or more days of school due to wheezing (OR=2.35, 95% CI=1.01-5.47), but those with ≥51.2 days were not at increased odds. In general, the exposure-response patterns for the exceedance day measures and wheeze outcomes were not consistent in children.

*Associations for traffic density/distance to roadway and asthma-like symptoms*

We observed few consistent associations between residence-based measures of traffic and wheeze outcomes in adults (Table 35 in Appendix). Adults with traffic density exposures in the 2$^{nd}$ and 3$^{rd}$ exposure quartiles were 23% (95% CI=0.94-1.62) and 30% (95% CI=0.99-1.71) more likely to report seeking medical help in the last year due to breathing problems compared to those in the lowest exposure quartile, however those in the highest TD exposure quartile did not appear to have increased odds.

We also did not observe consistent associations between traffic density and wheeze outcomes in children (Table 35 in Appendix). Children with asthma-like symptoms and living within 300 m of an interstate highway had two and a half times greater odds of ≥2 wheeze attacks (OR=2.55, 95% CI=1.16-5.57), and those living within 50 m of a major road had 73% (95% CI=0.98-3.07) greater odds of missing ≥2 days of school due to wheezing compared to children living farther from these roadways.

**Pollutant and Asthma-like Symptom Relationships after Adjusting for Vulnerability (Confounding) Factors**

In this part of the study, we tested the hypothesis if air pollution exposures, low SES status, and certain vulnerability factors associated with low SES exert independent adverse effects on individuals with asthma-like symptoms (Hypothesis 3). We presented findings multiple logistic regression results regarding associations between air pollution exposures and outcomes after including and excluding suspected confounders, such as insurance status, cigarette smoking, and delays in care.

*Associations between 2 or more wheeze attacks and $O_3$, adjusting for vulnerability factors in adults*

Under the base model, adults with asthma-like symptoms had an 11% (95% CI=0.94-1.30) suggested increase in the odds of having ≥2 wheeze attacks in the year prior to the interview as $O_3$ annual average concentration increased by 10 ppb (Table 36). The suggested positive relationship between having ≥2 wheeze attacks and $O_3$ was upheld under Model 2 (OR=1.12, 95% CI=0.95-1.32) and Model 3 (OR=1.12, 95% CI=0.95-1.31).

No other positive associations were observed under Model 1; however, the model demonstrated decreased odds for Latino respondents versus white respondents (OR=0.56, 95%

CI=0.39-0.78). The similar effect of being Latino was also evidenced under Model 3. Positive associations were observed between having two or more wheeze attacks and other vulnerability factors in Models 2 and 3. Under Model 2, respondents ages 35-64 had 63% (95% CI=1.08-2.44) greater odds than respondents 65 or older, and those who had ever smoked were 43% (95% CI=1.08-1.88) more likely to report ≥2 wheeze attacks in the last year than those who had never smoked. Employed respondents were less likely than unemployed respondents to have had ≥2 wheeze attacks in the last year (OR=0.52, 95% CI=0.37-0.73). Under Model 3, having a delay in care (OR=0.58, 95% CI=0.43-0.80) and the absence of household smoking (OR=0.54, 95% CI=0.37-0.79) had negative associations with having ≥2 wheeze attacks in the past year.

*Associations between 2 or more wheeze attacks and $O_3$, adjusting for vulnerability factors in children*

Among children with asthma-like symptoms, the odds of having ≥2 wheeze attacks had a suggested increase of 29% (95% CI=0.90-1.84) as annual average $O_3$ concentration increased by 10 ppb under the base model (Table 37). Under Models 2 and 3, the suggested association between ≥2 wheeze attacks and $O_3$ was maintained (OR=1.28, 95% CI=0.89-1.83 and (OR=1.27, 95% CI=0.86-1.89, respectively).

Under Model 1, a negative relationship was observed between having ≥2 wheeze attacks and poverty level. Children living between 200-399% of the FPL had over twice greater odds (OR=2.42, 95% CI=1.06-5.55) and children living below 200% of the FPL had over three times greater odds (OR=3.44, 95% CI=1.60-7.38) of having ≥2 wheeze attacks as children living at or above 400% of the FPL. Under Models 2 and 3, the relationship with poverty level persisted.

Table 36 (Highlights). Associations between two or more wheeze attacks and $O_3$ adjusting for vulnerability characteristics among CHIS adults with asthma-like symptoms[a]

| | | $O^3$ (per 10 ppb) | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | Model 1[b] (1121, 657)[e] | | | Model 2[c] (1115, 652)[e] | | | Model 3[d] (1105, 647)[e] | | |
| **Vulnerability Characteristic** | | OR | 95% C.I. | | OR | 95% C.I. | | OR | 95% C.I. | |
| 12-Month Pollutant Average[f] | | 1.11 | 0.94 | 1.30 | 1.12 | 0.95 | 1.32 | 1.12 | 0.95 | 1.31 |
| Age (Ref. ≥65) | | | | | | | | | | |
| | 18 - 34 | 0.85 | 0.57 | 1.27 | 1.17 | 0.72 | 1.88 | 0.63 | 0.40 | 0.98 |
| | 35 - 64 | 1.22 | 0.86 | 1.72 | 1.63 | 1.08 | 2.44 | 0.98 | 0.68 | 1.41 |
| Race (Ref. White) | | | | | | | | | | |
| | African American | 0.94 | 0.57 | 1.54 | 0.97 | 0.59 | 1.62 | 1.05 | 0.62 | 1.77 |
| | American Indian / Alaska Native | 1.83 | 0.73 | 4.59 | 2.00 | 0.78 | 5.17 | 1.85 | 0.72 | 4.79 |
| | Asian / Pacific Islander / Other | 1.02 | 0.63 | 1.65 | 1.08 | 0.67 | 1.73 | 1.07 | 0.64 | 1.78 |
| | Latino | 0.56 | 0.39 | 0.78 | 0.57 | 0.39 | 0.82 | 0.64 | 0.44 | 0.94 |
| Poverty (Ref. ≥400% FPL)[g] | | | | | | | | | | |
| | 0 - 199% FPL | 1.05 | 0.76 | 1.45 | 0.82 | 0.58 | 1.17 | 0.98 | 0.69 | 1.40 |
| | 200 - 399% FPL | 0.92 | 0.67 | 1.27 | 0.84 | 0.60 | 1.16 | 0.86 | 0.62 | 1.19 |
| Sex | | | | | | | | | | |
| | Female vs. Male | 0.99 | 0.76 | 1.28 | 0.96 | 0.73 | 1.26 | 0.94 | 0.71 | 1.23 |
| Work Status | | | | | | | | | | |
| | Employed vs Unemployed | | | | 0.52 | 0.37 | 0.73 | | | |
| Delay in Care | | | | | | | | | | |
| | Yes vs No | | | | | | | 0.58 | 0.43 | 0.80 |
| Household Smoking | | | | | | | | | | |
| | No vs Yes | | | | | | | 0.54 | 0.37 | 0.79 |

[a]For CHIS 2003, asthma-like symptoms is defined as reporting wheezing or whistling in the previous 12 months without an asthma diagnosis; only respondents who lived within 5 miles of an air monitoring station and lived in the same home or neighborhood for at least 9 months were included.
[b]Model 1 controlled for age, race, poverty level, and sex.
[c]Model 2 used the following vulnerability factors: insurance status, obesity, heart disease, smoking status, and work status, in addition to age, race, poverty level, and sex.
[d]Model 3 used the following vulnerability factors: urban/rural, usual source of care, delay in care, household smoking, dog or cat in home, cockroaches in home, household type, household crowding, diabetes, walking, in addition to age, race, poverty level and sex.
[e](Cases, Controls)
[f]Refers to the criteria pollutant noted in the column heading.
[g]FPL = Federal Poverty Level

Table 37 (Highlights). Associations between two or more wheeze attacks and vulnerability characteristics by $O_3$ among CHIS children with asthma-like symptoms[a]

| | | $O_3$ (per 10 ppb) (157, 153)[b] | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | Model 1[c] | | | Model 2[d] | | | Model 3[e] | | |
| **Vulnerability Characteristic** | | OR | 95% C.I. | | OR | 95% C.I. | | OR | 95% C.I. | |
| 12-Month Pollutant Average[f] | | 1.29 | 0.90 | 1.84 | 1.28 | 0.89 | 1.83 | 1.27 | 0.86 | 1.89 |
| Age (Ref. 6-11 years) | | | | | | | | | | |
| | < 6 years old | 0.66 | 0.35 | 1.25 | 0.69 | 0.37 | 1.29 | 0.65 | 0.34 | 1.25 |
| Race (Ref. White) | | | | | | | | | | |
| | African American | 0.32 | 0.06 | 1.69 | 0.26 | 0.04 | 1.60 | 0.38 | 0.08 | 1.81 |
| | American Indian / Alaska Native | 0.21 | 0.03 | 1.54 | 0.18 | 0.03 | 1.16 | 0.16 | 0.02 | 1.53 |
| | Asian / Pacific Islander / Other | 2.07 | 0.77 | 5.57 | 1.84 | 0.69 | 4.91 | 2.55 | 0.88 | 7.41 |
| | Latino | 0.53 | 0.25 | 1.15 | 0.46 | 0.20 | 1.07 | 0.75 | 0.34 | 1.65 |
| Poverty (Ref. ≥400% FPL)[g] | | | | | | | | | | |
| | 0 - 199% FPL | 3.44 | 1.60 | 7.38 | 3.62 | 1.59 | 8.23 | 4.88 | 2.05 | 11.63 |
| | 200 - 399% FPL | 2.42 | 1.06 | 5.55 | 2.40 | 1.06 | 5.47 | 2.61 | 1.10 | 6.23 |
| Sex | | | | | | | | | | |
| | Female vs. Male | 0.93 | 0.52 | 1.69 | 0.88 | 0.49 | 1.59 | 0.93 | 0.49 | 1.75 |
| Household Crowding | | | | | | | | | | |
| | No vs Yes | | | | | | | 2.32 | 1.02 | 5.27 |

[a]For CHIS 2003, asthma-like symptoms is defined as reporting wheezing or whistling in the previous 12 months without an asthma diagnosis; only respondents who lived within 5 miles of an air monitoring station and lived in the same home or neighborhood for at least 9 months were included.
[b](Cases, Controls)
[c]Model 1 controlled for age, race, poverty level, and sex.
[d]Model 2 used the following vulnerability factors: household smoking, dog or cat in home, cockroaches in home, and insurance status, in addition to age, race, poverty level, and sex.
[e]Model 3 used the following vulnerability factors: urban/rural, delay in care, housing type, and household crowding, in addition to age, race, poverty level, and sex.
[f]Refers to the criteria pollutant noted in the column heading.
[g]FPL = Federal Poverty Level

## Sensitivity Analyses

We performed analyses stratifying on: (1) length of residence in the same neighborhood, (2) employment status in adults, (3) residential distance to nearest monitoring station (3-, 5- and 10- miles), and (4) asthma medication use. Although associations between annual average $O_3$ exposure and odds of reporting asthma attacks in the previous year were similar for adults residing in the same neighborhood for <3 versus ≥3 years, associations between annual average $O_3$ and $PM_{2.5}$ exposure and odds of reporting daily asthma medication use in adults appeared isolated to individuals residing in the same neighborhood for at least 3 years (Table 38). In children, associations between annual average $NO_2$ and odds of daily asthma medication use also appeared isolated to longer-term residents (≥3 years). However, the sample sizes for respondents residing <3 years in the same neighborhood were smaller and the 95% confidence intervals for all point estimates overlapped widely.

Table 38. Association (OR (95% C.I.)) between asthma outcomes and 12-month pollutant exposures for CHIS 2003 adults and children with current asthma stratified by length of residence[a]

| | | | Lived in Neighborhood < 3 Years | | | | Lived in Neighborhood ≥ 3 years | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | Health Outcome | Pollutant | Cases | Non-Cases | OR[b] | 95% C.I. | Cases | Non-Cases | OR[b] | 95% C.I. |
| Adults | | | | | | | | | | |
| | Asthma Attack[c] | | | | | | | | | |
| | | $O_3$ | 179 | 259 | 1.22 | [0.90, 1.61] | 786 | 1,323 | 1.20 | [1.04, 1.39] |
| | Daily Asthma Medication | | | | | | | | | |
| | | $O_3$ | 120 | 171 | 0.99 | [0.70, 1.40] | 695 | 631 | 1.28 | [1.07, 1.54] |
| | | $PM_{2.5}$ | 77 | 97 | 0.99 | [0.64, 1.52] | 417 | 399 | 1.36 | [1.10, 1.67] |
| Children | | | | | | | | | | |
| | Daily Asthma Medication | | | | | | | | | |
| | | $NO_2$ | 35 | 72 | 1.21 | [0.62, 2.35] | 136 | 226 | 1.50 | [1.05, 2.15] |

[a]For CHIS 2003 current asthma is defined as reporting an asthma attack in the previous 12 months or answering yes to the question, "Do you still have asthma?"; only respondents who lived within 5 miles of an air monitoring station and lived in the same neighborhood for at least 9 months were included.
[b]Adjusted for age, race, poverty level, and sex.
[c]Respondents ever diagnosed with asthma who lived within 5 miles of an air monitoring station and in the same home or neighborhood for at least 9 months were included.

When stratifying on employment status, the association between annual average $PM_{10}$ and odds of ED visits in adults with  appeared isolated to employed individuals (Table 39). This was the opposite of our prior expectation, assuming residence-based exposure measures are less misclassified for unemployed individuals who spend more time at home and that this misclassification is non-differential. One possible explanation for this finding is that employed individuals have other risk factors (e.g., co-morbidities, obesity) which make them more susceptible to the effects of $PM_{10}$. Or associations in employed individuals may reflect, in part, time spent commuting, since in-vehicle air pollution exposures have been shown to be higher than ambient exposures (Fruin et al., 2004, Westerdahl et al., 2005, Zhu et al., 2007).

Table 39. Association (OR (95% C.I.)) between asthma outcomes and $PM_{10}$ pollutant for CHIS 2003 adults with current asthma stratified by employment status[a]

| | | Employed | | | | Unemployed | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | Cases | Non-Cases | OR[b] | 95% C.I. | Cases | Non-Cases | OR[b] | 95% C.I. |
| $PM_{10}$ | | | | | | | | | |
| | ED Visits | 107 | 641 | 1.44 | [1.10, 1.89] | 105 | 444 | 1.01 | [0.78, 1.33] |

[a]For CHIS 2003 current asthma is defined as reporting an asthma attack in the previous 12 months or answering yes to the question, "Do you still have asthma?"; only respondents who lived within 5 miles of an air monitoring station and lived in the same neighborhood for at least 9 months were included.
[b]Adjusted for age, race, poverty level, and sex.

Additionally, since we had address information for most respondents, we were able to examine the influence of residential distance from the monitoring station on our study results. Specifically, we ran logistic regression models for the outcome of ED visits for respondents living within 3-, 5-, and 10-miles of an air monitoring station for each pollutant (Table 40). For

adults, the point estimates for annual average $PM_{10}$ and $PM_{2.5}$ were higher for the 3- and 5-mile linkage distances, while odds ratio estimates for annual average $O_3$ followed the opposite pattern, but 95% CIs overlapped widely in all cases. The most marked difference in odds ratio point estimates across linkage distances was observed for annual average $NO_2$, with the highest association observed in adults living within 3 miles of a monitoring station. In children, crude odds ratio estimates for all pollutants were greater for individuals residing within 3 miles of a monitoring station, however, these patterns did not persist for $PM_{2.5}$ and $NO_2$ in adjusted models and 95% CIs for all associations were wide due to the smaller sample sizes available.

Table 40. Association (OR (95% C.I.)) between ED visits and 12-month pollutant exposures for 3-, 5-, and 10-mile linkage distances to monitors for CHIS 2003 adults and children with current asthma[a]

| | | Adults (≥18 years old) | | | | | | Children ( <18 years old) | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Exposure | Distance to Station (miles) | Cases (n) | Non-cases (n) | Crude OR | 95% C.I. | Adj. OR[b] | 95% C.I. | Cases (n) | Non-cases (n) | Crude OR | 95% C.I. | Adj. OR[b] | 95% C.I. |
| $O_3$ | | | | | | | | | | | | | |
| | 3 | 148 | 772 | 1.03 | [0.81, 1.32] | 1.07 | [0.82, 1.38] | 70 | 226 | 1.04 | [0.68, 1.58] | 1.12 | [0.71, 1.79] |
| | 5 | 245 | 1,372 | 1.14 | [0.93, 1.41] | 1.19 | [0.96, 1.47] | 120 | 417 | 0.86 | [0.61, 1.20] | 0.80 | [0.55, 1.17] |
| | 10 | 392 | 2,193 | 1.16 | [0.98, 1.38] | 1.13 | [0.95, 1.36] | 197 | 711 | 0.83 | [0.63, 1.09] | 0.80 | [0.59, 1.09] |
| $PM_{10}$ | | | | | | | | | | | | | |
| | 3 | 133 | 668 | 1.30 | [1.06, 1.60] | 1.16 | [0.94, 1.44] | 58 | 198 | 1.36 | [1.00, 1.84] | 1.27 | [0.89, 1.82] |
| | 5 | 212 | 1,092 | 1.29 | [1.09, 1.52] | 1.20 | [1.00, 1.43] | 97 | 339 | 1.13 | [0.89, 1.43] | 1.00 | [0.74, 1.34] |
| | 10 | 367 | 2,018 | 1.17 | [1.03, 1.33] | 1.08 | [0.94, 1.24] | 180 | 662 | 1.06 | [0.86, 1.29] | 1.00 | [0.78, 1.27] |
| $PM_{2.5}$ | | | | | | | | | | | | | |
| | 3 | 94 | 425 | 1.45 | [1.10, 1.91] | 1.23 | [0.91 ,1.66] | 42 | 131 | 1.45 | [0.92, 2.30] | 1.18 | [0.69 ,2.00] |
| | 5 | 160 | 830 | 1.36 | [1.09, 1.71] | 1.22 | [0.96, 1.56] | 73 | 262 | 1.19 | [0.84, 1.68] | 1.01 | [0.69, 1.49] |
| | 10 | 286 | 1,547 | 1.28 | [1.08, 1.52] | 1.15 | [0.96, 1.37] | 148 | 523 | 1.26 | [0.98, 1.61] | 1.15 | [0.86, 1.54] |
| $NO_2$ | | | | | | | | | | | | | |
| | 3 | 122 | 616 | 1.74 | [1.30, 2.32] | 1.52 | [1.12, 2.06] | 67 | 187 | 1.45 | [0.93, 2.25] | 1.28 | [0.80, 2.06] |
| | 5 | 200 | 1,115 | 1.33 | [1.06, 1.69] | 1.18 | [0.92, 1.50] | 113 | 356 | 1.23 | [0.86, 1.75] | 1.17 | [0.81, 1.69] |
| | 10 | 331 | 1,893 | 1.35 | [1.12, 1.64] | 1.24 | [1.01, 1.51] | 185 | 621 | 1.27 | [0.95, 1.71] | 1.20 | [0.90, 1.61] |

[a]For CHIS 2003 current asthma is defined as reporting an asthma attack in the previous 12 months or answering yes to the question, "Do you still have asthma?"; only respondents who lived within 5 miles of an air monitoring station and lived in the same neighborhood for at least 9 months were included.
[b]Adjusted for age, race, poverty level, and sex.

Medication use can be treated as an adverse outcome because it is a measure of asthma severity. However, use of daily medication can also be expected to reduce the frequency of other adverse outcomes such as ED visits and absences from work. To investigate whether air pollution effects on ED visits or absences from work are different among those taking asthma medication and those not taking medication, we conducted stratified analyses by medication use (Table 41 and 42). As anticipated, the results suggested that associations between annual average $O_3$, $PM_{10}$ and $PM_{2.5}$ exposure and odds of ED visits and having two or more work absences appeared to be greater among those not taking daily medication except for $PM_{2.5}$ exposure and odds of ED visits. However, all confidence intervals for the stratified odds ratios crossed the null. Therefore, the results suggested that asthma medication use could modify adverse effects of air pollutant exposures, but no firm conclusions could be drawn due to a lack of statistical power to formally test the differences.

Table 41. Associations (OR (95% CI) between 12-month pollutant averages and ED visits stratified by asthma medication use in CHIS 2003 adults with current asthma[a]

| | Daily Asthma Medication | | | | | | | | | | | | Overall | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Yes | | | | | | No | | | | | | | | | | | |
| Pollutant | Cases | Non-Cases | Crude OR | 95% C.I. | Adj. OR[b] | 95% C.I. | Cases | Non-Cases | Crude OR | 95% C.I. | Adj. OR[b] | 95% C.I. | Cases | Non-Cases | Crude OR | 95% C.I. | Adj. OR[b] | 95% C.I. |
| $O_3$ (per 10 ppb) | 176 | 639 | 1.03 | [0.81, 1.33] | 1.08 | [0.83, 1.40] | 69 | 733 | 1.22 | [0.84, 1.77] | 1.23 | [0.84, 1.80] | 245 | 1372 | 1.14 | [0.93, 1.41] | 1.19 | [0.96, 1.47] |
| $PM_{10}$ (per 10 µg/m$^3$) | 156 | 503 | 1.27 | [1.03, 1.58] | 1.14 | [0.90, 1.44] | 56 | 589 | 1.26 | [0.95, 1.67] | 1.23 | [0.90, 1.68] | 212 | 1092 | 1.29 | [1.09, 1.52] | **1.20** | [1.00, 1.43] |
| $PM_{2.5}$ (per 5 µg/m$^3$) | 116 | 378 | 1.35 | [1.02, 1.79] | 1.20 | [0.88, 1.62] | 44 | 452 | 1.21 | [0.81, 1.81] | 1.09 | [0.71, 1.65] | 160 | 830 | 1.36 | [1.09, 1.71] | 1.22 | [0.96, 1.56] |

[a] For CHIS 2003, current asthma is defined as reporting an asthma attack in the previous 12 months or answering yes to the question, "Do you still have asthma?"; only respondents who lived within 5 miles of an air monitoring station and lived in the same neighborhood for at least 9 months were included.
[b]Adjusted for age, race, poverty, and sex

Table 42. Associations (OR (95% CI) between 12-month pollutant averages and missing two or more days of work stratified by asthma medication use in CHIS 2003 adults with current asthma[a]

| | Daily Asthma Medication | | | | | | | | | | | | Overall | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Yes | | | | | | No | | | | | | | | | | | |
| Pollutant | Cases | Non-Cases | Crude OR | 95% C.I. | Adj. OR[b] | 95% C.I. | Cases | Non-Cases | Crude OR | 95% C.I. | Adj. OR[b] | 95% C.I. | Cases | Non-Cases | Crude OR | 95% C.I. | Adj. OR[b] | 95% C.I. |
| $O_3$ (per 10 ppb) | 106 | 551 | 1.06 | [0.79, 1.41] | 1.00 | [0.73, 1.36] | 70 | 657 | 1.07 | [0.75, 1.54] | 1.20 | [0.84, 1.72] | 176 | 1208 | 1.09 | [0.88, 1.37] | 1.15 | [0.91, 1.46] |
| $PM_{10}$ (per 10 µg/m$^3$) | 88 | 446 | 1.26 | [0.96, 1.65] | 1.15 | [0.85, 1.56] | 54 | 531 | 1.46 | [0.98, 2.18] | 1.47 | [0.93, 2.32] | 142 | 977 | 1.36 | [1.08, 1.72] | **1.28** | [1.00, 1.65] |
| $PM_{2.5}$ (per 5 µg/m$^3$) | 68 | 336 | 1.19 | [0.84, 1.69] | 1.09 | [0.76, 1.56] | 50 | 407 | 1.47 | [1.00, 2.17] | 1.26 | [0.83, 1.92] | 118 | 743 | 1.34 | [1.03, 1.74] | 1.23 | [0.94, 1.60] |

[a] For CHIS 2003, current asthma is defined as reporting an asthma attack in the previous 12 months or answering yes to the question, "Do you still have asthma?"; only respondents who lived within 5 miles of an air monitoring station and lived in the same neighborhood for at least 9 months were included.
[b]Adjusted for age, race, poverty, and sex

# IV. DISCUSSION

In this study, we linked ambient air monitoring and traffic data to California Health Interview Survey data. This study tested the hypotheses: 1) Among those with asthma or asthma-like symptoms, vulnerable sub-populations in California (e.g., racial/ethnic minorities and low-income individuals) have higher exposures to air pollution; 2) Individuals with asthma exposed to higher levels of air pollution are more likely to report adverse asthma outcomes, such as: asthma attacks or episodes, asthma emergency department (ED) visits, use of daily medication to control asthma, school or work absences, and daily/weekly asthma symptoms. Individuals with asthma-like symptoms (defined here as individuals without physician-diagnosed asthma who reported wheezing) and exposed to higher levels of air pollution are more likely to report: wheezing or whistling sound in the chest, attacks of wheezing or whistling, seeking medical care for such symptoms, and work/school days missed due to such symptoms; 3) Air pollution exposures, low socioeconomic status (SES), and certain "vulnerability factors" associated with low SES, exert independent adverse effects on individuals with asthma or asthma-like symptoms. The vulnerability factors examined were: **co-morbidity** (such as diabetes or heart disease); **access to care** (health insurance status, usual source of care); **disease management/asthma severity** (taking daily medication to control asthma, receiving an asthma management plan); **health behaviors** (being overweight/obese, smoking, walking outdoor, engaging in physical activity); **exposure to indoor triggers** (environmental tobacco smoke and indoor allergens, cockroaches, dogs and cats); and **housing conditions** (single family dwelling or apartment, crowding); and 4) Higher pollutant exposures interact with these vulnerability factors resulting in greater air pollution impacts on asthma in vulnerable sub-populations (racial/ethnic minorities, low-income individuals). The findings supported these hypotheses. In the following sections we discuss the strengths and limitations of our study and compare our results to relevant findings in the literature.

## Disparities in Exposure to Air Pollutants among Californians with Asthma

In general, we observed that respondents living below 200% of the federal poverty level (FPL) and minority respondents had higher estimated pollutant exposures and lived nearer to highways or major roadways and in areas of higher traffic density. Adults and children with current asthma living below 200% of the federal poverty level (FPL) had higher annual average exposures to $NO_2$, $PM_{10}$, and $PM_{2.5}$ than those living at or above 400% of the FPL. Racial/ethnic minorities, such as Latinos, African Americans and Asian/Pacific Islanders/others had higher $PM_{2.5}$, $PM_{10}$, and $NO_2$ exposures than whites; however, white adults and children had higher exposures to ozone than respondents in either of these three minority groups. Mean traffic density measures were higher for both Latino and African American children than for white children.

Our findings of disparities across pollutant and traffic exposures are similar to those reported by previous investigators. Gunier et al. (2003) reported that Californians in the lowest quartile of median family income were more likely to live in high-traffic areas than those in the highest income quartile. Children of color have also been previously shown to be more likely to live in high traffic areas than white children (Gunier, Hertz et al. 2003; Houston, Wu et al. 2004). Green et al. (2004) reported elementary schools in California with high proportions of economically-disadvantaged and non-white children were more likely to be located within

proximity to roadways with high traffic counts (Green, Smorodinsky et al. 2004). Other studies in Southern California using air toxics emission inventory data found that transportation sources were the most important sources for lifetime cancer risk, especially for racial minorities (Morello-Frosch, Pastor et al. 2001; Pastor, Morello-Frosch et al. 2005). Limited numbers of studies have used air monitoring data to assess disparities in exposures. A study in Sweden reported inverse relationships between $NO_2$ exposures at residences and schools and children's economic statuses (Chaix, Gustafsson et al. 2006).

Disparities in air pollution exposures that we and other investigators have observed stem from a long history of social and economic injustice, including racial segregation, housing discrimination, and land-use inequities (Houston, Wu et al. 2004). For instance, Jerrett et al. (2001) reported lower housing values in Hamilton, Canada were strongly associated with higher concentrations of particulate pollution(Jerrett, Burnett et al. 2001). Our observations related to $O_3$ were also consistent with previous studies. For example, $O_3$ levels were substantially higher in southern Mexico City, a higher socioeconomic status area, compared to the lower SES northern city (Checkley, West et al. 2011). Similarly, respondents in our study living farther from urban areas, such as in suburbs, had higher levels of annual exposure to $O_3$. This could be explained by the fact that it takes time for $O_3$ to form through photochemical reactions. $O_3$ is usually higher in areas downwind of sources of $NO_X$ and VOCs; for example, the highest levels in the LA Basin are in eastern areas/suburbs. $O_3$ can also be lower in areas closer to heavy traffic due to scavenging by NO from traffic (Godish 1991).

## Pollutant Effects on Asthma Outcomes

Among adults, we observed associations between annual average pollutant exposures and several asthma outcomes: frequent asthma symptoms (daily/weekly symptoms), asthma attacks or episodes, use of daily medication to control asthma, school or work absences due to asthma, and asthma ED visits, in the 12 months prior to the CHIS 2003 interview. In line with previous studies, asthma attacks were associated with higher exposures to $O_3$ among adults in our study (Slaughter, Lumley et al. 2003). Also for adults, ED visits, using daily asthma medication, and missing 2 or more days of work due to asthma were associated with higher exposures to three of the four pollutants in the study ($O_3$, $PM_{10}$, $PM_{2.5}$). These findings are consistent with previous literature showing a relationship between ED visits and higher levels of $O_3$ (Romieu, Meneses et al. 1995; Tolbert, Mulholland et al. 2000; Friedman, Powell et al. 2001), $PM_{10}$ (Meng, Rull et al. 2010) and $PM_{2.5}$ (Norris, YoungPong et al. 1999) and between daily/weekly asthma symptoms and higher exposures to $PM_{2.5}$ in adults (Ward and Ayres 2004; Meng, Rull et al. 2010).

Since much of the literature on the effects of air pollution on asthma has focused on children, our study will help to fill gaps in the literature for adult asthma. Our findings, which show an association between air pollution and missed work days due to asthma, are an important contribution to the literature since to the best of our knowledge there are few previous studies showing this association. As fewer studies have been conducted on asthma medication use and air pollution, our results will support and add to existing studies supporting an association (Thurston, Lippmann et al. 1997; Gent, Triche et al. 2003; Slaughter, Lumley et al. 2003; Gent, Koutrakis et al. 2009).

Associations between $NO_2$ and asthma outcomes were more clearly seen among children. Among children in our study, daily asthma medication and missing 2 or more days of school/day care were associated with higher exposures to $NO_2$. This is consistent with prior studies linking $NO_2$ to asthma medication use in children (Gauderman, Avol et al. 2005; Schildcrout, Sheppard et al. 2006) and missed school days due to asthma (O'Connor, Neas et al. 2008).

With regard to days of exceedance of state or federal standards, we found associations with asthma outcomes in adults and children, primarily for respondents in the highest quartiles of exceedance days. Among adults, exceedances of state $O_3$ standards were associated with increased odds for asthma attacks, visiting the ED, and using a daily asthma medication. We also found exceedances of the state $PM_{10}$ standard were associated with increased odds of asthma ED visits in adults. Exceedances of the federal $PM_{2.5}$ standard were associated with increased odds of ED visits and daily/weekly asthma symptoms. Among children, $O_3$ exceedances (state 1-hr standard) were associated with increased odds of missing 2 or more days of school. In previous studies, the number of exceedance days of state or federal standards is often used to examine short-term exposure effects; however our findings add to the existing knowledge that many days of exceedance of the standards may also have chronic effects on individuals with asthma, which confirms the importance of maintaining federal and state standards to protect the respiratory health of California's population.

Based on these latest analyses using CHIS 2003 data, we only observed few consistent positive associations between traffic density and residential proximity to traffic and asthma outcomes among respondents with current asthma or respondents with asthma-like symptoms. These findings are in contrast to our earlier studies in which we estimated strong positive associations between residential traffic density and asthma symptoms for CHIS 2001 respondents living in Los Angeles and San Diego Counties. For example, in our previous studies we estimated approximately two-fold greater odds of daily or weekly asthma symptoms (OR=2.11; 95% CI=1.38–3.23) in adult respondents with a lifetime asthma diagnosis, comparing individuals in the highest to the lowest quintile of traffic density (Meng, Wilhelm et al. 2007). Similarly, children with a lifetime diagnosis of asthma and in the highest quintile of traffic density were estimated to have 3 times higher odds of ED visits or hospitalizations (OR=3.27, 95% CI=1.08–9.89) than children in the lowest quintile of traffic density (Meng, Wilhelm et al. 2007; Wilhelm, Meng et al. 2008). The discrepancies in results may be due to several factors. First, our CHIS 2001 analyses focused on Los Angeles and San Diego Counties, as those were the two counties where residential cross-street information was collected from respondents. The mean and maximum traffic density values for subjects residing in those two highly urbanized areas were slightly higher than the traffic density values estimated here for respondents throughout the entire state of California. When we isolated our current analyses to LA and SD counties, we still did not observe associations with daily or weekly asthma symptoms in adults. However, we observed a 15% increase in odds of asthma ED visits in adults per interquartile increase in traffic density (OR=1.15, 95% CI=1.03-1.28) and an approximately 2-fold increase in odds of ED visits for those in the highest compared to lowest traffic density exposure quartile (OR=2.34, 95% CI=1.15-4.78). We did not observe associations between traffic density and odds of ED visits in children, even after restricting analyses to LA and SD Counties, but the sample size available was smaller than in our previous CHIS 2001 study, since previously we included all

respondents with lifetime asthma and here we include only current asthma. Also, the CHIS 2003 sample is smaller than CHIS 2001 in general (42,000 versus 55,000 households, respectively).

Second, previously we used Highway Performance Monitoring System (HPMS) traffic count data obtained directly from Caltrans to estimate traffic density and did not impute values to un-counted roadway segments. For this study, the Caltrans roadmap with linked HPMS data was no longer available and therefore we used TeleAtlas Dynamap traffic data and imputed values to roadway segments with missing traffic counts. By assigning the median traffic count for a given roadway category to roadway segments within the same roadway category without traffic counts across the entire state, we may have introduced additional error into our traffic density measures. Further investigation is needed to identify a better method for assessing traffic exposures for all Californians.

## Pollutant and Asthma Outcome Relationships after Adjusting for Vulnerability Factors

In addition to pollutant exposures, several other characteristics were related to increased odds of asthma outcomes (ED visits, taking a daily asthma medication, or missing 2 or more days of work). Odds of having these asthma outcomes were higher among African Americans and Latinos compared to whites. ED visits were more likely among respondents living below 200% of the FPL compared to those living at or above 400% of the FPL, asthma medication use was more likely among respondents ages 65 or older, and missing 2 or more days of work was more likely among women than men.

Other vulnerability factors, such as having heart disease and having adult onset of asthma, increased the odds of ED visits and asthma medication use among adults with current asthma. Also, having health insurance, having a usual source of care, or being a previous or current smoker increased the possibility of taking daily medications for asthma. Other factors, such as no secondhand smoking at home decreased the possibility of missing at least 2 days of work due to asthma.

Among children, those living below 200% and between 200-399% of the FPL consistently had higher odds of using daily asthma medication than those living at or above 400% of the FPL. Living in a mobile home increased the odds of using a daily asthma medication.

These findings contribute to the existing literature that, in addition to pollutant exposures, vulnerability factors, such as access to care (Meng, Babey et al. 2006) and other behavioral risk factors such as smoking or secondhand smoking (Silverman, Boudreaux et al. 2003), are associated with severe asthma. Our findings were consistent with other studies that suggest that comorbidities, especially cardiovascular disease, may also contribute to increases in negative health outcomes related to asthma (Gouveia and Fletcher 2000; Aga, Samoli et al. 2003; Anderson, Atkinson et al. 2003; Sandstrom, Frew et al. 2003; Filleul, Rondeau et al. 2004; Gauderman, Avol et al. 2004; Meng, Wilhelm et al. 2007).

There are some counterintuitive findings, such as the increased odds of an ED visit for those with an asthma management plan or a daily asthma medication. This may be related to the fact that CHIS is a cross-sectional survey, which introduces temporal ambiguity between outcomes and adjustment variables. In this case, individuals may be more likely to take daily medication or receive a disease management plan after an ED visit. Also, these findings could be due to the fact that these two measures may be indicators of disease severity, which is highly correlated with ED visits. The other counterintuitive findings, such as the presence of

cockroaches in the home and experiencing delays in care for any medical reason decreasing the odds of having daily medication or missing 2 or more work days, may be explained by the observation that these respondents were likely to be low-income populations; therefore, they may not have been able to afford medication nor had paid sick days, so they could not afford to miss work.

### Pollutant Interactions with Poverty and Race/Ethnicity for Asthma Outcomes

Another major contribution of this study is the detection of significant interactions for poverty and race/ethnicity, indicating that some racial/ethnic and income groups may be more vulnerable to the effects of air pollutant exposures on asthma outcomes. African American and Asian/Pacific Islander adults may be more vulnerable to the effects of $NO_2$ on respiratory health. Also, minority children appeared to have increased vulnerability to $NO_2$ and $PM_{10}$. Specifically, for the same increase in $NO_2$ exposure, African American and Asian/PI/other adults had greater increases in odds of two or more missed days of work due to asthma compared to white adults. African American adults also had a greater increase in odds of daily/weekly asthma symptoms for a similar increase in $NO_2$ exposure. American Indian/Alaska Native and Asian/PI/other children had greater increases in odds of daily/weekly symptoms than white children with similar exposure to $NO_2$. For children we also observed significant interactions between race/ethnicity and $PM_{10}$, with Latino children having a greater increase in odds of using daily asthma medication for the same increase in $PM_{10}$ levels, and African-American and Asian/PI/other children having greater increases in odds of experiencing daily/weekly symptoms for the same increase in $PM_{10}$ exposure compared to white children. Additionally, we found significant interactions for household poverty level and $NO_2$ exposure; among children we found a greater increase in odds of ED visits among those living below 200% of the FPL compared to those living at or above 400% of the FPL for the same increase in $NO_2$ exposure.

As Lipfert (2004) and others have pointed out, low socioeconomic status is a double-edged sword that fosters living in areas of increased pollution and also makes individuals more vulnerable to pollutant effects (Sexton and Adgate 1999; O'Neill, Jerrett et al. 2003; Lipfert 2004). Although a limited number of studies have addressed whether SES modifies the health effects of air pollution among those in disadvantaged circumstances, some of these studies provide support for our findings. In a study of chronic effects, neighborhood income level modified the health effects of air pollution (Finkelstein, Jerrett et al. 2003). They found that people with low incomes and high exposure were 2.3 times more likely to die from causes associated with air pollution exposures than those in the same exposure groups with high incomes. Our previous study using CHIS 2001 also had similar findings. We found greater estimated traffic effects for those with asthma in poverty, whereas the estimates for those with asthma above the poverty level moved closer toward the null (Meng, Wilhelm et al. 2008).

The increased vulnerability of these low SES sub-populations may result from many factors. In addition to pollutant exposures, these lower SES groups suffer from the burden of reduced health from material deprivation and psychosocial stress. Given the potential financial burden of needing daily asthma medication, it may be harder for respondents with low SES to obtain prescribed asthma medications. Without medication to temper their asthma symptoms, low SES individuals may also be more likely to experience severe asthma outcomes. A previous study by Ungar et al., reported a 14% increase in asthma exacerbations, defined as hospital or

ED visits, for each percentage point increase in the proportion of out-of-pocket household income spent on asthma medications ($p<.001$) (Ungar, Paterson et al. 2011). The results of our sensitivity analyses similarly suggested that respondents not taking a daily asthma medication may be more affected by pollutant exposures and be more likely to experience severe asthma outcomes, such as ED visits or missing two or more days of school/work. Additionally, previous literature has demonstrated that low SES individuals may be more likely to use EDs as the first source of care, even for non-emergencies, further inflating the disparity in ED visit use between those of high and low SES (O'Brien, Stein et al. 1997; Hong, Baumann et al. 2007). Nutrition can also play a role, as a lower consumption of anti-oxidants and nutrients to prevent inflammation among these sub-populations may be a contributing factor (Sienra-Monge, Ramirez-Aguilar et al. 2004). Moreover, low SES groups often live in older housing, which may not be well insulated, and thus have potentially higher intrusion of outdoor pollutants, such as motor vehicle exhaust (Houston, Wu et al. 2004). Additionally, exposures to other indoor allergens, such as dust mites and fungal spores that are believed to induce asthma symptoms (Zhong 1996) are more common in households with low SES status (Sarpong, Hamilton et al. 1996). Psychosocial stress has been linked to asthma morbidity (Wright and Steinbach 2001; Wright, Mitchell et al. 2004; Clougherty, Levy et al. 2007) and was also found to be higher among lower SES individuals than higher SES individuals with asthma (Chen, Fisher et al. 2003). Our findings contribute to the existing literature since these interactions have not been demonstrated in other studies. However, further studies are needed to better explore these relationships and to determine factors contributing to these differential effects.

## Asthma-like Symptoms among Californians

Exposure disparities among respondents with asthma-like symptoms were overall very similar to those found for respondents with current asthma. In general, adults and children living below 400% of the FPL had higher pollutant exposures with some exceptions, notably $O_3$ for children and adults and $PM_{10}$ for children. Latinos had higher annual average pollutant concentrations for all pollutants except $O_3$. We observed associations for wheeze and some pollutants, and our results are suggestive of associations between pollutants and two or more wheeze attacks as well as seeking medical help for wheezing. On average, associations for wheeze outcomes and pollutants appeared weaker than those for the asthma outcomes. This may be due to outcome misclassification, since wheezing is a broad outcome that may include people with undiagnosed asthma, viral illnesses, chronic conditions, or other respiratory issues. Respondents who mentioned COPD, emphysema, or bronchitis when asked about wheezing or whistling sounds in their chest in the past year were excluded from the wheeze outcomes, but this was only if they mentioned having one of these conditions. The literature on asthma-like symptoms is sparse. One study found environmental tobacco smoke and low-socioeconomic status to be associated with asthma-like symptoms (Yeatts, Davis et al. 2003).

## Study Strengths and Limitations

Currently, California only has surveillance capacity for asthma hospitalizations and ED visits, therefore, only reflecting severe asthma outcomes. CHIS provides a representative sample of Californians and the ability to examine air pollution associations with many other outcome measures among those with current asthma that affect a much larger population than

asthma mortality and hospitalizations/ED visits, such as medication use, frequency of symptoms, and school/work days missed while adjusting for many potential confounders. For instance, we had information on many potential confounding risk factors for asthma such as socioeconomic status, asthma disease management, behavior-related factors (e.g., smoking), access to health care, housing conditions and indoor air pollution exposures (e.g., secondhand smoking), and co-morbidities. Thus, this study provided an opportunity to examine the independent, as well as combined, effects of these factors on health outcomes. In addition to asthma outcomes, the study also evaluated associations with the prevalence of asthma-like symptoms among Californians with undiagnosed asthma or other respiratory diseases.

California has a large, ethnically diverse populations and covers geographic areas with both high and low air pollution levels in comparison to the rest of the nation. Therefore, it is very important to address a major goal of CARB's Environmental Justice Policy "to better characterize air pollution exposures in communities and to better assess health impacts, especially non-cancer effects, cumulative effects, and effects from long-term low-level exposures on vulnerable populations." To our knowledge, this is the first study to test the following hypotheses: 1) Among those with asthma or asthma-like symptoms, vulnerable sub-populations in California (e.g., racial/ethnic minorities and low-income individuals) have higher exposures to air pollution; 2) Individuals with asthma or asthma-like symptoms exposed to higher levels of air pollution are more likely to report adverse health outcomes; 3) Air pollution exposures, low socioeconomic status, and certain vulnerability factors exert independent adverse effects on individuals with asthma or asthma-like symptoms; and 4) Higher pollutant exposures interact with vulnerability factors, resulting in greater air pollution impacts on asthma in vulnerable sub-populations.

Some limitations associated with using CHIS data should be noted. First, this study is based on one year of CHIS survey data (2003) with a limited sample of Californians with asthma or asthma-like symptoms. Therefore, cautions need to be taken when generalizing the findings to the entire state population and to the impact of air pollution over years. Also, CHIS is a cross-sectional survey, which may raise concerns regarding temporal ambiguity between our outcome and pollutant measures. Because the CHIS 2003 survey collected information on duration of residence in the same house and neighborhood, we used 12-month pollutant data prior to the interview date and limited our study sample to those living in the same neighborhood for at least 9 months. Thus, even though we were not able to ascertain whether exposures occurred before outcome events in some cases (e.g. ED visits), we were able to assure that the exposure measurement periods were contemporaneous with the outcome measurement periods. Another limitation to note is that the study outcomes (such as prior asthma diagnosis) were self-reported and not verified by objective clinical measures. While clinical measurements of airway responsiveness appear to reflect the activity and severity of asthma at the time of measurement, it is generally accepted that data on long-term prevalence of symptoms and exacerbations may be better obtained by questionnaires (Eder, Ege et al. 2006).

There may also be concerns regarding selection bias due to non-response. For instance, Californians with low-SES may be less likely to respond to the survey. Selection bias usually occurs when the exposure could systematically influence the selection/response of cases and/or controls. For example, bias may result from a higher or lower response rate in exposed

cases than exposed controls. However, we believe that it is unlikely that CHIS respondents were aware of their personal exposure status in order to self-select differentially i.e. that it is most likely both cases and controls were selected independently of their knowledge of exposure status, since exposure status was not determined by interview retrospectively (vulnerable to recall bias), but calculated from routinely collected air monitoring and traffic data. In a subsequent survey (CHIS 2007), CHIS has conducted a special survey procedure in which follow up could be done with persons who were selected for the sample but did not respond to attempts to interview them as part of the telephone survey to try to assess the non-response bias. This survey found relative to the responders, that non-responders were more likely to be younger, Latino, tended to live in households with children, tended to be less educated, and have lower income. When low-SES Californians are the least likely to respond to the survey and also the most likely to be exposed, it will indirectly lead to differential bias by exposure status. Furthermore, people with health problems may also be more or less likely to respond for various reasons. Though selection bias is particularly relevant in case-control studies, even when the design is population-based like CHIS, it could still be an important issue for the reasons mentioned above.

Since most of the questions relevant to the study in CHIS asked respondents to recall what happened in the previous 12 months, there might be recall bias or error in the self-reported asthma morbidity indicators. Errors in recall might lead us to categorize some cases and non-cases improperly. Previous studies have shown that people are able to recall frequent events (such as frequent asthma symptoms) or rare but clinically significant episodes (such as ED visits or being diagnosed with asthma) very well (Pless and Pless 1995). However, this might not be true for certain measures, for instance, respondents might not be able to recall the number of days of school/work missed due to asthma accurately for more than the proximate past. As a result, we decided to use the measure of 2 or more school days missed instead of number of work/school days missed, which may be more error prone. We would expect this error to be similar for exposed and unexposed cases, i.e., non-differential with regard to exposure status.

Since CHIS is a telephone-based survey, the rapid growth of cellular telephone use over the past decade may create coverage problems for CHIS. Cell phones may generate two issues that may lead to non-coverage bias in telephone surveys. To assess non-coverage bias in CHIS, the landline RDD sample was supplemented with a sample of adults living in households with only cell phones in 2007. For the cell-phone-only sample, a sample of telephone numbers designated for cellular use was drawn and screened; only cell phone users that did not have a landline telephone at home were eligible to complete the adult survey. CHIS found relative to the landline sample, the cell-phone-only sample had a slightly higher proportion of non-Latino African Americans and a lower proportion of non-Latino whites than the landline sample. The household income of the cellular phone respondents is lower, perhaps reflecting a higher likelihood of having lower education and being single. The cell-phone-only sample is less likely to be unemployed by 11.7 percentage point. Again, if we believe the patterns of cell-phone-only households applies to CHIS 2003, it will indirectly lead to differential bias by exposure status when low-SES Californians are the least likely to be included in the survey and also the most likely to be exposed.

We mainly relied on residence-based air pollution exposure estimates and therefore lack personal exposure measures. CHIS did not collect information on respondents' work locations. Thus, our exposure measures did not take into account inter-individual variability in exposures due to personal mobility, and indoor, commuting and occupational exposures, and other factors, especially for non-elderly adults. However, previous cohort studies that included multiple communities assigned exposure based on community-average pollution concentrations and considered long-term health effects have shown that the results are relatively unaffected by a lack of personal exposure measures (Neuman, Graham et al. 2011). We did a sensitivity analysis, as recommended by the Research Screening Committee, to examine the potential importance of the resulting exposure misclassification on our air pollution effect estimates. Based on stratified analyses, we observed that the association between $PM_{10}$ and increased odds of ED visits in adults with asthma appeared isolated to employed individuals (Table 37), which is the opposite of what one would expect if exposure measures were less misclassified for unemployed individuals and this misclassification is non-differential. Employed individuals could have other risk factors (e.g., co-morbidities) that make them more vulnerable to $PM_{10}$ effects. Or associations in employed individuals may reflect, in part, time spent commuting, since in-vehicle air pollution exposures have been shown to be higher than ambient exposures (Fruin et al., 2004, Westerdahl et al., 2005, Zhu et al., 2007).

People in developed countries tend to spend the majority of their time indoors, and thus exposure to outdoor air pollution is modified by time spent indoors. One important factor influencing indoor and personal exposures to pollutants is the fraction of outdoor air that penetrates indoors, which is a function of pollutant type and home ventilation characteristics, including use of air conditioning. CHIS did not ask questions about the age of housing structures and use of air conditioning. The American Housing Survey (AHS) collected such data for a limited number of metropolitan cities, and we could not link that data to CHIS respondents. We originally planned to use these data to identify certain characteristics of people that have air conditioning (e.g., high SES) so that we could use those characteristics to extrapolate air conditioning use information to the CHIS population for stratified analyses. However, since the use of air conditioning also depends heavily on meteorology and topographic conditions of an area, e.g. residents of coastal areas are less likely to use air conditioning, we decided not to use the AHS data for extrapolating air conditioning use. Indoor pollutant exposures may be elevated in low income housing due to multiple sources, such as cigarette smoking, mold, and gas appliance combustion and small apartment sizes (Zota, Adamkiewicz et al. 2005). For example, levels of $NO_2$ and CO have been found to be substantially higher in low income, inner-city residences relative to the U.S. average (Schwab 1990). Also, subjects living in source and receptor areas of $O_3$ may experience different health effects, especially among those low-income subjects who receive higher $O_3$ exposures. Additionally, exposures to cockroaches, dust mites, and fungal allergens that are believed to induce asthma symptoms (Zhong 1996) are more common in households with generally poor living conditions (Sarpong, Hamilton et al. 1996). In addition to adult and adolescent active smoking habits, CHIS 2003 asked if anyone smokes cigarettes, cigars, or pipes anywhere inside the home, and if yes, about how many days per week. CHIS 2003 also ascertained if any dogs/cats are allowed inside the home and whether any cockroaches were present inside the home in the past 12 months, as well as type of housing. Thus we assessed exposures to these indoor pollutants and controlled for them as potential

confounders in our analyses. However, CHIS did not ask questions about all the other possible asthma triggers, such as age of housing, mold, dust mites, and use of gas appliances. As a result, we could not control for these possible triggers in this study.

## V. SUMMARY AND CONCLUSIONS

Although several studies have linked air pollution to asthma morbidity, studies are still needed to identify vulnerable sub-populations with a higher burden of asthma and to investigate whether higher pollutant exposures and possibly increased vulnerability to pollutants among these sub-populations contribute to the excess burden. Linking existing air pollutant data from ambient monitors, traffic data, and California Health Interview Survey (CHIS) 2003 data allowed us to conduct a study to address these issues. This study furthers the California Air Resources Board's (CARB) Vulnerable Population Research Program that aims to protect all California residents, particularly individuals considered especially susceptible, from the adverse effects of air pollution.

To investigate the effects of air pollution on those with asthma and asthma-like symptoms in California and to identify potentially vulnerable subgroups, we conducted a cross-sectional study linking California Health Interview Survey (CHIS) 2003 data to existing air pollutant and traffic data. We considered three populations in our analyses, CHIS 2003 respondents with lifetime asthma (N=5,620 adults and 1,889 children), current asthma (N=3,587 adults and 1,224 children), and those not diagnosed with asthma who experienced asthma-like symptoms (N=4,413 adults and 1,109). Respondents living in their current neighborhood for less than nine months were excluded. Using Geographic Information System (GIS) software, we linked these respondents' residential addresses to the nearest government air monitoring stations for each of four criteria pollutants ($O_3$, $PM_{10}$, $PM_{2.5}$, and $NO_2$). We then calculated annual pollutant averages for the 12-month period prior to respondents' interview dates. Additionally, we calculated the number of days concentrations measured at the nearest air monitor exceeded federal and state standards for these pollutants. To capture exposure to traffic pollutants, we assessed traffic density and distance to roadway as proxies for traffic exposure based on residential address. Once the exposures were calculated for respondents, we performed logistic regression analyses on respondents with asthma and respondents with asthma-like symptoms, separately for children and adults. Logistic regressions and interaction terms were used to evaluate increased vulnerability to pollutants among sub-populations. We also conducted pollutant-outcome analyses adjusting for several potential confounders related to vulnerability, such as smoking, obesity, heart disease, and having a usual source of health care.

In conclusion, we observed disparities in exposure to air pollutants by federal poverty level, and race/ethnicity among Californians with current asthma. In general, higher annual average exposures were observed for lower income groups and racial/ethnic minorities for $NO_2$, $PM_{10}$, and $PM_{2.5}$. In contrast, annual average $O_3$ exposure was generally lower or the same in these groups compared to higher income individuals and whites. Similar exposure disparities were observed for respondents with asthma-like symptoms. Among adults, we observed increases in odds of having asthma attacks, using daily asthma medication, missing 2 or more work days due to asthma, and asthma-related emergency department visits with increasing annual average pollutant concentrations for $O_3$, $PM_{10}$, and $PM_{2.5}$. Among children, use of daily

asthma medication and school/day care absences were associated with higher exposures to annual average $NO_2$ concentration. We also observed associations with the number of days exceeding federal or state standards for $O_3$, $PM_{10}$, and $PM_{2.5}$. We observed few consistent associations between residence-based measures of traffic and asthma outcomes in adults. We also were able to adjust for several potential confounders in our analyses and found that pollutant associations remained. Some of the novel findings of this work are the interaction between race/ethnicity and household federal poverty level with annual average pollutant exposures for $NO_2$ and $PM_{10}$, suggesting that racial/ethnic minority and low-income groups have greater increases in adverse health effects at the same level of increase in exposures. In respondents with undiagnosed asthma, positive associations were observed between asthma-like symptoms and annual air pollutant averages and exceedance measures, and again only a few associations were seen with traffic density and distance roadway measures.

## VI. RECOMMENDATIONS

Our results provide much needed information on the effects of long-term air pollution exposure on chronic severe asthma and asthma-like symptoms in uniquely vulnerable populations, such as racial/ethnic minorities, and low-income Californians. The results of this project indicated that current air quality in California needs to be further improved in order to protect sensitive populations, such as those suffering from asthma or asthma-like symptoms; also, more actions need to be taken to protect vulnerable sub-populations.

Further regulatory efforts are needed to reduce emissions and identify contributing sources and toxic constituents of air pollution. In addition to regulatory interventions to reduce emissions, interventions at the community level should also be given attention, for example, by requiring minimum distances to pollutant sources, e.g. freeways. Locating schools, day care centers, work places, homes, sports fields and parks away from busy roadways and other emission sources should be part of the requirements for community development. Additional monitoring of air pollution from mobile sources, for example near freeways and major roads, would be a tremendous asset in assessing the health effects of traffic related pollution. Individual level interventions are also needed to modify pollutant exposure and/or dose and to help individuals mitigate the health effects of air pollution. For instance, information on control of air pollution exposures, such as reducing outdoor activities when the air quality index is in the unhealthy range and exercising away from major roadways should be widely spread. Our study also indicates that further studies are needed to explore the relationships of socioeconomic status and race with air pollution and respiratory health effects. Other important areas for future work include research identifying factors that may increase vulnerability to pollutant effects and the testing of innovative strategies to reduce individual exposures and vulnerability to air pollution through community-based or family-focused interventions.

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## VIII. LIST OF INVENTIONS REPORTED AND COPYRIGHTED MATERIALS PRODUCED

Not applicable.

# IX. GLOSSARY OF TERMS, ABBREVIATIONS, AND SYMBOLS

µg- microgram
µm- micrometer
AADT- annual average daily traffic
AI- American Indian
AN- Alaska Native
BMI- body mass index
BRFSS- Behavioral Risk Factor Surveillance System
CARB- California Air Resources Board
CHIS- California Health Interview Survey
CI- confidence interval
CO- carbon monoxide
COPD- Chronic Obstructive Pulmonary Disorder
ED- emergency department
EPA- Environmental Protections Agency
FPL- federal poverty level
ft- feet
GIS- Geographic Information System
HPMS- Highway Performance Monitoring System
hr- hour
km- kilometer
L- length
m- meter
NHIS- National Health Interview Survey
$NO_2$- nitrogen oxide
NTAD- National Transportation Atlas Database
$O_3$- ozone
OR- odds ratio
PAR- population attributable risk
PI- Pacific Islander
PM- particulate matter
$PM_{10}$- particulate matter less than 10 µm in aerodynamic diameter
$PM_{2.5}$- particulate matter less than 2.5 µm in aerodynamic diameter
ppb- parts per billion
RDD- random-digit dial
SES- socioeconomic status
$SO_2$- sulfur dioxide
std. error- standard error
TD- traffic density
UC- University of California
VMT- Vehicle Meters Traveled

# X. APPENDIX

Table 6. Weighted distributions of annual pollutant averages, exceedance days, and traffic density (within 750 feet) for CHIS 2003 adults and children with current asthma[a]

| | Adults (≥18 years) | | | | | | | | Children (< 18 years) | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | n | Missing | Min | Max | Mean | Std Error of Mean | Median | 95th Percentile | n | Missing | Min | Max | Mean | Std Error of Mean | Median | 95th Percentile |
| **Pollutant Averages** | | | | | | | | | | | | | | | | |
| $O_3$ (ppb) | 1,617 | 242 | 22.8 | 63.5 | 41.6 | 0.27 | 40.1 | 54.7 | 537 | 62 | 23.0 | 64.2 | 41.3 | 0.49 | 39.5 | 54.4 |
| $PM_{10}$ (μg/m$^3$) | 1,304 | 224 | 12.3 | 80.1 | 28.6 | 0.34 | 27.0 | 45.2 | 436 | 67 | 13.0 | 80.1 | 30.0 | 0.61 | 29.6 | 46.2 |
| $PM_{2.5}$ (μg/m$^3$) | 990 | 703 | 4.1 | 27.5 | 16.0 | 0.18 | 15.4 | 23.4 | 335 | 216 | 7.4 | 26.2 | 16.8 | 0.34 | 17.8 | 23.5 |
| $NO_2$ (ppb) | 1,315 | 482 | 1.5 | 36.1 | 21.1 | 0.27 | 20.0 | 35.0 | 469 | 127 | 1.6 | 36.0 | 22.0 | 0.48 | 21.3 | 35.1 |
| **Exceedances (in days)** | | | | | | | | | | | | | | | | |
| $O_3$ 1-Hr (State) | 1,621 | 236 | 0 | 122 | 22.4 | 0.87 | 9.8 | 75.3 | 540 | 59 | 0 | 122 | 24.9 | 1.63 | 11.7 | 70.9 |
| $O_3$ 8-Hr (State) | 1,617 | 242 | 0 | 153 | 31.1 | 1.11 | 16.0 | 97.9 | 537 | 62 | 0 | 153 | 33.5 | 2.10 | 16.7 | 98.6 |
| $O_3$ 8-Hr (Federal) | 1,617 | 242 | 0 | 114 | 16.8 | 0.77 | 4.0 | 65.1 | 537 | 62 | 0 | 114 | 18.4 | 1.39 | 4.7 | 63.8 |
| $PM_{10}$ (State) | 1,304 | 224 | 0 | 66 | 7.2 | 0.37 | 2.9 | 25.6 | 436 | 67 | 0 | 65 | 7.8 | 0.65 | 3.1 | 26.7 |
| $PM_{10}$ (Federal) | 1,304 | 224 | 0 | 4 | 0.1 | 0.02 | 0.0 | 0.0 | 436 | 67 | 0 | 4 | 0.1 | 0.02 | 0.0 | 0.7 |
| $PM_{2.5}$ (Federal) | 990 | 703 | 0 | 54 | 15.5 | 0.49 | 12.8 | 41.3 | 335 | 216 | 0 | 54 | 17.5 | 0.99 | 14.2 | 48.1 |
| **Traffic density (VMT/day/meter$^2$ )** | | | | | | | | | | | | | | | | |
| 750 feet buffer[b] | 2940 | 1 | 0.1 | 583.0 | 66.0 | 2.29 | 47.4 | 195.2 | 1018 | 0 | 1.1 | 793.4 | 70.1 | 4.06 | 45.7 | 284.4 |

[a]For CHIS 2003, current asthma is defined as reporting an asthma attack in the previous 12 months or answering yes to the question, "Do you still have asthma?"; only respondents who lived within 5 miles of an air monitoring station and lived in the same home or neighborhood for at least 9 months were included.

[b]Based on imputed Tele Atlas traffic data.

Table 7. Frequencies for distance to roadway measures for CHIS 2003 adults and children with current asthma[a]

| | Adults (≥18 years) | | Children (< 18 years) | |
|---|---|---|---|---|
| Roadway Measure[b] | n | % (Weighted) | n | % (Weighted) |
| <300 m from a State Highway | 134 | 5.4 | 45 | 5.4 |
| <300 m from an Interstate Highway | 281 | 9.9 | 102 | 12.1 |
| <50 m from a Major Road | 584 | 20.5 | 172 | 19.1 |
| <50 m from a Minor Road | 2,546 | 87.1 | 894 | 90.2 |

[a]For CHIS 2003, current asthma is defined as reporting an asthma attack in the previous 12 months or answering yes to the question, "Do you still have asthma?"; only respondents who lived in the same home or neighborhood for at least 9 months and geocoded based on address and nearest cross-streets were included.
[b]Based on imputed Tele Atlas data.

Table 8. Pearson correlations between annual average air pollutant concentrations, exceedance measures, distance to roadway measures and traffic density[a]

| | $O_3$ (ppb) | $PM_{10}$ ($\mu g/m^3$) | $PM_{2.5}$ ($\mu g/m^3$) | $NO_2$ (ppb) | Traffic Density (750 ft buffer) (VMT/day/meters$^2$) | Distance to State Highway (meters)[b] | Distance to Interstate (meters)[b] | Distance to Major Roads (meters)[b] | Distance to Minor Roads (meters)[b] | $O_3$ 1-Hr (State) (days) | $O_3$ 8-Hr (Federal) (days) | $O_3$ 8-Hr (State) (days) | $PM_{10}$ (Federal) (days) | $PM_{10}$ (State) (days) | $PM_{2.5}$ (Federal) (days) |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| $O_3$ (ppb) | 1 | | | | | | | | | | | | | | |
| $PM_{10}$ ($\mu g/m^3$) | 0.49 | 1 | | | | | | | | | | | | | |
| $PM_{2.5}$ ($\mu g/m^3$) | 0.39 | 0.78 | 1 | | | | | | | | | | | | |
| $NO_2$ (ppb) | 0.04 | 0.56 | 0.72 | 1 | | | | | | | | | | | |
| Traffic Density (750 ft buffer) (VMT/day/meters$^2$)[b] | -0.06 | 0.03 | 0.05 | 0.13 | 1 | | | | | | | | | | |
| Distance to State Highway (meters)[b] | 0.21 | -0.02 | -0.10 | -0.18 | 0.06 | 1 | | | | | | | | | |
| Distance to Interstate (meters)[b] | 0.14 | -0.02 | -0.12 | -0.18 | -0.26 | -0.01 | 1 | | | | | | | | |
| Distance to Major Roads (meters)[b] | 0.05 | 0.01 | -0.06 | -0.06 | -0.22 | -0.05 | 0.22 | 1 | | | | | | | |
| Distance to Minor Roads (meters)[b] | 0.00 | -0.04 | -0.04 | -0.05 | 0.01 | 0.04 | 0.14 | -0.04 | 1 | | | | | | |
| $O_3$ 1-Hr (State) (days) | 0.82 | 0.59 | 0.49 | 0.25 | -0.01 | 0.22 | 0.06 | 0.02 | 0.01 | 1 | | | | | |
| $O_3$ 8-Hr (Federal) (days) | 0.83 | 0.56 | 0.45 | 0.13 | -0.03 | 0.23 | 0.10 | 0.03 | 0.01 | 0.98 | 1 | | | | |
| $O_3$ 8-Hr (State) (days) | 0.89 | 0.56 | 0.43 | 0.12 | -0.05 | 0.23 | 0.13 | 0.04 | 0.01 | 0.95 | 0.97 | 1 | | | |
| $PM_{10}$ (Federal) (days) | 0.19 | 0.43 | 0.06 | -0.02 | -0.04 | -0.04 | 0.10 | 0.05 | -0.01 | 0.15 | 0.16 | 0.15 | 1 | | |
| $PM_{10}$ (State) (days) | 0.44 | 0.81 | 0.49 | 0.22 | -0.02 | 0.02 | 0.03 | 0.03 | -0.02 | 0.56 | 0.56 | 0.54 | 0.60 | 1 | |
| $PM_{2.5}$ (Federal) (days) | 0.15 | 0.51 | 0.69 | 0.45 | 0.05 | -0.05 | -0.11 | -0.09 | -0.07 | 0.20 | 0.17 | 0.19 | -0.04 | 0.32 | 1 |

[a]For CHIS 2003, current asthma is defined as reporting an asthma attack in the previous 12 months or answering yes to the question, "Do you still have asthma?"; only respondents who lived within 5 miles of an air monitoring station, lived in the same home or neighborhood for at least 9 months, and geocoded by address or nearest cross streets were included.
[b]Based on imputed Tele Atlas traffic data or Tele Atlas Dynamap 2000 roadway map .

Table 10. Characteristics of CHIS 2003 children and adults with current asthma[a]

| | | Adults | | | Children | | |
|---|---|---|---|---|---|---|---|
| | | n | Pop. N | % (wtd.) | n | Pop. N | % (wtd.) |
| Sex | | | | | | | |
| | Male | 960 | 591,941 | 34.7 | 658 | 491,096 | 58.4 |
| | Female | 2,383 | 1,115,836 | 65.3 | 495 | 350,513 | 41.6 |
| Age (yr) | | | | | | | |
| | 0-5 | - | - | - | 222 | 177,434 | 21.1 |
| | 6-11 | - | - | - | 469 | 315,611 | 37.5 |
| | 12-17 | - | - | - | 462 | 348,564 | 41.4 |
| | 18-34 | 664 | 491,308 | 28.8 | - | - | - |
| | 35-64 | 1,964 | 939,448 | 55.0 | - | - | - |
| | 65+ | 715 | 277,021 | 16.2 | - | - | - |
| Race/Ethnicity | | | | | | | |
| | Latino | 368 | 294,678 | 17.3 | 245 | 226,661 | 26.9 |
| | American Indian/Alaska Native | 73 | 34,409 | 2.0 | 31 | 24,224 | 2.9 |
| | Asian/Other | 286 | 205,060 | 12.0 | 139 | 101,540 | 12.1 |
| | African American | 282 | 143,060 | 8.4 | 125 | 122,906 | 14.6 |
| | White | 2,334 | 1,030,571 | 60.3 | 613 | 366,277 | 43.5 |
| Education[b] | | | | | | | |
| | Less than 25 Years of Age | 225 | 194,646 | 11.4 | - | - | - |
| | High School Education or Less | 1,035 | 585,913 | 34.3 | 404 | 322,053 | 38.3 |
| | College or Vocational School | 1,604 | 734,275 | 43.0 | 595 | 421,236 | 50.1 |
| | Graduate School | 479 | 192,942 | 11.3 | 154 | 98,319 | 11.7 |
| Work Status | | | | | | | |
| | Employed | 1,945 | 1,051,873 | 61.8 | - | - | - |
| | Unemployed | 1,385 | 649,135 | 38.2 | - | - | - |
| Federal Poverty Level | | | | | | | |
| | 0-199% | 1,041 | 555,437 | 32.5 | 365 | 323,103 | 38.4 |
| | 200-399% | 837 | 407,082 | 23.8 | 376 | 259,102 | 30.8 |
| | ≥400% | 1,465 | 745,258 | 43.6 | 412 | 259,404 | 30.8 |
| Insurance Status | | | | | | | |
| | Uninsured All/Part of the Year | 393 | 274,915 | 16.1 | 71 | 41,168 | 4.9 |
| | Insured All of the Year | 2,950 | 1,432,862 | 83.9 | 1,082 | 800,440 | 95.1 |
| Usual Source of Care | | | | | | | |
| | Yes | 3,108 | 1,541,916 | 90.3 | 1,061 | 761,728 | 90.5 |
| | No | 235 | 165,861 | 9.7 | 92 | 79,881 | 9.5 |
| Delay in Needed Care in Last 12 Months | | | | | | | |
| | Yes | 723 | 359,191 | 21.0 | 101 | 68,460 | 8.1 |
| | No | 2,620 | 1,348,586 | 79.0 | 1,052 | 773,148 | 91.9 |
| Number of Doctor Visits in the Past Year | | | | | | | |
| | 0-1 | 587 | 345,241 | 22.1 | 251 | 181,385 | 22.9 |
| | 2-5 | 1,361 | 708,038 | 45.4 | 664 | 481,506 | 60.8 |
| | 6+ | 1,088 | 506,905 | 32.5 | 173 | 128,836 | 16.3 |
| Age at Asthma Diagnosis | | | | | | | |
| | 0-11 | 985 | 578,871 | 33.9 | 1,050 | 761,521 | 90.5 |
| | 12-17 | 309 | 186,378 | 10.9 | 103 | 80,087 | 9.5 |
| | 18-64 | 1,824 | 850,537 | 49.8 | - | - | - |
| | 65+ | 225 | 91,991 | 5.4 | - | - | - |
| Daily Asthma Medication | | | | | | | |
| | Yes | 1,653 | 813,586 | 47.6 | 421 | 311,250 | 37.0 |
| | No | 1,690 | 894,190 | 52.4 | 732 | 530,358 | 63.0 |
| Asthma Management Plan | | | | | | | |
| | Yes | 1,326 | 642,716 | 37.6 | 527 | 342,473 | 40.7 |
| | No | 2,017 | 1,065,060 | 62.4 | 626 | 499,136 | 59.3 |

[a]For CHIS 2003, current asthma is defined as reporting an asthma attack in the previous 12 months or answering yes to the question, “Do you still have asthma?"; only those who lived in the same home or neighborhood for at least 9 months were included.

[b]Representative of educational attainment level of the adult respondent or adult responding on behalf of the child.

Table 10: Characteristics of CHIS 2003 children and adults with current asthma[a] (continued)

| | | Adults | | | Children | | |
|---|---|---|---|---|---|---|---|
| | | n | Pop. N | % (wtd.) | n | Pop. N | % (wtd.) |
| Self-reported Health Status | | | | | | | |
| | Good/Very Good/Excellent | 2,258 | 1,147,937 | 67.2 | 969 | 683,856 | 81.3 |
| | Poor/Fair | 1,085 | 559,840 | 32.8 | 184 | 157,753 | 18.7 |
| Heart Disease (Adults) | | | | | | | |
| | Yes | 477 | 193,285 | 11.3 | - | - | - |
| | No | 2,866 | 1,514,491 | 88.7 | - | - | - |
| Congestive Heart Failure (Adult) | | | | | | | |
| | Yes | 96 | 26,817 | 31.7 | - | - | - |
| | No | 132 | 57,865 | 68.3 | - | - | - |
| Diabetes | | | | | | | |
| | Yes | 324 | 162,681 | 9.5 | - | - | - |
| | No | 2,979 | 1,528,892 | 89.5 | - | - | - |
| | Borderline | 40 | 16,204 | 1.0 | - | - | - |
| Body Mass Index (Adult) | | | | | | | |
| | Underweight/Normal | 1,267 | 654,162 | 38.3 | - | - | - |
| | Overweight/Obese | 2,076 | 1,053,615 | 61.7 | - | - | - |
| Body Mass Index (Teen) | | | | | | | |
| | Underweight/Normal | - | - | - | 290 | 221,431 | 63.5 |
| | Overweight/Obese | - | - | - | 172 | 127,133 | 36.5 |
| Smoking Status (Adult) | | | | | | | |
| | Current/Previous Smoker | 1,625 | 773,413 | 45.5 | - | - | - |
| | Never Smoker | 1,705 | 927,596 | 54.5 | - | - | - |
| Smokers in the Home | | | | | | | |
| | Yes | 358 | 190,362 | 11.1 | 69 | 64,252 | 7.6 |
| | No | 2,985 | 1,517,415 | 88.9 | 1,084 | 777,356 | 92.4 |
| Walking for Transportation or Leisure | | | | | | | |
| | Yes | 2,291 | 1,192,768 | 71.1 | - | - | - |
| | No | 976 | 484,724 | 28.9 | - | - | - |
| Dogs/Cats in the Home | | | | | | | |
| | Yes | 1,669 | 815,216 | 47.7 | 533 | 344,173 | 40.9 |
| | No | 1,674 | 892,560 | 52.3 | 620 | 497,436 | 59.1 |
| Cockroaches in the Home | | | | | | | |
| | Yes | 324 | 209,029 | 12.2 | 156 | 130,013 | 15.4 |
| | No | 3,019 | 1,498,748 | 87.8 | 997 | 711,596 | 84.6 |
| Rural/Urban | | | | | | | |
| | Urban | 1,208 | 682,214 | 39.9 | 402 | 342,472 | 40.7 |
| | 2nd City | 956 | 470,474 | 27.5 | 384 | 235,309 | 28.0 |
| | Suburban | 549 | 338,098 | 19.8 | 200 | 176,753 | 21.0 |
| | Town/Rural | 630 | 216,991 | 12.7 | 167 | 87,075 | 10.3 |
| Time at Current Address/Neighborhood | | | | | | | |
| | 9 months-<1 yr | 47 | 29,301 | 1.7 | 21 | 15,668 | 1.9 |
| | 1-<3 yr | 529 | 313,186 | 18.3 | 232 | 181,218 | 21.5 |
| | 3+ yr | 2,767 | 1,365,290 | 79.9 | 900 | 644,722 | 76.6 |
| Housing Type | | | | | | | |
| | House | 2,237 | 1,138,493 | 66.7 | 877 | 597,592 | 71.0 |
| | Apartment, Duplex, or Mobile Home | 1,106 | 569,284 | 33.3 | 276 | 244,017 | 29.0 |
| Household Crowding (CHIS 2003) | | | | | | | |
| | Yes | 319 | 277,822 | 16.3 | 279 | 280,846 | 33.4 |
| | No | 3,024 | 1,429,955 | 83.7 | 874 | 560,762 | 66.6 |

[a]For CHIS 2003, current asthma is defined as reporting an asthma attack in the previous 12 months or answering yes to the question, "Do you still have asthma?"; only those who lived in the same home or neighborhood for at least 9 months were included.

[b]Representative of educational attainment level of the adult respondent or adult responding on behalf of the child.

Table 11 (Detailed). Disparities in weighted mean annual pollutant concentrations by various demographic characteristics in CHIS 2003 children and adults with current asthma using bivariate analysis[a]

| | | $NO_2$ annual average (ppb) | | $O_3$ annual average (ppb) | | $PM_{10}$ annual average (μg/m$^3$) | | $PM_{2.5}$ annual average (μg/m$^3$) | |
|---|---|---|---|---|---|---|---|---|---|
| Demographics | | Adult mean | Child mean | Adult mean | Child mean | Adult mean | Child mean | Adult mean | Child mean |
| Household Federal Poverty Level (FPL) | 0 - 199 % FPL | 22.4*** | 24.1*** | 41.1 | 41.0 | 29.9** | 30.6* | 16.7*** | 17.5** |
| | 200 - 399 % FPL | 20.7 | 20.6 | 42.1 | 42.0 | 28.1 | 31.3** | 16.4** | 16.9* |
| | ≥ 400% FPL† | 20.1 | 20.2 | 41.8 | 41.1 | 27.8 | 28.0 | 15.0 | 15.3 |
| | | | | | | | | | |
| Race/ethnicity | Latino | 24.2*** | 23.9*** | 41.1* | 40.6* | 31.3*** | 30.8 | 17.9*** | 17.2* |
| | American Indian / Alaska Native | 19.2 | 23.9 | 42.0 | 47.0 | 29.4 | 32.1 | 15.3 | 19.3 |
| | Asian / Pacific Islander / Other | 22.6*** | 23.8** | 40.4* | 38.1*** | 28.8 | 28.8 | 16.2 | 16.6 |
| | African American | 21.0 | 22.1* | 39.3*** | 40.0* | 29.3 | 31.7 | 16.2* | 17.8* |
| | White† | 19.6 | 19.7 | 42.5 | 43.1 | 27.6 | 29.1 | 15.1 | 15.7 |
| | | | | | | | | | |
| Urban/Rural | Urban† | 24.4 | 25.1 | 38.9 | 37.8 | 28.6 | 29.3 | 16.2 | 17.0 |
| | Second City | 17.4*** | 18.7*** | 42.8*** | 44.3*** | 28.2 | 28.8 | 15.1** | 15.6 |
| | Suburban | 20.1*** | 19.6*** | 44.6*** | 45.5*** | 30.2* | 33.7** | 17.1 | 17.9 |
| | Town/ Rural | 10.6*** | 13.7*** | 47.6*** | 43.5** | 27.7 | 28.6 | 10.8*** | 13.9*** |
| | | | | | | | | | |
| Sex | Male | 20.8 | 22.6 | 41.2 | 41.4 | 27.7* | 30.9 | 15.7 | 17.4* |
| | Female† | 21.2 | 21.1 | 41.9 | 41.2 | 29.1 | 28.9 | 16.2 | 15.9 |
| | | | | | | | | | |
| Age (in years) | 0-5 | | 22.7 | | 41.9 | | 31.5 | | 17.8 |
| | 6-11 | | 22.3 | | 40.7 | | 29.6 | | 16.5 |
| | 12-17† | | 21.2 | | 41.6 | | 29.8 | | 16.6 |
| | 18-34 | 22.0* | | 41.4 | | 29.6* | | 16.5 | |
| | 35 - 64 | 21.0 | | 41.8 | | 28.3 | | 15.8 | |
| | 65 and above† | 19.9 | | 41.6 | | 27.8 | | 15.6 | |

[a]For CHIS 2003, current asthma is defined as reporting an asthma attack in the previous 12 months or answering yes to the question, "Do you still have asthma?"; only respondents who lived within 5 miles of an air monitoring station and lived in the same home or neighborhood for at least 9 months were included.

†Reference Group

*p < 0.05, **p < 0.01, ***p < 0.001

Table 12. Disparities in traffic and distance to roadways by various demographic characteristics in CHIS 2003 children and adults with current asthma using bivariate analysis[a]

| | | Teleatlas Traffic Density Within 750-ft Buffer (VMT/day/meter$^2$)[b] | | State Highway < 300 m[b] | | Interstate Highway < 300 m[b] | | Major Road < 50 m[b] | |
|---|---|---|---|---|---|---|---|---|---|
| | | Adult | Child | Adult | Child | Adult | Child | Adult | Child |
| **Demographics** | | mean | mean | % | % | % | % | % | % |
| Household Federal Poverty Level (FPL) | 0 - 199 % FPL | 66.6 | 84.6*** | 7.0 | 9.5* | 9.2 | 16.4** | 26.4*** | 24.7* |
| | 200 - 399 % FPL | 68.6 | 70.2* | 5.2 | 3.3 | 11.0 | 12.8 | 23.7 * | 20.7 |
| | ≥400% FPL† | 64.1 | 52.7 | 4.3 | 2.6 | 9.8 | 6.3 | 17.5 | 15.3 |
| Race/Ethnicity | Latino | 67.0 | 93.6*** | 6.3 | € | 7.5 | 18.1* | 24.2 | 19.9 |
| | American Indian / Alaska Native | 58.6 | 93.0 | € | € | € | € | 27.1 | € |
| | Asian / Pacific Islander / Other | 88.9 | 54.3 | € | € | 12.2 | € | 19.5 | 17.3 |
| | African American | 76.6* | 89.8** | € | € | 13.6 | € | 22.8 | 26.0 |
| | White† | 60.2 | 53.2 | 4.7 | 4.1 | 9.8 | 8.5 | 21.3 | 19.8 |
| Urban/Rural | Urban† | 80.6 | 84.6 | 4.4 | 4.7 | 10.9 | 11.3 | 22.9 | 27.1 |
| | 2nd City | 59.4*** | 57.4** | 4.5 | 4.8 | 8.9 | 9.0 | 22.5 | 14.2** |
| | Suburban | 59.5** | 68.8 | 4.1 | 2.8 | 10.3 | 18.0 | 17.3 | 18.1 |
| | Town/Rural | 40.6*** | 46.4*** | 13.3*** | 14.1 | 6.9* | 10.8 | 24.0 | 15.6* |
| Sex | Male | 65.3 | 68.3 | 3.3** | 5.3 | 9.0 | 12.1 | 23.0 | 18.6 |
| | Female† | 66.4 | 72.7 | 6.5 | 5.4 | 10.4 | 12.0 | 21.2 | 23.3 |
| Age | 0-5 | | 74.1 | | 6.9 | | 13.4 | | 15.5 |
| | 6-11 | | 73.1 | | 6.9 | | 14.9 | | 22.4 |
| | 12-17† | | 65.7 | | 3.3 | | 8.9 | | 21.5 |
| | 18-34 | 64.4 | | 7.2 | | 8.9 | | 22.4 | |
| | 35 - 64 | 65.8 | | 4.2 | | 10.0 | | 22.6 | |
| | 65 and above† | 69.1 | | 6.7 | | 11.3 | | 18.1 | |

[a]For CHIS 2003, current asthma is defined as reporting an asthma attack in the previous 12 months or answering yes to the question, "Do you still have asthma?"; only respondents who lived within 5 miles of an air monitoring station and lived in the same neighborhood for at least 9 months were included.
[b]Based on imputed Tele Atlas data or Tele Atlas Dynamap 2000 roadway map.
†Reference Group
€ Unstable values (CV > 30%)
* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table 13. Disparities in weighted prevalence of asthma outcomes by various demographic characteristics in CHIS 2003 children and adults with current asthma using bivariate analysis[a]

| | | Asthma Attack[b] | | ED Visits | | Daily Asthma Medication | | Missed ≥2 days of work/school[c] | | Daily/weekly asthma symptoms | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Adults | Children | Adults | Children | Adults | Children | Adults | Children | Adults | Children |
| **Demographics** | | % | % | % | % | % | % | % | % | % | % |
| Household Federal Poverty Level (FPL) | 0 - 199 % FPL | 40.3*** | 32.1* | 24.1 *** | 26.4** | 53.8*** | 45.7*** | 14.6 | 54.3*** | 35.0*** | 12.9 |
| | 200 - 399 % FPL | 32.6 | 37.1 | 12.8 | 23.0* | 46.4 | 34.4 | 13.9 | 47.12* | 31.0* | 12.9 |
| | ≥400% FPL † | 32.9 | 40.4 | 12.6 | 14.7 | 43.7 | 28.7 | 11.8 | 33.2 | 25.2 | 9.0 |
| Race/ethnicity | Latino | 32.6 | 29.8* | 26.8*** | 25.5 | 44.5 | 45.4** | 21.9*** | 54.1 | 22.2** | 11.5 |
| | American Indian / Alaska Native | 47.4 | 38.0 | € | € | 56.2 | € | € | € | 34.0 | € |
| | Asian / Pacific Islander / Other | 36.8 | 36.8 | 18.0 | € | 41.0 | 32.5 | 16.7* | 35.0 | 25.2* | 12.8 |
| | African American | 34.3 | 44.2 | 19.3 | 32.3* | 59.4** | 37.6 | 19.7** | 48.8 | 30.2 | - |
| | White † | 35.0 | 37.9 | 12.7 | 18.8 | 48.0 | 31.5 | 8.8 | 42.7 | 32.7 | 11.6 |
| Urban/Rural | Urban † | 33.5 | 35.0 | 17.0 | 25.0 | 45.8 | 39.6 | 15.6 | 45.1 | 27.7 | 10.4 |
| | Second City | 34.8 | 36.3 | 15.9 | 19.6 | 51.6 | 37.5 | 11.3* | 49.1 | 31.5 | 15.4 |
| | Suburban | 36.5 | 37.5 | 16.5 | 24.2 | 46.1 | 35.8 | 13.2 | 49.3 | 28.1 | 8.9 |
| | Town and Rural | 38.0 | 36.8 | 15.2 | 9.7*** | 47.5 | 27.7* | 9.6* | 29.2* | 35.3* | 12.6 |
| Sex | Male | 25.9*** | 36.6 | 13.8 * | 23.7 | 45.4 | 35.9 | 10.3* | 47.2 | 26.1* | 10.9 |
| | Female † | 41.8 | 35.3 | 17.8 | 19.0 | 48.8 | 38.5 | 14.7 | 43.4 | 31.7 | 12.8 |
| Age | 0-5 | | 55.2*** | | 44.3*** | | 37.7 | | 51.2 | | |
| | 6-11 | | 41.1*** | | 26.7*** | | 38.1 | | 42.9 | | 8.9* |
| | 12-17 † | | 23.4 | | 5.8 | | 35.7 | | - | | 16.1 |
| | 18-34 | 29.7 | | 13.9 | | 35.2*** | | 13.9 | | 20.3*** | |
| | 35 - 64 | 38.5 | | 18.7* | | 48.4*** | | 13.8 | | 32.3* | |
| | 65 and above † | 33.9 | | 13.1 | | 67.3 | | € | | 38.1 | |

[a]For CHIS 2003, current asthma is defined as reporting an asthma attack in the previous 12 months or answering yes to the question, "Do you still have asthma?"; only those who lived in the same home or neighborhood for at least 9 months were included.
[b]Respondents ever diagnosed with asthma who lived in the same home or neighborhood for at least 9 months were included.
[c]Data not collected for teen respondents
†Reference Group
€ Unstable values (CV > 30%)
*p < 0.05, **p < 0.01, ***p < 0.001

Table 14 (Detailed). Associations (OR (95% CI)) for 12-month pollutant averages and respiratory outcomes in CHIS 2003 adults and children with current asthma[a]

| | Asthma Attack[b] | | | | | | ED Visit for Asthma | | | | | | Daily Asthma Medication | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Pollutant | Cases | Non-Cases | Crude OR | 95% C.I. | Adj. OR[d] | 95% C.I. | Cases | Non-Cases | Crude OR | 95% C.I. | Adj. OR[d] | 95% C.I. | Cases | Non-Cases | Crude OR | 95% C.I. | Adj. OR[d] | 95% C.I. |
| Adults | | | | | | | | | | | | | | | | | | |
| $O_3$ (per 10 ppb) | 965 | 1582 | 1.20 | [1.06, 1.36] | **1.20** | [1.05, 1.36] | 245 | 1372 | 1.14 | [0.93, 1.41] | 1.19 | [0.96, 1.47] | 815 | 802 | 1.21 | [1.03, 1.41] | **1.22** | [1.04, 1.43] |
| $PM_{10}$ (per 10 μg/m$^3$) | 770 | 1272 | 1.07 | [0.94, 1.21] | 1.04 | [0.92, 1.18] | 212 | 1092 | 1.29 | [1.09, 1.52] | **1.20** | [1.00, 1.43] | 659 | 645 | 1.10 | [0.95, 1.28] | 1.12 | [0.96, 1.30] |
| $PM_{2.5}$ (per 5 μg/m$^3$) | 592 | 974 | 1.09 | [0.95, 1.27] | 1.07 | [0.93, 1.24] | 160 | 830 | 1.36 | [1.09, 1.71] | 1.22 | [0.96, 1.56] | 494 | 496 | 1.19 | [1.00, 1.42] | **1.26** | [1.05, 1.52] |
| $NO_2$ (per 10 ppb) | 782 | 1313 | 1.01 | [0.87, 1.18] | 0.99 | [0.84, 1.15] | 200 | 1115 | 1.33 | [1.06, 1.69] | 1.18 | [0.92, 1.50] | 667 | 648 | 1.01 | [0.85, 1.21] | 1.08 | [0.90, 1.30] |
| Children | | | | | | | | | | | | | | | | | | |
| $O_3$ (per 10 ppb) | 315 | 515 | 0.91 | [0.72, 1.14] | 0.88 | [0.69, 1.12] | 120 | 417 | 0.86 | [0.61, 1.20] | 0.80 | [0.55, 1.17] | 192 | 345 | 0.87 | [0.65, 1.15] | 0.88 | [0.66, 1.17] |
| $PM_{10}$ (per 10 μg/m$^3$) | 245 | 430 | 0.97 | [0.79, 1.19] | 0.97 | [0.77, 1.22] | 97 | 339 | 1.13 | [0.89, 1.43] | 1.00 | [0.74, 1.34] | 154 | 282 | 1.04 | [0.81, 1.33] | 0.97 | [0.75, 1.26] |
| $PM_{2.5}$ (per 5 μg/m$^3$) | 196 | 318 | 0.99 | [0.76, 1.28] | 0.96 | [0.74, 1.26] | 73 | 262 | 1.19 | [0.84, 1.68] | 1.01 | [0.69, 1.49] | 132 | 203 | 1.29 | [0.94, 1.78] | 1.20 | [0.87, 1.65] |
| $NO_2$ (per 10 ppb) | 282 | 430 | 0.93 | [0.72, 1.19] | 0.97 | [0.74, 1.27] | 113 | 356 | 1.23 | [0.86, 1.75] | 1.17 | [0.81, 1.69] | 171 | 298 | 1.46 | [1.07, 2.00] | 1.36 | [0.99, 1.87] |

[a]For CHIS 2003, current asthma is defined as reporting an asthma attack in the previous 12 months or answering yes to the question, "Do you still have asthma?"; only respondents who lived within 5 miles of an air monitoring station and lived in the same neighborhood for at least 9 months were included.
[b]Respondents ever diagnosed with asthma who lived within 5 miles of an air monitoring station and in the same home or neighborhood for at least 9 months were included.
[c]Data not collected for teen respondents.
[d]Adjusted for age, race, poverty level, and sex.

Table 14 (Detailed). Associations (OR (95% CI)) for 12-month pollutant averages and respiratory outcomes in CHIS 2003 adults and children with current asthma[a] (continued)

| | Missed ≥2 School/Work Days Due to Asthma[c] | | | | | | Daily/Weekly Asthma Symptoms | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Pollutant | Cases | Non-Cases | Crude OR | 95% C.I. | Adj. OR[d] | 95% C.I. | Cases | Non-Cases | Crude OR | 95% C.I. | Adj. OR[d] | 95% C.I. |
| Adults | | | | | | | | | | | | |
| $O_3$ (per 10 ppb) | 176 | 1208 | 1.09 | [0.88, 1.37] | 1.15 | [0.91, 1.46] | 504 | 1113 | 1.06 | [0.90, 1.25] | 1.03 | [0.87, 1.22] |
| $PM_{10}$ (per 10 μg/m$^3$) | 142 | 977 | 1.36 | [1.08, 1.72] | **1.28** | [1.00, 1.65] | 415 | 889 | 1.00 | [0.86, 1.17] | 1.03 | [0.89, 1.20] |
| $PM_{2.5}$ (per 5 μg/m$^3$) | 118 | 743 | 1.34 | [1.03, 1.74] | 1.23 | [0.94, 1.60] | 316 | 674 | 1.07 | [0.89, 1.28] | 1.15 | [0.96, 1.39] |
| $NO_2$ (per 10 ppb) | 147 | 985 | 1.41 | [1.07, 1.86] | 1.24 | [0.93, 1.65] | 410 | 905 | 0.96 | [0.80, 1.16] | 1.03 | [0.85, 1.25] |
| Children | | | | | | | | | | | | |
| $O_3$ (per 10 ppb) | 152 | 184 | 1.22 | [0.87, 1.71] | 1.18 | [0.83, 1.68] | 60 | 477 | 0.84 | [0.51, 1.37] | 0.77 | [0.48, 1.21] |
| $PM_{10}$ (per 10 μg/m$^3$) | 121 | 139 | 1.16 | [0.83, 1.62] | 1.07 | [0.77, 1.49] | 50 | 386 | 0.93 | [0.67, 1.29] | 0.89 | [0.63, 1.25] |
| $PM_{2.5}$ (per 5 μg/m$^3$) | 96 | 109 | 1.25 | [0.88, 1.79] | 1.17 | [0.80, 1.70] | 44 | 291 | 1.45 | [0.91, 2.30] | 1.32 | [0.85, 2.05] |
| $NO_2$ (per 10 ppb) | 132 | 166 | 1.40 | [0.98, 1.98] | 1.35 | [0.94, 1.96] | 51 | 418 | 1.17 | [0.73, 1.87] | 1.13 | [0.72, 1.75] |

[a]For CHIS 2003, current asthma is defined as reporting an asthma attack in the previous 12 months or answering yes to the question, "Do you still have asthma?"; only respondents who lived within 5 miles of an air monitoring station and lived in the same neighborhood for at least 9 months were included.
[b]Respondents ever diagnosed with asthma who lived within 5 miles of an air monitoring station and in the same home or neighborhood for at least 9 months were included.
[c]Data not collected for teen respondents.
[d]Adjusted for age, race, poverty level, and sex.

Table 15. Associations (OR (95% CI)) between air pollution exceedance days and asthma outcomes in CHIS 2003 adults and children with current asthma[a]

| | Asthma Attack[b] | | | | ED visit for asthma | | | | Daily Asthma Medication | | | | Missed ≥2 school/work days due to asthma[c] | | | | Daily/weekly asthma symptoms | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Exceedances in days | Cases | Non-Cases | OR[d] | 95% C.I. | Cases | Non-Cases | OR[d] | 95% C.I. | Cases | Non-Cases | OR[d] | 95% C.I. | Cases | Non-Cases | OR[d] | 95% C.I. | Cases | Non-Cases | OR[d] | 95% C.I. |
| Adults | | | | | | | | | | | | | | | | | | | | |
| $O_3$ 1-Hr (State) - Ref: < 0.8 days | | | | | | | | | | | | | | | | | | | | |
| 0.8 days - < 8.7 days | 271 | 470 | 1.11 | [0.82, 1.51] | 67 | 400 | 1.10 | [0.64, 1.89] | 222 | 245 | 0.86 | [0.59, 1.25] | 53 | 354 | 1.26 | [0.67, 2.35] | 128 | 339 | 0.56 | [0.38, 0.82] |
| 8.7 days - < 36.7 days | 257 | 413 | 1.35 | [0.98, 1.84] | 63 | 370 | 1.27 | [0.75, 2.16] | 216 | 217 | 1.17 | [0.80, 1.70] | 45 | 325 | 1.65 | [0.89, 3.06] | 134 | 299 | 0.80 | [0.54, 1.19] |
| ≥ 36.7 days | 256 | 376 | **1.40** | [1.03, 1.91] | 76 | 318 | **1.91** | [1.14, 3.18] | 217 | 177 | **1.52** | [1.03, 2.24] | 52 | 281 | 1.59 | [0.87, 2.92] | 135 | 259 | 0.96 | [0.65, 1.42] |
| $O_3$ 8-Hr (State) - Ref: < 1.9 days | | | | | | | | | | | | | | | | | | | | |
| 1.9 days - < 14.3 days | 260 | 425 | 1.19 | [0.89, 1.61] | 56 | 383 | 0.79 | [0.46, 1.36] | 212 | 227 | 0.90 | [0.63, 1.31] | 43 | 338 | 0.88 | [0.47, 1.64] | 122 | 317 | 0.55 | [0.38, 0.81] |
| 14.3 days - <51.2 days | 222 | 392 | 1.19 | [0.87, 1.62] | 57 | 325 | 1.07 | [0.64, 1.78] | 195 | 187 | 1.18 | [0.81, 1.71] | 47 | 282 | 1.30 | [0.73, 2.31] | 120 | 262 | 0.79 | [0.54, 1.17] |
| ≥ 51.2 days | 285 | 402 | **1.44** | [1.07, 1.94] | 82 | 355 | 1.42 | [0.88, 2.28] | 238 | 199 | **1.50** | [1.04, 2.18] | 50 | 319 | 1.24 | [0.70, 2.20] | 145 | 292 | 0.87 | [0.59, 1.27] |
| $PM_{10}$ (State) - Ref: < 1.6 days | | | | | | | | | | | | | | | | | | | | |
| 1.6 days - < 3.5 days | 193 | 300 | 1.29 | [0.92, 1.80] | 49 | 276 | 1.50 | [0.86, 2.60] | 160 | 165 | 0.83 | [0.56, 1.23] | 32 | 245 | 1.02 | [0.52, 2.00] | 97 | 228 | 0.71 | [0.47, 1.09] |
| 3.5 days - < 6.6 days | 140 | 230 | 1.25 | [0.86, 1.79] | 43 | 189 | 1.58 | [0.89, 2.79] | 106 | 126 | 0.93 | [0.62, 1.39] | 27 | 181 | 1.74 | [0.89, 3.40] | 70 | 162 | 1.03 | [0.66, 1.61] |
| ≥ 6.6 days | 223 | 347 | 1.19 | [0.86, 1.66] | 74 | 289 | **1.77** | [1.05, 2.97] | 197 | 166 | 1.16 | [0.79, 1.72] | 50 | 262 | 1.70 | [0.91, 3.18] | 118 | 245 | 0.95 | [0.63, 1.43] |
| $PM_{2.5}$ (Federal) - Ref: 4.8 days | | | | | | | | | | | | | | | | | | | | |
| 4.8 days - < 12.0 days | 105 | 201 | 0.75 | [0.50, 1.12] | 24 | 169 | 0.88 | [0.43, 1.78] | 103 | 90 | 1.11 | [0.70, 1.79] | 11 | 147 | 0.31 | [0.12, 0.80] | 61 | 132 | 1.20 | [0.73, 1.97] |
| 12.0 days - < 23.9 days | 205 | 294 | 1.08 | [0.75, 1.55] | 58 | 268 | 1.22 | [0.67, 2.22] | 155 | 171 | 0.95 | [0.61, 1.46] | 40 | 248 | 0.90 | [0.46, 1.75] | 96 | 230 | 1.02 | [0.66, 1.59] |
| ≥ 23.9 days | 133 | 222 | 1.06 | [0.72, 1.56] | 46 | 168 | **2.01** | [1.10, 3.68] | 114 | 100 | 1.49 | [0.92, 2.39] | 37 | 148 | 1.69 | [0.85, 3.35] | 76 | 138 | **1.66** | [1.02, 2.68] |
| Children | | | | | | | | | | | | | | | | | | | | |
| $O_3$ 1-Hr (State) - Ref: < 0.8 days | | | | | | | | | | | | | | | | | | | | |
| 0.8 days - < 8.7 days | 89 | 150 | 0.64 | [0.33, 1.25] | 40 | 116 | 1.85 | [0.74, 4.61] | 55 | 101 | 0.62 | [0.31, 1.26] | 47 | 50 | **2.73** | [1.09, 6.84] | 14 | 142 | 0.87 | [0.31, 2.44] |
| 8.7 days - < 36.7 days | 90 | 143 | 0.68 | [0.35, 1.34] | 35 | 112 | 1.48 | [0.56, 3.88] | 49 | 98 | 0.70 | [0.34, 1.43] | 40 | 50 | 1.98 | [0.78, 5.02] | 19 | 128 | 0.68 | [0.25, 1.86] |
| ≥ 36.7 days | 80 | 143 | 0.73 | [0.37, 1.43] | 30 | 113 | 1.09 | [0.41, 2.90] | 55 | 88 | 0.87 | [0.43, 1.78] | 45 | 50 | **3.00** | [1.20, 7.51] | 15 | 128 | 0.65 | [0.23, 1.79] |
| $O_3$ 8-Hr (State) - Ref: < 1.9 days | | | | | | | | | | | | | | | | | | | | |
| 1.9 days - < 14.3 days | 84 | 134 | 0.95 | [0.53, 1.69] | 33 | 118 | 0.76 | [0.36, 1.60] | 57 | 94 | 0.86 | [0.44, 1.70] | 42 | 48 | 1.97 | [0.85, 4.59] | 14 | 137 | 0.48 | [0.18, 1.32] |
| 14.3 days - <51.2 days | 76 | 134 | 0.56 | [0.31, 1.02] | 27 | 103 | 0.67 | [0.30, 1.49] | 46 | 84 | 0.83 | [0.40, 1.71] | 40 | 43 | 1.97 | [0.81, 4.81] | 15 | 115 | 0.45 | [0.17, 1.25] |
| ≥ 51.2 days | 87 | 152 | 0.86 | [0.48, 1.54] | 34 | 116 | 0.66 | [0.30, 1.47] | 54 | 96 | 0.93 | [0.48, 1.80] | 43 | 54 | 1.89 | [0.81, 4.43] | 19 | 131 | 0.63 | [0.25, 1.57] |
| $PM_{10}$ (State) - Ref: < 1.6 days | | | | | | | | | | | | | | | | | | | | |
| 1.6 days - < 3.5 days | 53 | 108 | 0.68 | [0.36, 1.31] | 18 | 86 | 1.12 | [0.44, 2.85] | 38 | 66 | 1.01 | [0.49, 2.11] | 28 | 35 | 1.10 | [0.42, 2.89] | 9 | 95 | 1.50 | [0.56, 4.06] |
| 3.5 days - < 6.6 days | 50 | 85 | 0.77 | [0.41, 1.46] | 22 | 66 | 1.71 | [0.72, 4.07] | 32 | 56 | 0.96 | [0.45, 2.05] | 23 | 30 | 1.13 | [0.43, 3.00] | 10 | 78 | 1.01 | [0.36, 2.79] |
| ≥ 6.6 days | 79 | 140 | 0.79 | [0.43, 1.46] | 35 | 99 | 1.08 | [0.44, 2.64] | 51 | 83 | 0.82 | [0.41, 1.66] | 43 | 39 | 1.61 | [0.64, 4.03] | 19 | 115 | 1.41 | [0.55, 3.61] |
| $PM_{2.5}$ (Federal) - Ref: 4.8 days | | | | | | | | | | | | | | | | | | | | |
| 4.8 days - < 12.0 days | 31 | 51 | 1.01 | [0.45, 2.29] | 12 | 39 | 0.46 | [0.14,1.54] | 16 | 35 | 0.72 | [0.27, 1.92] | 14 | 19 | 0.53 | [0.16, 1.78] | 2 | 49 | 0.49 | [0.09, 2.62] |
| 12.0 days - < 23.9 days | 63 | 99 | 0.97 | [0.48, 1.93] | 19 | 81 | 0.78 | [0.30, 2.05] | 49 | 51 | 2.09 | [0.93, 4.68] | 24 | 37 | 0.45 | [0.17, 1.17] | 16 | 84 | 1.68 | [0.58, 4.86] |
| ≥ 23.9 days | 55 | 82 | 0.94 | [0.47, 1.89] | 25 | 70 | 0.92 | [0.38, 2.23] | 38 | 57 | 1.35 | [0.61, 2.99] | 28 | 31 | 0.81 | [0.32, 2.08] | 14 | 81 | 1.11 | [0.41, 2.97] |

[a]For CHIS 2003, current asthma is defined as reporting an asthma attack in the previous 12 months or answering yes to the question, "Do you still have asthma?"; only respondents who lived within 5 miles of an air monitoring station and lived in the same home or neighborhood for at least 9 months were included. The categories represent the distribution of days over the exceedance measured across quartiles.

[b]Respondents ever diagnosed with asthma who lived within 5 miles of an air monitoring station and in the same home or neighborhood for at least 9 months were included.

[c]Data not collected for teen respondents.

[d]Adjusted for age, race, poverty level, and sex.

Table 16. Associations (OR (95% CI)) for traffic density/distance to roadway and asthma outcomes in CHIS 2003 adults and children with current asthma[a]

| | Asthma Attack[b] | | | | ED Visit for Asthma | | | | Daily Asthma Medication | | | | Missed ≥2 School/Work Days Due to Asthma[c] | | | | Daily/Weekly Asthma Symptoms | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Exposure | Cases | Non-Cases | OR[d] | 95% C.I. | Cases | Non-Cases | OR[d] | 95% C.I. | Cases | Non-Cases | OR[d] | 95% C.I. | Cases | Non-Cases | OR[d] | 95% C.I. | Cases | Non-Cases | OR[d] | 95% C.I. |
| Adults | | | | | | | | | | | | | | | | | | | | |
| Tele Atlas Traffic < 750ft Continuous | 1,733 | 2,858 | 1.03 | [0.97,1.09] | 438 | 2,502 | 1.08 | [0.97,1.21] | 1455 | 1,485 | 1.02 | [0.95,1.09] | 294 | 2,205 | 1.05 | [0.95,1.16] | 929 | 2,011 | 1.00 | [0.93,1.07] |
| Tele Atlas Traffic < 750ft (25th < 50th percentile)[e,f] | 437 | 736 | 1.05 | [0.84, 1.33] | 91 | 640 | 0.89 | [0.59, 1.36] | 352 | 379 | 0.92 | [0.69, 1.22] | 70 | 547 | 1.19 | [0.74, 1.92] | 227 | 504 | 1.05 | [0.78, 1.42] |
| Tele Atlas Traffic < 750ft (50th < 75th percentile)[e,f] | 433 | 686 | 1.08 | [0.86, 1.37] | 120 | 618 | **1.48** | [1.02, 2.16] | 359 | 379 | 0.98 | [0.74, 1.31] | 77 | 549 | 1.24 | [0.77, 1.98] | 238 | 500 | 1.06 | [0.79, 1.43] |
| Tele Atlas Traffic < 750ft (≥75th percentile)[e,f] | 408 | 653 | 1.07 | [0.84, 1.35] | 111 | 571 | 1.05 | [0.70, 1.58] | 350 | 332 | 0.89 | [0.66, 1.19] | 71 | 510 | 0.91 | [0.58, 1.45] | 219 | 463 | 0.99 | [0.74, 1.34] |
| State Highway < 300 m[f] | 83 | 132 | 0.82 | [0.55, 1.23] | 25 | 109 | 0.77 | [0.42, 1.41] | 73 | 61 | 0.91 | [0.55, 1.52] | 12 | 102 | 0.73 | [0.32, 1.66] | 96 | 185 | 1.16 | [0.80, 1.68] |
| Interstate Highway < 300 m[f] | 172 | 248 | 1.06 | [0.79, 1.43] | 54 | 227 | 1.51 | [0.91, 2.48] | 159 | 122 | 1.34 | [0.95, 1.90] | 30 | 209 | 1.10 | [0.64, 1.91] | 195 | 425 | 0.89 | [0.67, 1.17] |
| Major Road < 50 m[f] | 369 | 563 | 1.10 | [0.90, 1.35] | 97 | 523 | 1.02 | [0.69, 1.50] | 308 | 312 | 0.96 | [0.75, 1.24] | 57 | 486 | 0.66 | [0.44, 0.99] | 832 | 1,830 | 0.82 | [0.55, 1.24] |
| Children | | | | | | | | | | | | | | | | | | | | |
| Tele Atlas Traffic < 750ft Continuous | 589 | 990 | 1.03 | [0.93,1.14] | 203 | 815 | 0.98 | [0.88,1.09] | 372 | 646 | 1.04 | [0.94,1.16] | 258 | 347 | 1.08 | [0.94,1.24] | 112 | 906 | 0.93 | [0.76,1.15] |
| Tele Atlas Traffic < 750ft (25th < 50th percentile)[e,f] | 159 | 273 | 0.95 | [0.62, 1.45] | 59 | 202 | 0.97 | [0.50,1.87] | 104 | 157 | 1.17 | [0.69, 1.97] | 72 | 94 | 1.37 | [0.72, 2.60] | 34 | 227 | 0.93 | [0.42, 2.04] |
| Tele Atlas Traffic < 750ft (50th < 75th percentile)[e,f] | 140 | 246 | 0.91 | [0.60, 1.40] | 46 | 200 | 0.93 | [0.48,1.81] | 93 | 153 | 1.58 | [0.94, 2.66] | 60 | 80 | 1.23 | [0.63, 2.43] | 22 | 224 | 0.94 | [0.42, 2.12] |
| Tele Atlas Traffic < 750ft (≥75th percentile)[e,f] | 130 | 208 | 0.92 | [0.59, 1.44] | 51 | 175 | 0.92 | [0.49,1.73] | 88 | 138 | 1.43 | [0.83, 2.47] | 71 | 77 | 1.59 | [0.84, 3.02] | 24 | 202 | 0.93 | [0.38, 2.30] |
| State Highway < 300 m[f] | 26 | 45 | 0.76 | [0.36, 1.60] | 9 | 36 | 0.73 | [0.19, 2.74] | 16 | 29 | 0.45 | [0.17, 1.22] | 11 | 17 | 0.80 | [0.27, 2.34] | No cases | | | |
| Interstate Highway < 300 m[f] | 63 | 88 | 1.18 | [0.67, 2.07] | 25 | 77 | 1.13 | [0.54, 2.37] | 39 | 63 | 0.79 | [0.42, 1.50] | 28 | 39 | 1.23 | [0.60, 2.52] | 9 | 93 | 0.87 | [0.32, 2.37] |
| Major Road < 50 m[f] | 100 | 190 | 0.98 | [0.67, 1.42] | 34 | 150 | 0.95 | [0.55, 1.62] | 68 | 116 | 1.24 | [0.78, 1.97] | 50 | 57 | 1.34 | [0.77, 2.33] | 15 | 169 | 0.73 | [0.33, 1.61] |

[a]For CHIS 2003, current asthma is defined as reporting an asthma attack in the previous 12 months or answering yes to the question, "Do you still have asthma?"; only respondents who lived within 5 miles of an air monitoring station and lived in the same home or neighborhood for at least 9 months were included.
[b]Respondents ever diagnosed with asthma who lived within 5 miles of an air monitoring station and in the same home or neighborhood for at least 9 months were included.
[c]Data not collected for teen respondents.
[d]Adjusted for age, race, poverty level, and sex
[e]Reference: < 25$^{th}$ percentile; Units: vehicles per meter/day/meter$^2$
[f]Based on imputed Tele Atlas data or Tele Atlas Dynamap 2000 roadway map.

Table 17 (Detailed). Associations between ED visits and pollutants ($O_3$, $PM_{10}$, and $PM_{2.5}$) adjusting for vulnerability characteristics among CHIS adults with current asthma[a]

| | $O_3$ (per 10 ppb) | | | | | | | | | $PM_{10}$ (per 10 μg/m$^3$) | | | | | | | | | $PM_{2.5}$ (per 5 μg/m$^3$) | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Model 1[b] (245, 1372)[c] | | | Model 2[b] (245, 1365)[c] | | | Model 3[b] (240, 1337)[c] | | | Model 1[b] (212, 1092)[c] | | | Model 2[b] (212, 1085)[c] | | | Model 3[b] (209, 1058)[c] | | | Model 1[b] (160, 830)[c] | | | Model 2[b] (160, 825)[c] | | | Model 3[b] (157, 807)[c] | | |
| **Vulnerability Characteristic** | OR | 95% C.I. | | OR | 95% C.I. | | OR | 95% C.I. | | OR | 95% C.I. | | OR | 95% C.I. | | OR | 95% C.I. | | OR | 95% C.I. | | OR | 95% C.I. | | OR | 95% C.I. | |
| 12-Month Pollutant Average[d] | 1.19 | 0.96 | 1.47 | 1.18 | 0.95 | 1.47 | 1.15 | 0.91 | 1.45 | 1.20 | 1.00 | 1.43 | 1.18 | 0.99 | 1.42 | 1.25 | 1.03 | 1.51 | 1.22 | 0.96 | 1.56 | 1.21 | 0.95 | 1.54 | 1.20 | 0.92 | 1.56 |
| Age (Ref. ≥65) | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| 18 - 34 | 1.09 | 0.61 | 1.93 | 1.49 | 0.78 | 2.85 | 2.05 | 1.00 | 4.20 | 1.00 | 0.53 | 1.88 | 1.44 | 0.72 | 2.90 | 1.91 | 0.88 | 4.16 | 0.94 | 0.45 | 1.93 | 1.66 | 0.76 | 3.63 | 1.31 | 0.57 | 2.99 |
| 35 - 64 | 1.91 | 1.15 | 3.20 | 2.26 | 1.30 | 3.94 | 2.33 | 1.29 | 4.19 | 1.76 | 0.99 | 3.13 | 2.19 | 1.20 | 4.00 | 2.29 | 1.19 | 4.41 | 1.64 | 0.86 | 3.15 | 2.20 | 1.13 | 4.26 | 1.88 | 0.94 | 3.76 |
| Race (Ref. White) | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| African American | 1.86 | 1.04 | 3.32 | 1.76 | 0.97 | 3.20 | 1.40 | 0.73 | 2.68 | 1.42 | 0.74 | 2.71 | 1.30 | 0.67 | 2.53 | 1.16 | 0.58 | 2.29 | 1.92 | 0.99 | 3.71 | 1.95 | 0.98 | 3.87 | 1.38 | 0.71 | 2.67 |
| American Indian / Alaska Native | 1.51 | 0.43 | 5.29 | 1.46 | 0.42 | 5.16 | 0.96 | 0.29 | 3.16 | 1.36 | 0.38 | 4.93 | 1.27 | 0.33 | 4.88 | 0.83 | 0.24 | 2.91 | 0.97 | 0.16 | 5.95 | 0.89 | 0.14 | 5.58 | 0.59 | 0.10 | 3.34 |
| Asian / Pacific Islander / other | 1.26 | 0.71 | 2.26 | 1.38 | 0.77 | 2.47 | 1.41 | 0.79 | 2.51 | 0.80 | 0.42 | 1.54 | 0.84 | 0.44 | 1.59 | 0.90 | 0.45 | 1.79 | 0.96 | 0.47 | 1.96 | 1.08 | 0.52 | 2.23 | 0.92 | 0.46 | 1.84 |
| Latino | 2.03 | 1.27 | 3.23 | 2.08 | 1.30 | 3.35 | 2.23 | 1.32 | 3.78 | 2.42 | 1.46 | 4.00 | 2.46 | 1.47 | 4.12 | 2.40 | 1.37 | 4.22 | 2.03 | 1.18 | 3.49 | 2.38 | 1.35 | 4.22 | 2.05 | 1.09 | 3.88 |
| Poverty (Ref. ≥400% FPL)[e] | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| 0 - 199% FPL | 1.93 | 1.26 | 2.97 | 1.62 | 1.02 | 2.57 | 1.82 | 1.12 | 2.95 | 2.02 | 1.27 | 3.20 | 1.72 | 1.06 | 2.80 | 1.95 | 1.15 | 3.31 | 1.84 | 1.11 | 3.03 | 1.51 | 0.90 | 2.54 | 1.64 | 0.93 | 2.90 |
| 200 - 399% FPL | 1.14 | 0.69 | 1.89 | 1.07 | 0.64 | 1.80 | 1.11 | 0.68 | 1.83 | 1.34 | 0.79 | 2.27 | 1.31 | 0.76 | 2.25 | 1.37 | 0.79 | 2.39 | 1.09 | 0.57 | 2.06 | 1.09 | 0.58 | 2.05 | 0.98 | 0.51 | 1.90 |
| Sex | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Female vs. Male | 1.45 | 0.97 | 2.18 | 1.46 | 0.97 | 2.21 | 1.37 | 0.89 | 2.10 | 1.28 | 0.83 | 1.97 | 1.23 | 0.80 | 1.88 | 1.25 | 0.80 | 1.96 | 1.45 | 0.89 | 2.37 | 1.50 | 0.91 | 2.45 | 1.51 | 0.89 | 2.55 |
| Currently Insured | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Yes vs No | | | | 0.95 | 0.57 | 1.57 | | | | | | | 1.06 | 0.60 | 1.89 | | | | | | | 1.31 | 0.68 | 2.52 | | | |
| Obese | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| No vs Yes | | | | 0.80 | 0.55 | 1.15 | | | | | | | 0.70 | 0.47 | 1.03 | | | | | | | 0.83 | 0.53 | 1.30 | | | |
| Heart Disease | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Yes vs No | | | | 1.69 | 1.01 | 2.84 | | | | | | | 2.07 | 1.22 | 3.52 | | | | | | | 2.12 | 1.20 | 3.73 | | | |
| Smoker | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Ever vs Never | | | | 1.26 | 0.87 | 1.81 | | | | | | | 0.94 | 0.64 | 1.38 | | | | | | | 1.47 | 0.93 | 2.32 | | | |
| Work Status | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Employed vs. Unemployed | | | | 0.81 | 0.54 | 1.21 | | | | | | | 0.76 | 0.50 | 1.17 | | | | | | | 0.67 | 0.41 | 1.11 | | | |
| Urban/Rural (Ref. Town/ Rural) | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Urban | | | | | | | 0.84 | 0.41 | 1.70 | | | | | | | 0.72 | 0.36 | 1.46 | | | | | | | 0.84 | 0.20 | 3.53 |
| Second City | | | | | | | 0.98 | 0.49 | 1.96 | | | | | | | 0.72 | 0.36 | 1.41 | | | | | | | 1.06 | 0.27 | 4.19 |
| Suburban | | | | | | | 0.83 | 0.39 | 1.74 | | | | | | | 0.62 | 0.29 | 1.32 | | | | | | | 0.71 | 0.16 | 3.11 |
| Usual Source of Care | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Yes vs No | | | | | | | 0.95 | 0.50 | 1.79 | | | | | | | 0.98 | 0.48 | 1.99 | | | | | | | 0.72 | 0.33 | 1.56 |
| Delay in Care | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Yes vs No | | | | | | | 1.30 | 0.85 | 1.99 | | | | | | | 1.32 | 0.83 | 2.12 | | | | | | | 0.94 | 0.54 | 1.64 |
| Onset of Asthma | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Adult vs Child | | | | | | | 1.65 | 1.08 | 2.54 | | | | | | | 1.50 | 0.94 | 2.39 | | | | | | | 1.28 | 0.78 | 2.09 |
| Daily Asthma Medication | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| No vs Yes | | | | | | | 0.35 | 0.23 | 0.52 | | | | | | | 0.30 | 0.20 | 0.47 | | | | | | | 0.29 | 0.17 | 0.49 |
| Asthma Management Plan | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| No vs Yes | | | | | | | 0.52 | 0.36 | 0.76 | | | | | | | 0.56 | 0.37 | 0.84 | | | | | | | 0.63 | 0.40 | 1.00 |
| Household Smoking | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| No vs Yes | | | | | | | 0.85 | 0.46 | 1.55 | | | | | | | 1.28 | 0.66 | 2.46 | | | | | | | 1.10 | 0.54 | 2.27 |
| Dog or Cat in Home | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| No vs Yes | | | | | | | 1.01 | 0.67 | 1.50 | | | | | | | 1.01 | 0.66 | 1.57 | | | | | | | 1.18 | 0.72 | 1.93 |
| Cockroaches | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Yes vs No | | | | | | | 0.89 | 0.50 | 1.60 | | | | | | | 0.99 | 0.53 | 1.85 | | | | | | | 0.90 | 0.47 | 1.73 |
| Housing Type (Ref. House) | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Duplex or Apartment | | | | | | | 0.77 | 0.37 | 1.61 | | | | | | | 0.94 | 0.46 | 1.96 | | | | | | | 0.82 | 0.30 | 2.24 |
| Mobile Home | | | | | | | 0.58 | 0.28 | 1.19 | | | | | | | 0.80 | 0.41 | 1.59 | | | | | | | 0.62 | 0.23 | 1.65 |
| Household Crowding | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| No vs Yes | | | | | | | 1.16 | 0.68 | 2.00 | | | | | | | 1.11 | 0.60 | 2.04 | | | | | | | 1.20 | 0.63 | 2.27 |
| Diabetes (Ref. Pre-Diabetes/ | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Yes | | | | | | | 1.72 | 0.18 | 16.51 | | | | | | | 1.32 | 0.14 | 12.48 | | | | | | | 0.81 | 0.08 | 8.32 |
| No | | | | | | | 0.87 | 0.10 | 7.88 | | | | | | | 0.63 | 0.07 | 5.61 | | | | | | | 0.45 | 0.05 | 4.19 |
| Walking | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Yes vs No | | | | | | | 0.75 | 0.51 | 1.11 | | | | | | | 0.87 | 0.57 | 1.34 | | | | | | | 0.99 | 0.61 | 1.61 |

[a]For CHIS 2003, current asthma is defined as reporting an asthma attack in the previous 12 months or answering yes to the question, "Do you still have asthma?"; only respondents who lived within 5 miles of an air monitoring station and lived in the same home or neighborhood for at least 9 months were included.

[b]Only variables with listed values are included for each model.

[c](Cases, Non-cases)

[d]Refers to the criteria pollutant noted in the column heading.

[e]FPL = Federal Poverty Level

Table 18 (Detailed). Associations between daily asthma medication and pollutants ($O_3$, $PM_{10}$, and $PM_{2.5}$) adjusting for vulnerability characteristics among CHIS adults with current asthma[a]

| | $O_3$ (per 10 ppb) | | | | | | | | | $PM_{10}$ (per 10 μg/m$^3$) | | | | | | | | | $PM_{2.5}$ (per 5 μg/m$^3$) | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Model 1[b] (815, 802)[c] | | | Model 2[b] (811, 799)[c] | | | Model 3[b] (789, 788)[c] | | | Model 1[b] (659, 645)[c] | | | Model 2[b] (654, 643)[c] | | | Model 3[b] (634, 633)[c] | | | Model 1[b] (494, 496)[c] | | | Model 2[b] (491, 494)[c] | | | Model 3[b] (479, 485)[c] | | |
| **Vulnerability Characteristic** | OR | 95% C.I. | | OR | 95% C.I. | | OR | 95% C.I. | | OR | 95% C.I. | | OR | 95% C.I. | | OR | 95% C.I. | | OR | 95% C.I. | | OR | 95% C.I. | | OR | 95% C.I. | |
| 12-Month Pollutant Average[d] | 1.22 | 1.04 | 1.43 | 1.23 | 1.05 | 1.46 | 1.24 | 1.05 | 1.48 | 1.12 | 0.96 | 1.30 | 1.12 | 0.96 | 1.30 | 1.23 | 1.04 | 1.45 | 1.26 | 1.05 | 1.52 | 1.30 | 1.07 | 1.57 | 1.44 | 1.18 | 1.76 |
| Age (Ref. ≥65) | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| 18 - 34 | 0.32 | 0.21 | 0.48 | 0.52 | 0.33 | 0.83 | 0.30 | 0.19 | 0.48 | 0.32 | 0.20 | 0.51 | 0.51 | 0.30 | 0.87 | 0.33 | 0.19 | 0.55 | 0.40 | 0.24 | 0.66 | 0.80 | 0.44 | 1.44 | 0.41 | 0.23 | 0.73 |
| 35 - 64 | 0.55 | 0.39 | 0.78 | 0.76 | 0.52 | 1.12 | 0.51 | 0.35 | 0.74 | 0.53 | 0.36 | 0.79 | 0.72 | 0.46 | 1.13 | 0.50 | 0.32 | 0.77 | 0.56 | 0.36 | 0.88 | 0.91 | 0.55 | 1.49 | 0.52 | 0.32 | 0.85 |
| Race (Ref. White) | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| African American | 1.49 | 0.92 | 2.41 | 1.54 | 0.95 | 2.49 | 1.52 | 0.92 | 2.51 | 1.85 | 1.07 | 3.19 | 1.98 | 1.15 | 3.41 | 2.16 | 1.22 | 3.84 | 1.62 | 0.92 | 2.84 | 1.87 | 1.05 | 3.32 | 1.76 | 0.97 | 3.18 |
| American Indian / Alaska Native | 1.88 | 0.74 | 4.75 | 1.89 | 0.77 | 4.66 | 1.77 | 0.74 | 4.24 | 2.46 | 0.92 | 6.62 | 2.56 | 0.97 | 6.77 | 2.47 | 1.02 | 5.99 | 1.85 | 0.55 | 6.29 | 1.85 | 0.57 | 6.00 | 1.84 | 0.59 | 5.73 |
| Asian / Pacific Islander / other | 0.75 | 0.47 | 1.20 | 0.86 | 0.54 | 1.38 | 0.71 | 0.44 | 1.16 | 0.72 | 0.41 | 1.26 | 0.81 | 0.46 | 1.42 | 0.86 | 0.48 | 1.53 | 0.77 | 0.45 | 1.31 | 0.84 | 0.49 | 1.42 | 0.87 | 0.50 | 1.52 |
| Latino | 0.81 | 0.54 | 1.20 | 0.90 | 0.59 | 1.36 | 0.90 | 0.58 | 1.39 | 1.06 | 0.66 | 1.68 | 1.17 | 0.72 | 1.90 | 1.33 | 0.80 | 2.21 | 0.65 | 0.40 | 1.06 | 0.79 | 0.48 | 1.29 | 0.78 | 0.45 | 1.36 |
| Poverty (Ref. ≥400% FPL)[e] | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| 0 - 199% FPL | 1.30 | 0.94 | 1.79 | 1.16 | 0.82 | 1.65 | 1.41 | 0.98 | 2.03 | 1.16 | 0.81 | 1.65 | 1.06 | 0.72 | 1.57 | 1.28 | 0.86 | 1.90 | 1.28 | 0.86 | 1.91 | 1.12 | 0.73 | 1.72 | 1.45 | 0.94 | 2.22 |
| 200 - 399% FPL | 0.96 | 0.68 | 1.36 | 0.96 | 0.68 | 1.36 | 0.96 | 0.68 | 1.36 | 0.92 | 0.62 | 1.35 | 0.94 | 0.63 | 1.38 | 0.93 | 0.62 | 1.38 | 0.85 | 0.55 | 1.30 | 0.87 | 0.56 | 1.34 | 0.89 | 0.57 | 1.37 |
| Sex | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Female vs. Male | 1.12 | 0.85 | 1.48 | 1.11 | 0.84 | 1.48 | 1.09 | 0.82 | 1.47 | 0.95 | 0.69 | 1.30 | 0.98 | 0.71 | 1.35 | 0.94 | 0.67 | 1.31 | 1.06 | 0.75 | 1.51 | 1.07 | 0.75 | 1.53 | 0.98 | 0.68 | 1.41 |
| Currently Insured | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Yes vs No | | | | 1.56 | 1.02 | 2.38 | | | | | | | 1.42 | 0.86 | 2.35 | | | | | | | 1.86 | 1.11 | 3.11 | | | |
| Obese | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| No vs Yes | | | | 0.89 | 0.65 | 1.21 | | | | | | | 1.01 | 0.71 | 1.45 | | | | | | | 1.00 | 0.70 | 1.44 | | | |
| Heart Disease | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Yes vs No | | | | 1.77 | 1.16 | 2.69 | | | | | | | 1.81 | 1.12 | 2.91 | | | | | | | 1.73 | 1.05 | 2.84 | | | |
| Smoker | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Ever vs Never | | | | 1.41 | 1.07 | 1.86 | | | | | | | 1.31 | 0.97 | 1.77 | | | | | | | 1.47 | 1.04 | 2.06 | | | |
| Work Status | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Employed vs. Unemployed | | | | 0.75 | 0.55 | 1.02 | | | | | | | 0.78 | 0.55 | 1.12 | | | | | | | 0.58 | 0.39 | 0.86 | | | |
| Urban/Rural (Ref. Town/Rural) | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Urban | | | | | | | 0.95 | 0.56 | 1.60 | | | | | | | 0.79 | 0.45 | 1.38 | | | | | | | 1.24 | 0.44 | 3.50 |
| Second City | | | | | | | 1.25 | 0.74 | 2.11 | | | | | | | 1.07 | 0.61 | 1.86 | | | | | | | 1.91 | 0.67 | 5.42 |
| Suburban | | | | | | | 0.82 | 0.47 | 1.45 | | | | | | | 0.70 | 0.37 | 1.31 | | | | | | | 0.90 | 0.30 | 2.66 |
| Usual Source of Care | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Yes vs No | | | | | | | 2.28 | 1.35 | 3.85 | | | | | | | 1.83 | 1.02 | 3.28 | | | | | | | 3.07 | 1.57 | 5.99 |
| Delay in Care | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Yes vs No | | | | | | | 0.76 | 0.55 | 1.05 | | | | | | | 0.68 | 0.47 | 0.97 | | | | | | | 1.03 | 0.68 | 1.56 |
| Onset of Asthma | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Adult vs Child | | | | | | | 1.11 | 0.83 | 1.48 | | | | | | | 1.15 | 0.83 | 1.59 | | | | | | | 1.31 | 0.92 | 1.88 |
| Asthma Management Plan | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| No vs Yes | | | | | | | 0.46 | 0.35 | 0.61 | | | | | | | 0.44 | 0.32 | 0.60 | | | | | | | 0.41 | 0.29 | 0.59 |
| Household Smoking | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| No vs Yes | | | | | | | 0.84 | 0.54 | 1.31 | | | | | | | 0.76 | 0.47 | 1.22 | | | | | | | 0.65 | 0.38 | 1.12 |
| Dog or Cat in Home | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| No vs Yes | | | | | | | 0.89 | 0.67 | 1.19 | | | | | | | 0.73 | 0.52 | 1.01 | | | | | | | 0.80 | 0.56 | 1.14 |
| Cockroaches | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Yes vs No | | | | | | | 0.58 | 0.38 | 0.88 | | | | | | | 0.54 | 0.33 | 0.87 | | | | | | | 0.59 | 0.36 | 0.97 |
| Housing Type (Ref. House) | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Duplex or Apartment | | | | | | | 1.28 | 0.70 | 2.34 | | | | | | | 1.43 | 0.78 | 2.65 | | | | | | | 1.40 | 0.62 | 3.20 |
| Mobile Home | | | | | | | 1.19 | 0.67 | 2.12 | | | | | | | 1.26 | 0.70 | 2.25 | | | | | | | 1.33 | 0.60 | 2.99 |
| Household Crowding | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| No vs Yes | | | | | | | 0.67 | 0.43 | 1.05 | | | | | | | 0.88 | 0.52 | 1.48 | | | | | | | 0.89 | 0.53 | 1.51 |
| Diabetes (Ref. Pre-Diabetes/ Borderline Diabetes) | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Yes | | | | | | | 0.20 | 0.05 | 0.85 | | | | | | | 0.35 | 0.06 | 1.93 | | | | | | | 0.46 | 0.08 | 2.56 |
| No | | | | | | | 0.21 | 0.05 | 0.83 | | | | | | | 0.41 | 0.08 | 2.11 | | | | | | | 0.47 | 0.09 | 2.40 |
| Walking | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Yes vs No | | | | | | | 0.96 | 0.71 | 1.29 | | | | | | | 0.80 | 0.58 | 1.11 | | | | | | | 0.84 | 0.59 | 1.20 |

[a]For CHIS 2003, current asthma is defined as reporting an asthma attack in the previous 12 months or answering yes to the question, "Do you still have asthma?"; only respondents who lived within 5 miles of an air monitoring station and lived in the same home or neighborhood for at least 9 months were included.

[b]Only variables with listed values are included for each model.

[c](Cases, Non-cases) Refers to the criteria pollutant noted in the column heading.

[d]Refers to the criteria pollutant noted in the column heading.

[e]FPL = Federal Poverty Level

Table 19 (Detailed). Associations between missing 2 or more days of work pollutants ($O_3$, $PM_{10}$, and $PM_{2.5}$) adjusting for vulnerability characteristics among CHIS adults with current asthma[a]

| | $O_3$ (per 10 ppb) | | | | | | | | | $PM_{10}$ (per 10 µg/m$^3$) | | | | | | | | | $PM_{2.5}$ (per 5 µg/m$^3$) | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Model 1[b] (176, 1208)[c] | | | Model 2[b] (176, 1208)[c] | | | Model 3[b] (175, 1185)[c] | | | Model 1[b] (142, 977)[c] | | | Model 2[b] (142, 977)[c] | | | Model 3[b] (141, 957)[c] | | | Model 1[b] (118, 743)[c] | | | Model 2[b] (118, 743)[c] | | | Model 3[b] (118, 727)[c] | | |
| **Vulnerability Characteristic** | OR | 95% C.I. | | OR | 95% C.I. | | OR | 95% C.I. | | OR | 95% C.I. | | OR | 95% C.I. | | OR | 95% C.I. | | OR | 95% C.I. | | OR | 95% C.I. | | OR | 95% C.I. | |
| 12-Month Pollutant Average[d] | 1.15 | 0.91 | 1.46 | 1.15 | 0.91 | 1.44 | 1.13 | 0.88 | 1.44 | 1.28 | 1.00 | 1.65 | 1.30 | 1.01 | 1.68 | 1.25 | 0.96 | 1.63 | 1.23 | 0.94 | 1.60 | 1.23 | 0.94 | 1.60 | 1.24 | 0.93 | 1.66 |
| Age (Ref. ≥65) | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| 18 - 34 | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - |
| 35 - 64 | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - | - |
| Race (Ref. White) | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| African American | 3.79 | 2.02 | 7.13 | 3.61 | 1.92 | 6.79 | 3.81 | 1.82 | 7.99 | 3.60 | 1.73 | 7.47 | 3.45 | 1.69 | 7.05 | 3.25 | 1.43 | 7.42 | 4.69 | 2.24 | 9.81 | 4.45 | 2.11 | 9.41 | 5.50 | 2.35 | 12.89 |
| American Indian / Alaska Native | 1.73 | 0.51 | 5.85 | 1.62 | 0.46 | 5.79 | 1.86 | 0.52 | 6.71 | 1.83 | 0.55 | 6.08 | 1.67 | 0.45 | 6.16 | 1.93 | 0.57 | 6.52 | 1.13 | 0.18 | 7.28 | 1.01 | 0.15 | 6.86 | 1.34 | 0.21 | 8.53 |
| Asian / Pacific Islander / other | 2.06 | 1.07 | 3.97 | 1.98 | 1.01 | 3.87 | 2.18 | 1.11 | 4.30 | 1.15 | 0.49 | 2.68 | 1.05 | 0.46 | 2.42 | 1.14 | 0.47 | 2.78 | 1.74 | 0.82 | 3.70 | 1.69 | 0.78 | 3.64 | 1.92 | 0.91 | 4.05 |
| Latino | 3.42 | 2.06 | 5.68 | 3.30 | 1.99 | 5.49 | 3.86 | 2.25 | 6.64 | 3.60 | 2.00 | 6.47 | 3.51 | 1.94 | 6.36 | 3.82 | 2.03 | 7.18 | 3.28 | 1.76 | 6.10 | 3.31 | 1.76 | 6.23 | 4.21 | 2.12 | 8.33 |
| Poverty (Ref. ≥400% FPL)[e] | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| 0 - 199% FPL | 0.57 | 0.34 | 0.93 | 0.59 | 0.34 | 1.04 | 0.45 | 0.24 | 0.87 | 0.58 | 0.33 | 1.02 | 0.67 | 0.36 | 1.23 | 0.54 | 0.27 | 1.08 | 0.37 | 0.21 | 0.67 | 0.39 | 0.21 | 0.74 | 0.36 | 0.16 | 0.78 |
| 200 - 399% FPL | 1.13 | 0.67 | 1.89 | 1.16 | 0.69 | 1.96 | 1.25 | 0.75 | 2.10 | 1.30 | 0.73 | 2.32 | 1.47 | 0.82 | 2.63 | 1.53 | 0.85 | 2.76 | 0.92 | 0.50 | 1.70 | 1.00 | 0.53 | 1.90 | 1.17 | 0.64 | 2.15 |
| Sex | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Female vs. Male | 2.26 | 1.39 | 3.68 | 2.14 | 1.31 | 3.51 | 2.05 | 1.24 | 3.40 | 2.14 | 1.25 | 3.65 | 1.99 | 1.16 | 3.40 | 2.13 | 1.20 | 3.80 | 2.23 | 1.24 | 4.03 | 2.11 | 1.15 | 3.88 | 2.18 | 1.18 | 4.03 |
| Currently Insured | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Yes vs No | | | | 1.28 | 0.70 | 2.35 | | | | | | | 1.70 | 0.83 | 3.50 | | | | | | | 1.55 | 0.70 | 3.45 | | | |
| Obese | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| No vs Yes | | | | 0.82 | 0.53 | 1.27 | | | | | | | 0.91 | 0.56 | 1.49 | | | | | | | 0.81 | 0.47 | 1.39 | | | |
| Heart Disease | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Yes vs No | | | | 0.82 | 0.40 | 1.69 | | | | | | | 0.78 | 0.34 | 1.78 | | | | | | | 0.92 | 0.41 | 2.06 | | | |
| Smoker | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Ever vs Never | | | | 0.76 | 0.49 | 1.20 | | | | | | | 0.64 | 0.39 | 1.05 | | | | | | | 0.81 | 0.47 | 1.40 | | | |
| Urban/Rural (Ref. Town/Rural) | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Urban | | | | | | | 0.68 | 0.28 | 1.68 | | | | | | | 0.50 | 0.19 | 1.36 | | | | | | | 0.25 | 0.05 | 1.29 |
| Second City | | | | | | | 0.59 | 0.24 | 1.46 | | | | | | | 0.48 | 0.18 | 1.30 | | | | | | | 0.26 | 0.05 | 1.33 |
| Suburban | | | | | | | 0.59 | 0.22 | 1.57 | | | | | | | 0.52 | 0.17 | 1.58 | | | | | | | 0.21 | 0.04 | 1.17 |
| Usual Source of Care | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Yes vs No | | | | | | | 1.54 | 0.71 | 3.34 | | | | | | | 2.05 | 0.76 | 5.53 | | | | | | | 1.76 | 0.72 | 4.33 |
| Delay in Care | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Yes vs No | | | | | | | 0.89 | 0.56 | 1.41 | | | | | | | 0.71 | 0.41 | 1.22 | | | | | | | 0.47 | 0.25 | 0.89 |
| Onset of Asthma | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Adult vs Child | | | | | | | 0.77 | 0.48 | 1.22 | | | | | | | 0.75 | 0.45 | 1.25 | | | | | | | 0.72 | 0.43 | 1.20 |
| Daily Asthma Medication | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| No vs Yes | | | | | | | 0.44 | 0.28 | 0.69 | | | | | | | 0.44 | 0.26 | 0.74 | | | | | | | 0.49 | 0.28 | 0.86 |
| Asthma Management Plan | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| No vs Yes | | | | | | | 0.76 | 0.50 | 1.15 | | | | | | | 0.89 | 0.56 | 1.43 | | | | | | | 0.97 | 0.59 | 1.59 |
| Household Smoking | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| No vs Yes | | | | | | | 0.48 | 0.25 | 0.94 | | | | | | | 0.55 | 0.26 | 1.15 | | | | | | | 0.50 | 0.23 | 1.10 |
| Dog or Cat in Home | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| No vs Yes | | | | | | | 0.97 | 0.59 | 1.60 | | | | | | | 1.19 | 0.69 | 2.06 | | | | | | | 0.69 | 0.38 | 1.23 |
| Cockroaches | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Yes vs No | | | | | | | 2.10 | 1.16 | 3.79 | | | | | | | 1.46 | 0.76 | 2.82 | | | | | | | 1.46 | 0.73 | 2.91 |
| Housing Type (Ref. House) | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Duplex or Apartment | | | | | | | 0.48 | 0.19 | 1.25 | | | | | | | 0.77 | 0.23 | 2.63 | | | | | | | 1.64 | 0.37 | 7.29 |
| Mobile Home | | | | | | | 0.45 | 0.18 | 1.14 | | | | | | | 0.65 | 0.21 | 2.08 | | | | | | | 1.26 | 0.29 | 5.39 |
| Household Crowding | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| No vs Yes | | | | | | | 0.96 | 0.52 | 1.76 | | | | | | | 0.98 | 0.49 | 1.96 | | | | | | | 1.05 | 0.53 | 2.09 |
| Diabetes (Ref. Pre-Diabetes/ Borderline Diabetes) | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Yes | | | | | | | - | - | - | | | | | | | 1.02 | 0.06 | 16.36 | | | | | | | 0.88 | 0.06 | 12.12 |
| No | | | | | | | - | - | - | | | | | | | 1.58 | 0.11 | 21.85 | | | | | | | 1.13 | 0.10 | 13.04 |
| Walking | | | | | | | | | | | | | | | | | | | | | | | | | | | |
| Yes vs No | | | | | | | 1.08 | 0.68 | 1.72 | | | | | | | 1.18 | 0.69 | 2.00 | | | | | | | 1.11 | 0.60 | 2.06 |

[a]For CHIS 2003, current asthma is defined as reporting an asthma attack in the previous 12 months or answering yes to the question, "Do you still have asthma?"; only respondents who lived within 5 miles of an air monitoring station and lived in the same home or neighborhood for at least 9 months were included.

[b]Only variables with listed values are included for each model.

[c](Cases, Non-cases) Refers to the criteria pollutant noted in the column heading.

[d]Refers to the criteria pollutant noted in the column heading.

[e]FPL = Federal Poverty Level

Table 20 (Detailed). Associations between daily asthma medication and $PM_{2.5}$ adjusting for vulnerability characteristics among CHIS 2003 children with current asthma[a]

| | | $PM_{2.5}$ (per 5 μg/m$^3$) (132, 203)[b] | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | Model 1[c] | | | Model 2[c] | | | Model 3[c] | | |
| **Vulnerability Characteristic** | | OR | 95% C.I. | | OR | 95% C.I. | | OR | 95% C.I. | |
| 12-month pollutant average | | 1.20 | 0.88 | 1.63 | 1.20 | 0.87 | 1.64 | 1.28 | 0.93 | 1.76 |
| Age (Ref. 6-11) | | | | | | | | | | |
| | ≤ 6 | 0.89 | 0.40 | 1.99 | 0.89 | 0.40 | 1.99 | 1.04 | 0.46 | 2.36 |
| | 12 - 17 | 1.07 | 0.56 | 2.07 | 1.07 | 0.55 | 2.09 | 1.51 | 0.74 | 3.12 |
| Race (Ref. White) | | | | | | | | | | |
| | African American | 1.16 | 0.46 | 2.92 | 1.15 | 0.43 | 3.07 | 1.03 | 0.40 | 2.71 |
| | American Indian / Alaska Native | 1.11 | 0.15 | 8.44 | 1.11 | 0.14 | 8.82 | 0.84 | 0.11 | 6.26 |
| | Asian / Pacific Islander / Other | 0.92 | 0.34 | 2.49 | 0.97 | 0.36 | 2.60 | 0.86 | 0.32 | 2.29 |
| | Latino | 1.43 | 0.66 | 3.10 | 1.49 | 0.68 | 3.26 | 1.31 | 0.57 | 3.00 |
| Poverty (Ref. ≥400% FPL)[d] | | | | | | | | | | |
| | 0 - 199% FPL | 2.64 | 1.22 | 5.72 | 2.51 | 1.14 | 5.56 | 3.21 | 1.40 | 7.39 |
| | 200 - 399% FPL | 3.00 | 1.46 | 6.14 | 2.86 | 1.40 | 5.87 | 3.56 | 1.66 | 7.60 |
| Sex | | | | | | | | | | |
| | Female vs. Male | 0.96 | 0.52 | 1.75 | 0.96 | 0.52 | 1.76 | 0.92 | 0.51 | 1.69 |
| Household smoking | | | | | | | | | | |
| | No vs Yes | | | | 0.70 | 0.21 | 2.30 | | | |
| Dog or cat in home | | | | | | | | | | |
| | No vs Yes | | | | 1.03 | 0.53 | 1.99 | | | |
| Cockroaches | | | | | | | | | | |
| | No vs Yes | | | | 1.01 | 0.48 | 2.15 | | | |
| Currently insured | | | | | | | | | | |
| | Yes vs No | | | | 0.79 | 0.24 | 2.60 | | | |
| Rural/urban (Ref. Town and Rural) | | | | | | | | | | |
| | Urban | | | | | | | 0.44 | 0.08 | 2.38 |
| | Second City | | | | | | | 0.59 | 0.11 | 3.12 |
| | Suburban | | | | | | | 0.23 | 0.04 | 1.30 |
| Delay in care | | | | | | | | | | |
| | Yes vs No | | | | | | | 1.99 | 0.65 | 6.04 |
| Asthma management plan | | | | | | | | | | |
| | No vs Yes | | | | | | | 0.38 | 0.19 | 0.75 |
| Housing type (Ref. Mobile Home) | | | | | | | | | | |
| | Duplex or Apartment | | | | | | | 9.75 | 0.94 | 101.42 |
| | Mobile Home | | | | | | | 11.88 | 1.20 | 117.91 |
| Household crowding | | | | | | | | | | |
| | No vs Yes | | | | | | | 1.13 | 0.57 | 2.26 |

[a]For CHIS 2003, current asthma is defined as reporting an asthma attack in the previous 12 months or answering yes to the question, "Do you still have asthma?"; only respondents who lived within 5 miles of an air monitoring station and lived in the same home or neighborhood for at least 9 months were included.
[b](Cases, Non-cases)
[c]Only variables with listed values are included for each model.
[d]FPL = Federal Poverty Level

Table 24. Interaction between mean $NO_2$ annual exposure and Federal Poverty Level (FPL) or race/ethnicity on odds ratios (OR (95% CI)) of various asthma outcomes in CHIS 2003 children and adults with current asthma[a,b]

| | | ED Visits (Children) | | | Missed ≥ 2 Work Days (Adults) | | | Daily/Weekly Symptoms (Adults) | | | Daily/Weekly Symptoms (Children) | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Interaction | | OR | 95% C.I. | | OR | 95% C.I. | | OR | 95% C.I. | | OR | 95% C.I. | |
| $NO_2$ (10 ppb) by Household FPL | | | | | | | | | | | | | |
| | 0-199% FPL | 2.14 | 1.22 | 3.74 | | | | | | | | | |
| | 200-399% FPL | 0.77 | 0.41 | 1.45 | | | | | | | | | |
| | ≥400% FPL† | 0.66 | 0.33 | 1.34 | | | | | | | | | |
| | | | | | | | | | | | | | |
| $NO_2$ (10 ppb) by Race | | | | | | | | | | | | | |
| | Latino | | | | 1.20 | 0.72 | 2.03 | 0.63 | 0.38 | 1.03 | 1.18 | 0.57 | 2.45 |
| | American Indian / Alaska Native | | | | 1.13 | 0.23 | 5.53 | 0.33 | 0.07 | 1.52 | - | - | - |
| | Asian / Pacific Islander / Other | | | | 2.72 | 1.32 | 5.61 | 1.32 | 0.74 | 2.38 | 4.52 | 0.57 | 35.52 |
| | African American | | | | 1.86 | 0.87 | 3.96 | 2.21 | 1.13 | 4.33 | 0.95 | 0.27 | 3.37 |
| | White† | | | | 0.73 | 0.47 | 1.12 | 1.02 | 0.81 | 1.29 | 0.44 | 0.21 | 0.93 |

[a]For CHIS 2003, current asthma is defined as reporting an asthma attack in the previous 12 months or answering yes to the question, "Do you still have asthma?"; only respondents who lived within 5 miles of an air monitoring station and lived in the same home or neighborhood for at least 9 months were included.
[b]Adjusted for age, race, poverty level, and sex.
†Reference group

Table 25. Interaction between mean $PM_{10}$ annual exposure and race/ethnicity on odds ratios (OR (95% CI)) of various asthma outcomes in CHIS 2003 children and adults with current asthma[a,b]

| | | Daily Asthma Medication | | | Daily/Weekly Symptoms | | |
|---|---|---|---|---|---|---|---|
| Interaction | | OR | 95% C.I. | | OR | 95% C.I. | |
| $PM_{10}$ (10 $\mu g/m^3$) by Race | | | | | | | |
| | Latino | 1.33 | 0.87 | 2.04 | 0.51 | 0.25 | 1.03 |
| | American Indian / Alaska Native | 0.43 | 0.12 | 1.50 | 0.98 | 0.49 | 1.94 |
| | Asian / Pacific Islander / Other | 0.36 | 0.16 | 0.80 | 2.07 | 0.93 | 4.60 |
| | African American | 1.40 | 0.54 | 3.63 | 2.48 | 0.89 | 6.91 |
| | White† | 0.74 | 0.51 | 1.08 | 0.70 | 0.41 | 1.17 |

[a]For CHIS 2003, current asthma is defined as reporting an asthma attack in the previous 12 months or answering yes to the question, "Do you still have asthma?"; only respondents who lived within 5 miles of an air monitoring station and lived in the same home or neighborhood for at least 9 months were included.
[b]Adjusted for age, race, poverty level, and sex.
†Reference group

Table 26. Weighted distributions of annual pollutant averages, exceedance days, and traffic density (within 750 feet) for CHIS 2003 adults and children with asthma-like symptoms[a]

| | Adults | | | | | | | | Children | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | n | Missing | Min | Max | Mean | Std Error | Median | 95th Percentile | n | Missing | Min | Max | Mean | Std Error | Median | 95th Percentile |
| **Pollutant Averages** | | | | | | | | | | | | | | | | |
| $O_3$ (ppb) | 2044 | 298 | 22.9 | 65.7 | 41.7 | 0.25 | 40.1 | 55.0 | 441 | 52 | 22.9 | 63.5 | 42.0 | 0.56 | 39.7 | 55.7 |
| $PM_{10}$ ($\mu g/m^3$) | 1614 | 285 | 7.9 | 82.8 | 30.1 | 0.34 | 29.2 | 47.7 | 348 | 54 | 12.8 | 82.8 | 29.9 | 0.72 | 28.9 | 45.1 |
| $PM_{2.5}$ ($\mu g/m^3$) | 1253 | 862 | 4.1 | 26.9 | 16.5 | 0.17 | 17.0 | 23.4 | 274 | 167 | 6.6 | 26.2 | 16.5 | 0.36 | 16.9 | 23.4 |
| $NO_2$ (ppb) | 1687 | 616 | 1.4 | 36.1 | 22.0 | 0.22 | 21.4 | 34.9 | 364 | 107 | 5.0 | 36.0 | 22.3 | 0.50 | 21.3 | 35.0 |
| **Exceedance (in days)** | | | | | | | | | | | | | | | | |
| $O_3$ 1-Hr (State) | 2,045 | 290 | 0.0 | 131.0 | 23.6 | 0.84 | 10.3 | 77.3 | 442 | 50 | 0.0 | 122.0 | 24.6 | 1.82 | 11.3 | 76.7 |
| $O_3$ 8-Hr (Federal) | 2,044 | 298 | 0.0 | 130.0 | 17.7 | 0.73 | 4.3 | 64.5 | 441 | 52 | 0.0 | 114.0 | 18.5 | 1.64 | 4.6 | 65.9 |
| $O_3$ 8-Hr (State) | 2,044 | 298 | 0.0 | 160.0 | 32.1 | 1.05 | 16.7 | 97.7 | 441 | 52 | 0.0 | 153.0 | 33.9 | 2.40 | 17.2 | 99.6 |
| $PM_{10}$ (Federal) | 1,614 | 285 | 0.0 | 4.0 | 0.1 | 0.02 | 0.0 | 0.6 | 348 | 54 | 0.0 | 4.0 | 0.1 | 0.04 | 0.0 | 0.8 |
| $PM_{10}$ (State) | 1,614 | 285 | 0.0 | 66.0 | 8.2 | 0.44 | 3.2 | 26.8 | 348 | 54 | 0.0 | 64.0 | 7.9 | 0.99 | 3.8 | 26.2 |
| $PM_{2.5}$ (Federal) | 1,253 | 862 | 0.0 | 53.6 | 16.1 | 0.45 | 13.6 | 45.0 | 274 | 167 | 0.0 | 53.6 | 16.6 | 1.23 | 11.9 | 46.1 |
| **Traffic Density (VMT/day/meter$^2$)** | | | | | | | | | | | | | | | | |
| 750 feet buffer[b] | 3,624 | 5 | 1.0 | 745.3 | 65.8 | 1.81 | 45.6 | 216.2 | 831 | 2 | 0.5 | 637.2 | 64.1 | 3.96 | 43.4 | 226.1 |

[a]For CHIS 2003, having asthma-like symptoms is defined as reporting wheezing or whistling in the previous 12 months without an asthma diagnosis; only respondents who lived within 5 miles of an air monitoring station and lived in the same home or neighborhood for at least 9 months were included.
[b]Based on imputed Tele Atlas traffic data.

Table 27. Frequencies for distance to roadway measures for CHIS 2003 adults and children with asthma-like symptoms[a]

| | Adults (≥18 years) | | Children (< 18 years) | |
|---|---|---|---|---|
| Roadway Measure[b] | n | % (Weighted) | n | % (Weighted) |
| <300 m from a State Highway | 189 | 6.1 | 46 | 5.3 |
| <300 m from an Interstate Highway | 344 | 10.4 | 81 | 10.8 |
| <50 m from a Major Road | 717 | 19.7 | 150 | 17.8 |
| <50 m from a Minor Road | 3,253 | 89.8 | 752 | 90.7 |

[a]For CHIS 2003, having asthma-like symptoms is defined as reporting wheezing or whistling in the previous 12 months without an asthma diagnosis; only respondents who lived in the same home or neighborhood for at least 9 months, and geocoded by address or nearest cross streets were included
[b]Based on Tele Atlas Dynamap 2000 roadway map.

Table 28. Correlations between annual average air pollutant concentrations, exceedance measures, distance to roadways and traffic density among CHIS 2003 respondents with asthma-like symptoms[a]

| | $O_3$ (ppb) | $PM_{10}$ ($\mu g/m^3$) | $PM_{2.5}$ ($\mu g/m^3$) | $NO_2$ (ppb) | Traffic Density (750 ft buffer) (VMT/day/meters$^2$)[b] | Distance to State Highway (meters)[b] | Distance to Interstate (meters)[b] | Distance to Major Roads (meters)[b] | Distance to Minor Roads (meters)[b] | $O_3$ 1-Hr (State) (days) | $O_3$ 8-Hr (Federal) (days) | $O_3$ 8-Hr (State) (days) | $PM_{10}$ (Federal) (days) | $PM_{10}$ (State) (days) | $PM_{2.5}$ (Federal) (days) |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| $O_3$ (ppb) | 1 | | | | | | | | | | | | | | |
| $PM_{10}$ ($\mu g/m^3$) | 0.48 | 1 | | | | | | | | | | | | | |
| $PM_{2.5}$ ($\mu g/m^3$) | 0.34 | 0.74 | 1 | | | | | | | | | | | | |
| $NO_2$ (ppb) | -0.02 | 0.50 | 0.71 | 1 | | | | | | | | | | | |
| Traffic Density (750 ft buffer) (VMT/day/meters$^2$)[b] | -0.11 | 0.00 | 0.00 | 0.05 | 1 | | | | | | | | | | |
| Distance to State Highway(meters)[b] | 0.20 | 0.01 | -0.08 | -0.13 | -0.01 | 1 | | | | | | | | | |
| Distance to Interstate (meters)[b] | 0.22 | 0.03 | -0.09 | -0.21 | -0.25 | 0.02 | 1 | | | | | | | | |
| Distance to Major Roads (meters)[b] | 0.18 | -0.01 | -0.09 | -0.09 | -0.16 | -0.05 | 0.19 | 1 | | | | | | | |
| Distance to Minor Roads (meters)[b] | 0.07 | -0.01 | 0.01 | -0.05 | -0.01 | -0.02 | 0.17 | -0.02 | 1 | | | | | | |
| $O_3$ 1-Hr (State) (days) | 0.82 | 0.57 | 0.44 | 0.21 | -0.10 | 0.22 | 0.14 | 0.10 | 0.08 | 1 | | | | | |
| $O_3$ 8-Hr (Federal) (days) | 0.83 | 0.54 | 0.39 | 0.09 | -0.11 | 0.21 | 0.19 | 0.11 | 0.08 | 0.98 | 1 | | | | |
| $O_3$ 8-Hr (State) (days) | 0.88 | 0.55 | 0.39 | 0.08 | -0.12 | 0.22 | 0.23 | 0.13 | 0.08 | 0.95 | 0.97 | 1 | | | |
| $PM_{10}$ (Federal) (days) | 0.20 | 0.45 | 0.08 | 0.00 | -0.02 | -0.03 | 0.11 | 0.01 | -0.02 | 0.17 | 0.17 | 0.16 | 1 | | |
| $PM_{10}$ (State) (days) | 0.45 | 0.83 | 0.48 | 0.20 | -0.04 | 0.04 | 0.08 | 0.00 | 0.00 | 0.57 | 0.57 | 0.55 | 0.64 | 1 | |
| $PM_{2.5}$ (Federal) (days) | 0.15 | 0.46 | 0.71 | 0.47 | 0.04 | -0.02 | -0.09 | -0.10 | -0.02 | 0.17 | 0.13 | 0.15 | -0.03 | 0.27 | 1 |

[a]For CHIS 2003, having asthma-like symptoms is defined as reporting wheezing or whistling in the previous 12 months without an asthma diagnosis; only respondents who lived within 5 miles of an air monitoring station, lived in the same home or neighborhood for at least 9 months, and geocoded by address or nearest cross streets were included.
[b]Based on imputed Tele Atlas traffic data or Tele Atlas Dynamap 2000 roadway map.

Table 30. Characteristics of CHIS 2003 children and adults with asthma-like symptoms[a]

| | | Adults | | | Children | | |
|---|---|---|---|---|---|---|---|
| | | n | Pop. N | % (wtd.) | n | Pop. N | % (wtd.) |
| Sex | | | | | | | |
| | Male | 1,815 | 1,201,580 | 50.5 | 501 | 382,240 | 54.9 |
| | Female | 2,314 | 1,176,605 | 49.5 | 450 | 314,279 | 45.1 |
| Age (yr) | | | | | | | |
| | 0-5 | - | - | - | 388 | 286,760 | 41.2 |
| | 6-11 | - | - | - | 280 | 198,531 | 28.5 |
| | 12-17 | - | - | - | 283 | 211,229 | 30.3 |
| | 18-34 | 781 | 672,761 | 28.3 | - | - | - |
| | 35-64 | 2,502 | 1,340,825 | 56.4 | - | - | - |
| | 65+ | 846 | 364,599 | 15.3 | - | - | - |
| Race/Ethnicity | | | | | | | |
| | Latino | 615 | 547,086 | 23.0 | 270 | 238,571 | 34.3 |
| | American Indian/Alaska Native | 98 | 54,492 | 2.3 | 14 | 7,356 | 1.1 |
| | Asian/Other | 346 | 225,489 | 9.5 | 111 | 102,324 | 14.7 |
| | African American | 264 | 160,225 | 6.7 | 64 | 51,602 | 7.4 |
| | White | 2,806 | 1,390,893 | 58.5 | 492 | 296,666 | 42.6 |
| Education[b] | | | | | | | |
| | <25 years old | 263 | 244,568 | 10.3 | | | |
| | High School Education or Less | 1,614 | 1,030,693 | 43.3 | 381 | 297,992 | 42.8 |
| | College or Vocational School | 1,803 | 897,931 | 37.8 | 447 | 322,239 | 46.3 |
| | Graduate School | 449 | 204,993 | 8.6 | 123 | 76,289 | 11.0 |
| Work Status | | | | | | | |
| | Employed | 2,570 | 1,587,771 | 67.5 | - | - | - |
| | Unemployed | 1,530 | 764,934 | 32.5 | - | - | - |
| Federal Poverty Level | | | | | | | |
| | 0-199% | 1,420 | 926,384 | 39.0 | 344 | 295,620 | 42.4 |
| | 200-399% | 1,072 | 587,447 | 24.7 | 242 | 164,372 | 23.6 |
| | ≥400% | 1,637 | 864,354 | 36.4 | 365 | 236,527 | 34.0 |
| Insurance Status | | | | | | | |
| | Uninsured All/Part of the Year | 777 | 582,943 | 24.5 | 79 | 70,052 | 10.1 |
| | Insured All of the Year | 3,352 | 1,795,242 | 75.5 | 872 | 626,468 | 89.9 |
| Usual Source of Care | | | | | | | |
| | Yes | 3,611 | 1,993,423 | 83.8 | 871 | 622,911 | 89.4 |
| | No | 518 | 384,761 | 16.2 | 80 | 73,608 | 10.6 |
| Delay in Needed Care in Last 12 Months (CHIS 2003) | | | | | | | |
| | Yes | 1,060 | 612,860 | 25.8 | 125 | 85,653 | 12.3 |
| | No | 3,069 | 1,765,325 | 74.2 | 826 | 610,866 | 87.7 |
| Number of Doctor Visits in the Past Year | | | | | | | |
| | 0-1 | 1,161 | 759,972 | 33.8 | 209 | 161,582 | 25.0 |
| | 2-5 | 1,633 | 939,624 | 41.8 | 542 | 388,566 | 60.0 |
| | 6+ | 1,054 | 550,124 | 24.5 | 140 | 97,062 | 15.0 |
| Self-reported Health Status | | | | | | | |
| | Good/Very Good/Excellent | 2,753 | 1,525,954 | 64.2 | 845 | 602,278 | 86.5 |
| | Poor/Fair | 1,376 | 852,231 | 35.8 | 106 | 94,241 | 13.5 |
| Heart Disease (Adults) | | | | | | | |
| | Yes | 571 | 268,072 | 11.3 | - | - | - |
| | No | 3,558 | 2,110,113 | 88.7 | - | - | - |

[a]For CHIS 2003, having asthma-like symptoms is defined as reporting wheezing or whistling in the previous 12 months without an asthma diagnosis; only respondents who lived in the same home or neighborhood for at least 9 months were included.
[b]Representative of educational attainment level of the adult respondent or adult responding on behalf of the child.

Table 30. Characteristics of CHIS 2003 children and adults with asthma-like symptoms[a] (continued)

| | | Adults | | | Children | | |
|---|---|---|---|---|---|---|---|
| | | n | Pop. N | % (wtd.) | n | Pop. N | % (wtd.) |
| Congestive Heart Failure (Adult) | | | | | | | |
| | Yes | 112 | 53,894 | 41.3 | - | - | - |
| | No | 181 | 76,690 | 58.7 | - | - | - |
| Diabetes | | | | | | | |
| | Yes | 346 | 182,636 | 7.7 | - | - | - |
| | No | 3,717 | 2,156,359 | 90.7 | - | - | - |
| | Borderline | 66 | 39,189 | 1.7 | - | - | - |
| Body Mass Index (Adult) | | | | | | | |
| | Underweight/Normal | 1,534 | 891,647 | 37.5 | - | - | - |
| | Overweight/Obese | 2,595 | 1,486,538 | 62.5 | - | - | - |
| Body Mass Index (Teen) | | | | | | | |
| | Underweight/Normal | - | - | - | 200 | 157,263 | 74.5 |
| | Overweight/Obese | - | - | - | 83 | 53,966 | 25.5 |
| Smoking Status (Adult) | | | | | | | |
| | Current/Previous Smoker | 2,542 | 1,396,319 | 59.3 | - | - | - |
| | Never Smoker | 1,558 | 956,386 | 40.7 | - | - | - |
| Smokers in the Home | | | | | | | |
| | Yes | 708 | 407,822 | 17.1 | 67 | 49,370 | 7.1 |
| | No | 3,421 | 1,970,363 | 82.9 | 884 | 647,150 | 92.9 |
| Walking for Transportation or Leisure | | | | | | | |
| | Yes | 2,745 | 1,611,122 | 68.9 | - | - | - |
| | No | 1,317 | 727,414 | 31.1 | - | - | - |
| Dogs/Cats in the Home (CHIS 2003) | | | | | | | |
| | Yes | 1,945 | 1,030,316 | 43.3 | 391 | 266,096 | 38.2 |
| | No | 2,184 | 1,347,869 | 56.7 | 560 | 430,423 | 61.8 |
| Cockroaches in the Home (CHIS 2003) | | | | | | | |
| | Yes | 604 | 482,364 | 20.3 | 179 | 176,233 | 25.3 |
| | No | 3,525 | 1,895,821 | 79.7 | 772 | 520,286 | 74.7 |
| Urban/Rural | | | | | | | |
| | Urban | 1,619 | 1,039,998 | 43.7 | 358 | 295,795 | 42.5 |
| | 2nd city | 1,146 | 623,796 | 26.2 | 272 | 174,321 | 25.0 |
| | Suburban | 547 | 387,498 | 16.3 | 162 | 141,262 | 20.3 |
| | Town or Rural | 817 | 326,893 | 13.7 | 159 | 85,141 | 12.2 |
| Time at Current Address/Neighborhood | | | | | | | |
| | 9 months-<1 yr | 62 | 41,823 | 1.8 | 15 | 15,609 | 2.2 |
| | 1-<3 yr | 651 | 424,912 | 17.9 | 302 | 249,010 | 35.8 |
| | 3+ yr | 3,416 | 1,911,450 | 80.4 | 634 | 431,900 | 62.0 |
| Household Crowding (CHIS 2003) | | | | | | | |
| | Yes | 556 | 559,918 | 23.5 | 270 | 259,513 | 37.3 |
| | No | 3,573 | 1,818,266 | 76.5 | 681 | 437,007 | 62.7 |
| Housing Type | | | | | | | |
| | House | 2,710 | 1,500,907 | 63.1 | 691 | 478,939 | 68.8 |
| | Apartment, Duplex, or Mobile Home | 1,419 | 877,278 | 36.9 | 260 | 217,580 | 31.2 |

[a]For CHIS 2003, having asthma-like symptoms is defined as reporting wheezing or whistling in the previous 12 months without an asthma diagnosis; only respondents who lived in the same home or neighborhood for at least 9 months were included.
[b]Representative of educational attainment level of the adult respondent or adult responding on behalf of the child.

Table 31. Disparities in weighted mean annual pollutant concentrations by various demographic characteristics in CHIS 2003 children and adults with asthma-like symptoms using bivariate analysis[a]

| | | $NO_2$ annual average (ppb) | | $O_3$ annual average (ppb) | | $PM_{10}$ annual average ($\mu g/m^3$) | | $PM_{2.5}$ annual average ($\mu g/m^3$) | |
|---|---|---|---|---|---|---|---|---|---|
| Demographics | | Adults mean | Children mean | Adults mean | Children mean | Adults mean | Children mean | Adults mean | Children mean |
| Household Federal Poverty Level (FPL) | 0 - 199 % FPL | 23.3*** | 24.1 | 41.7 | 42.0 | 32.4*** | 30.9 | 17.4*** | 17.1* |
| | 200 - 399 % FPL | 21.1 | 20.8*** | 42.0 | 41.9 | 32.4* | 29.5 | 16.4** | 16.1 |
| | ≥400% FPL† | 21.1 | 20.3 | 41.3 | 42.3 | 29.5 | 28.5 | 15.3 | 15.4 |
| Race/ethnicity | Latino | 24.3*** | 24.8*** | 41.4 | 40.6 | 33.7*** | 32.6* | 18.2*** | 17.9** |
| | American Indian / Alaska | 19.2 | 19.0 | 41.0 | 41.6 | 28.4 | 29.6 | 14.3 | 16.5 |
| | Asian / Pacific Islander / Other | 22.4* | 21.3 | 41.0 | 44.4 | 28.1 | 27.5 | 15.1 | 14.6 |
| | African American | 23.3*** | 24.0* | 38.9*** | 40.4 | 32.8** | 28.0 | 17.3** | 15.4 |
| | White† | 20.5 | 19.3 | 42.4 | 43.2 | 28.6 | 28.6 | 15.7 | 15.5 |
| Urban/Rural | Urban† | 24.7 | 25.6 | 38.8 | 39.0 | 29.2 | 29.0 | 16.6 | 16.8 |
| | Second City | 18.4*** | 18.0*** | 44.2*** | 44.2*** | 30.7* | 28.9 | 15.9 | 14.6** |
| | Suburban | 19.8*** | 19.9*** | 44.7*** | 45.3*** | 33.0*** | 33.9* | 17.7* | 17.8 |
| | Town/Rural | 11.0*** | 18.8** | 48.1*** | 47.7*** | 26.0 | 28.6 | 10.1*** | 9.2*** |
| Sex | Male | 21.9 | 22.0 | 41.7 | 42.7 | 29.3* | 29.5 | 16.2 | 16.5 |
| | Female† | 22.2 | 22.8 | 41.7 | 41.2 | 30.7 | 30.3 | 16.8 | 16.5 |
| Age (in years) | 0-5 | | 21.7 | | 41.3 | | 28.9 | | 16.0 |
| | 6-11 | | 23.7 | | 43.2 | | 31.8 | | 17.6 |
| | 12-17† | | 22.1 | | 42.1 | | 29.5 | | 16.1 |
| | 18-34 | 22.2 | | 41.6 | | 30.3 | | 16.8 | |
| | 35 - 64 | 22.0 | | 41.8 | | 30.1 | | 16.4 | |
| | 65 and above† | 21.8 | | 41.4 | | 29.7 | | 16.3 | |

[a]For CHIS 2003, having asthma-like symptoms is defined as reporting wheezing or whistling in the previous 12 months without an asthma diagnosis; only respondents who lived within 5 miles of an air monitoring station and lived in the same home or neighborhood for at least 9 months were included.

†Reference Group

*p < 0.05, ** p < 0.01, *** p < 0.001

Table 32. Disparities in weighted prevalence of asthma-like symptoms by various demographic characteristics in CHIS 2003 children and adults using bivariate analysis[a]

| | | Wheeze[b] | | Missed ≥ 2 school/work days due to wheezing[c] | | ≥2 wheeze attacks[c] | | Sought medical help for breathing problem | |
|---|---|---|---|---|---|---|---|---|---|
| | | Adults | Children | Adults | Children | Adults | Children | Adults | Children |
| **Demographics** | | % | % | % | % | % | % | % | % |
| Household Federal Poverty Level (FPL) | 0 - 199 % FPL | 13.4*** | 9.4 | 17.3 | 45.2 | 60.4 | 54.8 | 41.9 | 65.1 |
| | 200 - 399 % FPL | 11.7** | 9.2 | 15.4 | 47.9 | 63.7 | 56.3 | 40.0 | 66.6 |
| | ≥400% FPL† | 9.8 | 11.2 | 17.3 | 44.0 | 62.7 | 44.3 | 41.6 | 65.1 |
| Race/ethnicity | Latino | 9.8*** | 9.0 | 24.5*** | 50.7 | 51.7*** | 48.6 | 46.1* | 69.1 |
| | American Indian / Alaska Native | 26.4*** | € | € | € | 74.9 | € | 36.0 | € |
| | Asian / Pacific Islander / Other | 7.2*** | 10.6 | 11.6 | 29.6* | 67.7 | 75.5*** | 43.0 | 56.6 |
| | African American | 13.2 | 10.3 | 17.6 | 52.5 | 62.4 | € | 49.0* | 49.7 |
| | White† | 13.1 | 10.4 | 14.3 | 45.5 | 64.8 | 48.1 | 38.5 | 68.2 |
| Urban/Rural | Urban† | 11.0 | 9.9 | 16.8 | 43.3 | 62.2 | 48.9 | 43.3 | 65.6 |
| | Second City | 12.2* | 9.4 | 18.9 | 51.4 | 61.8 | 62.2* | 41.6 | 66.9 |
| | Suburban | 10.0 | 10.3 | 14.4 | 45.9 | 63.1 | 48.2 | 39.5 | 68.9 |
| | Town/Rural | 14.4*** | 10.1 | 16.1 | 40.9 | 60.9 | 47.8 | 36.5* | 56.6 |
| Sex | Male | 11.6 | 10.9* | 13.6*** | 42.7 | 62.9 | 51.7 | 32.8*** | 65.7 |
| | Female† | 11.3 | 8.9 | 20.3 | 48.6 | 61.1 | 51.8 | 50.3 | 65.2 |
| Age | 0-5 | | 14.0*** | | 44.8 | | 46.7* | | 82.8*** |
| | 6-11 | | 8.0 | | 46.4 | | 59.1 | | 73.6*** |
| | 12-17† | | 8.4 | | - | | - | | 34.2 |
| | 18-34 | 10.7 | | 18.3 | | 59.2 | | 35.5*** | |
| | 35 - 64 | 12.0 | | 17.0 | | 63.1 | | 42.7 | |
| | 65 and above† | 11.1 | | € | | 64.0 | | 48.6 | |

[a]For CHIS 2003, having asthma-like symptoms is defined as reporting wheezing or whistling in the previous 12 months without an asthma diagnosis; only respondents who lived in the same home or neighborhood for at least 9 months were included.

[b]Respondents without an asthma diagnosis who lived within 5 miles of an air monitoring station and had lived in the same home or neighborhood for at least 9 months were included. Respondents with wheezing symptoms were designated as having asthma-like symptoms.

[c]Data not collected for teen respondents.

†Reference Group

€ Unstable values (CV > 30%)

*p < 0.05, ** p < 0.01, *** p < 0.001

Table 33 (Detailed). Associations (OR (95% CI)) for 12-month pollutant averages and asthma-like outcomes in CHIS 2003 adults and children with asthma-like symptoms[a]

| | Wheeze[b] | | | | Missed ≥2 School/Work Days Due to Wheezing[c] | | | | ≥2 Wheeze Attacks[c] | | | | Sought Medical Help for Breathing Problem | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Pollutant | Cases | Non-Cases | OR[d] | 95% C.I. | Cases | Non-Cases | OR[d] | 95% C.I. | Cases | Non-Cases | OR[d] | 95% C.I. | Cases | Non-Cases | OR[d] | 95% C.I. |
| Adults | | | | | | | | | | | | | | | | |
| $O_3$ (per 10 ppb) | 2,044 | 14,824 | **1.09** | [1.01, 1.18] | 258 | 1,296 | 0.91 | [0.74, 1.12] | 1,121 | 657 | 1.11 | [0.94, 1.30] | 739 | 1,039 | 1.06 | [0.91, 1.24] |
| $PM_{10}$ (per 10 µg/m$^3$) | 1,614 | 11,229 | **1.09** | [1.01, 1.18] | 198 | 1,027 | 0.84 | [0.66, 1.06] | 912 | 488 | 1.10 | [0.94, 1.29] | 577 | 823 | 1.09 | [0.94, 1.27] |
| $PM_{2.5}$ (per 5 µg/m$^3$) | 1,253 | 9,331 | 1.07 | [0.97, 1.17] | 162 | 797 | 0.88 | [0.69, 1.12] | 691 | 396 | 1.00 | [0.83, 1.19] | 462 | 625 | 1.06 | [0.89, 1.26] |
| $NO_2$ (per 10 ppb) | 1,687 | 12,534 | 0.93 | [0.86, 1.02] | 222 | 1,061 | 0.73 | [0.56, 0.96] | 905 | 566 | 1.09 | [0.91, 1.30] | 622 | 849 | 0.93 | [0.78, 1.11] |
| Children | | | | | | | | | | | | | | | | |
| $O_3$ (per 10 ppb) | 441 | 4,109 | 1.09 | [0.92, 1.29] | 143 | 167 | 0.96 | [0.67, 1.36] | 157 | 153 | 1.29 | [0.92, 1.81] | 295 | 146 | 0.99 | [0.72, 1.36] |
| $PM_{10}$ (per 10 µg/m$^3$) | 348 | 3,102 | 0.95 | [0.80, 1.13] | 112 | 129 | 0.75 | [0.53, 1.07] | 113 | 128 | 1.15 | [0.82, 1.60] | 223 | 125 | 0.80 | [0.58, 1.10] |
| $PM_{2.5}$ (per 5 µg/m$^3$) | 274 | 2,601 | 0.97 | [0.80, 1.18] | 87 | 107 | 0.90 | [0.59, 1.37] | 97 | 97 | 1.25 | [0.86, 1.82] | 172 | 102 | 1.16 | [0.73, 1.83] |
| $NO_2$ (per 10 ppb) | 364 | 3,504 | 0.94 | [0.79, 1.11] | 113 | 146 | 1.25 | [0.82, 1.90] | 134 | 125 | 1.02 | [0.69, 1.50] | 243 | 121 | 1.33 | [0.90, 1.98] |

[a]For CHIS 2003, having asthma-like symptoms is defined as reporting wheezing or whistling in the previous 12 months without an asthma diagnosis; only respondents who lived within 5 miles of an air monitoring station and lived in the same home or neighborhood for at least 9 months were included.
[b]Respondents without an asthma diagnosis who lived within 5 miles of an air monitoring station and had lived in the same home or neighborhood for at least 9 months were included. Respondents with wheezing symptoms were designated as having asthma-like symptoms.
[c]Data not collected for teen respondents.
[d]Adjusted for age, race, poverty level, and sex.

Table 34. Associations (OR (95% CI)) for annual days exceeding air pollution standards and asthma-like outcomes comparing quartiles in CHIS 2003 adults and children with asthma-like symptoms[a]

| | Wheeze[b] | | | | Missed ≥2 School/Work Days Due to Wheezing[c] | | | | ≥2 Wheeze Attacks[c] | | | | Sought Medical Help for Breathing Problem | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Exceedances in days | Cases | Non-Cases | OR[d] | 95% C.I. | Cases | Non-Cases | OR[d] | 95% C.I. | Cases | Non-Cases | OR[d] | 95% C.I. | Cases | Non-Cases | OR[d] | 95% C.I. |
| Adults | | | | | | | | | | | | | | | | |
| $O_3$ 1-Hr (State) - Ref. < 0.8 days | | | | | | | | | | | | | | | | |
| 0.8 days < 8.7 days | 489 | 3,892 | 0.84 | [0.70, 1.00] | 63 | 306 | 1.10 | [0.65, 1.84] | 257 | 167 | 1.13 | [0.78, 1.63] | 189 | 235 | 0.98 | [0.68, 1.42] |
| 8.7 days - < 36.7 days | 579 | 4,215 | 0.835 | [0.70, 0.99] | 76 | 369 | 0.777 | [0.48, 1.27] | 341 | 169 | 1.32 | [0.92, 1.89] | 193 | 317 | 0.74 | [0.52, 1.05] |
| ≥ 36.7 days | 507 | 3,577 | 1.01 | [0.85, 1.21] | 56 | 334 | 0.73 | [0.44, 1.23] | 284 | 157 | 1.29 | [0.89, 1.86] | 181 | 260 | 0.90 | [0.63, 1.30] |
| $O_3$ 8-Hr (State) - Ref. < 1.9 days | | | | | | | | | | | | | | | | |
| 1.9 days - <14.3 days | 479 | 3,752 | 0.88 | [0.74, 1.05] | 63 | 297 | 0.97 | [0.58, 1.62] | 249 | 167 | 1.16 | [0.81, 1.67] | 181 | 235 | 0.90 | [0.62, 1.28] |
| 14.3 days - < 51.2 days | 539 | 4,110 | 0.91 | [0.77, 1.08] | 76 | 336 | 0.86 | [0.52, 1.40] | 311 | 152 | 1.42 | [0.99, 2.04] | 180 | 283 | 0.78 | [0.55, 1.11] |
| ≥ 51.2 days | 562 | 3,526 | 1.16 | [0.97, 1.38] | 63 | 368 | 0.73 | [0.43, 1.24] | 320 | 174 | 1.30 | [0.91, 1.87] | 204 | 290 | 0.96 | [0.67, 1.38] |
| $PM_{10}$ (State) - Ref. < 1.6 days | | | | | | | | | | | | | | | | |
| 1.6 days - < 3.5 days | 383 | 2,850 | 1.01 | [0.84, 1.22] | 53 | 250 | 1.10 | [0.62, 1.94] | 218 | 126 | 0.96 | [0.64, 1.44] | 142 | 202 | 0.96 | [0.64, 1.43] |
| 3.5 days - <6.6 days | 301 | 2,405 | 0.90 | [0.73, 1.11] | 40 | 193 | 0.61 | [0.33, 1.16] | 177 | 83 | 1.07 | [0.69, 1.67] | 111 | 149 | 0.87 | [0.56, 1.35] |
| ≥ 6.6 days | 490 | 2,864 | **1.26** | [1.04, 1.53] | 60 | 321 | 0.60 | [0.33, 1.09] | 279 | 146 | 0.99 | [0.65, 1.49] | 182 | 243 | 1.20 | [0.80, 1.80] |
| $PM_{2.5}$ (Federal) - Ref. < 4.8 days | | | | | | | | | | | | | | | | |
| 4.8 days - < 12.0 days | 258 | 1,897 | 1.15 | [0.91, 1.46] | 29 | 167 | 0.81 | [0.41, 1.62] | 151 | 76 | 1.13 | [0.69, 1.87] | 93 | 134 | 1.06 | [0.65, 1.73] |
| 12.0 days - < 24.9 days | 391 | 2,839 | 1.16 | [0.93, 1.44] | 46 | 253 | 0.69 | [0.38, 1.28] | 198 | 137 | 0.78 | [0.49, 1.24] | 145 | 190 | 1.12 | [0.73, 1.73] |
| ≥ 24.9 days | 301 | 2,263 | 1.07 | [0.85, 1.35] | 48 | 194 | 0.91 | [0.49, 1.71] | 177 | 90 | 1.09 | [0.67, 1.76] | 114 | 153 | 1.09 | [0.70, 1.72] |
| Children | | | | | | | | | | | | | | | | |
| $O_3$ 1-Hr (State) - Ref. < 0.8 days | | | | | | | | | | | | | | | | |
| 0.8 days < 8.7 days | 114 | 1,056 | 1.19 | [0.81, 1.74] | 35 | 42 | 1.24 | [0.50, 3.07] | 40 | 37 | **2.79** | [1.10, 7.10] | 74 | 40 | 0.70 | [0.32, 1.54] |
| 8.7 days - < 36.7 days | 128 | 1,161 | 1.24 | [0.85, 1.81] | 44 | 47 | 1.62 | [0.70, 3.77] | 40 | 51 | 1.87 | [0.78, 4.53] | 84 | 44 | 0.86 | [0.37, 2.00] |
| ≥ 36.7 days | 110 | 1,069 | 1.19 | [0.86,1.73] | 33 | 46 | 1.14 | [0.49, 2.62] | 55 | 24 | **3.98** | [1.56, 10.11] | 74 | 36 | 0.93 | [0.41, 2.10] |
| $O_3$ 8-Hr (State) - Ref. < 1.9 days | | | | | | | | | | | | | | | | |
| 1.9 days - <14.3 days | 110 | 1,018 | 1.34 | [0.92, 1.96] | 33 | 39 | 1.24 | [0.52, 2.98] | 30 | 42 | 1.25 | [0.50, 3.09] | 76 | 34 | 1.29 | [0.59, 2.81] |
| 14.3 days - < 51.2 days | 116 | 1,110 | 1.20 | [0.83, 1.75] | 42 | 43 | **2.35** | [1.01, 5.47] | 42 | 43 | 1.71 | [0.75, 3.93] | 79 | 37 | 1.26 | [0.57, 2.80] |
| ≥ 51.2 days | 123 | 1,086 | 1.32 | [0.91, 1.91] | 37 | 50 | 0.98 | [0.43, 2.19] | 55 | 32 | 1.80 | [0.77, 4.20] | 79 | 44 | 1.12 | [0.53, 2.38] |
| $PM_{10}$ (State) - Ref. < 1.6 days | | | | | | | | | | | | | | | | |
| 1.6 days - < 3.5 days | 88 | 692 | 1.21 | [0.80, 1.83] | 30 | 28 | 1.60 | [0.56, 4.55] | 218 | 126 | 0.96 | [0.64, 1.44] | 60 | 28 | 1.28 | [0.51, 3.26] |
| 3.5 days - <6.6 days | 77 | 651 | 1.38 | [0.87, 2.18] | 31 | 32 | 1.10 | [0.40, 3.08] | 177 | 83 | 1.07 | [0.69, 1.67] | 53 | 24 | 0.70 | [0.27, 1.81] |
| ≥ 6.6 days | 106 | 948 | 1.08 | [0.70, 1.67] | 26 | 45 | 0.56 | [0.22, 1.46] | 34 | 37 | 0.65 | [0.24, 1.75] | 61 | 45 | 0.67 | [0.26, 1.71] |
| $PM_{2.5}$ (Federal) - Ref. < 4.8 days | | | | | | | | | | | | | | | | |
| 4.8 days - < 12.0 days | 57 | 493 | 1.62 | [0.99, 2.65] | 12 | 22 | 0.37 | [0.13, 1.10] | 151 | 76 | 1.13 | [0.69, 1.87] | 33 | 24 | 1.28 | [0.41, 4.04] |
| 12.0 days - < 24.9 days | 78 | 753 | 1.26 | [0.81, 1.95] | 27 | 28 | 0.77 | [0.29, 2.07] | 198 | 137 | 0.78 | [0.49, 1.24] | 55 | 23 | 1.75 | [0.67, 4.57] |
| ≥ 24.9 days | 66 | 633 | 1.19 | [0.73, 1.95] | 22 | 33 | 0.64 | [0.24, 1.76] | 31 | 24 | 1.30 | [0.49, 3.48] | 40 | 26 | 0.79 | [0.30, 2.09] |

[a]For CHIS 2003, having asthma-like symptoms is defined as reporting wheezing or whistling in the previous 12 months without an asthma diagnosis; only respondents who lived within 5 miles of an air monitoring station and lived in the same home or neighborhood for at least 9 months were included.

[b]Respondents without an asthma diagnosis who lived within 5 miles of an air monitoring station and had lived in the same home or neighborhood for at least 9 months were included. Respondents with wheezing symptoms were designated as having asthma-like symptoms.

[c]Data not collected for teen respondents

[d]Adjusted for age, race, poverty level, and sex.

Table35. Associations (OR (95% CI)) for traffic density/distance to roadway and asthma-like outcomes in CHIS 2003 adults and children with asthma-like symptoms[a]

| | Wheeze[b] | | | | Missed ≥2 School/Work Days Due to Wheezing[c] | | | | ≥2 Wheeze Attacks[c] | | | | Sought Medical Help for Breathing Problem | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Exposure | Cases | Non-Cases | OR[d] | 95% C.I. | Cases | Non-Cases | OR[d] | 95% C.I. | Cases | Non-Cases | OR[d] | 95% C.I. | Cases | Non-Cases | OR[d] | 95% C.I. |
| Adults | | | | | | | | | | | | | | | | |
| Tele Atlas Traffic <750ft Continuous | 3624 | 26356 | 0.99 | [0.96, 1.02] | 444 | 2312 | 0.97 | [0.90, 1.04] | 2027 | 1146 | 0.99 | [0.93, 1.05] | 1310 | 1863 | 0.96 | [0.90, 1.02] |
| Tele Atlas Traffic < 750ft (25th < 50th percentile)[e,f] | 967 | 6,702 | 1.07 | [0.93, 1.22] | 122 | 623 | 1.22 | [0.83, 1.79] | 542 | 320 | 0.82 | [0.62, 1.09] | 358 | 504 | 1.23 | [0.94, 1.62] |
| Tele Atlas Traffic < 750ft (50th < 75th percentile)[e,f] | 880 | 6,328 | 1.04 | [0.90, 1.19] | 116 | 562 | 1.28 | [0.86, 1.90] | 500 | 260 | 1.02 | [0.76, 1.35] | 325 | 435 | 1.30 | [0.99, 1.71] |
| Tele Atlas Traffic < 750ft (≥75th percentile)[e,f] | 824 | 6,194 | 0.98 | [0.85, 1.13] | 101 | 535 | 0.98 | [0.65, 1.49] | 447 | 271 | 0.91 | [0.67, 1.22] | 297 | 421 | 1.07 | [0.80, 1.43] |
| State Highway < 300 m[g] | 189 | 1,158 | 1.10 | [0.89, 1.37] | 19 | 125 | 0.64 | [0.35, 1.18] | 111 | 52 | 1.13 | [0.70, 1.83] | 64 | 99 | 1.21 | [0.79, 1.84] |
| Interstate Highway < 300 m[g] | 344 | 2,552 | 1.00 | [0.84, 1.19] | 41 | 224 | 0.75 | [0.46, 1.23] | 184 | 119 | 0.78 | [0.55, 1.11] | 112 | 191 | 0.77 | [0.53, 1.12] |
| Major Road < 50 m[g] | 717 | 4,923 | 0.99 | [0.88, 1.12] | 93 | 451 | 1.10 | [0.77, 1.57] | 404 | 211 | 1.16 | [0.89, 1.51] | 251 | 364 | 0.93 | [0.72, 1.20] |
| Children | | | | | | | | | | | | | | | | |
| Tele Atlas Traffic <750ft Continuous | 831 | 7,463 | 1.00 | [0.93, 1.08] | 273 | 301 | 1.08 | [0.94, 1.25] | 284 | 290 | 1.13 | [1.00, 1.27] | 561 | 270 | 0.94 | [0.80, 1.10] |
| Tele Atlas Traffic < 750ft (25th < 50th percentile)[e,f] | 205 | 2,036 | 0.87 | [0.65, 1.15] | 64 | 75 | 1.07 | [0.58, 1.99] | 79 | 60 | **2.15** | [1.10, 4.21] | 134 | 71 | 1.46 | [0.74, 2.90] |
| Tele Atlas Traffic < 750ft (50th < 75th percentile)[e,f] | 205 | 1,859 | 0.71 | [0.53, 0.93] | 68 | 76 | 0.89 | [0.48, 1.65] | 69 | 75 | 1.57 | [0.84, 2.96] | 139 | 66 | 0.80 | [0.42, 1.50] |
| Tele Atlas Traffic < 750ft (≥75th percentile)[e,f] | 187 | 1,572 | 0.92 | [0.68, 1.23] | 64 | 66 | 1.20 | [0.63, 2.30] | 60 | 70 | 1.39 | [0.72, 2.71] | 130 | 57 | 0.91 | [0.48, 1.73] |
| State Highway < 300 m[g] | 46 | 310 | 1.03 | [0.65, 1.63] | 12 | 19 | 0.59 | [0.21, 1.65] | 14 | 17 | 0.62 | [0.24, 1.64] | 30 | 16 | 0.98 | [0.41, 2.31] |
| Interstate Highway < 300 m[g] | 81 | 700 | 1.09 | [0.76, 1.58] | 35 | 24 | 1.69 | [0.76, 3.76] | 34 | 25 | **2.55** | [1.16, 5.57] | 53 | 28 | 0.51 | [0.23, 1.17] |
| Major Road < 50 m[g] | 150 | 1,263 | 0.93 | [0.70, 1.23] | 58 | 51 | 1.73 | [0.98, 3.07] | 52 | 57 | 0.89 | [0.50, 1.57] | 107 | 43 | 0.85 | [0.49, 1.47] |

[a]For CHIS 2003, asthma-like symptoms is defined as reporting wheezing or whistling in the previous 12 months without an asthma diagnosis; only respondents who lived in the same home or neighborhood for at least 9 months, and geocoded by address or nearest cross streets were included. Measures based on Tele Atlas traffic data.
[b]Respondents without an asthma diagnosis who lived within 5 miles of an air monitoring station and had lived in the same home or neighborhood for at least 9 months were included. Respondents with wheezing symptoms were designated as having asthma-like symptoms.
[c]Data not collected for teen respondents
[d]Adjusted for age, race, poverty level, and sex.
[e]Reference: <25$^{th}$ percentile
[f]Units: vehicle meters traveled/day/meter$^2$
[g]Based on imputed Tele Atlas traffic data or Tele Atlas Dynamap 2000 roadway map.

Table 36 (Detailed). Associations between two or more wheeze attacks and $O_3$ adjusting for vulnerability characteristics among CHIS adults with asthma-like symptoms[a]

| | | $O^3$ (per 10 ppb) | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | Model 1[b] (1121, 657)[c] | | | Model 2[b] (1115, 652)[c] | | | Model 3[b] (1105, 647)[c] | | |
| **Vulnerability Characteristic** | | OR | 95% C.I. | | OR | 95% C.I. | | OR | 95% C.I. | |
| $O_3$ 12-Month Average (per 10 ppb) | | 1.11 | 0.94 | 1.30 | 1.12 | 0.95 | 1.32 | 1.12 | 0.95 | 1.31 |
| Age (Ref. ≥65) | | | | | | | | | | |
| | 18 - 34 | 0.85 | 0.57 | 1.27 | 1.17 | 0.72 | 1.88 | 0.63 | 0.40 | 0.98 |
| | 35 - 64 | 1.22 | 0.86 | 1.72 | 1.63 | 1.08 | 2.44 | 0.98 | 0.68 | 1.41 |
| Race (Ref. White) | | | | | | | | | | |
| | African American | 0.94 | 0.57 | 1.54 | 0.97 | 0.59 | 1.62 | 1.05 | 0.62 | 1.77 |
| | American Indian / Alaska Native | 1.83 | 0.73 | 4.59 | 2.00 | 0.78 | 5.17 | 1.85 | 0.72 | 4.79 |
| | Asian / Pacific Islander / Other | 1.02 | 0.63 | 1.65 | 1.08 | 0.67 | 1.73 | 1.07 | 0.64 | 1.78 |
| | Latino | 0.56 | 0.39 | 0.78 | 0.57 | 0.39 | 0.82 | 0.64 | 0.44 | 0.94 |
| Poverty (Ref. ≥400% FPL)[d] | | | | | | | | | | |
| | 0 - 199% FPL | 1.05 | 0.76 | 1.45 | 0.82 | 0.58 | 1.17 | 0.98 | 0.69 | 1.40 |
| | 200 - 399% FPL | 0.92 | 0.67 | 1.27 | 0.84 | 0.60 | 1.16 | 0.86 | 0.62 | 1.19 |
| Sex | | | | | | | | | | |
| | Female vs. Male | 0.99 | 0.76 | 1.28 | 0.96 | 0.73 | 1.26 | 0.94 | 0.71 | 1.23 |
| Currently Insured | | | | | | | | | | |
| | Yes vs No | | | | 0.71 | 0.50 | 1.02 | | | |
| Obese | | | | | | | | | | |
| | No vs Yes | | | | 0.94 | 0.70 | 1.25 | | | |
| Heart Disease | | | | | | | | | | |
| | Yes vs No | | | | 1.28 | 0.84 | 1.94 | | | |
| Smoker | | | | | | | | | | |
| | Ever vs Never | | | | 1.43 | 1.08 | 1.88 | | | |
| Work Status | | | | | | | | | | |
| | Employed vs Unemployed | | | | 0.52 | 0.37 | 0.73 | | | |
| Urban/Rural (Ref. Town and Rural) | | | | | | | | | | |
| | Urban | | | | | | | 1.63 | 0.88 | 3.02 |
| | 2nd City | | | | | | | 1.40 | 0.75 | 2.62 |
| | Suburban | | | | | | | 1.55 | 0.80 | 3.01 |
| Usual Source of Care | | | | | | | | | | |
| | Yes vs No | | | | | | | 1.03 | 0.70 | 1.53 |
| Delay in Care | | | | | | | | | | |
| | Yes vs No | | | | | | | 0.58 | 0.43 | 0.80 |
| Household Smoking | | | | | | | | | | |
| | No vs Yes | | | | | | | 0.54 | 0.37 | 0.79 |
| Dog or Cat in Home | | | | | | | | | | |
| | No vs Yes | | | | | | | 0.82 | 0.62 | 1.10 |
| Cockroaches | | | | | | | | | | |
| | Yes vs No | | | | | | | 0.96 | 0.66 | 1.40 |
| Housing type (Ref. House) | | | | | | | | | | |
| | Duplex or Apartment | | | | | | | 0.68 | 0.32 | 1.46 |
| | Mobile Home | | | | | | | 0.65 | 0.31 | 1.38 |
| Household Crowding | | | | | | | | | | |
| | No vs Yes | | | | | | | 0.95 | 0.65 | 1.40 |
| Diabetes (Ref. Pre-Diabetes/ Borderline | | | | | | | | | | |
| | Yes | | | | | | | 0.62 | 0.17 | 2.21 |
| | No | | | | | | | 0.75 | 0.22 | 2.53 |
| Walking | | | | | | | | | | |
| | Yes vs No | | | | | | | 0.99 | 0.74 | 1.32 |

[a]For CHIS 2003, asthma-like symptoms is defined as reporting wheezing or whistling in the previous 12 months without an asthma diagnosis; only respondents who lived within 5 miles of an air monitoring station and lived in the same home or neighborhood for at least 9 months were included.
[b]Only variables with listed values are included for each model.
[c](Cases, Controls)
[d]FPL = Federal Poverty Level

Table 37. Associations between two or more wheeze attacks and vulnerability characteristics by $O_3$ among CHIS children with asthma-like symptoms[a]

| | | $O_3$ (per 10 ppb) (157, 153)[b] | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | Model 1[c] | | | Model 2[c] | | | Model 3[c] | | |
| **Vulnerability Characteristic** | | OR | 95% C.I. | | OR | 95% C.I. | | OR | 95% C.I. | |
| 12-Month Average | | 1.29 | 0.90 | 1.84 | 1.28 | 0.89 | 1.83 | 1.27 | 0.86 | 1.89 |
| Age (Ref. 6-11 years) | | | | | | | | | | |
| | < 6 years old | 0.66 | 0.35 | 1.25 | 0.69 | 0.37 | 1.29 | 0.65 | 0.34 | 1.25 |
| Race (Ref. White) | | | | | | | | | | |
| | African American | 0.32 | 0.06 | 1.69 | 0.26 | 0.04 | 1.60 | 0.38 | 0.08 | 1.81 |
| | American Indian / Alaska Native | 0.21 | 0.03 | 1.54 | 0.18 | 0.03 | 1.16 | 0.16 | 0.02 | 1.53 |
| | Asian / Pacific Islander / Other | 2.07 | 0.77 | 5.57 | 1.84 | 0.69 | 4.91 | 2.55 | 0.88 | 7.41 |
| | Latino | 0.53 | 0.25 | 1.15 | 0.46 | 0.20 | 1.07 | 0.75 | 0.34 | 1.65 |
| Poverty (Ref. ≥400% FPL)[d] | | | | | | | | | | |
| | 0 - 199% FPL | 3.44 | 1.60 | 7.38 | 3.62 | 1.59 | 8.23 | 4.88 | 2.05 | 11.63 |
| | 200 - 399% FPL | 2.42 | 1.06 | 5.55 | 2.40 | 1.06 | 5.47 | 2.61 | 1.10 | 6.23 |
| Sex | | | | | | | | | | |
| | Female vs. Male | 0.93 | 0.52 | 1.69 | 0.88 | 0.49 | 1.59 | 0.93 | 0.49 | 1.75 |
| Household Smoking | | | | | | | | | | |
| | No vs Yes | | | | 0.55 | 0.12 | 2.47 | | | |
| Dog or Cat in Home | | | | | | | | | | |
| | No vs Yes | | | | 1.11 | 0.56 | 2.22 | | | |
| Cockroaches | | | | | | | | | | |
| | Yes vs No | | | | 0.73 | 0.36 | 1.47 | | | |
| Currently Insured | | | | | | | | | | |
| | Yes vs No | | | | 0.38 | 0.11 | 1.26 | | | |
| Urban/Rural (Ref. Town and Rural) | | | | | | | | | | |
| | Urban | | | | | | | 1.20 | 0.36 | 3.95 |
| | 2nd City | | | | | | | 1.19 | 0.37 | 3.83 |
| | Suburban | | | | | | | 0.92 | 0.26 | 3.19 |
| Delay in Care | | | | | | | | | | |
| | No vs Yes | | | | | | | 0.43 | 0.15 | 1.20 |
| Housing type (Ref. House | | | | | | | | | | |
| | Duplex or Apartment | | | | | | | 0.38 | 0.04 | 3.71 |
| | Mobile Home | | | | | | | 0.59 | 0.06 | 6.23 |
| Household Crowding | | | | | | | | | | |
| | No vs Yes | | | | | | | 2.32 | 1.02 | 5.27 |

[a]For CHIS 2003, asthma-like symptoms is defined as reporting wheezing or whistling in the previous 12 months without an asthma diagnosis; only respondents who lived within 5 miles of an air monitoring station and lived in the same home or neighborhood for at least 9 months were included.
[b]Only variables with listed values are included for each model.
[c](Cases, Controls)
[d]FPL = Federal Poverty Level

## Summary of Findings for CHIS 2003 Respondents with Current Asthma

*Demographic/Descriptive*

- One-third of adults (32.5%) and more than a third of children (38.4%) with current asthma lived below 200% of the FPL.
- About 40% of adults and more than half of children (56.5%) with current asthma were minorities.

*Pollutant Exposure Disparities*

Adults

- In general, adult respondents with current asthma living below 200% of the FPL had higher estimates of annual average exposure to $NO_2$, $PM_{10}$, and $PM_{2.5}$, than those living at or above 400% of the FPL.
- Adult respondents in minority populations had higher estimated exposures to several criteria pollutants. Specifically, Latino adults had higher estimated exposures to $NO_2$, $PM_{10}$, and $PM_{2.5}$; African American adults had higher estimated exposures to $PM_{2.5}$; and Asian/PI/Other adults had higher estimated exposures to $NO_2$ compared to white respondents. However, they all had lower $O_3$ exposures than their white counterparts.
- African American Adults were more likely to live in areas with higher traffic density than white adults.

Children

- On average, children with current asthma living between 0-199% of the FPL had a higher estimated mean annual exposure to criteria pollutants ($NO_2$, $PM_{10}$, and $PM_{2.5}$) and were more likely to live in places with greater traffic density or live near highways or major roadways than children living at or above 400% of the FPL.
- Minority children had higher estimated mean annual exposures to several criteria pollutants. Specifically, Latino and African American children had higher estimated mean annual exposures to $NO_2$ and $PM_{2.5}$, and Asian/PI/Other children had higher estimated exposure to $NO_2$.
- Mean traffic density measures were higher for both Latino and African American children than for white children.

*Associations of Air Pollution Exposure with Asthma Outcomes*

Adults

- We observed increased odds of experiencing an asthma attack in the previous year among adults with lifetime asthma.
- We observed increased odds of ED visits, using daily asthma medication, and missing 2 or more days of work due to asthma with the increase in annual average $O_3$, $PM_{10}$, and $PM_{2.5}$ among adults with current asthma.

Children

- We observed increased odds of daily asthma medication use and missing 2 or more days of school/day care with the increase of annual average $NO_2$.

*Associations of Exceedance Days with Asthma Outcomes*

Adults

- Exceedances of state 1-hr $O_3$ standards (i.e. over 36.7 days per year) were associated with increased odds for asthma attacks among adults with lifetime asthma, and visiting the ED and using a daily asthma medication among adults with current asthma.
- Exceedances of the state 24-hr $PM_{10}$ standard (i.e. over 6.6 days per year) were associated with increased odds of asthma ED visits.
- Exceedances of the federal 24-hr $PM_{2.5}$ standard (i.e. over 23.9 days per year) were associated with increased odds of ED visits and daily/weekly asthma symptoms.

Children

- $O_3$ exceedances (state 1-hr standard) were associated with increased odds of missing 2 or more days of school.

*Association of Traffic Measures with Asthma Outcomes*

- An interquartile increase in traffic density within 750 feet of respondent's homes was associated with an increase in odds of reporting asthma ED visits in the past year among adults with current asthma.
- Living within 300 m of an interstate highway was associated with a suggested increase in the odds of visiting the ED in the past year, as well as a suggested increase in the odds of needing a daily asthma medication among adults.

*Vulnerability Factors*

Adults

- Positive associations between pollutants ($O_3$, $PM_{10}$, and $PM_{2.5}$) and asthma outcomes (ED visits, taking a daily asthma medication, or missing 2 or more days of work) persisted after adjusting for potential vulnerability factors.
- In addition to pollutant exposures, several other characteristics were related to increased odds of asthma outcomes: being African American or Latino, living below 200% of the FPL,, being a smoker, having heart disease and having adult onset asthma.
- Some characteristics, such as, not having an asthma management plan and using daily asthma medication, decreased odds of ED visits or missing 2 or more days of work. :.

Children

- Positive associations between daily asthma medication and $PM_{2.5}$ persisted after adjusting for potential vulnerability factors.
- Children living below 200% and between 200-399% of the FPL consistently had higher odds of using a daily asthma medication than those living at or above 400% of the FPL.
- Not having an asthma management plan decreased odds of using daily asthma medication.

*Interactions*

Adults

- For the same level of increase in annual average $NO_2$, African American and Asian/PI/other adults had greater increases in missing two or more days of work due to asthma compared to white adults.
- African American adults had a greater increase in daily/weekly asthma symptoms for the same level of increase in annual average $NO_2$.

Children

- For the same level of increase in annual average $NO_2$, American Indian/Alaska Native and Asian/PI/other children had greater increases in daily/weekly symptoms compared to white children.
- Latino children had a greater increase in using daily asthma medication, and African-American and Asian/PI/other children had a greater increase in experiencing daily/weekly symptoms for the same increase in annual average $PM_{10}$ as white children.
- Children living below 200% of the FPL had a greater increase in ED visits compared to those living at or above 400% of the FPL for the same increase in annual average $NO_2$.

## Summary of Findings for CHIS 2003 Respondents with Asthma-like Symptoms

*Demographic/Descriptive*

Adults

- 41.5% of adults with asthma-like symptoms were minorities.
- More than one-third (39.0%) of adults with asthma-like symptoms lived below 200% of the FPL.

Children

- 57.4% of children with asthma-like symptoms were minorities.
- Children with asthma-like symptoms fell below 200% of the FPL 42.4% of the time.

*Pollutant Exposure Disparities*

Adults

- In comparison to those living at 400% of the FPL or above, adults with asthma-like symptoms living below 200% of the FPL had higher estimated average annual exposure to all criteria pollutants except for $O_3$.
- Latino and African American adults had higher estimated average annual exposure to all criteria pollutants except for $O_3$ than white adults.

Children

- In comparison to those living at 400% of the FPL or above, children with asthma-like symptoms living below 200% of the FPL had higher estimated average annual exposure to $NO_2$ and $PM_{2.5}$.
- Latino and African American children had higher annual average concentrations of $NO_2$ than white children.
- Latino children also had higher annual average concentrations of $PM_{10}$ and $PM_{2.5}$ than white children.

*Associations between Air Pollution Exposure and Asthma-like Symptoms*

Adults

- Increases in $O_3$, $PM_{10}$, and $PM_{2.5}$ annual averages were associated with increased odds of wheeze.
- Increases in $O_3$ and $PM_{10}$ annual averages appeared to be associated with having 2 or more wheeze attacks.

Children

- Increased annual average $O_3$ appeared to be associated with increased odds of wheeze and having 2 or more wheeze attacks.

# EXHIBIT 4

# STATUS OF RESEARCH ON POTENTIAL MITIGATION CONCEPTS TO REDUCE EXPOSURE TO NEARBY TRAFFIC POLLUTION

**August 23, 2012**

California Environmental Protection

**Air Resources Board**

# Introduction

Air Resources Board (ARB) staff has prepared this document to provide information on scientific research that has been conducted on various building-related and site mitigation concepts suggested as potentially effective approaches for reducing the traffic-related exposures of those living near high traffic roadways. While it provides useful information for consideration of potential mitigation approaches, this paper is not intended as guidance for any specific project, and does not provide a methodology for determining appropriate mitigation measures for purposes of compliance with the California Environmental Quality Act. This review looked only at the current status of air pollution research, and does not address other potential community benefits of the concepts, such as the aesthetic and noise reduction benefits of adding vegetation or sound walls.

The State's current set-back requirement for schools (500 feet [ft]; PRC 21151.8) and the ARB's recommendations on siting for housing and other sensitive uses (e.g., 500 ft from major roadways and 1000 ft from busy distribution centers and rail yards; ARB 2005a) are intended to help protect the public from exposure to traffic emissions. Such emissions have been associated with a variety of serious health impacts in epidemiological studies, including exacerbation of respiratory and cardiovascular diseases and conditions, increased asthma and bronchitis in children, and increased risk of premature death. Traffic pollutant concentrations near high traffic roadways have been found to be 2 to 10 times higher than levels at a distance from the roadways. Also, recent studies have shown elevated traffic pollutant levels at greater distances from the roadway than previously measured.

ARB and the U.S. EPA continue to adopt increasingly stringent regulations limiting emissions from vehicles of all types, which have substantially reduced, and will continue to reduce, vehicle emissions. However, recently adopted regulations have compliance dates extending as far as 2025 for full implementation, and fleet turnover to zero or near-zero technologies will take 20 to 30 years. New reductions in vehicle emissions are improving regional air quality throughout California, including near roadways. As the ARB and the air districts work to reduce emissions from diesel PM and other pollutants, the impact of proximity will also be reduced. However, the differential exposure to high air pollution near high traffic roadways compared to other locations makes the siting of housing in those locations a continuing health concern. Recognizing that unhealthful levels of air pollution is a long term problem, ARB is funding research to identify advanced technologies to further reduce vehicle emissions, to better understand traffic related air pollution exposures, and to explore the benefits of high efficiency filtration in California homes.

As communities plan for more compact development, the potential health impacts of infill projects will need to be considered. Infill development can reduce urban sprawl and has other potential health and environmental benefits. It also has the potential to increase exposure to traffic pollution due to the proximity of the infill areas to established traffic routes.

## Status of Research on Traffic Exposures and Health Impacts

Measurements of air pollutants near roadways show a consistent finding of elevated levels based on proximity. Black carbon, often used as an indicator of diesel exhaust, and ultrafine particles (particles less than 0.1 microns in size), which are emitted in very high numbers from vehicles, are often 2 to 10 times (or more) higher near roadways and freeways (Zhu et al., 2002a, 2002b, 2006; Kuhn et al., 2005; Westerdahl et al., 2005; Ntziachristos et al., 2007; Kozawa et al., 2009a). Concentrations of PM2.5 (particles 2.5 microns or less in diameter) near busy roadways can be about 20% higher than levels at a distance (Zhu et al., 2002a; Kim et al., 2004; Janssen et al., 2001). Nitrogen oxides also are elevated near roadways, usually about 2 to 3 times the levels measured at a distance from the roadway (Kim et al., 2004; Singer et al., 2004; Kozawa et al., 2009a; Durant et al., 2010).

Previous studies of near roadway pollutant levels showed that concentrations of pollutants emitted from vehicles were highest right at the roadway and decreased substantially in the first 300-500 feet from the roadway (Zhu et al., 2002b; Knape 1999). These results were consistent with health studies that showed a stronger association of health impacts for those living within 300-500 ft of the roadway compared to those living farther than 500 ft from the roadway (Brunekreef et al., 1997; Venn et al., 2001; English et al., 1999). More recent studies have shown a somewhat longer plume of increased pollutant concentrations farther from the roadway. Using data collected mostly during the day and near roadways, a meta-analysis of many studies found that for almost all pollutants, elevated levels of pollutants caused by the increased contributions from roadways returns to background levels at 160 - 570 meters (m; 525 – 1870 ft; Karner et al., 2010). The range of distances needed to reach background is usually a result of local meteorological conditions, which can vary significantly; however, a more constant observation is a steep concentration gradient observed closest to the roadway, within 500 ft, with a more gradual and extended decline at further distances. Another meta-analysis found that the “spatial extent of impact” of motor vehicles can extend up to 400 m (1312 ft) for black carbon and particles and 500 m (1640 ft) for nitrogen dioxide ($NO_2$; Zhou and Levy 2007). Levels of traffic pollutants near roadways vary due to many factors, including traffic type and density, wind direction and speed, local and roadway topography, and time of day and season (Zhu et al., 2004; Kuhn et al., 2005; Moore et al., 2007; Ning et al., 2007; Hu et al., 2009; Kozawa et al., 2009a, 2009b).

In a major 2008 review of the scientific literature by the Health Effects Institute (HEI), proximity to busy roadways was found to be associated with a variety of adverse health impacts, the strongest association being exacerbation of asthma, with others including asthma onset in children, impaired lung function, and increased heart disease (HEI, 2010). More recent studies have added to the list of effects and heightened concern regarding exposure to traffic emissions. Respiratory and cardiovascular effects seen in these studies include an increased risk of new-onset chronic obstructive pulmonary disease (Andersen et al., 2010), a faster progression of atherosclerosis in those living within 100 m of highways in Los Angeles (Künzli et al., 2010), increased risk of

premature death from circulatory disease (Jerrett et al., 2009), and increased incidence of new heart disease (Kan et al., 2008). Other effects include increased risk of low birth weight (Brauer et al., 2008; Llop et al., 2010) and increased risk of pre-term delivery (Wilhelm and Ritz, 2003; Wilhelm et al., 2011) for mothers living very near heavy traffic, lower immune function in post-menopausal women living within 150 m of arterial roads (Williams et al., 2009), and increased risk of Type 2 diabetes in post-menopausal women (Krämer et al., 2010).

Children appear to be particularly vulnerable to the adverse effects of traffic emissions. Epidemiological studies have found significant associations of children living near high traffic areas with decreased lung function (Brunekreef et al., 1997; Gauderman et al., 2007), increased medical visits and hospital admissions for childhood asthma (English et al., 1999; Lin et al., 2002), increased wheezing (Venn et al., 2001), and increased childhood asthma and bronchitis (Kim et al., 2004; Gauderman et al., 2005; McConnell et al., 2006), including development of new asthma cases (McConnell et al., 2010; Gehring et al., 2010). Children living near busy roadways are especially likely to experience elevated exposures because they would also play outdoors in the neighborhood and typically would attend nearby schools. Their higher breathing rates per unit of body mass relative to adults (Adams, 1993) and their developing immune, neurological, and respiratory systems make them especially susceptible to impacts from air pollution.

ARB's recommendation to avoid siting sensitive land uses such as new housing within 500 ft of busy roadways was based on the traffic exposure and health studies completed as of 2005. More recent studies confirm the relationship, and indicate that in some situations an elevated risk extends well past 500 ft. A few studies have measured elevated pollutant levels at distances well beyond 1000 ft (305 m; Karner et al., 2010; Zhou and Levy, 2007). For example, Hu and colleagues (2009) found that in the pre-dawn hours in Los Angeles, elevated ultrafine particle number concentration, nitric oxide, and particle-bound polycyclic aromatic hydrocarbons extended at least 1200 m (3937 ft) downwind of the freeway and did not reach background levels until a distance of 2600 m (8530 ft). More importantly, results from the Southern California Children's Health Study on the association of residential distance to traffic and lung function development, performed in the same general location as the Hu et al. study, found adverse health effects in children living as far as 1500 m (4921 ft) from roads (Gauderman et al., 2007). These are not unique findings; in the HEI (2010) report mentioned above, the authors noted that studies showed that people living up to 500 m (1640 ft) from heavy traffic are most at risk from the health effects of traffic pollution.

## Status of Research on Mitigation Concepts

Various building and site mitigation approaches have been suggested as potential means to reduce exposure to traffic pollution near roadways. A review by ARB staff found that there has been limited study of most of these approaches. Building measures examined include high efficiency filtration for residences through either central, in-duct type filtration or portable air cleaners; and external building design

measures, such as locating the air intakes for ventilation systems on the opposite side of the building from outdoor sources, reducing the size and number of openable windows on the side of the building nearest the outdoor sources, or housing people in tall buildings. Site mitigation measures examined include the use of sound walls and vegetation as barriers. These measures are all assessed further below. Studies of elevated and below-grade roadways and freeway caps (also called freeway decks, lids or covers), which are covers over a sunken roadway that produce a road tunnel, also were reviewed, but studies were limited and results variable, and these measures are not feasible or are impractical for most new housing developments. Traffic measures such as those to reduce vehicle miles traveled also were considered; most such measures are typically integrated into roadway and community planning for regional benefits.

## Building-related Measures: Filtration

No single building-related measure has been identified as adequate to reduce entry of pollutants from nearby roadways to the extent expected from set-back under common conditions. However, the use of high efficiency filtration appears to be relatively effective in most circumstances, as discussed below. It is especially appropriate for new homes because new homes in California must have mechanical ventilation systems [CCR 2008, Title 24, Section 150(o)], and those systems purposely pull outdoor air into the home that often is not filtered at all or is poorly filtered. High efficiency filtration also appears useful in existing homes without mechanical ventilation as discussed below. Mechanical ventilation systems and the Code requirement are discussed further in the Addendum at the end of this paper.

### Background for Filtration

Outdoor-generated pollutants enter and leave buildings through three primary mechanisms: mechanical ventilation systems, which actively draw in outdoor air through an intake vent and distribute it throughout the building; natural ventilation (opening of doors or windows), which is the typical ventilation mode for most homes and small commercial buildings in California; and infiltration, which is the passive entry of unfiltered, outdoor air through small cracks and gaps in the building shell. Both natural ventilation and infiltration allow unfiltered air into the building and reduce the effectiveness of any filtration device.

Filter efficiency is rated using several scales, the most common of which is the Minimum Efficiency Reporting Value (MERV) rating system (ASHRAE 52.2-2007 as cited in EPA 2009). Flat fiberglass filters are the most common filters used in residential heating and air systems, and are rated at only MERV 1 to 4; they remove only a portion of the largest particles in the airstream that passes through the filter. MERV 5 to 8 filters are medium efficiency filters that remove some additional types of particles such as mold spores and cat and dog dander, but they still do not remove the finer particles produced on roadways. MERV 9 to 12 filters begin to remove particles smaller than PM2.5. Higher efficiency MERV 13 to 16 filters are rated to remove a portion of the ultrafine and submicron particles emitted from vehicles. True HEPA (high efficiency particle

arrestance) filters (equivalent to MERV 17 to 20) remove 99.97% to 99.999% of particles less than 0.3 microns, but these generally have not been available for residential applications. High efficiency filters associated with central heating, ventilating and air conditioning (HVAC) systems must be carefully selected to assure the mechanical system can handle the increased airflow resistance. Additional information on MERV ratings, the size particles they remove, and typical applications are provided in Table 1 in the Addendum at the end of this paper.

### High Efficiency Filtration with Mechanical Ventilation

Because mechanical ventilation has not been used in residential buildings until recently, there has been limited assessment of its impact on entry of particles and other pollutants into homes. However, a few recent studies of homes and schools have shown that high efficiency filtration in mechanical ventilation systems can be effective in reducing levels of incoming outdoor particles. In a seven-home study in northern California, Bhangar et al. (2010) found that the two homes with active filtration in a mechanical system had a notably lower portion of indoor particles from outdoors when the systems were on (filtration active) than when they were turned off (no filtration). In a modeling study of Korean residential units with mechanical ventilation, Noh and Hwang (2010) found that filters rated lower than MERV 7 were insufficient for reducing contaminants that enter through the ventilation filter, and concluded that filters should exceed MERV 11. In a school pilot study, a combination of MERV 16 filters used as a replacement for the normal panel filter in the ventilation system and in a separate filtration unit reduced indoor levels of outdoor-generated black carbon, ultrafine particles and PM2.5 by 87% to 96% in three southern California schools (SCAQMD, 2009). Use of the MERV 16 panel filter alone in the HVAC system achieved average particle reductions of nearly 90%. In a study of a single school in Utah, indoor submicron particle counts were reduced to just one-eighth of the outdoor levels in a building with a mechanical system using a MERV 8 filter (Parker et al., 2008). The investigators noted that the building shell and other mechanical system components appeared to play a significant role in the submicron particle removal as well.

These findings are similar to those from earlier studies of mechanically ventilated office buildings (e.g., Jamriska et al., 2000; Fisk et al., 1998). Fisk et al. (2000) concluded that use of higher efficiency filters instead of normal filters can reduce indoor numbers of submicron particles by 90% and that there is evidence of a large rate of removal of submicron indoor particles by processes (e.g., deposition) other than ventilation and filtration.

Because most of the studies discussed above were conducted in buildings with few or no indoor sources of submicron particles, the measured efficiencies of filters for reducing indoor concentrations of submicron particles from all sources may be overestimated. Many other studies have identified activities such as unvented cooking, cigarette smoking, and use of unvented gas appliances as indoor sources of submicron particles (ARB, 2005b, studies cited). These would only be removed by filtration to the extent the indoor air is re-circulated through the filters.

### High Efficiency Portable Air Cleaning Devices

Portable or stand-alone air cleaners are generally not as capable as in-duct air cleaners and those associated with mechanical ventilation systems for cleaning large areas such as an entire home (Consumer Reports, 2007). However, when they are appropriately sized for the space to be treated, and when they use high efficiency or HEPA filters, portable air cleaners can significantly reduce particles in the treated area and serve as an adjunct to other pollutant reduction measures (Hacker and Sparrow, 2005; Shaughnessy et al., 1994; Shaughnessy and Sextro, 2006; Skulberg et al., 2005; Ward et al., 2005). In the pilot study conducted in three southern California schools (discussed above), a large stand-alone air cleaner with MERV 16 filters reduced black carbon, ultrafine particles and PM2.5 counts by 90% or more, and PM2.5 mass by 75%, when the HVAC system was not running (SCAQMD, 2009). Barn et al. (2008) found median removal efficiencies of 55% to 65% for PM2.5 from fires and wood burning by a HEPA air cleaner in 21 winter homes and 17 summer homes. In other work, Fisk et al. (2002) estimated an 80% reduction in outdoor fine mode particles with stand-alone air cleaners using filters in the MERV 11 to 13 range.

Because new California homes are now required to have mechanical ventilation, stand-alone air cleaners are less relevant to the assessment of measures for new California home construction. However, highly efficient portable air cleaners may be useful in reducing indoor exposure to pollutants in existing homes that do not have mechanical ventilation, and in homes that use bathroom exhaust type mechanical ventilation systems, which by their design cannot incorporate filtration of the incoming air because the supply air enters through leakage points throughout the building.

### Removal of Gaseous Pollutants

There are limited options for effective removal of gaseous pollutants such as volatile organic chemicals, or VOCs, and $NO_2$ in central systems, and although the number and variety of technologies are increasing, there has been only limited research to date on their effectiveness. However, a few studies have examined the effectiveness of stand-alone filtration technologies intended to remove gaseous pollutants from the airstream (Shaughnessy and Sextro, 2006). The most comprehensive study was conducted by Chen et al. (2005), who tested the initial performance of 15 air cleaners with a mixture of 16 representative VOCs in a chamber study. Sorption filtration (e.g., activated carbon) removed some but not all VOCs (light and very volatile gases such as aldehydes and dichloromethane were not well removed). However, devices that included sorption media such as activated alumina impregnated with potassium permanganate showed better VOC removal efficiencies. In the schools study discussed above, the stand-alone unit used in one of the schools included charcoal sorbent for removal of gaseous pollutants; it removed 52% of the benzene indoors and 15% of total VOCs when operated with the HVAC turned off (SCAQMD, 2009). In a children's daycare center in Finland, Partti-Pellinen et al. (2000) found that up to 50% to 70% of nitrogen oxides could be removed by chemical filtration using a combination of charcoal, aluminum oxide and potassium permanganate, while another study found about 50% $NO_2$ removal by a HEPA air cleaner with large quantities of carbon in the adsorption bed, but little or no removal by other types of air cleaners (Shaughnessy et al., 1994).

Results from these studies show effectiveness for some technologies but are not conclusive due to their limited number and scope, including a relative lack of real world measurements. Additionally, some investigators have found that some filters re-emit VOCs that have been removed over time, or emit reaction products from the matter collected on the filter (Daisey and Hodgson, 1989; Fisk, 2007; Destaillats et al., 2011; Hyttinen et al., 2006, 2007).

### Limitations of High Efficiency Filtration

Although they can substantially reduce indoor concentrations of pollutants, mechanical filtration systems alone are insufficient to fully protect occupants from particles and other emissions from nearby roadways, for several reasons.

- First, most people tend to open their windows or doors at least part of each day (Offermann, 2009; Phillips et al., 1990), and such natural ventilation involves no filtration of incoming air and can diminish any pollutant reductions attained through the use of the mechanical system. The effectiveness of high efficiency filtration in homes whose occupants open their doors and windows regularly has not been quantified.
- Second, as higher MERV filters are used, greater attention must be paid to the increased air flow resistance that occurs with some filter types; mechanical system motors must be sufficiently sized to accommodate the air flow needs.
- Third, studies have shown that homeowners are not provided with sufficient information regarding use and maintenance of their central HVAC systems, or do not read and follow instructions for maintaining their filters (EPA, 2009; Offermann, 2009). Filtration is only effective if filters are well-fitted and are replaced or maintained according to the manufacturer's recommendations, and duct leakage is minimized (Thatcher et al., 2001; Wallace et al., 2004). Older (aged) filters have been associated with increased irritant health symptoms and decreased work performance in studies of filtration maintenance in workplaces (Clausen, 2004; Seppänen and Fisk, 2002; Wargocki et al., 2004).
- Finally, as discussed above, gaseous pollutants are not removed by most particle filters, and the technologies for VOC removal in residential applications are limited and still evolving.

### Expected Benefits of High Efficiency Filtration

High efficiency filtration has been used in homes and schools only recently, and there is a range of highly variable building characteristics, filtration technologies, and occupant behaviors that determine the effectiveness of high efficiency filters in reducing the overall levels of pollutants indoors. Accordingly, it is difficult to accurately quantify the actual reduction in particulate matter that would be achieved by introducing high efficiency filtration on a widespread basis across the population of California homes and schools. For example, while filters with a MERV 16 rating remove more than 95% of particles from 0.3 to 3 microns in diameter, only those particles in the airstream actually passing through the filter are removed. Factors that determine the fraction of particles removed from the air in a building include the airflow rate through the unit, the amount

of time that the system is “on”, the extent to which windows and doors are opened, and other factors. While results from the studies conducted in homes and schools to date appear promising, those studies usually limited the opening of windows and doors or followed other specific protocols. Thus, although a substantial reduction in particles would be expected, the reduction that would be realized across the wide variety of conditions in California homes and schools cannot be confidently estimated.

Two kinds of programs are currently being implemented that will provide critical information needed to help confirm and quantify the effectiveness of high efficiency filtration. First, ARB is funding two key studies of high efficiency filtration in homes. Second, several local air quality management districts and school districts are implementing programs to install high efficiency filtration devices in a substantial number of schools in California, and collecting data regarding the performance of the filtration units. These are discussed below.

### ARB’s Planned High Efficiency Filtration Research

ARB is funding a project entitled “Reducing In-Home Exposure to Air Pollution” to measure the exposure reduction and energy use of combinations of mechanical ventilation and filtration systems in order to identify compatible, low-energy systems that are effective at reducing indoor exposures to indoor, and incoming outdoor, pollutants. The study will be conducted by Drs. Brett Singer and Iain Walker of Lawrence Berkeley National Laboratory. The investigators plan to evaluate 15 current and new systems, and test seven of the most promising systems in a test home near a major roadway in an area with high ambient ozone and PM2.5 levels. They will measure fine and ultrafine particles, ozone, VOCs, $NO_2$ and black carbon, both indoors and outdoors, along with energy consumption and the performance of systems as filters age. This project is needed because new California homes are now required to have mechanical ventilation as discussed above, and the most widely used, low energy mechanical ventilation systems, bathroom exhaust systems, do not filter the incoming air; hence, the occupants’ indoor exposure to outdoor air pollutants can potentially increase with these systems.

ARB is also funding a second study entitled “Benefits of High Efficiency Filtration to Children with Asthma”. Dr. Deborah Bennett from the University of California at Davis will conduct this 4-year study of 200 children with asthma in Fresno and Riverside to quantify the exposure and asthma reduction benefits of high efficiency filtration in their homes. One intervention group will have high efficiency filters or filtration systems installed in their homes’ central heating and air conditioning systems. The second group will have high efficiency portable air cleaners placed in the child’s bedroom and in the main living area. Filters with a MERV rating of 15 or higher will be used. Improvements in asthma symptoms will be evaluated in a randomized cross-over design, with each participant receiving high efficiency air filtration for a year and no filtration for a year, allowing the investigators to identify the improvements related to the air filtration. During the control periods, “sham” filters with little or no particle removal capability will be used. Half of the homes with portable air cleaners will also have filters that remove ozone and VOCs. The extent to which particulate matter (PM10, PM2.5

and ultrafine particles), ozone, black carbon, and nitrogen oxides are reduced will be measured. Key asthma health endpoints will also be examined, including unplanned utilization of the healthcare system for asthma-related illness, short-term medication use, symptom diaries, peak exhaled flow, spirometry and exhaled nitric oxide.

### Current Programs Using High Efficiency Filtration

Several programs have been completed or are underway in the State to install and/or test high efficiency filters, primarily in schools, to reduce exposures to pollutants from heavy traffic and/or port-related emissions. Since 2008, the South Coast Air Quality Management District (SCAQMD) has approved $3 million for installation of high efficiency air filtration devices in a total of 18 schools and one community center in the Long Beach and Los Angeles Unified School Districts, San Bernardino and the Boyle Heights area (Kwon, 2012). SCAQMD also has agreed to oversee implementation of a program to utilize $5.4 million in settlement funds to install and maintain high performance air filtration devices at about 47 schools in Wilmington and San Pedro. Installation of the filtration devices was scheduled to begin in summer 2012. Detailed site assessments of the schools are underway prior to installation in order to determine the best filtration device for each classroom and to facilitate assessment of actual improvements in classroom air.

Also, the Bay Area Air Quality Management District (BAAQMD) is conducting a school air filtration project in five schools for about $300,000 (Smith, 2012). In 2010, a contractor completed installation of high efficiency air filtration equipment at five elementary schools located in the Bay View Hunters Point neighborhood of San Francisco. The filtration equipment is designed to reduce exposure inside the schools to particles from outdoor sources, as well as indoor-based particles such as some allergens. Initial monitoring results indicate that there has been a substantial reduction of particulate matter (up to about 50% to 75% for PM2.5 and higher for very small particles) inside the classrooms as a result of the newly installed high performance filters (IQAir, 2012).

To date, these programs appear successful, but overall cost, changes to the operation of the classrooms' central HVAC systems (such as running the system continuously rather than allowing it to switch on and off based on temperature needs) and other considerations (noise, drafts) may reduce the feasibility of the current technologies for use in all classrooms and require further refinements. However, because of the similarities of schools to homes with mechanical ventilation systems, one would expect comparable reductions in particle levels from high efficiency HVAC filtration in new and retrofitted homes.

### Cost of High Efficiency Filtration

About a dozen companies offer high efficiency filtration devices incorporated into, or suitable for, residential mechanical ventilation systems, and most offer just one or two models. The devices are rated from MERV 11 to 16, plus several are true HEPA filters (equivalent to about MERV 17 to 20). Initial costs range from about $200 to $2800 for a

very high end system; however, most cost less than $500. This range does not include installation, although in a new home the added cost over the installation of the mechanical system itself would be expected to be minimal. Annual filter replacement and/or maintenance cost ranges from about $25 to $255 per year, depending on MERV rating, number of filter changes needed per year, and whether the system includes a carbon filter for VOCs (which increases the cost of filter replacement, as these typically need to be replaced several times per year).

For existing homes and those that are renovated and do not have a mechanical ventilation system, either higher efficiency filters in the central heating and air system or portable high efficiency filtration devices could be used. High efficiency filters for central systems that can accept them cost about $20. However, the increased airflow resistance may cause the central system to be less efficient. Effective, high efficiency portable units range in purchase cost from about $200 to $1250 depending on the size of the room or space to be treated and the specific technologies included (e.g., MERV rating and charcoal or other VOC removal filters) and would typically not involve any installation costs. Replacement filters and maintenance range from about $75 to $500 per year, again depending on the types of filters included and how dirty the air is, which would determine the frequency of filter changes needed. To adequately treat the living areas of most homes (e.g., bedrooms, family room, living room), two or more portable units may be needed.

## External Building Design Measures

### Moving Air Intakes

Research focused on assessing external building design measures is generally not readily available. Locating air intakes for mechanical ventilation systems on the opposite side of the building from the nearby outdoor source and prevailing wind direction seems logical. However, the reduction of pollutant entry in such a case would depend on the distance of the intake from the outdoor source, the consistency of the prevailing wind direction, and any local geographical or structural objects that might produce wind turbulence or eddies near the building and the air intake. One particle expert has noted that moving the intake would likely only be beneficial when the outdoor source is very near the intake and the intake is moved fairly far away; otherwise, because particles tend to disperse quickly and particle plumes “flow” around buildings, elevated particle concentrations around the building will be fairly consistent (Thatcher, 2010). This view appears at least partially substantiated by an Australian study that found that the concentration of submicron particles was consistently high and relatively undiluted around a building that was within 15 m of the roadway (Morawska et al., 1999). However, because this option has received little scientific study, and because all new California homes are required to use mechanical ventilation, which will often include a supply air intake, this option warrants further study to determine whether there are conditions under which strategic placement of air intakes might provide some benefits.

### Reducing Openable Windows

Reducing the size and number of openable windows on the side of the building nearest the outdoor source would likely do little to reduce entry of particles and other pollutants into homes. Furthermore, this potential measure may not be acceptable to homeowners, who often open windows to take advantage of the breeze, from which the benefit arises primarily from opening windows on the prevailing wind side of the building. Windows opened only on the opposite side may result in little air movement in the home. In regions of the State where window opening currently replaces air conditioning in the summer evening and nighttime periods, there could be substantial energy and cost penalties for the increased use of mechanical air conditioning to cool the home. Additionally, increased indoor air stagnation and condensation may occur, which can result in mold issues. Thus, for all of these reasons, this option does not appear practical for single family dwellings. This measure might be acceptable in multi-family dwellings, depending on the specific building design and the ventilation systems used. However, inclusion of a sufficient number of windows (even if unopenable) would allow more daylight into the building, which would reduce energy use for indoor lighting and provide the satisfaction and efficiency benefits that accompany daylighting (Heschong Mahone Group, 2003a, 2003b).

### Taller Buildings

Housing people in taller buildings has also been suggested as a possible exposure reduction measure. However, one of the few relevant studies of multi-story buildings near busy roadways found that vertical differences in fine and ultrafine particle concentrations outside buildings with 9 to 26 stories were not significant and can be highly variable, depending on other local sources and local meteorological conditions (Morawska et al., 1999). A second study, conducted in New York, found significant decreases for outdoor black carbon and non-volatile polycyclic aromatic hydrocarbons for floors 6 to 32 during the non-heating season only (Jung et al., 2011). Additionally, floors 3 to 5 showed the highest median outdoor concentrations for all pollutants measured, although the trend was not statistically significant and the elevated pollutants were believed to come from nearby rooftop exhausts. Thus, multi-story housing may reduce exposure in some situations but requires further research to determine conditions under which tall buildings might provide a reliable approach to reduce exposure near busy roadways.

## Site-related Measures

The primary site-related measures reviewed by ARB staff were sound walls and vegetation barriers.

### Sound Walls

Sound walls appear to reduce pollutant concentrations near the roadway; near-road concentrations (within 15-20 m [49-66 ft]) have shown reductions up to about 50% (Ning et al., 2010; Baldauf et al., 2008; Bowker et al., 2007; Hagler et al., 2012). However, in some studies higher levels of pollution were seen behind the barrier and at a distance from the sound walls and roadways, although in some of these studies the higher levels

appear related to other sources of pollution (Ning et al., 2010; Bowker et al., 2007; Hagler et al., 2010; Baldauf et al., 2008). In one of the few field measurement studies of sound walls, conducted along two southern California freeways, Ning et al. (2010) found that concentrations at farther distances (about 80 to 100 m from the roadway) were typically greater for the portions of the roads with sound walls, and background levels behind sound walls were not reached until 250 to 400 m as compared to 150 to 200 m without sound walls. Modeling and tracer studies (Heist et al., 2009; Finn et al., 2009) showed that barriers reduced air pollution downwind of the barrier, although in some cases trapping of pollution and increased levels on the road would occur (Hagler et al., 2011; Finn et al., 2009). Nearby buildings and structural barriers can also affect the attenuation and dispersion of pollution from roadways, but results vary with different meteorological conditions (Bowker et al., 2007; Hagler et al., 2010; Hagler et al., 2012).

### Vegetation Barriers

Results for vegetation alone are more variable than those for sound walls. Vegetation can remove some gaseous pollutants by uptake or absorption, and particles are removed primarily by interception (impaction or physical adherence; Nowak et al., 2006; Fujii et al., 2008; Smith, 1990; Pardyjak et al., 2008; Baldauf et al., 2008). However, particles can be resuspended, apparently even at very low wind speeds (Fujii et al., 2008; Smith, 1990). Vegetation may restrict dispersion and increase concentrations on-road in street canyons with closer spacing of trees, particularly in low wind conditions (Gromke, 2011; Gromke and Ruck, 2007, 2009; Buccolieri et al., 2009). Another study has further shown the complexity of the effects of vegetation; investigators found different results depending on particle size and wind speed, and a non-linear increase of particle removal with increased leaf area density, which varies by tree species and season (Steffens et al., 2012). Gaps in vegetation barriers can have a significant negative impact on their effectiveness (Hagler et al., 2012), which needs to be addressed in future California research because California roadside vegetation tends to be less dense than that in the eastern U.S., where most previous field studies have been conducted. Also, some types of vegetation can trigger asthma and allergy attacks, and some emit reactive VOCs that contribute to the formation of ozone.

### Sound Walls and Vegetation Combined

A combination of sound walls and vegetation appears to be more effective than either one alone. The two used together have been shown to disperse pollutants more consistently and to greater distances than either alone, with up to about a 60% reduction in near roadway levels (Baldauf et al., 2008; Bowker et al., 2007). While sound walls alone and sound walls combined with vegetation show promise, the increase in concentrations on-road and at a distance seen in some studies can increase exposures of others in the population and thus redistributes, rather than removes, pollutants. Additionally, the complexity of pollutant movement under varying conditions makes accurate prediction of exposure reduction difficult. Specific conditions under which sound walls and vegetation can reliably and consistently reduce exposures to air pollution have not been identified, especially in California.

**Reduction of Indoor-generated Pollutants to Reduce Overall Exposure**

Particles, $NO_2$ and other pollutants emitted by vehicles and other outdoor sources also have indoor sources that can produce higher indoor concentrations at times (ARB, 2005b, Section 2, and sources cited). Therefore, a reduction in indoor emissions and exposures can reduce the overall health impact of exposure to outdoor pollutants because the total exposure (indoor plus outdoor) to those pollutants experienced by the building occupants would be reduced. A number of studies have identified unvented cooking, cigarette smoking, the use of unvented gas appliances, burning of candles and incense, and woodburning as indoor sources of fine and ultrafine particles (Bhangar et al., 2010; ARB, 2005b; Fortmann et al., 2001; Wallace, 1996; Wallace, 2005; Wallace et al., 2008). High fine and ultrafine particle counts have been measured from such indoor sources. In homes with such sources, average indoor concentrations and occupants' personal exposures to fine and ultrafine PM are dominated by those indoor sources. Thus, measures to reduce indoor sources can help to significantly reduce occupants' peak and overall daily exposures to key pollutants emitted from both traffic and indoor sources.

## Summary of Research Review

ARB has developed and adopted increasingly stringent regulations limiting emissions from passenger cars, trucks and buses, which have substantially reduced, and will continue to reduce, vehicle emissions. However, recently adopted regulations have compliance dates extending as far as 2025 for full implementation, and fleet turnover to zero or near-zero technologies will take 20 to 30 years. The set-back of buildings from high traffic roadways remains the most certain approach for preventing the residual health risk from traffic pollution exposures for those living closest to the roadways because it distances them from the highest pollutant concentrations. Research conducted since the publication of ARB's recommendations in 2005 further supports the use of set-back.

There are two mitigation measures that can be effective for exposure reduction. Increased filtration of air and reduction of indoor pollution sources potentially can reduce the overall pollution burden in homes. These measures warrant consideration especially in light of recent studies showing that the pollutant plumes at times can extend beyond 1000 ft (305 m) from the roadway. For most residential applications near busy roadways, high efficiency (MERV 13 to 16, or higher) pleated particle filters would generally be considered the most effective approach to filtration because they can remove the very small particles emitted by motor vehicles without emitting ozone, formaldehyde, or other harmful byproducts. Based on a limited number of studies, such high efficiency filtration has been shown to reduce indoor PM2.5 and ultrafine particle levels by up to 90% relative to incoming outdoor levels when doors and windows are kept mostly closed. Purchase costs for high efficiency filtration devices or systems that are compatible with residential mechanical ventilation systems (which are now required

in new residential construction in California) range from $200 up to $2800, but most are available for under $500. Because Title 24 now requires mechanical ventilation for new residential construction, enhanced filtration can help avoid increased exposures to outdoor pollutants that may occur. The use of high efficiency air filters in central heating and air systems or stand-alone air cleaning devices can also reduce exposures in existing homes and homes that use certain types of mechanical ventilation systems that cannot accommodate central filtration.

While research shows that high efficiency filtration can be effective, it has several limitations. Filtration cannot remove all incoming outdoor pollutants because of normal building leakage and the fact that most people open windows and doors at least a portion of the day, allowing entry of unfiltered air. Additionally, not all pollutants are filtered by the filter media. Moreover, studies show irregular homeowner maintenance of filters and central systems, and regular maintenance is critical for effective removal of pollutants. ARB is funding two studies that should help further identify the approximate reduction in exposure that high efficiency filtration can provide in homes. High efficiency filtration is already being used or is planned for use in over 70 schools in California; these programs should provide comparable information for high efficiency filtration in classrooms.

The benefits are less clear for most of the other potential mitigation measures examined. Studies have shown that the use of sound walls alone, or sound walls and vegetation together, can reduce near roadway concentrations by about 50% and 60%, respectively. However, the extent of exposure reduction is quite variable under different conditions of meteorology and topography, and increased levels of pollutants can occur on-road and at a distance from the roadway. Thus, unlike the situation with filtration, pollutants are primarily redistributed rather than removed; while individuals living near the roadway would benefit, those traveling on the road or living at a distance could experience elevated exposures at times. The effectiveness of vegetation alone is even more variable, and has not been well-quantified. Furthermore, vegetation with low allergenic potential and low reactive VOC formation needs to be identified and tested, and other limitations of vegetation as a pollution barrier need to be better understood. Research is needed that identifies the specific conditions under which sound walls and vegetation can consistently provide a reliable exposure reduction benefit with limited disbenefits. In particular, California field studies are needed because of the significant differences in California meteorology, building practices, and flora from those of the eastern U.S.

The limited studies conducted to date on other potential mitigation concepts are not promising, although further research may identify situations in which they are generally effective. Placement of air intakes on the side of the building opposite the roadway may make little difference in terms of exposure, due to rapid particle movement around buildings. Locating windows only on the side of the building opposite the roadway reduces indoor daylighting, air circulation and cooling, and may do little to reduce exposure. Finally, taller buildings do not necessarily experience substantially reduced pollutant levels at higher floor levels, depending on local meteorology and other nearby

sources of pollution. However, further research on placement of air intakes and housing in taller buildings may identify conditions under which these measures reliably reduce exposures. Research is warranted on these measures and the measures discussed above as effective or showing promise in order to further identify cumulative measures that together can assure sufficient exposure reduction and health protection for those living near busy roadways.

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# ADDENDUM

## *Current California Building Code Requirements*

Section 150(o) of Title 24 of the California Code of Regulations (CCR 2008) requires mechanical ventilation in all new residential construction in California built after January 1, 2010. Section 150(o) allows the requirement to be met through a variety of system types (CEC 2010). "Exhaust only" type systems increase the entry of unfiltered outdoor air through leakage points in the building shell and can result in negative pressure indoors, thus increasing the possibility of backdrafting of combustion emissions from gas water heaters, fireplaces and other combustion appliances. These are the most widely used systems in California. "Supply systems" typically use a small motor to bring outdoor air in through a ducted supply and can include high efficiency filters to filter the air as it is brought in, prior to circulation of the air throughout the home. Combination (supply and exhaust) systems are available, with some linked to the central heating and air system; these include filtration of incoming outdoor air. However, the Code requires only a MERV 6 air filter (an increase to MERV 8 is proposed in the 2012 revisions to Title 24), which does not remove the smaller particles emitted by vehicles which are the particles of greatest concern. In future construction, the type of mechanical system used in new homes will have a major impact on the entry of outdoor pollutants indoors − if filtration is not included or is weak, indoor exposures to outdoor pollutants likely will increase.

**Table 1. MERV Ratings***

| MERV Rating | Average Particle Size Efficiency (PSE), microns – % Removal | | | Typical Controlled Contaminant or Material Sources (ASHRAE 52.2) | Typical Building Applications |
|---|---|---|---|---|---|
| | **0.3-1.0** | **1.0-3.0** | **3.0-10.0** | | |
| 1-4 | | | <20% | **> 10 Microns**<br>Textile Fibers<br>Dust Mites, Dust, Pollen | Window AC units<br>Common Residential<br>Minimal Filtration |
| 5 | | | 20-35 | **3.0 to 10.0 Microns**<br>Cement Dust, Mold Spores, Dusting Aids | Industrial Workplace<br>Better Residential<br>Commercial |
| 8 | | | >70 | | |
| 9 | | <50 | >85 | **1.0 to 3.0 Microns**<br>Legionella, Some Auto Emissions, Humidifier Dust | Hospital Laboratories<br>Better Commercial<br>Superior Residential |
| 12 | | >80 | >90 | | |
| 13 | <75 | >90 | >90 | **0.3 to 1.0 Microns**<br>Bacteria, Droplet Nuclei (sneeze), Most Tobacco Smoke, Insecticide Dust | Superior Commercial<br>Smoking Lounge<br>Hospital Care<br>General Surgery |
| 16 | >95 | >95 | >95 | | |
| 17** | ≥ 99.97 | | | **<0.3 Microns (HEPA/ULPA filters)****<br>Viruses, Carbon Dust, Fine Combustion Smoke | Clean Rooms<br>Carcinogenic & Radioactive Matls.,<br>Orthopedic Surgery |
| 18** | ≥ 99.99 | | | | |
| 19, 20** | ≥ 99.999 | | | | |

* Adapted from EPA 2009; originally from ANSI/ASHRAE Standard 52.2-2007.
** Not part of the official ASHRAE Standard 52.2 test, but added by ASHRAE for comparison purposes.

# EXHIBIT 5

# IDENTIFYING URBAN DESIGNS AND TRAFFIC MANAGEMENT STRATEGIES FOR SOUTHERN CALIFORNIA THAT REDUCE AIR POLLUTION EXPOSURE

Contract #12-308
Final Report

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# i. Abstract

This study attempts to explain explicitly the direct and quantitative effects of complicated urban built-environment on near-road dispersion and levels of on-road air pollution at scales from a few meters away from the center of the street to several city blocks. This was studied using ultrafine particle concentrations ([UFP]) as a surrogate for on-road air pollution, as it is an excellent proxy for motor vehicle emissions at short time scales. We emphasize built-environments that arise around transit, transportation mode shifting, and transit oriented developments (TODs); types of development that are needed to move California communities toward improved public health combined with SB 375 goals of sustainability. Transit oriented developments are a type of community development that combines is a type of mixed-use community development that includes housing, office, retail and/or other amenities integrated into a walkable neighborhood, located within a half-mile of quality public transportation.

Seven measurement sites in the greater Los Angeles area with different built-environments but similar mesoscale meteorology were explored. At the sub-block scale, the data was used to explore the decay of air pollution away from intersections to find the optimal location of transit stops relative to the intersection, which lies about 40 m away from the center of the intersection. We also developed a detailed statistical micro-dynamics model that is able to closely reproduce second-by-second observations of UFP, and can disentangle the contributions of on-coming and on-going traffic, light and heavy-duty vehicles, traffic movements, building heights and other related features that impact pedestrian exposure. Next, we developed an approach to address several of the challenges associated with making high-resolution maps from mobile air pollution concentration measurements, including the question of how many repeats of a mobile monitoring route are needed to determine representative concentrations.

We find that the largest impact of the built environment is at the scale of several blocks. On the scale of a small neighborhood (several large city blocks), we find that after controlling for traffic, for most sampling days and sites, [UFP] were higher in the morning than those in the afternoon due to limited dispersion capacity combined with a relatively stable surface layer. In the calm mornings, the areal aspect ratio ($Ar_{area}$) developed in this study for real urban configurations showed a strong relationship with block-scale UFPs. $Ar_{area}$ includes the building area-weighted building height (or effective building height), the amount of open space, and the building footprint. In the afternoon, however, when wind speeds were generally higher and turbulence was stronger, the standard deviation of vertical velocity fluctuations, $\sigma_w$, was the most effective factor controlling [UFP]. The surrounding built environment appears to play an indirect role in observed [UFP] by affecting surface-level micrometeorology. The effects are substantial; controlling for traffic, differences in $Ar_{area}$, and building heterogeneity were related to differences in [UFP] by factors of two to three among the five study sites. These results have significant implications for pedestrian exposure to motor vehicle emissions as well as transit-oriented urban planning.

Moving to the scale of a single street, part of the study focused on street canyon-like urban built-environments. This work suggests that for this specific configuration, street-level concentrations of vehicle-related air pollutants can be estimated with a model that assumes that vertical turbulent transport of emissions dominates the governing processes. We developed a semi-empirical Vertical Dispersion Model (VDM), to describe the data collected in street canyons located in Hannover, Germany and Los Angeles. VDM indicates that magnification of concentrations relative to those in the absence of buildings is most sensitive to the aspect ratio of the street (the ratio of the effective building height to the street width). VDM estimates also indicate that the presence of the buildings in a street canyon configuration can potentially magnify street-level concentrations by as much as a factor of 3.5 relative to those in the absence of buildings. We translated the VDM equations into an

easy to use spreadsheet tool and python code that allows city planners to use VDM to conduct sensitivity analysis, generate concentration estimates, and develop mitigation strategies that aim to reduce the pedestrian exposure to air pollution within TODs. This tool is made available through the California Air Resources Board.

# ii. Executive Summary

This report investigates the impact of the built environment on concentrations of roadway pollutants, specifically ultrafine particles. The report emphasizes the configurations that arise around transit, mode shifting and transit-oriented development, and types of development that are needed to move California communities toward improved public health combined with SB 375 goals of sustainability. As higher density communities and transit-oriented developments are built, there is potential to create situations that expose more people to more roadway emissions. We seek to understand features of the built environment that may be adjusted to avoid or mitigate potential unintended consequences. Built environment effects are considered on several different scales: (1) sub-street scale within few meter of a road, (2) street scale looking at entirety of a single street, and (3) multi-block scale that spans over several blocks. The analyses were based on extensive field measurements made in several communities in the Los Angeles area during 2013, 2014 and 2015, most of it at high spatial and temporal resolution. In some cases, other datasets were used either from a 2008 study, also in Downtown Los Angeles and also supported by CARB, or from a longer term study in Hanover, Germany performed by other investigators. The communities in the Los Angeles area included four sites in Downtown Los Angeles, and sites in Temple City, Beverly Hills and Koreatown.

Exposure to elevated levels of roadway pollutants has been associated with a wide range of adverse health outcomes. Freshly emitted vehicular pollution is a complex mixture of gases and particles, of which ultrafine particles (UFP) is a major component. While the components of fresh vehicle emissions that cause adverse health effects is not well established, UFP are both an excellent proxy for roadway emissions and may potentially be a significant contributor to roadway-related toxicity. The analyses presented below begin at the sub-street scale and conclude at the multi-block scale. A summary for urban planners and policymakers is also provided.

## ii.1. Sub Street-Scale Results

### ii.1.1. Siting Transit Stops: Decay of Pollutants around Intersections

We investigated the characteristics of cross-intersection concentration profiles of ultrafine particles (UFP) with 5 m spatial resolution. This was performed using 1,744 profiles covering 90 m before and after the center of each intersection. Cross-intersection UFP profiles were measured with a mobile monitoring platform at 10 signalized intersections at six urban sites with distinct built environments during both mornings and afternoons. Measurements were made within 1.5 m of the sidewalk at breathing height (1.5 m above ground level) to approximate sidewalk exposures. UFP profiles were strongly influenced by high emission events from accelerating vehicles and showed elevated concentration peaks within 30 m of intersection centers followed by sharp decreases in concentrations with distance. The elevation of UFP near the intersection was accompanied by more frequent and larger transient concentration spikes. Thus, people that stay longer at the intersection have an increased chance of being exposed to these short-term extremely high concentrations. The concentrations decay to somewhat lower levels before the intersection (the ‘near’ side), than after the intersection (the ‘far’ side). However, as siting transit stops after intersections is preferred for smooth traffic flow, we focus on the ‘far’ side. Simple time-duration exposure calculations suggest moving a bus stop from 20 to 40 m to after the intersection reduces transit-users’ exposure levels to the total UFP inversely proportional to the elevation magnitude near the intersection, by an amount that varies widely, from hardly reducing exposure to cutting it by more than half.

### ii.1.2. Development of a Statistical Model to Explain Micro-dynamics of Pollutant Concentrations on Roadways

We developed a novel micro-modeling approach to quantify the impacts of the factors controlling concentrations of roadway pollutants at the time-scale of seconds and spatial scale of meters. The statistical model characterizes how UFP concentrations vary around a Mobile Measurement Platform (MMP) as it travels along an urban transect in downtown Los Angeles. The data we use to estimate our statistical model comes from 11 MMP sampling transects on Broadway Street in and near downtown Los Angeles in 2008. Our model includes factors such as the state of motion, speed and land position of the MMP, the number of on-going and on-coming light duty (light-duty vehicles and motor-cycles), heavy duty (trucks), and buses encountered by the MMP along the transect. We also characterize the built environment adjacent to the MMP, including adjacent building heights and the presence of intersections. When the MMP was stopped at intersections, we measured the queuing time and queue position as well as the number and type of vehicles crossing in front of the MMP on the perpendicular street. We further characterized the acceleration events for the platoons of vehicles traveling in the on-going and on-coming directions as well as those crossing the intersection from the left or right. Finally, our model includes wind direction and speed as well as the time of day when the transects occurred.

The model is designed to expressly provide valid attribution of UFP concentrations to specific factors in the presence of many other potentially confounding factors. For example, we are able to characterize the effect on UFP concentration of the number and type of passing vehicles in both an on-going or on-coming direction, while controlling other factors. This high-resolution modeling framework is especially helpful at intersections to decompose the respective multi-directional traffic events (stopping, queuing, and accelerations) on UFP concentrations. We also show how our model can be used to characterize free-flow traffic patterns versus the stop-and-go dynamics along a transect.

For each of the factors discussed above, we estimate the average effect (direction and size) of the traffic event on the UFP concentrations, decomposing these average effects into their temporal profile, which may include important sign and magnitude changes, resulting from opposing effects such as vehicle-induced turbulence and emissions, both of which often increase with increasing vehicle size. The model reproduces the data very well. The most important factors are the type of vehicle and its activity (on-coming, on-going accelerating, crossing etc.).

### ii.1.3. Mapping Pollutant Concentrations in Urban Areas

Mobile air pollution monitoring offers an opportunity to “map” pollutants with much higher spatial resolution than sparse stationary monitors, but the data they produce presents some unique challenges. They also raise the important question of how many repeats along a given route are needed to determine representative concentrations along the route. We develop a framework to address the challenges and constraints to developing higher spatial resolution maps from mobile data. The challenges include the non-uniform spatial resolution and distribution of the measurements; that measurements are made at slightly different locations in each pass of the mobile monitoring platform along a specific route (each “run”); in some cases, the poor precision of global positioning system coordinate data; potential for over/underweighting data; and varying urban background concentrations. We find that use of a reference grid and piecewise cubic Hermite spline interpolation between measurements to give equal weight to each sampling “run” at each grid reference point addresses many of the challenges effectively. A background correction was implemented to facilitate averaging over several sessions. For 1 s time resolution data collected at normal city driving speeds, we show that concentration maps of 5 m spatial resolution can be obtained by including up to 21% interpolated values. Finally, we use UFP concentrations to consider the minimum number of sampling runs needed to make a representative concentration map

with a specific spatial resolution, finding that generally between 15 to 21 repeats of a particular route under similar traffic and meteorological conditions is sufficient.

## ii.2. The Multi-Block Scale

The built environment at the multi-block scale has a markedly larger influence than it does at the street scale. In our multi-block scale study, we attempt to explain explicitly the direct and quantitative effects of complicated urban built-environment on near-road dispersion and levels of vehicular emissions at the scale of several city blocks (here, the "multi-block scale", again based primarily on ultrafine particle concentrations. For this study, we used five measurement sites in the greater Los Angeles area, with different built environments but similar mesoscale meteorology. The built environments varied from one with all 1-story buildings, one with a clearly defined street canyon, and three sites with very heterogeneous morphology characterized by a mix of one or two very tall buildings and low developments (parking lots or parks). After controlling for traffic throughout the study area, for most sampling days and sites, morning UFPs were higher than those in the afternoon due to limited dispersion capacity combined with a relatively stable surface layer in the morning. In the calm mornings, $Ar_{area}$ developed in this study for real urban configurations showed a strong relationship with multi-block-scale average UFP concentrations. $Ar_{area}$ includes the building-area-weighted building height, the low development, and the building footprint. In the afternoon, however, when wind speeds were generally higher and turbulence was stronger, vertical turbulence intensity $\sigma_w$ was the most effective factor controlling the concentrations of vehicular pollution. The surrounding built environment appears to play an indirect role in observed UFP concentrations by affecting surface-level micrometeorology. The effects are substantial; controlling for traffic, differences in $Ar_{area}$, and building heterogeneity were related to differences in UFP concentrations of factors of two to three among our five study sites.

## ii.3. Street Canyon Dispersion Modeling

We developed a semi-empirical dispersion, referred to as the Vertical Dispersion Model (VDM), to describe data collected in street canyons located in Hannover, Germany and Los Angeles, USA. The data collected in Hannover indicated that street-level concentrations of vehicle-related pollutants are governed by vertical turbulent transport of emissions. The analysis of measurements made in field studies conducted in Los Angeles showed that the effects of urban buildings on pollutant dispersion can be parameterized in terms of the effective aspect ratio of the street, which is the ratio of the frontal area weighted height of buildings facing the street to the width of the street. The dispersion model, VDM, relates roof-level concentrations to street-level concentrations using traffic flow rate, the effective aspect ratio of the street, and roof level turbulence as inputs. This formulation allows VDM to uses outputs from commonly used models such as AERMOD to estimate street-level concentrations in urban areas. Thus, this model can be used by urban planners to examine the impact of alternate TOD designs on street level concentrations associated with near surface emissions. We illustrate the application of VDM by estimating street-level concentrations of ultra-fine particles at the locations in Los Angeles where we conducted field studies. VDM estimates indicate that the presence of the buildings can potentially magnify street-level concentrations by as much as a factor of 3.5 relative to those in the absence of buildings.

We translated the VDM equations into a spreadsheet tool and python code that allows city planners to use VDM to conduct sensitivity analysis and generate concentration estimates. The spreadsheet tool is useful for development of mitigation strategies that aim to reduce the effective street aspect ratio or reduce emissions within the TOD through traffic management. The tool is made available through the California Air Resources Board.

## ii.4. Summary for Planners

Results from this study that are relevant to urban planning around transit and transit oriented development are summarized in a table below. These measures are not exclusive and should be considered in concert with other measures supported by related research and established guidelines, such as those requiring siting of sensitive population at least 500 feet from freeways, and statewide goals to reduce greenhouse gas emissions from transportation by reducing vehicle miles travelled and supporting development that will make it possible to replace car trips with bike, walking, and transit trips.

We emphasize that purpose of the table below is to assess the effects of specific features of the built-environment and what parameters should be considered when designing a TOD. This information should be considered in conjunction with other strategies not discussed here in planning decisions, including considerations of the vehicle emissions reduction, effective strategies to reduce vehicle miles traveled, and other factors.

In summary, this study suggests several strategies in developing a TOD to reduce exposure to pedestrians:

- Avoid development of high rises in close proximity
- Reduce building density (areal aspect ratio) by including open spaces, such as parks and parking lots, among the building environment
- Separate pedestrian walkway from streets and intersections expected to have substantial on-road traffic
- Develop transit systems to reduce on-road traffic during early mornings and late evenings
- Siting bus stops away from major on-road sources and intersections

Additionally, this study suggests several mitigation methods in existing TOD to reduce exposure to pollutants in urban areas:

- Develop a mitigation strategy to reduce vehicle traffic on streets with large aspect ratios
- Develop a mitigation strategy to divert pedestrians away from streets and intersections with heavy traffic
- Develop a mitigation strategy to divert pedestrians away from streets with large aspect ratios

Detailed information that provides evidences to these concluding statements can be found in the following report.

**Table for ii.4. General recommendations to reduce pedestrian and residential air pollution exposure in built environment.**

| Management | Suggested Direction | Approx. Size of Effect | Atmospheric Conditions & Notes |
|---|---|---|---|
| Areal aspect ratio ($Ar_{area}$), which combines building area-weighted height, building footprint, and the amount of open space | Lower building volumes and more open space result in lower pollutant concentrations. | The difference between very dense and low density built environments is approximately a factor of three. | Important under calm conditions (in the mornings at our sites). |
| Building Heterogeneity | Isolated tall (high-rise) buildings result in lower concentrations than homogeneous shorter or many taller buildings with similar volume. | Highly heterogeneous built environments can decrease concentrations by up to approximately a factor of two relative to completely homogeneous built environments. | Important under unstable conditions with moderate winds (afternoons at our sites). Not critical when the atmosphere is stable. |
| Street Canyons (relatively contiguous walls of buildings) | Heterogeneous building forms avoid hotspots created by street canyons. | Tall street canyons (~50 m) can increase local traffic air pollution concentrations by up to about 50% relative to open space. | |
| Bus Stop Siting | Siting bus stops further from intersection will reduce exposures. | From no effect to more than a factor of two reduction from moving the site from 20 to 40 meters from the intersection on the "far" side. | Pollutant concentrations usually peak near the center of the intersection, although there is a high degree of variability. |

## iii. Publication from this study

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DeShazo, et al. (2017) Modeling the Micro-dynamics of Ultrafine Particle Concentrations Caused by Traffic Events at Street Intersections.

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# List of Abbreviations

| | |
|---|---|
| ARB/CARB | California Air Resources Board |
| AGL | above ground level |
| BW | Broadway |
| CPBM | Canyon Plume Box Model |
| CPC | condensation particle counter |
| DTLA | Downtown Los Angeles |
| HEV | high emitting vehicle |
| IBL | internal boundary layer |
| LA | Los Angeles |
| MMP | Mobile Monitoring Platform |
| OSPM | Operational Street Pollution Model |
| SB375 | Senate Bill 375, the Sustainable Communities and Climate Protection Act of 2008 |
| TC | Temple City |
| TOD | transit oriented development |
| UBL | urban boundary layer |
| UCL | urban canopy layer |
| UFP | ultrafine particles |
| VDM | Vertical Dispersion Model |

# iv. Introduction

Air pollution emissions from motor vehicles are spatially-heterogeneous in urban areas. A large fraction of an individual's exposure to air pollutants can be attributed to relatively short periods of time spent on and near roadways, which often have highly elevated air pollutant concentrations compared to areas even at moderate distances away from roadways (Behrentz et al. 2005; Fruin et al. 2004; Marshall et al. 2005). However, because of the lack of adequate air pollutant measurement data near local roadways, studies of health effects attributed to transportation-related air pollutants have generally used proximity to freeways or arterial roadways as a surrogate for vehicle-related air pollution (Brugge et al. 2007; Ren et al. 2008; Volk et al. 2011; Zhou and Levy 2007). Despite this generalization, near roadway air pollution studies have shown moderate increases in a long list of adverse health outcomes, including increased incidence of cancer (Pearson et al. 2000), asthma (Janssen et al. 2003), general mortality (Hoek et al. 2002), heart attacks (Tonne et al. 2007), autism (Volk et al. 2011), pre-term birth (Ren et al. 2008) and other adverse outcomes associated with air pollution exposure near roadways.

While California has made tremendous progress in reducing vehicular emissions, evidence of the dangers of roadway air pollutant exposure is growing, highlighting the need to ensure that implementation of regional and local sustainability strategies under programs like Senate Bill 375 (SB 375) are consistent with ARB's criteria and toxic pollution exposure reduction policy goals. SB 375 requires Metropolitan Planning Organizations (MPOs) to develop Sustainable Community Strategies (SCS) as part of their Regional Transportation Plan to demonstrate how they will achieve regional greenhouse gas reduction (GHG) targets. Many MPOs are adopting Sustainable Communities Strategies that seek to direct new development and population growth to transit corridors. For example, the Southern California Association of Governments has adopted its first Sustainable Communities Strategy in compliance with SB 375 in which they propose placing more than 50 percent of new growth in High Quality Transit areas or transit oriented development.

Transit oriented developments (TODs) are built environments in which high density residential developments are located close to public transportation and local businesses. TODs have the potential of improving environmental sustainability by reducing emissions associated with transportation, and promoting healthy activities such as walking and cycling (Boarnet and Crane, 2007). TODs are being promoted to reduce GHGs and air pollutant emissions associated with transportation because these environments are associated with reduced use of personal cars. TODs are also expected to reduce concentrations of air pollutants averaged over city scales. However, there is concern that high-density development will instead increase exposure of pedestrians, residents and other users of TODs due to increased densities of vehicle emissions and building morphologies associated with higher density development. Therefore, policies to encourage greater residential density around transit corridors may lead to the unintended effect of greater pedestrian exposure to roadway air pollutants. This problem may be especially acute at public transit stops deliberately located on high-volume arterial roadways to increase the passenger connectivity, accessibility, and multi-modal travel. As a result, the traditional policy response has been to move pedestrians and residents away from roadway emissions, a less feasible approach within many transit environments.

Moreover, actual exposure of pedestrians within such transit environments depends upon several factors including air pollution dispersion dynamics that vary with site-specific street-building morphologies, transit-stop locations, wind speed, temperature, and spatial proximity and intensity of emissions, traffic management, as well as, potentially, pedestrian enclosures, vegetation, and other features of the built environment. Transportation and urban planners routinely make an array of decisions that may affect pedestrian and residential air quality. With respect to local emissions

sources, transportation planners implement local traffic controls (such as signals, stoplights, and maximum speeds) on the transit arterial roadways that determine spatially where vehicles accelerate and how fast they travel. At the neighborhood scale, these planners make routing decisions that influence local traffic volume and composition on these same transit arterial roadways. These decisions together determine the precise location and timing of air pollutant emissions within a transit-stop environment. Despite the growing literature on near-roadway exposures, little is known about how to proactively design and plan for these transit environments to minimize air pollution exposures. In addition, models currently being used by air quality planners at regional levels have undergone limited evaluation with data under stable atmospheric conditions that typically represents urban built environments. To effectively evaluate future TODs, there is a need to develop a tool that can help planners better understand and evaluate how California's building morphologies affect pedestrian exposures, and a need to develop a guideline that can help the planners design the most optimal TODs to reduce pedestrian exposure to harmful air pollution emissions. To address these concerns, this study was constructed to address the following objectives:

1) Develop a guideline for TOD planners to reduce pedestrian exposure to air pollution in urban built environments by extending the qualitative understanding of the influence of the built environments on street level air pollution concentration through field measurements and statistical modeling
2) Develop a dispersion model that can be used to provide TOD planners quantitative links among the variables that control dispersion in complex urban environments and how best to reduce air pollution exposures in said environment

Our study seeks to help develop tools to minimize these exposures by advancing the understanding of factors controlling spatially-variable pollutant concentrations around roadways. The products include a rich measurements data set, and a refined predictive atmospheric dispersion model for the complex urban landscape. Further, these are used together with available literature to provide decision-support tools and information for transportation and urban planners to protect public health, pursuant to ARB's mission and goals of protecting the public from harmful exposure to air pollution and reducing GHGs. Specifically, the report disseminates evaluative and remedial decision-support tools for planners that focus on more effectively siting transit stops, managing intersection traffic flow, and shaping new TODs to reduce pedestrian air pollution exposure.

This study focuses on design and operational features that may both play a major role in determining pedestrian air pollution exposure and could be modified through local planning and transportation decisions. These meta-features include: 1) traffic volume, composition, and traffic calming strategies, 2) building heights around the arterial roadway, and 3) building set-backs from the arterial roadway. The result are summarized as tools and guidelines that transportation and urban planning decision makers can use to guide everyday decisions that impact the exposure of pedestrians in TODs.

The project uses a three-pronged approach combining extensive real-time measurements of air pollutants such as ultrafine particles, statistical analysis of real-time data and its relation to the morphology of the built environment and traffic management strategies, and development of a dispersion model for evaluating the impact of built-environment design features. Measurement data were collected with a mobile measurement platform, which provided a rich data set by repeatedly sampling for brief stationary periods in a dense matrix of locations on three-to-four approximately mile-long "transects". These measurements were augmented by additional instrumentation to characterize micro-meteorology and pollutant levels aloft.

The data were also combined with existing street-level pollutants measured using mobile measurement platform, and was used to explore relationships between building morphology, traffic

management strategies, and pollutant concentrations on length scales of several meters away from the roadway. To provide predictive ability and explore the potential improvements to pollutant levels that may be anticipated from potential land use changes, a semi-empirical model was developed. The Operational Street Pollution Model (OSPM) developed by the Danish National Environmental Research Institute (Berkowicz et al., 2000) was used as the starting point for this modeling exercise. OSPM incorporates the main features of our current understanding of dispersion in street canyons, and has undergone evaluation with real-world observations made in European cities (Berkowicz et al., 1997, During et al., 2011). However, this study indicated that OSPM did not account for the inhomogeneous building environments of typical U.S. cities, such as Los Angeles, in which tall buildings are interspersed among two or three story buildings and open spaces. This necessitated the development of a model specific for California conditions. Collectively, this research will identifies and quantifies the impacts of alternative policy solutions that have been less recognized but promises to reduce pedestrian air pollutant exposures in transit environments.

The report is presented in two sections that describe the methodology, and the results from the two different analytical approaches used to explain the complex data:

- Field Measurements and Statistical Modeling
- Dispersion Modeling

The results from the two approaches reinforce each other. The results from the statistical approach provide semi-quantitative guidance on the design of built environments to reduce exposure to vehicle related pollutants. The dispersion model translates the data from the field studies into a practical tool that can be used by planners to estimate the quantitative impact of strategies to reduce exposure to these pollutants.

# v. Field Measurements and Statistical Modeling

## v.1. Introduction

Fresh vehicular emissions contain a wide range of particle- and gas-phase species, including carbon monoxide, nitric oxide, a variety of volatile and semi-volatile organics, and ultrafine particles. Because such emissions are emitted and diluted together, their individual impacts are difficult to separate. In general, the best tracer of near roadway air pollution is ultrafine particles (UFP) as it is most prominently produced during burning of fuels in internal combustion engines. UFP can be measured reliably with 1 second time resolution with wide dynamic concentration ranges (5 – 6 orders of magnitude). As dispersion near intersections and in city blocks takes place in seconds rather than minutes, the high time resolution is essential in understanding pedestrian exposure to air pollution emissions. Additionally, because UFP are incorporated into larger particle sizes on relatively short time scales under dynamic nucleation events, in urban areas UFP tend to have steady background concentrations upon which the impact of local sources (such as motor vehicles) are extremely clear. Actual exposure of pedestrians in transit-served environments depends upon several factors including air pollution dispersion dynamics that vary with site-specific street-building morphologies, transit stop locations, wind speed, temperature, spatial proximity and intensity of emissions, and traffic management, as well as pedestrian enclosures, vegetation, and other features of the built environment. Despite a growing literature on near-roadway exposure, little is known about how to proactively design and plan for these transit-served environments in order to minimize air pollution exposure.[1]

Transportation and urban planners routinely make an array of decisions that may impact pedestrian and residential air pollutant exposures.[2] With respect to local emissions sources, transportation planners implement local traffic controls (such as signals, stop lights and maximum speeds) on the arterial roadways that determine spatially where vehicles accelerate and how fast they travel. At the multi-block scale, these planners make routing decisions that influence local traffic volume and composition on these same transit arterial roadways. These decisions together determine the precise location and timing of air pollutant emissions within a transit-stop environment. Through building, zoning, and street design ordinances, urban planners' decisions influence the height and spacing of buildings as well as how far setback they are from the arterial roadway. The resulting street-building morphology around a transit stop then interacts with the prevailing winds (and other meteorological conditions) to determine whether, and how quickly, emissions are dispersed.

It is, therefore, desirable to develop a set of comprehensive recommendations on how to reduce pedestrian and residential air pollution exposures that transportation and urban planners can reference when making future development plans. These may include traffic controls and urban building configuration, which impact emissions and dispersion, respectively. Within the transit environment, urban planners also decide where pedestrian density will be greatest through decisions about where to site transit stops, sidewalks, and parks.

Several studies investigating the influence of the built environment on street level concentrations have been published, mostly focusing on deep street canyons. Three recent studies have taken the

[1] We recognize there are best design practices such as enclosed/covered bus stops, closed canopy pedestrian byways, pedestrian byways below or above ground, and urban forest canopy parks, but these practices were not evaluated in this study. See Zhou and Levy, 2007; Brugge, et al., 2007; Karner et al., 2010 for a meta-analysis of this literature as it relates to near-roadway exposure.

[2] We focus here on exposures to pedestrians, but the findings of our research will also inform exposure to residents in transit-adjacent housing (Marshall, et al., 2005; Zhu, et al., 2005).

first step toward understanding dispersion of traffic-related pollutants in urban areas with inhomogeneous building morphology, which is consistent with the focus of this study (Boarnet et al. 2011; Buonanno et al. 2011; Karra et al. 2011). Buonanno et al. (2011) focused on particles, including UFP, measured in a town in central Italy; Karra et al. (2011) studied dispersion of CO in Nicosia, Cyprus; and Boarnet et al. (2011) examined $PM_{2.5}$ in downtown Los Angeles, California. Boarnet et al. (2011) further examined the factors governing $PM_{2.5}$ concentrations measured on sidewalks next to arterial roadways in five cities in southern California. The measurements indicated that the most effective controlling factors for sidewalk $PM_{2.5}$ concentrations are daily variations, time of day, wind speed and direction, and temperature. They also found that traffic and built environment variables, while statistically significant, accounted for only a small amount of the observed variation; however, it should be noted that their built environment variables were classified rather than quantified. After accounting for these most effective controlling factors, they concluded that street canyons with higher than 5-story buildings are related to high $PM_{2.5}$ concentrations, and adjacent paved lots were negatively associated with $PM_{2.5}$ concentrations. Boogard et al. (2011) conducted an extensive study in the Netherlands in which five species, including particle number concentrations and black carbon, were measured over 6 weeks at 8 urban roadside locations in five cities. Although their results did not discern the roles of meteorology and emissions, the two streets with buildings lining one or both sides of the street showed the largest road contributions.

While these studies provide insight into air pollution in built environments, the measurements lack the spatial resolution and completeness to discern contributions of detailed urban morphology and traffic control at a level that could inform highly-local planning decisions about the built environment and traffic flow. Minimizing exposure to transportation-related air pollution is not fully considered currently in the process of planning for TODs (Haughey and Sherriff 2010).

There are several relevant spatial scales that must be investigated to fully understand the impact of the built environment on near-road pollutant concentrations. Here, we focus on a spatial scales ranging from a few meters away from the roadway to several city blocks that span over a network of local roads. We develop quantitative links among the variables that control dispersion in complex urban environments, including building morphology, traffic flow rates, and micrometeorology using field measurements and statistical modeling. We consider data from five sites in the greater Los Angeles area, each with similar vehicle fleet composition and similar meteorology, but markedly different built environments and traffic flow patterns. Ambient air measurements were performed both in the early morning and in the mid-afternoon, which have significantly different atmospheric stability and wind profiles.

## v.2. Background and Related Work

### v.2.1. Current research needs for TOD planners

A growing number of studies are focusing on exposures to vehicle-related air pollutants in urban areas from various perspectives (Boarnet et al. 2011; Chan et al. 2002; Hagler et al. 2012; Kaur et al. 2007; Kinney et al. 2000; Ryan et al. 2008; Steffens et al. 2012; Tong et al. 2012). Several focus on exposure experienced by pedestrians, cyclists and automobile occupants (Adams et al. 2001a; Adams et al. 2001b; Kaur et al. 2007), and others examine the difference in air pollution exposure experienced on minor versus major thoroughfares (Boogaard et al. 2011). Researchers are also investigating the influence of barriers, including vegetation (Hagler et al. 2012; Steffens et al. 2012) and sound walls, on air pollution exposure (Baldauf et al. 2008; Bowker et al. 2007; Finn et al. 2010; Hagler et al. 2012; Ning et al. 2010). Most of these studies have focused on air pollutants that are not strongly correlated to freshly emitted roadway pollutants (such as $PM_{2.5}$). Instead, studies using roadway air pollutants that are strongly associated to fresh motor vehicle emissions, such as

UFPs, are becoming more common (Hagler et al. 2012; Kaur et al. 2007; Ning et al. 2010). Land use regression models, an alternative to dispersion models, have also been utilized to relate these field measurement data of roadway air pollutants to exposures in different micro-environments (Rivera et al. 2012; Ryan et al. 2008). These models are valuable in several applications, including epidemiological studies, as they are easier to relate to geo-coded health data. However, they do not have predictive capability to inform block-level building and traffic design decisions.

Nevertheless, the importance of the detailed characteristics of buildings along streets on air pollutant concentrations is clear. For example, Tong et al. (2012) conducted a modeling study using ambient air monitoring data (Patel et al. 2009) focusing on black carbon, an excellent tracer for diesel traffic, to perform an analysis of the influence of buildings surrounding a school located in close proximity to a highway. The authors concluded that exposure to black carbon would have been about half if the buildings located between the school and the highway had not been there. Additional studies by Boogaard et al. (2011) and Boarnet et al. (2011), as presented previously, also provide insight into the importance of roadside building characteristics on air pollution concentrations.

While these studies provide information on air quality in built environments, the measurements lack the spatial resolution and completeness to refine and valid dispersion models for realistic urban landscapes. In addition, these studies have not been designed to inform highly-local planning decisions based on the morphology of built environment and local traffic flow regimes. Minimizing exposure to transportation-related air pollution is currently overlooked in the process of planning for TODs (e.g., (Haughey and Sherriff 2010). Furthermore, no design tools are available for urban planners and transportation planners that incorporate the goals of minimizing air pollution exposures for residents and pedestrians in TODs.

### v.2.2. Overview: mobile platform studies

Instrumented vehicles, or mobile platforms, was first used in the 1980s and have been more widely implemented in the early twenty first century. They have been used for several research goals: (a) to measure air pollutant levels on-board vehicles (i.e., "in-cabin" concentrations) under realistic driving conditions; (b) measure air pollutant concentrations on roadways (rather than taking measurements alongside roadways from fixed sites); (c) to collect stationary ambient air measurements at a set of locations in close proximity to either a source (e.g., airports) or receptor of interest; (d) to characterize the decay of air pollutant levels with increasing distances away from roadways and other concentrated sources; (e) to look for "hot spots" and areas of anomalously elevated air pollutant concentrations in residential and other areas; and (f) to directly sample in-situ motor vehicle plumes ("chase" studies). Within the past five decades, there has been intense focus on measurement of UFPs because of its toxicity and its potential to affect human health. One of the direct antecedents for the present project, Westerdahl et al. (Westerdahl et al. 2005) and Fruin et al. (Fruin et al. 2008) utilized the California Air Resources Board's (ARB's) non-polluting (electric) mobile platform together with multiple scanning mobility particle sizers to measure UFP and associated air pollutant concentrations on freeways and residential streets in Los Angeles, California. These studies showed freeway on-road UFP concentrations to be largely driven by truck emissions while hard accelerations of gasoline-powered vehicles appeared to be the most common source of high UFP concentrations on arterials. In other in-vehicle studies of UFP, Miguel and co-workers (Zhu et al. 2007) conducted mobile monitoring in Los Angeles using a passenger car equipped with a high-efficiency particle arrestance (HEPA) filter system, including measurement of in-cabin and on-road measurements for both freeways and surface streets. Hitchins et al. (2000) and Kittelson et al. (2004b) also measured high concentrations of UFP on and near roadways. Several mobile monitoring studies that included UFP measurements have been conducted in Europe (Pirjola et al. 2004; Weijers et al. 2004) and in the eastern United States (Canagaratna et al. 2004; Kittelson

et al. 2004a). All of these studies demonstrate the usefulness of mobile platforms while supporting the notion that UFPs are useful tracer for motor vehicle activities.

### v.2.3. Overview: Studies investigating air pollution gradients near freeways

The existence of strong air pollution gradients near freeways have been recognized at least since the 1980s, with early studies focusing on gas phase pollutants (Rodes and Holland 1981). Hitchins et al. (2000) measured concentrations of fine and ultra-fine particles at a distance of 15 to 375 m away from a major roadway. They found that particle concentrations decayed to about half of the peak value (at the closest point to the roadway) at approximately 100-150 m downwind away from the roadway (Hitchins et al. 2000), which provides evidences of a sharp gradient of fine and ultrafine particles. Similar studies were also conducted by Zhu et al. (Zhu et al. 2002; Zhu et al. 2002a), who measured UFP, CO, and black carbon (BC) along the upwind (200 m) and downwind (300 m) sides of a freeway in Los Angeles during the daytime. Peak concentrations were observed immediately adjacent to the freeway, with concentrations of air pollutants returning to upwind background levels about approximately 300 m downwind of the freeway.

The few near-roadway studies conducted at nighttime indicated larger areas of impact than during the daytime. Nighttime UFP concentrations were reported by Zhu et al. (Zhu et al. 2006) , who conducted measurements upwind (300 m) and downwind (500 m) of a freeway from 22:30 - 04:00. Although traffic volumes were much lower at night, particle number concentrations were about 80% higher 30 m downwind of the freeway compared with the daytime, with UFP concentrations of ~50,000 $cm^{-3}$ approximately 500 m downwind of I-405, a major Los Angeles freeway. Fruin and Isakov (Fruin and Isakov 2006) measured UFP concentrations in Sacramento, California, near the US Highway-50 freeway between 23:00 and 01:00 and found 30-80% of maximum centerline concentrations (measured on a freeway overpass) 800 m downwind. These differences suggests that air pollution gradients near freeways can vary not only based on the on-road traffic densities, but also based on meteorology that affects mixing and dynamic nucleation events downwind of the source.

### v.2.4. Previous results from the Air Resources Board Mobile Platform in Southern California

A steady stream of recent results have been produced by researchers at ARB, UCLA and a handful of additional institutions using the same ARB-maintained mobile platform (Choi et al. 2013b; Choi et al. 2016; Choi et al. 2012b; Choi et al. 2014; Fruin et al. 2008; Hu et al. 2009a; Hu et al. 2009b; Kozawa et al. 2008; 2009; Paulson et al. 2012; Quiros et al. 2012; Westerdahl et al. 2005; Westerdahl et al. 2008).

In a study published by Kozawa et al. (2009), researchers at ARB and UCLA investigated into the impact of goods movement in the communities of Wilmington and West Long Beach, California. The researchers analyzed residential multi-block measurements taken in a reference zone between 160 and 600 m away from (and to the west of) the I-710 freeway into the adjacent communities. The community level concentrations for BC, UFP, and NO were compared to concentrations measured within 150 m of the I-710 freeway. Data were analyzed for the morning and afternoon in both the winter and summer seasons.

In the summer season, the mean concentrations of BC, UFP and NO within the residential multi-block (away from the freeway or heavily-traveled surface streets), were approximately 2.5 $\mu g\ m^{-3}$, 15,000 $cm^{-3}$ and 25 ppb, respectively, during morning monitoring period, and 1 $\mu g\ m^{-3}$, 20,000 $cm^{-3}$ and 12 ppb, respectively, during the afternoon monitoring period. The comparable means for BC, UFP and NO in the winter were 3 $\mu g\ m^{-3}$, 25,000 $cm^{-3}$ and 35 ppb, respectively, in the morning, and 1.5 $\mu g\ m^{-3}$, 23,000 $cm^{-3}$ and 12 ppb, respectively, in the afternoon. In summary, the mean BC concentrations at the Wilmington community were comparable to the basin-wide mean

concentrations of 1-2 μg $m^{-3}$. In addition, the mean UFP and NO concentrations ranged between 15,000 and 25,000 $cm^{-3}$ and between 10 and 35 ppb, respectively, as a function of time of day and season.

Ratios of BC, UFP and NO concentrations for measurements made within 150 m or less of the I-710 freeway divided by the residential multi-block concentrations for the same monitoring periods ranged between approximately 1 and 7 depending on the air pollutant, the time of day, the season , and the wind direction. When both areas were downwind of the I-710 freeway, the mean ratios for the near-roadway micro-environment divided by the residential multi-block micro-environment were approximately 2, 2 and 3 for BC, UFP and NO, respectively.

### v.2.5. Overview: Longitudinal studies

Only few mobile platform studies of on-road or near-road air pollutant concentrations have been performed before the year 2000 due to limitations that involve instrument power and space constraints. so available longitudinal comparisons are limited. The earliest comprehensive on-road and in-vehicle measurements were conducted by Shikiya et al. (1989b) in Los Angeles in 1987/1988. The next major on-road study in California was conducted in 1997 (Rodes 1998) and allowed comparison of VOC concentrations in Los Angeles. The Los Angeles VOC results, when compared to the in-vehicle study conducted in 1987 (Shikiya et al. 1989a), reflected the significant reductions in vehicle emissions that occurred in the intervening decade. The in-vehicle aromatic VOC and CO concentrations measured in Rodes et al. (1998) were equal to or lower than the ambient concentrations measured by Shikiya et al. (1989a).

Mobile platform measurements of particles are more recent. Initial studies relied on optical counters, and comparisons are difficult across instrument types. Integrated mass measurements inside vehicles have been few due to the difficulty in obtaining sufficient mass over the time scale of hours. Rodes et al., (Rodes 1998) made some of the first measurements of on-road black carbon (BC), using an Aethalometer, thus providing one of the earliest potential "baselines" of on-road BC concentrations available for comparison for Los Angeles and Sacramento. They also sampled integrated $PM_{2.5}$ and $PM_{10}$ with filters and analyzed for metals. However, due to the study design of Rodes in which diesel vehicles were targeted, direct comparisons of BC concentrations require the adjustments described by Fruin et al. (Fruin et al. 2004). With these adjustments, comparisons of BC concentrations can be made back to 1997 if driving the same route at the same time of day. Available locations include arterial roadway and freeway routes in Sacramento and Los Angeles.

UFPs, generally measured by condensation particle counters (CPCs) have until recently been difficult to use in moving vehicles due to their sensitivity to motion. The first available measures of on-road UFP number in California are the studies of Westerdahl et al. (2005). They drove fixed freeway and arterial roadway routes in 2003 that have periodically re-driven these routes several times per year since then. Measurements also included NO, $NO_2$, BC, particle-bound PAH, CO, $CO_2$, and $PM_{2.5}$. These studies found fairly good day-to-day repeatability in the relationship between things like diesel truck volume and concentrations of BC, NO, and UFP. While day-to-day differences in absolute concentrations depend strongly on meteorology (and therefore time of day as well), their dependence on traffic conditions is more stable, thus lending itself to longitudinal comparisons and trend analysis.

## v.3. Methods

### v.3.1. Sampling sites and built-environmental characteristics

Extensive field experiments, including mobile and stationary measurements of vehicular pollutants and traffic, were conducted at four sites in and around downtown Los Angeles (DTLA) and at a site in Temple City, located 20 km east of DTLA, for 16 days between July and November 2013 (Figure 1, Table 1). Each sampling site represents a distinct urban built environment with a different building morphology (e.g., building heights and areas, intersection configurations, street widths, building densities, and overall homogeneity) and traffic patterns (e.g., traffic flow rates, traffic densities, fleet compositions, and traffic-light cycle periods). Each sampling site covered a 2-by-2 (or 2-by-3) block area centered on a main intersection where stationary sampling of pollutants and traffic monitoring were conducted, depending on availability of instruments. All sites were located more than 800 m from the nearest freeway, well outside the range of freeway influence during the daytime. As UFP are relatively short-lived and upwind areas for all sites consisted of similarly developed urban areas for many kilometers, the influence of areas farther than the neighboring several streets are not expected to be discernible in this dataset.

**Figure 1. Map of measurements sites in the South Coast Air Basin. Red stars denote the sampling sites that cover 2-by-2 (or 2-by-3) blocks centered by the main intersection where traffic recording and stationary measurements were conducted in 2013.**

**Table 1. Description of field measurements including built-environments, measurement dates, instrumentation, and sampling design.**

| Sites | Built-Env. | Date | Instrumentation | Sampling strategy |
|---|---|---|---|---|
| Broadway & 7th St. Site1 | Street canyon with tall buildings (H>40m) at both side of the street. Highly trafficked on both streets. | 7/1<br>7/2<br>7/3<br>7/5 | [1] ARB-MMP;<br>2 DM; 2 sonic tower (roof & surface);<br>4 traffic recording cameras | [A] 2 DM were paired across the street, staying about 5 min. at mid-blocks and intersections (quasi-stationary) |
| | | 11/13 | [2] Light-MMP;<br>*3 DM;* CPC; OPS; 2 surface sonic tower;<br>4 traffic cam | [B] 2 DM were stationary at the Intersections<br>1 CPC&OPS stayed at the sonic tower<br>1 DM stationary across the sonic tower |
| Temple City & Las Tunas Site5 | All short buildings (H < 6 m) around the site Moderately trafficked (Las Tunas > Temple) | 8/6 | [1] but 1 sonic tower | [A] |
| | | 9/17<br>9/18 | [3] Light-MMP;<br>1 DM; CPC; OPS; 2 sonic tower (roof & | [C] 1 DM at intersection (staying 5 light cycles at each corner) and CPC & OPS |

| | | | | |
|---|---|---|---|---|
| | | | surface);<br>4 Traffic cameras | stayed next to sonic tower |
| Olive & 12th St.<br>Site2 | One tall building at one corner of intersection + many open space<br>Sparse traffic | 9/24 | [3] | [C] |
| | | 9/25 | | [C] but 1 DM was stationary across the sonic tower |
| Vermont & 7th St.<br>Site3 | One tall building at intersection<br>Large traffic on Vermont | 10/7<br>10/14 | [3] | [C] |
| | | 11/18 | [2] | [B] |
| Wilshire & Carondelet<br>Site4 | Two tall buildings<br>Modest traffic on Wilshire | 11/1 | [2] | [A] and additional DM was stationary across the sonic tower |
| | | 11/6 | [2] | [B] |
| | | 11/20 | [2] | [B] |

*ARB-MMP*: California Air Resources Board mobile monitoring platform
*Light-MMP*: Electric vehicle equipped with a DiSCmini
*DM*: DiSCmini ultrafine particle counter, *CPC*: condensation particle counter, *OPS*: Optical particle sizer

The Broadway and 7th St. site (*Site1*) located in DTLA (34.04519°N / 118.25639°W) is a street canyon environment surrounded by tall commercial buildings on both sides of the streets. Building heights were > 40 m with little, if any, gaps between buildings. The block lengths/street widths (measured from building face to building face on the two sides of the street) of Broadway and 7th streets are 190 m/26 m and 100 m/22 m, respectively (see Table 2). The Olive and 12th St. site (*Site2*) is located 1 km southeast of *Site1* (34.03943°N / 118.26226°W). The intersection is occupied by a 130 m tall isolated skyscraper surrounded by large open spaces and low-story buildings. This site had low traffic flows and short queues (see Table 3). The block lengths/street widths of Olive and 12th St. are 180 m/28 m and 95 m/17 m, respectively. The Vermont and 7th St. site (*Site3*) located 4 km northwest of *Site1* (34.05976°N / 118.29164°W) is similar to *Site2*, but surrounding buildings are more densely patched and open spaces are smaller. In addition, Vermont Ave. in *Site3* is one of the busiest arterials in Los Angeles. The block lengths/street widths of Vermont Ave. and 7th St. are 190 m/30 m and 95 m/25 m, respectively. The Wilshire and Carondelet St. site (medium-sized buildings on one side, *Site4*) is located 3 km northwest from *Site1* and 1 km east from *Site3* (34.06012°N / 118.28054°W). *Site4* represents a typical city environment in the Los Angeles area, consisting of a mixture of open space and moderately-sized buildings. The whole block of the south side of Wilshire Blvd. is occupied by 30 m and 50 m tall buildings while the north side is open or occupied by 5 to 10 m tall buildings. The block lengths/street widths of *Site4* are 75 m/37 m (Wilshire) and 160 m/17 m (Carondelet). Finally, the Temple City and Las Tunas Blvd. site (a low and flat residential site, *Site5*) in Temple City (34.10669°N / 118.06090°W) is surrounded mostly by one-story single family homes and small commercial buildings (< 6 m in height). The block lengths/street widths of Temple City and Las Tunas Blvd. are 175 m/24 m and 115 m/30 m, respectively. Table 3 presents the height of urban canopy (mean building area-weighted building heights), where lower numbers indicate higher building morphology (e.g., *Site1* has a street canyon and tall buildings while *Site5* has a low/flat urban configuration). The distributions of buildings and building morphology around sampling sites are presented in Figure 2, Figure 3, and Table 2 presented below.

**Table 2. Built environments (Sites1-5) in the mobile sampling areas.**

| | Broadway & 7th (Site1) | Olive St. & 12th St. (Site2) | Vermont & 7th St. (Site3) | Wilshire & Carondelet (Site4) | Temple City & Las Tunas (Site5) |
|---|---|---|---|---|---|
| # of buildings | 59 | 34 | 90 | 44 | 143 |
| Max. building height (m) | 58 | 129 | 80 | 57 | 8 |
| Mean building height, $H_{bldg}$ (m) | 34 | 21 | 11 | 18 | 5 |

| | | | | | |
|---|---|---|---|---|---|
| Bldg. area weighted height, $H_{area}$ (m) | 40 | 42 | 25 | 24 | 6 |
| Bldg. homogeneity, $H_{area}/H_{bldg}$ (dimensionless) (1=perfectly homogeneous) | 1.16 | 2.01 | 2.21 | 1.39 | 1.09 |
| Mean building ground area ($m^2$) | 1,030 | 1,395 | 585 | 992 | 225 |
| Street width (m) | 26 (BW) / 22 ($7^{th}$) | 28 (Olive) / 17 ($12^{th}$) | 30 (Ver) / 25 ($7^{th}$) | 17 (Car) / 37 (Wil) | 24 (TC) / 30 (LT) |
| Simple Aspect ratio ($H_{area}/W_{street}$) | 1.7 | 1.9 | 0.9 | 0.9 | 0.2 |
| Block length (m) | 190 (BW) / 100 ($7^{th}$) | 180 (Olive)/ 95 ($12^{th}$) | 190 (Ver) / 95 ($7^{th}$) | 160 (Car) / 75 (Wil) | 175 (TC) / 115 (LT) |
| Ratio occupied by bldg. | 0.72 | 0.42 | 0.33 | 0.46 | 0.30 |

**Table 3. Characteristic traffic patterns observed for each site during the measurement periods. Values in parentheses are standard deviations.**

| | Morning | | | | | Afternoon | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Date | Light cycle sec | Traffic rate #·min$^{-1}$ | HDV/ MDV # /cycle | Queue length # /cycle | Traffic Ratio | Light cycle sec | Traffic rate #·min$^{-1}$ | HDV/ MDV # /cycle | Queue length # /cycle | Traffic Ratio |
| | Site1 (Street Canyon) | | | | | | | | | |
| 7/1 | 69(4) | 29(2) | 1.4(1.2) | 20(5) | 0.47 | | | | | |
| 7/2 | | | | | | 89(4) | 41(8) | 0.9(0.9) | 31(8) | 0.51 |
| 7/3 | 69(5) | 29(6) | 1.0(1.2) | 18(5) | 0.44 | 89(3) | 43(8) | 0.9(0.9) | 29(5) | 0.52 |
| 7/5 | 70(4) | 22(5) | 0.9(1.1) | 12(4) | 0.48 | 89(4) | 35(5) | 0.5(0.9) | 29(6) | 0.49 |
| 11/13 | 69(2) | 34(5) | 0.9(1.0) | 21(5) | 0.56 | 69(1) | 28(5) | 1.4(1.2) | 20(5) | 0.47 |
| | Site2 (Isolated skyscraper with low traffic rates) | | | | | | | | | |
| 9/24 | 69(2) | 22(7) | 0.6(0.8) | 3(2) | 0.81 | 77(10) | 12(3) | 0.6(0.6) | 2(1) | 0.75 |
| 9/25 | 69(3) | 27(7) | 0.9(0.9) | 6(3) | 0.87 | 69(1) | 10(3) | 1.0(0.7) | 3(2) | 0.75 |
| | Site3 (Isolate skyscrapers with high traffic rates) | | | | | | | | | |
| 10/7 | 89(3) | 47(6) | 2.0(1.2) | 22(6) | 0.81 | 89(1) | 51(7) | 1.4(1.1) | 28(6) | 0.75 |
| 10/14 | 91(12) | 47(7) | 1.4(1.1) | 27(7) | 0.81 | 90(11) | 47(6) | 1.2(1.0) | 27(7) | 0.77 |
| 11/18 | 89(1) | 54(7) | 1.6(1.1) | 33(7) | 0.77 | 89(2) | 51(6) | 1.1(1.0) | 29(6) | 0.76 |
| | Site4 (One-side medium height buildings) | | | | | | | | | |
| 11/1 | 110(44) | 30(5) | 1.2(1.3) | 4(2) | 0.95 | 98(34) | 29(9) | 1.0(0.9) | 5(2) | 0.94 |
| 11/6 | 100(30) | 35(6) | 0.8(0.9) | 4(2) | 0.93 | 107(36) | 29(4) | 0.8(0.9) | 6(3) | 0.92 |
| 11/20 | 100(30) | 35(6) | 0.9(0.9) | 5(3) | 0.91 | 97(23) | 30(5) | 1.1(1.0) | 6(2) | 0.89 |
| | Site5 (Low and Flat) | | | | | | | | | |
| 8/6 | 71(6) | 45(7) | 1.7(1.9) | 24(9) | 0.44 | 79(9) | 64(10) | 1.2(1.1) | 50(15) | 0.48 |
| 9/17 | | | | | | 70(8) | 49(10) | 1.6(1.5) | 27(8) | 0.46 |
| 9/18 | 81(8) | 61(10) | 1.8(1.6) | 41(13) | 0.49 | 69(1) | 55(9) | 1.4(1.1) | 33(10) | 0.48 |

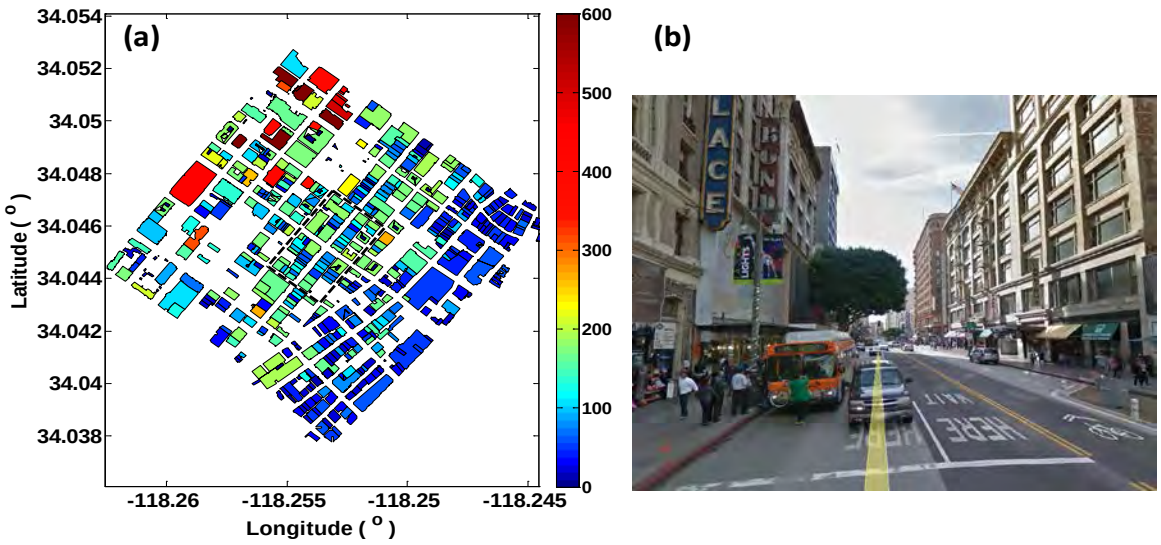



**Figure 2. (a) Map of building heights and morphology in DTLA. Color bar represents the building heights in feet. (b) The street view on Broadway St. captured in Google Earth. The building distributions and street views for the other sites are presented in the Supplementary Information.**

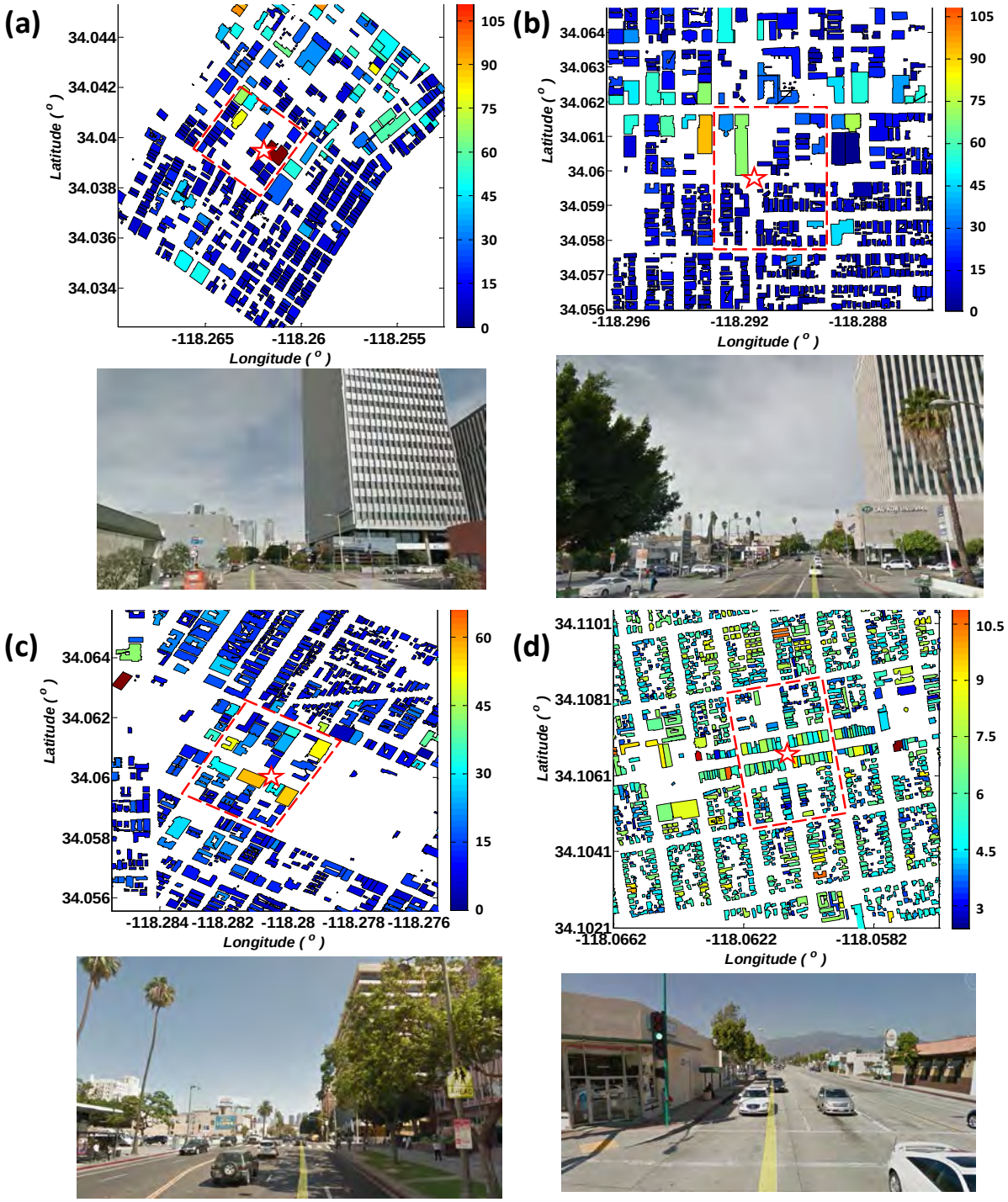



**Figure 3. Built environments of sampling sites: (a) *Site 2* (Olive & 12th St.), (b) *Site 3* (Vermont & 7th St.), (c) *Site 4* (Wilshire & Carondelet St.) in Los Angeles, and (d) *Site 5* (Temple City & Las Tunas) in Temple City. Top plots represent building distributions and heights (by colors in feet). The dashed line shows the sampling area. Bottom photos show the street view of the streets around the intersections. Color bar represents building height in meters.**

The 2014 summer sampling site was a 2 km-long section of Wilshire Blvd. located in Beverly Hills, CA (Figure 4). This site includes 5 signalized intersections and is surrounded by a variety of building configurations, with different traffic flow rates on the cross streets. Traffic was recorded at the two largest intersections, Doheney and La Cienega Blvd. that crossed Wilshire Blvd. Other sites were single intersections at which traffic was recorded in all four directions. The data from this site are discussed in the latter sections.

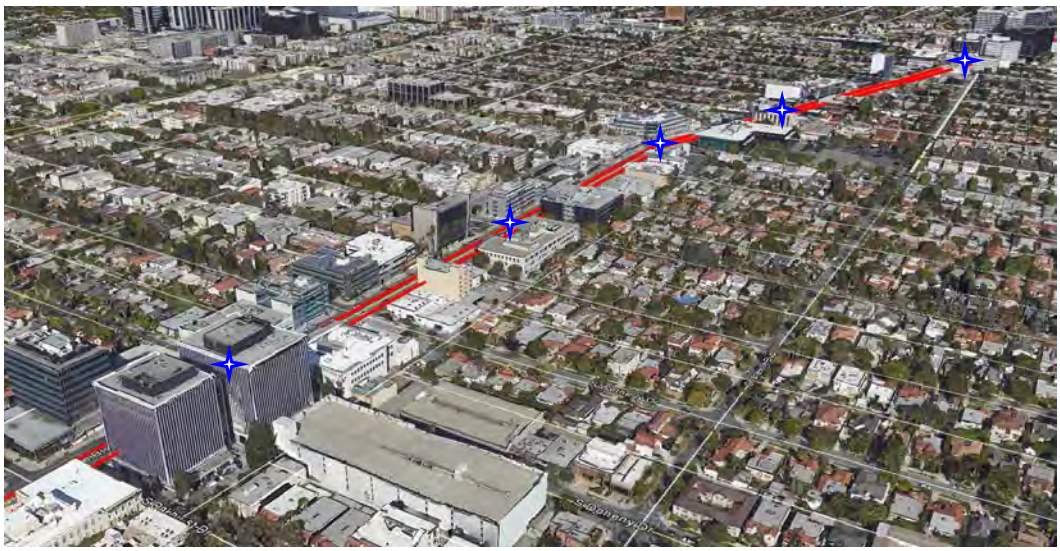


**Figure 4. Map of the Beverly Hills sampling route. Red line represents the mobile monitoring platform route and blue stars denote the locations of signalized intersections.**

### v.3.2. Instrumentation and Sampling Design

A fully-equipped Toyota RAV4 electric sub-SUV, maintained by ARB, served as a mobile monitoring platform (ARB-MMP). A suite of fast response instruments in the ARB-MMP measures various air pollutants, including UFP number concentrations and size distributions, nitrogen oxides (NOx), carbon monoxide (CO), carbon dioxide ($CO_2$), particle-bound polycyclic aromatic hydrocarbons (PB-PAH), particulate matter with aerodynamic diameters less than 2.5 µm ($PM_{2.5}$), and black carbon (BC) and have been used in a series of near-/on-road air quality studies (Choi et al. 2012a; Choi et al. 2013a; Hu et al. 2012; Kozawa et al. 2009, and others). When the ARB-MMP was not available due to maintenance, an electric vehicle (Chevrolet Volt or Nissan Leaf) equipped with a DiSCmini was used instead. The DiSCmini is a fast diffusion size classifier that measures UFP number concentration (20-700 nm size range) and the mean size of UFP collected every second. Many of the measurements were performed with a DiSCmini hand held particle counter (Matter Aerosol AG). As this instrument is relatively new, evaluations are only available for laboratory-generated nanoparticles under controlled indoor conditions (Bau et al. 2015; Mills et al. 2013).

A global positioning system, or GPS (GPSMAP 76CS, Garmin or BT-Q1000XT, Qstarz International Co., Ltd., depending on availability), was employed to record MMP positions every second, and the corrections of the GPS data were made using a line reference technique as described in detail in a companion paper (Ranasinghe et al. 2015). In this study, however, site-by-site comparisons are the main focus, and thus the highly spatially resolved data of the MMP were not used.

In general, the instruments in the MMP have different response times due to the characteristics of the instruments and differences in inlet length and flow rates. Air was drawn through a 6" diameter galvanized steel manifold installed through a window of the rear passenger space located 1.5 m above ground level Sampling ports for each instrument were located in the middle of the manifold with short (0.5–2 m) sampling tubing (1/4" Teflon for gases and 1/4" conductive tubing for particles and 1/2" conductive tubing for FMPS). For each instrument, flow and zero checks were performed before and after each measurement session. To account for any slight day-to-day differences in response time, a time-lag correlation method was used in post-data processing to synchronize the response time of the instruments (Choi et al., 2012). Concentration data and MMP position data were recorded at 1 s time resolution. Table 5 below summarizes information on the time-response of the individual instruments employed in this study. A complete description of the MMP calibration procedures is available in Hu et al. (2009b).

**Table 4. Monitoring instruments on the mobile monitoring platform.**

| Instrument | Measurement Parameter | Response time[a] (Inlet to record) |
|---|---|---|
| TSI Portable CPC, Model 3007 | Sub-micrometer particle number count (10 nm–1 μm) | 4 s |
| TSI FMPS, Model 3091 | Size-segregated particle count (5.6–560 nm) | 9 s |
| TSI DustTrak, Model 8520 | $PM_{2.5}$ mass | 5 s |
| EcoChem PAS 2000 | Particle-bound PAH | 10 s |
| Teledyne API Model 300E | CO | 21 s |
| LI-COR, Model LI-820 | $CO_2$ | 7 s |
| Teledyne-API Model 200E | NO | 22 s |
| Magee Scientific Aethalometer AE42 | Black carbon | 21 s |
| Vaisala Sonic Anemometer and Temperature/RH Sensor | Surface winds, temperature, and relative humidity | - |
| Garmin GPSMAP 76CS | Location and speed | - |
| Eurotherm Chessell Graphic DAQ Recorder | Data-logger | - |

[a] Response time is an averaged value for smoke test results (Choi *et al.*, 2013).

In all cases, the inlet for instruments was located on the passenger side of the vehicle near the roofline, in as close proximity to the sidewalk as practical. The same post-data processes described in Choi et al. (2012a) were performed to synchronize instruments and precisely account for the response time (a time-lag correlation method on a twice-daily basis).

A combination of mobile and stationary measurements was conducted depending on the availability of instruments (see Table 1). Intensive measurements were conducted for ~ 2 hrs twice a day, once in the early morning (06:00 - 09:00) and once in mid-afternoon (13:00 - 17:00). These periods represent two distinct meteorological conditions: limited mixing in the mornings vs. vigorous vertical mixing due to surface heating in the afternoons. A schematic of the sampling design is shown in Figure 5. For the entire sampling period, the MMP drove four-leaf clover shaped routes around the main intersection, typically completing 7 to 12 repeats of the route for each morning or afternoon. To supplement mobile measurements, a pair of DiSCminis were deployed on pedestrian sidewalks to measure UFP number concentration. The DiSCmini pair, being positioned across the street from one another, sampled for 5 to 10 minutes at the mid-blocks and intersections on one street and then moved to other mid-block or intersection locations (see Figure 5 and Figure 6). The objective of mobile sampling was to obtain highly resolved spatial distributions of pollutant concentrations, whereas paired measurements of UFP are useful for investigating street canyon and other effects caused by in-canopy circulation in different built environments. Also, paired DiSCmini measurements were taken in the immediate location of pedestrians at a height near breathing zones and therefore acted as a surrogate for direct pedestrian exposure to vehicular emissions.

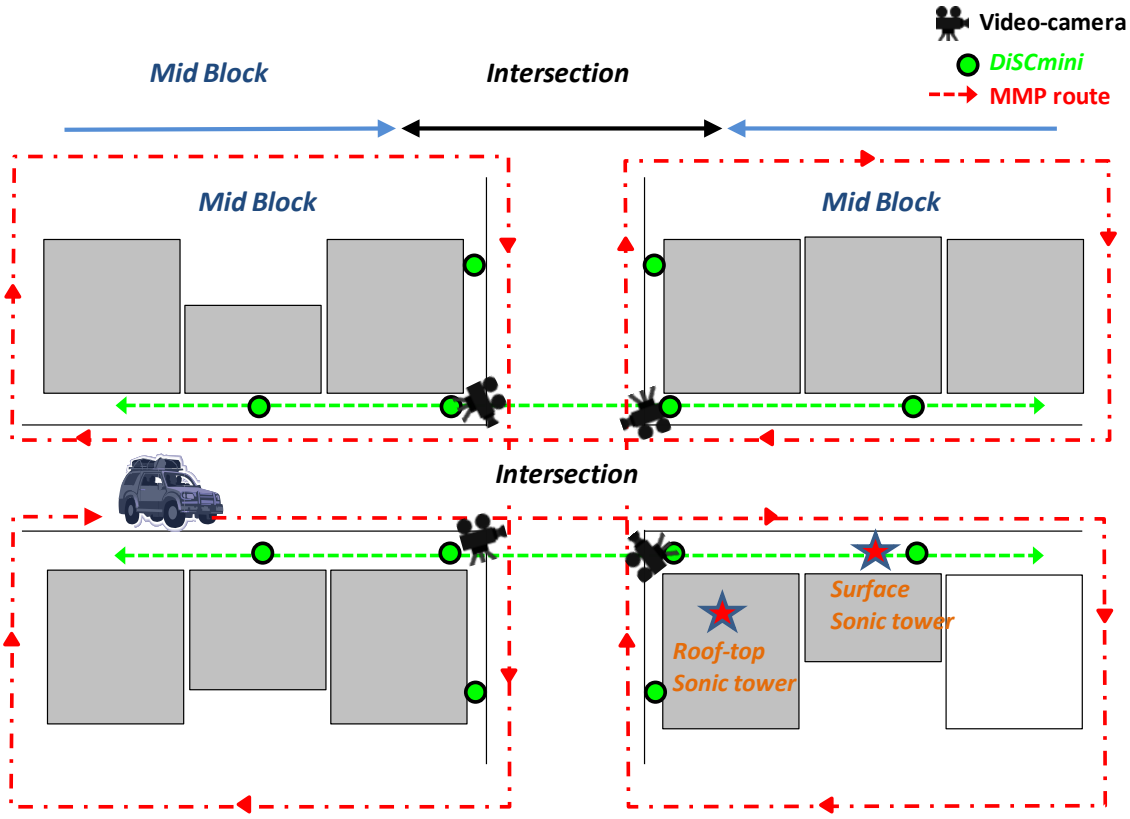



**Figure 5. A schematic of the intersection sampling design. Green circles denote the location of a DiSCmini pair (across the street) for 5-minute stationary measurements. Red stars represent the location of surface and roof-top (only when roof-top access was possible) sonic towers. The actual positions and spatial scales are different from this illustration.**

Systematic in-field inter-comparison between DiSCmini, Condensation Particle Counter (CPC, TSI 3007), and Scanning Mobility Particle Sizer (SMPS, TSI 3080) verified that the results from the DiSCmini for both particle number and size were in good agreement with conventional particle instruments. In this study, all DiSCmini data were converted to corresponding CPC values to be compared directly, because the CPC has more widely and conventionally been used in UFP air quality studies.

**Figure 6. The sampling route of the mobile monitoring platform (MMP) in downtown Los Angeles. BW denotes Broadway and EB, WB, NB, and SB represent eastbound, westbound, northbound and southbound, respectively. Map source: Google Earth.**

### v.3.3. Collecting meteorological data using MMP

The MMP was parked intermittently at various locations for 5 min periods to collect meteorological data from its sonic anemometer mounted on the roof of the MMP. In each measurement session, video recordings of the traffic were made at the central intersection using cameras mounted at each of the four corners of the intersection. Detailed information on the traffic signal light changes and the traffic counts for all four traffic flow directions were obtained manually by reviewing the video records. All data processing was done in MATLAB R2012a (The Mathworks, Inc.). Table 6 below summarizes the meteorological information that was collected at Site1 (Broadway and 7th).

**Table 5. Measurement periods and surface meteorology at BW-7th.**

| Date | Measurement Period | BW | | | 7th | | |
|---|---|---|---|---|---|---|---|
| | | Wind speed (m $s^{-1}$) | Wind direction# | Traffic flow (vehicles $s^{-1}$) | Wind speed (m $s^{-1}$) | Wind direction# | Traffic flow (vehicles $s^{-1}$) |
| 7/1/2013 | 09:15–11:45 | 0.96 | SW | 0.09 | 1.08 | ESE-NE* | 0.15 |
| | 15:30–18:00 | 0.91 | SW | + | + | + | + |
| 7/3/2013 | 08:15–11:00 | 1.34 | SSE–SW* | 0.10 | 1.06 | ESE–NE* | 0.13 |
| | 16:00–18:00 | 1.80 | SW–S* | 0.18 | 1.45 | NE | 0.18 |
| 7/5/2013 | 08:45–11:00 | 1.23 | * | 0.95 | 0.94 | ESE–NW* | 0.08 |
| | 15:30–18:15 | 1.13 | SSW–NW* | 0.14 | 1.48 | NE | 0.14 |

# NE (northeasterly), ESE (east-southeasterly), SSE (south-southeasterly), S (southerly), SSW (south-southwesterly), SW (southwesterly), NW (northwesterly).
* variable wind (wind direction was spread over two or more quadrants).
+ data not available.

### v.3.4. Site, built environment, and traffic characteristics

The site, the built environment, and the traffic characteristics all play a role in the variability of UFP concentrations on and near a roadway. Figure 7 presents an aerial view of the Broadway transect that was examined in this study. In addition, Table 7 below provides definitions to many of the variables on Broadway transect (and any other transects) that can potentially impact the UFP concentration measurements while using MMP and semi-stationary monitoring. Table 8 provides information and summary of the statistics on the key variables described in Table 7 and how variant each of these variable parameters are on Broadway transect. This provides additional insight into the resulting data and how they may affect the ambient air measurements.

**Figure 7. Map of the Broadway Transect.**

**Table 6. Variable Definitions.**

| All variables are continuous unless otherwise indicated | |
|---|---|
| Variable | Definition |
| Ultrafine particle concentration | Number of particles less than 100 nanometers in diameter measured in thousands per cubic centimeter of sampled air |
| Idle-to-moving | Binary variable indicating whether MMP is in queue |
| Speed | Measured speed of MMP measured in meters per second |
| Lane number | Lane position of MMP relative to right-hand curb. |
| Light-duty | Observed number of light-duty vehicles in visual range of MMP |
| Medium & Heavy-duty | Observed number of medium- or heavy-duty vehicles in visual range of MMP |
| Buses | Observed number of buses in visual range of MMP |
| Acceleration event | Binary variable indicating whether an acceleration event occurred in visual range of MMP |
| On-going Traffic | Indicates traffic conditions observed heading in same direction as MMP |
| On-coming Traffic | Indicates traffic condition observed heading in opposite direction as MMP |
| Crossing from the left traffic | Indicates traffic condition observed crossing direction of MMP from left |
| Crossing from the right traffic | Indicates traffic condition observed crossing direction of MMP from right |
| Intersection | Binary variable indicating no neighboring buildings to MMP (indicates MMP was in an intersection) |
| Average building height (m) | Observed average height of buildings directly neighboring MMP |
| Building height differential (m) | Height of building to the east relative to building to the west |
| North-south wind component | Wind speed in meters per second; positive if prevailing wind is from north, negative if from south |
| East-west wind component | Wind speed in meters per second; positive if prevailing wind is from east, negative if from west |
| Position | Linear reference unit measured in meters |

As shown in Table 7, the mean level and standard deviation of *UFP* was 10.40 and 0.79 thousand/cm$^3$ respectively with a minimum value of zero and maximum value of 14.14. Later in our study, the variable, UFP, will be lagged to explore the correlation of recently measured UFP concentrations and contemporaneous UFP concentrations. We will define these lagged variables as UFP-1, UFP-2, UFP-3, etc. to indicate that the UFP concentrations were 1, 2 and 3, seconds prior to the contemporaneous UFP measurement. These sensitivity analysis provides additional confidence in the results during detailed analysis of the MMP and stationary measurements.

**Table 7. Summary Statistics of Variables.**

| | Mean | Std. Dev. | Min | Max |
|---|---|---|---|---|
| Ultrafine Particle Concentration (thousands/cm3) | 10.4 | 0.79 | 0 | 14.14 |
| *MMP State of Motion and Speed* | | | | |
| Idle-to-moving | 0.27 | 0.45 | 0 | 1 |
| Speed (m/s) | 7.53 | 8.52 | 0 | 174.83 |
| Lane number | 3.86 | 0.47 | 1 | 5 |
| *On-going Traffic* | | | | |
| Light-duty | 0.054 | 0.33 | 0 | 9 |
| Medium & heavy-duty | 0.004 | 0.066 | 0 | 1 |
| Buses | 0.006 | 0.082 | 0 | 2 |
| Acceleration event | 0.014 | 0.118 | 0 | 1 |
| *On-Coming Traffic* | | | | |
| Light-duty | 0.276 | 0.57 | 0 | 4 |
| Medium & heavy-duty | 0.012 | 0.109 | 0 | 1 |
| Buses | 0.028 | 0.165 | 0 | 2 |
| Acceleration event | 0.013 | 0.112 | 0 | 1 |
| *Crossing from the Left Traffic* | | | | |
| Light-duty | 0.06 | 0.252 | 0 | 2 |
| Medium & heavy-duty | 0.003 | 0.054 | 0 | 1 |
| Buses | 0.003 | 0.052 | 0 | 1 |
| Acceleration event | 0.003 | 0.052 | 0 | 1 |
| *Crossing from the Right Traffic* | | | | |
| Light-duty | 0.064 | 0.266 | 0 | 3 |
| Medium & heavy-duty | 0.005 | 0.073 | 0 | 1 |
| Buses | 0.001 | 0.037 | 0 | 1 |
| Acceleration event | 0.004 | 0.062 | 0 | 1 |
| *Built Environment* | | | | |
| Intersection | 0.12 | 0.32 | 0 | 1 |
| Average building height (m) | 16.62 | 15.53 | 0 | 71.24 |
| Building height differential (m)[a] | -3.88 | 20.51 | -61.57 | 61 |
| *Meteorology* | | | | |
| North-south wind component (m/s)[b] | -1.89 | 2.01 | -3.91 | 1.31 |
| East-west wind component (m/s)[b] | -0.68 | 1.7 | -2.99 | 1.79 |

[a] Difference in height of neighboring buildings is positive if the building to the east is taller, and negative if the building to the west is taller.
[b] North-south wind component is positive if the north-south component of the prevailing wind is northerly, negative if southerly. East-west wind component is positive if the east-west component of the prevailing wind is easterly, negative if westerly.

***MMP Street Position and Velocity.*** We collected a wide range of independent variables that may be correlated with changes in UFP concentrations. The variable *speed* indicates the velocity of the MMP measure in meters per second (m/s) with a mean of 7.53 m/s. The variable *idle-to-moving* indicates that the MMP is stationary in a queue of traffic, most frequently waiting to cross an intersection. *Lane number* indicates what street lane the MMP occupies, relatively to the right hand curb with the parking lane nearest the curb being 1 and middle left-hand turning lane being 5. The mean value for lane in 3.86 indicating that the MMP was most often traveling in lane 3, near-center travel lane. Later in our analysis we will employ a variable *position* which indicates the linear

coordinates of the MMP along a linear transect representing the entire transect. We will discuss the use of this locator variable in later analysis.

***Traffic Conditions.*** Figure 2 may serve as a visual aid in understanding the definition of variables that includes i) traffic conditions around the MMP and ii) the adjacent built and transportation environment. As the MMP traveled along Broadway we characterized traffic that was a) on-going, for traffic traveling in the same southward direction as the MMP, b) on-coming traffic, for vehicles traveling northward on Broadway, approaching and then passing the MMP on its left side, c) when the MMP approached or was stopped before an intersection, d) as MMP crossed the intersection from the left, and e) traffic crossing the intersection from the right. For each of these traffic directions, we further characterized traffic in terms of i) acceleration events, indicating that traffic had transitioned from a stationary position at the traffic light to a state of motion as traffic accelerated. We also characterized the number of vehicles moving in each direction by type of vehicle, indicating the number of ii) light-duty vehicles, iii) medium and heavy-duty vehicles and iv) buses. Table 2 reveals that the on-going light-duty traffic counts are higher compared to on-coming traffic counts. For example, on-going light-duty vehicles exhibited a mean count of 0.330 per second while on-coming light-duty vehicles exhibited a mean of 0.109 per second. The traffic crossing the MMP from the left and from the right exhibited comparable counts of 0.252 and 0.266 per second, respectively for light-duty vehicles.

***Adjacent Built Environment.*** As the MMP travelled southward we also characterized the built environment features immediately adjacent at the time scale of one second. We chose to characterize the adjacent *average building height*, mean of 16.62, with a minimum of zero for parking lots or vacant land and a maximum of 71 meters for tall buildings. We also characterized the *building height differential*, subtracting the height on the west side from the east side as measured in meters. While the mean of the building height differential was only -3.88 meters, the minimum was -61.57 meters while the maximum was 61 meters. The variable *intersection* indicated when the MMP was in an intersection. The variable's mean of 0.12 in Table 2 suggests that the MMP was in an intersection approximately 12% of the time spent traversing the transect.

## v.4. Results and Discussion

### v.4.1. The effects of the traffic patterns, micro-meteorology, and built environment on street level UFP concentrations at a block scale

#### *v.4.1.1. Characteristics of traffic patterns, micro-meteorology, and built environments*

Observed traffic characteristics at each of the sites are shown in Table 3. The basic traffic light stops were 69 or 89 seconds; these changed actively depending on traffic conditions. Traffic rates ($vehicles \cdot min^{-1}$) were comparable or higher during the afternoon sampling compared to the morning sampling periods, except at *Site2*. The highest traffic rates were observed at *Site3* and *Site5* in both the mornings and the afternoons. Although the traffic rates were comparable between *Site3* and *Site5*, traffic density at *Site3* was significantly higher due to unequal distributions of traffic between the two streets and the denser arterial-street-network in this commercial/business district. Of the five sampling sites, *Site1* and *Site5* had equal traffic between north-south and east-west bound streets while *Site3* and *Site4* showed significant disparity in traffic rates. *Site2* also had unequal traffic distributions, but the overall traffic rate was comparatively small. Heavy- and medium-duty vehicles were encountered infrequently for all sampling sites (< 1.5 $vehicle \cdot min^{-1}$).

Observed meteorology including detailed surface micrometeorology is summarized in Table 8 and Figure 8. Morning meteorology was generally calm for all sampling sites, with mean wind speeds below 1.4 m·s$^{-1}$ with 1σ values within 0.4 m·s$^{-1}$; with the exception of 9/25/2013 at *Site2*, where the wind was exceptionally strong at 1.9 (±0.6) m·s$^{-1}$. Friction velocity ($u_*$), vertical wind fluctuation ($\sigma_w$), and turbulence kinetic energy (*TKE*) appeared to be similar among the sites in the morning (see Table 9). In the afternoon, wind speeds increased up to 3.3 m·s$^{-1}$ with 1σ values within 0.6 m·s$^{-1}$. Thus, meteorological conditions were not variable for the 2-hour sampling periods. Turbulence parameters for afternoon sampling periods varied more widely between sites. For instance, *Site1*, *Site4*, and *Site5* had less turbulent surface conditions than *Site2* and *Site3*. As noted earlier *Site2* and *Site3* have more heterogeneous building morphology with one or two isolated tall buildings together with large open areas and/or low building areas than other sampling sites. This heterogeneous building configuration may generate more intense turbulence near the intersections as discussed later.

**Table 8. Surface micro-meteorological conditions observed during sampling periods. Values in parentheses represent standard deviations of the respective parameters.**

| | Morning | | | | | Afternoon | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Date | Temp. (°C) | [a]Wind speed m/s | $u_*$ m/s | $\sigma_w$ m/s | TKE m$^2$ s$^{-2}$ | Temp. (°C) | [a]Wind speed m/s | $u_*$ m/s | $\sigma_w$ m/s | TKE m$^2$ s$^{-2}$ |
| | Site1 (Street canyon) | | | | | | | | | |
| 7/1 | 25.9 (±1.7) | 1.0 (±0.2) | 0.23 | 0.37 | 0.47 | 32.2 (±2.1) | 1.1 (±0.3) | 0.23 | 0.40 | 0.46 |
| 7/2 | 26.1 (±1.4) | 1.2 (±0.3) | 0.27 | 0.40 | 0.61 | 23.5 (±1.2) | 1.7 (±0.3) | 0.29 | 0.52 | 1.02 |
| 7/3 | 23.0 (±1.3) | 1.2 (±0.1) | 0.17 | 0.35 | 0.47 | 22.5 (±1.0) | 1.7 (±0.2) | 0.36 | 0.57 | 0.97 |
| 7/5 | 20.6 (±1.4) | 0.9 (±0.2) | 0.19 | 0.30 | 0.47 | 24.0 (±0.6) | 1.6 (±0.2) | 0.15 | 0.48 | 1.21 |
| 11/13 | 24.2 (±0.4) | 0.8 (±0.2) | 0.20 | 0.27 | 0.18 | 29.9 (±0.6) | 0.5 (±0.1) | 0.12 | 0.19 | 0.08 |
| | Site2 (Isolated skyscraper with low traffic rates) | | | | | | | | | |
| 9/24 | 26.6 (±0.4) | 0.8 (±0.2) | 0.15 | 0.24 | 0.23 | 28.3 (±0.2) | 2.1 (±0.6) | 0.72 | 0.73 | 1.90 |
| 9/25 | 21.7(±0.5) | 1.9 (±0.6) | 0.88 | 0.97 | 1.83 | 24.9 (±0.4) | 3.3 (±0.4) | 0.48 | 0.59 | 1.84 |
| | Site3 (Isolated skyscrapers with high traffic rates) | | | | | | | | | |
| 10/7 | 22.8 (±0.5) | 1.4 (±0.4) | 0.13 | 0.41 | 0.77 | 27.8 (±0.7) | 2.6 (±0.5) | 0.40 | 0.68 | 2.11 |
| 10/14 | 17.9 (±1.4) | 0.7 (±0.1) | 0.35 | 0.38 | 0.31 | 28.6 (±0.4) | 1.9 (±0.3) | 0.42 | 0.61 | 1.26 |
| 11/18 | 15.4 (±0.4) | 0.9 (±0.2) | 0.23 | 0.36 | 0.39 | 20.2 (±0.4) | 2.6 (±0.3) | 0.27 | 0.70 | 1.60 |
| | Site4 (One-side medium height buildings) | | | | | | | | | |
| 11/1 | 17.2 (±1.7) | 1.2 (±0.4) | 0.16 | 0.39 | 0.54 | 29.1 (±0.4) | 0.9 (±0.2) | 0.43 | 0.56 | 0.51 |
| 11/6 | 15.1 (±1.4) | 0.9 (±0.1) | 0.20 | 0.37 | 0.35 | 26.3 (±0.3) | 0.6 (±0.2) | 0.27 | 0.27 | 0.25 |
| 11/20 | 16.0 (±0.3) | 0.5 (±0.1) | 0.19 | 0.23 | 0.17 | 19.1 (±0.2) | 1.7 (±0.3) | 0.13 | 0.38 | 0.74 |
| | Site5 (Low and flat) | | | | | | | | | |
| 8/6 | | | | | | 29.4 (±0.6) | 1.5 (±0.1) | 0.45 | 0.63 | 1.07 |
| 9/17 | 21.4 (±0.5) | 0.7 (±0.1) | 0.10 | 0.26 | 0.24 | 30.3 (±1.1) | 1.1 (±0.2) | 0.26 | 0.40 | 0.47 |
| 9/18 | 20.0(±0.6) | 0.6 (±0.1) | 0.18 | 0.27 | 0.23 | 29.0 (±0.8) | 1.0 (±0.2) | 0.23 | 0.39 | 0.41 |

a. Wind speeds represent the ground level values obtained with sonic anemometer measurements. Thus wind direction is strongly influenced by localized built environment, and not shown in this table. Prevailing wind direction over the urban canopy obtained from nearby weather station is also presented.

| | Morning | | | Afternoon | | |
|---|---|---|---|---|---|---|
| *Date* | Temp. (°C) | Wind speed (m/s) | Wind direction (degree) | Temp. (°C) | Wind speed (m/s) | Wind direction (degree) |
| | *Site1 (Street canyon)*[a] | | | | | |

| | | | | | | |
|---|---|---|---|---|---|---|
| 7/1 | 27.7 (±1.1) | 1.0 (±0.2) | 178 (±29) | 33.9 (±2.2) | 1.1 (±0.2) | 199 (±10) |
| 7/2 | N/A | N/A | N/A | 26.3 (±1.2) | 1.6 (±0.3) | 189 (±6) |
| 7/3 | N/A | N/A | N/A | N/A | N/A | N/A |
| 7/5 | 23.3 (±0.9) | 1.1 (±0.3) | 121 (±79) | 26.0 (±0.5) | 2.3 (±0.4) | 250 (±9) |
| 11/13 | N/A | N/A | N/A | N/A | N/A | N/A |
| | *Site2 (Isolated skyscraper with low traffic rates)*[b] | | | | | |
| 9/24 | 23.9 (±2.8) | 0.7 (±1.2) | 30(-)[c] | 27.4 (±0.6) | 2.1 (±0.6) | 270 (-) |
| 9/25 | 20.0 (±1.1) | 1.9 (±0.6) | 100(-) | 23.7 (±0.3) | 1.5 (-) | 200 (-) |
| | *Site3 (Isolated skyscrapers with high traffic rates)*[b] | | | | | |
| 10/7 | 25.2 (±2.8) | Calm | Calm | 26.5 (±0.8) | 1.7 (±0.3) | 260 (-) |
| 10/14 | 17.2 (±3.1) | Calm | Calm | 26.7 (±0.6) | 1.5 (-) | 290 (-) |
| 11/18 | 15.5 (±1.4) | Calm | Calm | 19.2 (±0.3) | 1.8 (±0.4) | 280 (-) |
| | *Site4 (One-side medium height buildings)*[b] | | | | | |
| 11/1 | 17.2 (±4.2) | Calm | Calm | 29.2 (±0.3) | 1.4 (±1.3)[d] | 280 (-) |
| 11/6 | 19.6 (±3.3) | 1.0 (±0.9) | N/A | 25.6 (±1.6) | 1.0 (±0.9) | 260 (-) |
| 11/20 | 16.6 (±0.7) | Calm | Calm | 17.9 (±0.1) | - | - |
| | *Site5 (Low and flat)*[a] | | | | | |
| 8/6 | N/A | N/A | N/A | N/A | N/A | N/A |
| 9/17 | N/A | N/A | N/A | 29.1(±1.1) | 2.0 (±0.3) | 186 (±17) |
| 9/18 | 20.1 (±0.8) | 1.0 (±0.2) | 40 (±48) | 28.2 (±0.6) | 2.3 (±0.6) | 199 (±14) |

a. Data from sonic anemometer installed on the roof of the building
b. Weather station data located at the University of Southern California (USC)
c. (-) denotes lack of data
d. Inferred from incomplete data from USC weather station

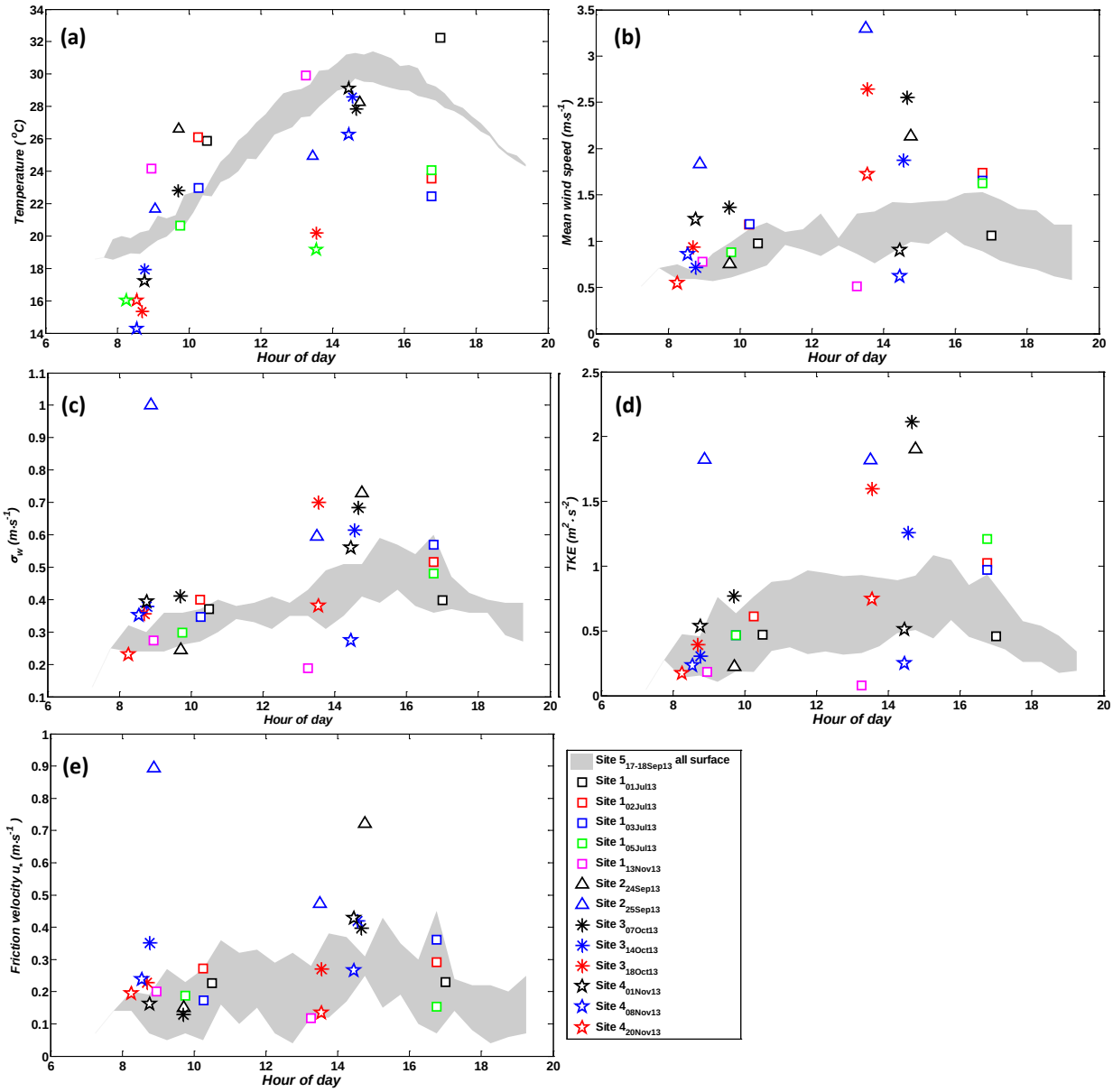



**Figure 8. Micro-meteorological characteristics for sampling sites on individual days (specified by colors): (a) temperature, (b) mean wind speed, (c) vertical fluctuation of winds, (d) turbulence kinetic energy, and (e) friction velocity in the ground level. The shaded areas represent diurnal variations of data obtained at *Site5*, and squares, triangles, asterisks, and stars denote representative values for the *Site1*, *Site2*, *Site3*, and *Site4*, respectively.**

To quantitatively investigate the built-environmental effects on street-level pollutant distributions, the key built-environmental factors were defined and calculated: the number of buildings in the sampling area; the mean building height (Eq. 1); building area-weighted height (Eq. 2); building heterogeneity (Eq. 3), street width, block length, and ratio of the area occupied by buildings to the total sampling area (building density; Eq. 4):

$$\textit{Mean building height},\ H_{bldg} = \frac{\sum_{i=1}^{N} H_i}{N} \tag{1}$$

$$\text{Building area-weighted building height}, H_{area} = \frac{\sum_{i=1}^{N} (S_i \times H_i)}{\sum_{i=1}^{N} S_i} \tag{2}$$

$$\textit{Building heterogeneity} = H_{area} / H_{bldg}\ \text{(1= perfectly homogeneous)} \tag{3}$$

$$Building\ density = \frac{\sum_{i=1}^{N} S_i}{\text{sampling area}} \text{(1= entirely covered by buildings, 0=open space)} \quad (4)$$

where, $N$ is number of buildings in the sampling area, $H_i$ and $S_i$ are height and area of the $i^{th}$ building, respectively. *Sampling area* is defined as the area of the rectangle covering the sampling area, as shown in Figure 2. We note that a simple arithmetic mean of $H_{bldg}$ can be significantly lowered when a sampling area consists of one very large isolated skyscraper and many small short buildings such as in *Site2* and *Site3*. Thus, we use $H_{area}$, which is defined as the building area-weighted building height (Eq. 2). Consequently, the dimensionless ratio of $H_{area}$ to $H_{bldg}$ represents the building heterogeneity; this has a value of 1 for perfectly homogeneous building morphology and higher values for more heterogeneous building morphology. *Site1* and *Site5* have the most homogeneous built environments (heterogeneity of 1.16 and 1.09, respectively) but are very different: *Site1* has all tall buildings (>40m height street canyon) and *Site5* has all small one-story buildings (lowest building canopy of 6 m). Tall buildings on one side and small ones on the other side gave *Site4* an intermediate homogeneity of 1.39. *Site2* and *Site3* were the most heterogeneous (2.01 and 2.21, respectively). These quantitative parameters can be compared directly with our observed UFP concentrations to find the direct effects of built environments.

#### *v.4.1.2. General Features of UFP concentrations*

To compare the representative levels of pollutants due to roadway emissions in various built environments, concentrations obtained from mobile measurements within each sampling area were averaged. Due to significant differences in meteorology between early mornings and afternoons (e.g., boundary layer depth, vertical mixing capacity, prevailing winds, and possibly secondary formation of nucleation mode particles), the results from the morning and the afternoon sampling were analyzed and discussed separately.

Figure 9 shows the daily mean UFP concentrations ([UFP] hereafter) for each sampling site. In general, [UFP] were higher in the morning than in the afternoon due to lower boundary layer heights with less turbulence, which limit vertical dispersion of emissions and increase pollutant residence time in the surface layer. Exceptions were *Site5*, *Site2* on September 5th, and *Site1* on July 5th, which showed higher concentrations in the afternoon. This cannot be explained by either emissions or dispersion because traffic flow rates were comparable to morning sampling events and the near surface atmospheric condition was more turbulent with a deeper boundary layer in the afternoon. The estimated boundary layer heights from vertical temperature profiles observed at Los Angeles International Airport (18 km southwest from *Site1*) were at least two times greater in the afternoon than morning sampling events on the following days: 236 m vs. 798 m on 9/5/2013; 174 m vs. 361 m on 9/17/2013; and 298 m vs. 486 m on 9/18/2013 (data on 7/5/2013 are not available).

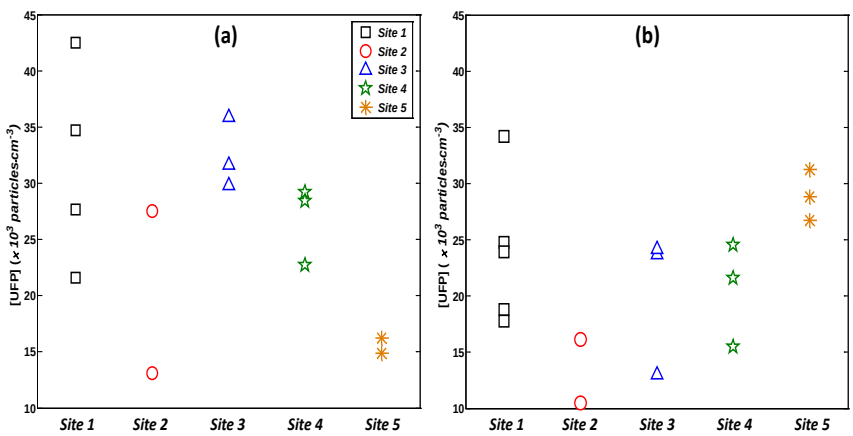



**Figure 9. Daily averaged [UFP] in the (a) morning and (b) afternoon sampling events at site.**

We hypothesize that enhanced afternoon concentrations were caused by photochemical secondary production of UFP (Hu et al. 2012; Ning et al. 2007). The interesting feature is that the afternoon elevation in the transient high-spikes-removed [UFP] was observed only when the morning [UFP] were less than $2\times10^4$ *particles·cm*$^{-3}$. On the other days, morning and afternoon [UFP] were linearly related to one another (Figure 10). In the morning of July 5th at *Site1*, however, $PM_{2.5}$ values were extraordinarily high (above the upper limit of DustTrak, > 1 mg/m$^3$), presumably due to Independence Day fireworks on the evening of July 4th. Thus, lower [UFP] in the morning could be due to lower traffic (24% lower) and/or an increased coagulational sink for fresh UFP from the dramatically increased $PM_{2.5}$ (Choi and Paulson 2016). *Site1*, the street canyon site in DTLA, had the highest [UFP] in both morning and afternoon, likely due to limited mixing with upper ambient air. *Site1* has a fairly tall, homogeneous building height and large simple aspect ratio, $A_r$=1.7 (defined as the ratio of $H_{area}$ to the mean street width, Table 2). *Site5* had the lowest [UFP] in the morning presumably because the small $H_{area}$ and $A_r$ (Table 2) help enhance the vertical mixing. *Site2* also had low [UFP] compared to the other sites, even though the simple $A_r$ at this site is highest ($A_r$=1.9). This can be explained by relatively low traffic flow rates at this site, combined with a negligible number of motor vehicles in queues during red lights at intersections. We also note that the simple $A_r$ does not account for open spaces (e.g. the gaps between buildings or large parking lots). The high $A_r$ at *Site2* derives from two tall isolated skyscrapers but this sampling site also has vast open parking areas (Figure 3), as shown by the minimal number of buildings in the selected area (Table 2). Morning [UFP] at *Site3* were comparable to *Site1* but sharply decreased in the afternoon, reaching levels similar to *Site4* that is lower than *Site1*. The elevated concentrations in the morning at *Site3* were likely due to both the heavy traffic flows and traffic density (Table 3). However, given that traffic flow rate and traffic density at *Site3* were similar during both the morning and the afternoon and higher than those at *Site1* and *Site4*, lower afternoon [UFP] (relative to *Site1* and *Site4*) cannot be readily explained with only traffic parameters. Consequently, these observations strongly suggest that [UFP] for each site were controlled by different factors (discussed quantitatively in later sections) depending on meteorological and built-environmental conditions.

#### v.4.1.2.1. Identification and replacement of transient high-spikes from high-emitting or accelerating vehicles

The UFP time-series obtained with the MMP includes a significant portion of transient concentration spikes (ca. 10 to 15%) due to high emitting vehicles (HEV) encountered during measurements (Choi et al. 2013a). These short-lived spikes can be higher than the baseline by factors of ~3 to 50 and affect the mean concentrations. Here we used an approach developed by Choi et al. (2013a) to separate the transient HEV spikes from the baseline variations.

The initial baseline variations were obtained from least squares quadratic polynomial fitting resistant to outliers as a smoothing function. Then, the standard deviation of initial baseline-subtracted UFP ($\sigma_{\Delta UFP}$) was calculated. The initial threshold value ($C_{T0}$) was defined as 3 times of initial $\sigma_{\Delta UFP}$ and the new threshold value ($C_{T1}$) was calculated using 3× $\sigma_{\Delta UFP<CT0}$ for the dataset of $\Delta UFP < C_{T0}$. This step was repeated until the threshold values converged on the specific constant value. In general, the threshold values converged within 8 iterations. The UFP concentrations higher than the final $C_T$ were flagged as transient spikes and replaced with corresponding baseline values if needed. Figure 10 below represents the morning and afternoon [UFP] comparisons after the removal of transient high-spikes from the time-series at each of the sampling sites.

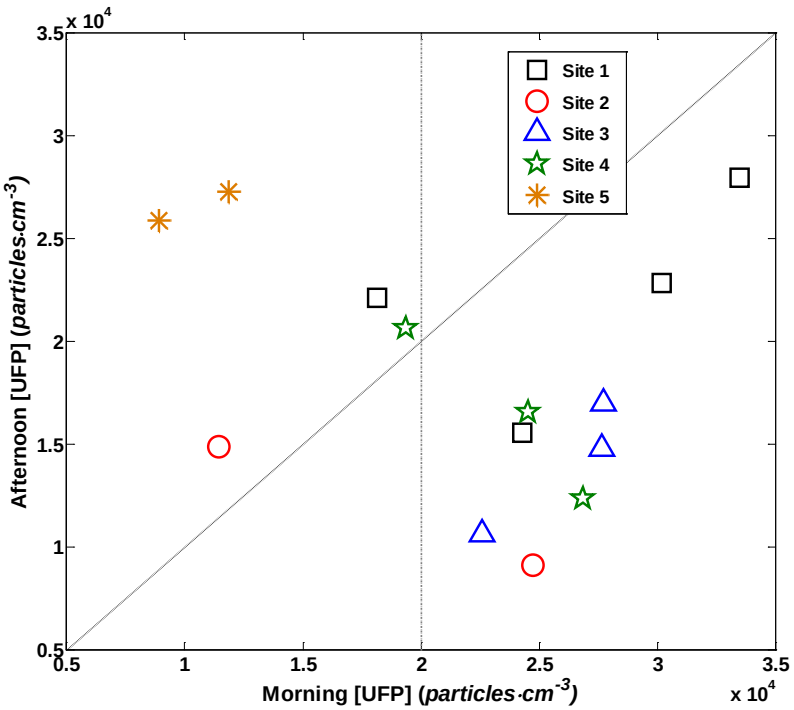



**Figure 10. The mean [UFP] of the transient high-spikes-removed time-series over the sampling areas in the morning vs. afternoon. Site 5 showed significant increases in [UFP] in the afternoon compared to the morning values despite comparable traffic volumes and more favorable atmospheric dispersive capacity.**

#### *v.4.1.3. Elevated emissions at the intersections*

One of the objectives of this field study was to investigate variations in pedestrian exposure to roadway emissions at different locations. In this respect, the study present general quantitative impacts of vehicle acceleration at intersections. Figure 11 shows the mean [UFP] at intersection corners (measured with stationary DiSCminis) vs. the average for the whole sampling area (measured with the MMP) at each of the sampling sites. The intersection averages were consistently higher than the whole sampling area average in both morning and afternoon sampling events for all sites except *Site3* afternoon. In the morning, [UFP] at the intersections was higher than the sampling area average by 24%, 10%, 5%, 11% and 55% at *Site1*, *2*, *3*, *4* and *5*, respectively; in the afternoon, intersections corners were higher by 36%, 31%, -14%, 18% and 31%, respectively. Traffic at *Site3* was concentrated on Vermont Ave. and, due to a long queue that covered the entire sampling blocks, acceleration events occurred over the whole sampling section of Vermont Ave., likely causing less significant intersection impacts.

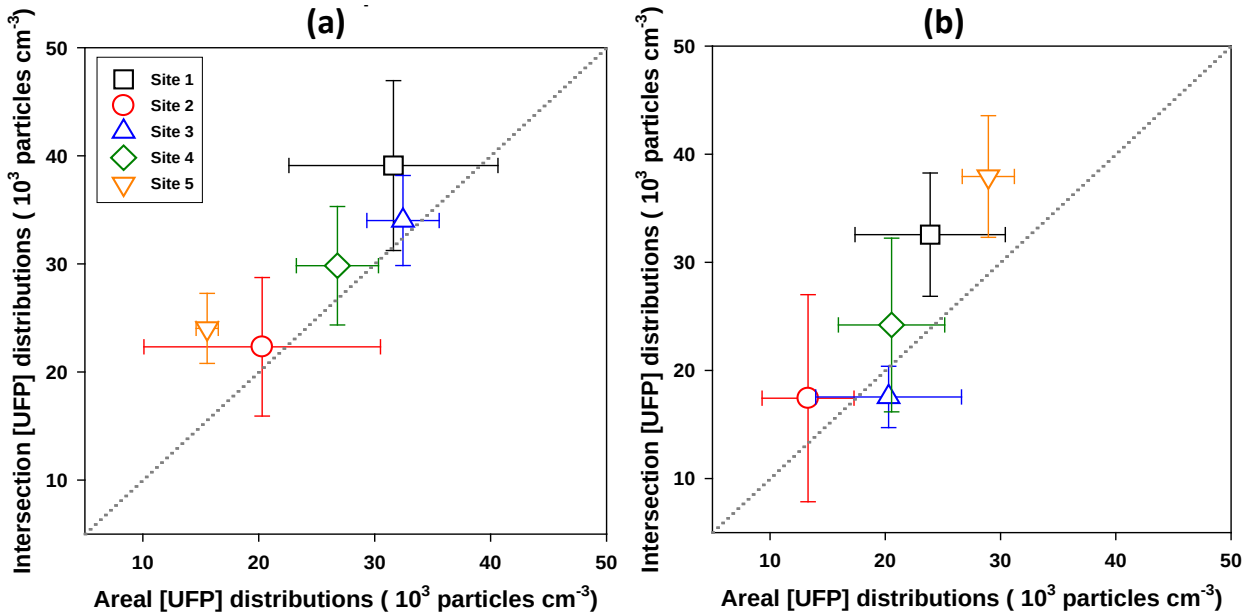



**Figure 11. The mean intersection vs. area-wide [UFP] distributions (a) in the morning and (b) in the afternoon sampling events at each sampling site. Vertical and horizontal bars denote standard deviations.**

Consistently higher [UFP] at the corners of intersections provides clear evidence that acceleration of vehicles at intersections increases pedestrian exposure to UFP. This is consistent with the argument in Klems et al. (2010) that the dominant period of transient spikes in UFP time-series matches traffic-light cycles. Although Klems et al. (2010) addressed only occurrences and periods of spikes from the intersection accelerations, this study, in addition, observed that the spike-removed baseline levels obtained with the same method in Choi et al. (2013a) were higher at intersection corners compared to the values over the sampling area: 29/33%, 15/38%, 2/3%, 7/18%, and 61/27% for *Sites1*, *2*, *3*, *4*, and *5* in the morning/afternoon, respectively (Figure 12). This implies, perhaps unsurprisingly, that higher emissions from vehicle accelerations at intersections are quickly mixed with ambient air and, at steady-state, result in persistently higher [UFP] in the intersection areas.

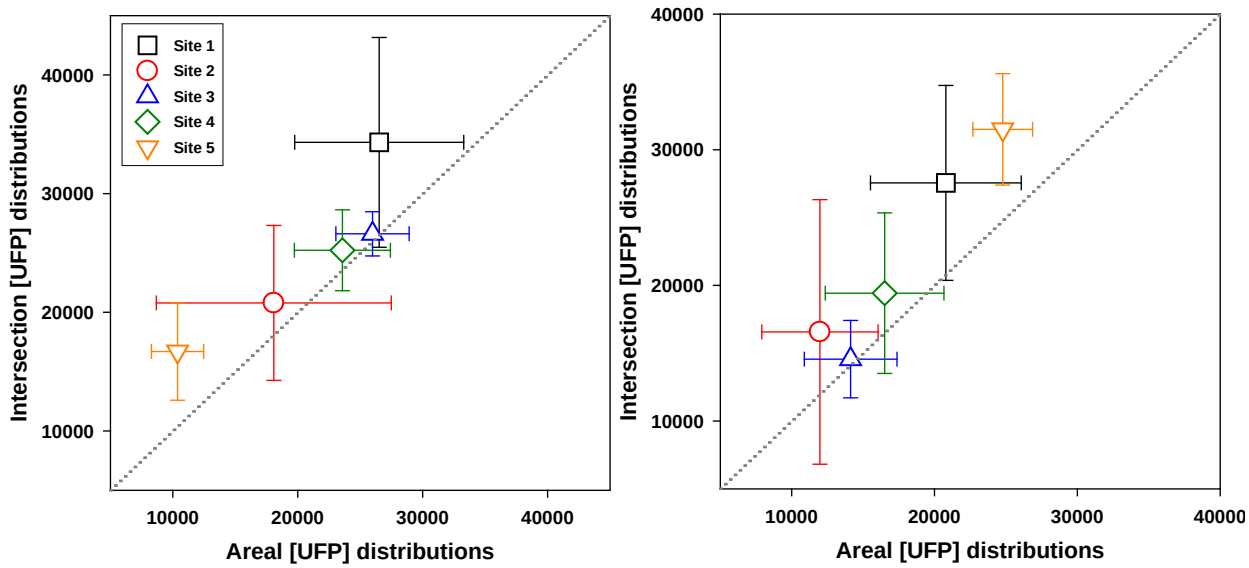



**Figure 12. Comparisons of [UFP] variations in baseline time-series at the intersections and over the sampling area at (a) in the morning and (b) in the afternoon. Horizontal and vertical bars denote 1σ variations.**

Given that the MMP route includes intersection areas (Figure 2) and the peak concentrations due to acceleration do not necessarily appear exactly at the corners (they can instead appear before and/or after intersections) (Ranasinghe et al. 2015), the concentration difference at intersection vs. over the sampling area can be higher than the values presented above. More complete analyses for the intersection impacts (including the locations and shapes of intersection peaks) will be presented separately.

### *v.4.1.4. Factors controlling near-roadway UFP concentrations*

#### v.4.1.4.1. Calm morning conditions

Most morning sampling events were calm, and meteorological variations between sites were not sufficient to explain the wide [UFP] variations among sampling sites (Figure 8). Traffic differences were noticeable between sites, but day-to-day variations at a single site were relatively insignificant. It appears that, in general, higher traffic rates led to higher levels of UFP, except at the two sites with extreme built-environments: the street canyon (*Site1*) and the low, flat canopy (*Site5*). The tall, homogeneous building canopy in the street canyon had higher [UFP] compared to observed traffic flow rates, and the opposite was true for the homogeneous built environment with the lowest building canopy (Figure 13).

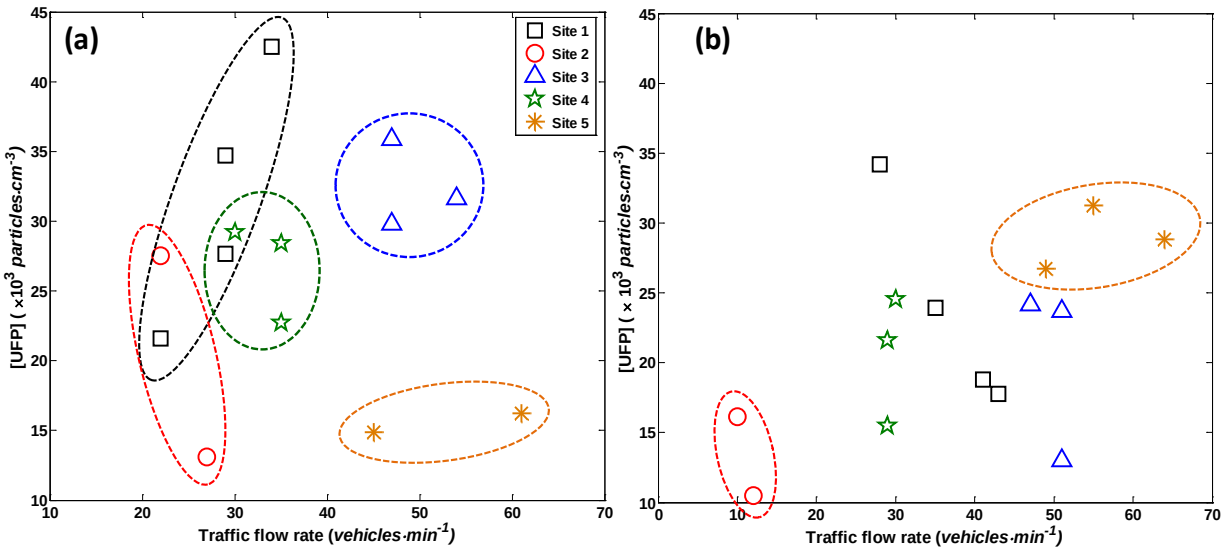



**Figure 13. Daily [UFP] as a function of traffic flow rates (vehicles·min-1) (a) in the morning and (b) in the afternoon sampling events. Ovals show a group of sampling site.**

A noticeable positive correlation was found between [UFP] and building area-weighted building height, $H_{area}$, particularly in the morning (Figure 14). *Site1* and *Site2* have similar $H_{area}$ values of around 40 m, however, the high $H_{area}$ at *Site2* results from few very tall buildings (~130 m) on a site with many large open parking lots around the intersection (Table 2 and Figure 3a), while *Site1* is largely surrounded by ~40 m buildings. To better capture the characteristics of different built-environment, a block-scale areal aspect ratio ($Ar_{area}$) was developed (Eq. 5):

$$Ar_{area} = \frac{H_{area}}{L_{diag} \times \left(1 - \sum S_{bldg} / A_{site}\right)} = \frac{H_{area}}{L_{diag} \times \left(A_{open} / A_{site}\right)} = \frac{H_{area}}{L_{open}} \qquad (5)$$

where $\Sigma S_{bldg}$ is the sum of the building ground areas, $A_{site}$ is the area of the sampling site, $L_{diag}$ is the diagonal block length, and $L_{open}$ and $A_{open}$ are the length scale and area of open space, respectively.

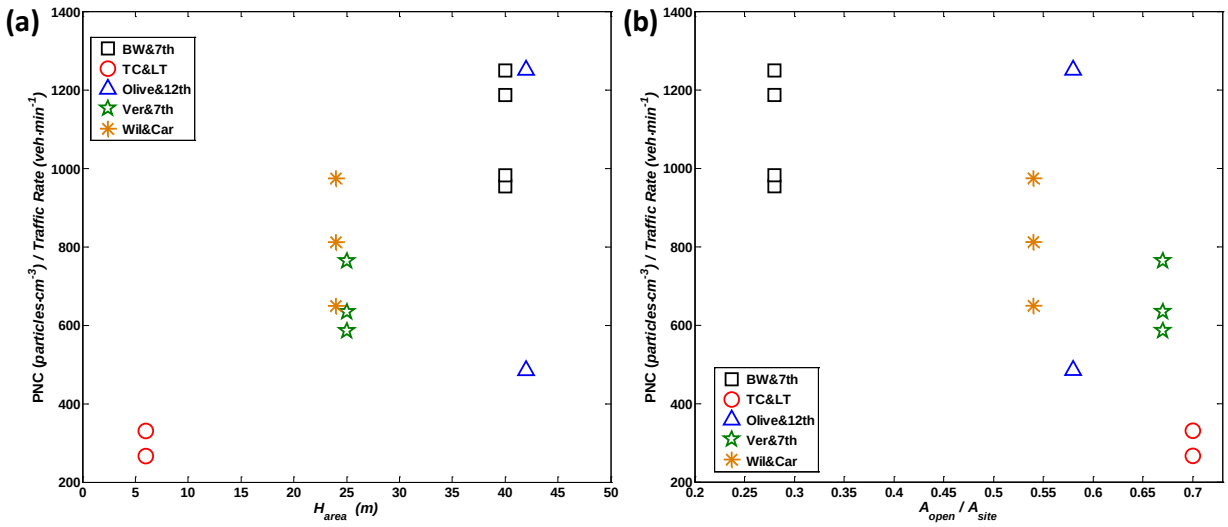



**Figure 14. Comparisons of traffic corrected [UFP] vs. building area weighted building heights ($H_{area}$) and the ratio of open space to sampling area ($A_{open}/A_{site}$) for the morning sampling events.**

The traffic-corrected [UFP], which is defined simply as observed [UFP] divided by observed traffic flow rate, showed a strong relationship with $Ar_{area}$ (Figure 15 and Eq. 6):

$$\frac{[UFP]}{Traffic\ flow\ rate} = 286 \times \log(Ar_{area}) + 1193 \qquad (R^2 = 0.67) \tag{6}$$

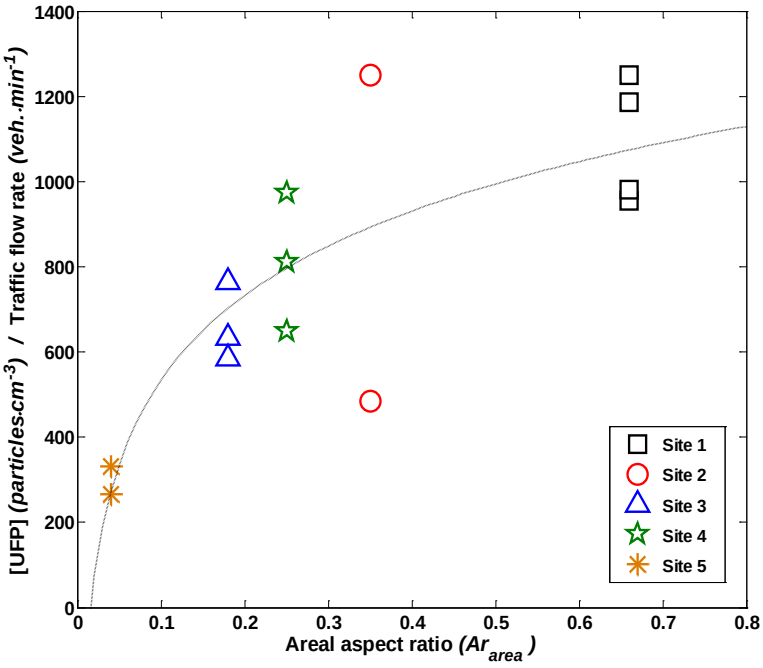



**Figure 15. Relationship between area aspect ratio ($Ar_{area}$) and [UFP] normalized to traffic flow rates in the morning (R2=0.67).**

Due to a log form of the best fit curve, [UFP] increase sharply with $Ar_{area}$ in a low $Ar_{area}$ regime, but in a high $Ar_{area}$ regime, the slope of [UFP] elevation with $Ar_{area}$ is dampened. The log form of the best fit implies that once the aspect ratio is above a critical level, recirculation cells form in the lower part of building canopy (Liu et al. 2004). Once the in-canopy recirculation cells are a dominant feature, additional increases in aspect ratio have a weaker effect on ground-level vehicular pollutants because recirculation cells separate ground-level in-canopy air from upper ambient air. More details about air flow impacts on spatial distributions of air pollutants will be presented in a

separate study (Ranasinghe et al. 2015). Consequently, our results suggest that built environments, particularly the areal aspect ratio ($Ar_{area}$) and traffic conditions, determine the spatial patterns of UFP levels under calm morning conditions.

#### v.4.1.4.2. Unstable afternoon conditions

In the afternoon, the areal aspect ratio does not explain the [UFP] between sampling sites as well as it does in the morning (Figure 14). This is not surprising given an increased meteorological influence due to more diverse meteorology between sampling sites/days in the afternoons. Differences in conditions such as deeper boundary layer depth, stronger turbulence intensities (Choi et al. 2011; Hussein et al. 2006; Seibert et al. 2000), and additional UFP source from photochemical secondary production may affect [UFP] (Hu et al. 2012; Ning et al. 2007).

In the afternoon, vertical fluctuations of winds ($\sigma_w$) are the strongest factor in determining UFP levels, as shown by the straightforward relationships between [UFP] and $\sigma_w$ ($R^2$=0.43, Figure 16). As the surface atmosphere becomes more turbulent (higher $\sigma_w$), UFP levels decrease due to stronger atmospheric dispersion. The effect of $\sigma_w$ on [UFP] becomes more evident when [UFP] are corrected for traffic flow rates; $R^2$ values increase up to 0.83 (Figure 16b). Note that two data points obtained from *Site2* clearly departed from the trend and thus were excluded from the curve fitting analyses. *Site2* has very infrequent traffic with traffic flow rates of only 1/3 to 1/5 of other sampling sites (Table 2). Relatively high [UFP] despite minimal vehicular emissions at this site are likely caused by an influx from nearby busy streets combined with a contribution from secondary production. This implies that horizontal wind fields play a critical role in understanding the heterogeneous spatial distributions of pollutants, particularly on streets with little pollution of their own.

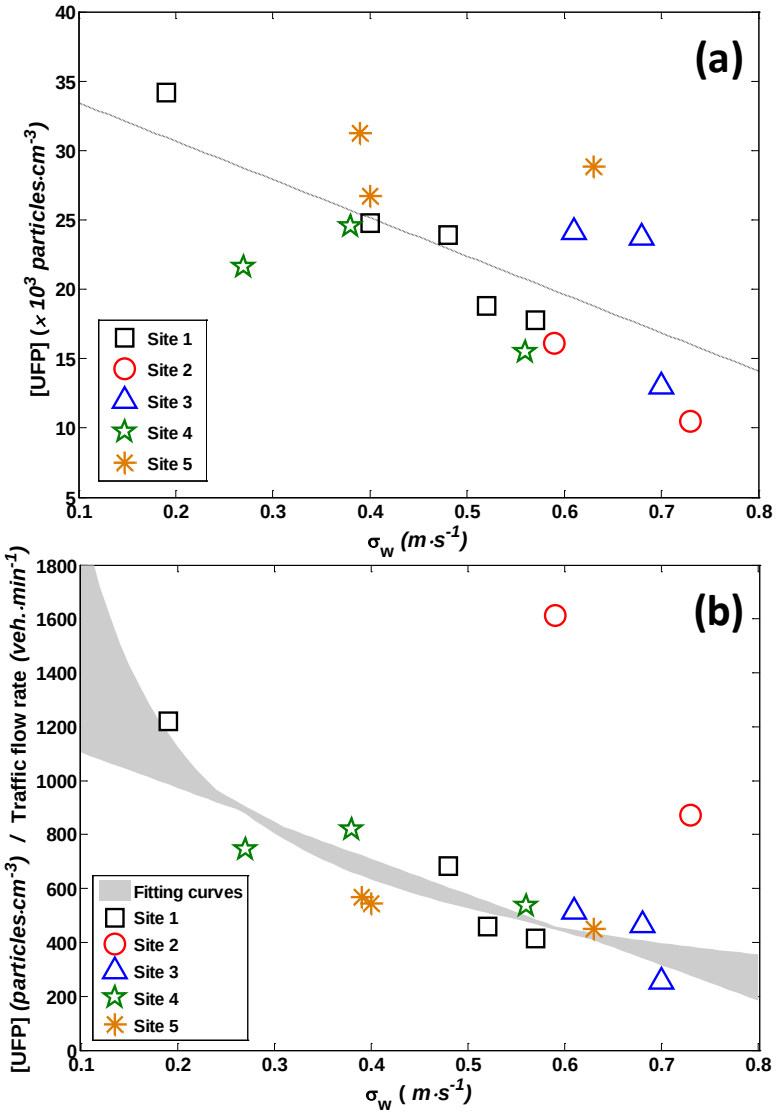



**Figure 16. Relationship between [UFP] and $\sigma_w$ for afternoonsampling events. (a) [UFP] vs $\sigma_w$ and (b) [UFP] normalized by observed traffic flows vs. $\sigma_w$. The grey area represents the range of best fit curves as described in the text. The values for *Site2* are removed from the analysis due to very low traffic counts on the street and subsequent likely contributions from nearby streets and other sources (see text.)**

There is not an obvious theoretical basis from which to derive a quantitative relationship between $\sigma_w$ and traffic normalized [UFP], and our data do not span a large enough range to suggest the best form. Thus, several types of simple curve fits were applied: linear, exponential, logarithmic, and power (Eq. 7). The linear form resulted in slightly lower $R^2$ value compared to others. Although the fitted values disperse widely at both ends, all fits showed a good agreement within observed $\sigma_w$ range; the shaded area in Figure 16b shows the maximum and minimum values of the curve fits.

$$\begin{aligned}\frac{[UFP]}{Traffic\ flow\ rate} &= -1315\cdot\sigma_w + 1236 && (R^2 = 0.74)\\ &= 1645\cdot\exp(-2.21\cdot\sigma_w) && (R^2 = 0.80)\\ &= -563\cdot\log(0.76\cdot\sigma_w) && (R^2 = 0.81)\\ &= 296\cdot\sigma_w^{-0.83} && (R^2 = 0.83)\end{aligned} \tag{7}$$

The strong relationships between traffic-normalized [UFP] and $\sigma_w$ emphasize the role of surface micro-meteorology in determining afternoon air pollution levels. However, we hypothesize that

built environments also affect air pollutant distributions indirectly by altering the turbulence intensities. To support this hypothesis, the comparisons between heterogeneity of building morphology for each site and observed surface turbulence parameters are shown in Figure 17.

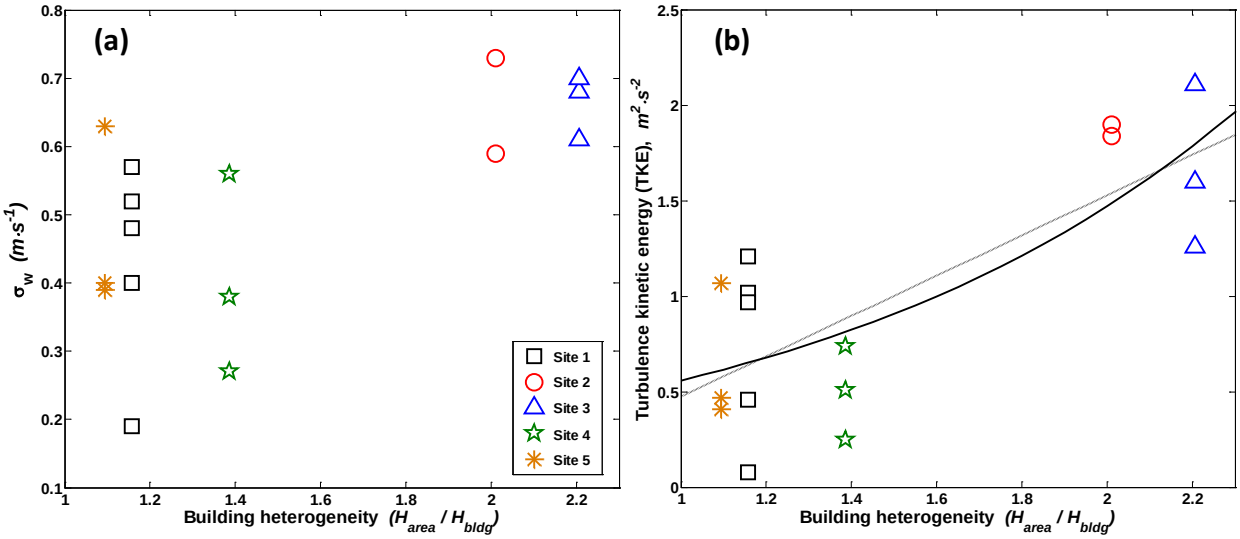



**Figure 17. Afternoon relationships between building heterogeneity vs. turbulence intensities: (a) vertical fluctuation of winds and (b) total turbulence kinetic energy (TKE) defined as $TKE = 1/2\sqrt{\sigma_u^{\ 2} + \sigma_v^{\ 2} + \sigma_w^{\ 2}}$. Dotted and solid lines in (b) represent the best fits in linear ($R^2$=0.60) and exponential ($R^2$=0.60) forms, respectively, for illustration of the increase trends of TKE with building heterogeneity.**

The fluctuations of vertical winds that showed the strongest relationships with the afternoon [UFP] appear to be somewhat related to building heterogeneity (Figure 17a). However, the daily variations of $\sigma_w$ for each sampling site are so large compared to the magnitude of the observed range that the relationships are not strong. On the other hand, the most heterogeneous sites, *Site2* and *Site3*, had consistently stronger $\sigma_w$ than other more homogeneous sites. As expected, the surface level turbulence kinetic energy for each site sharply increased with building heterogeneity (Figure 17b). This relationship implies that a heterogeneous building configuration enhances surface level turbulence, intensifying atmospheric dispersive capacity and reducing surface air pollutant levels under unstable daytime conditions.

## v.4.2. Developing high spatial resolution concentration maps using mobile air quality measurements

### *v.4.2.1. High spatial resolution concentration maps*

The 5 m spatial resolution maps shown in Figure 18 are the result of careful consideration of several underlying data processing issues of mobile monitoring data. With the use of a background correction, we were able to average data over sampling events on different days, and thus over a higher number of runs. After averaging the data over varying effects of micro-meteorology, traffic volume, traffic fleet composition, and background concentrations over different sampling events on different days, resulting UFP concentration maps retain the robust block and sub-block scale features of the concentration variation, making them a potentially useful tool in identifying pollution hot-spots at the block or the sub-block scale.

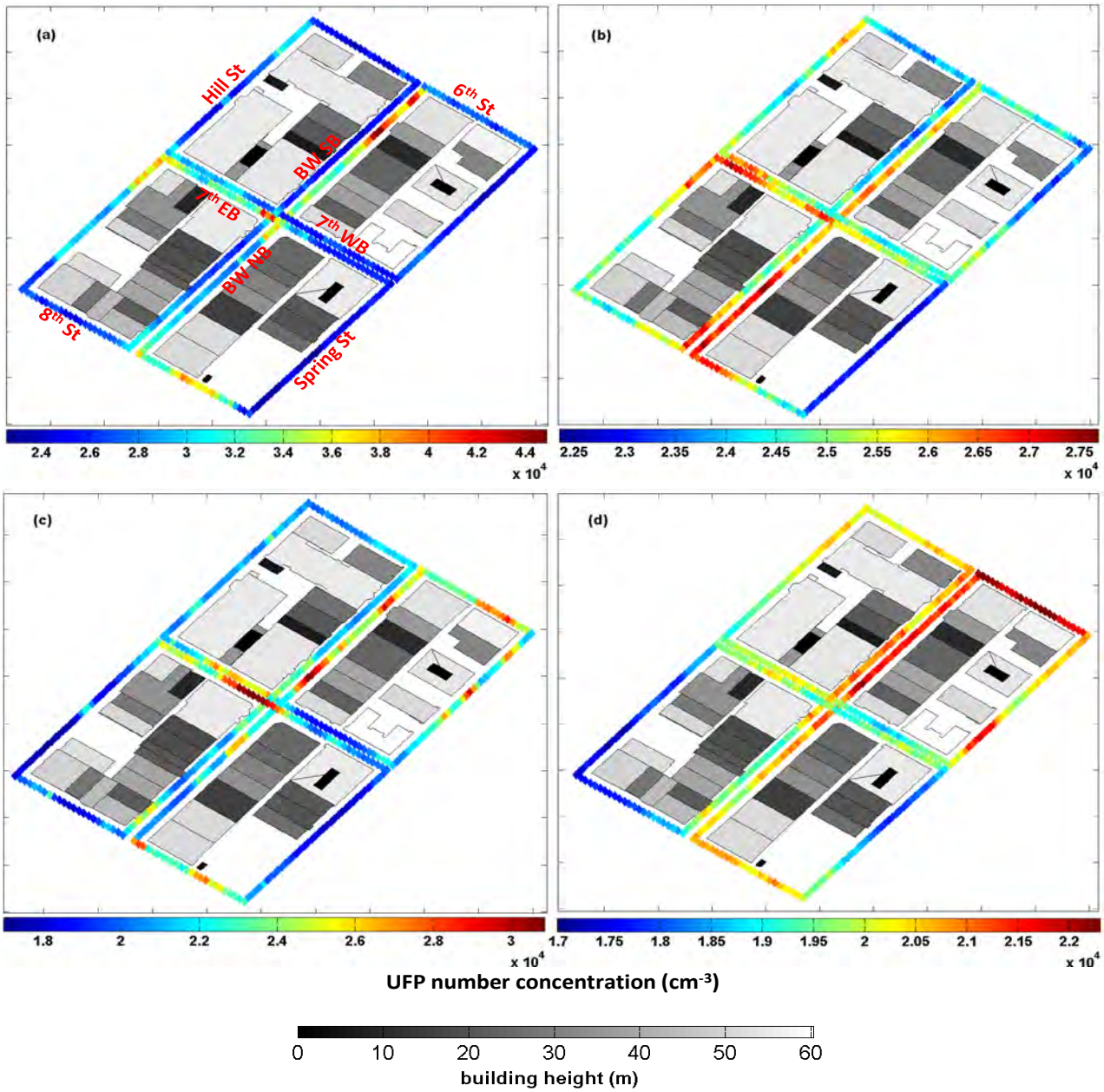



**Figure 18. Spatial variation of background corrected UFP concentrations averaged over (a, b) morning and (c, d) afternoon sampling events over three days for (a, c) data including HEV-related spikes and (b, d) data excluding HEV-related spikes. The spatial resolution of the maps is 5 m. The heights of the buildings in the nearby area is shown in gray scale.**

Figure 18 shows the UFP concentration maps at 5 m spatial resolution for the full data set including HEV-related spikes ("raw", Figure 18(a) and Figure 18(c)) and also for the data with HEV-related spikes removed ("spikes removed", Figure 18(b) and Figure 18(d)). The dominant feature of the "raw" concentration maps are the 'hot-spots' that appear at and near intersections, including both the area where queues form and where vehicles accelerate away from intersections. Once the HEV-related spikes are removed, features appear that reveal the influence of the built environment on street level UFP concentrations. While "raw" concentration maps are important in exposure analysis, maps with HEV spikes removed help understand various other factors influencing small spatial scale variations of the UFP concentration.

The "spikes removed" data reveal features at both the block- and the sub-block scales. Figure 18(d) shows that at the block-scale, 6th street in the afternoon shows the highest concentrations despite having low average traffic volume compared to other streets. On 7th street, in both morning and afternoon, there are generally higher concentrations on the east-bound side compared to the west-

bound side, despite having nearly the same traffic flow in both directions. Moreover, Figure 18(b) shows that at the sub-block scale, in the morning on Broadway north-bound near the intersection of 8th and BW, the south end of the block has elevated concentration in comparison to the queue forming at the north end. A similar situation can be noted on 8th street, just west of the intersection of BW and 8th, where the east end of the block shows elevated concentration in comparison to the queue forming at the west end. Many of these features can be explained by the surface level wind flow patterns that are heavily influenced by the local built environment, traffic patterns, and non-vehicle local sources. More detailed analyses of the effects of surrounding building morphology, micro-meteorological variations, and air flow patterns due to the built environment and traffic patterns on concentration distributions at different scales will be presented separately (Ranasinghe et al., in prep.).

#### *v.4.2.2. High Estimation of the minimum number of runs needed for representative concentration values*

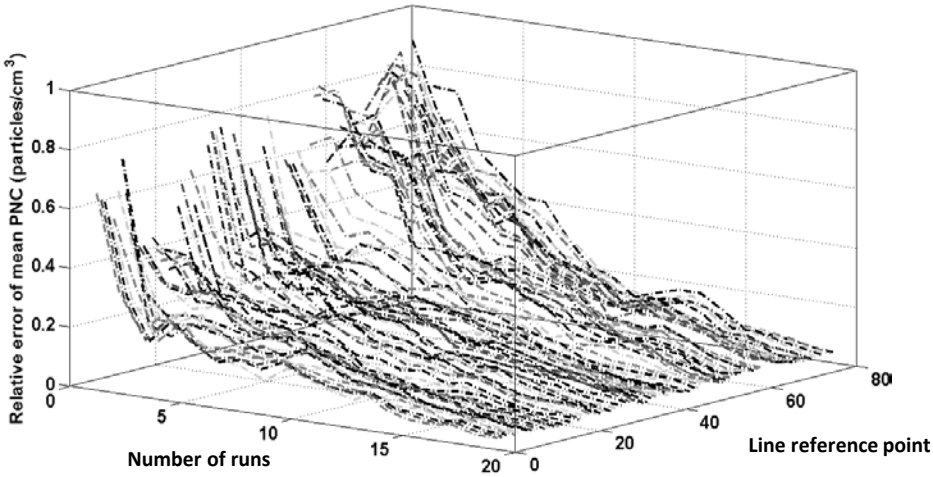
Due to transient and small spatial scale variations in air pollution concentrations, a single run of mobile measurements is clearly unable to capture a representative concentration field of an area. This raises the question of how many repeated measurements are needed to estimate a representative concentration field. Clearly this question is dependent on variability in meteorological as well as traffic conditions; features that in some cases might require very large amounts of sampling. In this study, typical morning and afternoon conditions at *Site1* was investigated. The average wind speeds on BW were 1.2 ± 0.2 m $s^{-1}$ for mornings and 1.3 ± 0.5 m $s^{-1}$ for afternoons. The most prevalent wind direction on BW was SW in both morning and afternoon sampling events. On 7th, the average wind speeds were 1.0 ± 0.1 m $s^{-1}$ for mornings and 1.5 ± 0.1 m $s^{-1}$ for afternoons. The most prevalent wind directions on 7th were ESE in the mornings and NE in the afternoons (Table 9). To investigate this question, the following exercise was performed on the UFP number concentration data set.

**Table 9. Average surface meteorology at Broadway and 7th (*Site1*). Here, $u_*$ is the friction velocity, $\sigma_w$ is the variance of vertical wind velocity and TKE is the turbulent kinetic energy*.**

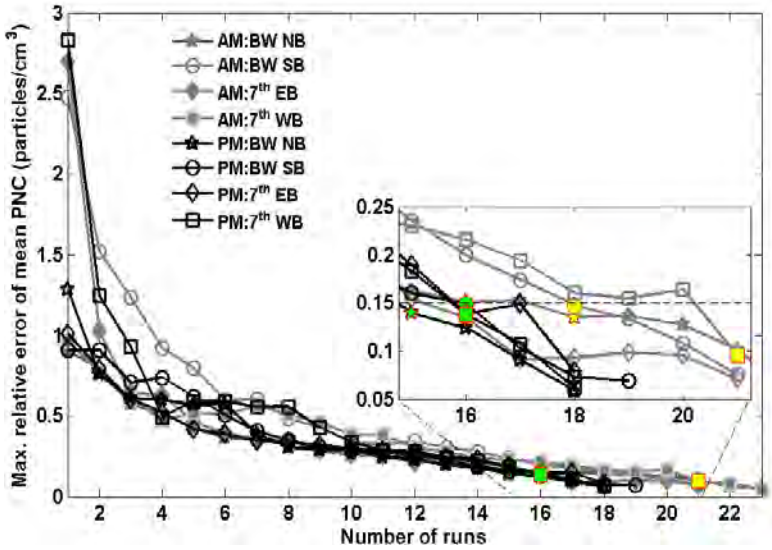
| Date | Temp. (°C) | $u_*$ (m $s^{-1}$) | $\sigma_w$ (m $s^{-1}$) | TKE ($m^2$ $s^{-2}$) | Temp. (°C) | $u_*$ (m $s^{-1}$) | $\sigma_w$ (m $s^{-1}$) | TKE ($m^2$ $s^{-2}$) |
|---|---|---|---|---|---|---|---|---|
| | Morning | | | | Afternoon | | | |
| 7/1/2013 | 25.9 | 0.23 | 0.37 | 0.47 | 32.2 | 0.23 | 0.40 | 0.46 |
| 7/3/2013 | 23.0 | 0.17 | 0.35 | 0.47 | 22.5 | 0.36 | 0.57 | 0.97 |
| 7/5/2013 | 20.6 | 0.19 | 0.30 | 0.47 | 24.0 | 0.15 | 0.48 | 1.21 |

First, all morning runs and all afternoon runs from the background-corrected concentration data set were collected separately. Each of these sets had runs spanning several days; many with fairly similar meteorological and traffic conditions (Table 5). For mornings, up to 22 runs were available for BW south-bound and 7th east-bound and 24 runs for BW north-bound and 7th west-bound. For afternoons, up to 19 runs were available for BW south-bound and 20 runs for other streets. For each street, at each line reference point, runs were selected at random (without replacement) and the mean concentration was calculated using an increasing number of runs, up to one less than the total number of runs available. This process was repeated 10 times for each street, choosing runs in different random order. For the sets of 10 repeated mean UFP concentration calculations at different reference points and for different number of averaged runs, the relative error (standard deviation normalized by mean) was calculated and plotted (Figure 19(a)). As shown in Figure 19(a), the rate of decrease in relative error varies among reference points along a given street. For simplicity, the maximum relative error along each street is considered and plotted against number of runs averaged for HEV "spikes removed" data (Figure 19(b)) and for HEV "spikes retained" data (Figure 19(c)). The minimum number of runs needed for the relative error to drop below 0.15 is calculated (the green or yellow symbols on each plot in Figure 19(b) and Figure 19(c)) and considered as the minimum number of runs needed for a representative UFP concentration value.

(a)




(b)




(c)

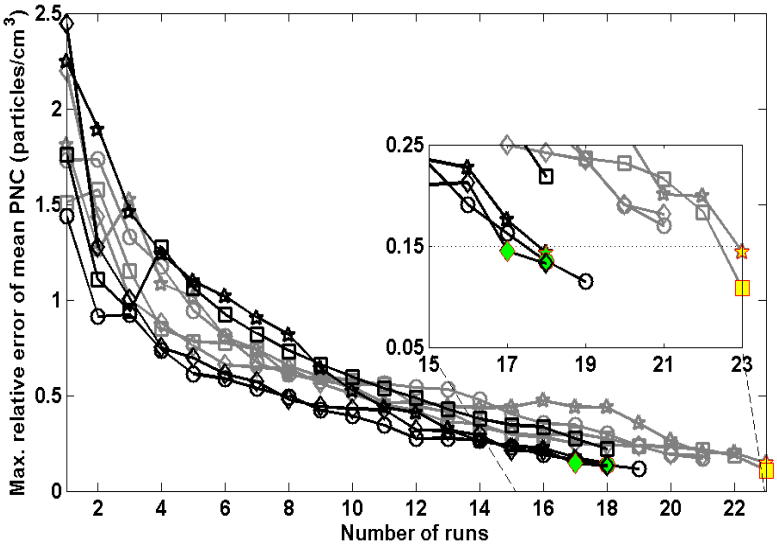



**Figure 19. (a) The relative error of repeated calculations of mean concentration of HEV "spike removed" data, for different numbers of averaged afternoon runs included in the averaging (x-axis), at each line reference points along a single example street (BW SB) (y-axis.) (b, c) The variation of maximum relative error along different street segments vs. the number of averaged runs for morning (AM) and afternoon (PM) sampling events (b) for HEV "spikes removed" data and (c) for HEV "spikes retained" data. The green and yellow symbols denote the points at which the relative error is at or below 0.15. The spatial resolution of the maps considered is 5 m.**

The estimate of the minimum number of runs needed for representative UFP concentration values at 5 m spatial resolution varies somewhat from street to street and is dependent on the data filters applied (Figure 19(b) and Figure 19(c)). For HEV "spikes removed" data, the maximum relative error along each street vs. number of averaged runs (Figure 19(b)) drops rapidly (in the first 2–7 runs). The maximum relative error along streets also drops rapidly, from the initial values of 282–90% to 50% at 4–9 runs, after which it decreases more slowly, reaching 15% at 15–21 runs. Hence the estimate of the minimum number of runs needed for representative concentrations at 5 m spatial resolution ranges between 16–21 runs for the mornings and between 15–16 runs for the afternoons (Figure 19(b)). For mornings when 16 runs are included, the average relative error considering all four streets is 11%. For afternoons when 15 runs are included, the average relative error considering all four streets is 9%. The morning sampling events usually have low wind speeds (Table 9). Consequently, the TKE and variance of $\sigma_w$ are lower in the mornings in comparison to the afternoons (Table 9), denoting lower atmospheric turbulence and mixing rates. The need for more runs for the morning sessions can be attributed to the lower mixing rates, resulting in a stronger influence of local sources on pollutant concentrations.

The inclusion of transient and large HEV spikes generally increases the minimum number of runs needed for all the streets and for both AM and PM sampling events (Figure 19(c)). Similar to the HEV "spikes removed" data, morning sampling needs more runs compared to afternoon sampling. For HEV "spikes retained" data, the maximum relative error along each street vs. number of runs averaged drops slowly compared to HEV "spikes removed" data. The maximum relative error along streets also drops from the initial values of 244–143% to 50% at 8–14 runs and subsequently drops below 15% with additional runs. For all the streets, maximum relative error drops below 22% at 21–23 runs for the mornings and at 17–18 runs for the afternoons. Hence, we conclude that the minimum number of runs needed for representative UFP concentrations at 5 m spatial resolution is at least 21–23 runs for the mornings and at least 17–18 runs for the afternoons.

These results apply only to UFP concentrations because the minimum number of runs needed for representative concentration values depends on the magnitude of variance of the data set. Hence the results depend on the air pollutant considered. We also showed that the results depend on the data filters applied (Figure 19(c)). For both HEV "spikes removed" and "spikes retained" data sets, the initial values of the maximum relative error markedly decreased when spatial resolution was decreased to 10 m. The minimum number of runs needed for representative concentration values generally decreased for all the streets and for both AM and PM sampling events.

In their effort to assess the minimum number of runs needed for representative concentrations at the street-scale, Van Poppel et al. (2013) and Peters et al. (2013) used data from moderately sized sets of mobile monitoring runs (20–24 runs), selecting different numbers of runs at random (without replacement) and averaging them to calculate the street means or medians. They used 1 s time resolution data collected by a MMP travelling at an average speed 2.7 m $s^{-1}$. The minimum number of runs needed to obtain representative concentrations was defined as the point at which these mean/median values calculated using a sub-set of runs came within a certain percentage deviation (15%–25%) of their “representative values”. They defined the “representative values” as the mean/median of all available runs. Peters et al. (2013) using a 15% deviation percentage concluded that for UFP concentrations the number of runs needed was 16 and 18 for the two sites considered. Van Poppel et al. (2013) used a portion of the data set used in Peters et al. (2013) study and concluded that for UFP concentrations, a 25% deviation could be achieved from 10–16 and 8–16 runs depending on the street, for analysis without and with background correction, respectively. In a continuation of this work, Van den Bossche et al. (2015) used a large dataset (96–256 runs) of BC measurements for a similar exercise. BC was measured at 1 s time resolution but as discussed earlier, the spatial resolution of these data is variable and complex due to the use of a post-data processing technique (ONA). Allowing replacement in the random selection of runs and employing

a background correction, trimmed mean, and 25% deviation, they concluded the number of runs needed is 14–61 depending on the street and also showed that the required number of runs rose to 108 when considering a spatial resolution of 20 m. Prior studies (Peters et al., 2013; Van Poppel et al., 2013) conducted with small UFP data sets are different from this study in terms of both the way in which the minimum number of required runs is defined and in the spatial scale considered. Despite these differences, our estimate of the minimum number of runs needed for representative UFP concentration values is also comparable with these two prior studies.

### v.4.3. Statistical modeling of the micro-dynamics of UFP concentrations caused by traffic at street intersections

A statistical model was developed to understand the UFP concentrations being measured by MMP to determine how on-road sources and micro-dynamic parameters affect the measurements. By constructing a statistical model, it is possible to perform sensitivity tests in which it would produce UFP concentration results under various scenarios constructed to determine how best to reduce emission/exposure on- and near-roadways. The following sections of this report describes the fundamental of the statistical model and results that reflect the MMP measurements conducted in this study.

#### *v.4.3.1.* Statistical model development

The concentration of UFP at a point in time and space along the transect is assumed to be a function of both contemporaneous and past variations in emissions of UFPs and in environmental conditions influencing the dispersion thereof. If at every time, $t$ :

- $y_t$ is the concentration of UFP
- $X_t$ is a vector of observed factors affecting the concentration of UFP
- $\epsilon_t$ is the cumulative effect of unobserved factors affecting the concentration of UFP

Then the concentration of UFP at time $t$ may be written:

$$y_t = f(X_t, X_{t-1}, X_{t-2}, \dots, \epsilon_t)$$

Or, if $f(\cdot)$ is assumed to be linear in terms (which may include higher order terms, such as quadratics or interactions of factors), we have the infinitely distributed lag model:

$$y_t = \alpha + \beta_t \cdot X_t + \beta_{t-1} \cdot X_{t-1} + \beta_{t-2} \cdot X_{t-2} + \dots + \epsilon_t$$

$$y_t = \alpha + \sum_{l=0}^{\infty} [(\beta_{t-l}] \cdot X_{t-l}) + \epsilon_t$$

where $\alpha$ is the intercept term. This model may be estimated by ordinary least squares provided that the time series of UFP concentrations is stationary (and thus less prone to producing spurious results). With this formulation:

- $\beta_{t-l}^{k}$ is the "dynamic multiplier"—the effect of factor $k$ , observed at lag length $l$ , on contemporaneous UFP
- $\sum_{t=0}^{\infty} \beta_{t-l}^{k}$ is the "cumulative multiplier"—the cumulative effect of all past observations of factor $k$ on contemporaneous UFP or, alternately, the cumulative effect of a single observation of factor $k$ on contemporaneous and all future observations of UFP

Two issues remain, however. First, data on infinite lags of the factors affecting the concentration of UFP are unavailable. Second, the estimates of the coefficients will be biased if the unobserved effect, t, is correlated over time. This is likely, particularly with high frequency data. So rather than estimate the dynamic and cumulative multipliers directly, we conduct consistent ordinary least squares estimation by including lagged values of UFP (and, implicitly, lagged values of the unobserved effect) as additional regressors. This yields the autoregressive distributed lag model:

$$y_t = \delta + \sum_{l=1}^{q}(\theta_{t-l} \cdot y_{t-l}) + \sum_{l=0}^{r}(\delta_{t-l} \cdot X_{t-l}) + v_t$$

where $q$ and $r$ are the chosen lag lengths for lagged values of UFP and the observable factors affecting the concentration of UFP, respectively. (The optimal choice of $q$ and $r$ will vary depending on the application; we discuss our choices in this setting further below.)

Dynamic and cumulative multipliers of interest may then be recovered as functions of the estimated coefficients. This proceeds by transforming the estimated Eq. 3 into Eq. 2 in this sub-section through successive substitution of the estimated Eq. 3 for $y_{t-l}$. Standard errors for the multipliers may then be approximated using the delta method. Simulations of synthetic scenarios may then be conducted using the estimated Eq. 3 by setting all factors (other than those specified by the scenario) to their long-run means and forecasting the resulting evolution of the concentration of UFP. The above discussion suggests that estimation should be conducted using the autoregressive distributed lag model so long as $y_t$ is stationary within runs, and $\epsilon_t$ is serially correlated within runs.

#### v.4.3.1.1. Statistical model specification

Our preferred specification for the model is present in Table 10. The numbers of various lags are selected to maximize explanatory power, while minimizing concerns about collinearity and overfitting. An augmented Dickey-Fuller test rejects at the 1 percent significance level, for each of the twelve runs, the null hypothesis that the concentration of UFP is non-stationary. Moreover, the first-and second-order partial autocorrelations of the residuals of the infinitely distributed lag model are statistically different from zero at the 5 percent level for all runs (and at the 1 percent level for all but one run); consequently at least two lags of the concentration of UFP should be included as additional explanatory variables.

This specification of the autoregressive distributed lag model features fifteen lags of the concentration of UFP. It includes contemporaneous and two lags of the following variables: i) mobile monitoring platform (MMP) lane, ii) MMP idle status, iii) MMP speed, iv) building height (average), v) building height (east-west difference).

This model also contains contemporaneous and eight lags of the following variables: i) intersection status, ii) acceleration events (ongoing, oncoming, left-crossing or right-crossing traffic), iii) light-duty vehicle (ongoing, oncoming, left-crossing or right-crossing traffic), iv) heavy-duty vehicle (ongoing, oncoming, left-crossing or right-crossing traffic), and v) bus (ongoing, oncoming, left-crossing or right-crossing traffic). Finally, the model includes a fourth-order polynomial in position along the transect index. Because the Goldfeld-Quandt test rejects at the 1 percent significance level, for most combinations of runs, the null hypothesis that the unobserved effects have equal variances, heteroskedasticity-robust standard errors are used throughout.

**Table 10. Model estimated UFP concentrations**

| | Coef. | Robust Std. Err. | t | 95% Conf. Interval | |
|---|---|---|---|---|---|
| Ultrafine particles | - | - | - | - | - |
| *L1* | 1.570023 | (0.032944) | 47.66 | 1.505429 | 1.634618 |
| *L2* | -0.97724 | (0.06129) | -15.94 | -1.09742 | -0.85707 |
| *L3* | 0.462292 | (0.058039) | 7.97 | 0.348494 | 0.576091 |
| *L4* | -0.1962 | (0.051342) | -3.82 | -0.29687 | -0.09553 |
| *L5* | 0.111163 | (0.052347) | 2.12 | 0.008525 | 0.213801 |
| *L6* | -0.0746 | (0.052223) | -1.43 | -0.177 | 0.027793 |
| *L7* | 0.047506 | (0.046826) | 1.01 | -0.04431 | 0.139319 |
| *L8* | -0.01174 | (0.044584) | -0.26 | -0.09915 | 0.075682 |
| *L9* | -0.02807 | (0.046768) | -0.6 | -0.11977 | 0.063625 |
| *L10* | 0.043222 | (0.045103) | 0.96 | -0.04521 | 0.131656 |
| *L11* | -0.00584 | (0.047927) | -0.12 | -0.09981 | 0.088138 |
| *L12* | -0.06238 | (0.052001) | -1.2 | -0.16434 | 0.039584 |
| *L13* | 0.064465 | (0.045479) | 1.42 | -0.02471 | 0.153637 |
| *L14* | -0.00614 | (0.037542) | -0.16 | -0.07975 | 0.067465 |
| *L15* | 0.003634 | (0.020206) | 0.18 | -0.03598 | 0.043253 |
| MMP lane number | 0.021661 | (0.023973) | 0.9 | -0.02534 | 0.068665 |
| *L1* | -0.03214 | (0.038736) | -0.83 | -0.10809 | 0.043809 |
| *L2* | 0.020199 | (0.031948) | 0.63 | -0.04244 | 0.08284 |
| MMP speed | -0.00108 | (0.000974) | -1.11 | -0.00299 | 0.000827 |
| *L1* | -0.00027 | (0.000519) | -0.52 | -0.00128 | 0.00075 |
| *L2* | 0.001176 | (0.000605) | 1.95 | -9.17E-06 | 0.002362 |
| MMP speed$^2$ | 9.63E-06 | (9.14E-06) | 1.05 | -8.29E-06 | 2.76E-05 |
| MMP speed × MMP speed [L1] | -2.41E-06 | (2.33E-05) | -0.1 | -4.80E-05 | 4.33E-05 |
| MMP speed × MMP speed [L2] | -4.3E-05 | (4.09E-05) | -1.05 | -0.00012 | 3.74E-05 |
| Light-duty (on-going) | -0.00498 | (0.010035) | -0.5 | -0.02466 | 0.014696 |
| *L1* | 0.009632 | (0.012011) | 0.8 | -0.01392 | 0.033183 |
| *L2* | 0.002232 | (0.010139) | 0.22 | -0.01765 | 0.02211 |
| *L3* | -0.00399 | (0.009377) | -0.43 | -0.02237 | 0.014398 |
| *L4* | 0.004014 | (0.008987) | 0.45 | -0.01361 | 0.021635 |
| *L5* | 0.03278 | (0.019689) | 1.66 | -0.00583 | 0.071385 |
| *L6* | -0.00351 | (0.01187) | -0.3 | -0.02678 | 0.019768 |
| *L7* | -0.0252 | (0.008029) | -3.14 | -0.04095 | -0.00946 |
| *L8* | -0.01705 | (0.010656) | -1.6 | -0.03794 | 0.003844 |
| Light-duty (on-coming) | 0.008546 | (0.008049) | 1.06 | -0.00724 | 0.024328 |
| *L1* | 0.006847 | (0.008713) | 0.79 | -0.01024 | 0.02393 |
| *L2* | -0.00211 | (0.007518) | -0.28 | -0.01685 | 0.012628 |
| *L3* | -0.00817 | (0.007961) | -1.03 | -0.02378 | 0.007441 |
| *L4* | -0.00085 | (0.007741) | -0.11 | -0.01602 | 0.014331 |
| *L5* | 0.016506 | (0.007749) | 2.13 | 0.001311 | 0.0317 |
| *L6* | 0.002587 | (0.007536) | 0.34 | -0.01219 | 0.017363 |
| *L7* | 0.001244 | (0.007086) | 0.18 | -0.01265 | 0.015137 |
| *L8* | -0.00774 | (0.006849) | -1.13 | -0.02117 | 0.005689 |

| | Coef. | Robust Std. Err. | t | 95% Conf. Interval | |
|---|---|---|---|---|---|
| Light-duty (cross from left) | -0.01417 | (0.009027) | -1.57 | -0.03187 | 0.00353 |
| *L1* | -0.00858 | (0.00935) | -0.92 | -0.02691 | 0.00975 |
| *L2* | -0.00891 | (0.010808) | -0.82 | -0.0301 | 0.012282 |
| *L3* | 0.017274 | (0.010686) | 1.62 | -0.00368 | 0.038227 |
| *L4* | -0.00414 | (0.011633) | -0.36 | -0.02694 | 0.018673 |
| *L5* | 0.002213 | (0.011937) | 0.19 | -0.02119 | 0.025617 |
| *L6* | 0.004934 | (0.009314) | 0.53 | -0.01333 | 0.023196 |
| *L7* | 0.017519 | (0.010199) | 1.72 | -0.00248 | 0.037518 |
| *L8* | 0.012175 | (0.012117) | 1 | -0.01158 | 0.035933 |
| Light-duty (cross from right) | 0.001778 | (0.00944) | 0.19 | -0.01673 | 0.020287 |
| *L1* | -0.00587 | (0.010189) | -0.58 | -0.02585 | 0.014112 |
| *L2* | -0.00602 | (0.009618) | -0.63 | -0.02488 | 0.012841 |
| *L3* | -0.00683 | (0.008405) | -0.81 | -0.02331 | 0.009653 |
| *L4* | -0.0074 | (0.009283) | -0.8 | -0.02561 | 0.010797 |
| *L5* | 0.005945 | (0.009092) | 0.65 | -0.01188 | 0.023772 |
| *L6* | 0.005077 | (0.013298) | 0.38 | -0.021 | 0.031152 |
| *L7* | 0.00422 | (0.008896) | 0.47 | -0.01322 | 0.021662 |
| *L8* | -0.00144 | (0.013014) | -0.11 | -0.02696 | 0.024073 |
| Heavy/medium-duty (on-going) | -0.00531 | (0.029522) | -0.18 | -0.06319 | 0.052579 |
| *L1* | 0.027436 | (0.065685) | 0.42 | -0.10135 | 0.156226 |
| *L2* | 0.004115 | (0.031977) | 0.13 | -0.05858 | 0.066813 |
| *L3* | -0.0637 | (0.038713) | -1.65 | -0.13961 | 0.012207 |
| *L4* | 0.027891 | (0.034249) | 0.81 | -0.03926 | 0.095044 |
| *L5* | -0.02322 | (0.027525) | -0.84 | -0.07719 | 0.030746 |
| *L6* | 0.059098 | (0.026915) | 2.2 | 0.006325 | 0.111871 |
| *L7* | 0.073935 | (0.032116) | 2.3 | 0.010964 | 0.136906 |
| *L8* | 0.018336 | (0.032131) | 0.57 | -0.04466 | 0.081336 |
| Heavy/medium-duty (on-coming) | -0.05056 | (0.023199) | -2.18 | -0.09605 | -0.00507 |
| *L1* | 0.039984 | (0.033778) | 1.18 | -0.02625 | 0.106212 |
| *L2* | 0.003812 | (0.036426) | 0.1 | -0.06761 | 0.075233 |
| *L3* | -0.03505 | (0.031004) | -1.13 | -0.09584 | 0.025739 |
| *L4* | 0.007392 | (0.031211) | 0.24 | -0.0538 | 0.068587 |
| *L5* | -0.04074 | (0.031396) | -1.3 | -0.1023 | 0.020822 |
| *L6* | 0.021185 | (0.034177) | 0.62 | -0.04583 | 0.088197 |
| *L7* | 0.011189 | (0.024192) | 0.46 | -0.03625 | 0.058624 |
| *L8* | -0.00397 | (0.023871) | -0.17 | -0.05078 | 0.042833 |
| Heavy/medium-duty (cross from left) | -0.04469 | (0.024822) | -1.8 | -0.09336 | 0.003982 |
| *L1* | -0.04284 | (0.036507) | -1.17 | -0.11442 | 0.028737 |
| *L2* | 0.025378 | (0.023068) | 1.1 | -0.01985 | 0.070608 |
| *L3* | 0.007655 | (0.042993) | 0.18 | -0.07664 | 0.091952 |
| *L4* | 0.073666 | (0.04015) | 1.83 | -0.00506 | 0.152388 |
| *L5* | -0.00061 | (0.024931) | -0.02 | -0.04949 | 0.048272 |
| *L6* | 0.061759 | (0.0572) | 1.08 | -0.05039 | 0.173912 |
| *L7* | -0.01299 | (0.029304) | -0.44 | -0.07045 | 0.044471 |
| *L8* | -0.01243 | (0.027829) | -0.45 | -0.06699 | 0.042139 |

| | Coef. | Robust Std. Err. | t | 95% Conf. Interval | |
|---|---|---|---|---|---|
| Heavy/medium-duty (cross from right) | -0.01111 | (0.023332) | -0.48 | -0.05686 | 0.034637 |
| *L1* | -0.00384 | (0.029673) | -0.13 | -0.06202 | 0.054345 |
| *L2* | -0.06229 | (0.029217) | -2.13 | -0.11958 | -0.00501 |
| *L3* | -0.00332 | (0.030497) | -0.11 | -0.06312 | 0.056471 |
| *L4* | 0.019426 | (0.028825) | 0.67 | -0.03709 | 0.075944 |
| *L5* | -0.0046 | (0.031342) | -0.15 | -0.06605 | 0.056858 |
| *L6* | 0.01017 | (0.038304) | 0.27 | -0.06493 | 0.085273 |
| *L7* | 0.060909 | (0.08433) | 0.72 | -0.10444 | 0.226257 |
| *L8* | -0.01557 | (0.02606) | -0.6 | -0.06667 | 0.035528 |
| Bus (on-going) | 0.008687 | (0.02706) | 0.32 | -0.04437 | 0.061745 |
| *L1* | 0.028092 | (0.030012) | 0.94 | -0.03075 | 0.086938 |
| *L2* | -0.04942 | (0.029853) | -1.66 | -0.10796 | 0.00911 |
| *L3* | -0.00908 | (0.035777) | -0.25 | -0.07923 | 0.061065 |
| *L4* | -0.01559 | (0.031622) | -0.49 | -0.07759 | 0.046415 |
| *L5* | -0.02942 | (0.047037) | -0.63 | -0.12164 | 0.06281 |
| *L6* | -0.01523 | (0.030386) | -0.5 | -0.07481 | 0.044351 |
| *L7* | 0.009399 | (0.036993) | 0.25 | -0.06314 | 0.081933 |
| *L8* | 0.003148 | (0.038153) | 0.08 | -0.07166 | 0.077955 |
| Bus (on-coming) | 0.042706 | (0.029711) | 1.44 | -0.01555 | 0.100961 |
| *L1* | -0.02875 | (0.021672) | -1.33 | -0.07125 | 0.013739 |
| *L2* | 0.031345 | (0.026528) | 1.18 | -0.02067 | 0.083358 |
| *L3* | 0.030845 | (0.0268) | 1.15 | -0.0217 | 0.083393 |
| *L4* | -0.03489 | (0.023118) | -1.51 | -0.08022 | 0.010436 |
| *L5* | -0.05048 | (0.021203) | -2.38 | -0.09206 | -0.00891 |
| *L6* | 0.042297 | (0.022479) | 1.88 | -0.00178 | 0.086372 |
| *L7* | -0.01136 | (0.022346) | -0.51 | -0.05518 | 0.03245 |
| *L8* | 0.008167 | (0.018929) | 0.43 | -0.02895 | 0.045281 |
| Bus (cross from left) | 0.03628 | (0.051009) | 0.71 | -0.06373 | 0.136295 |
| *L1* | 0.01695 | (0.056579) | 0.3 | -0.09399 | 0.127886 |
| *L2* | -0.01409 | (0.034951) | -0.4 | -0.08262 | 0.054436 |
| *L3* | 0.006299 | (0.052042) | 0.12 | -0.09574 | 0.10834 |
| *L4* | 0.052658 | (0.036783) | 1.43 | -0.01946 | 0.12478 |
| *L5* | 0.040381 | (0.038769) | 1.04 | -0.03563 | 0.116396 |
| *L6* | 0.077589 | (0.061751) | 1.26 | -0.04349 | 0.198667 |
| *L7* | 0.087997 | (0.059011) | 1.49 | -0.02771 | 0.2037 |
| *L8* | -0.08822 | (0.054626) | -1.62 | -0.19533 | 0.018884 |
| Bus (cross from right) | -0.11105 | (0.109929) | -1.01 | -0.32659 | 0.104489 |
| *L1* | -0.00908 | (0.037571) | -0.24 | -0.08274 | 0.064588 |
| *L2* | 0.028099 | (0.030309) | 0.93 | -0.03133 | 0.087527 |
| *L3* | -0.01513 | (0.023961) | -0.63 | -0.06211 | 0.031852 |
| *L4* | 0.033668 | (0.037116) | 0.91 | -0.03911 | 0.106443 |
| *L5* | 0.031641 | (0.033618) | 0.94 | -0.03428 | 0.097557 |
| *L6* | 0.04424 | (0.03808) | 1.16 | -0.03043 | 0.118906 |
| *L7* | -0.00868 | (0.028001) | -0.31 | -0.06358 | 0.046227 |

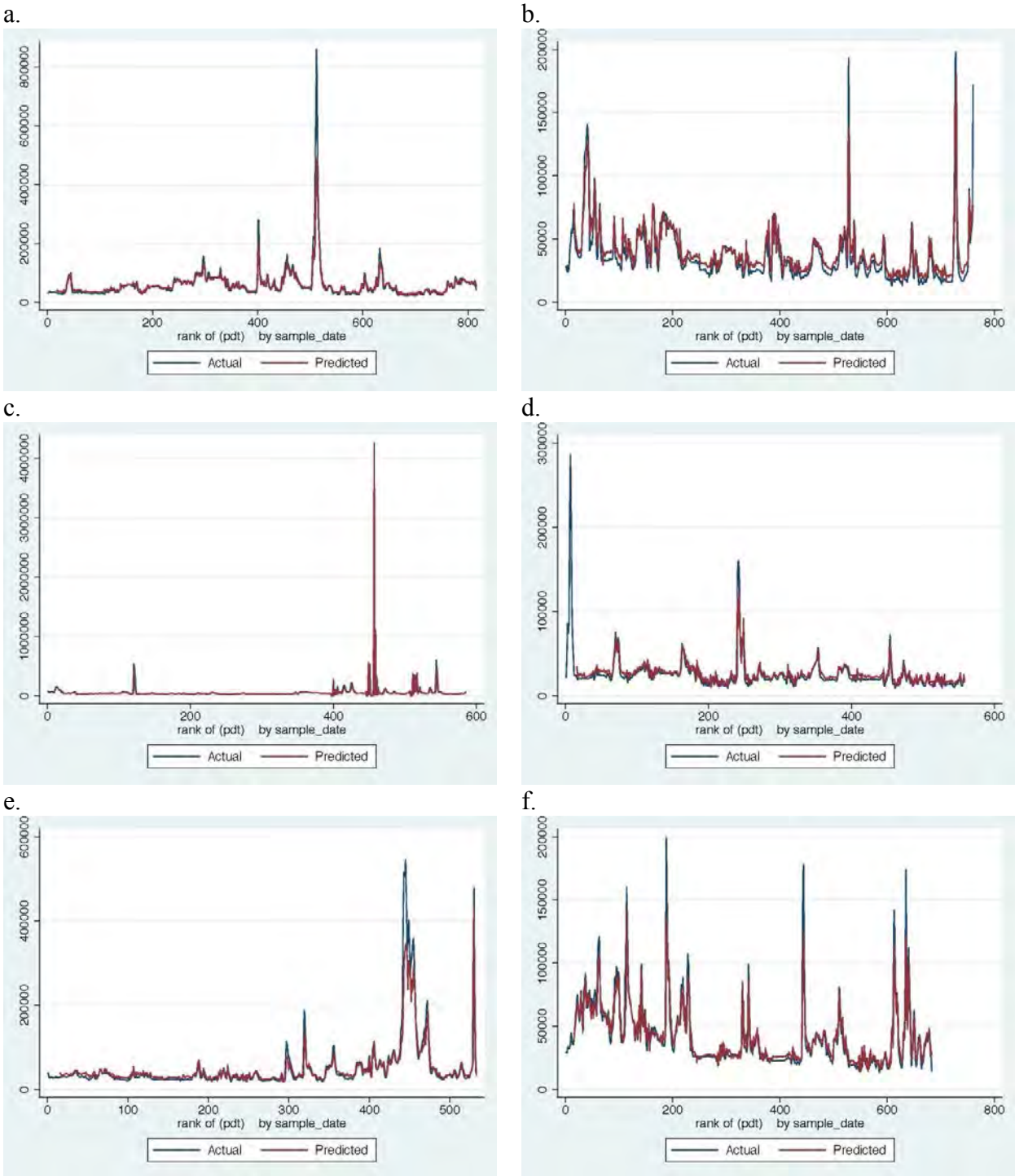
| | Coef. | Robust Std. Err. | t | 95% Conf. Interval | |
|---|---|---|---|---|---|
| *L8* | -0.00914 | (0.024205) | -0.38 | -0.0566 | 0.038321 |
| Acceleration (on-going) | -0.01689 | (0.038817) | -0.44 | -0.093 | 0.05922 |
| *L1* | 0.110624 | (0.046376) | 2.39 | 0.019694 | 0.201553 |
| *L2* | 0.027547 | (0.058163) | 0.47 | -0.0865 | 0.141589 |
| *L3* | 0.190339 | (0.141579) | 1.34 | -0.08726 | 0.467938 |
| *L4* | 0.209642 | (0.09996) | 2.1 | 0.013648 | 0.405637 |
| *L5* | 0.077272 | (0.055305) | 1.4 | -0.03117 | 0.18571 |
| *L6* | 0.042518 | (0.036491) | 1.17 | -0.02903 | 0.114067 |
| *L7* | 0.075313 | (0.041) | 1.84 | -0.00508 | 0.155703 |
| *L8* | 0.056461 | (0.044719) | 1.26 | -0.03122 | 0.144143 |
| Acceleration (on-coming) | 0.02797 | (0.039676) | 0.7 | -0.04983 | 0.105764 |
| *L1* | -0.07029 | (0.048921) | -1.44 | -0.16621 | 0.02563 |
| *L2* | -0.03298 | (0.07294) | -0.45 | -0.176 | 0.110035 |
| *L3* | -0.14594 | (0.142616) | -1.02 | -0.42557 | 0.133691 |
| *L4* | -0.22217 | (0.088564) | -2.51 | -0.39582 | -0.04852 |
| *L5* | -0.03385 | (0.064731) | -0.52 | -0.16077 | 0.093067 |
| *L6* | -0.01779 | (0.046737) | -0.38 | -0.10943 | 0.073843 |
| *L7* | -0.01851 | (0.044124) | -0.42 | -0.10503 | 0.068002 |
| *L8* | 0.019328 | (0.052231) | 0.37 | -0.08308 | 0.121739 |
| Acceleration (cross from left) | 0.020756 | (0.030988) | 0.67 | -0.04 | 0.081516 |
| *L1* | -0.02112 | (0.030586) | -0.69 | -0.08109 | 0.038854 |
| *L2* | 0.050372 | (0.06047) | 0.83 | -0.06819 | 0.168937 |
| *L3* | 0.049612 | (0.033572) | 1.48 | -0.01621 | 0.115438 |
| *L4* | 0.131799 | (0.224735) | 0.59 | -0.30885 | 0.572444 |
| *L5* | -0.05979 | (0.054833) | -1.09 | -0.1673 | 0.04772 |
| *L6* | 0.124471 | (0.107919) | 1.15 | -0.08713 | 0.336069 |
| *L7* | -0.0268 | (0.031938) | -0.84 | -0.08942 | 0.035826 |
| *L8* | 0.025801 | (0.030489) | 0.85 | -0.03398 | 0.085582 |
| Acceleration (cross from right) | -0.01862 | (0.039225) | -0.47 | -0.09553 | 0.058293 |
| *L1* | -0.02311 | (0.051862) | -0.45 | -0.1248 | 0.078575 |
| *L2* | -0.00725 | (0.027757) | -0.26 | -0.06168 | 0.047174 |
| *L3* | -0.06603 | (0.035083) | -1.88 | -0.13482 | 0.002757 |
| *L4* | 0.188406 | (0.149858) | 1.26 | -0.10543 | 0.482237 |
| *L5* | -0.01735 | (0.044845) | -0.39 | -0.10528 | 0.070574 |
| *L6* | 0.055406 | (0.036781) | 1.51 | -0.01671 | 0.127523 |
| *L7* | 0.023112 | (0.025406) | 0.91 | -0.0267 | 0.072926 |
| *L8* | -0.02465 | (0.028053) | -0.88 | -0.07965 | 0.030358 |
| Intersection | -0.02876 | (0.015911) | -1.81 | -0.05996 | 0.002436 |
| *L1* | 0.015379 | (0.01671) | 0.92 | -0.01738 | 0.048141 |
| *L2* | -0.01167 | (0.015824) | -0.74 | -0.0427 | 0.019359 |
| *L3* | 0.013785 | (0.01475) | 0.93 | -0.01514 | 0.042707 |
| *L4* | -0.01144 | (0.014643) | -0.78 | -0.04015 | 0.017268 |
| *L5* | 0.012445 | (0.013926) | 0.89 | -0.01486 | 0.03975 |
| *L6* | -0.01066 | (0.013016) | -0.82 | -0.03618 | 0.014865 |
| *L7* | 0.038222 | (0.014022) | 2.73 | 0.010728 | 0.065716 |

| | Coef. | Robust Std. Err. | t | 95% Conf. Interval | |
|---|---|---|---|---|---|
| *L8* | -0.00132 | (0.012158) | -0.11 | -0.02516 | 0.022518 |
| Avg. building height | -0.00051 | (0.00056) | -0.91 | -0.00161 | 0.000587 |
| *L1* | 0.000082 | (0.000693) | 0.12 | -0.00128 | 0.001441 |
| *L2* | 0.000438 | (0.000523) | 0.84 | -0.00059 | 0.001463 |
| Building height diff. | 4.56E-05 | (0.000376) | 0.12 | -0.00069 | 0.000783 |
| *L1* | 0.000312 | (0.000375) | 0.83 | -0.00042 | 0.001047 |
| *L2* | 0.000363 | (0.000294) | 1.23 | -0.00021 | 0.000938 |
| North-south wind | -0.00656 | (0.004016) | -1.63 | -0.01443 | 0.001315 |
| North-south wind × Avg. building height | 0.000356 | (0.000146) | 2.43 | 6.91E-05 | 0.000642 |
| East-west wind | 0.004917 | (0.004085) | 1.2 | -0.00309 | 0.012927 |
| East-west Wind × Building height diff | -0.00029 | (0.000231) | -1.28 | -0.00075 | 0.000158 |
| Linear position | -9.1E-05 | (0.000101) | -0.9 | -0.00029 | 0.000106 |
| Linear position$^2$ | 7.44E-08 | (7.87E-08) | 0.94 | -8.00E-08 | 2.29E-07 |
| Linear position$^3$ | -2.36E-11 | (2.34E-11) | -1.01 | -6.94E-11 | 2.23E-11 |
| Linear position$^4$ | 2.53E-15 | (2.35E-15) | 1.08 | -2.08E-15 | 7.13E-15 |
| Constant | 0.617515 | (0.091564) | 6.74 | 0.437983 | 0.797046 |

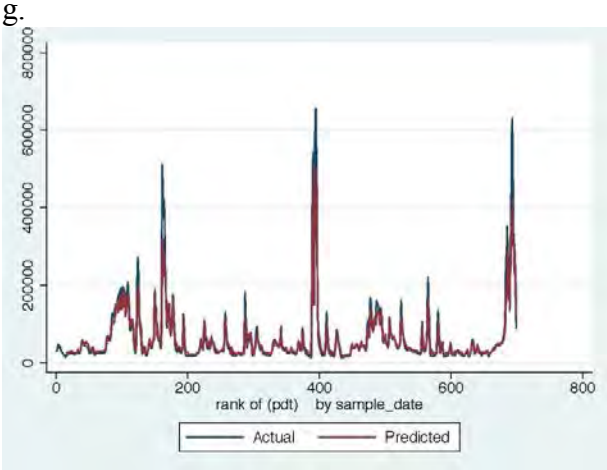
### *v.4.3.2. Out of sample model prediction: assessing accuracy*

To test for overfitting of our model, we perform 12-fold cross validation of the model iteratively leaving out each run, and using the remaining runs to predict the concentration of UFP for the withheld run. The resulting correlations of the actual and predicted concentrations range from 0.68 to 0.95, suggesting that the model predicts quite well out-of-sample.

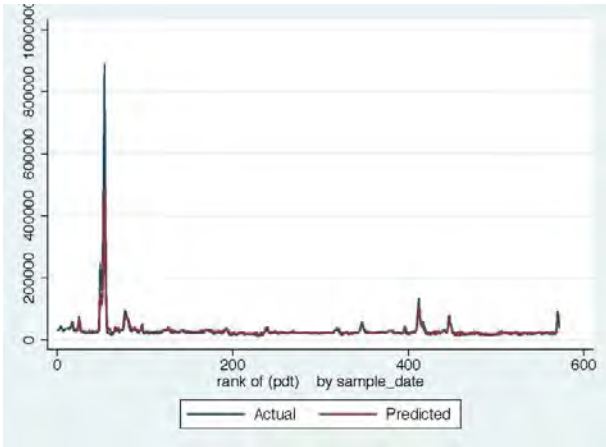
**Figure 20. Actual versus Predicted UPF Concentrations for AM and PM Transects.**



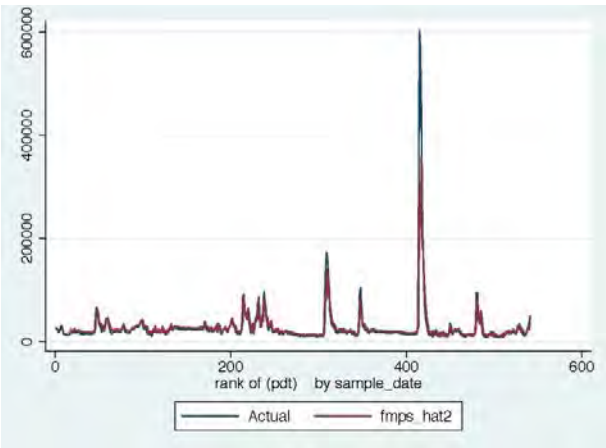
g.



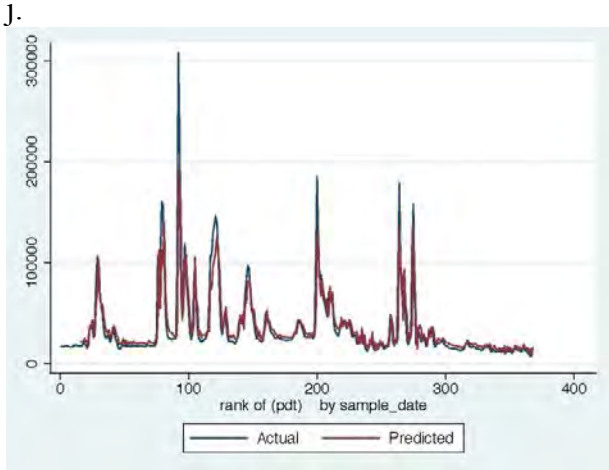

h.




i.




j.




### *v.4.3.3. Model development and general results*

Our empirical model presented in Table 10 contains over a hundred estimated parameters, several of which involved lagged variables as well as interaction effects between numerous variables. To ease the interpretation of this model we present the estimated cumulative effects on UFP of focal variables in Table 11. We estimate the cumulative effect of a given variable on UFP, e.g., the number of on-going light-duty vehicles in front of the MMP, by placing all the other parameters at their mean values. We then estimate the predicted concentration of UFP when the variable of interest is set to zero, e.g., no on-going light-duty vehicles, and compared that with predicted UFP concentration when the variable of interest is set to its mean level, e.g., on-going light-duty vehicles. In addition, we bound these mean cumulative effects by estimating their 95% confidence intervals.

**Table 11. Cumulative Impacts of Traffic Events.**

| All effects are in thousands of particles per $cm^3$. | | | | |
|---|---|---|---|---|
| | Impact | Std. Error | 95% Conf. Interval | |
| *MMP State of Motion and Speed* | | | | |
| Idle-to-moving | -28.65 | (-11.4) | -50.99 | -6.31 |
| Speed (m/s) | -15.32 | (-5.74) | -26.56 | -4.07 |
| Lane number | 15.73 | (-4.47) | 6.97 | 24.49 |
| *On-going Traffic* | | | | |
| Light-duty | 9.78 | (-14.01) | -17.69 | 37.24 |
| Medium & Heavy-duty | 142.76 | (-90.31) | -34.26 | 319.78 |

| All effects are in thousands of particles per $cm^3$. | | | | |
|---|---|---|---|---|
| | Impact | Std. Error | 95% Conf. Interval | |
| Buses | -25.66 | (-155.91) | -331.25 | 279.92 |
| Acceleration event | -58.7 | (-122.98) | -299.74 | 182.34 |
| *On-Coming Traffic* | | | | |
| Light-duty | -0.99 | (-16.77) | -33.85 | 31.87 |
| Medium & heavy-duty | -18.8 | (-57.18) | -130.88 | 93.28 |
| Buses | -21.31 | (-71.44) | -161.34 | 118.72 |
| Acceleration event | -158.72 | (-127.01) | -407.66 | 90.22 |
| *Crossing from the Left Traffic* | | | | |
| Light-duty | 30.05 | (-34.74) | -38.04 | 98.15 |
| Medium & heavy-duty | 80.79 | (-76.07) | -68.31 | 229.89 |
| Buses | 92.64 | (-111.96) | -126.8 | 312.08 |
| Acceleration event | 26.96 | (-98.47) | -166.05 | 219.97 |
| *Crossing from the Right Traffic* | | | | |
| Light-duty | -10.94 | (-58.6) | -125.8 | 103.92 |
| Medium & heavy-duty | -30.58 | (-162.81) | -349.68 | 288.53 |
| Buses | 54.04 | (-59.01) | -61.61 | 169.69 |
| Acceleration event | 52.59 | (-50.29) | -45.98 | 151.17 |
| *Built Environment* | | | | |
| Intersection | 320.15 | (-120.69) | 83.6 | 556.7 |
| Average building height (m) | 1.7 | (-4.5) | -7.13 | 10.52 |
| Building height differential (m)[a] | 233.06 | (-270.62) | -297.36 | 763.48 |

[a] Difference in height of neighboring buildings is positive if the building to the east is taller, and negative if the building to the west is taller.

As narrative strategy, we begin by describing variables that would become apparent as the MMP begins its transect sampling run. Thus, we started with variables associated with the i) MMP's state of motion and speed, ii) the on-going and on-coming traffic, iii) dynamics as it approaches and passes through an intersection, and finally, iv) the effects of the built environment and its interactions with meteorology.

***MMP State of Motion and Speed.*** As the MMP commences its transect sampling run, our model predicts that as it moves from *idle to moving* that the UFP concentrations will decline by -29 thousand/$cm^3$ with 95% intervals of -51 and -6. Notice that both confidence intervals are negative, indicating with a high degree of confidence that this change produces consistently lower UFP concentration. Similarly, as the *MMP speed* increases an additional meter per second, the measured UFP decline by 15 thousand/$cm^3$ with confidence intervals of -27 and -4. Both declines in UFP associated with changes in forward motion of the MMP may arise either by mixing or a partial vortex forming around the intake opening. As the MMP encounters slower moving traffic, causing it to *change lanes,* this is associated with an increase in UFP on the order of 16 thousand/$cm^3$ and are consistently positive (95% CI: 7 to 25 thousand/$cm^3$).

***On-going Vehicles.*** As the MMP travels along Broadway, it will encounter on-going traffic in immediately adjacent lanes and in front of it. The effect of adding the mean number of *on-going light-duty vehicles* is to increase the predicted UFP by 10 thousand/cm$^3$ with an 95% confidence interval of -18 and 37 thousand/cm$^3$. The predicted effect of adding the mean number of *on-going heavy and medium vehicles* was to increase UPF by over 142 thousand/cm$^3$ with a confidence interval of -34 and 320 thousand/cm$^3$. Notice that effect of adding on-going heavy duty vehicles as compared to light-duty vehicles is to increase UFP concentrations by more than a factor of 10. In contrast, the predicted effect increasing the mean number of *on-going buses* is to decrease UFP to -26 thousand/cm$^3$, with wide 95% confidence intervals of -331 and 280 thousand/cm$^3$. Thus, is likely due to both the high fraction of natural gas buses and their ability to entrain a larger volume of cleaner air from aloft into their wakes.

***On-coming Vehicles.*** The MMP also passes on-coming traffic in the opposite two lanes. For on-coming vehicles we anticipate two potentially countervailing effects. On-coming traffic releases fresh emissions which would increase UFP concentration, albeit very briefly as these vehicles pass by the MMP. This rapid passing of these on-coming vehicles is also associated with increased turbulence and mixing of air which may dilute UFP concentrations around the MMP leading to a lowering of UFP concentrations.

The cumulative impacts (Table 11) suggest that the mean effect of all types of on-coming vehicles is associated with lower UFP. However, while the upper 95% confidence interval for each type of vehicle includes positive values but at concentrations that are lower than those associated with comparable on-going vehicles. For example, the effect of adding the mean number of *on-coming light-duty vehicles* was to decrease the predicted UFP by 1 thousand/cm$^3$ with a 95% confidence interval of -34 and 32 thousand/cm$^3$. The predicted effect of adding the mean value of *on-coming heavy and medium vehicles* was to decrease UPF by over 19 thousand/cm$^3$ with a confidence interval of -131 and 93 thousand/cm$^3$. The predicted effect for the mean number of *on-coming buses* is a -22 thousand/cm$^3$ with 95% confidence intervals of -116 and 119 thousand/cm$^3$. Notice that size of these effects increases progressively with the body size of vehicles (light-duty vehicle, mid to large truck, bus) which may be associated with increasingly larger induced turbulence effects.

***Dynamics at Intersections***. The emission and dispersion dynamics at intersections are complex, as vehicles approach, enter and leave the intersection from four directions. From each direction, some vehicles will queue and idle at a red stop light before accelerating from that stationary position through the intersection as part of a platoon of vehicles. We find that measured UFP concentrations are highest while the MMP is in intersections as compared to when it is traveling along streets bounded by buildings. When entering an *intersection*, our model predictions UFP will increase by mean amount of 320 thousand/cm$^3$ with 95% confidence intervals of 84 and 557.

Our model enables us to explore several questions related to the UFP concentrations measured by the MMP while it is both i) stopped at an intersection and ii) as it traverses the intersection. For context, consider the MMP as it approaches an intersection from an on-going direction as in Figure 2. Our model enables us to consider a setting in which the MMP first comes to a stop in a queue at a red light. Traffic is waiting to cross into the intersection from the left, which will pass closest to the queuing MMP. Traffic is also waiting to cross into the intersection from the right in lanes that are relatively further away. Recall that the prevailing wind direction is from the southwest. Thus, when vehicles crossing from the right accelerate from a stopped position, the wind will tend to move their fresh emissions toward the queuing MMP. However, when vehicles crossing from the left accelerate from a stopped position, these fresh emissions are generally released downwind of the MMP. In keeping with these predictions, acceleration events for traffic crossing from the right are

associated with UFP increase of 58 thousand/cm$^3$ when vehicles crossing from the left accelerate, the associated UFP increase is much smaller 27 thousand/cm$^3$.

These queuing vehicles also crossed in front of the queuing MMP. Light-duty vehicles crossing from the left were associated with an increase of 30 thousand/cm$^3$ while heavy duty vehicles were associated with an increase of 81 thousand/cm$^3$. Buses crossing from the left were associated with an UFP concentration increase of 92 thousand/cm$^3$. There is no agreement whether these increases in UFP are contributions from these specific vehicles or whether these types of vehicles created different turbulence patterns that spread local emissions that had accumulated previously.

What it is curious, is that some types of vehicles crossing from the right produce different effects. Light-duty vehicles and heavy duty vehicles crossing from the right were associated with lower (not higher) UFP concentrations in the queuing MMP. When crossing from the right, light-duty vehicles and heavy-duty vehicles were associated with -11 thousand/cm$^3$ and -31 thousand/cm$^3$ respectively. Right-crossing buses, as with left-crossing buses, were associated with increase in UFP concentration of 53 thousand/cm$^3$. Especially for variables associated with intersection dynamics, it is important to notice how large the confidence intervals are.

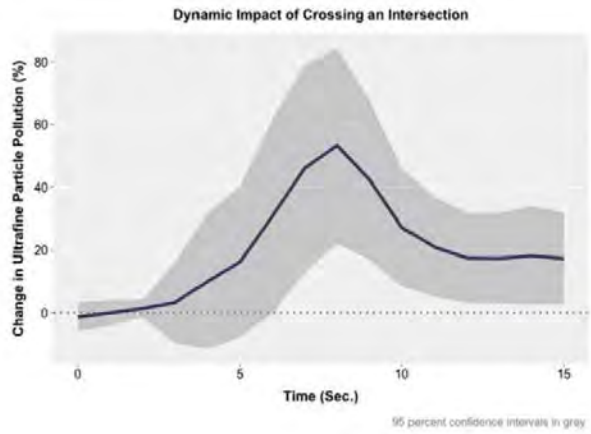
#### *v.4.3.4. Temporal profiles of traffic events*

In the previous section, we summarized the effects of various traffic impacts as simple averages, while in reality each event transpired over a period of time. Including higher temporal resolution within our model enables us to describe for each of these events how the UFP concentration varies over time. In the forgoing analysis, we examine the temporal profile of selected events over 15 second intervals by setting all the variables at their mean levels before re-setting the relevant event from zero to its mean level. Below we have chosen a few events to illustrate how valuable this type of analysis may be.

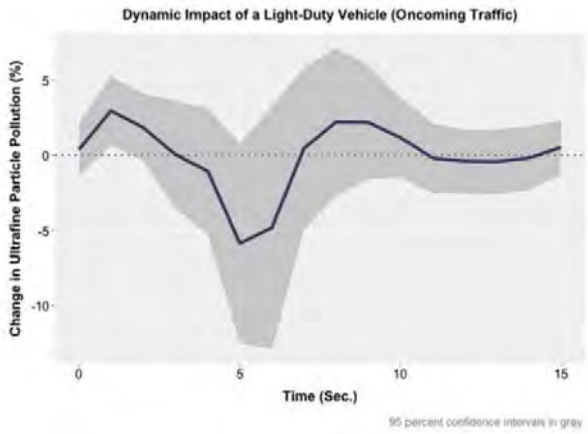
***Intersections.*** We begin with Figure 21, which describes the UFP impacts of the MMP crossing an intersection. The y-axis represents the percentage change from the UFP mean prior to the MMP entering an intersection. The solid line represents the means UFP level while the shaded area indicates the 95% confidence intervals. Using the estimated model to simulate the effects of MMP passing through the intersection, we observed that UFP concentrations peaks at over 50% of their pre-event mean between 5 and 10 seconds after entering the intersection. Even after 15 seconds, the simulations reveal the UFP concentrations are 20% above their pre-event, and statistically-significantly so. This suggests a longer time period is needed to fully characterize the time profile of intersection effects, which we will explore in the following section.

**Figure 21. Time Profiles of Selected Traffic Events.**

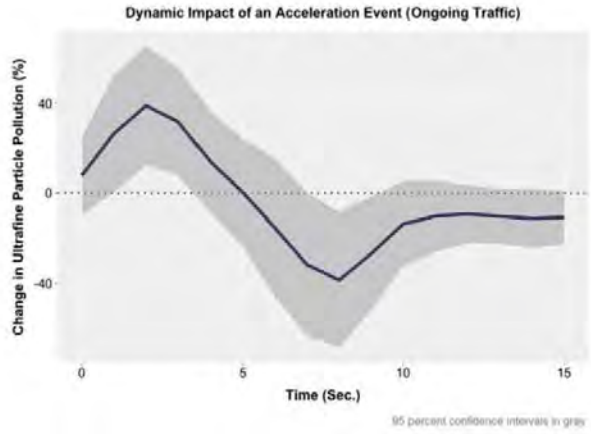
a.



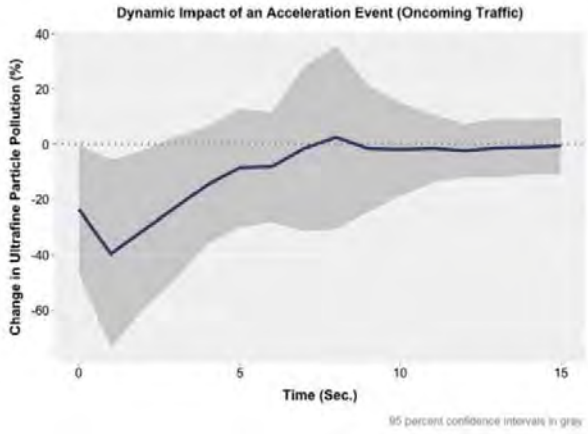

b.



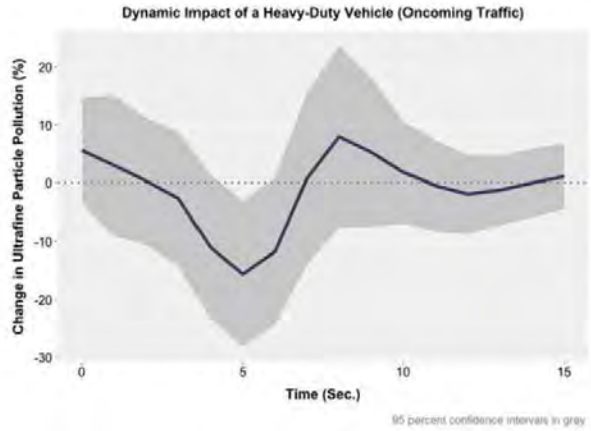

c.



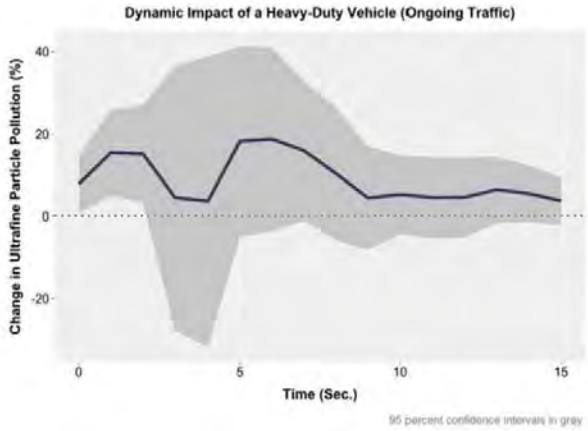

d.




e.




f.




***On-coming traffic: Heavy and Light Duty.*** In the previous section, recall that the cumulative impact of all types of on-coming vehicles was associated with a decrease UFP. However, we identified two countervailing effects of coming traffic: i) an increase in fresh emission and ii) an increase vehicle induced turbulence which might entrain cleaner air and reduce UFP concentrations. Below we compare the time profiles of heavy and light duty vehicles. Heavy duty vehicles are likely to travel more slowly and be larger that light duty vehicles, thus presenting different patterns of vehicle induced turbulence.

The cumulative impact on UFP concentration from an *on-coming heavy duty vehicle* was a -19 thousand/cm$^3$ with a 95% confidence interval of -131 and 93 thousand/cm$^3$ (Table 11). However, Figure 5e reveals significant systematic temporal variation in UFP concentrations for on-coming heavy duty vehicles. Within the first second of the on-coming heavy duty vehicles being visually identified the UFP concentrations decreased, reaching its lowest point at the 5th second before increasing to about 8% above pre-event concentrations. By the 10th second, concentrations levels have returned to their pre-event level.

The UFP concentration for an *on-coming light duty vehicle* was a -1 thousand/cm$^3$ with a 95% confidence interval of -34 and 32 thousand/cm$^3$ (Table 11). Figure 21 shows a systematic m-shaped temporal variation in UFP concentrations for on-coming light duty vehicles. Within the first second of the on-coming light duty vehicles being visually identified, a small (2-3%) but statistically significant increase in the UFP concentration was observed. At about the 4 second mark, on-coming light duty vehicles are then associated with a drop of over 5% in UFP concentration, and this persists until second 7. After this, concentrations rebound, and positive correlation up to 2-3% concentration level until they level off at second 7 to prior background levels. While small in magnitude, the aggregate effect of numerous on-coming light duty vehicles may prove to be significant.

***Acceleration events for On-going and Oncoming Traffic.*** Acceleration events, which includes all vehicle types, occurs when traffic that was stopped at a red light accelerates. As shown in Table 11, the cumulative impacts of on-going acceleration events were -59 thousand/cm$^3$. Figure 21 presents the dynamic impacts of an acceleration event for on-going traffic. For the first 5 seconds, UFP concentrations are strongly correlated with acceleration, with the peak mean level rising to 40% of pre-event levels before falling and are statistically different from zero at the 95% level. Between the 5th and 10th second, UFP concentrations decreased by 40%; again this difference is statistically different from zero at the 95% level. Focusing only on the simple mean of -59 thousand/cm$^3$ for on-going traffic masks this consistent and significant lateral s-pattern in UFP concentrations.

As shown in Table 11, the cumulative impacts of on-coming acceleration events were -159 thousand/cm$^3$. This impact is one directional; Figure 5d shows a time profile in which UFP concentrations drop immediately (within 1 second) to a low of 40% but with the 95% confidence interval including -70%. The concentration then increases steadily until approximately the 7th second where it stabilizes at pre-event levels, suggesting vehicle-induced entrainment of cleaner air dominates an increase in UFP from the vehicle plume.

#### *v.4.3.5. Simulating changes in traffic composition*

The estimated model from Table 10 can also be used to characterize larger scale travel-patterns and traffic composition scenarios and their associated UPF concentration transect profile. We begin with a baseline scenario in which the MMP travels 400 m through an intersection without stopping and starting again. We then add a stop-start scenario at the intersection to this same 400 m transect. This simulation enables us to characterize the predicted UFP concentration profile distinctly in terms of time (per second) versus distance (per meter) revealing important differences. Finally, we use the estimated model to consider how the stop-start scenario differs if there is a significant increase in the number of light duty vehicles.

***Our Baseline assumptions and UFP Predictions.*** The baseline simulation is over a 400 meter transect representative of the broader Broadway environment. It contains a 12-meter intersection from meter 103 to 115. This case represents a rational scenario in which a vehicle is not required to stop at an intersection. The MMP cruises for 225 m/30 s. at constant speed (7.5 m/s) across an intersection. No acceleration instances occur, no cross-traffic is encountered, and all neighboring buildings are 15 m high. Wind variables and location fixed effects (building height and differences)

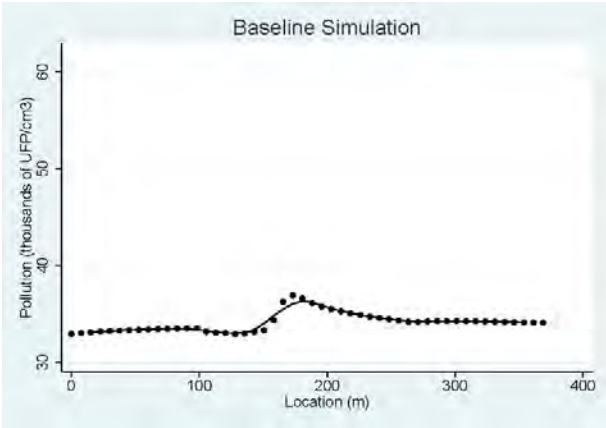
are set to long-run averages. On-going and on-coming traffic are also set to long-run averages. The resulting UFP concentration profile is presented in Figure 22a. The starting UFP concentration is approximately 32 thousand/cm$^3$. Shortly after passing through the intersection UFP concentrations increase slightly to approximately 37 thousand/cm$^3$ before returning to the starting baseline.

***"Stop-and-start" Simulation.*** The MMP travels for 10 seconds at 7.5 m/s, then decelerates for 10 seconds, before stopping at the intersection for 30 seconds. As soon as the MMP comes to a complete stop, cross-traffic accelerates into the intersection. On-coming, on-going, left- and right-cross-traffic (when occurring) are set to long-run averages. As soon as the MMP starts accelerating, on-going and on-coming traffic accelerate into the intersection. It then accelerates through the intersection for 10 seconds and cruises for 10 seconds returning to a velocity of 7.5 m/s.

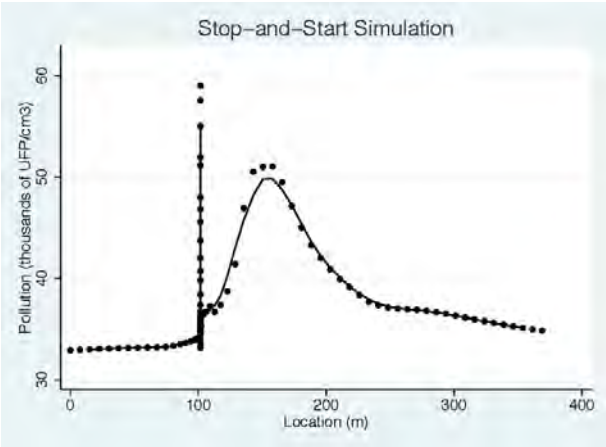
The UFP concentration profile associated with this simulation may be expressed in terms of location (or distance such meters) as shown in Figure 22b. Figure 22b shows a marked increase of almost 60 thousand/cm$^3$ in measured UFP concentrations at 103 m, when the MMP is stopped for 30 s in front of the red light (and cross traffic). Notice that in this stopped position, the measured UFP are nearly twice the baseline concentration of 32 thousand/cm$^3$. The simulation reveals a significant second spike of UFP concentrations for the next 85 m as the MMP accelerates with a platoon of other vehicles passing through the fresh emissions of the crossing traffic. This second peak of UFP rises in concentration to almost 50 thousand/cm$^3$. This simulation illustrates the spatial structure and origins of the more aggregate intersection plume of emissions shown earlier in Figure 21.

**Figure 22. Baseline Free-flow Simulation and Simulation of Stop-start events at an intersection.**

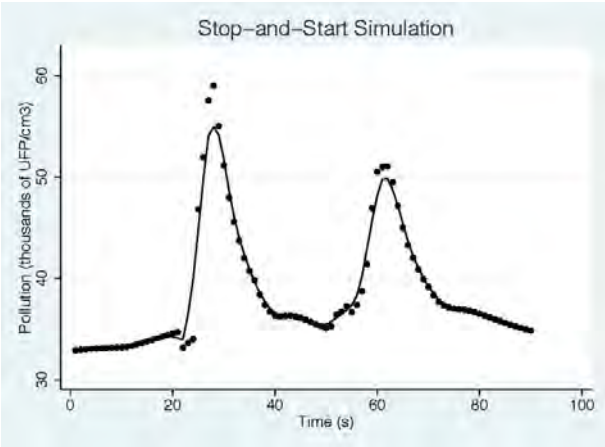
a.



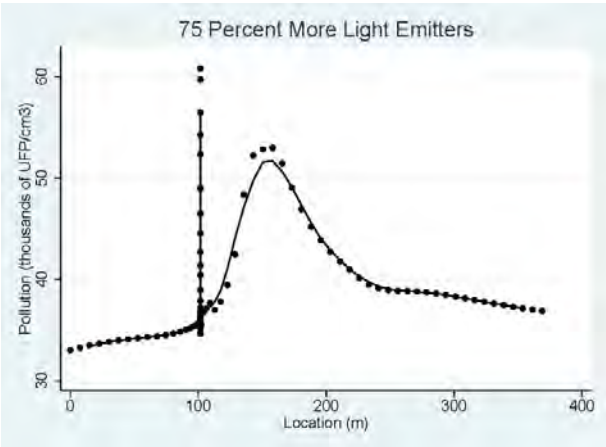

b.



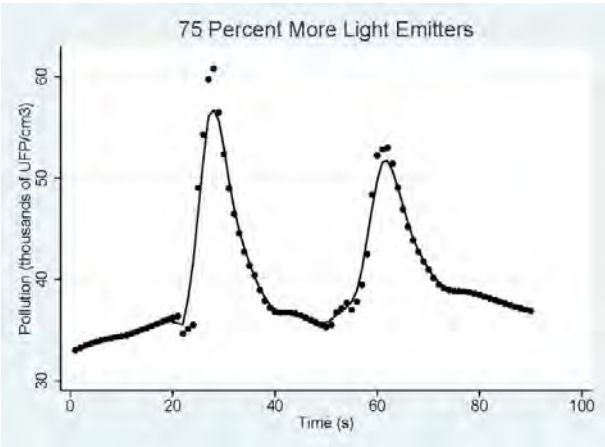

c.




d.




e.




The UFP concentration profile can also be characterized in terms of time (i.e., seconds) as shown in Figure 22c. This time-denominated profile also shows a bi-modal distribution of UFP but unlike the location-denominated profile, this distribution is smoother. What we learn from this presentation of the data is that UFP emission spikes sharply just after the 20 s mark as soon as the MMP has come to full stop. What is obscured by the distance-denominated profiles, but is revealed here is that UFP concentrations then steadily decline over the next 30 s the MMP is stationary at the stop light. As the MMP accelerated over the next 10 s and moved across the intersection UFP concentrations steadily increased. The second emission plume peaks just as the MMP is exiting the intersection (at the 60 s mark) and then steadily declines.

In principle, this model should also be able to simulate changes in UFP associated with changes in the composition of traffic around the MMP. In our last simulation, we evaluate the effects on an increase of 75% in light duty vehicles. This is shown in Figure 22d and Figure 22e, which represents both the distance- and time-denominated UFP profiles. The levels of concentrations can be directly compared to the Figures 6B and 6C which have the average number of the light duty vehicles. The increase in UFP appears small: The peaks in both profiles increase by about 3 thousand/cm$^3$ which is about a 10 percent increase. Given that number of light duty vehicles increase by 75% this seems like a small increase. We also explore increasing the number of heavy duty vehicle by a comparable amount. Because heavy duty vehicles were very infrequently encountered, and thus represented a very small base, even doubling them did not significantly alter the UFP profiles in our simulations.

### v.4.4. Cross-intersection profiles of UFP in various environments: implications for pedestrian exposure and transit stop siting

#### *v.4.4.1. Data analysis and cross-intersection concentration profiles*

Producing representative profiles from MMP data presents a few challenges associated with over/underweighting data and dealing with day-to-day variations in background concentrations. The acquisition of correct positioning information of the MMP is critical in obtaining highly resolved concentration profiles. Handheld GPS units have approximately 3 to 5 m accuracy with a wide area augmentation system (WAAS). Nonetheless, under specific conditions such as street canyons shadowing effects can result in poor reception of satellite signals (Birmili et al. 2013; Misra and Enge 2006; Ranasinghe et al. 2016). These wandering GPS position data were corrected with a line referencing technique developed by Ranasinghe et al. (2016). Briefly, a reference line consisting of evenly spaced points was assigned for each direction of the streets. Five-meter spatial resolution for the points on the reference line was chosen, based on the mean MMP speed driven for all sessions (3.0 m·s$^{-1}$ with 1σ = 2.9 and 3.4 m·s$^{-1}$ for the morning and afternoon sampling periods, respectively). Each GPS data point was assigned to the closest reference point. When multiple data points are assigned to one reference point (e.g., when the MMP stopped or was moving slowly), these data were averaged. This avoids the problem of overweighting a run with many data points compared to a run when the MMP passed by a reference point without stopping. The other overweighting problem occurs when some runs have no data assigned to a particular reference point, due to a fast-moving MMP. To avoid this problem, the concentration profile for each individual run was interpolated using Piecewise-Cubic-Hermite-Interpolation scheme (PCHIP) (Fritsch and Carlson 1980). PCHIP was successfully applied to interpolate data points while faithfully preserving the concentration profiles (Ranasinghe et al. 2016).

To obtain representative concentration profiles for intersections many concentration profiles were collected in different built- and traffic-environments. In addition, to avoid the distortions from daily concentration variations on averaging the profiles, the minimum value of each profile was subtracted from the profile before averaging. Then, to put the data back on a meaningful scale, the mean of the minimum values from all collected profiles was added to obtain mean concentration profiles. In the figures, the MMP driving direction is represented by a (-) sign when it approaches the intersection, zero at the center of intersections, and (+) signs after passing by the intersection. Thus, distance in the averaged profiles consistently represents the traffic direction in which the MMP was headed, and not the direction relative to the compass. The distance covers 90 m on either side of the intersection, which was chosen because the average block length is 180±64 m (Table 12) and earlier work has suggested this range captures most of the variability around intersections (Goel and Kumar 2015). Sampling sites are shown in Figure 2, Figure 3, and Figure 4 in Section 2.1.

**Table 12. Sampling dates and information of the intersections investigated in this study.**

| | Bev | BW&7th | Olv&12th | Ver&7th | Wil&Car | TC&LT |
|---|---|---|---|---|---|---|
| Street | (1) varying<br>(2) Wilshire Blvd. | (1) Broadway St.<br>(2) 7th St. | (1) Olive St.<br>(2) 12th St. | (1) Vermont Ave.<br>(2) 7th St. | (1) Carondelet St.<br>(2) Wilshire Blvd. | (1) Temple City Blvd.<br>(2) Las Tunas Blvd. |
| Street width | (1) varying<br>(2) 30-38 m | (1) 26 m<br>(2) 22 m | (1) 28 m<br>(2) 17 m | (1) 30 m<br>(2) 25 m | (1) 17 m<br>(2) 37 m | (1) 24 m<br>(2) 30 m |
| Traffic flow rates (A.M.) | (1) varying<br>(2) 24 | (1) 12<br>(2) 15 | (1) 21<br>(2) 4 | (1) 39<br>(2) 10 | (1) 2<br>(2) 31 | (1) 25<br>(2) 28 |
| Traffic flow rates (P.M.) | (1) varying<br>(2) 47 | (1) 20<br>(2) 20 | (1) 8<br>(2) 3 | (1) 38<br>(2) 12 | (1) 2<br>(2) 27 | (1) 26<br>(2) 29 |
| Traffic density (qualified) | Long queue length in one direction (WB in A.M. and EB in P.M.) | Medium queue lengths but slow vehicle speeds | Minimal queue lengths | Long Queue lengths covered the whole block particularly in NB | Low queue lengths | Long queue lengths but rapidly dissipated due to effective traffic flow |
| Distance between traffic lights | (1) varying[a]<br>(2) 330 m[b] | (1) 200 m<br>(2) 125 m | (1) 180 m<br>(2) 125 m | (1) 224 m<br>(2) 174 m[c] | (1) 190 m<br>(2) 100 m | (1) 200 m<br>(2) 135 m |
| Number of profiles obtained for each run | 2 (east-, west-bound) × 5 intersections =10 | 4 (north-, south-, east-west-bound) | 3 (north-, south-, east-bound) | 4 (north-, south-, east-west-bound) | 4 (north-, south-, east-west-bound) | 4 (north-, south-, east-west-bound) |
| Sampling dates | 6/17/2014<br>6/19/2014<br>6/24/2014<br>6/36/2014 | 7/1/2013<br>7/2/2013<br>7/3/2013<br>7/5/2013 | 9/24/2013<br>9/24/2013 | 10/7/2013<br>10/14/2013<br>11/18/2013 | 11/1/2013<br>11/6/2013<br>11/20/2013 | 8/6/2013<br>9/17/2013<br>9/18/2013 |

### *v.4.4.2. Highly resolved UFP profiles around intersections*

In this section, we show [UFP] profiles around signalized intersections obtained by averaging individual profiles that were collected for varying traffic, spatial, and temporal spans. The averaging spans expanded from each traffic direction for each site into the entire traffic directions for each site and into the entire sites for the morning and afternoon sampling events.

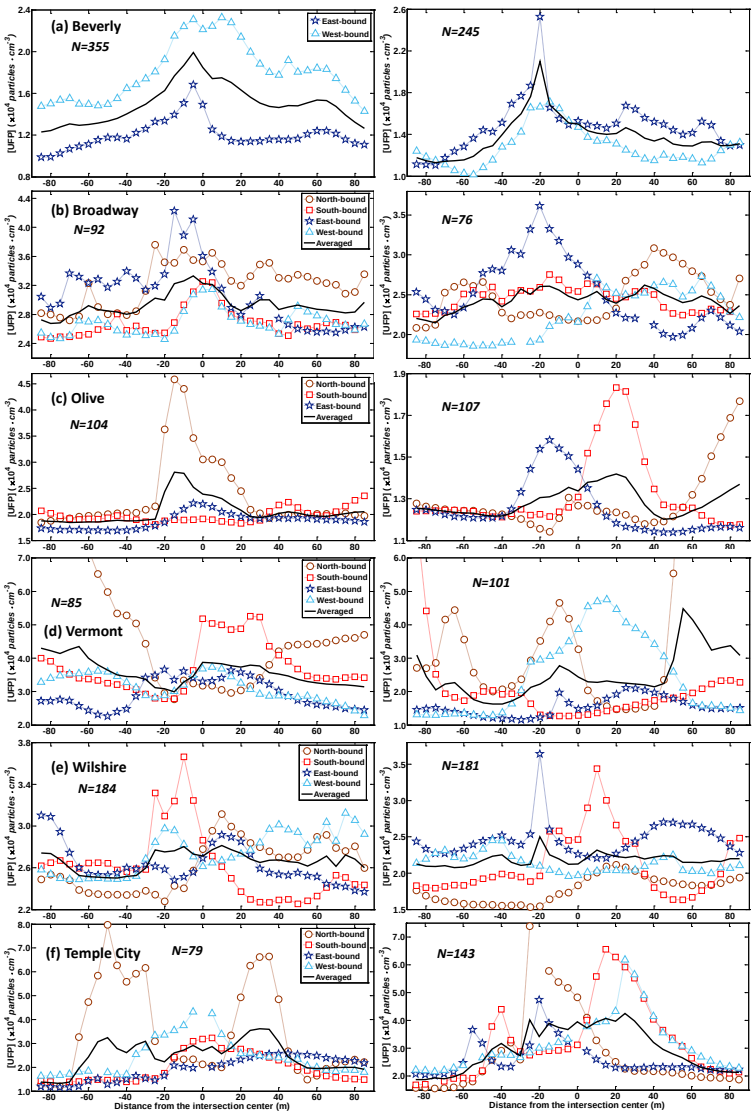



**Figure 23. The mean UFP profiles across the intersection for each direction of the site (broken lines with symbols) and the mean profile of all directions for each site (black solid lines). N denotes the number of the total profiles obtained for the site. Left and right panels show the results for AM and PM sampling events, respectively. The % values in parentheses represent UFP elevation at the peak relative to the base value.**

Figure 23 and Table 13 show the statistical results of averaging [UFP] profiles of each direction for each site. The first striking feature of the mean [UFP] profiles of each traffic direction for each site is that highly elevated [UFP] occurs commonly near the intersections but at varying locations (mostly < 30 m from the intersection center) within a relatively short distance range (roughly less than 60 m of peak width) (Figure 23). The only exception was Vermont & 7$^{th}$ site, at which the profiles showed more complicated shapes. Vermont Ave. is one of the busiest arterial streets in Los Angeles, and for both morning and afternoon sampling events, the road experienced traffic congestion along the entire sampling block (particularly for the north-bound). Thus, vehicles repeatedly stopped and accelerated throughout, likely causing higher [UFP] all along the roadway. In this respect, we confined the distance range to identify the intersection peak within ±50 m from the intersection center.

**Table 13. Basic statistics of the intersection UFP profiles of each direction for each site.**

| Site | Direction | Morning | | | Afternoon | | |
|---|---|---|---|---|---|---|---|
| | | Peak position (m) | Base position (m) | Conc. Diff. between peak and base (%)[a] | Peak position (m) | Base position (m) | Conc. Diff. between peak and base (%)[a] |
| Beverly | EB | -5 | -85 | 70.4 | -20 | -75 | 128.4 |
| | WB | 10 | -55 | 62.9 | -15 | -55 | 69.0 |
| Broadway | EB | -15 | 60 | 65.3 | -20 | 50 | 84.0 |
| | WB | 5 | -75 | 27.4 | 10 | -55 | 45.8 |
| | NB | -15 | -70 | 38.3 | 40 | -85 | 48.1 |
| | SB | 0 | -80 | 31.8 | -15 | 55 | 22.8 |
| Olive | EB | -5 | -45 | 31.0 | -15 | 50 | 38.8 |
| | NB | -15 | 35 | 137.9 | -5 | -15 | 14.3 |
| | SB | 0 | -25 | 5.8 | 20 | 75 | 55.8 |
| Vermont | EB | -20 | -50 | 62.0 | 25 | -30 | 83.2 |
| | WB | 5 | 85 | 63.0 | 15 | -50 | 270.0 |
| | NB | -50 | -15 | 217.5 | -10 | 30 | 215.8 |
| | SB | 25 | -20 | 88.4 | -30 | -5 | 60.6 |
| Wilshire | EB | 10 | 85 | 23.0 | -20 | 15 | 65.2 |
| | WB | -20 | -45 | 19.4 | -45 | -5 | 25.3 |
| | NB | 10 | -20 | 36.8 | 20 | -25 | 38.0 |
| | SB | -10 | 50 | 62.4 | 10 | 50 | 110.2 |
| eTemple City | EB | 40 | -70 | 122.7 | -20 | -75 | 90.3 |
| | WB | -5 | -45 | 154.7 | 25 | -75 | 127.0 |
| | NB | 30 | -75 | 447.3 | -25 | -65 | 226.6 |
| | SB | 5 | -40 | 132.6 | 15 | -85 | 181.6 |
| Mean[b] (std) | | 15 (15) | 54 (23) | 86 (93) | 20 (10)[b] | 46 (26) | 95 (72) |

The mean relative elevations of the peak [UFP] near the intersection was 86% (1σ = 93%) and 95% (1σ = 72%) compared to the profile minima for the morning and afternoon session, respectively (Table 13), although they ranged widely (6 ~ 447% for the morning and 23 ~ 270% for the afternoon session). The mean position of the concentration peaks is 15 (±15) m and 20 (±10) m from the center of the intersection for the morning and afternoon sampling events, respectively. The mean location of the minimum concentrations occurred 54 (±23) m and 46 (±26) m from the center of the intersection for the morning and afternoon sampling events, respectively. It should be noted that the peak distance is the absolute value, although the peak and minimum concentrations occurred in both far and near sides of the intersection. Peaks appeared equally before or after the intersection; 57% were on the near side on the morning, and 50% in the afternoon. The minimum position, however, appeared more on the near side of the intersections; 76 and 66% appearing on the near side in the morning and afternoon, respectively. This implies that there would be a reduction in exposure, were bus stops sited before the intersection. However, we also acknowledge that there are several other factors that favour the far side for placing traffic stops, including better traffic flow and shorter stop times for transit vehicles, both of which tend to lower air pollutant emissions and/or exposure of transit users.

The varying location and magnitude of [UFP] peak resulted from complex interactions between the built environment, traffic (as well as vehicle composition), drivers' behavior, winds, and other meteorological conditions. This complicated feature of intersection peak locations and magnitudes should be noted when stationary measurements for pedestrian exposure to vehicular emissions are conducted near the intersection.

The black solid lines in Figure 23 represent the results of averaging all of the directions for each site together. Averaging more profiles together (beyond the first 10 or 20, which have a lot of variance)

results in progressively smaller differences between the peak and minimum concentrations, due to variations in the location of the peak concentration. As a result, while averaging more data together results in more generalized profiles, it underestimates potential exposures around the intersection. For example, the elevation of [UFP] (concentration difference between the peak and minimum values relative to the minimum, Δ[UFP]) is significantly increased when Δ[UFP] was obtained from averaging all Δ[UFP] for individual directions, compared to one obtained from the profile of averaging all of the directions for the entire sites. The mean increase in [UFP] near the intersection was 50 and 66% when Δ[UFP] was calculated from the latter case in the morning and afternoon sampling events, respectively, while these values increased to 86 and 95% when we averaged Δ [UFP] obtained from the profiles of each traffic directions (the former case). Nonetheless, the common feature of the mean concentration profiles for each sampling site is similar to those for individual traffic directions that [UFP] were higher near the intersections and had larger variations than locations about 40 m or more from the intersections (Figure 23 and Table 14).

**Table 14. Basic statistics of intersection [UFP] profiles for each site. The values in parentheses are averages from all profiles of each traffic direction for each site.**

| | | Bev | BW&7th | Oliv&12th | Ver&7th | Wil&Car | TP&LT | AVG |
|---|---|---|---|---|---|---|---|---|
| A.M. | Peak conc. | 19,910 | 33,311 | 28,062 | 38,792 | 28,145 | 36,115 | 30,723 |
| | Min. conc. | 12,292 | 26,760 | 18,455 | 29,923 | 25,017 | 13,218 | 20,944 |
| | Mean. conc. | 14,815 | 29,316 | 20,605 | 35,869 | 26,626 | 24,777 | 25,366 |
| | Conc. Difference between peak and min. conc. | 7,618 (7,967) | 6,551 (10,474) | 9,607 (12,711) | 8,869 (21,306) | 3,128 (9,030) | 22,897 (31,780) | 9,779 (16,401) |
| | Conc. difference relative to min. conc. | 62% (67%) | 24% (41%) | 52% (58%) | 45% (82%) | 13% (35%) | 173% (214%) | 50% (86%) |
| | Peak conc. location | -10 m[a] | -10 m | -15 m | 0 m | 10 / -10 m | 30 m[b] | |
| | Baseline location | -85 m | -80 m | -70 m | -15 m | -45 m | -75 m | |
| P.M. | Peak conc. | 20,989 | 26,224 | 14,193 | 29,430 | 24,990 | 52,469 | 28,049 |
| | Min. conc. | 11,280 | 21,455 | 12,013 | 16,278 | 20,776 | 28,714 | 18,419 |
| | Mean conc. | 13,815 | 24,256 | 12,865 | 25,010 | 21,848 | 39,646 | 22,907 |
| | Conc. Difference between peak and min. conc. | 9,709 (10,602) | 4,769 (10,098) | 2,180 (5,759) | 11,493 (23,164) | 4,214 (10,793) | 23,756 (43,582) | 9,353 (18,103) |
| | Conc. difference relative to min. conc. | 86% (99%) | 22% (50%) | 18% (36%) | 71% (158%) | 20% (60%) | 83% (155%) | 66% (95%) |
| | Peak conc. location | -20 m | 35 /-20 m | 20 m | -10 m | -20 m | 25 / -25 m | |
| | Baseline location | -75 m | -75 m | -45 m | -40 m | -65 m | -70 m | |

a. Positive length denotes the distance after the intersection center
b. Negative length means the distance before the intersection center

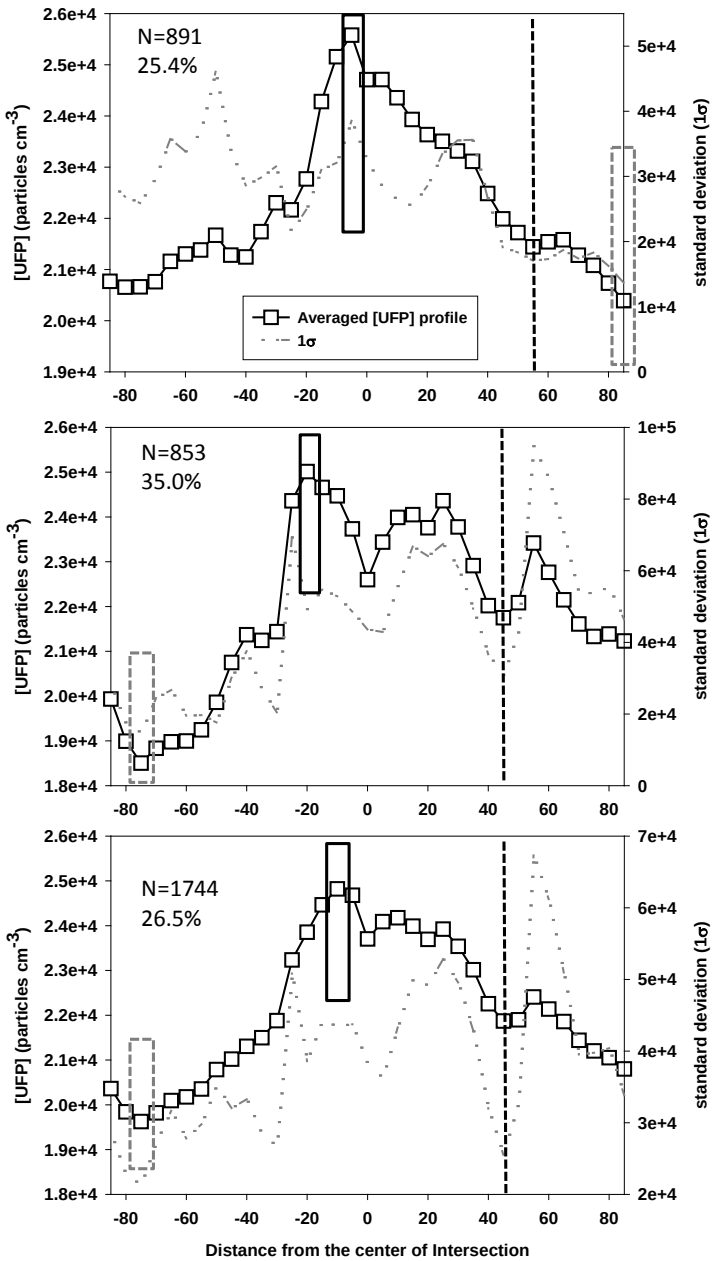



**Figure 24. Same as Figure 23 but for (a) the entire A.M., (b) the P.M., and (c) the entire periods. Dark gray rectangles denote the location of the peak concentration and gray dashed rectangle indicate the base location. See text for the vertical gray dotted lines.**

While more optimized values could potentially be developed for more specific situations, we produced several averages to produce simplified, general results. In this respect, the averaged cross-intersection profiles for the mornings (891 profiles) and afternoons (853 profiles) of the all sites were calculated (Figure 24a, b). Similar to the mean profiles for each site and each traffic direction, the peak concentrations occurred within 30 m before and after the center of intersection and sharply decreased by 45~55 m from the intersection. The peak concentrations near the intersections were 25% and 35% higher than the baseline values for the mornings and afternoons, respectively. Given that the magnitude of morning and afternoon averaged profiles were similar, all transect data (1744 individual profiles) were also averaged to produce a single mean intersection profile. Considering the large number of conditions represented, the mean profile is expected to show the general pattern of cross-intersection pollutant concentration variations regardless of time and location at least for the Los Angeles area. The peak concentration occurred 10 m before the intersection, and was 27% higher than the baseline value at 75 m before the intersection. Shoulder peaks occurred at 10 m and

25 m after the center of intersection and had higher standard deviations and sharply decreased with distance from the intersection up to 45 m, although [UFP] kept decreasing more slowly with farther distance.

***Variability in the profiles.*** The common feature of the mean cross-intersection [UFP] profiles was that larger variability was observed at and around the intersection peak locations than at distances farther away from the intersection (grey dashed lines in Figure 24). Thus, although the mean peak concentration was about 27% higher than that at 75 m away from the intersection center, the concentrations at some moments much higher than the mean value; the 1σ standard deviation at the peak is about double the mean concentration.

This section discusses the concentration distributions at two extreme cases: at the intersection peak and the baseline where the lowest concentration occurred in the mean profile. For the overall [UFP] average profile (Figure 24c), the peak concentration was recorded at 10 m before the center of intersection (indicated with a black rectangle) and the baseline value was set about 75 m before the intersection (indicated with a grey dotted rectangle), each of which includes 1,744 data points (N=1,744). Using the collected data for two locations, cumulative distribution plots were obtained (Figure 25). The linear fit obtained with observation data between lower 25% and 75% of the dataset was considered as a baseline of cumulative distribution (red and black dotted lines) because in this range data distribute quasi-linearly ($R^2$ > 0.99 for both the peak and base locations). In this way, the transient [UFP] spikes above the baseline that likely originated from excessive emissions events can be quantified. In addition, the baseline fit provides the information with respect to [UFP] variations in lower concentration part. The baseline for the peak location (a red dotted line in Figure 25) was slightly higher (slope = 2.80 ± 0.01×$10^4$) than that for the base location (slope = 2.51 ± 0.01×$10^4$). The difference in the interceptions between two baselines was negligible (4,603 vs. 4,233 for the peak and base locations, respectively). Overall, the baseline difference between two cases was only 10% relative to the base case (calculated by integrating the baseline fits from lower 25% through 75% of data).

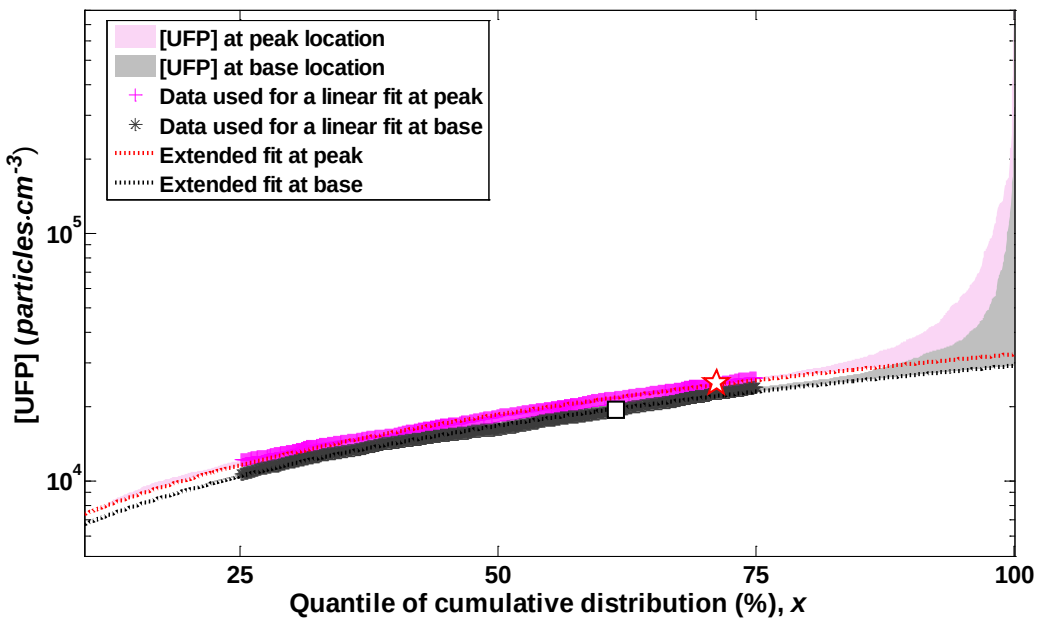



**Figure 25. Cumulative [UFP] distribution plot at the peak (pink area) and base locations (gray area) indicated as black and gray dashed rectangles in Fig. (2c). Data are from the mean [UFP] profile for the entire sampling periods (N=1744). Red and black dotted lines show a linear fit for data between lower 20 to 70% (pink crosses and gray asterisks) for the peak and base location, respectively. Red Star and the white denote the mean values at the peak and base location, respectively.**

The major differences between two cases occurred in the upper 15% of the dataset. The observations sharply shot over the fitted baseline around the upper 15% of data for both the peak and base locations, however, larger deviations were shown for the peak location than the base case. These large deviations from the fitted baseline were caused most likely by elevated emissions from high emitting vehicles or simultaneous accelerating of several more moderately polluting vehicles in a queue, because differences in traffic flow rates and fleet compositions between the two locations are negligible due to their close proximity (65 m from one another).

An accelerating effect in the upper quintile region at the peak location (Excessive Emission Impact, EEI) is defined as the ratio of the integration of difference between observed concentrations and fitted baseline at the peak to that at the base location:

$$EEI = \frac{\int [UFP]_{diff,\ peak} dr}{\int [UFP]_{diff,\ base} dr}$$

where $[UFP]_{diff}=[UFP] - [UFP]_{fit(20\% - 70\%)}$ (shaded areas between fitted line and UFP distribution) and subscripts peak and base represent the peak and base location, respectively. The *EEI* value for the UFP profile averaged for the entire profiles was 2.2. Considering the magnitude of [UFP] at the peak is dampened in the averaged profile due to varying locations of intersection peaks, acute effects due to high emission events can also be underestimated in the above analysis. Indeed, the mean *EEI* from the profiles of individual directions significantly increased: 9.6 (±8.0) for morning and 16.3 (±16.5) for afternoon. This implies that under some conditions (e.g., when traffic signal changes), pedestrian can possibly be exposed to more intensive and frequent excessive vehicular emissions near the intersections than mid-blocks.

***Traffic effects on the magnitude of intersection UFP elevation.*** We attempt to relate traffic patterns to the magnitude of UFP elevations at the peak locations, which varied widely by sites as well as by traffic directions at the same site. Variables for traffic patterns include (1) traffic flow rates per traffic-signal cycle (vehicles·min$^{-1}$), (2) vehicle number waiting for green light at the moment the signal changed from red to green (queue length; Q-length), and (3) the queue length considering the proportion of high emitting vehicles (HEV; defined as heavy and medium duty diesel vehicles in this study). The third variable (HEV-Q-length) was defined as:

$$\text{HEV-Q-length} = (\mathbf{Q-length}) \times \frac{\mathbf{TFR}_{tot} - \mathbf{TFR}_{HEV}}{\mathbf{TFR}_{tot}} + \mathbf{20} \times (\mathbf{Q-length}) \times \mathbf{TFR}_{HEV}$$

where $TFR_{tot}$ and $TFR_{HEV}$ represent traffic flow rates of total vehicles and HEV, respectively. The weighting value of 20 for HEV was introduced based on re-construction of the literature review (Choi et al. 2013a; Kumar et al. 2011). HEV weighting on traffic flow rate did not change the results (not shown), thus this is not included in the discussion.

All three variables showed positive correlations with both relative and absolute UFP elevations at the intersection (relative UFP elevation is defined as the ratio of the concentration difference between the peak and minimum locations to the minimum concentration, and absolute elevation is simply the concentration difference between the peak and minimum locations) (Figure 26). However, *TFR* correlated (in an exponential form) less significantly with UFP elevations than the other traffic variables, and these correlations disappeared for the afternoon sampling events (Figure 26a, b). Whereas, Q-length was linearly correlated with UFP elevations better than *TFR*; the relationships were kept in the afternoon sampling events as well (Figure 26cd). The best correlations were obtained between HEV weighted Q-length and UFP elevations for the morning periods, although afternoon relationships were comparable to those of Q-length (Figure 26e, f). A wide scatter of these 1:1 plots was likely produced by a large variability of emissions from

individual vehicles due to different driving behaviour and vehicle maintenance as well as vehicle composition. A slight improvement in correlations by weighting HEV effects on Q-length is likely due to a small portion of HEV in a vehicle composition at the study sites (2.6±1.0% and 2.5±1.9% of the total traffic for the morning and afternoon periods, respectively).

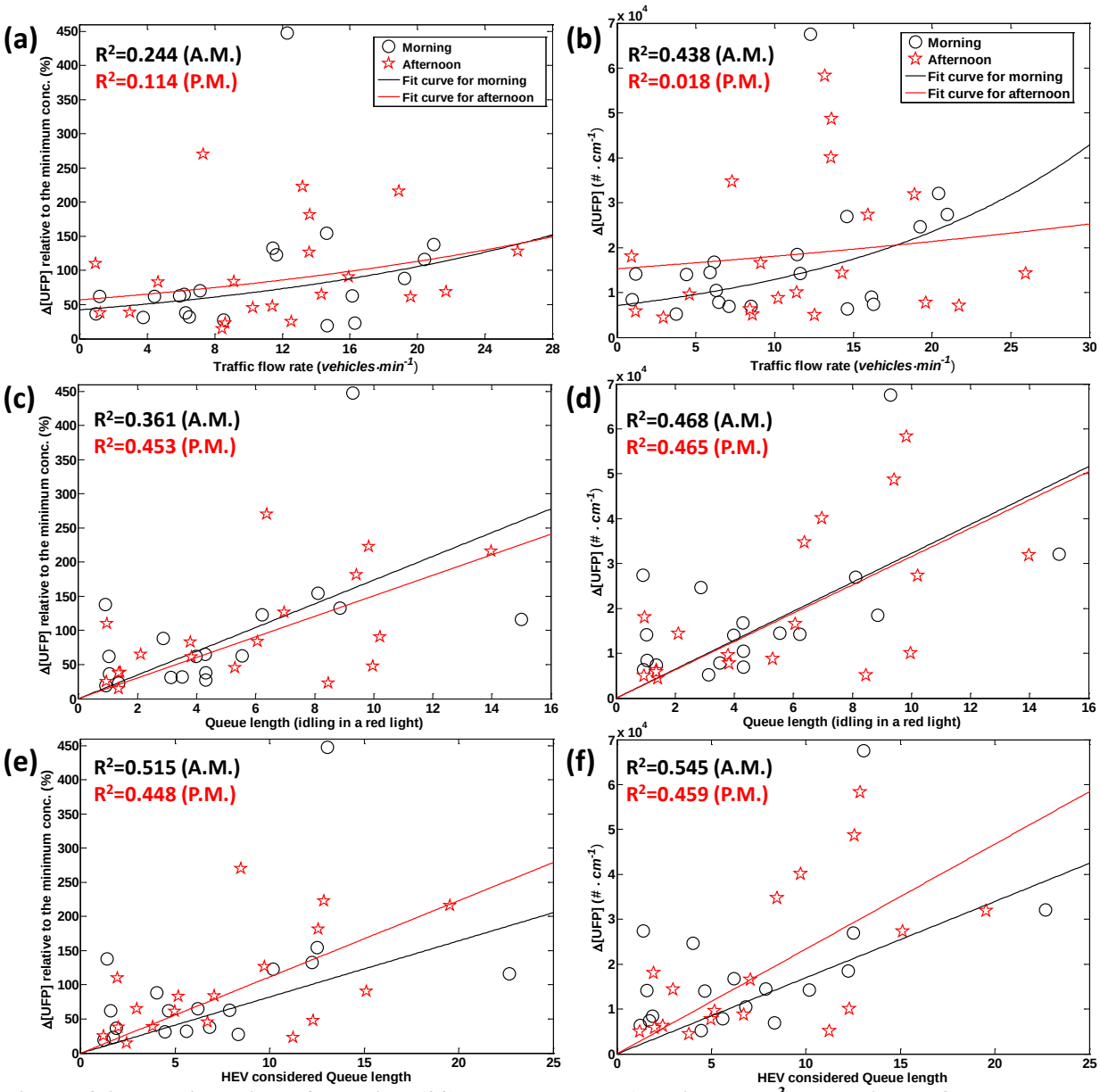



**Figure 26. Relationships of relative (%) and absolute (particles·cm$^{-3}$) elevations of UFP at the peak location with traffic parameters (traffic flow rate, queue length, and HEV considered queue length). The left panel (a, c, e) is for relative [UFP] elevation and the right panel for absolute concentration increase at the peak locations. Queue length is defined as the number of vehicles waiting for the green signal at the moment when traffic signal changes from red to green.**

Consequently, we believe that HEV weighted Q-length is likely a more important factor controlling UFP elevations at the intersections, which is reasonable considering that heavy-duty diesel trucks emit much more UFP than gasoline-powered vehicles under certain conditions and a bigger group of vehicles accelerating simultaneously would emit more UFP.

### *v.4.4.3. Implications to pedestrian exposure and transit-oriented development*

Here, for the case of bus stop siting taken alone, we estimate the exposure levels of transit users to elevated [UFP] around the intersections using a simple time-duration model of pedestrians moving through and/or waiting in the zones of low and high UFP concentrations for two cases: when bus

stops are located 40 m vs. 20 m after the center of intersection. The 'exposure level' is represented by the sum of [UFP] exposure for the period of activities ($\sum[UFP]$). The 40 m distance was chosen based on the mean distance of the local minimum in the concentration profile and 20 m is based on the mean distance of the concentration peaks. A mean pedestrian walk speed of 1.4 $m \cdot s^{-1}$ (Knoblauch et al. 1996; Parise et al. 2004) was assumed, and the UFP concentrations were set constant in both the affected zone (high UFP concentration was assumed within 20 m before and after the intersection) and beyond the affected zone (low UFP). The transit users' behaviour was assumed that (1) people spend 10 s for disembarkment, (2) walk to the intersection at constant speed, (3) spend 45 s (half of a traffic light cycle) to traverse a single crosswalk at the intersection, (4) walk to the bus stop for transfer, and (5) wait for a bus for 10 minutes at the bus stop.

The results suggest that pedestrian experiences more exposure in the case of 20 m bus-stop location compared to the 40 m case unless intersection [UFP] is not elevated. The extent of increased risk is also proportional to the extent of UFP elevation at the intersection. We note that absolute differences in concentrations are of less value than relative differences because the absolute UFP concentrations vary widely depending on location as well as in time, since vehicle fleets, maintenance, and fuels are continuously evolving, both within and between different countries and regions. Additionally, other directly emitted air pollutants such as $NO_x$ or VOCs may be of interest. However, using the current data set as an example, in case the intersection [UFP] = $3.0 \times 10^4$ particles $cm^{-3}$ and local minimum [UFP]= $2.0 \times 10^4$ particles $cm^{-3}$ (50% lower at 40 m), the total exposure levels were $1.4 \times 10^7$ particles $cm^{-3}$ vs. $2.0 \times 10^7$ particles $cm^{-3}$ at the 40 m and 20 m bus stop location, respectively. This corresponds to 38% reduction in UFP exposure when the bus stop is located at 40 m from the intersection. This result indicates that locating bus stops around 40 m and farther away from the center of intersections reduces transit user's exposure to intersection emission events.

# vi. Dispersion Modeling

## vi.1. Introduction

Regional planning efforts indicate that TODs are expected to reduce regional air pollution (~10 km). However, there is concern that high building density can reduce dispersion of pollutants relative to that in terrain without buildings. Thus, TOD residents living or working in close proximity to urban streets may be exposed to elevated concentrations of vehicle emitted pollutants. This report describes the results of a research study designed to estimate the effect of building morphology on dispersion and hence air quality within a TOD.

Results from field studies (Hanna et al., 2014), laboratory experiments (Barlow and Belcher, 2002), and numerical simulations (Hang et al., 2012; Ketzel et al., 2000) have provided valuable insight into the mechanisms that govern dispersion of pollutants in the urban canopy. This information is the basis for semi-empirical dispersion models such as the Canyon Plume Box Model (CPBM) (Yamartino and Wiegand, 1986), and the Operational Street Pollution Model (OSPM) (Berkowicz et al., 1997). These models apply primarily to street canyons between relatively uniform buildings, which are common in Europe where these models originate. They are not applicable to the inhomogeneous building structures that characterize urban area cores in the United States, because the inhomogeneous environments produce complex flow structures inconsistent with the street canyon model formulation (Karra et al., 2011; Nelson et al., 2007).

Inhomogeneous built environments with tall buildings can induce flows that are significantly different from the idealized flows assumed in street canyon models. A wind tunnel study of an urban neighborhood with a single tall tower found enhanced vertical dispersion in the wake of the tower (Brixey et al., 2009). Another wind tunnel model of Manhattan found strong transport of contaminants up the lee sides of several of the tallest buildings (Heist et al., 2004). Results of the Joint Urban 2003 field study showed that the flow within an inhomogeneous street canyon was complex, with different flow structures resulting from slightly differing wind directions (Nelson et al., 2007). Large downdrafts and updrafts that could transport pollutants vertically were also observed.

Because of the complexity of the flows in such areas, the objective of the study reported here is limited to capturing the essential features of dispersion in the presence of buildings through a semi-empirical dispersion model. The model was developed through analysis of data from 1) a five year measurement program conducted in a street canyon in Hannover, Germany 2) field studies conducted in Los Angeles. The results of the analysis have been summarized in the form of a semi-empirical dispersion model that relates street-level pollutant concentrations to local emissions, building morphology, and micrometeorological variables.

The semi-empirical model used in this study can be contrasted with comprehensive models such as computational fluid dynamics (CFD) models. CFD models are designed to be general purpose because they solve the governing equations directly and can be adapted to a broad range of problems. However, they are numerically cumbersome and their results are difficult to interpret. Most importantly, they have not undergone adequate evaluation with real-world data (Di Sabatino et al., 2007). On the other hand, the relatively simple structure of the semi-empirical model allows calibration with field data to develop a practical tool that provides concentration estimates consistent with observed values.
The semi-empirical model described here seeks to explain first order effects of buildings on dispersion using only a small number of essential built environment and meteorological variables. It is designed to allow city planners and policymakers to support decisions related to the impact of TOD design on exposure to traffic emissions.

Here, we provide an overview of our knowledge of dispersion in urban environments, and describe the analysis of near road concentration data collected at an urban site in Hannover, Germany using several semi-empirical dispersion models. The analysis provides insight into the meteorological variables that govern dispersion in an urban built environment. This provides motivation for our study of dispersion in environments with non-uniform building height and spatial inhomogeneity, and also motivates the need for the field measurements that were conducted in Los Angeles. This is followed by a development and evaluation of the semi-empirical dispersion model with data collected in the field measurements conducted in Los Angeles.

Finally we provide the conclusions and the implications for the design of TODs. In this section, we present a method to estimate the impact of a planned TOD on street level concentrations of vehicle related emissions. This method can be readily used in combination with commonly used regulatory models, such as AERMOD (Cimorelli et al., 2005), to estimate the magnitude of concentration hotspots that can be created through the interaction between emissions and flow effects induced by urban buildings.

## vi.2. Background and related work

The impact of the built environment on dispersion of emissions in urban areas has been studied over the past 40 years using results from field, tracer, wind tunnels and numerical simulations. Models to estimate the magnitude of this impact have typically characterized urban morphology in terms of the "street canyon" shown in Figure 27. The street canyon is a long street with uniform buildings on both sides, forming a canyon. When the rooftop wind blows close to perpendicular to the canyon, one or multiple vortices form within the canyon. A single vortex results in the wind direction at street level pointing in the opposite direction to that at the rooftop, from the windward side to the leeward side. When the rooftop wind is at an angle, a helical vortex forms within the canyon.

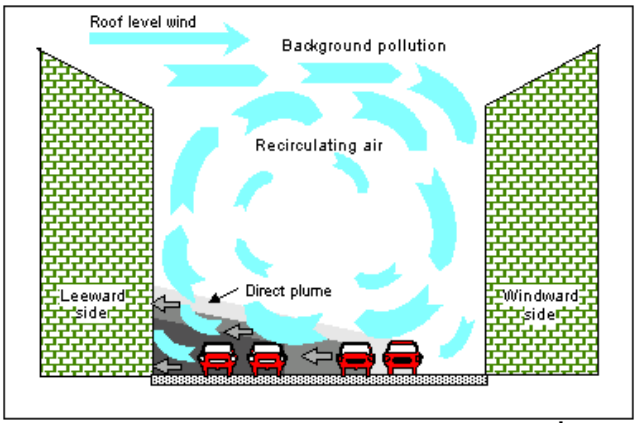



**Figure 27. Street Canyon Schematic (Source: http://envs.au.dk/en/knowledge/air/Models/OSPM/)**

The existence of a vortex within the street canyon depends on the aspect ratio, the ratio of building height to street width. When the aspect ratio is small, no consistent vortex forms within the canyon, and when the aspect ratio is large, multiple vortices may form. The widely used *o*perational *s*treet *p*ollution *m*odel (OSPM, Berkowicz, 2000) uses the vortex to divide the street canyon into two regions in modeling dispersion. This is discussed in more detail in a later section.

Some studies show the importance of the rooftop wind speed in determining dispersion in street canyons. Other studies indicate that vertical pollutant transport occurs due to an unstable shear layer that develops at the top of the canyon (Louka et al., 2000). The unsteady fluctuations of the shear layer cause intermittent recirculation in the canyon, thus intermittently flushing pollutants out of the canyon. Nakamura and Oke, (1988) measured temperatures within a 17 m tall street canyon. They found that the temperature difference between roof and canyon floor was usually less than 0.5 °C.

During the day the floor was warmer than the roof and during the night the floor was cooler. The temperature differences are very small, a fact that is attributed to rapid mixing within the canyon. The air is unstable or near neutral within the canyon, even during the night. During the day the vertical canyon surfaces provide large surface area to absorb solar radiation. The building material stores heat, with the result that during the night the canyon can remain warm. In the context of an entire urban area this is known as the urban heat island. The implication for dispersion is that the turbulence levels are likely to be larger within the urban area during nighttime than outside the urban area, and thus dispersion is also likely to be larger in the urban area than in a rural area at night.

The existing work on modeling street canyons is summarized by Vardoulakis et al., (2003). Existing models can be classified as: empirical regression models, semi-empirical box models, semi-empirical Gaussian plume models, Lagrangian particle models, unsteady Gaussian puff models, and CFD models. We are most interested in the semi-empirical box and Gaussian plume models because they require easily measured input variables and capture only the essential mechanisms of dispersion in cities. The Canyon Plume Box Model (CPBM) (Yamartino and Wiegand, 1986) was one of the first street canyon models that was evaluated with field data. The Operational Street Pollution Model (OSPM) (Berkowicz, 2000) borrowed the major ideas in CPBM and introduced simplifications that has made it more accessible to the non-expert user. Because OSPM is the most widely used street canyon model, we describe it next.

## vi.3. Methods

### vi.3.1. Operational street pollution model (OSPM)

Dispersion of pollutants in urban areas is governed by complex flows that result from the interaction of boundary-layer flow with buildings in the urban canopy. The flows can assume a number of forms even within the idealized street canyon shown in Figure 27. The street canyon refers to the structure formed by a long street lined on both sides by buildings of uniform height. The wind can be channeled along the street canyon if the upper level wind is at an angle to the axis of the canyon. When the rooftop wind is perpendicular to the street axis, the flow inside the canyon forms a vortex in which the wind direction at street level can be opposite to that at roof level. The length of the recirculating vortex at ground-level depends on the height of the upwind building. At oblique wind angles, the flow can also appear in the form of a helical vortex with its axis along the street. At intersections, the flow can separate at building edges and to give rise to vortices with vertical and horizontal axes (See Belcher, 2005 for a review of urban flows; Tiwary et al., 2011).

The foundation of OSPM is a model for dispersion in a street canyon. The model treats the road as a long area source with the emissions distributed evenly over the width of the road. Concentrations associated with emissions from vehicles in a street canyon are assumed to consist of two components: 1) a direct plume of emissions that is transported by the surface wind, and 2) a well-mixed region corresponding to the re-circulating flow in the canyon. The vortex divides the street into two regions; within the vortex, the surface wind blows towards the leeward side, and outside the vortex, the surface wind blows towards the windward side of the canyon. The direct plume contributions to the leeward and the windward side receptors depend on the fraction of road emissions within these two regions. If the vortex, whose length depends on the height of the upwind buildings and wind speed, covers the entire street width, there is no direct contribution to the windward receptor. Conversely, if the vortex length is small compared to the width of the road, the leeward receptor sees a small direct contribution, while the windward receptor is subject to most of the direct contribution. The second component of the concentration, the well mixed concentration within the vortex, depends on emissions into the vortex and the outflow from the top and the sides of the vortex, which is idealized as a trapezoid. In general, the total concentration, $C$, at a receptor is given by

$$C = C_d + C_m + C_b, \tag{1}$$

where $C_d$ is the direct plume contribution, $C_m$ is the well mixed concentration in the vortex, and $C_b$ is the background concentration corresponding to sources outside the road. We can illustrate the structure of OSPM by considering the special case when the wind blows perpendicular to the road, and the canyon is deep enough that the vortex covers the entire width of the road. Then, the direct contribution, $C_d$, on the leeward side of the canyon is given by

$$C_d = \sqrt{\frac{2}{\pi}} \frac{Q}{\sigma_w W} \ln\left(1 + \frac{\sigma_w W}{h_0 u_s}\right), \tag{2}$$

where $Q$ is the emission per unit length of the road, $\sigma_w$ is the standard deviation of the vertical velocity fluctuations at street level, $W$ is the width of the road, $h_0$ is the height over which the emissions are mixed next to the vehicle, and $u_s$ is the wind speed at the height of emissions, which is taken to be 2 m. For this special case, the windward receptor is only affected by the well mixed concentration given by

$$C_m = \frac{Q}{\sigma_{wt} W}. \tag{3}$$

where $\sigma_{wt}$ is the standard deviation of the vertical velocity fluctuations at the top of the canyon, which is related to the rooftop wind speed, and traffic induced turbulence at the base of the street canyon. The structure of the recirculating vortex in the street canyon is a function of the wind angle with respect to the street axis, and the height of the upwind building. The wind speed at emission level, $u_s$, is related to the rooftop wind speed, $u_t$, through an extension of the logarithmic profile to the emission height,

$$\begin{aligned} u_s &= u_t \frac{\ln(h_0 / z_0)}{\ln(H / z_0)} (1 - 0.2 p \sin\Phi) \\ p &= \max\left(\frac{H_{upwind}}{H}, 1\right) \end{aligned} \tag{4}$$

where $H$ is the average height of the canyon, $\Phi$ is the angle between the wind at roof top and the street canyon axis, and $z_0$ is the urban roughness length. Notice that when $H_{upwind} = 0$, the building has no effect on the wind speed at emission height. Similarly, the wind is allowed to flow through the canyon when $\Phi = 0$. The standard deviation of vertical velocity fluctuations near ground-level is taken to consist of two components: 1) that related to wind speed at emission height, and 2) that generated by vehicle motion:

$$\sigma_w = \left((\alpha u_s)^2 + \sigma_{wc}^2\right)^{1/2}, \tag{5}$$

where $\alpha = 0.1$ and $\sigma_{wc}$ is associated with turbulence induced by vehicle motion. OSPM parameterizes this component in terms of vehicle speed and dimensions, width of the road, and traffic flow rate. The emission rate, $Q$, is taken to be a function of the traffic flow rate, the mix of vehicles, and the emission factors for each of the pollutants. For the simplest case of one type of vehicle with a flow rate of $N$ (vehicles/s) and emission factor, $e_f$, (g/(m.vehicle)), the emission rate is given by

$$Q = Ne_f \tag{6}$$

In OSPM, $\sigma_w$, the standard deviation of the vertical velocity fluctuation, is related to the surface wind speed, $u_s$, and traffic induced turbulence, and the surface wind speed, in turn, is related to the rooftop wind speed. The inputs to OSPM are: 1) average height and width of the street canyon; the upwind and downwind buildings can have different heights, 2) the orientation of the street canyon, 3) the rooftop wind speed and direction, 4) the emission rate, which can be calculated internally within OSPM given the mix of vehicles and traffic flow rates, 5) the traffic parameters required to estimate traffic induced turbulence, and 6) the background concentrations as a function of time.

The implicit assumption is that the mean wind speed is the primary meteorological variable determining the magnitude of the dispersion. As we show later, the rooftop and near surface vertical turbulent velocities or the average rooftop vertical velocity provided better explanations of near road concentrations. Additionally, the assumption of a constant turbulent intensity of 0.1, used to estimate the near surface turbulence from the wind speed, is questionable based on observations of much larger turbulent intensities made in this study as well as those in other field measurements in Oklahoma City and Manhattan (Hanna et al., 2007).

In the next section we describe the analysis of near road concentration data collected in Hannover, Germany. The data is used to evaluate several dispersion models in order to identify the meteorological variables that govern dispersion in Hannover. The results also guided the development of the model to describe the data collected in the field studies conducted in Los Angeles.

## vi.4. Results and Discussion

### vi.4.1. Operational Analysis of long term measurements of near road concentrations of vehicle emissions to determine the primary variables governing dispersion in urban streets

The German Niedersächsisches Ministerium für Umwelt, Energie und Klimaschutz operates an air quality monitoring network throughout Germany. We obtained data from two monitoring sites that record gaseous pollutants, $PM_{2.5}$, and $PM_{10}$ concentrations as well as meteorological data. The two sites, HRSW and HRVS, are located in Hannover on Göttinger Strasse. Figure 28 presents a map of the buildings and site locations. The sites are positioned at two heights, one (HRVS) near ground level within the urban canopy and one (HRSW) on a building roof 34 m above the street site. Concentration measurements were made at 1.5 m AGL and 35.5 m AGL. The buildings adjacent to Göttinger Strasse are 20 m tall and have almost no gaps between them, thereby forming a 20 m tall, 25 m wide and 200 m long street canyon. Göttinger Strasse runs along a line that points 17 degrees west of north. The street level HRVS site was instrumented with a sonic anemometer at 10 m AGL to measure 30 minute average wind speed, temperature, turbulent fluxes of momentum and heat, and vertical and horizontal turbulent velocity. The roof level HRSW site measured 30-minute average wind speed, wind direction, solar radiation, temperature, humidity, pressure, and precipitation at 42 m AGL. Automatic traffic counts classified into passenger cars and trucks were made. Data was obtained from January 1, 2003 through December 31, 2007.

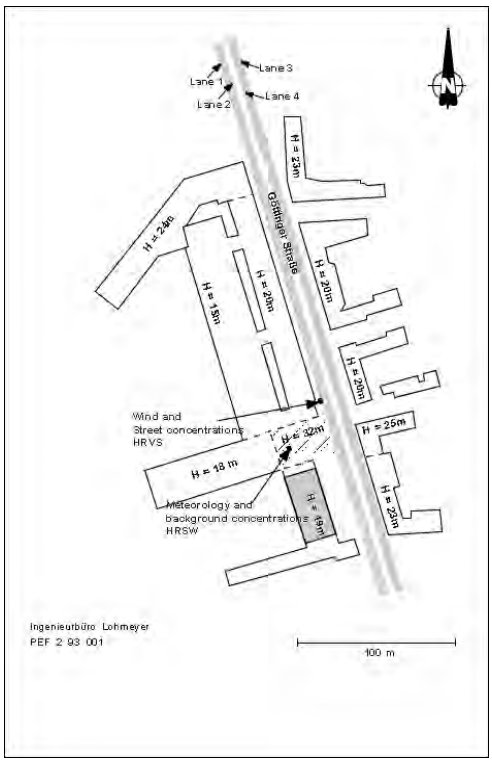



**Figure 28. Map of Göttinger Strasse showing locations of concentration and micro meteorology stations and building heights.**

We used the $NO_x$ concentration measurements for model comparison because $NO_x$ emission factors can be estimated within a factor of two. These emission factors were determined using the EPA Emfac 2007 emission model (CARB, 2015). We used the average emission factors for light and heavy duty trucks for the truck portion of the traffic and that of light duty vehicles for the passenger car portion. The emission resulting emission factors for the year 2003 are 0.465 g/km and 6.18 g/km for light duty vehicles and trucks respectively. Model performance was quantified through the geometric mean, $m_g$ and standard deviation $s_g$ of the ratio of the measured to the estimated concentrations, the correlation coefficient, $r^2$ and the fraction of measurements within a factor of two of model estimates, $fact2$ .

Figure 29 compares estimates from equation (2) using $h_0 = 2\,m$ with corresponding observed concentrations. The scatter plot shows that the model overestimates measurements by a factor of two, and the correlation between model estimates and observations is 0.37. The other panels of Figure 29 show the variation of the modeled and observed concentrations, normalized by emission rate, with 10 m wind speed, standard deviation of vertical velocity fluctuations, and wind direction. The variation of the observations is generated by binning data based on the variables on the x-axes and computing the average value in each bin. Error bars show standard deviations of the data within each bin. These figures show that the model systematically overestimates observations when the wind speed is less than about 1 m/s. We see that the normalized measured concentration varies linearly with $1/\sigma_w$. The model shows similar sensitivity to $1/\sigma_w$, except for very small values of $\sigma_w$ where the model underestimates concentrations.

The bottom right panel of Figure 29 shows that the model predicts the correct magnitude of concentrations when the wind direction is parallel to the street, and overestimates when the wind direction is perpendicular to the street. The model reproduces the observed variation of concentration with wind direction even though the model equations are independent of wind direction because the magnitude of the wind speed varies with direction. The model produces larger

concentrations when the wind direction is perpendicular to the street because the wind speed is smaller during these conditions.

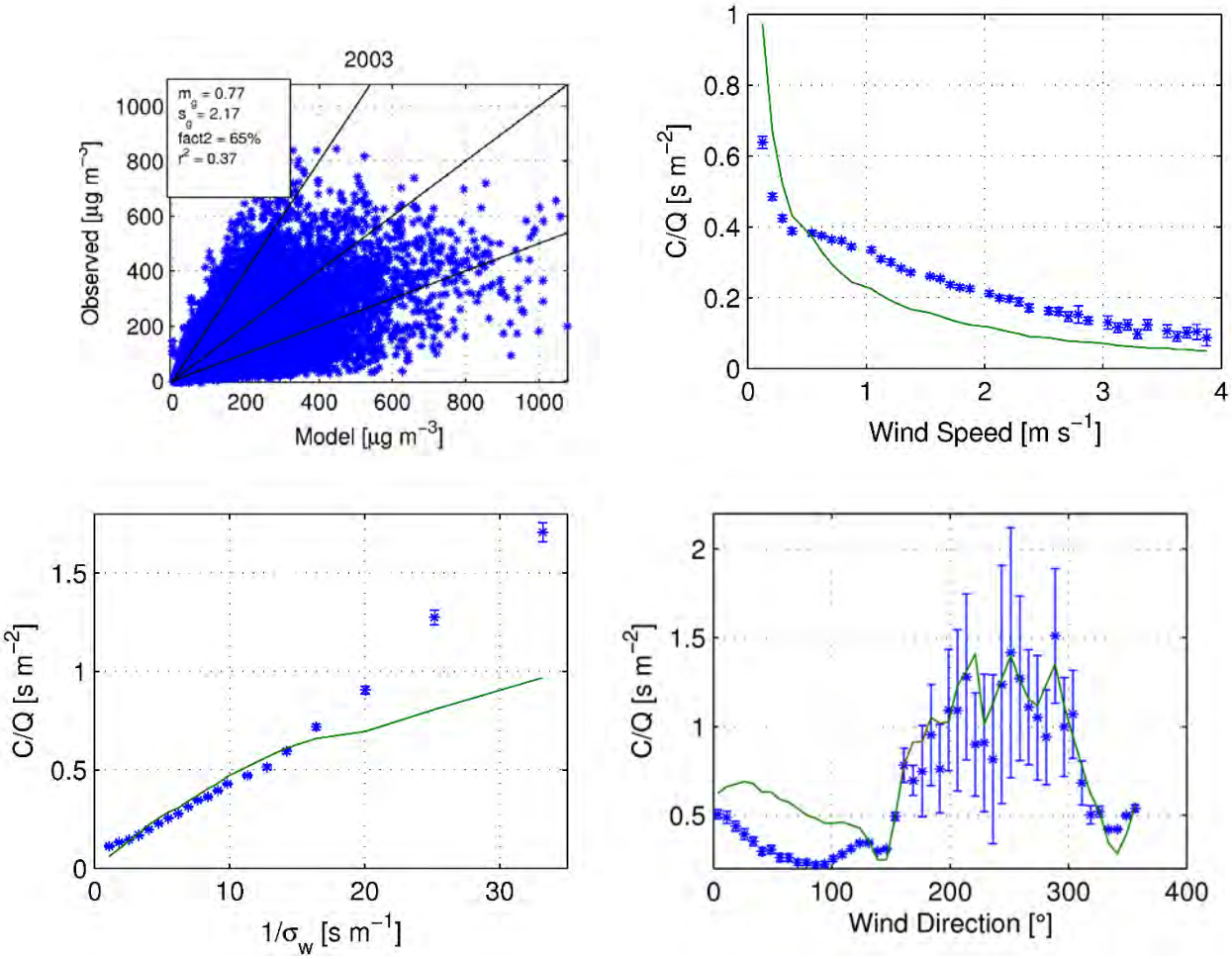



**Figure 29. Comparison of equation (2) with observations during 2003. Top left shows scatter plot. Top right shows variation of observations (blue) and model (green) with wind speed. Bottom left shows variation with $1/\sigma_w$. Bottom right shows variation with wind direction. Wind directions parallel to the street are 163 and 343 degrees.**

Figure 30 compares concentrations measured in 2003 with estimates from the model when the log term containing the explicit dependence on surface wind speed is neglected:

$$C_s = \alpha \frac{Q}{\sigma_w W} + C_r \qquad \textbf{(7)}$$

where $\alpha$ is a parameter whose value is determined empirically. The value of $\alpha$ that best describes the data using the assumed emission factors is 0.9. Equation (7), which is the leading term of Equation (2), is similar to the OSPM recirculating contribution model (Berkowicz et al., 1997), although in OSPM, the rooftop value of $\sigma_w$ is prescribed, and is determined from the rooftop wind speed through a constant turbulent intensity.

The model bias is smaller than that corresponding to equation (2), with an $m_g$ of 1.02. The correlation coefficient of $r^2 = 0.62$ is also better than that from equation (2). The fraction of data within a factor of two of the observations is 72%. The bottom left panel of Figure 30 shows that the model has the same sensitivity to $\sigma_w$ as the observations, although it tends to underestimate while equation (2) is unbiased except for very small values of $\sigma_w$. The top right panel of Figure 30 shows

that model also has the correct sensitivity to wind speed, and has less bias than equation (2) when the wind speed is less than 1 m/s. We conclude that the model without the explicit wind speed dependence performs better than that given by equation (2) under low wind speed conditions. The improved performance under low wind speed conditions is also seen on the bottom right panel of Figure 8, which shows concentration plotted against wind direction. In the figure we see that model overestimates less than equation (2) during perpendicular flow conditions, when the wind speed is smallest.

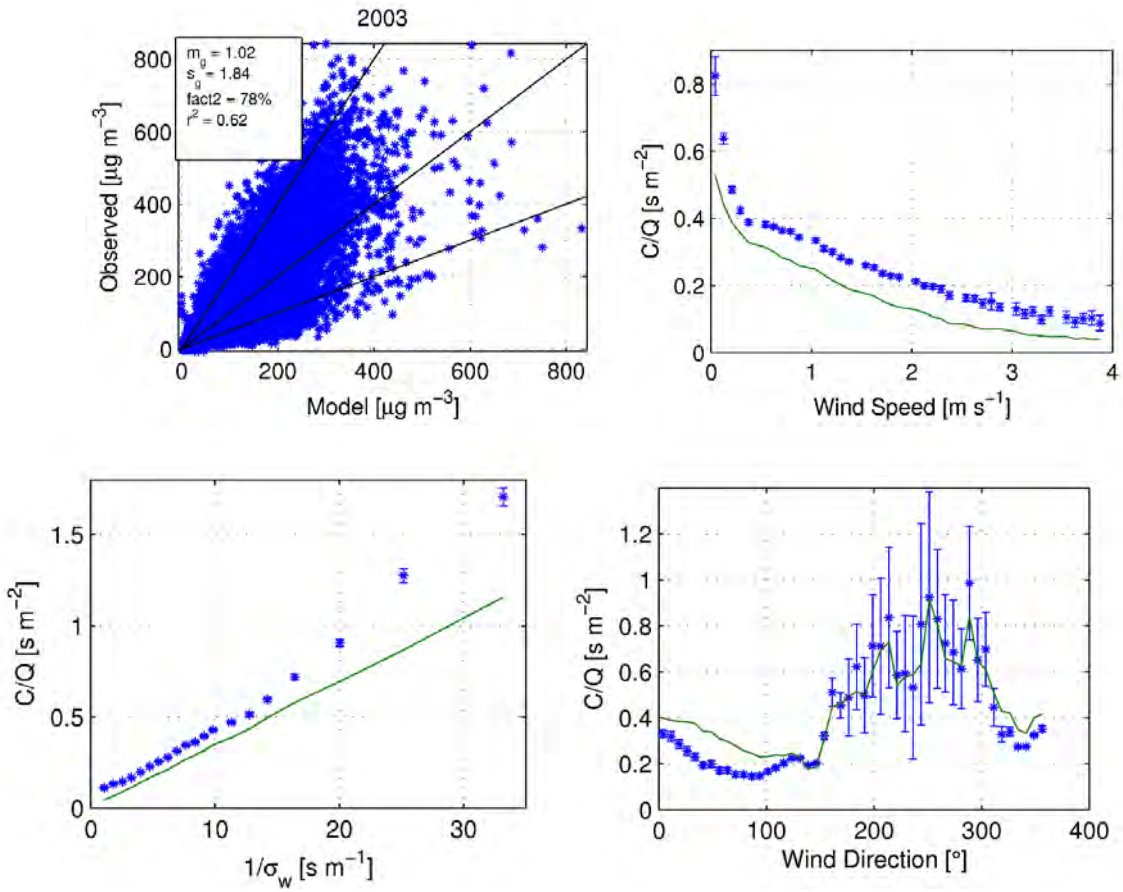



**Figure 30. Comparison of estimates from equation (2) without explicit wind speed dependence with corresponding observations during 2003. Top left shows scatter plot. Top right shows variation of observations (blue) and model (green) with wind speed. Bottom left shows variation with $1/\sigma_w$. Bottom right shows variation with wind direction.**

Based on this comparison we conclude that the vertical turbulent velocity explains most of the variance of the observed concentrations, while the explicit inclusion of wind speed does not improve predictions of near road concentrations. The importance of the vertical turbulent velocity and insensitivity to wind speed was observed previously in near road concentrations of vehicle emitted NO (Venkatram et al., 2007). It is surprising that the near road concentrations are not more strongly correlated with the street level wind speed because the standard deviation of vertical velocity fluctuations is normally strongly correlated with wind speed. However, in urban environments where mean wind speeds are small the horizontal turbulent velocities are usually very large relative to the mean wind speed, and the mean wind varies significantly depending on the presence of nearby buildings (Hanna et al., 2007). The result is that horizontal motion is better characterized as a turbulent dispersion rather than advection by a strong mean flow. Thus, the concentrations are primarily determined by turbulent transport in the vertical direction. This view of urban dispersion is somewhat inconsistent with the street canyon picture of a strong recirculating vortex flow.

This analysis of data from Hannover indicates that the street level vertical turbulent velocity is the dominant variable controlling dispersion within a street. The next section develops a model based on this result.

## vi.4.2. Development of a model to understand the impact of buildings on near-road concentrations using field measurements in Los Angeles

The analysis of measurements of near road concentrations of vehicle emissions in Göttinger Strasse, Hannover, Germany determined the primary meteorological variables that govern dispersion of vehicle emissions in urban environments. The analysis could not examine the impact of building morphology on concentrations because the data was obtained at a single site where the buildings were relatively uniform in height. The field studies conducted in Los Angeles provided the information required to include the effects of building morphology on dispersion of street-level emissions.

### *vi.4.2.1. Design of field measurements in Los Angeles*

The design of the Los Angeles field measurements is based on the knowledge gained from the analysis of data from Göttinger Strasse in Hannover. The analysis shows that the standard deviation of vertical velocity fluctuations measured at approximately half the building height is the primary variable governing near road concentrations. Thus, the Los Angeles field study included measurements of the near surface and rooftop turbulent velocities. The field study also included measurements of mean wind speed and wind direction in order to evaluate the performance of other dispersion models such as OSPM. OSPM estimates the near road concentration as the sum of the direct plume contribution from the road and the recirculating contribution, due to trapping of pollutants within the street canyon. The recirculating contribution is governed by the rooftop standard deviation of vertical velocity fluctuations. In OSPM (see section 2 for details), the rooftop turbulence and surface wind speed and turbulence levels are estimated from the rooftop mean wind speed. Thus, our field study included measurements of the turbulence and mean winds at the urban rooftop as well as at the surface.

For most practical applications, the rooftop wind speed and direction within the urban area have to be estimated from measurements of wind speed at a nearby local monitoring station, most likely located at a nearby airport. This is because 1) meteorological variables are not routinely measured in urban areas, and 2) it is difficult to make representative measurements in urban areas with tall buildings because flow around individual buildings can be significantly influenced by the wake effects of individual buildings. Thus, it is necessary to relate urban values that govern dispersion to upwind rural meteorology. The wind speed and turbulence from the upwind rural area are modified by the built environment as the air passes from an upwind monitoring station to the urban site of interest. The wind speed at the urban rooftop, and therefore the turbulence and near surface mean wind as well, are thus a function of the overall built environment characteristics between the rural reference location and the urban site. The modification of the meteorological variables between the reference and urban sites is modeled using the internal boundary layer (IBL) model described in a later section. The field studies conducted in Los Angeles were designed to evaluate the IBL model by including measurements of the mean wind and turbulent variables at a rural reference location upwind of the urban field sites.

We used ultrafine particles (UFP) as the tracer to evaluate the dispersion models. There are several reasons for this choice. First, UFP is associated with negative health effects (Knibbs et al., 2011). Second, at the time scales of interest, dispersion is the primary mechanism for reducing UFP particle number concentrations (Ketzel and Berkowicz, 2004). Third, we have access to several TSI3022A condensation particle counters that provide high resolution measurements of UFP.

Field measurements pose significant challenges to isolating the effects of the buildings on dispersion because variability in uncontrolled factors such as traffic emission rate can overwhelm the signal due to the presence of buildings. The evaluation of the dispersion model requires accurate estimates of the emission rates, which are difficult to determine in practice. During the typical congested driving conditions of urban environments, the local traffic within a street is often accelerating or idling, which increases the uncertainty in estimating emission rates (Smit et al., 2008). Emission models of gaseous pollutants and particle mass are usually accurate within a factor of two or three (Smit et al., 2010). Ultrafine particle number (UFP), which we use as the primary measured pollutant in the field study, has emission factors that vary by about an order of magnitude (Kumar et al., 2011). Because of the uncertainty in the emission estimates, UFP concentration measurements were made at two sections on the same street when possible: one section with tall buildings adjacent to the street, and another where there are no buildings or very short adjacent buildings. This design ensures that local vehicle emissions are similar at the two locations, allowing us to directly compare concentrations at the open and building sections to isolate the building effect. The experiment design conducted in practice has depended on availability of instruments, and the overall design has evolved as we gained knowledge from previous experiments. The measurements were made at various time periods between September 2013 and July 2014. Figure 31 shows the location of the field sites. Table 15 gives an overview of the dates during which measurements were made and summarizes the building morphology of the sites. Appendix A gives an overview of the building morphology of the various field locations in this campaign the details of each experiment and the instrumentation used at each of the field sites.

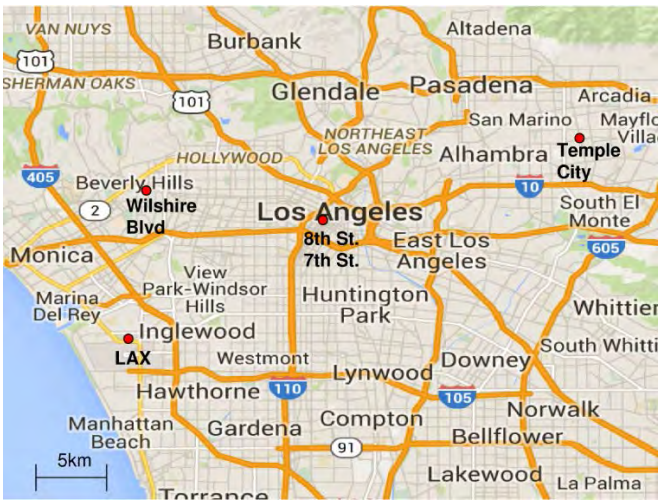



**Figure 31. Field study locations in Los Angeles County. Map Data: Google.**

**Table 15. Overview of measurement locations**

| Location | Dates | | Building Morphology |
|---|---|---|---|
| | Micrometeorology | UFP | |
| 8th St., Los Angeles | 4/23/14 – 5/13/14 | 5/7/14, 5/9/14 | Simultaneous open site and deep street canyon (*H/W=2.0*). |
| Wilshire Blvd., Beverly Hills | 5/19/14 – 7/1/14 | 5/30/14 | Variable building heights up to 50 m |
| Temple City Blvd., Temple City | 1/13/14 – 2/14/14 | 1/15/14 – 1/17/14 | Uniform height shallow street canyon (*H/W=0.2*). |
| 7th St./Broadway, Los Angeles | 9/20/13 | 9/20/13 | Deep street canyon. Two perpendicular streets. |

### *vi.4.2.2. Instrumentation*

We used Campbell Scientific CSAT3 3D sonic anemometers (Figure 31)to record the three components of wind speed and the sonic temperature at a sampling frequency of 10 Hz. The data were processed using the method described in (Kaimal and Finnigan, 1994) to yield the 30 or 60

minute averaged values of the turbulent heat flux, surface friction velocity, standard deviation of the vertical and horizontal turbulent velocities, wind speed, wind direction, temperature, and Monin-Obukhov Length.

TSI 3022A condensation particle counters (Figure 31) were used to record ultrafine particle number concentrations at a sampling rate of 1 Hz. Samples were drawn through a copper and Tygon tube with the tube sampling inlet set at 1 m above ground level. The instruments measured the concentration of particles with diameters greater than 10 nm (50% detection efficiency is 10 nm). The inlet flow rate is $25\ cm^3 s^{-1}$ and the flow rate through the detector is $5\ cm^3 s^{-1}$. Power was supplied by $12\ V\ 100\ A\ hr$ lead acid deep cycle batteries through $12\ V\ to\ 120\ V$ AC power inverters. Data loggers were constructed to record data from the serial port and store it on SD cards. AQMesh five gas concentration monitors (Figure 31) were used to measure concentrations of carbon monoxide, nitrogen oxide, nitrogen dioxide, ozone, and sulfur dioxide at the 8th St and Temple City field sites.

**Figure 32. Instruments used in Los Angeles filed measurements. Left - Campbell scientific CSAT3 sonic anemometer. Center - Condensation particle counter. Right - AQMesh concentration monitor (at top of photo).**

#### *vi.4.2.3. Vertical dispersion model*

Based on the analysis of the data collected in Hannover, Germany, we formulated the Vertical Dispersion Model (VDM) by assuming the horizontally averaged concentration in the street canyon is governed primarily by vertical transport, so that the flux of pollutants at the surface is matched by vertical turbulent diffusion:

$$Q \sim KW\left(\frac{C_s - C_r}{H}\right) \qquad \textbf{(8)}$$

where $Q$ is the emission rate per unit length of the street canyon, $W$ is the width of the canyon, and $H$ is the equivalent height of the canyon. If we assume that the background makes similar contributions to $C_s$ and $C_r$, we can evaluate Equation (8) with observations by using the difference between the observed $C_s$ and $C_r$. The eddy diffusivity $K$ is expressed as

$$K = \sigma_w l \qquad \textbf{(9)}$$

where $\sigma_w$ is the vertical average, between the surface and roof, of the standard deviation of vertical velocity fluctuations, and $l$ is a mixing length. The vertically averaged $\sigma_w$ is estimated from the measured surface, $\sigma_{ws}$, and roof, $\sigma_{wr}$, values from

$$\frac{1}{\sigma_w} = \frac{1}{2}\left(\frac{1}{\sigma_{wr}} + \frac{1}{\sigma_{ws}}\right) \tag{10}$$

If we assume that the size of the large turbulent eddies dominating vertical mixing is limited by the smaller of the street width and height, $l$ is proportional to the smaller of $W$ and $H$, so that

$$l \sim \left(\frac{WH}{W+H} + h_0\right) \tag{11}$$

where $h_0$ is a mixing length associated with the size of the source. Substituting Equations (9) and (11) into Equation (8) yields

$$C_s = \frac{Q}{\beta\sigma_w W}\frac{H\left(1+a_r\right)}{H+h_0\left(1+a_r\right)} + C_r \tag{12}$$

where the aspect ratio $a_r = H/W$. Equation (12) has the same form as Equation (4), except that it contains the aspect ratio, *H/W*. The value of $\beta$ that is consistent with the aspect ratio of Göttinger Str. and the best fit value for $\alpha = 0.9$ in Equation (4) is 1.7. However, we find that a value of 1.0 provides a better fit to the Los Angeles data. Note that the parameter β combines the proportionality constants implied in Equations (8) and (11). If measurements at roof level are not available, we can estimate $C_r$ by assuming that the local emissions are matched by vertical transport at roof level:

$$Q = \gamma C_r \sigma_{wr} W \tag{13}$$

where the value of $\gamma$ is determined by fitting the model to field measurements described in the next section. Substituting Equation (13) into (12) yields

$$C_s = \frac{Q}{\gamma\sigma_{wr}W}\left[1 + \frac{\gamma}{\beta}\frac{\sigma_{wr}}{\sigma_w}\frac{H\left(1+a_r\right)}{H+h_0\left(1+a_r\right)}\right] \tag{14}$$

Equations (12) and (14) are referred to as the Vertical Dispersion Model (VDM). Equation (12) can be used if $\sigma_w$ and $C_r$ are known. In the field studies conducted in Los Angeles, information at roof level was not available at several of the sites, and it was necessary to estimate $\sigma_{wr}$. We can estimate $\sigma_{wr}$ by assuming that turbulent kinetic energy produced at roof level, per unit length of street, $u_{*r}^2 U_r W$, is dissipated over the volume of the street:

$$u_{*r}^2 U_r W \sim \sigma_{wr}^3 WA \sim \frac{\sigma_w^3}{l} WH \tag{15}$$

where $l$ is the length scale of the large turbulent eddies within the canyon, and $u_{*r}^2$ and $U_r$ are the shear stress and the mean wind speed at roof level, both of which are correlated with $\sigma_{wr}$. If $l$ is similar to the form given by Equation (11), we can write the semi-empirical expression

$$\frac{\sigma_{wr}}{\sigma_w} = \left(1 + \eta \frac{H}{W}\right)^{1/3} \quad \textbf{(16)}$$

where $\eta$=0.4 provides the best fit with the data as shown in a later section. The ratio of rooftop and average $\sigma_w$ is nearly constant because the 1/3 power in Equation (16) results in low sensitivity to the aspect ratio.

The models described in this section were evaluated with data from field studies conducted at several locations in Los Angeles, California, the details of which are presented in in section 3. At most of these locations, the buildings lining these streets varied substantially in height. So the application of the dispersion model depended on defining an effective building height, *H*. We found that the following definition provided the best agreement between model estimates and observations:

$$H = \frac{1}{L}\sum_i H_i B_i \quad \textbf{(17)}$$

where $L$ is the street length, $H_i$ and $B_i$ are the height and width (along the length of the street) of building $i$, and the sum is taken over all the buildings on one side the street. Equation (17) can be interpreted as the area-weighted building height: the sum of the frontal area of the buildings divided by the street length. Then, the equivalent building height used in Equation (11) is the average over both sides of the street.

We assume that the modeled concentration represents an average over the street canyon within one city block. For the effective building height to be consistent with the model, it is calculated from the geometry of all the buildings bordering the street canyon within one city block. The use of the block length for defining the scale for horizontal inhomogeneity is arbitrary, but the assumption of horizontal homogeneity within one city block has been used in models such as SIRANE (Soulhac et al., 2011), and comparisons with observations indicate that this is a useful assumption.

Figure 33 evaluates the leading term, $1/(\sigma_w W)$, of the VDM with observations made at the building section of 8th St. on May 9th, 2014. The surface concentration in the figure is the average of the concentration on both sides of the street. The vertical concentration differences shown on the left panel are 30 minute averages normalized by the emission rate calculated using the traffic flow rate and constant emission factor of $10^{14}\ \mathrm{veh}^{-1} km^{-1}$. This emission factor is the same order of magnitude as that observed in an urban street canyon (Ketzel et al., 2003). We assumed that the background concentrations at the surface and the roof were the same, so that the concentration difference represented only the component associated with emissions from the street canyon.

The left panel shows that the observations are correlated with $1/(\sigma_w W)$, which is proportional to the predictions of the VDM. The right panel assumes that the emissions are constant, corresponding to the daily average traffic flow. Neglecting the variation of traffic flow surprisingly improves the correlation between model estimates and observations of $(C_s - C_r)/Q$. This illustrates the uncertainty in relating UFP emissions to the traffic flow rate and a constant emission factor, since variation in vehicle speed and acceleration can significantly affect the emission factor (Kittelson et al., 2004). It is clear from the analysis that the concentration difference between the surface and the

roof is governed by the standard deviation of the vertical velocity fluctuations. This is consistent with the assumption underlying the VDM.

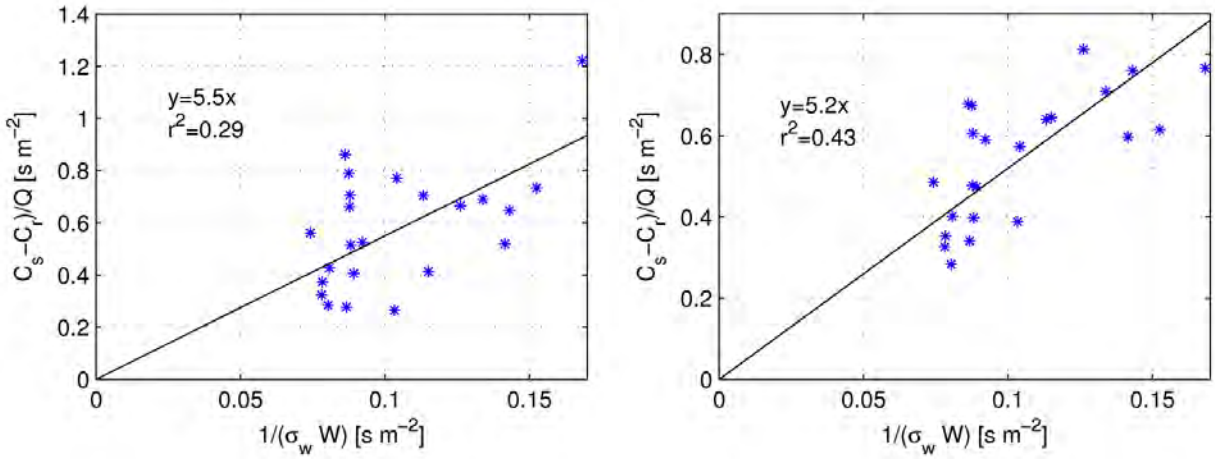



**Figure 33: Evaluation of the VDM with 30 minute average data from the building section of 8th Street (LA). Left panel: the emission rate is taken to be the 30 minute average value. Right panel: the emission rate is the daily average value. Surface concentrations are averages of both sides of the street.**

We next describe the evaluation of the VDM at the other sites in Los Angeles where the field studies were conducted. This required estimating the effective aspect ratio of the field study sites. We obtained building height and outline information from the Los Angeles Count GIS data portal (Los Angeles County, 2008), which was used to calculate the built environment parameters shown in Table 16 for each site. The table also shows the average hourly traffic recorded during the field measurements.

**Table 16. Summary of area weighted building height, street width, and aspect ratio of all sites.**

| Site | Area weighted building height (m) | Street width (m) | Aspect ratio | Average Traffic (veh/hr) |
|---|---|---|---|---|
| 8th St Building Section | 43.25 | 20.0 | 2.16 | 722 |
| 8th St Mid Section | 34.5 | 20.0 | 1.73 | 722 |
| Broadway | 35.9 | 26.0 | 1.38 | 1013 |
| 7th St | 45.8 | 25.0 | 1.83 | 1162 |
| Temple City | 6.00 | 30.0 | 0.20 | 1849 |
| Wilshire Blvd Building | 36.0 | 30 | 1.20 | 2772 |
| Wilshire Blvd Open | 8.25 | 30 | 0.28 | 2772 |

Figure 34 shows the evaluation of equation (12) with the vertical concentration differences at all the sites in the Los Angeles field study where rooftop concentration measurements were made. We have used a value of $\beta = 1.0$ for the VDM model estimates. The slope of the line that fits the data is consistent with a UFP emission factor of $2 \times 10^{14}\ veh^{-1}\ km^{-1}$. This value is within the range reported in literature (Ketzel et al., 2003). The model underestimates the concentrations on Broadway, but most of the model estimates are within a factor of two of the observations.

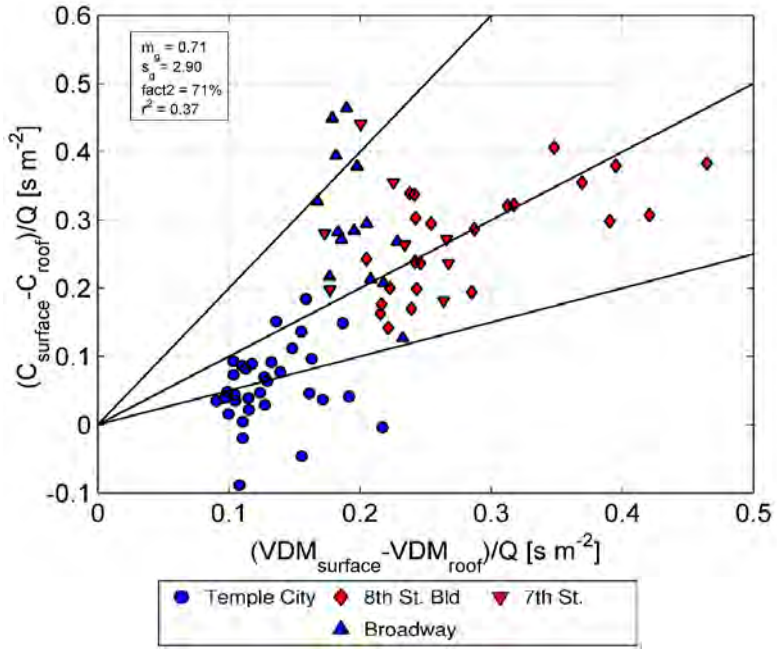



**Figure 34. Evaluation of VDM model with data collected in the Los Angeles field studies. The concentration has been normalized by the daily average emission rate assuming an emission factor of $2 \times 10^{14}\ veh^{-1}\ km^{-1}$.**

### *vi.4.2.4. Evaluation of VDM with the local contribution*

At several of the field sites, we did not have access to the roof of a tall building to make the measurements required to eliminate the background concentration using the vertical difference in concentrations. A model for the background, such as that in Pournazeri et al., (2014) could be used to estimate the background; however, it is difficult to apply such a model because it requires a detailed particle emission inventory for the LA basin. This inventory is not yet available in view of the large uncertainty in estimating emissions of ultrafine particles. Moreover, the "open" sites have no well-defined rooftop concentration since there is no building height at which to measure the concentration. For this reason, we developed an alternative method to analyze the data that does not rely on measurements of the roof concentration. Instead of using the vertical concentration difference to evaluate equation (12), we determined the contribution of local emissions to the total concentration observed at the surface monitors, and compared only this “local contribution” with the estimates from equation (14).

The UFP concentration time series contains information about the local vehicle emissions in the form of large amplitude short lived spikes superimposed on the slowly varying baseline. These local emission events from high emitting vehicles produce large concentration spikes that can be separated from the total concentration. We filtered the signal to separate the slowly varying component from the spikes, which contained information about local emissions. A moving average filter with a window size larger than the time scale of the spikes does not adequately separate the two components because the concentration distribution is highly skewed, making the average an inadequate measure of the baseline concentration. Instead of the moving average, we used a windowed percentile to separate the components. We defined the baseline as the concentration that is below a chosen percentile of the concentration distribution. Then, within each time window of a chosen length, each data point was classified as either baseline or spike if the concentration was below or above the percentile cutoff of the window. The baseline was then constructed by linearly interpolating between the points that are classified as baseline, and the spikes were separated by subtracting the baseline from the total. This type of analysis is common for analyzing UFP time series, especially in analysis of mobile monitoring data (Bukowiecki et al., 2002).

Figure 35 shows the result of this procedure with the baseline at the 10th percentile for several hours of data collected in 8th St on May 9th, 2014. The window size was taken to be 10 minutes to capture several spikes, each of which lasted for about a minute. This procedure resulted in a clear separation of the spikes and baseline concentrations.

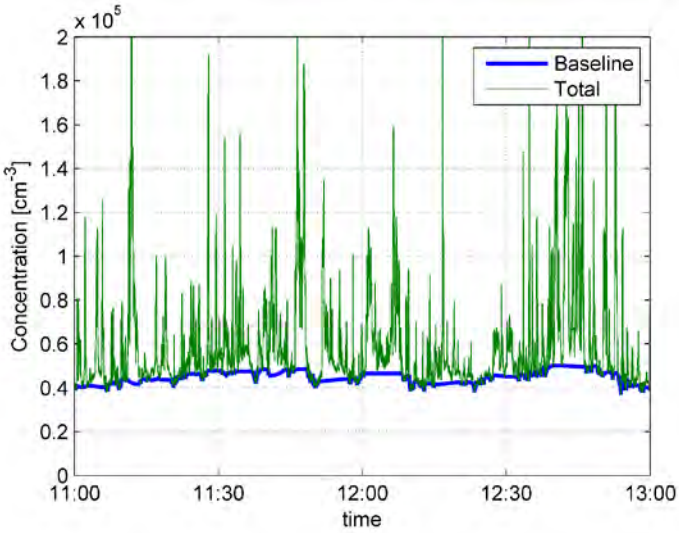



**Figure 35. Baseline and total concentration.**

A windows size of 10 minutes resulted in an adequate separation of the concentration signals, but can be considered to be somewhat arbitrary. We calculated the 30-minute average of the baseline concentration for window sizes of 5, 10, and 15 minutes, and found no significant difference in the results. This is shown in Figure 36 for data collected at 8th St. Because the cutoff percentile is also an arbitrary choice we chose different cutoff percentiles and compared the resulting 30-minute average baselines. Figure 36 shows that the baseline is affected by the choice of cutoff, with larger cutoffs producing larger baselines. The spikes are also sensitive to the choice of cutoff. However, this does not affect the analysis significantly because different cutoffs only result in a shift of the concentrations by a constant amount. The important feature of this procedure is that it removes the variability caused by variation in the baseline.

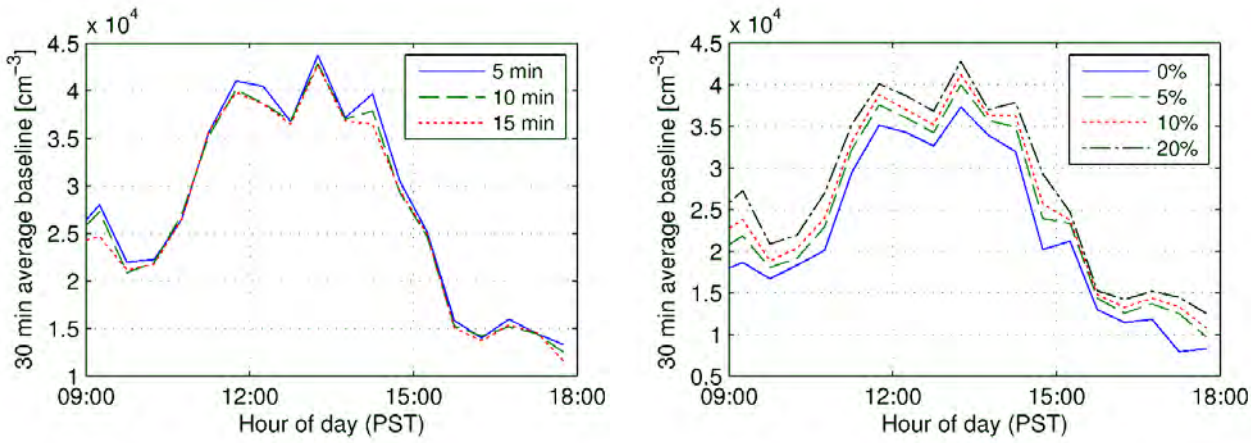



**Figure 36. Sensitivity of calculated baseline to window size (left) and cutoff percentile (right).**

The VDM was applied to the data from the field studies using the best fit parameters $h_0 = 2\,m$, $\gamma = 1.0$, and $\beta = 1.0$. The values of $\beta$ and $\gamma$ were determined by matching the observed and modeled concentrations from the Los Angeles data. The value of $\beta = 1.0$ is smaller than the value of 1.7 that corresponds to the value determined from the best fit of equation (14) to the Göttinger Strasse data. This uncertainty in parameter values is acceptable in the light of the uncertainty in

NOx emission estimates, which are mostly within a factor of two of observations (Smit et al., 2010). This uncertainty range suggests that $\beta$ lies between 0.85 and 3.4. We have chosen $\beta = 1.0$ because this produces a UFP emission factor consistent with literature values. The value of $\gamma = 1.0$ produces a good fit between the surface local contribution and Equation (14) and also produces the correct magnitude of the rooftop value of the local contribution through Equation (13).

Figure 37 indicates that the model provides a good description of the measured local contributions of UFP at most of the sites. This implies that local contributions are primarily governed by the ratio of equivalent building height to street width and the standard deviation of the vertical velocity fluctuations. The slope of the fit line is consistent with an emission factor of $1 \times 10^{14}\ veh^{-1}\ km^{-1}$, which is within the range reported by (Ketzel et al., 2003).

The observed local contribution at the 8th St open site is much larger than that predicted by the model. The emissions have to be approximately five times larger than that estimated based on the traffic flow rate on 8th St to account for the model underestimation. We suspect that emissions from adjacent streets influence the concentration at the open site, which implies that the VDM estimate represents the concentration averaged over an area larger than a single street, and the effective building height for the open site should encompass the buildings within a larger area than the region directly adjacent to the street. To examine this possibility, the building height of the 8th St open section was changed from its initial value of zero to the area weighted building height of the 8th St building section. The right panel of Figure 37 shows that now the model has almost no bias for the 8th St open site. Thus, the model underestimation for the open site is likely due to underestimation of the effective building height. This also means that concentrations in a relatively open site among buildings are affected by the presence of adjacent buildings.

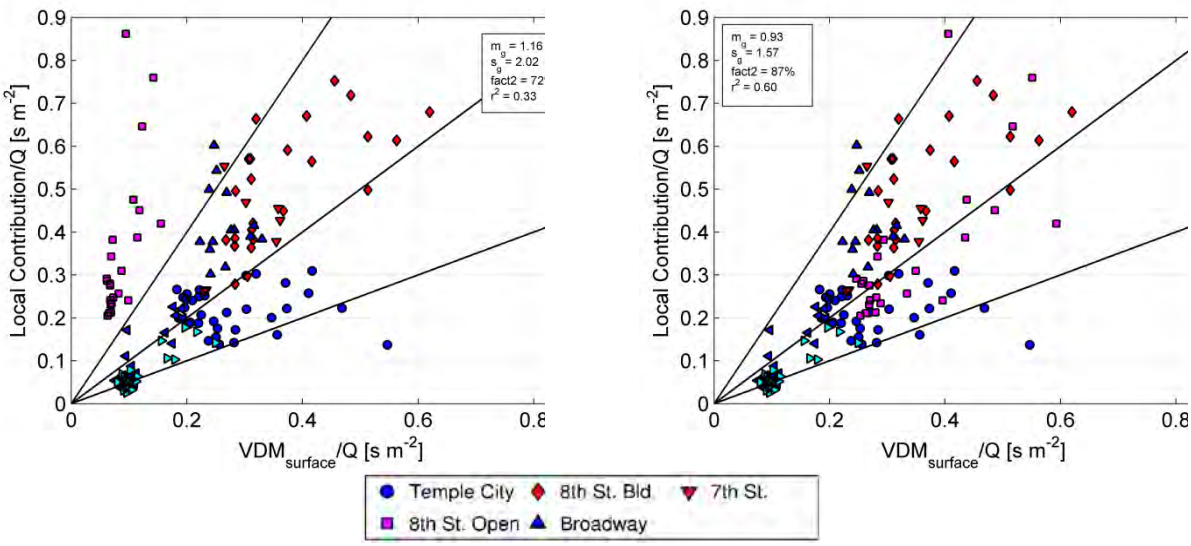



**Figure 37. Comparison of estimates from VDM with 30-minute averaged local contributions. Concentration is normalized by daily average emission rate, assuming an emission factor of $10^{14}\ veh^{-1}\ km^{-1}$. Left – The 8$^{th}$ St open section building height is zero. Right - The building height of the 8th St open section has been set equal to that of the 8th St building section.**

Our analysis of data from field studies conducted in urban areas suggests that vertical mixing governs near surface concentrations within the urban canopy. This conclusion is supported by observations analyzed in (Hanna et al., 2014), which show that data from field studies conducted in Manhattan, NY, indicate rapid vertical mixing in the presence of buildings. We show that modeling the air quality impact of vehicular emissions within cities with significant building height variability reduces to estimating the effective aspect ratio of the street, the ratio of the mean street width and the area weighted building height, and the roof level $\sigma_w$. The effective aspect ratio plays the major

role in magnifying concentrations relative to those that would have been measured in the absence of buildings. Concentrations are relatively insensitive to the mean wind speed at the rooftop or surface. This supports the conclusion that vertical turbulent transport rather than advection by the mean wind dominates dispersion in cities.

We have provided evidence to show that the simple vertical dispersion model provides an adequate description of the variation of local contributions within a street lined by buildings of varying heights. However, in order to estimate the impact of buildings on concentrations that would have been observed in the absence of buildings, we need to quantify the effects of buildings on the primary meteorological variable governing dispersion within the VDM, the standard deviation of the vertical velocity fluctuations, $\sigma_w$. This requires a model that will trace the evolution of the urban boundary layer as it travels from a rural area to the urban site of interest. This model needs to incorporate the morphology of buildings upwind of the urban site. While progress has been made (Luhar et al., 2006) in developing such a model, the model estimates are not yet reliable. However, we can obtain insight into the problem through empirical information from the field studies. This is addressed next.

#### *vi.4.2.5. Micro-meteorology*

The relationships among the $\sigma_w$ measured on 8th street and those at the upwind LAX location are illustrated in Figure 38. The top left panel shows that the standard deviation of the vertical velocity fluctuations at roof level and at street level are highly correlated. The street level $\sigma_w$ is about 0.67 of the roof level values, which is consistent with Equation (16). The observed ratio is similar to the ratios of 0.63 and 0.54 found in the MSG05 (Hanna et al., 2007) and MID05 (Hanna and Zhou, 2009) measurements in Manhattan. The top right panel justifies the assumption used to derive Equation (16): the shear production of turbulence at roof level, $u_*^2 U_r$, is proportional to $\sigma_w^3$ at roof level.

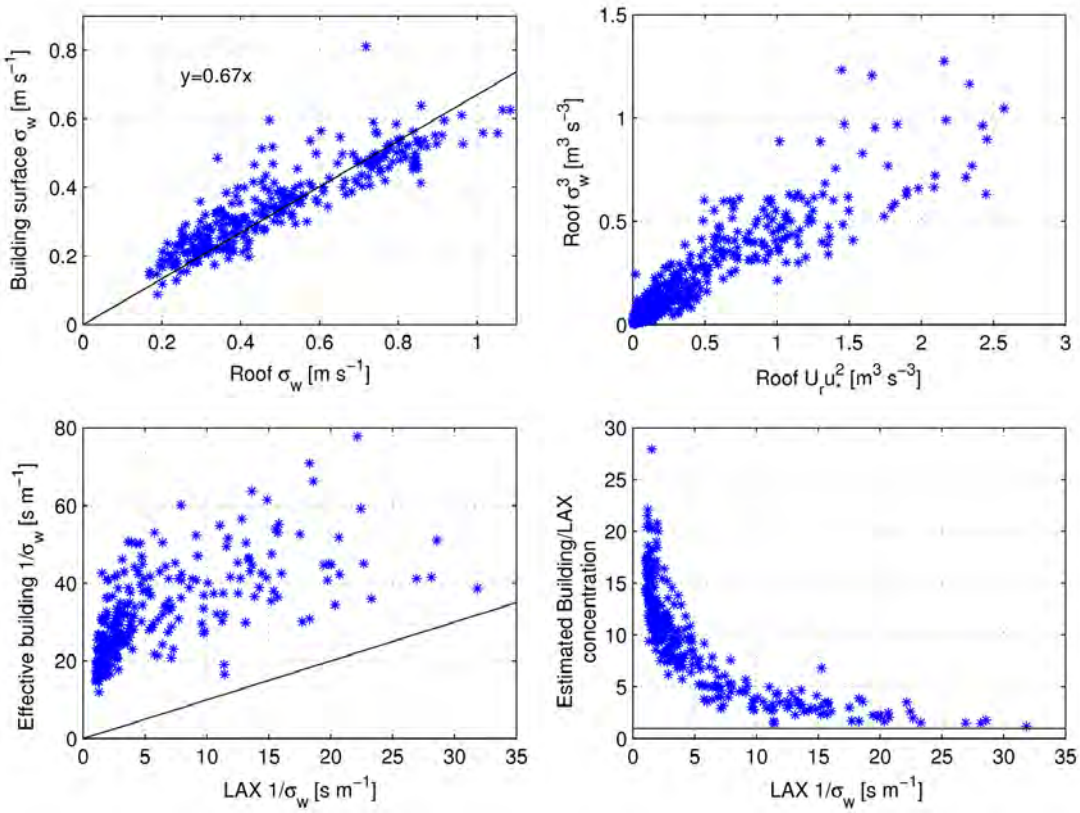



**Figure 38. Micrometeorology measured at Los Angeles 8th St. site. Micrometeorology is determined from the 8th St. "building" and "roof" sonic anemometers.**

The bottom two panels of Figure 38 illustrate the effect of the building aspect ratio on the effective $\sigma_w$. The bottom left panel compares $1/\sigma_w$ measured at the upwind LAX site to the effective value at the building site,

$$\frac{1}{\sigma_{weff}} = \frac{1}{\sigma_{wr}} + \frac{1}{\sigma_w}\left(\frac{\gamma}{\beta}\frac{H(1+a_r)}{(H+h_0(1+a_r))}\right) \tag{18}$$

derived from Equation (14). Notice the role of the aspect ratio in decreasing the effective $\sigma_w$. We see that the effective building values of $1/\sigma_w$ (a measure of concentration) are always larger than those at the upwind LAX site, but they are not correlated for upwind values less than about 0.2 m/s; while the LAX $\sigma_w$ decrease, the effective building $\sigma_w$ values fluctuate around 0.03 m/s. The bottom right panel of Figure 38 shows that, based on the modeled concentrations, the building always magnifies concentrations relative to the open terrain at LAX, but the magnification decreases to unity when the $\sigma_w$ at the LAX site is about 0.03 m/s.

By assuming that turbulent kinetic energy produced at roof level is dissipated inside the canyon, we have derived the tentative relationship given in Equation (16), with the best fit to the data when $\eta = 0.4$. Figure 39 shows that this equation provides a useful estimate of the near surface $\sigma_w$ using the measured values at roof level at several sites in Los Angeles.

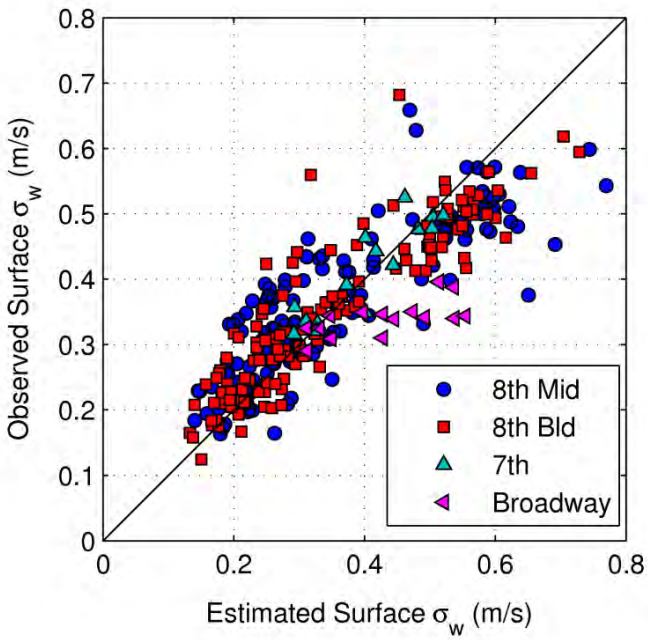



**Figure 39. Estimates of vertical velocity fluctuations measured at street level compared with measured values at several sites in Los Angeles. The black line is the 1 to 1 line.**

The relationship between the $\sigma_w$ measured at the roof level and the upwind site can, in principle, be estimated using an internal boundary layer model (Luhar et al., 2006). Figure 40 compares the LAX and 8$^{th}$ St. roof values of $\sigma_w$. For wind blowing from LAX to 8$^{th}$ St., which occurs during daytime, the $\sigma_w$ measured at the rooftop is about 1.2 times the LAX value. For other wind directions, the two values of $\sigma_w$ are not well correlated, and during nighttime the roof $\sigma_w$ can be much larger than that at LAX. This variability in relationships between the upwind $\sigma_w$ and the roof top value suggests using measurements as a guide to specifying the value of $\sigma_w$.

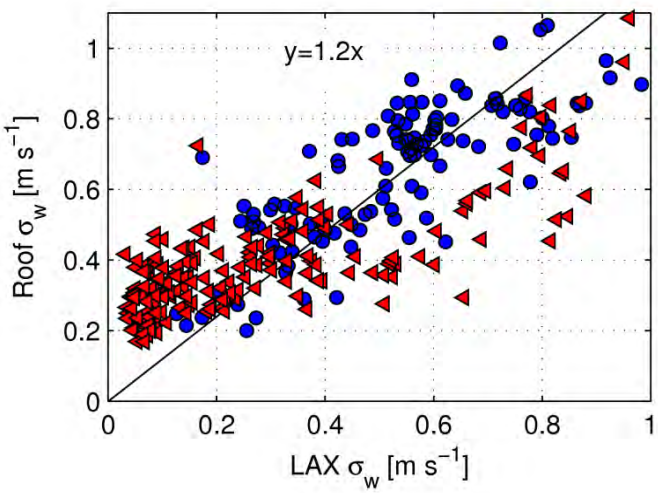



**Figure 40. Relationship between vertical velocity fluctuations measured at LAX and those at roof level on 8th St. ● - Data filtered so the roof site is downwind of the LAX site. (Wind direction at LAX within 20 degrees of the heading from LAX to 8th St.) ◀ - All other data.**

### *vi.4.2.6. Using VDM to Assess Exposure to Traffic Emissions in TODs*

VDM provides city planners with the tools necessary to evaluate the impact of the built environment on exposure to traffic emissions in TODs. The model can be used in two ways: (1) to understand the effect of changing model input variables on the air pollution concentration (sensitivity analysis), and (2) to estimate the air pollution concentrations for real-world cities and alternative TOD designs using detailed emission and meteorological inputs and parameters. This section shows the results of the sensitivity analysis and describes how VDM can be used to generate detailed air pollution concentration estimates. For these purposes, the VDM equations have been translated into a tool that consists of a python code and an excel spreadsheet that provide the overall model formulation and an easy to use graphical interface. This spreadsheet tool allows city planners to use VDM to conduct sensitivity analysis and generate practical concentration estimates.

The spreadsheet tool uses readily available meteorological inputs and user specified traffic counts, emission factors, and building parameters. The process used by the tool to estimate concentrations of local traffic emissions is as follows:

1) Derive estimates of the surface roughness length of the city. This is done using methods of MacDonald et al. (1998) to convert the plan area fraction, frontal area fraction, and average building height into estimates of the zero plane displacement height and surface roughness length through equations (19) and (20):

$$\frac{d}{\overline{H}} = 1 + 4.43^{-\lambda_p}(\lambda_p - 1) \qquad \textbf{(19)}$$

$$\frac{z_{0urban}}{d} = \left(1 - \frac{d}{\overline{H}}\right)\exp\left[-\left(0.5\frac{C_D}{\kappa^2}\left(1 - \frac{d}{\overline{H}}\right)\lambda_f\right)^{-0.5}\right] \qquad \textbf{(20)}$$

where $z_{0urban}$ is the surface roughness length, $d$ is the zero plane displacement height, $\overline{H}$ is the average building height, $\lambda_p$ and $\lambda_f$ are the plan and frontal area fractions, $C_D$ is the building drag coefficient that is taken to be 1.2, and $\kappa$ is the Von Karman constant.

2) Estimate the urban rooftop standard deviation of vertical velocity fluctuations, $\sigma_{wr}$, from measurements at an upwind rural or airport reference location. We use a simplified form of the internal boundary layer model (Luhar et al., 2006) to convert the standard deviation of vertical velocity fluctuations from the rural area to the corresponding rooftop value:

$$\frac{\sigma_{wr}}{\sigma_{wrural}} = \left( \frac{z_{0urban}}{z_{0rural}} \right)^{0.14} \quad \textbf{(21)}$$

where $\sigma_{wrural}$ is the standard deviation of vertical velocity fluctuations at the rural or airport reference location and $z_{0rural}$ is the surface roughness length at the reference location. $\sigma_{wrural}$ can be taken from standard AERMOD (Cimorelli et al., 2005) meteorological input files produced by the AERMET preprocessor. These input files are readily available for many meteorological measurement sites in California.

3) Compute the concentration of local traffic emissions (from within the TOD) at the building rooftop using equation (13). Alternatively, the rooftop concentration can be calculated using AERMOD and input into the spreadsheet. For this option, AERMOD is run without considering the buildings explicitly. The impact of buildings in AERMOD is only included through the standard AERMOD urban options.

4) Compute the surface concentration of local traffic emissions using equation (12) and equation (16) using the user-specified area-weighted building height and street width.

The user can specify emission inputs in the spreadsheet by providing traffic flow rates and emission factors. The tool includes built-in emission factors for $PM_{2.5}$, UFP, and $NO_x$.

The model calculated results are displayed in the spreadsheet using several figures. First, the diurnal variation of the concentrations is shown. Second, the variation of the concentration and the magnification, the ratio of air pollution concentration with and without buildings, with building height are shown. The user can also export data from the spreadsheet to conduct additional analysis.

We now provide some basic results that can be examined by planners using the spreadsheet tool. Figure 41 shows the sensitivity of the predicted magnification to changes in the area-weighted building height generated using the spreadsheet. The magnification increase quickly with building height at first and then increases linearly with the ratio of area-weighted building height to street width. This result provides insight into the effect of increasing building heights within a TOD.

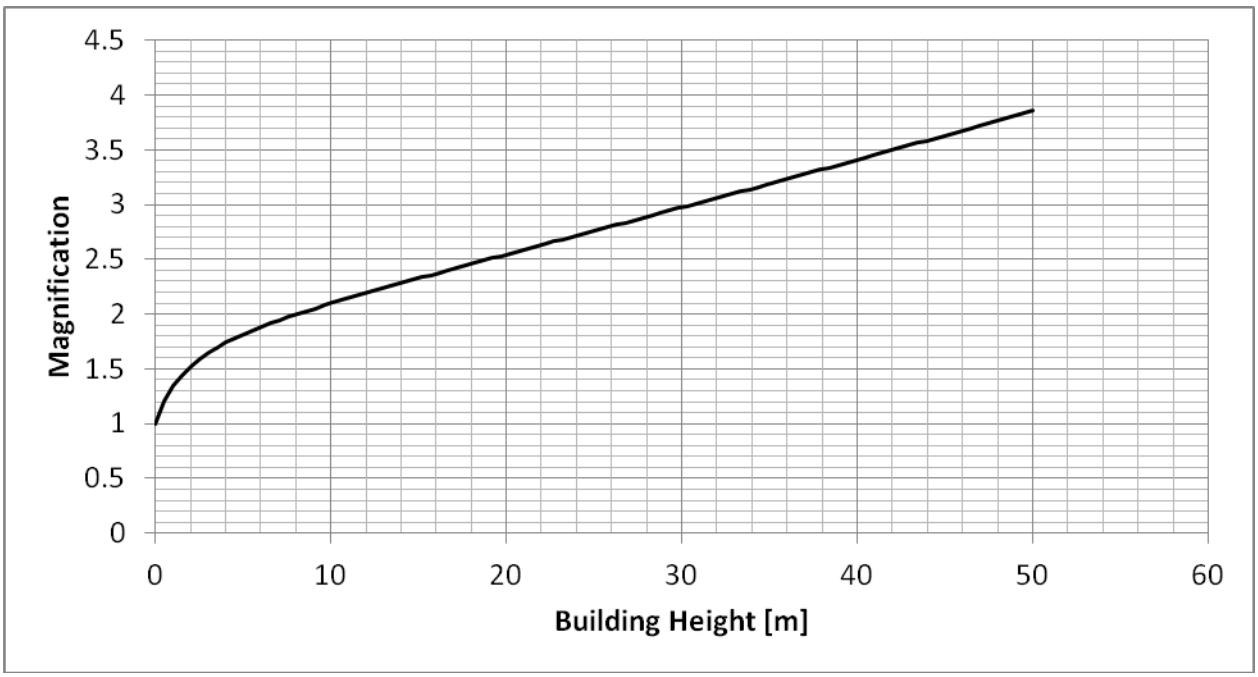



**Figure 41: Sensitivity of magnification (ratio of concentration with buildings to that with no buildings) to the area-weighted building height. The street width is 30 m.**

Figure 42 shows the diurnal variation of the VDM-estimated $PM_{2.5}$ concentration for a typical urban area. These results can be incorporated into exposure estimates by city planners. They can, for example, be combined with time-activity patterns and building indoor/outdoor ratios to estimate exposure to local traffic emissions for residents who live in the TOD.

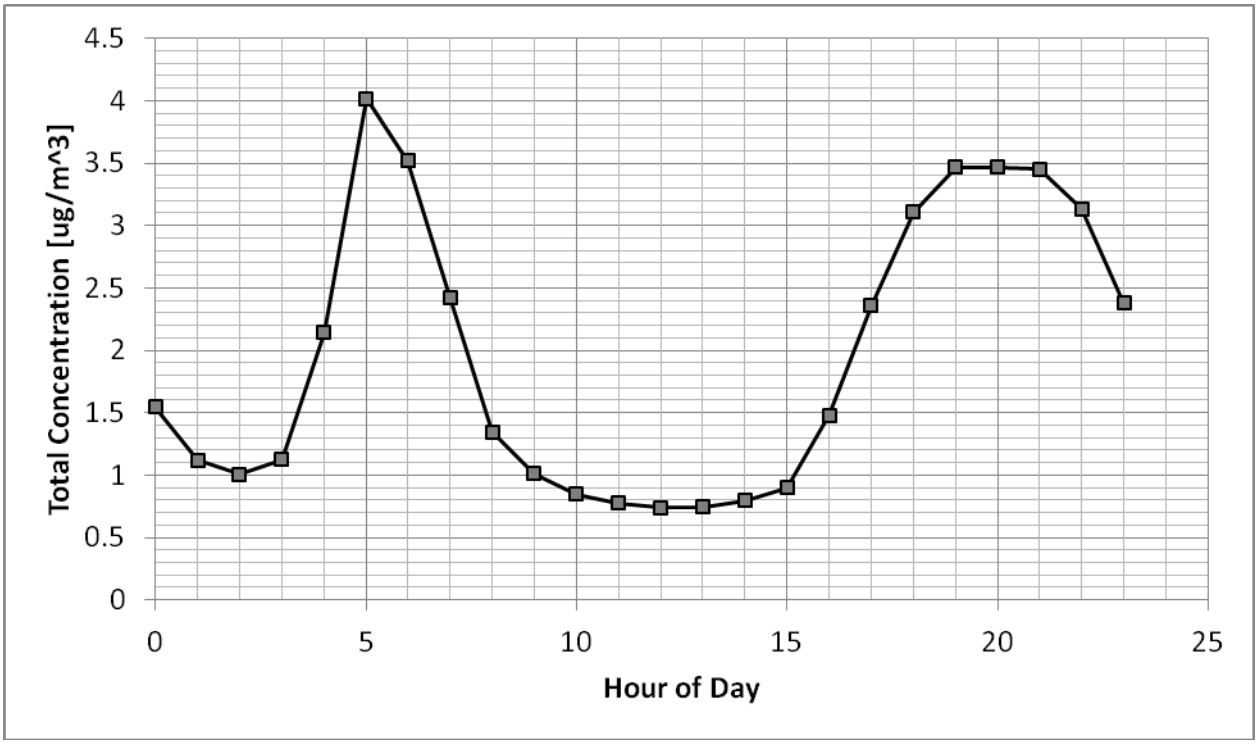



**Figure 42: Variation of PM2.5 concentration with hour of day predicted by the VDM spreadsheet. The area-weighted building height is 28 m and street width is 30 m. Traffic is typical of "arterial" roads.**

#### *vi.4.2.7. Implications to pedestrian exposure and transit-oriented development*

The VDM indicates two general strategies that can be used to mitigate exposure to elevated concentrations of traffic emissions. First, methods can be implemented that limit the effective street aspect ratio; the ratio of the area-weighted building height to street width. The expected result of reducing the aspect ratio is a reduction in exposure to air pollution concentration as indicated in Figure 41. To reduce the aspect ratio, planners can set limits on building heights, require that buildings be spaced apart or include open areas in the TOD design, or increase the street width.

Second, the local traffic within the TOD can be limited to reduce traffic emissions and exposure to air pollution concentrations. Air pollution concentrations are directly proportional to traffic emission rate and so a given reduction in traffic corresponds to an equal reduction in air pollution concentration. The implementation of traffic management strategies can be designed by the planner based on the expected air pollution concentrations calculated by the VDM. Traffic management can also be combined with changes to building parameters that limit the effective aspect ratio.

Finally, conversion of streets within TODs into pedestrian zones completely removes the impact of local traffic on exposure to air pollution concentrations and may provide significant reductions in total exposures for TOD residents.

These strategies must be implemented by planners based on knowledge of the expected urban background air pollution concentrations. The magnitude of the reduction in concentrations of local traffic emissions that is expected due to implementation of mitigation strategies should be considered in context of the air pollution contribution from background sources.

## vii. Conclusions

California is on a path to transition towards more sustainable communities, and state, regional, and local governments are making important investments to support the future transportation needs of the growing population, including integrated transportation and land use planning, as well as providing accessibility to sustainable transportation choices and transit-oriented developments. However, these developments may also put public closer to traffic air pollution corridors, including heavily trafficked urban cores. Therefore, it is important to study and understand the potential air quality exposures experienced in these urban cores, and identify potential strategies in built-environment design, land use planning, and traffic management that can help reduce public exposure to traffic air pollutants in the transit-oriented developments.

Urban environments are very complex, especially the heterogeneous built environments typical of California. We have shown, however, that it is possible to isolate the impact of the many aspects of heterogeneous fleets, urban driving modes, and the built environment on air quality in these environments. Depending on the spatial and temporal scale that is relevant to the question being asked, different features of the urban environment play a larger or smaller role.

The project utilized two distinct data collection and analysis approaches to evaluate the design and operational considerations in Transit Oriented Developments using comprehensive air quality measurement studies in Los Angeles County, CA. One team collected and analyzed high frequency mobile measurements using statistical analysis and modeling. The other team collected comprehensive dataset using stationary air pollution and meteorological monitors to develop a dispersion model that can be used to estimate the impact of urban morphology on air pollution associated with traffic emissions.

The study highlights that although direct emissions from the fleet are of primary importance and dominate the concentrations at the 1-second time scale in micro dynamics models, built environment factors play a much larger role when averaged over longer time (minutes) and length scales (tens of meters). The results of this study can inform the design of transit-oriented developments to reduce exposure to traffic emissions.

Our research findings have important implications for urban planning, traffic management policies and air pollution exposure of pedestrians and vehicle occupants in urban centers. Broadly, our data provide quantitative insights into how the built environment and traffic flows influence pedestrian exposure to vehicle pollution, and offer urban planners and traffic managers strategies to reduce street-level pollutant concentrations.

Our findings that lower traffic flows and fewer stops reduce near-roadway pollution are consistent with earlier studies and shed light on the potentials of traffic management policies that may be employed to mitigate pedestrian exposure. In addition to the obvious dependence on traffic volumes, different built environment characteristics are important at different times of day, because atmospheric stability varies with time of day.

For calm mornings, the study found that the area aspect ratio ($Ar_{area}$) is an important factor in controlling the block-scale vehicular pollutant concentrations. Higher values of $Ar_{area}$, correspond to more building volume and less open space, and limited mixing with the above ambient air. On the other hand, turbulence intensities played a major role in dispersing vehicular emissions in the afternoon. The built environment affects surface turbulence intensities, enhances vertical mixing and thus plays an indirect role in controlling block-scale [UFP concentration]. An isolated tall building surrounded by open space or short buildings is likely help to reduce the levels of vehicular pollution by up to a factor of two. Thus, urban planning focused on decreasing $Ar_{area}$ and increasing

heterogeneity of building distributions is expected to substantially improve near-roadway air quality and reduce pedestrian exposure to vehicular emissions.

In general, the project suggests several broad mitigation methods to reduce exposures to pollutant concentrations:

1) Avoid creating street canyons by placing tall buildings next to one another, or reduce emissions most where building aspect ratios are large. Emissions can be reduced by reducing traffic flow or by regulating traffic in built up areas to avoid acceleration of vehicles, which produces high emissions.
2) Including open spaces among the buildings and building heterogeneity, not only in the center of the TOD but also within several surrounding blocks will also reduce exposures.
3) Separate pedestrian and heavy vehicle traffic into different streets.
4) Reduce traffic during early morning and late evening hours when turbulence levels are low.

Our results and recommendations on TOD design are consistent with the other studies on the impact of the built environment on air pollution (Klein et al., 2007: Driving physical mechanisms of flow and dispersion in urban canopies. International Journal of Climatology, 27, 1887-1907, P. Klein, B. Leitl, and M. Schatzmann; Yuan et al., 2014: Improving air quality in high-density cities by understanding the relationship between air pollutant dispersion and urban morphologies. Building and Environment, 71, 2014, 245-258. C. Yuan, E. Ng, and L.K. Norford). These studies also show that dispersion of vehicle emissions is governed by building aspect ratio and turbulence levels. We have contributed to advancing the state-of-the-science by developing methods that provide quantitative estimates of air pollution levels using these variables as inputs.

## vii.1. General Recommendations for TOD Design

Table 17 below summarizes the overall size of the effects of different features of the built environment investigated in this report.

### vii.1.1. Built Environment Considerations

#### *vii.1.1.1. Single street scale*

Analysis of data collected in Hannover and in the field studies conducted in this project show that near-road concentrations of vehicle related pollutants are governed by the ratio of frontal-area weighted building height to street width and the vertical average of the standard deviation of vertical velocity fluctuations primarily. These relationships are summarized in a dispersion model referred to as the Vertical Dispersion Model (VDM). We translated the VDM equations into an easy to use spreadsheet tool and python code that allows city planners to use VDM to conduct sensitivity analysis, generate concentration estimates, and develop mitigation strategies. The model provides understanding on the roles of the following features in governing air quality in built environments. This tool is made available through the California Air Resources Board.

<u>Effective aspect ratio:</u> Analysis using the VDM dispersion model indicates that the effective aspect ratio, $a_r$, plays a major role in determining the effect of buildings on the impact of vehicle emissions on street level concentrations. The aspect ratio is defined by the equations:

$$a_r = \frac{H}{W} \; ; H = \frac{\sum_i A_i}{2L}$$

where $W$ is the width of the street, $A_i$ is the area of a buildings facing the street on *both* sides projected on a vertical plane, and $L$ is the length of the block. Thus, the effective height, $H$, of buildings on a street lined with buildings of uniform height with little space between them will be close to the actual height of buildings. On the other hand, $H$ of a street lined with buildings with widely varying heights will weigh the buildings with the largest areas obstructing the flow across the street. Furthermore, open spaces between buildings, corresponding to zero frontal areas, will reduce the effective height and hence the aspect ratio, $a_r$.

Street Canyons: Dispersion analysis in urban areas suggests that vertical mixing governs near surface concentrations within the urban canopy, consistent with observations analyzed in (Hanna et al., 2014), which show that data from field studies conducted in Manhattan, NY, indicate rapid vertical mixing in the presence of buildings. In the study at Los Angeles, 8th St, 50 m tall buildings next to the 20 m wide street are associated with a 50% increase in the UFP concentration due to local emissions relative to the section of the road, which had no buildings directly adjacent, resulting in a 25% increase in the total concentration.

Building Heterogeneity: The dispersion model shows that the presence of buildings lining a street decreases dispersion of local vehicle emissions relative to open terrain, and thus increases street-level concentrations by an increment that is proportional to $(1 + a_r)$. The modeling results indicate that at the sites in Los Angeles where the field studies were conducted, the presence of buildings increases the concentration by a factor of 1.19 to 1.58 relative to the concentration that would be measured in the absence of buildings. This increase is related to the magnification of concentrations associated with local emissions: the presence of the buildings can potentially magnify street-level concentrations associated with local emissions by as much as a factor of 4 relative to those in the absence of buildings. This implies that built-up areas with large aspect ratios create local hot spots in the presence of local emissions.

#### *vii.1.1.2. Multi-block scale*

The built environment at the multi-block scale has a markedly larger influence than it does at the street scale, and further approaching TOD design from the point of view of several blocks rather than a single street or intersection offers more options to design for lower exposure in TODs. Concentrations are generally higher in the morning, a time when our results showed lower building volumes and/or interspersed open space can result in substantially lower pollutant concentrations. In the afternoons, more building heterogeneity is helpful. Thus, avoiding street canyons and designing heterogeneous built environments that include open space within a block or two of busy roadways or intersections may be the best overall approach. While our research at the multi-block scale is novel, it would benefit from additional study to verify and expand on these important findings.

### vii.1.2. Street design and land use considerations

Intersection design: Analysis of mean cross-intersection [UFP] profiles suggests larger variability at and around the intersection peak locations than at distances farther away from the intersection. While comparing the concentration distributions profiles around traffic intersections, the overall [UFP] peak concentration was recorded at 10 m before the center of intersection. This implies that under some conditions (e.g., when traffic signal changes), pedestrian can possibly be exposed to more intensive and frequent excessive vehicular emissions near the intersections than mid-blocks.

Since the concentrations varied widely by sites as well as by traffic directions at the same site, the traffic patterns were related to the magnitude of UFP elevations at the peak locations. It was found that are UFP elevations at the intersections were affected by several important factors, including the traffic flow rates per traffic-signal cycle (vehicles·min$^{-1}$), the vehicle number waiting for green light at the moment the signal changed from red to green (queue length; Q-length), and the queue length

considering the proportion of high emitting vehicles (HEV; defined as heavy and medium duty diesel vehicles in this study). This is reasonable considering that heavy-duty diesel trucks emit much more UFP than gasoline-powered vehicles and a bigger group of vehicles accelerating simultaneously would emit more UFP.

Transit Stop Siting: The study analyzed the UFP concentrations experienced by transit users depending on the location of transit stops with respect to traffic intersections. The results suggest that pedestrian experiences more exposure when a bus-stop is located 20 m away from an intersection, when compared to 40 m distance. The total exposure levels were $1.4\times10^{7}$ particles$\cdot cm^{-3}$ vs. $2.0 \times 10^{7}$ particles$\cdot cm^{-3}$ at the 40 m and 20 m bus stop location, respectively. This corresponds to 38% reduction in UFP exposure when the bus stop is located at 40 m from the intersection. This result indicates that locating bus stops around 40 m and farther away from the center of intersections reduces transit user's exposure to intersection emission events.

### vii.1.3. Other features of the built environment and other topics for future research

Several other aspects of urban design not investigated in this study clearly also have the potential to impact exposures of users in complex urban environments. These include street configurations such as "complete streets", lane configurations (one way, configurations that reduce or create more stop/starts and accelerations), mode shifting, proximity to other major roadways, airports, freight movements, traffic management strategies and placement of vegetation. While some of these topics have been investigated to a sufficient level that the magnitude of the effects are understood fairly well, the potential gains from several of them are poorly understood and in need of additional research.

**Table 17. General recommendations to reduce pedestrian and residential air pollution exposure in built environment.**

| Management | Suggested Direction | Approx. Size of Effect | Atmospheric Conditions & Notes |
|---|---|---|---|
| Areal aspect ratio ($Ar_{area}$), which combines building area-weighted height, building footprint, and the amount of open space | Lower building volumes and more open space result in lower pollutant concentrations. | The difference between very dense and low density built environments is approximately a factor of three. | Important under calm conditions (in the mornings at our sites). |
| Building Heterogeneity | Isolated tall (high-rise) buildings result in lower concentrations than homogeneous shorter or many taller buildings with similar volume. | Highly heterogeneous built environments can decrease concentrations by up to approximately a factor of two relative to completely homogeneous built environments. | Important under unstable conditions with moderate winds (afternoons at our sites). Not critical when the atmosphere is stable. |
| Street Canyons (relatively contiguous walls of buildings) | Heterogeneous building forms avoid hotspots created by street canyons. | Tall street canyons (~50 m) can increase local traffic air pollution concentrations by up to about 50% relative to open space. | |
| Bus Stop Siting | Siting bus stops further from intersection will reduce exposures. | From no effect to more than a factor of two reduction from moving the site from 20 to 40 meters from the intersection on the "far" side. | Pollutant concentrations usually peak near the center of the intersection, although there is a high degree of variability. |

# viii. Acknowledgement and References

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## viii.2. References

### viii.2.1. References for Section II

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# ix. Appendix: Supporting information for Section vi.4

***Los Angeles Field Measurements.*** Los Angeles - 8th St, April 23 - May 13 2014 measurements were made near the 8th and Hill St intersection in Los Angeles between April 23 and May 13, 2014. An overview of the experiment is shown in Figure A1. The site was chosen because 8th St has a section where there are no buildings next to the road, the "parking lot" site, and a section where there are tall buildings directly next to the road, the "building" site. This design helps remove the effect of emission variation: because the open and building sites have very similar traffic we can directly compare the concentrations at the site to determine the effect of the buildings at the "building" section on the concentration. Sonic anemometers were placed at an upwind rural location near LAX airport, at a rooftop location on the 50 m tall Union Lofts building, located at the 8th and Hill St intersection, at the parking lot and building sites, and at the mid-section between the two sites.

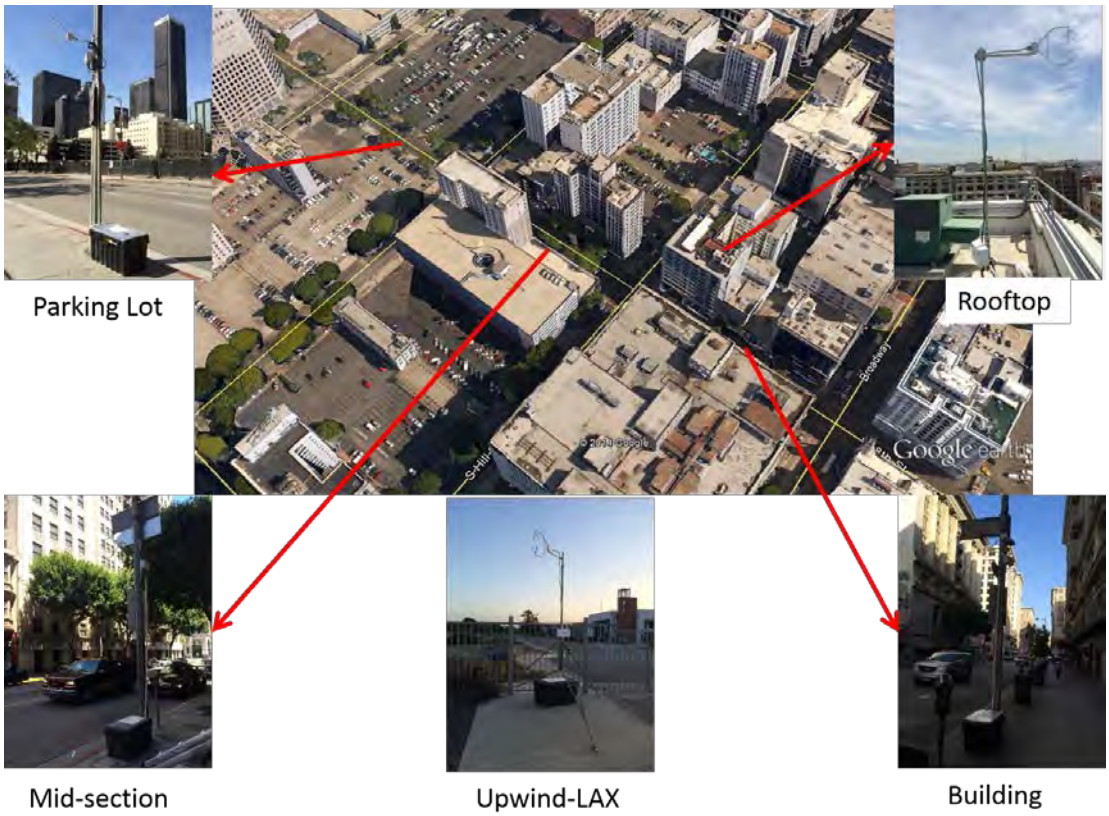



**Figure A1: Overview of 8th St field study.**

The upwind rural sonic anemometer was mounted on a tripod at 3.15 m above ground level (AGL). The bottom center picture in Figure A1 shows this anemometer, looking downwind. There were no buildings upwind of the sonic essentially all the way to the Pacific Ocean several kilometers to the west. The street level sonic anemometers were mounted to light poles next to 8th St at about 4 m AGL (Figure A1). See Figure A4 and Table A1 for detailed instrument locations. The anemometers were attached to 2 foot beams attached to the poles and oriented with the sensor pointed toward the street, except for the sonic next to the parking lot, which was oriented southwest because the predominant wind direction is southwesterly. We attempted to place the anemometers away from major obstructions. However, 8th St has several large trees next to the road, and the instrument at location 1 (Table A1) was placed about 10 m downwind of a tree, and instrument location 2 was at a section of road where there were trees upwind and downwind. Locations 3 and 4, the locations of the primary sonic anemometer measurements, were far from any trees or other obstacles.

**Figure A2: Sonic anemometer and AQMesh monitor mounted to light pole next to 8th St.**

Condensation particle counters were placed at the locations shown in Figure A4. The CPCs were placed on May 7th and May 9th between about 8:00 and 18:00 PDT, but no rooftop CPC (number 360) was used on May 7th because the instrument was not available. The CPCs were co-located (Figure A3) for 30 minutes before the start of measurements on both May 7th and 9th to derive inter-instrument calibration factors used to adjust the data. The results of the calibration are shown in Appendix B.

**Figure A3: Co-location of CPCs before start of measurements on 8th St.**

Traffic data for 8th St was obtained from automatic traffic detectors run by city of LA. The traffic was recorded at the 8th St and Olive St intersection located midway between the open and building sites.

**Figure A4: Instrument locations on 8th St. See Table A1 for symbol definitions.**

**Table A1: Detailed instrument locations on 8th St.**

| ID | Instrument | Location | Latitude | Longitude | Heading (° from north) | Height (inches) | Dates (month/day/year) |
|---|---|---|---|---|---|---|---|
| 1 | Sonic 0984 | Building Region | 34.04413 | --118.25533 | 19 | 146 | 4/23-5/1 |
| 2 | Sonic 0984 | Middle region | 34.04482 | -118.25603 | 190 | 157 | 5/1-5/13 |
| 3 | Sonic 0245 | Building region | 34.04403 | -118.25541 | 204 | 160 | 4/23-5/13 |
| 4 | Sonic 1055 | Parking lot | 34.04523 | -118.25713 | 37 | 155 | 4/22-5/13 |
| 5 | Sonic 2564 | Upwind LAX airport | 33.95494 | -118.40472 | 36 | 124 | 5/7-5/13 |
| 6 | Sonic | 8th St. roof of union lofts | 34.044285 | -118.255326 | 18 | 95 | 5/1-5/13 |
| 1 | AQMesh 82150 | Building region | | | | 152 | 5/1-5/9 |
| 3 | AQMesh 89150 | | | | | 166 | 5/1-5/9 |
| A | CPC 483 | Near sonic 0245 | 34.04403 | -118.25541 | | 59 | 5/7-5/9 |
| B | CPC 498 | ACross street from sonic 0245 | 34.044213 | -118.255361 | | 59 | 5/7-5/9 |
| C | CPC 494 | Near sonic 1055 | 34.045406 | -118.25738 | | 59 | 5/7 and 5/9 |
| D | CPC 502 | ACross street from sonic 1055 | 34.045506 | -118.257285 | | 59 | 5/7 and 5/9 |
| E | CPC 360 | Roof of union lofts building | 34.044398 | -118.255464 | | 59 | 5/9 |

***Los Angeles - 7th St and Broadway, September 20, 2013.*** Figure A5 shows an overhead view of the locations of measurements made in Los Angeles on September 20. Table A2 shows the detailed locations of the instruments. Four sonic anemometers were mounted on tripods at several locations: one on a tripod about 3 m above the top of the 55 m tall Van Nuys apartment building next to 7th St, one at 2.4 m AGL on the southwest side of 7th St, one at 2.4 m AGL on the northwest side of Broadway, and one at 2.4 m AGL at a park, Rancho Cienega Recreation center, approximately 10 km west (upwind) of the 7th St site. The upwind sonic was placed as far from buildings and trees as possible on a flat baseball field. The buildings surrounding the park were 1 or 2 stories tall. The rooftop sonic was placed on a section of the rooftop about 10 m higher than the rest of the roof near the edge of the building (Figure A6). A small structure stands about 7 m to the east of the sonic.

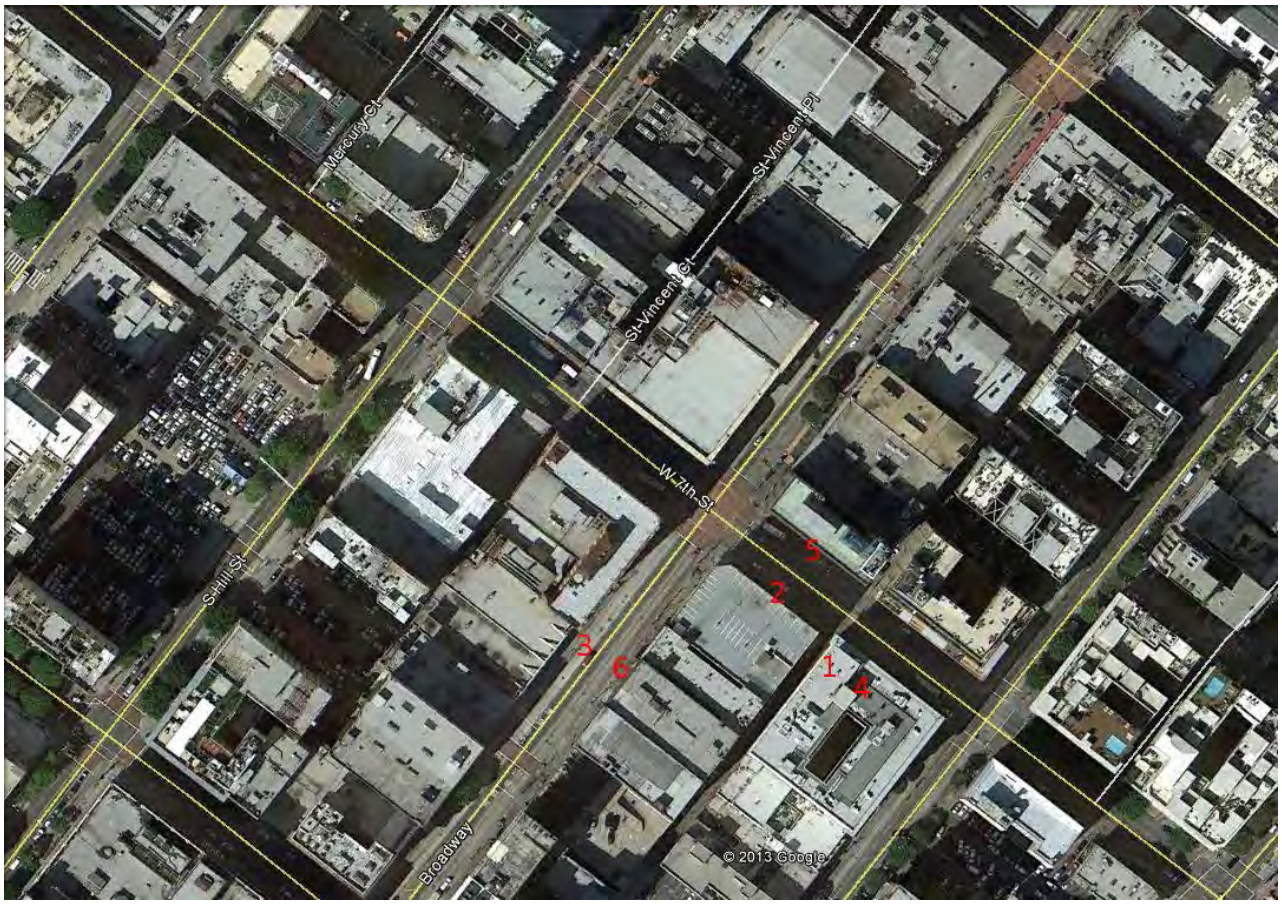



**Figure A5: Instrument locations at 7th St and Broadway field site. See Table 6 for symbol definitions.**

**Table A2: Detailed instrument locations at 7th St and Broadway.**

| ID | Instrument | Location | Latitude | Longitude | Heading (° from north) | Height |
|---|---|---|---|---|---|---|
| 1 | Sonic | Roof | 34.044645 | -118.25297 | 83 | 57.4 |
| 2 | Sonic | 7th St | 34.04495 | -118.253129 | 210 | 2.4 |
| 3 | Sonic | Broadway | 34.044639 | -118.253956 | 322 | 2.4 |
| 2 | DiscMini 2 | 7th St South | 34.044888 | -118.253035 | | 1 |
| 3 | CPC 483 | Broadway west | 34.044719 | -118.253955 | | 1 |
| 4 | CPC 481 | Roof | 34.044623 | -118.252775 | | 56 |
| 5 | DiscMini 1 | 7th St North | 34.045004 | -118.252914 | | 1 |
| 6 | CPC 360 | Broadway East | 34.044634 | -118.253786 | | 1 |
| | Sonic | Upwind Park | 34.023285 | -118.35091 | 75 | 2.4 |

**Figure A6: Location of rooftop sonic anemometer near the edge of a section of roof about 10 m above the rest of the roof. A small structure sites about 7 m to the east of the sonic.**

Three TSI 3022A CPCs and two Matter Aerosol DiSCminis were used to measure concentrations of ultrafine particle number. Two CPCs were placed on opposite sides of Broadway, and one on top of the 55 m tall Van Nuys apartment building. The two DiSCminis were placed on opposite sides of 7th St. The CPCs and DiSCminis were calibrated relative to each other by co-locating the instruments for about 30 minutes and adjusting the data using the resulting calibration factors. Appendix B shows the calibration plots along with the regression coefficients for each instrument.
Traffic at the 7th St and Broadway intersection was recorded using a camera attached to a tripod on the roof of the Van Nuys building. Additionally, traffic data was obtained from the city of LA's automatic traffic counting system. The traffic data for 7th St was obtained from detectors located at 7th St and San Pedro intersection, and for Broadway from detectors at Broadway and Pico St. The manual and automatic counts were compared, and the automatic counts were adjusted to match the magnitude of the manual counts.

***Temple City - Las Tunas Dr and Temple City Blvd.*** This site is characterized by fairly uniform single story buildings. Measurements of particle concentrations and turbulence were made between January 13 and February 13, 2014 in Temple City at the Las Tunas Dr and Temple City Blvd intersection. Figure A7 shows the locations of the CPCs and sonic anemometers at the site. One anemometer was placed on the roof of a 6.1 m tall building near the intersection and another was mounted to a light post at the south side of the street near mid-block. An additional sonic was placed at a rural upwind site at Rose Hill Park. CPCs were placed at the four corners of the block and on both sides of the street near mid-block on January 15, 16, and 17. The CPCs were co-located to derive calibration factors. Results are given in Appendix B. Traffic was counted using a video camera near mid-block. Table A3 gives a summary of the instrument locations.

**Figure A7: Temple City experiment site. For marker definitions see Table 7.**

**Table A3: Detailed instrument locations at Temple City site.**

| ID | Instrument | Location | Latitude | Longitude | Heading (° from north) | Height | Dates (month/day/year) |
|---|---|---|---|---|---|---|---|
| 1 | Sonic | South Center | 34.106693 | -118.060151 | 88 | 2.4 | 1/13/14-2/13/14 |
| 2 | Sonic | Roof | 34.1069 | -118.060712 | 354 | 8.4 | 1/13/14-2/13/14 |
| 1 | CPC 502 | South Center | 34.106693 | -118.060151 | 322 | 1 | 1/15/14,1/16/14,1/17/14 |
| 2 | CPC 498 | Roof | 34.106874 | -118.060758 | | 7 | 1/15/14,1/16/14,1/17/14 |
| 6 | CPC 499 | South West | 34.10662 | -118.060669 | | 1 | 1/15/14,1/16/14,1/17/14 |
| 7 | CPC 480 | South East | 34.106761 | -118.059658 | | 1 | 1/15/14,1/16/14,1/17/14 |
| 8 | CPC 497 | North East | 34.106975 | -118.059708 | | 1 | 1/15/14,1/16/14,1/17/14 |
| 9 | CPC 494 | North Center | 34.106903 | -118.060259 | | 1 | 1/15/14,1/16/14,1/17/14 |
| 10 | CPC 483 | North West | 34.106834 | -118.060711 | 75 | 1 | 1/15/14,1/16/14,1/17/14 |

***Beverly Hills - Wilshire Blvd.*** The measurements on Wilshire Blvd, Beverly Hills were similar to those made on 8th St, except no rooftop measurements were made. Figure A8 shows an aerial view of the site, showing CPC locations near the “parking lot” and “building” locations, which have only short buildings and 50 m tall buildings directly next to the road, respectively. Sonic anemometers were placed next to the street at the "parking lot" and "building" locations, and at the same upwind LAX site used in the Los Angeles 8th St field study. Four CPCs were placed at the location shown in Figure A8. Table A4 gives details of the instrument locations.

**Figure A8: View of Wilshire Blvd field site. For marker definitions see Table 8.**

**Table A4: Detailed instrument locations at Beverly Hills site.**

| ID | Instrument | Location | Latitude | Longitude | Heading (° from north) | Height | Dates (month/day/year) |
|---|---|---|---|---|---|---|---|
| 1 | Sonic 0245 | Building Region North | 34.067162 | -118.390488 | 50 | 4.14 | 5/19/14-7/1/14 |
| 3 | Sonic 0984 | Open Region South | 34.06696 | -118.3923 | 95 | 4.06 | 5/19/14-7/1/14 |
| 1 | CPC 494 | Building Region North | 34.067162 | -118.390488 | | 1 | 5/30/14 |
| 2 | CPC 483 | Building Region South | 34.066954 | -118.390392 | | 1 | 5/30/14 |
| 3 | CPC 498 | Open Region South | 34.06696 | -118.3923 | | 1 | 5/30/14 |
| 4 | CPC 502 | Open Region North | 34.067162 | -118.392417 | | 1 | 5/30/14 |
| | Sonic 1055 | Upwind near LAX | 33.95494 | -118.40472 | | 3.15 | 5/19/14-7/1/14 |

***Calibration of Condensation Particle Counters.*** On each day that measurements were made, prior to locating the TSI3022A condensation particle counters at field locations, the instruments were co-located at one location next to the street. The data collected during the co-location was used to derive inter-instrument calibration factors that were used to adjust the concentrations measured during the experiments.

Figure B1 shows scatter plots comparing concentrations measured by three of the CPCs at the 8th St field site on May 7th, 2014 with one CPC used as the reference. Figure B2 shows a similar comparison for May 9th, 2014. Figure B3 shows scatter plots comparing concentrations measured by the four CPCs and one DiscMini at the Broadway field site on September 20, 2013. Figure B4 shows the calibration plots for the CPCs used at the Temple City field site on January 16, 2014. Calibration plots for the CPCs used at the Wilshire Blvd site are shown in Figure B5.

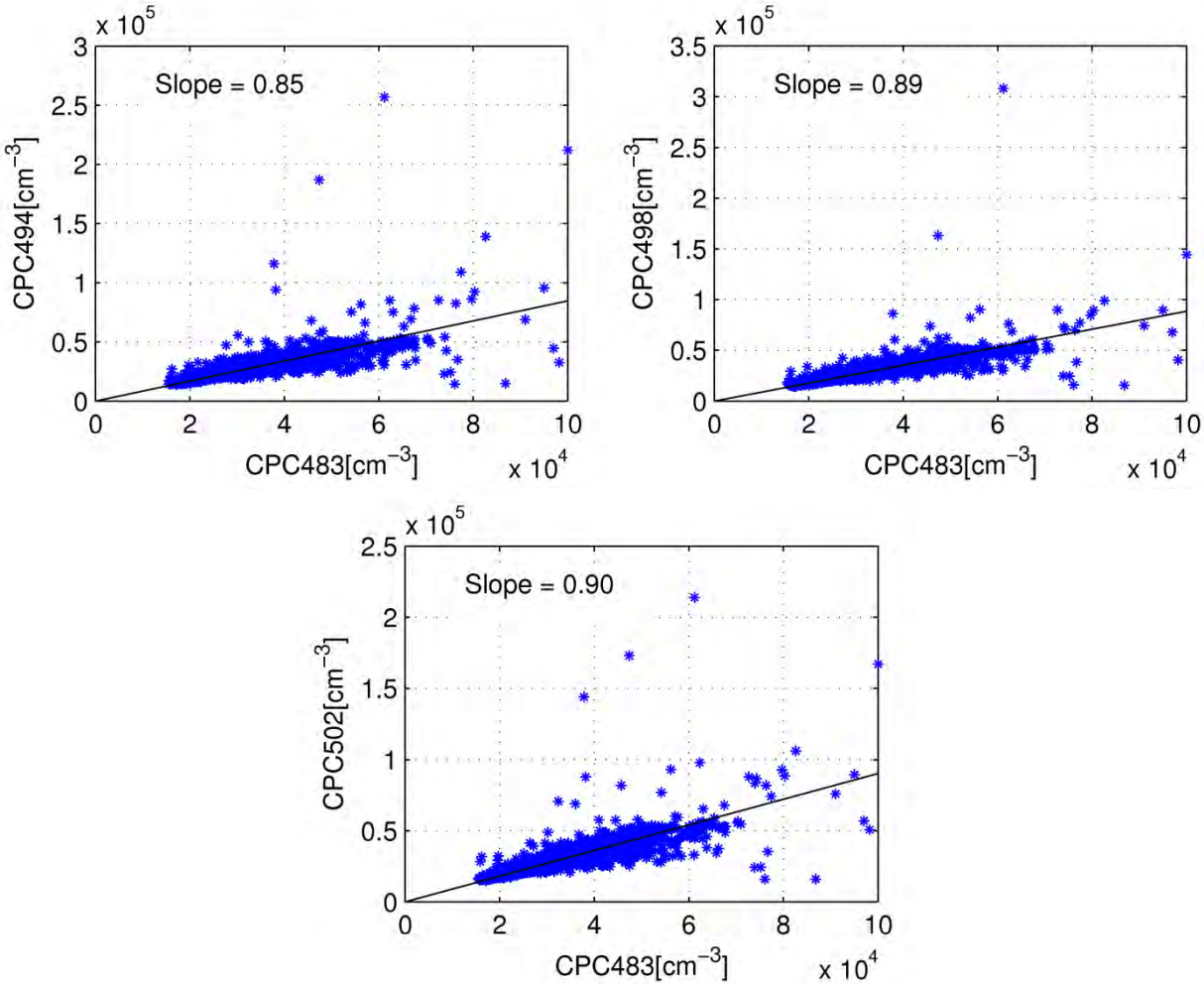



**Figure B1: Calibration derived from co-location of instruments next to 8th St on May 7th.**

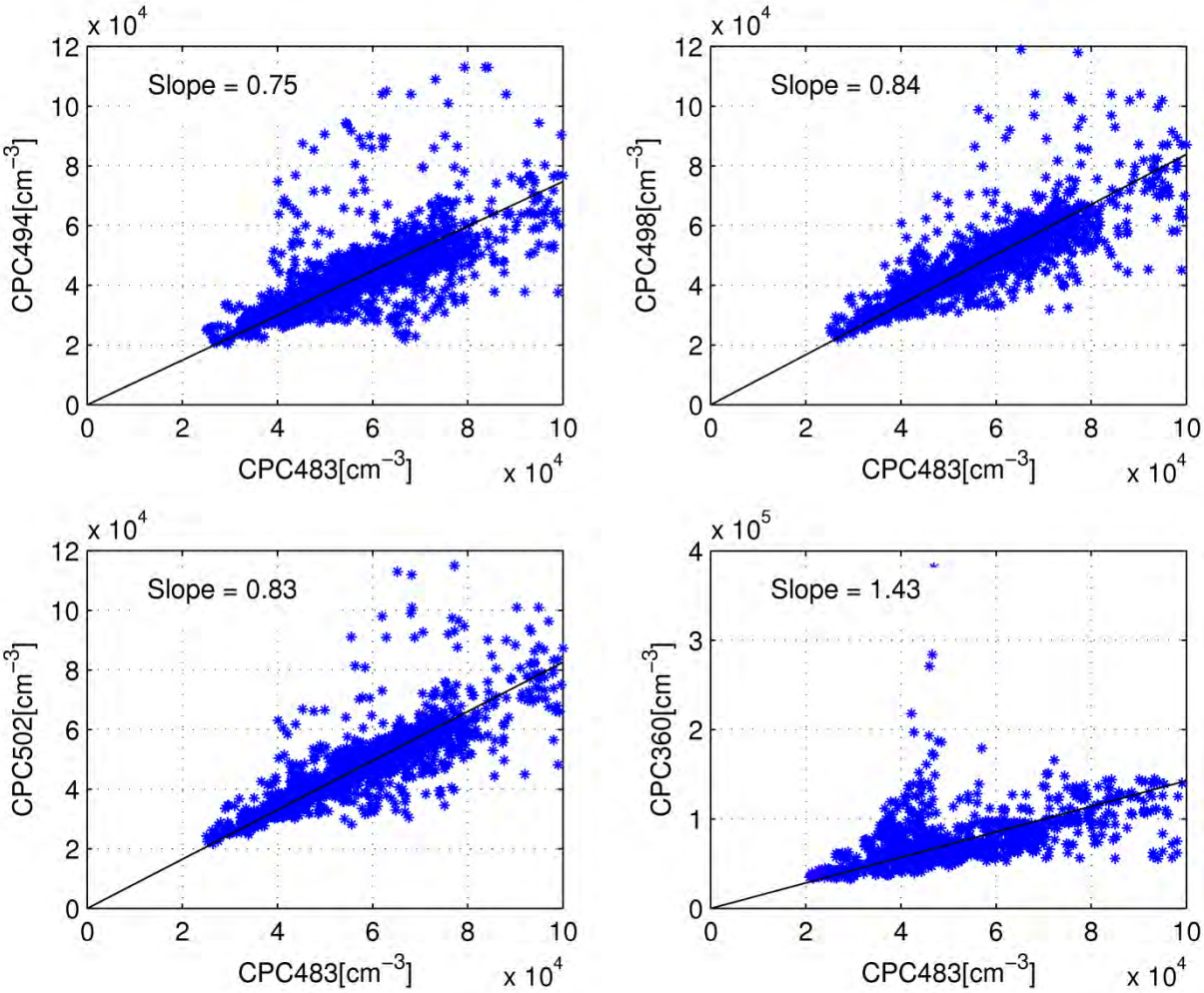



**Figure B2: Calibration derived from co-location of instruments next to 8th St on May 9th.**

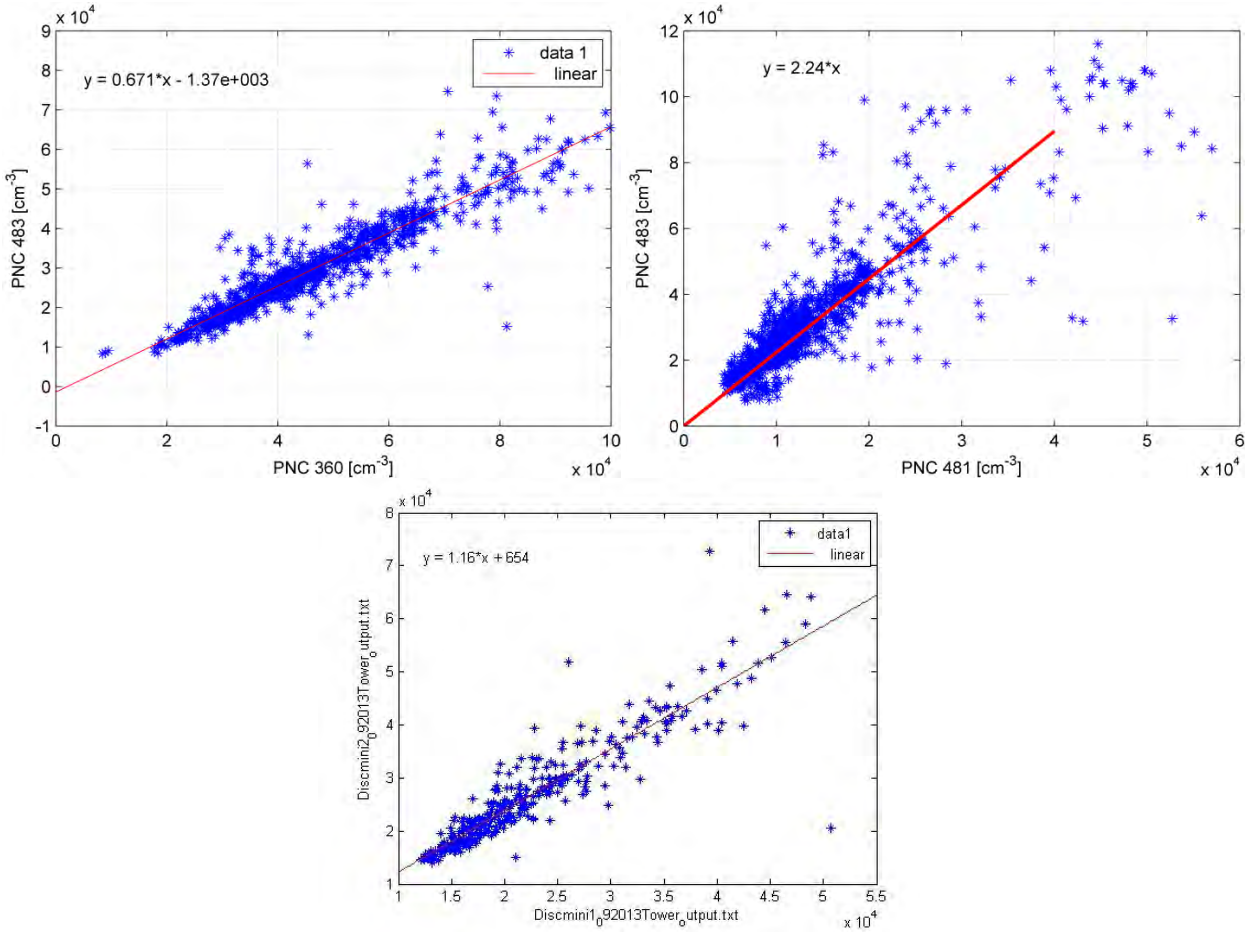
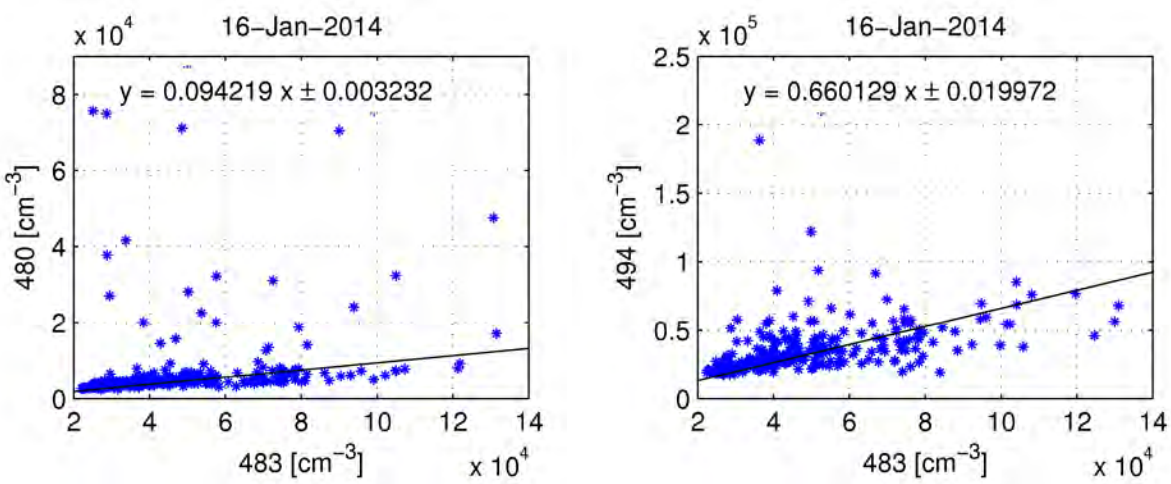



**Figure B3: Calibration derived from co-location of instruments next to Broadway on September 20th. The calibration factor between DiSCmini 2 and CPC 483 was 1.34 (not shown).**



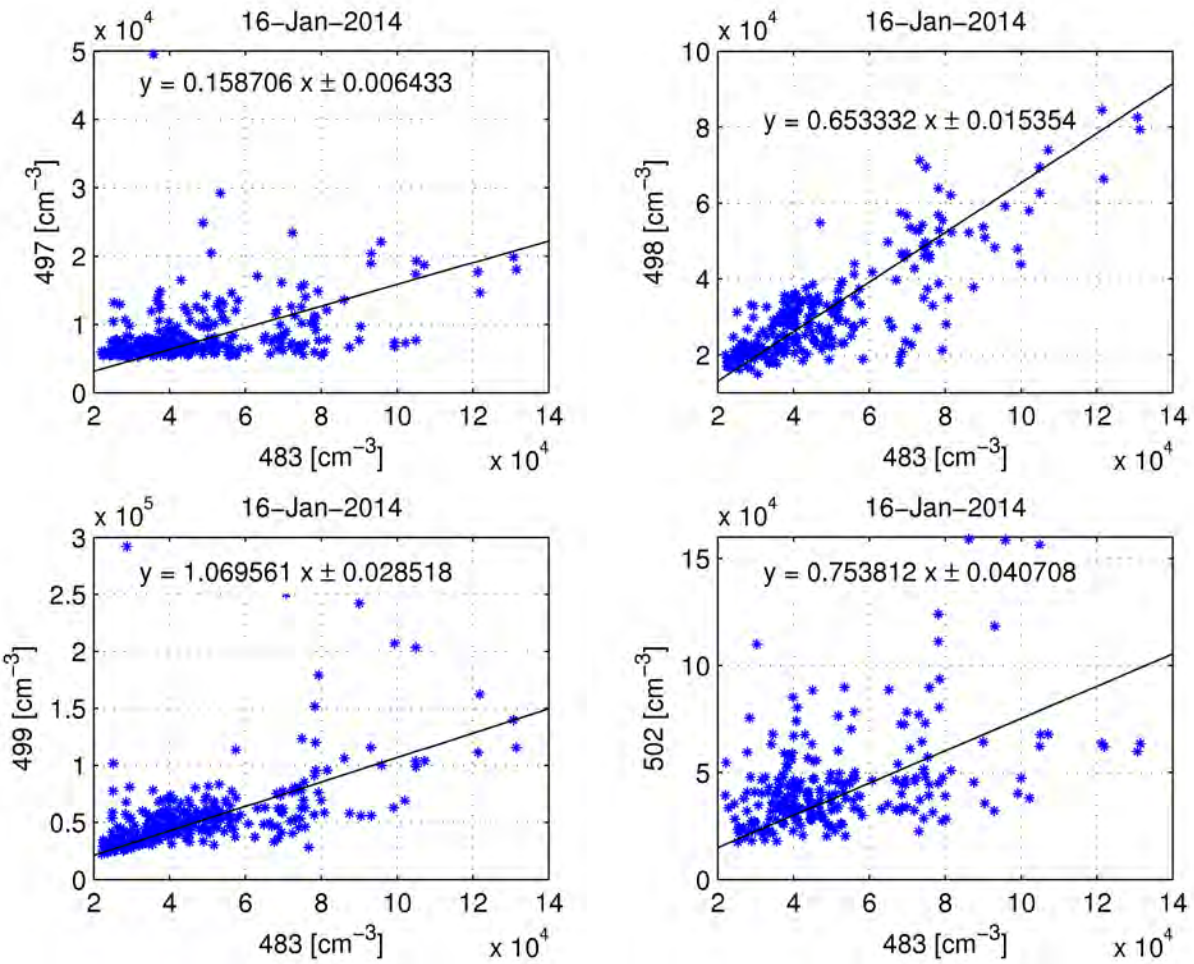
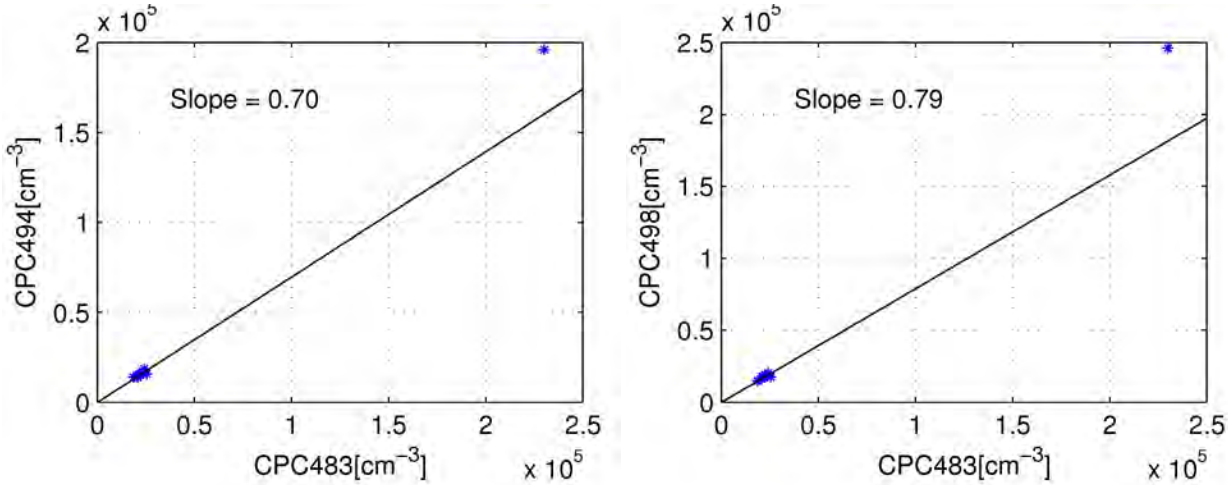



**Figure B4: Calibration derived from co-location of instruments next to Temple City Blvd on January 16, 2014.**



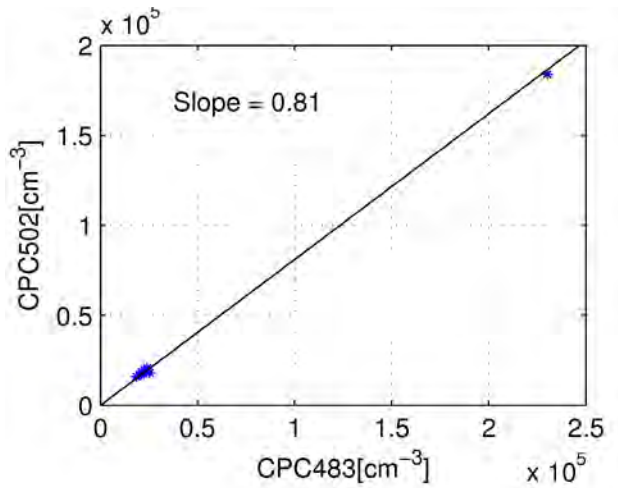



**Figure B5: Calibration derived from co-location of instruments next to Wilshire Blvd on May 30, 2014.**

# EXHIBIT 6

Union of Concerned Scientists

FACT SHEET

# Inequitable Exposure to Air Pollution from Vehicles in California

## *Who Bears the Burden?*

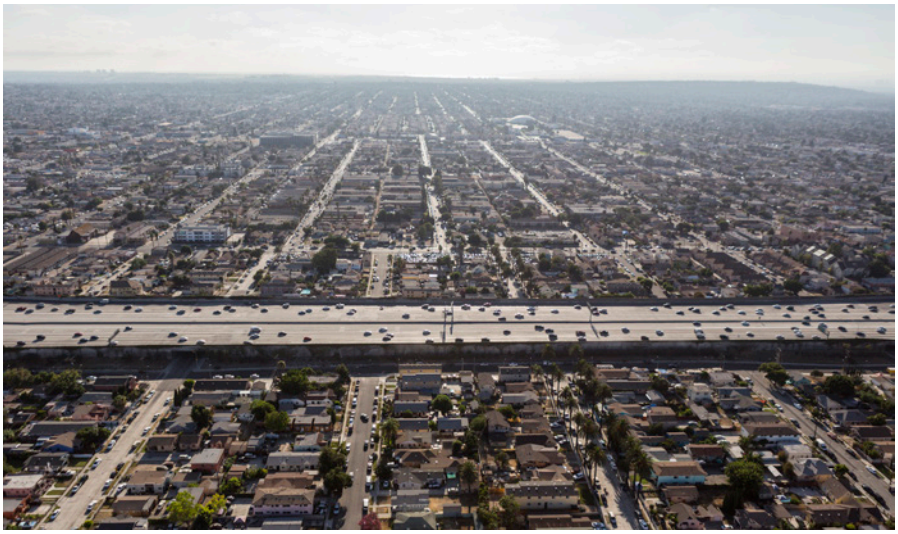
**HIGHLIGHTS**

*This analysis explores the significant contributions of cars, trucks, and buses to particulate matter air pollution in California and the disproportionate effects on communities of color and low-income communities. Advancing clean transportation policies—such as those that encourage vehicle electrification, cleaner fuels, and reduced driving—will help reduce air pollution emissions. Additionally, policies and investments should be evaluated for their ability to reduce the current inequities in exposure to vehicular air pollution borne by low-income Californians and communities of color. This report helps to inform such strategies.*

Transportation constitutes a significant source of both global warming emissions and air pollution in California. This analysis from the Union of Concerned Scientists (UCS) quantifies the formation of particulate matter (PM) air pollution from on-road vehicles and identifies the locations and populations most at risk regarding this pollution. The analysis measures the annual average concentration of particulate matter using a 2014 estimate of emissions as input data (EPA 2014). Research links exposure to particulate matter smaller than 2.5 micrometers in diameter ($PM_{2.5}$)—20 times smaller than even fine human hair—to increased illness and death, primarily from heart and lung diseases. These particles are small enough to penetrate deeply into the lungs, and the smallest particles can even enter into the bloodstream. The use of cars, trucks, and buses in California both directly produces $PM_{2.5}$ and also produces gases that lead to the formation of additional $PM_{2.5}$.

This analysis of particulate matter from cars, trucks, and buses in California finds the following:

- On average, African American, Latino, and Asian Californians are exposed to more $PM_{2.5}$ pollution from cars, trucks, and buses than white Californians. These groups are exposed to $PM_{2.5}$ pollution 43, 39, and 21 percent higher, respectively, than white Californians.
- Exposure to $PM_{2.5}$ from cars, trucks, and buses is not equally distributed across the state (Figure 1, p. 2). People living in Los Angeles County are exposed to 60 percent more vehicle pollution than the state average and 250 percent more than the San Francisco Bay Area.



trekandshoot/iStockphoto

*Millions of California residents live near major highways (such as Highway 110 in Los Angeles County, shown here) and are exposed to high levels of vehicular air pollution. African American and Latino Californians are disproportionately exposed to more of this pollution, as are low-income households.*

- The lowest-income households in the state live where $PM_{2.5}$ pollution is 10 percent higher than the state average, while those with the highest incomes live where $PM_{2.5}$ pollution is 13 percent below the state average.
- Californians living in households without a personal vehicle are also exposed to much higher levels of vehicle pollution than other households because they tend to live in urban areas surrounded by vehicle traffic.

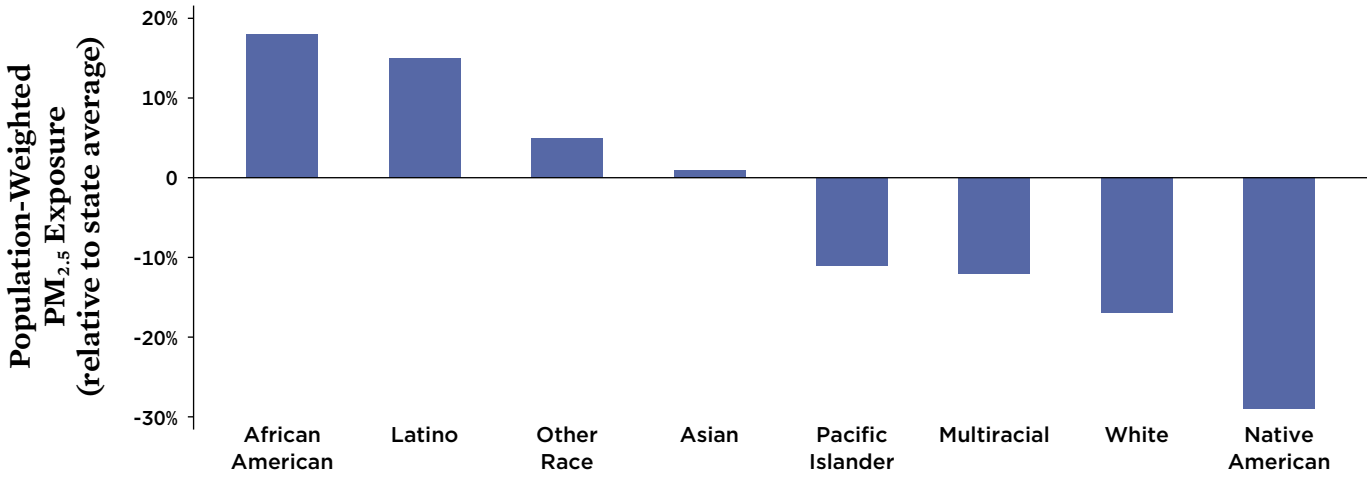
Cleaner technologies are now available to replace gasoline, diesel, and other combustion-powered[1] vehicles, making some of these local transportation emissions avoidable. By supporting strategies such as cleaner fuels, the reduction of miles driven, and the electrification of vehicles—and by targeting clean technology deployment to benefit the most affected communities—both air pollution and carbon emissions that cause climate change can be reduced, while addressing the inequity of $PM_{2.5}$ exposure.

## Why Is Particulate Matter Air Pollution a Problem?

Some $PM_{2.5}$ pollution forms directly during combustion, from sources such as fires, power plant emissions, and vehicle exhaust. Additional $PM_{2.5}$ comes from sources such as road and construction dust. However, much of the $PM_{2.5}$ forms indirectly through the reactions of pollutant gases in the atmosphere (Fine, Sioutas, and Solomon 2008). These gases include ammonium, nitrogen oxides, sulfur oxides, and volatile organic compounds. Most of these pollutants are emitted in vehicle exhaust, though volatile organic compounds also come from the evaporation of gasoline during refueling and from leaks in vehicles' fuel tanks and lines.

Exposure to $PM_{2.5}$ has significant negative health impacts; it has been estimated that fine particulate air pollution is responsible for the vast majority of the 3 million to 4 million annual deaths attributed to air pollution worldwide. While $PM_{2.5}$ is not the only air pollutant that adversely affects health, it is estimated to be responsible for approximately 95 percent of the global public health impacts from air pollution (Landrigan et al. 2018; Lelieveld et al. 2015). Both acute and chronic exposure to $PM_{2.5}$ have been linked to illness and death (Brook et al. 2010). Short-term exposure to elevated levels of $PM_{2.5}$ can exacerbate lung and heart ailments, cause asthma attacks, and lead to both increased hospitalizations and mortality from cardiovascular diseases (Orellano et al. 2017; Pope and Dockery 2006). Chronic exposure to $PM_{2.5}$ also causes increased death rates attributed to cardiovascular diseases, including heart attacks, and has been linked to other adverse impacts such as lung cancer (Fine, Sioutas, and

FIGURE 1. Disproportionately High Exposure for African Americans and Latinos in California




*African American and Latino Californians have 19 and 15 percent higher exposure to $PM_{2.5}$, respectively, than the state average, while white Californians are exposed to 17 percent lower concentrations.*

Note: The following US Census Bureau–defined racial groups were used in the analysis: White; Black or African American; American Indian or Alaska Native; Asian; Native Hawaiian or Other Pacific Islander; Hispanic; Latino; and Some Other Race. In the chart above, Latino includes census respondents who select Hispanic, Latino, or both; Other Race includes census respondents who select Some Other Race as their only race.

SOURCES: US CENSUS BUREAU 2018; EPA 2014.

> ***Fine particulate air pollution is responsible for the vast majority of the 3 million to 4 million annual deaths attributed to air pollution worldwide.***

Solomon 2008). Chronic exposure to $PM_{2.5}$ in children has also been linked to slowed lung-function growth, development of asthma, and other negative health impacts (ALA 2018a; Gehring et al. 2015; Gauderman et al. 2004).

On-road vehicles are a significant source of harmful emissions in California. The burning of fossil fuels such as gasoline and diesel has negative effects: it produces climate-changing emissions such as carbon dioxide and it reduces air quality. $PM_{2.5}$ pollution is of particular concern in California, as the state has seven of the 10 most polluted US cities in terms of $PM_{2.5}$ pollution (ALA 2018b).

$PM_{2.5}$ air pollution (and the exposure to that pollution) varies greatly within the state, and significant variability exists within each region, leading to disparities in exposure to pollution linked to factors such as race and income level. This analysis quantifies the documented, lived experiences of communities of color and supports solutions to reduce air pollution that have been in existence and advocated for decades.

## Analysis of $PM_{2.5}$ Pollution from On-Road Transportation

The concentration of $PM_{2.5}$ at any particular location depends on several variables. These include the location of the $PM_{2.5}$ and precursor $PM_{2.5}$-forming emissions (from tailpipes and refueling locations). Weather patterns and geography also play a role in the generation of secondary $PM_{2.5}$ particles from other air pollutants. In addition, weather and geography determine the movement of $PM_{2.5}$ pollution. Exposure itself depends on the location of both the pollution and the people inhaling the pollution.

To estimate the average annual exposure and health impacts of particulate matter air pollution from cars, trucks, and buses, UCS modeled $PM_{2.5}$ concentrations in California resulting from emissions from vehicle tailpipes and vehicle refueling[2] (Tessum, Hill, and Marshall 2017). We estimated ground-level pollution exposure at the census tract level[3] and combined that information with population and demographic data to understand how exposure to $PM_{2.5}$ varies among groups and locations.

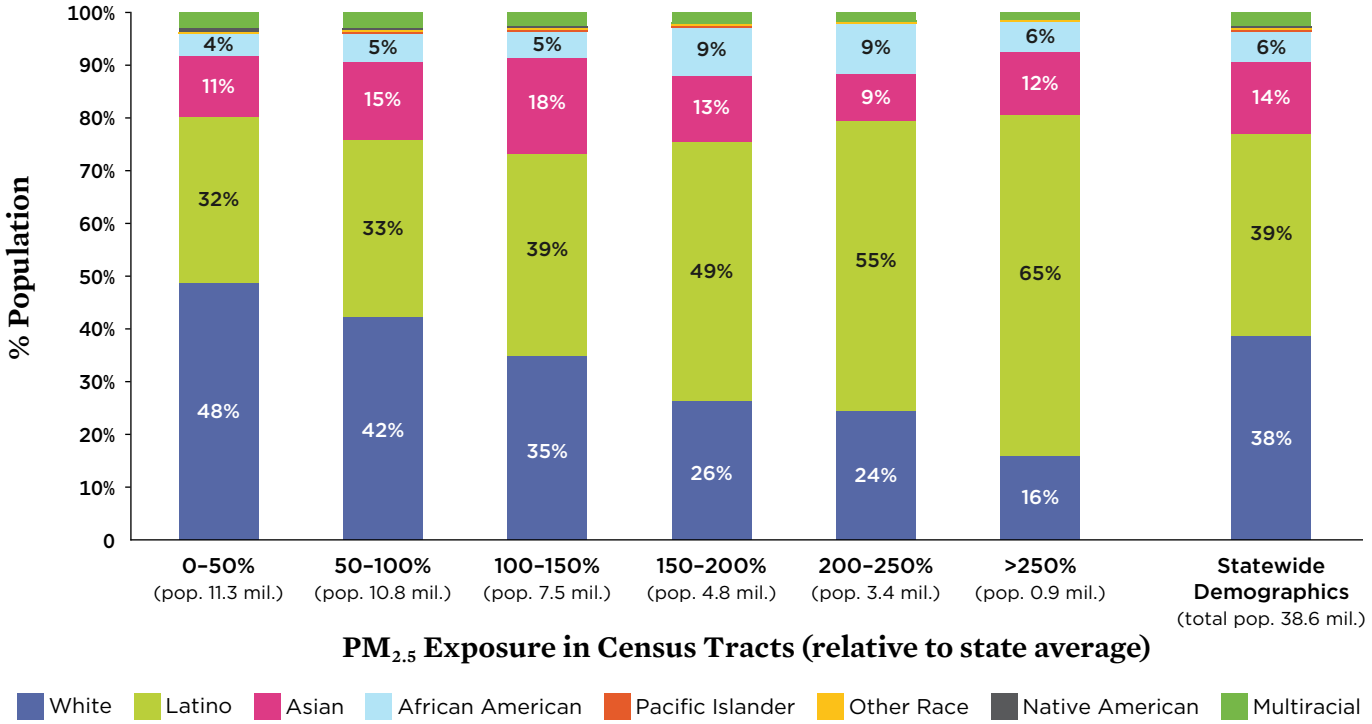
The human health impacts from $PM_{2.5}$ pollution depend not only on the concentration of pollution but also on the number of people exposed. Elevated $PM_{2.5}$ levels in densely populated regions of the state will have a greater public health impact than the same pollution concentration in unpopulated areas. Therefore, to compare $PM_{2.5}$ levels between regions of the state (and between demographic groups), we used population-weighted $PM_{2.5}$ concentrations. For example, to determine the average exposure for a resident of a particular county, we multiplied the concentration in a census tract by the population in the tract. We then divided the sum of these values for the county by the county population to determine the average exposure for a resident of the county. We used a similar process to find the average exposure for demographic groups within the state.

Other types of transportation—such as airplanes, marine vessels, and trains—are not included in these estimates, and their emissions would add to the exposures shown in this work. Operations at freight facilities and ports are also excluded from the $PM_{2.5}$ concentration and exposure modeling. These other transportation and freight emissions can cause significant health impacts—especially for those who live closest to these facilities—leading to known environmental justice concerns (Hricko 2008). The contributions of on-road vehicles to local $PM_{2.5}$ exposure—and related issues of environmental justice—are less well known and yet affect many Californians.

## $PM_{2.5}$ Exposure from Cars, Trucks, and Buses Causes Significant Health Impacts

Based on the location of the particulate matter air pollution and the total population exposed, research estimates that $PM_{2.5}$ from on-road transportation leads to approximately 3,100 premature deaths per year in California due to cardiovascular disease, heart attacks, and other illnesses (Tessum, Hill, and Marshall 2014; Krewski et al. 2009). For comparison, in 2017, 1,829 homicides were reported in the state, or about 40 percent less than the estimated deaths due to $PM_{2.5}$ pollution from cars and trucks (Bulwa 2018). The number of pollution-related deaths is only slightly lower than the 3,600 traffic fatalities reported statewide in 2016 (CAOTS 2017). Given the projected increase in death rate, this pollution has an annual cost of $29 billion, based on the US Environmental Protection Agency's estimate of the value of risks to human life (EPA 2018).

FIGURE 2. Areas with Higher $PM_{2.5}$ Pollution Have a Higher Fraction of People of Color




*Higher $PM_{2.5}$ exposure in an area is correlated with a higher fraction of people of color. In the census tracts with the highest level of on-road vehicle pollution, more than half of the population identifies as Latino while less than a quarter identifies as white. In contrast, the population of Latino and white Californians statewide is nearly equal.*

Note: The following US Census Bureau–defined racial groups were used in the analysis: White; Black or African American; American Indian or Alaska Native; Asian; Native Hawaiian or Other Pacific Islander; Hispanic; Latino; and Some Other Race. In the chart above, Latino includes census respondents who select Hispanic, Latino, or both; Other Race includes census respondents who select Some Other Race as their only race.

SOURCES: US CENSUS BUREAU 2018; EPA 2014.

## Greater $PM_{2.5}$ Pollution for Latinos and African Americans, Low-Income Households

The $PM_{2.5}$ pollution burden from cars, trucks, and buses is inequitably distributed among racial groups in California (Figure 1). Latino community members are, on average, exposed to 15 percent higher $PM_{2.5}$ concentrations than the average Californian, and African Americans in California experience concentrations 18 percent higher than the average Californian. White Californians have average exposure that is 17 percent lower than the mean for the state. Thus, African Americans in California are, on average, burdened with $PM_{2.5}$ pollution 43 percent higher than that affecting white community members.

Pollution burdens can also be seen at the community level (Figure 2). In census tracts with average annual $PM_{2.5}$ concentrations less than half the state average, whites make up 48 percent of the population, while only constituting 38 percent of the state's total population. In contrast, the most polluted census tracts have a higher proportion of people of color. More than 60 percent of people in these highest burden areas are Latino, compared with a state population that is just 39 percent Latino. The difference in exposure to harmful $PM_{2.5}$ pollution can be seen when considering the racial composition of the communities with the highest pollution burden from cars and trucks—and about 1 in 10 Californians live in these communities, where $PM_{2.5}$ pollution is greater than twice the state average. The inequities and disparities are clear.

Research also links inequitable disparities in household income to pollution exposure, with less affluent households having higher exposure to $PM_{2.5}$ pollution from on-road transportation. On average, households with the lowest incomes (less than $20,000 per year) are exposed to more than 25 percent more particulate matter air pollution than the highest-income households (greater than $200,000 per year). Another measure shows the difference in exposure in census tracts designated

as "low-income qualified"[4] by the US Department of Housing and Urban Development (HUD). In HUD-designated low-income areas, average $PM_{2.5}$ pollution levels from on-road vehicles were 32 percent higher than outside these areas.

## Those without Cars Experience Higher Amounts of $PM_{2.5}$ Pollution

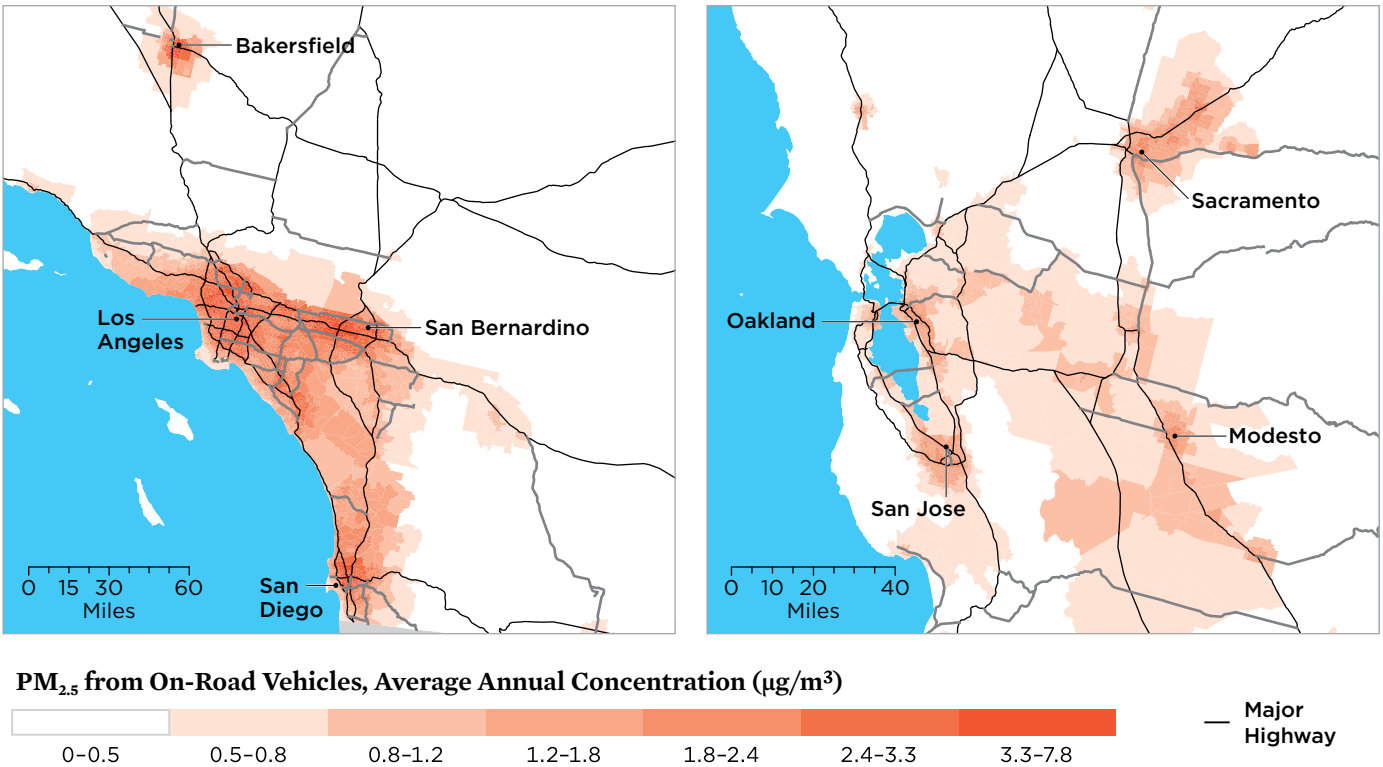
Another group that, on average, is exposed to higher amounts of particulate matter air pollution includes those in households without a car. People in this category face exposure to pollution levels 19 percent above the state average. Many of these households are located in urban areas of the state with higher population densities, where car, truck and bus pollution is more concentrated.

California has proposed strategies to reduce air pollution and climate-changing emissions from personal transportation. These include efforts to reduce personal car driving, as well as land use policies such as transit-oriented development and the addition of high-density housing in existing urban areas (CARB 2017). These strategies could increase the number of people who live in high-density urban housing and have greater access to alternative modes of travel, such as public transit, walking, or biking. It will be important to institute the complementary policies envisioned in California's strategy (see p. 7), such as vehicle electrification, to reduce air pollution from transportation and avoid increasing the number of people exposed to elevated levels of $PM_{2.5}$.

## Particulate Pollution from On-Road Vehicles Is Highest in Southern California

Los Angeles County ranks highest in average $PM_{2.5}$ concentration from on-road vehicles; however, the individual census tracts with the highest $PM_{2.5}$ concentrations in the state are in the city of Bakersfield (Kern County) (Figure 3). Importantly,

FIGURE 3. $PM_{2.5}$ Pollution Concentrations from On-Road Vehicles




*Higher levels of fine particulate matter air pollution are found in pockets of southern California (left) and the Bay Area (right). Northern California's air pollution is less concentrated than Southern California's metropolitan areas.*

SOURCES: US CENSUS BUREAU 2018; EPA 2014.

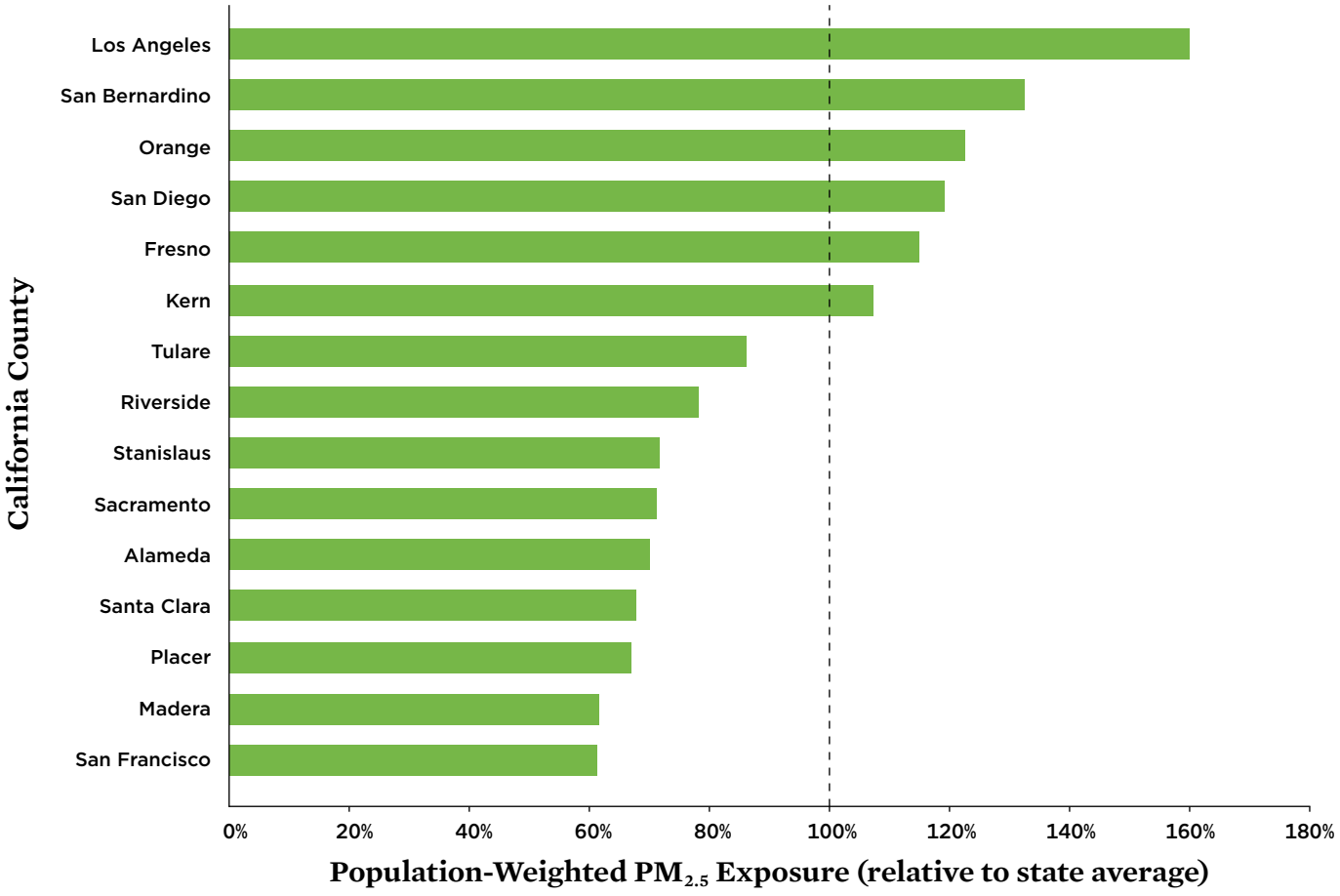
we note that the precision of the air quality model output for this analysis is one square kilometer. This is sufficient to see pollution differences within a city. However, specific locations, such as busy intersections or shipping facilities, would not be distinguishable at this resolution, and hyperlocal $PM_{2.5}$ concentrations could be higher than shown in this analysis.

## $PM_{2.5}$ Exposure from Cars and Trucks Varies Greatly within California

As noted earlier, Los Angeles County has the highest average $PM_{2.5}$ pollution exposure from cars and trucks in the state: on average, 60 percent higher than the mean value for the state (Figure 4). One quarter of the population in Los Angeles County experiences pollution levels that are more than double the state average. And because Los Angeles County is the most populous in the state, this higher level of pollution affects millions of people. Only six counties have an average exposure from on-road transportation that is greater than the state average, but four of them (Los Angeles, Orange, San Bernardino, and San Diego) are in the top five most populous counties in California, with a combined population of almost 19 million people.

Other areas, such as the San Francisco Bay Area, have zones of higher pollution but have much lower average exposure to vehicle-related particulate pollution compared with the state average. The worst regions of the Bay Area (such as downtown Oakland and San Jose) have annual average $PM_{2.5}$ concentrations equal to the average across Los Angeles County.

FIGURE 4. California Counties with Highest Population-Weighted $PM_{2.5}$ Exposure




*Six counties, which are among the most populated counties in the state, have $PM_{2.5}$ exposure levels above the state average.*

SOURCES: US CENSUS BUREAU 2018; EPA 2014.

It is important to note that these results are for a specific subset of pollution sources (on-road vehicles) and for one class of air pollutants ($PM_{2.5}$). Therefore, these results do not indicate total impacts of air pollution in a region or for a demographic group. For example, while San Diego has higher predicted $PM_{2.5}$ concentrations from on-road vehicles than does Fresno County, the overall air pollution in Fresno is often worse than that in the San Diego region. The San Joaquin Valley (where Fresno is located) is noncompliant with federal $PM_{2.5}$ air quality standards, while San Diego is currently within the compliance standard for $PM_{2.5}$ air pollution. The difference between the two regions is caused by other sources of particulate matter air pollution from agriculture, the environment, and stationary sources such as factories, power plants, and refineries. Off-road transportation sources, such as trains and aviation, also cause additional $PM_{2.5}$ burdens.

David Reichmuth/UCS

*A growing number of California communities (such as Oakland, served by AC Transit) are adopting fuel-cell and battery electric buses. Because they don't have tailpipe emissions, electric-drive buses play an important role in reducing transportation-related air pollution in the state and can improve air quality in the communities they serve.*

## Opportunities to Reduce Harmful Impacts of Vehicle Use

Particulate matter air pollution from on-road transportation such as diesel and gasoline vehicles places significant health burdens on Californians, and those burdens are inequitably distributed. However, there are opportunities to greatly reduce the exposure to $PM_{2.5}$ by reducing tailpipe and refueling emissions, making these burdens avoidable.

Electrification of vehicles, both passenger and freight, could greatly reduce emissions. Battery-electric and fuel cell vehicles in particular have no tailpipe emissions (however, there are minor amounts of $PM_{2.5}$ emissions from tire and brake wear) and completely avoid the need for, and emissions associated with, gasoline refueling. Electricity generation and hydrogen production can produce emissions; however, California has renewable content standards for both hydrogen for transportation and electricity that will limit additional emissions (Wisland 2018; California State Senate 2006).

More efficient and lower-emissions conventional vehicles are also important for reducing air pollution. Gasoline vehicles with higher fuel economy need less refueling, potentially reducing the amount of volatile organic compounds evaporating during refueling and from spillage. And fuel-saving technologies, such as start-stop systems that reduce idling, can also contribute to reduced tailpipe emissions.

Decreasing the amount of driving, especially in higher population areas, is also a potential strategy to reduce harmful air pollution and improve public health. Land use decisions are important to reducing the need for driving, and policies that encourage use of public transit, walking, or biking in the place of private passenger auto use could reduce $PM_{2.5}$ generation. This is especially true if the transit options are low-emissions, such as electric rail and buses.

Importantly, state and local governments must take targeted actions to reduce emissions in and near densely populated neighborhoods and in the communities of color and low-income communities that are currently burdened with a disproportionate share of pollution from cars and trucks. Existing actions such as California's Clean Vehicle Rebate Project, low- and zero-emissions vehicle regulations, regional

***Electrifying passenger and freight vehicles could greatly reduce emissions—a critical solution for communities that are currently burdened with a disproportionate share of vehicle pollution.***

Kathy deWitt/Alamy Stock Photo

*Increasing development in metropolitan areas means even more California residents are living near polluted traffic corridors. Policies aimed at reducing transportation-related emissions should prioritize densely populated neighborhoods, as well as communities of color and low-income communities that are currently burdened by a disproportionate level of vehicle pollution.*

Sustainable Communities Strategies, and the Sustainable Freight Action Plan exemplify steps the state has taken to reduce air pollution from vehicles. Policymakers have also taken specific actions aimed at reducing burdens on the most heavily affected communities, for example, by instituting requirements for the government to invest a minimum percentage of revenue generated from the state's cap-and-trade program in disadvantaged communities.[5] Clean vehicle incentive programs—which provide greater financial incentives for lower-income households and for deployments in disadvantaged communities—as well as programs to accelerate the retirement of the oldest, highest polluting vehicles are also being implemented (CARB 2018).

While Californians can make a difference by choosing cleaner vehicles, much of the pollution comes from sources outside an individual's direct control. The state needs regulations, incentives, and other policies to reduce vehicle emissions. Equity and meaningful involvement of disadvantaged communities should be key considerations in designing policies and strategies to reduce pollution from vehicles. The state will need to continue to make progress on reducing emissions and should prioritize actions that reduce the inequitably distributed burden of air pollution in California. This analysis provides evidence of the need for and importance of these types of programs and can help inform and shape future actions to reduce pollution exposure and environmental inequities in California.

***David Reichmuth*** *is a senior engineer in the UCS Clean Vehicles Program.*

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*Organizational affiliations are listed for identification purposes only. The opinions expressed herein do not necessarily reflect those of the organizations that funded the work or the individuals who reviewed it. The Union of Concerned Scientists bears sole responsibility for the report's contents.*

**ENDNOTES**

1. The vast majority of on-road emissions come from gasoline- and diesel-powered vehicles; however, some emissions come from compressed natural gas– and ethanol-powered (E85) vehicles.
2. Details on the modeling approach using the reduced-complexity InMAP model can be found at *www.ucsusa.org/air-quality-methodology*. The InMap model is available at *www.spatialmodel.com/inmap*.
3. Census tracts are groupings of, on average, 4,000 persons.
4. In Low-Income Housing Tax Credit Qualified Census Tracts, 50 percent of households have incomes below 60 percent of the area median gross income or have a poverty rate of 25 percent or more.
5. "Disadvantaged communities" are defined by the California Environmental Protection Agency as census tracts identified by the California Communities Environmental Health Screening Tool as being the most burdened by pollution and most vulnerable to its effects, when considering socioeconomic characteristics and underlying health status.

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# EXHIBIT 7

# Inequity in consumption of goods and services adds to racial–ethnic disparities in air pollution exposure

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**Fine particulate matter ($PM_{2.5}$) air pollution exposure is the largest environmental health risk factor in the United States. Here, we link $PM_{2.5}$ exposure to the human activities responsible for $PM_{2.5}$ pollution. We use these results to explore "pollution inequity": the difference between the environmental health damage caused by a racial–ethnic group and the damage that group experiences. We show that, in the United States, $PM_{2.5}$ exposure is disproportionately caused by consumption of goods and services mainly by the non-Hispanic white majority, but disproportionately inhaled by black and Hispanic minorities. On average, non-Hispanic whites experience a "pollution advantage": They experience ∼17% less air pollution exposure than is caused by their consumption. Blacks and Hispanics on average bear a "pollution burden" of 56% and 63% excess exposure, respectively, relative to the exposure caused by their consumption. The total disparity is caused as much by how much people consume as by how much pollution they breathe. Differences in the types of goods and services consumed by each group are less important. $PM_{2.5}$ exposures declined ∼50% during 2002–2015 for all three racial–ethnic groups, but pollution inequity has remained high.**

air quality | environmental justice | fine particulate matter | input–output | life cycle assessment

Fine particulate matter ($PM_{2.5}$) exposure is a major health risk factor in the United States, responsible for 63% of deaths from environmental causes and 3% of deaths from all causes (1). It is a risk factor that is inequitably distributed among demographic groups, including racial–ethnic groups, owing in part to differences in pollution concentrations at locations of residence (2, 3). The extent to which differences in consumption of goods and services by racial–ethnic groups contribute to observed disparities in exposure is unknown, as is whether racial–ethnic groups have benefited equitably from recent improvements in $PM_{2.5}$ air quality.

Here, we explore racial–ethnic disparities in the causation and effect of exposure to $PM_{2.5}$ in the United States. We do this by investigating links among pollution, the parties responsible for its emission, and the health impacts that result. First, we estimate mortality from $PM_{2.5}$ for all emission sources in the United States. Next, we attribute these emissions to the end-use activities and to the end-user parties ultimately responsible for their generation. Finally, we compare results among racial–ethnic groups to explore what we term "pollution inequity": the extent to which groups disproportionately contribute to or bear the burden of pollution.

We estimate mortality impacts in the United States from $PM_{2.5}$ exposure using spatially explicit emissions data from all pollutant emission sources (4), the Intervention Model for Air Pollution (InMAP) air quality model (5), and spatially explicit population and health data (ref. 6; see *Materials and Methods*). We consider emissions of primary $PM_{2.5}$ and of secondary $PM_{2.5}$ precursors, both of which contribute to increased atmospheric $PM_{2.5}$ concentrations. Our approach yields estimates of premature deaths caused by $PM_{2.5}$ exposure in the United States for each year during 2003–2015, disaggregated by 5,435 emissions source types, at a spatial resolution varying between 1 and 48 km depending on population density. We aggregate impacts into 15 emitter groups. (See *Materials and Methods*; *SI Appendix*, Tables S1–S14 show the largest emitter types in the 14 anthropogenic and domestic emitter groups.)

We estimate a population-weighted average ambient $PM_{2.5}$ exposure concentration of 7.7 $\mu g \cdot m^{-3}$ for the United States in 2015, causing 131,000 premature deaths (Fig. 1 and *SI Appendix*, Fig. S1; see *SI Appendix*). Of these, 102,000 are caused by US anthropogenic emissions and 29,000 by other sources, largely wildfires and natural biogenic emissions (26,000), with minor contributions from Canadian and Mexican emissions (3,000). The total number of deaths reported here is higher than a commonly cited estimate of 93,000 (1), but at the low end of the range of a recently published estimate of 121,000–213,000 deaths (7), which uses a concentration–response relationship similar to the one employed here (6). (*SI Appendix*, Table S15 reports estimates of $PM_{2.5}$ mortalities using several concentration–response functions.)

Responsibility for air pollution is typically assigned to its emitters (8) (e.g., a factory), but it can also be ascribed to end uses (e.g., the purchase and use of manufactured goods) by end users (e.g., individual consumers) that ultimately result in its release (Fig. 1). Here, we connect $PM_{2.5}$ air pollution and its health impacts to end uses

## Significance

**Racial–ethnic disparities in pollution exposure and in consumption of goods and services in the United States are well documented. Some may find it intuitive that, on average, black and Hispanic minorities bear a disproportionate burden from the air pollution caused mainly by non-Hispanic whites, but this effect has not previously been directly established, let alone quantified. Our "pollution inequity" metric is generalizable to other pollution types and provides a simple and intuitive way of expressing a disparity between the pollution that people cause and the pollution to which they are exposed. Our results are timely, given public debate on issues relating to race, equity, and the regulation of pollution.**

Author contributions: C.W.T., J.D.M., and J.D.H. designed research; C.W.T., K.A.M., D.A.P., N.P.S., S.K.T., J.D.M., and J.D.H. performed research; C.W.T., D.A.P., J.D.M., and J.D.H. contributed new reagents/analytic tools; C.W.T., K.A.M., J.D.M., and J.D.H. analyzed data; and C.W.T., J.S.A., A.L.G., N.Z.M., K.A.M., D.A.P., S.P., N.P.S., S.K.T., J.D.M., and J.D.H. wrote the paper.

The authors declare no conflict of interest.

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Data deposition: The model used in this paper has been deposited in Zenodo, https://zenodo.org/record/2549859.

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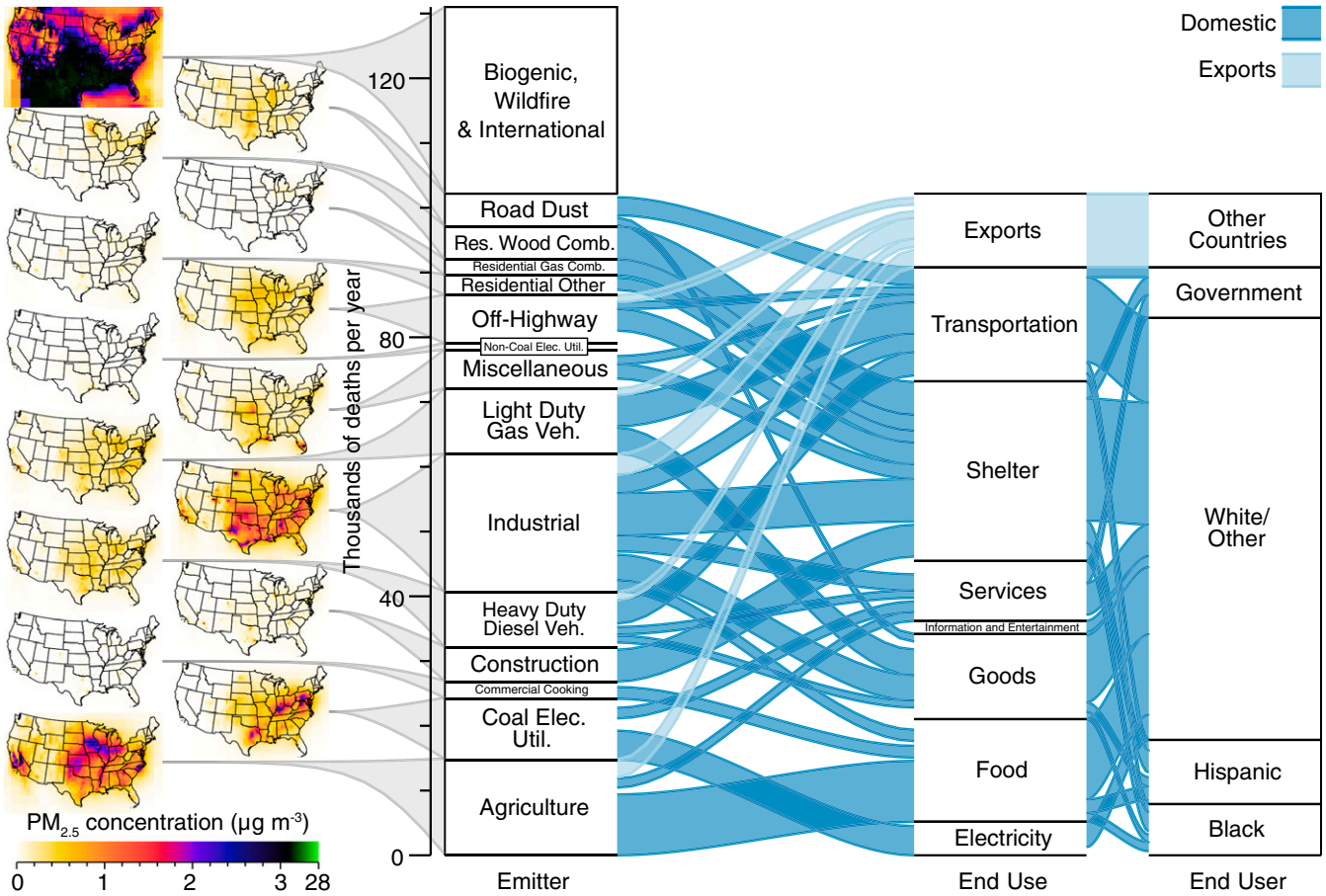


**Fig. 1.** Sources of US mortality from $PM_{2.5}$. $PM_{2.5}$ concentrations resulting from emissions from each emitter group [maps on *Left*; color scale contains a discontinuity at the 99th percentile of concentrations (i.e., 3.1 $\mu g \cdot m^{-3}$)] and relationships among $PM_{2.5}$ health impacts as attributed to emitters (*Left* bar), end uses (*Middle* bar), and end users (*Right* bar). The height of the bar on the *Left* shows the number of $PM_{2.5}$-attributable premature deaths caused by the physical production of emissions from each group of emitters, the height of the *Middle* bar shows the number of deaths caused by demand for each group of end uses, and the height of the bar on the *Right* shows the number of deaths caused by different types of end users. The blue connecting lines show relationships among emitters, end uses, and end users; connecting lines representing <1,000 deaths are not shown. (Detailed relationships between end uses and emitters for each racial–ethnic end-user group are shown in Fig. 2; time trends are shown in *SI Appendix*, Fig. S4.)

and end users by coupling economic input–output relationships to pollution emission sources (https://www.bea.gov/industry/io_annual.htm). Our approach allows us to attribute responsibility to (*i*) emitter entities that physically emit air pollutants; (*ii*) end uses that lead to air pollution emissions, often through intermediate economic transactions; and (*iii*) end users. We track 19 end-user types, which we aggregate here into four groups (personal consumption by each of three racial–ethnic groups, as well as government consumption), and 389 end-use categories, which we aggregate here into seven groups (electricity, food, goods, information and entertainment, services, shelter, and transportation).

Of 102,000 premature deaths from domestic anthropogenic emissions, we estimate 11,000 (11%) are caused by demand for goods that are exported (Fig. 1). Of the remaining 91,000 premature deaths caused by end uses within the United States, 83,000 (91%) are attributed to personal consumption (i.e., individual consumers); the remaining 8,000 (9%) are caused by pollution related to governmental expenditures.

To determine racial–ethnic inequity, we disaggregate personal consumption and exposure to $PM_{2.5}$ by race–ethnicity. Here, "exposure" is the population-weighted average ambient concentration at places of residence. We focus on the subset of impacts (83,000 premature deaths) that we can attribute to consumption by individuals in the United States, excluding the 48,000 premature deaths caused by governmental end uses, exports, and nonanthropogenic sources. (Racial–ethnic disparities in overall exposure to $PM_{2.5}$ from all sources are shown in *SI Appendix*, Fig. S2.) We consider persons self-identifying as black or African-American (hereafter, "black"; 12% of the population), Hispanic or Latino ("Hispanic"; 17% of the population), and the remainder [non-Hispanic white (62% of the population) plus all other race–ethnicity groups (8% of the population); hereafter, "white/other" (70% of the population)].

We define and quantify pollution inequity for a group $g$ ($I_g$) as the fractional difference between a racial–ethnic group's exposure to $PM_{2.5}$ caused by all groups ($E_g$) and that group's population-adjusted contribution to the overall $PM_{2.5}$ exposure of all groups ($C_g$) (Eq. **1**):

$$\left[I_g = \frac{E_g}{C_g} - 1\right]. \qquad [1]$$

Positive values for pollution inequity indicate that a group experiences more exposure than it causes (on average and after adjusting for population sizes); negative values indicate the opposite.

We find that blacks are exposed to 6.0 $\mu g \cdot m^{-3}$ of $PM_{2.5}$ ($E_g$), which is 21% greater than the overall population average exposure of 5.0 $\mu g \cdot m^{-3}$, while their population-adjusted consumption causes $PM_{2.5}$ exposure of 3.8 $\mu g \cdot m^{-3}$ to the overall population ($C_g$), which is 23% less exposure than average (Fig. 2). We therefore estimate for blacks a pollution inequity of 56% (Fig. 3*A*; $6.0\,\mu g \cdot m^{-3}/3.8\,\mu g \cdot m^{-3} - 1 = (1+0.21)/(1-0.23) - 1 = 56\%$). Hispanics are exposed to 12% more $PM_{2.5}$ than average (5.5 $\mu g \cdot m^{-3}$),



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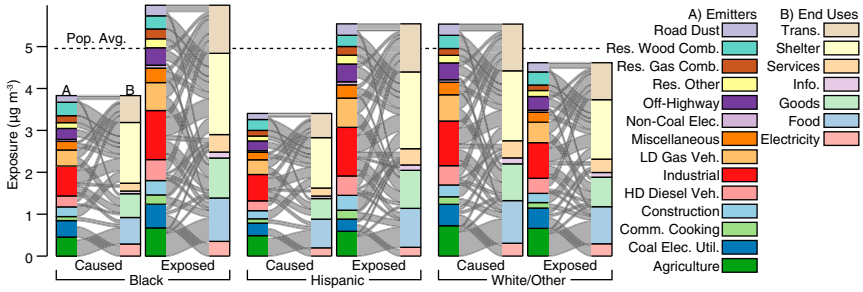


**Fig. 2.** Average $PM_{2.5}$ exposure experienced and caused by racial–ethnic groups. Total exposure to $PM_{2.5}$ caused by population-adjusted group consumption ("caused," or $C_g$) and group exposure to $PM_{2.5}$ caused by total personal consumption ("exposed," or $E_g$), stratified by racial–ethnic group. Pollution inequity is the percent difference between a group's "exposed" and "caused" bars. Each group of bars shows the (*A*) emitters and (*B*) end uses responsible for the exposure, with gray connecting lines showing relationships among emitters and end uses. Connecting lines representing <0.04 $\mu g \cdot m^{-3}$ are not shown.

but cause 31% less exposure than average (3.4 $\mu g \cdot m^{-3}$), for a pollution inequity of 63%. Whites/others are exposed to 7% less $PM_{2.5}$ than average (4.6 $\mu g \cdot m^{-3}$), but cause 12% more exposure than average (5.5 $\mu g \cdot m^{-3}$), for a pollution inequity of –17%.

Blacks are more exposed than whites/others to pollution from every emitter group (Fig. 2). The same holds for Hispanics, with the exceptions of $PM_{2.5}$ originating from agriculture, from coal electric utilities, and from residential wood combustion, for which they are exposed to 11%, 40%, and 14% less, respectively, than whites/others. Those three types of emissions are concentrated in regions of the United States with relatively low Hispanic populations (Fig. 1). Whites/others consume more—and cause more exposure—than do blacks and Hispanics across all seven end-use categories; the end uses representing the greatest differences in consumption-caused exposure are food (for which whites/others cause 61% and 49% more exposure than blacks and Hispanics, respectively), transportation (74% and 93%), and services (118% and 114%).

Differences in consumption across groups are comparable or larger contributors to pollution inequity than are differences in exposure across groups. Consumption differences account for 52%, 73%, and 63% of overall pollution inequity for blacks, Hispanics, and whites/others, respectively (Fig. 3*A*). Previous analyses have found that when considering only differences in locations of residence, exposure disparities by race are much larger than disparities by income (9, 10). Our results suggest that income, to the extent that it correlates with consumption, is an important factor in determining how much pollution a person causes, even if it may be statistically less important as a determinant of exposure. We also find that differences in racial–ethnic groups' contribution to exposure are driven more by differences in their overall amount of consumption (magnitude effect) than by differences in the types of goods and services they consume (composition effect) (Fig. 4 and *SI Appendix*, Fig. S3).

Exposure to $PM_{2.5}$ caused by personal consumption by all three racial–ethnic groups decreased by an average of 51% during 2003–2015 (Fig. 3*B*, *SI Appendix*, Fig. S4, and Movie S1), even as personal consumption expenditures increased (*SI Appendix*, Fig. S4 and Tables S16–S18). Furthermore, absolute differences in exposure caused by overall consumption decreased among groups, as did absolute differences in overall exposure caused by each group's consumption (Fig. 3*B*). Pollution inequity has remained high, however, decreasing by 23% for blacks (from 73% in 2003 to 56% in 2015) but increasing by 5% for Hispanics (from 60% in 2003 to 63% in 2015; Fig. 3*C*). Increases in consumption during 2003–2015 were larger for blacks than Hispanics for most sectors of the economy; two notable examples are spending on shelter (17% and 2% increases, respectively) and goods (21% and 6%, respectively) (*SI Appendix*, Tables S16–S18). Decreases in absolute exposure differences were primarily caused by decreases in the $PM_{2.5}$ concentrations where blacks and Hispanics live, rather than by blacks and Hispanics moving to locations with lower $PM_{2.5}$ concentrations (*SI Appendix*, Fig. S2).

Here, we have described linkages between human end-use activities and air pollution—and the racial–ethnic disparities therein. We find that, in the United States, $PM_{2.5}$ air pollution is disproportionately induced by the racial–ethnic majority and disproportionately inhaled by racial–ethnic minorities. All have benefited from recent reductions in atmospheric $PM_{2.5}$ concentrations. Our analysis shows for the first time how pollution inequity is driven by differences among racial–ethnic groups in both exposure and the consumption that leads to emissions. Still, questions remain about the spatial context of pollution inequity, its underlying causes, how best to address it, and its generalizability. For example, little is known about the "spatial scale" of inequity, such as whether consumers tend to live near to or far from the people exposed to the pollution resulting from their consumption. Further information on this issue would clarify whether this inequity could best be investigated and addressed at the city, state, or national level. Another open question is whether the patterns of pollution inequity described here are observed for other pollutants, times, or locations (e.g., in other countries). The pollution inequity metric defined here could be used to explore such questions and to inform discussion of inequity in other environmental burdens, including climate change, for which inequities can occur across continents and generations, in addition to across race–ethnicities.

## Materials and Methods

Environmentally extended economic input–output analysis has been used to track air pollutant and greenhouse gas emissions induced by economic demand within and among national economies (11–16). Fewer studies have reported air quality-related health impacts induced by domestic (17, 18) and international trade (19–21). Here, we explore relationships among human end-use activities in the United States, $PM_{2.5}$-related health impacts caused by those activities, the corresponding consumption–exposure inequity among race–ethnicity groups, and related temporal trends.

Unlike analyses of greenhouse gas emissions for use in climate-change impact assessment, analyses of health impacts from non-greenhouse gas air pollution strongly benefit from spatial differentiation. For example, within the United States, health impacts per unit of emissions of $PM_{2.5}$ and its precursors

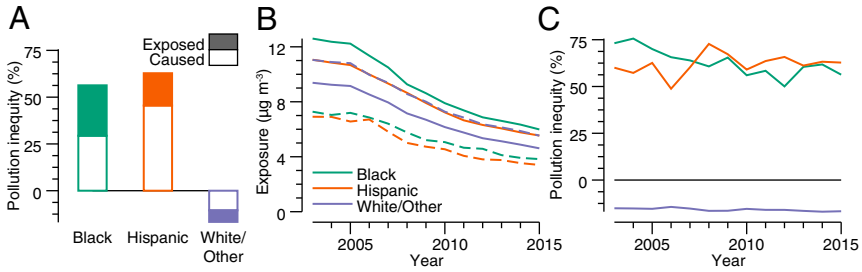


**Fig. 3.** Pollution inequity contributions and trends. (*A*) Contributions of differences in consumption (caused, or $C_g$) and location of residence (exposed, or $E_g$) to pollution inequity. (*B*) Exposure of each racial–ethnic group to $PM_{2.5}$ caused by the total combined personal consumption of all groups (exposed or $E_g$; solid lines) and total-population exposure to $PM_{2.5}$ caused by each group's population-adjusted consumption (caused, or $C_g$; dashed lines), 2003–2015. (*C*) Pollution inequity levels, 2003–2015.



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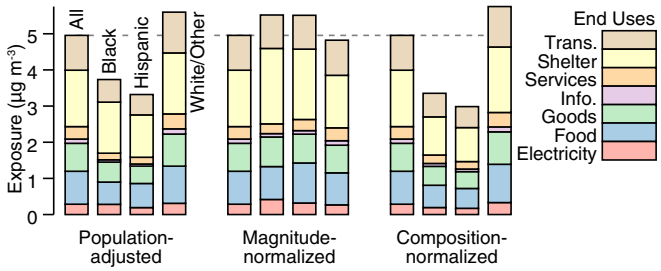


**Fig. 4.** Effect of magnitude and composition of consumption on $PM_{2.5}$ exposure. Population-adjusted $PM_{2.5}$ exposure (*Left*): actual population-adjusted exposure (as seen in Fig. 2). Magnitude-normalized $PM_{2.5}$ exposure (*Middle*): hypothetical exposure in which the overall magnitude of per capita consumption for each race–ethnicity is adjusted to match "All" without changing the composition of goods and services consumed. Composition-normalized $PM_{2.5}$ exposure (*Right*): hypothetical exposure where the composition of goods and services consumed by each race–ethnicity is adjusted to match All without changing the overall magnitude of consumption.

vary greatly across emission locations (22, 23). Spatial resolution is especially important when quantifying disparities in exposure among demographic groups (9). Therefore, to create a spatially explicit environmentally extended economic input–output model for the United States, we couple economic input–output (https://www.bea.gov/industry/io_annual.htm) and consumption (https://www.bls.gov/cex/) data with spatially explicit emissions data (4), the InMAP air quality model (5), and spatially explicit population and health data (ref. 6; https://www.census.gov/programs-surveys/acs/technical-documentation/table-and-geography-changes/2015/5-year.html; https://www.cdc.gov/nchs/data_access/cmf.htm). The resulting model relates air pollution emissions, concentrations, and health impacts with economic activity in the United States at a spatial scale varying between 1 and 48 km, depending on population density and emissions density. We refer to the model as the Extended InMAP Economic Input–Output (EIEIO) model, which is freely available at the Zenodo repository (24). InMAP is able to spatially resolve both the entire contiguous United States and within-city concentration gradients, which is critical for quantifying within- and among-city differences in exposure.

EIEIO uses economic data to trace human end-use activities that directly and indirectly cause air pollutant emissions and the resulting human exposure to $PM_{2.5}$. The model tracks relationships between final "end users," the activities or "end uses" they are participating in (or "final demand for commodities," in economic input–output terminology) that induce air pollution emissions, and the "emitter" entities that are physically releasing air pollutant emissions. EIEIO also tracks "intermediate uses." Intermediate uses are purchases by businesses to produce something that they are selling, whereas end uses are purchases or activities for reasons other than producing something to be directly sold. For example, the purchase of electricity to heat a home is an end use, whereas the purchase of electricity to manufacture fertilizer is an intermediate use. Our analysis includes both the emissions caused by an end use itself (e.g., tailpipe emissions from driving a car) and the emissions from economic activities in support of the end use (e.g., emissions from the production of gasoline to fuel the car).

EIEIO tracks 19 end-user types, 389 end-use categories, and 5,434 categories of emitters. For ease of display and communication, we present results here in groups of four users, seven uses, and 14 emitters; further details are in *SI Appendix*. Mappings from the use and emitter categories to corresponding groups are in Tessum et al. (24). Unless otherwise noted, all results are for year 2015.

Methods are described below and in Tessum et al. (24). The model source code includes a graphical interface that can be used for exploratory analysis and visualization. Results here were generated using a 2018-vintage Google Compute Engine instance with 32 CPU cores, 208 GB of RAM, and a 500-GB hard drive.

**Economic Production.** To relate final economic demand for commodities to economic activity or production in individual industries, we use the following US Bureau of Economic Analysis (BEA) Input–Output Accounts Data (https://www.bea.gov/industry/io_annual.htm):

*i*) Final demand ($d_f$): Economic activity that leads to the final consumption of a good or service and that is not induced by economic activity in another sector of the economy. This can include demand for exports.
*ii*) Import final demand ($d_{fi}$): Economic activity that leads to the final consumption of an imported good or service and that is not induced by economic activity in another sector of the economy.
*iii*) Total requirements ($R_t$): Direct plus indirect purchases from an industry required to produce a dollar of output of a commodity (25).
*iv*) Total domestic requirements ($R_{t,d}$): Domestic (i.e., within the United States) direct plus indirect purchases from an industry required to produce a dollar of output of a commodity.
*v*) Total import requirements ($R_{t,i}$): Calculated as $R_{t,i} = R_t - R_{t,d}$.

where $d_f$ and $d_{f,i}$ are vectors with one entry for each of 389 commodity sectors, and $R_t$, $R_{t,d}$, and $R_{t,i}$ are matrices with one row for each of 389 industry sectors and one column for each of 389 commodity sectors.

We calculate economic production, $\rho$, caused by final demand as in Eq. **2**:

$$\rho = Rd_f, \quad [2]$$

where $R$ is one of $R_t$, $R_{t,d}$, or $R_{t,i}$ depending on whether total, domestic, or international economic production is desired. For imports, $d_f$ is replaced with $d_{fi}$. $\rho$ is a vector with one entry for each industry sector.

BEA input–output data are disaggregated to the detailed level of 389 industries and 389 commodities for year 2007, and to the summary level of 71 industries and 73 commodities for years 1997–2015. To perform calculations for years other than 2007, we scale the detailed 2007 data as in Eq. **3**:

$$v_{d,i,c,y} = \frac{v_{d,i,c,2007}\, v_{s,i,c,y}}{v_{s,i,c,2007}}, \quad [3]$$

where $v_{d,i,c,y}$ is a value at the detailed level of aggregation for industry $i$ and commodity $c$ for the year of interest, $v_{d,i,c,2007}$ is the corresponding value at the detailed level of aggregation for year 2007, and $v_{s,i,c,y}$ and $v_{s,i,c,2007}$ are values for the corresponding summary level of aggregation for the year of interest and 2007, respectively.

Some negative values for final demand exist in the BEA input–output data tables. These typically relate to divestments or reductions in amounts of stocks. Because our objective is to use economic relationships to model air pollution emissions and impacts, and divestments or stock reductions do not cause negative emissions in the same way that investments and increases in stocks can be said to cause positive emissions, we set all negative final demand values to zero.

**Demographic-Specific Personal Consumption Demand.** BEA input–output data report final demand from personal expenditures, but the data do not disaggregate consumption by racial or ethnic groups. To calculate demographic-specific consumption, we match categories in the US Bureau of Labor Statistics Consumer Expenditure Survey (CES) (https://www.bls.gov/cex/) to the BEA input–output sectors, then use the demographic information in the CES data to allocate BEA personal expenditures among demographic groups. The CES data report expenditures separately for the following: Hispanics or Latinos; Not Hispanic or Latino: whites and all other races; and Not Hispanic or Latino: blacks or African-Americans.

As of this writing, CES data are available for the years 2003–2015. EIEIO does not account for geographic variation in consumption amounts or in the proportions of goods and services consumed.

**Augmented Personal Consumption.** In addition to personal consumption (causing 46,000 premature deaths from $PM_{2.5}$), we also attribute BEA private expenditure final demand categories to individual end users and allocate the expenditures among demographic groups. We do this by directly adding final demand for "Residential private fixed investment" (16,000 premature deaths from $PM_{2.5}$) to personal consumption, as individuals are the ultimate end users of residential buildings. The remaining private expenditure categories include expenditures on nonresidential structures (9,400 deaths), nonresidential equipment (9,400 deaths), and intellectual property (500 deaths), as well as changes in inventory (1,700 deaths). Because consumption activities provide the revenue streams that organizations use to make capital investments and to generate inventory, albeit with time lags that we do not account for here, we consider these expenditures—and the resulting air pollution—to be caused by personal consumption. Therefore, we attribute these additional categories of demand to demographic groups proportionate to each group's overall fraction of combined personal consumption and residential investments. Although government expenditures are also ultimately funded by individuals, the taxes that fund the government are compulsory, and relationships between individual tax contributions and government spending decisions are uncertain. Therefore, we do not attribute government expenditures to individuals, but instead track and display them as their own category.

**Emission Factors.** We create spatially explicit emissions factors—in units of mass per time of emissions of primary $PM_{2.5}$ and secondary $PM_{2.5}$ precursors [oxides of nitrogen ($NO_x$), oxides of sulfur ($SO_x$), ammonia ($NH_3$), and volatile



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organic compounds (VOCs)] per dollar—for each of the 5,434 EPA source classification codes (SCCs) in the year 2014 US National Emissions Inventory (NEI), version 1 (4). Each emissions record in the NEI contains an SCC that specifies the type of source creating the emissions. First, we match each SCC to one or more of the 389 BEA industries. Some sources of emissions cannot be directly matched to BEA industries because they do not result from economic transactions. We match these sources to the BEA industry to which it is most closely related. The largest source of these nontransactional emissions is the personal use of light-duty vehicles, which we match to the "automobile manufacturing" industry based on the assumption that the individuals and entities that drive light-duty vehicles and create the resulting emissions are the same as the individuals and entities that purchase automobiles. Other nontransactional sources of emissions include leisure activities such as barbecuing and operating recreational vehicles, which we attribute to relevant residential or recreational industries. The cross-walk between SCCs and BEA industries can be found in Tessum et al. (24). We use this cross-walk to map the economic production vector, $\rho$, which has one element for each BEA industry, to vector $\hat{\rho}$, which has one element for each SCC equal to the sum of economic production in the BEA industry or industries that the SCC is matched to. $\hat{\rho}$ double counts economic production in some cases, but is used in a way that ensures emissions are not double counted.

Next, we process the NEI emissions (excluding emissions occurring in Canada and Mexico, which are tracked separately) using the InMAP Air Emissions Preprocessor program, also included in Tessum et al. (24). We assign each emissions record to the BEA industry or industries it belongs to and allocate the emissions to a spatial grid with cell edge lengths varying between 1 and 48 km, depending on population density and emission density. [The grid employed by InMAP is described further by Tessum et al. (5).] We allocate county-specific emissions to grid cells within counties using spatial surrogates, as described by the US EPA (4).

Finally, we calculate spatially explicit emissions factors by dividing the emissions from each SCC by the total domestic economic production in the matched industry or industries (i.e., $\hat{\rho}$) resulting from domestic and export final demand. The result is a series of emissions factor matrices, $E_p$, where $p$ is one of the pollutants in (primary $PM_{2.5}$, $NO_x$, $SO_x$, $NH_3$, VOC). Each emissions factor matrix has one row for each spatial grid cell, one column for each SCC, and dimensions of [mass·time$^{-1}$·\$$^{-1}$].

For analysis years other than 2014, we adjust the 2014 NEI emissions according to state- and source-group-specific annual trends in emissions published by the US EPA (https://www.epa.gov/air-emissions-inventories/air-pollutant-emissions-trends-data). To quantify health impacts from non–human-related emissions sources, we also include combined biogenic and wildfire emissions from year 2005, as processed by Tessum et al. (26). Further information is in *SI Appendix*. We calculate spatially explicit emissions of a pollutant $p$ ($e_p$) induced by human activity (using economic final demand as a surrogate for human activity) as shown in Eq. **4**:

$$e_p = E_p \hat{\rho}, \quad [4]$$

where $e_p$ is a vector with length equaling the number of spatial grid cells and dimensions of [mass·time$^{-1}$].

**$PM_{2.5}$ Concentrations.** Primary $PM_{2.5}$ and secondary $PM_{2.5}$ precursors are emitted into the atmosphere where they are transported by wind, transformed by chemistry, and ultimately inhaled by humans or otherwise removed. We account for these phenomena using InMAP, version 1.2.1 (5); InMAP creates spatially explicit estimates of ambient $PM_{2.5}$ concentrations caused by the emissions estimated by EIEIO. For computational expedience, we use InMAP to create a set of source–receptor matrices, which describe linear relationships between (*i*) emissions in each of many source locations and (*ii*) concentrations in each of many receptor locations. We create the InMAP source–receptor matrix (ISRM) by running separate InMAP simulations that estimate the ground-level changes in $PM_{2.5}$ concentrations of emissions of $SO_x$, $NO_x$, VOCs, $NH_3$, and primary $PM_{2.5}$ in each of ∼50,000 InMAP grid cells. This is repeated three times to consider emissions plume height ranges of 0–57, 240–380, and 760–1,000 m, for a total of ∼150,000 simulations. The result can be represented as a rank-four tensor describing independent linear relationships between emissions and $PM_{2.5}$ concentrations for discrete combinations of pollutant emitted, emissions source location, emissions plume height, and concentration receptor location. By using linear interpolation to calculate impacts for sources with plume heights that do not fall within the modeled height ranges, ISRM can quickly calculate $PM_{2.5}$ concentrations resulting from arbitrary combinations of emissions sources and locations. ISRM model performance evaluation is in *SI Appendix*.

Ground-level concentrations of $PM_{2.5}$ depend on the height and location of emissions; therefore, instead of directly using the $E_p$ matrices to calculate concentration impacts, we create a separate series of matrices for the concentration factor, $C_p$, for each emitted pollutant, $p$, by using the ISRM to calculate total concentrations from the NEI emissions records associated with each SCC—while accounting for individual plume heights from each emissions record—and dividing the result by the total transformed domestic economic production, $\hat{\rho}$. The resulting matrices, $C_p$, have one row for each spatial grid cell, one column for each SCC, and units of micrograms per cubic meter per dollar. Total $PM_{2.5}$ concentration impacts ($c$) of economic final demand are calculated by summing impacts from each emitted pollutant as in Eq. **5**:

$$c = \sum_p \{C_p \hat{\rho}\}, \quad [5]$$

where $c$ is a vector with length equaling the number of spatial grid cells and units of micrograms per cubic meter.

**Health Impacts.** Air pollution-related health impacts from economic final demand are a function of population counts, underlying incidence rates, and concentration–response relationships, in addition to the $PM_{2.5}$ concentrations themselves.

***Population counts.*** Population counts are based on data from the US Census Bureau American Community Survey (ACS) 5-Year Estimates (https://www.census.gov/programs-surveys/acs/technical-documentation/table-and-geography-changes/2015/5-year.html) for midpoint years 2007–2014, plus the year 2000 decennial census, downloaded from the National Historical Geographic Information System (27) at census block-group spatial resolution. We calculate health impacts for several race–ethnicity categories:

*i*) Total population (314 million people in our study domain, as of 2014).
*ii*) People of all races who are Hispanic or Latino; we refer to this group as Hispanic (54 million people).
*iii*) People who are not Hispanic or Latino and are black or African American alone; we refer to this group as black (39 million people).
*iv*) All people who are not in the Hispanic or black groups; we refer to this group as white/other; this group includes 196 million whites, 15 million Asians or Pacific Islanders, 2 million American Indians, and 8 million Others/Multiple Races.

Population counts for years 2001–2006 are estimated using spatially explicit interpolation with 2000 and 2007 as the endpoints, years 1997–1999 use year 2000 population counts without modification, and year 2015 uses year 2014 population counts without modification. Data for years 2007–2014 are directly available from ACS. We use the total population count to calculate total health impacts, and we use the separate counts for each demographic group to calculate inequity in $PM_{2.5}$ exposure. The racial–ethnic groups used here were chosen to align with the demographic groups in the Consumer Economics Survey (https://www.bls.gov/cex/). We use population counts for people of all ages, rather than restricting the analysis to a specific age range. One reason for this is that publicly available US Census data do not include both race–ethnicity and age information at the block-group spatial resolution. We allocate population counts to spatial grid cells, using area weighting for census block groups that overlap more than one grid cell. The resulting vectors, $p_g$, where $g$ is the set of demographic groups above, have one row for each grid cell and units of [persons].

***Underlying incidence rates.*** We use county-specific data for baseline all-cause mortality rates from the US Centers for Disease Control and Prevention (https://www.cdc.gov/nchs/data_access/cmf.htm) for years matching the population years above. We use mortality rates for the full population, rather than for a specific age range. Following Apte et al. (28), we calculate the county-average underlying mortality incidence rate, $I_o$, as in Eqs. **6** and **7**:

$$I_{o,c} = \frac{I_c}{\overline{\mathrm{HR}}_c}, \quad [6]$$

$$\overline{\mathrm{HR}}_c = \frac{\sum_{i=1}^{N_c} P_i \times \mathrm{HR}(C_i) f_{i,c}}{\sum_{i=1}^{N} P_i}, \quad [7]$$

where $I_c$ is the reported mortality rate in a given county; $\overline{\mathrm{HR}}_c$ is the average mortality hazard ratio caused by $PM_{2.5}$ in county $c$; $i$ is one of $N_c$ grid cells in county $c$; $P_i$ is population count in grid cell $i$; $\mathrm{HR}(C_i)$ is the result of the concentration–response relationship described below for total $PM_{2.5}$ concentration $C_i$, calculated as described in *$PM_{2.5}$ Concentrations*; and $f_{i,c}$ is the area fraction of grid cell $i$ that overlaps with county $c$. The term $I_{o,c}$ represents a hypothetical mortality incidence rate in the absence of ambient $PM_{2.5}$. For health impact calculations, we assume that the underlying incidence rate for all racial–ethnic groups is the same as the population average. We calculate a US population-average $I_{o,c}$ of 763 deaths per 100,000 people per year in 2014.

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***Concentration–response relationship.*** We represent the effect of changes in $PM_{2.5}$ concentration on mortality rates using the relationship described by Nasari et al. (6) and Burnett et al. (7), as in Eq. **8**:

$$\mathrm{HR}(C) = \exp\left(\frac{\gamma * \ln(C+1)}{1+\exp[-(C-\delta)/\lambda]}\right), \quad [8]$$

where $\mathrm{HR}(C)$ is the hazard ratio of mortality incidence at $PM_{2.5}$ concentration $C$—in units of micrograms per cubic meter—compared with a hypothetical underlying incidence rate, $I_o$, in the absence of ambient $PM_{2.5}$. $\gamma$, $\delta$, and $\lambda$ are empirically determined constants. Nasari et al. use an ensemble version of Eq. **8**, where $\gamma$, $\delta$, and $\lambda$ take many combinations of values and the prediction of each combination is weighted by its performance in predicting health outcomes in the American Cancer Society cohort. To reduce model complexity and computational expense, we use a deterministic version of the relationship, where $\gamma = 0.0478$, $\delta = 6.94$, and $\lambda = 3.37$ are determined using nonlinear regression to predict the expected value of the ensemble prediction. The relationship used here and by Nasari et al. (6) differs from the relationship presented by Burnett et al. (7) in that it is derived from the US-based American Cancer Society cohort rather than from 41 global cohorts.

The term $\mathrm{HR}(C)$ is a nonlinear function; therefore, the impact of a change in concentration depends on the initial concentration. It follows that if a number of emissions sources are consecutively added or subtracted from an area, their health impact per unit emission will depend on the order that they were added or subtracted. We assume that the impact of each unit $PM_{2.5}$ is equal to the average per-unit impact of $PM_{2.5}$ in a given location, as in Eq. **9**:

$$\overline{\mathrm{HR}}_i = \frac{\mathrm{HR}(C_{t,i})}{C_{t,i}}, \quad [9]$$

where $\overline{\mathrm{HR}}_i$ is the average per-unit concentration hazard ratio at location $i$, and $C_{t,i}$ is the total concentration at location $i$.

As a sensitivity analysis, we also use three other hazard ratio models based on the work of Krewski et al. (29) and Lepeule et al. (30), which all take the form shown in Eq. **10**:

$$\mathrm{HR}(C) = \exp(\beta \times \max[0, C - C_o]), \quad [10]$$

where $\beta$ is an empirically determined constant. We use two $\beta$ values reported by Krewski et al. (29): $\beta = \ln(1.06)/10$ and $\beta = \ln(1.078)/10$. We also use $\beta = \ln(1.14)/10$ as reported by Lepeule et al. (30). $C_o$ represents the lowest observed concentration: 5 $\mu g \cdot m^{-3}$ for Krewski et al. (29) and 8 $\mu g \cdot m^{-3}$ for Lepeule et al. (30); our method assumes that for concentrations below this threshold, the risk of $PM_{2.5}$-caused premature mortality is zero.

***Health impact calculation.*** We calculate the health impacts of air pollution using Eq. **11**:

$$M(C_i) = p_i \sum_{c}^{n} I_{o,c} f_{i,c} \overline{\mathrm{HR}}_i, \quad [11]$$

where $M(C_i)$ is the number of mortalities caused by the concentration of pollution $(C_i)$ at location $i$, $p_i$ is the population count in grid cell $i$, $I_{o,c,i}$ is the underlying incidence rate for one of $n$ counties $(c)$ overlapping grid cell $i$, and $f_{i,c}$ is the fraction of grid cell $i$ that overlaps county $c$. We then calculate the $PM_{2.5}$ health impacts, $d$, of economic final demand by combining Eqs. **5** and **11** in Eq. **12**:

$$d = M\left(\sum_{p}\{C_p \hat{\rho}\}\right), \quad [12]$$

where $d$ is a vector with length equaling the number of spatial grid cells and units of [deaths].

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