NRDC
THE EARTH'S BEST DEFENSE

NATURAL RESOURCES DEFENSE COUNCIL

Via Email (eircomment.conventionstadium@lacity.org) and U.S. Mail

May 21, 2012

Case # ENV 2011-0585-EIR
Environmental Analysis Unit
Department of City Planning
200 North Spring Street, City Hall, Room 750
Los Angeles, CA 90012

Re: Comments on Convention and Event Center Project Draft Environmental Impact Report (DEIR), State Clearinghouse No. 2011031049

Dear Environmental Analysis Unit:

On behalf of the Natural Resources Defense Council (NRDC) and its members and activists who live and work in the City of Los Angeles and the surrounding areas, we provide the following comments on the Draft Environmental Impact Report (DEIR) for the proposed Convention and Event Center project, also referred to as the Farmers Field stadium project (the "Project").

NRDC has closely followed the development of the Project and hopes that it will be successful as the greenest and most transit-friendly in the country, and will be in compliance with the letter and spirit of Senate Bill 292 as well as the public commitments that AEG, the Project's sponsor, has made to the Clinton Climate Initiative. However, we are disappointed that the DEIR does not include any analysis of measures that could lead to the project achieving carbon neutrality or fewer people driving their cars to and from the stadium (trip ratio), as SB 292 requires; nor does it include many of the measures in AEG's promises to the Clinton Initiative. We also have concerns about air quality, health risk, green construction practices and sustainability relating to the Project.

SB 292 and CEQA

The California Environmental Quality Act (CEQA) requires that the environmental effects of a project be analyzed and that all reasonably feasible mitigation be implemented. SB 292 did not weaken CEQA or make any changes in what must be analyzed in an EIR. Nor did SB 292 make any changes in the general rule that mitigation measures must be enforceable.

www.nrdc.org
1314 Second Street
Santa Monica, CA 90401
TEL 310 434-2300
FAX 310 434-2399
100% Postconsumer Recycled Paper
NEW YORK • WASHINGTON, DC • SAN FRANCISCO • BEIJING • CHICAGO

Here, while the DEIR includes a list of transportation-related measures that might affect the transportation mode shift and carbon neutrality of the Project, there is no analysis of the actual or potential effectiveness of those measures. Nor is there any commitment that the project proponent, AEG, will put any of these measures in place. These failures are inconsistent with CEQA and with SB 292.

Under the provisions of SB 292, which became law in 2011 and is now codified as Section 21168.6.5 of the Public Resources Code, AEG received the benefit of an expedited judicial review process for any challenge to the Project's EIR in return for promising carbon neutrality and mode shift greater than any other U.S. football stadium. Specifically, subsection (h) of SB 292 provides as follows:

> It is the intent of the Legislature that the *project* minimize traffic congestion and air quality impacts that may result from private automobile trips to the stadium through the requirements of this division as supplemented, pursuant to subdivision (i), by the implementation of measures that will do both of the following:
> (1) Achieve and maintain carbon neutrality by reducing to zero the net emissions of greenhouse gases, as defined in subdivision (g) of Section 38505 of the Health and Safety Code, from private automobile trips to the stadium.
> (2) Achieve and maintain a trip ratio that is no more than 90 percent of the trip ratio at any other stadium serving a team in the National Football League. (emphasis added)

The bill's use of the word "project" was intentional and is highly relevant here. Under CEQA, an EIR must review a "project," and it is well established that the project is the "whole of an action." Guidelines § 15378(a) (definition of "project"). Because the bill clearly describes these measures as features *of the project*, failing to include them in the draft EIR is a violation of CEQA as amended by SB 292.

This analysis is reinforced by the language in subsection (i)(1) of the bill:

> As a condition of approval of the *project* subject to this section, the lead agency shall require the applicant to implement measures that will meet the requirements of this division and paragraph (1) of subdivision (h) by the end of the first season during which a National Football League team has played at the stadium. (emphasis added)

The key term "project" is used again here. More importantly, this language makes it clear that these measures must be included in the City Council's action approving the project, which is expected to occur at the same time the final EIR is certified. The reference in subsection (i)(1) to "this Division" refers to Division 13

of the Public Resources Code, entitled "Environmental Quality," and includes all of CEQA. Thus, as a condition of project approval, the DEIR must tell the public and the City what the mode shift and carbon neutrality provisions are, how effective they will be, and how they will be enforced. SB 292 does not eliminate or change these CEQA requirements.

In particular, the DEIR should analyze the benefits to air quality and GHG emissions from a reasonable range of options, including:

- Ticket bundling and/or season ticket options for access. Given that the majority of teams in the NFL sell out most of their games to season ticket holders, providing options for these patrons in a controlled format that is part of the purchase of season tickets can provide valued patrons with options that save money, enhance the guest experience of the event, or both. For example, instead of selling "parking passes" along with season seats, season ticketholders to Los Angeles NFL football would be provided a series of choices as part of a philosophy of "it's your money, it's your experience." A subscriber would pay for season parking but receive a debit card instead of parking passes. The debit card would be able to be used on public transit, special event shuttle/transit, or even on premium event transport that provides special programming (*i.e.*, a former NFL player or coach discussing the upcoming game with riders). The card would also be accepted at stadium sanctioned souvenir and concession shops, etc. This is the most "market-based" and seemingly passive approach; however, given the expense involved in attending an NFL game coupled with having several choices on how one spends their own money to access the event could prove to be a powerful incentive to people to make different access choices for different games/events.

- Park and ride (Hollywood Bowl style, per bus at capacity). Mode split at the Bowl is approximately 30%.

- Metro buses and other regional bus lines (*e.g.*, the Big Blue Bus) at capacity, assuming that a game-day suitable schedule can be worked out.[1]

- Metro Rail at capacity, assuming that a game-day suitable schedule can be worked out.

- Subway at capacity, assuming that a game-day suitable schedule can be worked out.

[1] Current night and weekend schedules for Metro buses, Metro Rail, Metrolink, DASH and many regional bus operations are unlikely to be able to handle crowds at a Farmers Field event, and so these schedules would need to be modified. The DEIR should identify the cost and other issues associated with this.

- Metrolink at capacity, assuming that a game-day suitable schedule can be worked out.
- Charter buses.
- Shuttles or DASH buses to take people from Union Station to the stadium, assuming that a game-day suitable schedule can be worked out.
- Timing of transfers from Union Station to Metro Rail, buses or the DASH to the stadium.
- Bicycle use.
- Walking.
- Ridesharing.

CEQA also requires disclosure of AEG's backup for the estimates in the DEIR for initial mode use for transit, walking and biking, as well as any plans AEG has to disseminate public transit and ridesharing information to the public. Considerable skepticism was expressed at the May 16, 2012 public hearing on the DEIR as to whether AEG's estimates of transit ridership at other stadiums were accurate or relevant.

With respect to carbon offsets, CEQA requires disclosure of AEG's specific plans, to the extent there are any, for carbon offsets in the Pico-Union neighborhood and in the South Coast air basin in general. Any offsets used to comply with SB 292 should be verified in the same way that cap and trade offsets are verified pursuant to AB 32. *See* Cal. Health and Safety Code § 38560(d);[2] *see also* 17 Cal. Code Regs. § 95977 (requirement for third-party verification of offsets).

These same data are required whether the carbon neutrality and mode shift measures are viewed as mitigation measures or features of the Project. These

[2] Section 38560(d) provides in part as to greenhouse gas reductions, including offsets: "(1) The greenhouse gas emission reductions achieved are real, permanent, quantifiable, verifiable, and enforceable by the state board. (2) For regulations pursuant to Part 5 (commencing with Section 38570), the reduction is in addition to any greenhouse gas emission reduction otherwise required by law or regulation, and any other greenhouse gas emission reduction that otherwise would occur. 3) If applicable, the greenhouse gas emission reduction occurs over the same time period and is equivalent in amount to any direct emission reduction required pursuant to this division." *See also* Section 38505(k), stating that offsets must "result in the same greenhouse gas emission reduction, over the same time period, as direct compliance with a greenhouse gas emission limit or emission reduction measure adopted by the state board."

measures are certainly feasible[3] since AEG agreed to carry them out to meet the targets in SB 292. Moreover, common sense tells us that these could be important mitigation measures to reduce the Project's significant and allegedly unavoidable impacts on air quality and public health locally and regionally. *See* Sec. IV.F, 1-81–1-82. But the DEIR does not undertake this analysis, in violation of CEQA. *See* Guidelines § 15126.4(a); *see also Communities For A Better Environment v. City of Richmond*, 184 Cal. App. 4th 70, 92 (2010) ("Formulation of mitigation measures should not be deferred until some future time." (Guidelines, § 15126.4(a)(1)(b).) An EIR is inadequate if "[t]he success or failure of mitigation efforts ... may largely depend upon management plans that have not yet been formulated, and have not been subject to analysis and review within the EIR.").

The Clinton Global Initiative

AEG made a number of significant public promises to the Clinton Global Initiative about the environmental benefits of the Project[4] and has been publicly praised for these promises by President Clinton[5]. Unfortunately, many of these commitments do not appear in the DEIR.

Below, in italics, are the commitments that AEG made to the Clinton Global Initiative and a discussion of their treatment (if any) in the DEIR.

> *AEG Commitment to Clinton Global Initiative: This commitment expands in detail and scope upon an existing commitment made by AEG to make Farmers Field Stadium carbon neutral in terms of fan transportation. AEG also committed to make Farmers Field Stadium the best in the NFL in terms of highest percent of non-auto fan travel to venue. This commitment also addresses the design, construction, and operations of the venue.*

There are no enforceable measures in the DEIR concerning carbon neutrality or percentage of non-auto fan travel.

---

[3] CEQA Guidelines 15364 defines "feasible" as "capable of being accomplished in a successful manner within a reasonable period of time, taking into account economic, environmental, legal, social, and technological factors."

[4] The Clinton Global Initiative website is: http://www.clintonglobalinitiative.org/commitments/default.asp. AEG's commitments about the Project may be found by entering "AEG" into the Commitment Maker / Partner search box on this page: http://www.clintonglobalinitiative.org/commitments/commitments_search.asp?Section=Commitments.

[5] See http://greeninghollywood.wordpress.com/2011/09/21/clinton-global-initiative-recognizes-aeg-farmers-field/

AEG Commitment to Clinton Global Initiative: 100 percent climate neutral for greenhouse gas emissions from energy consumption and mechanical operations of the stadium

Farmers Field will achieve climate neutrality through the installation of best available technology for HVAC, lighting, and all major electrical appliances and control systems; through on-site generation of clean and renewable energy; through the purchase of renewable energy credits and through educating employees on energy efficiency through their Environmental Management system. Special projects under analysis include: additional solar panels on garages, 100% LED lighting throughout building, and a Carbon Offset Ticket.

There is no analysis in the DEIR of how carbon neutrality will be accomplished. Indeed, the DEIR asserts that no mitigation is required with respect to the effects of the project on climate change. IV.F.2-53.

AEG Commitment to Clinton Global Initiative: Recycling and Waste Diversion

- 90 percent solid waste diverted during construction

- 75 percent generated during operation

Farmers Field will divert waste from landfill through a robust recycling, the donation of durable goods, and implementing a front of house composting program that includes sourcing biodegradable concessions packaging.

The DEIR only promises 50 percent diversion of solid waste during operation and 50-75% during construction.

AEG Commitment to Clinton Global Initiative: 100 percent Carbon Neutral for greenhouse gas emissions from private automobile trips to stadium

Best in NFL by 10% in terms of trip ratio of fans to private automobiles

There are no enforceable commitments in the DEIR for either of these promises.

AEG Commitment to Clinton Global Initiative: Education

Farmers Field and L.A. LIVE will feature educational environmental signage and messaging throughout venues and event scoreboards.

There are no enforceable commitments in the DEIR relating to AEG's educational environmental signage policy.

Green Construction

All of the mitigation measures designed to reduce the air pollution from construction of the project discussed in the DEIR are strong measures that will effectively reduce emissions. *See* DEIR at IV.F.1-73–74. We support all of the discussed mitigation measures. However, there are more feasible and effective mitigation measures that also must be included.

First, while Mitigation Measure F.1-3 requires "trucks and vehicles in loading and unloading queues" to "have their engines turned off after 5 minutes when not in use" (*see* DEIR at IV.F.1-73), there must also be idling limits to 5 minutes or less for trucks and vehicles that are not just in loading and unloading queues and also for construction equipment.

Second, trucks and equipment hauling material such as debris or any fill material operating at the project site or traveling to and from the Project site must be fully covered.

Third, while the DEIR includes mitigation measures requiring the use of cleaner construction equipment, the DEIR does not include any mitigation measures requiring the use of cleaner heavy-duty trucks, despite the fact that, similar to construction equipment, heavy-duty trucks operating at the site and traveling to and from the site will run predominantly on diesel fuel and emit harmful emissions, including diesel particulate matter. To mitigate the air emissions from heavy-duty trucks, the following mitigation measures must be included:

Prior to December 31, 2012, all on-road heavy-duty diesel trucks with a gross vehicle weight rating ("GVWR") of 19,500 pounds or greater shall meet or exceed the EPA 2007 on-road emission standards for particulate matter ("PM") (0.01 g/bhp-hr); or shall be equipped with a California Air Resources Board ("CARB") verified Level 3 diesel particulate filter.

From January 1, 2014 and onwards, all on-road heavy-duty diesel trucks with a GVWR of 19,500 pounds or greater shall comply with U.S. Environmental Protection Agency ("EPA") 2007 on-road

emission standards for PM and NOx (0.01 g/bhp-hr and at least 1.2 g/bhp-hr, respectively).

Sustainability

In the DEIR, AEG promises 35% water reduction, 14% energy performance improvement, 50% solid waste diverted during operation, 12 electric vehicle charging stations, 250 bicycle spaces, and a solar array system in the LA Live Way Garage. Similar promises were made to the Clinton Global Initiative. However, the details of these proposals are unclear or missing and we believe that more can and must be done. Below we list measures that other venues have put in place (including at the Portland Rose Garden, which is operated by AEG) that AEG can and must implement here.

Clean Energy: AEG has not made a commitment to use only renewable/clean energy. Portland's Rose Garden gets 100% of its electricity from renewable sources.

Lincoln Financial Field in Philadelphia plans to build 11,000 solar panels and 14 micro wind-turbines combined with a dual-fuel cogeneration plant that uses heat, biodiesel and natural gas to serve all energy needs and sell energy back to the grid. Currently, they buy 100% of their electricity from renewable sources (14 million kWh in wind energy credits annually from NativeEnergy), and have an on-site solar energy system at their NovaCare Complex training facility that generates 16,000 kWh per year. Eagles' employees also receive incentives to switch from conventional power to wind energy at home.

In 2011 Century Link Field in Seattle installed a 3,750-panel solar array that spans 2.5 acres of the event center's roof. The system is projected to produce 830,000 kWh annually. The panels use cutting-edge thin-film technology that capture both direct and reflected sunlight across a 360-degree cylinder surface, taking advantage of the event center's reflective roofing material.

NASCAR's Pocono Raceway in Long Pond, Pennsylvania installed a 25-acre solar farm with a 3-MW system, providing electricity for the entire raceway facility and 1000 homes nearby—between 3 to 4 million kWh per year. This installation, operating since August 2010, is currently the largest solar installation at a major US sports venue and the first that powers a major US sports facility entirely by on-site renewable energy.

The Kaohsiung World Stadium in Taiwan is the first stadium in the world to be 100% powered by solar. Completed in 2011, the stadium's 8,844-panel PV array generates an estimated 1.14 million kWh per year, much of which is sent back to the local grid to power nearby communities.

The Philadelphia Phillies have been a member of EPA's Green Power Partnership since 2008, and have purchased 20 million kilowatt-hours of Green-e certified renewable energy credits (RECs) to cover 100% of their energy needs over the last five years.

The Cleveland Indians installed an innovative helical wind micro-turbine in March piloting a new wind technology in partnership with Cleveland State University's Fenn College of Engineering, estimated to produce 40,000 kWh annually. The Indians also have a 42-panel solar photovoltaic (PV) array that produces 10,000 kWh each year.

Diversion of waste during operation: AEG states in the DEIR that it will divert 50% of waste during operations; AEG promised the Clinton Initiative that it would divert 75% of such waste.

- The San Francisco Giants at AT&T Park divert over 85% of their waste for recycling and composting.

- The Seattle Mariners currently divert over 81% of their waste for recycling and composting. Much of their success has resulted from eliminating most items that cannot be recycled and composted from their procurement altogether—a strategy that they hope will result in a 90% + diversion rate this year.

- Century Link Field diverted 70% of its waste for recycling and composting in 2011, and aims to divert 80% in 2012–2013.

- The Rose Garden diverts 60% of its waste from landfills through its recycling and composting programs, totaling over 800 tons annually.

- Lincoln Financial Field diverts 65% of the stadium's waste from landfills as of 2012.

- Australia's ANZ Stadium has a 100% closed-loop recycling program where all trash is collected and separated for recycling. All of their products are made from recycled materials and can be recycled themselves.

Composting: AEG has only promised front of house (attendees, voluntary) composting but not back of house (staff) composting. Colorado Convention Center provides for both. Lincoln Financial Field even composts its grass cuttings and recycles the oil from their kitchens and vendors for biodiesel. Safeco Field, Century Link Field, and AT&T Park have comprehensive front and back of house composting programs that contribute significantly to their high diversion rates. The San Diego Padres have partnered with Buster Biofuels to provide biodiesel fuel for local school buses by recycling oil and grease from concessions at the ballpark.

Construction: AEG's construction will use a minimum of 20% recycled materials and 5% fast growing materials. Other stadia have exceeded this rate:

- Citi Field contains 12,500 tons of structural steel, about 95 percent of which is recycled. The stadium also has 65,000 square feet of porous pavers and a large drainage bed near the commuter bus parking lot to reduce storm water runoff. It has metered hands-free faucets, toilet flush-o-meters and waterless urinals, which conserve millions of gallons of water each year.

- The Miami Marlins' LEED Silver construction of Marlins Park incorporated over 40% of pre- and post-consumer recycled materials. They also sourced 51% of their construction materials from sources within 500 miles of the stadium site.

During construction of MetLife Stadium in New York, all equipment ran on low-sulfur diesel fuel and had filters to reduce emissions. The stadium includes structural steel and rebar made from 24,280 tons of scrap. Its 8,000 piles were also made from scrap. About 10 percent of the exterior aluminum wall slats contain recycled material, and general seating is made of 20 percent post-consumer plastics.

Ford Field used 20 million pounds of recycled steel, recycled glass floors, recycled rubber for parking lots, and bamboo for floors and elevators. New Meadowlands Stadium has seats made from partially-recycled plastic and scrap iron. New England Patriots took crushed old stadium concrete and reused it for parking lots. Some stadia have used PEFC-certified timber. *See* Green Sports Alliance, London Olympic Park Awarded FSC Certification, http://www.greensportsalliance.org/news-feed/london-olympic-park-awarded-fsc-certification (last visited May 18, 2012).

Green Roofs: Farmers Field will have a retractable roof, but the new convention center can have a green roof. Washington, D.C.'s Nationals Ballpark (6,300 square feet) and Prince's Park Stadium in the United Kingdom have green roofs, as will the new Santa Clara Stadium near San Francisco (opening in 2014). The 2.5-acre green roof atop the Target Center, home to the Minnesota Timberwolves, captures about a million gallons of stormwater per year, saving $10,000 annually in stormwater charges. The New York Mets' administration building at Citi Field features a 15,000-square foot green roof.

Construction waste: The Farmers Field Stadium has committed to recycling 50–75% of all construction waste. AEG promised the Clinton Initiative that it would recycle 90%. In comparison, the Rose Garden in 1995 recycled more than half of its construction waste, amounting to nearly 36,000 tons. *See* http://infohouse.p2ric.org/ref/26/25329.pdf. The Miami Marlins' LEED Silver construction of Marlins Park diverted 98% of all construction wastes from landfills

to recycling facilities.

Event Center Food: AEG does not discuss this issue in the DEIR. The Rose Garden provides a line of locally produced food and beverages and has 95% compostable/100% recycled food and beverage containers. The current Los Angeles Convention Center has a 100% environmentally friendly approach to food: biodegradable or composted service ware, a food waste diversion program, and a sustainable menu with 100% free range organic grass-fed beef and sustainable seafood. Lincoln Financial Field uses corn-based cups that can biodegrade in 50 days as well as corn-plastic dishes/utensils. Safeco Field uses compostable food and service ware specified by their composting hauler, and offers local and organic food and beverages.

At the new Yankee stadium, management uses biodegradable beverage cups; it also composts material — including grass clippings from the outfield — reducing trash hauled to landfills by 40 percent. After each homestand, the stadium donates boxes of unused food through a program called Rock and Wrap It Up. Management also recycles the waste cooking oil from the stadium — 20,000 gallons last year was converted to 18,000 gallons of biodiesel fuel.

Electronic Ticketing/Recycled Paper: AEG has made no commitment to use electronic ticketing or print on recycled paper. The Rose Garden provides electronic ticketing. Lincoln Financial Field uses recycled paper for tickets and programs. All Red Sox publications, tickets and print are recycled paper (including media guides, pocket schedules, yearbooks, programs, etc).

Other Measures: Lincoln Financial Field's website allows fans to see the actual kilowatt hours from the team's solar panels to promote awareness. At MetLife Stadium, home to the New York Giants and Jets, a "Solar Ring" installation atop the stadium will generate 25 times the power needed to operate the stadium on game days with a 1500-panel solar array. The Eagles buy offsets for the carbon emissions they accumulate while flying across the country and allow ticket holders to purchase renewable energy offsets when purchasing tickets.

The Minnesota Twins earned several LEED points-including an exemplary performance and innovation point-for its comprehensive Green Cleaning program. In 2011, the Twins reduced the usage of chemical cleaning compounds by 66 percent over 2010, using a total of 73 percent of cleaning compounds that met the USGBC's LEED standards. Progressive Field features a solar pavilion with real-time energy generation

Local and Regional Air Quality and Health Risk

The DEIR projects significant air quality impacts locally and regionally.

> Regional operational emissions would still exceed the SCAQMD daily emission threshold for regional NOX, VOC, PM10 PM2.5, and CO after implementation of feasible mitigation measures. Therefore, operation of the Proposed Project would have a significant and unavoidable impact on regional air quality. Cumulative operational air quality impacts would also remain significant. (IV.F 1-81)

> Localized operational emissions would also still exceed their respective state and federal thresholds. Therefore, with respect to localized emissions from operational activities, PM10 and NOX impacts would be significant and unavoidable. (IV.F 1-81)

> During Proposed Project operations significant localized PM10 concentration levels would occur in certain areas surrounding the Project Site. (IV.F. 1-82)

The Project-related pollutants that cause these exceedances are overwhelmingly from mobile sources – that is, fans driving to and from the stadium. 95% of the PM2.5, 97% of the SOx and 88% of the NOx from "event days" are from these sources.[6] But instead of reducing the use of mobile sources, the DEIR shows that AEG is facilitating them by, for example, proposing improvements to the Hollywood Freeway and building additional parking structures.

Remarkably, given the predicted air pollution levels,[7] pollution from mobile sources other than trucks using the Project's loading docks is not analyzed in the

[6] Table IV.F.1-7 at IV.F.1-41.

[7] Actual levels may be greater than analyzed because of the DEIR's failure to consider a scenario or scenarios that involve an NFL stadium event with an overlapping basketball or hockey capacity event at the Staples Center. Since there are two pro basketball and one pro hockey teams based at Staples, and since hockey and basketball seasons overlap all but the first month of the football season and since each of the basketball and hockey teams play upwards of 40 home games, it is not unlikely that such combination events would occur and they actually might be fairly frequent. Combination event scenarios that should have been considered include afternoon basketball or hockey with evening football, afternoon football with evening basketball or hockey, simultaneous afternoon football with either basketball or hockey and simultaneous evening football with either basketball or hockey. AEG claims that it will be able to coordinate events at Staples Center, the Convention Center and Farmers Field to eliminate such overlap, but does not say

Health Risk Assessment in the DEIR, making that document essentially worthless as well as in violation of CEQA Guideline 15126.2(a) and *Bakersfield Citizens for Local Control v. City of Bakersfield*, 124 Cal.App.4th 1184, 1219-1220 (2004).

It is our understanding that AEG's reason for not including non-diesel emissions in the Health Risk Assessment is that gasoline-driven vehicles would not emit substantive amounts of air toxics. This same reasoning was rejected in *Bakersfield Citizens*, in which an EIR found adverse air quality impacts derived "primarily from automobile emissions during operation and from architectural coatings and construction equipment during construction phase" but those emissions were not analyzed for health risk. The Court of Appeal rejected this approach, explaining:

> Guidelines section 15126.2, subdivision (a) requires an EIR to discuss, inter alia, "health and safety problems caused by the physical changes" that the proposed project will precipitate. Both of the EIR's concluded that the projects would have significant and unavoidable adverse impacts on air quality. It is well known that air pollution adversely affects human respiratory health. (See, e.g., Bustillo, *Smog Harms Children's Lungs for Life, Study Finds*, L.A. Times (Sept. 9, 2004).) Emergency rooms crowded with wheezing sufferers are sad but common sights in the San Joaquin Valley and elsewhere. Air quality indexes are published daily in local newspapers, schools monitor air quality and restrict outdoor play when it is especially poor and the public is warned to limit their activities on days when air quality is particularly bad. Yet, neither EIR acknowledges the health consequences that necessarily result from the identified adverse air quality impacts. Buried in the description of some of the various substances that make up the soup known as "air pollution" are brief references to respiratory illnesses. However, there is no acknowledgement or analysis of the well-known connection between reduction in air quality and increases in specific respiratory conditions and illnesses. After reading the EIR's, the public would have no idea of the health consequences that result when more pollutants are added to a nonattainment basin. On remand, the health impacts resulting from the adverse air quality impacts must be identified and analyzed in the new EIR's.

It is well-known that non-diesel mobile source air emissions such as ozone and its precursors (NOx, SOx, VOCs) contribute to respiratory and other health problems. *See, e.g., Traffic-related air pollution and asthma in economically disadvantaged and high traffic density neighborhoods in Los Angeles County,*

---

how, or what enforcement measures will be in place in the face of resistance by the sports leagues or national television networks.

California, available at http://www.arb.ca.gov/research/single-project.php?row_id=64715; the California Air Resources Board Children's Health Study, available at http://www.arb.ca.gov/research/chs/chs.htm#new; Gauderman, et al., *The Effect of Air Pollution on Lung Development from 10 to 18 Years of Age*, 351 N Eng Journal Med 1057-1067 (2004); Health Effects Institute, *Traffic-Related Air Pollution: A Critical Review of the Literature on Emissions, Exposure, and Health Effects* (2010), available at http://pubs.healtheffects.org/view.php?id=334; USEPA *Health Effects of Ozone in the General Population*, available at http://www.epa.gov/apti/ozonehealth/population.html; USEPA, *Health Effects of Nitrogen Dioxide*, available at http://www.epa.gov/air/nitrogenoxides/health.html; USEPA, *Health Effects of Sulfur Dioxide*, available at http://www.epa.gov/air/sulfurdioxide/health.html; US EPA, *Ozone Health Risk Assessment for Selected Urban Areas* (2007), available at http://www.epa.gov/ttnnaaqs/standards/ozone/data/2007-01_ozone_ra_final_tsd.pdf; US EPA, *Risk and Exposure Assessment to Support the Review of the NO2 Primary National Ambient Air Quality Standard* (2008), available at http://www.epa.gov/ttn/naaqs/standards/nox/data/20081121_NO2_REA_final.pdf; US EPA, *Risk and Exposure Assessment to Support the Review of the SO2 Primary National Ambient Air Quality Standards: First Draft* (2008), available at http://www.epa.gov/ttnnaaqs/standards/so2/data/2008_06_rea_firstdraft.pdf.[8]

Here are the primary results of the Children's Health Study, as reported[9] by CARB, the study's sponsor:

- Air pollution harms children's lungs for life. Children exposed to higher levels of particulate matter, nitrogen dioxide, acid vapor and elemental carbon, had significantly lower lung function at age 18, an age when the lungs are nearly mature and lung function deficits are unlikely to be reversed.

- Children that were exposed to current levels of air pollution had significantly reduced lung growth and development when exposed to higher levels of acid vapor, ozone, nitrogen dioxide and particulate matter which is made up of very small particles that can be breathed deeply into the lungs.

---

[8] See also Delfino, *Repeated hospital encounters for asthma in children and exposure to traffic-related air pollution near the home*, 102 Annals of Allergy, Asthma & Immunology 138-144(February, 2009); Guaderman, *Childhood Asthma and Exposure to Traffic and Nitrogen Dioxide*, 16 Epidemiology 737-743 (2005); Brugge, *Near-highway pollutants in motor vehicle exhaust: A review of epidemiologic evidence of cardiac and pulmonary health risks*, Environmental Health (2007). Copies of these articles are being submitted with this comment letter.

[9] See http://www.arb.ca.gov/research/chs/over.htm.

- Children living in high ozone communities who actively participated in several sports were more likely to develop asthma than children in these communities not participating in sports.

- Children living in communities with higher concentrations of nitrogen dioxide, particulate matter, and acid vapor had lungs that both developed and grew more slowly and were less able to move air through them. This decreased lung development may have permanent adverse effects in adulthood.

- Children who moved away from study communities had increased lung development if the new communities had lower particulate matter levels, and had decreased lung development if the new communities had higher particulate matter levels.

- Days with higher ozone levels resulted in significantly higher school absences due to respiratory illness.

- Children with asthma who were exposed to higher concentrations of particulate matter were much more likely to develop bronchitis.

In view of these data and studies, the Health Risk Analysis in the DEIR needs to be re-done to analyze the risk posed by non-diesel emissions from mobile sources associated with the Project.

Moreover, the air pollution effects reported in the DEIR, which will have their greatest impact on the nearby Pico-Union neighborhood, are not unavoidable. For example, the mobile source emissions modeling is based on an assumed[10] 26-mile trip length for fans attending events at the new stadium. That trip length can be substantially lessened, or essentially eliminated, if affordable, efficient public transit and other measures that we have discussed in this letter are put in place. Claiming that failure to analyze such measures is a conservative approach does not mask the fact that the air quality, and health, problems that will be inflicted on the Pico-Union neighborhood and others by the Project can be mitigated.

[10] "The DEIR analysis was based on information provided by the Traffic Consultant in which it was assumed 10 miles for worker commute, 8 miles for patrons at the Convention Center, and 26 miles for events at the Event Center. Appendix M-1 at 200.

In conclusion, the DEIR needs to be rewritten and then re-circulated when the problems we have identified are fixed. Thank you for considering our comments.

Very truly yours,

David Pettit
Senior Attorney
Director, Southern California Air Program

Damon Nagami
Staff Attorney

Environmental Health

BioMed Central

Review

Open Access

Near-highway pollutants in motor vehicle exhaust: A review of epidemiologic evidence of cardiac and pulmonary health risks

Doug Brugge*[1], John L Durant[2] and Christine Rioux[3]

Address: [1]Tufts Community Research Center, Tufts University School of Medicine, Boston, MA, USA, [2]Department of Civil and Environmental Engineering, Tufts University, Medford, MA 02155, USA and [3]Interdisciplinary PhD Program, Tufts University, Medford, MA 02155, USA

Email: Doug Brugge* - dbrugge@aol.com; John L Durant - john.durant@tufts.edu; Christine Rioux - Christine.rioux@tufts.edu

* Corresponding author

Published: 9 August 2007

Environmental Health 2007, **6**:23 doi:10.1186/1476-069X-6-23

Received: 2 January 2007
Accepted: 9 August 2007

This article is available from: http://www.ehjournal.net/content/6/1/23

Abstract

There is growing evidence of a distinct set of freshly-emitted air pollutants downwind from major highways, motorways, and freeways that include elevated levels of ultrafine particulates (UFP), black carbon (BC), oxides of nitrogen (NOx), and carbon monoxide (CO). People living or otherwise spending substantial time within about 200 m of highways are exposed to these pollutants more so than persons living at a greater distance, even compared to living on busy urban streets. Evidence of the health hazards of these pollutants arises from studies that assess proximity to highways, actual exposure to the pollutants, or both. Taken as a whole, the health studies show elevated risk for development of asthma and reduced lung function in children who live near major highways. Studies of particulate matter (PM) that show associations with cardiac and pulmonary mortality also appear to indicate increasing risk as smaller geographic areas are studied, suggesting localized sources that likely include major highways. Although less work has tested the association between lung cancer and highways, the existing studies suggest an association as well. While the evidence is substantial for a link between near-highway exposures and adverse health outcomes, considerable work remains to understand the exact nature and magnitude of the risks.

Background

Approximately 11% of US households are located within 100 meters of 4-lane highways [estimated using: [1,2]]. While it is clear that automobiles are significant sources of air pollution, the exposure of near-highway residents to pollutants in automobile exhaust has only recently begun to be characterized. There are two main reasons for this: (A) federal and state air monitoring programs are typically set up to measure pollutants at the regional, not local scale; and (B) regional monitoring stations typically do not measure all of the types of pollutants that are elevated next to highways. It is, therefore, critical to ask what is known about near-highway exposures and their possible health consequences.

Here we review studies describing measurement of near-highway air pollutants, and epidemiologic studies of cardiac and pulmonary outcomes as they relate to exposure to these pollutants and/or proximity to highways. Although some studies suggest that other health impacts are also important (e.g., birth outcomes), we feel that the case for these health effects are less well developed scientifically and do not have the same potential to drive public policy at this time. We did not seek to fully integrate the relevant cellular biology and toxicological literature, except for a few key references, because they are so vast by themselves.

We started with studies that we knew well and also searched the engineering and health literature on Medline. We were able to find some earlier epidemiologic studies based on citations in more recent articles. We include some studies that assessed motor vehicle-related pollutants at central site monitors (i.e., that did not measure highway proximity or traffic) because we feel that they add to the plausibility of the associations seen in other studies. The relative emphasis given to studies was based on our appraisal of the rigor of their methodology and the significance of their findings. We conclude with a summary and with recommendations for policy and further research.

Motor vehicle pollution

It is well known that motor vehicle exhaust is a significant source of air pollution. The most widely reported pollutants in vehicular exhaust include carbon monoxide, nitrogen and sulfur oxides, unburned hydrocarbons (from fuel and crankcase oil), particulate matter, polycyclic aromatic hydrocarbons, and other organic compounds that derive from combustion [3-5]. While much attention has focused on the transport and transformation of these pollutants in ambient air – particularly in areas where both ambient pollutant concentrations and human exposures are elevated (e.g., congested city centers, tunnels, and urban canyons created by tall buildings), less attention has been given to measuring pollutants and exposures near heavily-trafficked highways. Several lines of evidence now suggest that steep gradients of certain pollutants exist next to heavily traveled highways and that living within these elevated pollution zones can have detrimental effects on human health.

It should be noted that many different types of highways have been studied, ranging from California "freeways" (defined as multi-lane, high-speed roadways with restricted access) to four-lane (two in each direction), variable-speed roadways with unrestricted access. There is considerable variation in the literature in defining highways and we choose to include studies in our review that used a broad range of definitions (see Table 1).

It should also be noted that there may be significant heterogeneity in the types and amounts of vehicles using highways. The typical vehicle fleet in the US is composed of passenger cars, sports utility vehicles, motorcycles, pickup trucks, vans, buses, and small, medium, and large trucks. The composition and size of a fleet on a given highway may vary depending on the time of day, day of the week, and use restrictions for certain classes of vehicles. Fleets may also vary in the average age and state of repair of vehicles, the fractions of vehicles that burn diesel and gasoline, and the fraction of vehicles that have catalytic converters. These factors will influence the kinds and amounts of pollutants in tailpipe emissions. Similarly, driving conditions, fuel chemistry, and meteorology can also significantly impact emissions rates as well as the kinds and concentrations of pollutants present in the near-highway environment. These factors have rarely been taken into consideration in health outcome studies of near-highway exposure.

Based on our review of the literature, the pollutants that have most consistently been reported at elevated levels near highways include ultrafine particles (UFP), black carbon (BC), nitrogen oxides (NOx), and carbon monoxide (CO). In addition, $PM_{2.5}$, and PM_{10} were measured in many of the epidemiologic studies we reviewed. UFP are defined as particles having an aerodynamic diameter in the range of 0.005 to 0.1 microns (um). UFP form by condensation of hot vapors in tailpipe emissions, and can grow in size by coagulation. $PM_{2.5}$ and PM_{10} refer to particulate matter with aerodynamic diameters of 2.5 and 10 um, respectively. BC (or "soot carbon") is an impure form of elemental carbon that has a graphite-like structure. It is the major light-absorbing component of combustion aerosols. These various constituents can be measured in real time or near-real time using particle counters (UFP) and analyzers that measure light absorption (BC and CO), chemiluminescence (NOx), and weight ($PM_{2.5}$ and PM_{10}). Because UFP, NO_x, BC, and CO derive from a common source – vehicular emissions – they are typically highly inter-correlated.

Air pollutant gradients near highways

Several recent studies have shown that sharp pollutant gradients exist near highways. Shi et al. [6] measured UFP number concentration and size distribution along a roadway-to-urban-background transect in Birmingham (UK), and found that particle number concentrations decreased nearly 5-fold within 30 m of a major roadway (>30,000 veh/d). Similar observations were made by Zhu et al. [7,8] in Los Angeles. Zhu et al. measured wind speed and direction, traffic volume, UFP number concentration and size distribution as well as BC and CO along transects downwind of a highway that is dominated by gasoline vehicles (Freeway 405; 13,900 vehicles per hour; veh/h) and a highway that carries a high percentage of diesel vehicles (Freeway 710; 12,180 veh/h). Relative concentrations of CO, BC, and total particle number concentration decreased exponentially between 17 and 150 m downwind from the highways, while at 300 m UFP number concentrations were the same as at upwind sites. An increase in the relative concentrations of larger particles and concomitant decrease in smaller particles was also observed along the transects (see Figure 1). Similar observations were made by Zhang et al. [9] who demonstrated "road-to-ambient" evolution of particle number distributions near highways 405 and 710 in both winter and sum-

Table 1: Summary of near-highway pollution gradients

Citation	Location	Highway traffic intensity[a]	Pollutants measured[b]	Observed Pollution Gradients
Shi et al. 1999 (6)	Birmingham, UK	30,000 veh/d	UFP + FP (10-10^4 nm)	2–100 m [c]
Zhu et al. 2002 (8)	Los Angeles; Freeway 710	12,180 veh/h	UFP, CO, BC	17–300 m [c]
Zhu et al. 2002 (7)	Los Angeles; Freeway 405	13,900 veh/h	UFP, CO, BC	30–300 m [c]
Hitchins et al. 2002 (11)	Brisbane (Austr.)	2,130–3,400 veh/h	UFP + FP (15-2 × 10^4 nm), $PM_{2.5}$	15–375 m [c]
Fischer et al. 2000 (13)	Amsterdam	<3,000–30,974 veh/d	$PM_{2.5}$, PM_{10}, PPAH, VOCs	NA
Roorda-Knape et al. 1998 (14)	Netherlands	80,000–152,000 veh/d	$PM_{2.5}$, PM_{10}, BC, VOCs, NO_2	15–330 m [c]
Janssen et al. 2001 (15)	Netherlands	40,000–170,000 veh/d	$PM_{2.5}$, VOCs, NO_2	< 400 m [c]
Morawska et al. 1999 (12)	Brisbane (Austr.)	NA	UFP	10–210 m [c]

[a]As defined in article cited (veh/d = vehicles per day; veh/h = vehicles per hour).
[b]UFP = ultrafine particles; FP = fine particles; $PM_{2.5}$ = particles with aerodynamic diameter ≤ 2.5 um; PM_{10} = particles with aerodynamic diameter ≤ 10 um; BC = black carbon; PPAH = particle-bound polycyclic aromatic hydrocarbons; VOCs = volatile organic compounds
[c]Pollutant measurements were made along a transect away from the highway
NA = not applicable; measurements were not made.

mer. Zhang et al. observed that between 30–90 m downwind of the highways, particles grew larger than 0.01 um due to condensation, while at distances >90 m, there was both continued particle growth (to >0.1 um) as well as particle shrinkage to <0.01 um due to evaporation. Because condensation, evaporation, and dilution alter size distribution and particle composition, freshly-emitted UFP near highways may differ in chemical composition from UFP that has undergone atmospheric transformation during transport to downwind locations [10].

Two studies in Brisbane (Australia) highlight the importance of wind speed and direction as well as contributions of pollutants from nearby roadways in tracking highway-generated pollutant gradients. Hitchins et al. [11] measured the mass concentrations of 0.1–10 um particles as well as total particle number concentration and size distribution for 0.015–0.7 um particles near highways (2,130–3,400 veh/h). Hitchens et al. observed that the distance from highways at which number and mass concentrations decreased by 50% varied from 100 to 375 m depending on the wind speed and direction. Morawska et al. [12] measured the changes in UFP number concentrations along horizontal and vertical transects near highways to distinguish highway and normal street traffic contributions. It was observed that UFP number concentrations were highest <15 m from highways, while 15–200 m from highways there was no significant difference in UFP number concentrations along either horizontal or vertical transects – presumably due to mixing of highway pollutants with emissions from traffic on nearby, local roadways.

In addition to UFP, other pollutants – such as $PM_{2.5}$, PM_{10}, NO_2 (nitrogen dioxide), VOCs (volatile organic compounds), and particle-bound polycyclic aromatic hydrocarbons (PPAH) – have been studied in relation to heavily-trafficked roadways. Fischer et al. [13] measured $PM_{2.5}$, PM_{10}, PPAH, and VOC concentrations outside and inside homes on streets with high and low traffic volumes in Amsterdam (<3,000–30,974 veh/d). In this study, PPAH and VOCs were measured using methods based on gas chromatography. Fischer et al. found that while $PM_{2.5}$ and PM_{10} mass concentrations were not specific indicators of traffic-related air pollution, PPAH and VOC levels were ~2-fold higher both indoor and outdoor in high traffic areas compared to low traffic areas. Roorda-Knape et al [14] measured $PM_{2.5}$, PM_{10}, black smoke (which is similar to BC), NO_2, and benzene in residential areas <300 m from highways (80,000–152,000 veh/d) in the Netherlands. Black smoke was measured by a reflectance-based method using filtered particles; benzene was measured using a method based on gas chromatography. Roorda-Knape et al reported that outdoor concentrations of black smoke and NO_2 decreased with distance from highways, while $PM_{2.5}$, PM_{10}, and benzene concentrations did not change with distance. In addition, Roorda-Knape et al. found that indoor black smoke concentrations were correlated with truck traffic, and NO_2 was correlated with both traffic volume and distance from highways. Janssen et al. [15] studied $PM_{2.5}$, PM_{10}, benzene, and black smoke in 24 schools in the Netherlands and found that $PM_{2.5}$ and black smoke increased with truck traffic and decreased with distance from highways (40,000–170,000 veh/d).

In summary, the literature shows that UFP, BC, CO and NOx are elevated near highways (>30,000 veh/d), and that other pollutants including VOCs and PPAHs may also be elevated. Thus, people living within about 30 m of highways are likely to receive much higher exposure to

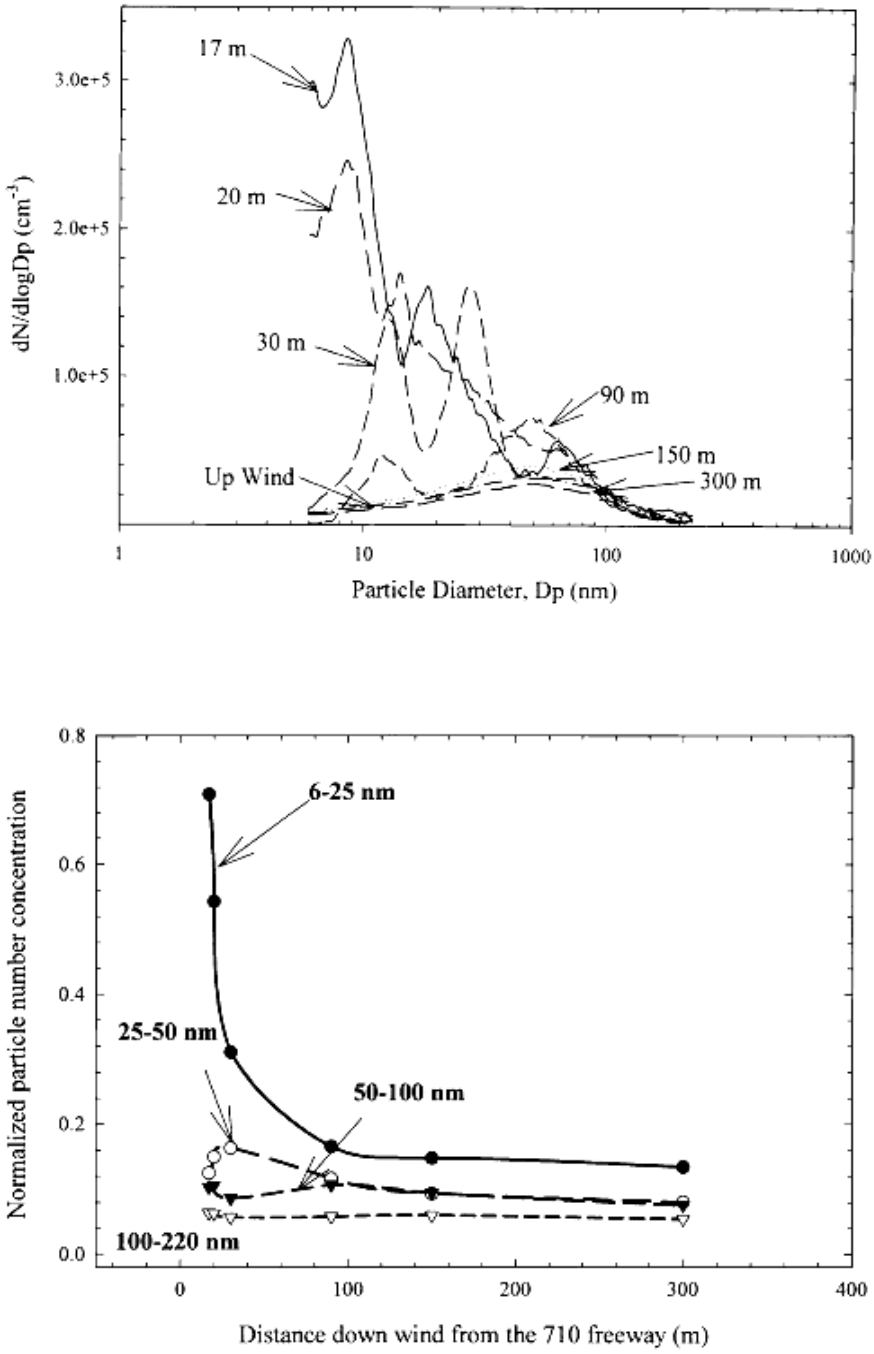

Figure 1
Ultrafine particle size distribution (top panel) and normalized particle number concentration for different size ranges (bottom panel) as a function of distance from a highway in Los Angeles. From Zhu et al. (8). Reprinted with permission from Elsevier.

traffic-related air pollutants compared to residents living >200 m (+/- 50 m) from highways.

Cardiovascular health and traffic-related pollution

Results from clinical, epidemiological, and animal studies are converging to indicate that short-term and long-term exposures to traffic-related pollution, especially particulates, have adverse cardiovascular effects [16-18]. Most of these studies have focused on, and/or demonstrated the strongest associations between cardiovascular health outcomes and particulates by weight or number concentrations [19-21] though CO, SO_2, NO_2, and BC have also been examined. BC has been shown to be associated with decreases in heart rate variability (HRV) [22,23] and black smoke and NO_2 shown to be associated with cardiopulmonary mortality [24].

Short-term exposure to fine particulate pollution exacerbates existing pulmonary and cardiovascular disease and long-term repeated exposures increases the risk of cardiovascular disease and death [25,26].

Though not focused on near-highway pollution, two large prospective cohort studies, the Six-Cities Study [27] and the American Cancer Society (ACS) Study [28] provided the groundwork for later research on fine particulates and cardiovascular disease. Both of these studies found associations between increased levels of exposure to ambient PM and sulfate air pollution recorded at central city monitors and annual average mortality from cardiopulmonary disease, which at the time combined cardiovascular and pulmonary disease other than lung cancer. The Six-Cities Study examined $PM_{2.5}$ and $PM_{10/15}$. The ACS study examined $PM_{2.5}$. Relative risk ratios of mortality from cardiopulmonary disease comparing locations with the highest and lowest fine particle concentrations (which had differences of 24.5 and 18.6 ug/m^3 respectively) were 1.37 (1.11, 1.68) and 1.31 (1.17, 1.46) in the Six Cities and ACS studies, respectively. These analyses controlled for many confounders, including smoking and gas stoves but not other housing conditions or time spent at home. The studies were subject to intensive replication, validation, and reanalysis that confirmed the original findings. $PM_{2.5}$ generally declined following implementation of new US Environmental Protection Agency standards in 1997 [17,29], yet since that time studies have shown elevated health risks due to long-term exposures to the 1997 PM threshold concentrations [29,30].

Much of the epidemiological research has focused on assessing the early physiological responses to short-term fluctuations in air pollution in order to understand how these exposures may alter cardiovascular risk profiles and exacerbate cardiovascular disease [31]. Heart rate variability, a risk factor for future cardiovascular outcomes, is altered by traffic-related pollutants particularly in older people and people with heart disease [22,23,32]. With decreased heart rate variability as the adverse outcome, negative associations between HRV and particulates were strongest for the smallest size fraction studied [33] (PM0.3–1.0); [34] (PM0.02–1). In two studies that included other pollutants, black carbon, an indicator of traffic particles, also elicited a strong association with both time and frequency domain HRV variables; associations were also strong for PM2.5 for both time and frequency HRV variables in the Adar et al study [[23]; this and subsequent near highway studies are summarized in Table 2], however, PM2.5 was not associated with frequency domain variables in the Schwartz et al. study [22].

Several studies show that exposure to PM varies spatially within a city [35-37], and finer spatial analyses show higher risks to individuals living in close proximity to heavily trafficked roads [18,37]. A 2007 paper from the Woman's' Health Initiative used data from 573 $PM_{2.5}$ monitors to follow over 65,000 women prospectively. They reported very high hazard ratios for cardiovascular events (1.76; 95% CI, 1.25 to 2.47) possibly due to the fine grain of exposure monitoring [18]. In contrast, studies that relied on central monitors [27,28] or interpolations from central monitors to highways are prone to exposure misclassification because individuals living close to highways will have a higher exposure than the general area. A possible concern with this interpretation is that social gradients may also situate poorer neighborhoods with potentially more susceptible populations closer to highways [38-40].

At a finer grain, Hoek et al. [24] estimated home exposure to nitrogen dioxide (NO_2) and black smoke for about 5,000 participants in the Netherlands Cohort Study on Diet and Cancer. Modeled exposure took into consideration proximity to freeways and main roads (100 m and 50 m, respectively). Cardiopulmonary mortality was associated with both modeled levels of pollutants and living near a major road with associations less strong for background levels of both pollutants. A case-control study [41], found a 5% increase in acute myocardial infarction associated with living within 100 m of major roadways. A recent analysis of cohort data found that traffic density was a predictor of mortality more so than was ambient air pollution [42]. There is a need for studies that assess exposure at these scales, e.g., immediate vicinity of highways, to test whether cardiac risk increases still more at even smaller scales.

Although we cannot review it in full here, we note that evidence beyond the epidemiological literature support the contention that $PM_{2.5}$ and UFP (a sub-fraction of $PM_{2.5}$) have adverse cardiovascular effects [16,17]. $PM_{2.5}$ appears

Table 2: Summary of near-highway health effects studies

Citation	Location	Highway traffic intensity[a]	Pollutants measured[b]	Distance from highway	Health Outcomes	Statistical association[e]
Schwartz et al. 2005 (22)	Boston	NA	$PM_{2.5}$, BC, CO	NA	Heart rate variability	Decreases in measures of heart rate variability
Adar et al. 2007 (23)	St. Louis, Missouri	NA	$PM_{2.5}$, BC, UFP	On highway in busses	Heart rate variability	Decreases in measures of heart rate variability
Hoek et al. 2002 (24)	Netherlands	NA	BC, NO_2	Continuous [d]	Cardio-pulmonary mortality, lung cancer	1.41 OR for living near road
Tonne et al. 2007 (41)	Worchester, Mass.	NA	$PM_{2.5}$	Continuous [d]	Acute myocardial infarction (AMI)	5% increase in odds of AMI
Venn et al. 2001 (49)	Nottingham, UK	NA	NA	Continuous [d]	Wheezing in children	1.08 OR for living w/ in 150 m of road
Nicolai et al. 2003 (58)	Munich, Germany	>30,000 veh/d	Soot, benzene, NO_2	Traffic counts within 50 m of house	Asthma, respiratory symptoms, allergy	1.79 OR for asthma and high traffic volume
Gauderman et al. 2005 (65)	Southern California		NO_2	Continuous [d]	Asthma, respiratory symptoms	Increased asthma closer to freeways
McConnell et al. 2006 (57)	Southern California	NA	NA	Continuous [d]	Asthma	Large risk for children living w/in 75 m of road
Ryan, et al. 2007 (59)	Cincinnati, Ohio	> 1,000 trucks/d	PM2.5	400 m	Wheezing in children	NA
Kim et al. 2004 (60)	San Francisco	90,000 – 210,000 veh/d	PM, BC, NO_x	School sites	Childhood asthma	1.07 OR for high levels of NO_x
Wjst et al. 1993 (68)	Munich, Germany	7,000–125,000 veh/d	NO_x, CO	School sites	Asthma, bronchitis	Several statistical associations found
Brunekreef et al. 1997 (69)	Netherlands	80,000 – 152,000 veh/d	PM_{10}, NO_2	Continuous[d]	Lung function	Decreased FEV with proximity to high truck traffic
Janssen et al. 2003 (74)	Netherlands	30,000–155,000 veh/d	$PM_{2.5}$, NO_2, benzene	< 400 m [c]	Lung function, respiratory symptoms	No association with lung function
Peters et al. 1999 (82)	Southern California	NA	PM_{10}, NO_2	NA	Asthma, bronchitis, cough, wheeze	1.54 OR of wheeze for boys with exposure to NO_2
Brauer et al. 2007 (67)	Netherlands	Highways and streets	$PM_{2.5}$, NO_2, soot	Modeled exposure	Asthma, allergy, bronchitis, respiratory symptoms	Strongest association was with food allergies
Visser et al. 2004 (91)	Amsterdam	> 10,000 veh/d	NA	NA	Cancer	Multiple associations
Vineis et al. 2006 (87)	10 Eurpoean countries	NA	PM_{10}, NO_2, SO_2	NA	Cancer	1.46 OR near heavy traffic, 1.30 OR for high exposure to NO_2
Gauderman et al. 2007 (73)	Southern California	NA	PM_{10}, NO_2	Continuous[d]	Lung Function	Decreased FEV for those living near freeway

[a]As defined in article cited (veh/d = vehicles per day; veh/h = vehicles per hour).
[b]UFP = ultrafine particles; FP = fine particles; $PM_{2.5}$ = particles with aerodynamic diameter ≤ 2.5 um; PM_{10} = particles with aerodynamic diameter ≤ 10 um; BC = black carbon; PPAH = particle-bound polycyclic aromatic hydrocarbons; VOCs = volatile organic compounds
[c]Pollutant measurements were made along a transect away from the highway
[d]Proximity of each participant to a major road was calculated using GIS software
[e]Statistical association between proximity to highway or exposure to traffic-generated pollutants and measured health outcomes
NA = not applicable; measurements were not made.

to be a risk factor for cardiovascular disease via mechanisms that likely include pulmonary and systemic inflammation, accelerated atherosclerosis and altered cardiac autonomic function [17,22,43-46]. Uptake of particles or particle constituents in the blood can affect the autonomic control of the heart and circulatory system. Black smoke, a large proportion of which is derived from mobile source emissions [30], has a high pulmonary deposition efficiency, and due to their surface area-to-volume ratios can carry relatively more adsorbed and condensed toxic air pollutants (e.g., PPAH) compared to larger particles [17,47,48]. Based on high particle numbers, high lung deposition efficiency and surface chemistry, UFP may provide a greater potential than $PM_{2.5}$ for inducing inflammation [10]. UFPs have high cytotoxic reactive oxygen species (ROS) activity, through which numerous inflammatory responses are induced, compared to other particles [10]. Chronically elevated UFP levels such as those to which residents living near heavily trafficked roadways are likely exposed can lead to long-term or repeated increases in systemic inflammation that promote arteriosclerosis [18,29,34,37].

Asthma and highway exposures

Evidence that near highway exposures present elevated risk is relatively well developed with respect to child asthma studies. These studies have evolved over time with the use of different methodologies. Studies that used larger geographic frames and/or overall traffic in the vicinity of the home or school [49-52] or that used self-report of traffic intensity [53] found no association with asthma prevalence. Most recent child asthma studies have,

instead, used increasingly narrow definitions of proximity to traffic, including air monitoring or modeling) and have focused on major highways instead of street traffic [54-59]. All of these studies have found statistically significant associations between the prevalence of asthma or wheezing and living very close to high volume vehicle roadways. Confounders considered included housing conditions (pests, pets, gas stoves, water damage), exposure to tobacco smoke, various measures of socioeconomic status (SES), age, sex, and atopy, albeit self-reported and not all in a single study.

Multiple studies have found girls to be at greater risk than boys for asthma resulting from highway exposure [55,57,60]. A recent study also reports elevated risk only for children who moved next to the highway before they were 2 years of age, suggesting that early childhood exposure may be key [57]. The combined evidence suggests that living within 100 meters of major highways is a risk factor, although smaller distances may also result in graded increases in risk. The neglect of wind direction and the absence of air monitoring from some studies are notable missing factors. Additionally, recent concerns have been raised that geocoding (attaching a physical location to addresses) could introduce bias due to inaccuracy in locations [61].

Studies that rely on general area monitoring of ambient pollution and assess regional pollution on a scale orders of magnitude greater than the near-roadway gradients have also found associations between traffic generated pollution (CO and NOx) and prevalence of asthma [62] or hospital admission for asthma [63]. Lweguga-Mukasa et al. [64] monitored air up and down wind of a major motor vehicle bridge complex in Buffalo, NY and found that UFP were higher downwind, dropping off with distance. Their statistical models did not, however, support an association of UFP with asthma. A study in the San Francisco Bay Area measured $PM_{2.5}$, BC and NO_X over several months next to schools and found both higher pollution levels downwind from highways and a linear association of BC with asthma in long-term residents [60].

Gauderman et al. [65] measured NO_2 next to homes of 208 children. They found an odds ratio (OR) of 1.83 (confidence interval (CI): 1.04–3.22) for outdoor NO_2 (probably a surrogate for total highway pollution) and lifetime diagnosis of asthma. They also found a similar association with distance from residence to freeway. Self-report was used to control for numerous confounders, including tobacco smoke, SES, gas stoves, mildew, water damage, cockroaches and pets which did not substantially affect the association. Gauderman's study suggests that ambient air monitoring at the residence substantially increases statistical power to detect association of asthma with highway exposures.

Modeling of elemental carbon attributable to traffic near roadways based on ambient air monitoring of $PM_{2.5}$ has recently emerged as a viable approach and a study using this method found an association with infant wheezing. The modeled values appear to be better predictors than proximity. Elevation of the residence relative to traffic was also an important factor in this study [66]. A 2007 paper reported on modeled NO_2, $PM_{2.5}$ and soot and the association of these values with asthma and various respiratory symptoms in the Netherlands [67]. While finding modest statistically significant associations for asthma and symptoms, it is somewhat surprising that they found stronger associations for development of sensitization to food allergens.

Pediatric lung function and traffic-related air pollution

Studies of association of children's lung function with traffic pollutants have used a variety of measures of exposure, including: traffic density, distance to roadways, area (city) monitors, monitoring at the home or school and personal monitoring. Studies have assessed both chronic effects on lung development and acute effects and have been both cross-sectional and longitudinal. The wide range of approaches somewhat complicates evaluation of the literature.

Traffic density in school districts in Munich was associated with decreases in forced vital capacity (FVC), forced expiratory volume in 1 second (FEV_1), FEV1/FVC and other measures, although the 2-kilometer (km) areas, the use of sitting position for spirometry and problems with translation for non-German children were limitations [68]. Brunekreef et al. [69] used distance from major roadways, considered wind direction and measured black smoke and NO2 inside schools. They found the largest decrements in lung function in girls living within 300 m of the roadways.

A longitudinal study of children (average age at start = 10 years) in Southern California reported results at 4 [70] and 8 years [71]. Multiple air pollutants were measured at sites in 12 communities. Due to substantial attrition, only 42% of children enrolled at the start were available for the 8-year follow-up. Substantially lower growth in FEV_1 was associated with PM_{10}, NO_2, $PM_{2.5}$, acid vapor and elemental carbon at 4 and at 8 years. The analysis could not indicate whether the effects seen were reversible or not [72]. In 2007, it was reported from this same cohort that living within 500 m of a freeway was reported to be associated with reduced lung function [73].

A Dutch study [74] measured $PM_{2.5}$, NO_2, benzene and EC for one year at 24 schools located within 400 m of major roadways. While associations were seen between symptoms and truck traffic and measured pollutants, there was no significant association between any of the environmental measures and FVC < 85% or FEV_1 < 85%. Restricting the analysis to children living within 500 m of highways generally increased ORs.

Personal exposure monitoring of NO_2 as a surrogate for total traffic pollutants with 298 Korean college students found statistically significant associations with FEV_1, FEV_1/FVC, and forced expiratory volume between 25 and 75% (FEV_{25-75}), but not with FVC. The multivariate regression model presented suggests that FEV_{25-75} was the outcome measure that most clearly showed an effect [75]. Cross-sectional studies of children in Korea [76] and France [77] also indicate that lung function is diminished in association with area pollutants that largely derive from traffic.

Time series studies suggest there are also acute effects. A study of 19 asthmatic children measured PM via personally carried monitors, at homes and at central site monitors. The study found deficits in FEV_1 that were associated with PM, although many sources besides traffic contributed to exposure. In addition, the results suggest that ability to see associations with health outcomes improves at finer scale of monitoring [78]. PM was associated with reduced FEV_1 and FVC in only the asthmatic subset of children in a Seattle study [79]. Studies have also seen associations between PM and self reported peak flow measurements [80,81] and asthmatic symptoms [82].

Cancer and near highway exposures

As noted above, both the Six-Cities Study [27] and the American Cancer Society (ACS) Study [28] found associations between PM and lung cancer. Follow-up studies using the ACS cohort [29,37] and the Six-Studies cohort [83] that controlled for smoking and other risk factors also demonstrated significant associations between PM and lung cancer. The original studies were subject to intensive replication, validation, and re-analysis which confirmed the original findings [84].

The ASHMOG study [85] was designed to look specifically at lung cancer and air pollution among Seventh-day Adventists in California, taking advantage of their low smoking rates. Air pollution was interpolated to centroids of zip codes from ambient air monitoring stations. Highway proximity was not considered. The study found associations with ozone (its primary pollutant of consideration), PM10 and SO2. Notably, these are not the pollutants that would be expected to be substantially elevated immediately adjacent to highways.

A case control study of residents of Stockholm, Sweden modeled traffic-related NO2 levels at their homes over 30 years and found that the strongest association involved a 20 year latency period [86]. Another case control study drawn from the European Prospective Investigation on Cancer and Nutrition found statistically significantly elevated ORs for lung cancer with proximity to heavy traffic (>10,000 cars per day) as well as for NO_2 and PM_{10} at nearby ambient monitoring stations [87]. Nafstad et al. [88] used modeled NO_2 and SO_2 concentrations at the homes of over 16,000 men in Oslo to test associations with lung cancer incidence. The models included traffic and point sources. The study found small, but statistically significant associations between NO_2 and lung cancer. Problems that run through all these studies are weak measures of exposure to secondhand tobacco smoke, the use of main roads rather than highways as the exposure group and modeled rather than measured air pollutants.

A study of regional pollution in Japan and a case control study of more localized pollution in a town in Italy also found associations between NO_2 and lung cancer and PM and lung cancer [89,90]. On the other hand, a study that calculated SIRs for specific cancers across lower and higher traffic intensity found little evidence of an association with a range of cancers [91].

The plausibility of near-highway pollution causing lung cancer is bolstered by the presence of known carcinogens in diesel PM. The US EPA has concluded after reviewing the literature that diesel exhaust is "likely to be carcinogenic to humans by inhalation" [92]. An interesting study of UFP and DNA damage adds credibility to an association with cancer [93]. This study had participants bicycle in traffic in Copenhagen and measured personal exposure to UFP and DNA oxidation and strand breaks in mononuclear blood cells. Bicycling in traffic increased UFP exposure and oxidative damage to DNA, thus demonstrating an association between DNA damage and UFP exposure *in vivo*.

Policy and research recommendations

Based on the literature reviewed above it is plausible that gradients of pollutants next to highways carry elevated health risks that may be larger than the risks of general area ambient pollutants. While the evidence is considerable, it is not overwhelming and is weak in some areas. The strongest evidence comes from studies of development of asthma and reduction of lung function during childhood, while the studies of cardiac health risk require extrapolation from area studies of smaller and larger geographic scales and inference from toxicology laboratory investigations. The lung cancer studies, because they include pollutants such as O_3 that are not locally concentrated, are not particularly strong in terms of the case for near-high-

way risk. There is a need for lung cancer research that uses major highways rather than heavily trafficked roads as the environmental exposure.

While more studies of asthma and lung function in children are needed to confirm existing findings, especially studies that integrate exposure at school, home and during commuting, to refine our knowledge about the association, we would point to the greater need for studies of cardiac health and lung cancer and their association with near highway exposures as the primary research areas needing to be developed. Many of the studies of PM and cardiac or pulmonary health have focused on mortality. Near highway mortality studies may be possible, but would be lengthy if they were initiated as prospective cohorts. Other possibilities include retrospective case control studies of mortality, cross sectional studies or prospective studies that have end points short of mortality, such as biological markers of disease. For all health end points there is a need for studies that adequately address the possible confounding of SES with proximity to highways. There is good reason to think that property values decline near highways and that control for SES by, for example, income, may be inadequate.

Because of the incomplete development of the science regarding the health risks of near highway exposures and the high cost and implication of at least some possible changes in planning and development, policy decisions are complicated. The State of California has largely prohibited siting of schools within 500 feet of freeways (SB 352; approved by the governor October 2, 2003). Perhaps this is a viable model for other states or for national-level response. As it is the only such law of which we are aware, there may be other approaches that will be and should be tried. One limitation of the California approach is that it does nothing to address the population already exposed at schools currently cited near freeways and does not address residence near freeways.

Conclusion

The most susceptible (and overlooked) population in the US subject to serious health effects from air pollution may be those who live very near major regional transportation route, especially highways. Policies that have been technology based and regional in orientation do not efficiently address the very large exposure and health gradients suffered by these populations. This is problematic because even regions that EPA has deemed to be in regional PM "attainment" still include very large numbers of near highway residents who currently are not protected. There is a need for more research, but also a need to begin to explore policy options that would protect the exposed population.

Abbreviations

UFP = ultra fine particles

BC = black carbon

NO_2 = nitrogen dioxide

NOx = oxides of nitrogen

CO = carbon monoxide

PM = particulate matter

$PM_{2.5}$ = particulate matter less than 2.5 um

PM_{10} = particulate matter less than 10 um

PPAH = particle bound polyaromatic hydrocarbons

EC = elemental carbon

VOC = volatile organic compounds

SO_2 = sulfur dioxide

ACS = American Cancer Society

SES = socioeconomic status

EPA = Environmental Protection Agency

OR = odds ratio

FEV_1 = forced expiratory volume in 1 second

FEV_1/FVC = ratio of FEV_1 and forced vital capacity

FEV_{25-75} = forced expiratory volume between 25 and 75

FVC = forced vital capacity

ug/m^3 = micrograms per cubic meter of air

m = meters

um = micrometers

veh/d = vehicles per day

veh/h = vehicles per hour

Competing interests

The author(s) declare that they have no competing interests.

Authors' contributions

DB took the lead on the manuscript. He co-wrote the background and wrote the sections on asthma, lung function and cancer and the conclusions. JLD wrote the section on air pollutants near roadways and contributed substantially to the background. CR wrote the section on cardiovascular health. All authors participated in editing and refining the manuscript and all read it multiple times, including the final version.

Acknowledgements

We thank Wig Zamore for useful insights into the topic. The Jonathan M Tisch College of Citizenship and Public Service partially supported the effort of Doug Brugge and Christine Rioux. Figure 1 was reproduced with permission of the publisher.

References

1. **American Housing Survey for the United States: 2003 Series H150/03** [http://www.census.gov/hhes/www/housing/ahs/ahs03/ahs03.html]. Accessed May 2007.
2. Massachusetts Fact Book; 2004.
3. Chambers LA: **Classification and extent of air pollution problems.** In *Air Pollution Volume I*. 3rd edition. Edited by: Stern AC. Academic Press, NY; 1976.
4. Rogge WF, Hildemann LM, Mazurek MA, Cass GR, Simoneit BRT: **Sources of fine organic aerosol. 2. Noncatalyst and catalyst-equipped automobiles and heavy-duty diesel trucks.** *Environmental Science Technology* 1993, **27:**636-651.
5. Graedel TE, Hawkins DT, Claxton LD: **Atmospheric Chemical Compounds: Sources, Occurrence, and Bioassay.** Academic Press, Inc., New York, NY; 1986.
6. Shi JP, Khan AA, Harrison RM: **Measurements of ultrafine particle concentration and size distribution in the urban atmosphere.** *The Science of the Total Environment* 1999, **235:**51-64.
7. Zhu Y, Hinds WC, Kim S, Sioutas C: **Concentration and size distribution of ultrafine particles near a major highway.** *Journal of the Air and Waste Management Association* 2002, **52(9):**1032-1042.
8. Zhu Y, Hinds WC, Kim S, Shen S, Sioutas C: **Study of ultrafine particles near a major highway with heavy-duty diesel traffic.** *Atmospheric Environment* 2002, **36:**4323-4335.
9. Zhang KM, Wexler AS, Zhu Y, Hinds WC, Sioutas C: **Evolution of particle number distribution near roadways. Part II: the 'Road-to-Ambient' process.** *Atmospheric Environment* 2004, **38:**6655-6665.
10. Sioutas C, Delfino RJ, Singh M: **Exposure assessment for atmospheric ultrafine particles (UFP) and implications in epidemiologic research.** *Environmental Health Perspectives* 2005, **113(8):**947-955.
11. Hitchins J, Morawska L, Wolff R, Gilbert D: **Concentrations of submicrometre particles from vehicle emissions near a major road.** *Atmospheric Environment* 2000, **34:**51-59.
12. Morawska L, Thomas S, Gilbert D, Greenaway C, Rijnders E: **A study of the horizontal and vertical profile of submicrometer particulates in relation to a busy road.** *Atmospheric Environment* 1999, **33:**1261-1274.
13. Fischer PH, Hoek G, van Reeuwijk H, Briggs DJ, Lebret E, van Wijnen JH, Kingham S, Elliott PE: **Traffic-related differences in outdoor and indoor concentrations of particles and volatile organic compounds in Amsterdam.** *Atmospheric Environment* 2000, **34:**3713-3722.
14. Roorda-Knape MC, Janssen NAH, De Hartog JJ, van Vliet PHN, Harssema H, Brunekreef B: **Air pollution from traffic in city districts near major motorways.** *Atmospheric Environment* 1998, **32:**1921-1930.
15. Janssen NAH, van Vliet PHN, Aarts F, Harssema H, Brunekreef B: **Assessment of exposure to traffic related air pollution of children attending schools near motorways.** *Atmospheric Environment* 2001, **35:**3875-3884.
16. National Research Council, Committee on Research Priorities for Airborne Particulate Matter: **Research priorities for airborne particulate matter, IV: continuing research progress.** National Academy Press, Washington, DC; 2004.
17. US Environmental Protection Agency: *Air quality criteria for particulate matter* Research Triangle Park; 2004.
18. Miller KA, Siscovick DS, Sheppard L, Shepherd K, Sullivan JH, Anderson GL, Kaufman JD: **Long-term exposure to air pollution and incidence of cardiovascular events in women.** *The New England Journal of Medicine* 2007, **356:**447-458.
19. Riedliker M, Cascio WE, Griggs TR, Herbst MC, Bromberg PA, Neas L, Williams RW, Devlin RB: **Particulate matter exposure in cars is associated with cardiovascular effects in healthy young men.** *American Journal of Respiratory and Critical Care Medicine* 2004, **169:**934-940.
20. Hoffmann B, Moebus S, Stang A, Beck E, Dragano N, Möhlenkamp S, Schmermund A, Memmesheimer M, Mann K, Erbel R, Jockel KH, Heinz Nixdorf RECALL Study Investigative Group: **Residence close to high traffic and prevalence of coronary heart disease.** *European Heart Journal* 2006, **27:**2696-2702.
21. Ruckerl R, Greven S, Ljungman P, Aalto P, Antoniades C, Bellander T, Berglind N, Chrysohoou C, Forastiere F, Jacquemin B, von Klot S, Koenig W, Kuchenhoff H, Lanki T, Pekkanen J, Perucci CA, Schneider A, Sunyer J, Peters A: **Air pollution and inflammation (IL-6, CRP, fibrinogen) in myocardial infarction survivors.** *Environmental Health Perspectives* 2007, **115:**1072-1080.
22. Schwartz J, Litonjua A, Suh H, Verrier M, Zanobetti A, Syring M, Nearing B, Verrier R, Stone P, MacCallum G, Speizer FE, Gold DR: **Traffic related pollution and heart rate variability in a panel of elderly subjects.** *Thorax* 2005, **60:**455-461.
23. Adar SD, Gold DR, Coull BA, Schwartz J, Stone P, Suh H: **Focused exposures to airborne traffic particles and heart rate variability in the elderly.** *Epidemiology* 2007, **18:**95-103.
24. Hoek G, Brunekreek B, Goldbohm S, Fischer P, van den Brandt PA: **Association between mortality and indicators of traffic-related air pollution in the Netherlands: a cohort study.** *The Lancet* 2002, **360:**1203-1209.
25. Peters A, von Klot S, Heier M, Trentinaglia I, Hormann A, Wichmann HE, Lowel H: **Exposure to traffic and the onset of myocardial infarction.** *The New England Journal of Medicine* 2004, **351:**1861-70.
26. Pope CA, Dockery DW: **Health effects of fine particulate air pollution: lines that connect.** *Journal of Air and Waste Management* 2006, **56(6):**709-742.
27. Dockery DW, Pope CA, Xu X, Spengler JD, Ware JH, Fay ME, Ferris BG, Speizer FE: **An association between air pollution and mortality in six U.S. cities.** *New England Journal of Medicine* 1993, **329:**1753-9.
28. Pope CA, Thun MJ, Namboodiri MM, Dockery DW, Evans JS, Speizer FE, Hath CW: **Particulate air pollution as a predictor of mortality in a prospective study of US adults.** *American Journal of Respiratory and Critical Care Medicine* 1995, **151:**669-674.
29. Pope CA, Burnett RT, Thun MJ, Calle EE, Krewski D, Ito K, Thurston GD: **Lung Cancer, Cardiopulmonary mortality, and long-term exposure to fine particulate air pollution.** *Journal of the American Medical Association* 2002, **287:**1132-1141.
30. Kunzli N, Jerrett M, Mack WJ, Beckerman B, LaBree L, Gilliland F, Thomas D, Peters J, Hodis HN: **Ambient air pollution and Atherosclerosis in Los Angeles.** *Environmental Health Perspectives* 2005, **113:**201-206.
31. Peters A: **Particulate matter and heart disease: Evidence from epidemiological studies.** *Toxicology and Applied Pharmacology* 2005:477-482.
32. Wheeler A, Zanobetti A, Gold DR, Schwartz J, Stone P, Suh H: **The relationship between ambient air pollution and heart rate variability differs for individuals with heart and pulmonary disease.** *Environmental Health Perspectives* 2006, **114:**560-566.
33. Chuang K, Chan C, Chen N, Su T, Lin L: **Effects of particle size fractions on reducing heart rate variability in cardiac and hypertensive patients.** *Environmental Health Perspectives* 2005, **113:**1693-1697.
34. Chan C, Chuang K, Shiao G, Lin L: **Personal exposure to submicrometer particles and heart rate variability in human subjects.** *Environmental Health Perspectives* 2004, **112:**1063-1067.
35. Brauer M, Hoek G, van Vliet P, Meliefste K, Fischer P, Gehring U, Heinrich J, Cyrys J, Bellander T, Lewne M, Brunekreef B: **Estimating long-term average particulate air pollution concentrations: application of traffic indicators and geographic information systems.** *Epidemiology* 2003, **14:**228-239.

36. Brunekreef B, Holgate ST: **Air pollution and health.** *Lancet* 2002, **360:**1233-1242.
37. Jerrett M, Finkelstein M: **Geographies of risk in studies linking chronic air pollution exposure to health outcomes.** *Journal of Toxicology and Environmental Health* 2005, **68:**1207-1242.
38. O'Neill MS, Jerrett M, Kawachi I, Levy JI, Cohen AJ, Gouveia N, Wilkinson P, Fletcher T, Cifuentes L, Schwartz J: **Workshop on Air Pollution and Socioeconomic Conditions. Health, wealth, and air pollution: advancing theory and methods.** *Environmental Health Perspectives* 2003, **111:**1861-1870.
39. Jerrett M, Burnett RT, Ma R, Pope CA, Krewski D, Newbold KB, Thurston G, Shi Y, Finkelstein N, Calle EE, Thun MJ: **Spatial Analysis of Air Pollution and Mortality in Los Angeles.** *Epidemiology* 2005, **16(6):**727-736.
40. Finkelstein M, Jerrett M, Sears MR: **Environmental inequality and circulatory disease mortality gradients.** *Journal of Epidemiology and Community Health* 2005, **59:**481-487.
41. Tonne C, Melly S, Mittleman M, Coull B, Goldberg R, Schwartz J: **A case-control analysis of exposure to traffic and acute myocardial infarction.** *Environmental Health Perspectives* 2007, **115:**53-57.
42. Lipfert FW, Wyzga RE, Baty JD, Miller JP: **Traffic density as a surrogate measure of environmental exposures in studies of air pollution health effects: Long-term mortality in a cohort of US veterans.** *Atmospheric Environment* 2006, **40:**154-169.
43. Pope CA, Burnett RT, Thurston GD, Thun MJ, Calle EE, Krewski D, Godleski JJ: **Cardiovascular mortality and long-term exposure to particulate air pollution – Epidemiological evidence of general pathophysiological pathways of disease.** *Circulation* 2004, **109:**71-77.
44. Brook RD, Franklin B, Cascio W, Hong Y, Howard G, Lipsett M, Luepker R, Mittleman M, Samet J, Smith SC, Tager I: **Air pollution and cardiovascular disease: a statement for healthcare professionals from the expert panel on population and prevention science of the American Heart Association.** *Circulation* 2004, **109:**2655-2671.
45. Sun Q, Wang A, Jin X, Natanzon A, Duquaine D, Brook RD, Aguinaldo JG, Fayad Z, Fuster V, Lippman M, Chen LC, Rajagopalan S: **Long-term air pollution exposure and acceleration of atherosclerosis and vascular inflammation in an animal model.** *Journal of the American Medical Association* 2005, **294:**3003-3010.
46. Sandhu RS, Petroni DH, George WJ: **Ambient particulate matter, C-reactive protein, and coronary artery disease.** *Inhalation Toxicology* 2005, **17:**409-413.
47. Oberdorster G: **Pulmonary effects of inhaled ultrafine particles.** *International Archives of Occupational and Environmental Health* 2001, **65:**1531-1543.
48. Delfino RJ, Sioutas C, Malik S: **Potential role of ultrafine particles in associations between airborne particle mass and cardiovascular health.** *Environmental Health Perspectives* 2005, **113:**934-946.
49. Venn A, Lewis S, Cooper M, Hubbard R, Hill I, Boddy R, Bell M, Britton J: **Local road traffic activity and the prevalence, severity, and persistence of wheeze in school children: combined cross sectional and longitudinal study.** *Occupational & Environmental Medicine* 2000, **57:**152-158.
50. Waldron G, Pottle B, Dod J: **Asthma and the motorways – One district's experience.** *Journal of Public Health Medicine* 1995, **17:**85-89.
51. Lewis SA, Antoniak M, Venn AJ, *et al.*: **Secondhand smoke, dietary fruit intake, road traffic exposures, and the prevalence of asthma: A cross-sectional study in young children.** *American Journal of Epidemiology* 2005, **161:**406-411.
52. English P, Neutra R, Scalf R, Sullivan M, Waller L, Zhu L: **Examining associations between childhood asthma and traffic flow using a geographic information system.** *Environmental Health Perspectives* 1999, **107:**761-767.
53. Heinrick J, Topp R, Gerring U, Thefeld W: **Traffic at residential address, respiratory health, and atopy in adults; the National German Health Survey 1998.** *Environmental Research* 2005, **98:**240-249.
54. Van Vliet P, Knape M, de Hartog J, Janssen N, Harssema H, Brunekreef B: **Motor vehicle exhaust and chronic respiratory symptoms in children living near freeways.** *Environmental Research* 1997, **74:**122-132.
55. Venn AJ, Lewis SA, Cooper M, Hubbard R, Britton J: **Living near a main road and the risk of wheezing illness in children.** *American Journal of Respiratory and Critical Care Medicine* 2001, **164(12):**2177-2180.
56. Venn A, Yemaneberhan H, Lewis S, Parry E, Britton J: **Proximity of the home to roads and the risk of wheeze in an Ethiopian population.** *Occupational and Environmental Medicine* 2005, **62:**376-380.
57. McConnell R, Berhane K, Yao L, Jerrett M, Lurmann F, Gilliland F, Kunzli N, Gauderman J, Avol E, Thomas D, Peters J: **Traffic susceptibility, and childhood asthma.** *Environmental Health Perspectives* 2006, **114:**766-772.
58. Nicolai T, Carr D, Weiland SK, Duhme H, von Ehrenstein O, Wagner C, von Mutius E: **Urban traffic and pollutant exposure related to respiratory outcomes and atopy in a large sample of children.** *European Respiratory Journal* 2003, **21:**956-963.
59. Ryan PH, LeMasters , Biswas P, Levin L, Hu S, Lindsey M, Bernstein DI, Lockey J, Villareal M, Hershey GKH, Grinshpun SA: **A comparison of proximity and land use regression traffic exposure models and wheezing in infants.** *Environmental Health Perspectives* 2007, **115:**278-284.
60. Kim JJ, Smorodinsky S, Lipsett M, Singer BC, Hodgson AT, Ostro B: **Traffic-related air pollution near busy roads: The East Bay children's respiratory health study.** *American Journal of Respiratory and Critical Care Medicine* 2004, **170:**520-526.
61. Ong P, Graham M, Houston D: **Policy and programmatic importance of spatial alignment of data sources.** *Am J Public Health* 2006, **96:**499-504.
62. Hwang BF, Lee YL, Lin YC, Jaakkola JJ, Guo YL: **Traffic related air pollution as a determinant of asthma among Taiwanese school children.** *Thorax* 2005, **60:**467-473.
63. Migliaretti G, Cadum E, Migliore E, *et al.*: **Traffic air pollution and hospital admissions for asthma: A case control approach in a Turin (Italy) population.** *International Archives of Occupational and Environmental Health* 2005, **78:**164-169.
64. Lweguga-Mukasa JS, Oyana TJ, Johjnson C: **Local ecological factors, ultrafine particulate concentrations, and asthma prevalence rates in Buffalo, New York, neighborhoods.** *Journal of Asthma* 2005, **42:**337-348.
65. Gauderman WJ, Avol E, Lurmann F, Kuenzli N, Gilliland F, Peters J, McConnell R: **Childhood asthma and exposure to traffic and nitrogen dioxide.** *Epidemiology* 2005, **16:**737-743.
66. Ryan PH, LeMasters GK, Biswas P, Levin L, Hu S, Lindsey M: **A comparison of proximity and land use regression traffic exposure models and wheezing in infants.** *Environmental Health Perspectives* 2007, **115:**278-284.
67. Brauer M, Hoek G, Smit HA, de Jongste JC, Gerritsen J, Postma DS, Kerkhof M, Brunekreef B: **Air pollution and development of asthma, allergy and infections in a birth cohort.** *European Respiratory Journal* 2007, **29:**879-888.
68. Wjst M, Reitmeir P, Dodd S, Wulff A, Nicolai T, von Loeffelholz-Colberg EF, von Mutius E: **Road traffic and adverse effects on respiratory health in children.** *British Medical Journal* 1993, **307:**596-307.
69. Brunekreef B, Janssen NA, de Hartog J, Harssema H, Knape M, van Vliet P: **Air pollution from truck traffic and lung function in children living near motorways.** *Epidemiology* 1997, **8:**298-303.
70. Gauderman WJ, McConnell , Gilliland F, London S, Thomas D, Avol E, Vora H, Berhane K, Rappaport EB, Lurmann F, Margolis HG, Peters J: **Association between air pollution and lung function growth in Southern California Children.** *American Journal of Respiratory and Critical Care Medicine* 2000, **162(4 Pt 1):**1383-1390.
71. Gauderman WJ, Avol E, Gilliland F, Vora H, Thomas D, Berhane K, McConnell R, Kuenzli N, Lurmann F, Rappaport E, Margolis H, Bates D, Peters J: **The Effect of Air Pollution on Lung Development from 10 to 18 Years of Age.** *New England Journal of Medicine* 2005, **351:**1057-67.
72. Merkus PJFM: **Air pollution and lung function.** *New England Journal of Medicine* 2005, **351:**2652.
73. Gauderman WJ, Vora H, McConnell R, Berhane K, Gilliland F, Thomas D, Lurmann F, Avol E, Kunzli N, Jarrett M, Peters J: **Effect of exposure to traffic on lung development from 10 to 18 years of age: A cohort study.** *The Lancet* 2007, **369:**571-577.
74. Janssen NA-H, Brunekreef B, van Vliet P, Aarts F, Meliefste K, Harssema H, Fischer P: **The relationship between air pollution from heavy traffic and allergic sensitization, bronchial hyper-**

responsiveness, and respiratory symptoms in Dutch school children. *Environmental Health Perspectives* 2003, **111:**1512-1518.
75. Hong Y-C, Leem J-H, Lee K-H, Park D-H, Jang J-Y, Kim S-T, Ha E-H: **Exposure to air pollution and pulmonary function in university students.** *International Archives of Occupational and Environmental Health* 2005, **78:**132-138.
76. Kim HJ, Lim DH, Kim JK, Jeong SJ, Son BK: **Effects of particulate matter (PM10) on pulmonary function of middle school children.** *Journal of the Korean Medical Society* 2005, **20(1):**42-45.
77. Penard-Morand C, Charpin D, Raherison C, Kopferschmitt C, Caillaud D, Lavaud F, Annesi-Maesano I: **Long-term exposure to background air pollution related to respiratory and allergic health in schoolchildren.** *Clinical and Experimental Allergy* 2005, **35:**1279-1287.
78. Delfino RJ, Quintana PJE, Floro J, Gastanaga VM, Samimi BS, Klienman MT, Liu LJ, Bufalino C, Wu C, McLaren CE: **Association of FEV1 in asthmatic children with personal and microenvironment exposure to airborne particulate matter.** *Environmental Health Perspectives* 2004, **112:**932-941.
79. Koenig JQ, Larson TV, Hanley QS, Rebolledo V, Dumler K, Checkoway H, Wang SZ, Lin D, Pierson WE: **Pulmonary function changes in children associated with fine particulate matter.** *Environmental Research* 1993, **63:**26-38.
80. Van der Zee SC, Hoek G, Boezen HM, Schouten JP, van Wijnen JH, Brunekreef B: **Acute effects of urban air pollution on respiratory health of children with and without chronic respiratory symptoms.** *Occupational and Environmental Medicine* 1999, **56(12):**802-813.
81. Pekkenen J, Timonen KL, Ruuskanen J, Reponen A, Mirme A: **Effects of ultrafine and fine particulates in urban air on peak expiratory flow among children with asthmatic symptoms.** *Environmental Research* 1997, **74:**24-33.
82. Peters JM, Avol E, Navidi W, London SJ, Gauderman WJ, Lurmann F, Linn WS, Margolis H, Rappaport E, Gong H, Thomas DC: **A study of twelve Southern California communities with differing levels and types of air pollution: Prevalence of respiratory morbidity.** *American Journal of Respiratory and Critical Care Medicine* 1999, **159(3):**760-767.
83. Laden F, Schwartz J, Speizer FE, Dockery DE: **Reduction in fine particulate air pollution and mortality: extended follow-up of the Harvard six-cities study.** *American Journal of Respiratory and Critical Care Medicine* 2006, **173:**667-672.
84. Health Effects Institute: **Reanalysis of the Harvard six cities study and the American Cancer Society study of particulate air pollution mortality.** *Final Version; Boston, MA* 2000.
85. Beeson WL, Abbey DE, Knutsen SF: **Long-term concentrations of ambient air pollutants and incident lung cancer in California adults: Results from the ASHMOG study.** *Environmental Health Perspectives* 1998, **106:**813-823.
86. Nyberg F, Gustavsson P, Jarup L, Bellander T, Berglind N, Jakobsson R, Pershagen G: **Urban air pollution and lung cancer in Stockholm.** *Epidemiology* 2000, **11:**487-495.
87. Vineis P, Hoek G, Krzyzanowski M, Vigna-Tagliani F, Veglia F, Airoldi L, Autrup H, Dunning A, Garte S, Hainaut P, Malaveille C, Matullo G, Overvad K, Raaschou-Nielsen O, Clavel-Chapelon F, Linseisen J, Boeing H, Trichopoulou A, Palli D, Peluso M, Krogh V, Tumino R, Panico S, Bueno-De-Mesquita HB, Peeters PH, Lund EE, Gonzalez CA, Martinez C, Dorronsoro M, Barricarte A, Cirera L, Quiros JR, Berglund G, Forsberg B, Day NE, Key TJ, Saracci R, Kaaks R, Riboli E: **Air pollution and risk of lung cancer in a prospective study in Europe.** *International Journal of Cancer* 2006, **119:**169-174.
88. Nafstad P, Haheim LL, Oftedal B, Gram F, Holme I, Hjermann I, Leren P: **Lung cancer and air pollution: A 27-year follow up of 16 209 Norwegian men.** *Thorax* 2003, **58:**1071-1076.
89. Choi K-S, Inoue S, Shinozaki R: **Air pollution, temperature, and regional differences in lung cancer mortality in Japan.** *Archives of Environmental Health* 1997, **52:**160.
90. Biggeri A, Barbone F, Lagazio C, Bovenzi M, Stanta G: **Air pollution and lung cancer in Trieste, Italy: Spatial analysis of risk as a function of distance from sources.** *Environmental Health Perspectives* 1996, **104:**750-754.
91. Visser O, van Wijnen JH, van Leeuwen FE: **Residential traffic density and cancer incidence in Amsterdam, 1989–1997.** *Cancer Causes & Control* 2004, **15:**331-339.
92. US Environmental Protection Agency: **Health Assessment Document for Diesel Engine Exhaust. Washington, DC.** 2002.
93. Vinzents PS, Meller P, Sorensen M, Knudsen LE, Hertel O, Jensen FP, *et al.*: **Personal exposure to ultrafine particulates and oxidative DNA damage.** *Environmental Health Perspectives* 2005, **113:**1485-1490.

Repeated hospital encounters for asthma in children and exposure to traffic-related air pollution near the home

Ralph J. Delfino, MD, PhD*; Joyce Chang, PhD*; Jun Wu, PhD*†; Cizao Ren, PhD*; Thomas Tjoa, MS, MPH*; Bruce Nickerson, MD‡; Dan Cooper, MD§; and Daniel L. Gillen, PhD¶

Background: Aggregate hospital encounters for asthma (admissions or emergency department visits) have been associated with daily regional air pollution. There are fewer data on relationships between repeated hospital encounters and traffic-related air pollution near the home.

Objective: To estimate the association of local traffic–generated air pollution with repeated hospital encounters for asthma in children.

Methods: Hospital records for 2,768 children aged 0 to 18 years (697 of whom had ≥2 encounters) were obtained for a catchment area of 2 hospitals in northern Orange County, California. Residential addresses were geocoded. A line source dispersion model was used to estimate individual seasonal exposures to local traffic–generated pollutants (nitrogen oxides and carbon monoxide) longitudinally beginning with the first hospital encounter. Recurrent proportional hazards analysis was used to estimate risk of exposure to air pollution adjusting for sex, age, health insurance, census-derived poverty, race/ethnicity, residence distance to hospital, and season. The adjustment variables and census-derived median household income were tested for effect modification.

Results: Adjusted hazard ratios for interquartile range increases in nitrogen oxides (4.00 ppb) and carbon monoxide (0.056 ppm) were 1.10 (95% confidence interval, 1.03–1.16) and 1.07 (1.01–1.14), respectively. Associations were strongest for girls and infants but were not significantly different from other groups. Stronger associations in children from higher-income block groups ($P < .09$ for trend) may have been due to more accurate data.

Conclusions: Associations for repeated hospital encounters suggest that locally generated air pollution near the home affects asthma severity in children. Risk may begin during infancy and continue in later childhood, when asthma diagnoses are clearer.

Ann Allergy Asthma Immunol. 2009;102:138–144.

INTRODUCTION

Many studies[1] show that children with asthma are susceptible to acute adverse changes in asthma outcomes from short-term increased exposure to ambient air pollutants measured at central regional sites at some distance from their residence. A few studies[2–5] have shown associations of pediatric asthma outcomes with personal exposure measurements of air pollutants. There are fewer data on whether certain air pollution sources cause these asthma associations largely because analyses have focused on temporal rather than spatial differences in exposure. Spatial heterogeneity of potentially toxic pollutant components are not well represented by data from ambient air monitoring sites, which have provided the bulk of exposure data in previous studies.[6,7]

A major contributor to air pollution exposure in urban areas is from mobile transportation sources. In southern California, for example, on-road emission sources alone contribute approximately 45% of volatile organic compounds, 63% of nitrogen oxide (NO_x), and 76% of carbon monoxide (CO) in the air.[8] There is a growing view that to accurately measure the magnitude of pediatric respiratory associations, air pollutant exposures are best evaluated closer to where children reside.[9] High home or school traffic density has been associated with prevalence of diagnosed asthma in epidemiologic studies.[10,11] Cohort studies[12–18] have shown associations between asthma incidence or early wheeze or cough without a cold and traffic-related air pollution near the homes of preschool children using geographic information system (GIS)–based exposure models.

Affiliations: * Department of Epidemiology, School of Medicine, University of California, Irvine; † Program in Public Health, College of Health Sciences, University of California, Irvine; ‡ Division of Pulmonary Medicine, Children's Hospital of Orange County, Orange, California; § Department of Pediatrics, School of Medicine, University of California, Irvine; ¶ Department of Statistics, School of Information and Computer Sciences, University of California, Irvine.

Disclosures: Authors have nothing to disclose.

Funding Sources: The project described was supported by the South Coast Air Management District (SCAQMD), through the University of California, Los Angeles, Asthma and Outdoor Air Quality Consortium (contract UCLA-040623), and by grant ES11615 from the National Institute of Environmental Health Sciences (NIEHS), National Institutes of Health (NIH). Asthma surveillance data were collected under grant FCI-CU3–13 from the Children and Families Commission of Orange County for the Asthma and Chronic Lung Disease Institute.

Disclaimer: The contents of this article are solely the responsibility of the authors and do not necessarily represent the official views of the NIEHS, NIH, or SCAQMD.

Received for publication October 1, 2008; Received in revised form November 25, 2008; Accepted for publication November 30, 2008.

Numerous experimental studies have provided evidence that exposure to chemicals capable of inducing airway oxidative stress, such as polycyclic aromatic hydrocarbons from diesel and auto exhaust, may play a role in the onset of allergic sensitization that could lead to asthma and in the acute exacerbation of respiratory allergic diseases, including asthma.[19] However, the impact of exposure to traffic on repeated episodes of asthma requiring hospital care is unclear.

Time-series studies have generally evaluated the relationship between central site air pollution measurements and aggregate (nonindividual) daily data for asthma emergency department (ED) visits and hospital admissions.[1] We conducted the first longitudinal study[20] of the relationship between repeated hospital encounters for individual children admitted with an asthma diagnosis and traffic-related air pollution in the outdoor home environment of these children. We found increased risk of repeated ED visits and hospitalizations for children 18 years and younger with a primary or secondary diagnosis of asthma in those living within 300 m of arterial roads or freeways. At highest risk were children in the top quintile of traffic density and those who had 750 m or more of arterial road and freeway length within 300 m of their residence. The present study advances this analysis of home traffic indices by using an improved GIS-based exposure evaluation method and air dispersion models. We aimed to estimate the risk of repeated hospital encounters in a cohort of children with a primary diagnosis of asthma in relation to individual exposures to local traffic–generated air pollution. Furthermore, given previous evidence,[21] we tested whether children of lower socioeconomic status are at increased risk from air pollution exposures.

METHODS

Population and Outcomes

We passively followed up patients aged 0 to 18 years admitted to the hospital or seen in the ED with a primary diagnosis of asthma. Hospital data were extracted from billing records at 2 hospitals primarily serving the urban core of Northern Orange County. The study region consists of census block areas located within 13 km of the Children's Hospital of Orange County (CHOC) or the University of California Irvine Medical Center (UCIMC) (within 2.5 km of each other). This region was determined by mapping all records and finding a high density of patients visiting the CHOC and the UCIMC from this catchment area. This provided a reasonable evaluation of repeated hospital utilization for individual patients.

We identified 2,768 patients seen at the CHOC or the UCIMC between January 1, 2000, and December 31, 2003. Hospital data included health insurance, sex, age, race/ethnicity, and home address. For the 2,768 identified patients there were 4,020 unique hospital encounters (ED visits or hospitalizations by a particular patient $\geq$8 days apart). We geocoded the home addresses of all the patients and linked this to US Census 2000 block group socioeconomic data and traffic data using the GIS. The institutional review boards of the UCIMC and the CHOC approved the study protocol and establishment of the hospital records surveillance system for respiratory illnesses.

Exposure Evaluation

Residential addresses at the first hospital encounter were successfully geocoded for 93% of the patients (Tele Atlas North America Inc, Boston, Massachusetts). California Department of Transportation traffic data for major roads and highways were linked to the home locations.

We applied CALINE4 dispersion models to estimate nitrogen dioxide (NO_2), NO_x (nitric oxide + NO_2), and CO concentrations at each residence from local traffic emissions of gasoline vehicles and diesel trucks within a 5-km radius of each residence. The 5-km radius was used previously, and reasonable agreement was observed between CALINE4-modeled and measured 2-week average NO_2 concentrations at 260 residences in southern California (R^2 = 0.3–0.9).[22] The CALINE4 model is a gaussian line source dispersion model designed to estimate local pollutant concentrations from motor vehicle emissions based on traffic volumes, roadway geometry, vehicle emission rates, and meteorologic conditions (wind speed and direction, atmospheric stability, and mixing heights).[23] Wind patterns affect the general direction and dispersion of pollutants, leading to different exposures for individuals on the upwind vs downwind side of traffic sources.[6] Average diurnal and day-of-week freeway and nonfreeway traffic variations were included. Emission factors were obtained from the California Air Resources Board's EMFAC2007 (v2.3) vehicle emissions model. Meteorologic data were obtained from the National Weather Service.

Exposures were updated for each participant every 6-month season from the time of entry into the study at first admission or ED visit (event) to the end of follow-up. Seasons were divided into 2 periods of southern California weather for CALINE4 estimates (warm season: May-October; cool season: November-April). Therefore, exposures during follow-up were estimated seasonally across the 4-year study.

Statistical Analysis

The relationship between hospital encounters and traffic-related air pollution (dispersion-modeled CO, NO_2, and NO_x) was tested using recurrent event proportional hazards models in SAS version 9.2 (SAS Institute Inc, Cary, North Carolina). We estimated the baseline hazard for admission separately for each recurrence time and assumed a common log–hazard ratio for the exposure association across all recurrence times.[24] We accounted for within-patient correlation of recurrent events by using robust variance estimates.[25] Only 3 patients had 11 to 12 readmissions, so we considered only 10 or fewer readmissions per patient in the analysis. Patients were considered to be at risk for recurrence from the time of first hospital encounter until the end of the observation period (December 31, 2003) or their 19th birthday. Time at risk

started at the first or a subsequent event and ended with each season (when time at risk begins with the next seasonal exposure) or at the next event (when time at risk begins again with the current seasonal exposure).

We adjusted for a priori–identified potential confounders available from hospital records: age group (0, 1–5, 6–18 years), sex, race/ethnicity, insurance status, and residence distance to hospital. We also controlled for neighborhood socioeconomic status using US Census 2000 percent of households below the poverty level, for which there was no clear evidence of effect modification. We tested covariates for effect modification in separate models including product terms (interactions) of the air pollutant with the potential effect modifier, including age group, sex, race/ethnicity, insurance status, season, residence distance to hospital, and another indicator of neighborhood socioeconomic status (median household income). Significance tests for product terms were evaluated using the Wald χ^2 test for each contrast. We assume that product term $P < .10$ indicates significant interaction, ie, that the association with air pollution differs in one group compared with a reference group (eg, boys vs girls). Results stratified by group were obtained from product term models. Hazard ratios and 95% confidence intervals were calculated for interquartile range increases in air pollutants to standardize and compare associations regardless of pollutant concentration ranges or units of measurement.

RESULTS

There were 2,071 children (74.8%) with 1 hospital encounter during follow-up and 697 (25.2%) with 2 or more (Table 1). There was an expected predominance of boys, and 1,666 children (60.2%) were 0 to 5 years old at their first hospital encounter and had 66.7% of readmissions. Seasonal air pollutant exposures are given in Table 2. Pollutants were strongly correlated ($R > 0.9$). The modeled concentrations of fresh traffic emissions equaled approximately 20% of ambient NO_x concentrations at the regional station (38 ppb). Other pollutants (CO and NO_2) shared a similar pattern. Nevertheless, fresh traffic-generated air pollution contributes greatly to the spatial heterogeneity of ambient pollution.[6]

Table 3 indicates the significant increased risks of repeated hospital encounters of 7% to 10% per interquartile range increase in traffic-related NO_x and CO exposures. Associations for NO_2 are approximately half that for NO_x and do not reach significance at $P < .05$. There is little difference in coefficients between adjusted and unadjusted models and between NO_x and CO. The remaining models include NO_x but not NO_2.

Table 4 gives the models stratified by sex and age group. Although the product terms (interactions) are not significant, the point estimates for CO and NO_x are stronger in girls than in boys and in infants than in older children. Hazard ratios for children aged 6 to 18 years are more positive than for those aged 1 to 5 years, but the lower 95% confidence limits dip below 1.0 in both groups.

Table 5 provides the models stratified by census block group poverty and median household income above vs below the median population distribution. Although hazard ratios were larger for those in block groups with more families below the poverty level, product terms were nonsignificant. Models stratified by median household income showed stronger and significant associations for both pollutants in those in the upper half of income distribution. We did not find significant differences by health insurance status, although coefficients were larger for those with private insurance (Table 5). Results by race/ethnicity showed a lower risk estimate for nonwhite patients attributable to black, Asian, and other patients (data not shown). Therefore, we combined these non-Hispanic nonwhite groups because of low sample sizes in each (Table 1) and compared regression estimates for them and for Hispanic patients with those of white patients (Table 5). There were no significant differences in associations between white and Hispanic patients for NO_x or CO. However,

Table 1. Demographic Characteristics of the 2,768 Study Participants

Characteristic	Readmission for asthma, No. (%)	
	No (n = 2,071)	Yes (n = 697)
Sex		
Female	892 (43.1)	277 (39.7)
Male	1,179 (56.9)	420 (60.3)
Age group at study entry, y		
0	311 (15.0)	197 (28.3)
1–5	890 (43.0)	268 (38.4)
6–18	870 (42.0)	232 (33.3)
Race		
White non-Hispanic	910 (43.9)	327 (46.9)
White Hispanic	904 (43.7)	317 (45.5)
Black	56 (2.7)	15 (2.2)
Asian	52 (2.5)	14 (2.0)
Other	94 (4.5)	17 (2.4)
Unknown	55 (2.7)	7 (1.0)
Insurance status at study entry		
Private	799 (38.6)	295 (42.3)
Government sponsored or self-pay	1,042 (50.3)	371 (53.2)
Unknown	230 (11.1)	31 (4.5)
Census block median household income (quartiles), $		
≤36,672	516 (24.9)	176 (25.2)
>36,672–45,000	517 (25.0)	176 (25.2)
>45,000–59,375	507 (24.5)	190 (27.3)
>59,375	531 (25.6)	155 (22.2)
Census block percentage below poverty (quartiles)		
≤6.3	527 (25.4)	164 (23.5)
>6.3–14.0	515 (24.9)	178 (25.5)
>14.0–23.3	509 (24.6)	173 (24.8)
>23.3	520 (25.1)	182 (26.1)
Residence distance to treating hospital, median, km		
≤6.36	1,022 (49.3)	362 (51.9)
>6.36	1,049 (50.7)	335 (48.1)

Table 2. Distribution of Traffic-Related Air Pollution Exposures by Season[a]

Exposure and season	Mean (SD)	Minimum	25th percentile	Median	75th percentile	Maximum	Interquartile range
NO_2, ppb							
Cool	5.24 (2.39)	0.66	3.72	4.86	6.42	17.9	2.70
Warm	5.66 (2.61)	0.71	4.00	5.15	6.72	26.0	2.72
NO_x, ppb							
Cool	8.10 (3.75)	1.00	5.70	7.52	9.98	27.0	4.29
Warm	6.35 (2.99)	0.76	4.45	5.75	7.59	29.7	3.14
CO, ppm							
Cool	0.114 (0.052)	0.014	0.081	0.106	0.140	0.378	0.060
Warm	0.103 (0.048)	0.013	0.072	0.093	0.123	0.482	0.051

Abbreviations: CO, carbon monoxide; NO_2, nitrogen dioxide; NO_x, nitrogen oxide.
[a] The cool season is November through April, and the warm season is May through October. Exposures are estimated from all person-times of observation during follow-up.

Table 3. Traffic-Related Air Pollution and Repeated Hospital Encounters for Asthma in 2,768 Children Aged 0 to 18 Years

Exposure	Unadjusted HR (95% CI)[a]	*P* value	Adjusted HR (95% CI)[b]	*P* value
NO_2	1.044 (0.992–1.098)	.10	1.042 (0.987–1.101)	.14
NO_x	1.094 (1.035–1.156)	.002	1.097 (1.034–1.164)	.002
CO	1.072 (1.016–1.131)	.01	1.073 (1.013–1.137)	.02

Abbreviations: CI, confidence interval; CO, carbon monoxide; HR, hazard ratio; NO_2, nitrogen dioxide; NO_x, nitrogen oxide.
[a] Values are for an interquartile range increase in the air pollutant (NO_2, 2.68 ppb; NO_x, 4.00 ppb; and CO, 0.056 ppm).
[b] Adjusted for sex, age group, race, health insurance status, residence distance to hospital, and poverty.

Table 4. Traffic-Related Air Pollution and Repeated Hospital Encounters for Children With Asthma by Sex and Age

Model	Patients, No.	Exposure	HR (95% CI)[a]	*P* value	Product term *P* value
Sex					
Male	1,599	NO_x	1.071 (0.991–1.158)	.08	.30
		CO	1.054 (0.978–1.137	.17	.45
Female	1,169	NO_x	1.136 (1.043–1.238)	.003	Reference
		CO	1.100 (1.011–1.197)	.02	Reference
Age group, y					
0	508	NO_x	1.197 (1.075–1.333)	.02	.22
		CO	1.158 (1.041–1.289)	.007	.32
1–5	1,158	NO_x	1.042 (0.952–1.140)	.18	.52
		CO	1.021 (0.933–1.117)	.65	.44
6–18	1,102	NO_x	1.090 (0.979–1.212)	.12	Reference
		CO	1.076 (0.972–1.191)	.16	Reference

Abbreviations: CI, confidence interval; CO, carbon monoxide; HR, hazard ratio; NO_x, nitrogen oxide.
[a] Values are for an interquartile range increase in the air pollutant (NO_x, 4.00 ppb; CO, 0.056 ppm) adjusted for sex, age group, race, health insurance status, residence distance to hospital, and poverty. Stratified results are from the product term models.

there were significantly smaller associations for non-Hispanic nonwhite patients than for white patients. There was no significant interaction for residence distance to hospital or for season (data not shown).

DISCUSSION

Overview of Findings and Implications

We found that residential exposure to traffic-related air pollution is associated with increased risk of hospital encounters for asthma in children. There was some evidence that infants and girls were at highest risk. These results are consistent with those of a cross-sectional study[26] and 2 case-control studies[27,28] showing increased risk of asthma hospitalizations or other medical care visits with increasing home traffic density indices. These results are also consistent with those of a longitudinal analysis of recurrent respiratory hospital encounters using traffic indices,[20] but associations in that study were not significant for patients with a primary diagnosis of asthma.

Table 5. Traffic-Related Air Pollution and Repeated Hospital Encounters for Children With Asthma by Socioeconomic Status and Race/Ethnicity

Model	Patients, No.	Exposure	HR (95% CI)[a]	*P* value	Product term *P* value[b]
Poverty[c]					
Median or less	1,384	NO_x	1.078 (0.999–1.163)	.05	Reference
		CO	1.054 (0.979–1.134	.16	Reference
Greater than the median	1,384	NO_x	1.116 (1.026–1.214)	.01	.39 (.70)
		CO	1.094 (1.006–1.190)	.04	.49 (.71)
Median household income[d]					
Greater than the median	1,383	NO_x	1.145 (1.054–1.244)	.001	Reference
		CO	1.120 (1.034–1.213)	.005	Reference
Median or less	1,385	NO_x	1.068 (0.983–1.160)	.12	.23 (.09)
		CO	1.041 (0.959–1.129)	.34	.20 (.07)
Insurance status					
Private	1,094	NO_x	1.136 (1.036–1.247)	.007	Reference
		CO	1.102 (1.006–1.206)	.04	Reference
Government sponsored or self-pay	1,413	NO_x	1.080 (1.005–1.160)	.04	.38
		CO	1.061 (0.989–1.138)	.10	.51
Unknown	261	NO_x	0.886 (0.569–1.379)	.59	.28
		CO	0.913 (0.591–1.412)	.68	.41
Race/ethnicity					
White	1,237	NO_x	1.145 (1.055–1.243)	.001	Reference
		CO	1.113 (1.027–1.205)	.009	Reference
Hispanic	1,221	NO_x	1.097 (1.008–1.193)	.03	.46
		CO	1.081 (0.996–1.173)	.06	.62
Non-Hispanic nonwhite	310	NO_x	0.829 (0.624–1.102	.20	.03
		CO	0.804 (0.601–1.074)	.14	.03

Abbreviations: CI, confidence interval; CO, carbon monoxide; HR, hazard ratio; NO_x, nitrogen oxide.

[a] Values are for an interquartile range increase in the air pollutant (NO_x, 4.00 ppb; CO, 0.056 ppm) adjusted for sex, age group, race, health insurance status, residence distance to hospital, and poverty. Stratified results are from product term models.

[b] The *P* value for interaction of air pollution with socioeconomic variables and race/ethnicity. The *P* value for trend from continuous poverty and median household income is given in parentheses.

[c] The median of Census 2000 block group percentage below the federal poverty level was 14%.

[d] The median of Census 2000 block group of median household income was $45,000.

The present results support the use of dispersion modeling for evaluating traffic-related exposures, but other methods involving direct home or neighborhood air pollutant measurements have been proposed to further limit exposure error.[6]

We did not include background pollutant concentrations because variability is likely low within the 13-km study radius. Dispersion-modeled gases are considered surrogates of other more toxic gases and particles emitted from nearby diesel trucks and automobiles, including ultrafine particles that have been found at notably higher concentrations near roadways along with black carbon, particle number, and CO.[29] The stronger associations for NO_x compared with NO_2 support this because NO_2 is strongly affected by photochemical reactions that can occur across time away from roadways, whereas NO_x is expected to capture traffic emissions more generally.[30] Key pollutants likely represented by NO_x and CO are those carried by ultrafine particles. Ultrafine particles carry more redox-active components than larger particles, which are more spatially homogenous.[7,31,32] Based in large part on experimental evidence, it has been hypothesized that particles from vehicular exhaust, especially in the ultrafine range, can trigger oxidative stress. When antioxidant responses are then overwhelmed, airway inflammation may follow, leading to increasing asthma symptoms in susceptible children.[19,33]

Potentially Susceptible Subgroups

There was no significant difference in association by sex or age group, and widened confidence intervals in stratified results suggest that subsample sizes may have limited the ability to compare these groups. Nevertheless, the largest associations were for infants, followed by children aged 6 to 18 years, in whom the diagnosis of asthma is clearest. There was limited evidence of stronger associations in girls, consistent with other studies of traffic-related air pollution and respiratory outcomes.[10,20,27,34] The underlying reasons for sex differences are unknown.

Significant associations for NO_x and CO in infants are intriguing. Approximately half of the repeated encounters in this group occurred between ages 1 and 3 years. These findings suggest that early-life exposures to traffic pollutants may affect asthma severity and development. This view is

supported by studies[12–18] of preschool children that have found increased risk of incident asthma or wheeze or cough without a cold from long-term exposures to local traffic–related air pollutants using GIS-based methods. A recent study[35] showed acute increases in wheeze occurrence with elevations in daily regional NO_2 and NO_x levels in infants and children followed up during their first 3 years of life. These findings are consistent with emerging views that gene-environment interactions during early life are important in the prognosis of early-onset wheeze and in the development of lung function deficits and asthma in later life.[36,37]

In contrast to previous findings,[20] we did not find stronger associations in children without insurance or with government-sponsored insurance than in children with private insurance. Instead, there were significantly stronger associations in patients living in census block groups in the upper half of the distribution of median household income. However, there was no significant difference in associations between white and Hispanic patients (the predominant minority group). Both groups showed significant or nearly significant associations for NO_x and CO. The remaining minorities (blacks, Asians, and others) showed significantly smaller null associations compared with white patients. The distribution of median household income by race/ethnicity did not explain these findings because only Hispanic patients showed significantly more families below the median income distribution (61%) compared with white patients (40%) and the remaining minorities (44%) ($P < .001$ by χ^2 test).

Evaluating community-level contextual factors may be important in understanding the heterogeneity in asthma expression and risk.[38] Environmental disparities, such as exposure to traffic and indoor allergens, have been proposed to explain the increased asthma burden in minority and lower socioeconomic groups.[39] Although the present findings do not support this hypothesis, the number of potentially important environmental factors that differ by communities is large[39] and mostly unmeasured in the present study. Furthermore, we speculate that these findings of smaller associations in children living in lower-income census block groups may have been attributable to 2 factors: (1) less consistent exposure as evaluated at study entry due to less stable residence and (2) less consistent outcome data due to more variable use of hospitals, including those not evaluated in this study. To test this possibility, we successfully contacted parents and administered a short survey for 250 randomly selected patients aged 0 to 8 years seen at the participating EDs or admitted to the hospitals with lower respiratory tract illnesses. Of 103 respondents with nonmissing data, those with survey-reported household annual incomes less than $30,000 (N = 58) were significantly more likely to have lived in the same residence for 12 or fewer months (29%) than were the 45 families making $30,000 or more (9%) ($P < .02$ by χ^2 test). In addition, those with annual household incomes less than $30,000 were more likely to have gone to a hospital not captured in the surveillance data (26%) than were families making at least $30,000 (13%) ($P < .12$ by χ^2 test).

There are several limitations to the present study design. First, some children used other hospitals, and, therefore, the outcome ascertainment is incomplete and the censoring assumption underlying the analysis is subject to some error. We also did not directly contact parents. Therefore, we could not ascertain any change in residence after the first event, leading to potential exposure error. Additional exposure error comes from unmeasured exposures occurring when children are away from their residence. We also did not have data on other known or suspected risk factors that may have confounded associations, including family history of asthma and environmental exposures (eg, second-hand smoke, aeroallergens, endotoxin, family size).[38,39] This may have led to misclassification of risk. For example, differences in the distribution of indoor and outdoor allergen triggers may have biased associations. Because the study used a retrospective cohort design, it was not possible to obtain allergen measurements during times at risk. Such measures could be used in future prospective cohort studies.

Finally, we could not confirm asthma diagnoses independently, which is especially important in younger patients, in whom lower respiratory tract illnesses can often induce asthma-like symptoms that resolve at later ages. Asthma diagnosis can be made using objective methods, such as spirometry, at school ages. Nevertheless, given other evidence,[12,40] it is conceivable that air pollutants also enhanced the propensity toward lower respiratory tract illness–related wheeze in the present population.

Conclusions

Traffic-related NO_x and CO were associated with repeated hospital encounters for asthma in children, suggesting that traffic-generated air pollution near the home affects asthma symptom severity. These findings suggest that this potential risk may begin during infancy. Early lower respiratory tract illness with recurrent wheeze symptoms can increase asthma risk in later childhood, when the diagnosis of asthma is clearer.[41,42] Evidence from the present study supports a possible role of pollutants from traffic emissions in this progression.

Cohort studies of asthma risk in children to date have focused on general populations or on children with family histories of atopy. Prospective environmental data are sparse for high-risk populations who present to the hospital with asthma exacerbations. Additional work with improved assessments of air pollutant exposures and asthma outcomes in such high-risk populations is likely to be fruitful given the present results.

REFERENCES

1. Kim JJ; American Academy of Pediatrics Committee on Environmental Health. Ambient air pollution: health hazards to children. *Pediatrics*. 2004;114:1699–1707.
2. Delfino RJ, Quintana PJE, Floro J, et al. Association of FEV_1 in asthmatic children with personal and microenvironmental exposure to airborne particulate matter. *Environ Health Perspect*. 2004;112:932–941.
3. Delfino RJ, Staimer N, Gillen D, et al. Personal and ambient air pollu-

tion is associated with increased exhaled NO in children with asthma. *Environ Health Perspect*. 2006;114:1736–1743.
4. Delfino RJ, Staimer N, Tjoa T, et al. Personal and ambient air pollution exposures and lung function decrements in children with asthma. *Environ Health Perspect*. 2008;116:550–555.
5. Koenig JQ, Jansen K, Mar TF, et al. Measurement of offline exhaled nitric oxide in a study of community exposure to air pollution. *Environ Health Perspect*. 2003;111:1625–1629.
6. Jerrett M, Arain A, Kanaroglou P, et al. A review and evaluation of intraurban air pollution exposure models. *J Expo Anal Environ Epidemiol*. 2005;15:185–204.
7. Sioutas C, Delfino RJ, Singh M. Exposure assessment for atmospheric ultrafine particles (UFP) and implications in epidemiological research. *Environ Health Perspect*. 2005;113:947–955.
8. South Coast Air Quality Management District. 2003 Air Quality Management Plan (AQMP). http://www.aqmd.gov/aqmp/aqmd03aqmp.htm. Accessed November 1, 2008.
9. Delfino RJ. Think globally, breath locally: why the worldwide health impact of air pollution on young children begins in our neighborhoods. *Thorax*. 2006;61:184–185.
10. McConnell R, Berhane K, Yao L, et al. Traffic, susceptibility, and childhood asthma. *Environ Health Perspect*. 2006;114:766–772.
11. Salam MT, Islam T, Gilliland FD. Recent evidence for adverse effects of residential proximity to traffic sources on asthma. *Curr Opin Pulm Med*. 2008;14:3–8.
12. Brauer M, Hoek G, Smit HA, et al. Air pollution and the development of asthma, allergy and infections in a birth cohort. *Eur Respir J*. 2007;29:879–888.
13. Clougherty JE, Levy JI, Kubzansky LD, et al. Synergistic effects of traffic-related air pollution and exposure to violence on urban asthma etiology. *Environ Health Perspect*. 2007;115:1140–1146.
14. Morgenstern V, Zutavern A, Cyrys J, et al. Atopic diseases, allergic sensitization, and exposure to traffic-related air pollution in children. *Am J Respir Crit Care Med*. 2008;177:1331–1337.
15. Nordling E, Berglind N, Melén E, et al. Traffic-related air pollution and childhood respiratory symptoms, function and allergies. *Epidemiology*. 2008;19:401–408.
16. Pierse N, Rushton L, Harris RS, Kuehni CE, Silverman M, Grigg J. Locally generated particulate pollution and respiratory symptoms in young children. *Thorax*. 2006;61:216–220.
17. Ryan PH, LeMasters G, Biagini J, et al. Is it traffic type, volume, or distance? wheezing in infants living near truck and bus traffic. *J Allergy Clin Immunol*. 2005;116:279–284.
18. Ryan PH, Lemasters GK, Biswas P, et al. A comparison of proximity and land use regression traffic exposure models and wheezing in infants. *Environ Health Perspect*. 2007;115:278–284.
19. Nel AE, Diaz-Sanchez D, Li N. The role of particulate pollutants in pulmonary inflammation and asthma: evidence for the involvement of organic chemicals and oxidative stress. *Curr Opin Pulm Med*. 2001;7:20–26.
20. Chang J, Delfino RJ, Gillen D, Tjoa T, Nickerson B, Cooper D. Repeated respiratory hospital encounters among children with asthma and residential proximity to traffic. *Occup Environ Med*. In press.
21. Laurent O, Pedrono G, Segala C, et al. Air pollution, asthma attacks, and socioeconomic deprivation: a small-area case-crossover study. *Am J Epidemiol*. 2008;168:58–65.
22. Gauderman WJ, Avol E, Lurmann F, et al. Childhood asthma and exposure to traffic and nitrogen dioxide. *Epidemiology*. 2005;16:737–743.
23. Benson P. *CALINE4: A Dispersion Model for Predicting Air Pollutant Concentrations Near Roadways*. Sacramento: California Department of Transportation; 1989.
24. Lin D, Wei L, Yang I, Ying Z. Semiparametric regression for the mean and rate functions of recurrent events. *J R Statist Soc B*. 2000;62:711–730.
25. Lin D, Wei L. Robust inference for the Cox proportional hazards model. *J Am Stat Assoc*. 1989;84:1074–1078.
26. Wilhelm M, Meng YY, Rull RP, English P, Balmes J, Ritz B. Environmental public health tracking of childhood asthma using California Health Interview Survey, traffic, and outdoor air pollution data. *Environ Health Perspect*. 2008;116:1254–1260.
27. English P, Neutra R, Scalf R, Sullivan M, Waller L, Zhu L. Examining associations between childhood asthma and traffic flow using a geographic information system. *Environ Health Perspect*. 1999;107:761–767.
28. Lin S, Munsie JP, Hwang SA, Fitzgerald E, Cayo MR. Childhood asthma hospitalization and residential exposure to state route traffic. *Environ Res*. 2002;88:73–81.
29. Zhu YF, Hinds WC, Kim S, Sioutas C. Concentration and size distribution of ultrafine particles near a major highway. *J Air Waste Manage Assoc*. 2002;52:1032–1042.
30. Atkinson R. Atmospheric chemistry of VOCs and NOx. *Atmos Environ*. 2000;34:2063–2101.
31. Li N, Sioutas C, Cho A, et al. Ultrafine particulate pollutants induce oxidative stress and mitochondrial damage. *Environ Health Perspect*. 2003;111:455–460.
32. Ntziachristos L, Froines JR, Cho AK, Sioutas C. Relationship between redox activity and chemical speciation of size-fractionated particulate matter. *Part Fibre Toxicol*. 2007;4:5.
33. Gilmour MI, Jaakkola MS, London SJ, et al. How exposure to environmental tobacco smoke, outdoor air pollutants, and increased pollen burdens influences the incidence of asthma. *Environ Health Perspect*. 2006;114:627–633.
34. Oftedal B, Brunekreef B, Nystad W, et al. Residential outdoor air pollution and lung function in schoolchildren. *Epidemiology*. 2008;19:129–137.
35. Andersen ZJ, Loft S, Ketzel M, et al. Ambient air pollution triggers wheezing symptoms in infants. *Thorax*. 2008;63:710–716.
36. Saglani S, Bush A. The early-life origins of asthma. *Curr Opin Allergy Clin Immunol*. 2007;7:83–90.
37. Stern DA, Morgan WJ, Halonen M, Wright AL, Martinez FD. Wheezing and bronchial hyper-responsiveness in early childhood as predictors of newly diagnosed asthma in early adulthood: a longitudinal birth-cohort study. *Lancet*. 2008;372:1058–1064.
38. Wright RJ, Subramanian SV. Advancing a multilevel framework for epidemiologic research on asthma disparities. *Chest*. 2007;132:757S–769S.
39. Gold DR, Wright R. Population disparities in asthma. *Annu Rev Public Health*. 2005;26:89–113.
40. Romieu I, Samet JM, Smith KR, Bruce N. Outdoor air pollution and acute respiratory infections among children in developing countries. *J Occup Environ Med*. 2002;44:640–649.
41. Castro-Rodriguez JA, Holberg CJ, Wright AL, Martinez FD. A clinical index to define risk of asthma in young children with recurrent wheezing. *Am J Respir Crit Care Med*. 2000;162:1403–1406.
42. Henderson J, Granell R, Heron J, et al. Associations of wheezing phenotypes in the first 6 years of life with atopy, lung function and airway responsiveness in mid-childhood. *Thorax*. 2008;63:974–980.

Requests for reprints should be addressed to:
Ralph J. Delfino, MD, PhD
Department of Epidemiology
School of Medicine
University of California, Irvine
100 Theory Dr
Ste 100
Irvine, CA 92617-7555
E-mail: rdelfino@uci.edu

Childhood Asthma and Exposure to Traffic and Nitrogen Dioxide

W. James Gauderman,[*] Edward Avol,[*] Fred Lurmann,[†] Nino Kuenzli,[*] Frank Gilliland,[*] John Peters,[*] and Rob McConnell[*]

Background: Evidence for a causal relationship between traffic-related air pollution and asthma has not been consistent across studies, and comparisons among studies have been difficult because of the use of different indicators of exposure.

Methods: We examined the association between traffic-related pollution and childhood asthma in 208 children from 10 southern California communities using multiple indicators of exposure. Study subjects were randomly selected from participants in the Children's Health Study. Outdoor nitrogen dioxide (NO_2) was measured in summer and winter outside the home of each child. We also determined residential distance to the nearest freeway, traffic volumes on roadways within 150 meters, and model-based estimates of pollution from nearby roadways.

Results: Lifetime history of doctor-diagnosed asthma was associated with outdoor NO_2; the odds ratio (OR) was 1.83 (95% confidence interval = 1.04–3.22) per increase of 1 interquartile range (IQR = 5.7 ppb) in exposure. We also observed increased asthma associated with closer residential distance to a freeway (1.89 per IQR; 1.19–3.02) and with model-based estimates of outdoor pollution from a freeway (2.22 per IQR; 1.36–3.63). These 2 indicators of freeway exposure and measured NO_2 concentrations were also associated with wheezing and use of asthma medication. Asthma was not associated with traffic volumes on roadways within 150 meters of homes or with model-based estimates of pollution from nonfreeway roads.

Conclusions: These results indicate that respiratory health in children is adversely affected by local exposures to outdoor NO_2 or other freeway-related pollutants.

(*Epidemiology* 2005;16: 737–743)

Previous studies have demonstrated a link between outdoor air pollution and the occurrence of symptoms in children already diagnosed with asthma.[1] However, results are not consistent with respect to whether air pollution causes asthma. Most studies have found little evidence to support an association between community-average exposures to air pollution and community asthma prevalence.[2] These study designs failed to account for the variability in exposure resulting from vehicular traffic in urban areas. Asthma has been associated with local variation in traffic patterns within communities in many,[3–7] but not all,[8–11] studies that have examined the impact of local traffic. One possible reason for the inconsistency in these recent studies is the use of different indicators of traffic-related pollution. Some have measured pollutant exposure at home, some have estimated traffic volume near the home, and some have estimated exposure to traffic-related pollutants at home based on dispersion models. Little work has been done to validate estimates of traffic exposure against measured pollution concentrations. Most studies have been conducted in European cities, which differ from U.S. cities in the layout of streets and homes, and also in the relative proportion of diesel- to gasoline-powered vehicles.

We evaluated several commonly available indicators of traffic exposure and compared them with nitrogen dioxide (NO_2) levels measured at the homes of subjects participating in the Children's Health Study. The Children's Health Study was initiated in 1993 with a cohort of school-aged children from 12 southern California communities representing a wide range in air quality. To date, this study has reported associations between air pollution and several outcomes, including lung function,[12–15] respiratory symptoms in asthmatics,[16,17] and asthma incidence.[18] These analyses have relied on com-

Submitted 12 October 2004; accepted 7 February 2005.

From the *Department of Preventive Medicine, University of Southern California Keck School of Medicine, Los Angeles, California; and †Sonoma Technology, Inc., Petaluma, California.

Supported in part by the California Air Resources Board (Contract A033-186), the National Institute of Environmental Health Sciences (5P30ES07048 and 1P01ES11627), the Southern California Particle Center and Supersite, the Environmental Protection Agency (grant R 82670801), and the Hastings Foundation.

Supplemental material for this article is available with the online version of the journal at www.epidem.com; click on "Article Plus."

Correspondence: W. James Gauderman, Department of Preventive Medicine, University of Southern California, 1540 Alcazar St., Suite 220, Los Angeles, CA 90089. E-mail: jimg@usc.edu.

ISSN: 1044-3983/05/1606-0737
DOI: 10.1097/01.ede.0000181308.51440.75

parisons of average health across communities in relation to the pollution levels measured at a central site monitor in each community. In 2000, we conducted a study to measure NO_2 levels at a random sample of children's homes within each of the study communities. We examine how local variation in NO_2 and indicators of exposure to traffic-related pollutants are related to each other, and whether they are associated with lifetime prevalence of asthma and asthma-related outcomes.

METHODS

Study Subjects

In calendar year 2000, we measured outdoor NO_2 levels at the homes of randomly selected participants in the Children's Health Study. Eligible children included those who were originally enrolled as fourth graders (average age = 10 years) in 1993 (cohort 1) or 1996 (cohort 2), with the additional criteria that in 2000, they were still actively participating in the study and had lived in the same home since study enrollment. We excluded 2 of the 12 study communities (Lompoc and Lake Arrowhead) from this study, because neither has any major sources of traffic. From the pool of 890 eligible subjects, we randomly sampled 229 children for NO_2 monitoring. Samplers were deployed outside each home for 2-week periods in the summer and fall of 2000. Valid measurements in both seasons were obtained at 208 (91%) of the homes. Reasons for invalid measurements included lost samplers, subjects who moved, and difficulties with field access or deployment. The study protocol was approved by the Institutional Review Board for Human Studies at the University of Southern California, and informed consent was provided by a parent or legal guardian for all study subjects.

Nitrogen Dioxide Sampling

Ambient NO_2 was sampled with Palmes tubes.[19] These diffusion-based samplers have been widely used in several microenvironmental and personal air quality studies.[20–22] We deployed samplers outside the homes of study subjects, thus avoiding previously identified confounders such as indoor nitrous acid formation, gas stoves, or wall heaters. Samplers were attached at the roofline eaves, signposts, or rain gutters at an approximate height of 2 meters above the ground, oriented in a downward position and protected by an oversized paper cup. Duplicate samplers and field travel blanks were randomly assigned to approximately 10% of the subjects' homes. Samplers were deployed for 2-week periods in both summer (mid-August) and fall (mid-November) in all communities. Deployment across communities was accomplished over a 4-day period at the start of the summer and fall field sampling periods. Within any 1 community, samplers at all locations were deployed within a 4-hour period, and 2 weeks later the samplers were retrieved within a 4-hour period. Samplers were transported to and from the field in cooled portable ice chests. The samplers were prepared for field use and analyzed at the Harvard School of Public Health.

Traffic Exposures

We characterized exposure of each study participant to traffic-related pollutants by 3 metrics: (1) proximity of the residence to the nearest freeway; (2) average number of vehicles traveling within 150 meters of the residence each day, including vehicles on freeways, arterials, major collector roads, and (where available) on minor collector roads; and (3) model-based estimates of traffic-related air pollution at the residence, derived from dispersion models that incorporate distance to roadways, vehicle counts, vehicle emission rates, and meteorologic conditions. Methods used to estimate each of these exposure factors are described subsequently.

Residence addresses were standardized and their locations geocoded using the TeleAtlas database and software (Tele Atlas Inc., Menlo Park, CA, www.na.teleatlas.com). We used the TeleAtlas MultiNet USA database, a comprehensive geo-positioning-satellite-accurate database of roadways, for all analyses because it is more accurate than the standard files available from the U.S. Census. To estimate distance to the nearest freeway, we used ERSI ArcGIS Version 8.3 (ESRI, Redland, CA, www.esri.com) software tools to calculate the distance from each residence to the nearest interstate freeway, U.S. highway, or limited access highway. In these calculations, each direction of travel was represented as a separate roadway, and the "distance to nearest freeway" was the shortest distance from the residence to the middle of the nearest set of lanes of the freeway.

To estimate vehicle counts near homes, annual average daily traffic volumes were obtained from the California Department of Transportation (CALTRANS) Highway Performance Monitoring System for the year 2000. The traffic volumes were transferred from the CALTRANS roadway network to the TeleAtlas networks using previously described methods.[23] The hourly traffic volumes on weekdays and weekend days were estimated from the annual average daily traffic volumes and the average diurnal and day-of-week freeway and nonfreeway traffic variations observed in Southern California. These data were used to calculate the daily average number of vehicles traveling within 150 meters of each residence, weighted by inverse distance from the home to each road. This local traffic density was expressed as traffic volume per square meter.

To obtain model-based estimates of traffic-related pollution exposure, we used the CALINE4 line-source air-quality dispersion model.[24] Principal model inputs included roadway link geometry, link traffic volumes, meteorologic conditions (wind speed and direction, atmospheric stability, and mixing heights), and vehicle emission rates. The 5-year

average joint distributions of wind speeds and directions were obtained from 1 surface-monitoring station in or near each study community. The dispersion model was applied to simulate the transport and dispersion of NO_x as a chemically inert pollutant. Although NO, NO_2, and ozone undergo rapid atmospheric chemical reactions immediately downwind of sources, NO_x can be treated as a chemically inert pollutant for the first hour of transport from sources because the time-scale for NO_x oxidation is 10 to 20 hours in urban atmospheres.[25] Vehicle NO_x emission rates were obtained from the California Air Resources Board's EMFAC2002 vehicle emissions model. Concentrations of NO_2 were estimated by applying the annual average ratio of observed NO_2 to NO_x for each hour of the day (from the community central site monitor) to the CALINE4 model's estimated NO_x concentrations. We estimated the contribution to residential exposure separately for freeway and for nonfreeway traffic.

Ambient NO_2 concentrations in the community are a result of meteorologic transport of pollutants into the community, local point and area source emissions, and local mobile source emissions. The CALINE4 model was used to model NO_2 from local traffic in each community and, therefore, always predicts concentrations lower than the total NO_2 from all sources. Separate regional modeling analysis has indicated that local mobile source emissions contribute 12% to 68% of the average NO_2 in the study communities.[23] For comparison purposes, we also generated exposure assignments based on fine particulate matter (PM) and carbon monoxide (CO) emission factors. Model-based estimates of NO_2, PM, and CO were very highly correlated with one another ($R > 0.90$), indicating that the NO_2-based estimates we use in this article should be considered an estimate of traffic-related pollution in general rather than simply exposure to this specific pollutant.

Questionnaire Data

When we originally enrolled subjects as fourth graders, each subject's parent or legal guardian completed a baseline medical history questionnaire. Asthma was defined as a "yes" response to the question "Has a doctor ever diagnosed your child as having asthma?" This questionnaire was also used to determine whether the child had recently (within the last 12 months) wheezed, recently wheezed during exercise, or was currently using any type of medication to control asthma. Questions about potential risk factors for asthma included parental income or education, environmental tobacco smoke exposure, in utero exposure to maternal tobacco smoking, and presence in the home of mildew, water damage, gas stove, pests, and pets.

Statistical Analysis

We used logistic regression to model the relationship of each traffic measure, including measured NO_2 at the home and the traffic indicators described previously, with baseline asthma prevalence in the 208 study participants. A natural-log transformation of each traffic indicator was used in these analyses, because the distribution of each variable was positively skewed. All models included adjustments for sex, race, Hispanic ethnicity, cohort (whether the subject was enrolled in 1993 or 1996), and indicator variables for study community. We considered separate models for 2-week average NO_2 concentrations measured in summer and in winter and for the 4-week average across seasons. Odds ratios (ORs) for asthma in analyses of measured NO_2 concentrations were scaled to an increase of 5.7 ppb, the average interquartile range (IQR) in 4-week average NO_2 within the 10 communities. ORs for the traffic indicators were also scaled to 1 IQR in exposure (specifically 1.2 km for distance to the nearest freeway; 2720 vehicles per m^2 per day for traffic volumes within 150 meters; and 0.64, 0.49, and 1.27 ppb for model-based estimates of NO_2 from freeways, nonfreeways, and all roads, respectively).

RESULTS

Doctor-diagnosed asthma was reported by 31 (15%) of the 208 children, with variability in prevalence across communities (Table 1). Overall community-average NO_2 levels measured at homes ranged from 12.9 ppb in Atascadero to 51.5 ppb in San Dimas, with similar patterns across communities in summer and winter. The NO_2 levels (average of summer and winter) measured at homes are shown in Figure 1. Within each community, there was substantial variation in NO_2 levels from home to home. Although the amount of variation in NO_2 was generally larger in more polluted communities, there were some exceptions. For example, there was little variation in the relatively high NO_2 community of Mira Loma, whereas there was considerable variation in the lower NO_2 community of Alpine.

The average NO_2 concentration measured at homes was associated with asthma prevalence (Table 2). For each increase of 5.7 ppb in average NO_2, the OR for asthma increased by 1.83 (95% CI = 1.04–3.21). Odds ratios were similar whether based on summer-only (1.55) or winter-only (1.50) measurements. The effect of average NO_2 was of similar magnitude after adjustment for several potential confounders, including socioeconomic status of participants and housing characteristics (Table 2).

Measured NO_2 concentrations at homes were correlated with residential distance from the nearest freeway and with model-based estimates of traffic-related pollution from roadways (Appendix Table, available with the online version of this article). In each community, we observed negative correlations between NO_2 concentration and distance of the home to the freeway. The overall correlation between NO_2 and freeway distance, adjusted for community, was $R = -0.54$. The corresponding correlations of measured NO_2

TABLE 1. Distribution of Lifetime History of Asthma and Measured NO_2 by Community (n = 208)

Community	No.	Asthma (%)	NO_2 (ppb)		
			Summer	Winter	Average†
Alpine (AL)	24	21	20.1	19.0	19.6
Atascadero (AT)	13	23	12.3	13.6	12.9
Lake Elsinore (LE)	22	5	17.6	27.4	22.5
Lancaster (LN)	16	19	16.9	22.0	19.5
Long Beach (LB)	20	10	34.6	50.5	42.5
Mira Loma (ML)	17	12	37.2	48.4	42.8
Riverside (RV)	30	20	37.9	42.8	40.3
San Dimas (SD)	34	15	52.0	51.0	51.5
Santa Maria (SM)	19	16	12.7	17.9	15.3
Upland (UP)	13	8	46.3	36.0	41.2

*Parent report of doctor-diagnosed asthma in the child.
†Mean in each community of NO_2 concentrations measured at homes for 2 weeks each in summer and winter. Average is the 4-week arithmetic average of summer and winter measurements.

with model-based estimates were 0.56 for pollution from freeways and 0.34 for pollution from nonfreeways. In each community, measured NO_2 was more strongly correlated with estimates of freeway-related pollution than with nonfreeway pollution. Measured NO_2 was less correlated with traffic counts within 150 meters of homes (R = 0.24), with inconsistent patterns of correlations from community to community.

Both distance to the freeway and the model-based estimate of freeway-related pollutants were associated with asthma history (Table 3). Asthma prevalence was higher with decreasing distance from the freeway; specifically when comparing the 25th to 75th percentile of freeway distance, the OR was 1.89 (95% CI = 1.19–3.02). For the comparison of 75th to 25th percentile of model-based pollutant exposure from freeways, the OR was 2.22 (1.36–3.63). Asthma was not associated with traffic volumes or with model-based exposure to nonfreeway roads. The associations observed with freeway distance and model-based pollution from freeways were robust to adjustment for all of the potential confounders shown in Table 2 (data not shown).

Measured NO_2 and the 2 freeway-related traffic indicators were also associated with recent wheeze, recent wheeze with exercise, and current use of asthma medication

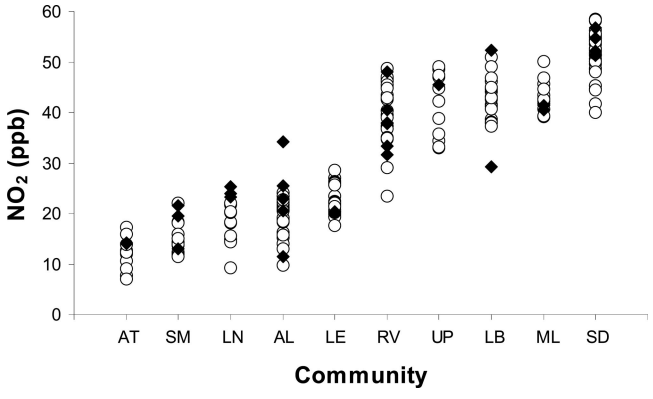

FIGURE 1. Four-week average of nitrogen dioxide measured at homes of asthmatic (solid black diamond) and nonasthmatic (open circle) children in 10 communities. See Table 1 for community abbreviations.

TABLE 2. Association Between 4-Week Average NO_2 at Homes and Asthma History, Adjusted for Several Potential Confounders

Description	OR* (95% CI)
Base model†	1.83 (1.04–3.21)
Base model, with additional adjustment for:	
Environmental tobacco smoke	1.93 (1.09–3.43)
In utero exposure to maternal smoking	1.85 (1.05–3.28)
Parental income	1.99 (1.11–3.57)
Parental education	1.90 (1.07–3.37)
Gas stove	1.87 (1.06–3.30)
Mildew	1.81 (1.01–3.23)
Water damage	1.82 (1.03–3.21)
Cockroaches	1.83 (1.04–3.21)
Pets	1.88 (1.06–3.33)

*Odds ratio per increase of 1 interquartile range (5.7 ppb) in NO_2.
†Base model includes adjustments for sex, race, Hispanic ethnicity, cohort, and community.

TABLE 3. Associations Between Exposure to Traffic at Home and Asthma History

Exposure Metric	Odds Ratio per IQR OR* (95% CI)
Distance to freeway	1.89 (1.19–3.02)
Traffic volume within 150 meters	1.45 (0.73–2.91)
Model-based pollution from:	
Freeways	2.22 (1.36–3.63)
Other roads	1.00 (0.75–1.33)
Freeways and other roads	1.40 (0.86–2.27)

*Odds ratio per change of 1 IQR. For distance to freeway, OR for the 25th percentile compared with the 75th percentile (ie, living closer compared with farther from the freeway). For remaining traffic variables, OR for the 75th percentile compared with the 25th percentile. All models were adjusted for sex, race, Hispanic ethnicity, cohort, and community.

(Table 4). For example, the OR per increase of 5.7 ppb in measured NO_2 was 1.72 (1.07–2.77) for recent wheeze and was 2.19 (1.20–4.01) for current use of asthma medication.

DISCUSSION

We found robust associations of several indicators of exposure to traffic-related air pollution at homes in southern California with lifetime history of asthma, current asthma medication use, recent wheeze, and recent exercise-induced wheeze. Residential distance to a freeway and model-based estimates of freeway traffic-emission exposure at homes were each associated with the prevalence of asthma. Each of these traffic metrics was also correlated with measured concentrations of NO_2, and measured NO_2 was associated with asthma. Taken as a whole, these results indicate that exposure to outdoor levels of NO_2 or other freeway-related pollutants was a significant risk factor for asthma.

A strength of this asthma study is that it used both measured pollution and multiple indicators of exposure to traffic at the same homes in a large number of communities. The results suggest that measuring NO_2 or another pollutant is important for validation of the use of traffic measures and for selection of the most appropriate indicator of traffic exposure for the population under study. Those few studies that have measured residential exposure or that have validated models of exposure using measurements of pollutants have generally shown associations with asthma,[6,7,26] whereas the failure to validate traffic indicators may explain inconsistent results from several other studies.[8–11] In our study, simple distance to a freeway was as strongly and precisely associated with asthma and wheeze as was NO_2. It remains to be seen whether the association with this simple and widely available indicator is replicable in other studies or could be used for estimating risk in communities without having to make additional measurements of traffic-related pollutants.

We did not find associations between respiratory health and other indicators of traffic near homes, including modeled pollution from nonfreeway roads and traffic volumes within 150 meters of homes. One possible explanation for this lack of association is that the contribution to pollution levels from these smaller roads (where tens or hundreds of vehicles travel each day) is trivial compared with freeways that dominate the transportation grid in southern California with daily average counts in our communities between 50,000 to 270,000 vehicles. In addition, vehicle counts are accurately measured on freeways but are only estimated on smaller roads where participants lived. Our results are in contrast to several recent (mostly European) studies that have reported associations of asthma with traffic counts in close proximity to the home.[6,7,27,28] These differences in results may be partly the result of differences in urban geography and closer proximity of homes in Europe to heavily traveled roadways.

There have been a few other studies of traffic and childhood asthma in the United States. One large study in southern California found no association of asthma prevalence with traffic counts within 550 feet of the home,[9] similar to our finding of no association with traffic volumes within 150 meters of the home. Consistent with our findings related to measured NO_2, a recent study in northern California[29] found an association between measured traffic-related pollutants at schools and childhood asthma.

TABLE 4. Associations Between Measured NO_2 and Asthma-Related Outcomes (n = 208)

Outcome	No.	Measured NO_2 OR* (95% CI)	Distance to Freeway OR* (95% CI)	Model-based Pollution From Freeways OR* (95% CI)
Lifetime history of asthma	31	1.83 (1.04–3.21)	1.89 (1.19–3.02)	2.22 (1.36–3.63)
Recent wheeze†	43	1.72 (1.07–2.77)	1.59 (1.06–2.36)	1.70 (1.12–2.58)
Recent wheeze with exercise†	25	2.01 (1.08–3.72)	2.57 (1.50–4.38)	2.56 (1.50–4.38)
Current asthma medication use	26	2.19 (1.20–4.01)	2.04 (1.25–3.31)	1.92 (1.18–3.12)

*Odds ratio per change of 1 IQR in exposure (see footnotes to Tables 2 and 4).
†Within the last 12 months.

The observed associations of traffic with asthma are biologically plausible. Increased oxidative and nitrosative stress associated with NO_2 exposure may impair respiratory responses to infection and thus result in lung injury and asthma exacerbation.[20,30] However, the association of NO_2 with asthma prevalence has been extensively evaluated in epidemiologic studies of exposure to indoor sources, often at levels considerably higher than the modest (5.7 ppb) IQR of exposure in our study, and the observed associations have not been consistent.[30,31] It is possible that outdoor NO_2, which occurs in a complex mixture that includes particulate matter and other pollutants known to affect respiratory health, is a marker of some other traffic-related pollutant(s) responsible for increasing asthma risk. For example, some field studies suggest that the concentration of fine particulate matter, especially black smoke (an indicator of diesel exhaust), varies with nearby high-traffic roads and with NO_2.[32–35] It has been hypothesized that particulate matter, especially diesel exhaust particulate, may contribute to the development of allergies and asthma.[36] Additional research is needed to study the health effects of specific pollutants that occur in complex mixtures of traffic emissions.

A possible limitation of this study is the assessment of asthma by questionnaire, which could be affected by access to care and differences in diagnostic practice among physicians.[37] However, we found associations of traffic indicators with recent wheeze and exercise-induced wheeze, 2 symptoms of asthma that are unlikely to be affected by access to care or diagnostic bias. Another limitation is the possibility of poor or biased reporting of asthma by parents. However, self-report of physician-diagnosed asthma has been found to reflect what physicians actually reported to patients, at least in adults, and validity as assessed by repeatability of response is good.[38] Self-report of physician diagnosis has been the main criterion for identifying asthma in epidemiologic studies of children and has been recommended as the epidemiologic gold standard because a more precise identification tool is not available.[39] Reporting bias is unlikely to have explained the observed associations, because parents were not aware of the specific focus of the study on air pollution at the time the questionnaire was completed. Biased participation with respect to disease status in this substudy is also unlikely, because the prevalence of doctor-diagnosed asthma in the sample of 208 children (15%, Table 1) was not very different from the asthma prevalence in the remaining 668 eligible children (13%, $P = 0.56$).

Another potential study limitation is that measured NO_2 and the traffic metrics were determined after the onset of asthma and extrapolated to earlier in life. However, the systems of freeways and other major roadways in the study communities have been in place and essentially unchanged for many years. We thus expect that the spatial pattern of exposure to traffic emissions from home to home was relatively similar over the lifetimes of these children. Bias could also have occurred if the families of asthmatic children had preferentially moved to a home near a freeway, but this seems unlikely. Additionally, our observed associations were robust to adjustment for factors known to be related to population mobility, housing location, and access to care, including race/ethnicity and indicators of socioeconomic status (as well as household characteristics). This robustness further suggests that our results were not the result of these potential confounders.

These results have both scientific and public health implications. They strengthen an emerging body of evidence that air pollution can cause asthma and that traffic-related pollutants that vary within communities are partly responsible for this association. The current regulatory approach that focuses almost exclusively on regional pollutants merits reevaluation in light of this emerging evidence and in light of the enormous costs associated with childhood asthma.[40] In addition, because NO_2 may be a surrogate for the pollutant or pollutants responsible for the observed effects, further study is indicated to identify the specific pollutant(s). In this regard, improved physical and chemical characterization of ambient ultrafine particles (including particle number concentration distributions, as well as more traditional chemical analyses) are topics of specific ongoing research interest in southern California and elsewhere.

ACKNOWLEDGMENTS

We are very grateful for input from the external advisory committee to this study, including David Bates, Morton Lippmann, Jonathan Samet, Frank Speizer, John Spengler, and Scott Zeger. We thank Tami Funk of Sonoma Technology for contributing to the assessment of residential distance to freeways, and Robert Weker of the Harvard School of Public Health for preparing and analyzing the Palmes tubes.

REFERENCES

1. Nicolai T. Air pollution and respiratory disease in children: what is the clinically relevant impact? *Pediatr Pulmonol Suppl*. 1999;18:9–13.
2. Clark NM, Brown RW, Parker E, et al. Childhood asthma. *Environ Health Perspect*. 1999;107(suppl 3):421–429.
3. Venn A, Lewis S, Cooper M, et al. Local road traffic activity and the prevalence, severity, and persistence of wheeze in school children: combined cross sectional and longitudinal study. *Occup Environ Med*. 2000;57:152–158.
4. Edwards J, Walters S, Griffiths RK. Hospital admissions for asthma in preschool children: relationship to major roads in Birmingham, United Kingdom. *Arch Environ Health*. 1994;49:223–227.
5. Hirsch T, Weiland SK, von Mutius E, et al. Inner city air pollution and respiratory health and atopy in children. *Eur Respir J*. 1999;14:669–677.
6. van Vliet P, Knape M, de Hartog J, et al. Motor vehicle exhaust and chronic respiratory symptoms in children living near freeways. *Environ Res*. 1997;74:122–132.
7. Nicolai T, Carr D, Weiland SK, et al. Urban traffic and pollutant exposure related to respiratory outcomes and atopy in a large sample of children. *Eur Respir J*. 2003;21:956–963.
8. Wjst M, Reitmeir P, Dold S, et al. Road traffic and adverse effects on respiratory health in children. *BMJ*. 1993;307:596–600.
9. English P, Neutra R, Scalf R, et al. Examining associations between childhood asthma and traffic flow using a geographic information

system. *Environ Health Perspect*. 1999;107:761–767.
10. Waldron G, Pottle B, Dod J. Asthma and the motorways—one district's experience. *J Public Health Med*. 1995;17:85–89.
11. Kramer U, Koch T, Ranft U, et al. Traffic-related air pollution is associated with atopy in children living in urban areas. *Epidemiology*. 2000;11:64–70.
12. Peters JM, Avol E, Gauderman WJ, et al. A study of twelve Southern California communities with differing levels and types of air pollution. II. Effects on pulmonary function. *Am J Respir Crit Care Med*. 1999; 159:768–775.
13. Gauderman WJ, McConnell R, Gilliland F, et al. Association between air pollution and lung function growth in southern California children. *Am J Respir Crit Care Med*. 2000;162:1383–1390.
14. Avol E, Gauderman W, Tan S, et al. Respiratory effects of relocating to areas of differing air pollution levels. *Am J Respir Crit Care Med*. 2001;164:2067–2072.
15. Gauderman WJ, Avol E, Gilliland F, et al. The effect of air pollution on lung function development in children aged 10 to 18 years. *New Engl J Med*. 2004;351:1057–1067.
16. McConnell R, Berhane K, Gilliland F, et al. Air pollution and bronchitic symptoms in southern California children with asthma. *Environ Health Perspect*. 1999;107:757–760.
17. McConnell R, Berhane K, Gilliland F, et al. Prospective study of air pollution and bronchitic symptoms in children with asthma. *Am J Respir Crit Care Med*. 2003;168:790–797.
18. McConnell R, Berhane K, Gilliland F, et al. Asthma in exercising children exposed to ozone: a cohort study. *Lancet*. 2002;359:386–391.
19. Palmes ED, Gunnison AF, DiMattio J, et al. Personal sampler for NO_2. *Journal of the American Industrial Hygiene Association*. 1976;37:570–577.
20. Linaker CH, Coggon D, Holgate ST, et al. Personal exposure to nitrogen dioxide and risk of airflow obstruction in asthmatic children with upper respiratory infection. *Thorax*. 2000;55:930–933.
21. Alm S, Mukala K, Pasanen P, et al. Personal NO2 exposures of preschool children in Helsinki. *J Expo Anal Environ Epidemiol*. 1998; 8:79–100.
22. Samet J, Lambert W, Skipper B, et al. Nitrogen dioxide and respiratory illnesses in infants. *Am Rev Respir Dis*. 1993;148:1258–1265.
23. Wu J, Lurmann F, Winer A, et al. Development of an individual exposure model for application to the Southern California children's health study. *Atmos Environ*. 2005;39:259–273.
24. Bensen P. *CALINE4—A Dispersion Model for Predicting Air Pollution Concentrations Near Roadways*. Sacramento: California Department of Transportation; 1989.
25. Trainer M, Parrish D, Goldan P, et al. Review of observation-based analysis of the regional factors influencing ozone concentrations. *Atmos Environ*. 2000;34:2045–2061.
26. Brauer M, Hoek G, Van Vliet P, et al. Air pollution from traffic and the development of respiratory infections and asthmatic and allergic symptoms in children. *Am J Respir Crit Care Med*. 2002;166:1092–1098.
27. Venn AJ, Lewis SA, Cooper M, et al. Living near a main road and the risk of wheezing illness in children. *Am J Respir Crit Care Med*. 2001;164:2177–2180.
28. Zmirou D, Gauvin S, Pin I, et al. Traffic related air pollution and incidence of childhood asthma: results of the Vesta case–control study. *J Epidemiol Community Health*. 2004;58:18–23.
29. Kim JJ, Smorodinsky S, Lipsett M, et al. Traffic-related air pollution near busy roads: the East Bay Children's Respiratory Health Study. *Am J Respir Crit Care Med*. 2004;170:520–526.
30. Health effects of outdoor air pollution. Part 2. Committee of the Environmental and Occupational Health Assembly of the American Thoracic Society. *Am J Respir Crit Care Med*. 1996;153:477–498.
31. Bates DV. Observations on asthma. *Environ Health Perspect*. 1995; 103(suppl 6):243–247.
32. Brauer M, Hoek G, van Vliet P, et al. Estimating long-term average particulate air pollution concentrations: application of traffic indicators and geographic information systems. *Epidemiology*. 2003;14:228–239.
33. Fischer PH, Hoek G, Van Reeuwijka H, et al. Traffic-related differences in outdoor and indoor concentrations of particles and volatile organic compounds in Amsterdam. *Atmos Environ*. 2000;34:3713–3722.
34. Roorda-Knape MC, Janssen NAH, de Hartog JJ, et al. Air pollution from traffic in city districts near major motorways. *Atmos Environ*. 1998;32: 1921–1930.
35. Seaton A, Dennekamp M. Hypothesis: ill health associated with low concentrations of nitrogen dioxide—an effect of ultrafine particles? *Thorax*. 2003;58:1012–1015.
36. Li N, Kim S, Wang M, et al. Use of a stratified oxidative stress model to study the biological effects of ambient concentrated and diesel exhaust particulate matter. *Inhal Toxicol*. 2002;14:459–486.
37. Samet JM. Epidemiologic approaches for the identification of asthma. *Chest*. 1987;91:74S–78S.
38. Ehrlich RI, Du Toit D, Jordaan E, et al. Prevalence and reliability of asthma symptoms in primary school children in Cape Town. *Int J Epidemiol*. 1995;24:1138–1145.
39. Burr ML. Diagnosing asthma by questionnaire in epidemiological surveys [Editorial]. *Clin Exp Allergy*. 1992;22:509–510.
40. Smith DH, Malone DC, Lawson KA, et al. A national estimate of the economic costs of asthma. *Am J Respir Crit Care Med*. 1997;156:787–793.