Wig Zamore
13 Highland Ave. #3
Somerville MA 02143

617-625-5630
wigzamore@rcn.com

April 26, 2006

Jamy Buchanan Madeja, Esq.,
Buchanan & Associates
31 Mount Vernon Street, Boston, MA 02108

Memo on Transportation, Air Quality and the Lovejoy Wharf Project
RE: Draft Environmental Impact Report / Final Project Impact Report
FOR: Strada234 Residents Group

Dear Ms. Madeja:

The purpose of this memo is to frame some of the primary transportation, air quality and public health issues that require far greater scrutiny in connection with the Lovejoy Wharf project. I am most concerned that the project environmental documents – the DEIR, FPIR and Chapter 91 filing - do not adequately address the large air pollution and health risks attendant to the shared location of Strada234 and Lovejoy Wharf in the midst of extraordinarily intense regional transportation facilities and proportionate air emissions.

As you know I have devoted a considerable amount of time the last several years to understanding the dynamics of regional transportation pollutants and their effects on local health. As a result, I have addressed the Boston Metropolitan Planning Organization's Regional Transportation Advisory Committee, the core graduate course in Occupational and Environmental Health at Tufts NEMC, the Massachusetts Ozone SIP Steering Committee and many other groups. On February 3 of this year I was one of eight people in the US given a few moments to address EPA's Clean Air Scientific Advisory Committee (CASAC) during their formal deliberative session on the national standards for particulate matter (PM). In their official role as advisors on the Clean Air Act, CASAC has subsequently written EPA Administrator Johnson recommending tighter national fine particulate matter standards, based on current science. Although not the focal point of this EPA PM criteria review, an earlier advisory letter from CASAC to Administrator Johnson had specifically noted the need for greater national attention to near highway health effects.

From my point of view the fundamental issue at Lovejoy Wharf is quite simple. The environment surrounding this project is severely stressed - to a degree that few members of government or the general public yet realize. Any incremental environmental burdens from the proposed project - and any failure to manage cumulative and interactive effects - will add to the burden borne by nearby residents. Therefore the project details - including vehicle idling at the entry to the mechanical garage, cold temperature "cold starts" after retrieval of vehicles from the garage, unnecessary ground floor retail reliance on private vehicle trips, the impacts of the project design in retarding dispersion of pollutants, et cetera – can only be responsibly considered within the context of a comprehensive understanding of the public health issues that envelope the project site. That baseline contextual analysis is entirely absent from the project filings. Cumulative effects are not accurately or responsibly portrayed.

Without a fundamental understanding of the serious impacts of the surrounding highways and diesel commuter rail, at the very least, it is not possible to judge whether the Lovejoy Wharf project is exacerbating, mitigating or even ignoring the risks affecting the current residents of Strada234 and the future residents of Lovejoy Wharf itself. These neighboring residential properties – one existing and one

proposed - lie directly between Interstate 93, with roughly 160,000 vehicles per day, and North Washington Street / Rutherford Avenue, with roughly 40,000 vehicle trips per day.

The Leverett Connector, the ramp to Storrow Drive, Causeway Street and other heavily traveled local streets also lie nearby. The highway portals in close proximity to these properties are an additional factor as they may serve to concentrate vehicular air pollutants. This is true whether or not the transverse jet turbines in the tunnel sections - substituted for dispersion cost effectiveness reasons midway through the Big Dig - are functioning or not.

Strada234 and Lovejoy Wharf lie within 100 meters of over 200,000 vehicles per day. They also lie within 200 to 300 meters of the nearly 200 diesel trains per day and the 50,000 or more diesel trains per year which enter or exit North Station.

In the last several years a large body of public health research has accumulated - much of it funded by US EPA and/or NIH – which raises the level of concern regarding residential sites located next to highways and heavy rail. Based on the best research yet published in North America and Western Europe, one might expect the following health outcomes for long term residents of these neighboring properties:

1. A 20% increase in all cause mortality as compared to a clean background location not affected by intense regional transportation emissions.

2. A 50% increase in acute myocardial infarction (heart attack) and lung cancer mortalities as compared to a clean background site.

The weight of evidence regarding the association of mobile pollutants with all cause, heart attack and lung cancer mortality is very large as a result of US cohort studies published since 1990 which have focused upon regional and city differences in long term air pollution levels, especially fine particulate matter and associated gases resulting from combustion processes. As epidemiologists have investigated mortality gradients closer and closer to primary air pollution sources they have tied their studies to markers of fresh pollutants – such as nitrogen oxides - and then simply to proximity to high volume sources. Unlike the large cohort studies, the best near highway mortality studies have been done in Canada and Europe but are considered to be generalizable to the United States.

Other health outcomes reported by reputable researchers for those who live within 100 meters of highways - neither yet widely replicated nor contradicted by other studies - are listed below:

3. Up to a 50% increase in Chronic Obstructive Pulmonary Disease (COPD) among adult women living near highways.

4. Up to a 100% increase in childhood asthma incidence for children who grow up in near highway locations, as well as more frequent exacerbation.

5, Up to a 100% increase in childhood cancers – by birth address – for children born and spending their first years of life next to intense transportation emissions sources.

6. Up to a 400% increase in children who never develop 80% of normal adult lung capacity, assuming they reside near intense regional transportation emissions from age 10 through age 18.

7. Increases in adverse birth outcomes - including low weight and premature delivery – have also been associated with various air pollutants.

(I have appended to the end of this memo a representative list of relevent air pollution and public health studies, including the abstracts for many of them and some specific comments. I am also supplying most of these that are available electronically, for the public record.)

The primary failure of the Lovejoy Wharf filings, in my opinion, is the failure to account for the extraordinary health risks associated with the impacts of nearby regional transportation emissions and the cumulative (and interactive) risks added by each project's incremental environmental burdens. The emissions of most concern are fresh ultrafine particles (below .1 micron in diameter), fresh fine particles (below 2.5 microns in diameter), and associated semi-volatile organic compounds (SVOCs), including air toxics. These pollutants have very steep concentration gradients in near source locations and the associated health effects are proportionally steep.

Recent studies of regional transportation corridors and nodes have added enormously to our understanding of near source particle evolution, particle counts, particle surface area, particle composition and penetration rates for nearby buildings. In brief, an overwhelming majority of ultrafine and fresh fine particulates from transportation sources are nucleated after supersaturated gases leave the exhaust streams of both gasoline (spark ignited) and diesel (compression ignited) vehicles. Within a few tens of meters of highways there can be between 50,000 and 200,000 ultrafines particles per cubic centimeter. These fresh ultrafine particles peak in total mass within about 100 meters downwind from transportation corridors and can significantly exceed urban background levels for up to 300 meters.

The fresh ultrafine particulates contain a very high level of volatile and semi-volatile organic compounds rich in toxic materials. They also have significant penetration rates into nearby buildings. Because the smaller particulates have much higher surface area as well as an diverse variety of harmful compounds, the dose and intake fraction affecting nearby residents is much larger than for other metropolitan area citizens. The chronic health effects suffered are proportional to the size of the emissions sources (as in vehicles or trains per day), proximity to those sources and the cumulative amount of time exposed. The juxtaposition of intense regional transportation corridors and nodes with very nearby residences creates the most serious environmental health problem currently faced in urban areas of the US.

More accurate contour gradients for carbon monoxide (CO), nitrogen oxides (NOx) and volatile organic compounds (VOCs) might help also. The fresh particulates and associated gases of greatest concern tend to have concentration gradients which are similar to those recorded for some of the overlapping pollutant classes. (A small detail that needs reconciling is the assumed atmospheric boundary layer for the emissions modeling. The wind studies in the project filings assume a boundary layer of 400 meters while the emissions models assume a boundary layer of 1000 meters. Depending on the other model characteristics, which are not detailed in the filings, a 150% expansion in the assumed boundary layer could result in a 60% understatement of calculated pollutant concentrations.) More fundamentally, the CO calculations and other limited pollutant discussions contained within the Lovejoy Wharf project documents, which have no connection to actual regional transportation levels in the vicinity of the project, are grossly inadequate.

The bureaucratic processes that typically frame development project impact documents in Massachusetts have unfortunately lagged the rapidly evolving real world understanding of these most serious environmental and health issues. In order for the Lovejoy Wharf project to fully disclose the environmental and health issues associated with the location of the project and then the project's impacts upon its neighbors, it is absolutely necessary that the project proponents first portray all the roadway vehicular volumes and diesel train volumes within 1000 meters of the project. Peak and average emissions levels for ALL pollutants of concern from these transportation sources need to be detailed and then run through local dispersion models. Finally, population exposure levels over the short and long term must be calculated, as well as portrayed much more clearly. This is the only reasonable context imaginable to me for a frank discussion of the health risks associated with the Lovejoy Wharf project.

A small measure of the serious nature of these nearby transportation emissions can be gained from several official documents recently issued by the California Air Resources Board (CARB) – their April 2005 **Air Quality and Land Use Handbook** and their October 2004 **Roseville Rail Yard Study**. Both of these documents use an older air toxics risk analysis framework which does not yet incorporate the more recent

near source health studies associated with fresh fine particulate matter, transportation emissions markers and proximity to regional transportation corridors and nodes. They therefore severely understate the extent of lung cancer and ischemic heart disease (including acute myocardial infarctions) associated with regional transportation emissions.

Nevertheless, the **CARB Land Use Manual** suggests that all California Planning Boards think twice before siting sensitive land uses – schools, hospitals, residences, active playgrounds, et cetera – within 500 feet of highways with over 100,000 vehicles per day. They note (Table 1.2 and subsequent discussion) that the relative health risk associated with highways far exceeds that associated with other common California air emissions sources. It is illegal in California to site a school within 500 feet of a highway. The **CARB Land Use Manual** and the **Roseville Rail Yard Study** also suggest that care should be taken in land use siting decisions within 1000 feet of large diesel rail facilities, and that commuter rail emissions typically exceed freight rail emissions. The Roseville Rail Yard is California's largest facility. It handles, and/or immediately abuts, roughly half the annual diesel rail volume of North Station.

Comment letters that I have submitted to EPA during the current criteria review of national ambient air quality standards (NAAQS) for particulate matter and the written notes associated with my recent comments to the Clean Air Scientific Advisory Committee to EPA are available from official Federal document sites. These public EPA particulate matter comments are repetitive, given the subject, and focus more specifically on the highway and diesel train emissions faced by the community of Somerville. They are, however, relevent to the Strada234 and Lovejoy Wharf project locations because Somerville and these North Station sites involve very similar highway and diesel train volumes – indeed, in many cases they are exposed to the very same vehicles.

Among the studies I am forwarding, the near roadway and city gradient studies that address lung cancer and heart attack mortality are most important. The near roadway morbidity studies, especially those from southern California that address pulmonary effects, are also especially relevent.

Regards,

Wig Zamore

Note: EPA's current Mobile Source Air Toxics (MSAT) rule documents (at regulations.gov) include a discussion of some serious flaws in current EPA recommended models. (This has been known for several months now.) More specifically, EPA has discovered through field testing that actual new advanced technology vehicle emissions in "cold temperature" cold start situations are several 100% higher than assumed. EPA had assumed the same "cold temperature" cold start emissions as at 70 degrees F. EPA will provide corrective technical guidance to the states and MPOs soon but the MSAT rule documents do seem to contain enough guidance to run more accurate emissions models in the meantime.

References for regional transportation, air pollution and public health. (Comments in blue italic.)

****

1. **Air Pollution and Noise from Interstate Route 93, Report No. 21195a,** April 1971, Prepared for Commonwealth of Massachusetts Department of Public Works, by Bolt Beranak and Newman Inc. with assistance from Environmental Research and Technology Inc.

   This study – which I could copy - detailed the large increases in Particulate Matter, Carbon Monoxide, Lead and Noise that would result in Somerville from the completion of I93 in the early 1970s. It is relevent to the North Station area as well but only as historic and background material. The Clean Air Act of 1970 and its predecessor in 1967 clearly stated that serious human health impacts could be expected from both the pre-existing levels of pollution in Somerville along Routes 28 and 38, and the increases from completing I93. Massachusetts rationalized that since EPA's regulations were expected to eliminate 90% of mobile pollution within 5 years, it did not matter. But EPA failed in that ambition. This study is not available from the State Transportation Library or the Office of Transportation but is in BBN's library.

2. **I-93 Somerville Corridor Study Recommendations,** December 1972, Prepared for Department of Public Works, Commonwealth of Massachusetts by Justin Gray Associates.

   This I-93 follow-up study was in the State Transportation Library at 10 Park Plaza. Justin Gray was a small but highly reputable planning policy group associated with Harvard. The study admits the environmental issues associated with the I93 completion and attempts to lay out mitigations options, most of which were not followed.

   Two other contemporaneous studies of note are:

3. **Air pollution and human health. Lave LB and Seskin EP**, Science. 1970 Aug 21;169(947):723-33. PMID: 5432570 [PubMed - indexed for MEDLINE]

   Seminal article that established accurate association of sulfates and particulates with mortality, based largely on research review (including English studies of rural versus urban mortalities). They followed with a full text of same name in 1977 and a subsequent article in 1979. There were some early predecessors to Lave and Seskin, including two AMA pieces on carcinogens in diesel and gasoline and an illuminating US Surgeon General's report on mobile pollution in 1962. Lave and Seskin calculated that mobile damages were very large but worried that fixing the problem might be too costly. They were close in their mortality calculations.

4. **Air Quality and Automobile Emission Control**, 4 Volumes, A Report by the Coordinating Committee on Air Quality Studies, Prepared for the Committee on Public Works, United States Senate, US Government Printing Office 1974

   Both Harvard and MIT have this. Important sections show knowledge that primary emissions affect local health strongly (urban gradients, not near roadway) and are more damaging than secondary emissions which primarily affect regional health. This study concluded that concentration of population in the urban core would decrease regional vehicle miles traveled on highways but would also seriously increase pollution exposures and therefore regional health impacts. Using TASSIM modeling of transportation and land use options in Boston and Los Angeles to get a handle on benefits and costs, this study nevertheless concluded that since EPA was going to eliminate 90% of mobile air pollution by 1976, many tactics were unnecessary. But EPA fell far short of its initial Clean Air Act of 1970 goals.

Lung cancer and heart attacks are the two leading causes of death in the US, each accounting for about 7.5% of annual mortality. They are also the two fatal diseases most highly associated with air pollution. For example, from 1989 through 2003 Somerville had 291 more lung cancer and heart attack deaths than the community would have had if it suffered these diseases at the same age-adjusted rate as the state of Massachusetts as a whole. It thus seems likely that Somerville has had 500 or so excess lung cancer and heart attack deaths since the completion of I93. (The arterial highways, Mystic Avenue and McGrath Highway, and the diesel commuter rail should share some of the blame for Somerville's transportation related health disparities.) US EPA, US OMB and California's Air Resources Board suggest that each premature death from environmental causes equals $6 to $8 million in damages, using a calculation of the "value of a statistical life".

5. **An association between air pollution and mortality in six U.S. cities.** Dockery DW, Pope CA 3rd, Xu X, Spengler JD, Ware JH, Fay ME, Ferris BG Jr, Speizer FE. N Engl J Med. 1993 Dec 9;329(24):1753-9. Environmental Epidemiology Program, Harvard School of Public Health, Boston, MA 02115.

This is the chronic study that caused everyone including EPA to sit up, take notice and (after the Pope 1995 ACS cohort study) add the new national fine PM standards in 1997. I have left off of this list the single city acute studies, many of which were done by Joel D. Schwartz. They preceded the chronic studies and contributed to the PM standards change. Eventually the single city acute studies were supplanted by compilations across many cities. The chronic studies are associated with higher levels of mortality than the acute and there is some overlap in mortality numbers. It is harder to deal with daily variations in fine PM levels than annual averages, especially when explaining the analytic techniques to policy makers and the general public, even though the physical association of episodic air pollution and immediate health impacts may appear more causal. Note that this Harvard Six Cities study looked at individual cities with centrally placed monitors and a variety of fine PM sources – all combustion based but including some industry, power and mobile. Because the geographic scale associated with each set of monitors is smaller than in the Pope ACS studies (see below), the correlation between the monitors and cohort health impacts is tighter. The reported mortality per 10 μg/m3 of fine PM (micrograms PM per cubic meter of air) is thus considerably higher. However, this Harvard study done from scratch does not have the same cohort size as Pope and loses some statistical power thereby. Nevertheless it is quite a piece of work.

BACKGROUND. Recent studies have reported associations between particulate air pollution and daily mortality rates. Population-based, cross-sectional studies of metropolitan areas in the United States have also found associations between particulate air pollution and annual mortality rates, but these studies have been criticized, in part because they did not directly control for cigarette smoking and other health risks. METHODS. In this prospective cohort study, we estimated the effects of air pollution on mortality, while controlling for individual risk factors. Survival analysis, including Cox proportional-hazards regression modeling, was conducted with data from a 14-to-16-year mortality follow-up of 8111 adults in six U.S. cities. RESULTS. Mortality rates were most strongly associated with cigarette smoking. After adjusting for smoking and other risk factors, we observed statistically significant and robust associations between air pollution and mortality. The adjusted mortality-rate ratio for the most polluted of the cities as compared with the least polluted was 1.26 (95 percent confidence interval, 1.08 to 1.47). Air pollution was positively associated with death from lung cancer and cardiopulmonary disease but not with death from other causes considered together. Mortality was most strongly associated with air pollution with fine particulates, including sulfates. CONCLUSIONS. Although the effects of other, unmeasured risk factors cannot be excluded with certainty, these results suggest that fine-particulate air pollution, or a more complex pollution mixture associated with fine particulate matter, contributes to excess mortality in certain U.S. cities.

PMID: 8179653 [PubMed - indexed for MEDLINE]

6. **Particulate air pollution as a predictor of mortality in a prospective study of U.S. adults.** Pope CA 3rd, Thun MJ, Namboodiri MM, Dockery DW, Evans JS, Speizer FE, Heath CW Jr. Am J Respir Crit Care Med. 1995 Mar;151(3 Pt 1):669-74. Department of Environmental Health, Harvard School of Public Health, Boston, Massachusetts.

The second blockbuster study of chronic PM exposure, this is based on the huge American Cancer Society cohort – 1.2 million people across the US, roughly half of whom had not moved when the initial study was done. This study used government (regional) monitor records and looked for the association of their levels with the health of metropolitan area populations that were spread over much larger areas than in the Harvard study. The cohort size gave statistical power but the less direct correspondence of central monitor readings and cohort experience led to lower mortality calculations per 10 μg/m3 of fine PM. Also, note that the lung cancer association is stronger in the 2002 follow-up, perhaps simply because there are more years of study in which the lung cancers could develop. This Pope ACS study pinned down the high number of cardiovascular deaths associated with fine PM. Another surprize in the Pope studies has been the additive effects of smoking and air pollution. There had been some expectation that smoking would obscure any ambient air impacts. Basically, the ambient PM impacts reported by Harvard and Pope were at an order of magnitude larger than expected.

Time-series, cross-sectional, and prospective cohort studies have observed associations between mortality and particulate air pollution but have been limited by ecologic design or small number of subjects or study areas. The present study evaluates effects of particulate air pollution on mortality using data from a large cohort drawn from many study areas. We linked ambient air pollution data from 151 U.S. metropolitan areas in 1980 with individual risk factor on 552,138 adults who resided in these areas when enrolled in a prospective study in 1982. Deaths were ascertained through December, 1989. Exposure to sulfate and fine particulate air pollution, which is primarily from fossil fuel combustion, was estimated from national data bases. The relationships of air pollution to all-cause, lung cancer, and cardiopulmonary mortality was examined using multivariate analysis which controlled for smoking, education, and other risk factors. Although small compared with cigarette smoking, an association between mortality and particulate air pollution was observed. Adjusted relative risk ratios (and 95% confidence intervals) of all-cause mortality for the most polluted areas compared with the least polluted equaled 1.15 (1.09 to 1.22) and 1.17 (1.09 to 1.26) when using sulfate and fine particulate measures respectively. Particulate air pollution was associated with cardiopulmonary and lung cancer mortality but not with mortality due to other causes. Increased mortality is associated with sulfate and fine particulate air pollution at levels commonly found in U.S. cities. The increase in risk is not attributable to tobacco smoking, although other unmeasured correlates of pollution cannot be excluded with certainty.

PMID: 7881654 [PubMed - indexed for MEDLINE]

7. **Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality.** Daniel Krewski, Richard T Burnett, Mark S Goldberg , Kristin Hoover, Jack Siemiatycki, Michael Jerrett, Michal Abrahamowicz, and Warren H White. Health Effects Institute 2000. Cambridge, Massachusetts.

The Harvard and ACS cohort studies were so important that complete independent reanalyses were desirable. Accordingly, EPA asked Health Effects Institute to assemble a team to carry it out. Daniel Krewski, who had overeseen a national risk analysis institute in Canada, was asked to lead the effort. The HEI studies, using more elaborate statistical approaches, concluded that the two seminal studies were accuarate in all substantive respects. Note that all three of these studies, like Lave and Seskin, considered sulfate and particulates, as well as a long list of potential confounders.

8. Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. Pope CA 3rd, Burnett RT, Thun MJ, Calle EE, Krewski D, Ito K, Thurston GD. JAMA. 2002 Mar 6;287(9):1132-41. Department of Economics, Brigham Young University, 142 FOB, Provo, UT 84602, USA. cap3@email.byu.edu.

This is the reigning and most influential "between region" PM cohort study. It is important in the current fine PM standards review. All mortality associations with PM are stronger in this follow-up study than in the original Pope ACS, especially the lung cancer. The data reported in the study are stronger than the abstract suggests.

CONTEXT: Associations have been found between day-to-day particulate air pollution and increased risk of various adverse health outcomes, including cardiopulmonary mortality. However, studies of health effects of long-term particulate air pollution have been less conclusive. OBJECTIVE: To assess the relationship between long-term exposure to fine particulate air pollution and all-cause, lung cancer, and cardiopulmonary mortality. DESIGN, SETTING, AND PARTICIPANTS: Vital status and cause of death data were collected by the American Cancer Society as part of the Cancer Prevention II study, an ongoing prospective mortality study, which enrolled approximately 1.2 million adults in 1982. Participants completed a questionnaire detailing individual risk factor data (age, sex, race, weight, height, smoking history, education, marital status, diet, alcohol consumption, and occupational exposures). The risk factor data for approximately 500 000 adults were linked with air pollution data for metropolitan areas throughout the United States and combined with vital status and cause of death data through December 31, 1998. MAIN OUTCOME MEASURE: All-cause, lung cancer, and cardiopulmonary mortality. RESULTS: Fine particulate and sulfur oxide--related pollution were associated with all-cause, lung cancer, and cardiopulmonary mortality. Each 10-microg/m(3) elevation in fine particulate air pollution was associated with approximately a 4%, 6%, and 8% increased risk of all-cause, cardiopulmonary, and lung cancer mortality, respectively. Measures of coarse particle fraction and total suspended particles were not consistently associated with mortality. CONCLUSION: Long-term exposure to combustion-related fine particulate air pollution is an important environmental risk factor for cardiopulmonary and lung cancer mortality.

PMID: 11879110 [PubMed - indexed for MEDLINE]

9. Reduction in Fine Particulate Air Pollution and Mortality: Extended follow-up of the Harvard Six Cities Study. Laden F, Schwartz J, Speizer FE, Dockery DW. Am J Respir Crit Care Med. 2006 Jan 19; [Epub ahead of print] Exposure, Epidemiology and Risk Program, Department of Environmental Health, Harvard School of Public Health, Boston, MA, USA; Channing Laboratory, Department of Medicine, Brigham and Women's Hospital and Harvard Medical School, Boston, MA, USA.

This follow-up study relies on public air pollution monitors as the original Harvard Six Cities installed monitors were abandoned before the study conclusion. The additional periods, as with Pope, seem to add accuracy and this study (like some from Europe) encouragingly shows that urban mortality goes down as the air gets cleaner. (However, some California researchers are finding a disturbing increase in ultrafine particulate concentrations near highways even though California has the most aggressive clean fuel and vehicle regulations in the US.)

Rationale: A large body of epidemiologic literature has found an association of increased fine particulate air pollution (PM2.5) with acute and chronic mortality. The effect of improvements in particle exposure is less clear. Objectives: Earlier analysis of the Harvard Six Cities adult cohort study showed an association between long-term ambient PM2.5 and mortality between enrollment in the mid-1970's and follow-up until 1990. We extended mortality follow-up for eight years in a period of reduced air pollution concentrations. Methods: Annual city-specific PM2.5 concentrations were measured between 1979-1988, and estimated for later years from publicly available data.

Exposure was defined as (1) city-specific mean PM2.5 during the two follow-up periods, (2) mean PM2.5 in the first period and change between these periods, (3) overall mean PM2.5 across the entire follow-up, and (4) year-specific mean PM2.5. Mortality rate ratios were estimated with Cox proportional hazards regression controlling for individual risk factors. Measurements and Main Results: We found an increase in overall mortality associated with each 10 microg/m(3) increase in PM2.5 modeled either as the overall mean (RR=1.16, 95%CI=1.07-1.26) or as exposure in the year of death (RR=1.14, 95%CI=1.06-1.22). PM2.5 exposure was associated with lung cancer (RR=1.27, 95%CI=0.96-1.69) and cardiovascular deaths (RR=1.28, 95%CI=1.13-1.44). Improved overall mortality was associated with decreased mean PM2.5 (10 microg/m(3)) between periods (RR=0.73, 95% CI=0.57-0.95). Conclusion: Total, cardiovascular, and lung cancer mortality were each positively associated with ambient PM2.5 concentrations. Reduced PM2.5 concentrations were associated with reduced mortality risk.

PMID: 16424447 [PubMed - as supplied by publisher]

10. **Daily Deaths Associated with Air Pollution in Six US Cities and Short-Term Mortality Displacement in Boston,** Joel Schwartz, pages 219 to 226 in **Revised Analysis of Time-Series Studies of Air Pollution and Health,** Zanobetti, Schwartz et alia, Health Effects Institute, Boston 2003. **Airborne Particles and Daily Deaths in 10 US Cities**, Joel Schwartz, pages 211 to 218, same. Many other similar studies in this single HEI Report.

These studies by Joel Schwartz within the HEI report are referenced by EPA's current PM criteria review health effects study by Abt Associates. In the Abt work, the official EPA estimate of Boston's annual premature mortality from chronic PM (594 deaths per year in Suffolk, Middlesex and Norfolk counties if a cut-off of 7.5 μg/m3 is used with an annual average level of 12.1 μg/m3 assumed) comes from Pope ACS Extended 2002 and the official estimate of Boston's annual premature mortality from acute PM (390 deaths per year if a cut-off of 3.5 μg/m3 is used) comes from the top Schwartz article listed above. Note that Boston's "design value" for annual average fine PM is 14.4 μg/m3. The whole HEI report is Volume II of the National Morbidity, Mortality and Air Pollution Study. Volume I came out in 2000, led by Jon Samet at Johns Hopkins. There were very high hopes for NMMAPS and it remains the premier multi-city acute PM study. But it was hurt by the need for reanalyses after it became apparent that the S-Plus software employed in the study had some issues handling concurvity within a GAM model (general additive regression analysis looks at one factor at a time, but many in succession). Most of the related studies are available from HEI's web-site. The acute PM study statistics are challenging.

11. **Lung cancer and air pollution: a 27 year follow up of 16 209 Norwegian men.** Nafstad P, Haheim LL, Oftedal B, Gram F, Holme I, Hjermann I, Leren P. Thorax. Division of Epidemiology, Norwegian Institute of Public Health, Oslo, Norway. per.nafstad@fhi.no 2003 Dec;58(12):1071-6.

This is the best European general traffic and lung cancer report. Note that this paper uses NOx concentration calculations rather than PM monitors. It also found much less association with SO2 (building heating systems origin) than NOx (traffic origin). Another European study further below (Nyberg) looks at local gradients of traffic pollution and lung cancer in Stockholm with higher calculated association as the scale of investigation is smaller.

BACKGROUND: The well documented urban/rural difference in lung cancer incidence and the detection of known carcinogens in the atmosphere have produced the hypothesis that long term air pollution may have an effect on lung cancer. The association between incidence of lung cancer and long term air pollution exposure was investigated in a cohort of Oslo men followed from 1972/73 to 1998. METHODS: Data from a follow up study on cardiovascular risk factors among 16 209 40 to 49 year old Oslo men in 1972/73 were linked to data from the Norwegian cancer register, the Norwegian death register, and estimates of average yearly air pollution levels at the

participants' home address in 1974 to 1998. Survival analyses, including Cox proportional hazards regression, were used to estimate associations between exposure and the incidence of lung cancer. RESULTS: During the follow up period, 418 men developed lung cancer. Controlling for age, smoking habits, and length of education, the adjusted risk ratio for developing lung cancer was 1.08 (95% confidence interval, 1.02 to 1.15) for a 10 micro g/m(3) increase in average home address nitrogen oxide (NO(x)) exposure between 1974 and 1978. Corresponding figures for a 10 micro g/m(3) increase in sulphur dioxide (SO(2)) were 1.01 (0.94 to 1.08). CONCLUSIONS: Urban air pollution may increase the risk of developing lung cancer.

PMID: 14645978 [PubMed - indexed for MEDLINE]

12. **Urban air pollution and lung cancer in Stockholm.** Nyberg F, Gustavsson P, Jarup L, Bellander T, Berglind N, Jakobsson R, Pershagen G. Epidemiology. 2000 Sep;11(5):487-95. Division of Environmental Epidemiology, Institute of Environmental Medicine, Karolinska Institute, Stockholm, Sweden.

Although the number of cases is fairly small, this paper is the best of its kind ever done and suggests that there is a very large increase in lung cancer, over 20 years, with statistical significance for the most traffic polluted areas of Stockholm (1.44 to 1.60 relative risk). The study suggests that never smokers and light smokers - up to a pack a day - both experience about a 50% increase in lung cancer mortality. The study uses NO2 as a marker for mobile pollution intensity.

We conducted a population-based case-control study among men 40-75 years of age encompassing all cases of lung cancer 1985-1990 among stable residents of Stockholm County 1950-1990. Questionnaires to subjects or next-of-kin (primarily wives or children) elicited information regarding smoking and other risk factors, including occupational and residential histories. A high response rate (>85%) resulted in 1,042 cases and 2,364 controls. We created retrospective emission databases for NOx/NO2 and SO2 as indicators of air pollution from road traffic and heating, respectively. We estimated local annual source-specific air pollution levels using validated dispersion models and we linked these levels to residential addresses using Geographical Information System (GIS) techniques. Average traffic-related NO2 exposure over 30 years was associated with a relative risk (RR) of 1.2 (95% confidence interval 0.8-1.6) for the top decile of exposure, adjusted for tobacco smoking, socioeconomic status, residential radon, and occupational exposures. The data suggested a considerable latency period; the RR for the top decile of average traffic-related NO2 exposure 20 years previously was 1.4 (1.0-2.0). Little association was observed for SO2. Occupational exposure to asbestos, diesel exhaust, and other combustion products also increased the risk of lung cancer. Our results indicate that urban air pollution increases lung cancer risk and that vehicle emissions may be particularly important.

PMID: 10955399 [PubMed - indexed for MEDLINE]

13. **Ambient air pollution and atherosclerosis in Los Angeles.** Kunzli N, Jerrett M, Mack WJ, Beckerman B, LaBree L, Gilliland F, Thomas D, Peters J, Hodis HN. Environ Health Perspect. 2005 Feb;113(2):201-6. Division of Environmental Health, Department of Preventive Medicine, Atherosclerosis Research Unit, Keck School of Medicine, University of Southern California, Los Angeles, California 90033-9013, USA. kuenzli@usc.edu

This is good background for the Jerrett LA American Cancer Society cohort study (below). This Kunzli study shows a relationship between fine particulate matter (PM) levels and build-up of arterial plaque in women's arteries. Like Michael Jerrett, Nino Kunzli was brought to LA (from Europe) to help with studies of mobile source pollution and health effects. Ever since C. Arden Pope's first American Cancer Society cohort study in 1995 showed very strong cardiovascular

impacts of fine PM, there has been a rush to find mechanisms. Those efforts quickly came to focus on cardiovascular inflammation, plaque build-up and ischemic heart disease leading to acute myocardial infarction (heart attack). A study published in December 2005 detailed the real creation of this condition in rodents using concentrated air pollutants. Pope had earlier found very high association of fine PM levels and C reactive protein levels (a very accurate biomarker for heart disease). It also seems that statins may work to mitigate inflammatory damage from fine PM.

Associations have been found between long-term exposure to ambient air pollution and cardiovascular morbidity and mortality. The contribution of air pollution to atherosclerosis that underlies many cardiovascular diseases has not been investigated. Animal data suggest that ambient particulate matter (PM) may contribute to atherogenesis. We used data on 798 participants from two clinical trials to investigate the association between atherosclerosis and long-term exposure to ambient PM up to 2.5 micron in aerodynamic diameter (PM2.5). Baseline data included assessment of the carotid intima-media thickness (CIMT), a measure of subclinical atherosclerosis. We geocoded subjects' residential areas to assign annual mean concentrations of ambient PM2.5. Exposure values were assigned from a PM2.5 surface derived from a geostatistical model. Individually assigned annual mean PM2.5 concentrations ranged from 5.2 to 26.9 microg/m3 (mean, 20.3). For a cross-sectional exposure contrast of 10 microg/m3 PM2.5, CIMT increased by 5.9% (95% confidence interval, 1-11%). Adjustment for age reduced the coefficients, but further adjustment for covariates indicated robust estimates in the range of 3.9-4.3% (p-values, 0.05-0.1). Among older subjects (greater than or equal to 60 years of age), women, never smokers, and those reporting lipid-lowering treatment at baseline, the associations of PM2.5 and CIMT were larger with the strongest associations in women 60 years of age (15.7%, 5.7-26.6%). These results represent the first epidemiologic evidence of an association between atherosclerosis and ambient air pollution. Given the leading role of cardiovascular disease as a cause of death and the large populations exposed to ambient PM2.5, these findings may be important and need further confirmation.

PMID: 15687058 [PubMed - indexed for MEDLINE]

14. **Spatial analysis of air pollution and mortality in Los Angeles.** Jerrett M, Burnett RT, Ma R, Pope CA 3rd, Krewski D, Newbold KB, Thurston G, Shi Y, Finkelstein N, Calle EE, Thun MJ. Epidemiology. 2005 Nov;16(6):727-36. Department of Preventive Medicine, Keck School of Medicine, University of Southern California, Los Angeles, California 90089-9011, USA. jerrett@usc.edu

This is Michael Jerrett's blockbuster. Using a subset of the ACS cohort just from Los Angeles, he shows much higher association of fine PM and mortality per 10 μg/m3 than previously found in the ACS metropolitan area studies by Pope. The pedigree of the co-authors who signed onto this study is unmatched. They also co-authored Harvard Six Cities 1993, Pope ACS 1995, Krewski HEI reanalyses 2000, Pope ACS 2002 and many other seminal studies. Michael Jerrett is one of the foremost geographically oriented environmental epidemiologists publishing in North America right now. He was brought down from Canada to help in LA. Although not an original goal of the study, Jerrett found a 44% increase in lung cancer mortality for zip codes whose centroids lay within 500 meters of southern California highway intersections.

BACKGROUND: The assessment of air pollution exposure using only community average concentrations may lead to measurement error that lowers estimates of the health burden attributable to poor air quality. To test this hypothesis, we modeled the association between air pollution and mortality using small-area exposure measures in Los Angeles, California. METHODS: Data on 22,905 subjects were extracted from the American Cancer Society cohort for the period 1982-2000 (5,856 deaths). Pollution exposures were interpolated from 23 fine particle (PM2.5) and

42 ozone (O3) fixed-site monitors. Proximity to expressways was tested as a measure of traffic pollution. We assessed associations in standard and spatial multilevel Cox regression models. RESULTS: After controlling for 44 individual covariates, all-cause mortality had a relative risk (RR) of 1.17 (95% confidence interval=1.05-1.30) for an increase of 10 mug/m PM2.5 and a RR of 1.11 (0.99-1.25) with maximal control for both individual and contextual confounders. The RRs for mortality resulting from ischemic heart disease and lung cancer deaths were elevated, in the range of 1.24-1.6, depending on the model used. These PM results were robust to adjustments for O3 and expressway exposure. CONCLUSION: Our results suggest the chronic health effects associated with within-city gradients in exposure to PM2.5 may be even larger than previously reported across metropolitan areas. We observed effects nearly 3 times greater than in models relying on comparisons between communities. We also found specificity in cause of death, with PM2.5 associated more strongly with ischemic heart disease than with cardiopulmonary or all-cause mortality.

PMID: 16222161 [PubMed - indexed for MEDLINE]

15. Association between mortality and indicators of traffic-related air pollution in the Netherlands: a cohort study. Hoek G, Brunekreef B, Goldbohm S, Fischer P, van den Brandt PA. Lancet. 2002 Oct 19;360(9341):1203-9.

Hoek and Brunekreef are among Europe's foremost air pollution and health researchers. They are very diverse. Brunekreef has been called many times to the US and has been tapped by Health Effects Institute – which is 50% owned by EPA and 50% by industry – to do more traffic and health investigations for them. HEI historically has garnered the most EPA funding of any outside policy and research group in the air / health realm. This Netherlands study found extraordinarily high relative risks for cardiopulmonary mortality among those living near very busy roadways – within 100 meters of highways and 50 meters of busy city streets: a 40% increase in all cause mortality and a 95% increase in cardiopulmonary deaths.

Institute for Risk Assessment Sciences, Environmental and Occupational Health group, Utrecht University, Utrecht, Netherlands. g.hoek@iras.uu.nl

BACKGROUND: Long-term exposure to particulate matter air pollution has been associated with increased cardiopulmonary mortality in the USA. We aimed to assess the relation between traffic-related air pollution and mortality in participants of the Netherlands Cohort study on Diet and Cancer (NLCS), an ongoing study. METHODS: We investigated a random sample of 5000 people from the full cohort of the NLCS study (age 55-69 years) from 1986 to 1994. Long-term exposure to traffic-related air pollutants (black smoke and nitrogen dioxide) was estimated for the 1986 home address. Exposure was characterised with the measured regional and urban background concentration and an indicator variable for living near major roads. The association between exposure to air pollution and (cause specific) mortality was assessed with Cox's proportional hazards models, with adjustment for potential confounders. FINDINGS: 489 (11%) of 4492 people with data died during the follow-up period. Cardiopulmonary mortality was associated with living near a major road (relative risk 1.95, 95% CI 1.09-3.52) and, less consistently, with the estimated ambient background concentration (1.34, 0.68-2.64). The relative risk for living near a major road was 1.41 (0.94-2.12) for total deaths. Non-cardiopulmonary, non-lung cancer deaths were unrelated to air pollution (1.03, 0.54-1.96 for living near a major road). INTERPRETATION: Long-term exposure to traffic-related air pollution may shorten life expectancy.

PMID: 12401246 [PubMed - indexed for MEDLINE]

16. Environmental inequality and circulatory disease mortality gradients. Finkelstein MM, Jerrett M, Sears MR. J Epidemiol Community Health. 2005 Jun;59(6):481-7. Program in Occupational

Health and Environmental Medicine and Institute of Environment and Health, McMaster University, Toronto, Canada. murray.finkelstein@utoronto.ca

Finkelstein and Jerrett had been studying Hamilton and Toronto in Ontario for quite some time. By adopting the simpler Hoek and Brunekreef paradigm of proximity to highways and busy urban roadways, they were able to find very high association of mobile sources and premature mortality that was not as evident in earlier studies using sophisticated GIS and kriging techniques that assumed equal harm from all fine PM mass within an urban area regardless of source. This study, though a bit problematic because of the special population, has added to their ability to separate socioeconomic risk from the risk of location, with location dominating the public health outcomes at this fine-grained scale.

STUDY OBJECTIVE: Studies in Europe and North America have reported that living in a disadvantaged neighbourhood is associated with an increased incidence of coronary heart disease. The aim of this study was to test the hypotheses that exposure to traffic and air pollution might account for some of the socioeconomic differences in mortality rates in a city where residents are covered by universal health insurance. DESIGN: Cohort mortality study. Individual postal codes used to derive: (1) socioeconomic status from census data; (2) mean air pollution levels from interpolation between governmental monitoring stations; (3) proximity to traffic from the geographical information system. Analysis conducted with Cox proportional hazards models. SETTING: Hamilton Census Metropolitan Area, Ontario, Canada, on the western tip of Lake Ontario (population about 480,000). PARTICIPANTS: 5228 people, aged 40 years or more, identified from register of lung function laboratory at an academic respirology clinic between 1985 and 1999. MAIN RESULTS: Circulatory disease (cardiovascular and stroke) mortality rates were related to measures of neighbourhood deprivation. Circulatory disease mortality rates were also associated with indices of long term ambient pollution at the subjects' residences (relative risk 1.06, 1.00 to 1.13) and **with proximity to traffic (relative risk 1.40, 1.08 to 1.81).** Subjects in more deprived neighbourhoods had greater exposure to ambient particulate and gaseous pollutants and to traffic. CONCLUSIONS: At least some of the observed social gradients in circulatory mortality arise from inequalities in environmental exposure to background and traffic air pollutants.

PMID: 15911644 [PubMed - indexed for MEDLINE]

17. **The effect of air pollution on lung development from 10 to 18 years of age.** 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. **N Engl J Med.** 2004 Sep 9;351(11):1057-67. Department of Preventive Medicine, University of Southern California, Los Angeles 90089, USA. jimg@usc.edu Erratum in: N Engl J Med. 2005 Mar 24;352(12):1276.

This study has been cited by many including the current EPA criteria review documents for PM. As with other near source studies, NO2 here is considered to be a marker of the general basket of traffic emissions and not necessarily the cause of all effects. Besides the very large increase in relative risk of lung capacity impairment in the communities with greater pollution levels, the study found that children who moved from a dirtier community to a cleaner community had proportionally less impairment.

BACKGROUND: Whether exposure to air pollution adversely affects the growth of lung function during the period of rapid lung development that occurs between the ages of 10 and 18 years is unknown. METHODS: In this prospective study, we recruited 1759 children (average age, 10 years) from schools in 12 southern California communities and measured lung function annually for eight years. The rate of attrition was approximately 10 percent per year. The communities represented a wide range of ambient exposures to ozone, acid vapor, nitrogen dioxide, and particulate matter. Linear regression was used to examine the relationship of air pollution to the forced expiratory volume in one second (FEV(1)) and other spirometric measures. RESULTS: Over the eight-year

period, deficits in the growth of FEV(1) were associated with exposure to nitrogen dioxide (P=0.005), acid vapor (P=0.004), particulate matter with an aerodynamic diameter of less than 2.5 microm (PM(2.5)) (P=0.04), and elemental carbon (P=0.007), even after adjustment for several potential confounders and effect modifiers. Associations were also observed for other spirometric measures. Exposure to pollutants was associated with clinically and statistically significant deficits in the FEV(1) attained at the age of 18 years. For example, the estimated proportion of 18-year-old subjects with a low FEV(1) (defined as a ratio of observed to expected FEV(1) of less than 80 percent) was 4.9 times as great at the highest level of exposure to PM(2.5) as at the lowest level of exposure (7.9 percent vs. 1.6 percent, P=0.002). CONCLUSIONS: The results of this study indicate that current levels of air pollution have chronic, adverse effects on lung development in children from the age of 10 to 18 years, leading to clinically significant deficits in attained FEV(1) as children reach adulthood. Copyright 2004 Massachusetts Medical Society

PMID: 15356303 [PubMed - indexed for MEDLINE]

18. Childhood asthma and exposure to traffic and nitrogen dioxide. Gauderman WJ, Avol E, Lurmann F, Kuenzli N, Gilliland F, Peters J, McConnell R. Epidemiology. 2005 Nov;16(6):737-43. Department of Preventive Medicine, University of Southern California Keck School of Medicine, Los Angeles 90089, USA. jimg@usc.edu

This second impressive Gauderman led study from the Children's Health series in southern California is very good on association of asthma and roadway proximity as well as the magnitude of effect from nearby highways as compared to busy city streets. Because the near source gradients are so steep, it is likely to be hard to sort out the effects of primary (fine PM, NO2, air toxics) versus secondary (ozone) air pollutants on childhood asthma in any study which does not have very good and fine-grained geographic control of cohort exposure levels. This very large California effort has many other interesting subparts including one which suggests that vitamins and healthy diet may be able to mitigate some of the outcomes. (Note that Somerville, Chelsea and downtown Boston children would be expected to have higher than average asthma only for those who live very close to the highways, and lower than average asthma everywhere else in these areas because of depressed ozone levels. The fresh traffic emissions which cause lung cancer and heart attack deaths to be elevated also lower local ozone levels as they borrow an oxygen atom from ozone to turn NO into NO2. In fact Somerville and Chelsea school children on the whole seem to have slightly lower average asthma rates than other communities with less fresh pollutants from traffic.)

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 (NO2) 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 NO2; 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 NO2 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 NO2 or other freeway-related pollutants.

PMID: 16222162 [PubMed - indexed for MEDLINE]

19. Long-term air pollution exposure and living close to busy roads are associated with COPD in women. Schikowski T, Sugiri D, Ranft U, Gehring U, Heinrich J, Wichmann EH, Kramer U. Respir Res. 2005 Dec 22;6(1):152 [Epub ahead of print].

This is included because it hits directly on the issue of adult breathing impairment near busy roadways. (In looking at MassCHIP's health information in the past, only lung cancer and acute myocardial infarction really pop off the pages in relation to transportation emissions and municipal mortalities, but it might be worth looking more thoroughly at the whole dataset again as there are more years available now than when I first looked at the health records for all 351 Massachusetts communtiies several years back.)

ABSTRACT: BACKGROUND: Lung function and exacerbations of chronic obstructive pulmonary disease (COPD) have been associated with short-term exposure to air pollution. However, the effect of long-term exposure to particulate matter from industry and traffic on COPD as defined by lung function has not been evaluated so far. Our study was designed to investigate the influence of long-term exposure to air pollution on respiratory symptoms and pulmonary function in 55-year-old women. We especially focused on COPD as defined by GOLD criteria and additionally compared the effects of air pollution on respiratory symptoms by questionnaire data and by lung function measurements. METHODS: In consecutive cross sectional studies conducted between 1985-1994, we investigated 4757 women living in the Rhine-Ruhr Basin of Germany. NO2 and PM10 exposure was assessed by measurements done in an 8 km grid, and traffic exposure by distance from the residential address to the nearest major road using Geographic Information System data. Lung function was determined and COPD was defined by using the GOLD criteria. Chronic respiratory symptoms and possible confounders were defined by questionnaire data. Linear and logistic regressions, including random effects were used to account for confounding and clustering on city level. RESULTS: The prevalence of COPD (GOLD stages 1-4) was 4.5%. COPD and pulmonary function were strongest affected by PM10 and traffic related exposure. A 7 mug/m3 increase in five year means of PM10 (interquartile range) was associated with a 5.1% (95% CI 2.5%-7.7%) decrease in FEV1, a 3.7% (95% CI 1.8%-5.5%) decrease in FVC and an odds ratio (OR) of 1.33 (95% CI 1.03-1.72) for COPD. Women living less than 100m from a busy road also had a significantly decreased lung function and COPD was 1.79 times more likely (95% CI 1.06-3.02) than for those living farther away. Chronic symptoms as based on questionnaire information showed effects in the same direction, but less pronounced. CONCLUSION: Chronic exposure to PM10, NO2 and living near a major road might increase the risk of developing COPD and can have a detrimental effect on lung function.

PMID: 16372913 [PubMed - as supplied by publisher]

20. Local variations in CO and particulate air pollution and adverse birth outcomes in Los Angeles County, California, USA. Wilhelm M, Ritz B. Environ Health Perspect. 2005 Sep;113(9):1212-21. Department of Epidemiology, School of Public Health, University of California, Los Angeles, CA 90095, USA.

There have been a lot of studies of birth outcomes and ambient air pollution. I am including two (here and below) by California researchers who are familiar with trying to tie these health effects to different air pollutants. Although many of the studies that have been done have found statitistical significance, they often do so for different pollutants and different trimesters. As with asthma and with fine PM generally, it is likely that multiple factors are involved, including socio-economic. It also seems likely to me that many of these studies, including this one, may be looking for fine-grained impacts using coarser-grained approaches. A better understanding of the geographic scales of influence and the gradients of the pollutants might lead to greater clarity

of association. Carbon monoxide, for example, can have very steep gradients near mobile sources, just as ultrafine particulates and many of the VOCs do. (Somerville, Chelsea and Boston have generally good birth outcomes - which speaks to the effective public health system here.)

We extended our previous analyses of term low birth weight (LBW) and preterm birth to 1994-2000, a period of declining air pollution levels in the South Coast Air Basin. We speculated that the effects we observed previously for carbon monoxide, particulate matter < 10 microm in aerodynamic diameter (PM10), and traffic density were attributable to toxins sorbed to primary exhaust particles. Focusing on CO, PM10, and particulate matter < 2.5 microm in aerodynamic diameter (PM2.5), we examined whether varying residential distances from monitoring stations affected risk estimates, because effect attenuation may result from local pollutant heterogeneity inadequately captured by ambient stations. We geocoded home locations, calculated the distance to the nearest air monitors, estimated exposure levels by pregnancy period, and performed logistic regression analyses for subjects living within 1-4 mi of a station. For women residing within a 1-mi distance, we observed a 27% increase in risk for high (> or = 75th percentile) first-trimester CO exposures and preterm birth and a 36% increase for high third-trimester pregnancy CO exposures and term LBW. For particles, we observed similar size effects during early and late pregnancy for both term LBW and preterm birth. In contrast, smaller or no effects were observed beyond a 1-mi distance of a residence from a station. Associations between CO and PM10 averaged over the whole pregnancy and term LBW were generally smaller than effects for early and late pregnancy. These new results for 1994-2000 generally confirm our previous observations for the period 1989-1993, again linking CO and particle exposures to term LBW and preterm birth. In addition, they confirm our suspicions about having to address local heterogeneity for these pollutants in Los Angeles.

PMID: 16140630 [PubMed - in process]

21. Birth outcomes and prenatal exposure to ozone, carbon monoxide, and particulate matter: results from the Children's Health Study. Salam MT, Millstein J, Li YF, Lurmann FW, Margolis HG, Gilliland FD. Environ Health Perspect. 2005 Nov;113(11):1638-44. Department of Preventive Medicine, University of Southern California, Keck School of Medicine, Los Angeles, California 90033, USA.

See note above.

Exposures to ambient air pollutants have been associated with adverse birth outcomes. We investigated the effects of air pollutants on birth weight mediated by reduced fetal growth among term infants who were born in California during 1975-1987 and who participated in the Children's Health Study. Birth certificates provided maternal reproductive history and residence location at birth. Sociodemographic factors and maternal smoking during pregnancy were collected by questionnaire. Monthly average air pollutant levels were interpolated from monitors to the ZIP code of maternal residence at childbirth. Results from linear mixed-effects regression models showed that a 12-ppb increase in 24-hr ozone averaged over the entire pregnancy was associated with 47.2 g lower birth weight [95% confidence interval (CI), 27.4-67.0 g], and this association was most robust for exposures during the second and third trimesters. A 1.4-ppm difference in first-trimester carbon monoxide exposure was associated with 21.7 g lower birth weight (95% CI, 1.1-42.3 g) and 20% increased risk of intrauterine growth retardation (95% CI, 1.0-1.4). First-trimester CO and third-trimester O3 exposures were associated with 20% increased risk of intrauterine growth retardation. A 20-microg/m3 difference in levels of particulate matter < or = 10 microm in aerodynamic diameter (PM10) during the third trimester was associated with a 21.7-g lower birth weight (95% CI, 1.1-42.2 g), but this association was reduced and not significant after adjusting for O3. In summary, O3 exposure during the second and third trimesters and CO exposure during the first trimester were associated with reduced birth weight.

PMID: 16263524 [PubMed - in process]

22. Roads, railways, and childhood cancers. Knox EG. J Epidemiol Community Health. 2006 Feb;60(2):136-41. Mill Cottage, Front Street, Great Comberton, Pershore, Worcestershire WR10 3DU, UK. E.G.Knox@btinternet.com.

EG Knox, a retired Professor, has been churning out a series of childhood cancer studies which simply look at the birth and death addresses of those children deceased by cancer who have migrated, and at the known mapped emissions sources in Britain, to find apparent levels of relative risk. His studies have been criticized by health statisticians hired by industry as lacking control of cohort characteristics, leading to possible inherent bias. However, the simple and consistent results showing very high association of birth addresses, proximity to mobile and other combustion sources, and childhood cancer frequency are compelling. They are consistent with more sophisticated analyses of susceptibility, and form at least a very strong hypothesis that should be confirmed or overturned by follow-up studies. Additionally, his work can fairly easily be presented to those residents most likely to be affected.

Study OBJECTIVES: To locate geographical sources of engine exhaust emissions in Great Britain and to link them with the birth addresses of children dying from cancer. To estimate the cancer initiating roles of nearby roads and railways and to measure effective ranges. DESIGN: Birth and death addresses of all children born between 1955 and 1980 in Great Britain, and dying from leukaemia or other cancer during those years, were linked to locations of railway stations, bus stations, ferry terminals, railways, roads, canals, and rivers. Nearest distances to births and deaths were measured, and migration data relating to children who had moved house were analyzed. Excesses of close to hazard birth addresses, compared with close to hazard death addresses, indicate a high prenatal or early postnatal risk of cancer initiation.Setting and SUBJECTS: Child cancer birth and death addresses and their map references were extracted from an earlier inquiry. Map references of putative hazards were downloaded from the Ordnance Survey national digital map of Great Britain. These data are recorded to a precision of one metre and have ground accuracies around 20 metres. MAIN RESULTS: Significant birth excesses were found within short distances of bus stations, railway stations, ferries, railways, and A,B class roads, with a relative risk of 2.1 within 100 m, tapering to neutral after 3.0 km. About 24% of child cancers were attributable to these joint birth proximities. Roads exerted the major effect. CONCLUSIONS: Child cancer initiations are strongly determined by prenatal or early postnatal exposures to engine exhaust gases, probably through maternal inhalation and accumulation of carcinogens over many months. The main active substance is probably 1,3-butadiene.

PMID: 16415262 [PubMed - in process]

23. Molecular evidence of an interaction between prenatal environmental exposures and birth outcomes in a multiethnic population. Perera FP, Rauh V, Whyatt RM, Tsai WY, Bernert JT, Tu YH, Andrews H, Ramirez J, Qu L, Tang D. Environ Health Perspect. 2004 Apr;112(5):626-30. Columbia Center for Children's Environmental Health, Mailman School of Public Health, Columbia University, 60 Haven Avenue #B-109, New York, NY 10032, USA. Fpp1@columbia.edu

Federica Perara is in some ways the opposite of EG Knox. She co-wrote the first book on fine PM in 1979, called Respirable Particles, and has been working out of the Mailman School at Columbia for many years in the field of molecular epidemiology. She has authored a series of very technical studies of the impact of PAHs, ETS and other complex pollutants on infants and children. Looking at New York City, and other communities around the world, she has found a high degree of association between mobile and other combustion pollutants with DNA adducts and myriad health outcomes. Her research suggests that exposure of infants and pregnant women to high levels of mobile source pollutants results in considerably higher susceptibility to

cancer and many other complex health outcomes. She has also found a cascading impact on infants that have highly polluted neighborhood air, other in-home exposures (like smoking) and some degree of deprivation. This paper is an example of her ongoing work.

Inner-city, minority populations are high-risk groups for adverse birth outcomes and also are more likely to be exposed to environmental contaminants, including environmental tobacco smoke (ETS), benzo[a]pyrene (BaP), and other polycyclic aromatic hydrocarbons (PAHs) found in urban air. In a sample of nonsmoking African-American and Dominican women, we evaluated the effects on birth outcomes of prenatal exposure to ETS, using questionnaire data and plasma cotinine as a biomarker of exposure, and environmental PAHs using BaP-DNA adducts as a molecular dosimeter. We previously reported that among African Americans, high prenatal exposure to PAHs estimated by prenatal personal air monitoring was associated with lower birth weight ($p = 0.003$) and smaller head circumference ($p = 0.01$) after adjusting for potential confounders. In the present analysis, self-reported ETS was associated with decreased head circumference ($p = 0.04$). BaP-DNA adducts were not correlated with ETS or dietary PAHs. There was no main effect of BaP-DNA adducts on birth outcomes. However, there was a significant interaction between the two pollutants such that the combined exposure to high ETS and high adducts had a significant multiplicative effect on birth weight ($p = 0.04$) and head circumference ($p = 0.01$) after adjusting for ethnicity, sex of newborns, maternal body mass index, dietary PAHs, and gestational age. This study provides evidence that combined exposure to environmental pollutants at levels currently encountered in New York City adversely affects fetal development.

PMID: 15064172 [PubMed - indexed for MEDLINE]

****

Additional references (roughly 24. through 42.) that focus on primary particulate nucleation and condensation, ultrafine particle concentration gradients near highways, ultrafine and semi-volatile health effects, and penetration of particulates and associated gases into nearby buildings. (Without additional comments but abstracts will be added.)