1. Pathophysiologic changes that involve inflammation, coagulation, and cardiac rhythm.

2. Subramania, R.
Motor Vehicle Traffic Crashes as a Leading Cause of Death in the United States, 2001.
National Highway and Traffic Safety Administration, Dec. 2003
http://www-nrd.nhtsa.dot.gov/pdf/nrd-30/NCSA/RNotes/2003/809-695.pdf

3. Fast Stats A to Z.
National Center for Health Statistics.
http://www.cdc.gov/nchs/fastats/acc-inj.htm

4.

Type of Injury	**Deaths Due to Injuries, 2000**
Road traffic	1,260,000
Suicide	815,000
Interpersonal violence	520,000
Drowning	450,000
Poisoning	315,000
War and conflict	310,000
Falls	283,000
Burns	238,000

World Health Organization, *The Injury Chartbook and Injury: A Leading Cause of the Global Burden of Disease,* May 12, 2003, http://www.who.int/mediacentre/releases/2003/pr40/en/

5. [11]
Samet, J.M., Zeger, S.L., Dominici, F., Curriero, F., Coursac, I., Dockery, D.W., Schwartz, J., & Zanobetti, A.
The National morbidity, mortality, and air pollution study: Part II
Morbidity and mortality from air pollution in the United States
Health Effects Institute; Research Report: Number 94, Part II; June 2000; p 82.
90 cities in different regions of the US, covering all areas. Daily PM_{10} values given for 1987 to 1994. Also O_3, SO_2, NO_2 and CO. Univariate analysis showed highest values for SO_2, with CO second, NO_2 third, and PM_{10} fourth. Distributed lag models give higher values, and authors note that the effects of pollution do not reach 0 until a lag of 5 days has occurred. Authors conclude: "Overall, this study provides strong evidence of association between PM_{10} levels and exacerbation of chronic heart and lung disease sufficiently severe to warrant hospitalization". Effect of PM_{10} on mortality generally higher in Northeast, industrial Midwest, and southern California than in other regions.

6. [210]
Zeghnoun, A., czernichow, P., Beaudeau, P., Hautemaniere, A., Froment, L., Le Tertre, a., & Quenel, P.
Short-term effects of air pollution on mortality in the cities of Rouen and Le Havre, France, 1990–1995Arch Environ Health 56; 327-335,2001
In Rouen, interquartile range increase of 60.5–94.1 micrograms/m^3 of ozone was associated with a 4.1 percent increase in total mortality; SO_2 (interquartile range17.6–36.4 mixcrograms/m^3)

associated with an 8.2 percent increase in respiratory mortality; NO_2 (interquartile range 25.3–42.2 micrograms/m^3)associated with a 6.1 percent increase in cardiovascular mortality. In Le Havre, SO_2 (interquartile range 11.3–35.6 micrograms/m3) was associated with 3 percent increase in cardiovascular mortality. Also an increase in cardiovascular mortality was associated with particulate pollution (PM less than or equal to 13 microns). Analysis used two pollutant models. Note problems of high collinearity between pollutants. Note that the region of Haute-Normandie where these cities are, is one of the most highly industrialized areas in France. Daily mortality in Rouen in summer averaged 9.58 and in winter 10.37; in Le Havre the figures were 6.00 and 6.67. Cardiovascular mortality was 4 times greater than respiratory in both cities. PM_{13} and NO_2 correlation coefficients were 0.58; and with SO_2 0.68; ozone was not correlated with any other pollutant. Note that the degree of temporal smoothing had a considerable influence on the indices.

7. [256]
McConnell, R., Berhane, K., Gilliland, F., London, S.J., Islam, T., Gauderman, W.J., AVOL, E., Margolis, H.G., & Peters, J.M.
Asthma in exercising children exposed to ozone: a cohort study; Lancet 2002; 359; 386–391
From the Southern California Children's Study.
Relevant numbers; 5762 children completed baseline questionnaires. 479 excluded because they were not at school when the questionnaire was administered; 883 excluded for a history of asthma; 312 excluded because of missing answers to "wheezing" questions; 26 excluded for chest illnesses such as cystic fibrosis; 527 excluded because they had less than one year of follow-up. This left 3535 children with no initial history of asthma. 2752 of these had no history of wheezing; 1934 played sports; 273 played three or more team sports. There were 46 low pollution communities (O_3 daytime mean 40.0 ppb); and 46 high pollution communities (O_3 mean 59.6 ppb). PM_{10} twice as high in high ozone communities (43.3 vs 21.6) and $PM_{2.5}$ three times higher (21.4 vs 7.6). NO_2 three times higher in high ozone communities (29.2 vs 10.8 ppb). It was shown that development of asthma prospectively was three times higher in children participating in more than 3 sports in high ozone communities, compared to children who did no sports in both communities or did fewer than 3 sports in high ozone communities. No differences in development of asthma if other pollutants were studied. Excellent discussion; 32 references. Convincing argument as to why standard cross-sectional comparisons might show no differences in prevalence of asthma.

8. [304]
Ballester, F., Saez, M., Perez-hoyos, S., Iniguez, C., Gandarillas, A., Tobias, A., Bellido, J., Taracido, M., Arribas, F., Daponte, A., Alonso, E., Canada, A., Guillen-Grima, F., Cirera, L., Perez-Boillos, M.J., Saurina, C., Gomez, F., Tenias, J.M.& The Emecam Group
The EMECAM project: a multicentre study on air pollution and mortality in Spain: combined results for particulates and for sulfur dioxide; Occup Environ Med 2002:59:300–30813
Spanish cities studied with data for the period 1990–1996. Poisson regression and generalized additive model. Results show 0.8 percent increase in total mortality per 10 microgram/m^3 increase in Black Smoke. Same increase in SO_2 associated with a 0.5 percent increase. Higher association seen for respiratory conditions (difficult to read exact figure from the graph). With 2 pollutant analyses, BS association not affected. SO_2 was reduced, but the association with SO_2 peaks was not affected. Authors conclude both associations were robust.

9. Kwon, H-J., Cho, S-H., Nyberg, F., & Pershagen, G.
Effects of ambient air pollution on daily mortality in a cohort of patients with congestive heart failure; Epidemiology 2001: 12; 413–419
Comparison between the general population and a cohort of patients with congestive heart failure. These were hospital admissions with a primary discharge diagnosis of congestive heart failure from a medical insurance file in Seoul for the period 1994 to 1996. This program covers 96 percent of the population. Daily number of deaths averaged 90.4 per day, and of patients with congestive heart failure averaged 1.0 per day. 694 male and 1,113 female patients with congestive heart failure. Pollutant data: 24 hour averages were PM_{10} mean 68.7 with 90^{th} percentile of 109.6; C0 (100 ppb) mean12.4; NO_2 ppb 24 hour mean 31.7 with 90^{th} percentile of 46.0; SO_2 ppb mean 13.4 with 90^{th} percentile of 25.1. One hour ozone in ppb as maximal hourly mean was 31.8 with 90^{th} percentile of 55.0. Correlation coefficients showed NO_2 & PM_{10} = 0.775; CO and NO_2 0.744; and SO_2 and PM_{10} was 0.699. Effects of pollutants were 2.5 to 4.1 times higher (depending on the pollutant) in the congestive heart failure cases than in the general population. Odds ratios for the general population were: For 42.1 micrograms/m^3 of PM_{10} = 1.014; for 0.59 ppm of CO = 1.022; for 14.6 ppb of NO_2 = 1.021; for 9.9 ppb of SO_2 = 1.020; and for 20.5 ppb of ozone = 1.010. The authors conclude: "The finding of a stronger association in the patients with congestive heart failure reinforces the evidence that a harmful effect of air pollution is mediated by cardiovascular mechanisms." Odds ratios for the cases were PM_{10} =1.058; CO = 1.054; NO_2 = 1.065; SO_2 = 1.070; and O_3 = 1.034.

10. Scientists have known for over a century that some air pollutants can cause global warming. The theory of global warming is quite simple: energy from the sun reaches the Earth. When it hits the ground, some of that light energy is transformed to infrared and bounces up. If it were able to pass through the atmosphere on its way up, it would leave the Earth a frozen and barren planet like Mars. Instead of leaving the upper atmosphere, however, the infrared is trapped by water vapor, carbon dioxide and other natural constituents of the air that act like the roof of a greenhouse. This " greenhouse effect" heats up the Earth's atmosphere to a level that supports life as we know it. Unfortunately some air pollutants also trap infrared heat, and the levels of these have been rising steadily for a century.

POLLUTANTS THAT CAUSE GLOBAL WARMING

Pollutant	Does it Cause Global Warming?	Does it Damage Human Health?	Time from Elimination to Climate Benefit
Carbon Monoxide	✔ Yes! Scavenges the hydroxyl radical, turns into CO_2	✔ Yes!	Hours, days
Ozone	✔ Yes! Roughly as powerful as methane.	✔ Yes!	Weeks
Fine Particles ("Black Carbon")	✔ Yes!	✔ Yes!	Weeks
Oxides of Nitrogen	✔ Yes! Creates ozone.	✔ Yes!	Weeks
Sulfur Dioxide	✖ No!	✔ Yes!	N/A

Carbon Dioxide	✔ Yes!	✔ Yes! Heat increase speeds ozone and fine particle formation.	30–95 years
Methane	✔ Yes! Second only to CO_2	✔ Yes! Forms "background" ozone	8–10 years

Most scientists believe that the Earth has been growing warmer and that this is attributable to human-caused pollution. A small minority of scientists say that global warming either is not occurring or is caused by natural factors, such as sunspots. Though many of these critics are supported directly or indirectly by the coal, oil, electricity and vehicle industries, they accuse those on the other side of bias because they are funded by governments.

If global warming is occurring, this should happen—	**Is It?**
Increase in atmospheric temperature.	Yes
Increase in sea temperatures.	Yes
Increase in soil temperature.	Yes
Decrease in stratospheric temperature.	Yes
Melting of glaciers.	Yes
Melting of sea ice.	Yes
Melting of polar ice.	Yes
Rise in ocean levels.	Yes
Shifts in plant and animal populations.	Yes
More violent and frequent weather events	Yes
If these increases are due to human activity, this should be found—	**Are they?**
Observed increases would agree with computer models	Yes
Models should accurately predict the past	Yes
Warming should vary by region	Yes

11. National Academy of Sciences, Committee on Tropospheric Ozone Formation and Measurement, *Rehtinking the Ozone Problem in Urban and Regional Air Pollution*, pp. 413–24, National Academy Press, 1991, Washington, D.C..

12. Deuel, H., Guthrie, P., Moody, W., Deck, L., Lange, S., Hameed, F., Castle, J., Mearns, L. Potential Impacts of Climate Change on Air Quality and Human Health
Air & Waste Management Association, June, 1999

13. [676]

Wjst, M., Reitmeir, P., Dold, S., Wulff, A., Nicolai, T., Von loeffelholz-Colberg, E.F., & Von Mutius, E.; Road Traffic and adverse effects on respiratory health in children; BMJ 307; 596–600, 1993.
There are 7445 fourth grade children aged 9–11 in Munich, of whom 6537 were examined in this survey. Density of car traffic ranged from 7000 to 125,000 cars per 24 hours. PEFR was 0.71 percent lower per increase of 25,000 cars. MEFR was also reduced. Response to cold air challenge not different however. Lifetime prevalence of asthma and recurrent bronchitis were not affected, but the cumulative presence of dyspnoea was slightly increased. This is the first report of an association with traffic and was quoted by Brunekreef in his later studies.

14. [415]
Hoek, G., Meliefste, K., Cyrys, J., Lewne, M., Bellander, T., Brauer, M., Fischer, P., Gehring, U., Heinrich, J., Van Vliet, P., & Brunekreef, B. Spatial variability of fine particle concentrations in three European areas; Atmospheric Environment 36; 4077–4088; (2002).
Data from Stockholm, Munich and different areas in the Netherlands. Comparisons between $PM_{2.5}$ recordings and the 'reflectance' of filters at 40–42 different sites. Annual average values of $PM_{2.5}$ were 11–20 micrograms/m^3 in Munich; 8–16 micrograms/m^3 in Stockholm; and 14–26 micrograms/m^3 in the Netherlands. $PM_{2.5}$ levels on average 17–18 percent higher at traffic sites than at urban background sites, and the absorption coefficient values were 31 percent to 55 percent increased above background. Authors note "that spatial variation of traffic-related air pollution may be underestimated if $PM_{2.5}$ only is measured." Plots show that $PM_{2.5}$ and reflectance values generally followed each other on temporal plots from the three regions. Technical issues discussed in detail and a well-written discussion of their findings relative to other similar studies of $PM_{2.5}$.

15. [680]
Nicolai, T., Carr, D., Weiland, S.K., Duhme, H., Von Ehrenstein, O., Wagner, C., & Von Mutius; Urban traffic and pollutant exposure related to respiratory outcomes and atopy in a large sample of children; Eur Respir J 2003; 21; 956–963.
Data from Munich. Random samples of 7,509 school children. Traffic exposure assessed by traffic count data and an emission model. Traffic counts were associated with current asthma, wheeze, and cough. In children with ETS exposure, traffic exposure also associated with a positive skin prick test. No pollutant associated with allergic sensitization, but cough associated with soot, benzene and NO_2; current asthma with soot and benzene; and current wheeze with benzene and NO_2. Authors note that effects of socioeconomic factors could not be ruled out.

16. [681]
Brunekreef, B., & Sunyer, J.
Editorial: Asthma, rhinitis and air pollution: is traffic to blame? Eur Respir J 2003; 21;913–915.
Comment on the previous two papers. Note: "Not surprisingly, the primary pollutants, carbon monoxide and NO_X contribute strongly and positively to this factor. However, at the same time, ozone has a negative loading, most likely related to the well-known fact that ozone concentrations are low in areas where primary emission concentrations are high. The interpretation then becomes complicated. Surely the associations found should not be interpreted as showing a protective effect of ozone but rather as suggesting an important role for primary combustion products from traffic."

17. [922]
Duki, M.I.Z., Sudarmadi, S., Suzuki, S., Kawada, T., & Tri-Tugaswati, A.
Effect of Air Pollution on Respiratory Health in Indonesia and its economic cost; Arch Environmental Health 58; 2003; 135–143.
16,663 pairs of junior high school students and their mothers in Indonesian cities surveyed by questionnaire. Prevalence rates of cough, phlegm, persistent cough, wheezing without a cold, and asthma significantly correlated with NO_2 emitted along large roads near their houses, and to a lesser extent with smoking. In Central Jakarta and Tangerang, the NO_2 levels were highest (37 ppb and 31 ppb measured for two periods of 3 consecutive days). It is calculated that reduction of NO_2 to a proposed level of 25 ppb "could yield savings in mean direct out-of-pocket expense per capita for treatment of the symptoms of 15,639–18,165 Indonesian rupiah (US% 6.80–7.90) and reduce average work/school days lost per capita by 3.1–5.5 days."

18. Stroke. 2003 Dec; 34(12):2776–80; Epub 2003 Nov 13. Stroke mortality associated with living near main roads in England and wales: a geographical study. Maheswaran R, Elliott P.; Public Health GIS Unit, School of Health and Related Research, University of Sheffield, UK. r.maheswaran@sheffield.ac.uk
Background and Purpose: Air pollution is associated with stroke, and road traffic is a major source of outdoor air pollution. Using proximity to roads as a proxy for exposure to road traffic pollution, we examined the hypothesis that living near main roads increases the risk of stroke mortality. Methods: We used a small-area ecological study design based on 113 465 census enumeration districts in England and Wales. Stroke mortality (International Classification of Disease, 9th revision, codes 430 through 438) in England and Wales from 1990 to 1992 for people >or=45 years of age was examined through the use of 1991 population denominators. Exposure was calculated as distance from each enumeration district population centroid to the nearest main road. We adjusted for age, sex, socioeconomic deprivation (using Carstairs index), regional variation, urbanization, and metropolitan area using Poisson regression. RESULTS: The analysis was based on 189 966 stroke deaths and a population of 19 083 979. After adjustment for potential confounders, stroke mortality was 7 percent (95 percent confidence interval [CI], 4 to 9) higher in men living within 200 m of a main road compared with men living >or=1000 m away. The corresponding increase in risk for women was 4 percent (95 percent CI, 2 to 6) and the risk for men and women combined was 5 percent (95 percent CI, 4 to 7). These raised risks diminished with increasing distance from main roads. Conclusions: Living near main roads is associated with excess risk of mortality from stroke, and if causality were assumed, approximately 990 stroke deaths per year would have been attributable to road traffic pollution. PMID: 14615623 [PubMed - indexed for MEDLINE]

19. [401]
Hoek, G., Brunekreef, B., Goldbohm, S., Fischer, P., & Van Den Brandt, P.; Association between mortality and indicators of traffic-related air pollution in the Netherlands: a cohort study; Lancet: Published online on Sept 24th, 2002.
Netherlands cohort study on diet and cancer (NLCS) started in 1986 with 120,852 participants aged 55–69 at enrollment: 48 percent were men. Exact address was known. Random sample of 5000 selected, and long term exposure to traffic was assessed (black smoke and NO_2) with an indicator variable for proximity of the home address to major roads. Those living within 100

metres of a freeway or within 50 metres of a major urban road were judged exposed. Statistical treatment used Cox's proportional hazards model with adjustment for potential confounders. 489 died during the follow-up period. Cardiopulmonary mortality was associated with living near a major road (relative risk was 1.95 with CI 1.09–3.52). Less consistently associated with estimated background ambient concentrations. Non cardiopulmonary non lung cancer deaths had no association to estimated air pollution. Confounder variables were as selected for the ACS study—smoking history known. Educational level also known. Corrections for socio-economic status also applied. Estimated mean background exposure to NO_2 was 36.1 microgram/m^3. Note that from US data loss of life expectancy is thought to be between 1 and 2 years between the most and least polluted exposures. Note that in this study there was no bias from self-reported exposures, and also that this study probably was less sensitive to spatial autocorrelation since exposure was estimated on a smaller spatial scale.

20. Epidemiology. 2001 Nov;12(6):649–53.
Daily mortality and air pollution along busy streets in Amsterdam, 1987–1998. Roemer WH, van Wijnen JH.; Department of Environmental Medicine, Amsterdam Municipal Health Service, Amsterdam, The Netherlands.
Time-series studies on the association between daily mortality and air pollution levels have been criticized because they use background air pollution measurement sites to estimate exposure of the whole population, including those living along busy roads. To evaluate whether the exposure of people living along busy roads is estimated with error, we calculated separate effect estimates with separate exposure estimates using background and traffic-influenced measurement stations. We used Poisson regression analysis with generalized additive models to correct for long-term trends, influenza, ambient temperature and relative humidity, and day of the week. Black smoke and nitrogen dioxide were associated with mortality (relative risk of 1.38 and 1.10, respectively, for an increase of 100 microg/m^3 on the previous day). Effect estimates were larger in the summer and in the population living along busy roads. Effect estimates were also larger using background stations rather than traffic stations. Overall, we found differences in the association between mortality and air pollution using different methods of exposure assessment; these differences are attributable to exposure misclassification for populations living along busy roads.

21. [514]
Hedley, A.J., Wong, C-M.M., Thach, T.Q., MA, S., Lam, T-H., & Anderson, H.R.; Cardiorespiratory and all-cause mortality after restrictions on sulfur content of fuel in Hong Kong: an intervention study; Lancet 2002: 360: 1646–1652.
In July 1990, Hong Kong mandated that all power plants and road vehicles had to use a fuel oil with no more that 0.5 percent sulfur by weight. Ambient SO_2 fell immediately. A significant decline in respiratory (3.9 percent) and cardiovascular mortality (2.0 percent) on an annual basis occurred, with no changes in deaths from other causes. Gain in life expectancy was 20 days for women and 41 days for men. Particulate levels did not change. Note that Katsouyanni in Europe noted an effect of SO_2 independently from $PM_{2.5}$. SO_2 ambient fell from a baseline level of 44.2 micrograms/m^3 to about 20.8 at one year after the fuel change, and 22.3 over the next 2–5 years. NO_2 levels fell a little, and O_3 levels and PM_{10} (at about 60 micrograms/m^3), were unaffected.

22. Ritz, B., Yu, F., Fruin, S., Chapa, G., Shaw, G.M., & Harris, J.A.; Ambient air pollution and risk of birth defects in Southern California; Am J Epidemiol 2002: 155;17-25.

The authors evaluated the effect of air pollution on the occurrence of birth defects ascertained by the California Birth Defects Monitoring Program in neonates and fetuses delivered in southern California in 1987–1993. By using measurements from ambient monitoring stations of carbon monoxide (CO), nitrogen dioxide, ozone, and particulate matter <10 μm in aerodynamic diameter, they calculated average monthly exposure estimates for each pregnancy. Conventional, polytomous, and hierarchical logistic regression was used to estimate odds ratios for subgroups of cardiac and orofacial defects. Odds ratios for cardiac ventricular septal defects increased in a dose-response fashion with increasing second-month CO exposure (odds ratio $(OR)_{2nd\ quartile}$ CO = 1.62, 95% confidence interval (CI): 1.05, 2.48; $OR_{3rd\ quartile}$ CO = 2.09, 95% CI: 1.19, 3.67; $OR_{4th\ quartile}$ CO = 2.95, 95% CI: 1.44, 6.05). Similarly, risks for aortic artery and valve defects, pulmonary artery and valve anomalies, and conotruncal defects increased with second-month ozone exposure. The study was inconclusive for other air pollutants. The authors' results are supported by the specificity of the timing of the effect and some evidence from animal data; however, this is the first known study to link ambient air pollution during a vulnerable window of development to human malformations.

23. [4]
Raaschou-Nielsen, O., Hertel, O., Thomsen, B.L., & Olsen, J.H.
Air Pollution from traffic at the residence of children with cancer
Am J Epidemiol 2001: 153; 433–443
1,989 children recorded with cancer at the Danish Cancer Registry. With diagnosis of leukemia, CNS tumors, or malignant lymphoma during 1968–1991 compared with 5,506 control children selected at random. Residential histories traced from 9 months before birth till the time of diagnosis. Information on traffic and configuration of streets collected, and average concentrations of benzene and NO_2 (indicators of traffic related air pollution) calculated. Risk of lymphomas (Hodgkin's disease) increased by 25 percent for a doubling of the calculated concentrations of benzene and NO_2. Good discussion of measurements and calculation of traffic exposure from NO_2.

24. Northeast States Clean Air Foundation, Hunts Point Cooperative Market: Advanced Truck Stop Electrification Project, http://www.cleanaircommunities.org/projects/huntspoint.html

25. New York City Department of Health and Mental Hygiene, "The Health of Hunts Point and Mott Haven," http://www.ci.nyc.ny.us/html/doh/pdf/data/2003nhp-bronxd.pdf

26. http://www.thepoint.org/reenvisioning/ACTION_W/WHYAREWE.HTM

27. [469]
Lena, T.S., Ochieng, V., Carter, M., Holguin-Veras, J., & Kinney, P.L.; Elemental carbon and $PM_{2.5}$ levels in an urban community heavily impacted by truck traffic; Environ Health Perspectives 110;1009–1015 (2002).
Hunts Point is a 650 acre peninsula on the southeastern shore of the Bronx, NY City, and is a hub of the transportation system. 10,000 trucks a day use it. Notes that mean EC concentrations may vary by a factor of 4, whereas $PM_{2.5}$ levels are reasonably homogenous. Detailed sampling design is described. Average hourly counts of large trucks were about 124; of small trucks about 95; and passenger cars about 200. Elemental carbon levels in micrograms/m^3 varied from about

2.60 to peak levels of 7.70. Straight line relationship between EC concentration and the number of large trucks per hour, such that baseline EC was about 2.5, and EC level of 7 corresponded to 3000 trucks/hour. $PM_{2.5}$ less closely related but still significant and went from about 20 micrograms/m^3 to about 27 micrograms/m^3 when the traffic was about 250 large trucks/hour. Interesting data.

28. [35]
Steerenberg, P.A., Nierkens, S., Fischer, P.H., Vanloveren, H., Opperhuizen, A., Vos, J.G., & Van Amsterdam, J.G.C.
Traffic-related air pollution affects peak expiratory flow, exhaled nitric oxide, and inflammatory nasal markers.
Arch Environ Health 56; 167–174; 2001
82 children attending elementary school either in Utrecht (more polluted) or in Bilthoven (classified as suburban) studied. Black smoke in Utrecht was 53 micrograms/m^3 compared to 18 in Bilthoven. NO_2 and CO were 1.5 and 1.8 times higher in Utrecht. Nasal lavage showed that levels of interleukin-98, urea, uric acid, albumin, and nitric oxide metabolites were significantly higher in the Utrecht children. PEFR was 5.3 ml/min less in Utrecht children. Coughing and respiratory disease levels appeared to be higher in suburban children, and parents of suburban children smoked more cigarettes/day. Odds ratios for different parameters reported. Authors conclude that children in area of higher traffic-related air pollution show increased levels of inflammatory nasal markers; increased response in PEFR; and increased levels of exhaled NO.

29. [294]
Gehring, U., Cyrys, J., Sedimeir, G., Brunekreef, B., Bellander, T., Fischer, P., Bauer, C.P., Reinhardt, D., Wichmann, H.E., & Heinrich, J.
Traffic-related air pollution and respiratory health during the first two years of life
Eur Respir J 2002, 19; 690–698
Study of Traffic-Related Air Pollution on childhood Asthma (TRAPCA). Geographic information systems based exposure modeling used to estimate exposure to traffic-related air pollutants in Munich at the birth addresses of 1,756 infants. Ranges in estimated exposures as follows: $PM_{2.5}$ 11.9–21.9 micrograms/m^3; $PM_{2.5}$ absorbance 1.38–4.39 x 10^{-5} m^{-1}; NO_2 19.5–66.9 micrograms/m^3. Significant associations found between these pollutants and cough without infection; and dry cough at night in the first year of life. In the second year, the effects were attenuated. $PM_{2.5}$ absorbance thought to be a marker for diesel exposure. 40 measurement sites for the pollutants were used for the modeling of exposure. In this age group, doctor diagnosed asthma was below 1 percent. There was a 70 percent prevalence of doctor diagnosed respiratory infections in the first year of life. 12.8 percent had gas cooking; 6.6 percent had home dampness; 30.8 percent had indoor molds; and 18.3 had pets at home. 23.4 percent had ETS at home. Associations were stronger in boys than girls. Review of previous similar studies. Future papers will deal with Dutch and Swedish data from the same study, but the Dutch data are said to have shown an association between traffic exposure and wheeze and asthma in the first two years of life (unreferenced).

30. Occup Environ Med. 1996 Apr; 53(4):241–7
Chronic respiratory symptoms in children and adults living along streets with high traffic density; Oosterlee A, Drijver M, Lebret E, Brunekreef B.

Objectives: To investigate if the population living along streets with high traffic density has a higher prevalence of chronic respiratory symptoms. Methods: A sample of 673 adults and 106 children (0–15 years), living along busy traffic streets in the city of Haarlem was compared with a control sample of 812 adults and 185 children living along quiet streets. Exposed and control streets were selected on the basis of model calculations of NO_2 concentrations. A postal questionnaire containing questions about respiratory symptoms and several potential confounders was used to collect information from the study subjects. Results: After adjustment for potential confounders, children living along busy streets were found to have a higher prevalence of most respiratory symptoms than children living along quiet streets. Adjusted odds ratios were significant for wheeze and for respiratory medication used. Risk ratios were higher for girls than for boys, with significant adjusted odds ratios between 2.9 and 15.8 for girls. In adults, only mild dyspnoea was more often reported by subjects living along streets with high traffic density.

31. Environ Res. 1997;74(2):122–32.
Motor vehicle exhaust and chronic respiratory symptoms in children living near freeways.
van Vliet P, Knape M, de Hartog J, Janssen N, Harssema H, Brunekreef B.
To examine whether motor vehicle exhaust from freeways has an effect on respiratory health of children, a cross-sectional study was conducted. Children attending schools situated less than 1000 m from major freeways in the Province of South Holland were asked to participate. The selected freeways carry between 80,000 and 150,000 vehicles per day. Separate counts for truck traffic indicated a range from 8000 to 17,500 trucks per day. At a total of 13 schools, 1498 children were asked to participate. From these children, 1068 usable questionnaires were obtained. Chronic respiratory symptoms reported in the questionnaire were analyzed with logistic regression. Distance from the freeway and (truck) traffic intensity were used as exposure variables. Cough, wheeze, runny nose, and doctor-diagnosed asthma were significantly more often reported for children living within 100 m from the freeway. Truck traffic intensity and the concentration of black smoke measured in schools were found to be significantly associated with chronic respiratory symptoms. These relationships were more pronounced in girls than in boys.

32. Kim, J.J., Smorodinsky, S., Lipsett, M., Singer, B.C., Hodgson, A.T., & Ostro, B.
Traffic-related air pollution near busy roads: the East Bay Children's Respiratory Health Study.Am. J. Respir. Crit. Care Med. 2004, doi:10.1164/rccm.200403-281OC
The team conducted a school-based cross-sectional study in the San Francisco Bay Area in 2001. Information on current bronchitis symptoms and asthma, home environment, and demographics were obtained by parental questionnaire (n=1,109). Concentrations of traffic pollutants (particulate matter (PM_{10}, $PM_{2.5}$), black carbon (BC), and nitrogen oxides (NO_X and NO_2)) were measured at ten school sites during several seasons. Although pollutant concentrations were relatively low, differences were observed in concentrations between schools nearby versus those more distant (or upwind) from major roads. Using a two-stage multiple logistic regression model, found associations between respiratory symptoms and traffic-related pollutants. Among those living at their current residence for at least one year, the adjusted odds ratios (OR) for asthma in relation to an interquartile difference in NO_X were OR = 1.07; (95 percent confidence interval, 1.00–1.14). Thus, we found spatial variability in traffic pollutants and associated differences in respiratory symptoms in a region with good air quality. Our findings support the hypothesis that traffic-related pollution is associated with respiratory symptoms in children.

33. Ned Tijdschr Geneeskd. 1997 Sep 20;141(38):1814–8
[Relationship between air pollution due to traffic, decreased lung function and airway symptoms in children][Article in Dutch]; de Hartog JJ, van Vliet PH, Brunekreef B, Knape MC, Janssen NA, Harssema H. Landbouwuniversiteit, departement Omgevingswetenschappen, Wageningen.
Objective: To assess whether air pollution by traffic was related to lung function and chronic respiratory symptoms in children living. Design: Descriptive. Setting: The province of South Holland, the Netherlands. Methods: In the period May through July of 1995 pulmonary function tests and questionnaires were obtained from 1,092 and 1,068 children respectively in six city districts near busy motorways in the province of South Holland. In the same period, indoor measurements were performed at 12 schools of NO_2, black smoke and PM_{10} dust density. Lung function data were analyzed by multiple linear regression and respiratory symptoms were analyzed by multiple logistic regression. As independent variables, distance between motorway and home, passenger car traffic density and lorry traffic density on the motorway, and black smoke and NO_2 concentrations in schools were taken. Results: Significant differences in lung function and respiratory symptoms were found between children living in different city districts. Lung function as well as symptoms were associated with lorry traffic density on the motorway. The validity of these findings was supported by associations between black smoke concentrations (representative for diesel soot) and lung function as well as respiratory symptoms. In contrast, there was no association between passenger car traffic counts or NO_2 and lung function or respiratory symptoms. Conclusion: The results suggest that air pollution by lorry traffic can lead to reduced lung function and to an increased prevalence of chronic respiratory symptoms in children living near major motorways.
34. Eur Respir J. 2002 Apr;19(4):690–8
Traffic-related air pollution and respiratory health during the first two years of life.
Gehring U, Cyrys J, Sedlmeir G, Brunekreef B, Bellander T, Fischer P, Bauer CP, Reinhardt D, Wichmann HE, Heinrich J.
GSF-National Research Centre for Environment and Health, Institute of Epidemiology, Neuherberg, Germany. gehring@gsf.de
As part of an international collaborative study on the impact of Traffic-Related Air Pollution on Childhood Asthma (TRAPCA), the health effects associated with long-term exposure to particles with a 50 percent cut-off aerodynamic diameter of 2.5 microm ($PM_{2.5}$), $PM_{2.5}$ absorbance, and nitrogen dioxide (NO_2) were analyzed. The German part of the TRAPCA study used data from subpopulations of two ongoing birth cohort studies (German Infant Nutrition Intervention Programme (GINI) and Influences of Lifestyle Related Factors on the Human Immune System and Development of Allergies in Children (LISA)) based in the city of Munich. Geographic information systems (GIS)-based exposure modelling was used to estimate traffic-related air pollutants at the birth addresses of 1,756 infants. Logistic regression was used to analyze possible health effects and potential confounding factors were adjusted for. The ranges in estimated exposures to $PM_{2.5}$, $PM_{2.5}$ absorbance, and NO_2 were 11.9–21.9 microg m(-3), 1.38–4.39 x 10(-5) m(-1), and 19.5–66.9 microg x m3, respectively. Significant associations between these pollutants and cough without infection (odds ratio (OR) (95% confidence interval (CI)): 1.34 (1.11–1.61), 1.32 (1.10–1.59), and 1.40 (1.12–1.75), respectively) and dry cough at night (OR (95% CI): 1.31 (1.07–1.60), 1.27 (1.04–1.55), and 1.36 (1.07–1.74), respectively) in the first year of life were found. In the second year of life, these effects were attenuated. There was some indication of an association between traffic-related air pollution and symptoms of cough. Due to

the very young age of the infants, it was too early to draw definitive conclusions from this for the development of asthma.

35. American Journal of Respiratory and Critical Care Medicine Vol 166. pp. 1092–1098, (2002)
Air pollution from traffic and the development of respiratory infections and asthmatic and allergic symptoms in children; Brauer M, Hoek G, Van Vliet P, Meliefste K, Fischer PH, Wijga A, Koopman LP, Neijens HJ, Gerritsen J, Kerkhof M, Heinrich J, Bellander T, Brunekreef B.; School of Occupational and Environmental Hygiene, University of British Columbia, Vancouver, British Columbia, Canada. brauer@interchange.ubc.ca
Despite the important contribution of traffic sources to urban air quality, relatively few studies have evaluated the effects of traffic-related air pollution on health, such as its influence on the development of asthma and other childhood respiratory diseases. We examined the relationship between traffic-related air pollution and the development of asthmatic/allergic symptoms and respiratory infections in a birth cohort (n approximately 4,000) study in The Netherlands. A validated model was used to assign outdoor concentrations of traffic-related air pollutants (nitrogen dioxide, particulate matter less than 2.5 micro m in aerodynamic diameter, and "soot") at the home of each subject of the cohort. Questionnaire-derived data on wheezing, dry nighttime cough, ear, nose, and throat infections, skin rash, and physician-diagnosed asthma, bronchitis, influenza, and eczema at 2 years of age were analyzed in relation to air pollutants. Adjusted odds ratios for wheezing, physician-diagnosed asthma, ear/nose/throat infections, and flu/serious colds indicated positive associations with air pollutants, some of which reached borderline statistical significance. No associations were observed for the other health outcomes analyzed. Sensitivity analyses generally supported these results and suggested somewhat stronger associations with traffic, for asthma that was diagnosed before 1 year of age. These findings are subject to confirmation at older ages, when asthma can be more readily diagnosed.

36. [60]
Tittanen, P., Timonen, K.L., Ruuskanen, J., Mirme, A., & Pekkanen, J.
Fine Particulate air pollution, resuspended road dust and respiratory health among symptomatic children
Eur Respir J 1999; 13; 266–273
"49 children with chronic respiratory symptoms aged 8–13 followed daily for six weeks in the spring in Kuopio Finland." Standard measurements of PM_{10} and $PM_{2.5}$ together with the number concentrations of particles from 0.01–10 microns in size PM_{10} was mostly resuspended soil and street dust and the concentration was estimated using aluminum content of the PM_{10} samples. Evening PEFR was significantly associated with the 1 day lagged number of particles in the size range 0.1–1.0 microns. All the particulate indices were significantly associated with an increased risk of cough. Main sources of particulate air pollution in Kuopio are traffic, peat-fired power plant, and corrugated cardboard mill. Medication use noted in diaries. Interesting data on intercorrelations of pollutants, with NO_X having a correlation coefficient of 0.79 with number of particles 0.01–0.1 size range, and 0.41 with $PM_{2.5}$
Port Alberni study is cited, but not discussed in detail.

37. [57]

Salvi, S., Blomberg, A., Rudell, B., Kelly, F., Sandstrom, T., Holgate, S.T., & Frew, A.
Acute inflammatory responses in the airways and peripheral blood after short-term exposure to diesel exhaust in healthy human volunteers
Am J Respir Crit Care Med 1999; 159; 702–709
15 healthy human volunteers exposed to air and diluted diesel exhaust for one hour with intermittent exercise. PM_{10} inhaled was 300 micrograms/m3; NO_2 was 1.6 ppm; NO 4.5 ppm; CO 7.5 ppm; total hydrocarbons 4.3 ppm; formaldehyde 0.26 milligram/m^3; suspended particles were 4.3 x 10^6 cm^3 6 hours after end of exposure, BAL performed. Results showed no effects on lung function; significant increase in neutrophils and B lymphocytes in BAL; together with increases in histamine and fibronectin; Bronchial biopsies showed increases in neutrophils, mast cells, CD4+ and CD8+, and T lymphocytes. Upregulation of endothelial adhesion molecules also found. Neutrophils and platelets increased significantly in peripheral blood following exposure. Authors conclude that "at high ambient concentration, acute short-term diesel exhaust exposure produces a well-defined and marked systemic and pulmonary inflammatory response in healthy human volunteers, which is underestimated by standard lung function measurements." Important data.

38. [83]
Giovagnoli, M.R., Alderisio, M., Cenci, M., Nofroni, I., & Vecchione, A.
Carbon and Hemosiderin-laden macrophages in sputum of traffic policemen exposed to air pollution.
Arch Environ Health 54; 284–290; 1999
164 Rome traffic policemen compared to 119 individuals from Perugia (unexposed). Sputum produced on three consecutive mornings fixed with 70 percent alcohol. 20 fields per two slides analyzed. Tenfold greater concentration of dust cells in Roman specimens. Increased macrophages and inflammatory cells also observed.

39. [957]
Holgate, S.T., Sandstrom, T., Frew, A.J., Stenfors, N., Nordenhall, C., Salvi, S., Blomberg, A., Helleday, R., & Sodenberg, M.
Health Effects of Acute Exposure to Air Pollution.
Part 1: Healthy and Asthmatic subjects exposed to diesel exhaust Health Effects Institute Research Report No. 112; December 2003
15 asthmatic subjects with mild atopic asthma and positive methacholine challenge with FEV_1 more than 70 percent of predicted. Control group of 25 volunteers of mean age 25 years. All were nonsmokers. Directly generated and diluted diesel exhaust exposure at about 100 micrograms/m^3 for 2 hours. No significant changes in bronchial wash fluid, but bronchial biopsy material showed a significant increase in IL-8 gene expression, and an increase in IL-8 protein in BAL fluid; Modest but significant increase in airway resistance in both groups and lymphocytes were increased in BAL samples. In the asthmatic subjects, there was also a significant increase in IL-10 staining in the biopsy tissues. Authors concluded: "This study demonstrated that modest concentrations of diesel exhaust have clear-cut inflammatory effects on the airways of nonasthmatic (control) subjects." No evidence of neutrophilic inflammation in the asthmatics.

40. For example, when eight persons were exposed for six hours to levels of oxides of nitrogen, then to an allergen, markers of inflammation—specifically, eosinophilic cationic protein—

increased. The authors concluded that "These results suggest that acute exposure to NO_2 at levels found at the curbside in heavy traffic during episodes of pollution, may 'prime' eosinophils for subsequent activation by allergen in individuals with a history of seasonal allergic rhinitis." In plainer language, the air pollution worsened and lengthened bouts of hay fever.

A study in Taiwan tends to confirm this. Comparing pollution levels with prevalence of doctor-diagnosed allergic rhinitis—again, hay fever to most of us—in 331,686 people, they found that as levels of carbon monoxide, oxides of nitrogen and ozone—or, in other words, traffic—rose, hay fever did the same.

[484]
Wang, J.H., Devalia, J.L., Duddle, J.M., Hamilton, S.A., & Davies, R.J.
Effect of six-hour exposure to nitrogen dioxide on early-phase nasal response to allergen challenge in patients with a history of seasonal allergic rhinitis; J Allergy Clin Immunol 1995; 96; 669–6768 cases studied.
Exposure to either air or 400 ppb NO_2 for 6 hours. Changes in nasal airways resistance (NAR) and eosinophil cationic protein (ECP), mast cell tryptase (MCT), neutrophil myeloperoxidase (MPO) and interleukin-8 (IL-8) in nasal lavage recorded before and after exposure. Air or NO_2 exposure did not alter these parameters. Allergen exposures, after both air and NO_2 increased levels of MCT. ECP levels increased after NO_2 exposure followed by allergen exposure. Authors suggest: 'These results suggest that acute exposure to NO_2 at levels found at the curbside in heavy traffic during episodes of pollution, may "prime" eosinophils for subsequent activation by allergen in individuals with a history of seasonal allergic rhinitis".

41. [679]
Lee, Y-L., Shaw, C-K., Su, H-J., Lai, J-S., Ko, Y-C., Huang, S-L., Sung, F-C., & Guo, Y.L.; Climate, traffic-related air pollutants and allergic rhinitis prevalence in middle-school children in Taiwan; Eur Respir j 2003; 21; 964–970.
331,686 nonsmoking children in schools within 2 km of 55 monitoring stations. Mean annual exposures were CO = 853 ppb; NO_X = 35.1 ppb; SO_2 = 7.57 ppb; PM_{10} = 69.2 micrograms/m3; O_3 = 21.3 ppb; temperature = 22.9□C; relative humidity = 76.2%. Prevalence of physician diagnosed allergic rhinitis was 28.6 percent in males and 19.5 percent in females. After adjustments, prevalence was associated with higher midsummer temperatures, and CO, NO_X, and O_3.

42. [101]
Zagury, E., Le Moullec, Y., & Momas, I.
Exposure of Paris taxi drivers to automobile air pollutants within their vehicles
Occup Environ Med 2000; 57; 406–410
Data collected from 29 randomly selected drivers between 27 Jan and 27 March 1997. CO measured over 8-hour period with portable monitor. Black smoke measured with cellulose filter and pump; NO_X measured with passive samplers. Levels measured:

Pollutant	Mean	Median	SD

CO	3.8 ppm	2.0	1.7 ppm
Black Smoke	168	164	53 microgram/m^3
NO_X	139	131	43 microgram/m^3
	(72.3)	(68.1)	(22.4) ppb

All of these values were higher than concentrations recorded in ambient air by ambient Parisian air monitoring network over the same period, and were close to or slightly exceeded the concentrations measured at fixed stations close to traffic. Authors note that the results justify "a medical follow-up of this occupational group". Figures show a one minute peak of high CO (30 ppm) for one driver; a peak of > 1100 micrograms/m^3 of NO in another; and a peak of 180 micrograms/m^3 of NO_2 recorded as the means of all observations. Influence of prevailing weather conditions noted. Also—

[227]
Weinhold, B.
Editorial: Pollutants lurk inside vehicles; Environ Health Perspectives 109; A422–A427, 2001
Review of data collected in Paris on pollutants outside and within vehicles. Refers to 1998 CARB Report. VOC concentrations, particularly benzene about twice as high in the vehicle as outside, with a considerable differential in CO. NO_X not discussed. Discussion of various mitigation and avoidance strategies.

43. [275]
Rank, J., Folke, J., & Jespersen, P.H.
Differences in cyclists and car drivers exposure to air pollution from traffic in the city of Copenhagen
Science of the Total Environment 279 (2001) 131–136
Two cyclists and two car drivers in two cars equipped with personal air samplers while driving for 4 hours on two different days in the morning traffic of Copenhagen. Air sample charcoal tubes analyzed for benzene, toluene, ethylbenzene and xylene content, and the air filters analyzed for particles. Days were in June and August. Cars not using air vent recirculation. Exposure of car drivers for particulate matter was twice that of cyclists; for benzene was three times higher in car drivers; and for the other organics was four times higher. Discussion notes that others have reported higher organic chemical exposures in cars than in ambient air. Note that WHO has established a life span risk of cancer over 70 years per million people of 0.13–0.23 microgram/m^3 of benzene. The values recorded in this study were 5–14 micrograms/m3 or 40 – 50 times higher than the WHO concentration. No calculation of delivered dose based on higher ventilation rate of cyclists; role of car heater does not seem to be precisely defined. Interesting comparisons with other observations. 20 references.

44. [797]
Jarvholm, B., & Silverman, D.
Lung cancer in heavy equipment operators and truck drivers with diesel exhaust exposure in the construction industry
Occup Environ Med 2003; 60; 516–520

Swedish computerized register of construction workers spanning 1971 to 1992 with 6,364 male truck drivers and 14,364 drivers of heavy construction vehicles selected as index group, and 119,984 carpenters and electricians were the reference group. An increased risk of lung cancer existed in the truck drivers but not in the operators of heavy equipment (use of cabins decreased risk of lung cancer). Truck drivers also had an increased risk of prostate cancer.

45. [492]
Adams, H.S., Nieuwenhuijsen, M.J., Colvile, R.N., Older, M.J., & Kendall, M.
Assessment of road users' elemental carbon personal exposure levels, London, UK; Atmospheric Environment 36 (2002) ; 5335–534
2400 fine particle ($PM_{2.5}$) personal exposure levels recorded during journeys by bus, bicycle, car and underground railway. EC contribution calculated by new optical method. Geometric means in summer were: cyclists 11.2 [16.4] micrograms/m^3; bus passengers 13.6 [18.6]; car drivers 21.6 [27.3]. Winter levels in brackets [ ]. EC/$PM_{2.5}$ ratios were 0.5–0.6 for cyclists and 0.7–0.8 for car drivers. Large differences noted between central high traffic density roads and less central routes. EC personal exposure levels were noted to be considerably higher than those reported from fixed site monitors. Data from Imperial College, London.

46. [241]
Bunn, H.J., Dinsdale, D., Smith, T., & Grigg, J.
Ultrafine particles in alveolar macrophages from normal children; Thorax 2001; 56; 932–934
22 children aged 3 months to 16 years. No respiratory symptoms; Nonbronchoscopic BAL procedure before elective surgery. Data from Leicester, UK; proximity of home to busy main road or residential street noted. All children's AM contained ultrafine carbonaceous particles (< 0.1 microns). Significantly more found in children who lived close to a busy traffic road. EM picture of carbonaceous ultrafine particles within a phagosome of an alveolar macrophage from a child aged 3 months. 10 percent of AM contained particles in AMs of children on busy roads, against 3.2 percent in children who lived on quiet roads.

47. [307]
Lin, S., Munsie, J.P., Hwang, S-A., Fitzgerald, E., & Cayo, M.R.; Childhood asthma hospitalization and residential exposure to State route traffic; Environmental Research Section A 88; 73–81 (2002)
417 white children aged 0–14 living in Erie County but not in Buffalo, who were admitted to hospital for asthma between Jan 1990 and December 1993. Controls were 461 children of similar age admitted over the same period for nonrespiratory conditions. Residential information linked to traffic information provided by the New York State Department of Transportation. After adjustment for age and poverty level, children hospitalized for asthma were more likely to live on roads with the highest tertile of vehicle miles traveled (within 200 metres) with an OR of 1.93, and were more likely to have trucks and trailers passing within 200 metres of their residence (OR = 1.43). No associations if distances were 500 metres. Comprehensive discussion

48. [914]
Gatshick, E., Laden, F., Hart, J.E., & Caron, A.; Residence near a major road and respiratory symptoms in U.S. Veterans
Epidemiology 14: 728–736; 2003

Data from southeastern Massachusetts collected by questionnaire from 2,628 veterans. GIS methodology to assess distance from a main road. After adjustments for smoking, age, and occupational exposures, men living within 50 metres of a main road were more likely to report persistent wheeze (OR=1.3) compared with those living more than 400 metres away. Risk of chronic phlegm was also increased, but no association with chronic cough. The association was not dependent on pre-existing doctor diagnosed chronic respiratory or cardiac disease. Mean age was 60.6 years. Median traffic volume was 9351 vehicles per day for subjects within 50 metres of the roadway. Good review of similar studies and relevant literature. Notes similar data from 3 groups of 1500 women in Tokyo, in whom the risk of wheeze was also noted to be elevated. 39 references.

49. Epidemiology. 1997 May;8(3):298–303
Air pollution from truck traffic and lung function in children living near motorways; Brunekreef B, Janssen NA, de Hartog J, Harssema H, Knape M, van Vliet P.
The contribution of motorized traffic to air pollution is widely recognized, but relatively few studies have looked at the respiratory health status of subjects living near busy roads. We studied children in six areas located near major motorways in the Netherlands. We measured lung function in the children, and we assessed their exposure to traffic-related air pollution using separate traffic counts for automobiles and trucks. We also measured air pollution in the children's schools. Lung function was associated with truck traffic density but had a lesser association with automobile traffic density. The association was stronger in children living closest (< 300 m) to the motorways. Lung function was also associated with the concentration of black smoke, measured inside the schools, as a proxy for diesel exhaust particles. The associations were stronger in girls than in boys. The results indicate that exposure to traffic-related air pollution, in particular diesel exhaust particles, may lead to reduced lung function in children living near major motorways.PMID: 9115026 [PubMed - indexed for MEDLINE]

50. Weiland SK, Mundt KA, Ruckmann A, Keil U. Self reported wheezing and allergic rhinitis in children and traffic density on street of residence. Ann Epidemiol 4:243–247 (1994).
(30.) Duhme H, Weiland SK, Keil U, Kraemer B, Schmid M, Stender M, Chambless L. The association between self-reported symptoms of **asthma** and allergic rhinitis and self reported traffic density on streets of residence in adolescents. Epidemiology 7:576–582 (1996).

51. U.S. National Library of Medicine, Medline Plus, "rheumatoid arthritis," http://www.nlm.nih.gov/medlineplus/ency/article/000431.htm.

52. U.S. National Library of Medicine, Medline Plus, "Chron's Disease," http://www.nlm.nih.gov/medline plus/ency/article/000249.htm#Symptoms.

53. [224]
Bonvallot, V., Baeza-Squiban, A., Baulig, A., Brulant, S., Boland, S., Muzeau, F., Barouki, R., & Marano, F.; Organic compounds from diesel exhaust particles elicit a proinflammatory response in human airway epithelial cells and induce cytochrome p. 450 1A1 expression; Am J Respir Crit Care Mol Biol 25, 515–521, 2001
Diesel exhaust particles (DEP) enhance inflammatory response in human bronchial epithelial cell cultures (16HBE). Effects of native DEP 9nDEP), organic extracts of DEP (OE-DEP), and

stripped DEP or carbonaceous particles (sDEP) and carbon black particles (CB) compared . OE-DEP and nDEP induce granulocyte macrophage colony-stimulating factor, whereas the carbonaceous core induces less intense effects. nDEP induces expression of CYP1A1, a cytochrome involved in PAH metabolism in in vitro airway epithelial cells. Detailed methodological description and results analysis. Data from 2 laboratories in Paris. Probably important observations.

54. Much attention in the medical research community is focused on identifying these proteins so they can be used in the treatment of cancer victims. One consequence of the chemical and radiation therapies used to treat cancers is a severe decline in white and other blood cells. If the proteins that induce production of granulocytes and macrophages can be isolated and produced, it can then be injected into patients in order to boost the effectiveness of their own immune systems. Some of these vaccines have successfully cured tumors in mice and are now being tested in human subjects. See—
Jaffee E.M., Hruban R.H., Biedrzycki B., Laheru D., Schepers K., Sauter P.R., Goemann M., Coleman J., Grochow L., Donehower R.C., Lillemoe K.D., O'Reilly S, Abrams R.A., Pardoll D.M., Cameron J.L., Yeo C.J.
Novel allogeneic granulocyte-macrophage colony-stimulating factor-secreting tumor vaccine for pancreatic cancer: a phase I trial of safety and immune activation.
J Clin Oncol. 2001 Jan 1;19(1):145–56
http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=11134207&dopt=Abstract

55. [359]
Juvin, P., Fournier, T., Boland, S., Soler, P., Marano, F., Desmonts, J-M., & Aubier, M.; Arch Environ Health 57; 53–60; 2002.
Cells from human lung epithelial cell line A-549 incubated with diesel exhaust particles or with inert particles. Phagocytosis studied with EM analysis and flow cytometry. Both kinds of particles engulfed by alveolar Type II cells, and diesel particles but not inert particles induced a dose and time dependent increase in granulocyte macrophage-colony-stimulating factor release and a transient inhibition of interleukin-8 release. Conclude that diesel exhaust exposure alters cytokine production – an effect not caused by inert particles (carbon black).

56. [245]
Hashimoto, K-I., Ishii, Y., Uchida, Y., Kimura, T., Masuyama, K., Morishma, Y., Hirano, K., Nomura, A., Sakamoto, T., Takano, H., Sagai, M., & Sekizawa, K.; Exposure to diesel exhaust exacerbates allergen-induced airway responses in guinea pigs; Am J Respir Crit Care Med 164, 1957–1963, 2001.
Guinea pigs exposed to air or diesel exhaust (3 mg/m^3) for 12 hr/day for 8 weeks with and without sensitization to ovalbumin. Results show that "exposure to diesel exhaust enhances mucus secretion and eosinophilic inflammation during the immediate airway response. Diesel exhaust exposure also increases airway permeability and airway inflammation during the late airway response." Results at lower exposures not described, so the lower level of effect is not known. Different components of diesel exhaust exposure not detailed in this paper, but may have bee3n described elsewhere. The paper is illustrated with excellent histologic illustrations of airway changes.

57. [246]
Vincent, R., Kumarathasan, P., Goegen, P., Bjarnson, S.G., Guenette, J., Berube, D., Adamson, I.Y., Desjardins, S., Burnett, R.T., Miller, F.J., & Battistini, B.; Inhalation Toxicology of Urban Particulate Matter: acute cardiovascular effects in rats; Research Report No. 104, Health Effects Institute, October 2001:p. 64. (New address: Charlestown Navy Yard, 120 Second Ave, Boston, MA 02129-4533, USA).
Exposures of rats to urban PM (resuspended Ottawa-derived PM_{10}) by inhalation for four hours to 48 mg/m^3; or water –leached particles (49 mg/m^3), or diesel soot (5 mg/m^3) or carbon black (5 mg/m^3). Observations by implanted telemetering devices. Lung cell labeling was low indicating an absence of acute lung injury. Urban PM_{10} caused an increase in blood pressure on day 2 after exposure, with elevations of plasma endothelin 3 at 2, 32, and 48 hours after exposure. Modified PM_{10} from which soluble components had been extracted did not affect blood pressure, but did affect the levels of endothelin 1, 2, & 3 at 2 hours after exposure. Exposure to diesel soot but not carbon black, caused an elevation of endothelin 3 at 36 hours after exposure, but did not affect the blood pressure. Methodology described in detail and all results very well illustrated. Critical statistical evaluation of significance of all differences. Excellent discussion of possible significance of the findings, emphasizing the observation that there is a vasopressor response in the rats without acute lung injury. Elevated endothelins have been shown to be associated with worsening in patients with congestive heart failure, so this induced change is important. Publication in this extended Report format allows a more detailed description of methods and results than would be the case in a submitted paper.

58. [798]
Yanagisawa, R., Takano, H., Inoue, K., Ichinose, T., Sadakane, K., Yoshino, S., Yamaki, K., Kumagai, Y., Uchiyama, K., Yoshikawa, T., & Morita, M.; Enhancement of acute lung injury related to bacterial endotoxin by components of diesel exhaust particles; Thorax 2003; 58; 605–612.
Intratracheal administration of different mixtures into mice. Results indicate that acute lung injury is caused by the carbonaceous fraction of diesel particles, rather than the extracted organic chemicals. Proinflammatory cytokines, chemokines, and Toll-like receptors are believed responsible for the induced effects. Results are well described and illustrated. Data from the National Institute for Environmental studies in Tsukuba in Japan.

59. [167]
Kleeman, M.J., Sshauer, J.J., & Cass, G.R.; Size and Composition Distribution of fine particulate matter emitted from motor vehicles; Environ Science & Technol: 34; 1132–1142; 2000.
Dilution source sampling system used to measure size-distributed chemical composition of fine particle emissions. It is impressive that from gasoline cars with and without catalytic converters, and from diesel engines, the size fraction is very constantly between 0.10 and 1.0 microns. Notes observations by Bagley et al (HEI Technical Report No 76 of 1996) that a 1991 diesel Cummins engine delivered lower overall particle mass emissions, but 15–35 times the number of particles as a 1988 Cummins engine (ultrafine particles made up the difference). For gasoline vehicles under various conditions, peaks of particle distributions seem to be between 0.1 and 0.2 microns, and the same is true of medium duty diesel vehicles. Sulphate particles seem to be a bit larger, peaking at about 0.9 microns. Interesting information.

60. [274]
Wahlin, P., Palmgren, F., & Van Dingenen, R.; Experimental studies of ultrafine particles in streets and the relationship to traffic; Atmospheric Environment 35 Supplement No. 1 (2001) S63–S69.
Notes that emissions of fine particles may have increased although the total mass of particles emitted from vehicles may have decreased. Data from Copenhagen and Odense with some measurements at street level and some on rooftops. 29 size fractions from 0.01 to 0.7 microns. Significant street level correlations between CO, NO_X, and ultrafine particles. Distribution of diesel particles thought to include smaller particles than those from petrol engines. Particle densities were up to 200,000 per cubic centimeter, with NO_X up to 170 ppb (324 micrograms/m^3) and CO up to about 2 ppm.

61. [59]
Berube, K.A., Jones, T.P., Williamson, B.J., Winters, C., Morgan, A.J., & Richards, R.J.; Physicochemical characterisation of diesel exhaust particles: factors for assessing biological activity; Atmospheric Environment 33 (1999) 1599–1614.
From Cardiff. Source was a 1985 Japanese ISEKI tractor burning Esso 2000 Diesel and a 20/30 mixture of Esso light engine oil. Operated at 2000 rpm. Details of methodology.
Define four basic shapes: 1. Spherulites (individual particles); 2. Chains or clusters of spherulites; 3. Spherules (large bodies of spherulites; 4. Flake-like bodies. Equivalent spherical diameter of spherulites was 0.23 microns; Distributions of particle size by number showed 10% were ultrafines; 89.5% were fine (0.1–2.0 microns) and 0.4% coarse (greater than 2.5 microns). But distribution by mass showed 0.01% ultrafine, 52.6% fine; and 47.4% coarse. Electron probe X-ray microanalysis showed presence of C,O,Na,Mg,K,Al,Si,P,S,Cl,and Ca along with a range of metals (Ti,Mn,Fe,Zn,&Cr). By analysis before and after sonication of particles in water, the mobile sorbed metals were Mg,P,Ca,Cr,Mn,Zn,Sr,Mo,Ba,Na,Fe,S, & Si.
Stress differences between sonicated and impacted diesel particles - these differences are likely to affect toxicity. Excellent pictures. A definitive article.

62. [4]
Lloyd, A.C., & Cackette, T.A.; Diesel Engines: Environmental Impact and Control; J Air & Waste Manage. Assoc 51; 809–847; 2001.
A Critical Review article that discusses all aspects of Diesel vehicles. Notes that between 1992 and 1997, in the US the Annual Vehicle Miles (in millions) by diesels increased in all categories; in vehicles less than 10,000 pounds, from about 20,000 to 30,000; and in vehicles 26,000 pounds and heavier from about 80,000 to about 117,000 (which would be a 46% increase). Health data well reviewed and emphasis is well balanced. Effect of high sulfur fuel noted and quantitated. No estimates of likely increases in personal exposure but notes importance of the question. Also deals with fuel leakage and spills of fuel.
A first class review in every way.

63. See, e.g., Diesel Technology Forum, “Demand for Diesels: The European Experience,” at http://www.dieselforum.org/whitepaper/downloads/EuropeanExperience.pdf.

64. [165]

Friedman, M.S., Powell, K.E., Hutwagner, L., Graham, L.M., & Teague, W.G.; Impact of changes in transportation and Commuting behaviors during the 1996 Summer Olympic Games in Atlanta on Air Quality and Childhood Asthma; JAMA 2001: 285; 897–905.
Comparison of the 17 days of the Olympic Games (July 19–Aug 4) to a baseline period consisting of the 4 weeks before and 4 weeks after the Olympic Games. Peak one hour level of O3 fell to 50–100 ppb during the games from a predicted value of about 70–120 in the comparison periods. PM_{10} (24 hour level) was 20–45, compared to levels of 30–70; NO_2 was only slightly lower running at about 30 ppb peak one hour level compared to values between 20–65. CO also slightly lower. Traffic density measurements showed decreases of 22 percent in weekday 1-hour morning peak traffic counts during the Olympic Games. Ozone levels fell slightly over the same period in three different places 60 km to 100 km from Atlanta; but these changes were only about one fifth of the drop in Atlanta. Citywide acute care visits and hospitalizations for asthma were logged. Results showed no changes in nonasthma diagnoses; decreases of 41 percent in Medicaid claims file, 44 percent decreases in HMO database; 11 percent decreases in two emergency pediatric departments; and decreases of 19 percent in Georgia Hospital Discharge Database. Lack of change in other diagnostic categories indicates that children did not leave Atlanta over the period of the Olympic Games.

65. [331]
Grosse, S.D., Matte, T.D., Sschwartz, J., & Jackson, R.J.; Economic gains resulting from the reduction in Children's exposure to Lead in the United States; Environ Health Perspectives 110;563–569; 2002.
Calculate improvements in worker productivity as a consequence of lowering the blood lead in children by 15 mug/dl which occurred between 1976 and 1999. Assumed change in cognitive function to be between 0.185 and 0.323 IQ points for each 1 mug/dl change in blood lead. Estimated economic benefit for each year's cohort of 3.8 million 2 year old children ranges from $110 billion to $319 billion.

66. "Curitiba, Brazilthree Decades of Thoughtful City Planning," http://www.dismantle.org/curitiba.htm

67. [1010]
Koop, G., & Tole, L.; Measuring the health effects of air pollution: to what extent can we really say that people are dying from bad air?; J Environmental Economics & Management 47 (2004) 30–54.
Data from Toronto from 1992–1997; six monitors for gases operational; for years 1992–1994, one particulate monitor at Bay/Wellesley; from 1996–1997, the only monitor was at Evans/Arnold streets, and for 1995 both monitors were available. Roughly one observation every three days, but 66.29 percent of the raw daily observations were missing. Bayesian Model Averaging method used for data analysis. 48 explanatory variables included. Note that if three lags are included with all possible variables, there would be 312 potential explanatory variables, and this number could be increased to 348 if different splines are used in the analysis. They express results for $PM_{2.5}$ in terms of one standard deviation, or per 8.75 micrograms/m^3 change in the variable. Authors conclude that point estimates are unreliable for policy decisions as the standard deviations are very large. But point estimates of the effect of numerous air pollutants on mortality all tend to be positive "albeit small". They also note that measures of uncertainty

associated with the point estimates become very large: “Indeed they become so large that the hypothesis that air pollution has no effect on mortality is not implausible”. Also: “We stress that our findings do not necessarily imply that air pollution does not have adverse health effects (or that air pollution abatement and regulatory policies should not take into account non-mortality related effects such as potential impacts for asthmatics and individuals with other respiratory illnesses).”

68. [929]
Liu, S. Krewski, D., Shi, Y., Chen, Y., & Burnett, R.T.; Association between gaseous ambient air pollutants and adverse pregnancy outcomes in Vancouver, Canada; Environmental Health Perspectives 111; 2003: 1773–1778.
Data from single live births in Vancouver from 1985 to 1998 analysed. Preterm low birth weight (LBW) and intrauterine growth retardation (IUGR) recorded as outcomes from Stats Canada registry. 13 census tracts in the greater Vancouver area with daily average and 1 hour concentrations of SO_2, NO_2, CO, and O_3. and for PM_{10} from 1994 to 1998. Mean daily averages for SO_2 were 4.9 ppb with 95^{th} percentile of 10.5; for NO_2 average was 19.4 ppb with 95^{th} percentile of 31.9 ppb; for CO was1.0 ppm,and 95^{th} percentile was 2.2 ppm; and for O_3, daily average was 13.4 ppb and 95^{th} percentile was 25.1 ppb.

LBW associated with SO_2 in first month of pregnancy with OR of 1.11 for a 5 ppb increase; Preterm birth associated with SO_2 (OR 1.09) for a 5 ppb increase, and to CO (OR 1.08) for a 1 ppm increase during the last month of pregnancy. IUGR was associated with SO_2 (OR 1.07) for a 5 ppb increase, and to NO_2 (OR 1.05) for a 10 ppb increase during the first month of pregnancy. Authors conclude that the relatively low pollution levels are associated with increased risks of the two outcomes.

69. [916]
Bero, L.; Implications of the tobacco industry documents for public health and policy; Annu. Rev. Public Health 2003: 24: 267–288.
Previously secret internal tobacco industry documents have now become available due to settlement agreements. “These documents show that the tobacco industry has been engaged in deceiving policy makers and the public for decades”. In a final section on “Corporate Interests and Public Health”, similar information on the asbestos industry, and the lead industry are documented. The author notes: “The historical similarities in behavior among the tobacco, asbestos, and lead industries indicate that public health researchers who are interested in the activities of chemical, pharmaceutical, or oil companies, for example, could learn much about how these industries operate by studying the internal tobacco industry documents”.
This is a detailed and compelling indictment.
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70. [3]
Hitchins, J., Morawska, L., Wolff, R., & Gilbert, D.; Concentrations of submicrometre particles from road emissions near a major road; Atmospheric Environment 34 (2000); 51–59.
Abstract only read. Data from Brisbane. When wind is blowing directly from the road, the concentration of fine and ultrafine particles decays to around half of the maximum (measured at

the closest point to the road) at a distance of approximately 100–150 meters from the road. For the wind blowing parallel to the road, the reduction to half the concentration occurs at 50–100 meters. PM2.5 levels decrease with distance to around 75 percent for wind from the road to 65 percent for wind parallel to the road at a distance of 375 meters.

71. Dann, T.; Ambient air concentrations of air in Canada (1989–1998), http://www.ccme.ca/assets/pdf/monitor_june27_99_e.pdf

72. [150]
Meneses, F., Romieu, I., Ramirez, M., Colome, S., Fung, K., Ashley, D., & Hernandez-Avila, M.; A survey of personal exposures to benzene in Mexico City; Arch Environ Health 1999; 54; 359–363.
45 volunteers measured during a workshift. Service station attendants were 359.5 micrograms/m^3; street vendors were 83.7; and office workers were 45.2 micrograms/m^3. The latter values were substantially higher than those reported by others for office workers elsewhere. Fuel used in Mexico City contains 1.5–2.0 percent of benzene. A few of the office workers were exposed to ETS. Vapor recovery program is being instituted in Mexico City.

73. Guthrie, J, Sabourin, R., Brunet, E.; Benzene in Canadian Gasoline: Report on the Effect of the Benzene in Gasoline Regulations 2002 .Environment Canada, http://www.ec.gc.ca/energ/fuels/reports/Benz_2002/BenzeneReport2002_e.cfm