1. The two principal oxides of nitrogen are nitric oxide (NO) and nitrogen dioxide (NO_2). Usually, NO is emitted from tailpipes or smokestacks, then transformed in the atmosphere into NO_2, which can be further converted to fine particle nitrates or gaseous nitric acids. However, in some engines (e.g., certain diesels), NO_2 is directly emitted. The nitrogen compound in which the ambient standard is expressed and for which monitors measure is NO_2. Nitric oxide and nitric dioxide are produced from molecular nitrogen in the gas phase through the Zeldovich mechanism, first proposed by Russian scientist Y. B. Zeldovich in 1947. The reactions account for much of the active nitrogen formed in hot exhaust gases from combustion sources and following the rapid heating of the air during lightning discharges. Zeldovich, Y. B., P. Y. Sadonikov, and D. A. Frank-Kamenetskii, 1947: Oxidation of nitrogen in combustion (M. Shelef, Transl.). *Acad. Sci. USSR,* Inst. Chem. Phys., Moscow Leningrad. American Meteorology Society, *Glossary of Metrology,* http://amsglossary.allenpress.com/glossary/search?id=active-nitrogen1

2. Roughly 32 percent of the total nitrogen entering the Chesapeake Bay, the nation's largest estuary, is from atmospheric sources, for example. Chesapeake Bay Program, "Bay Trends and Indicators," http://www.chesapeakebay.net/status.cfm?sid=126. Also, Lewis Linker, Robert Thomann, "The Cross-Media Models of the Chesapeake Bay: Defining the Boundaries of the Problem," http://www.epa.gov/owow/watershed/Proceed/linker.html

3. Nitrogen oxides ($NO_X = NO + NO_2$) do not directly affect Earth's radiative balance, but they catalyze tropospheric formation of ozone, which is a powerful greenhouse gas, through a sequence of reactions. United Nations Environment Program, Intergovernmental Panel on Climate Change, *Climate Change 2001:Working Group I: The Scientific Basis,* p. 4.2.3.3, http://www.grida.no/climate/ipcc_tar/wg1/141.htm.

4. Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, *Latest Findings on National Air Quality, 2001 Status and Trends*, Sep. 2002, p. 2, http://www.epa.gov/airtrends/reports.html.

5. Ward's Communications, *Ward's Motor Vehicle Facts and Figures 2001,* pp. 70-71, (Southfield, Mich., 2002).

6. Emissions in the year 2000 are estimated to have been about 32.6 terragrams of nitrogen, projected to be 110 in the year 2100. United Nations Environment Program, Intergovernmental Panel on Climate Change, *Climate Change 2001:Working Group I: The Scientific Basis*, p. 4.2.3.3, http://www.grida.no/climate/ipcc_tar/wg1/141.htm.

7. "Regional/Global Interactions in Atmospheric Chemistry," www-as.harvard.edu/chemistry/trop/presentations/powerpoints/djj2001/epa_oaqps.ppt

8. The most recent controlled exposure study [591], using a two-hour exposure with moderate exercise to just over 1000 micrograms/m^3 of NO_2 in healthy nonsmoking volunteers, showed that although this did not affect pulmonary function tests, it did cause changes in a variety of components of bronchoalveolar lavage, indicating that this concentration had resulted in significant effects at the level of the pulmonary endothelium, together with a mild inflammatory

response.

9. University of Birmingham, “Early Stages of Acute Inflammation,” http://medweb.bham.ac.uk/http/mod/3/1/a/acute.html.

10. [133]
SCHWARTZ, J.
Air Pollution and blood markers of cardiovascular risk
Environ Health Perspect 109 (suppl 3): 405–409; (2001)
Analysis of NHANES III data. Fibrinogen levels, platelet counts, and white blood cell (wbc) counts assessed. Regressions controlled for age, race, sex, body mass index, current smoking, and number of cigarettes/day. PM_{10} associated with all three outcomes; SO_2 associated with wbc counts; NO_2 with platelet counts and fibrinogen; and ozone exposure with nothing. Further analyses in two pollutant models indicated that PM_{10} was probably the dominant effector. Magnitude modest since there was a 13 microg/dl change in fibrinogen for an interquartile range of PM_{10}. Results lend support to the mortality studies.

11. University of Birmingham, “Early Stages of Acute Inflammation,” http://medweb.bham.ac.uk/http/mod/3/1/a/acute.html.

12. Barck C., Sandstrom T., Lundahl J., Hallden G., Svartengren M., Strand V., Rak S., Bylin G., "Ambient level of NO2 augments the inflammatory response to inhaled allergen in asthmatics" *Am. J. Respir. Crit. Care Med.*, 159:3, 760-767 (1999).
Thirteen subjects with mild asthma and allergy were exposed at rest to either purified air or 500 microg x m 3 NO2 for 30 min, followed 4 h later by an allergen inhalation challenge. The exposures (NO2 or air) were performed in random order and at least 4 weeks apart. Lung function during NO2/air exposure and allergen challenge was measured by plethysmography, and then hourly by portable spirometry after exposures. Subjective symptoms were recorded during and after exposure. Bronchoscopy with bronchial wash (BW) and bronchoalveolar lavage (BAL) was performed 19 h after allergen challenge. NO2+allergen enhanced the percentage of neutrophils in both BW and BAL compared to air+allergen (BW 19 vs. 11, P=0.05; BAL 3 vs. 1, P=0.02 median values).The levels of eosinophil cationic protein (ECP) in BW was higher after NO2+allergen compared to air+allergen (90 vs. 3.6 microg/l; P=0.02, median values). There was no NO2-associated effect on symptoms or pulmonary function. These data suggest that ambient NO2 can enhance allergic inflammatory reaction in the airways without causing symptoms or pulmonary dysfunction.

13. D.E. Gardner, “Oxidant-induced Enhanced Sensitivity to Infection in Animal Models and their Extrapolations to Man,” *Journal of Toxicology and Environmental Health* 13 (1984): 423–39; D.E. Gardner, et al., “Influence of Exposure Mode on the Toxicity of NO_2,” *Environmental Health Perspectives* 30 (1979): 23–29.

14. R.M. Rose, et al., “The Pathophysiology of Enhanced Susceptibility to Murine Cytomegalovirus Respiratory Infection During Short-term Exposure to 5 PPM Nitrogen Dioxide,” *American Review of Respiratory Disease* 137 (1988): 912–17.

15. M.J. Evans, et al., "Renewal of the Terminal Bronchiolar Epithelium in the Rat Following Exposure to NO_2 or O_3," *Laboratory Investigation.* 35 (1976): 246–57.

16. W. Harrington, et al., "Short-term Nitrogen Dioxide Exposure and Acute Respiratory Disease in Children.," *Journal of the Air Pollution Control Association* 35 (1985): 1061–67.

17. V. Hasselblad, et al., "Synthesis of Environmental Evidence: Nitrogen Dioxide Epidemiology Studies," *Journal of the Air Waste Management Association* 42 (1992): 662–71.

18. L.M. Neas, et al., "Association of Indoor Nitrogen Dioxide with Respiratory Symptoms and Pulmonary Function in Children," *American Journal of Epidemiol*ogy 135 (1991): 204–9.

19. A. Richters and V. Richters, "Nitrogen Dioxide: Inhalation, Formation of Microthrombi in Lungs and Cancer Metastasis," *Journal of Environmental Pathology, Toxicology, and Oncology* 9 (1989): 45–51. This is but one of a series of studies conducted by these researchers either together or with others. In another published study, Richter A. and Kuraitis K. exposed animals to air containing 0.40 +/- 0.05 ppm or 0.80 +/- 0.05 ppm of NO2. After the appropriate exposure periods, the animals were infused intravenously with B16 mouse melanoma cells. At 3 wk post-infusion the animals were killed and the lungs were examined for melanoma nodule development. The lungs of the NO2-exposed animals contained a significantly higher number of melanoma nodules than the lungs of control animals (P less than .0025). They concluded that the results "indicate that inhalation of ambient or near ambient levels of NO2 influences the metastasis of blood-borne cancer cells. This raises the possibility that similar events may occur in the human population." Arch Environ Health. 1981 Jan-Feb;36(1):36-9. Other studies by these researchers, members of the faculty of the School of Pathology at the University of Southern California, can be found at PubMed, http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?CMD=Display&DB=PubMed.

20. A. Richters and V. Richters, "Nitrogen Dioxide: Inhalation, Formation of Microthrombi in Lungs and Cancer Metastasis," *Journal of Environmental Pathology, Toxicology, and Oncology* 9 (1989): 45–51. This is but one of a series of studies conducted by these researchers either together or with others. In another published study, Richter A. and Kuraitis K. exposed animals to air containing 0.40 +/- 0.05 ppm or 0.80 +/- 0.05 ppm of NO2. After the appropriate exposure periods, the animals were infused intravenously with B16 mouse melanoma cells. At 3 wk post-infusion the animals were killed and the lungs were examined for melanoma nodule development. The lungs of the NO2-exposed animals contained a significantly higher number of melanoma nodules than the lungs of control animals (P less than .0025). They concluded that the results "indicate that inhalation of ambient or near ambient levels of NO2 influences the metastasis of blood-borne cancer cells. This raises the possibility that similar events may occur in the human population." Arch Environ Health. 1981 Jan-Feb;36(1):36-9. Other studies by these researchers, members of the faculty of the School of Pathology at the University of Southern California, can be found at PubMed, http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?CMD=Display&DB=PubMed.

21. [64]
INFANTE-RIVARD, C.

Childhood asthma and Indoor Environmental Risk factors
Am J Epidemiol 1993; 137; 834–44
Case-control study conducted in Montreal between 1988 and 1990 of three to four-year-old children diagnosed with asthma by a pediatrician. 457 cases of asthma recruited at a hospital emergency room. Similar number of controls chosen from family allowance files and matched with case children on age and census tract. A census tract is a small geostatistical unit including a mean of about 4,000 persons with maximum economic and social homogeneity. Telephone interviews conducted. Twenty percent feasibility sample chosen to wear an NO_2 badge during a 24-hour period. Independent risk factors for asthma were: mother's heavy smoking, use of humidifier in child's room; electric heating system in the home; ETS not quite significant. History of pneumonia, absence of breast feeding, and family history of asthma were all significant risk factors. Dose-response relation between NO_2 (in ppb) and asthma.

NO_2 comparisons:

Level	Cases (%)	Controls (%)
0 ppb	24.5	39.2
> 0.5 to < 10	18.0	43.0
> 10 to < 15	13.1	10.1
> 15 ppb	44.2	7.5
Gas Cooking	6.6	5.2
Humidifier in child's room	67.6	55.8
Pets	43.7	43.5

Analysis well described. Well referenced and discussion is interesting.

22. [466]
ZHANG, J., HU, W., WEI, F., WU, G., KORN, L.R., & CHAPMAN, R.S.
Children's respiratory morbidity prevalence in relation to air pollution in four Chinese cities
Environmental Health Perpsectives110; 961–967, 2002. Questionnaires used, and $PM_{2.5}$ and PM_{10} known, together with SO_2 and NO_2. Positive associations between morbidity prevalence and outdoor levels of PM of all size fractions, with the strongest being for PM_{10}–$PM_{2.5}$ fraction. Ambient levels of NO_2 and SO_2 were also associated with an increased prevalence of respiratory symptoms. Arithmetic means for four years for NO_2 varied from 38 micrograms/m^3 to a high of 230 in one urban area. PM_{10} values varied from a low of 81 to a high of 237 micrograms/m^3. Highest asthma prevalence was 4.2 percent and most were between 1.4 percent and 3.3 percent. Parental smoking levels were high.

23. [209]
BRAUN-FAHRLANDER, C., ACKERMANN-LIEBRICH, U., SCHWARTZ, J., GNEHM, H.P., RUTISHAUSER, M., & WANNER, H.U.
Air pollution and respiratory symptoms in preschool children
Am Rev Respir Dis 145, 42–47, 1992

Random sample of 625 Swiss children aged 0–5 years. Two cities in Switzerland; 1800 children from Basel and Zurich, a suburban community of Wetzikon, and a rural area Rafzerfeld in the canton of Zurich. Random sample drawn, and 625 included from Basel and Zurich. Participation spread throughout the year.

Typical Annual Mean Concentrations of Pollutants

	SO_2		NO2		O3*	TSP
	ug/m3	ppm^	ug/m3	ppm	hrs	ug/m3
Rural	8–12	.004	20–30	.025	600–800	35–40
Subs	30–40	.01	30–50	.04	350–450	40–45
Cities	50–70	.024	60–140	.10	80–150	50–55
AQS	30	.012	30	.03	1 hr 120 ug/m3	70
USNAAQS	80	.032	100	.10	1 hr 235 ug/m3	75

^ = of mean value

* = hrs above 120 ug/m3 April-September

Passive samplers measured NO_2 during a six-week period when the child was on a daily diary; these were used both inside and outside the home. Twenty percent of diaries were validated by comparison with pediatrician's notes. NO_2 measured outdoors but not indoors was associated with the duration of respiratory symptoms. TSP was a more significant predictor of duration of any symptom than NO_2. Six-week average TSP was associated with incidence of coughing episodes, and marginally significant as a predictor of upper respiratory episodes, and previous days' TSP was a significant predictor of the incidence of upper respiratory symptoms. Annual average NO_2 was associated with the duration of any episode and of upper respiratory episodes. Authors conclude that "incidence and duration of respiratory episodes are likely associated with particulate concentrations and duration may be associated with NO_2."

Questionnaire data (Percentage figures):

	Basel (%)	Zurich (%)
Asthma	1.4	2.0
Bronchitis	28.2	25.1
Tonsillitis	16.4	19.1
Otitis media	23.5	28.6
Pneumonia	4.2	0.5
Croup	11.0	12.6

189 under the age of 2; 237 from 2–5 years.
Upper respiratory symptoms had linear relationship to quartiles of TSP up to 100 micrograms/m^3.
Good discussion and references.

1–2 percent increase in clinic visits for 10 percent increase in NO_2

24. [261]
HWANG, J-S., & CHAN, C-C.
Effects of air pollution on daily clinic visits for lower respiratory tract illness
Am J Epidemiol 155, 1–10, 2002
From Taipei Institute of Statistical Science. Data from clinic visits in 50 townships and city districts aggregated so that daily rates of lower respiratory disease could be calculated for each area. "Monitoring Station Numbers" shown on a map, but it is not clear whether these indicate actual numbers of monitors. Statistical methods described in detail (two staged Bayesian approach) and appear to be sophisticated. Fifty different areas analysed; percent increase in clinic visits plotted against NO_2 lag 0 and lag 1. Most of the 95 percent confidence limits above 1, and average was about 1–2 percent increase per 10 percent increase in NO_2 level. Regressions most significant for people over 65, but data for children and adults aged 15–64 also significant. PM_{10}, SO_2, and CO also showed significant regression coefficients. Note that a community's annual PM_{10} level influenced the regression values. Very large database and excellent analytical methods used.

25. [102]
MEDINA, S., LE TETRE, A., QUENEL, P., LE MOULLEC, Y., LAMELOISE, P., GUZZO, J.C., FESTY, B., FERRY, R., & DAB, W.
Air pollution and doctor's house calls: results from the ERPURS system for monitoring the effects of air pollution on public health in Greater Paris, France, 1991–1995
Environmental Research 75, 73–84, 1997
Increase from 5^{th} to 95^{th} percentile in Black Smoke (7–51 microgram/m3) led to an RR of 1.32 for house calls. Relationship stronger for asthma visits in children (0–14). RR's for 24 hour SO_2 and NO_2 were in the same range. RR was 1.63 for ozone and myocardial conditions. Asthma visits and ozone showed an interaction with minimum temperature as an effect was only seen when temperature was 10°C or higher. In two pollutant models, only black smoke and ozone effects remained stable. House calls for asthma varied between 28/day and 0/day over the 1500 days of the study. Seasonal pattern removed by smoothing as shown by flat plot of residuals. Interesting data.
ERPURS project is "Evaluation des Risques de la Pollution Urbaine pour la Sante."

26. [305]
HAJAT, S., ANDERSON, H.R., ATKINSON, R.W., & HAINES, A.
Effects of air pollution on general practitioner consultations for upper respiratory diseases in London
Occup Environ Med 2002: 59: 294–299Non-parametric methods of analysis of time-series data, adjusting for seasonal factors, day of the week, holiday effects, influenza, weather, pollen concentrations and serial correlation. Data collected from London practices between January 1992 and December 1994. Two-day lag showed greatest effects. 10^{th}-90^{th} percentile change in SO_2 (13–

31 micrograms/m^3) resulted in 3.5 percent increase in childhood consultations. For the 10^{th}–90^{th} percentile change (16–47 micrograms/m^3) in PM_{10} in adults aged 15–64, there was a 5.7 percent increase, and in adults over 65 years, there was a 10.2 percent increase. Effects of NO_2 closely followed the effects for PM_{10}. Authors point out that although the effects are relatively small, the impact on demand for services could be considerable.

27. [50, 317]
YE, F., PIVER, W.T., ANDO, M., & PORTIER, C.J.
Effects of temperature and air pollutants on cardiovascular and respiratory diseases for males and females older than 65 years of age in Tokyo, July and August 1980–1995
Environmental Health Perspectives 109; 355–359 (2001)

28. [485]
BATES, D.V., BAKER-ANDERSON, M., & SIZTO, R.
Asthma attack periodicity: a study of hospital emergency visits in Vancouver
Environmental Research 51; 51–70; 1990
All visits to the Emergency Departments of nine hospitals serving just under 1 million people in Greater Vancouver, were logged from July 1^{st} 1984 to October 31^{st} 1996. There were about 25,000 visits a month in all age groups; 2.7 percent were for respiratory conditions and of these, 41.3 percent were for asthma. Data from 11 continuously operating monitoring stations also tabulated on a daily basis, giving mean hourly values for SO_2, NO_2, and O_3. Daily aerosol sulphate recorded at one station. Principal findings were:

1. There was a peak in asthma visits affecting children and the 15–60 age group but not those over 60, which occurred in the third week of September each year, causing at least a doubling of weekly visits for a three-week period. This was not related to temperature changes or to peaks in air pollutants. No cause could be identified.
2. In summer, asthma visits in the 15–60 age group are associated with SO_2 and SO_4 levels.
3. In winter, all respiratory visits but not asthma alone, are correlated with SO_2 levels on the same day, and lagged 24 hours and 48 hours.
4. In the 61+ age group, NO_2 levels were related to respiratory visits on the same day, and lagged 24 and 48 hours in the winter; asthma visits were also related to SO_2.
5. Respiratory visits were unrelated to temperature changes, but varied seasonally.
6. The correlation coefficients between SO_2 and NO_2 levels were 0.67 in May–October, and 0.61 in November–April.

29. [50, 317]
YE, F., PIVER, W.T., ANDO, M., & PORTIER, C.J.
Effects of temperature and air pollutants on cardiovascular and respiratory diseases for males and females older than 65 years of age in Tokyo, July and August 1980–1995
Environmental Health Perspect 109; 355–359; (2001)

30. [111]
PETERS, A., LIU, E., VERRIER, R.L., SCHWARTZ, J., GOLD, D.R., MITTELMAN, M., BALIFF, J., OH, J.A., ALLEN, G., MONAHAN, K., & DOCKERY, D.W.
Air Pollution and incidence of cardiac arrhythmia
Epidemiology 2000: 11: 11–17

Patients with implanted cardioverter defibrillators. 100 cases in Eastern Massachusetts. Pollution indices of particulate matter, black carbon, and gaseous air pollutants measured daily 1995–1997. A 26 ppb increase in NO_2 was associated with increased defibrillator interventions two days later (OR = 1.8: CI 1.1–2.9).

Patients with ten or more interventions experienced increased arrythmias in association with NO_2, CO, Black carbon, and fine particle mass. Levels of pollutants are given. The 95 percentile values were: PM_{10} = 37; $PM_{2.5}$ = 26.6: Black carbon = 2.84; CO = 0.97 ppm; O_3 = 0.036 ppm: SO_2 = 0.019 ppm; NO_2 = 0.037 ppm. Maximal values were about twice these, and the mean values were about half. Strongest associations were for NO_2. Two pollutant models with CO and black carbon suggested that NO_2 was dominant. Odds ratios appeared to be linearly related to pollutant level.

Hospital admissions for heart attack, as well as fibrinogen.

31. [184]
PEKKANEN, J., BRUNNER, E.J., ANDERSON, H.R., TIITANEN, P., & ATKINSON, R.W.
Daily concentrations of air pollution and plasma fibrinogen in London
Occup Environ Med 2000; 57; 818–822
4982 male and 2223 female office workers had blood samples collected in a cross sectional survey between September 1991 and May 1993. The fibrinogen content was determined, and the association of level with mean concentrations of air pollutants during the day of blood sampling and during the preceding three days was assessed.

After adjustment for weather "an increase in the 24 hour mean NO_2 during the previous day from the 10^{th} to the 90^{th} percentile (61.7 micrograms/m^3) was associated with a 1.5 percent higher fibrinogen concentration. Also associated with CO. Associations stronger in warm season (April to September), when PM_{10} and BS also became significantly associated. No association with SO_2 or ozone. NO_2 and CO also associated with 2.8 percent and 3.6 percent increase in hospital admissions for myocardial infarction (see Occup Environ Med 1997; 54; 535-540). Interesting data.

angina, heart attack

[50, 317]
YE, F., PIVER, W.T., ANDO, M., & PORTIER, C.J.
Effects of temperature and air pollutants on cardiovascular and respiratory diseases for males and females older than 65 years of age in Tokyo, July and August 1980–1995
Environmental Health Perspectives 109; 355–359 (2001)

32. [560]KUO, H.W., LAI, J.S., LEE, M.C., TAI, R.C., & LEE, M.C.
Respiratory effects of air pollutants among asthmatics in Central Taiwan
Arch Environ Health 57; 2002: 194–200
12,926 subjects studied from eight junior high schools. Data on monthly hospital admissions for respiratory illness collected from the National insurance bureau for one year. Twenty percent of the subjects had lung function tests. The prevalence rates for asthma were correlated significantly with NO_2 ® = 0.63) and with ozone ® = 0.51). Levels of NO_2 and PM_{10} were correlated

significantly with monthly hospital admissions. A deficit of 6–11 percent in the lung function tests was also recorded. Notes that asthma prevalence has increased significantly in Taiwan, rising from 1.34 percent to 5.82 percent between 1974 and 1990. Logistic analysis shown as follows:

Pollutant	Level	Adjusted OR for Asthma	95% CI
NO_2	<0.023ppm >0.023ppm	1 1.692	 1.15–2.48
SO_2	<0.005 ppm >0.005 ppm	1 0.558	 0.674–2127
O_3	<23 ppb > 23ppb	1 1.308	 0.318–1.769
PM_{10}	<65.9 micrograms/m^3 >65.9 micrograms/m^3	1 0.837	 0.640–1.401

Interesting and well-informed discussion.

33. [107]
WANG, T-N., KO, Y-C., CHAO, Y-Y., HUANG, C-C., & LIN, R-S.
Association between indoor and outdoor air pollution and adolescent asthma from 1995 to 1996 in Taiwan
Environ Research Section A 81; 239–247; 1999
Survey of 165,173 high school students aged 11–16 in two communities in Taiwan. ISAAC questionnaire used. Asthma defined as positive answer to any of the questions 1 to 5 in that questionnaire in the video program. Kaohsiung City and County had some more heavily polluted regions, and some cleaner. Annual means of CO (0.60 to 1.49 ppm), NO_2 (0.001 to 0.034 ppm), PM_{10} (19.4 to 112.81 micrograms/m^3), SO_2 (0 to 0.023 ppm) and O_3 (0.002 to 0.031 ppm). TSP varied from 112 to 237 micrograms/m^3 as annual mean.

Correction for ETS conducted. Prevalence slightly higher if ETS reported. Asthma prevalence varied around 13 percent. In case of PM_{10} for example, prevalence was 15.3 with values below 80, and 14.6 with values above. In the case of SO_2, value was 14.46 in areas with SO_2 less than 0.03 ppm, and 15.08 in areas where it was above this. NO_2 below 0.028 prevalence was 13.47, and if above this was 15.23. Asthma prevalence also varied with CO and with TSP (which was a better indicator than PM_{10}). Authors conclude: “We observed a statistically significant association between outdoor air pollution and asthma, after controlling for potential confound variables.” Models with TSP, NO_2, CO, O_3, and airborne dust were all significant, whereas PM_{10} and SO_2 were not. Note that asthma prevalence was increased by as much as 29 percent by the major outdoor pollutants.

34. [64]
INFANTE-RIVARD, C.
Childhood asthma and Indoor Environmental Risk factors
Am J Epidemiol, 1993; 137; 834–844
Case-control study conducted in Montreal between 1988 and 1990 of 3–4 year old children diagnosed with asthma by a pediatrician. 457 cases of asthma recruited at a hospital emergency room. Similar numbers of controls chosen from family allowance files and matched with case

children on age and census tract. A census tract is a small geostatistical unit including a mean of about 4,000 persons with maximum economic and social homogeneity. Telephone interviews conducted. 20 percent feasibility sample chosen to wear an NO_2 badge during a 24-hour period. Independent risk factors for asthma were: mother's heavy smoking, use of humidifier in child's room; electric heating system in the home; ETS not quite significant. History of pneumonia, absence of breast feeding, and family history of asthma were all significant risk factors. Dose-response relation between NO_2 (in ppb) and asthma.

NO_2 comparisons:

Level	Cases (%)	Controls (%)
0 ppb	24.5	39.2
> 0.5 to > 10	18.0	43.0
> 10 to < 15	13.1	10.1
> 15 ppb	44.2	7.5
Gas Cooking	6.6	5.2
Humidifier in child's room	67.6	55.8
Pets	43.7	43.5

Analysis well described. Well referenced and discussion is interesting.

35. [22]
SMITH, B.J., NITSCHKE, M., PILOTTO, L.S., RUFFIN, R.E., PISANIELLO, D.L., & WILSON, K.J.
Health Effects of daily indoor nitrogen dioxide exposure in people with asthma
Eur Respir J 2000; 16; 879–885
125 self reported asthmatics of all ages from < 14 years to > 50 years, over a 42- day period wore lapel badges at home and recorded seven different symptoms in a diary. Outdoor pollutant levels, spores and weather variables were also recorded during the study. Data from South Australia. GEE methodology used for analysis. 76 percent of males and 81 percent of females lived in houses with gas appliances. Of participants over the age of 14, 28 percent of men and 24 percent of women were cigarette smokers. Mean time +/- SD for self reported badge exposure was 4.5 hours/day (+/- 2.4). Personal time weighted average levels of NO_2 exposure ranged from 0–1,760 ppb. Range of median indoor NO_2 levels for each individual was 3.70–146.66 ppb. Within subjects interquartile ranged from 0.60–153.50 ppb.

Results showed that the following interactions were significant:
In < 14 year age group only:
1. NO_2 and chest tightness with one day lag (OR=1.29):
2. NO_2 and breathlessness on exertion with one day lag (OR=1.13);
3. NO_2 and daytime asthma attacks on same day (OR=1.13);
4. NO_2 and night asthma attacks on same day and with a one day lag (OR =1.15);

For age group 35–49 years:
5. NO_2 was associated with coughs with a one day lag (OR =1.15).
Authors conclude that "Daily personal exposures to NO_2 are associated with asthmatic symptoms in children". Useful details of method of distributing and collecting badges.

36. [58]
ATKINSON, R.W., ANDERSON, H.R., STRACHAN, D.P., BLAND, J.M., BREMNER, S.A., & DE LEON, P.
Short-term associations between outdoor air pollution and visits to accident and emergency departments in London for respiratory complaints
Eur Respir J 1999, 13, 257–265
Points out that ER visits include less severe cases, and are also unrestricted by bed availability. 12 London departments used; data from 1992–1994. Poisson regressions used for seasonal patterns, meteorological conditions, and influenza epidemics. NO_2, O_3, SO_2, CO, and particles as Black Smoke (BS) and PM_{10}. "Strong associations between visits for all respiratory complaints and increases in SO_2; 2.8 percent increase in number of visits for a 18 microgram increases, and a 3.0 percent increase for a 31 microgram/m^3 increase in PM_{10}. Also significant associations between asthma and SO_2, NO_2 and PM_{10}. No associations with O_3. Authors conclude there is a linkage. Map of hospital locations given. Daily visits hover between about 50 and 150; asthma visits are between 10 & 30, half of which are for age group 0–14. SO_2 level mean was 21.2 micrograms/m^3 for a 24-hour average (8.05 ppb). Unexpectedly strong association between asthma visits for children and NO_2, this being very strong for the warm season. Detailed tabulated data presented. Emphasize that the sample was larger than in most studies; that the complaint used in the study was that stated by the subject on presentation and did not represent the clinical diagnosis after assessment in the ER; also note that "The associations with NO_2 and SO_2 in children presenting with asthma are particularly strong".

Authors conclude that SO_2 associations for asthma are surrogates for associations with fine sulphate particles are incorrect. Note that CO might have been associated with cardiovascular events in the elderly as the majority of visits by them were for the nonspecific complaint of difficulty in breathing" (68 percent).

NOTE: The strong associations with asthma would seem to "correct" the Anderson conclusion on hospital admissions in earlier paper in Thorax.

37. [95]
HAJAT, S., HAINES, A., GOUBET, S.A., ATKINSON, R.W., & ANDERSON, H.R.
Association of air pollution with daily GP consultations for asthma and other lower respiratory conditions in London
Thorax 54; 597–605; 1999
Time series analysis. Between 268,718 and 295,740 registered patients in London using 45–47 practices contributing to the General Practice Research database during 1992–94. Associations found with NO_2, CO, and PM_{10} for asthma in children. In summer, for a 10^{th}–90^{th} percentile increase in NO_2 lagged by one day, asthma consultations increased 13.2 percent with NO_2, 11.4 percent with CO, and 9.0 percent with SO_2. In winter for lower respiratory disease, these percentages became NO_2, 7.2 percent; CO, 6.2 percent; and SO_2, 5.8 percent. Negative

associations with ozone noted.

In adults, only consistent association was with PM_{10} (increase of 9.2 percent).

Detailed air pollution data for different seasons. Correlation between SO_2 and NO_2 about 0.6 year round, and about the same between PM_{10} and SO_2. Correlation coefficient between PM_{10} and NO_2 was 0.73 all year; 0.78 in summer; and 0.69 in winter. Significant associations also noted for pollutants and consultations for patients over the age of 65. In summer for NO_2, the increase was 20 percent for asthma for the percentile gap. Note that results are likely to be underestimates.

Important data.

38. [552]
STIEB, D.M., JUDEK, S., & BURNETT, R.T.
Meta-analysis of time-series studies of air pollution and mortality: effects of gases and particles and the influence of cause of death, age, and season
J Air Waste Management Association 52; 470–484, 2002
Results from 109 studies from around the world analysed and single and multi-pollutant models with causes of death, age and season taken into account. Results showed an overall 2.0 percent change in all cause mortality followed a 31.3 microgram/m^3 increase in PM_{10}. The percentages were 1.7 percent for 1.1 ppm of CO; 2.8 percent per 24 ppb of NO_2; 1.6 percent per 31.2 ppb of O_3; and 0.9 percent per 9.4 ppb of SO_2, using the daily maximum value for ozone, but the 24-hour average for then other pollutants. Larger effect sizes were seen for respiratory mortality for all pollutants except O_3. Authors conclude: “This synthesis leaves little doubt that acute air pollution exposure is a significant contributor to mortality.” Extended description of statistical methodology used, and reasons for the selection of particular studies in relation to different pollutants are well described. Excellent figures of the percent excess in all cause mortality in different studies. 175 references.

39. [9]
FAIRLEY, D.
Daily mortality and air pollution in Santa Clara County, California: 1989–1996
Environmental Health Perspectives 1999: 107; 637–41
Data for 1989–1996.
Wood burning and ammonium nitrate each contribute about 40 percent of SCC’s wintertime $PM_{2.5}$. Particle size varies by season, with $PM_{2.5}$ being 70 percent of PM_{10} during the winter, but 50 percent for the year as a whole. Samet modeling strategy followed. List of correlations between pollutants given; CO and $PM_{2.5}$ was 0.435. NO_2 was higher at 0.662, and NO_3 was highest at 0.830. Results similar to those of earlier analysis for 1980–1986 data.

$PM_{2.5}$ significant as was CO when lagged. NO_3 also significant. Respiratory risks generally higher than for cardiovascular disease. No evidence of a threshold. Author concludes that current national air quality standards are not protective. Relative risks for $PM_{2.5}$ present in all seasons of the year, but NO_3 highest in summer.

40. [520]

VEDAL, S., BRAUER, M., WHITE, R., & PETKAU, J.
Air Pollution and daily mortality in a city with low levels of pollution
Environ Health Perspect 111: 45–51 (2003)
Analysis of data from Vancouver, Canada from January 1994 to December 1996.
Temporal patterns removed from the data at each of the available monitoring sites using time and cosine functions. Respiratory deaths defined as all deaths coded with ICD-9 codes of 460–519; and cardiovascular deaths as deaths with ICD-9 codes of 390–459.

50^{th} and 90^{th} percentiles of PM_{10} were 13 and 23 micrograms/m^3; for one hour maximal ozone they were 27 and 39 ppb. Seasonal analyses conducted, with summer ranging from May through September, and winter from October to April. Analyses avoided the recent difficulties with the GAM model. NO_2 levels for 50 percent and 90 percent percentiles were 16.1 and 22.9 ppb. Intercorrelations between pollutants are shown and were: SO_2 and NO_2 summer 0.80; PM_{10} and NO_2 (summer) 0.84; PM_{10} and SO_2 (winter) 0.78; NO_2 and CO (summer) 0.81; charts show spikes in NO_2 and SO_2 similar to those documented by Bates et al in 1984.

Charts show the relationships between five pollutants, total mortality, respiratory, and cardiovascular mortality in the two different seasons. Authors conclude in the Abstract: "The dominant associations were between ozone and total mortality and respiratory and cardiovascular mortality in the summer, and between NO_2 and total mortality in the winter, although some associations with PM_{10} may also have been present. We conclude that increases in low concentrations of air pollution are associated with increased daily mortality." They also raise the possibility that there may be some other factors present in the air pollution-meteorology mix rather than effects of the pollutants themselves, but do not suggest what these might be.

41. [67]
BREMNER, S.A., ANDERSON, H.R., ATKINSON, R.W., McMICHAEL, A.J., STRACHAN, D.P., BLAND, & BOWER, J.S.
Short term associations between outdoor air pollution and mortality in London 1992–1994
Occup Environ Med 1999; 56; 237–44
This analysis includes PM_{10} and CO. "No significant associations were found between any pollutant and all cause mortality, but, with the exception of ozone, all estimates were positive." PM_{10} had the largest effect on respiratory mortality (4 percent increase in deaths of all ages for a 10^{th}–90^{th} percentile increment). NO_2, O_3 and black smoke were associated with cardiovascular deaths, but there was no evidence of a PM_{10} association. SO_2 effect very close to significance. Notes that the effects of ozone in the earlier study were not replicated. Pollution variables were interesting: brackets indicate 10^{th}–90th percentiles and max value)
$N0_2$ 24 hour mean 33.7 ppb (22.3–46.3; 133.7)
NO_2 1 hour mean 50.3 ppb (34.3–70.3; 224.3)
0_3 8 hr mean 17.5 (4.4–30.1; 79.9)
O_3 1 hr. ppb mean 22.6 (6.0–36.5; 98.5)
SO_2 24 hr. uG/m^3 mean 21.2 (13.0–31.0; 82.2)
CO 24 hr. ppm mean 0.8 (0.5–1.3; 5.6)
PM_{10} 24 hr. uG/m^3 mean 28.5; (15.8–46.5; 99.8)
BS 24 hr uG/m^3 mean 12.7; (5.5–21.6; 69.8)

pneumonia mortality in the elderly associated with NO_X.

42. [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, 1990–1995
Arch Environ Health 56; 327–35,2001
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 microgram/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/m^3) was associated with a 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.

43. [130]
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; 413–19
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.

44. [282]
HONG, Y-C., LEE, J-T., KIM, H., HA, E-H., SCHWARTZ, J., & CHRISTIANI, D.C.
Effects of air pollutants on acute stroke mortality
Environ Health Perspectives 110; 187–91 (2002)
Data from Seoul, Korea over a 4-year period. 20 automated sampling stations. Daily 24-hour mean values calculated of all pollutants, but 8-hour average used for ozone. Time-series with generalized additive model. Increase of 1.5 percent in stroke mortality associated with PM_{10} interquartile range; also increase of 2.9 percent associated with increase in ozone concentrations on the same day. Associations also with NO_2 (3.1 percent), SO_2 (2.9 percent), and CO (4.1 percent) for each interquartile range of pollutant. Analysis showed that the pollutants were interactive with respect to their effects on stroke mortality. Stronger associations for PM_{10} in the elderly and in women than in other groups. Occurrence of “harvesting” considered possible.

45. [375]
SUNYER, J., BASAGANA, X., BELMONTE, J., & ANTO, J.M.
Effect of nitrogen dioxide and ozone on the risk of dying in patients with severe asthma
Thorax 2002: 57; 687–93
Patients over the age of 14 who died during the period 1985–1995 in Barcelona who had visited the emergency department of one of the four largest hospitals in the city; for asthma during 1985–1989. Total of 467 men and 611 women. “Air pollution was measured at the city monitoring stations which provide a mean for the entire city.” Daily values of PM_{10}—appears to have a mean value of 61.2 micrograms/m^3; Black smoke 40.0; 1-hour NO_2, 89.7 micrograms/m^3; 24- hour NO_2, 52.3 micrograms/m^3; 1-hour ozone, 69.3 micrograms/m^3; 8 hour ozone, 54.4 micrograms/m^3; SO_2 18.8 micrograms/m^3; Pollen and spores recorded weekly and measured by ‘the Cour method.’ Thought to be accurate for pollen grains, but fungal spores get damaged and are underestimates.

For cases with more than one admission always for asthma, the odds ratio for interquartile change was as follows:

NO_2, 1.688 (1.074 to 2.652): Ozone, 1.755 (0.984 to 3.133) with 95 percent confidence limits in brackets. These values were not much changed by correction for total pollen and for spore counts. “Patients with severe asthma—that is, those with more than one admission to the emergency department for an asthma exacerbation—had a higher risk of dying on days with higher levels of NO_2 regardless of the season, and O_3 in the warm season. These associations were not confounded by the weekly levels of pollen and fungal spores.”

46. [379]
MOSQUERON, L., MOMAS, I., & LE MOULLEC, Y.

Personal exposure of Paris office workers to nitrogen dioxide and fine particles
Occup Environ Med 2002: 59: 550–56
62 office workers all nonsmokers equipped with passive samplers for NO_2 (48 hours) and active pumps for 24 hours of $PM_{2.5}$. Simultaneous measurements in homes and offices. Time activity diaries were completed by all. Results show that personal $PM_{2.5}$ was higher than in home and ambient concentrations. Personal exposure to NO_2 (43.6 micrograms/m^3) was significantly higher than in home concentrations (35.1) but lower than the background outdoor level (60.1). Personal exposures to both were not significantly different from office levels. In home $PM_{2.5}$ affected by exposure to in-home tobacco smoke, and NO_2 in home concentration was affected moistly by the ambient level and gas cooking time.

47. [62]
EBELT, S., BRAUER, M., CYRYS, J., TUCH, T., KREYLING, W.G., WICHMANN, H-E., & HEINRICH, J.
Air quality in postunification Erfurt, East Germany: associating changes in pollutant concentrations with changes in emissions.
Environ Health Perspect 109: 325–33 (2001)
Airborne particle size distribution and gaseous pollutants followed through the 1990's when sharp reductions in emissions were occurring. Decreases (19–91 percent) noted in all pollutants except particles in the 0.01 to 0.03 size range. These were the smallest particles being measured. Number concentrations of these increased by 115 percent between 1991 and 1998 possibly due to the increase in diesel emissions, together with the reduction in large particles due to coal burning which might have been scavenging the smaller ones. SO_2 much reduced because open coal burning decreased. Morning particle peaks of all sizes were associated with NO and CO in both the 1991 and 1998 periods. Morning hour peaks in ultrafines were related to the increase in traffic.

Erfurt has population of 201,100. Wintertime inversions occur. Measurements made at Institute of Hygiene site 1 km south of the city center. Details of technical methods to measure particles in different size ranges. Trucks went from 2.4 percent of total fleet in 1991, to 8.7 percent in 1998, and diesel trucks and buses more than trebled in number (from 44,236 in 1991 to 125,315 in 1998). Close relationship between NO and CO and particles less than 2.5 microns shown.

48. [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; 406–10
Data collected from 29 randomly selected drivers between 27 January and 27 March 1997. CO measured over eight-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 micrograms/m^3
NO_X	139	131	43 micrograms/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.

49. [719]
RIEDIKER, M., WILLIAMS, R., DEVLIN, R., GRIGGS, T., & BROMBERG, P.
Exposure to particulate matter, volatile organic compounds, and other air pollutants inside patrol cars
Environ Sci Technol 2003, 37, 2084–93
25 work shifts in the autumn using two cars and a GPS system. Average pollutant levels were generally low compared to ambient air quality standards. CO was 2.7 ppm; NO_2 was 41.7 micrograms/m^3; O_3 was 11.7 ppb; $PM_{2.5}$ was 24 micrograms/m^3. VOC levels inside the cars were in the ppb range and they showed the imprint of gasoline. $PM_{2.5}$ was 24 percent lower than ambient and roadside levels. Authors conclude that combustion engine emissions from other vehicles were an important source of air pollutants inside the cars. Note that others have found $PM_{2.5}$ levels in vehicles higher by a factor of 2 than ambient levels (ref Adams, H.S et al: Sci Total Environ 2001; 279; 29–44). Good description of methodology used, and very useful discussion of the findings. Mean highest CO recorded was 5.9 ppm. Mean NO_2 in vehicles was 41.7 ppb, but the range was considerable—from1.6 to an outlier of 548 ppb.

50. [132]
RIJNDERS, E., JANSSEN, N.A.H., VAN VLIET, P.H.N., & BRUNEKREEF, B.
Personal and outdoor nitrogen dioxide concentrations in relation to degree of urbanization and traffic density
Environ Health Perspect 109 (suppl 3): 411–17; (2001)
Personal and home outdoor NO_2 measured in 241 children from six different primary schools in the Netherlands. Weekly average measurements in four different seasons. Exposure to traffic varied. For children living near highways, personal and outdoor NO_2 concentrations significantly decreased with increasing distance of the home from the highway. Traffic density and inner urban living also significant. "Because NO_2 can be considered a marker for air pollution from traffic, the more easily measured variables, degree of urbanization, traffic density, and distance to a nearby highway can all be used to estimate exposure to traffic-related air pollution." Classroom mean data for NO_2 shows values in very urban district twice as high as in nonurban. 50 percent higher NO_2 values in very urban compared to nonurban in outdoors school values. Very thorough study.

[217]
PALIN, L.A., BINOTTI, M., BONA, G., & PANELLA, M.
Letter to the Editor: Occupational & Environmental Medicine:
Comment on Linaker et al Occup Environ Med 2000; 57; 472–76
Quote observations on 310 children aged 5–14 in Novara a small town in northwest Italy using Palmes tubes for five days a week in each season of the year. Only factor related to levels

observed (mean of 6200 measurements was 42.7 micrograms/m^3) living along busy streets. Critical of only one measurement station of ambient concentration as used by Linaker et al.

[308]
ALILI, F., MOMAS, I., CALLAIS, F., LE MOUELLEC, Y., SACRE, C., CHIRON, M., & FLORI, J-P
Exposure to traffic pollution: comparison between measurements and a model
Archives Environmental Health 56; 552–58, 2001
27 Paris (canyon street) sites used. Model calculations based on traffic density and emissions compared to NO_2 measured with passive samplers over a six-week period. Highly significant correlation ®=0.83) between the two. Mean concentrations not significantly different. Authors used the OSPM model to calculate the concentrations based on partitioning the street into two sections by wind direction. Emissions calculated from overall traffic density data, which was measured during the period of the study by positioned vehicle counters. Mean NO_2 values were about 82–225 micrograms/m^3.

disagreement between ambient and personal monitors

[211]
GAUVIN, S., LE MOULLEC, Y., BREMONT, F., MOMAS, I., BALDUCCI, F., CIOGNARD, F., POILVE, M-P., ZMIROU, D., & VESTA INVESTIGATORS
Relationships between nitrogen dioxide personal exposure and ambient air monitoring measurements among children in three French Metropolitan areas: VESTA Study
Arch Environ Health 56; 336–41, 2001
Comparison between 48 hours of personal exposure to NO_2 (measured by a modified Palmes tube) and ambient measurements at the same time. This was poor in all three cities (Grenoble, Toulouse, and Paris). But when other ambient or indoor sources were taken into account, the r^2 values increased to 0.43, 0.50, and 0.37. The main variables taken into account were an index of traffic intensity and proximity, and use of a gas cooker at home. ETS explained 42.7 percent of personal exposures in Paris but not in the other two cities.

51. D. Brook Harper and Brian McConkey, “Environmental McCarthyism and the Precautionary Principle - Learning from the Past While Addressing Current Dilemmas,” in *2001: Sustaining the Global Farm*, D.E. Stott, R.H. Mohtar and G.C. Steinhardt (eds.), http://topsoil.nserl.purdue.edu/nserlweb/isco99/pdf/ISCOdisc/SustainingTheGlobalFarm/P220-Harker.pdf.

52. Chauncey Starr, “The Precautionary Principle Versus Risk Analysis,” *Risk Analysis* Vol. 23, No. 1, 2003, http://www.epri.com/attachments/290782_Starr_RiskArticle.pdf+precautionary+principle++%22Chauncey+Starr%22&hl=en&ie=UTF-8.

53. U.S. Environmental Protection Agency, “EPA announces next step to improve the New Source Review program,” August 27, 2003, http://www.epa.gov/newsroom/headline2_082703.htm.

54. Eric Pianin, “EPA Eases Clean Air Rule on Power Plants,” *The Washington Post*, A1, Aug. 28, 2003.

55. [596]
ROSNER, D., & MARKOWITZ, G.
Industry challenges to the Principle of prevention in Public Health: the Precautionary Principle in Historical perspective
Public Health Reports 117; 501–11; 2002
Authors are from the Center for the History & Ethics of Public Health, Columbia University and the CUNY Graduate Center, New York. Note that the Business Roundtable founded in 1972 as an association representing 200 of the U.S. largest corporations, vigorously opposed the Kyoto protocol, arguing that no policy commitments should be made until the economic consequences are thoroughly understood, and equating Precaution with economic and social stagnation.

This history outlines the century of struggle preceding the modern arguments over the principle of precaution, in terms of protecting public health. The analysis involves lead, silica, and vinyl chloride. Excellent history—note that a series of advertisements in National Geographic, Saturday Evening Post, and other magazines in the 1920’s extolled the use of lead in modern American life. Similar reviews of silica and vinyl chloride, documenting the fact that the hazards were well known to industry which however took exceptional steps to prevent the scientific facts from being known. Notes the role of apologists such as Edith Efron and Elizabeth Whelan who wrote that the arguments against a variety of chemicals only represented the views of the “extreme environmentalist movement” and had needlessly terrorized the public into believing that chemicals were unduly hazardous. The latter author called for “Americans to recognize the severity of the gap between science and popular public thought, and the dramatically unpleasant side effects that a continuing embracing of environmental alarmism will have for our country.” Notes that the American Council on Science and Health was mostly supported by industry, particularly those that had fought against a vinyl chloride standard.

This agency has denied the relationship between asbestos, agent orange, DDT, lead, and chemical food additives and environmental disease. The article ends with redefining the role of public health protection in the face of this dismal history.

69 references documenting the sources of the many quotations.

56. [228]
KRIEBEL, D., TICKNER, J., EPSTEIN, P., LEMONS, J., LEVINS, R., LOECHLER, E.L., QUINN, M., RUDEL, R., SCHETTLER, T., & STOTO, M.
The Precautionary Principle in Environmental Science
Environmental Health Perspectives 109, 871–76, 2001
Authors are from University of Massachusetts, USA. Notes four principle components of the Precautionary Principle:
1. taking preventive action in the face of uncertainty;
2 shifting the burden of proof to the proponents of an activity;
3. exploring a range of alternatives to possibly harmful actions; and,
4. increasing public participation in decision-making.

The 1998 consensus statement [1] characterized the principle as follows: “When an activity raises threats of harm to human health or the environment, precautionary measures should be taken even if some cause and effect relationships are not fully established scientifically.” It is a translation of a German term: “Vorsorgeprinzip.”

Discussion of Historical links, Motivating factors, and points of Opposition. Case illustrations include cellular phones; pesticides in schools; and polyvinyl chloride toys. Also discussion of Limitations of Conventional Scientific methods, under the headings of Hypothesis formulation; emphasis on independent effects, and not interactions; a narrow definition of uncertainty; setting Type 1 and Type II error rates; Type III errors (when an accurate answer is provided to the wrong problem); and Disciplinary Divisions. Under the heading Scientific Methods to inform Precautionary Policy, the authors note: “It is never easy to determine the moment in this process when there is sufficient evidence to act as if a causal connection exists, but scientists can and should play an important role in this decision, as they are the ones who know the data and the methods best.” This section has notes on “What is studied” and “Research Methods.”

The Discussion ends with Conclusions and Recommendations, and the linkage between science and policy is described under seven headings, noting that: “The Precautionary Principle, then, is meant to ensure that the public good is represented in all decisions made under scientific uncertainty. When there is substantial scientific uncertainty about the risk and benefits of a proposed activity, policy decisions should be made in a way that errs on the side of caution with respect to the environment and the health of the public.”

A very useful and well-written discussion.

57. [643]
STARR, C.
The Precautionary Principle versus Risk Analysis
Risk Analysis 23; 2003; 1–3
Notes strengths and weaknesses of adopting the Precautionary Principle as a basis for decision-making. States that no such verifiable principle exists; “It is a rhetorical statement that provides government a public welfare masquerade for an indefinite deferment of a long-term policy response, or allows the deferment of disclosure of near-term actions motivated by political pressures.” Instead, recommends a “comparative benefit/cost/risk analysis,” but notes “such early risk analyses have pragmatic uncertainties based on the limited available knowledge base and, accordingly, require judgmental application.” Interesting analogy to the Belmont 2002 horse race. Author is President of EPRI.

58. [112]
SLOVIC, P.
Trust, Emotion, Sex, Politics, and Science: Surveying the Risk-Assessment Battlefield
Risk Analysis 19: 689–701: 1999
Quotes:

> “Risk Assessment is inherently subjective and represents a blending of science and judgment with important psychological, social, cultural, and political factors”

> " The limitations of risk science, the importance and difficulty of maintaining trust, and the complex, sociopolitical nature of risk point to the need for a new approach—one that focuses upon introducing more public participation into both risk assessment and risk decision-making in order to make a decision process more democratic, improve the relevance and quality of technical analysis, and increase the legitimacy and public acceptance of the resulting decisions."

Summarizes recent studies of risk assessment by different groups, and points out that white males have a consistently lower estimate of risk than white females, black males, or black females who are all about the same across a wide spectrum of risks. These range from a low for Commercial air travel to a high of cigarette smoking. Air quality comes about in the middle. Very good discussion. Notes that research has shown that the public incorporates many factors into its risk equation.

"Danger is real, but risk is socially constructed.."

56 references.

59. David Rosner and Gerald Markowitz, "A "Gift of God"?: The Public Health Controversy Over Leaded Gasoline During the 1920's," *American Journal of Public Health*, pp. 121–25, Vol. 75, No. 4 (April, 1985).

60. DuPont preferred to use the name "Ethyl"—it disguised the additive's reliance on lead as its active ingredient. Lead, a silvery grey metal known to every child who's ever weighted a fishing line, is toxic in every known form and of utterly no nutritional value. Lead's lethal nature had been known for centuries—it was known by the early Greeks and Romans to cause madness, dementia and death, and is thought by some reputable scientists to have caused the decline and fall of the Roman Empire. See Clair Patterson, "An Alternative Perspective—Lead Pollution in the Human Environment: Origin, Extent, and Significance," in *Lead in the Human Environment*, National Academy of Sciences (Washington, D.C., 1980).

61. Rosner and Markowitz, p. 350. Well before the poisonings at Bayway, word had leaked to the public health community that massive quantities of a compound containing lead might soon be marketed by industry giants General Motors, Standard Oil and DuPont, leading the Surgeon General of the United States to write Pierre du Pont on December 20, 1922 for further information. Thomas Midgely, the DuPont-General Motors engineer who had developed the additive dismissed their concerns. "The average congested street will probably be so free from lead that it will be impossible to detect it," he wrote. In response to another letter, Midgely wrote "poisoning is almost impossible ... the exhaust does not contain enough lead to worry about, but no one knows what legislation might come into existence fostered by competition and fanatical health cranks." Rosner and Markowitz at pp. 99–101.

62. Richard L. CANFIELD RL, HENDERSON CR Jr., M.A., Deborah A., CORY-SLECHTA DA, COX C, JUSKO BS & LANPHEAR BP. Intellectual Impairment in Children with Blood Lead Concentrations below 10 µg per Deciliter NEngJMed 348;1517–26, 2003
Measured blood lead concentrations in 172 children at 6, 12, 18, 24, 36, 48, and 60 months of age

and administered the Stanford–Binet Intelligence Scale at the ages of 3 and 5 years. The relation between IQ and blood lead concentration was estimated with the use of linear and nonlinear mixed models, with adjustment for maternal IQ, quality of the home environment, and other potential confounders.

Results: Blood lead concentration was inversely and significantly associated with IQ. In the linear model, each increase of 10 µg per deciliter in the lifetime average blood lead concentration was associated with a 4.6-point decrease in IQ (P=0.004), whereas for the subsample of 101 children whose maximal lead concentrations remained below 10 µg per deciliter, the change in IQ associated with a given change in lead concentration was greater. When estimated in a nonlinear model with the full sample, IQ declined by 7.4 points as lifetime average blood lead concentrations increased from 1 to 10 µg per deciliter.

Conclusions: Blood lead concentrations, even those below 10 µg per deciliter, are inversely associated with children's IQ scores at three and five years of age, and associated declines in IQ are greater at these concentrations than at higher concentrations. These findings suggest that more U.S. children may be adversely affected by environmental lead than previously estimated.

63. [764]
FLORET, N., MAUNY, F., CHALLIER, B., ARVEUX, P., CAHN, J-Y., & VIEL, J-F.
Dioxin emissions from a solid waste incinerator and risk of non-Hodgkin Lymphoma
Epidemiology 14; 392–98, 2003
Data from Besancon in France where there is an incinerator that processed 67,000 metric tons of waste in 1998. Measurements of exhaust gas found levels of 16.3 ng international toxic equivalency factor (1-TEQ/m^3) whereas the European guide value for this is 0.1 ng 1-TEQ/m^3. Exposure to dioxin modeled and average dioxin exposures varied in different locations by a factor of 16 (<0.001 pg/m^3 to >0.004 pg/m^3 to a high of 0.0016 pg/m^3). 222 incident cases of non-Hodgkin's lymphoma compared to over 2000 controls. Odds ratio of highest exposure category was 2.3. Conclusion supports finding from Seveso of an increased risk of non-Hodgkin's lymphoma in that exposed population. NOTE: units of "pg" not explained in the paper as far as I could find.