First Things First

Figure 1 This is a picture of the Earth's air. Nitrogen and oxygen, make up 99 percent of the air. The other one percent contains pollutants that make people sick, kill forests and lakes and cause global warming.

WHAT THE HECK IS AIR POLLUTION ANYWAY?!

It is a waste—gasoline or coal that's incompletely burned, heat that is set loose in the air instead of being used, or poisons like lead or mercury that shouldn't have been there in the first place.

SO WHY SHOULD WE WORRY?

Because it kills us—not just us, but trees and lakes, forests and streams, kids and grandparents alike. If it is not poisonous enough to kill us outright, it hurts us—kids miss school—which means their parents must stay home from work and lose money. People must visit doctors, emergency rooms, and stay in hospitals.

If there's not enough to cause immediate sickness, it changes you in many smaller ways—lungs start to look like those of people who smoke, arteries harden, blood pressure increases. Some pollutants are so poisonous that they cause children to be less intelligent, make trouble at school and home—even kill people.

As hard as it is to believe, pollution even makes the world hotter—and if gets too hot, it may be impossible for all of us to live. Millions, even billions of people could be killed by forest fires, floods, diseases and simply the heat itself.

WHERE IS ALL THIS POLLUTION?

The pollution is everywhere—in schools and school buses, at home, in factories and hospitals, on desert islands, the North and South Poles, and even mountaintops. There is so much pollution and it moves around so easily that avoiding it is

Figure 2 Air pollution is everywhere, including New York City....
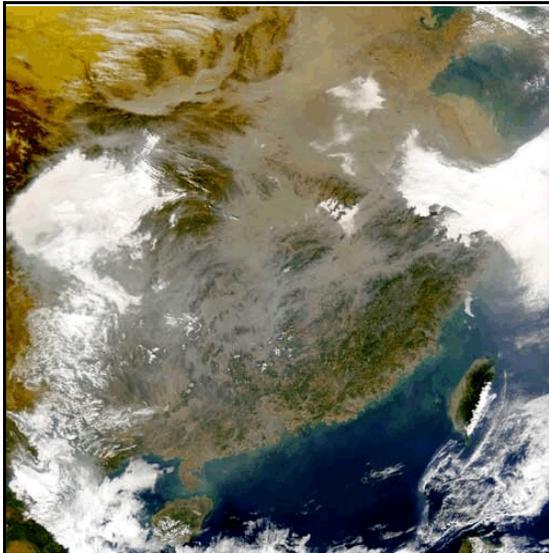
Figure 3 China, seen from a satellite.

impossible. Every living thing on Earth—human beings, plants and animals—must breathe, and when they do pollution enters the body. Some pollutants will burn holes in your lungs. Some pass through the lungs, into blood, hardening arteries and causing stroke and heart attack. Some lands on the nerves that transmit the sense of smell to the brain and move through them into the brain itself. This can happen within seconds in some cases, within minutes in others, but some injuries last for an entire lifetime, or end life altogether.

ARE THERE SOLUTIONS?

Yes, hundreds, even thousands of them. And since we can't very well stop breathing, and neither can trees or animals, the only way to protect ourselves is to stop the pollution from ever being created.

IS THIS ABOUT STOPPING POLLUTION?

Yes. This will explain what air pollution is, where it comes from, how it hurts us, and how to stop it. It will tell you about ways to make electricity for our homes, schools, offices and stores without any pollution, show pictures of cars and trucks that can us take us places with zero air pollution, and ways to live without making pollution.

## WHAT ARE THE AIR POLLUTANTS AND WHERE DO THEY COME FROM?

There are three sources of air pollution:

- The air we breathe, which transformed by heat into pollutants.
- Heat, whether from burning fuels or from sunlight.
- The thing that is burned, such as gasoline or coal, together with their impurities.
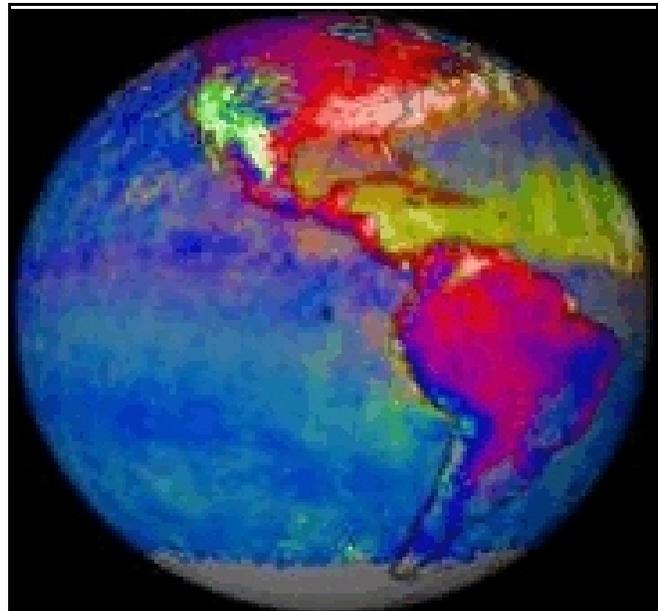
Figure 4 North and South America, shown in red.

AIR POLLUTION STARTS IN THE AIR
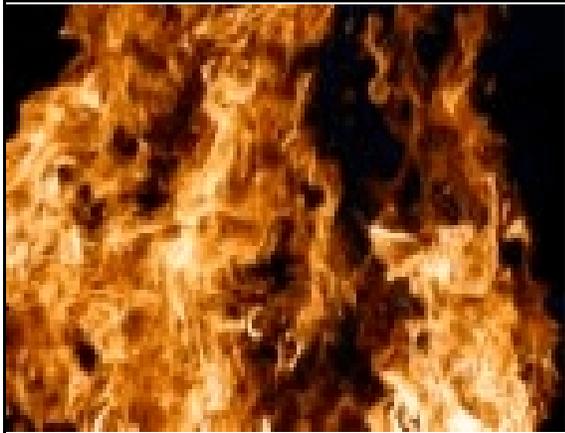
Figure 5 Burning, whether in car engines, fireplaces or factories creates the "shape shifting" air pollutant, oxides of nitrogen or NO_X. It causes asthma, sicknesses, acid rain, global warming and more—much, much more.

About 20 percent of the air is oxygen. We breathe that to stay alive. About 80 percent is nitrogen, which we also breathe. Even though we do not need the nitrogen to stay alive, it does not hurt us.

Flames transform

As long as temperature is normal, the oxygen and nitrogen stay apart. But when something is burned, like gasoline in a car, trees during a forest fire, coal at a factory—even something as simple as a candle—it causes oxygen and nitrogen to combine with one another, creating poisonous gases called oxides of nitrogen or NO_X. Also, some things that are burned—coal, for example—contain nitrogen, so they also create poisonous oxides of nitrogen, or NO_X, as it is usually called.

Scientists would not expose human beings to levels of NO_X that hurt people on purpose, but to understand how it works, they make animals in the laboratory breathe it. When these rats, mice and other animals breathe oxides of nitrogen, they catch more colds and other infections and die sooner than they should.[1, 2] In animals that breathe NO_X for several weeks, their lungs change and start to act like those of people who have emphysema.[3] This is a disease that makes it so hard to breathe that people often must carry oxygen containers and walk with canes, but even then can barely go across a room.

Sickness from NO_X

Children in the sixth grade or younger who breathe NO_X get more colds, cough a lot and wheeze—a whistling sound when they breathe in and out.[4] Because oxides of nitrogen are caused by burning something, places with kerosene heaters or where people cook on gas stoves can have high levels. Children who live in NO_X-polluted homes have difficulty breathing, cough a lot and get sick more often.[5]

NO_X, the shape-shifter

If NO_X stayed the same, that would be bad enough. But it doesn't. NO_X is a shape-shifter. It changes, again and again.
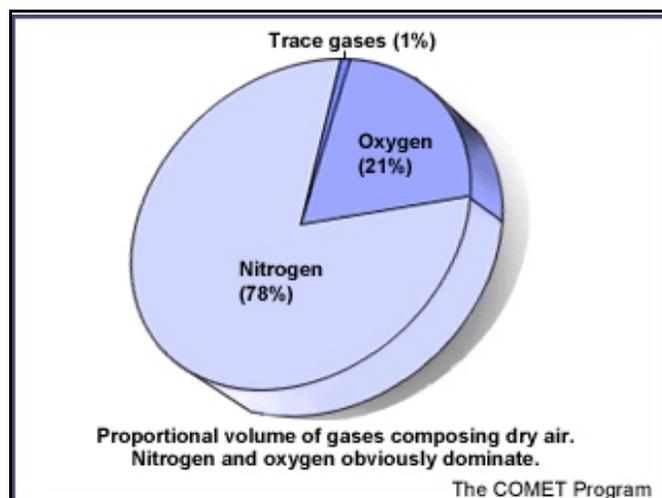
Figure 6 Oxygen and nitrogen in the air usually stay apart, but the heat from a fire—like in a car's engine or a factory burning coal—makes them combine, creating oxides of nitrogen, or NO_X, poisonous gases.

First, it turns into acids, then microscopically fine particles and finally ozone, or smog. By the time this chain of reactions halts, NO_X and its progeny have injured and killed humans, devastated forests and lakes, artificially enhanced plant growth—and subsequent death—in bays and sounds,[6] destroyed stratospheric ozone, and warmed the Earth.[7] NO_X is truly, in the words of Dr. Bert Brunekreef of Utrecht University, "The gas that won't go away." For this, humanity is vastly worse off—and matters are getting worse.

As illness caused by NO_X rises, children and their parents increase visits to the first line of defense in the medical community, general practice doctors;[8] hospital visits for asthma, bronchitis, pneumonia, angina—that is the chest pain that men and women with heart problems feel—and heart attack all increase.[9] So do visits to hospital emergency rooms by those aged 61 or older.[10]

New cases of asthma

One of the most frightening developments in the past twenty or so years has been the global rise in asthma among children. It is described as like trying to breathe through a straw, and some studies show that oxides of nitrogen can cause asthma.

Two studies were in Taiwan, where asthma rates have more than tripled since 1973. The scientists found that as NO_X levels increased there—and they were only about one-third of those in Los Angeles—so did asthma rates.[11, 12]   A third study, this one of 3- and 4-year-old children in Montreal, Canada showed that if mothers were heavy smokers or if NO_X increased, the children had higher rates of asthma.[13]

Once people have developed asthma, NO_X triggers both day and night asthma attacks, as well as other illnesses, such as chest tightness and breathlessness.[14] In London at a dozen different hospitals, when NO_X went up, so did asthma visits by children.[15] Different researchers examined the records of nearly 300,000 children, and again, when NO_X levels rose so did asthma visits—up 13.2 percent in summer and in winter, for emphysema, bronchitis and other lower respiratory disease, visits jumped 27.2 percent.[16]

Figure 7 Somewhat like this single-celled amoeba, NOx is a shape-shifter, changing from gas to particle to acid, and reacting to form ozone, or smog, damaging lungs, increasing deaths and poisoning lakes, streams, bays, all while causing global warming.

The cascade of injuries continues, bringing deaths. In London, when levels of NO_2 rose, so, too, did heart-related deaths, as well as death from pneumonia in the older people.[17] This was also true in Rouen and Le Havre, France: when NO_2 increased, there was a 6.1 percent jump in heart-related deaths,[18] and the same was true in Korea where some died of heart failure[19] and others from stroke.[20]

The next steps: fine particles and acids

As the nitrogen pollution continues to cook in the air, it turns into extremely small solid and liquid particles. Some are only 50 billionths of a meter—about 800 would fit on the width of a human hair—vastly too small to be seen even with an optical microscope.[21]

In its two new forms, nitrogen continues to leave immense damage in its wake. Nitric acid kills fish in lakes and streams, mleaving them so acid that they can no longer support populations of aquatic life. In soils, it poisons root systems.[22] Whether the NO_X-formed particles are themselves fatal is unclear.

Why asthma makes it hard to breathe

Air enters the respiratory system from the nose and mouth and travels through the bronchial tubes.

In an asthmatic person, the muscles of the bronchial tubes tighten and thicken, and the air passages become inflamed and mucus-filled, making it difficult for air to move.

In a non-asthmatic person, the muscles around the bronchial tubes are relaxed and the tissue thin, allowing for easy airflow.

Inflamed bronchial tube of an asthmatic

Normal bronchial tube

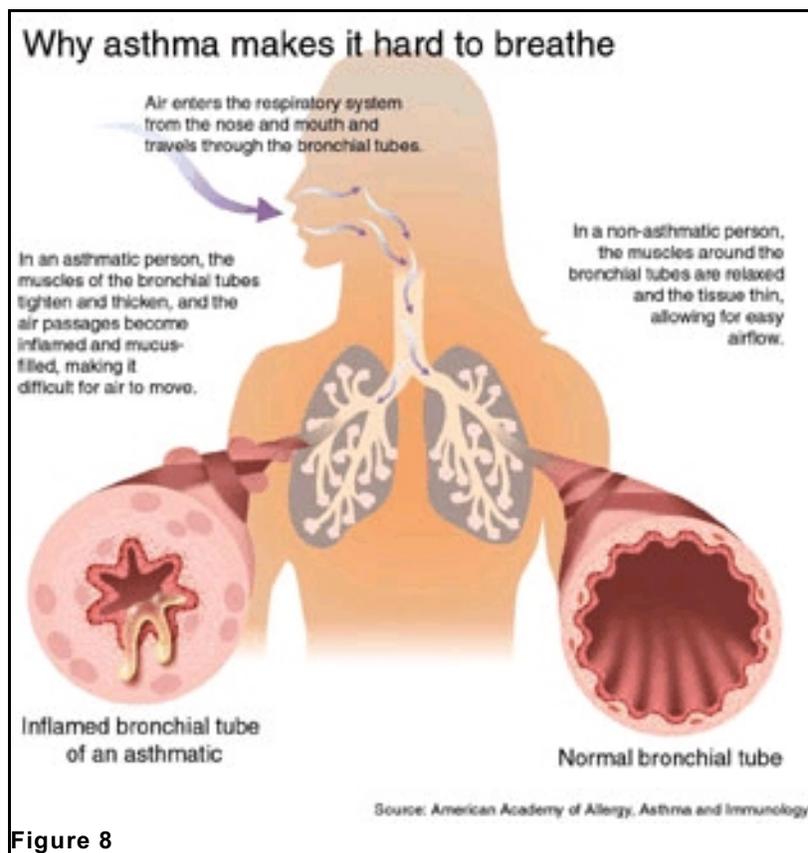Source: American Academy of Allergy, Asthma and Immunology

Figure 8

Then another pollutant, ozone (or smog) forms

Even now, NO_X is not finished with its dirty work. With sunlight providing energy, it reacts with gasoline fumes, gas from landfills, and other so-called hydrocarbons—some of these come from trees and plants—to form one of the most pervasive and harmful of all air pollutants, smog, often called ozone. It will be discussed later, but it is important to bear in mind that smog requires three ingredients: sunlight and hydrocarbons, both of which occur naturally; and NO_X, which is almost completely a result of human pollution (though some is formed by lighting bolts).

Since it is impossible to eliminate sunlight, and a pretty bad idea to cut down all the trees and plants, there is only one other practical solution left: eliminate oxides of nitrogen. Later, ways to do this will be discussed. Sadly, however, although NO_X can be reduced fairly cheaply and easily, that isn't happening.

Polluters like to *claim* that air pollution, including oxides of nitrogen and smog, are being reduced, but that is simply untrue, as the following chart shows.
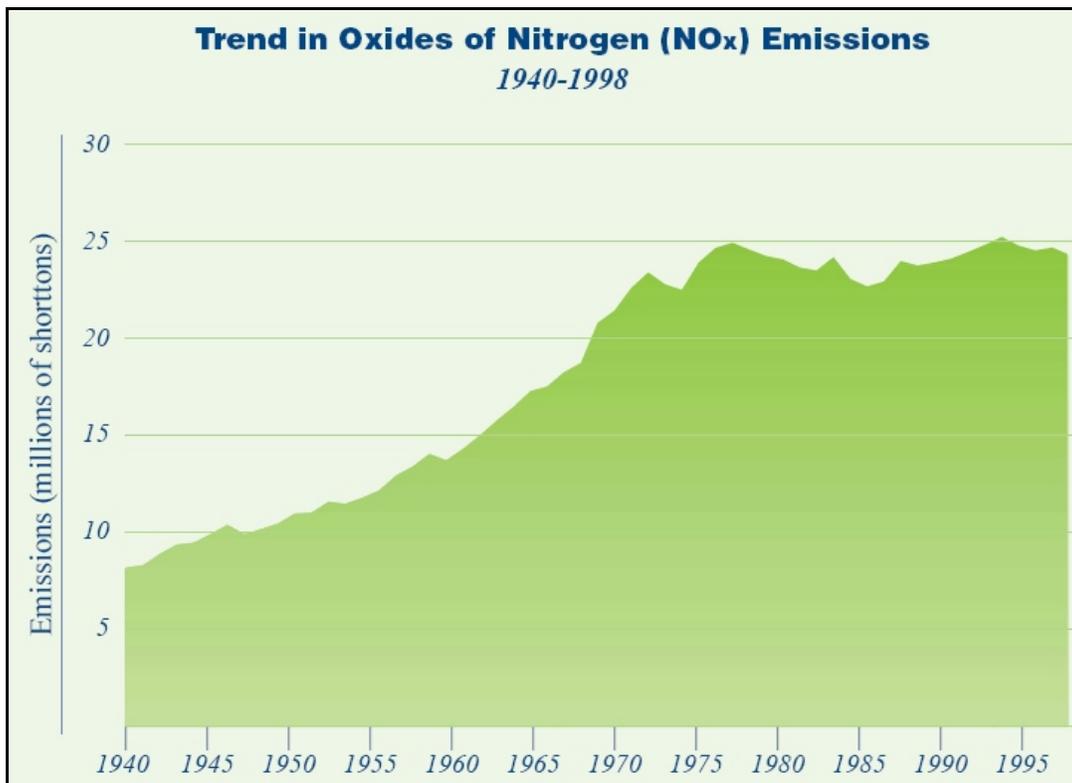
Figure 9 After increasing steadily until 1975, NO$_X$ emissions have stubbornly remained essentially unchanged, despite a roughly 90 percent reduction from cars on a per mile basis.

1. 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.

2. 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.

3. 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.

4. 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.

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

6. 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.

7. 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.

8. [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: 294–299
Non-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. 10th–90th percentile change in SO_2 (13–31 micrograms/m^3) resulted in 3.5 percent increase in childhood consultations. For the 10th–90th 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.

9. [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)

10. [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 1st 1984 to October 31st 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.

11. [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.

12. [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.

13. [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.

14. [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.

15. [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.

16. [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 10th–90th 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.

17. [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 10th–90th 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 10th–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.

18. [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.

19. [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 90th percentile of 109.6; C0 (100 ppb) mean12.4; NO_2 ppb 24 hour mean 31.7 with 90th percentile of 46.0; SO_2 ppb mean 13.4 with 90th percentile of 25.1. One hour ozone in ppb as maximal hourly mean was 31.8 with 90th 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.

20. [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.

21. Tolocka, M.P. et. al. Ultrafine nitrate particle events in Baltimore observed by real-time single particle mass spectrometry. Atmos. Env. Volume 38, Issue 20, June 2004.
Ambient particles in Baltimore, Maryland were characterized from April through November 2002 using the real-time single particle mass spectrometer, RSMS III. When particles containing nitrate were examined, two types of ultrafine particle events were revealed: a large burst of nominally "pure" nitrate particles in the 50–90 nm size range, and a smaller (and less frequent) burst of "pure" particles in the 50–90 nm size range that grew to 110–220 nm with time.

Coincident with both of these events was an increase in the number of mixed composition particles containing nitrate, suggesting that they were formed by condensation of ammonium nitrate onto pre-existing particles. Meteorological variables, particle number concentrations and continuous nitrate mass measurements were compared to the single particle data. Number and mass concentrations estimated from RSMS III correlated well with similar measurements with other techniques. Ultrafine nitrate particle events were observed during periods of low temperature and high relative humidity as expected from ammonium nitrate equilibrium considerations. During these events, the partitioning of ammonium nitrate to the particle phase strongly influenced the particle number concentration as well as the chemical composition.

22. Cronan, C.S & Schofield, C.L.
Aluminum Leaching Response to Acid Precipitation: Effects on High-Elevation Watersheds in the Northeast.
Science 20 April 1979: Vol. 204. no. 4390, pp. 304–306.
Atmospheric inputs of sulfuric acid and nitric acid to noncalcareous higher-elevation watersheds in the White Mountain and Adirondack regions lead to comparatively high concentrations of dissolved aluminum in surface and ground waters. This phenomenon appears to result from modern increases in soil aluminum leaching. Transport of this aluminum to acidified lakes can lead to fish mortality. Combined results from areas of silicate bedrock in the United States and Europe suggest that aluminum represents an important biogeochemical linkage between terrestrial and aquatic environments exposed to acid precipitation.