Health and Clean Air Newsletter

Spring--2003

(1) **[542]**
DOUGLAS, J.W.B., & WALLER, R.E.
Air pollution and respiratory infection in children
Br J Prev & Social Medicine 20; 1–8, 1966
3,866 children followed for a period of five years. All were adopted into other families. Categorized as living in high, moderate, low or very low regions of pollution. Logging of lower chest infections over a three-year period showed 12.9 percent incidence in high pollution region, versus 4.3 percent in very low region. This study had an important influence on the deliberations of the U.S. Senate Committee responsible for the first US Clean Air Act.

(2) **[541]**
GIRSCH, L.S., SHUBIN, E., DICK, C., & SCHULANER, F.A.
A study on the epidemiology of Asthma in Children in Philadelphia
J Allergy 39; 347–357, 1967
The: "Citizen's Guide to Air Pollution" on page 94 notes: "The first study of the relationship between hospital emergency visits and pollutants would appear to be one conducted in Philadelphia in 1965 by Girsch and his colleagues." Comparing 1,346 patient visits to the Emergency Department of the Children's Hospital, they concluded: 'There was a threefold greater incidence of bronchial asthma during days of noteworthy high pollution. This was compared to the observation that very few asthma attacks occurred during the ragweed season. It is remarkable that very little attention seems to have been paid to these data."

(3) Roughton FJW, Darling RC. "The effect of carbon monoxide on the oxyhemoglobin dissociation curve." *Am J Physiol* 1944;141:17–31.

(4) Longo LD, Hill EP. "Carbon monoxide uptake and elimination in fetal and maternal sheep." *Am J Physiol* 1977;232:H324–30; **and**, Bureau MA, Monette J, Shapcott D, Pare C, Mathieu J-l, Lippe J, Blovin D, Berthiaume Y, Begin R. "Carboxyhemoglobin concentration in fetal cord blood and in blood of mothers who smoked during labor." *Pediatrics* 1982;69:371–73.

(5) World Health Organization, Tobacco Free Initiative, http://www.emro.who.int/TFI/SharedWorld-Women-Mums.htm.

(6) U.S. Environmental Protection Agency, *National Air Quality and Emissions Trends Report, 1999*, Table A-2, pp. 132–35 (Washington, D.C. 2001).

(7) **[154]**
BOBAK, M.
Outdoor air pollution, low birth weight, and prematurity

Environmental Health Perspectives 108: 173–176; 2000
Study based on 67 districts of Czech Republic in 1991. Maternal exposures to SO_2 TSP, and NO_X in each trimester of pregnancy estimated as arithmetic means of all daily measurements taken by all monitors in the district of birth of each infant. Odds ratios of low birth weight, prematurity, and intrauterine growth retardation calculated. Low birth weight and prematurity associated with SO_2 and less strongly for TSP. No effect on intrauterine growth retardation. Low gestational age accounted for the association between SO_2 and low birth weight. Effect greatest in relation to SO_2 in first trimester.

(8) **[138]**
MAISONET, M., BUSH, T.J., CORREA, A., & JAAKKOLA, J.K.
Relation between ambient air pollution and low birth weight in the northeastern United States
Environ Health Perspect 109 (suppl 3); 351–356 (2001)
Low birth weight (LBW) evaluated in relation to ambient CO, PM_{10}, and SO_2 data. Births between January 1994 and December 1996 in six northeastern cities, Boston, Hartford, Philadelphia, Pittsburgh, Springfield, and Washington DC. LBW consisted of birth weight below 2,500 grams. Risk of LBW increased with increasing CO or SO_2 exposure. No association with PM_{10}. 26 references.

(9) **[254]**
RITZ, B., & YU, F.
The effect of ambient carbon monoxide on low birth weight among children born in southern California between 1989 and 1993.
Environ Health Perspect. 1999 Jan;107(1):17–25.

(10) **[212]**
HA, E-H., HONG, Y-C., LEE, B-E., WOO, B.-H., SCHWARTZ, J., & CHRISTIANI, D.C.
Is Air Pollution a risk factor for low birth weight in Seoul?
Epidemiology 2001; 12; 643–648
All full term births in Seoul over the period of two years from January 1996 to December 1997. Adjustment for gestational age, maternal age, parental educational level, parity, and infant sex. Smoothing plots used. The results showed that the adjusted relative risk of low birth weight was 1.08 for each interquartile increase for CO during the first trimester; also 1.07 for NO_2; 1.06 for SO_2; and 1.04 for TSP—all for interquartile increases in exposure. Exposure estimated from 21 monitoring sites—main pollutant is vehicle exhaust. 276,763 births included. 75^{th} percentile of NO_2 exposure was 35.4 ppb. Mean birth weight changes more or less linear with each pollutant. For NO_2, from lowest to highest was a drop in birth weight of about 30 gm.

(11) **[255]**
RITZ, B., YU, F., FRUIN, S., CHAPA, G., SHAW, G.M., & HARRIS, J.A.
Ambient Air Pollution and Risk of Birth Defects in Southern California
Am J Epidemiol 2002; 155; 17–25

California has a Birth Defects Monitoring Program; data from this between 1987 and 1993 were used; place of residence was compared to locally measured air pollutants –22 stations measured CO; 27 collected O_3 data; and 11 had PM_{10} monitors. Eligible cases were all live born infants and fetal deaths diagnosed between 20 weeks of gestation and one year after birth with isolated, multiple syndromic or chromosomal cardiac or orofacial cleft defects. Residence had to be within 10 miles of a monitoring station. Results for six groups analyzed—aortic defects, defects of atrium and septum; endocardial and mitral valve defects; pulmonary artery and valve defects; conotruncal defects such as tetralogy of Fallot; and ventricular septal defects. All cardiac defects confirmed by autopsy or by surgical reports, catheterization, or echocardiogram. Orofacial defects also subdivided.

Results showed that odds ratios for ventricular septal defects increased in a dose-response fashion with increasing second-month CO exposure (Odds Ratios over 2.0 in some cases). Similarly, risks for aortic artery and valve defects, pulmonary artery and valve anomalies, and conotruncal defects increased with second month ozone exposure. Animal data which supports these findings are summarized. Very thorough hierarchical two level regression model used to adjust for multiple comparisons. Control infants and fetuses numbered 10,649. There were 313 ventricular septal defects. 50 references.

(12) **[528]**
ELLIOT, J.G., CARROLL, N.G., JAMES, A.L., & ROBINSON, P.J.
Airway alveolar attachment points and exposure to cigarette smoke *in utero*
Am J Respir Crit Care Med 167; 45–49, 2003
32 infants who died from SIDS grouped according to their perinatal cigarette smoke exposure. Distance between alveolar attachments on the airways was measured, and was found to be greater in infants exposed to cigarette smoke in utero or both in utero and during the postnatal period, but not different in those with only postnatal exposure. Authors conclude: "These findings suggest that *in utero* cigarette smoke exposure may result in abnormal airway function due to a reduction of the forces opposing airway narrowing."

Figure illustrates method of measurement with photomicrograph of a membranous airway from an eight-month-old infant. Distance between attachments was 0.10 mm in those exposed and 0.08 mm in those not exposed. 82 airways examined in nonexposed group, 46 in those exposed *in utero* only, and 147 in those with both *in utero* and postnatal exposure.

(13) **[543]**
BATES, D.V.
The Effects of Air Pollution on Children
Environ Health Perspect 103 (Suppl 6): 49–53, 1995
Drew attention to the increased mortality in children in the London 1952 episode. Table as follows:

All	<4 weeks	4 weeks-1 year	1-14 years	Total
Week before episode 945	16	12	10	38
Week of the episode 2484	28	26	13	67

(14) **[100]**
BOBAK, M., & LEON, D.A.
The Effect of Air Pollution on Infant Mortality appears specific for respiratory causes in the postnatal period
Epidemiology 1999; 10; 666–670
All births registered in the Czech Republic between 1989 and 1991. For each infant death, 20 controls randomly selected from infants of the same sex born on the same day and alive when the case died. Exposure assigned as the arithmetic mean of all 24-hour air pollution measurements in the district of residence of each case and control for the period between the birth and death of the index case. 2,494 infant deaths. Respiratory deaths analyzed. Risk Rate ratios for a 50 microgram/m^3 increase in particles = 1.95; 1.74 for SO_2; and 1.66 for NO_2. Only particles showed a consistent association when all pollutants entered in one model. No evidence of an association between any pollutant and mortality from any other cause. Conclude: "the effects of air pollution on infant mortality are specific for respiratory causes in the postneonatal period, are independent of socioeconomic factors, and are not mediated by birth weight or gestational age."

The crude RR for SO_2 was 2.16; adjusted for socioeconomic was 1.94; adjusted for perinatal factors was 2.09; and adjusted for all covariates was 1.87. These values were higher than those for TSP. Similar RRs for NO_2 were 1.55; 1.71;; 1.60; & 1.78.

Respiratory deaths numbered about 133.

(15) **[103]**
CHAY, K.Y., & GREENSTONE, M.
The Impact of air pollution on infant mortality: evidence from geographic variation in pollution shocks induced by a recession
National Bureau of Economic Research, 1050 Massachusetts Avenue, Cambridge, MA 02138
Manuscript of about 40 typewritten pages with Figures and Tables sent to me by Dr. Michael Brauer. It had been sent to him by his brother, who is an economist in Washington, DC.

In 1980–1982, there was an economic recession, leading to substantial reductions in particulate pollution in some regions of the US. In this paper, neonatal mortality was assessed in relation to the reductions in TSP which occurred (regions being classified as those which had large reductions, medium reductions and small reductions). A large bank of data was analyzed, and it was shown that average income did not change over this period; that other variables that might have an influence on neonatal mortality did not change; and that mean birth weight and Apgar indices did not alter.

It is shown that there was a reduction in infant deaths (within the period of one day and one month of birth) during the period of lower pollution. This amounted to four to eight fewer infant deaths per 100,000 live births at the county level, for a 1 microgram/m^3 reduction in TSP [note that in the Abstract and in the MS except on page 41 where it is correctly written, this is miss-spelled "mg/m3"]. The authors, being economists, note that this corresponds to an elasticity of 0.35–0.45.

In a sub analysis of Pennsylvania, where the drop in TSP was generally large, they note: "In all of Pennsylvania, mean TSP's pollution was relatively stable at about 70–74 units from 1978–1980 and then declined precipitously to about 53 units by 1982–1983. At the same time, in 1978–1980 infant deaths within one year of birth attributable to "internal" causes (e.g. respiratory and cardiopulmonary deaths) were stable and occurred at the rate of 1315–1380 per 1000,000 live births. But from 1980–1982, the internal infant mortality rate declined from 1315 to 1131, and remained at this lower level in 1983–1984. While not controlling for all changes that may have occurred in the absence of the pollution decline, these numbers imply that a 1 microgram/m^3 decline in TSP's may result in about 10–11 fewer infant deaths per 100,000 births, which is an elasticity of 0.5–0.6."

Also: "Based on our quasi-experimental research design, we find a significant impact of pollution reductions on decreases in infant mortality rates at the county level, with a 1 microgram/m^3 decline in suspended particulates associated with about four to eight fewer infant deaths per 100,000 live births (a 0.35–0.45 elasticity). The results are driven almost entirely by fewer deaths occurring within one month and one day of birth, suggesting that pollution exposure adversely impacts the fetus before birth."

The paper contains figures that illustrate the changes, and many tables with a complex array of data from 1978 to 1984 addressing other factors that might have influenced the results. The data are convincing and the conclusions seem valid.

The paper is written in a somewhat roundabout way (perhaps characteristic of the style of economists?) but the complex data are well presented and discussed.

(16) **[139]**
CONCEICAO, G.M.S., MIRAGLIA, S.G.E.K., KISHI, H.S., SALDIVA, P.H.N., & SINGER, J.M.
Air Pollution and child mortality: a time-series study in Sao Paulo, Brazil
Environ Health Perspect 109 (suppl 3):347–350 (2001)
Mortality from respiratory causes of children under the age of 5. Daily levels of SO_2, CO, PM_{10}, and ozone used. Temperature and humidity data included. All three pollutants were associated with death rate, and the observed associations were dose dependent and "quite evident after a short period of exposure (2 days)." The estimated proportions of respiratory deaths attributed to CO, SO_2, and PM_{10} when considered individually, were around 15 percent, 13 percent, and 7 percent respectively. Reliability of death certification process is described.

(17) **[23]**

SMITH, K.R., SAMET, J.M., ROMIEU, I., & BRUCE, N.

Indoor air pollution in developing countries and acute lower respiratory infections in children

Thorax 2000; 55; 518–532

Review article. 116 references. 4.1 million deaths annually from acute respiratory illnesses in children under five in developing countries. This is compared with 3.0 million from intestinal disease, and 0.68 million from malaria. Measured particulate levels indoors recorded in Papua New Guinea, Kenya, India, Nepal, China, and the Gambia. Description of biomass cooking. Important review.

(18) **[35]**

STEERENBERG, P.A., NIERKENS, S., FISCHER, P.H., VANLOVEREN, H., OPPERHUIZEN, A., VOS, J.G., & VAN AMSTERDAM, J.G.C.

Traffic-related air pollution affects peak expiratory flow, exhaled nitric oxide, and inflammatory nasal markers

Arch Environ Health 56; 167–174; 2001

Eighty-two children attending elementary school either in Utrecht (more polluted) or in Bilthoven (classified as suburban) studied. Black smoke in Utrecht was 53 micrograms/m^3 compared to 18 in Bilthoven. NO_2 and CO were 1.5 and 1.8 times higher in Utrecht. Nasal lavage showed that levels of interleukin-98, urea, uric acid, albumin, and nitric oxide metabolites were significantly higher in the Utrecht children. PEFR was 5.3 ml/min less in Utrecht children. Coughing and respiratory disease levels appeared to be higher in suburban children, and parents of suburban children smoked more cigarettes/day. Odds ratios for different parameters reported. Authors conclude that children in area of higher traffic-related air pollution show increased levels of inflammatory nasal markers; increased response in PEFR; and increased levels of exhaled NO.

(19) **[94]**

NEAS, L.M., DOCKERY, D.W., KOUTRAKIS, P., & SPEIZER, F.E.

Fine Particles and Peak Flow in Children: acidity versus mass

Epidemiology 1999; 10; 550–553

Ninety-two children from one camp and 64 from another. These were day camps. Eighty-four were aged ten or older. Twenty-four were "ever diagnosed" asthmatics; 11 used an asthma inhaler. Each child performed three supervised PFR measurements twice daily, upon arrival a.m. and departure p.m. Particle strong acidity, and fine sulfate were both measured. PM_{10} averaged about 30 micrograms/m^3; ozone averaged about 56 ppb as a 12-hour average in daytime. Lower morning PEFR's associated with both inhalable particulate matter and fine sulfate particles. Effect was -2.79 l/min per 8 microgram/m^3 increase in sulfate. Particle strong acidity and the coarse particle mass were weakly associated with lower peak flows. Acutely lower peak flows were associated with fine sulfate particles, but only weakly with the acidity of these. Forty observation days.

Twelve hour ozone also associated with flow decrements. 20 ppb O_3 increment associated with -1.38 l/min change in PEFR.

(20) **[73]**

GOREN, A., HELLMANN, S., GABBAY, Y., & BRENNER, S.

Respiratory problems associated with exposure to airborne particles in the community
Arch Environ Health 54; 165–171, 1999

Rural area of Israel; one unpolluted and the other exposed to pollution from a cement factory and quarries. 638 children aged 7–13 in polluted community compared to 338 in clean community. Map of location of school, cement factory and quarry in polluted region. Graph of TSP and PM_{10} has erroneous ordinate, but peaks of PM_{10} might have exceeded 300 micrograms/m^3. PM_{10} level of 150 micrograms/m^3 said to be violated very often in polluted region. No particle analysis. Children in polluted region had: more respiratory symptoms, more cough, and more cough accompanied by sputum. Asthma also more prevalent. No differences in PFT's, but PEFR was lower in children in polluted region.

(21) **[113]**

VAN DER ZEE, S.C., HOEK, G., BOEZEN, H.M., SCHOUTEN, J.P., VAN WIJNEN, J.H., & BRUNEKREEF, B.

Acute effects of urban air pollution on respiratory health of children with and without chronic respiratory symptoms
Occup Environ Med 1999: 56: 802–813

Studies in three consecutive winters starting in 1992. PEFR and daily respiratory symptoms registered daily in panels of children aged 7–11 in areas in the Netherlands with high traffic density. Simultaneously, panels of children in rural areas studied. Daily measurements of PM_{10}, black smoke (BS), SO_2, and NO_2 performed in all areas. "In children with symptoms from both areas, significant associations were found between PM_{10}, BS, and sulphate concentrations and the prevalence of symptoms of the lower respiratory tract and decrements in PEFR."

Also noted that particle concentrations in the urban areas were associated with increased use of bronchodilators. A 100 microgram/m^3 increase in the five-day mean PM_{10} was associated with a twofold increase in the use of bronchodilators, a 50 percent increase in lower respiratory symptoms, and an 80 percent increase in decrements of PEFR. Much smaller effects in children without any pre-existing respiratory symptoms.

Numbers of Children

	With Symptoms		Without Symptoms	
	Urban	**Rural**	**Urban**	**Rural**
Winter 1992–1993	31	48	43	60
1993–1994	55	71	56	77
1994–1995	56	59	38	39

Authors also conclude that the use of medication for asthma does not prevent the adverse effects of particulate air pollution. Excellent discussion.

(22) Committee on Environmental Health, American Academy of Pediatric, "Ambient Air Pollution: Respiratory Hazards to Children," *Pediatrics,* 91:6 (June, 1993), http://www.aap.org/policy/04408.html.

(23) **[17]**
BURNETT, R.T., SMITH-DORION, M., STIEB, D., RAIZENNE, M.E., BROOK, J.R., DALES, R.E., LEECH, J.A., CAKMAK, S., & KREWSKI, D.
Association between ozone and hospitalization for acute respiratory diseases in children less than two years of age
Am J Epidemiol 2001: 153; 444–452
15-year period 1980–1994 in Toronto (including 6 cities of Toronto, North York, East York, Etobicoke, Scarborough & York). Daily number of emergency or urgent hospital admissions for croup, pneumonia, asthma and acute bronchitis/bronchiolitis. Gastroenteritis used as control (second leading cause of hospitalization). Prediction equations, previously validated, used to predict $PM_{2.5}$ from TSP, and sulfates also used in the summer. LOESS nonparametric smoothing method. Adjustment for weather described, and temporal trends and day of the week effects taken out. Mean values for ozone 45.2 ppb; $PM_{2.5}$ 18 micrograms/m^3; $PM_{10-2.5}$ 16.2; NO_2 ppb 44.1; SO_2 ppb 11.8; CO ppm 1.9. Mean admissions/day for respiratory problems 2.9; mean GI admissions 1.2:

> Percentage increase in daily admissions in May–August period was 14.2 percent associated with a 45.2 ppb increase in O_3, (daily one hour maximum.) if lag of one day used. If lag of 2 days, was 13.2 percent increase. Based on five-day average, 45.2 ppb increase in ozone associated with 34.8 percent increase. This became 29.4 percent after adjustment for either $PM_{10-2.5}$ or NO_2. Correlation of O_3 with $PM_{2.5}$ was 0.58 and with NO_2 was 0.52; with CO was 0.24.

Increases for asthma were 31.3 percent; croup 45.3 percent; acute bronchitis/bronchiolitis 45.7 percent; and 23.3 percent for pneumonia. Note of CO relationship since this showed strongest relationship after adjustment for ozone.

(24) Burnett et al. "Effects of Low Ambient Levels of Ozone and Sulfates on the Frequency of Respiratory Admissions to Ontario Hospitals." *Environ Res,* 1994; 65:172–94.

(25) **[297]**
GILLILAND, F.D., BERHANE, K., RAPPAPORT, E., THOMAS, D.C., AVOL, E., GAUDERMAN, W. J., LONDON, S.J., MARGOLIS, H.G., MCCONNELL, R., ISLAM, K.T., & PETERS, J.M.
The Effects of Ambient Air Pollution on School Absenteeism due to respiratory illness
Epidemiology 2001: 12; 43–54

"An increase of 20 ppb of O_3 was associated with an increase of 62.9 percent for illness-related absence rates, 82.9 percent for respiratory illnesses, 45.1 percent for upper respiratory illnesses, and 173.9 percent for lower respiratory illnesses with wet cough." The effects were larger in communities with lower long term PM_{10} values. A remarkable feature of this paper is the sophistication of the statistical analysis—rewrites the book on how such information should be handled. Lag effect is somewhat confusing, since a 15-day lag provided the best fit, but the peak effect occurred five days after the O_3 peak and fell off slowly after that.

(26) Air Pollution Causes Increased School Absences; Researchers in Utah discovered that increases of inhalable particulate matter (PM_{10}) resulted in a 40 percent increase in overall absences from school by children. Ransom M.R., Pope C.A., "Elementary School Absences and PM_{10} Pollution in Utah Valley," *Environ Res* 1992; 58:204–19.

(27) **[527]**
PARK, H., LEE, B., HA, E-H., LEE, J-T., KIM, H., & HONG, Y-C.
Association of air pollution with school absenteeism due to illness
Arch Pediatr Adolesc Med 2002; 156; 1235–1239
Data from Seoul. Pollution data: Mean values PM_{10} 68 micorgrams/m^3; NO_2 33 ppb; SO_2 9.2 ppb; CO 1.11 ppm; and ozone 22.8 ppb. Correlation coefficient between NO_2 and SO_2 was 0.68 and between NO_2 and PM_{10} was 0.76. Study period from March 2, 1996 to December 22, 1999. Average enrollment was 671 boys and 593 girls. "A teacher recorded an illness-related absence, by parent's report, in an attendance record for each class."

LOESS, a regression smoother, applied in a time-series analysis using a generalized additive model. Model selected using Akaike's information criteria. Average number of daily absences was 5.89, with 4.20 due to illness. Hence absence rate was 4.66 per 100 student days, and illness related absence rate was 3.69 per 1000 student days.
Relative risks of illness related absences per interquartile range of PM_{10} (42 micrograms/m^3) 1.06; per IQ range of NO_2 (14.5 ppb) was 1.02; per IQ range of ozone (15.9 ppb) was 1.08; per IQ range of SO_2 (5.68 ppb) was 1.09; and per IQ range of CO (0.52 ppm) was 0.96. No significant increased risk for non-illness-related absences.

(28) **[165]**
FRIEDMAN, M.S., POWELL, K.E., HUTWAGNER, L., GRAHAM, L.M., & TEAGUE, W.G.
Impact of changes in transportation and Commuting behaviors during the 1996 Summer Olympic Games in Atlanta on Air Quality and Childhood Asthma
JAMA 2001: 285; 897–905
Comparison of the 17 days of the Olympic Games (July 19–Aug 4) to a baseline period consisting of the 4 weeks before and 4 weeks after the Olympic Games. Peak one hour level of O_3 fell to 50–100 ppb during the games from a predicted value of about 70–120 in the comparison periods. PM_{10} (24 hour level) was 20–45, compared to levels of 30–70; NO_2 was only slightly lower running at about 30 ppb peak one-hour level compared to values between 20–65. CO also slightly lower. Traffic density measurements showed decreases of 22 percent in weekday 1-hour morning peak traffic counts during the Olympic Games. Ozone levels fell

slightly over the same period in three different places 60 km to 100 km from Atlanta; but these changes were only about one fifth of the drop in Atlanta.

Citywide acute care visits and hospitalizations for asthma were logged. Results showed no changes in nonasthma diagnoses; decreases of 41 percent in Medicaid claims file, 44 percent decreases in HMO database; 11 percent decreases in two emergency pediatric departments; and decreases of 19 percent in Georgia Hospital Discharge Database. Lack of change in other diagnostic categories indicates that children did not leave Atlanta over the period of the Olympic Games.

(29) **[362]**
HEINRICH, J., HOELSCHER, B., FRYE, C., MEYER, I., PITZ, M., CYRYS, J., WJST, M., NEAS, L., & WICHMANN, H-E.
Improved air quality in reunified Germany and decreases in respiratory symptoms
Epidemiology 2002 13; 394–401
Three study areas. Questionnaires administered to 7,632 children aged 5–14; collected in three phases: 1992–1993; 1995–1996; and 1998–1999. The region was the State of Sachsen-Anhalt formerly part of East Germany. Details of air pollution levels. Odds ratios (and confidence limits) for a 50 microgram/m^3 change in TSP were 3.0 (1.7–5.3) for bronchitis; 2.6 (1.0–6.6) for sinusitis; and 1.9 (1.2–3.1) for frequent colds. Effect sizes for a 100 microgram/m^3 increment in SO_2 were similar. Effect estimates noted to be stronger in children not exposed to gas stove emissions, visible molds or dampness, cats, or ETS. Impressive linear relationship with all points plotted between prevalence of bronchitis and TSP, such that prevalence was 35–40 percent when TSP below 40 micrograms/m^3 as an annual mean, and up to 62 percent when TSP was 75 microgram/m^3. There was a drop in bronchitis prevalence from 54 percent to 38 percent between the first and third surveys.

(30) Kopp, M.V., Bohnet, W., Frischer, T., Ulmer, C., Studnicka, M., Ihorst, G., Gardner, C., Forster, J., Urbanek, R., & Kuer, J.; "Effects of ambient ozone on lung function in children over a two-summer period," *Eur Respir J* 2000; 16; 893–900.

(31) University of Toronto, *The Mechanics of Respiration Supplementary Notes*, www.utoronto.ca/respgrp/MechSN.pdf.

(32) **[366]**
GAUDERMAN, W.J., GILLILAND, G.F., VORA, H., AVOL, E., STRAM, D., MCCONNELL, R., THOMAS, D., LURMANN, F., MARGOLIS, H.G., RAPPAPORT, E.B., BERHANE, K., & PETERS, J.M.
Association between air pollution and lung function growth in Southern California Children
Am J Respir Crit Care Med 166; 76–84; 2002

1,678 children enrolled as fourth graders in 1996 and followed for four years. Results essentially the same as for the first cohort, namely significant deficits in lung function growth associated with exposure to acid vapor, NO_2, $PM_{2.5}$, and elemental carbon. MMEF reduced by 11 percent and FEV1 by 5 percent across the observed range of acid exposure. Exposure to ozone associated with reduced growth of PEFR (the p value for this was = 0.006).

(33) **[13]**
JEDRYCHOWSKI, W., FLAK, E., & MORZ, E.
The adverse effect of low levels of ambient air pollutants on lung function growth in preadolescent children
Environ Health Perspect 107; 669–674; 1999
1,001 preadolescent children in Krakow, Poland. City center residence contrasted with control area with less pollution. Cohort prospective study over two years. Asthmatic children excluded. No differences in girls but boys in polluted region had lower FVC and FEV1 changes. TSP values polluted vs cleaner were 52.6/33.2 micrograms/m^3, and for SO_2 were 43.87 vs 31.77 micrograms/m^3. Too short a period of follow-up for the study to be definitive.

(34) Van Louveren H.S., Wagenaar S., Walvoort H.C., Vos J.G.; Effect of ozone on the defense to a respiratory Listeria monocytogenes infection in the rat. Suppression of macrophage function on cellular immunity and aggravation of histopathology in lung and liver during infection. *Toxicol Appl Pharmacol*, 1988; 94:374–393.

(35) Gardner D.E.; "Use of experimental airborne infections for monitoring altered host defenses," *Environ Health Perspect* 1982; 43:99–107.

(36) "Executive Summary," *Air Quality Criteria for Ozone and Related Photochemical Oxidants,* United States Environmental Protection Agency, (Washington, DC: Office of Research and Development), February 1994.

(37) Hyde, D., Plopper, C., Harkema, J., St. George, J., Tyler, W., Dungworth, D.; Ozone-induced structural change in monkey respiratory system; *Atmospheric Ozone Research and Its Policy Implications,* Eds. Schneider, T., Lee, S.D., Wolters, G.J.R. and Grant, L.D., Elsevier, 1989.

(38) **[232]**
PETERS, J.M., AVOL, E., NAVIDI, W2., LONDON, S.J., GAUDERMAN, W.J., LURMANN, F., LINN, W.S., MARGOLIS, H., RAPPAPORT, E., GONG, H. JR., & THOMAS, D.C.
A study of twelve southern California communities with differing levels and types of Air Pollution
Am J Respir Crit Care Med 1999: 159; 760–767.
Basic description of the study and analysis of questionnaire data; no very striking differences between the 12 communities in spite of considerable differences in air pollution.

(39) **[235]**

GAUDERMAN, W.J., LONDON, S.J., MARGOLIS, H.G., MCCONNELL, R., ISLAM, K.T., & PETERS, J.M.

The Effects of Ambient Air Pollution on School Absenteeism due to respiratory illness
Epidemiology 2001: 12; 43–54

"An increase of 20 ppb of O_3 was associated with an increase of 62.9 percent for illness-related absence rates, 82.9 percent for respiratory illnesses, 45.1 percent for upper respiratory illnesses, and 173.9 percent for lower respiratory illnesses with wet cough." The effects were larger in communities with lower long term PM10 values. A remarkable feature of this paper is the sophistication of the statistical analysis—rewrites the book on how such information should be handled. Lag effect is somewhat confusing, since a 15-day lag provided the best fit, but the peak effect occurred five days after the O_3 peak and fell off slowly after that.

(40) **[236]**

GAUDERMAN, W.J., MCCONNELL, R., GILLILAND, F., LONDON, S., THOMAS, D., AVOL, E., VORA, H., BERHANE, K., RAPPAPORT, E., LURMANN, F., MARGOLIS, H., & PETERS, J.

Association between Air Pollution and lung function growth in Southern California Children
Am J Respir Crit Care Med 162; 1383–90: 2000

This is the basic longitudinal analysis paper. Confirmed by identical data from second longitudinal cohort (presented at Advisory Committee Meeting in January 2001).

Four-year period of observation in three cohorts of children (n=3035). Deficit in lung growth associated with higher exposures to PM_{10}, $PM_{2.5}$, $PM_{10-2.5}$, NO_2, and inorganic acid vapor. No association with ozone. Comparing most polluted with least polluted environments, loss of FEV1 was 3.4 percent, and in MMEF was 5.0 percent. The deficits were larger in children spending more time outdoors. Data is well presented and graphs of adjusted annual lung function against pollutants are convincing. Deficits generally larger than those reported for ETS exposure.

(41) **[256]**

MCCONNELL, R., BERHANE, K., GILLILAND, F., LONDON, S.J., ISLAM, T., GAUDERMAN, W.J., AVOL, E., MARGOLIS, H.G., & PETERS, J.M.

Asthma in exercising children exposed to ozone: a cohort study
Lancet 2002; 359; 386–391

From the Southern California Children's Study. Relevant numbers;
5,762 children completed baseline questionnaires;
479 excluded because they were not at school when the questionnaire was administered;
883 excluded for a history of asthma;
312 excluded because of missing answers to "wheezing" questions;
26 excluded for chest illnesses such as cystic fibrosis:
527 excluded because they had less than one year of follow-up;
This left 3,535 children with no initial history of asthma; 2,752 of these had no history of wheezing; 1,934 played sports; 273 played three or more team sports;

There were 46 low pollution communities (O_3 daytime mean 40.0 ppb); and 46 high pollution communities (O_3 mean 59.6 ppb). PM_{10} twice as high in high ozone communities (43.3 vs 21.6) and $PM_{2.5}$ three times higher (21.4 vs 7.6). NO_2 three times higher in high ozone communities (29.2 vs 10.8 ppb).

It was shown that development of asthma prospectively was three times higher in children participating in more than 3 sports in high ozone communities, compared to children who did no sports in both communities or did fewer than 3 sports in high ozone communities. No differences in development of asthma if other pollutants were studied. Excellent discussion; 32 references. Convincing argument as to why standard cross-sectional comparisons might show no differences in prevalence of asthma.

(42) Galizia, A., & Kinney, P.L.; "Long-Term residence in areas of high ozone: associations with respiratory health in a nationwide sample of nonsmoking young adults," *Environ Health Perspectives* 107: 675–79; 1999.

(43) **[454]**
KUNZLI, N., LURMANN, F., SEGAL, M., NGO, L., BALMES, J., & TAGER, I.B.
Association between lifetime ambient ozone exposure and pulmonary function in college freshmen—results of a pilot study
Environ Research 72, 8–23, 1997
130 Berkeley freshmen aged 17–21 (all never smokers) participated twice; calculation of 8 different indices of lifetime ozone exposure (all were closely correlated). "FEF25-75 and FEF 75 percent decreased with both effective exposure and ecologic assignment of O^3 exposure." FEV1 and FVC not significantly related. This finding is described as being "consistent with biologic models of chronic effects of O^3 in the small airways" Note that the findings have to be confirmed in a larger sample that is representative of the target population. PM_{10} not related to function test changes. Acute and subacute effects of local air pollution were unlikely. Very detailed discussion but forgot to quote Richards study in young smokers.

(44) Committee on Environment and Public Works, U.S. Senate, *Legislative History of the Clean Air Act Amendments of 1970*, 93d Cong., 2d Session, 1974, Committee Print (Washington, D.C.: U.S. Government Printing Office, 1974) p. 358. The proposed tailpipe standards had been arrived at by estimating the aggregate reduction in emissions that would be required to reduce ambient concentrations to levels meeting the health-based standards, then back-calculating to the gram-per-mile limits cars would have to meet to achieve the reductions.

(45) **[542]**
DOUGLAS, J.W.B., & WALLER, R.E.
Air pollution and respiratory infection in children
Br J Prev & Social Medicine 20; 1–8, 1966

3,866 children followed for a period of five years. All were adopted into other families. Categorized as living in high, moderate, low or very low regions of pollution. Logging of lower chest infections over a three-year period showed 12.9 percent incidence in high pollution region, versus 4.3 percent in very low region. This study had an important influence on the deliberations of the U.S. Senate Committee responsible for the first US Clean Air Act.

(46) **[543]**
BATES, D.V.
The Effects of Air Pollution on Children
Environ Health Perspect 103 (Suppl 6): 49–53, 1995
Drew attention to the increased mortality in children in the London 1952 episode. Table as follows:

All	**<4 weeks**	**4 weeks-1 year**	**1-14 years**	**Total**
Week before episode 945	16	12	10	38
Week of the episode 2484	28	26	13	67

(47) **[298]**
MCCONNELL, R., BERHANE, K., GILLILAND, F., ISLAM, T., GAUDERMAN, W.J., LONDON, S.J., AVOL, E., RAPPAPORT, E.B., MARGOLIS, H.G., & PETERS, J.M.
Indoor risk factors for asthma in a prospective study of adolescents
Epidemiology 2002; 13; 288–295
3,535 Southern California school children with no history of asthma enrolled in 1993 and followed for five years. 265 reported a new diagnosis of asthma during the follow-up, and of these 163 had reported no history of wheeze at baseline. In those with no wheezing, an increased risk of developing asthma was associated with a humidifier, any pet, or specifically a dog in the home. An estimated 32 percent of new asthma cases could be attributed to pets. 23.6 percent of boys and 20.8 percent of girls reported a wheeze. 25 percent white, 19.8 percent Hispanic and 17.1 percent black. Air conditioning in 62.3 percent of homes in those with wheeze, and in 59.9 percent of homes in those with no wheeze. Well written discussion. 52 references.

(48) **[298]**
MCCONNELL, R., BERHANE, K., GILLILAND, F., ISLAM, T., GAUDERMAN, W.J., LONDON, S.J., AVOL, E., RAPPAPORT, E.B., MARGOLIS, H.G., & PETERS, J.M.
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(49) **[528]**

ELLIOT, J.G., CARROLL, N.G., JAMES, A.L., & ROBINSON, P.J.

Airway alveolar attachment points and exposure to cigarette smoke *in utero*

Am J Respir Crit Care Med 167; 45–49, 2003

32 infants who died from SIDS grouped according to their perinatal cigarette smoke exposure. Distance between alveolar attachments on the airways was measured, and was found to be greater in infants exposed to cigarette smoke in utero or both in utero and during the postnatal period, but not different in those with only postnatal exposure. Authors conclude: "These findings suggest that *in utero* cigarette smoke exposure may result in abnormal airway function due to a reduction of the forces opposing airway narrowing."

(50) **[366]**

GAUDERMAN, W.J., GILLILAND, G.F., VORA, H., AVOL, E., STRAM, D., MCCONNELL, R., THOMAS, D., LURMANN, F., MARGOLIS, H.G., RAPPAPORT, E.B., BERHANE, K., & PETERS, J.M.

Association between air pollution and lung function growth in Southern California Children

Am J Respir Crit Care Med 166; 76–84; 2002

1,678 children enrolled as fourth graders in 1996 and followed for four years. Results essentially the same as for the first cohort, namely significant deficits in lung function growth associated with exposure to acid vapor, NO_2, $PM_{2.5}$, and elemental carbon. MMEF reduced by 11 percent and FEV1 by 5 percent across the observed range of acid exposure. Exposure to ozone associated with reduced growth of PEFR (the p value for this was = 0.006).

(51) **[545]**

Pope, C.A. III

Respiratory hospital admissions associated with PM_{10} pollution in Utah, Salt Lake, and Cache Valleys

Arch Environ Health 46, 90–97, 1991