Health and Clean Air Newsletter

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1 Methuen & Co. (New York, 1987).

2 Although larger particles dominate the visible smoke that once characterized smokestacks and tailpipes, "haze" is dominated by particles in the smaller size ranges. The visibility impacts of particulate matter in urban settings maximizes in the PM2.5 size range, especially between 0.2-2 microns. Because most combustion particles are in the PM2.5 size range, some emission controls have tended to reduce haze as well as smoke. Much of this improvement has been due primarily to reductions in elemental carbon (aka soot, black carbon), which are efficient visibility reducers. Thus, while visibility of one sort–the dark plumes and smoky exhausts–has improved, haze remains a major challenge in many urban and rural areas. Ultrafine particles (less than 0.1 micron) do not reduce visibility per se, but they do grow into the fine particle range rather quickly and thus also reduce visual range.

3 Different researchers using varying methodologies have arrived at similar conclusions. Perhaps the seminal modern study was "Increased Mortality in Philadelphia Associated With Daily Air Pollution Concentrations," [SCHWARTZ, J &DOCKERY, D. *American Review of Respiratory Disease*, Vol 145, pgs. 600-604: 1992] in which the authors concluded that of the two million deaths each year in the U.S., particles accounted for 3 percent, or 60,000. In response to criticisms by some that time-varying factors such as season and day of week had not been sufficiently controlled, a reassessment, "A Case-Crossover Analysis of Air Pollution and Mortality in Philadelphia," [NEAS, L., SCHWARTZ, J. & DOCKERY, *D. Environ Health Perspect* 107:629-631: 1999] was conducted. The authors said the reassessment "confirms the general conclusion of the previous Poisson regression analysis of an association of TSP with daily mortality in Philadelphia, Pennsylvania." Earlier work was undertaken by Lave and Seskin [LAVE, L. B. & SESKIN, E.P. "Does Air Pollution Shorten Lives?" *Statistical and Mathematical Aspects of Pollution Problems*. Ed. John W. Pratt. New York: Marcel Drekker, 1974], concluding that the answer is affirmative. In 1980, using a newly assembled database, Lave and a colleague again examined the relationship between mortality and measures of sulfates and suspended particulates in 96 cities, counties, and metropolitan areas. The researchers found a "strong, consistent, and statistically significant association between sulfates and mortality." [CHAPPIE, M. & LAVE, L., "The Health Effects of Air Pollution: A Reanalysis," National Center for Environmental Economic, U.S. Environmental Protection Agency, [http://yosemite.epa.gov/EE/epa/eerm.nsf/vwRepNumLookup/EE-0192?OpenDocument.] Some studies have been conducted on a smaller scale. The Ohio River Basin Energy Study, a five-year analysis finished in 1981 concluded that the annual air pollution death toll in the six Midwestern states of Illinois, Indiana, Kentucky, Ohio, West Virginia, and Pennsylvania might range up to 25,000. [Office of Environmental Engineering and Technology, U.S. Environmental Protection Agency, Ohio River Basin Energy Study (ORBES) Main Report, p. 102, Washington, 1981]. See also "Particulate Air Pollution and Daily Mortality: A Synthesis," [*Public Health Reviews* 1991/1992 Vol. 19 (1992), pgs. 39-60], in which Schwartz reviews a number of studies finding an association between exposure to particles and death.

4 POPE, C.A. III., BURNETT, R.T., THUN, M.J., CALLE, E.E., KREWSKI, D., ITO, K., & THURSTON, G.D. "Lung Cancer, Cardiopulmonary mortality, and long-term exposure to fine particulate air pollution." *JAMA 2002*: Vol. 287; 1132-1141. American Cancer Society cohort recruited in 1982. Analysis of over 500,000 people in an average of 51 metropolitan districts. Interesting data showing reductions in PM2.5 from 1979-1983 and from 1999 to 2000, values ranging from 10 to 30 in the first period, and from 5 to 20 in the second. Nonparametric smoothed response functions shown for the three categories of diagnosis; conclude that for a 10 microgram/m3 change in PM10, all cause mortality increased by 4%; cardiopulmonary mortality increased by 6%, and lung cancer mortality increased by 8%. 95% confidence levels of all indices of RR were above 1.0. Coarse particle fraction and TSP not consistently associated with mortality. Other pollutants considered were sulfate, sulfur dioxide, nitrogen dioxide, carbon monoxide, and ozone. Numbers of metropolitan areas that could be considered varied with the different pollutants. Cox proportional hazards model with inclusion of a metropolitan-based random effects component in a two stage analysis. The continuous smoking variables included nine different indices (such as "current smokers years of smoking squared" and eight others). Controls also devised for educational level and occupational exposures. A 2 dimensional term was inserted to account for spatial trends. Higher regressions were noted in men than in women, and lower educational status was associated with higher risks. Risks in never smokers were also generally higher than in former or current smokers. Authors conclude: "The findings of this study provide the strongest evidence to data that long-term exposure to fine particulate air pollution common to many metropolitan areas is an important risk factor for cardiopulmonary mortality."

5 Maps can be found at http://capita.wustl.edu/CAPITA/CapitaReports/USVisiTrend/fig1.html, based on data collected by researchers at Washington University in St. Louis, MO., http://capita.wustl.edu/CAPITA/CapitaReports/USVisiTrend/usvstrd0.html.

6 U.S. Environmental Protection Agency, *National Air Quality and Emission Trends Report: 1999*, chapter 6, Visibility, www.epa.gov/oar/aqtrnd99/PDF%20Files/Chapter6.pdf.

7 KLEEMAN, M.J., SCHAUER, J.J., & CASS, G.R. "Size and Composition Distribution of fine particulate matter emitted from motor vehicles." *Environ Science & Technol:* Vol: 34; pgs. 132-1142:2000. Dilution source sampling system used to measure size-distributed chemical composition of fine particle emissions. It is impressive that from gasoline cars with and without catalytic converters, and from diesel engines, the size fraction is very constantly between 0.10 and 1.0 microns. Notes and observations by Bagley et al (*HEI Technical Report No 76* of 1996) that a 1991 diesel Cummins engine delivered lower overall particle mass emissions, but 15-35 times the number of particles as a 1988 Cummins engine (ultrafine particles made up the difference). For gasoline vehicles under various conditions, peaks of particle distributions seem to be between 0.1 and 0.2 microns, and the same is true of medium duty diesel vehicles. Sulfate particles seem to be a bit larger, peaking at about 0.9 microns. Interesting information.

8 BUNN, H.J., DINSDALE, D., SMITH, T., & GRIGG, J. "Ultrafine particles in alveolar macrophages from normal children." *Thorax* 2001; 56; 932-93422. Children aged 3 months to 16 years. No respiratory symptoms; Nonbronchoscopic BAL procedure before elective

surgery. Data from Leicester, UK; proximity of home to busy main road or residential street noted. All children's AM contained ultrafine carbonaceous particles (< 0.1 microns). Significantly more found in children who lived close to a busy traffic road. EM picture of carbonaceous ultrafine particles within a phagosome of an alveolar macrophage from a child aged 3 months. 10% of AM contained particles in AMs of children on busy roads, against 3. BUNN, H.J., DINSDALE, D., SMITH, T., & GRIGG, J. Ultrafine particles in alveolar macrophages from normal children *Thorax* 2001; 56; 932-93422 children aged 3 months to 16 years. No respiratory symptoms; Nonbronchoscopic BAL procedure before elective surgery. Data from Leicester, UK; proximity of home to busy main road or residential street noted. All children's AM contained ultrafine carbonaceous particles (< 0.1 microns). Significantly more found in children who lived close to a busy traffic road. EM picture of carbonaceous ultrafine particles within a phagosome of an alveolar macrophage from a child aged 3 months. 10% of AM contained particles in AMs of children on busy roads, against 3.2% in children who lived on quiet roads.

9 HAUSER, R., GODLESKI, J.L., HATCH, V., & CHRISTIANI, D.C. "Ultrafine particles in human lung macrophages." *Arch Environ Health* 56; 150-156: 2001. 14 healthy current nonsmokers, of whom 11 were utility workers; 3 non-maintenance employees of a university. Macrophages isolated from BAL fluid. EM used to count particles within macrophages, and these were found in all subjects. Average was between 34 to 231 ultrafine particles per cubic microgram of cell cytoplasm. Numbers were associated with FEV1 as percent of predicted. Authors note: "The demonstration of ultrafine particles in all 14 subjects independent of occupational exposure, suggests that there is environmental exposure to ultrafine particles." Illustration of intracellular ultrafines ranging in size from 6-60 nm; these are compared to cultured macrophages which contain no particles. 4 of the subjects were welders, but they did not have more particles than the nonwelders.

10 Karcher, B., R.P. Turco, F. Yu, M.Yu, M.Y. Danilin, D.K. Weisenstein, R.C. Miake-Lye, and R. Busen. "A Unified Model for Ultrafine Aircraft Particle Emissions." *J. of Geophy. Res.*, 105, D24, pp. 29,379-29,386: 2002. Also, Material Safety Data Sheet, Chevron Jet Fuel, http://library.cbest.chevron.com/lubes/chevmsdsv9.nsf/db12c751d5603b418825681e007cddb3/0af8b9b4d5605ecf8825652c005f0c6f?OpenDocument.

11 *Ward's Motor Vehicle Facts and Figures 2001*, "Travel Trends, Vehicle Miles of Travel and Fuel Consumption." pp. 70–71.

12 Peter Brimblecombe, *The Big Smoke*, p. 8, Methuen & Co. (London, 1987).

13 U.S. Environmental Protection Agency, *Air Quality Criteria for Particulate Matter*, Volume I, p. 2-23 (March 2001).

14 U.S. Environmental Protection Agency, *Air Quality Criteria for Particulate Matter*, Volume I, p. 2-35 (March 2001).

15 For an excellent summary of the anthrax attacks and a wide variety of related information, see the web site of the Centers for Disease Control, http://www.bt.cdc.gov/Agent/Anthrax/Anthrax.asp.

16 BRAUER, M., AVILA-CASADO, C., FORTOUL, T.I., VEDAL, S., STEVENS, B., & CHURG, A. "Air pollution and retained particles in the lung." *Environ Health Perspect* 2001, 109; 1039-1043. Comparison between 11 autopsy lungs in never smoking women in Mexico City, and 11 control residents of Vancouver, BC. Average PM10 levels given as 66 micrograms/m3 in Mexico City and 14 micrograms/m3 in Vancouver. Particles counted on electron microscopy. Total retained particles were 2,055 x 10^6 particles/gram of dried lung from Mexico City lungs and 279 x 10^6 particles/g dry lung from Vancouver residents. Lungs from Mexico City contained numerous chain-aggregated, masses of ultrafine carbonaceous spheres, some of which contained sulfur and aggregates of ultrafine aluminum silicate. These constituted 25% of the total particles in the Mexico City lungs but were only rarely seen in the Vancouver lungs.

17 Inglesby TV, et. al. "Anthrax as a Biological Weapon." JAMA 1999; 88: http://jama.ama-assn.org/issues/v281n18/ffull/jst80027.html

18 SEATON, A., SOUTAR, A., CRAWFORD, V., ELTON, R., McNERLAN, S., CHERRIE, J., WATT, M., AGIUS, R., & STOUT, R. "Particulate air pollution and the blood." *Thorax* 54; 1027-1032: 1999. 112 individuals over the age of 60 in Edinburgh and Belfast provided blood samples over 18 months-108 provided the maximum of 12 samples. Estimated personal exposures to PM10 over the previous three days showed negative correlations with Hb concentration, PCV, and red blood cell count, platelets, and Factor VII levels. Rise of C-reactive protein also documented. Suggest that inhalation of PM10 may produce a sequestration of red cells in the circulation. Possible changes in red cell adhesiveness. Suggest that these changes may be linked to cardiovascular effects. PM10 levels in Belfast reached 80 micrograms/m3 in Belfast on two occasions, but averaged about 25-30. In Edinburgh, levels were lower as no peak exceeded 60 micrograms/m3, and the annual average was nearer 15-20 micrograms/m3. Possible occurrence of hemodilution was excluded by measurements of albumin in the blood which showed no dilution had occurred. Rise of C-reactive protein may indicate that an inflammatory response has occurred, although no increase in white cells was noted (NOTE - prevalence of banded neutrophils not measured). Link blood changes to possible ischemic damage in those with vulnerable coronary circulations.

19 PEKKANEN, J., TIMONEN, K.L., TITTANEN, P., MIRME, A., RUUSKANEN, J., & VANNINEN, E. "Daily variations of particulate air pollution and ST-T depressions in subjects with stable coronary heart disease. The Finnish ULTRA Study." *Am J Respir Crit Care Med* 161: A24: 2000. 23 women and 24 men aged 54-83 with stable coronary heart disease studied with biweekly clinic visits for 6 months. Ambulatory ECG recorded on these visits. PM2.5 and ultrafines recorded each day. Exercise test done and was more indicative of change. 94 ST-T depressions observed. There was a consistent association between either ultrafine particles or PM2.5 with an increased risk of ST-T depression

20 DEVLIN, R.B., CASCIO, W., KEHRL, H., & GHIO, A. "Changes in heart rate variability in young and elderly humans exposed to concentrated ambient air particles." *Am J Respir Crit Care Med* 161: A239: 2000. 14 young (18-35) and 14 elderly (65-80) volunteers exposed to concentrated Chapel Hill particles for 2 hours. Changes in time and frequency

domains measured. No changes in young subjects, but elderly group developed significant decrements in both time and frequency domains immediately after exposure. Changes persisted for at least 24 hours.

21 VAN EEDEN, S.F., TAN, W.C., SUWA, T., MUKAE, H., TERASHIMA, T., FUJII, T., QUI, D., VINCENT, R., & HOGG, J.C. "Cytokines involved in the systemic inflammatory response induced by exposure to particulate matter air pollutants (PM10)." *Am. J Respir Crit Care Med* 164; 826-830: 2001. Human alveolar macrophages (AM) harvested from bronchial lavage specimens (BAL) from a noninvolved segment or lobe of lungs resected for small peripheral tumours. These were more than 90% viable. All specimens tested for endotoxin contamination. Urban PM10 preparation (EHC 93) came from filters from Ottawa. Identified cytokines also measured in the blood of young army cadets exposed to Asian smoke in April 1998 in Singapore (see {10607}). AM Cells were incubated with residual oil fly ash (ROFA), ambient urban particles (EHC 93) inert carbon particles, and latex particles of different sizes (0.1, 1.0, and 10 microns) for 24 hours. The latex, inert carbon and ROFA particles all showed a similar maximum TNF (tumour necrosis factor alpha) response, whereas EHC 93 showed a greater maximum response that was similar to lipopolysaccharide (LPS). EHC 93 (Ottawa PM10) also resulted in a broad spectrum of proinflammatory cytokines, (IL-6, MIP-1alpha, and GM-CSF), with no difference in the anti-inflammatory cytokine IL-10. Analysis of blood samples taken during the exposure of the army cadets in Singapore to the PM10 from the Asian fires of 1998, showed elevated levels of IL-1beta, IL-6, and GM-CSF during the exposure time. Authors conclude: "These results show that a range of different particles stimulate AM (human alveolar macrophages) to produce proinflammatory cytokines and these cytokines are also present in the blood of subjects during an episode of acute atmospheric air pollution. We postulate that these cytokines induced a systemic response that has an important role in the pathogenesis of the cardiopulmonary adverse health effects associated with atmospheric pollution."

22 DONALDSON, K., STONE, V., SEATON, A., & MACNEE, W. "Ambient particle inhalation and the cardiovascular system; potential mechanisms." *Environmental Health Perspectives* 109 (Supplement 4), 523-527: 2001. Notes that particles increase calcium flux on contact with macrophages. Oxidative stress is likely because of the very large particle surface area, and this can be augmented by oxidants generated by recruited inflammatory leukocytes. Notes that blood viscosity, fibrinogen, and C-reactive protein are elevated on PM10 exposure. Report on their study of elderly individuals in whom PM10 exposure resulted in an increase in C-reactive protein, which is an index of inflammation. Postulate that oxidative stress in the lungs may affect the cardiovascular system by increasing permeability, and by causing atheromatous plaque rupture or endothelial erosion; clotting factors might change and favor thrombus formation. Detailed discussion of blood viscosity and fibrinogen. Suggest that fibrinogen, CRP, and factor VII are part of the acute-phase response mediated by cytokines released during inflammatory reactions. Note that in a survey of 388 British men aged 50-69, the prevalence of coronary artery disease increased 1.5 fold for each doubling of C-reactive Protein level. They have also reported increased **CRP in association with increases in urban PM10. Useful and provocative review.**

23 This list was compiled by one of the most accomplished of fine particle researchers, Dr. Joel Schwartz of Harvard University and presented in testimony before the U.S. Congress. See Testimony of Joel Schwartz, http://www.senate.gov/~epw/105th/schwartz.htm.

24 Pope, C. Arden, III, Michael J. Thun, Mohan M. Namboodiri, Douglas W. Dockery, John S. Evans, Frank E. Speizer, and Clark W. Health, Jr. "Particulate Air Pollution as a Predictor of Mortality in a Prospective Study of U.S. Adults." *Am. J Respir Crit Care Med* 151, 669-74: 1995.

25 Dockery, Douglas W., C. Arden Pope III, Xiping Xu, et al., "An Association Between Air Pollution and Mortality in Six U.S. Cities." *New England Journal of Medicine* Vol 329, 1753–59: 1993.

26 PETERS, A., DOCKERY, D.W., MULLER, J.E., & MITTLEMAN, M.A. "Increased particulate air pollution and the triggering of myocardial infarction." *Circulation* 2001; 103; 2810-2815. 772 patients with MI in Greater Boston interviewed between Jan 95 and May 96. Hourly concentrations of PM2.5, carbon black, and gaseous air pollutants were measured. Case-crossover approach used. For each subject, one case period was matched to 3 control periods exactly 24 hours apart. Conditional logistic regression analyses carried out, with Odds Ratios for a change in air pollution from the 5th to the 95th percentile calculated. Mean age 61.6 years; 164 were under age 50, 365 between 50-69; and 243 over 70 years of age. 63% were male. 31% had a prior myocardial infarction. 72% were "ever smokers" and 32% current smokers. Odds ratios for MI and PM2.5 were above 1.2 for up to 2 hours before onset; and about 1.1 for 3-5 hours before. Using both the values of PM2.5 in the previous 2 hour period, and a delayed response associated with a 24 hour average exposure 1 day before the symptoms, an odds ratio of 1.48 was associated with an increase of 25 micrograms/m3 in PM2.5 during the 2 hour period, and of 1.69 for an increase of 20 micrograms/m3 PM2.5 in the 24 hour period 1 day before the onset. Remarkable to see such a strong effect in what is a relatively small sample.

27 POPE, C.A. III., BURNETT, R.T., THUN, M.J., CALLE, E.E., KREWSKI, D., ITO, K., & THURSTON, G.D. "Lung Cancer, Cardiopulmonary mortality, and long-term exposure to fine particulate air pollution." *JAMA* 2002; 287; 1132-1141. American Cancer Society cohort recruited in 1982. Analysis of over 500,000 people in an average of 51 metropolitan districts. Interesting data showing reductions in PM2.5 from 1979-1983 and from 1999 to 2000, values ranging from 10 to 30 in the first period, and from 5 to 20 in the second. Nonparametric smoothed response functions shown for the three categories of diagnosis; conclude that for a 10 microgram/m3 change in PM10, all cause mortality increased by 4%; cardiopulmonary mortality increased by 6%, and lung cancer mortality increased by 8%. 95% confidence levels of all indices of RR were above 1.0. Coarse particle fraction and TSP not consistently associated with mortality. Other pollutants considered were sulfate, sulfur dioxide, nitrogen dioxide, carbon monoxide, and ozone. Numbers of metropolitan areas that could be considered varied with the different pollutants. Cox proportional hazards model with inclusion of a metropolitan-based random effects component in a two stage analysis. The continuous smoking variables included nine different indices (such as "current smokers years of smoking squared" and eight others). Controls also devised for educational level and occupational exposures. A 2 dimensional term was inserted to account for spatial trends. Higher regressions were noted in men than in women, and lower educational status was associated with higher risks. Risks in never smokers were also

generally higher than in former or current smokers. Authors conclude: "The findings of this study provide the strongest evidence to date that long-term exposure to fine particulate air pollution common to many metropolitan areas is an important risk factor for cardiopulmonary mortality."

28 SAMET, J.M., ZEGER, S.L., DOMINICI, F., CURRIERO, F., COURSAC, I., DOCKERY, D.W., SCHWARTZ, J., & ZANOBETTI, A. "The National morbidity, mortality, and air pollution study: Part II Morbidity and mortality from air pollution in the United States Health Effects Institute." *Research Report: Number 94*, Part II; June 2000 p. 82. 90 cities in different regions of the US, covering all areas. Daily PM10 values given for 1987 to 1994. Also O3, SO2, NO2 and CO. Univariate analysis showed highest values for SO2, with CO second, NO2 third, and PM10 fourth. Distributed lag models give higher values, and authors note that the effects of pollution do not reach 0 until a lag of 5 days has occurred. Authors conclude: "Overall, this study provides strong evidence of association between PM10 levels and exacerbation of chronic heart and lung disease sufficiently severe to warrant hospitalization." Effect of PM10 on mortality generally higher in Northeast, industrial Midwest, and southern California than in other regions.
This study is summarized in the EPA criteria document.

29 SAMOLI, E., SCHWARTZ, J., WOJTYNIAK, B., TOULOUMI, G., SPIX, C., BALDUCCI, F., MEDINA, S., ROSSI, G., SUNYER, J., BACHAROVA, L., ANDERSON, H.R., & KATSOUYANNI, K. "Investigating regional differences in short-term effects of air pollution on daily mortality in the APHEA project; a sensitivity analysis for controlling long-term trends and seasonality." *Environmental Health Perspect* 109; 349-353: (2001).

30 Western European cities: Athens, Barcelona, Cologne, London, Lyon, Milan, and Paris;and five central eastern European cities Bratislava: Cracow, Lodz, Poznan, and Wroclaw--as previously analyzed in the APHEA study. Days with Black smoke > 200 micrograms/m3 were excluded. Poisson regression methods. Loess smoothing regression smoother, which is a generalization of a weighted moving average. Window chosen that minimized Akaike's Information Criterion. Black smoke and SO2 were the pollutants analyzed. Increase in BS of 50 micrograms/m3 associated with 2.2% increase in mortality when analysis restricted to days with BS less than 200, and 3.1% increase if restricted to days with BS less than 150 micrograms/m3. SO2 effect for similar increase was 5.0% and 5.6% respectively. This increase occurred only in the eastern European cities. Authors conclude that "part of the heterogeneity in the estimates of air pollution effects between western and central-eastern cities reported in previous publications was caused by the statistical approach used and the inclusion of days with pollutant levels above 150 micrograms/m3."

31 BURNETT, R.T., BROOK, J., DANN, T., DELOCIA, C., PHILIPS, O., CAKMAK, S., VINCENT, R., GOLDBERG, M.S., & KREWSKI, D. "Association between particulate- and gas-phase components of urban air pollution and daily mortality in eight Canadian Cities." *Inhalation Toxicology*, 12 (Supplement 4): 15-39, 2000. Analysis of time series data from Montreal, Ottawa-Hull, Toronto, Windsor, Winnipeg, Edmonton, Calgary, and Vancouver. Important feature is detailed compositional analysis of the PM10 collected in

each city. PM2.5 a stronger predictor of mortality that PM10-2.5. Size-fractionated particulate mass explained 28% of the total health effect of the mixture, with the remaining effects being accounted for by the gases. Carbon represented half the mass of the particulate matter. 47 elemental concentrations reported. Good discussion of statistical issues and notes that slightly stronger associations resulted from coadjustment method compared to pre-adjustment of both mortality and air pollution data. Correlations between pollutants are given. Separate risk relationships not given for different cities, but pollutant values are given for each city. Sulfur showed the highest correlation with fine mass, with Pb, Si, FE, K, Zn, Mn, P and SE modestly correlated with PM2.5. Both particulates and gases associated with mortality.

32 Wollf G.T., In Response to the PM Debate, Regulation 20:1, 1997. The author, a General Motors Corporation scientist chaired the U.S. Environmental Protection Agency's Clean Air Science Advisory Committee.

33 (1)Schwartz J. "Is there harvesting in the association of airborne particles with daily deaths and hospital admissions?" *Epidemiology* 2001; 12:55-61. (2) Zanobetti A, Schwartz J, Samoli E et al. "The temporal pattern of mortality responses to air pollution: a multi-city assessment of mortality displacement." *Epidemiology* 2002; 13:87-93. (3) Zeger SL, Dominici F, Samet J. "Harvesting-resistant estimates of air pollution effects on mortality." *Epidemiology* 1999; 10:171-175. (4) Schwartz J. "Harvesting and long term exposure effects in the relation between air pollution and mortality." *Am J Epidemiol* 2000; 151:440-48.

34 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/m3 increase in particles = 1.95; 1.74 for SO2; and 1.66 for NO2. 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 SO2 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 NO2 were 1.55; 1.71;; 1.60; & 1.78. Respiratory deaths numbered about 133. Important observations.

35 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 SO2, CO, PM10, 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, SO2, and PM10 when considered individually, were around 15%, 13%, and 7% respectively. Reliability of death certification process is described.

36 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 4-8 fewer infant deaths per 100,000 live births at the county level, for a 1 microgram/m3 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 1982-83. At the same time, in 1978-80 infant deaths within one year of birth attributable to "internal" causes (eg respiratory and cardiopulmonary deaths) were stable and occurred at the rate of 1315-1380 per 1000,000 live births. But from 1980-82, the internal infant mortality rate declined from 1315 to 1131, and remained at this lower level in 1983-84. While not controlling for all changes that may have occurred in the absence of the pollution decline, these numbers imply that a 1 microgram/m3 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/m3 decline in suspended particulates associated with about 4-8 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.

37 Dockery, D. W., and C. A. Pope III. "Acute Respiratory Effects of Particulate Air Pollution," *Annual Review Public Health, 1994*, vol 15, 1994, 107–32.

38 Euler GL, Abbey DE, Magie AR, Hodkin JE. "Chronic obstructive pulmonary disease symptom effects of long term cumulative exposure to ambient levels of total suspended particulates and sulfur dioxide in California Seventh-Day Adventist residents." *Arch Environ Health* 1987; 42:213–22.

39 Ware J, Ferris Jr. B, Dockery D, Spengler J, Stram D, Speizer F. "Effect of ambient sulfur oxides and suspended particles on respiratory health of preadolescent children." *Am Rev Respir Dis* 1986;133:834–42.

40 Dockery DW, Speizer FE, Stram DO, Ware JH, Spengler JD. "Effects of inhalable particles on respiratory health of children." *Am Rev Respir Dis* 1989; 139:587–94.

41 Bates DV, Sizto R. "Air pollution and hospital admissions in southern Ontario: The acid summer haze effect." *Environ Res* 1987;43:317–31.

42 Samet JM, Speizer FE, Bishop Y, Spengler JD, Ferris Jr BG. "The relationship between air pollution and emergency room visits in an industrial community." *J Air Pollut Control Assoc* 1981; 31:236–40.

43 C. Arden Pope III, "Respiratory Disease Associated with Community Air Pollution and a Steel Mill, Utah Valley." *American Journal of Public Health*, Vol. 79 (May 1989), pgs. 623-628.

44 Beck BD, Brain JD, Bohannon De. "The pulmonary toxicity of an ash sample from Mt. St. Helens volcano." *Exp Lung Res* 1981; 2:289–301.

45 Beck BD, Brain JD, Wolfthal SF. "Assessment of lung injury produced by particulate emissions of space heaters burning automotive waste oil." *Ann Occup Hyg* 1988;32:257–65.

46 Beck BD, Brain JD. "Prediction of the pulmonary toxicity of respiratory combustion products from residential wood and coal stoves." In: eds. *Residential Wood and Coal Combustion*. Louisville, Ky: Air Pollution Control Association, 1982; 264–80.

47 Beck BD, Brain JD, Bohannon DE. "The pulmonary toxicity of an ash sample from Mt. St. Helens volcano." *Exp Lung Res* 1981;2:289–301.

48 SCHWARTZ, J., NORRIS, G., LARSON, T., SHEPPARD, L., CLAIBORNE, C., & KOENIG, J. "Episodes of high coarse particle concentrations are not associated with increased mortality." *Environ Health Perspectives* 1999; 107; 339-342. Gary Norris' PhD thesis work showing that mortality in Spokane was associated with PM10 when this rose from vehicle sources, but was not raised when the PM10 increases were due to duststorms.

49 Centers for Disease Control and Prevention, *National Vital Statistics Report* 48:11, Table 12, http://www.cdc.gov/nchs/.

50 Section 109(d)(1) of the law requires the Administrator "at five-year intervals...make such revisions in such criteria and standards and promulgate such new standards as may be appropriate....The Administrator may review and revise criteria or promulgate new standards earlier or more frequently than required under this paragraph."

51 See, e.g., U.S. Department of Justice, Press Release, "U.S. FILES CLEAN AIR LAWSUIT AGAINST DUKE ENERGY," Dec. 22, 2000, http://www.usdoj.gov/opa/pr/2000/December/710enrd.htm.

52 JEFF NESMITH, "Bush Announces New Air Pollution Plans," Cox Newspapers, Feb. 15, 2002, http://www.coxnews.com/washingtonbureau/staff/nesmith/.

53 According to one press account, "(EPA Administrator Christine Todd) Whitman says her agency's staff is busy developing provisions to replace the New Source Review program." Continuing, "At a congressional hearing in March, Whitman noted that if Congress approves Clear Skies, the NRS program would be "redundant," with respect to power plants." "Clear Skies" may be ahead for electric power plants." *Environmental Science and Technology*, April 3.

54 LEVY, J.I., SPENGLER, J.D., HLINKA, D., SULLIVAN, D., & MOON, D. "Using CALPUFF to evaluate the impacts of power plant emissions in Illinois: model sensitivity and implications." *Atmospheric Environment* 36 (2002) 1063-1075. Nine power plants in Illinois studied using the CALPUFF model and meteorological data from NOAA's Rapid Update cycle model. Population-weighted annual average concentration increments associated with current emissions were 0.04 micrograms/m3 of primary PM2.5, 0.13 micrograms/m3 of secondary sulfate particles, and 0.10 micrograms/m3 of secondary nitrate particles. Plots of calculated impacts plotted against distances from the source. Maximum increment of 0.6 micrograms/m3 close to the facilities. Attached note gives basis of calculating avoided health impacts per year if lower target emission levels were reached: Mortality 190: Emergency room visits 2,532; Asthma attacks 13,290;Restricted activity days 168,900.Each of these is broken down by county. Table of estimated emission rates for each of the nine plants. 40% of the primary PM2.5 total exposure is located within 50 Km of the power plants. Population at risk is 18 million age 30 or older. Authors note: "Pre-1980 coal-fired power plants currently contribute about half of the electricity generation in the US and are responsible for 97% of power plant SO2, and 85% of power plant NO2 emissions"

55 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: 675-679; 1999520. Nonsmoking Yale students examined. Symptoms of cough, phlegm, wheeze apart from colds, and composite respiratory symptom index (RSI) used to assess symptoms. FVC, FEV1 and flow rates recorded. High exposure group were those who had lived for four or more years in a US county with a 10 year average summer season daily 1 hour maximum ozone of > 80 ppb. Controlling for covariates, those from higher ozone region had lower FEV1 (-3.1%), FEF25-75 (-8.1%) and nearly significant FEF75 difference. Stronger associations for men than for women. All symptoms were increased in higher ozone group, with Odd Ratio of 2.0 for RSI package. Wheeze had odds ratio of 1.97. High ozone areas were in Arizona; Los Angeles; Connecticut; Maryland; New Jersey; New York; and Utah. Note that in men, deficit in

FEF25-75 was – 13%; and in FEV1 was – 4.7%:In women, ozone exposure was associated with lower FEV1, (- 0.26%) , FEF25-75 (- 1.96%), and FEF75 (- 2.0%) but the differences did not reach statistical significance.

56 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 O3 was associated with an increase of 62.9% for illness-related absence rates, 82.9% for respiratory illnesses, 45.1% for upper respiratory illnesses, and 173.9% 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 O3 peak and fell off slowly after that. .

57 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.

58 MISHRA, V.K., RETHERFORD, R.D., & SMITH, K.R. "Biomass cooking fuels and prevalence of tuberculosis." *India Int J Infect Dis* 1999; 3; 119-129. Analysis of 260,162 people aged 20 and over in a Family Health Survey conducted in India in 1992-3. "Results strongly suggest that use of biomass fuels for cooking substantially increases the risk of tuberculosis in India."