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Analysis of Ozone Layer Components

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Ozone O3 is formed by processes involving nitrogen oxides NOx and volatile organic compounds VOCs, which are catalyzed by UV radiation from sunshine. Each VOC may generate varying levels of ozone. One kilogram me of ethane, for example, creates half the quantity of ozone as one kilogram me of formaldehyde. Ozone levels are typically maximum on warm, bright, windless days. Ozone concentrations peak in the morning and/or afternoon and fall in the evening during rush hour. Motor vehicles approximately 40% of emissions; industrial activities, notably the chemical and petroleum sectors; and any usage of paints, varnishes, and solvents another 40% for these sources combined are among the sources of VOCs. Other notable producers of VOCs are service stations, pesticide application, dry cleaning, fuel combustion, and open burning [1].

In adults and children, ozone is a potent oxidant and respiratory tract irritant, producing shortness of breath, chest discomfort when breathing deeply, wheezing, and coughing. Ozone levels have decreased by 21% since 1980 as the Environmental Protection Agency EPA and state and municipal governments collaborated to enhance air quality. On the effects of O3 on respiratory symptoms, respiratory function, and airway inflammation in humans, a considerable and relatively consistent body of evidence has gathered, particularly on transitory reactions to acute exposure. Mucociliary and early alveolar zone particle clearance, functional responses in macrophages and epithelial cells, and alterations in lung cell secretions are among the other lung function responses to acute and subacute exposure that have been examined, mostly in animals. Sub chronic and chronic animal exposure methods have been linked to structural alterations in the smaller conductive airways and the more proximal gas exchange area. Chronic impacts might occur as a consequence of accumulated harm or as a result of adaptive mechanisms to repeated daily or intermittent exposure.

Health Effects

Pulmonary Function

During forced expiratory movements, inhaling O3 induces concentration-dependent mean decreases in exhaled volume and flow rate. David Bates, M.D., demonstrated in 1972 that patients exposed to ambient O3 levels while exercising had alterations in lung function. 2 Mortimer et al.3 discovered that a two-day lag had the largest impact on peak expiratory flow rate PEFR in a trial of 846 urban children ages 4-9 years with asthma, with a five-day dispersed lag having an even higher effect. They gathered data from eight cities in the United States with an O3 mean of 48 ppb and a high of 58 ppb. O3 or NO2 levels were linked to respiratory symptoms [2].

It is also well documented that repeated daily one- or two-hour doses at a level that provides a functional response with a single exposure result in an improved reaction on the second day, decreased responses on days 3 and 4, and almost no response by day 5.4,5 This functional adaptation to exposure goes away around a week after the exposure stops. Multiday exposures also reduced inflammation, as measured by polymorphonuclear cells PMNs, fibronectin, and IL-4 in lavage fluid. 6 Peak functional response occurred on the first day of recurrent 6.6 hour per day exposure to 120 ppb O3, with progressively lower responses

on the second, third, and fourth days of exposure. Nevertheless, methacholine challenge reactivity peaked on the second day and remained higher for the whole five days of exposure, and there were long-term abnormalities in small airway function. O3 might produce an increase in reactivity in asthma sufferers. The long-term alterations in airway responsiveness and small airway function have a significant health impact.

Kinney and colleagues measured lung function in 154 Tennessee kids up to six times during a two-month period in late winter and early spring. Significant relationships were seen in child-specific regressions of lung function vs maximal 1-hour O3 concentration the previous day. While children at school are predicted to have low activity levels, the unusually high response coefficients might be due to potentiation by other pollutants or a lack of seasonal adaptation. Kingston-Harriman, Tennessee, is well-known for its high levels of aerosol acidity. Decreases in peak expiratory flow rates were linked with ambient O3 concentrations in a study of children with moderate to severe asthma at a summer camp in the Connecticut River Valley. Asthmatic children had low levels of physical activity, therefore their O3 intake was poor, and they had reduced reserve functional capacity. The degree of health risk associated with these functional deficiencies is significant. Field investigations of functional reactions of individuals participating in outdoor recreational activities in the presence of various amounts of O3 have also been conducted. Spektor et al. measured pre- and post-exercise respiratory performance in thirty young individuals who participated in daily outdoor exercise for about one-half hour per day in an area with regional summer haze but no local point sources. The magnitudes of the functional decrements per unit of ambient O3 concentration were comparable to those seen in controlled chamber exposure trials with participants exposed while exercising hard for one or two hours.

Functional decrements proportional to ambient O3 concentrations have also been seen in elite cyclists in the Netherlands11 and hikers on Mount Washington in New Hampshire. Hikers with a history of asthma or wheezing exhibited four times the reactions to O3, PM2.5, and aerosol acidity as the rest. Pollutant levels were fairly low on average, with O3 averaging 4.0 ppb, PM2.5 at 10 mg/m3, and acidity at 0.3 mg/m3 H2SO4 equivalent. Brauer observed that throughout the summer, lung function in fifty-eight outdoor agricultural labourers in British Columbia decreased by a mean of 54 ppb and a maximum of 84 ppb. When tested twice a day for two weeks in the summer, ozone was linked to lower evening peak expiratory flow rates, with a five-day cumulative lag exposure demonstrating the highest impact in 473 nonsmoking women ages 19-43 years in Virginia. The morning peak expiratory flow rate decrements were associated to PM2.5 and acid exposure. During six weeks, mail carriers had their PEFR assessed twice a day; the night PEFR drop was linked to 8-hour O3 levels with a lag of 0-2 days, adjusting for PM, temperature, humidity, sex, age, and illness status.

Airway Reactivity and Inflammation

Ozone is an irritating gas that enhances airway reactivity, which may be assessed by response to methacholine inhalation. Airway hyperreactivity does not affect all participants, and asthmatic and atopic patients may be more vulnerable. Ozone may promote bronchial hyper-reactivity by interacting with particles and other gaseous contaminants. Horstman and colleagues18 found that 6.6-hour exposures to 80, 100, and 120 ppb increased methacholine responsiveness by 56, 89, and 121% in healthy participants, respectively. Seven asthmatic patients were treated to 0.12 ppm ozone for one hour before being exposed to allergen; the low dosage of O3 enhanced bronchial reactivity to allergen, lowering the quantity of allergen necessary to elicit a 15% reduction in FEV1.

Jorres et al. subjected twenty-four patients with moderate stable allergic asthma, twelve subjects with allergic rhinitis without asthma, and ten healthy subjects to 250 ppb O3 or filtered air FA for three hours while doing intermittent activity. As compared to filtered air, FEV1 reduced by 13% in asthma patients, and the dosage of methacholine or allergen more than doubled. When O3 or filtered air were followed by allergen inhalation in patients with rhinitis, mean FEV1 dropped by 7.8% and 1.3%, respectively. Seltzer and colleagues exposed 10 healthy people to 0.4 or 0.6 ppm ozone or air and then performed bronchoalveolar lavage BAL 3 hours later. Ozone exposure enhanced bronchial hyperreactivity,

methacholine responsiveness, neutrophils and prostaglandins in the BAL, and reactions were larger with higher ozone doses. In its Research Triangle Park, North Carolina, location, the EPA developed a Health Effects Research Laboratory where ozone-induced inflammation in the lower airways could be studied directly using technologies such as bronchoalveolar lavage. They found an exposure-response association at three levels of exposure, with inter-individual heterogeneity.

They started with 0.4 ppm O3 exposure for two hours with activity, followed by BAL the following day on eleven healthy people. Neutrophils rose eightfold, while BAL fluid biochemical alterations included increases in neutrophil elastase, fibronectin, and prostaglandins. They also performed a nasal lavage NL and found an increase in neutrophils both immediately after the NL and the next day at the time of the BAL. The O3 dosage was then reduced to 0.10 or 0.08 ppm O3, with exercise lasting 6.6 hours and followed with BAL the following day. 24 Both degrees of exposure produced lower respiratory tract inflammation, with increased neutrophils, prostaglandins, fibronectin, interleukin-6, and reduced alveolar phagocytosis. Aris and colleagues verified these investigations at the University of California-San Francisco, where fourteen individuals were exposed to 0.20 ppm O3 for 4 hours with exercise and BAL of the proximal airway done to test the irritating nature of O3 gas. 25 They discovered a rise in neutrophils as well as the same biochemical markers, including interleukin-8, which is chemotactic for neutrophils.

They also performed mucosal biopsies for histology, confirming neutrophil infiltration following O3 exposure vs filtered air. The time course of O3-related effects after one hour of exposure to 0.3 ppm on three different days revealed that FEV1 decreased immediately; proximal airway neutrophilia peaked at 6 hours and lasted until the following day. Kinney and colleagues reported on fifteen United States Coast Guard joggers on Governors Island in New York Harbor, comparing ambient summer ozone exposure to that in the winter using BAL. 27 Summer BAL fluids included greater amounts of lactic dehydrogenase, IL-8, and prostaglandin E2. Asthmatics may be more vulnerable to the irritating effects of 6 hours of O3 inhalation and exercise. Five asthmatics and five healthy people were subjected to 0.20 ppm O3 in chamber experiments and had no change in FEV1, but had substantial increases in neutrophils, IL-6, and IL-8 levels the following day. There was significant inter-individual heterogeneity in these chamber trials, and methacholine tests for bronchial hyper-reactivity were unable to predict who would respond to O3. There was no additional effect from allergen exposure including pulmonary function, BAL neutrophils, or cytokines in a study of fourteen asthmatics exposed to O3 at 0.20 ppm for 1 hour with exercise, indicating a negative study, but a subgroup of nine individuals were more sensitive to allergen exposure and had more neutrophilia. A linear association between neutrophils at 6-hour or 18-hour post-O3 exposure BALs based on a meta-analysis of twenty-one papers [3], [4].

Two weeks of inhalation budesonide a corticosteroid pretreatment with 800 mg twice a day showed no protection against inhaled O3 in terms of pulmonary function, methacholine reactivity, or neutrophil recruitment. investigated the pulmonary effects of O3 on healthy persons with and without antioxidant supplementation and found that antioxidants mitigated the O3-induced functional decrements but not its impact on raising neutrophils and IL-6 in BAL fluid. In vitro, one-hour exposures of alveolar macrophages under 0.4 ppm O3 enhanced the production of inflammatory cytokines IL-1b, IL-6, IL-8, and TNF-a roughly fourfold above control without affecting cell survival.

Rats given a single dosage of O3 displayed enhanced proliferation of bronchial and alveolar epithelial cells, as determined by proliferating cell nuclear antigen PCNA. Corticosteroids lowered this from 19.2% to 10.9% p 0.5 and decreased neutrophil influx. Using transgenic knockout mice exposed to O3 in chamber tests, matrix metalloproteinases MMPs were shown to have a role in O3-mediated lung damage, with MMP-9 protecting but MMP-7 not. Moreover, the tumour necrosis factor-a receptor, the key transcription NF-kB, and the signalling routes to 6the nucleus were all required for O3s inflammatory effects, as shown by O3 treatment in transgenic mice missing these genes.

Epidemiological Studies of Populations Exposed to Ozone in Ambient Air databases such as the Georgia Medicaid claims file and peak daily O3 levels During the games, concentrations fell by 28% and traffic fell by 23%. An An epidemiologic study of 271 asthmatic children aged 12 years who were attending a

summer camp was conducted. A 50 ppb rise in 1 hr O3 was associated with respiratory symptoms wheeze increased by 35% and chest tightness increased by 47% at a camp in southern New England. O3 levels greater than 0.063 ppm during an 8-hour peak on the same day were linked with a 30% increase. Chest stiffness increased OR 1.64, 95% CI 1.23-2.17. There were also one-day delays. persistent coughing with shortness of breath OR 1.33, 95% CI 1.09–1.62. These findings were only significant for the half of the group that utilised

Medication for maintenance. Increasing bronchodilator usage was linked to maximum concentration of same-day O3. No respiratory symptoms nor bronchodilators were present. PM2.5 levels were linked to marijuana usage. As a result, asthma severity might be used to categorise the group. O3 has two degrees of sensitivity to air pollution. A two-year longitudinal investigation of In Detroit, inner-city schoolchildren discovered a startling 8-hour peak O3 level. connection with upper respiratory illness, while the subgroup of corticosteroid-treated asthmatic children exhibited substantial correlations with both O3 and PM. decreased FEV1. The majority thirty-one of Atlantas hospital emergency rooms took part in the Atlanta Particles and Health Study SOPHIA Between 1993 and 2000, 11% of 4.5 million ER visits were related to respiratory problems. PM10, O3, NO2, and CO were each linked to 1%-3% of the causes.

increases in visits for upper respiratory infection per standard deviation rise in A 20 ppb rise in NO2 was related with a 3.5% increase in Visits for chronic obstructive pulmonary disease COPD. The asthma risk ratios Visits were strongest with delays of five to eight days, and with O3 at There are one- and two-day delay. Asthma connections were greater in the summer months in the case of O3 and PM2.5. Hospitalizations for asthma in children from birth to age California Air Resources Board home air pollution levels were associated to 19 years. From 1983 until 2000, the Resources Board discovered a time-independent, constant the relationship between ambient O3 levels and quarterly hospital discharge for asthma Reduced lung function and increased respiratory symptoms, such as

Asthma aggravation occurs with rising ambient O3, particularly in children. Burnett et al.49 found that the impact was highest at a one- or two-day lag in an examination of respiratory hospital admissions in fourteen Canadian cities but the biggest of all for a four-day dispersed lag. They researched hospitals. Admissions for acute respiratory hospitalizations in Toronto from 1980 to 1994 2 year old youngsters utilising daily time series to adapt for day affects

of the week, season, and weather. The increase was 35% CI 19-52%. Throughout May to August, daily admissions for respiratory diagnoses were related with a 5-day moving average daily 1-hour maximum O3 concentration of 0.045 ppm. August. Newborn respiratory morbidity was discovered in eleven Canadian cities. to be associated with gaseous pollutants, accounting for 9.61% of all pollutants combined. Modifying variables such as ambient temperature, aeroallergens, and other co-pollutants such as particles may all play a role in this association. Summertime hospital admissions and emergency department visits for respiratory problems may be attributed to ozone air pollution. According to this research, O3 may contribute for one to three more summertime respiratory hospital admissions every 100 ppb O3, per million people. Yang et al recently observed substantial connections between O3 respiratory hospital admissions and children under the age of three and the elderly in Vancouver, Canada, where the 24-hour average O3 concentration was just 13 ppb.

Mortality

In a Denver epidemiological research conducted between 1993 and 1997, daily measurements of temperature, PM10, and gaseous pollutants were compared to contemporaneous data on hospital admissions for those over the age of 65. According to the findings, O3 was linked to an increased risk of hospitalisation for acute myocardial infarction, coronary atherosclerosis, and pulmonary heart disease. A cross-sectional study of thirty-six US cities collected respiratory hospital admissions as well as ozone and PM10 data from 1986 to 1999.

During the warm season, a 5 ppb rise in O3 resulted in a 0.27% 95% CI 0.08-0.47% increase in COPD admits and a 0.41% 95% CI 0.26-0.57% increase in pneumonia admissions. At the same time period, a 10 mg/m3 rise in PM10 caused increases in COPD of 1.47% and pneumonia of 0.84%. Thurston and Ito examined data from previous time-series mortality studies. had not taken into account the ambient temperature. The combined study resulted in a14 1.036 for every 100 ppb rise in daily 1-hour maximum O3 95% CI 1.023- 1.050. The subgroup of research that defined the nonlinearity of the

The temperature-mortality relationship generated a total estimate of RR 14 1.056. per 100 ppb 95% confidence interval: 1.032-1.081. This suggests that previous time-series studies The impacts of O3 air pollution on premature mortality have been underestimated using linear temperature-mortality parameters. Weather control is required for O3 research. This is especially problematic since high O3 days are often rather hot. Schwartz, He employed a case-crossover technique, matching a day by temperature with O3 was used as the independent variable in a study of more than He discovered that when matching on temperature, there was an association of 0.23% CI 0.01-0.44% with a 10 ppb rise in maximum hourly O3 concentrations. The discovery was limited to the summer months, was unaffected by PM, and was comparable in size to seasonal matching and temperature control using regression splines. Findings O3 epidemiology studies are essential since they show an increase in mortality.

While developing a health standard, mortality dominates the cost-benefit assessments. The NMMAPS National Mortality and Morbidity Air Pollution Study was employed. Data about ambient O3 from the EPAs Atmospheric Information Retrieval System AIRS. A positive connection was identified in all but two localities, and a statistically significant association was revealed for seven communities and the ninety-five as a whole. The 95-community impact was largest on the same day, and significantly important for one- and two-day delays, and considerably more so when The dispersed lag of six days was taken into account National and international Estimates of the short-term effects of O3 on mortality by community were not affected by the addition of PM10 or PM2.5 in time-series models. The EPA hired three different groups of scientists to complete the work.

Nitrogen Oxides

Since nitrogen dioxide NO2 is a predecessor to the creation of O3, controls for the O3 standard must account for this important NAAQS-regulated pollutant. Since there are additional gases than nitrogen dioxide, it is often referred to as NOx. Nitric oxide NO, nitrous oxide N2O, nitro peroxide NO2 and N2O4, and nitrogen trioxide N2O3 are also implicated. Inner cities with automotive and truck traffic, electrical utilities, refineries, and gas-fired interior ranges are also potential sources. Between 1980 and 2006, annual average outdoor concentrations declined by 41%, and average maximum hourly amounts in the United States were about 30 ppb, with maxima reaching 200 ppb, particularly along high-traffic roadways. In 1971, an annual average norm of 53 ppb was established.

They are irritating gases, much as ozone. Normal subjects exposed to NO2 levels of 7,500 ppb for 1-2 hours in a chamber exhibited enhanced nonspecific airway hyper responsiveness. 64 After 30-60 minutes of exercise, levels as low as 300 ppb might decrease pulmonary function in asthmatics. COPD patients exhibit higher airway resistance during exercise at 1,600 ppb. Indoor NO2 exposure from petrol stoves has been linked to an increase in respiratory infections in children under the age of two in selected Harvard Six Cities Study families. There was no difference in pulmonary function, however wheezing and breathing problems were higher in homes using electric stoves. Asthmatic youngsters in Tucson, Arizona, had considerable decreases in the peak flow brought on by petrol burners. According to a meta-analysis of paediatric respiratory illnesses linked to NO2 exposure, there is a 20% increase in the likelihood of developing a respiratory ailment for every additional 15 ppb of indoor NO2 exposure. Epidemiology studies connected elevated NO2 levels to more emergency hospital visits, higher asthma symptoms, and worse lung function. Clinical research showed that asthma patients had higher airway response to allergen exposure. 68 The EPA published a new 1-hour NO2 standard in January 2009 at a concentration of 100 ppb. The 1-hour standard would take on the shape of the 98th percentile of the yearly distribution of daily maximum 1-hour average concentrations, averaged over three years.

Sulfur Dioxide and Acid Rain

Sulfur dioxide SO2 is a colorless gas that is highly soluble and reactive. Electric power plants account for approximately 65% of SO2 emissions into the atmosphere, with the remaining 35% coming from metal processing, industrial sources, and fuel combustion sources. SO2 can In water or water vapour, dissolve to form various acidic sulphates. Point sources such as smoke stacks from industrial and power plants disperse SO2 in a 20-kilometer radius, resulting in 1-hour average levels of 0.2-0.3 ppm in North America. Between 1983 and 2002, the outdoor ambient SO2 concentration decreased by 54%, owing primarily to reductions in power plant and industrial emissions. Emissions have decreased from 31,161,000 tonnes in 1970 to 18,867,000 tonnes in 1999, owing primarily to flue gas desulfurization from the addition of calcium oxide lime, which reacts with SO2 to form calcium sulfite [5], [6]. Following the 2007 diesel truck regulations, more stringent regulation limits on the amount of sulphur in fuels will reduce ambient SO2. Indoor space heaters that use kerosene are a source of SO2 pollution in the home. Because ambient SO2 levels have decreased in comparison to ozone, particulate matter PM, and hazardous air pollutants, there has been less importance placed on controlling SO2 for health effects.

SO2 is a key chemical precursor in the formation of sulfate-containing PM and acidic aerosols acid rain, both of whom have significant environmental consequences. mixtures of combustion-related gases in the air, such as SO2 from power plants and industrial facilities, and nitrogen.1 Oxides from power plants, automobiles, and other sources of combustion cause Nucleation, or the formation of fine and ultrafine particles in the atmosphere. Secondary PM formation, with the recognised precursors primarily SO2 and nitrogen oxides, accounts for a significant portion of total PM exposure in the environment.

United States of Americas northeast. SO2 can combine with industrial ultrafine particles to produce form larger fine particles. Only 2% of SO2 absorbed or scrubbed in the nasal turbinates reaches the glottis. This improves with mouth exercise. breathing, where SO2 can be absorbed in the respiratory aqueous lining It can pass through the epithelium and reach the lower respiratory tract, where its irritant properties can be felt. Bronchoconstriction and airway inflammation may occur. Acid rain is a broad term that refers to a combination of wet and dry deposition. Deposited material from the aerobic environment higher than normal concentrations of amounts of nitric and sulfuric acids. The chemical forerunners, or precursors, of Acid rain is caused by both natural sources such as volcanoes and decaying vegetation, as well as man-made sources such as sulphur dioxide SO2 and nitrogen oxides NOx emissions from fossil fuel combustion. In the

In the United States, approximately two-thirds of all SO2 and one-quarter of all NOx are produced. from electric power generation that relies on burning fossil fuels like coal. Acid rain occurs when these gases react in the atmosphere with water, oxygen, and other elements. and other chemicals to produce a variety of acidic compounds. As a result, there is a mild A sulfuric acid and nitric acid solution. When sulphur dioxide and nitrogen oxides are present are emitted by power plants and other sources, and the prevailing winds blow these compounds that span state and national borders, sometimes hundreds of miles miles. Acid rain can be harmful to plants, aquatic species, and humans. Infrastructure.

SO2 Health Effects

SO2 associated with exposure to increased mortality in several studies. due to all causes and to cancer specifically. For instance, consider the study of 6The relative risk RR of all-cause mortality in the American Cancer Society cohort that reported the link between mortality and criteria air pollutants 1.25 95% CI 1.13-1.37 from sulphate exposure

and was higher at the county level, with an RR of 1.5.2. The National Mortality and Morbidity Air Pollution Study NMMAPS also investigated SO2 and discovered no significant associations with total mortality.3 A study of 40,704 SO2-exposed pulp and paper workers discovered a However, the overall standardised mortality ratio was reduced to 0.89 95% CI 0.87-0.96. lung cancer has a marginally higher

rate of 1.08 95% CI 0.98-1.18.4 6The lung cancer risk was reduced after adjusting for occupational co-exposures. increased when compared to unstressed workers rate ratio 14 1.49; 95% CI 1.14–1.96. There was some evidence of a positive relationship between The relationship between cumulative SO2 exposure and lung cancer mortality has been studied. These findings support the notion that SO2 exposure increases mortality. SO2 is a respiratory irritant at 10 ppm, causing cough, dyspnea, eye and throat irritation, and reflex bronchial constriction. The month of July Hong Kong mandated in 1990 that all power plants and road vehicles use fuel oil with a sulphur content of no more than 0.5% by weight.

There was a decrease in seasonal deaths over the next twelve months. by a cool season death rate peak between thirteen and twenty-four months, before resuming the expected pattern in years 3-5. There were decreases. In the average annual trend, deaths from all causes 2.1%, p 14 0.001, respiratory 3.9%, and cardiovascular 2.0% were observed. The average increase in life expectancy per year The exposure to the lower pollutant concentration was twenty days per year for Females have forty-one days, while males have forty-one days. In the two years following the intervention. In children, there was a reduction in chronic bronchitic symptoms and bronchial hyperresponsiveness. Over a five-year period, SO2 decreased by 45%, and respirable Particulates have been decreasing for two years. Daily SO2 concentrations were significantly associated with daily mortality in twelve Canadian cities, with an average concentration of only 5 mg/L. m3 Daily mortality in a district of Chongqing, China, was studied from for associations with daily ambient sulphur from January to December 1995 dioxide and fine particles. Particulate matter with a diameter of less than 2.5 mm. PM2.5 was monitored for seven months, while SO2 was monitored for the same length of time.

The researchers discovered a link between daily ambient SO2 levels and respiratory and cardiovascular mortality. disease. For example, consider the impact of a 100 mg/m3 0.04 ppm increase in daily intake. SO2 levels were associated with a relative risk of 1.20 95% CI 1.11-1.30 of cardiovascular mortality, with a three-day lag. The SO2 connection remained. When PM2.5 is controlled, the system becomes more robust. There were no correlations found between The daily ambient PM2.5 intensity and any cause of death. a shortcomingThe absence of measurements of carbon monoxide, ozone, and other pollutants was a limitation of this study. or nitrous oxide. Chongqing is surrounded by trees, has a population of 30 million people, and relies on high-sulfur coal for energy. Sulfur levels range from 4% to 12%.

The European Air Pollution Health Effects Approach was discussed in a report. Sunyer et al. investigated the short-term impacts of SO2 levels on cardiovascular disease hospital admissions in seven European countries. Daily Admissions for cardiovascular diseases increased by 0.7% 95% CI: 0.1 to 1.3 per 1,000. Specifically, ischemic heart disease increased significantly with increased SO2 levels of the same day and the day before, with each 10 mg/m3 increase of SO2. subjects under the age of 65. Even after accounting for, this was still significant. PM10 variations. Study participants over 65 years old had significant daily cardiovascular Admissions for PM10 alone 1.3%, CI 0.7-1.8 for each 10 mg/m3 increase in PM10. This study demonstrates that SO2 pollution can cause ischemic heart disease. A study of 7,319 admissions for asthma in kids aged 6 to 15 in Toronto Sulfur dioxide concentrations were related to asthma hospitalizations in girls and CO exposure and asthma hospitalizations in boys over a 12-year period from 1981 to 1993.9 Nitrogen dioxide was positively related to asthma. acceptance of both sexes. For CO and NO2, the lag time was two to three days. for boys, and six to seven days for SO2 and NO2 with girls. These results

After PM adjustment, remained. Using information from the Harvard Measurements of pollutants at the time were total, according to the Six Cities Study. total suspended particulates that are less than [TSP], total suspended particulates, and all three pollutants were significantly linked to cough in preadolescent children. TSP was linked to bronchitis and lower respiratory illnesses. In South Korea, Lee et al. assessed the connections between low birth weight and air pollution.11 Low birth weight tended to rise with CO exposure [7]–[9]. Pregnancy exposure to SO2 and NO2 occurs between months 3 and 5, PM10 between months 2 and 4, and PM10 between months 2 and 5. a historical cohort 92,288 full-term births were examined in a Taiwan study in relation to SO2 exposure. in the initial trimester. Higher exposure levels,

greater than 0.011 parts per million in term pregnancies is linked to a 26% rise in the risk of low birth weight, versus SO2 exposures less than 0.007 ppm.12 No significant elevated

Risk for other air pollutants was noted. studies utilizing human exposure chambers exposing healthy subjects to at 1-2 ppm, the effects of SO2 include a decrease in the forced expiratory volume in Asthmatics exhibit bronchoconstriction between 0.5 and 0.1 ppm, respiratory symptoms, and 1 second FEV1. exposure to sulfuric acid aerosol in non-smokers Exercise in healthy volunteers did not result in an inflammatory response as measured by bronchoalveolar lavage, and there was no indication that antimicrobial defence had changed.13 Carlisle and Sharp reviewed the function of athletic training. exercise can make it more dangerous to be exposed to ambient air pollution outside, especially PM, volatile organic compounds, ozone, nitrogen oxides, and carbon monoxide.

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