Implications of Air Pollution Effects on Athletic Performance

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Atmospheric Chemistry Background

There are a large number or chemical compounds that are present in a polluted atmosphere and that alone or in combination are important to consider for their potential effect on the respiratory system and impact on athletic performance. A general categorization or description of the level of pollution in terms of the concentration of one or more compounds or by type such as oxidizing compounds is inadequate and misleading. A useful initial categorization of pollutant compounds according to their mechanism of production, primary or secondary, is often made. For health effects, considerations of the physical state, gaseous or particulate, and the solublity and reactivity of the pollutant is also important. Pollutant compounds or substances that are emitted directly from a source and that undergo little or no chemical change in the atmosphere from source to receptor are termed primary pollutants. These include gases such as CO, CO_2, SO_2, NO and particulate material such as lead, graphite carbon(soot), fly ash, etc. Those pollutants that are formed by chemical reaction of emitted and natural precursors in the atmosphere are termed secondary pollutants. These include O_3, a gas, and HNO_3, H_2SO_4, PAN plus a host of other inorganic and organic compounds, that may exist in gaseous or particulate states. The main source directly or indirectly for

all of these pollutants is the combustion of fuels by industrial, utility, business, transportation, or domestic use. These emissions in an urban/industrialized airshed add to the natural background and regional scale pollution levels.

Some of these pollutants are of little concern with respect to respiratory health due to their lack of effects at the low observed levels (CO_2) or due to the fact that they are not inhaled (particles larger than $10\,\mu$m). Others are of known concern due to their water solubility and irritant effect on the upper respiratory system (SO_2 and H_2SO_4 and other acidic compounds) or due to their potential for oxidizing reactions in the lower respiratory system (O_3, PAN, HNO_3). Some of these are monitored routinely, others, due to technical difficulties, are not. And still others due to their low concentrations and untested health effects are not yet of concern.

Korean Air Quality Background

Seoul lies in an east—west oriented river valley near sea level about 50 km inland from the west coast of Korea. The valley is bounded by ridges to an elevation of up to several hundred meters on the north and lower ·on the south. The climate is similar to that of other mid—latitude industrialized regions in terms of temperature, precipitation, cloudiness and fog occurrence. Due to its latitude of 37N, it receives more solar insolation than most of industrialized Europe or North America, however, but less than the South Coast Basin or Los Angeles area of Southern California.

The major sources of air pollution in South Korea are from varied industrial, automotive and domestic sources similar to other industrialized regions (Kim, 1984). Distinguishing source features are that industrial sources are increasing in this developing country and that domestic emissions are the result of combustion of coal briquettes. Sulfur dioxide emissions increased 5 % in the period 1978—1980 primarily due to an increase in coal combustion.

The latter source is widespread, difficult to control and likely to be a significant source of carbon monoxide as well as sulfur oxides. There are several industrial centers in Korea including Seoul and other industrial centers in Japan and China within a radius of 1,500 km that could cause significant regional background levels of pollutants to exist over Korea.

An extensive network of monitors for sulfur dioxide exists in Seoul and other major cities in Korea. A similar monitoring network for other gaseous pollutant compounds and particulate material is not in place yet. Sulfur dioxide measurements in Seoul show annual mean levels of 80 to 90 ppbv with no trend evident between 1978 and 1981 (Kim, 1985). The highest concentrations of SO_2, 130 to 160 ppbv, occur during the winter season and with the excess presumably due to emissions from increased domestic heating. Minimum concentrations occur from May through October with levels of 30 to 70 ppbv. Observations at seven stations within a 5 km radius of the baseball stadium in SE Seoul for a single month during the less polluted season, October 1983, show a mean concentration of 22 ppbv with a station to station variation of a factor of two above and below that mean. Variation of SO_2 concentrations as a function of week or day of week at any one station was much less.

Prediction of shorter term, peak values of SO_2 concentrations that are of importance with respect to public health concerns, especially aggravation of asthma or other sensitive bronchial reactions, cannot be quantified from long term averages. But concentrations several fold higher than those reported above are highly probable over periods of an hour or less. Similarly, with the exception of particulate sulfate compounds, predictions of the concentration of other pollutant parameters are not possible from the SO_2 data base. Since most of the particulate sulfate in industrial urbanized regions is the result of atmospheric oxidization of the SO_2 precursor, an estimate of the expected concentration of particulate sulfate can be made based on the observed SO_2/particulate SO_4 ratio in other industrialized regions (Hidy, et al., 1978). This ra-

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tio varies as a function of atmospheric chemistry parameters and distance from source.   A value of three to five for this ratio is not unreasonable for the urban Seoul area.   Assuming a value of four results in a estimated annual mean particulate sulfate concentration of about $60\,\mu g\,m^{-3}$ as sulfuric acid with proportional variability througout the year.   The molecular form of the sulfate ion will depend on the available gaseous NH_3.

Pulmonary Functional Effects

During the past decade experiments have been carried out to investigate the health effects of exposure to the ambient air pollutants described above. Although the earliest studies were carried out with the subjects seated at rest, most recent controlled human studies have been carried out during intermittent exercise.   The intermittent exercise paradigm serves as an appropriate model of normal activity levels seen in the outdoors.   A survey of the results of these intermittent exercise exposures leads to the conclusion that exercise exaggerates the pulmonary effects of air pollutant inhalation. One of the first studies to demonstrate this was carried out by Hazucha et al. (1973) who reported a marked decrease in pulmonary function among healthy subjects performing light exercise while exposed to 0.37 ppm of ozone (O_3).   DeLucia and Adams (1977) studied the effects of graded exercise on lung function and blood biochemistry in six men after 1 hour of exposure to 0.30 ppm of ozone via a mouthpiece.   The workloads were 25 percent, 45 percent, and 65 percent of each individual's maximum oxygen uptake $(\dot{V}_{02}$ max). Ventilation volume and $\dot{V}_{02}$ were unaffected by even the most severe exposure/ exercise protocols.   However, most subjects demonstrated signs of toxicity (symptoms such as congestion, wheezing, and headache) during the most stressful protocols.   In addition, vital capactiy, forced expiratory volume in 1 sec, and midmaximum flow rate decreased significantly after inhalation for 1 hr of 0.30 ppm at 65 percent $\dot{V}_{02}$ max.   Discernible, though not statistically signifi-

cant, changes in lung function were seen at exposures to 0.12ppm O_3. A similar relationship between exercise and pulmonary functional changes has been seen following exposure to SO_2. Koenig and co workers (1981) have shown that when asthmatic adolescent subjects were exposed to 1.0ppm SO_2 during intermittent exercise pulmonary functional changes were 3-22 times greater than following exposure at rest. Also some subjects reported wheezing and shortness of breath. Sheppard et al. (1981) have shown significant increases in specific airway resistance (SR_{aw}) in adult asthmatic subjects following exposure to 0.5ppm SO_2. Although these results from asthmatic subjects may not seem relevant for persons involved in competitive sports, Pierson and associates (1984) found that approximately 15% of the members of the 1984 US Olympic team had some degree of exercise-induced bronchospasm (EIB).

Obviously persons with clinical asthma or EIB are at risk with respect to lung effects caused by the increased dose of pollutants inhaled during periods of high ventilation such as exercise. Inhalation of pollutants during exercise increase the dose (calculated as the product of concentration, time and ventilation rate) and also change the deposition pattern of the pollutant gases and particles. Kleinman (1984) has developed a mathematical model of SO_2 deposition and absorption using anatomical, physiological and physicochemical parameters measured in controlled laboratory experiment with animal and human subjects. The model takes into account minute ventilation $(\dot{V}_E)$, partitioning between oral and nasal breathing, and differences in SO_2 scrubbing in oral and nasal pathways. Based on this model, it is predicted that higher ventilatory rates and decreased time of contact with upper airways occurring during exercise would result in delivering of higher doses of gases such as SO_2 to sensitive target sites within the respiratory system. It has been shown that during quiet breathing through the nose greater than 99% of SO_2 is taken up in the nasal passages and less than 1% can be measured at the oropharynx (Speizer & Frank, 1966). Therefore circumstances which require breathing other than quietly through the nose remove a major defense mechanism

against water soluble gases such as SO_2.

There is evidence that healthy trained athletes such as marathon runners and competetive cyclists also show decreased respiratory function when exposed to ambient air pollutant during vigorous exercise. One early epidemiological study investigating the effect of O_3 exposure on athletic performance was conductd by Wayne et al. (1967). This study reported that the proportion of runners who failed to improve their running times rose as concentration of oxidant increased from 0.03 to 0.30 ppm. More recent controlled human studies also provide data on the relationship between athletic performance and air pollutant levels. Adams and Schelegle (1983) studied ten healthy, well −trained long−distance male runners following one hour exposure to 0.0, 0.20 or 0.35 ppm O_3 during exercise simulating either training or competition. Significant decreases in forced vital capacity (FVC) and forced expiratory volume in one second (FEV_1) were seen following the O_3 exposures. The authors concluded that the statistically significant pulmonary function impairment observed following 0.20 ppm O_3 exposure suggests that endurance athletes may be a group susceptible to the effects of a given O_3 concentration due to their increased ventilatory rate.

Folinsbee et al. (1984) studied the pulmonary effects of exposure to 0.21 ppm O_3 in seven well trained men and women who were competitive distance cyclists. The subjects were exposed for one hour to air or 0.21 ppm O_3 at 75 % of maximal O_2 consumption. Significant decreases in FVC were seen following the ozone exposure. Also the subjects reported symptoms of laryngeal and tracheal irritation and soreness and chest tightness during a deep inspiration.

The effects of SO_2 exposure on exercise performance also have been studied (Linn et al, 1983). Twenty three asthmatic volunteers were studied during exposure to 0, 0.2, 0.4 or 0.6 ppm SO_2. Exposures included 5 minutes of heavy exercise at an average ventilatory rate of $48 \, lm^{-1}$. Significant decreases in pulmonary function were seen following exposures to either 0.4 or 0.6

ppm SO_2. Unfortunately, there are no data from controlled human studies of high performance athletes exposed to SO_2 at concentrations recorded in Seoul.

On the basis of these above studies it is obvious that some subjects show significant decrements in lung function following vigorous exercise at low levels of O_3 or SO_2. There has been much discussion whether an "adaptation" of lung response occurs with repeated exposure to ozone (Bromberg & Hazucha, 1982). The observation that Los Angeles residents showed miminal response to 0.4 ppm O_3 whereas subjects from Montreal showed significant responses to the same concentration (Hackney et al, 1976) first suggested that some alteration of response or " adaptation " may occur in populations chronically exposed to O_3 in ambient air. Subsequent reports from laboratory studies with human volunteers indicated that with repeated daily exposures to O_3, decrements in the O_3−induced lung function changes are seen. Farrell and coworkers (1979) studied 14 normal human subjects who were exposed for 3 hours on 5 consecutive days to 0.4 ppm O_3. Significant decreases in FVC were seen following exposure on day 1, 2 and 3 ; however there was no significant difference in FVC on the fourth and fifth days of O_3 exposure.

Linn and co−workers (1982) exposed eleven healthy subjects to 0.47 ppm O_3 for 2 hours on four consecutive days. Ten of the eleven subjects developed an "adaptation" to the O_3 exposure by the third day of exposure as measured by FEV_1 changes. One subject did not show an antenuated response to the repeated exposure. The adaptation in the ten subjects (smaller decreases in FEV_1) was partly lost after a 4 day interval between exposures and totally lost after a 7−day interval. These authors conclude that adaptation may be of relatively little importance to health since it may fail to develop and appears to be lost rapidly. Thus it is by no means certain what lung response can be expected in residents of areas with continuous oxidant levels. Also it is not at all clear that "adaptation" to low levels of O_3 is protective. This response may reflect the presence or development of underlying changes or in-

jury to the lung (Bromberg & Hazucha, 1982).

There also is one report of tolerance to repeated SO_2 exposures in adult asthmatic subjects (Sheppard, et al. 1983). Subjects were exposed to 0.5 ppm SO_2 during 3-minute of eucapnichyperventilation for three sessions at 30-minute intervals. The magnitude of increase in SR_{aw} was diminished after the second and third exposures. It was concluded that repeated exposures to a low concentration of SO_2 over a short period can induce tolerance to the bronchomotor effect of SO_2.

The relationship between dietary vitamin E levels and pulmonary susceptibility to ozone has been reviewed recently (Chow, 1983). Animal studies indicate that vitamin E, an antioxidant, can prevent some morphological and biochemical effects of O_3 exposure which have been attributed to ozone-initiated peroxidation.

Hackney and co-workers investigated the protective action of vitamin E in human volunteers. Subjects received 800 or 1,600 IU of vitamin E prior to 2 hour exposures to 0.5 ppm O_3 at rest (Posin et al., 1979) or during intermittent exercise (Hackney et al., 1981). Neither study showed any indication of added protection from vitamin E against O_3-induced blood chemistry or lung function change following O_3 exposure. To the best of our knowledge, there is no evidence that ozone related lung effects in human populations could be decreased by increased dietary vitamin E intake.

In addition to air pollutants, the temperature and relative humidity (RH) of inhaled air can influence respiratory function. The beneficial effect of inspiring hot, humid air (37℃~100% RH) during exercise was reported in 1977 by Chen and Horton. Conversely, Strass et al. (1977) showed that exercise-induced bronchospasm is enhanced by breathing cold (−11 to −15℃) air. These authors reported that atopic subjects showed a 17% decrease in FEV_1 following exercise exposure to the cold air. More recently Anderson (1984) has reviewed the literature on environmental influences on EIB and concluded that water loss rather than heat loss is the most important stimulus for EIB.

These data indicate that ambient humidity and temperature can influence respiratory function and consequently athletic performance.

Management of Allergic Athlete

The key to management of allergic athletes is the identification of problems specific to athletes with allergic diseases. The major problem of allergic athletes is exercise − induced bronchospasm (EIB). This bronchospasm many times is under−appreciated or misinterpreted and requires specific management for optimal performance for any athlete, especially Olympic athletes. The prevalence of EIB among patients with asthma is approximately 90 to 95 %. All other allergic athletes tested have a 30 to 35 % prevalence of EIB.

Personal medical history is important, but many times athletes or patients are not aware that their breathing difficulty is related to EIB. The histories have a reliability of only 60 %, i.e., 40 % of patients reporting negative history will have EIB.

There are three methods of testing that are most important at the present time :

(a) Free−range running for a period of 6 to 8 minutes, which is adequate to cause a pulse rate that is commensurate with approximately 85 % oxygen consumption for that individual, is widely used. Free−range running, however, has the disadvantage of being subjected to varying air temperature and relative humidity as well as airborne pollutants, both natural and man−made, i.e., pollens and/or sulfur dioxide and ozone.

(b) Treadmill testing is commonly used and is readily available in most laboratories and hospitals. The patient runs for 6 to 8 minutes to attain a pulse rate commensurate with 85 % oxygen comsumption.

(c) The cyclometer is another instrument used less commonly. Its variability in producing EIB has curtailed its use in many clinics.

Other tests that are important include the methacholine challenge test,

which is actually more sensitive than exercise testing. This is a good diagnostic test for patients who have cough or other less direct symptoms but may have bronchial reactivity. It is administered in a dose ranging method and can be very helpful in finding patients with latent bronchial hyperreactivity. A new method recently introduced is that of eucapnic voluntary hyperventilation with a 5% carbon dioxide, 20% oxygen and 75% nitrogen mixture with a relative humidity of $< 1\%$. This methodology, using dry dehumidified air, can induce bronchospasm and detect bronchial hyperreactivity by acutely increasing water loss in the airways.

Other Allergic Problems

The identification of other allergic problems in competitive athletes is important to proper management. Specific problems that can cause difficulty include : (a) Rhinitis and /or pollinosis or hay fever, especially for patients who perform outdoors and in events during the pollen season where aeroallergens are present at the time of competition. This is especially so for endurance events where high volumes of air are inspired over a relatively short period of time : e.g., a marathon runner will inhale and exhale in 2 1/2 hours what would be a normal tidal volume for 2 months. (b) Sinusitis is a problem that plagues athletes, with symptoms of low—grade headaches, cough and a general feeling of fatigue. It is often overlooked and can have a significant effect upon an athlete. (c) Hives or urticaria, and especially cholinergic urticaria, are vexing problems for athletes, due to discomfort, and can diminish performance. Also, cold urticaria for certain athletes, especially in aquatic sports or winter sports, can be a very serious management problem. (d) Exercise-induced anaphylaxis is another life threatening situation that is both difficult to manage and very frightening for the athlete. It seems to be related to food intake prior to exercise and the distinguishing feature is the development of angioedema or urticaria and upper airway laryngeal edema, but

no wheezing. The management of these patients is primarily one of identification and supportive measures, advice to exercise in a fasting state, and always to exercise with a companion. (e) Drug reactions also can occur and be very devastating, especially with such medications as aspirin or nonsteriodal anti—flammatory drugs and antibiotics. Other factors can affect allergic athletes and either exacerbate or initiate their existing clinical status. These include infections, expecially respiratory infections, sinusitis or rhinopharyngitis, pyoderma and other cutaneous infections. Fungal infections can occur both in the skin and alimentary tract as well as the genitourinary tract, especially in patients who have been on prolonged courses of antibiotic therapy. Stress is another factor that is usually an intregal factor for athletes, especially if complicated by hives, urticaria, shortness of breath or sneezing. The nutritional state of an athlete can also be a very important factor, especially water and protein needs of strenuously exercising athletes including distance runners, mountain climbers and other endurance performers. Many athletes are very prone to fat diets and other maneuvers that may not be scientifically based or safe.

Management Protocols

The appropriate management of an allergic athlete follows a careful individual assessment of the various allergic disorders and other factors that may affect that athlete. This starts with a careful history and physical examination and a pre—participation physical assessment.

The management of EIB can be accomplished several ways. (a) Appropriate choice of a sport and/or the use of a warm—up protocol can many times help an athlete with EIB participate effectively, even at world class competitive levels. (b) Pharmacologic management of EIB can be divided into two catagories :

(1) Athletes with normal resting airway function. The drug of choice is

albuterol or one of the other selective B_2 agonist aerosols taken 10 minutes prior to competition. Cromolyn sodium also is very effective when taken, again 10 minutes prior to competition, by turbospinhaler, especially if EIB is predominantly caused by the large airways. Theophylline in a rapid release formulation can be administered at 5 mg/kg one hour prior to exercise. It has the disadvantage of requiring that it be taken one hour prior to exercise and also must be a rapid release formulation to be effective.

(2) Athletes with abnormal resting airway function or athletes who fail to respond to the maneuvers above. The cornerstone of therapy is the administration of a sustained released theophylline preparation, in a dosage of 12 to 20 mg kg^{-1} dy^{-1} to attain a serum blood level of 7.5 to 20 μg ml^{-1}. Once an athlete has achieved an adequate theophylline level, the use of albuterol or cromolyn 10 minutes prior to exercise will usually block EIB effectively. Some asthmatics with severe airway involvement will require a combination of albuterol and cromolyn administered prior to exercise after they have adequate theophylline doses. Some will require concomitant corticosteriod therapy to stabilize their airways.

The appropriate treatment of other allergic problems, including rhinitis, hay fever and especially sinusitis, are important to overall management of allergic athletes. The identification of urticaria and its appropriate pre−medication with hydroxyzine or cyproheptadine is important. Patients with exercise anaphylaxis, and the allergic athlete, should be encouraged to exercise only in the company of companions and only in a fasting state. They also should carry epinephrine(Epipen) when they exercise. The management of other factors such as stress and infection are important for an allergic athlete and should not be overlooked, especially prior to competition. The United States Olympic Committee has found that a pre−participation examination prior to a world event was very important and patients with respiratory tract infections required appropriate vigorous therapy to avoid disabling affects of respiratory infections during international competition. This protocol worked

effectively with participating athletes at the 1984 Olympic games and should meet with even more success in 1988.

Drug testing and the use of banned drugs has unfortunately become a major part of national and international competition. The XXIII Olympiad in Los Angeles in 1984 amply demonstrated that the Olympic movement is mounting a vigorous anti—doping campaign and seems to be succeeding with a vigorous drug testing program. Many of the national athletic unions are now adopting a strong stance against doping and are testing and disqualifying athletes for the use of banned medications.

The most commonly abused drugs are androgenic steriods and amphetamine—like stimulants. These are banned for most national competitive events and should be avoided by athletes. On the other side of the coin, medications that have been described above for the management of allergic asthma, rhinitis, hay fever and urticaria should be carefully delineated by a team physician and the national governing body of the sport in which the athlete is participating.

Thus, it can be seen that knowledge of air chemistry and specific pollutant effects on lung function become important to the competitive athlete. Also, all athletes, and allergic athletes especially, need to understand the special stresses that exercising in an urban environment can place on physiological parameters. Responsible management can mitigate the effects of inhalation of air pollutants.

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