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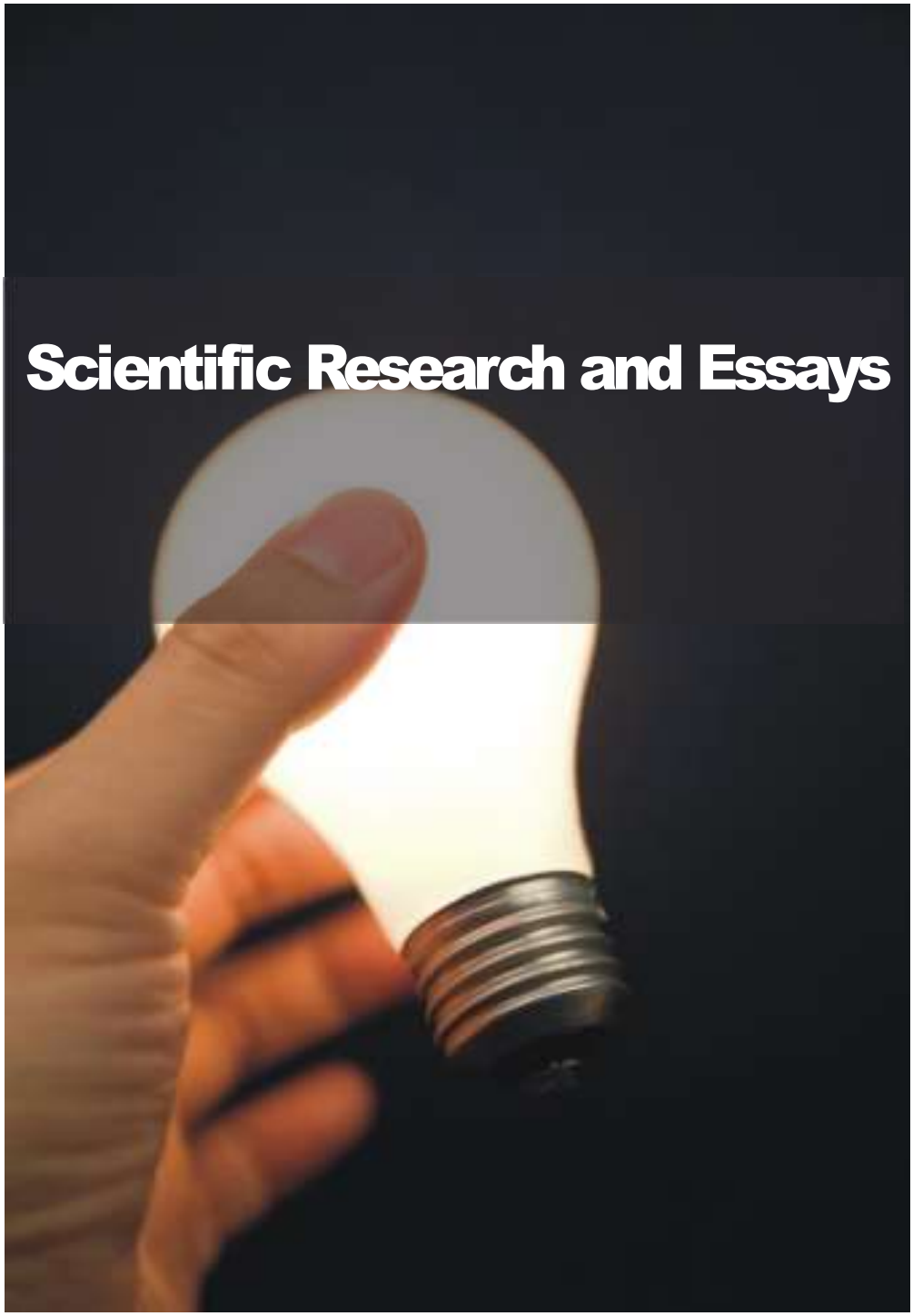

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Principal air pollutants and their effects on athletes' health and performance: A critical review

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The aim of this review was to assess the health effects of the criteria air pollutants: Sulfur dioxide (SO_2), carbon oxides (CO_X), nitrogen dioxide (NO_2), particulate matter (PM) and ozone (O_3) together with photochemical smog and global warming in relation to exercise with particular focus on athletes. In this review, respiratory and cardiovascular health impacts of the specified principal air pollutants have been seen. It will also give information about ambient air quality standard to estimate air quality and take appropriate measure for training athletes according to the US Environmental protection agency. The effect of photochemical smog has been discussed as a result of the synergistic effect of ozone (O_3), nitrogen dioxide (NO_2), volatile organic compounds (VOCS) and peroxyacetyl nitrate (PAN). A wide spectrum illness that arises from global warming that needs precautions have been seen in this review. In general this review will provide information for athletes, coaches, managers, sports medicine team, health community as well as other concerned bodies.

Key words: Principal air pollutants, athletes, asthmatics, respiratory effect, cardiovascular effect, acute exposure, chronic exposure.

INTRODUCTION

Pollution nowadays is the main problem in the world. This issue has always been the agenda of everyone for those who concerned for the health of the world. Some areas of the world are in the worst pollution, which exposed people to poor health, even death. For example, in Russia -Dzerzinsk, 300,000 people, Norilsk, 134,000 people, In China Linfen, Shanxi province, 200,000 people, In Haina, Dominican republic 85,000 people, In India Ranipet, 3,500,000 people are potentially affected by chemicals and toxic byproducts like Sarin, VX gas, fly-ash, carbon oxides, nitrogen oxides, $PM_{2.5}$, PM_{10}, volatile organic compounds, arsenic, particulates including Strontium-90, Caesium-137, Sulfur dioxide, heavy metals (nickel, copper, cobalt, lead, selenium), phenols, hydrogen sulfide (Blacksmith Institute, 2006). Most of these are air pollutants and such environmental pollution creates undeniable health impacts on human beings.

WHO (2003) classified the health effects of air pollution into two. The first one is the effects attributed to short-term exposure, which includes: daily mortality**,** respiratory and cardiovascular hospital admissions**,** respiratory and cardiovascular emergency department visits**,** respiratory and cardiovascular primary care visits**,** use of respiratory and cardiovascular medications**,** days of restricted

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activity**,** work absenteeism**,** school absenteeism**,** acute symptoms (wheezing, coughing, phlegm production, respiratory infections) and physiological changes (for example lung function). The other category is effect attributed to long-term exposure, such as: mortality due to cardiovascular and respiratory disease**,** chronic respiratory disease incidence and prevalence (asthma, COPD, chronic pathological changes)**,** chronic changes in physiologic functions**,** Lung cancer**,** chronic cardiovascular disease**,** intrauterine growth restriction (low birth weight at term, intrauterine growth retardation, small for gestational age).

Both in the short and long term respiratory and cardiac impacts have been linked to air pollution and exercise intensity (Mroszczyk, 2012) and yet elite athletes are rarely aware of the health risk associated with training or competing in an environment with poor air quality. For example, Cutrufello et al. (2012), Singh (2013) and Spitz (2014) reported that about 2 million deaths per year may be attributed to air pollution. It is the main cause of cardio respiratory infection, especially for special populations like active individuals and sports men due to speedy and high consumption rate of gas in inhaled air.

The volume of air inspired or expired with each normal breath (tidal volume) is about 500 ml in the adult male. But in women pulmonary volumes and capacities are about 20-25% less than men, and they are greater in large size and athletic people than in small size and inactive people (Guyton, 2006).

The metabolic demands of exercise increase minute ventilation and therefore the rate of inhalation of pollutants increase. The type of sport that the athletes involved has also different exposure potential for polluted air. For example, summer Olympic sports (triathlon, running, cycling, soccer, rowing, tennis, and swimming), Winter Olympic sports (Cross-country skiing, downhill skiing, hockey, figure skating, and speed skating) and Paralympics are sports with the highest potential exposure to poor indoor or outdoor air quality (Piturro, 2008).

In this connection, athletes become at high risk of inhaling polluted air because of two major reasons. The first one is the 10 to 20 times (NFHS, 2011) or 10 to 15 times (Kargarfard et al*.,* 2009; El-Hadedy and Zaiton, 2012) increase in the inspiration of the normal volume of air per minute during exercise and increased velocity of respiration exposes them to air pollutants. For comparison, the typical inactive individual inhales about 600 liters of air each hour. During exhaustive activity, however, the volume can be as high as 7,000 liters (Bonini et al., 2006). This implies that exercise in polluted air greatly increases lung surface contact with airborne pollutants. The second is that athletes could breathe in the mouth than in their nose (El-Hadedy and Zaiton, 2012). Breathing through the nose filter some percent of inhaled pollutants, but during exercise air intake usually comes through the mouth that enables air pollutants more directly get into the lungs. Apart from the health effect, polluted air decrease in the performance of athletes. For example, in a research done with 8 km running, the athletes' performance was significantly decreased in hot, humid and ozone-polluted condition (Elisa, 2009). Another study results from Kargarfard et al*.* (2009) showed that there are significant decreases in all of the respiratory parameters: Expiratory reserve volume (ERV), Inspiratory capacity (IC)- forced expiratory volume in 1 s (FEV_1), Maximum voluntary ventilation (MVV), Forced vital capacity (FVC) of active individuals in polluted air compared with non-polluted air.

The prevalence of asthma can be taken as one form of risk in elite athletes. Its prevalence has been reported to range between 3.7 and 22.8% (Bonini et al., 2006) indicating that asthma and asthma-like symptoms seem to be more common in elite athletes compared with inactive individuals. Asthma is most commonly found in athletes performing endurance events such as cycling, swimming, or long distance running (Bonini et al., 2006; Helenius and Haahtela, 2000). According to Helenius (2000), asthma risk is closely associated with atrophy and its severity. When the two risk factors, sporting event and atrophy were combined in a logistic regression model, the relative risk of asthma was 25-fold in an atrophic speed and power athlete, 42-fold in an atrophic long-distance runner, and even 97-fold in atrophic swimmers compared with non-atrophic controls (Helenius and Haahtela, 2000). Olympic cyclists, who spend many hours training on the road and, who are often exposed to PM, NO_X and ozone (O_3) have a high prevalence of asthma (17%) (Fitch, 2012; Kippelen et al., 2012).

Generally, results of reports indicate that even a brief acute exposure to moderate levels of air contamination may promote modest but significant physiological abnormalities in clinically healthy young adult individuals during exercise (El-Hadedy and Zaiton, 2012). Athletes like that of asthmatic individuals, infants and elderly people; are potentially sensitive populations with respect to inhalation of ambient air pollutants that greatly increase their dose of gaseous or particulate air pollutants through increased ventilatory rate (Carlisle and Sharp, 2001; Kim et al., 1991; Singh, 2013). A study by Das and Chatterjee (2013) in its part reported that there exists a significant negative correlation between air pollutant parameters and hemoglobin concentration. This is to mean that inhalation of polluted air adversely affects hemoglobin concentration and this effect is pronounced in active individuals like athletes.

AMBIENT AIR QUALITY STANDARDS

The Air Quality Index (AQI) is the national standard method for reporting air pollution levels. An index such as the air quality index (AQI) is necessary because there are several air pollutants, each with different typical ambient

Table 1. Pollution standard index (PSI).

Index value	Air quality index (AQI)	Color
Up to 50	Good	Green
51 to 100	Moderate	Yellow
101 to 150	Unhealthy for Sensitive Groups*	Orange
151 to 200	Unhealthy	Red
201 to 300	Very unhealthy	Purple
> 301	Hazardous	Maroon

Source: US EPA (2000); Chen (2013). *Refers to asthmatics, allergic and active people.

Table 2. Pollutant characteristics in polluted and low polluted atmosphere.

Pollutants/Characteristics	Low polluted	High polluted
Humidity (%)	< 48.5	> 49.5
Temperature (°C)	< 9.8	> 9.6
Altitude (m)	> 1626	< 1600
Carbon monoxide, CO (ppm)	< 2.4	> 35.4
Ozone, O_3 (ppb)	< 1.6	> 10.1
Particulate matter, PM_{10} (ppm)	< 20	> 248
Nitrogen dioxide, NO_2 (ppb)	< 18.3	> 45.4
Sulphur dioxide, SO_2 (ppb)	< 18.2	> 46.9
Pollution standard index (PSI)	< 50	> 200

Kargarfard et al. (2009, 2011).

concentrations and each with different levels of harm, and to report actual concentrations for all of them would be confusing (Illinois, 2012).

For example, the U.S. Environmental Protection Agency (EPA) has set National Ambient Air Quality Standards (NAAQS) for six principal air pollutants (criteria air pollutants) considered harmful to public health: Sulfur dioxide (SO_2), carbon monoxide (CO), nitrogen dioxide (NO_2), particulate matter (PM), ozone (O_3), and lead. Some of these pollutants, such as PM, are commonly associated with increased incidence of asthma, decreased lung function and cardiovascular complications, while others affect health in different ways like effects of lead on the developing nervous system (Table 1).

Researchers in the area measured high polluted and low polluted atmospheres based on the EPA criteria air pollutants and the results are reported as shown in Table 2.

Thus, according to the report, the PSI of the highly polluted atmosphere is > 200 (Table 2) which means it is very unhealthy (Table 1).

High level of air pollutants may lead to decrease in the maximal oxygen consumption (VO_2 max) which could be because of low levels of oxygen transport from the pulmonary alveoli (Brook et al., 2010, Liux et al., 2008, Olivera et al., 2006). The most common air pollutants are: carbon monoxide, nitrogen oxides, sulfur oxides, particulate matters with a diameter of less than 10 µm (PM_{10}), and ozone (Table 1). The aim of this review was, therefore, to assess the health and performance effects of sulfur dioxide (SO_2), carbon oxides (CO_X), nitrogen dioxide (NO_2), particulate matter (PM) and ozone (O_3) and global warming in relation to exercise with particular focus on athletes.

EFFECT OF PRINCIPAL AIR POLLUTANTS AND THEIR INTERACTION IN THE BODY

Particulate matter

Particulate matter (PM) includes soluble or insoluble solid and liquid materials present in the air in the form of particles which are small enough (less than 10 µm) to remain in suspension for some hours or days (WHO, 2003). Particles less than 10 µm (PM_{10}) are capable of entering the respiratory tract and reaching the deeper parts of the lung up to alveoli, and deposited if less than 2.5 µm ($PM_{2.5}$) in diameter. However, particles of

diameter less than 0.5 µm ($PM_{0.5}$) are least likely to be deposited in the respiratory tract, as they are too small to either impact on, or diffuse into, the walls effectively and are exhaled before they can be deposited (Manhan, 2003; WHO, 2003). Sulfate, mineral dust (including trace elements), nitrate, Black carbon as well as black smoke leads to the accumulation of total suspended particulates, and they are the main contributors for the deposition of PM_{10} and $PM_{2.5}$ (WHO, 2003).

Such deposition is exacerbated by exercise. For example, a comparison study conducted between rural and inner city areas indicates that personal exposure to PM of people exercising at the roadside in the city is higher than that of the sedentary person and those exercising in rural locations and significant health and performance effects have been seen on athletes' in the city (Carlisle and Sharp, 2001).

Air polluted by car exhaustions, combustion of engines, especially freshly generated PM_{10}, is considered highly injurious to the airways, especially dehydrated and damaged airways are believed to be more vulnerable to the poisonous effects of PM_{10}, and PM_1 (Kippelen et al., 2012). Athletes respiratory tract becomes dehydrated during high intensity trainings which exacerbates the damage of both upper and lower respiratory tract due to PM.

Among healthy athletes, it is generally reported that daily low-dose exposures to air pollution, and then PM, does not have any significant effect on short-term pulmonary function (Williams, 2011). Nonetheless, it is concluded that PM exposure and exercise on competitive athlete is still susceptible to pulmonary inflammation, decreased lung function, increased risk of asthma and decreases in exercise performance (Cutrufello et al., 2012; Spitz, 2014).

Carbon oxide

Carbon monoxide (CO) emissions in urban areas now a day is greater than the emissions of all other pollutants combined. The ability to generate prolonged energy depends on the extraction and transportation of oxygen to skeletal muscle for cellular respiration. However, the presence of CO can alter the ability of hemoglobin to bind and transport oxygen to cells because CO can compete with oxygen to the sites of hemoglobin (Hadgson, 2010). Consequently, hemoglobin will combine with CO to form carboxyhaemoglobin (COHb) and it has a 230 times greater affinity for CO than for oxygen (Singh, 2013). Finally, CO puts its significant potential effect by altering the blood's carrying and transporting capacity of oxygen to body cells (Oliveira, et al., 2006; Singh, 2013).

The World Health Organization (WHO, 2000) calculated the relation between CO concentration and blood COHb for a lightly exercising subject. COHb values are reduced by a factor of two for a person at rest and increased by a similar factor by heavy exercise like endurance athletes. Thus a heavily exercising subject can expect to have 1.6% COHb after one hour in 20 mg/L CO (Carlisle and Sharp, 2001). Levels of 2.7% COHb and upwards result in evidence of impaired behavior (Colls, 1997; Carlisle and Sharp, 2001). In another study it is reported that exhaustive exercise performed for 30 minutes in high traffic areas is equivalent to smoking 10 cigarettes (Marr and Ely, 2010). Such prolonged exposure and exhaustive exercise to heavy traffic areas brought a 5% concentration of COHb in the blood (Parminder, 2013). This implies that athletes should be warned not to do exercise in the area and time of high traffic.

Sulfur oxides

Polluted air contains oxides of sulfur, commonly sulfur dioxide (SO_2) that results primarily from the industrial combustion of coal, with soft coal (pyrite) containing the highest levels of sulfur (Hadgson, 2010) and organically bound sulfur in coal and fuel oil (Manahan, 2003). These sources emit millions of tons of SO_2 into the global atmosphere annually and are largely responsible for acid rain (Manahan, 2003).

SO_2 tends to adhere to air particles and enter the inner respiratory tract, where they are not effectively removed (Carlisle and Sharp, 2001). In the respiratory tract, it combines readily with water, that is, 1 volume of water dissolves 45 volumes of SO_2 at 15°C (WHO, 2006) to form sulfurous acid and then sulfuric acid resulting in irritation of mucous membranes and bronchial constriction (Manahan, 2003; Hadgson, 2010) by stimulating the vagus nerve (the tenth cranial nerve) in the pharynx region of the throat, behind the tongue (Kargarfard et al., 2009; Singh, 2013) where it results in reductions in forced expiratory volume (FEV1) or other indices of ventilatory capacity, increases in specific airway resistance, and symptoms such as wheezing or shortness of breath (Kim et al., 1991; WHO, 2000; Singh, 2013). Such effects are enhanced by exercise, which increases the volume of air inspired thereby allowing SO_2 to penetrate further into the respiratory tract (WHO, 2000; El-Hadedy and Zaiton, 2012). These effects in turn increase the sensitivity of the airway to other airborne toxicants (Hodgson, 2010), and enhanced if penetration to lower regions is increased through the mouth rather than nose breathing and through exercise that raises the amount and depth of inhalation (WHO, 2000; Carlisle and Sharp, 2001; El-Hadedy and Zaiton, 2012). The minimum concentration evoking changes in lung function in exercising asthmatics is of the order of 4 mg/L. However, SO_2 begins to affect lung function in normal healthy adults at concentrations between 1 and 2 mg/L (Borresen, 2008).

Numerically, asthmatics are generally ten times more sensitive to SO_2 than non-asthmatics, especially when exercising. At concentrations of 500 ppb SO_2, exercising asthmatics experience pronounced changes (as much as 100%) in airways resistance after five minutes of exercise (Carlisle and Sharp, 2001). Thus, with the recommended air quality standard for SO_2 being 0.1 mg/L (15 minute average) (Carlisle and Sharp, 2001), it is unlikely to have an effect on athletes with normal lung function, but in asthmatics (Carlisle and Sharp, 2001; Borresen, 2008). It is also reported that a decreases in FEV_1 of 50 to 60% are observed in most exercising asthmatics exposed to 0.25 mg/L SO_2 (Folinsbee, 2001; Campbell et al., 2011). Subjects showed significant respiratory changes and asthmatic attacks with exposure to SO_2 at concentrations as low as 0.1mg/L for 10 minutes or longer (Campbell et al., 2011). SO_2 exposures in the range of 100–400 mg/L in air pose an immediate danger of death (National Academies, 2004; Borresen, 2008). Lower concentrations (<40 mg/L), however, have been associated with death in specific incidents (Borresen, 2008). In addition to lung effects, cardiovascular, gastrointestinal, neurological, and hematological effects have been observed in humans exposed to SO_2 by inhalation (National Academies, 2004). For example, human, non-asthmatic subjects exposed to 1–8 mg/L SO_2 showed an increased pulse rate, which could be aggravated through exercise (Campbell et al., 2011). Therefore, SO_2 is clearly an important irritant for exercising asthmatics and may cause problems for the asthmatic athlete, but higher concentrations of SO_2, more than the quality standard 0.1 mg/L for 15 minute average, could affect the non-asthmatic athlete.

Nitrogen oxide

Nitrogen oxides (NOx) enter the atmosphere primarily as NO, produced in combustion processes from organically bound nitrogen endogenous to fossil fuels (particularly coal, heavy fuel oil, and shale oil) and from atmospheric nitrogen under the conditions that exist in an internal combustion engine, and photochemical processes in the atmosphere tend to convert NO to NO_2 (Manahan, 2003).

Nitrogen dioxide (NO_2), a gas found in photochemical smog, is also a pulmonary irritant and is known to lead to pulmonary edema and hemorrhage (Manahan, 2003). The main issue of concern is its contribution to the formation of photochemical smog and ozone, although nitrogen oxides also contribute to acid deposition (Hadgson, 2010). Inhalation of air containing 200 to 700 mg/L of NO_2 can be fatal (Hadgson, 2010). The biochemical action of NO_2 includes disruption of some enzyme systems, such as lactic dehydrogenase (Barnes et al., 1991). Nitrogen dioxide probably acts as an oxidizing agent similar to, though weaker than, ozone. Included is the formation of free radicals, particularly the hydroxyl radical HO. Like ozone, it is likely that NO_2 causes lipid peroxidation. This is a process in which the C=C double bonds in unsaturated lipids are attacked by free radicals and undergo chain reactions in the presence of O_2, resulting in their oxidative destruction (Manahan, 2003). Bonini et al. (2006) reported that there is little negative effect as a result of exposure to NO_2 in normal studied subjects. However, asthmatics have been shown to experience significant increases in cold air hyperventilation with short-term NO_2 exposures of 0.5 mg/L; however, NO_2 levels in urban environments are usually below 0.15 mg/L (Carlisle and Sharp, 2001) which is very low compared to the NO_2 concentration in excess of 1.0 mg/L that required to bring changes in pulmonary function in healthy adults (Klaunig, 2004; Barnes, 1991; WHO, 2006). For example, significant increases in airway resistance have been reported with exposure to 5.0 mg/L NO_2 (Barnes, 1991; WHO, 2006). Other studies on the pulmonary effect of NO_2 showed that inhalation of 0.18 or 0.30 mg/L NO_2 for 30 min including 16 min of heavy exercise does not adversely affect the pulmonary function (Kim et al., 1991), and no significant pulmonary function change in both healthy and asthmatic adolescent subjects exposed to 0.12 or 0.18 mg/L NO_2 for 30 min at rest followed by 10 min of moderate exercise (Koenig et al., 1991). Numerous studies on people with asthma, chronic obstructive pulmonary disease (COPD) or chronic bronchitis have shown that exposure to low levels of nitrogen dioxide can cause small decrements in forced vital capacity (FVC) and forced expiratory volume in 1 second (FEV_1) or increases in airway resistance. Generally, the lowest level of NO_2 exposure reported to show a direct effect on pulmonary function in asthmatics, was 0.3 mg/L for two and half hours exposure (Graham, 2001; WHO, 2006).

Ozone

Ozone (O_3), a secondary air pollutant, is produced from the photochemical reaction of nitrogen oxides (NOx) and volatile organic compounds (VOCs) under sunlight (Akimoto et al., 2006; Manahan, 2003; Hadgson, 2010; Singh, 2013) and the influx from the stratosphere (Akimoto et al., 2006). Emission of NOx and VOCs are increasing dramatically as the world experiences rapid industrialization (Akimoto, 2003). O_3 is one of the pollutants of concern because it is associated with extensive health effects, most notably with the respiratory system, and it can affect both forests and agricultural crops (Sillman, 2003). Offcourse, distinguishing “good” (stratospheric) and “bad” (tropospheric) O_3 is highly essential. Tropospheric O_3 occurs from 0 to 10 miles above the earth’s surface, and is harmful. But stratospheric O_3, located about 30 miles above the earth’s surface, is responsible for filtering out incoming UV radiation and thus is beneficial (Manahan, 2003;

Sillman, 2003). In this review, tropospheric O_3, its formation occurs through the following sequence of reactions (Akimoto et al., 2006; Manahan, 2003; Sillman, 2003; Hadgson, 2010), is considered. The sequence is almost always initiated by the reaction of various VOC or CO with the OH radical (1 and 2).

$$VOC_1 + OH + [O_2] \longrightarrow RO_2 + H_2O \quad (1)$$

$$CO + OH + [O_2] \longrightarrow HO_2 + CO_2 \quad (2)$$

This is followed by the conversion of NO to NO_2 (through reaction with HO_2 or RO_2 radicals), which also regenerates OH (3 and 4).

$$RO_2 + NO + [O_2] \longrightarrow VOC_2 + HO_2 + NO_2 \quad (3)$$

$$HO_2 + NO \longrightarrow OH \bullet + NO_2 \quad (4)$$

NO_2 is photolyzed to generate atomic oxygen, which combines with O_2 to create O_3 (5 and 6).

$$NO_2 + hv \longrightarrow NO + O \quad (5)$$

$$O + O_2 + M \longrightarrow O_3 + M \quad (6)$$

Here, VOC_1, VOC_2 are primary and secondary volatile organic compounds, respectively; RO_2, Represents alkyl or any organic chain attached to O_2.

The primary toxicological concern with O_3 involves the lungs as its exposure increases the activity of free-radical-scavenging enzymes in the lung (Akimoto et al., 2006), indicative of ozone's ability to generate the reactive oxidant species responsible for oxidative stress. Like nitrogen dioxide and ionizing radiation, O_3 in the body produces free radicals that can be involved in destructive oxidation processes, such as lipid peroxidation (Manahan, 2003; Singh, 2013). Exposure to ozone can cause chromosomal damage and also appears to have adverse immunological effects (Manahan, 2003; WHO, 2006). Radical-scavenging compounds, antioxidants, and compounds containing sulfhydryl groups can protect organisms from the effects of ozone (Manahan, 2003; Akimoto et al., 2006).

Exposure to elevated ozone concentrations has been reported to give rise to symptoms that include cough, chest pain, difficulty in breathing, headache, eye irritation and a decrease in forced expiratory volume in one second, and all of these effects are likely to impact upon performance (Carlisle and Sharp, 2001; Florida-James et al., 2004;; Bennett, 2007). These responses are exacerbated during exercise because of the following reasons: one is the increment of the absolute amount of O_3 to be inhaled, second, the rise in the uniformity of ventilation throughout all lung tissues, and the third one is "nasal scrubbing" (Absorbing gases during quiet breathing through the nose) is compromised (Hazucha, 1996; Kargarfard et al., 2009). Athletes, for example, are vulnerable to the effects of inhaled O_3 because of their exercise patterns (Helenius and Haahtela, 2000). Both expired volume and Volume of O_2 are both dramatically increased with the onset of physical activity, whether it is heavy, short term or less intense and prolonged warm up, training, and competition (Kargarfard et al., 2009). The respiratory discomfort associated with O_3 exposure may cause decreased maximal work performance (Carlisle and Sharp, 2001).

Quantitatively, it is reported that a concentration of 0.1 mg/L causes decrease in lung function at an exercise intensity equating to a minute ventilation of 70 L/min (Florida-James et al., 2004). Different study results cited in OEHHA (1999) reported that impairment of lung function and subsequent impairment of exercise performance were measured in exercising adult athletes (age 19-30) exposed to 0.2 mg/L ozone for 1 h. A decrease in post-exercise forced expiratory volume in 1 second (FEV_1) of 21.6% was observed; a 5.6% decrease in FEV_1 was observed in athletes following a 1 h exposure to 0.12 mg/L ozone with exercise. Significant reductions in peak minute ventilation, oxygen uptake, and tidal volume were observed in athletes exposed to 0.2 mg/L ozone, but not in those exposed to 0.12 mg/L, and athletes exercising in elevated ozone, exhibited reduced endurance and lung function (Kargarfard et al., 2009). For asthmatics exposed to 0.08 mg/L and higher of ozone, the forced expiratory volume (FEV) is reduced during physical exercise (Akimoto et al., 2006).

In a similar study, 3% decrease was observed in FEV_1 in male children (age 8-11) following a 2.5 h exposure to 0.12 mg/L O_3 with intermittent exercise (OEHHA, 1999). No significant increase in cough was noted as a result of O_3 exposure. A study cited in OEHHA (1999) reported that significant O_3 associated decrements in forced vital capacity (FVC), FEV_1, peak expiratory flow rate (PEFR), forced expiratory flow (FEF) 25-75, and the ratio of FEV_1/FVC in healthy adults following outdoor exercise in ambient ozone concentrations of 0.021 - 0.124 mg/L for an average of 29 minutes.

Photochemical Smog

Photochemical smog is a reddish brown haze that is often seen in many urban areas. It is created by sunlight-promoted reactions in the lower atmosphere that has a devastating effect on the environment (Manahan, 2003). Once O_3 and NO_2 together with volatile organic compounds (VOCs) are in the troposphere they combined with hydrocarbons to form another secondary pollutant Peroxyacetyl nitrate (PAN), the oxygen and Nitrogen containing compound found in the troposphere (Manahan, 2003; Illerup, 2008). VOCs consist of a group of more than 100 chemicals formed during incomplete

combustion of fuel and other organic substances, many of which are carcinogenic (Sharman, 2004; Carlisle and Sharp, 2001).

The typical smog episode occurs in hot, sunny weather under low humidity conditions with the characteristic symptoms of brown haze in the atmosphere that causes reduced visibility, eye irritation, and respiratory distress in humans. Prolonged effects on laboratory mice exposed to community photochemical smog levels (0.14 mg/L average of the daily "total oxidant" maxima) over a 16 month period showed an increase in lung tumor development (Louis, 1967). In general, the effect of photochemical smog is the synergistic effect of O_3, NO_2, VOCs and PAN, mostly O_3 as it constitutes about the 90 % of the smog (Illerup, 2008).

Global warming

The release of Greenhouse gases, those that absorb heat radiates from Earth's surface and release some of it back towards the Earth, increasing the surface temperature. These gases benefit humans by maintaining a stable, moderate temperature. A substantial increase in greenhouse gases could harm human life. These gases are: carbon dioxide (CO_2), methane (CH_4), nitrous oxide (N_2O), chlorofluorocarbons (CFCs), ozone (O_3) hydrochlorofluorocarbons (HCFCs), hydrofluorocarbons (HFCs), perfluorocarbons (PFCs) and sulphur hexafluoride (SF_6) (Harrison, 1999; Manahan, 2003). A report by the Intergovernmental Panel on Climate Change (IPCC) shows that global emissions of greenhouse gases have risen to unprecedented levels despite a growing number of policies to reduce climate change (IPCC, 2014). These policies are majorly focused on the reduction, sequestration and storage of carbon dioxide (CO_2) as far as the global warming and climate change issues are considered (Vercruyssen, 2007).

Global warming creates hot weather which can be the cause of heat stress on three aspects of sports participation; comfort, performance, and health/safety. When exercising, the human body can reach temperatures of 41˚C (from normal body temperature of 37°C), and it may be difficult for the body to cool itself down in a hot, humid environment. Typical signs of heat stress include: nausea, cramps, clammy skin, dizziness and confusion (Gassewitz and Radomski, 2008). The body temperature may be cooled by radiation (transfer by electromagnetic waves), convection (wind or air movement), conduction (by contact) or evaporation (by sweating). The most common method of cooling when the surrounding temperature is hot is through sweating. One litter sweat evaporation results in 600 kcal heat loss (Gassewitz and Radomski, 2008). During exercise, sweat can be produced at a rate of over two liters per hour, and sweat losses of over 6 liters per hour have been recorded in marathon runners (Gassewitz and Radomski, 2008)

When the temperature increased above an optimum, performance decreased and the more the temperature increases, the larger the decreases in running speeds. For example, the optimal temperature at which women's maximal running speed attained was 9.9°C, and an increase of 1°C from this optimal temperature will result in a speed loss of 0.03% (Helou et al., 2012). Hot weather and its first measurable impact is the reduction of physical performance (Kenefick, 2007; Maughan et al., 2007; Hargreaves, 2008) as it is detrimental for the cardiovascular, muscular and central nervous systems (Helou et al., 2012). Moreover; warm weather enhances the risk of exercise induced hyperthermia like heat cramp, heat exhaustion and heat stroke. During exercise some of the energy is transformed to external work, but the efficiency of this energy is usually less than 20-25%. Therefore, 75-100% of the liberated energy appears as heat in the active muscle tissue. The amount of heat generated in the body must be dissipated to the environment, or else the heat content and the temperature of the body will increase and endanger the homeostatic setting of the body (Michael et al., 2003). This situation will exacerbate when exercising in hot weather and exposes to heat related illness.

Heat related illnesses represent a spectrum of disease ranging from heat cramps, exhaustion and edema all leads to heat stroke and death; Heat stroke is typically present in warm, humid conditions with elements of overexertion and dehydration on the part of the athlete with high mortality rates if unrecognized and emergency actions have not been taken (Francis et al., 2005).

CONCLUSION

1. Air pollutants are constantly emitted into the atmosphere and affect the health condition of the people specially asthmatics allergic and active individuals.
2. This effect on athletes is pronounced as metabolic demands of exercise increase minute ventilation and therefore the rate of inhalation of pollutants increase.
3. The synergetic effects of principal air pollutants i.e. sulfur dioxide (SO_2), carbon oxides (CO_X), nitrogen dioxide (NO_2), particulate matter (PM), ozone (O_3) and global warming affects both health and performances of athletes.

RECOMMENDATION

1. Policy makers should give emphasis on establishing a station for regular surveillance and monitoring of air quality index
2. Athletes, coaches, managers or sport professionals and any other concerned body should be aware of air pollution impact on athletes.
3. A careful look and regular check up on the daily report

of air quality index is critical before exercise.
4. Sport academies should be built outside of towns with low traffic areas.
5. If trainings performed in town, it should be done during low traffic times.

CONFLICT OF INTERESTS

The authors have not declared any conflict of interests.

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