# The effect of air pollutants on heart rate during Physical Activity

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**Abstract:** In multiple epidemiologic investigations, air pollution has been associated with increased heart rate and decreased heart rate variability. Recent studies on air pollution and heart rate have suggested that autonomic imbalances, as evidenced by increases in heart rate and decreases in heart rate variability, may specifically contribute to the increased mortality. The main air pollutants are divided into 5 main categories, including Particular mater, O3, NO2, SO2 and CO that they can increase the oxygen demand of the heart muscle by increasing heart rate. There are many reports that show the relationship between particulate air pollution and heart rate. The elevated heart rate and myocardial ischemia seen with exposure to air pollutions prior to exercise are important to consider in exercise populations with compromised cardiovascular function, who may not be too able to accommodate the additional cardiovascular strain. Therefore, it is important to consider how air pollution will affect exercise testing and prescription by those involved in cardiac rehabilitation programs. Individuals should take into account the method of transport to an exercise location. In particular, competing athletes and those with heart disease could consider avoiding traffic prior to exercise to minimize the health risk and potential effects on exercise performance.
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## Introduction

The growing air pollution in large cities, especially in developing countries threatens the health of the residents of these cities seriously. Air pollutants are substances that affect the quality of the environment. Today the Contamination of the environment around athletes and ordinary people who work in an open environment, during activities is a concern for them.

Pulmonary irritants such as PM stimulate the sympathetic nervous system, and inhibit the parasympathetic nervous system, causing an increase in heart rate (Paton., 1998; Neas., 2000; Brook et al., 2010). An elevated resting heart rate has been recognized as a risk factor for all-cause mortality as well as for cardiovascular mortality independent of other major risk factors (Wannamethee et al., 1995; Shaper et al., 1993; Dyer et al., 1980). Moreover Epidemiologic investigations have observed that exposure to particulate air pollution is associated with increased mortality and morbidity, especially cardiovascular morbidity and mortality (Pope and Dockery., 1999). Acceleration of heart rates and diminished heart rate variability in association with air pollution may lead to cardiac events (Seaton et al., 1995, Pope et al., 1999a; Pope et al., 1999b; Liao et al., 1999; Gold et al., 2001). One study reported that heart rate variability started to decrease within hours of exposure (Liao et al., 1999).

Three reasons why athletes are at special risk of inhaling pollutants have been put forward by McCafferty (McCafferty., 1981). firstly, when sports activities, according to the increased metabolic needs, the amount of air that enters the respiratory system, may even be more than 20 times (Lippi et al., 2008) So a very large volume of polluted air, Enters the lungs compared to the rest and Exacerbates its harmful effects on health. Secondly, during exercise, most air enters through the mouth, that limit clearing the nasal breathing air (Basrur., 2003). Thirdly, the increased airflow velocity carries pollutants deeper into the respiratory tract. Furthermore, pulmonary diffusion capacity has been shown to increase with exercise (Turcotte et al., 1992; Turcotte et al., 1997; Stokes et al., 1981; Fisher and Cerny., 1982); it may therefore be postulated that the diffusion of pollutant gases increases with exercise.

In a cross - sectional study, Cakmak et al found a significant associations between exposures to elevated levels of air pollutants and increased heart rate (Cakmak et al., 2011). Pope et al showed that Daily pulse rate, as well as the odds of having a pulse rate elevated by more than 5 or 10 beats/min, were associated with exposure to particulate air pollution on the previous 1 to 5 d. The association between

pulse rate and PM10 was small compared with the overall variability in pulse rate (Pope et al., 1999a).

Weichenthal et al examined the association between Traffic-related air pollutants and acute changes in heart rate (HRV) during cycling and their findings suggest that short-term exposure to traffic-related air pollution may contribute to changes in the autonomic regulation of the heart in the hours immediately after cycling (Weichenthal et al., 2011). Compared to FA, Giles et al found that pre-exercise exposure to DE (diesel exhaust) significantly increased heart rate during exercise. This study addressed how the respiratory system and heart rate are affected by air pollution exposure prior to exercise, and suggested that a 60-min exposure to DE (300 µg/m3 of PM2.5) prior to exercise significantly attenuated exercise-induced bronchodilation and increased heart rate during exercise (Giles et al., 2012). Nazari et al showed Air pollution did not affect heart rate in resting position; however, in training position, its effect was significant on heart rate (Nazari et al., 2014).

**Five major of air pollutants**

The main air pollutants are divided into 5 main categories, including Particular mater, O3, NO2, SO2 and CO.

Table 1- Authorized standards of five air pollution According to two units of measurement

| pollutant | Time of measurement | Time measurement | amount of pollutants | Unit of measurement 1 | Unit of measurement 2 |
|---|---|---|---|---|---|
| CO | Hourly<br>Hourly | 8<br>1 | 9<br>35 | ppm<br>ppm | (10(mg/m3))<br>(40(mg/m3)) |
| SO2 | Yearly<br>Hourly | 24 | 140<br>32 | ppb<br>ppb | (365(µg/m3))<br>(80(µg/m3)) |
| NO2 | Yearly<br>Hourly | 1 | 210<br>53 | ppb<br>ppb | (400(µg/m3))<br>(100(µg/m3)) |
| PM10 | Yearly<br>Hourly | 24 | 150<br>50 | µg/m3<br>µg/m3 | |
| O3 | Hourly | 1 | 12 | ppb | (234(µg/m2)) |

**1-Particular matter**

Particular matter are including fine solid material (soluble or non-soluble) or liquid that as a percentage of Remain in the air for hours or even days. Health effects of particulate matter on human depends on the size, concentration and their chemical compounds (Schwela, 2000).

Zereba et al reported that increased levels of PM10 particles are associated with increased heart rate (Zereba et al., 2001). Peters et al, showed that increasing the concentration of Particular maters to 75 micrograms per cubic meter, increased heart rate 12/1 beats per minute (Peters et al., 1999). In researches of Ibald-Mulli et al, 131 subjects were tested and it was found that tiny particles maters reduce heart rate (Ibald-Mulli et al., 2004).

To address the possible mechanisms for PM-cardiovascular disease mortality, Liao et al. examined the cardiac autonomic response to daily variations in PM in 26 elderly (mean age 81) individuals for 3 consecutive weeks. Results show that the odds ratio (95% confidence interval) of low heart rate variability high frequency for high (vs. not high) pollution days was 3.08 (1.43, 6.59) (Duanping et al., 1999).

Pope et al observed associations between exposure to particulate air pollution and HR and HRV. They reported mean HR was positively associated with PM10. Higher levels of PM10 were associated with reduced SDNN, the estimate of overall HRV, and reduced SDANN, the estimate of long-term components of HRV. In contrast, higher levels of PM10 were associated with increased r-MSSD, an estimate of the short-term components of HRV. The associations between HRV and PM10 persisted even after controlling for mean HR (Pope et al., 1999b). By the same token Dockery et al showed that Elevated PM10 levels in the previous 1–5 days have been associated with an increase in resting heart rate (Dockery et al., 1999).

while Rundell et al did not find that heart rate was affected during exercise in a high PM1 environment (Rundell and Caviston, 2008) and 12 healthy volunteers exercise exposed to diesel exhaust gases of the vehicle under Donaldson et al and it was observed that heart rate did not change (Donaldson et al., 2009), that these results did not match with the results listed above, however, the differing results between various studies could be related to exposure duration, exposure timing (before exercise vs. during exercise) and fuel type (diesel vs. gasoline or etc).

**2- Ozone**

Ozone, a ubiquitous constituent of the upper atmosphere and toxic contaminant predominant in the photochemical smog of numerous metropolitan areas, is one of the most potent oxidising agents in the atmosphere (Mustafa and Tierney., 1978; Stokinger and Coffin., 1968) and it is a secondary pollutant with high chemical activity. At concentrations typical of photochemical smog episodes, it readily reacts with various cellular constituents causing damage to the respiratory tracts of animals (Jaffe., 1968; Mautz et al., 1985; Mustafa and Tierney., 1978), as well as to agricultural crops (Menzel., 1970; Stokinger and Coffin., 1968). The total amount of ozone inhaled in a given exposure time (usually 1 to 2 hours) has been increased by employing heavier exercise.

Gong and colleagues conducted a study to determine the effects of O3, on cardiac function. Heart rate, when exposed to O3, was higher than exposed to air. These effects in people who have coronary disease, was more important (Gong et al., 1998). Maximal oxygen uptake was shown by Folinsbee et al to decrease by 10.5% following a 2-hour exposure to 0.60 ppm ozone with moderate intermittent exercise (Folinsbee et al., 1977). Then Adams showed that Decrements in VO2max were accompanied by decreases in maximum heart rate (HRmax) (Adams., 1987). And finally no significant O3 effects on heart rate (HR) were found in volunteers with coronary artery disease (CAD) after 40 min exercise in 0.2 or 0.3 ppm O3 in superko et al's researches (superko et al., 1984), and because of a decrease in maximum exercise time, maximum heart rate may even be reduced (Foxcroft and Adams., 1986).

**3- Nitrogen dioxide (NO2)**

Investigations on the effects of exposure of human subjects to nitrogen dioxide (N02) have mainly focused on pulmonary function and lung inflammation.

Research of gold and colleagues on the effects of NO2 on heart rate Showed that NO2 was not associated with increased heart rate and decreased heart rate variability (Gold et al. 2001).

**4- Sulfur dioxide (SO2)**

Paula Santos et al. have assessed the effects of air pollution on heart rate variability in healthy vehicular traffic controllers in the city of Sa˜o Paulo, using 24 h electrocardiographic. Pollution impacted on HRV indicators during the winter. SO2 effects on SDNN and SDANN were mainly negative. Changes were observed for SO2 concentration on the concurrent day (lag 0) and the prior 4 and 5 days. The r-MSSD, an estimator of short-term variation in heart rate modulated by the parasympathetic nervous system and correlated with HF, was acutely affected by SO2 (Paula Santos et al., 2005), as reported by other authors (Pope et al., 1999b; Liao et al., 1999; Gold et al., 2000; Magari et al., 2001). Peters and colleagues showed that exposure to SO2, increases heart rate 1.75 beats per minute compared to a healthy environment (Peters et al., 1999).

**5- Carbon monoxide (CO)**

CO is a colourless and odourless toxic gas which is among the most common air pollutants. If we breathe air containing carbon monoxide, some of it combines with hemoglobin in blood and forms carboxyhemoglobin (Carlisle and Sharp., 2001). The junction of carbon monoxide is exactly where the oxygen binds but carbon monoxide binding affinity to hemoglobin 200 times more than binding affinity of oxygen to hemoglobin (Carlisle and Sharp., 2001).

COHb levels of 5 % can increase heart rate during exercise (Kizakevich et al., 2000), with the magnitude of increase rising substantially when COHb reaches 15 % (Pirnay et al., 1971). This increase could be due to the relative hypoxemia caused by the COHb displacing oxygen. The effects of raised COHb on exercise performance have indicated a significantly lower VO2MAX, anaerobic threshold, and oxygen pulse (VO2/heart rate), and a significantly higher heart rate and pulse pressure (Hopkins., 1990).

During the 1985 air pollution episode, increases in heart rate determined by a resting electrocardiogram were present in a random sample. Even after adjusting for cardiovascular risk factors and meteorologic parameters, the increases in heart rate were apparent. Elevated heart rates were consistently observed in association with concentrations of SO, total suspended particulates, and CO. The effects of the episode seemed to be stronger in women than in men (Peters et al., 1999).

Adams et al researched on Exercise air versus carbon monoxide exposure. They showed There was no significant difference in rest heart rate between the exposure days (60 * 1 .8 beats/min for air versus 60. 1 .8 beats/min for carbon monoxide). During exercise There was a trend for maximal heart rate to be higher after carbon monoxide (105± 3 .7 beats/min for air versus 108 ± 3.5 beats/min for carbon monoxide, p = 0.06); but maximal rate pressure Product (20.389 ± 1080 for air versus 20.515 ± 1 .039 for carbon monoxide) and peak work load (312 ± 28 versus 303 ± 26 kp-m) were indistinguishable on the 2 exposure days (Adams et al 1988).

**Discussions**

Because high levels of air pollutants increase symptoms during exercise, this could also discourage an individual from exercising, thus reducing physical activity levels and health status. Therefore, to avoid exposure to air pollution, individuals should be encouraged to exercise away from traffic.

Researchers could consider direct measures of cognitive performance following exposure to PM. Given the pathophysiologic basis of impaired vascular function with PM exposure, the effects during exercise warrant further investigation. Many exercise studies do not have a control resting condition making it hard to disentangle the effects of exercise from air pollution; therefore, future research should take this into account. More information on the cardiovascular, respiratory, and systemic responses to air pollution exposure are needed, particularly for PM. How these responses change with temperature, exercise intensity, sex, and different cohorts such as the elderly, the young, and those with pre-existing diseases would be important. For laboratory-based studies the role of pollutant synergy/co-exposure during exercise is important to understand.

It is advisable to exercise whenever possible in open rural or park land. High momentary peaks can occur in the levels of any of the pollutants. Try to avoid the rush hour when NOx, CO, and VOCs are likely to accumulate. If it is cold and smoggy, exercise indoors. Windy weather tends to dilute and disperse the pollutants.

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