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Association between blood pressure and physical activity in air pollution

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ABSTRACT: Routine physical activity has been shown to reduce blood pressure, decrease blood coagulation, improve coronary blood flow, and augment cardiac function. There are excellent reviews which cover other cardio vascular effects of air pollution which may include alterations in flow - mediated vasodilation, heart rate variability, and hospital admissions and mortality due to cardio vascular disease. Various research studies on the effects of air pollution on blood pressure, lead to different results. However, this inconsistency was also because of difference in type of pollutants or intensity of exercise protocol or difference in age and fitness of the participants.

***Keywords*:** physical activity, blood pressure, air pollution.

INTRODUCTION

An excellent review of the topic by Thompson and colleagues revealed that acute, dynamic exercise may result in transient changes in the form of reductions in triglyceride levels, increases in HDL cholesterol level, decreases in blood pressure (for 12–16 hours), reductions in insulin resistance and improvements in glucose control (Thompson et al., 2001). These acute changes indicate the important role individual exercise sessions have on health status.

But is it useful exercise in polluted air? Most research to date have examined lung function and Changes in ECG and oxygen saturation and heart rate and blood pressure during exercise in polluted environments in healthy subjects, have been less studied.

In humans, environmentally relevant concentrations of PM2.5 and O3 acutely augment brachial artery vasoconstriction (Brook et al., 2002). Rats exposed to the same pollutants also show elevated concentrations of plasma endothelin-1, a powerful vasoconstrictor (Bouthillier et al., 1998). These data are of particular interest to cardiovascular risk in humans, because augmented vasomotor tone (vasoconstriction) has the potential to increase myocardial afterload and ischaemia. Each time the heart contracts, a pressure wave travels through conduit arteries of low resistance to peripheral arteries of higher resistance. A portion of the pressure wave is reflected back to the heart (wave reflection) and the intensity of this reflection is dependent on the tone of the large conduit arteries. Increased large artery vasoconstriction causes increased wave reflection, such that there is an early return to the heart of the arterial pressure waveform. This early returning waveform boosts central (aortic) systolic blood pressure (afterload), in addition to diminishing the time and pressure of coronary artery perfusion, thus promoting ischaemia (Nichols and O'Rourke, 1998). If regular exercise in a polluted environment exacerbates this effect, risk would be enhanced, because central, and not peripheral, blood pressure correlates with left ventricular hypertrophy (Marchais et al., 1993), carotid intima media thickness (Boutouyrie et al., 1999) and all-cause mortality (Safar et al., 2002).

Various research studies on the effects of air pollution on blood pressure, lead to different results

Several, but not all studies of air pollution and blood pressure have found positive associations. Drechsler- Parks using non-invasive cardio-graphic impedance for cardiac output, has examined healthy subjects after 2 hours of exposure to NO_2 environments with 0.60ppm concentration and in O_3 environment with 0.4ppm concentration and

combination of NO_2 and O_3. He found out that a combination of NO_2 and O_3 decreased cardiac output compared with the individual gases, but there was no change in blood pressure (Drechsler- parks, 1995).

Volpino et al, Examined the relationship between air pollution and cardiovascular function at rest and during exercise test between the Police traffic. 130 people, including 68 Policemen Street and 62 police inside the office were examined. There was no significant difference between the two groups in terms of smoking, drug use, etc. Subjects were tested with ECG and spirometry test by bicycle ergometer and have been showed significant changes in cardiopulmonary function. Policemen Street had high diastolic blood pressure at rest and during exercise test. (Volpino et al., 2004).

Linn et al, concluded about 30 subjects and showed with $1mg/m^3$ increase in particulate matter (PM) increased the amount of systolic blood pressure and diastolic blood pressure on the value of 172/0 mm Hg and 095/0 mm Hg respectively. PM_{10} was related to blood pressure but this relation was significant in $PM_{2/5}$ (Linn et al., 1999), and they found that systemic blood pressure in healthy subjects is slightly reduced exposure to NO_2 (Linn et al., 1985).

Gong et al, Conducted a study on the effects of O_3, on the heart function. Categories of subjects with high blood pressure and those without hypertension were exercised for 3 hours with cardiac and arterial catheterization in a medium containing 0/3 ppm O_3. No difference was observed in the indices of cardiac function. However, heart rate, speed of pressure generated, the slope of the alveolar-arterial oxygen pressure was higher when exposed to O_3, relative to the air (Gong et al., 1998).

Ibald-Mulli et al's research showed that air pollution increases systolic blood pressure, especially in people with the disease of high viscosity of plasma or heart palpitations (Ibald-Mulli et al., 2001), While superfine particles significantly reduces systolic and diastolic blood pressure, mainly among those with previous myocardial infarction (Ibald-Mulli et al., 2004).

Researches on 62 subjects with heart disease revealed that air pollution increased systolic blood pressure, diastolic blood pressure and mean arterial blood pressure (Zanobetti et al., 2004). After exercising of 12 healthy subjects exposed to emissions from diesel vehicles, there is no change in systolic and diastolic blood pressure (Donaldson et al., 2009). And Brauer et al's research revealed that exposure to carbon monoxide, is inversely associated with systolic blood pressure (Brauer et al., 2001). Also Polluted air did not have a significant effect on systolic and diastolic blood pressure in rest but its effect was significant in activity (nazari et al., 2014).

Rest blood pressure was slightly higher after air as compared with carbon monoxide exposure (157 ± 4.2 mm Hg for air versus 152 ± 4 .0 mm Hg for carbon monoxide, $p < 0.05$). Maximal blood pressure achieved during exercise did not d41cr by exposure (192 ± 4.6 mm fig for air versus 139 ± 5 .1 mm Hg for carbon monoxide) (Adams et al., 1988).

Phenanthraquinone, a constituent of diesel exhaust, inhibits nitric oxide production in bovine endothelial cells, increases blood pressure and suppresses nitric-oxide-mediated vasodilatation in rats (Kumagai et al., 2001). Rundell et al found no conduit artery basal vasoconstriction in healthy young males for up to 24 h after acute 30 min high PM exposure from 2-cycle gasoline engine exhaust during exercise (Rundell et al., 2010). There was a significant vasoconstrictive response after exercise while inhaling 4-cycle gasoline engine exhaust (Rundell et al., 2007) and Brook et al found significant ($p = .007$) vasoconstriction of the brachial artery after 2-h exposure of concentrated ambient particles (150 μg·m−3, PM2.5) plus ozone (120 ppb) (Brook et al., 2002). An acute vasoconstrictive response related to increased plasma ET-1 was identified after 2-h diesel exhaust exposure (Peretz et al., 2008). Recently, relationships between diastolic blood pressure increase and particle concentrations in two inhalation studies were identified, suggesting a basal vasoconstrictive response in the peripheral vasculature (Brook et al., 2009).

Cakmak et al's study shows that exposures to elevated levels of air pollutants were associated with higher resting blood pressure (Cakmak et al., 2011). Among sixty-four retirees studied by ambulatory blood pressure monitoring for ten days, a 5.2 mg/m3 increase in five-day averaged organic carbon was associated with increased systolic and diastolic blood pressure of 8.2 (95% CI 3.0,13.4) and 5.8 mmHg (95%CI3.0–8.6), respectively (Delfino et al.,2010). No statistically significant effects were observed for PM10 effects in particular blood pressure changes (Brauer et al., 2001; Linn et al., 1999; Brook et al., 2002).

In the investigation of superko et al, a group of six patients with documented coronary heart disease, specifically, symptom-limited angina, were exposed to filtered air and to 0.20 and 0.30 ppm ozone for 40 minutes while completing a simulation of their prescribed cardiac rehabilitation exercise session. In none of the patients was pulmonary function, exercise ventilatory pattern (ventilation volume or respiratory frequency, oxygen uptake), or cardiovascular response (heart rate, systolic blood pressure, rate-pressure product, or electrocardiographic results) to ozone exposure of 40 minutes altered in a statistically significant manner (superko et al., 1984).

Paula Santos has assessed the effects of air pollution on heart rate variability and blood pressure in 48 healthy subjects aged 31 to 55, vehicular traffic controllers in the city of Sa˜o Paulo, using 24 h electrocardiographic and blood pressure monitoring. The present study showed that effects of air pollution on blood pressure were remarkable.

CO had both acute and lagged effects, whereas SO2 effects were mainly lagged. They observed that an increase in carbon monoxide by 1/1 PPM, provides an increase in systolic blood pressure and diastolic blood pressure by 6.2 mm Hg and 8/1 mm Hg respectively. In summary, this results show that in adult and healthy workers directly exposed to automotive traffic-generated air pollution, increases in primary gaseous pollutants were associated with changes in blood pressure and HRV. Despite the high correlation between primary pollutants, the effects for PM10 and NO2 were not found. (Paula Santos et al., 2005).

However, this inconsistency the differing results between various studies could be related to difference in environments, type of pollutants or intensity of exercise protocol or difference in age and fitness of the participants. The authors suggest that medication intake and disease status, both affecting the autonomic control of the heart, may explain the differences between various studies (Linn et al., 1999; Ibald-Mulli et al., 2001).

CONCLUSION

Air pollution can cause blood clots, coronary atherosclerosis, angina and even infarction (Vermylen et al., 2005). Other complications such as tachycardia, hypertension and anemia caused by contamination with heavy metals (particularly mercury, nickel and arsenic) (Huang, and Ghio, 2006). Particle pollution also can increase the oxygen demand of the heart muscle (Pekkanen et al., 2002) by increasing blood pressure (Ibald-Mulli et al., 2001). After breathing PM, its components (metals and organic particles) enter the bloodstream and stimulate stress oxidative reactions inside the vessels, thus directly causes narrowing of the blood vessels (Huang and Ghio, 2006) and ultimately increases the blood pressure. PM2.5, because of its smaller size and chemical activity, can penetrate the small airways and alveoli of the lung and enter the blood circulation (Nemmar et al., 2002). Exact biologic mechanisms by which recent PM2.5 exposure may increase risk of acute cerebrovascular events are not known but possible mechanisms include acute arterial vasoconstriction (Brook et al., 2002), acute increases in blood pressure (Urch et al., 2005) and acute increases in plasma viscosity (Peters et al., 1997).

There are many hypotheses to explain the effects of air pollutants on cardiovascular diseases. Among them, changes in blood markers associated with increased cardiovascular risks (Seaton et al., 1995; Schwartz, 2001) ischaemic response in the myocardium (Pekkanen et al., 2002), and effects on the autonomic nervous system (Peters et al., 2000; Pope et al., 1999a; Pope et al., 1999b; Liao et al., 1999; Gold et al., 2000; Magari et al., 2001; Ibald-Mulli et al., 2001) which are related to blood pressure and HRV changes. The cardiovascular system and HR are permanently under the influence of the sympathetic and parasympathetic nervous systems, with a predominance of the latter. Mental stress, physical exertion, and stimuli promoted by outdoor air pollution, tobacco smoke, and weather variables may change the balance between the two systems, decreasing parasympathetic influences and increasing the sympathetic tone. This new situation leads to increases in blood pressure and HR, decreases in HRV, and lowers the ventricular fibrillation threshold (Kennedy, 1997). Hence, the risks of cardiac arrhythmia, sudden death, and other cardiovascular events are higher (Stone and Godleski, 1999; Kennedy, 1997; Huikuri et al., 2001; Van den Hoogen et al., 2000; Tsuji et al., 1994; Clement et al., 2003).

Although the blood pressure variations may have no acute clinical implications in healthy individuals, minor blood pressure variations may trigger arrhythmia, myocardial infarction, and stroke in those with cardiovascular co-morbidities (Kennedy, 1997; Van den Hoogen., 2003; Peters et al., 2001; Hong et al., 2002).

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