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RESPIRATORY PHYSIOLOGY

Effects of Polluted Air on Cardiovascular and Hematological Parameters After Progressive Maximal Aerobic Exercise

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Abstract
Purpose Exercising or doing physical activity in polluted air could expose a person to the adverse health effects of air pollution. This study aimed to compare the cardiovascular and hematologic indices following an incremental exercise test (shuttle run) under clean versus polluted air conditions.
Methods Nineteen male athletes aged 21–27 years were assigned to either a trained athletes group (TA, $n = 10$) or a complete training cessation/detrained athletes group (DA, $n = 9$) at least 3–4 months after their competitive season. All participants performed the multi-stage shuttle run test on two separate days in either polluted air (37.4 carbon monoxide part per million) or clean air (2.5 carbon monoxide part per million) condition.
Results When compared to the clean air environment, progressive incremental exercise in polluted air condition significantly ($p < .05$) decreased maximal oxygen uptake (VO_{2max}), red blood cell count, and hematocrit for both TA and DA groups. Meanwhile, the participants' mean corpuscular hemoglobin, mean red blood cell volume, white blood cells, and platelets in these two groups increased significantly ($p < .05$) when they were exercised in the polluted air ambiance. Maximal heart rate and heart rate recovery showed significant ($p = .04$) increases only in the DA group. However, hemoglobin concentration remained unchanged in both groups.
Conclusion Acute exposure to high concentrations of pollutants during exercise resulted in decline in cardiovascular functions and hematological parameters in healthy athletes.

Keywords Air pollution · Blood parameters · Cardiovascular parameters · Progressive exercise

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Introduction

In recent decades, air pollution in metropolitan cities has become a major health concern to the residents. Exposure to polluted air has many damaging effects on human health. Polluted air contains materials which may enter the human bloodstream through the nose, mouth, skin, and digestive system. Most of the air pollutants appear in the blood have no markedly negative effects [1, 2]. However, studies on animals demonstrated that fine particles sediment onto alveolar surfaces will then be phagocytosed by macrophages. In turn, this leads to the production of

mediators which enter the blood stream and stimulate bone marrow to increase the production of white corpuscles [3, 4]. Air pollution is now one of the biggest bioenvironmental problems in the world. A huge amount of pollutants, especially carbon monoxide exhausted from vehicles, is released into the air every day [5, 6]. This has a huge impact on the quality of air we are breathing into our lungs. Increased level of carbon monoxide in the blood impairs oxygen uptake and transfer by erythrocytes [7]. The blood cells are very sensitive to bioenvironmental poisons and some research has shown that in the cold months of the year, red blood cell counts may increase because of the higher levels of air pollution [6].

While regular physical activity has been shown to have various physiological benefits and improve well being, lack of physical activity can increase the incidence of diseases, mortality, and morbidity rates [8]. This is further accentuated by air pollution which has been associated with high blood pressure, respiratory difficulties, and death in both children and adults. About 800,000 people in the world had died each year because of diseases related to air pollution [9]. Despite the potential benefits of physical activity, exercising vigorously for extended periods of time in polluted air put individuals at risk of inhaling more pollutants and thus increases the appearance of pollutants in the blood stream [10, 11].

Most sports programs and physical activities that are performed in urban environments take place in the open air with close proximity to the roads that have heavy or congested traffic. It is estimated that greenhouse gas emissions by automobiles are the largest cause of urban pollution, and the toxic compounds from such pollutants lead to various health problems such as cardiovascular disease, respiratory disease, and cancer [12]. Although individuals should not ignore regular sport and its benefits, they should not exercise in highly polluted areas. However, to assess the detailed effects of air pollution on participants in sport or exercise remains challenging [13]. A number of the adverse effects of air pollution on daily fluctuations in the level of pollutants in the air have been connected with a wide array of health issues, including decreased lung function, in a significant number of studies. This is particularly true among certain segments of the population (e.g., athletes and physically active individuals) that are deemed susceptible [14].

It is important to examine the well being of the athletes who live and train in such large cities and metropolitan areas as Tehran and Isfahan where the air pollution is greater than ever. For this reason, the purpose of the present investigation was to assess the short-term effects of progressive maximal aerobic exercise in polluted air on cardiovascular parameters such as VO_2max, maximal heart rate, recovery heart rate, hematological parameters including complete blood count, as well as red and white blood cell counts (both of which are sensitive to acute effects of air pollution) between two groups of young male trained and detrained athletes.

Methods

Participants

Nineteen non-smoking male athletes between the ages of 21 and 27 years volunteered to participate in the study. Ten participants were randomly assigned to a trained athletes (TA) group, and nine participants were assigned to the detrained athletes (DA) group. Participants were included in the study once they met all the following inclusionary criteria: apparently healthy, non- smokers, and participating in regular aerobic training at least 3–4 days per week for 3–4 years for the TA group and not participating in regular aerobic training during the week for at least 3–4 months for the DA group. Procedures were performed in accordance with the Helsinki Declaration for the ethical treatment of human participants. Written informed consent was provided to all participants prior to their participation in the study. The research was approved by the University of Isfahan's Ethic Committee.

Study Design

The study made use of a quasi-experimental design with pretest and posttest measurements without a control group. Prior to the study, participants were familiarized with all experimental methods and procedures. The participants reported to the laboratory in the morning after an overnight fast for eight hours. Immediately before the tests, blood samples were collected from a forearm vein. Participants participated in a pacer test (Shuttle run test Type 2) on two separate days with different pollutant standard indexes (PSI). PSI was directly derived from the Isfahan Meteorological Bureau in the morning of test day. The Bureau used the data collected from the Azadi Square pollution detector, which is the nearest detector to University of Isfahan, Isfahan, Iran, and test place. The critical pollutant in the polluted day was carbon monoxide. At least two hours after having breakfast, participants from both groups were asked to perform a maximal incremental exercise until exhaustion using the multi-stage shuttle run test Type 2 in a clean air environment (with PSI of 62). In a subsequent visit (7 days later), the participants were asked to perform another maximal incremental exercise until exhaustion using the multi-stage shuttle run test Type 2, but this time in a polluted air ambiance (with PSI of 213). The test procedure was randomized in order to avoid

Table 1 Pollutants density and pollution standard index of clean air and polluted air conditions

	Low-polluted area	High-polluted area
Humidity (%)	55.7	57.2
Temperature (°C)	6.3	6.1
Altitude (m)	1,626	1,626
CO (ppm)	2.3	37.4
O_3 (ppb)	1.5	11.2
PM_{10} (μg/m^3)	19	250
NO_2 (ppb)	18.7	47.5
SO_2 (ppb)	18.4	46.9
PSI	<50	>200

ppm part per million, *ppb* part per billion, *CO* carbon monoxide (acceptable level: 9 ppm); O_3 ozone (acceptable level: 0.08 ppb); PM_{10} particular matter 10 (acceptable level: 50 μg/m^3), NO_2 nitrogen dioxide (acceptable level: 0.05 ppb), SO_2 sulfur dioxide (acceptable level: 0.03 ppb), *PSI* pollution standard index (good: 0–50; moderate: 51–100; unhealthy: 101–199; very unhealthy: 200–299; and hazardous: >299)

familiarization and the two tests were interspersed by one week. The PSI was used to determine air quality (Table 1). Physical and physiological assessments and blood sample collection were performed immediately before and after exercise. Maximal heart rate was recorded following the test and recovery heart rate was recorded 3 min after the end of the test using a heart rate monitor (Polar Electro S810, Finland).

Study Location

Isfahan city is located about 340 km south of Tehran, at a longitude of 51° 40′ and latitude of 32° 38′. The city's average altitude above sea level is approximately 1,570 meters. The area of the city is approximately 18,585 hectares and its population is estimated at around 2 million [6]. At present, air pollution is the greatest bioenvironmental threat in Iran, with the cities of Tehran and Isfahan being most affected. Cars release around 1.5 million tons of contaminants each year, especially carbon monoxide, into the air of these cities [5, 6].

Measurements

Anthropometric Variables

Body mass was accurately measured to half a kilogram with the participants wearing light clothing and no shoes (SECA700; CA, USA). Seca height gages were used to measure height to the nearest half a centimeter. From these two variables, BMI was calculated and recorded [15].

Measurement of VO_{2max}

To estimate VO_{2max} of the participants, the 20-m multi-stage shuttle run test was conducted and absolute VO_2max was reported in ml/min [16, 17].

Blood Samples

The participants were asked to refrain from any exercise 24 h before the test. Venous blood samples (5 ml) were taken from an antecubital vein of participants for a full blood test which included red blood cells (RBC), hematocrit (HTC), hemoglobin count (HB), mean corpuscular hemoglobin (MCH), mean corpuscular volume (MCV), white blood cell (WBC), and platelet (PLT) counts. The blood specimens were measured by the American hematology system H3 (Homolog 3) method (i.e., the peroxidase content was measured when the blood cells were passing in front of a light).

Exercise Protocol

The 20-m multi-stage shuttle run test had an initial running velocity of 8.0 km per hour (km/h), then increased to 9.0 km/h for level 2, and then increased by 0.5 km/h each minute thereafter [18]. VO_{2max} was calculated using the following formula:

$$VO_{2max} = 6 \times (\text{Velocity}) - 27.4$$

Immediately after the exercise test, the participants' maximal heart rate (HR_{max}) was measured using a Polar pulse meter which was worn on a belt around the chest with the receiver on the participant's wrist. Three minutes after the end of the test, the recovery heart rate was measured with the same aforesaid procedures.

Statistical Analysis

All data were expressed as mean ± standard deviations. The normality of the distribution of the variables was tested using a Kolmogorov–Smirnov test. Independent t-tests were used to compare the trained and detrained groups prior to commencing with the study. Paired t-tests were used to compare the two experimental states (i.e., clean air and polluted air) in both the TA and DA groups. SPSS for Windows (Version 18.0) was used for data analysis, and the significant level was set to $p < .05$.

Results

This purpose of this study was to compare the cardiovascular and hematologic indices following an incremental

exercise test under clean and polluted air conditions. The distribution of environmental pollution levels (under clean and polluted air conditions) during the study period is depicted in Table 1. The temperature and humidity of both testing conditions were identical, but the pollutants density and PSI of the polluted air environment were much higher than those in the clean air atmosphere. Table 2 shows the physical and physiological characteristics in trained and detrained athletes at the baseline evaluation. Significant ($p = .001$) differences were found in VO_{2max} values at baseline between the TA and DA groups under the clean air condition.

Comparing cardiovascular parameters between the groups exercising in clean or polluted air.

The mean values of the cardiovascular parameters of the trained and detrained groups in clean and polluted air conditions as well as the associated statistical analyses are presented in Table 3. When compared to the clean air condition, the participants completed the progressive incremental exercise in the acute polluted air environment showed a decline in VO_{2max} by 3.44 % (from 52.40 to 50.60 ml kg^{-1} min^{-1}, $p = .005$) in the TA group and a drop of 4.55 % (from 43.93 to 41.93 ml kg^{-1} min^{-1}, $p = .020$) in the DA group. Significant ($p = .04$) increases in maximal heart rate (MHR) were found only in the DA group (from 167.78 to 181.11 beats min^{-1}), whereas no significant ($p > .05$) differences were observed in the heart rate recovery (HRR) between the polluted air and clean air conditions for both the TA and DA groups (Table 3).

Comparing hematological parameters between groups exercising in clean or polluted air.

When compared to the clean air condition, the red blood cell count of those participants exercised in the polluted air environment decreased significantly ($p = .003$) both in the TA group (from 5.53 to 5.14) and the DA group (from 5.66 to 5.34), whereas the hematocrit dropped by 4.66 % (from 45.26 to 43.15, $p = .026$) and 3.40 % (from 45.02 to 43.49, $p = .054$) for TA and DA groups, respectively. On the other hand, the mean corpuscular hemoglobin increased significantly by 4.98 % (from 27.73 to 29.11, $p = .002$) and 5.61 % (from 27.08 to 28.60, $p = .001$) for these two groups, respectively. Likewise, the mean red blood cell volume increased by 2.51 % (from 81.60 to 83.65, $p = .001$) and 2.34 % (from 79.66 to 81.55, $p = .004$), whereas the white blood cells inflated by 21.53 % (from 96.60 to 117.40, $p = .045$) and 10.00 % (from 92.33 to 101.56, $p = .005$) for the TA and DA groups, respectively. The same was true for the platelet count, which increased dramatically from 309.43 to 365.10 (+18.00 %, $p = .024$) in the TA group and from 296.78 to 379.78 (+27.63 %, $p = .001$) in the DA group. Nonetheless, no significant ($p < .05$) changes were detected in hemoglobin in any of these groups. There were also no significant ($p < .05$) differences in any of the measured hematological parameters between the two groups after the progressive aerobic exercise in either the clean or polluted air condition. A comparison of the hematological parameters between the TA and DA groups tested under the two different environments is presented in Table 4.

Discussion

Exercise and physical activity in polluted air could cause a person to be exposed to the adverse health effects of air pollution. This study aimed to compare the cardiovascular and hematologic indices following an incremental exercise test (shuttle run) in clean versus polluted air conditions. As shown in our findings, progressive aerobic exercise under polluted air rather than clean air condition can cause a reduction in VO_{2max}. The reason for such change is because of the decrease in the amount of oxygen transferred in the blood. Since large amounts of oxygen are needed to produce sufficient energy during long-term aerobic activities, any inhibition in transferring oxygen to the active tissues leads to marked deterioration in the physical performance [19–21]. In the study of Brook et al., the heart rates measured immediately after the shuttle test and after a 3-min recovery were significantly higher after exercise in the polluted air than in the clean air condition [22]. The findings of the current study are inconsistent with that of Brook et al. probably because the participants in our study had a better air exchange mechanisms or they are much "fitter" than the subjects in Brook et al.'s study.

This increase in both MHR and HRR could be due to a fall in oxygen levels in the blood when breathing in the

Table 2 Physical and physiological characteristics in trained and detrained athletes at the baseline evaluation

Characteristics	Trained ($n = 10$) Mean ± SD	Detrained ($n = 9$) Mean ± SD	p
Age (years)	22.60 ± 1.90	23.44 ± 0.53	.210
Height (m)	1.82 ± 0.66	1.77 ± 0.57	.770
Mass (kg)	73.70 ± 7.42	72.67 ± 7.76	.070
BMI (kg/m^2)	22.25 ± 1.88	23.34 ± 2.51	.300
VO_2max (ml kg^{-1} min^{-1})	52.40 ± 4.05	43.93 ± 3.28	.001**

BMI body mass index, *VO_{2max}* maximum oxygen consumption

** $p < .01$

Table 3 Comparison of physiological parameters between athletes in trained and detrained groups under clean and polluted air conditions

Variables	Athletes	Clean air mean ± SD	Polluted air mean ± SD	*p*	Within-group % change
VO_{2max} (ml kg^{-1} min^{-1})	Trained	52.40 ± 4.05	50.60 ± 4.69	.005**	−3.44
	Detrained	43.93 ± 3.28	41.93 ± 3.50	.022*	−4.55
MHR (beats min^{-1})	Trained	166.00 ± 9.66	169.00 ± 14.49	.541	+1.81
	Detrained	167.78 ± 8.33	181.11 ± 16.92	.042*	+7.94
HRR (beats min^{-1})	Trained	101.00 ± 8.76	104.00 ± 12.65	.434	+3.00
	Detrained	112.22 ± 8.33	115.56 ± 10.14	.282	+3.34

MHR maximal heart rate, *HRR* heart rate recovery, % Change = (polluted air value − clean air value)/clean air value × 100

* $p < .05$; ** $p < .01$

Table 4 Comparison of hematological parameters between athletes in trained and detrained groups under clean and polluted air conditions

Variables	Athletes	Clean air mean ± SD	Polluted air mean ± SD	*p*	Within-group % change
RBC (10^{12}/L)	Trained	5.53 ± 0.25	5.14 ± 0.37	.003**	−7.05
	Detrained	5.66 ± 0.25	5.34 ± 0.21	.003**	−5.65
HCT (%)	Trained	45.26 ± 2.51	43.15 ± 2.82	.026*	−4.66
	Detrained	45.02 ± 1.64	43.49 ± 1.80	.054*	−3.40
HB (g/L)	Trained	15.03 ± 0.96	15.08 ± 0.92	.765	+0.33
	Detrained	15.30 ± 0.60	15.24 ± 0.63	.650	−0.39
MCH (pg/cell)	Trained	27.73 ± 1.26	29.11 ± 1.24	.002**	+4.98
	Detrained	27.08 ± 1.13	28.60 ± 1.54	.001**	+5.61
MCV (fL)	Trained	81.60 ± 3.29	83.65 ± 2.60	.001**	+2.51
	Detrained	79.66 ± 2.56	81.55 ± 3.71	.004**	+2.37
WBC (10^{12}/L)	Trained	96.60 ± 42.50	117.40 ± 35.11	.045*	+21.53
	Detrained	92.33 ± 15.98	101.56 ± 13.31	.005**	+10.00
Platelet count (10^{9}/L)	Trained	309.40 ± 79.54	365.10 ± 63.05	.024*	+18.00
	Detrained	296.78 ± 72.29	379.78 ± 73.57	.001**	+27.63

RBC red blood cell count, *HCT* hematocrit, *HB* hemoglobin count, *MCH* mean corpuscular hemoglobin, *MCV* mean corpuscular volume, *BWC* blood white cell count, and % Change = (polluted air value−clean air value)/clean air value × 100

* $p < .05$; ** $p < .01$

polluted air environment, which hindered oxygen transportation to the active muscles [5, 23]. Progressive aerobic exercise in polluted air in both TA and DA groups had different effects on blood factors. In this regard, decreases were observed in RBC, and HCT, and increases were found in MCH, MCV, WBC, and PLT counts. It seems that contaminants in polluted air, especially fine particulates, cause a fall in RBC count. Some researchers have confirmed that these fine particles can have a destructive effect on corpuscles, particularly when doing physical activity in polluted air when inflow to the lungs is more demanding and rigorous [6, 21]. Changes in HCT can also be interpreted in the same way as the reduced number of RBC [8]. An increase in MCV in both trained and detrained athletes is in contrast with other studies that showed no changes or a decreased in MCV [19]. The possible cause of these divergent results is the difference in levels of air pollution and the exposure time to the polluted air [21]. However, the likely explanation for the increase in mean corpuscular is the enduring destruction of red blood cells by pollution. This process affects old red blood cells the most since old red blood cells are smaller than younger erythrocytes and they are thus more vulnerable. Whatever the reason, the increase in MCV when exercising in polluted air is consistent with previous studies [24].

This study also showed an increase in WBC in both trained and detrained athletes after exercising in polluted air, which is consistent with a study on military firefighters [26]. The leukocytosis is probably caused by tissue destruction, increased lung infection, and increased production of antibodies following increased exposure to air pollutants [19, 20, 25]. We also observed increased platelets numbers in

both trained and detrained athletes after exercising in polluted air, which is consistent with most previous studies [3, 27]. The current study showed that there was a significant drop in mileage rate when exercising in polluted air in both groups, a result that has been found previously [6, 8]. The reason for this inferior performance is because of the deterioration in oxygen distribution and lung function that occurs during exercise in polluted air [6]. Carbon monoxide (CO) is a poisonous gas which has no color or odor, and it can lead to hypoxia via multiple mechanisms. Varying physiological qualities like lung capacity, volume of dead space, and diffusion coefficient of an individual's lung along with his or her breathing rate, are all believe to play a role in the variance of CO intake. The presence of CO causes less oxygen to be transferred from hemoglobin to myoglobin, which in turn forces the heart to beat more frequently in order to compensate for the lack of oxygenation. As a result of the lowering of maximum cardiac output and maximal arteriovenous oxygen difference, both the maximum oxygen uptake (VO_{2max}) and total work output also see a marked decrease [28]. Predicting the likelihood and extent of risk of CO poisoning for athletes who train in areas with high concentrations of air pollutants is a difficult exercise due to the fact that the concentration of CO in any given area is dependent upon its movement based on weather patterns such as temperature and prevailing winds.

The clinical findings of this study are limited by the small number of participants. As a result, the findings of this study may lack generalizability. The measurement of pollution exposure in this study was only relied on the report from the Isfahan Meteorological Bureau in the morning of the test. A better measurement of the PSI proximate to the testing location may provide more accurate results. Furthermore, there may be some other potential confounding environmental variables that may affect the outcome of this study. Future studies should include a larger sample size from various populations, and to be conducted in other months of the year because of the PSI is higher due to the temperature inversion and increased use of personal vehicles in Iran. In conclusion, physiological and blood parameters in both trained and detrained subjects were impacted when undertaking exercise in the polluted air condition. These physiological and hematological changes can adversely affect physical performance. However, the magnitude of damage done by polluted air to the cardiovascular and respiratory systems and other organs and the threat to the health of people, especially athletes, remain unknown. Nevertheless, athletes who train in polluted areas should be made aware of the destructive effects of these pollutants.

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Conflict of interest None of the authors of this paper had any personal or financial conflicts of interest.

Ethical Approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

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