HEMODYNAMIC PHYSIOLOGICAL RESPONSE TO ACUTE EXPOSURE TO AIR POLLUTION IN YOUNG ADULTS ACCORDING TO THE FITNESS LEVEL

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Abstract

BACKGROUND: The purpose of this study was to determine the impact of acute exposure to air pollution on the hemodynamic parameters and physical fitness components in two groups of healthy men differing in fitness (trained and untrained) and the correlation of parameters between the areas.

METHODS: Thirty four healthy college student males (18 low-fitness, mean age 20.44±2.43 years and 16 high-fitness, age 22.19 ± 2.07 years) who were students of the Isfahan University participated in this study. First, two environments including with high and moderate concentrations of ambient air pollution were determined on the basis of the environmental protection agency. Then, all participants performed a Canadian Aerobic Fitness test (CAFT) to determine maximal oxygen uptake (VO2max) in sport sciences laboratory. Each participant also performed 2 sub-maximal exercise tests in two environments including polluted. The tests consisted of three phases: phase A, in non-polluted air area (laboratory), and phase B, very polluted air area, C, moderate polluted air area. All 3 exercise tests were completed within a 1-week period interval between phases. Maximal oxygen uptake (VO2max), maximal heart rate (MHR), systolic blood pressure (SBP), diastolic blood pressure (DBP), and other anthropometric values were measured at end sub-maximal exercise test. Data were analyzed using one-way analysis of variance (ANOVA) with repeated measures and correlation.

RESULTS: At baseline, there were no significant differences between the groups in age, height, weight, DBP, but body mass index(BMI), body fat, resting heart rate(RHR) and SBP was significantly lower in subjects with high fitness ($F_{1,32} = 10.96$, $P < 0.002$, $F_{1,32} = 13.91$, $P < 0.001$, $F_{1,32} = 21.29$, $P < 0.001$, $F_{1,32} = 13.72$, $P < 0.001$, respectively). Although, baseline MHR and VO2max were higher in subjects with high-fitness than in students with low-fitness ($F_{1,32} = 10.07$, $P < 0.01$, $F_{1,32} = 74.23$, $P<0.001$, respectively). For both low-fitness and high-fitness subjects the mean physiological and hemodynamic measurements at baseline and after exercise were significantly associated with concentrations of ambient air pollution category ($P<0.05$).

CONCLUSION: Although statistical significance was found for a number of hemodynamic parameters and physical fitness components in trained and untrained subjects, we speculate that the very small differences in the physiological responses to exercising in urban regions which are often in contact with air pollution ,are of little practical significance and would not affect the performance.

Keywords: Air pollution, VO2max, Systolic and Diastolic blood pressure.

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Introduction

The health effects of air pollution are increasingly recognized as a major public health concern. Air pollution in all of its forms - including sulfur dioxide, ozone, fine particles, carbon monoxide, and nitrogen oxides – is a serious threat for human health (1). It is

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well documented that the physiological deposition and absorption of pollutants have detrimental effects on health (2). Clinicians need to be aware of the health hazards of air pollution for the general population and particularly for susceptible individuals.

Air pollution is the biggest environmental problem that Iran currently faces especially in Tehran and Isfahan as the two cities with highest levels of air pollution. About 1.5 million tons of pollutants are produced in Tehran annually, and carbon monoxide from car exhaust making up a large percentage of these pollutants (1, 2)

Regular physical activity plays a significant role in the human well-being at all life stages, spanning from childhood to the senior years. It has favorable impact on lipid profile, increases insulin receptor density , increases fibrinolytic activityand reduces blood pressure (3-5).

A well-designed exercise program may enhance the immediate physical, psychomotor and intellectual attainments ,even in young ages (6, 7). Physical inactivity is associated with increased morbidity and premature mortality (3-5, 8). A study estimated that 21,000 premature deaths and 2.5% of the total direct health costs in Canada ($2.1 billion)were attributable to physical inactivity (8).

Several studies have shown an inverse relation between fitness level and the incidence of coronary heart disease (CHD) and the number of its risk factors (3, 4, 5, 9).

Cardiorespiratory fitness refers primarily to the capacity of heart and lungs to deliver oxygen to skeletal muscles, and maximal aerobic power is an indicator of the maximal capacity of oxygen delivery. Individuals with a high maximal aerobic power can undertake demanding physical task without suffering fatigue (10). In the present study, maximal oxygen uptake was estimated using a Canadian Aerobic Fitness test (CAFT) and is expressed as estimated aerobic fitness.

Many physical activity and exercise programs involved training in outdoor areas, which in urban environments may be near roadways, in close proximity to motor vehicles. Automobile emissions are estimated to be the greatest single contributor to urban air pollution, and their toxic constituents contribute to several diseases as CHD, respiratory diseases and cancers (11).

Air pollution may affect athletic performance (11). During aerobic exercise, even at relatively low intensities, inspired air is taken in predominantly through the mouth, and there is a major increase in minute ventilation and diffusion capacity. These factors augment the respiratory uptake of airborne contaminants, with increased penetration to the lower gas exchange regions of the lungs. The total amount of ultra fine particulate matter deposited in the respiratory tract of humans during moderate exercise is about five times than at rest (12).

A study showed higher values in agility, leg muscle power and handgrip strength in footballers of less air pollutant zones than higher air pollutant zones (13). Although people should not be deterred from regular exercise and its established benefits, but they should be informed that during exercising they should avoid high-polluted areas (14).

A large body of scientific evidences demonstrated that particulate air pollution, even at the levels experienced in major urban centers, adversely affects the health of children and adults (15-18).

Such adverse effects include an increase in cardiovascular and respiratory deaths, reduced lung function, acute and chronic bronchitis, asthma attacks, emergency room visits, increased hospitalizations for respiratory and cardiac causes, elevated mortality rates, and increased incidence and duration of respiratory symptoms among children and elderly people as well as athletes (19).

Although the mechanisms of relationship between exercise and adverse impacts arising from breathing polluted air, remains to be determined. Numerous studies have shown increased health hazards with increased activity levels, notably in individuals with underlying health conditions (19-21).

A chamber study using a controlled indoor environment with varying levels of air pollutants showed that increasing the intensity of exercise while exposed to ozone adversely impacts lung function and increases reporting of symptoms (20). Another chamber study showed that exposure to ozone during exercise had significantly greater adverse impact on the lung function of men with chronic obstructive lung disease than healthy individuals (21).

A review of British studies among athletes concluded that outdoor exposure to carbon monoxide (CO) such as occurring near traffic, is detrimental to athletic performance,and that ozone might adversely impact lung function and athletic performance. Furthermore, it revaled that among those athletes with asthma, even low levels of SO2 may result in wheezing, chest tightness and increased airway resistance. Asthmatics are generally ten times more sensitive to SO2 than non-asthmatics, especially when exercising (22). There is no evidence-based guideline about the ways to reduce the adverse impact of exercise in polluted outdoor environments. However, some notes such as intermittent exercise with rest periods, prolonged exercise at lower intensity, and/ or shifting exercise to indoor environments have been suggested accordingly. A study on pulmonary function response to equivalent doses of ozone consequent to intermittent and continuous exercise did not document significant difference in lung function due to ozone between continuous and intermittent exercise, however by considering resting periods during exercise, some subjective symptoms had reduced (23).

Because of limited and controversial findings in this field, the current study aimed to determine the impact of acute exposure to air pollution on the hemodynamic physiological parameters in two groups of healthy men differing in fitness. The main objectives of this study were to investigate short-term effects of air pollution on physiological function, to find physiological function parameters that are both sensitive to the acute effects of air pollution and suitable for screening of groups of healthy men according to their fitness level, and to study these associations in an environmental setting typical for the current air pollution situation in many cities in Iran.

Materials and Methods

Study population: The study population comprised low-fitness and high-fitness male college student aged 18 to 26 years from the Isfahan University.

Participants: Thirty four healthy college student males (18 low-fitness, mean age 20.44 ± 2.43 years and 16 high-fitness, age 22.19 ± 2.07 years) who were studying in the Isfahan University, participated in this study in January 2009. All participants were informed about the possible risks associated with participating in this study and gave informed consent prior to participation. Based on self-reported information gathered from a screening questionnaire, none of the participants had a history of smoking, and none were currently taking any medications. All subjects were free from the signs and symptoms of any overt chronic disease. The high-fitness or trained students in the study had minimum three years training, with experience in competitions and they were undergoing regular practice and training. But the low- fitness or untrained students did neither regular practice and training nor any regular physical activity program. All subjects from both the regions of the city included in this study, and were from the same socioeconomic and nutritional status.

Selection of Place: The air of Isfahan (in the center of Iran) is predominantly affected by industrial emissions, particularly steel plant and motor traffic because of a high proportion of diesel trucks and cars, leading to elevated concentrations during stagnant conditions. Details of the air monitoring project have been described before (2). Two zones of the city, namely Darvazeh Shiraz and Takhti squares were selected because the air quality of these two zones were monitored through the environmental protection agency for the parameters suspended particulate matter (SPM), respirable particulate matter (RPM), sulphur dioxide (SO2), and oxides of nitrogen such as nitrogen dioxide (NO2) (2).

Physical and physiological measurements: Before beginning the testing, participants filled in a questionnaire on readiness for physical activity (9). First, baseline anthropometric and physiological measurements including weight, height, body mass index (BMI), Fat mass (%) resting heart rate (RHR), maximal heart rate (MHR), systolic blood pressure (SBP) and diastolic blood pressures (DBP) were registered and then the Canadian Aerobic Fitness test (CAFT) was administered between 9 to 12 am for determine of maximal oxygen uptake (VO2max). Weight was determined on a balance scale in light clothing to an accuracy of 100 g. Height was measured without shoes by a non-elastic measuring tape against a wall to an accuracy of 0.1 cm. BMI was calculated by Meltzer's equation (24).

Exercise Protocol: The participants underwent a sub-maximal-exercise fitness test, called the Canadian Aerobic Fitness test (CAFT) that estimates maximal oxygen uptake (VO2max). The test is based on oxygen cost of the last exercise stage (VO2), age, sex, height and weight (25).

All participants then performed CAFT to determine peak oxygen consumption (VO2peak). CAFT consists of a series of stepping sequences of progressively increasing intensity done on double steps 20.3 cm in height to a six- count recorded musical rhythm. The test begins with a 3-minute warm-up stage (stage A) at an intensity corresponding to 65% - 80% of the average aerobic power excepted for subject. If a predetermined post exercise heart rate is not exceeded and no adverse sign or symptoms are noted, the subject performs a second 3-minute stage (stage B) at an intensity corresponding to 65% - 80% of the average aerobic power excepted for his or her age group. In the absence of adverse sign or symptoms, and provided that the ceiling heart rate is not surpassed, a final 3-minute stage (stage C) is performed at an intensity corresponding to 65% - 80% of the average aerobic power excepted for subject (25).

In this study, we measured the blood pressure (BP) immediately after recording the maximal heart rate at 10 seconds after exercise (i.e., between 15 and 45 seconds after exercise). Moreover, we predicted the VO2max (in milliliter per kilogram per minute) from the performance on the CAFT using the following formula: VO2max = 42.5 + 16.6 (VO2) – 0.12(W)

– 0.12(H) – 0.24(A), where VO2 is the average oxygen cost of the last exercise stage completed, W is body weight (in kilograms), H is the post-exercise heart rate (in beats per minute) and A is the age (in years) (9, 25). On the basis of participants' norms (25), subjects with VO2max at or below the 30th percentile for their age group were classified as being in the low- fitness category, from 31st to the 69th percentile in the moderate-fitness category and at or above the 70th percentile in the high-fitness category.

Each participant also performed 3 CAFT in three environments including polluted and non-polluted. The tests consisted of three phases: phase A, in non-polluted air area (laboratory), and phase B, very polluted air area, C, moderate polluted air area. All three exercise tests were completed within a 1-week period interval between phases. Participants were instructed to abstain from vigorous exercise for 24 hours prior to testing; from consuming diuretic agents including caffeine; and from eating for at least 24 hours prior to testing to avoid possible acute effects on exercise capacity, blood pressure, and heart rate.

Data analysis and statistical methods: All variables were first tested for normality. Values are given as means ± SD. Kolmogorov-Smirnov (normality) test was done for quantifying the discrepancy between the distribution of data and an ideal Gaussian distribution.

Repeated-measures ANOVA was used for comparison of data within the groups. Statistical analysis was performed using the Statistical Package for Social Sciences (SPSS), version17 for Macintosh. Statistical significance was set at P<0.05.

Results

Demographic and physiological information which showed mean age, height, weight, BMI, body fat, RHR, MHR, VO2max, SBP and DBP for all categories with low-fitness and high-fitness ,are presented in Table 1. Comparison of low and high-fitness groups showed no significant difference in age, height and weight, but BMI and body fat were significantly lower in subjects with high fitness (F 1,32= 10.96, P <0.002, F 1,32=13.91, P <0.001, respectively) (Table 1).

With respect to baseline physiological measures (Table 1), baseline RHR and systolic BP were lower in subjects with high-fitness than in subjects with low-fitness (F 1,32= 21.29, P <0.001, F 1,32=13.72, P <0.001, respectively), but basal MHR and VO2max were higher in students with high-fitness than in students with low-fitness (F 1,32= 10.07, P <0.01, F 1,32=74.23, P <0.001, respectively),

There were statistically significant differences in all physiological parameters including VO2max, systolic and diastolic (SBP and DBP) except MHR in low-fitness subjects with respect to the polluted environments and in a relatively clean environment after CAFT (F 2,34=14.72, P <0.001, F 2,34=689.99, P < 0.001, F 2,34=14.72, P <0.001, F 2,34= 3.23, P > 0.05 respectively) (Table 2).

Statistically significant differences were documented in all physiological parameters include MHR, VO2max, SBP and DBP in high-fitness subjects with respect to the polluted environments and in a relatively clean environment after CAFT (F 2,30= 15.21, P < 0.001, F 2,30=3.53, P < 0.04, (F 2,30= 4.65, P < 0.001, F 2,30=102.91, P < 0.001, respectively) (Figure 1).

Table 1. Baseline physical and physiological characteristics of the subjects

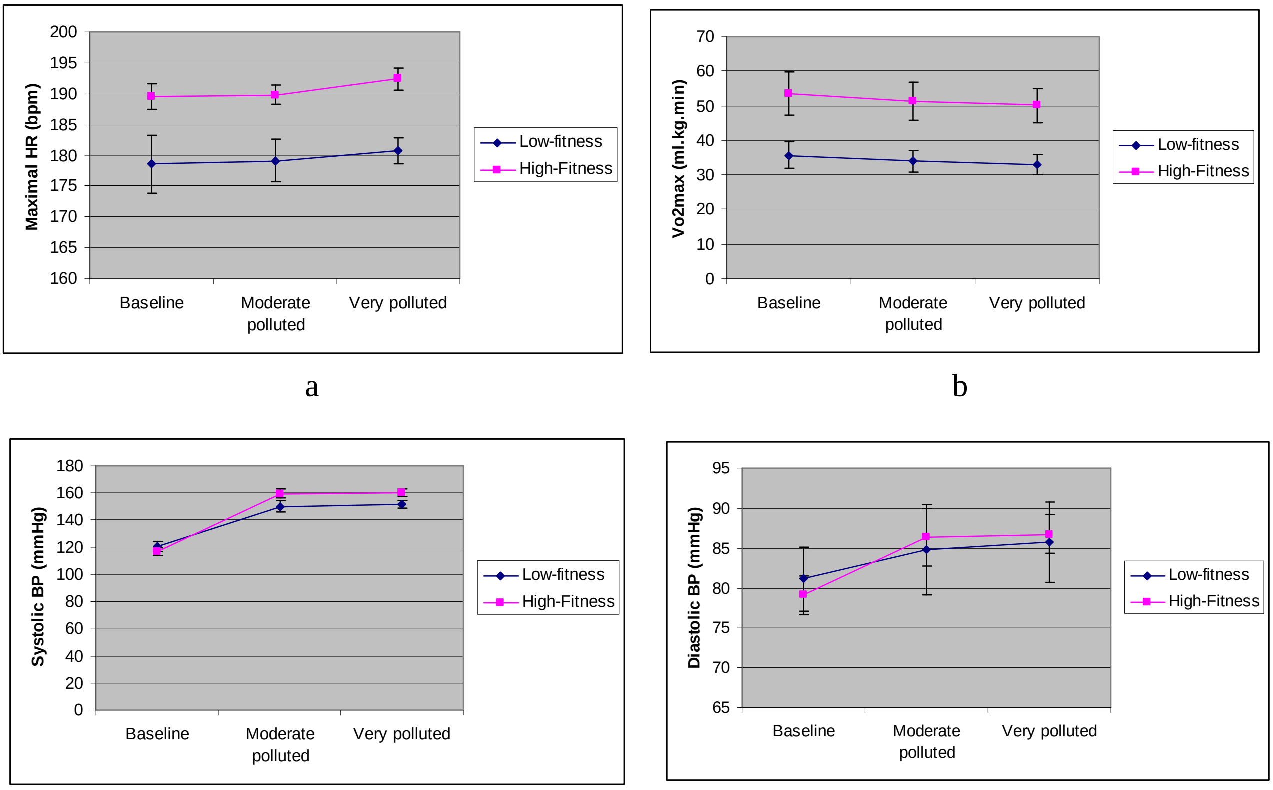
Variable	Low-fitness	High-fitness
No. of subjects	18	16
Age (yr)	21.17±2.57	22.19±2.07
Height (cm)	171.83±7.44	174.69±8.04
Body mass (kg)	72.50±7.01	70.94±7.66
Fat mass (%)***	17.41±2.29	14.31±2.55
Body mass index (Kg/m^2) **	24.53±1.42	23.18±0.86
Resting heart rate (bpm) ***	74.72±3.46	70.25±1.84
Maximal heart rate (bpm) **	178.56±4.68	189.56±2.16
VO2max (mL kg-1 min-1) ***	35.68±3.82	53.62±6.16
Systolic BP (mmHg)***	121.00±3.69	116.81±3.10
Diastolic BP (mmHg)	81.11±4.06	79.12±2.45

Values are means±SD. VO2max, maximal oxygen uptake, ** P <0.01, *** P <0.001

Table 2. Physiological measures of subjects of low-fitness and high-fitness in a polluted environment and in a relatively clean environment after CAFT

Variable	**Low-fitness**			**High-fitness**		
	Baseline	Very polluted	Moderate polluted	Baseline	Very polluted	Moderate polluted
MHR (bpm)	178.56±4.68	180.74±2.11	179.11±3.50	189.56±2.16***	192.38±1.86	189.81±1.52
VO2max (mL kg-1 min-1)	35.68±3.82***	33.03±2.99	33.98±3.18	53.62±6.16*	50.11±5.02	51.41±5.54
SBP (mmHg)	121.00±3.69***	151.56±2.77	150.22±3.96	116.81±3.10***	160.31±3.18	159.50±3.41
DBP (mmHg)	81.11±4.06 ***	85.78±5.02	84.77±5.65	79.12±2.45***	86.75±2.50	86.38±3.58

MHR, maximal heart rate; VO2max, maximal oxygen uptake; SBP, systolic blood pressure; DBP, diastolic blood pressures; exercise. * P >0.05, ***P >0.001, *vs.* baseline training. Values are means±SD.



a b

c d

Figure 1 (a-d). Comparison of some characteristics at baseline in environments with high and moderate concentrations of ambient air pollution according to the fitness level of participants

Discussion

This study examined short-term effects of urban air pollution in morning hours on hemodynamic physiologic responses in two groups of healthy young individuals differing in fitness.

In fact, most of the short-term effects of air pollution on health have been obtained for individuals with previous respiratory or cardiovascular diseases. However, exercise is a condition that increases the dose of inhaled pollution, thus people exercising in environments pervaded by air contaminants are probably at increased risk, due to an exercise-induced amplification in respiratory uptake, lung deposition and toxicity of inhaled pollutants (26).

The importance of our results resides in the fact that even a very short-term period of exposure to environmental air pollutants exposure (about of 1 h) decreases the cardiorespiratory performance and increases selected physiological parameters of two groups of healthy young adults with differing in fitness, as indicated by VO2max (Tables 2).

The results showed that the sub-maximal physiological response to brief acute exposure to air pollutants appeared to be more dramatically worsened than the maximal response, demonstrating that air pollutants can represent an environmental stressor inducing deleterious effects when cardiorespiratory performance is analyzed for the same workload (i.e., at the sub-maximal level).

As a results of the physiological changes that occur during sub-maximal exercise, it has been postulated that endurance athletes may have greater than average susceptibility and exposure to air pollutants. Three reasons why athletes are at special risk of inhaling pollutants have been put forward by McCafferty. Firstly, there is a proportionate increase in the quantity of pollutants inhaled with increases minute ventilation (VE) during exercise. Secondly, a large fraction of air is inhaled through the mouth during exercise. Thirdly, the increased air flow velocity carries pollutants deeper into the respiratory tract.

When examining demographics for two groups, significant differences not found between the groups of healthy men differing in fitness in age, height, weight, but BMI and body fat was significantly lower in subjects with high fitness (F 1,32= 10.96, P <0.002, F 1,32=13.91, P <0.001, respectively). With respect to baseline physiological measures, baseline RHR and systolic BP are lower in subjects with high-fitness than in subjects with low-fitness (F 1,32= 21.29, P <0.001, F 1,32=13.72, P <0.001, respectively), but Basal MHR and VO2max are higher in subjects with high-fitness than in subjects with low-fitness (F 1,32= 10.07, P <0.01, F 1,32=74.23, P <0.001, respectively), although groups did not differ in diastolic BP (F 1,32=2.89, P >0.05).

The lower body fat, basal HR, systolic BP and diastolic BP in males with high-fitness than in males with low-fitness together with the significantly higher MHR and vo2max in the CAFT confirm that both groups are very different, as the former has better physically fitness.

There were statistically significant differences in all physiological parameters including VO2max, SBP and DBP, except MHR in low-fitness subjects with respect to the polluted environments and in a relatively clean environment after CAFT (F 2,34=14.72, P <0.001, F 2,34=689.99, P <0.001, F 2,34=14.72, P <0.001, F 2,34= 3.23, P >0.05 respectively) (Table 2).

We also revealed that there was statistically significant differences in all physiological parameters include MHR, VO2max, SBP and DBP in high-fitness subjects with respect to the polluted environments and in a relatively clean environment after CAFT (F 2,30= 15.21, P <0.001, F 2,30=3.53, P <0.04, (F 2,30= 4.65, P <0.001, F 2,30=102.91, P <0.001, respectively) (Table 2).

Our results demonstrated that exercising in urban regions which are often in contact with air pollution ,increased the MHR, SBP and DBP at exercise in two groups of healthy men differing in fitness. Whereas, after of acute exercise training in contact with air pollution,the VO2max was decreased in two groups of healthy men differing in fitness.

Carbon monoxide (CO) is the most significant primary air pollutant, altering the ability of hemoglobin to carry oxygen (27). Hemoglobin also combines with CO to form carboxyhemoglobin, and has 230 times greater affinity for CO than for O2, which indicates that atmospheric air pollutants (especially, CO) have a great potential to alter oxygen transport in blood (28).

Several studies have reported that strenuous exercise in heavy air pollution and traffic for 30 minutes can increase the level of COHb 10-fold, which is the equivalents of smoking 10 cigarettes. There is no doubt than Co is detrimental to athletes performance and there is much evidence of this. With CO in bloodstream, less O2 is released from haemoglobin to myoglobin, and therefore, to compensate, the heart must work harder and beat more frequently. Maximum cardiac output and maximal arteriovenouse difference are lowered, resulting in a decrease in maximum oxygen uptake (Vo2max) and work output. The formation COHb is reversible, and exposure to clean air removes most of the gas from the body, with a half life of three to four hours (28-30). The effects of raised COHb on performance have indicated a significantly lower VO2max, anaerobic threshold, and oxygen pulse (Vo2/heart rate), and a significantly higher heart rate and pulse pressure.

In view of other evidences on air pollution and blood pressure, probably the null findings seem more likely. In fact, some studies conducted among similar populations, showed an increase of SBP in association with an increase of high concentrations of ambient air pollution (29, 30).

In addition to the inflammatory mechanism hypothesis, an increase of the sympathetic tonus in association with particulate air pollution and/or an increase of the vascular tonus following an increase of the endotheline were proposed as potential mechanisms that can explain this increase of the SBP (29).

Recent studies have confirmed a relationship between blood pressure and exposure to air pollution. Population-based studies have shown increases in both systolic blood pressure and pulse pressure [29, 31] with increasing levels of ambient pollution exposure.

Controlled exposure studies to concentrated ambient particles and ozone have demonstrated an increase in diastolic blood pressure during a two-hour exposure [32].

Also, we observed a marked difference in systolic blood pressure with exercise. In both groups, blood pressure increased during exercise compared with the baseline average, although this increase was less during the exposure to air pollution in two environments.

Exercise induced increases in systolic blood pressure have been linked to myocardial infarction [33] as well as stroke [34], and increased blood pressure is an established major risk factor for the development of both atherosclerosis and cardiovascular mortality [34, 35].

Therefore we predict that the use of a facemask in a susceptible population has the potential to reduce the incidence of acute cardiovascular events as well as myocardial ischemia [36, 37].

The present study had several important limitations. First, the most significant limitation of this study is the unavailability of direct measurements of air quality in Isfahan.

Second, the subjects were untrained and trained healthy young adults, and the number of subjects was small. Therefore, further studies will be needed to generalize our findings. Third, this study did not employ blood sampling. Thus, we could not measure metabolic risk factors for cardiovascular disease before and after the exercise training program.

A further limitation of our study is its cross-sectional design and the unavailable repeated measures in time for each subject. Blood pressure is an extremely variable parameter and depends on various factors such as medication, physical activity, age, sex, and meteorology, for which we tried to adequately control in this analysis, but we still must be careful in interpretation of these results.

With respect to the effects of particulate air pollution on the SBP, it would not be appropriate to draw any conclusions regarding the clinical relevance of these findings, because there is not enough epidemiologic evidence. Further research applying other methods, such as time series studies, is needed to investigate the effects of ambient particulate matter on cardiovascular health and to establish further evidence on how particles affect the control of blood pressure and how these effects contribute to adverse health outcomes.

Based on our analysis of air pollution levels in Isfahan, and review of exposure assessment and health effects studies by other investigators, we offer the following guidance to Isfahan residents for minimizing exposure to air pollutants while maximizing physical activity:

On Smog Alert Days and When the AQI is 50 or Higher:

1- Moderate physical activity outdoors on smog alert and poor air quality days, even if you are healthy. Ways of moderating activity are to shift from vigorous activity levels to moderate or light activity levels, reducing the duration of activity and introducing more rest breaks. Drink plenty of water before, during and after exercise.

2- For all people, but especially those with heart or breathing problems (including asthma), it is important they monitor any symptoms they experience with different activity levels and as the air quality index (AQI) increases. Examples of symptoms to look for include coughing, wheezing, chest tightness, pain with breathing deeply, and difficulty breathing. Anyone experiencing symptoms should reduce their level of activity.

3- On smog alert days, consider exercising indoors in a smoke-free environment, and if available, one that is air-conditioned

On Non-Alert Days (When the AQI is Less than 50):

1- Continue to monitor any symptoms experienced with different activity levels and as the air quality index (AQI) increases. Reduce activity level outdoors when AQI values are above those known to trigger each individual's symptoms.

2- If possible, schedule routine vigorous exercise, such as running and jogging, for early in the morning (before 7 a.m.) and in low traffic areas (such as residential neighborhoods and parks) .Most importantly, however, it is essential that persons involved in health promotion emphasize the health benefit of physical activity in ensuring wellbeing, including the prevention of disease. Routine physical activity on a year round basis is to be encouraged, while alerting the public to ways of moderating vigorous physical activity outdoors on those days when smog alerts occur. While there is a need to protect that segment of the population already engaged in routine vigorous activity from air pollution during smog alerts, it is critical to recognize the health benefit of more modest measures to increase physical activity in those persons who tend to be sedentary. There is a need to increase awareness that introducing more light activity (such as with slow pace walking, easy gardening, stretching) and moderate activity (such as with brisk walking, bicycling, raking leaves) to persons who are generally inactive is very beneficial to improving their health. To date, there appears to be no evidence that light or moderate physical activity during smog alert conditions poses a health risk to people without other underlying medical conditions. However, there is some evidence in the literature that prolonged vigorous activity when air quality is poor can pose a health risk, especially to those persons with pre-existing respiratory conditions. More research is required in this area. In the long term, it is important to ensure that barriers to physical activity, such as limited physical education opportunities in schools, access to recreational or sports facilities, and availability of green space are addressed in a comprehensive way to achieve public health goals. Accelerated action on improving air quality is of paramount important for many reasons, one of them being to ensure that poor air quality does not become another barrier for enhancement of physical activity.

However, due to the dissimilarities among studies carried out in different places with specific air pollution characteristics and different populations, further investigations are required on a research topic that is far from concluded.

Conclusion

Our results demonstrated that exercising in urban regions are often in contact with air pollution, increased the MHR, SBP and DBP at exercise in two groups of healthy men differing in fitness. Whereas, after acute exercise training in contact with air pollution the VO2max was decreased in two groups of healthy men differing in fitness. The results of the survey show that significantly more information and education is required in people about implications of exercising in polluted environments not only through coaches and sports associations but also through physicians, clinicians and other health professionals.

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References

1. Khalilzadeh S, Khalilzadeh Z, Emami H, Masjedi MR. Air pollution status and cardiorespiratory admissions in Tehran. National Research Institute of Tuberculosis and Lung Disease, Iran 2009; 8(1): 35-40.
2. Kargarfard M, Rouzbahani R, Rezanejad S, Poursafa P. The effect of air pollution on cardio respiratory performance of active individuals. ARYA Atherosclerosis 2009; 5(2): 69-75.
3. Rodrigues AN, Perez AJ, Carletti L, Bissoli NS, Abreu GR. The association between cardiorespiratory fitness and cardiovascular risk in adolescents. J Pediatr (Rio J) 2007; 83(5): 429-35.
4. Koley S. Association of cardio respiratory fitness, body composition and blood pressure in collegiate population of amritsar, Punjab, India. The Internet Journal of Biological Anthropology 2007; 1(1).
5. Physical Activity Fundamental To Preventing Disease.[2002 June 20]. Available from URL: http://aspe.hhs.gov/health/reports/physicalactivity.
6. Shanmugam C, Maffulli N. Sports injuries in children. Br Med Bull 2008; 86: 33-57.
7. Adirim TA, Cheng TL. Overview of injuries in the young athlete. Sports Med 2003; 33(1): 75-81.
8. Katzmarzyk PT, Gledhill N, Shephard RJ. The economic burden of physical inactivity in Canada. CMAJ 2000; 163(11): 1435-40.
9. Jette M, Sidney K, Quenneville J, Landry F. Relation between cardiorespiratory fitness and selected risk factors for coronary heart disease in a population of Canadian men and women. CMAJ 1992; 146(8): 1353-60.
10. Bouchard C, Shephard RJ. Physical activity, fitness, and health: the model and key concepts. in: Bouchard C, Shephard RJ, editors. Physical activity, fitness, and health: international proceedings and consensus statement. Illinois: Human Kinetics Publishers; 1994: 77-88.
11. Shephard RJ. Athletic performance and urban air pollution. Can Med Assoc J 1984; 131(2): 105-9.
12. Daigle CC, Chalupa DC, Gibb FR, Morrow PE, Oberdorster G, Utell MJ, et al. Ultrafine particle deposition in humans during rest and exercise. Inhal Toxicol 2003; 15(6): 539-52.
13. Das P, Debnath P, Chatterjee P, Chatterjee P. Study of selected motor ability variables of school going boys in two different pollutant zones. Biomedicine 2005; 25(1): 21-6.
14. Sharman JE. Clinicians prescribing exercise: is air pollution a hazard? MJA 2005; 182(12): 606-7.
15. Burnett RT, Cakmak S, Brook JR. The effect of the urban ambient air pollution mix on daily mortality rates in 11 Canadian cities. Can J Public Health 1998; 89(3): 152-6.
16. Burnett RT, Smith-Doiron M, Stieb D, Cakmak S, Brook JR. Effects of particulate and gaseous air pollution on cardiorespiratory hospitalizations. Arch Environ Health 1999; 54(2): 130-9.
17. Stieb DM, Judek S, Burnett RT .Meta-analysis of time-series studies of air pollution and mortality: effects of gases and particles and the influence of cause of death, age, and season. J Air Waste Manag Assoc 2002; 52(4): 470-84.
18. Vedal S, Brauer M, White R, Petkau J. Air pollution and daily mortality in a city with low levels of pollution. Environ Health Perspect 2003; 111(1): 45-52.
19. American Lung Association. annotated bibliography of recent studies on the health effects of Air Pollution.[Online]. 2001. Available from URL: http://www.sbcapcd.org/sbc/o3biblio.pdf
20. Adams WC. Ozone dose-response effects of varied

equivalent minute ventilation rates. J Expo Anal Environ Epidemiol 2000; 10(3): 217-26.

21. Gong H, Jr., Shamoo DA, Anderson KR, Linn WS . Responses of older men with and without chronic obstructive pulmonary disease to prolonged ozone exposure. Arch Environ Health 1997; 52(1): 18-25.

22. Carlisle AJ, Sharp NC. Exercise and outdoor ambient air pollution. Br J Sports Med 2001; 35(4): 214-22.

23. McKittrick T, Adams WC. Pulmonary function response to equivalent doses of ozone consequent to intermittent and continuous exercise. Arch Environ Health 1995; 50(2): 153-8.

24. Meltzer AA, Mueller WH, Annegers JF, Grimes B, Albright DL. Weight history and hypertension. J Clin Epidemiol 1988; 41(9): 867-74.

25. Nieman DC. Fitness and sports medicine: an introduction. 2nd ed. New York: Bull Pub. Co; 1990.

26. Folinsbee LJ. Effects of air pollutants on exercise. In: Garrett WE, Kirkendall DT, editors. Exercise and sport science. Philadelphia: Lippincott Williams & Wilkins; 2000: 285-97.

27. Hlastala MP, McKenna HP, Franada RL, Detter JC. Influence of carbon monoxide on hemoglobin-oxygen binding. J Appl Physiol 1976; 41(6): 893-9.

28. Armstrong LE. Performing in extreme environments. 1st ed. Champaign: Human Kinetics; 2000.

29. Ibald-Mulli A, Stieber J, Wichmann HE, Koenig W, Peters A. Effects of air pollution on blood pressure: a population-based approach. Am J Public Health 2001; 91(4): 571-7.

30. Blood pressure and heart rate associated with PM2.5 in a cardiac rehabilitation Proceedings of The 98th American Thoracic Society Annual Conference; 2002 May 17–23; Atlanta, GA, USA; 2002.

31. Auchincloss AH, Diez Roux AV, Dvonch JT, Brown PL, Barr RG, Daviglus ML, et al. Associations between recent exposure to ambient fine particulate matter and blood pressure in the Multi-ethnic Study of Atherosclerosis (MESA). Environ Health Perspect 2008; 116(4): 486-91.

32. Urch B, Silverman F, Corey P, Brook JR, Lukic KZ, Rajagopalan S, et al. Acute blood pressure responses in healthy adults during controlled air pollution exposures. Environ Health Perspect 2005; 113(8): 1052-5.

33. Mundal R, Kjeldsen SE, Sandvik L, Erikssen G, Thaulow E, Erikssen J .Exercise blood pressure predicts mortality from myocardial infarction. Hypertension 1996; 27(3 Pt 1): 324-9.

34. Kurl S, Laukkanen JA, Rauramaa R, Lakka TA, Sivenius J, Salonen JT. Systolic blood pressure response to exercise stress test and risk of stroke. Stroke 2001; 32(9): 2036-41.

35. Hippisley-Cox J, Coupland C, Vinogradova Y, Robson J, Minhas R, Sheikh A, et al. Predicting cardiovascular risk in England and Wales: prospective derivation and validation of QRISK2. BMJ 2008; 336(7659): 1475-82.

36. Mills NL, Tornqvist H, Gonzalez MC, Vink E, Robinson SD, Soderberg S, et al. Ischemic and thrombotic effects of dilute diesel-exhaust inhalation in men with coronary heart disease. N Engl J Med 2007; 357(11): 1075-82.

37. Chuang KJ, Coull BA ,Zanobetti A, Suh H, Schwartz J, Stone PH, et al. Particulate air pollution as a risk factor for ST-segment depression in patients with coronary artery disease. Circulation 2008; 118(13): 1314-20.