

THE EFFECT OF AIR POLLUTION ON CARDIO RESPIRATORY PERFORMANCE OF ACTIVE INDIVIDUALS

Mahdi Kargarfard⁽¹⁾, Reza Rouzbahani⁽²⁾, Saber Rezanejad⁽³⁾, Parinaz Poursafa⁽⁴⁾

Abstract

BACKGROUND: Although much has been discussed regarding the improvement of quality of life with regular physical exercise, we need studies on the cardio respiratory response evaluated on the basis of O₂ uptake, transport, and utilization in areas with high concentrations of pollutants in the atmosphere. The purpose of this study was to determine the effect of air pollution on respiratory and cardiac performance of active individuals in the environments with polluted air and non-polluted air.

METHODS: Twenty healthy non-smoker athlete undergraduate male students (Mean \pm SD: age 21.70 \pm 2.10 yr, height 175.80 \pm 6.78 cm, weight 65.58 \pm 4.23 kg and BMI 24.44 \pm 2.32) volunteered to participate in the study. First, two environments including polluted and non-polluted were determined on the basis of the environmental protection agency. Then, the subjects were performed on a field cooper test. The tests consisted of two phases: phase A, in non-polluted air area and phase B, in polluted air area, with a 7-day interval between phases. Finally, respiratory volumes and capacities were measured.

RESULTS: The results of analysis by paired t-test showed that there were significant decreases in all of the respiratory parameters (ERV, IC, FVC, FEV₁, MVV, FEV₂₅₋₇₅, FEV₁/FVC), in polluted air compared with non-polluted air (P < 0.05).

The heart rate measures in two group showed that the mean of heart rate in polluted area was (89 \pm 4) more than non polluted environment (83 \pm 5) and this was significant at P=0.028.

CONCLUSION: Therefore, the acute exposure to polluted air may cause a significant reduction in the respiratory and cardiac performance of active individuals.

Keywords: air pollution, respiratory and cardiac performance, active individuals.

ARYA Atherosclerosis Journal 2009, 5(2): 71-77

Date of submission: 11 Mar 2009, *Date of acceptance:* 18 Jun 2009

Introduction

Regular physical activity has a positive influence on the health of people at all stages of their life. The greater the activity level, the higher the respiratory rate and resultant intake of air.¹ Therefore, mostly in large urban centers and industrial areas, "in the fight for health and longevity", people are forced to exercise in areas with inadequate conditions, extreme temperatures, little ventilation, low impact absorption soil, and, above all, a direct contact with air pollution.² Air pollution is the contamination of our atmosphere. Man-made air pollution sources include: shipping, ports, automobile Emissions, airplane Exhaust, everyday Use of Toxic Chemicals.^{3,4} Man-made pollutants include a mixture of vapors and gases found in both outdoor and indoor environments. The most common gaseous pollutants are: carbon dioxide, car-

bon monoxide, hydrocarbons, nitrogenoxides, sulfur oxides and ozone. Even though air pollution is usually a greater problem in cities, pollutants contaminate air everywhere, even indoors. These contaminating substances include various gases and tiny particles, or particulates that can harm human health. Indoor air pollution is caused by the use of toxic cleaning products, cigarette smoking, the use of certain construction materials, and home furnishings.^{3,4} During the past 20 years, concern has increased about possible problems associated with exercising in polluted air. The air in many cities is contaminated with small quantities of gases and particles not naturally found in the air we breathe. When air becomes stagnant or when a temperature inversion occurs, some of these pollutants reach concentrations that significantly impair athletic performance.^{3,8,9}

1) PhD, Associate Professor of Physical Education and Sport Sciences, University of Isfahan, Isfahan, Iran.

2) MD, MPH, Community Medicine Specialist, Researcher, Isfahan University of Medical Sciences, Isfahan, Iran.

3) MSc in Physical Education and Sport Sciences, Researcher, University of Isfahan, Isfahan, Iran.

4) MSc, Environmental Protection Engineer, Science and Research University, Tehran, Iran.

Corresponding author: Dr Reza Rouzbahani, E-mail: re.rouzbeh@gmail.com

Several studies have reported that quality of the air we breathe affects the quality of our health. Air quality has an impact on the health of our lungs and the entire cardio respiratory system. In addition to oxygen, the air contains other substances such as pollutants, which can be harmful to health.^{3,8,9} Once the pollutants are released into the atmosphere, they are transported by wind, rain and snow, pushing the substances back down to the earth and causing a detrimental environmental cycle between air, land and water contamination.^{3,4} The inhalation of those pollutants may have harmful effects on the lungs and other organs of the body. When pollutants are inhaled, they trigger an increase in "reactive oxygen species" superoxidizing molecules that damage cells, cause inflammation in the lungs, and spark the cascade of harmful effects in the heart and cardiovascular system. Recent research suggests that ultrafine air pollutants, such as those coming from car exhaust, may pass into the blood stream and damage the heart and blood vessels directly. Hearts directly exposed to ultrafine air pollutants show an immediate decrease in both coronary blood flow and the heart's pumping function, as well as a tendency to develop arrhythmias, according to studies conducted at the Heart Institute.^{3,4}

The respiratory system is particularly sensitive to air pollutants because it is made up of a mucous membrane covering its internal surface.^{3,8,9} Ozone can cause damage to the alveoli air sac in the lungs where exchange of oxygen and carbon dioxide is done.^{10,11}

Many of us live in areas which have high levels of air pollution- or must travel to such areas for competitions. What's unfortunate for athletes is that heavy smog can impair their performance. As a result, it's important for coaches to know how to reduce the effects of pollution on our athletes.^{4,12} The amount of air pollutants that people take into their lungs depends on many factors, including their activity level and resultant respiratory rate, their mode of breathing, and the concentration of pollutants at that particular time and microenvironment. Vigorous exercise is known to increase the respiratory rate and change the mode of breathing from through the nose to through the mouth, thereby by-passing the ability of the nose to filter some pollutants.¹ Co, a product of car exhaust, is 230 times more likely to bind with hemoglobin than is oxygen. As a result, high Co concentrations reduce the blood's ability to transport and release oxygen, reducing the volume and intensity of exercise, an athlete can perform. Co is the form of air pollution with the greatest negative effect on athletic performance. Other pollutants such as sulfur dioxide, cigarette smoke, dust and ozone have been poorly studied. Although they're all respiratory irritants and

may reduce airflow in the lungs, it isn't known whether they impair performance in healthy individuals. However, they certainly will affect those who already have respiratory problems.^{4,13}

Athletes are particularly vulnerable to pollutant effects because they inhale 10 to 15 times the air volume as compared with the sedentary. The typical inactive individual inhales about 600 liters of air each hour. During strenuous activity the volume can be as high as 7,000 liters. So, exercise in polluted air vastly increases lung surface contact with airborne pollutants.^{14,15}

Numerous studies have reported associations between increases in ambient air pollution and risk of cardiorespiratory mortality and morbidity, but the underlying mechanisms for these associations are still not clear.^{7,10,12-18} Increased incidence of acute myocardial infarction (MI) has been observed following exposure to as little as 2 hours to several days of elevated particulate air pollution, as well as "time spent in traffic" in several U.S. and European cities. Although relative risks associated with pollution are small compared with those of known clinical risk factors, traffic pollution or proxies for traffic pollution, have been repeatedly implicated as risk factors for acute cardiorespiratory disease.¹⁸ The effects of short-term increases in levels of ambient particulate matter (PM) have been associated with triggering of cardiac arrhythmias, myocardial infarction, heart rate variability, inflammatory response measured by C-reactive protein (CRP), blood viscosity, decompensation of heart failure patients, exacerbation of myocardial ischemia as well as other blood markers (e.g., hemoglobin, fibrinogen, platelet counts, white cell counts).^{16,17,18} These observed effects would provide a mechanism by which chronic exposure to ambient air pollution is associated with risk of coronary heart disease (CHD).

Studies have demonstrated that athletes exercising in elevated ozone, exhibited reduced endurance and lung function. High carbon monoxide levels have a synergistic effect, further decreasing performance.^{19,20} Numerous panel studies investigating short term changes in lung function in children, have been performed. Meta-analyses indicated adverse effects of ozone and particulate matter on lung function. However, most have focused on daily mean levels of air pollution and, if investigating effect on lung function, have concentrated on parameters such as peak expiratory flow (PEF) that are easily collected in children. Fine particles of different origin and chemical composition are supposed to differ in their health impact.²¹ Air pollutants may, in addition to other responses, cause lung cell damage, inflammatory responses, im-

pairment of pulmonary host defenses, and acute changes in lung function and respiratory symptoms as well as chronic changes in lung cells and airways. Acute and chronic exposure to air pollutants is also associated with increased mortality and morbidity.²²

It has been shown that increase in the intensity of exercise while exposed to ozone, adversely impacts lung function and increases reporting of symptoms. A study of adult hikers, found that with prolonged hiking in an outdoor setting, exposure to ozone, PM_{2.5} and acid aerosols was associated with significant impacts on pulmonary function.²³ A review of British studies involving athletes, concluded that outdoor exposure to Co (such as occurs near traffic) is detrimental to athletic performance and that ozone adversely impacts lung function and likely athletic performance.^{1,24} Increasing evidence points to the importance of canceling all games and practices when pollution levels put athletes health at risk. Hopefully, current and future research will provide a better understanding of the limitations imposed by air pollutants.⁵ It is necessary for those who exercise under such polluted conditions to have enough knowledge of their negative effects. In line with these issues, it is also a must for trainers to apply helpful strategies while performing matches, for prevention of putting individuals in risky conditions and to keep them in appropriate atmosphere. Hence it is necessary for athletes and executive agents to delve into influencing issues and direct the condition towards a better one in which no effect of bad negative polluted air can influence individuals. The purpose of this study was to determine Respiratory and Cardiovascular effects of air pollutants on active individuals

Materials and Methods

Study design and Subjects:

Target population included all healthy non-smoker athlete undergraduate male students from the University of Isfahan who were participated in the study. This was a interventional study. Twenty healthy non-smoker athlete undergraduate male students between 20 and 26 years of age with similar socio-economic background volunteered to participate in the study. They were informed of the purpose and methods of this study, as well as its benefits and possible dangers to health before giving written consent to them. Also, the individuals were allowed to withdraw from it at any time. After this orientation, each subject signed an informed term of consent. They were in good mental and physical condition. None of the subjects had history of chronic diseases including cardiorespi-

ratory diseases and endocrine or metabolic diseases. The study was proved by the university physician.

The age of each subject was calculated from the date of birth as recorded in his institute. Weight and height of each subject were measured by using a weighing scale (Seca, Model Germany) fitted with a height measuring stand to the nearest 0.1 cm and 0.05 kg respectively. Body mass index (BMI) were calculated by the following formula: $BMI (Kg/m^2) = (Weight \text{ in Kg}) / (Height \text{ square in Meters})^2$.²⁴ Skin folds and anthropometric measurements (viz., girths and widths) were measured by Holtain Skin fold Caliper with constant tension (Holtain Ltd., UK) and measuring tape with anthropometric rod, respectively, according to the guidelines of Johnson and Nelson (1982). The participants trained 3 times a week throughout the year.

Exercise tests:

First, two environments including polluted and non-polluted were determined on the basis of the environmental protection agency. Then, the subjects were performed on a field cooper test at the same time of day (between 9:00 and 11:00 am), and the time of the test was identical for each subject. The tests consisted of two phases: phase A, in non-polluted air area, and phase B, in polluted air area, with a 7-day interval between phases. Finally, respiratory volumes and capacities and heart rate were measured in field by using spirometry system linked to computer (Jagger-Masterscope Rotary for PC-connection only).²⁶ The two sessions were separated by at least one week. The subjects were asked not to participate in any physical activity in the 24 hours before each session.

Procedure:

For testing, two places with the same climate (altitude, temperature and humidity) were chosen on the basis of information gathered via environmental protection agency of Isfahan, Bakhtiyardasht station as clean environment and Azadi square station as polluted environment.

It is to be mentioned that environment protection agency has used pollutant standard index to determine pollutant and clean environment on the basis of five original pollutants including SO₂, O₃, NO₂, CO and PM₁₀ (Table 1).

According to table 1 significant difference was found between the mean values of air pollutants in two mentioned environment. The subjects carried out the test in two stages, first time in clean environment (Bakhtiyardasht station) and the second time, in the morning, at the same time as stage one, in polluted area (Azadi square station).

Table 1. Characteristics of two environments including polluted and non-polluted were determined on the basis of the environmental protection agency

Environment	Co p.p.m*	O ₃ p.p.b**	PM ₁₀ µg/m ³	No ₂ p.p.b	So ₂ p.p.b	PSI
Non-polluted	2.4	1.6	20	18.3	18.2	50>
Polluted	35.4	10.1	248	45.4	46.9	<200

* Part per million ** Part per billion

Statistical Analysis:

Means (SD) were determined. Paired t-test analyses were undertaken for the statistical treatment of the data. Statistical analysis was performed using the Statistical Package for Social Sciences (SPSS), version 17 for Macintosh. Statistical significance was accepted ($P < 0.05$).

Results

The mean and SD values of the physical parameters of healthy non-smoker active individuals are presented in Table 2. The BMI values showed that the subjects were non-obese, non- overweight and almost thin according to the available classification.^{24,26} Table 1 represents the values of different two environments including polluted and non-polluted measurements in the both environments.

The heart rate measures in two group showed that the mean of heart rate in polluted area was

(89 ± 4) more than non polluted environment (83 ± 5) and this was significant at $P < 0.05$.

Table 2. Physical characteristics of subjects

Variable	Mean \pm SD
Age (yr)	21.70 \pm 2.10
Height (cm)	175.80 \pm 6.7
Body mass (kg)	65.85 \pm 4.23
Body mass index (Kg/m ²)	24.44 \pm 2.32

The results of analysis by paired t-test and significant differences in Table 3 showed that there were significant decreases in all of the respiratory parameters (ERV, IC, FVC, FEV₁, MVV, FEV₂₅₋₇₅, FEV₁/FVC), in polluted air compared with non-polluted air ($P < 0.05$).

Table 3. Respiratory performance variables of athlete undergraduate male students in a polluted environment and in a non - polluted environment

Variables	Non- polluted environment	Polluted environment	Paired t-test		
			Degree of free	T value	Significant
ERV*	1.35 \pm 0.16	1.19 \pm .13	19	4.86	0.001
IC*	3.56 \pm 0.26	3.27 \pm 0.20	19	4.25	0.001
FVC*	5.07 \pm 0.48	4.77 \pm 0.40	19	6.45	0.001
FEV ₁ *	4.53 \pm 0.30	4.16 \pm 0.18	19	6.54	0.001
MVV*	155.58 \pm 9.19	149.40 \pm 5.40	19	3.28	0.004
FEV ₂₅₋₇₅ *	5.33 \pm 0.52	4.91 \pm 0.44	19	4.15	0.001
FEV ₁ /FVC*	90.05 \pm 8.86	87.72 \pm 7.46	19	2.11	0.048

Data are reported as mean \pm SD and Paired t-test results for 20 healthy athlete undergraduate male students who exercised in both environments.

ERV = Expiratory Reserve Volume; IC = Inspiratory Capacity; FVC= Forced Vital Capacity; FEV₁ = Forced Expiratory Volume in First Second; MVV = Maximum Ventilatory Volume; FEV₂₅₋₇₅ = Forced Expiratory Volume₂₅₋₇₅; FEV₁/FVC = percent of Forced Expiratory Volume in First Second on Forced Vital Capacity.

* $P < 0.05$ compared with Non - polluted environment (paired t-test).

Discussion

The purpose of this study was to determine the effect of air pollution on respiratory performance of active individuals in the environments with polluted and non-polluted air. The results of analysis by paired t-test to the respiratory function responses showed that there were significant decreases in all of the respiratory parameters Expiratory reserve volume (ERV), Inspiratory capacity (IC), FEV1 - forced expiratory volume in 1 second, Maximum voluntary ventilation (MVV), Forced vital capacity (FVC) (ERV, IC, FVC, in polluted air compared with non-polluted air ($P < 0.05$) that these results have been seen in past researches.^{5,6,28-34}

Results showed that when polluted air inhalation, especially SO₂, can produce acute bronchial tube constriction, by stimulating the vagus nerve in the pharynx region of the throat, behind the tongue. This bronchospasm results in an increased resistance to the air flow.²⁸ Interestingly, this airway resistance (Raw) increases at rest, as the volume of inspired air increases.³² Two factors are involved: (a) increased ventilation per minute (VE) augments the total volume of SO₂ delivered to sensitive bronchial sites; and (b) increased VE decreases the percentage of air that is inspired through the nose, bypassing an important defense against SO₂, nasal "scrubbing" of soluble gases. Nasal scrubbing is a very effective process that absorbs more than 99% of SO₂ during quiet breathing.³⁵ Both of these factors, therefore, allow a greater volume of SO₂ into the deeper segment of the pulmonary tree and theoretically should increase bronchospasm.²⁸

Because exercise increases VE, the foregoing information logically suggests that Raw will increase if SO₂ is inhaled during labor, sport specific competition, or athletic competition. And, if Raw increases, it is theoretically reasonable that physical performer will suffer, because maximal ventilation will be reduced. These have been reviewed by various authors, who have drawn the conclusions about the effects of SO₂ inhalation on exercise performance in healthy adults that some, individuals show significant decrements in lung function following low levels of SO₂ exposure.^{4,32, 35}

So, anatomically and physiologically, the effects of ozone on the bronchial tubes and lungs apparently result from stimulation of irritant receptors in the pharynx region of the throat, behind the tongue, which in turn causes a hyperirritable state in the vagus nerve and involuntary inhibition of inspiration.^{34,36} This neurochemical response results in rapid, shallow breathing during O₃ and exercise exposure, as well as

a reduction in the amount of ambient air that reaches the alveoli.³⁷⁻⁴⁰ Both of these factors enhance one's sensation of breathlessness during exercise.³⁶ The pulmonary function testes (PFT) that indicate impairment (i.e., reduced capacity) are FVC, FEV1, and Raw. Because these responses may alter the diffusion of O₂ from the alveoli into the capillary bloodstream, it is possible that this is one mechanism by which O₃ reduces exercise performance. Bates, in fact, proposed this mechanism (i.e., reduced alveolar ventilation-perfusion ratio) along with two others: a decreased O₂ saturation in arterial blood, and an increased energy requirement of respiratory muscular effort.⁴⁰ So in human, O₃ inhalation impairs pulmonary function, causes respiratory discomfort, and increases the number of reported clinical symptoms.²⁹ These responses are exacerbated during exercise because (a) the absolute amount of O₃ inhaled increases, (b) the uniformity of ventilation throughout all lung tissue increases, and (c) "nasal scrubbing" (i.e., absorbing gases during quiet breathing through the nose) is compromised.³⁶

Conclusion

In summary, the acute exposure to polluted air, may cause a significant reduction in the respiratory performance of active individuals. Our results 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.

Therefore, Clinicians should not encourage people to exercise in polluted air, since epidemiological studies indicate that the benefits of regular exercise outweigh potential harm.^{17,18,41,42} However, given the evidence linking air pollution to disease, together with the possibility that exercise near road traffic may intensify harmful effects, it is advisable to avoid or minimize exposure to air-borne contaminants. Accordingly, we recommend that physicians and other health professionals advise people undertaking an exercise program to exercise outdoors in parks and recreation areas away from busy roadways or industrial sites. This advice does not take into consideration regional differences in ambient pollutant levels that vary with the time of day. Therefore, in large cities where the ambient atmospheric levels of particulate matter regularly exceed national air safety standards, it may be useful to limit exercise sessions to the hours of the day when air pollution is likely to be less concentrated (i.e. early hours of the morning). Importantly, some populations may be especially sensitive to air pollution (i.e. children, elderly, diabetics or those with

existing heart or lung disease) and care should be given to offer prescriptive advice to these people in particular.⁴¹ Also, the elderly and patients with cardiovascular disease shouldn't exercise outside on days with increased air pollution levels. On much polluted days, they should consider staying inside, and during the winter, they should limit exposure to fireplace smoke. Of course, the real solution is to reduce air pollution.

Acknowledgements

We would like to acknowledge the financial support from the Researcher and Technology Center and Grant Agency of the University of Isfahan.

References

- Campbell M, Li Q, Gingrich S, Howard D, Macfarlane R, Cheng SH. Health benefits of physical activity. In: Basrur SH, Editor. Air pollution and physical activity: Examination of Toronto air data to guide public advice on smog and exercise. Toronto: Public Health; 2003. p. 3-4.
- Oliveira R.S, Barros Neto T.L, Braga A.L.F, Raso V, Pereira LAA, Morette SR, et al. Impact of acute exposure to air pollution on the cardio respiratory performance of military firemen. *Braz J Med Biol Res* 2006; 39(12): 1643-9.
- Stern AC. Air pollution: Air quality management. New York: Academic Press; 1977. p. 343-54.
- Armstrong LE. Air pollution: Exercise in the city. In: Armstrong LE, Editor. Performing in extreme environments. Champaign, IL: Human Kinetics; 2000. pp. 197-229.
- Wilmore JH, Costill DL. Cardiovascular and respiratory function. In: Wilmore JH, Costill DL, Editors. Physiology of sport and exercise. Champaign, IL: Human Kinetics; 1994. p. 121.
- Mittleman MA. Air pollution, exercise, and cardiovascular risk. *N Engl J Med* 2007; 357(11): 1147-9.
- Puvacic Z. Epidemiologic aspects of bronchial carcinoma. *Med Arh* 1995; 49(3-4 Suppl 1): 17-9.
- Raphaelo S. Air pollutants may affect the respiratory system and cardiovascular system. Ezine articles [Online]. 2004; Available from: URL: <http://ezinearticles.com/?id=1224025>
- Braga AL, Saldiva PH, Pereira LA, Menezes JJ, Conceicao GM, Lin CA, et al. Health effects of air pollution exposure on children and adolescents in Sao Paulo, Brazil. *Pediatr Pulmonol* 2001; 31(2): 106-13.
- Adamson IY, Vincent R, Bjarnason SG. Cell injury and interstitial inflammation in rat lung after inhalation of ozone and urban particulates. *Am J Respir Cell Mol Biol* 1999; 20(5): 1067-72.
- Avol EL, Linn WS, Venet TG, Shamoo DA, Hackney JD. Comparative respiratory effects of ozone and ambient oxidant pollution exposure during heavy exercise. *J Air Pollut Control Assoc* 1984; 34(8): 804-9.
- Oliveira RS, Barros Neto TL, Braga AL, Raso V, Pereira LA, Morette SR, et al. Impact of acute exposure to air pollution on the cardiorespiratory performance of military firemen. *Braz J Med Biol Res* 2006; 39(12): 1643-9.
- Adir Y, Merdler A, Ben Haim S, Front A, Harduf R, Bitterman H. Effects of exposure to low concentrations of carbon monoxide on exercise performance and myocardial perfusion in young healthy men. *Occup Environ Med* 1999; 56(8): 535-8.
- Braun-Fahrlander C, Ackermann-Lieblich U, Wanner HU, Rutishauser M, Gnehm HE, Minder CE. Effects of air pollutants on the respiratory system in young children. *Schweiz Med Wochenschr* 1989; 119(41):1424-33.
- Chimenti L, Morici G, Paterno A, Bonanno A, Vultaggio M, Bellia V, et al. Environmental conditions, air pollutants, and airway cells in runners: A longitudinal field study. *J Sports Sci* 2009; 27(9): 925-35.
- Wellenius GA, Batalha JR, Diaz EA, Lawrence J, Coull BA, Katz T, et al. Cardiac effects of carbon monoxide and ambient particles in a rat model of myocardial infarction. *Toxicol Sci* 2004; 80(2): 367-6.
- Sharman JE, Cockcroft JR, Coombes JS. Cardiovascular implications of exposure to traffic air pollution during exercise. *QJM* 2004; 97(10):637-43.
- Chen LH, Knutsen SF, Shavlik D, Beeson WL, Petersen F, Ghamsary M, et al. The association between fatal coronary heart disease and ambient particulate air pollution: Are females at greater risk? *Environ Health Perspect* 2005; 113(12): 1723-9.
- Chitano P, Hosselet JJ, Mapp CE, Fabbri LM. Effect of oxidant air pollutants on the respiratory system: insights from experimental animal research. *Eur Respir J* 1995; 8(8): 1357-71.
- Moshammer H, Hutter HP, Hauck H, Neuberger M. Low levels of air pollution induce changes of lung function in a panel of schoolchildren. *Eur Respir J* 2006; 27(6): 1138-43.
- DeLucia AJ, Adams WC. Effects of O₃ inhalation during exercise on pulmonary function and blood biochemistry. *J Appl Physiol* 1977; 43(1): 75-81.
- Folinsbee LJ. Human health effects of air pollution. *Environ Health Perspect* 1993; 100: 45-56.
- Korrick SA, Neas LM, Dockery DW, Gold DR, Allen GA, Hill LB, et al. Effects of ozone and other pollutants on the pulmonary function of adult hikers. *Environ Health Perspect* 1998; 106(2): 93-9.
- Robert AR, Scott OR. Exercise in differing environments. In: Robert AR, Scott OR, Editors. Fundamental principles of exercise physiology for fitness performance, and health. London: Mc Graw Hill Education; 1999. 398-9.

25. Meltzer AA, Mueller WH, Annegers JF, Grimes B, Albright DL. Weight history and hypertension. *J Clin Epidemiol* 1988; 41(9): 867-74.
26. Sandstrom T. Respiratory effects of air pollutants: experimental studies in humans. *Eur Respir J* 1995; 8(6): 976-95.
27. Chatterjee S, Chatterjee P, Bandyopadhyay A. Skin-fold thickness, body fat percentage and body mass index in obese and non-obese Indian boys. *Asia Pac J Clin Nutr* 2006; 15(2): 231-5.
28. Kleinman MT. Sulfur dioxide and exercise: relationships between response and absorption in upper airways. *J Air Pollut Control Assoc* 1984; 34(1):32-7.
29. Bennett WD, Hazucha MJ, Folinsbee LJ, Bromberg PA, Kissling GE, London SJ. Acute pulmonary function response to ozone in young adults as a function of body mass index. *Inhal Toxicol* 2007; 19(14): 1147-54.
30. Timonen KL, Pekkanen J, Tiittanen P, Salonen RO. Effects of air pollution on changes in lung function induced by exercise in children with chronic respiratory symptoms. *Occup Environ Med* 2002; 59(2): 129-34.
31. Nadel JA, Salem H, Tamplin B, Tokiwa Y. Mechanism of bronchoconstriction during inhalation of dioxide. *J Appl Physiol* 1965; 20: 164-7.
32. Frank NR, Amdur MO, Worcester J, Whittenberger JL. Effects of acute controlled exposure to SO₂ on respiratory mechanics in healthy male adults. *J Appl Physiol* 1962; 17: 252-8.
33. Horvath SM. Impact of air quality in exercise performance. *Exerc Sport Sci Rev* 1981; 9: 265-96.
34. Gibbons SI, Adams WC. Combined effects of ozone exposure and ambient heat on exercising females. *J Appl Physiol* 1984; 57(2): 450-6.
35. Speizer FE, FRANK NR. The uptake and release of SO₂ by the human nose. *Arch Environ Health* 1966; 12(6): 725-8.
36. Hazucha MJ, Madden M, Pape G, Becker S, Devlin R, Koren HS, et al. Effects of cyclo-oxygenase inhibition on ozone-induced respiratory inflammation and lung function changes. *Eur J Appl Physiol Occup Physiol* 1996; 73(1-2): 17-27.
37. Foxcroft WJ, Adams WC. Effects of ozone exposure on four consecutive days on work performance and VO₂max. *J Appl Physiol* 1986; 61(3): 960-6.
38. DeLucia AJ, Adams WC. Effects of O₃ inhalation during exercise on pulmonary function and blood biochemistry. *J Appl Physiol* 1977; 43(1): 75-81.
39. Savin WM, Adams WC. Effects of oxone inhalation on work performance and VO₂ max. *J Appl Physiol* 1979; 46(2): 309-14.
40. Bates D V. Effects of irritant gases on maximal exercise performance. In: Cerretelli P, Whipp BJ, editors. *Exercise Bioenergetics and Gas Exchange*. Amsterdam: Elsevier North Holland Biomedical Press; 1980. pp. 337-44.
41. Lee IM, Hsieh CC, Paffenbarger RS, Jr. Exercise intensity and longevity in men. The Harvard Alumni Health Study. *JAMA* 1995; 273(15): 1179-84.
42. Sesso HD, Paffenbarger RS, Jr., Lee IM. Physical activity and coronary heart disease in men: The Harvard Alumni Health Study. *Circulation* 2000; 102(9): 975-80.