

Assessment of Allergic Biomarkers; Total Immunoglobulin E Antibodies Levels and Peripheral Blood Eosinophil among Public Transporter Drivers with Traffic-Related Respiratory Diseases in Tehran

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ABSTRACT

Background: Traffic air pollution can be induced or developed the different respiratory diseases. Megacity has potential magnitude in the production of high density and large- volume traffic triggering factors in the outdoor environment. High concentrations of Formaldehyde and polycyclic aromatic hydrocarbons (PAHs) are found in traffic density of ambient air in urban area. It can be stimulated both allergic state and diseases. The PAHs originates from the motor engine are as pro-inflammatory compounds which can enhance Immunoglobulin E (IgE) responses. The purpose of the study was evaluation of allergic markers among public drivers in Tehran, to assess the respiratory diseases.

Materials and Methods: A total of 151 subjects were sequentially enrolled among public drivers with traffic related respiratory diseases. The total IgE antibodies in serum were measured according to manufacture recommendation.

Results: The mean age recorded 47.66 ± 8.82 standard deviation (SD). The peripheral eosinophil proportion was 3.19 ± 2.16 SD. The mean IgE antibody levels were 205.89 ± 238.67 SD. Allergic state was found in 47% of target population. Frequencies of traffic-related air pollution diseases (TRAPD) in chronic obstructive pulmonary disease (COPD) 34%, asthma 25%, rhinitis 21% and bronchitis 21%, respectively. Allergic state distributed high frequency in asthma, rhinitis, COPD and bronchitis diseases, respectively.

Conclusion: Allergic biomarkers of IgE antibody and peripheral eosinophilia were widely distributed among TRAPD. They observed more frequency on the allergic base diseases than non-allergic small airway diseases. It may be reflected the actual role of traffic -related air pollution on the sensitization of all categories of TRAPD.

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► *Implication for health policy/practice/research/medical education:* Total Immunoglobulin E Antibodies Levels and Peripheral Blood Eosinophil among Public Transporter Drivers

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1. Introduction:

Traffic air pollution can be induced or developed small airway diseases (1) and chronic obstructive pulmonary disease (COPD) (2, 3). It can stimulate the allergic state as a pre-risk factor (4) in development of allergic diseases (5, 6) and however, it is sensitized individual to adult-onset asthma (7).

Urban pollution is complex compounds. Incomplete combustion of fossil-fuel produces gaseous and particulate emissions. Polycyclic aromatic hydrocarbons (PAHs) are detected 46-90% within the traffic air pollution (8). The PAHs from the motor engine activities is as pro-inflammatory compounds and enhanced Immunoglobulin E (IgE) responses as a biomarker of allergy (9). It seems to be responsible in inducing allergic airway (10, 11) and asthma (12).

Tehran is the capital of Iran, a big city with heavy volume and highly density traffic (13). Heath's issue of urban citizen is extremity distributed (14). Drivers of the public service are long-standing exposure and at-risk of health status.

The purpose of this study was evaluation of allergic markers among public drivers in Tehran, to assess the respiratory diseases.

2. Materials and Methods:

This study was cross-sectional. It finalized at Shahid Beheshti University of Medical Sciences (SBUMS), Tehran-Iran, in 2012.

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Setting of this study was designed in Loghman Hakim general teaching hospital. Tehran; the capital city of Iran was the field of study.

Participations followed the inclusion criteria. They consisted as males, public transporter drivers, background of the work duration up to five years and respiratory symptoms or known small airway diseases. We categorized traffic-related air pollution diseases (TRAPD) in two conditions; allergic disease (asthma and rhinitis) and small airway disease (COPD and subclinical chronic bronchitis). Selection of the participants occurred through the chest clinic. All the numbers within the group enrolled based upon the call and encourage by the previously selected driver co-workers. Men had high frequency of the public drivers in our country. Entire subjects from this study had work-time over ten hours per day and six days per week.

Exclusion criteria matched lack of interest, unable to follow the study designed, smoker, hubble bubble, opium user and being actuality another disorder.

One physician examined the participants and followed with standard chest X-ray and pulmonary function test. Diagnosed and treated patients were accepted as the respiratory disease and not followed.

An open questionnaire was distributed among participations. The topics included demographic data major history, respiratory symptoms, rhinitis symptoms, allergic history, habits and medications.

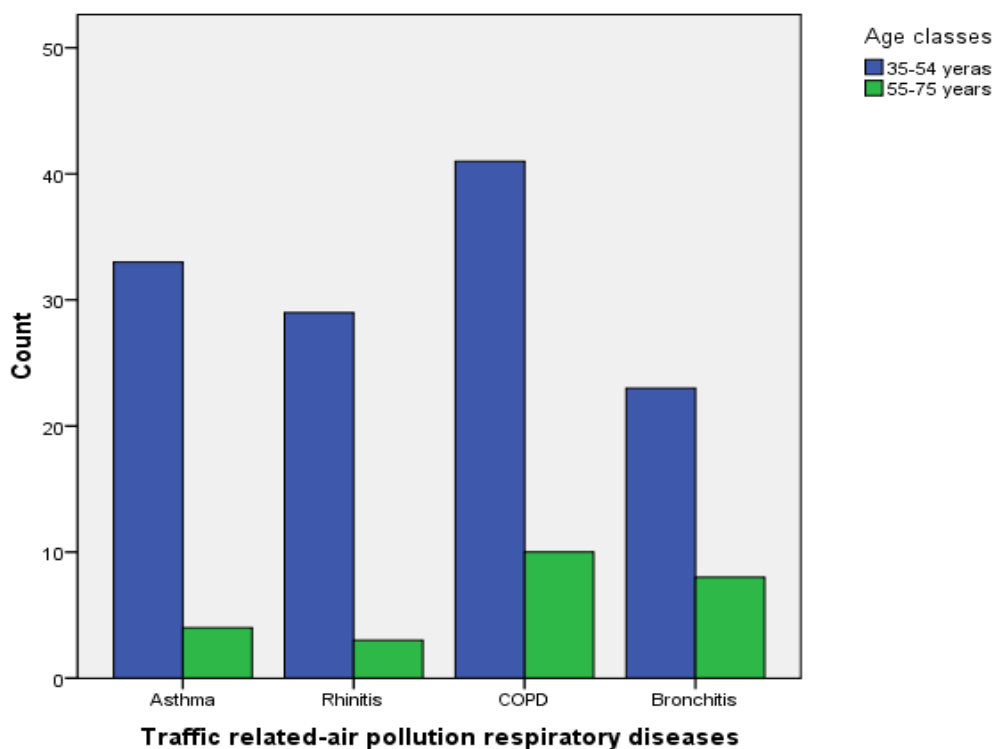


Fig. 1. It demonstrates the frequency distribution of traffic- related air pollution diseases with age classes.

Bronchial asthma and COPD were documented contracted on the American Thoracic Society (ATS) (15) and Global Initiative for Chronic Obstructive Lung

Disease (GOLD) (16) guidelines, as respectively. Rhinitis allergic evaluated based on the history and symptom and relation to trigger factors.

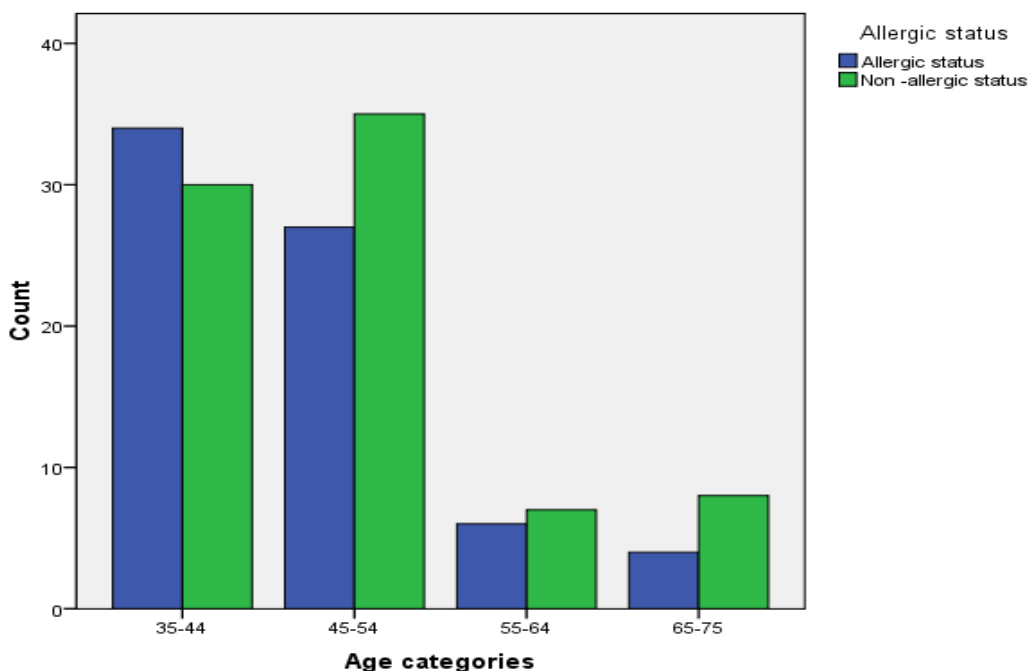


Fig. 2. It reveals distribution of age categories with allergic status in target population.

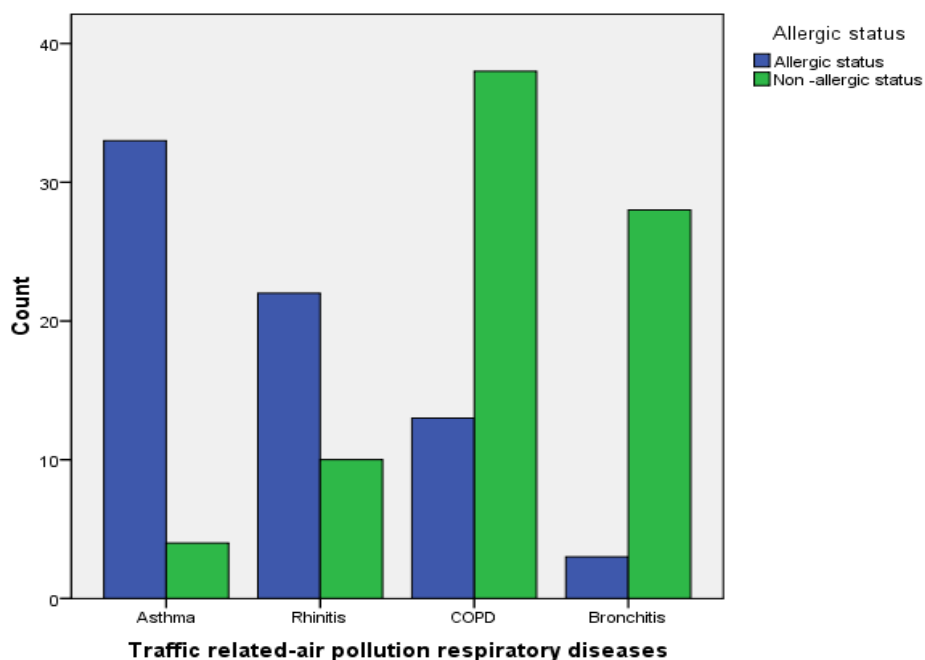


Fig. 3. It shows status allergic status with SAD subtypes.

Whole blood sample was obtained all the subjects at fasting state from the vein. They were transfers to unique laboratory. Total IgE antibody measured with Enzyme-Linked Immunosorbent Assay

(ELISA) according to the Manufacture’s protocol (Padtan Elm, Iran Co Ltd). Set point of allergic state was up to 180 Iu/mL. Complete blood count was carried out with trade name of Sysmex kx-21. Cut of point

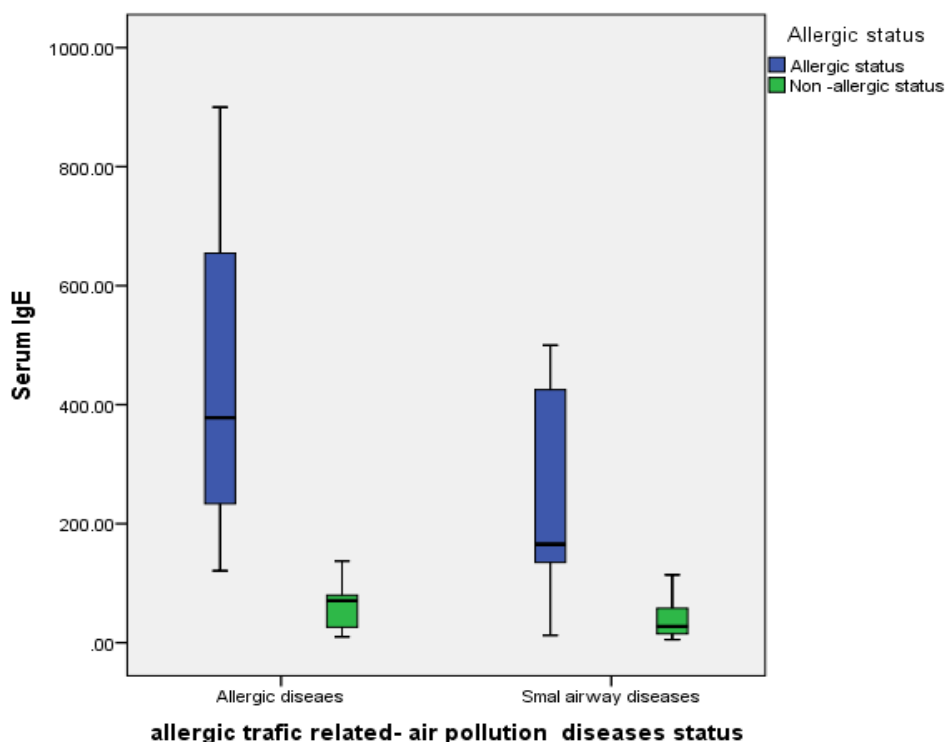


Fig. 4. It presents frequency of IgE status with subgroups of traffic- related air pollution diseases (TRAPD).

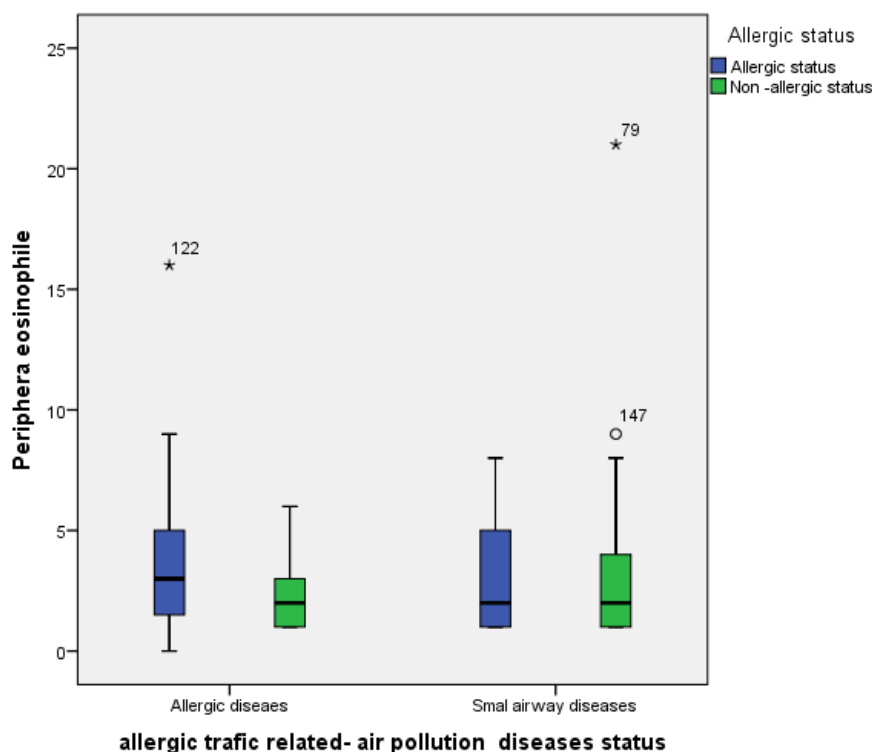


Fig. 5. It presents frequency of IgE status with subgroups of traffic- related air pollution diseases (TRAPD).

for eosinophilia defined on the upper normal rang >2%.

Data analyzed with SPSS program V. 18.0. Frequency distribution was evaluated with percent. Test of normality; Kolmogorov-Smirnov, Shapiro-Wilk were $p < 0.001$. Effect size between subsets was 0.83. Compare of mean was performed with nonparametric tests of independent-sample T test; Mann-Whitney U and Crosstab. Association was carried out with Chi-square test; Fisher's exact test and correlation; Spearman's rho. P value was set at a significant level in through of the study (< 0.05 two tailed).

3. Results:

A total of 151 public drivers, male patients completed criteria for the study. The mean age was 47.66 ± 8.82 SD, ranged over 35-75, median was 45 years. Frequency of population in the age categories disclosed cumulative percent 83 in the range over 35-54 years. It was the first class of the target population.

Figure 1 demonstrates the frequency distribution of TRAPD with age classes. Frequencies of (TRAPD) in COPD were 34%, asthma 25%, rhinitis 21% and bronchitis 21%, respectively. Figure 2 reveals distribution of age categories with allergic status in target population. 47% of target population was allergic. Percent of allergic status in subsets of focus population for asthma was 47%, rhinitis 31%, COPD 18%, bronchitis 4%, respectively.

Figure 3 shows allergic status with SAD subtypes. Mean of IgE antibody concentrations in serum was 205.89 ± 238.67 SD Iu/mL. Serum IgE distributed in more frequency in the less than 54 years age (83%). Of those, 48% and 38% were within first and second age classes, respectively. However, high frequency of allergic level of IgE antibodies located at the allergic TRAPD group 78%. Figure 4 presents IgE status of the TRAPD group. There were relevant differences between IgE level in serum with TRAPD ($p < 0.001$). However,

marked positive correlation was detected between serum allergic biomarkers (IgE and PE), ($r=0.2$, $p=0.004$).

Mean \pm SD of peripheral eosinophilia (PE) proportion was 3.19 ± 2.69 percent, ranged between 0-21 percent with 3% median. It found 52% in the first class of age and in 52% within TRAPD. Figure 3 reveals peripheral eosinophilia status with TRAPD. Fisher's exact test was reflected significant differences between PE with TRAPD ($p<0.03$).

Figure 5 shows TRAP diseases with allergic status. Allergic-TRAPD was distributed within 35-54 years class was 46%. It was significant statistically ($p<0.001$). Frequency of asthma and rhinitis within former age class was 25% and 21%, separately. Moderate strength Spearman's correlation was detected between allergic status with allergic-TRAPD ($r=0.6$, $p<0.001$).

4. Discussion:

TRAPD is one of the causative factors in the urban environmental area. Biomass fuels and motor vehicle's ambient particulate matter are the main component of TRAPD. They are triggering inflammation and inducing oxidative stress in at risk-subjects (17). The hazard effects are related to the volume and density of the pollutants (18). In addition, they can be inception or exacerbation of allergic diseases (19).

Rapid urbanization causes to change Tehran, capital of Iran to the metropolitan. It had one of the worst atmospheric air pollution in the world (20).

Bronchial asthma is a chronic inflammatory airway disease. TRAPD suggested a role in induction of adult-onset asthma (21, 22). Prevalence of asthma is varied between outdoor environmental pollutants of the inner-cities. It reported 2.8% in Mashhad, Iran (23) and 5% in Mexico (24). Incidence of asthma-related air pollution is not clear in adults. However, there is a self-report prevalence of adult asthma in close-living to traffic 8.7% (25).

Asthma can be developed by the allergic state as a pre-risk factor (26). TRAPD can be potentiating allergic reactions (27) and allergic respiratory diseases (28). Significant IgE levels in serum of asthmatic public transport drivers (PTDs) may support the recent concept.

PTDs are at high risk of occupational diseases, mortality (29) and disability. They are spent so much of work time in the air pollution environment. Their workplace is exposed against hazardous air pollution. COPD is a chronic airway disease with irreversible airway obstruction. It is leading cause of death around the world. Smoking has a potential risk factor in development of the diseases (30). However, 25-45% of COPD patients are established particularly in the developing countries and non-smoking (31). Approximately, half of the worldwide people are exposed against fuel biomass smoke (31). Air pollution is a causative factor in development of COPD (32, 33).

Asthma-COPD phenotype is an accepted subject among COPD patients with an asthma pattern (34, 35). They are associated with raised immunoglobulin E antibody and bronchial hyper-responsiveness. Outcome of the recent phenotype of COPD is not clearly evaluated in relation to morbidity and mortality.

Chronic bronchitis (CB) is a clinical definition in the epidemiological study. Induction of bronchitis is depended to duration and intensity of exposure-response agents. The CB is induced in exposing against the TRAPD (25, 36). It is in a natural course encountered a risk factor in development of COPD but may be absent in the established the diseases (37). Our outcome for the study does not meet the criteria of obstructive pattern of COPD patients, and subclinical CB defined as a separate entity. Chronic bronchitis-asthma can be an overlap disease (38, 39). It is contributed with associated symptoms and clinical features (40), and developed in air pollution

conditions (38, 39). However, atopy may be predisposed in development of CB (41). It appears in the residency in the TRAPD conditions and raised risk of allergic sensitization (42).

There is a relationship between allergy inflammatory response and air pollution. They have been reported as a predisposal factor in developing atopy, allergic sensitization state and diseases (43, 44). Rhinitis may be an associated component of atopic and allergic condition of the subjects or presented as alone.

5. Conclusion:

Allergic biomarkers (IgE antibody) and peripheral eosinophilia were widely distributed among TRAPD. They observed more frequency on the allergic base diseases than non-allergic small airway diseases. In addition, allergic base or allergic phenotype diseases detected more frequency in the middle age subjects; less than 54 years. The end point resulting may be reflected the actual role of traffic-related air pollution on the changed direction threshold allergic status toward raised new set point between public drivers with SAD. However, it has been an apparent role in inducing or development of allergic sensitization in the transporter drivers mainly with non-allergic bas.

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References

1. Kelly FJ, Fussell JC. Air pollution and airway disease. *Clin Exp Allergy*. 2011;41(8):1059-71.
2. Ko FW, Hui DS. Air pollution and chronic obstructive pulmonary disease. *Respirology*. 2012;17(3):395-401.
3. Zeng G, Sun B, Zhong N. Non-smoking-related chronic obstructive pulmonary disease: a neglected entity?. *Respirology*. 2012;17(6):908-12.
4. Jung KH, Yan B, Moors K, Chillrud SN, Perzanowski MS, Whyatt RM, Hoepner L, Goldstein I, Zhang B, Camann D, Kinney PL, Perera FP, Miller RL. Repeated exposure to polycyclic aromatic hydrocarbons and asthma: effect of seroatopy. *Ann Allergy Asthma Immunol*. 2012;109(4):249-54.
5. Bernstein DI. Diesel exhaust exposure, wheezing and sneezing. *Allergy Asthma Immunol Res*. 2012;4(4):178-83.
6. Takizawa H. Impact of air pollution on allergic diseases. *Korean J Intern Med*. 2011;26(3):262-73.
7. Künzli N, Bridevaux PO, Liu LJ, Garcia-Esteban R, Schindler C, Gerbase MW, Sunyer J, Keidel D, Rochat T; Swiss Cohort Study on Air Pollution and Lung Diseases in Adults. Traffic-related air pollution correlates with adult-onset asthma among never-smokers. *Thorax*. 2009;64(8):664-70.
8. Narváez RF, Hoepner L, Chillrud SN, Yan B, Garfinkel R, Whyatt R, Camann D, Perera FP, Kinney PL, Miller RL. Spatial and temporal trends of polycyclic aromatic hydrocarbons and other traffic-related airborne pollutants in New York City. *Environ Sci Technol*. 2008;42(19):7330-5.
9. Al-Daghri NM, Alokail MS, Abd-Alrahman SH, Draz HM, Yakout SM, Clerici M. Polycyclic aromatic hydrocarbon exposure and pediatric asthma in children: a case-control study. *Environ Health*. 2013;12(1):1.
10. Aubier M. [Traffic-related pollutants and their impact on allergic respiratory diseases *Bull Acad Natl Med*. 2009;193(6):1303-13.
11. Carlsten C, Melén E. Air pollution, genetics, and allergy: an update. *Curr Opin Allergy Clin Immunol*. 2012;12(5):455-60.
12. Ebtakar M. Air pollution induced asthma and alterations in cytokine patterns. *Iran J Allergy Asthma Immunol*. 2006;5(2):47-56.
13. Naddafi K, Hassanvand MS, Yunesian M, Momeniha F, Nabizadeh R, Faridi S, Gholampour A. Health impact assessment of air pollution in megacity of Tehran, Iran. *Iranian J Environ Health Sci Eng*. 2012;9(1):28.
14. Azizi MH. Impact of traffic-related air pollution on public health: a real challenge. *Arch Iran Med*. 2011;14(2):139-43.

15. Pellegrino R, Viegi G, Brusasco V, Crapo RO, Burgos F, Casaburi R, Coates A, van der Grinten CP, Gustafsson P, Hankinson J, Jensen R, Johnson DC, MacIntyre N, McKay R, Miller MR, Navajas D, Pedersen OF, Wanger J. Interpretative strategies for lung function tests. *Eur Respir J*. 2005;26(5):948-68.
16. GOLD - the Global initiative for chronic Obstructive Lung Disease. Available at: http://www.goldcopd.org/uploads/users/files/GOLD_Report_2013.pdf
17. Hogg JC, van Eeden S. Pulmonary and systemic response to atmospheric pollution. *Respirology*. 2009;14(3):336-46.
18. Alnawaiseh NA, Hashim JH, Md Isa Z. Relationship Between Vehicle Count and Particulate Air Pollution in Amman, Jordan. *Asia Pac J Public Health*. 2012 Aug 16. [Epub ahead of print]
19. Leung TF, Ko FW, Wong GW. Roles of pollution in the prevalence and exacerbations of allergic diseases in Asia. *J Allergy Clin Immunol*. 2012;129(1):42-7.
20. Naddafi K, Hassanvand MS, Yunesian M, Momeniha F, Nabizadeh R, Faridi S, Gholampour A. Health impact assessment of air pollution in megacity of Tehran, Iran. *Iranian J Environ Health Sci Eng*. 2012;9(1):28.
21. Künzli N, Bridevaux PO, Liu LJ, Garcia-Esteban R, Schindler C, Gerbase MW, Sunyer J, Keidel D, Rochat T; Swiss Cohort Study on Air Pollution and Lung Diseases in Adults. Traffic-related air pollution correlates with adult-onset asthma among never-smokers. *Thorax*. 2009;64(8):664-70.
22. Jacquemin B, Schikowski T, Carsin AE, Hansell A, Krämer U, Sunyer J, Probst-Hensch N, Kauffmann F, Künzli N. The role of air pollution in adult-onset asthma: a review of the current evidence. *Semin Respir Crit Care Med*. 2012;33(6):606-19.
23. Boskabady MH, Kolahdoz GH. Prevalence of asthma symptoms among the adult population in the city of Mashhad (north-east of Iran). *Respirology*. 2002;7(3):267-72.
24. García-Sancho C, Fernández-Plata R, Martínez-Briseño D, Franco-Marina F, Pérez-Padilla JR. Adult asthma in Mexico City: a population-based study. *Salud Publica Mex*. 2012;54(4):425-32.
25. Lindgren A, Strohm E, Montn emery P, Nihl en U, Jakobsson K, Axmon A. Traffic-related air pollution associated with prevalence of asthma and COPD/chronic bronchitis. A cross-sectional study in Southern Sweden. *Int J Health Geogr*. 2009;8:2.
26. Boulay ME, Boulet LP. The relationships between atopy, rhinitis and asthma: pathophysiological considerations. *Curr Opin Allergy Clin Immunol*. 2003;3(1):51-5.
27. Salvi, Sundeep. Pollution and allergic airways disease. *Curr Opin Allergy Clin Immunol*. 2001;1(1):35-41.
28. Laumbach RJ, Kipen HM. Respiratory health effects of air pollution: update on biomass smoke and traffic pollution. *J Allergy Clin Immunol*. 2012;129(1):3-11.
29. Brunekreef B, Beelen R, Hoek G, Schouten L, Bausch-Goldbohm S, Fischer P, Armstrong B, Hughes E, Jerrett M, van den Brandt P. Effects of long-term exposure to traffic-related air pollution on respiratory and cardiovascular mortality in the Netherlands: the NLCS-AIR study. *Res Rep Health Eff Inst*. 2009;(139):5-71.
30. Eisner MD, Anthonisen N, Coultas D, Kuenzli N, Perez-Padilla R, Postma D, Romieu I, Silverman EK, Balmes JR; Committee on Nonsmoking COPD, Environmental and Occupational Health Assembly. An official American Thoracic Society public policy statement: Novel risk factors and the global burden of chronic obstructive pulmonary disease. *Am J Respir Crit Care Med*. 2010;182(5):693-718.
31. Salvi SS, Barnes PJ. Chronic obstructive pulmonary disease in non-smokers. *Lancet*. 2009;374(9691):733-43.
32. Schikowski T, Sugiri D, Ranft U, Gehring U, Heinrich J, Wichmann HE, Krämer U. Long-term air pollution exposure and living close to busy roads are associated with COPD in women. *Respir Res*. 2005;6:152.
33. Zeng G, Sun B, Zhong N. Non-smoking-related chronic obstructive pulmonary disease: a neglected entity? *Respirology*. 2012;17(6):908-12.
34. Rathod VP, Kapoor P, Pillai KK, Khanam R. Assessment of asthma and chronic obstructive pulmonary disorder in relation to reversibility, IgE, eosinophil, and neutrophil count in a University Teaching

- Hospital in South Delhi, India. *J Pharm Bioallied Sci.* 2010;2(4):337-40.
35. Soler-Cataluña JJ, Cosío B, Izquierdo JL, López-Campos JL, Marín JM, Agüero R, Balóira A, Carrizo S, Esteban C, Galdiz JB, González MC, Miravittles M, Monsó E, Montemayor T, Morera J, Ortega F, Peces-Barba G, Puente L, Rodríguez JM, Sala E, Sauleda J, Soriano JB, Viejo JL. Consensus document on the overlap phenotype COPD-asthma in COPD. *Arch Bronconeumol.* 2012;48(9):331-7.
36. Künzli N, Kaiser R, Medina S, Studnicka M, Chanel O, Filliger P, Herry M, Horak F Jr, Puybonnieux-Textier V, Quénel P, Schneider J, Seethaler R, Vergnaud JC, Sommer H. Public-health impact of outdoor and traffic-related air pollution: a European assessment. *Lancet.* 2000;356(9232):795-801.
37. Lu M, Yao W, Zhong N, Zhou Y, Wang C, Chen P, Kang J, Huang S, Chen B, Wang C, Ni D, Wang X, Wang D, Liu S, Lu J, Shen N, Ran P. Chronic obstructive pulmonary disease in the absence of chronic bronchitis in China. *Respirology.* 2010;15(7):1072-8.
38. Björnsson E, Plaschke P, Norrman E, Janson C, Lundbäck B, Rosenhall A, Lindholm N, Rosenhall L, Berglund E, Boman G. Symptoms related to asthma and chronic bronchitis in three areas of Sweden. *Eur Respir J.* 1994;7(12):2146-53.
39. Uzun K, Ozbay B, Ceylan E, Gencer M, Zehir I. Prevalence of chronic bronchitis-asthma symptoms in biomass fuel exposed females. *Environ Health Prev Med.* 2003;8(1):13-7.
40. Bahous J, Cartier A, Ouimet G, Pineau L, Malo JL. Nonallergic bronchial hyperexcitability in chronic bronchitis. *Am Rev Respir Dis.* 1984;129(2):216-20.
41. Terho EO, Koskenvuo M, Kaprio J. Atopy: a predisposing factor for chronic bronchitis in Finland. *J Epidemiol Community Health.* 1995;49(3):296-8.
42. Heinrich J, Topp R, Gehring U, Thefeld W. Traffic at residential address, respiratory health, and atopy in adults: the National German Health Survey 1998. *Environ Res.* 2005;98(2):240-9.
43. Salvi S. Pollution and allergic airways disease. *Curr Opin Allergy Clin Immunol.* 2001;1(1):35-41.
44. Proietti L, Spicuzza L, Polosa R. Urban air pollution at the crossroads of the allergic pandemic. *Ann Ital Med Int.* 2003;18(2):64-72.