Environmental Health Engineering and Management Journal

Original Article

CrossMark
click for updates

Health impact assessment of short-term exposure to NO_2 in Kermanshah, Iran using AirQ model

Yusef Omidi[1], Gholamreza Goudarzi[2], Ali Mirza Heidari[3], Seyed Mohammad Daryanoosh[1*]

[1]MSc, Department of Environmental Health Engineering, School of Health, Lorestan University of Medical Sciences, Khorramabad, Iran
[2]Associate Professor, Environmental Technologies Research Center, Ahvaz Jundishapur University of Medical Sciences, Ahvaz, Iran
[3]BSc, Department of Environmental Health Engineering, School of Public Health, Ahvaz Jundishapur University of Medical Sciences, Ahvaz, Iran

Abstract

Background: Epidemiological studies have illustrated that exposure to atmospheric nitrogen oxides (NO_2/NO_X) is responsible for an increased risk of acute or chronic diseases such as cancer. In this study the health risks associated with nitrogen dioxide (NO_2) were assessed in the city of Kermanshah, the capital of Kermanshah province, Iran.

Methods: Data on hourly NO_2 levels that had been measured with the Environ tech model M200 was taken from the Kermanshah Environmental Protection Agency (KEPA). The AirQ2.2.3 model was used to quantify data based on baseline incidence (BI), relative risk (RR), and attributable proportion (AP). The number of cases of cardiovascular mortality (CM), acute myocardial infarction (MI), and hospital admissions for chronic obstructive pulmonary diseases (HACOPD) was estimated.

Results: The results of the current study show that there were 33, 16, and 13 cases of CM, acute MI, and HACOPD, respectively, attributable to NO_2 exposure. Furthermore, 26.85% of health impacts occurred on days when NO_2 levels did not exceed 40 μg/m³. For every 10 μg/m³ increase in the NO_2 level, the risk of CM, MI, and HACOPD rose about 0.2%, 0.36%, and 0.38%, respectively.

Conclusion: In order to reduce the number of cases of mortality and morbidity caused by exposure to NO_2, several immediate steps should be taken by the government to control emissions from various sources, particularly car exhaust, to reduce the levels of NO_2 in the atmosphere.

Keywords: Health impact, Myocardial infarction, COPD, NO_2, Kermanshah

Citation: Omidi Y, Goudarzi G, Heidari AM, Daryanoosh SM. Health impact assessment of short-term exposure to NO_2 in Kermanshah, Iran using AirQ model. Environmental Health Engineering and Management Journal 2016; 3(2): 91–97. doi: 10.15171/EHEM.2016.06.

*Correspondence to:
Seyed Mohammad Daryanoosh
Email:
mohammad_daryanoosh@yahoo.com

Introduction

Air pollution due to industrialization and urbanization is one of the major environmental issues of the current century, especially in developing countries (1-4). Because of its adverse effects on human health, exposure to air pollution is related to increased rates of mortality and morbidity (5-8). Air pollution is a mixture of different atmospheric pollutants such as particulate matter, nitrogen dioxide (NO_2), carbon monoxide, ozone, sulfur dioxide, etc (7,9). In recent decades, researchers have considered short-term and long-term exposure to air pollutants and the outcome of such exposure on human health (10-12). Among different atmospheric pollutants, NO_2 is a gas with oxidant properties that is capable of diminishing the air quality in many urban and industrial contexts as well as the indoor air of homes with domestic combustion devices. Nitrogen oxide (NO) is the main precursor of NO_2 in the presence of atmospheric oxidants such as ozone (O_3). In polluted

air, NO can be quickly oxidized by O_3 and then converted into NO_2 through a photochemical reaction (13-16). The main source of NO_2 in the urban atmosphere is gas released from road traffic. Other anthropogenic sources of NO_2 include power stations, fossil fuels, waste incinerates, industrial boilers, and household heaters. Furthermore, the major sources of NO_2 in indoor environments are natural gas cookers and smoke resulting from burning cigarettes (14,17). According to the results of epidemiological studies in Europe and around the world, approximately 5%-7% of lung cancer cases among ex-smokers and non-smokers can be associated with exposure to high levels of air pollutants containing NO_2 or being in the proximity of roads with heavy traffic (17,18). A few studies have demonstrated that short-term exposure to NO_2 can cause mortality in exposed populations because of cardiovascular diseases (14,19). Jenkins et al stated that exposure to an NO_2 level of 400 ppb for 3 hours alone or in combina-

tion considerably increased sensitivity to inhaled allergens compared with contact to air without additive effects (20). Some epidemiological studies have indicated that ambient air pollution is related to cardiovascular diseases, especially myocardial infarction (MI) (21). Acute MI is a serious sickness that typically leads to death and increases the health costs of a society. Goudarzi et al demonstrated the association between increasing NO_2 levels and acute MI among people in Ahvaz, Iran (22). Chronic obstructive pulmonary disease (COPD) is an inflammatory airway disease. NO_2 is a slight soluble gas that is maintained in the small airways of the lungs. Thus, patients with COPD or asthma attacks are more sensitive to low levels of NO_2 (23). Ghozikali et al showed that approximately 0.9% of total hospitalizations associated with COPD resulted from exposure to NO_2 (18). Fattore et al showed that exposure to atmospheric NO_2 caused cardiovascular mortality (CM) in the inhabitants of the cities of Mazzano and Rezzato, Italy (14). Because of the effects of air pollution on human health demonstrated by data from various studies, the World Health Organization (WHO), European governments, and other associations have started shaping environmental policies to investigate the impacts of atmospheric pollution on public health (24,25). Another study by Yang et al showed an association between hospital admissions and an increase of 32 µg/m³ in NO_2 concentration in the air of Kaohsiung, Taiwan (26). The current study assessed the health impacts of exposure to NO_2 on the inhabitants of Kermanshah, Iran during 2014-2015.

Methods

Study area

Kermanshah (34°19′N, 47°7′E), the capital city of Kermanshah province, is located in western Iran. It has a population of about 870 000 persons and is the ninth most crowded city in Iran. It is well-known for its cultural sites and monuments and is also a good passage for transportation between Iran and Iraq. Kermanshah is one of the most polluted cities in Iran because of several huge industrial factories such as petrochemical refinery and Kermanshah Oil Refining Company (KORC) that are located in the center of the city (27) and also because of the high number of automobiles (28) (see Figure 1). One air pol-

lution-monitoring station situated in Ziba Park and Kermanshah Environmental Protection Agency (KEPA) were responsible for measuring NO_2 concentrations in the air. The hourly NO_2 concentrations for one year, from March 2014 to March 2015, were taken from the KEPA for use in the present study.

Relative risk and baseline incidence

The Air Quality Health Impact Assessment (AirQ2.2.3) software was developed to calculate the health effects of short-term contact with atmospheric pollutants on a resident population in a specific area for one year. In epidemiological studies, relative risk (RR) is the increase in chances of developing a sickness caused by exposure to a pollutant. The values of RR (per 10 µg/m³) defined by the WHO and baseline incidence (BI, per 100 000 people) associated with different types of mortality and morbidity cases are shown in Table 1. The amounts of RR and related BI have been extracted from AirQ2.2.3 data files developed by the WHO European Center for Environment and Health based on various conducted peer-reviewed studies (29,30).

Standardization of NO2 data

The Environ tech model M200 was used to measure NO_2. This system continuously monitored atmospheric NO_2 concentrations and involved a cross-flow modulated semi-decompression chemiluminescence method. Raw data obtained from KEPA was based on volumetric unit (ppm or ppb), while the AirQ software accepted data based on gravimetric units (µg/m³). Thus for conversion, the data from air quality monitoring was saved in a Microsoft Excel spreadsheet. All processing mechanisms for converting volumetric data to gravimetric units and other processes include averaging, coding, modifying pressure and temperature, and data filtering were carried out using the mentioned software. For filtering, the data was sorted into averages of diurnal concentrations, briefed by sorting amounts from lower to higher, and then the number of days in the range of NO_2 concentrations were identified. Data was converted by the ideal gas formula presented in Eq. 1:

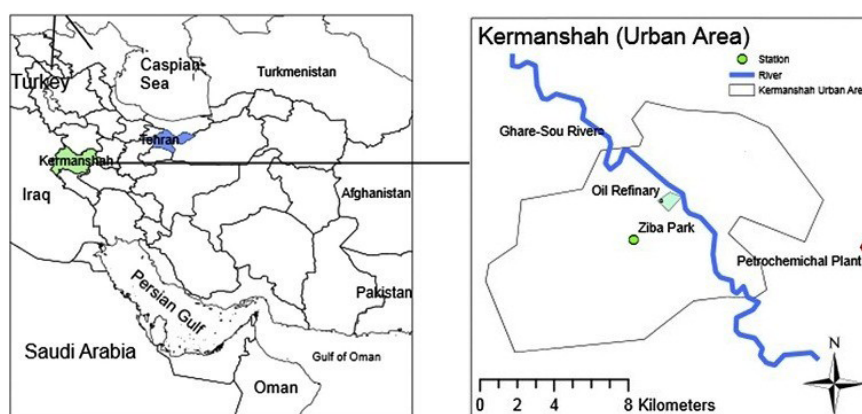$$P_1 V_1 / T_1 = P_2 V_2 / T_2 \qquad (1)$$

Figure 1. Kermanshah city and sampling station.

Table 1. RR with 95% CI used for investigating the health effects of NO_2

Health effect		BI	RR (95% CI) per 10 µg/m³
Mortality	CM	497	1.0020 (1.00-1.0040) (14)
Morbidity	Acute MI	132	1.0036 (1.0015-1.0084) (17)
	HACOPD	101.4	1.0038 (1.0004-1.0094) (18)

Abbreviations: RR, relative risk; NO_2, nitrogen dioxide; BI, baseline incidence; CM, cardiovascular mortality; HACOPD, hospital admissions for chronic obstructive pulmonary diseases; MI, myocardial infarction.

where parameters of P_1, V_1, and T_1 are the initial pressure, volume, and absolute temperature, respectively. Also, P_2, V_2, and T_2 are the final amounts of pressure, volume, and absolute temperature under standard conditions, respectively.

Concentration response equations

The AirQ2.2.3 software model was proposed by the WHO for assessing potential outcomes of exposure to different pollutants in the atmosphere on the health of people living in a given place and period (5). The estimation was based on the population attributable risk proportion (AP) defined as the portion of the health effect on a specified inhabitant exposed for a certain time to a specific atmospheric pollutant (14,31). The AP can be estimated using Eq. (2):

$$AP = \sum\{[RR(c)-1] \times P(c)\} / \sum[RR(c) \times P(c)] \qquad (2)$$

where AP is the attributable proportion of the health end-point and RR(c) is the RR for certain human health effect in category "c" (e.g., residential or industrial) of contact that can be obtained from the exposure-response functions taken from different studies. P(c) is the exposed group of people (5,14).

The rate attributable to the exposure can be estimated using the following equation, if the baseline frequency of the health effect in the population under evaluation has been determined:

$$IE = I \times AP \qquad (3)$$

where IE and I are the rate of the health impact attributable to the exposure and the BI of the health effect on investigated inhabitants, respectively. If the population rate is known, the number of estimated excess cases associated with the exposure may be calculated using Eq. (4):

$$NE = IE \times N \qquad (4)$$

where NE is the number of excess persons associated with the contact and N is the number of people under study.

Input adjustment

Mortality and morbidity cases were assessed by running the AirQ model version: 2.2.3. The different parameters were considered, and concentration intervals of NO_2 were recorded into 10 µg/m³ categories related to equivalent exposure categories. The model assumes that the NO_2 measured data is representative of the mean exposure of the people. Finally, the cumulative numbers of excess cases for CM, acute MI, and HACOPD were detected using AirQ2.2.3 software based on RR and BI presented above

in Table 1.

Results

The annual average, annual maximum, summer and winter averages, summer and winter maximums, and 98th percentile of NO_2 concentration taken from running the AirQ2.2.3 software are illustrated in Figure 2. Obviously, the annual average concentration of NO_2 in Kermanshah was equal to 75.76 µg/m³. The average summer NO_2 level was 63.08 µg/m³, which is lower than the winter average of 77.34 µg/m³. The maximum annual NO_2 concentration was observed in the winter with a concentration of 198.42 µg/m³. This value was higher than the summer concentration with a maximum of 142.93 µg/m³.

Table 2 illustrates the association between percentages of the population attributable risk (AP) and the cumulative number of excess cases of exposure to atmospheric NO_2 among people of Kermanshah. As seen, the cumulative numbers of CM, MI, and HACOPD cases in a central RR and AP percentages of 1.20%, 2.15%, and 2.27% per 100 000 people were estimated to be 33, 16, and 13 persons, respectively. According to the model's default for NO_2, in low RR (RR=1), the value of estimated AP is zero. Therefore, the cumulative number of excess cases for CM is zero.

Figure 3 shows the results of quantifying the health outcomes of exposure to NO_2 in Kermanshah obtained from the model. This figure illustrates diagrams based on the
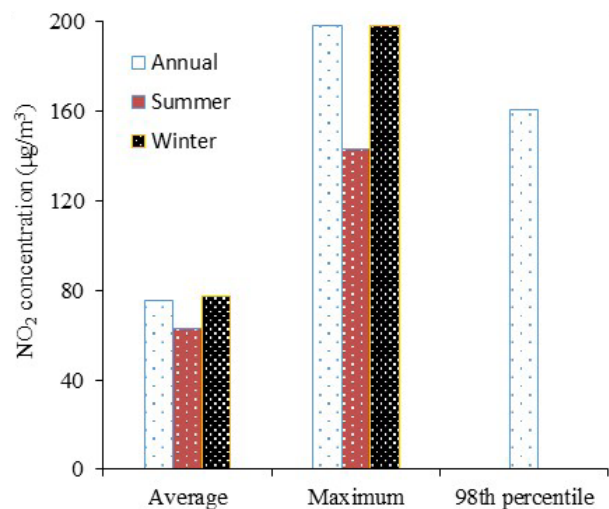

Figure 2. The highest and the lowest concentrations of NO_2 in Kermanshah.

Table 2. Estimated AP percentages and number of excess cases due to short-term exposure to NO_2 levels above 10 µg/m³

Health effects	Estimated AP (%)	NO. of excess cases
CM	1.2085 (0.000-2.3881)	33 (0.0-65)
Acute MI	2.1515 (0.9091-4.8867)	16 (7-35)
HACOPD	2.2715 (0.2441-5.4369)	13 (2-30)

Abbreviations: NO_2, nitrogen dioxide; CM, cardiovascular mortality; HACOPD, hospital admissions for chronic obstructive pulmonary diseases; MI, myocardial infarction; AP, attributable proportion.
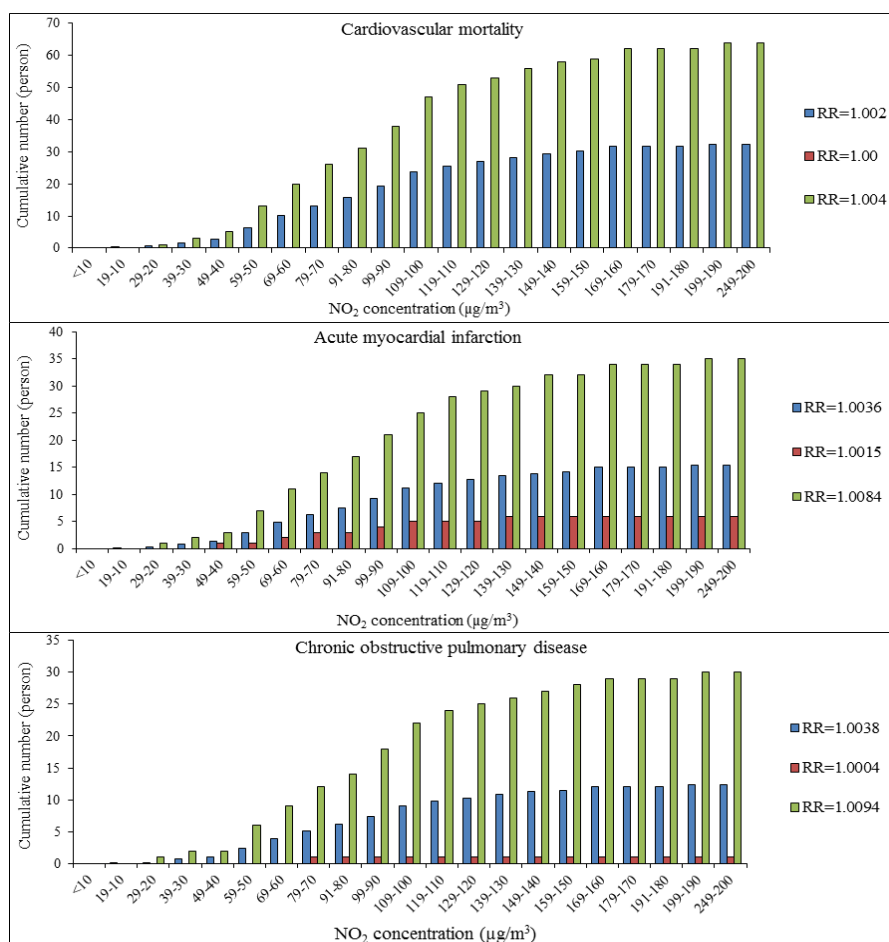[a] Taken from the lower and upper of RR amounts.

Figure 3. Relationship between cumulative number of health effects and NO₂ concentrations

cumulative number of each health outcome and the cumulative number of estimated excess cases at the low, middle, and upper ranges (5%, 50%, and 95%), respectively. In the three curves of RR (associated with upper, central, and lower) in each diagram, the upper curve corresponded to a 95% RR (overestimate), the idle curve related to the central RR, and the lower curve corresponded to a 5% of RR (underestimate). The cumulative numbers of 30.7, 14.6, and 11.7 cases of CM, MI, and HACOPD, respectively, can be attributed to exposure concentrations of NO₂ above 40 µg/m³. The results also showed that about 1.2% (95% CI: 0.00%-2.4%) of CM, 2.2% (95% CI: 0.91%-4.5%) of MI, and 2.3% (0.24%-5.4%) of HACOPD cases were attributed to NO₂ levels over 10 µg/m³. With each increase of 10 µg/m³ in NO₂ level, the risk of CM, MI, and HACOPD rises about 0.2%, 0.36%, and 0.38%. Also, 92.33% of health endpoints of exposure to NO₂ occurred on days with levels over 10 µg/m³. Furthermore, 26.85% of health outcomes happened in days with NO₂ concentrations not exceeding 40 µg/m³.

Discussion

In this study, the method suggested by the WHO for assessing health impacts of atmospheric pollutants was used to investigate the effects of short-term exposure to NO₂ on the health of Kermanshah inhabitants, one of the most densely populated cities of western Iran. For this objective, AirQ2.2.3 software was applied. This tool has been used in several epidemiological studies around the world to evaluate the health effects attributable to NO₂ on mortality and morbidity rates (5,6,14,17,32). Three ranges of RR (lower, central, and upper ranges) were considered for each endpoint based on the default of AirQ software. The annual, summer, and winter averages of NO₂ levels were 75.76, 63.08, and 77.34 µg/m³, respectively. The primary standard for NO₂ published by National Ambient Air Quality Standard (NAAQS) is 100 µg/m³. The current WHO air quality guideline recommends an annual average concentration of 40 µg/m³ for NO₂ exposure. Statistical analysis showed that the annual average of NO₂ in Kermanshah was lower than the value recommended by NAAQS but higher than the WHO guideline. The annual, summer, and winter maximums and the 98th percentile of NO₂ were equal to 198.42, 142.93, 198.42, and 160.78 µg/m³, respectively. These values were determined in a similar study (51.53, 37.29, 66.32, 179.54, 106.01, 179.54, and 130.03 µg/m³) in Ahvaz, Iran (17) and are approximately consistent with the findings of the present study. The annual maximum NO₂ concentration was observed in winter with a value of 198.42 µg/m³, which is higher than the summer maximum level (142.93 µg/m³). This increase can be associated with the use of gas fuel for heating homes

and the stability of the atmosphere during the winter season (17). The maximum percentage of days that people in Kermanshah were exposed to different levels of NO_2 during one year (from March 2014 to March 2015) was detected in the concentration range of 50-59 $\mu g/m^3$. The cumulative number of excess cases for CM, MI, and HACOPD based on corresponding BI values per one hundred thousand people as defined by the WHO, in central RR and estimated AP were estimated to be 1.208%, 2.151%, and 2.271% (33, 16, 13 persons), respectively. The cumulative number of excess cases for CM and MI were also investigated in 2011-2012 in Kermanshah (17) and found to be equal to 44 and 17 persons, respectively. This result shows reductions of about 25% and 5.8% in mortality and morbidity cases due to exposure to NO_2 compared with the findings of the current study. The cumulative number of HACOPD cases caused by exposure to NO_2 in Tabriz, Iran was 11.6 persons (33). This number is lower than the results of the current study. In APs equal to 1.8%, 2.4%, and 2.2%, the cumulative number of CM and respiratory mortality cases in Rezzato, Italy were 2.4 and 2.2 persons due to exposure to NO_2 during one year, respectively (14). NO_2 is most retained in the small airways of the lungs, because it is not highly soluble and thus has a greater airway deposition. It remains a pollutant of current concern. In many experimental studies, there was a relationship between COPD and more sensitive to NO_2 than healthy control subjects (23). In another study in Suwon city, the number of CM in the BI value of 84.5 per 10^5 people and in the centerline RR (RR = 1.004) was estimated to be 23.9 persons (9). That number is lower than the results of the current study. For the population of more than 870 000 in Kermanshah, about 13, 6.2, and 5 cases of mortality due to CM, MI, and HACOPD annually can be attributed to NO_2 levels higher than 100 $\mu g/m^3$. In another study in Tabriz, the rates of mortality and morbidity caused by exposure to NO_2 in a population of one and a half million persons were assessed. The results illustrated that the cumulative numbers of excess cases for mortality because of cardiovascular diseases, MI, and HACOPD were 34.2, 16.3, and 13.2 persons (18), which is consistent with the estimated results of the current study. CM had the lowest RR in the central group (RR = 1.0020) compared with MI (RR = 1.0036) and HACOPD (RR = 1.0038), but a high number of these health effects were because of a higher BI quantity (497 per 10 000 persons). The results of the present study illustrate that 1.2% (95% CI: 0.0%-2.4%) of CM, 2.2% (95% CI: 0.91%-4.5%) of MI, and 2.3% (95% CI: 0.24%-5.4%) of HACOPD were attributed to exposure to NO_2 concentrations higher than 10 $\mu g/m^3$. In an APHEA project in Europe, the short-term impacts of air pollution, such as HACOPD, were evaluated. The RR for a 50 $\mu g/m^3$ increase in the diurnal average of NO_2 concentration was 1.02% (95% CI: 1.00%-1.05%) (34). In another study, an increase in NO_2 levels was associated with an 11% increase in daily hospitalizations for cardiorespiratory diseases (35). In Sao Paulo, Brazil, the atmospheric NO_2 had a significant relationship with increased HACOPD (36). For each 10 $\mu g/m^3$ increase in the NO_2 level, the risk of CM, MI, and HACOPD rose about 0.2%, 0.36%, and 0.38%, respectively. In Bushehr, Iran, each 10 $\mu g/m^3$ increase of NO_2 concentration led to an increased risk of mortality and morbidity cases of about 0.4% in the year 2011-2012 (17). In another study in Tabriz, the results showed that with each 10 $\mu g/m^3$ increase in NO_2 concentration, the risk of HACOPD increased 0.38% during 2008-2009 (37). In Toronto, Canada, study results illustrated that there were 7.7 cases of COPD hospitalization, 40.4% of which were because of exposure to NO_2 (38). The results of this study showed that 7.67% of the health endpoints attributed to NO_2 occurred on days with pollutant levels not exceeding 20 $\mu g/m^3$. In Ahvaz, Iran, 82% of MI cases occurred on days with an atmospheric NO_2 level of less than 110 $\mu g/m^3$ (22). In the present study, 87.12% of MIs occurred in NO_2 levels not exceeding 110 $\mu g/m^3$. In Shiraz, Iran NO_2 had a positive correlation with respiratory deaths in persons aged 18 to 60 years (39). A rapid slope in the NO_2 level range of less than 110 $\mu g/m^3$ is observable. This slope is attributed to the fast increase of the number of mortality and morbidity cases due to exposure to different concentrations of NO_2 in Kermanshah. These recent findings are in agreement with those of 2011-2012 study conducted in Kermanshah (17).

Limitations

In this suggested approach, the study only focused on the health effects of a single pollutant without regarding the simultaneous contact with other contaminants. The interactions between different pollutants are not well evaluated. Moreover, since AirQ is an ecologically-based method and not an epidemiological approach, it does not consider intra-individual differences within the people investigated.

Conclusion

According to the current results, there was a decrease in the cumulative number of mortality and morbidity cases in Kermanshah for 2014-2015 compared with 2011-2012 due to lower NO_2 concentrations or to a decrease in the number of days in which people were exposed to high NO_2 concentrations. The results of this study are in line with those of previous studies; they illustrate this approach, and the software offers an easy and valuable model which is helpful in decision-making. Although the results are consistent with other studies throughout the world, more studies with specific or national RR and BI values based on climate, geographical, and statistical features are required. There is a need to prevent various source emissions, especially automobile exhaust, to reduce NO_2 levels in the atmosphere. To reduce the health endpoints of exposure to NO_2, health training should be performed for the public, especially for vulnerable individuals, to advise them to reduce their activity levels on polluted days. An attempt should be made by governmental bodies to control emissions from various sources, particularly from car exhaust, to reduce the concentration of NO_2 in the atmosphere.

Ethical issues
The authors certify that all data collected during the study is presented in this manuscript, and no data from the study has been or will be published separately.

Competing interests
The authors declare that they have no competing interests.

Authors' contributions
YO performed the literature search and wrote the manuscript. GG conceived and designed the study. SMD and AMH processed the data using the model. All authors critically reviewed, refined, and approved the manuscript.

References
1. Goudarzi G, Geravandi S, Foruozandeh H, Babaei AA, Alavi N, Vosoughi Niri M, et al. Cardiovascular and respiratory mortality attributed to ground-level ozone in Ahvaz, Iran. Environ Monit Assess 2015; 187(8): 1-9
2. Nourmoradi H, Goudarzi G, Daryanoosh SM, Omidi-Khaniabadi F, Jourvand M, Omidi-Khaniabadi Y. Health impacts of particulate matter in air by AirQ model in Khorramabad city, Iran. J Bas Res Med Sci 2015; 2(2): 44-52.
3. Yavari A, Sotoudeh A, Parivar P. Urban Environmental Quality and Landscape Structure in Arid Mountain Environment. Int J Environ Res 2007; 1(4): 325-40.
4. Nourmoradi H, Omidi-khaniabadi y, Goudarzi G, Jourvand M, Nikmehr K. Investigation on the Dust Dispersion (PM10 and PM2.5) by Doroud Cement Plant and Study of Its Individual Exposure Rates. Journal of Ilam University of Medical Sciences 2016; 24(1): 64-75. [In Persian].
5. Gurjar BR, Jain A, Sharma A, Agarwal A, Gupta P, Nagpure AS, et al. Human health risks in megacities due to air pollution. Atmos Environ (1994) 2010; 44(36): 4606-13.
6. Gharehchahi E, Mahvi AH, Amini H, Nabizadeh R, Akhlaghi AA, Shamsipour M, et al. Health impact assessment of air pollution in Shiraz, Iran: a two-part study. Journal of Environmental Health Sciences and Engineering 2013; 11(11): 2-8.
7. Zhou M, Liu Y, Wang L, Kuang X, Xu X, Kan H. Particulate air pollution and mortality in a cohort of Chinese men. Environ Pollut 2014; 186: 1-6.
8. Daryanoosh SM, Goudarzi G, Khaniabadi YO, Armin H, Bassiri H, Khaniabadi FO. Effect of exposure to PM$_{10}$ on cardiovascular diseases hospitalizations in Ahvaz, Khorramabad and Ilam, Iran during 2014. Iranian Journal of Health, Safety & Environment 2016; 3(1): 428-33.
9. Jeong S. The Impact of air pollution on human health in Suwon city. Asian J Atmos Environ 2013; 7(4): 227-33.
10. Nourmoradi H, Khaniabadi YO, Goudarzi G, Daryanoosh SM, Khoshgoftar M, Omidi F, et al. Air quality and health risks associated with exposure to particulate matter: a cross-sectional study in Khorramabad, Iran. Health Scope 2016; 5(2): e31766.
11. Raaschou-Nielsen O, Andersen ZJ, Jensen SS, Ketzel M, Sørensen M, Hansen J, et al. Traffic air pollution and mortality from cardiovascular disease and all causes: a Danish cohort study. Environ Health 2012; 11: 60.
12. Borge R, Paz Ddl, Lumbreras J, Pérez J, Vedrenne M. Analysis of contributions to NO$_2$ ambient air quality levels in Madrid city (Spain) through modeling. Implications for the development of policies and air quality monitoring. Journal of Geoscience and Environment Protection 2014; 2(1): 6-11.
13. Health effects of outdoor air pollution. Committee of the Environmental and Occupational Health Assembly of the American Thoracic Society. Am J Respir Crit Care Med 1996; 153(1): 3-50.
14. Fattore E, Paiano V, Borgini A, Tittarelli A, Bertoldi M, Crosignani P, et al. Human health risk in relation to air quality in two municipalities in an industrialized area of Northern Italy. Environ Res 2011; 111(8): 1321-7.
15. Ghiyathoddin M. Air pollution -sources, effectsand control. Tehran: Tehran University Press; 2006. [In Persian].
16. Najafpoor AA, Hosseinzadeh A, Allahyari S, Javid AB, Esmaily H. Modeling of CO and NOx produced by vehicles in Mashhad, 2012. Environmental Health Engineering and Management Journal 2014; 1(1): 45-9.
17. Zallaghi E, Goudarzi G, Haddad MN, Moosavian SM, Mohammadi MJ. Assessing the effects of nitrogen dioxide in urban air on health of west and southwest cities of Iran. Jundishapur J Health Sci 2014; 6(4): e23469.
18. Ghozikali MG, Borgini A, Tittarelli A, Amrane A, Naddafi K, Mohammadyan M, et al. Quantification of the health effects of exposure to air pollution (NO2) in Tabriz, Iran. Fresenius Environmental Bulletin 2015; 24(11): 4142-8.
19. Williams M, Atkinson R, Anderson H, Kelly F. Associations between daily mortality in London and combined oxidant capacity, ozone and nitrogen dioxide. Air Qual Atmos Health 2014; 7(4): 407-14.
20. Jenkins HS, Devalia JL, Mister RL, Bevan AM, Rusznak C, Davies RJ. The effect of exposure to ozone and nitrogen dioxide on the airway response of atopic asthmatics to inhaled allergen: dose- and time-dependent effects. Am J Respir Crit Care Med 1999; 160(1): 33-9.
21. Environmental Protection Agency. Integrated Science Assessment for Oxides of Nitrogen - Health Criteria (Second External Review Draft). USEPA; 2015.
22. Goudarzi G, Mohammadi MJ, Angali KA, Neisi AK, Babaei AA, Mohammadi M, et al. Estimation of health effects attributed to NO2 exposure from the use of AirQ model in Ahvaz. Arch Hyg Sci 2012; 1(2): 59-66.
23. Yang Q, Chen Y, Krewski D, Burnett R, Shi Y, McGrail K. Effect of short-term exposure to low levels of gaseous pollutants on chronic obstructive pulmonary disease hospitalizations. Environ Res 2005; 99(1): 99-105.
24. Touloumi G, Katsouyanni K, Zmirou D, Schwartz J, Spix C, de Leon AP, et al. Short-term effects of ambient oxidant exposure on mortality: a combined analysis within the APHEA project. Air pollution and health: a European approach. Am J Epidemiol 1997; 146(2): 177-85.
25. Stieb DM, Judek S, Burnett RT. Meta-analysis of time-series studies of air pollution and mortality: update in relation to the use of generalized additive models. J Air Waste Manag Assoc 2003; 53(3): 258-61.
26. Yang C, Chen Y, Yang C, Ho S. Relationship between ambient

air pollution and hospital admissions for cardiovascular diseases in Kaohsiung, Taiwan. J Toxicol Environ Health A 2004; 67(6): 483-93.

27. Marzouni MB, Alizadeh T, Banafsheh MR, Khorshiddoust AM, Ghozikali MG, Akbaripoor S, et al. A comparison of health impacts assessment for PM_{10} during two successive years in the ambient air of Kermanshah, Iran. Atmos Pollut Res 2016; In press. doi: 10.1016/j.apr.2016.04.004.

28. Akbary M, Farahbakhshi M. Analyzing and Tracing of Dust Hazard in Recent Years in Kermanshah Province. Int J Environ Res 2015; 9(2): 673-82

29. Poloniecki JD, Atkinson RW, de Leon AP, Anderson HR. Daily time-series for cardiovascular hospital admissions and previous day s air pollution in London, UK. Occup Environ Med 1997; 54(8): 535-40.

30. Burnett RT, Dales RE, Brook JR, Raizenne ME, Krewski D. Association between ambient carbon monoxide levels and hospitalization for congestive heart failure in the elderly in 10 Canadian cities. Epidemiology 1997; 8(2): 162-7.

31. Moustris KP, Proias GT, Larissi IK, Nastos PT, Koukouletsos KV, Paliatsos AG. Health impacts due to particulate air pollution in Volos city, Greece. J Environ Sci Health A Tox Hazard Subst Environ Eng 2016; 51(1): 15-20.

32. Mohammadi A, Azhdarpoor A, Shahsavani A, Tabatabaee H. Investigating the health effects of exposure to criteria pollutants using AirQ2.2.3 in Shiraz, Iran. Aerosol and Air Quality Research 2016; 16(4): 1035-43.

33. Ghanbari Ghozikali M, Heibati B, Naddafi K, Kloog I, Oliveri Conti G, Polosa R, et al. Evaluation of chronic obstructive pulmonary disease (COPD) attributed to atmospheric O3, NO2, and SO2 using AirQ model (2011–2012 year). Environ Res 2016; 144(Pt A): 99-105.

34. Anderson HR, Spix C, Medina S, Schouten JP, Castellsague J, Rossi G, et al. Air pollution and daily admissions for chronic obstructive pulmonary disease in 6 European cities: results from the APHEA project. Eur Respir J 1997; 10(5): 1064-71.

35. Burnett RT, Cakmak S, Brook JR, Krewski D. The role of particulate size and chemistry in the association between summertime ambient air pollution and hospitalization for cardiorespiratory diseases. Environ Health Perspect 1997; 105(6): 614-20.

36. Gouveia N, de Freitas CU, Martins LC, Marcilio IO. Respiratory and cardiovascular hospitalizations associated with air pollution in the city of Sao Paulo, Brazil. Cad Saude Publica 2006; 22(12): 2669-77. [In Portuguese].

37. Ghozikali MG, Mosaferi M, Safari GH, Jaafari J. Effect of exposure to O_3, NO_2, and SO_2 on chronic obstructive pulmonary disease hospitalizations in Tabriz, Iran. Environ Sci Pollut Res Int 2015; 22(4): 2817-23.

38. 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.

39. Dadbakhsh M, Khanjani N, Bahrampour A. Death from respiratory diseases and air pollutants in Shiraz, Iran (2006-2012). Journal of Environment Pollution and Human Health 2015; 1(3): 4-11.