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RESEARCH ARTICLE

Asthma disease as cause of admission to hospitals due to exposure to ambient oxidants in Mashhad, Iran

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Abstract Nowadays, asthma is one of the most common chronic respiratory diseases, worldwide. Many reports have emphasized the correlation between the short-term exposure to the ambient air pollutants and acute respiratory diseases, especially among children with asthmatic symptoms. The aim of this study was to evaluate the relationship between the exposure to three atmospheric antioxidants (NO_2, SO_2, and O_3) and hospital admission due to asthmatic disease (HAAD) in the city of Mashhad, Iran. The concentrations of atmospheric antioxidants were obtained from the real-time monitoring stations located in the city. The collected data were employed for developing predictive models in the AirQ software. In order to investigate the association between short-term exposure to air pollutants and HAAD, the study participants were categorized into two age groups: less than 15 and from 15 to 64 years old. The results indicated that in people less than 15 years increase in NO_2 (attributable proportion (AP) = 3.775%, 95% CI 0.897–6.883%), SO_2 (AP = 3.649%, 95% CI 1.295–5.937%), and O_3 (AP = 0.554%,95% CI 0.00–3.321) results in increase in HAAD. While for those aged between 15 and 64 years, the AP was 4.192% (95% CI 0.450–7.662%) for NO_2; 0.0% (95% CI 0.00–1.687%) for SO_2; and 0.236% (95% CI 0.00–1.216%) for O_3. The number of asthmatic cases who were less than 15 years admitted to the hospitals during the study period was higher than that of those within the age groups between 15 and 64 years as a consequence of exposure to NO_2 (101 vs. 75), SO_2 (98 vs. 0), and O_3 (15 vs. 3), respectively. To the best of our knowledge, the AirQ model has not been applied before to estimate the effect of atmospheric antioxidant exposure on hospital admission because of asthma disease. Eventually, this model is proposed to be applicable for other cities around the world.

Keywords Asthma syndromes · Admission to hospitals · Air oxidant pollutants · AirQ software · Mashhad · Iran

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Introduction

Asthma is the most common chronic respiratory disease influencing 5 to 18% of people worldwide, especially children, and characterized by recurrent attacks of breathlessness and wheezing, which vary in severity and frequency from person to person (Bousquet et al. 2007; To et al. 2013). Asthmatic symptoms may occur several times in a day or week in affected

individuals and, for some people, become worse during physical activity or at night. During an asthma attack, the lining of the bronchial tubes swells, causing the airways to narrow and reducing the flow of air into and out of the lungs. Recurrent asthma symptoms frequently cause sleeplessness, daytime fatigue, reduced activity levels, and school and work absenteeism. Asthma has a relatively low fatality rate compared to other chronic diseases (Health and Organization 2008). Previous studies have emphasized a correlation between short-term exposure to the ambient air pollutants such as particulate matters ($PM_{2.5}$ and PM_{10}), ozone (O_3), and nitrogen dioxide (NO_2) and acute respiratory syndromes embodied particularly in children with asthma (Trasande and Thurston 2005). Moreover, previous reports showed a constantly increasing trend in the outpatient visits and hospitalizations due to incidence of asthma, possibly attributable to the ambient air pollutant exposure (Riedl and Diaz-Sanchez 2005). Numerous studies introduced possible sources of air pollutants which can create asthma, for instance, fossil fuel combustion may cause airway inflammation through oxidative stress mechanisms (Riedl and Diaz-Sanchez 2005).

Also, the organic and nonorganic pollutants at high concentrations, especially those located on ultrafine particles (< 0.1 μm), are expected to intensify asthma symptoms (Ntziachristos et al. 2007), (Knol et al. 2009; McCreanor et al. 2007) and reduction of lung function (McCreanor et al. 2007). High traffic density in proximity to homes and schools cannot be ignored as a risk factor for incidence and prevalence of asthma (Delfino et al. 2009; Salam et al. 2008). However, in Iran like the rest of the world, outdoor airborne pollutants are emitted from a wide variety of mobile and stationary sources listed as follows but the list is not exhaustive: motor vehicles, industries, home heating sources, and bushfires (Miri et al. 2016a, c). Natural phenomena can also have a role in the creation of asthma, since dust storms with associated drought can occasionally cause extreme air pollution events (Khaniabadi et al. 2017; Maleki et al. 2016; Miri et al. 2017; Nikoonahad et al. 2017). Exposure to aeroallergens such as pollen has also been reported to be a major trigger of asthma exacerbations (Murray et al. 2006).

Nitrogen dioxide (NO_2) and sulfur dioxide (SO_2) are classified as primary criteria air pollutants while ozone (O_3) is classified as secondary. Nitrogen dioxide (NO_2) is acidic gas and forms via oxidation processes in which released nitrogen oxide reacts with atmospheric oxidizers such as O_3. The major sources of NO_2 are burning of organic fuels in road vehicles, heaters, and industrial activities. Sulfur dioxide (SO_2) is a highly reactive corrosive gas and mainly emitted from fossil fuel combustion in mining and industrial facilities. NO_2 and SO_2 are associated with adverse effects on respiratory health (USEPA 2016).

The WHO European Center for Environment and Health has proposed the AirQ software model as a valid and reliable tool to estimate the potential health effects of air pollution by assigning score points for each criteria pollutant and to enable assessment of scenarios characterized by varied pollutants (Conti et al. 2017). AirQ software is a Windows software that collects, manages, and displays results from air quality information data and noise levels. It was designed to calculate the magnitude of the impacts of air pollution on health in a given population level (Conti et al. 2017). Similar studies were applied this model to assess health effect of air pollutants in other Iranian cities (Miri et al. 2016b, 2017; Mokhtari et al. 2015; Nikoonahad et al. 2017) and Italy (Fattore et al. 2011; Ferrante et al. 2012).

Therefore, there is a dire need to evaluate HAAD due to exposure to atmospheric NO_2, SO_2, and O_3 in Mashhad as the largest city northeast of Iran with more than 2,700,000 inhabitants, using the AirQ software, developed by the WHO European Center for Environment and Health management.

Material and methods

Study area

The city of Mashhad is considered as the capital of the province Khorasan Razavi and is the second megacity in Iran. It lies in longitude 59°35′E to 59°74′E and latitude 36°14′N to 36°48′N. According to the 2016 census, its population was 2,766,258 inhabitants (Statistical Centre of Iran 2015). Generally, Mashhad has a young population with 669,434 individuals less than 15 years old, 1,609,962 inhabitants within the age group between 15 and 64 years old, and 763,487 individuals are more than 64 years old. The geographical locations of Mashhad and its air pollution monitoring stations are shown in Fig. 1.

AirQ software and exposure assessment

AirQ software 2.2.3 is a tool for health risk assessment of air pollution in different areas within a specified period developed by the WHO European Center. In this software, assessment is conducted based on the attributable proportion (AP). AP refers to the fraction of the health effect in a well-defined population which can be related. Two assumptions are considered in AP application. The first one assumes a demonstrated causal relation between exposure and health effect, while the second one proposes that no major confounding effects are considered in this association. AP is calculated using the following equation:

$$AP = \left(\sum\{[RR(c)-1] \times P(c)\} \right)/\left(\sum[RR(c) \times P(c)]\} \right) \quad (1)$$

Where, RR is a relative risk for a given health endpoint in category "c" of exposure obtained from the exposure-response functions derived from epidemiological study, and
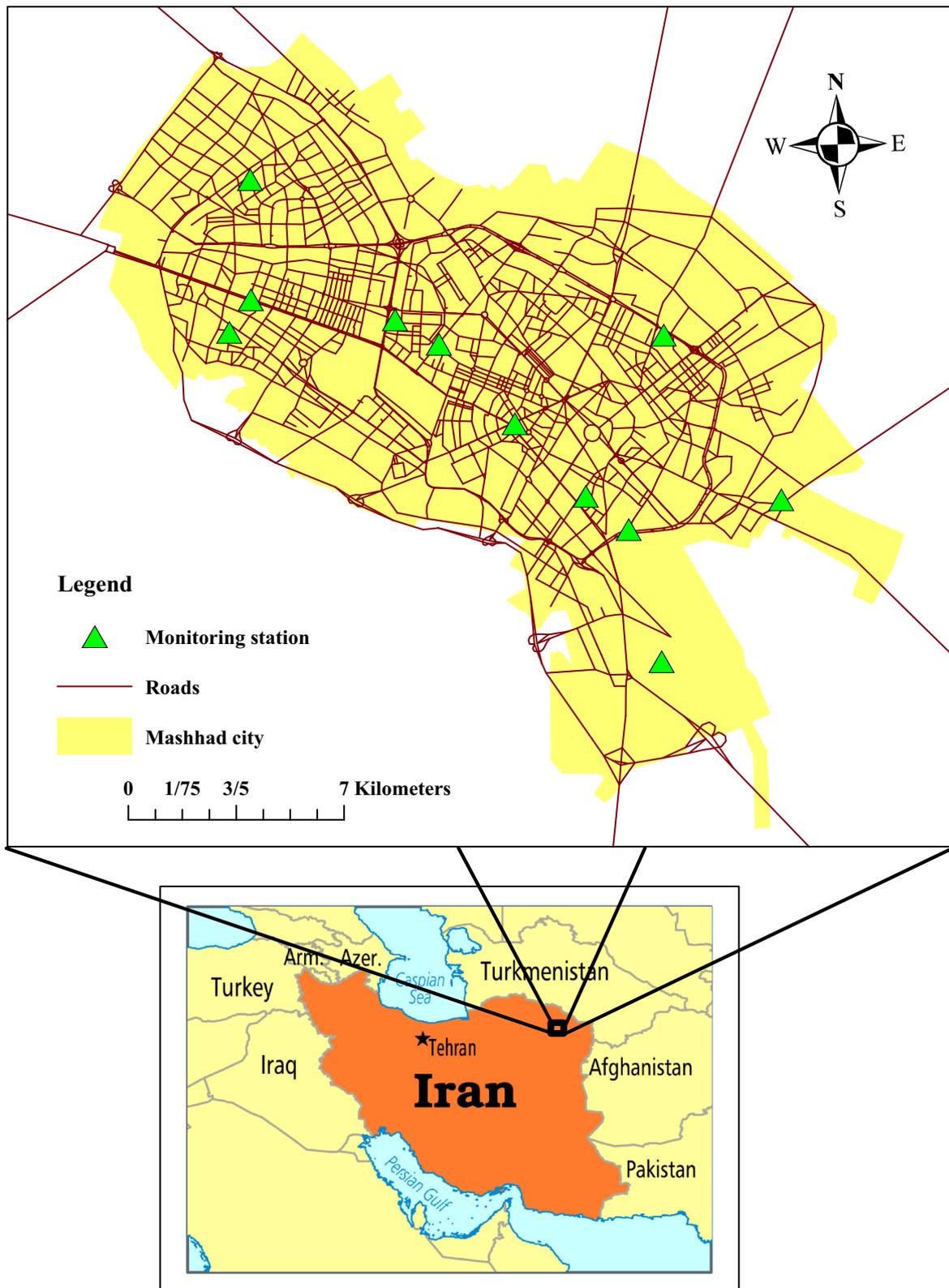
Fig. 1 Study area and air pollution monitoring stations

P (c) represents the proportion of the population in category c of exposure.

If the baseline frequency of the health effect is ascertained, the rate attributed to the exposure can be calculated as follows:

$$IE = 1 \times AP \qquad (2)$$

Where, IE is the amount of health effect attributable to the exposure, and I is the baseline frequency of the health effect in the investigated population. Finally, the number of cases attributable to the exposure can be estimated as follows, if the size of the population is known:

$$NE = IE \times N \qquad (3)$$

Where, NE implies the number of cases attributed to the exposure and N implies the size of the investigated population (Gharehchahi et al. 2013; Hassanvand et al. 2013; Naddafi et al. 2012).

RR reflects the rate of the impact on the health as exposure to air pollutants is changeable. RR is the finding of time series studies in which the concentration changes of air pollutants and paralleled health effects changes are evaluated during a long period of time (Fattore et al. 2011).

Based on our knowledge, there has been no time series survey carried out in Iran yet. Hence, the WHO default data available by AirQ software were used in this study.

The amounts of RR for NO_2, SO_2, and O_3 were obtained from previous performed studies in the APHEA project (Touloumi et al. 1996).

The main drawback of RR collected from previous studies conducted in other regions is an increase of the model prediction error. However, it remains possible that valuable information provided about the impacts of air pollutants in the study area help policy makers and stockholders in order to mitigate the possible health effects and to apply necessary management.

For the baseline incidence (BI) rate default, AirQ software was used for hospital admissions asthma < 15 years and hospital admissions asthma > 15–64 years investigated in this study.

Air pollution data and exposure assessment

There are 12 air pollution monitoring stations in Mashhad City under the management of the local Environmental Protection Agency. In the present study, a 1-year period of air pollution monitoring data from March 2014 to March 2015 was obtained from the Environmental Protection Agency. In order to prevent invalid evaluation of health effects, WHO exclusion criteria were applied to exclude some cases out of the data taken from the stations. Finally, stations 9, 7, and 4 remained to use their data for NO_2, SO_2, and O_3, respectively. The remained stations cover all areas of the city and were active during the study period. To consider temperature and pressure in the study area, air pollution data conversion from parts per billion to micrograms per cubic meter was conducted and after classification at 10 μ/m^3 intervals entered into the AirQ software. The exposure days for each of the pollutants at specified intervals as well as the annual and seasonal mean, maximum, and 98th percentile of pollutants were calculated and inserted into the software. The daily average was used for the NO_2, SO_2, and 1-h average "moving average" was utilized for O_3.

Result and discussion

The descriptive statistical analysis of NO_2, SO_2, and O_3 concentrations is shown in Table 1. In summary, the annual mean and maximum concentrations of NO_2 were 87.09 and 911.79 $\mu g/m^3$, which were more than those of the WHO guideline (40 $\mu g/m^3$) (WHO 2006). The annual mean and maximum concentrations of SO_2 were 73.86 and 120.54 $\mu g/m^3$ which were 3.69 and 6.02 times more than those of the WHO guideline (20 $\mu g/m^3$) (WHO 2006). The most probable explanations for high concentrations of these pollutants were heavy vehicle traffic and the many industrial facilities around the study area. The annual 1-h average concentration of O_3 in

Table 1 Summarized data of oxidant pollutant in Mashhad in years 2014–2015 entered to AirQ software

	NO_2 (annual average 24 h)			SO_2 (annual average 24 h)			O_3 (annual average 1 h)		
	Station low	Station high	Average	Station low	Station high	Average	Station low	Station high	Average
Annual mean	32.8	315.3	87.09	22.2	407.0	73.86	24.2	85.8	56.40
Winter mean	47.1	72.6	68.35	21.0	843.0	109.91	21.6	85.6	48.10
Summerr mean	21.2	551.7	109.11	23.4	31.8	38.41	27.6	89.0	66.65
Percentile 98th	128.6	3152.3	473.09	44.7	71.2	89.44	41.5	209.4	91.89
Annual maximum	169.2	5184.7	911.79	90.5	150.54	120.54	99.8	517.3	153.65
Winter maximum	166.0	167.4	157.81	90.5	150.54	120.54	98.0	507.7	153.65
Summer maximum	36.2	5082.9	911.79	50.9	72.8	98.89	55.5	139.6	103.25

Table 2 Baseline incidence, relative risk (RR), estimated AP expressed as percentage, and number of excess cases in a year attributed to exposure above 10 $\mu g/m^3$ for NO_2, SO_2, and O_3

Health endpoints	BI[a]	RR (95% CI) per 10 $\mu g/m^3$	Pollutant	AP (uncertainty range)[b]	No. of excess cases (uncertainty range)[b]
Hospital admissions asthma < 15 years	100	1.0052 (1.0012–1.0098)	NO_2	3.775 (0.897–6.883)	101(24–185)
	100	1.015 (1.0052–1.025)	SO_2	3.649 (1.295–5.937)	98(35–160)
	100	1.0012 (1–1.0074)	O_3	0.554 (0.00–3.321)	15(0–90)
Hospital admissions asthma > 15–64 years	66	1.0058 (1.0006–1.011)	NO_2	4.192 (0.450–7.662)	75 (8–137)
	66	1 (1–1.0068)	SO_2	0.00 (0.00–1.687)	0 (0–30)
	66	1.003 (1–1.0156)	O_3	0.236 (0.00–1.216)	3 (0–15)

[a] $_a$Raw rate per 100,000 inhabitants

[b] Obtained using the lower and upper RR values

summer was more than that in winter and less than WHO guideline (100 $\mu g/m^3$) (WHO 2006).

Table 2 presents BI, RR from the APHEA project (Touloumi et al. 1996), and the result of hospital admission asthma disease (HAAD) estimated by the AirQ model. AP for HAAD < 15 years old attributed to NO_2, SO_2, and O_3 were 3.775% (95% CI 0.897–6.883%), 3.649% (95% CI 1.295–5.937%), and 0.554% (95% CI 0.00–3.321%), respectively. Also, AP for HAAD of 15–64 years were 4.192% (95% CI 0.450–7.662%), 0.00% (95% CI 0.00–1.687%), and 0.236% (95% CI 0.00–1.216%) were related to NO_2, SO_2, and O_3 with concentrations over 10 $\mu g/m^3$, respectively. The number of excess cases for HAAD <15 years were 101 cases for NO_2, 98 cases for SO_2, and 15 cases for O_3. According to these data, the number of HAAD related to NO_2 is more than that to other oxidant pollutants, and the effect of this pollutant on age group less than 15 years old is more than that on age group of 15–64 years old (Table 2). This result may be explained by the fact that the children and teenagers are more susceptible to inflammatory markers and oxidative stress related to air pollution and, therefore, the risk of asthma is higher in this age group (Delfino et al. 2014). Clark et al. (2010) demonstrated that as the level of exposure to ambient air pollution in early life increased, higher risks of asthma can be observed. In their study, NO_2 was the most important pollutant associated with risk estimation. They also reported that SO_2 in industrial areas was associated with asthma risk. Many other studies also indicated that traffic-related air pollution and especially NO_2 have adverse effect on asthma disease (Brauer et al. 2007; Morgenstern et al. 2008; Mortimer et al. 2008; Strickland et al. 2010).

Chen et al. (2016) studied the impact of air pollution on HAAD in a time series analysis and reported that the largest effect associated with ozone was an 11.7% (95% CI 5.8–17.9%) increase in the risk of HAAD for children per 10 ppb increase in O_3 concentration. The other obtained finding was 12.5% (95% CI 6.6–18.7%) increase in risk of HAAD per 10 ppb increase in NO_2 concentration based on a 5-day cumulative effect. In a meta-analysis study by Ji et al. (2011) with 96 peer-reviewed published articles from 1990 to 2008 reported

significant associations with ozone observed for HAAD for combined age groups, whereas no significant association was found between HAAD and ozone levels for children or adults (15–64 years) separately. Figure 2 indicates the cumulative number of excess cases in the central relative risk value. As we can see in this figure, for HAAD < 15 years, increase in concentration of SO_2 until range of 60–69 $\mu g/m^3$ resulted in increase in the cumulative number of hospitalizations, but no increase in hospitalization was found for a concentration more than this range. The main reason is that the number of days with concentration more than this range is just 1 day. For NO_2, the rate for HAAD < 15 years and HAAD > 15–64 years was uniform but in concentration higher than 350 $\mu g/m^3$, sudden increase in HAAD was observed.

The RR value indicates increasing risk due to exposure to a pollutant. In the present study, increase in RR for HAAD < 15 years was associated with increase in NO_2 (0.52% per 10 $\mu g/m^3$), SO_2 (1.5% per 10 $\mu g/m^3$), and O_3 (0.12% per 10 $\mu g/m^3$). For HAAD > 15–46 years, these increases were 0.58% and 0 and 0.3%; with regard to RR values, it can be concluded that SO_2 has the highest impact on HAAD < 15 years.

Compared with $SO2$, the bigger number of hospitalizations due to NO_2 can be explained by a higher concentration of this pollutant in the study area. The main source for NO_2 is heavy traffic which can be seen most of the time throughout the study area (Delfino et al. 2014; WHO 2006).

Another study in Mashhad on human health effect related to these air pollutants reported that 0.99% of the total mortality was attributed to SO_2 (130 cases), 2.21% was attributed to NO_2 (290 cases), and 2.09% was attributed to O_3 (274 cases). Also, hospital admissions due to chronic obstructive pulmonary disease (CODP) attributable to O_3, NO_2, and SO_2 exposure in a year were 79, 58, and 33 cases, respectively (Miri et al. 2016b).

The major limitation with the AirQ software model was the difficulty in estimation of the health effects caused by mixture of pollutants, and their synergistic effects cannot be taken into account. This limitation can be interpreted as a lack of knowledge about the toxicity mechanism of the different compounds. In this

Fig. 2 Relationship between oxidant pollutant concentrations and cumulative number of HAAD < 15 years (**a**) and HADD > 15–64 years (**b**) in central RR
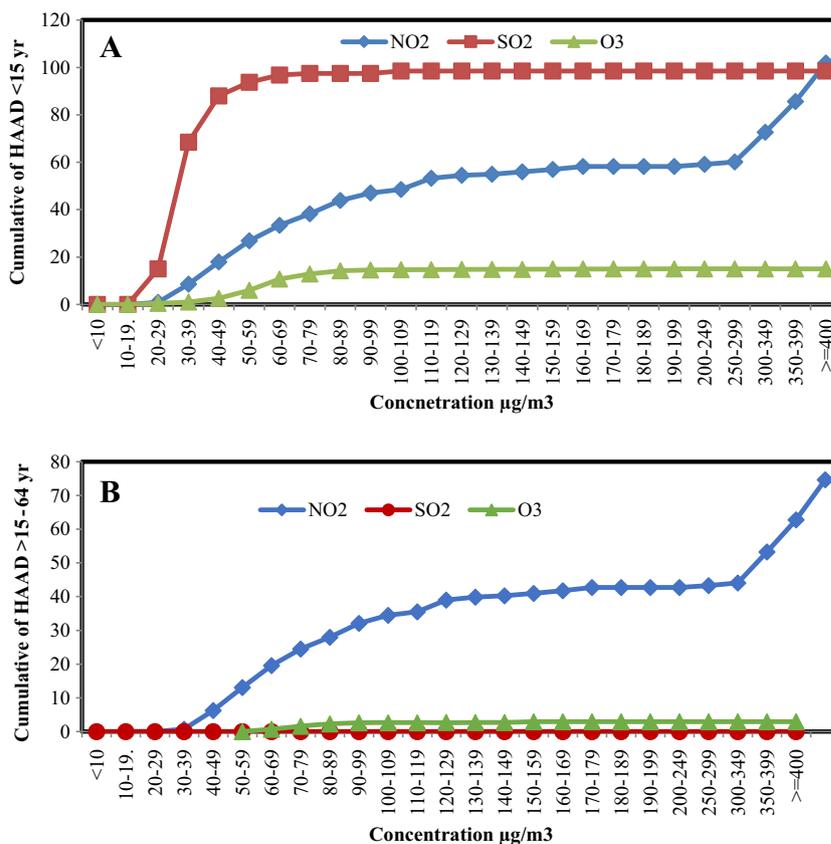
case, it is often assumed that coexposure to different pollutants results in the additive effect compared to the imposed effect by individual pollutants.

The other possible source of error in this study was the stationary monitoring stations which may lead to overestimation of exposure for inhabitants far from the stations. The available solution for this challenge was using the mobile monitoring stations. The last supposed deficiency is owed to a lack of information regarding RR in the study area and inevitably using RR obtained in European countries and the USA. Since RR can be different according to characteristics of a population, so the prediction error can be increased (WHO 2001).

Conclusion

To the best of our knowledge, this is the first study that evaluates the impact of air pollution related to oxidant HAAD using the AirQ model. The results showed that the annual mean concentrations of NO_2 and SO_2 were more than WHO guideline. We found that for every 10 $\mu g/m^3$ increase in pollutant concentration, there was the highest increase in RR in relation to SO_2 and this pollutant had the most effect on HAAD < 15 years in the study area. The most cumulative number of excess cases was attributed to the NO_2 pollutant. Our result suggested that the government and decision makers

should apply management programs such as public transportation development and fossil fuel substitution to reduce air pollutants as much as possible. Although there are some limitations with the AirQ model, this model is a very useful and simple model to investigate health impact due to exposure to air pollution, and it can help decision makers to organize future health programs in metropolitan cities all over the world. Finally, although some studies have used this model to estimate health impacts due to exposure to air pollution, we also suggest this model for evaluation of HAAD.

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