Cardiopulmonary mortality and COPD attributed to ambient ozone

Yusef Omidi Khaniabadi, Philip K. Hopke, Gholamreza Goudarzi, Seyed Mohammad Daryanoosh, Mehdi Jourvand, Hassan Basiri

A R T I C L E   I N F O

Keywords: Health impact, Mortality, COPD, Ozone, Kermanshah

A B S T R A C T

Tropospheric ozone is the second most important atmospheric pollutant after particulate matter with respect to its impact on human health and is increasing of its concentrations globally. The main objective of this study was to assess of health effects attributable to ground-level ozone (O₃) in Kermanshah, Iran using one-hour O₃ concentrations measured between March 2014 and March 2015. The AirQ program was applied for estimation of the numbers of cardiovascular mortality (CM), respiratory mortality (RM), and hospital admissions for chronic obstructive pulmonary disease (HA-COPD) using relative risk (RR) and baseline incidence (BI) as defined by the World Health Organization (WHO). The largest percentage of person-days for different O₃ concentrations was in the concentration range of 30–39 µg/m³. The health modeling results suggested that ~2% (95% CI: 0–2.9%) of cardiovascular mortality, 5.9% (95% CI: 2.3–9.4) of respiratory mortality, and 4.1% (CI: 2.5–6.1%) of the HA-COPD were attributed to O₃ concentrations higher than 10 µg/m³. For each 10 µg/m³ increase in O₃ concentration, the risk of cardiovascular mortality, respiratory mortality, and HA-COPD increased by 0.40%, 1.25%, and 0.86%, respectively. Furthermore, 88.8% of health effects occurred on days with O₃ level less than 100 µg/m³. Thus, action is needed to reduce the emissions of O₃ precursors especially transport and energy production in Kermanshah.

1. Introduction

Air pollution is one of the most important of the environmental problems in the world, especially in developing countries (Nourmoradi et al., 2015; Tominz et al., 2005; Wang et al., 2009). Ambient air pollution and indoor air pollutants are serious threats in which exposure to ozone (O₃), nitrogen dioxide (NO₂), sulfur dioxide (SO₂), carbon monoxide (CO), lead (Pb), particulate matter (PM), and air toxins can cause both the acute and chronic health effects (Kelly, 2003; Neisi et al., 2016; Sousa et al., 2008; Taiwo et al., 2014). According to the World Health Organization (WHO), approximately 800,000 deaths per year globally occur due to the respiratory and cardiovascular disease, and lung cancer caused by the air pollution (Nourmoradi et al., 2016; Zarandi et al., 2015). Many time-series and case-crossover studies have been performed on the short and long-term impacts of air pollution and found increased hospital admissions due to adverse cardiovascular and respiratory effects, acute asthma attacks, reduced longevity, and mortality (Bayram et al., 2001; Shahsavani et al., 2012).

Dust is the major environmental issue in southwestern of Iran including Kermanshah, Ahvaz, and Ilam. Many studies on dust properties and its impact on human and experimental rats have been well documented (Dianat et al, 2016a; Dianat et al, 2016b; Goudarzi et al, 2014; Maleki et al, 2016; Naimabadi et al, 2016; Radmanesh et al, 2016; Rezaei et al, 2014; Soleimani et al, 2015; Soleimani et al, 2016). Although the MED storms are the main concern in this part of Iran, there is a scarcity of investigations about gaseous pollutants such as carbon monoxide, nitrogen dioxide, sulfur dioxide, and tropospheric ozone. Tropospheric O₃ is the second most important air pollutant after PM and its atmospheric concentration continues to rise (Dehghani et al., 2014). Relationships have been observed between O₃ exposure and pulmonary function (Andersen et al., 2011; Ghozikali et al, 2016; Gryparis et al., 2004). Epidemiological studies showed that the exposure in the range of 160–360 µg/m³ O₃ levels for a duration of 1–8 h may reduce lung function (Zarandi et al., 2015). Ground level ozone disrupts photosynthesis in plants and damages crop yields (Van Dingenen et al., 2009). Ozone has an important role in the climate...
change, global warming, atmospheric chemistry, and air quality deterioration (Chen and Hopke, 2009; Goudarzi et al., 2015; Mohnen et al., 1993). The concentration of tropospheric O3 is driven by the concentrations of its precursors. The precursors’ concentrations depend on the anthropogenic and biogenic sources (Dadbakhsh et al., 2015; Ohara et al., 2007).

Kermanshah, the capital city of Kermanshah Province, is the second largest city in western Iran with a population in excess of 870,000 persons. It is among the most polluted cities of Iran because of the presence of several major industrial factories and a high number of automobiles (Akbary and Farahbakhshi, 2015). The main objective of the present study was to investigate the health impacts of exposure to ground level O3 in Kermanshah.

2. Materials and methods

2.1. Study area

This study was conducted in Kermanshah (34°19′N, 47°3′E), the capital city of Kermanshah Province in Iran (Fig. 1). It is 1312 m above sea level and is located in a cold, semi-arid climate with the annual average precipitation and temperature of 447.7 mm and 14.3 °C, respectively. The vegetative cover is steppe grasslands with few trees. The temperature may drop to 10 °C in the winter and can rise to 44 °C in the summer (Omidi et al., 2016). The Kermanshah’s Environmental Protection Agency (KEPA) operates an air monitoring station that measures O3 concentrations along with other criteria pollutants. An ozone monitor (model 400 A, Mansouri and Hamidian, 2013) was used for continuous measuring of O3 and the values were reported as hourly

Fig. 1. Location of Kermanshah and sampling station (Marzouni, M.B., et al., 2016).
values. These hourly O₃ data were available from KEPA for exposure estimates in the evaluation of health endpoints.

2.2. AirQ2.2.3 software

The Air Quality Health Impact Assessment (AirQ2.2.3 model) is a program developed by WHO to assess the health outcomes of air pollutants (Nourmoradi et al., 2015; Goudarzi et al., 2016; Shahsavani et al., 2012). The health impacts assessment model combines air quality data with epidemiological information such as relative risk (RR), attributed proportion (AP), and baseline incidence (BI) to estimate the health endpoint rates attributable to that air pollutant. The results of the model are mortality and morbidity estimates (Fattore et al., 2011).

In this study, AirQ2.2.3 (http://www.euro.who.int/en/health-topics/environment-and-health/air-quality/activities/airq-software-tool-for-health-risk-assessment-of-air-pollution) was applied (Zhou et al., 2014). The assessment is based on the attributable proportion that is identified as the portion of the health effects in a particular population attributable to a given air pollutant (Nourmoradi et al., 2015; Shakour et al., 2011). The AP value can be calculated by using Eq. (1):

\[ AP = \sum \left[ \frac{\left[ RR \times PE \right] / \sum \left[ P(c) \right]}{\sum \left[ P(c) \right]} \right] \]  

where AP is the attributable proportion of the health impacts, RR is the relative risk for a particular health effect for people in a given exposure category and can be obtained from the functions of exposure-response. \( P(c) \) is the population of the exposed group in each exposure category (Lawrence et al., 2007; Nourmoradi et al., 2015). The quantity attributable to the population exposure can be estimated by Eq. (2), if the baseline incidence of the health impact in the population under study is known (Nourmoradi et al., 2015).

\[ IE = I \times AP \]  

where IE is the frequency of exposure within a given concentration range and I is the baseline frequency of the given health effect in the studied population (Fattore et al., 2011). If the population size is known, the number of excess cases attributable to exposure can be calculated by the following equation:

\[ NE = IE \times N \]  

where NE and N are the number of cases attributable to exposure and the population size under study, respectively (Daryanoosh et al., 2016a, 2016b; Goudarzi et al., 2010).

2.3. Input adjustment

In order to assess of health impacts of O₃ by AirQ2.2.3 tool, the O₃ concentration values measured from March 2014 to March 2015 were obtained from the KEPA monitoring station. The data were in volumetric units (ppm or ppb), while the AirQ software tool requires the data based on gravimetric unit (µg/m³). Therefore, the data was converted to gravimetric units for AirQ model (the simple conversion ratio: 1 ppb is 2 µg/m³) (Chen and Hopke, 2009). The data were sorted from lower to higher, and then the number of days in the various O₃ concentration intervals was determined (Table 1).

The raw data were used for estimation of mortality and morbidity rates. For this purpose, these steps were performed successively:

1. Using an Excel spreadsheet, the gaseous concentration data were converted to standard temperature and pressure;
2. The concentrations were converted from volumetric to gravimetric units.
3. Hourly average values were calculated.
4. The data were sorted and the ozone frequency distribution was calculated.

5. AirQ was used to estimate the CM, RM and HA-COPD rates from exposure to O₃ for each exposure category.

2.4. Exposure assessment

The measured O₃ concentrations were expressed as hourly averages. The values of RR and BI were estimated according to the AirQ model’s default to associating the health impacts of O₃. This model assumes that measured concentrations were representative of the mean population exposure. The RR gives the increase in the chance of the harmful effect related to a given chance in the exposure concentrations, and obtained from time-series studies where day-to-day changes in air pollutants over long time intervals were associated with daily mortality, hospital admissions, and another health indicators (Maleki et al., 2016; Daryanoosh et al., 2016a, 2016b).

The number of excess cases of cardiovascular mortality (CM), respiratory mortality (RM), and hospital admissions due to chronic obstructive pulmonary diseases (HA-COPD) from exposure to O₃ was assessed based on RR and BI values obtained from published epidemiological studies on short-term health impacts of air pollution (Table 2).

3. Results

Table 3 presents the maximum 1-h average, the summer and winter maximum 1-h averages, and 98th percentile average of ground-level O₃ concentrations. The maximum annual 1-h average was 297.2 µg/m³. The maximum winter 1-h average was 328 µg/m³ and was higher than the maximum summer value (266 µg/m³). The 98th percentile 1-h average was 261 µg/m³.

Table 4 provides the attributable proportion (AP) percentages and the effects of exposure to O₃ on the number of CM, RM, and HA-COPD based on the estimated RR and BI with 95% confidence intervals (95% CI). The number of estimated excess cases for CM, RM, and HA-COPD for the intermediate RR values were 83, 32, and 36 persons, respectively, in Kermanshah. Fig. 2 shows the results of quantification of the health risks due to

<table>
<thead>
<tr>
<th>Conc. interval [µg/m³]</th>
<th>Number of days</th>
<th>Conc. interval [µg/m³]</th>
<th>Number of days</th>
<th>Conc. interval [µg/m³]</th>
<th>Number of days</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt; 10</td>
<td>0</td>
<td>80–89</td>
<td>9</td>
<td>160–169</td>
<td>0</td>
</tr>
<tr>
<td>10–19</td>
<td>26</td>
<td>90–99</td>
<td>2</td>
<td>170–179</td>
<td>2</td>
</tr>
<tr>
<td>19–29</td>
<td>43</td>
<td>100–109</td>
<td>2</td>
<td>180–189</td>
<td>1</td>
</tr>
<tr>
<td>30–39</td>
<td>67</td>
<td>110–119</td>
<td>0</td>
<td>190–199</td>
<td>5</td>
</tr>
<tr>
<td>40–49</td>
<td>65</td>
<td>120–129</td>
<td>3</td>
<td>200–249</td>
<td>11</td>
</tr>
<tr>
<td>50–59</td>
<td>55</td>
<td>130–139</td>
<td>5</td>
<td>250–299</td>
<td>5</td>
</tr>
<tr>
<td>60–69</td>
<td>30</td>
<td>140–149</td>
<td>5</td>
<td>300–349</td>
<td>2</td>
</tr>
<tr>
<td>70–79</td>
<td>22</td>
<td>150–159</td>
<td>5</td>
<td>350–399</td>
<td>0</td>
</tr>
</tbody>
</table>

Table 1 The number of days in the various O₃ concentration intervals.

Table 2 Baseline incidence and relative risk with confidence intervals (95% CI) obtained from different studies.

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Health effect</th>
<th>BI</th>
<th>RR for ozone (95% CI) per 10 µg/m³</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mortality</td>
<td>Cardiovascular mortality</td>
<td>1.004 (1.000–1.006)</td>
<td>(Fattore et al., 2011)</td>
</tr>
<tr>
<td></td>
<td>Respiratory mortality</td>
<td>1.008 (1.004–1.012)</td>
<td>(Fattore et al., 2011)</td>
</tr>
<tr>
<td>Morbidity</td>
<td>HA-COPD</td>
<td>1.0066 (1.004–1.013)</td>
<td>(Ghosizkali et al., 2014)</td>
</tr>
</tbody>
</table>

a Baseline incidence.
b Relative risk.
exposure to atmospheric O3 in Kermanshah. This figure shows the cumulative number of each health endpoint at three levels of relative risk (5%, 50%, and 95%). The figure also provides the cumulative number of excess cases for each O3 concentration interval (µg/m³) in three relative risk categories, upper, central, and lower. The intermediate set of bars in each chart links to 50% relative risk. Lower bars will underestimate (relative risk of 5%), and the upper curve will overestimate (relative risk of 95%) the likely relative risk.

The cumulative number of excess CM, RM, and HA-COPD cases for the 50% RR category (intermediate bars) are 83, 32, and 36 persons, respectively. The results also show that about 2% (95% CI: 0.0–2.9%) of CM, 5.9% (95% CI: 2.3–9.4) of RM, and 4.1% (CI: 2.5–6.1%) of HA-COPD, respectively, can be attributed to O3 concentrations greater than 10 µg/m³. For each 10 µg/m³ increase in O3, the risk of CM, RM, and HA-COD increased by 0.4%, 1.25%, and 0.86%, respectively. In addition, 18.8% of health impacts due to O3 occurred at concentrations less than 20 µg/m³, and 88.8% of estimated excess cases occur on days with O3 concentrations less than 100 µg/m³.

4. Discussion

The AirQ2.2.3 software tool has been applied in epidemiological studies in many locations to evaluate the health impacts of air pollutants (e.g., Dehghani et al., 2014; Yari et al., 2016; Jeong, 2013; Krzyzanowski, 1997). Cardiovascular mortality (CM), respiratory mortality (RM), and hospital admissions due to chronic obstructive pulmonary disease (HA-COPD) attributed to short-term exposure of tropospheric O3 were estimated. The hourly annual maximum, summer maximum, winter maximum, and 98th percentile in Ahvaz, Iran were 72.6, 84.4, 60.5, and 121.2 µg/m³, respectively (Goudarzi et al., 2013), similar to the results of the present study. The highest 24-h average O3 concentrations to which people in Kermanshah were exposed were in the range of 30–39 µg/m³. Comparable values in Shiraz during 2013 were 70–79 µg/m³ (Mohammadi et al., 2016).

The relative risk (RR) and attributable proportion (AP) for CM, RM,
and HA-COPD caused by exposure to tropospheric O$_3$ were calculated using baseline incidence rates (BI) of 497, 66, and 101 per 100,000 persons, respectively. For our population of 870,000 and based on these annual BI values, about 83, 32, and 36 persons CM, RM, and HA-COPD, respectively, can be estimated annually for O$_3$ levels above 10 µg/m$^3$.

Martuzzi et al. (2006) investigated the health impacts of O$_3$ in 13 Italian cities with a total population of approximately 9 million during the period of 2002–2004. The total estimated mortality in relation to O$_3$ exposure was 516 persons annually. In Shiraz, the cumulative number of excess cases attributable to O$_3$ was estimated to be 218 and 85 persons for CM and RM, respectively (Mohammadi et al., 2016). In a study in Suwon, South Korea with a population of about 1,118,000 people, the cumulative number of excess cases due to exposure to O$_3$ was 43 persons (Jeong, 2013). In Tabriz, Iran with 1,500,000 population, the cumulative number of excluding accidental cases of 47 persons was obtained for HA-COPD in 2008–2009 (Ghozikali et al., 2014). For each 10 µg/m$^3$ increase in O$_3$ level, the risk of HA-COPD increased about 0.58% in Tabriz (Ghozikali et al., 2014). There was an association between increase of O$_3$ level and rise of HA-COPD risk in Minnesota, USA (Schwartz, 1994). The results of another study conducted in Ahvaz found that 3.52% (95% CI: 0.05–5.58%) of mortality and morbidity were associated with O$_3$ concentrations above 10 µg/m$^3$ (Goudarzi et al., 2013).

Burnett et al. (1997) reported that a rise of 23 µg/m$^3$ in O$_3$ (an interquartile range) resulted in an 11% increase in hospital admissions due to respiratory disease and an 8% increase in cardiac admissions. In another study in Sao Paulo, Brazil, exposure to O$_3$ has a significant correlation with the increase of HA-COPD cases (Gouveia et al., 2006). More than 92% of short-term health risks are in relation to the days where the background O$_3$ concentration was lower than 150 µg/m$^3$. Also, higher than 55.3% cases of mortality and morbidity were attributable to O$_3$ concentrations lower than 40 µg/m$^3$. While in the study of Ghozikali et al., more than 96% of HA-COPD was associated with O$_3$ concentrations less than 100 µg/m$^3$. At O$_3$ concentrations below 30 µg/m$^3$, HA-COPD was not observed (Ghozikali et al., 2014).

The impact assessment of air pollution on public health is an important topic because air pollution continues to be a risk factor for human health, especially in Iran where air pollutant concentrations continue to rise. Local analyses of the health effects of air pollution are limited, so the use of the AirQ model and similar software is necessary to provide estimates of the potential health outcomes. Our study also has limitations as follows:

- Generally, in quantitative assessment of health impacts of air pollution, the interactions between different contaminants are not well evaluated and this information is rarely available.
- In the approach used here, the health impacts are focused on a single pollutant without considering the simultaneous exposure to the multiple pollutants to which the public is actually exposed.
- Another limitation is the RR estimates that were derived in studies of different populations in comparison to that under investigation. Additionally, since this model is an ecologically based approach and not an epidemiological approach, it does not consider intra-individual differences because of different behaviors among the inhabitants investigated (i.e. active or passive smoking, proximity to major roads, morbidity during the days, etc.).

A further limitation is potential exposure misclassification. This approach assumes that concentrations measured at the central monitoring point are representative of the exposure of all people living in Kermanshah. Particularly, while for O$_3$ the RR are typically based on epidemiological studies conducted on the European population. However, the transferability of the health outcome estimates from the evidentiary e.g., the US cohort to the locally studied people is reasonable if no compelling evidence that the target population and the evidentiary population differ in their responses to the air pollution.

5. Conclusions

The results of this study showed that high O$_3$ concentrations and/or increased number of days on which people are exposed to this pollutant likely result in adverse health outcomes in Kermanshah. The results of our study are in agreement with those of prior studies and show that this method and model offers a valuable and easy-to-use software that will be helpful to air quality managers and senior officials. Based on the estimated cumulative number of individuals affected by the O$_3$, it can be concluded that cardiovascular death had the major role in the total mortality. Although this results were consistent with other studies conducted throughout the world, additional studies to better specific the RR and BI values based on actual conditions in Kermanshah are necessary. For ozone, precursor species like NOx and reactive VOCs will need to be controlled. Various source emissions including transport and energy production facilities need to be reduced in order that ozone concentrations are reduced. Health training is needed to provide members of the general public especially individuals with chronic lung and heart diseases, elderly and children, to reduce their activities on the days with unhealthy air quality.

Acknowledgement

The authors wish to thank the Kermanshah’s Environmental Protection Agency (KEPA) for supply of air pollution data. The authors also would like the thank Anis Omidi Khaniabadi (an Engineer of Shahid Chamran University of Ahvaz) for her help in the preparation of the figures by Photoshop Software for this manuscript.

References
