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MONITORING THE IMPACT OF AMBIENT OZONE ON HUMAN HEALTH USING TIME SERIES ANALYSIS AND AIR QUALITY MODEL APPROACHES

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ABSTRACT

This study examined the health impacts of O₃ in Ahvaz (Iran). Ozone data were obtained from the Iranian Environmental Protection Agency and the time series were analyzed while the health endpoints from O₃ exposure were calculated using the **Air Q** model. The time series analysis showed that air pollutants levels were associated with five steps delay of O₃ and zero step delay of moving average ARMA (5, 0). The results of **Air Q** model revealed cumulative cases of cardiovascular mortality and myocardial infarction related to surface O₃. The number of cases attributable to O₃ exposure for cardiovascular mortality and myocardial infarction were estimated at 182 and 51 people per year, respectively. The finding of this study showed that, the distribution of O₃ data has a correlated structure over time. Ground-level O₃ was found to be positively correlated with an increased risk of cardiovascular mortality and acute myocardial infarction in Ahvaz.

KEYWORDS:

Time series analysis, **Air Q** Model, Health Endpoint, Relative Risk, Iran.

INTRODUCTION

Air pollution remains the most serious environmental problem with adverse effects on human health [1-6]. In a typical urban environment, the population is exposed to about 200 air pollutants or classes of air pollutants [7, 8]. Surface ozone (O₃)

is an important atmospheric pollutant, a highly reactive trace gas and the third most important greenhouse gas in terms of radiative forcing contributing to climate change [9, 10]. Ozone is produced as a result of photochemical reactions between oxides of nitrogen (NO_x) and volatile organic compounds (VOC_s) [11-15]. Ground-level ozone is produced and destroyed in a cyclical set of chemical reactions involving NO_x, VOC_s, heat and sunlight [11, 12, 16]. As a result, differences in NO_x, VOC emissions and weather patterns contribute to daily, seasonal and yearly differences in O₃ concentrations in cities and remote areas [11, 12, 16]. The lifetime of tropospheric O₃ is long enough, i.e. a few days in the boundary layer to a few months in the free troposphere, to allow transport from regional to hemispheric scale affecting areas far from the source regions [11, 12, 17-20]. The severity, frequency and duration of O₃ peaks exhibit complex patterns and source-receptor relationships, such that the most efficient strategies to reduce local or regional O₃ exposures are not always obvious [11, 12, 17-19]

Time series and case-crossover analyses are the most common methods used to estimate the short-term effects of air pollution on human health [21, 22]. There have been numerous community time series studies on the effect of air pollution on mortality and morbidity [23-25, 22, 26]. Development and use of statistical and other quantitative methods in environmental sciences have been a major interface between environmental scientists and statisticians [27]. The common descriptive statistical approach, used for air quality measurement and modeling, is a simple method developed to understand the behavior and variability of air quality [28]. Time series analysis is a useful tool for a

better understanding of cause and effect relationship in environmental pollution ([29-34]. In the present study, we have intended to use the Box-Jenkins method to analysis the time series models. Many studies have tried to detect trends in air pollution over time, using different techniques, e.g. nonlinear time series prediction or Mann-Kendall test [35, 18, 36]. On the other hand, some studies have tried to relate air pollution to human health through time series and epidemiological studies focused on the health effects, increases in hospital admissions or deaths, at the population level and may be used to calculate the short-term health impacts of air pollutants [37, 24, 38].

Studies on health effects of O₃ using the time series analysis and air quality approach is rarely reported. The objective of this study was to assess the potential effects of O₃ exposure on human health in Ahvaz city (Northern Iran) using the time series analysis and **Air Q2.2.3** model.

MATERIALS AND METHODS

The study Area. Ahvaz has is the capital city of Khuzestan province with a population of approximately 1 million people and a surface area of 140 km² (Figure 1). It is located between 49°29' E and 31°45' N [39-47].

Methodology. In this study, we carried out the health assessment of O₃ using the time series analysis from MINITAB version 16 statistical package and the World Health Organization (WHO) air quality health impact assessment tool, Air Q2.2.3 model. Hourly ozone data for the year 2011 were obtained from the Iranian Environmental Protection

Agency (Iranian EPA) while the meteorological data were collected from the Iranian Bureau of Meteorology over the same time period.

(1) Time series analysis. Time series models have two advantages over other methods such as regression and polynomial regression [48]. The first is that, values are modeled on past events or past stochastic terms (shocks) for easier interpretation and monitoring of air characteristics. The second advantage is, forecasting or estimation of future values and means to predict future values are based on past values and stochastic structure of data [37, 38, 30].

The time series analysis was a set of observations that are arranged chronologically. It consists of sequential data points taken at equal increments over the time. The Auto Correlation Function (ACF) and the Partial Autocorrelation Function (PACF) are two important instruments to identify q and p , respectively. The order of time series model (Box-Jenkins steps) consists of three steps. The first step is the identification of p and q , the second one is the estimation of parameters and the third one is the verification of the model adequacy [49, 50]. For interpretation purposes, it is often useful to plot autocorrelation function (ACF) against lag time, k . The ACF is a simple graphical method useful to find the time relationship of an event. ACF is defined as following:

$$P_y(k) = \frac{R_y(k)}{R_y(0)} \quad (1)$$

where $P_y(k)$ and $R_y(k)$ are the autocorrelations of the time series variable y_t and the auto covariance of y_t at time lag k , respectively [51, 52].

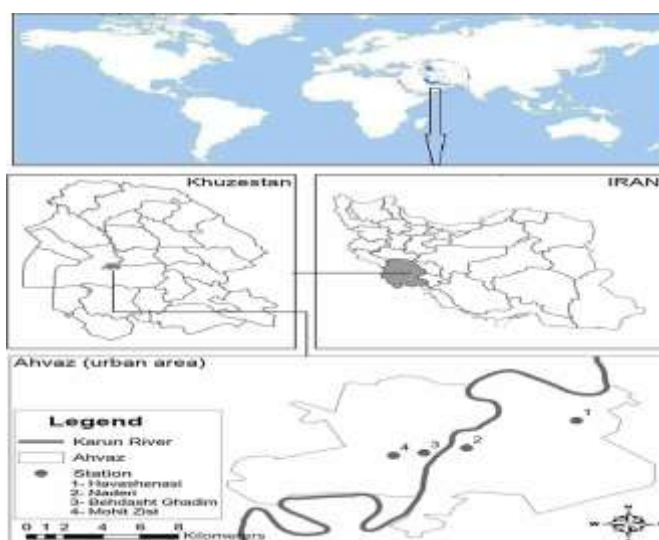


FIGURE 1
Map of the study area showing the monitoring stations

PACF allows examining the effect of a variable value at a given past sampling period on its current value, while filtering out intervening effects of other past sampling periods on its behavior. PACF can be defined by the following ratio [51]:

$$PACF = \frac{COV((y_t - y_t^1)(y_{t+k} - y_t^1))}{\left(\sqrt{VAR(y_t - y_t^1)}\sqrt{VAR(y_{t+k} - y_t^1)}\right)} \quad (2)$$

where, COV is the covariance, VAR is the variance, y_t is the time series variable, k is the time lag, and y_{t+k}^1 is the estimate of the last coefficient in an autoregression AR(k) model. The sample autocorrelation coefficient [53] is written as:

$$r_k = \frac{\sum_{t=1}^{N-k} (X_t - \bar{X})(X_{t+k} - \bar{X})}{\sum_{t=1}^N (X_t - \bar{X})^2} \quad (3)$$

where N is the total length of record, k is lag time, X_t is observation at time t and \bar{X} is the mean of the series. The r_k lies between -1 and +1; the values above zero denote correlation at the given lag. The values of k range from 0 to N . ACF can show whether the observation depends on time or not. When ACF decays rapidly to zero after a few lags, it may be an indication of stationary in the time series, while a slow decay of ACF may be the indication of non-stationary [54, 55]. A strict stationary means that there is no systematic change in mean (no trend) and variance, and strictly periodic variations have been removed. Quantitatively, this means that the joint distribution of $X(t_1), \dots, X(t_n)$ is the same as the joint distribution of $X(t_1+t), \dots, X(t_n+t)$, for all t_1, \dots, t_n . This also indicates that shifting the time of origin by t has no effect on the joint distributions, which only depends on the time intervals between t_1, t_2, \dots, t_n . Second-order stationary is for weak stationary. In other words, a finite memory of the series leads to a gradual decline of the envelope of the ACF [56]. Another way to investigate whether a series is time dependent or not is time series regression [57]. The polynomial time regression between dependent variables, Y_t and time, t is written as follows:

$$Y_t = \beta_0 + \beta_1 t + \beta_2 t^2 + \dots + \beta_p t^p + \varepsilon_t \quad (4)$$

where, β_0 to β_p are the parameters of the regression equation and least square point estimates of them may be obtained by regression techniques. Detailed statistical inference on the significance of regression and parameters can be seen elsewhere in [57]. A broad class of time series model is ARMA (p, q), which combines the two models: AR (Auto Regressive) and MA (Moving Average) [50]. ARMA (p, q) is given as follows:

$$\left(\sum_{i=1}^p \phi_i B^i\right) Y_t = \left(\sum_{i=1}^q \theta_i B^i\right) \varepsilon_t \quad (5)$$

where, B is the backshift operator ($B_i y_t = y_{t-i}$), ϕ, θ are the parameters of autoregressive and moving average models, respectively. \bar{Y}_t is the mean of the

air characteristics like O_3 and ε_t denoted as error term which is assumed a normal distribution with zero mean and constant variance σ_2 . P and q are the orders of AR and MA, respectively.

(2) **Air Q Model Data analysis.** Air Q model is based on statistical equations. The cardiovascular mortality and acute myocardial infarction associated with O_3 air pollution in Ahvaz were calculated with the Air Q2.2.3 model, utilizing the relative risk and baseline incidence from the World Health Organization (WHO) database. Attributable proportion was calculated as following formula:

$$AP = \frac{SUM\{[RR(c)-1] \times p(c)\}}{SUM[RR(c) \times p(c)]} \quad (6)$$

Where, $p(c)$ is the exposure population of city in a particular category [58]. RR denotes the relative risk for a given health endpoint. Relative risk is a ratio of the probability of the event occurring in the exposed group versus a non-exposed group [59].

$$RR = \frac{\text{Probability of event when exposed}}{\text{probability of event when non - exposed}} \quad (7)$$

The number of cases attributable to the exposure can be estimated as the following equation knowing the size of the population:

$$NE = IE \times N \quad (8)$$

Where, NE denotes the number of cases attributed to the exposure and N denotes the size of the investigated population.

Attributable proportion was multiplied at baseline incidence and divided to 10^5 . Obtained value should be multiplied at population ($\times 10^6$) [39]. The results will be the excess cases of mortality or morbidity attributed to a given pollutant (e.g. ozone).

The final model is given as follows:

$$O_{3t} = 2.95 + 0.78O_{3t-1} + 0.24O_{3t-5} + a_t - 0.16a_{t-1} \quad (9)$$

where $a_t \approx N(0, 116.1)$, N stand for normal distribution.

RESULTS

Time series analysis. Time series plots (TSP) of O_3 concentrations over time during different seasons are presented in Figures 2 and 3. The primary and secondary standards of O_3 according to the National Ambient Air Quality Standard (NAAQS) for 8 hours are $140 \mu\text{g}/\text{m}^3$. The guidelines applied worldwide are based on the expert evaluation of current scientific evidence for O_3 ($100 \mu\text{g}/\text{m}^3$ for 8-hour mean, [60]). In Ahvaz the annual O_3 mean was $224 \mu\text{g}/\text{m}^3$ in 2011 whereas a mean value of $122 \pm 10.8 \mu\text{g}/\text{m}^3$ was observed in rural sites around the western Mediterranean basin (O_3 hot spot) over the time-period 2000-2010 [61]. This O_3 value was strongly higher than the WHO and NAAQS standards based on 8-hour mean.

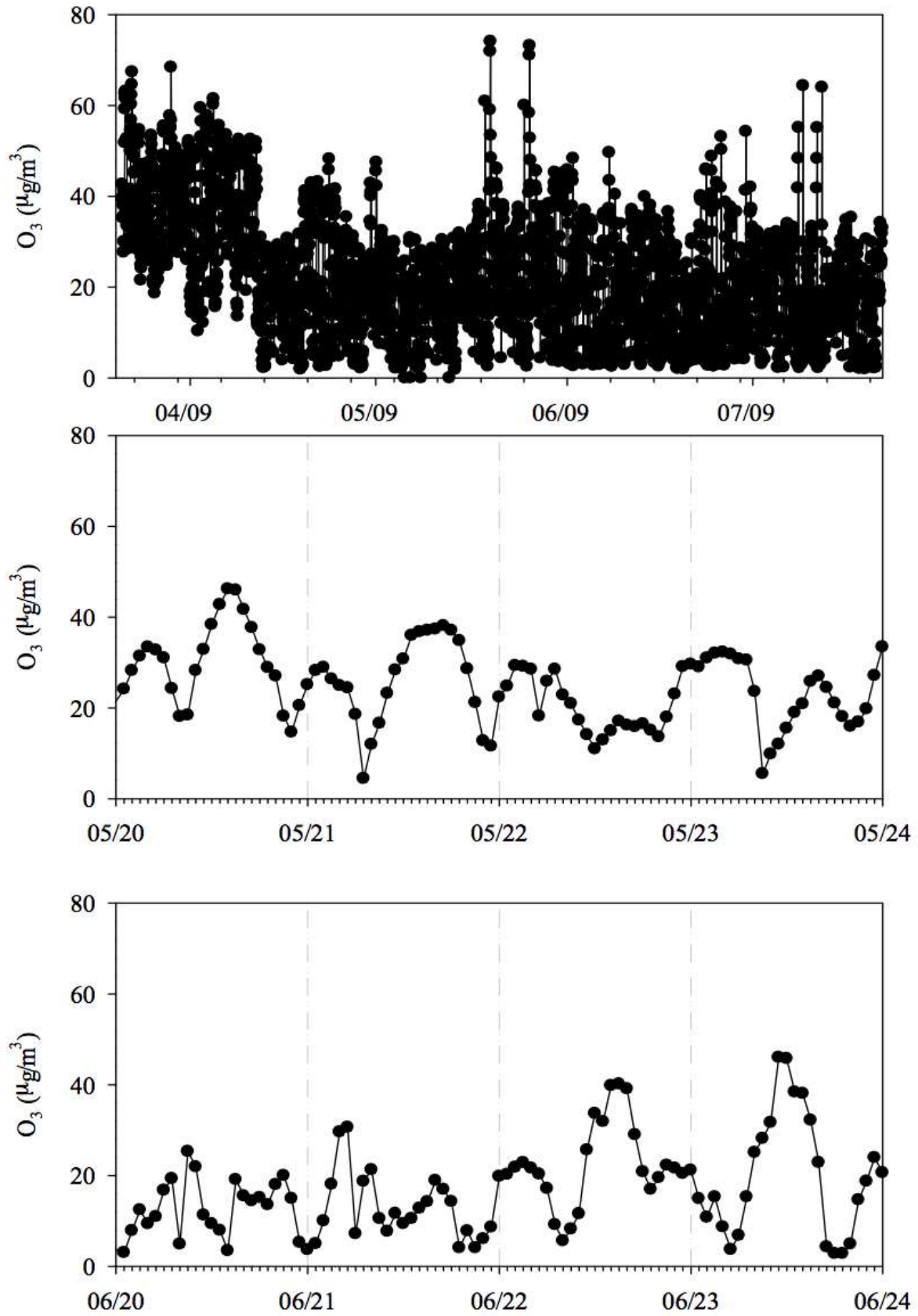


FIGURE 2
Time series plot of ozone concentration (Spring-Summer)

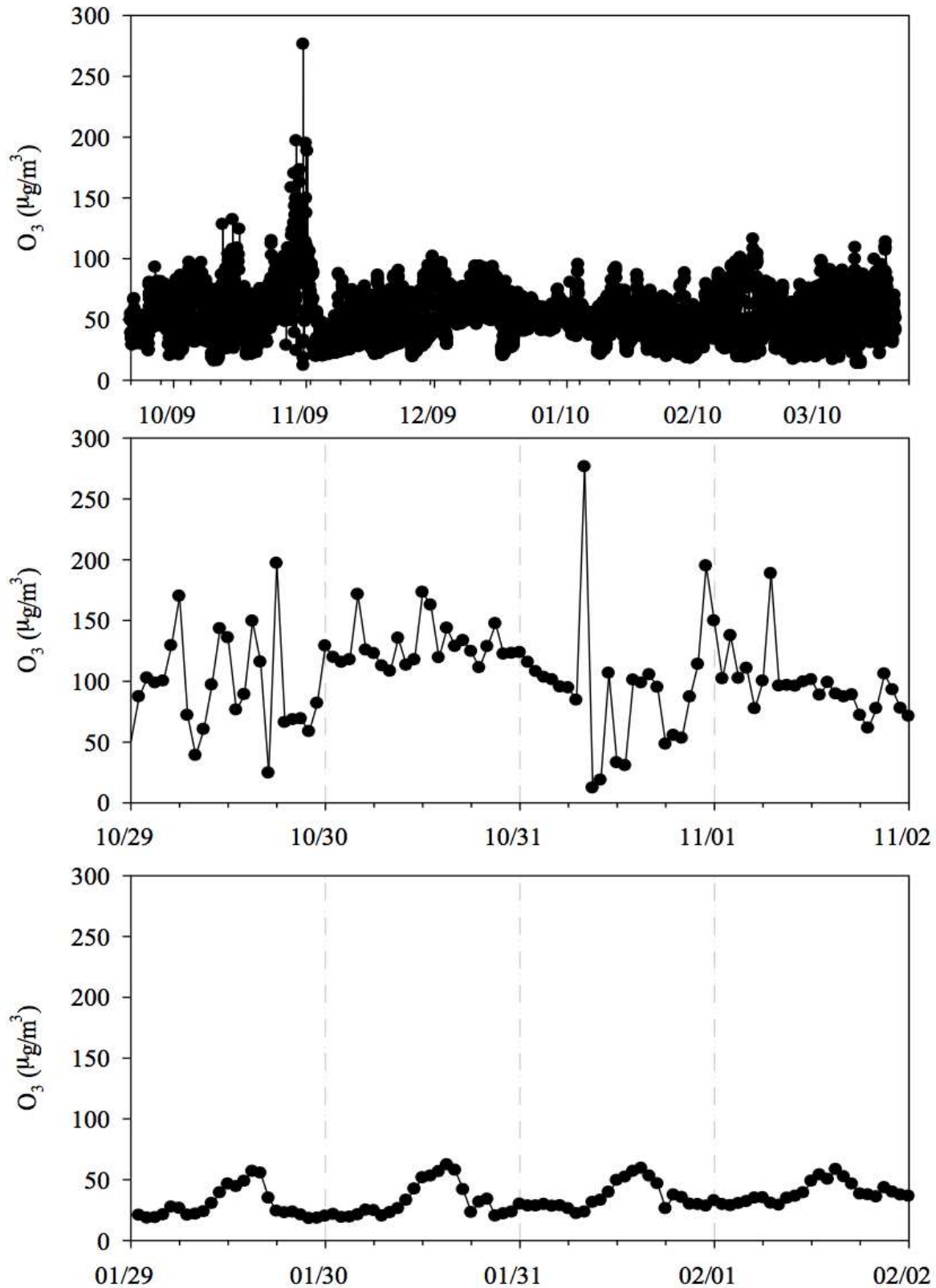


FIGURE 3
Time series plot of ozone concentration (Fall-Winter)

Figures 4 and 5 show the time series autocorrelation for O_3 concentrations. Both figures suggest $p=5$ and $q=1$ and the autocorrelation had a decay

pattern indicating no moving average term in model (Fig. 4) and the most significant correlation are

observed for the first five lags of partial correlations (Fig. 5).

We have carried out ARMA (5, 1) or AR (5) and the estimation of parameters are given in Table 1. Minitab result showed that the coefficients of second, third and fourth order of autoregressive are not significant ($p > 0.05$). Therefore, we preferred to remove those terms. Furthermore, Ljung –Box results are given for both models, which in turn suggest no seasonal pattern in the model.

Health assessment. Relative risk and estimated attributable proportion percentage for cardiovascular mortality and myocardial infarction were calculated and presented in Table 2. Baseline incidence (BI) for this health endpoints for O_3 were 497 and 66 per 10^5 [59]. The number of cardiovascular mortality was 182 (RR=1.004 and AP=3.56%) while the number of myocardial infarction was 51 (RR=1.008 and AP=7.01%) at the centerline of relative risk.

Autocorrelation function for Ozone

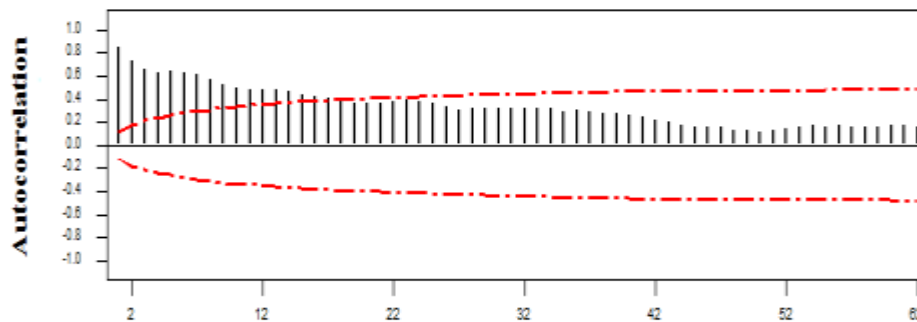


FIGURE 4

Time series autocorrelation for ozone concentration

Partial Autocorrelation function for Ozone

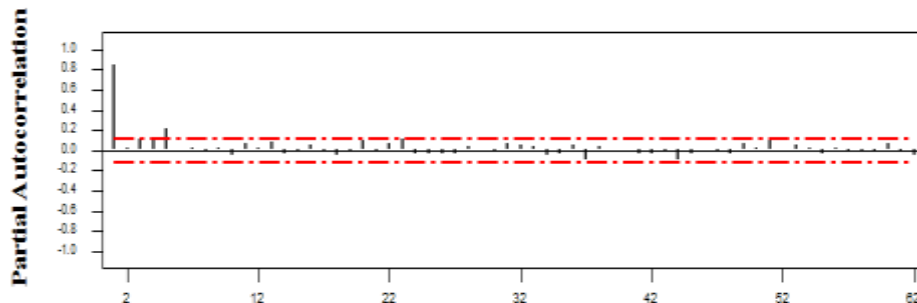


FIGURE 5

Time series partial autocorrelation function for ozone concentration

TABLE 1
Time series of ozone concentration

Type	Coefficient	SE Coefficient	T	P
AR 1	0.7762	0.0562	13.81	0.000
AR 2	-0.0505	0.0717	-0.70	0.482
AR 3	0.0155	0.0720	0.22	0.829
AR 4	-0.0573	0.0722	-0.79	0.428
AR 5	0.2394	0.0565	4.24	0.000
MA 1	0.1582	0.0591	2.68	0.008
Constant	2.9498	0.6174	4.78	0.000
Mean	38.511	8.060		
Residuals: SS = 34728.6 (back forecasts excluded)				
		MS = 116.1	DF = 299	
Modified	Box-Pierce	(Ljung-Box)	Chi-Square	Statistic
Lag	12	24	36	48
Chi-Square	4.1	15.8	25.4	34.1
DF	6	18	30	42
p-value	0.667	0.609	0.707	0.803

AR: Auto Regressive
MA: Moving Average

TABLE 2
Estimated relative risk indicators and the components attributable to ozone cases which are related to cardiovascular mortality and respiratory mortality

Parameter	RR (medium)	Estimated AP (%)	Number of excess cases (person)
Cardiovascular mortality	1.004	3.56	182
Myocardial infarction	1.008	7.01	51

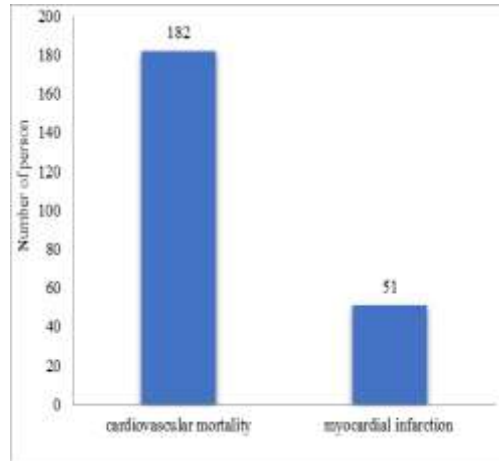


FIGURE 6
Cases of cardiovascular mortality and myocardial infarction from high ozone concentration

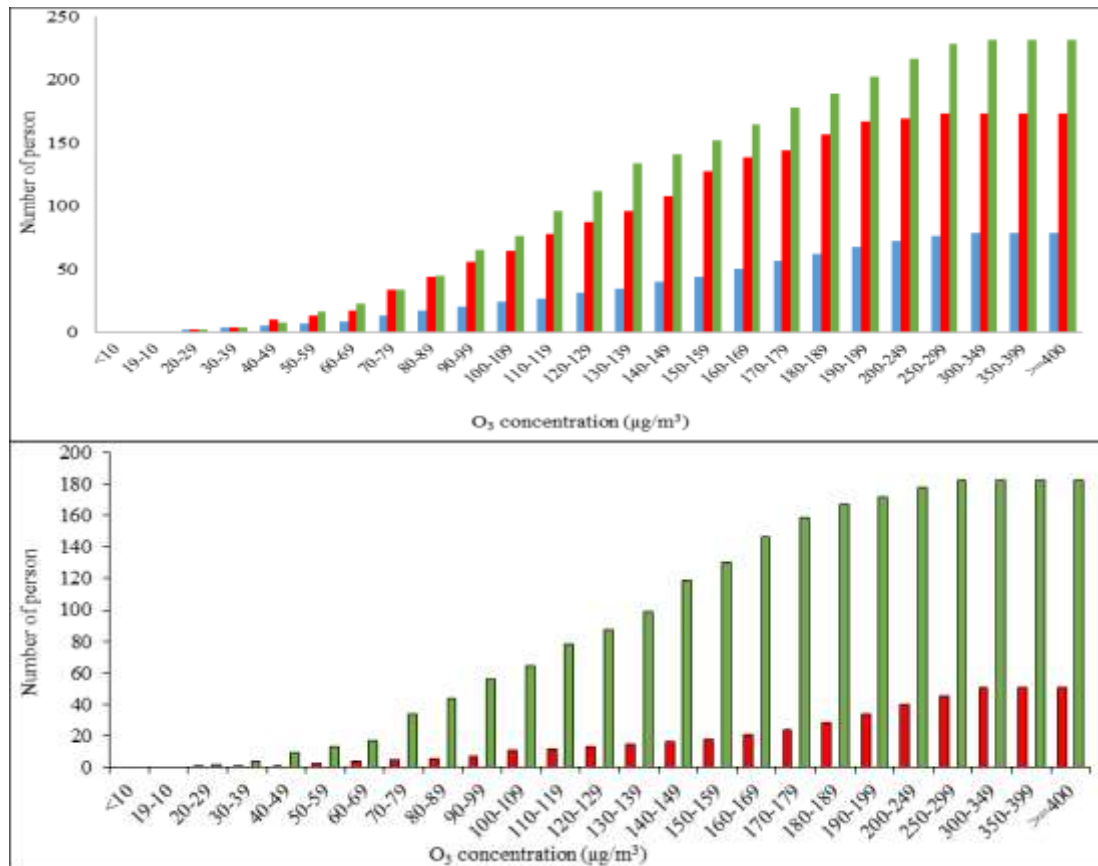


FIGURE 7
Trends of relationship between cumulative of cardiovascular mortality and myocardial infarction versus ozone concentration (low in blue, central in red, and high in green)

Figure 6 shows the cumulative cardiovascular mortality and myocardial infarction data versus O_3 . During the study period (year 2011), cumulative cases of cardiovascular mortality were estimated at 182 while 51 persons were estimated as myocardial infarction patients within one year of exposure. The trends of relationship between cumulative cases of cardiovascular mortality and myocardial infarction due to O_3 concentrations were illustrated in Figure 7. It was observed that 67% of cardiovascular mortality cases occurred on days with O_3 concentration lower than $140 \mu\text{g}/\text{m}^3$ and 61% of cases occurred on days where O_3 levels do not exceed $150 \mu\text{g}/\text{m}^3$. On the other hands, 46% of myocardial infarction cases occurred on days with O_3 levels lower than $160 \mu\text{g}/\text{m}^3$ while 71% of cases occurred on days where O_3 concentration are lower than $250 \mu\text{g}/\text{m}^3$. It was also observed that cases of myocardial infarction rose to 82% when O_3 concentration was higher than $300 \mu\text{g}/\text{m}^3$.

DISCUSSION

Based on the results of our study, there was a significant association between the surface O_3 level and human health. Su et al. (2006) observed in their study the adverse effects of fine particulate air pollution on cardiovascular hospital emergency room visits obtained from time-series study (PDL model) in Beijing, China [62].

Results of this present study are similar to studies reported from different regions of world. Three year data on daily ozone concentrations and daily number of deaths from 23 cities/areas were collected. Their studies revealed no significant effects during the cold half of the year. For the warm season, an increase in the 1-hour ozone concentration by $10 \mu\text{g}/\text{m}^3$ was associated with 0.33% (95% confidence interval, 0.17–0.52) increase in the total daily number of deaths, 0.45% (95% CI, 0.22–0.69) in the number of cardiovascular deaths and 1.13% (95% CI, 0.62–1.48) in the number of respiratory deaths [63]. High percentage of the observed health endpoints in this study was associated with high O_3 concentration in Ahvaz. In another study, McConnell et al. (2002) investigated the relation between newly diagnosed asthma and team sports in a cohort of children exposed to different concentrations of ozone. Their findings showed that in communities with high O_3 concentrations, the relative risk of developing asthma in children who engaged in three or more sports was 3.3 (95% CI 1.9–5.8), compared with children playing no sports [64]. A 2006 study linked exposures to high O_3 levels for as little as one hour to a particular type of cardiac arrhythmia that itself increases the risk of premature death and stroke [65]. Based on the results of this study, the number of cases of health effects was relatively higher because of greater concentration in

Ahvaz city. A study in the southwestern France found that exposure to elevated O_3 levels for one to two days increased the risk of acute myocardial infarction for middle-aged adults without heart disease [19].

Citizens are more exposed to high O_3 levels in many rural areas than people living in the cities [6]. This study showed higher O_3 concentration in Ahvaz than in Spain, Italy and France (western Mediterranean basin) and Tehran [12]. Most important results from the study are the significant overrun of exposure metrics in comparison with the objectives of legislative air quality directives. [12] evaluated the health effects of O_3 by Air Q model in Tehran city and found that, 3.57% of all cardiovascular and respiratory mortalities were attributed to O_3 concentrations over $20 \mu\text{g}/\text{m}^3$ [12]. Also, the O_3 impact at concentrations higher than $70 \mu\text{g}/\text{m}^3$ resulted to 0.6% of all causes of mortality in eight major Italian cities [66]. However, in contrary to this study, the study of Ritz et al. had reported no effect in the risk of preterm birth associated with ambient O_3 . We estimated outcomes attributed to surface O_3 which may be underestimated due to the existence of other pollutants into the air [67]. Future investigations in collaboration with international scientific teamwork are needed to explore more details for this kind of studies toward mitigating the impact of O_3 or other pollutants on inhabitant in megacities.

It was considered that the 8-h guideline would protect against acute elevated 1-h exposures [68]. The current standards consider only acute health effects, e.g. lung inflammation, increased numbers of daily hospital admissions, and mortality, for cardiovascular and respiratory diseases, including exacerbations of asthma and chronic obstructive pulmonary diseases and do not account for possible chronic effects at long-term O_3 exposure levels [69]. To protect population and vegetation against the adverse effects of O_3 , suitable standards and consistent standards are needed.

CONCLUSIONS

This study had assessed the health impacts of air pollution, using time series analysis and AirQ model in Ahvaz (Iran). Statistical model is an applicable method to evaluate the behavior of pollutant and its dispersion. In this study, data showed that the O_3 has a correlated structure over time. ARMA model showed that the surface O_3 at each time could be estimated based on the previous value AR (5) and the second last value of MA (0). In this context, the model developed in this study, AirQ model, helps decision-makers in the quantitative assessment of new policies that will affect air quality. Comparison between local (Ahvaz) and WHO epidemiological parameters (relative risk and baseline incidence) suggested that local input data

should be used in environmental decision-making, because default values could lead to very different policy decisions. If new morbidity and mortality indicators (O₃-induced injury) are available, they can be easily incorporated into the system to estimate the O₃ health effects. Accordingly, cost-effective measures and management schemes should be considered to abate air pollution concentrations and/or reduce the exposure of general population to air pollutants. This study established that, O₃ exposure was associated with an increased risk of cardiovascular mortality and acute myocardial infarction in Ahvaz, Iran. We recommend further studies to evaluate the model of emissions and a method to evaluate the behavior of the surface O₃ in all megacities of Iran.

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The authors have no conflict of interests.

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