

Estimation of Health Effects Attributed to NO₂ Exposure Using AirQ Model

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Background & Aims of the Study: As an air pollutant, NO₂ reacts with ammonia, moisture and other compounds to form small particles. These small particles may cause or aggravate respiratory diseases and exacerbate existing heart diseases, leading to increased hospital admissions and premature death. In this study, the AirQ2.2.3 model was used to evaluate adverse health effects caused by NO₂ exposure in Ahvaz city (Iran) during 2009.

Materials & Methods: The adverse health effects of NO₂ as an air pollutant in Ahvaz city at 2009 were calculated by AirQ2.2.3 utilizing relative risk and baseline incidence related to health end point defaults. NO₂ data were taken from Ahvaz Department of Environment. These data were in volumetric base. Health effects are being related to the mass of pollutants inhaled and this is why the AirQ model was on gravimetric basis. So, there was a conflict between AirQ model and "Ahvaz Department of Environment (ADoE)" data. Conversion between volumetric and gravimetric units (correction of temperature and pressure), coding, processing (averaging) and filtering are implemented for solving such problem.

Results: Approximately 3% of total cardiovascular mortality, acute myocardial infarction, and hospital admission for chronic obstructive pulmonary disease happened when the NO₂ concentrations was more than 20 µg/m³. Low percentage of the observed health endpoints was associated with low concentration of measured NO₂. The lower level of relative risk value may be achieved if some control strategies for reducing NO₂ emission are used.

Conclusions: Therefore, the higher relative risk value can depict mismanagement in urban air quality.

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Background

Anthropogenic air pollution has been and continues to be a serious problem. Its potentially harmful pollutant levels are produced in environments which can be harmful for human health. According to World

Health Organization estimations, 800,000 people are dying worldwide each year because of cardiovascular and respiratory diseases, which are attributed to air pollution. Approximately 150,000 of these deaths occur in south of Asia.

The results of studies on short and long term effects of air pollution were estimated in the form of excess hospital admissions rates increase the number of consultation with physician, asthma attacks, cardiopulmonary disease, death, and number of the years of life lost (YOLL) (1-9).

Nitrogen dioxide (NO₂) is produced in a combustion process which takes place mostly in transportation, power stations, heating plants, and industrial processes. The summation of NO₂ and NO is called NO_x. The major source of NO_x is road traffic. Although 50% of total NO_x emission is caused by road traffic, this percentage can be higher in megacities and metropolitans (10-12).

There are chronic and acute health effects, which may be appeared due to exposure to low and high NO₂ concentrations, respectively. NO₂ can hurt lower parts of respiratory system and at high concentration, which may be occurred rarely at unprecedented industrial events, can lead to serious lung damages. High concentration of NO₂ can result in airway inflammation and this is why even healthy individuals show some anomalies in the respiratory system function. People with chronic obstructive pulmonary disease (COPD) or asthma attacks are more sensitive to the low concentration of NO₂ (13-14).

Epidemiological studies have shown an association between NO₂ concentrations and health endpoints. In a multicenter case-crossover study a 9.6 µg/m³ increase in the NO₂ concentrations was associated with the hospitalization for cardiovascular disease. The results showed that increase in hospital admission for cardiovascular disease, cardiac failure, ischemic heart disease, and myocardial infarction were attributed to increase in the NO₂ levels (15).

Again in another case-crossover study which was conducted in Spain, there was an association between NO₂ levels and mortality in

patients suffered from asthma exacerbation. In this study, a 22.9 µg/m³ increase in the NO₂ concentration (minimum 5.2 µg/m³) was associated with mortality (16). Positive associations were found between increases of

32 µg/m³ at temperatures above 20°C and hospital admissions for both primary intracerebral hemorrhage and ischemic stroke (17). Another study has shown associations between hospital admissions for cardiovascular diseases and an increase of 32 µg/m³ in NO₂ levels (18). Elevated odd ratios due to hospital admission for an increment of 10.5 µg/m³ NO₂ levels were observed among susceptible groups in a cross-over study (19).

Recently, several studies have reported adverse health effects below the current exposure limits for NO₂, particularly in children and the elderly individuals. In a study, with long-term exposure (1-year average), 1.17, 1.57 were calculated as relative risk (RR) values with an increase of 16 µg/m³ in NO₂ concentrations for all causes of death and cardiopulmonary disease, respectively. Again for the same increase of NO₂ concentrations in a long-term exposure study (5-year average), RR values of 1.19 and 1.74 were calculated for all causes of mortality and cardio pulmonary disease, respectively (20).

Aims of the study: AirQ software was proved to be a valid and reliable tool to estimate the potential short-term effects of air pollution, predicts health endpoints attributed to criteria pollutants, and allows the examination of various scenarios in which emission rates of pollutants are varied. The aim of this study was to assess the potential effects of NO₂ exposure on human health in Ahvaz city (located in Southwest Iran) during year 2009 using the AirQ2.2.3 model.

Materials & Methods

In this retrospective study, a specialized model, “the air quality health impact

assessment tool (AirQ2.2.3 model)", was used to assess the potential effects of NO₂ exposure on human health in Ahvaz city (located in Southwest Iran) during year 2009. Since the design of the model's default is carried out by the World Health Organization in Eastern Mediterranean Region, so we can generalize it for Ahvaz city.

The acute cardiovascular mortality, acute myocardial infarction and COPD associated with NO₂ air pollution in Ahvaz in 2009 were calculated by AirQ2.2.3 model utilizing relative risk and baseline incidence from World Health Organization (WHO) data.

NO₂ data were taken from "Ahvaz Department of Environment (ADoE)". Stations were "Downtown", "Old School of Public Health", "Bureau of Meteorology", and "Head office of ADoE".

These data were on volumetric base. Health effects are being related to the mass of pollutants inhaled and this is why the AirQ model was on gravimetric basis. So, there was a paradox between AirQ model and ADoE data.

Conversion between volumetric and gravimetric units (correction of temperature and pressure), coding, processing (averaging), and filtering were implemented for solving such problem, as follows:

1- Correction for non-standard temperature and pressure: Raw air quality monitoring data were saved in a Microsoft

Office Excel spreadsheet. All processing mechanisms, such as correction, coding, averaging and filtering were performed in this software.

The temperature and pressure are unlikely to be standard, so, we also needed to be able to convert gravimetric units in the Standard Temperature and Pressure (STP) into other temperatures and pressures. In STP, we have 1 m³ air containing a certain mass of material. When the temperature and pressure change, the volume of the gas changes, but it still contains the same mass of material. Therefore, we

needed to find the new volume from the Ideal Gas Equation:

$$\frac{P_1 V_1}{T_1} = \frac{P_2 V_2}{T_1}$$

Where P₁, V₁, and T₁ are the initial pressure, volume and absolute temperature; and P₂, V₂ and T₂ are the final pressure, volume and absolute temperature. In our case:

P₁=1 atmosphere; V₁=1 m³; T₁= 273.15 K; and we needed to find V₂.

Therefore, rearranging equation above:

$$V_2 = T_2/P_2 \times P_1 V_1/T_1 = T_2/273.15 P_2$$

2- Coding: We wrote a code in a column after date column in spreadsheet Excel in order to facilitate mechanism of averaging. In the first cell of code column the formula for coding was: B3= Left (A3, 5)

To know what this formula means, it should be noted that Excel computes from date of Microsoft starting up till the first time of monitoring.

Table 1) Code specification for NO₂ concentrations regarding time intervals

	A (Date)	B (Code)	C (NO ₂ , µg/m ³)
1	03/21/2007 00:00	39162	164.01
2	03/21/2007 01:00	39162	174.57
3	03/21/2007 02:00	39162	165.98
22	03/21/2007 21:00	39162	110.12
23	03/21/2007 22:00	39162	107.91
24	03/21/2007 23:00	39162	73.47
25	03/22/2007 00:00	39163	81.77
26	03/22/2007 01:00	39163	53.90

3- Averaging: The routine method to provide 24-hour average is performed by clicking on f(x) icon. The insert function is appeared. The routine way takes more time when there is a huge number of data. To obtain daily average of the pollutant at each station in the city, it is necessary to write (IF (B26<>B27; AVERAGE (C3:C26)) command. According to code

column, the value of codes will be changed after 24 hours, so that there is a special code for each day. The formula can generalize to the next cells and columns in both directions (horizontal and vertical).

A slight changing in the command sentence of formula would be recommended if it spread horizontally. For example, the above conditional sentence must alter to (IF (B26<>B27; AVERAGE (D3:D26)). Comparison of these two conditional formulas implies that C changes to D for providing average in the next column, which belongs to the levels of NO₂ at the adjacent station. Up and down dragging the conditional sentence will act to compute daily average of NO₂ in all stations. Huge numbers of cells (23 from 24 cells) will be filled by the word “false” which means the first part of conditional sentence is not correct. Therefore, average value is placed on 24 number cells, which are repeated in every 24 cell intervals.

3- Primary filtering: Primary filtering was used for deleting “false” cells. The command Data/Filter/Auto filter/Does not equal to false, is implemented to achieve 365 average values of NO₂ concentrations at each station.

4- Secondary filtering: According to model database, it is suggested to specify the highest and the lowest stations before using secondary filtering.

A station is considered as the highest when the average value of this station is greater than that of other stations and vice versa for the lowest. Therefore, the lowest station should be selected as the station that has the lowest yearly average for NO₂ concentrations in the city.

In the next step, the column of all stations for NO₂ concentrations was built to make an appropriate background for secondary filtering. Then, the air quality data were made from filtering on the column of all stations.

The aim of secondary filtering on the average column of all stations was to find the concentration intervals of NO₂ in year 2009.

Data analysis: AirQ model is based on Statistical equations. Sample community was Ahvaz city which its approximate population was considered one million people.

The collected data was used for NO₂ pollutant in AirQ model. Attributable proportion was calculated as following formula:

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

Where p(c) is population of city, RR is relative risk (RR).

In statistics and mathematical epidemiology, relative risk (RR) is the risk of an event (or of developing a disease) relative to exposure. Relative risk is a ratio of the probability of the event occurring in the exposed group versus a non-exposed group.

$$RR = \frac{\text{Incidence in the exposed}}{\text{Incidence in the non-exposed}}$$

Attributable proportion was multiplied at baseline incidence and divided to 105. Obtained value should be multiplied at population (n=106). The results will be the excess cases of mortality or morbidity attributed to given pollutant (NO₂).

Results

The primary standard of NO₂ according to National Ambient Air Quality Standard (NAAQS) is 100 µg/m³ (21). World Health Organization (AQQ WHO) has recommended 40 and 200 µg/m³ as annual and hourly averages of NO₂ concentrations, respectively (22). The table 2 shows that annual average of NO₂ concentration in Ahvaz was 27 µg/m³ in 2009, which is lower than AQQ WHO and also much lower than NAAQS standards.

The “bureau of Meteorology” and “Head Office of ADoE” had the highest and the lowest NO₂ concentrations during 2009, respectively. The yearly average, summer average, winter average and 98 percentile of NO₂

concentrations in these stations have been presented in table 2.

Table 2) Highest and lowest concentrations of NO₂ (µg/m³) corresponding to stations for use in AirQ2.2.3 model (year 2009)

stations	Ahvaz	Bureau of Meteorology	Head Office of ADoE*
Parameter			
Annual mean	27	66	18
Summer mean	6	-	16
Winter mean	43	72	19
98 percentile	115	369	50

Ahvaz Department of Environment

RR and estimated attributable proportions (AP) percentage for acute myocardial infarction (MI) have been shown in table 3. According to model's default, the baseline incidence (BI) of this health endpoint for NO₂ was 132 per 100,000 people, so, the number of estimated number of excess cases was estimated 9 at centerline of relative risk (RR=1.0036 and AP=0.6977%).

Table 3) Relative risks, attributable proportions and number of persons suffering from acute myocardial infarction due to NO₂ exposure

Indicator	RR (average)	Estimated AP (%)	No. of excess cases (persons)
Lower	1.0015	0.2919	3.7
Average	1.0036	0.6977	8.9
Upper	1.0084	1.6129	20.7

Figure 1 shows the cumulative number of acute MI versus NO₂ concentrations. Cumulative number of this health endpoint were estimated by this model was 9 in 2009. Sixty percent of this number has occurred in the days with NO₂ concentrations lower than 70 µg/m³. It should be noted that 82% of above mentioned number are corresponded to the days with concentrations below 110 µg/m³.

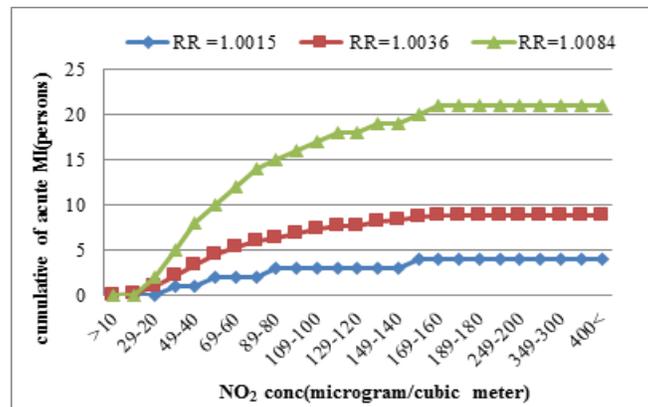


Figure 1) Relationship between cumulative number of acute myocardial infarction (MI) and NO₂ concentrations

The RR and estimated AP percentage were presented in table 4 for acute cardiovascular mortality. BI obtained from model's default was 497 per 100,000 people for this health endpoint. Therefore, estimated number of excess cases were calculated 19 at centerline of relative risk (RR=1.002 and AP=0.3888%).

Table 4) Relative risks, attributable proportions and number of persons due to NO₂ exposure (cardiovascular mortality)

Indicator	RR (average)	Estimated AP (%)	No. of excess cases (persons)
Lower	1	0.000	0.000
Average	1.002	0.3888	18.7
Upper	1.004	0.7746	37.3

Cumulative cases of cardiovascular mortality attributed to NO₂ concentrations has been illustrated in figure 2 with three ranges of relative risk. Thirty seven persons were estimated by the model as total cumulative number of cardiovascular death within one year of exposure. Sixty percent of these cases have occurred in days with NO₂ levels not exceeding 70 µg/m³.

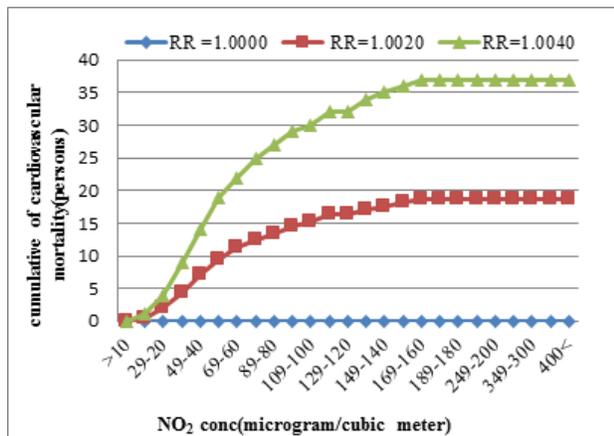


Figure 2) Relationship between cumulative number of cardiovascular mortality and NO₂ concentrations

Relative risk and estimated AP percentage has been shown in table 5 for acute hospital admission of COPD. BI of this health endpoint is 101.4 per 100,000, the estimated number of excess cases were 8 at centerline of relative risk (RR=1.0038 and AP=0.7361%).

Table 5) Relative risks, Attributable proportions and pulmonary disease due to NO₂ exposure

Indicator	RR (average)	Estimated AP (%)	No. of excess cases (persons)
Lower	1.0004	0.078	0.8
Average	1.0038	0.7361	7.2
Upper	1.0094	1.0094	17.7

Hospital admission of COPD versus NO₂ concentrations has shown in figure 3. In this case the lowest line (RR=1.0004) is not attached to X axis and it shows a little difference with figure 2. Estimated cases which attributed to NO₂ for hospital admission for COPD at lower, central and higher level of RR were 1, 8 and 18, respectively.

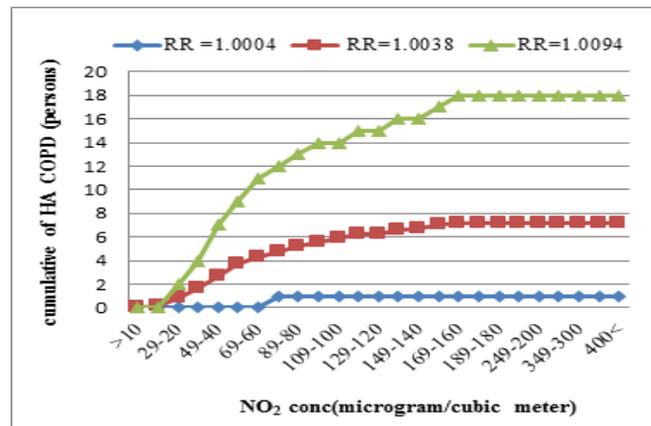


Figure 3) Relationship between cumulative number of hospital admission for chronic obstructive pulmonary disease and NO₂ concentrations

Discussion

Figures 1 to 3 have illustrated NO₂ concentrations versus related health endpoint. As the results showed, three ranges of relative risk based on model's default were considered for assessing health effects of NO₂. Furthermore, BI values were also taken from default of the model. For our population of one million people and base on BI of 497 per 100,000 people each year, some 4970 cardiovascular mortality cases can be expected annually, out of this number, 19 cases can be attributed to the NO₂ concentrations above 20 µg/m³. Eighty two percent of MI cases occurred in days with pollution not exceeding 110 µg/m³, Seventy seven percent of cardiovascular mortalities occurred in days with pollutant not exceeding 100 µg/m³ and 66% of COPD cases occurred in days with pollutant not exceeding 80 µg/m³. Although cardiovascular mortality had the lowest RR in centerline compared to MI and COPD high number of this health endpoint case was due to its higher BI value (497 per 100,000). On the other hand, greater number of acute cardiovascular mortality cases compared to MI cases was related to higher BI for acute cardiovascular mortality. Even in comparison between MI and COPD, BI had more importance than RR. In the interpretation of RR, it should be noted that a value of 1 for this

epidemiological parameter implies no impact on human health. In addition, severe effects can be expected when it exceeds from 1. Lack of cardiovascular mortality was referred to calculated RR value 1 and that is why the lowest line is attached on the X axis. It was also associated with being zero for both estimated AP and estimated number of excess cases. The lower level of RR value might be achieved if some control strategies for reducing NO₂ emission are used. Therefore, the higher RR value can depict mismanagement in urban air quality.

Gudarzi et al. exploited AirQ model to estimate the NO₂ hygienic effects in Tehran (capital of Iran). Based on their results, almost 3.4% of all cases of whole cardiovascular and respiratory mortalities are attributed to the NO₂ concentrations greater than 60 µg/m³ (23). Zalaghi et al. exploited AirQ model to estimate the NO₂ hygienic effects on Bushehr and Kermanshah. Based on their results almost 4 and 3 percent of all cases of cardiovascular and respiratory mortalities are attributed to the NO₂ concentrations greater than 40 µg/m³. (24).

To summarize, approximately 3 percent of total cardiovascular mortality, acute myocardial infarction, and hospital admission for COPD happened when the NO₂ concentration was over 20 µg/m³. Low percentage of the observed health endpoints was associated with low concentration of measured NO₂, and as it was mentioned previously, NO₂ concentration was lower than WHO and NAAQS guidelines' values.

Footnotes

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Conflict of Interest:

The authors declare no conflict of interest.

References

1. Bateson TF, Schwartz J. Who is sensitive to the effects of particulate air pollution on mortality? A case-cross over analysis of effect modifiers. *Epidemiology* 2004;15(2):143-9.
2. Oberdörster G, Finkelstein J, Ferin J, Godleski J, Chang LY, Gelein R, et al. Ultrafine particles as a potential environmental health hazard: studies with model particles. *Chest* 1996;109(3 Suppl):68S-69S.
3. Izzotti A, Parodi S, Quaglia A, Farè C, Vercelli M. The relationship between urban air pollution and short-term mortality: quantitative and qualitative aspects. *Eur J Epidemiol* 2000;16(11):1027-34.
4. Pope CA 3rd, Burnett RT, Thun MJ, Calle EE, Krewski D, Ito K, et al. Lung cancer, Cardiopulmonary, Mortality, and long term exposure to fine particulate air pollution. *JAMA* 2002;287(9):1132-41.
5. Schwartz, J., (2000). The distributed lag between air pollution and daily deaths. *Epidemiol* 11:320-6.
6. Reed MD, Gigliotti AP, McDonald JD, Seagrave JC, Seilkop SK, Mauderly JL. Health effects of subchronic exposure to environment levels of diesel exhaust. *Inhal Toxicol* 2004;16(4):177-93.
7. Pan Z, Molhave L, Kjaergaard SK. Effects on eyes and nose in humans after experimental exposure to airborne office dust. *Indoor Air* 2000;10(4):237-45.
8. Krzyzanowski M, Cohen A, Anderson R. Quantification of health effects of exposure to air pollution. *Occup Environ Med* 2002;59(12):791-3.
9. World Health Organization. AQG Air Quality Guidelines for Europe, 2nd edition. Copenhagen: WHO Regional Office for Europe, WHO Regional Publications, European Series, No. 91;2000. p:175-9.
10. QUARG. Air Quality in the United Kingdom. First report, Quality of Urban air review group. London: Department of Environment; 1993. p:331-2.
11. Jol A, Kielland G. Air pollution in Europe 1997: Executive summary. Copenhagen: European

Environment Agency, 1997 (EEA Environmental Monograph, No. 4); p:107.

12. Salway AG, Goodwin JWL, Eggleston HS, Murrells TP. UK emissions of air pollutants 1970–1994. National Environmental Technology Centre, AEA Technology, Culham; 1996. Report AEA/RAMP/20090001/R/003.

13. Expert Panel on Air Quality Standards. Nitrogen dioxide. London: The Stationery Office. 1996; Versions: 3 & 6:1.

14. Update and revision of the air quality guidelines for Europe: Meeting of the Working Group on Classical Air Pollutants. Copenhagen: WHO Regional Office for Europe; 1995.

15. Barnett AG, Williams GM, Schwartz J, Best TL, Neller AH, Petroschevsky AL, Simpson RW. The effects of air pollution on hospitalizations for cardiovascular disease in elderly people in Australian and New Zealand cities. *Environ Health Perspect* 2006;114(7):1018–23.

16. Sunyer J, Basagana X, Belmonte J, Anto JM. Effect of nitrogen dioxide and ozone on the risk of dying in patients with severe asthma. *Thorax* 2002;57(8):687-93.

17. Tsai SS, Goggins WB, Chiu HF, Yang CY. Evidence for an association between air pollution and daily stroke admissions in Kaohsiung, Taiwan. *Stroke* 2003;34(11):2612–6.

18. Yang CY, Chen YS, Yang CH, Ho SC. Relationship between ambient air pollution and hospital admissions for cardiovascular diseases in Kaohsiung, Taiwan. *J Toxicol Environ Health A* 2004;67(6):483-93.

19. Yang Q, Chen Y, Shi Y, Burnett RT, McGrail KM, Krewski D. Association between ozone and respiratory admissions among children and the elderly in Vancouver, Canada. *Inhal Toxicol* 2003;15(13):297–1308.

20. Gehring U, Heinrich J, Krämer U, Grote V, Hochadel M, Sugiri D, et al. Long-term exposure to ambient air pollution and cardiopulmonary mortality in women. *Epidemiology* 2006;17(5):545–51.

21. US Environmental Protection Agency. National Ambient Air Quality Standards (NAAQS): for air pollutant. US Environmental Protection Agency; 2010. Available from: <http://www.epa.gov/air/criteria.html> (Accessed 12 August 1999).

22. World Health Organization. WHO Air quality guidelines for particulate matter, ozone, nitrogen dioxide and sulfur dioxide: Summary of risk assessment, Global update. World Health Organization 2005. [Available at: www.euro.who.int/document/e90038.pdf]; p:16-7.

23. Goudarzi G, Nadafi K, Mesdaghiniya A. Quantification of health effects of air pollution in Tehran and determining the impact of a comprehensive program to reduce air pollution in Tehran on the third axis. PhD Dissertation, Tehran University of Medical Sciences, Tehran, Iran 2007.

24. Zalaghi E. Survey of health Effects of Air Pollution Ahvaz, Bushehr and Kermanshah with Use of AIRQ Model. MSc Dissertation, Islamic Azad University, Science and Research Branch, Ahvaz, Iran 2010.