



# Physiological Equivalent Temperature Index and mortality in Tabriz (The northwest of Iran)

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## ABSTRACT

There are few epidemiological studies about climate change and the effect of temperature variation on health using human thermal indices such as the Physiological Equivalent Temperature (PET) Index in Iran. This study was conducted in Tabriz, the northwest of Iran and Distributed Lag Non-linear Models (DLNM) combined with quasi-Poisson regression models were used to assess the impacts of PET on mortality by using the DLNM Package in R Software. The effect of air pollutants, time trend, day of the week and holidays were controlled as confounders. There was a significant relation between high (30 °C, 27 °C) and low (−0.8 °C, −9.2 °C and −14.2 °C) PET and total (non-accidental) mortality; and a significant increase in respiratory and cardiovascular deaths in high PET values. Heat stress increased Cumulative Relative Risk (CRR) for total (non-accidental), respiratory and cardiovascular mortality significantly (CRR<sub>Non Accidental Death, PET=30 °C, lag 0–30</sub> = 1.67, 95%CI: 1.31–2.13; CRR<sub>Respiratory Death, PET=30 °C, lag 0–13</sub> = 1.88, 95%CI: 1.30–2.72; CRR<sub>Cardiovascular Death, PET=30 °C, lag0–30</sub> = 1.67 95%CI: 1.16–2.40). Heat stress increases the risk of total (non-accidental), respiratory mortality, but cold stress decreases the risk of total (non-accidental) mortality in Tabriz which is one of the cold cities of Iran.

## 1. Introduction

Over the past few decades, there has been growing concerns and scientific debates about the negative effects of extreme temperature events on human health around the world (Parry et al., 2007; Solomon, 2007). Weather changes are a risk factor for health and many studies have been conducted to show the relation between environmental variables (especially air temperature) and mortality (Nastos and Matzarakis, 2012). Heat stress is associated with heart rate changes (Ghotbi Ravandi et al., 2016), and exposure to temperature outside of the thermal comfort zone can increase the risk of death and hospital admissions (Xu et al., 2013).

Thermal indices are used to examine and understand the effects of temperature stress on human health (Ndetto and Matzarakis, 2015). If a reasonable balance is created between the heat generated by the body and the heat lost, then the human is in a thermal comfort condition (Brager et al., 2004; Roshan et al., 2016b). In this case, there is no burden on the body's thermal adjustment system. By increasing thermal

stress (cold or heat), the cardiovascular system's activity increases to regulate body heat. After several days of thermal stress, "thermal adaptation" occurs, which makes it easier to deal with temperature stress. Exposure to extreme temperature stress increases the risk of physiological disorders, and physiologic disorders lead to health threats and even death in people with cardiorespiratory problems, children, and the elderly (Laschewski and Jendritzky, 2002). Ambient air temperature and the apparent temperature are the most common direct indicators of thermal stress (Xu et al., 2013). The increasing need for validated methods to estimate open air thermal comfort zones for meteorological services, urban planning, tourism and health; has led to the development of thermal indicators (Jendritzky et al., 2012; Roshan et al., 2016a). Brager et al. (2004) emphasizes that people's thermal comfort conditions are not solely determined by simple atmospheric and meteorological variables; but other parameters such as air humidity, wind speed, average radiant temperature, and physiological factors such as type of physical activity and clothing affect it as well.

The Physiological Equivalent Temperature is part of the Munich

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Energy-Balance Model for Individuals (MEMI) outputs. Although the Physiological Equivalent Temperature Index is one of the most comprehensive indicators for assessing meteorological conditions such as thermo-physiological stress (Esmaili et al., 2011), the use of the PET index in assessing the effect of thermal change or stress on mortality has been rare (Lin et al., 2012). Considering the lack of epidemiological studies in Iran that have addressed the effect of thermal changes on human health by human thermal indices such as PET, this study aimed to investigate the impact of PET index changes on mortality in Tabriz (northwestern Iran).

## 2. Materials and methods

### 2.1. Study site

Tabriz is the fifth largest city and one of the historical capitals of Iran and the capital of the East Azerbaijan Province (<http://www.i-dem.ir/Pages/Eng/AboutTabriz.aspx>, n.d.). In the 2017 census, its population was more than 1,600,000 people (<https://www.amar.org.ir>, 2017). Tabriz is situated at an altitude of 1361 m above sea level, and is georeferenced as 38°5'N and 46°16'E (<http://www.tabriz.clima-temps.com/map.php>, n.d.).

### 2.2. Data

Tabriz's death records were inquired from the death registration system of the Health Deputy at Tabriz University of Medical Sciences according to the International Classification of Death. ICD10 codes from 2010 to 2015 for a period of 6 years, were classified into age and gender subgroups. Mortality due to external factors such as accidents, suicide and murder (codes S and thereafter) were excluded and only death records from A00 to R99 were included in this study (Linares et al., 2015). Death cases were divided into three general categories as follows:

- A. Non-Accidental Deaths (NAD)(A00-R99)
- B. Respiratory Deaths(RD)(J00-J99)
- C. Cardiovascular Deaths(CVD)(I00-I99)

Meteorological data on daily ambient air temperature, wind speed, relative humidity and cloudiness was obtained from the Meteorological Organization of the Province. Cloudiness was reported for 8 times a day at 0, 3, 6, 9, 12, 15, 18 and 21 h.

Data about the concentration of air pollutants was acquired from the Environmental Protection Agency of the Province. Tabriz had 7 weather stations some of which were inactive at some point of time. The average of the recorded data of all active stations per day (24-h average of pollutants) were used in this study. The percent of missing data in NO<sub>2</sub>, SO<sub>2</sub> and PM<sub>10</sub> were 23%, 12% and 4%, respectively. The EM (Expectation-Maximization) method, which is a statistical method for missing imputation (Lin, 2010), was used to estimate missing data by SPSS software (version 22).The meteorological and mortality data was complete and did not have any missing.

### 2.3. PET index

PET index was presented in 1999 as a global benchmark for thermal stress assessment (Höppe, 1999). It could be deemed as the room temperature where the human body experiences a degree of thermal stress equal to the one experienced outdoors (Roshan et al., 2016b). In this study, the Rayman software was used to calculate the PET index values (Matzarakis et al., 2010, 2007).

The required variables to calculate the PET index include: A) geographical variables such as altitude and latitude of the area under study, B) meteorological variables such as dry air temperature (°C), relative humidity (%), wind speed(m/s)and cloudiness(octas); and C) individual

**Table 1**

Physiologically Equivalent Temperature (PET) for different grades of thermal sensation and physiological stress on human beings (during standard conditions).

PET (°C) in Iran <sup>a</sup>	Thermal sensation	Physiological stress level
< -10.7	Very cold	Extreme cold stress
-10.7 to -0.7	Cold	Strong cold stress
-0.7-8.8	Cool	Moderate cold stress
8.8-17.7	Slightly cool	Slight cold stress
17.8-27	Comfortable	No thermal stress
27-35.1	Slightly warm	Slight heat stress
35.1-43	Warm	Moderate heat stress
43-50.8	Hot	Strong heat stress
> 50.8	Very hot	Extreme heat stress

<sup>a</sup> Farajzadeh H. Evaluation and analysis of Climatic comfort conditions for tourism in Iran using bioclimatic Indices. Dissertation, Kharazmi University. 2017.

variables such as height, weight, age, gender, clothing and physical activity (W/m<sup>2</sup>).

The individual variables are the same as the physiological parameters that are included in the model. Given that the physiological characteristics vary in different people, standard parameters as follows were used in the model. Male gender, height = 175 cm, weight = 75 kg, age = 35 years, clothing = 0.9 (Clo), and level of physical activity = 80 W/m<sup>2</sup> (Roshan et al., 2016b).

Rayman software calculated 8 values per day for the PET index and then the eight values were averaged. After calculating the mean daily PET index, the PET classification in Table 1, presented for Iran's climatic conditions by Farajzadeh (2017), was used. In this table, the thermal comfort zone is set between 17.8 and 27 °C, which means in this zone no thermal stress is imposed on humans.

### 2.4. Statistics

Distributed Lag Non-linear Models (DLNM) combined with quasi-Poisson regression models were used to assess the impact of the PET index on mortality. The long term and seasonal trend of daily mortality was adjusted by using a natural cubic spline of time with 7 degrees of freedom (df) per year.

PM<sub>10</sub>, SO<sub>2</sub>, NO<sub>2</sub>and were controlled by using the stratified distributed lag model with 7 day lags (0–7) and 3 df. We created different breaks in the data, and eventually based on the Quasi Akaike Information Criteria (QAIC) we made 3 strata (lag 0–2, lag3–5 and lag 5–7) in our data, to use in constrained DLNM. Air pollutants are potential confounders in the association between environmental stressors and mortality (Buadong et al., 2009). We also controlled for the day of the week and holidays as categorical variables. The DLNM is developed based on a 'cross-basis' function, which allows simultaneous estimation of the non-linear effects across lags. It shows the relationship between PET and mortality at each mean daily value of PET and its lags. DLNM also calculates the cumulative effect of lagged variables (Gasparrini

**Table 2**

Descriptive statistics for Physiologically Equivalent Temperature (PET), Non Accidental Death (NAD), Respiratory Death (RD), Cardiovascular Death (CVD), air pollutants and age groups.

Variable	Mean (SD)	Median	N
PET(°C)	9(11.8)	8.9	2191
PM <sub>10</sub> (µg/m <sup>3</sup> )	77(38)	71.7	2191
SO <sub>2</sub> (µg/m <sup>3</sup> )	66(46)	60.5	2191
NO <sub>2</sub> (µg/m <sup>3</sup> )	47(33)	43.5	2191
NAD	18.8(5)	19	41,293
RD	1.9(1.5)	2	4269
CVD	8.6(3.5)	8	18,886
Age < 65	5.3(2.6)	5	11,616
Age 65–74	3.6(1.9)	3	8033
Age > =75	9.8(3.6)	9	21,623

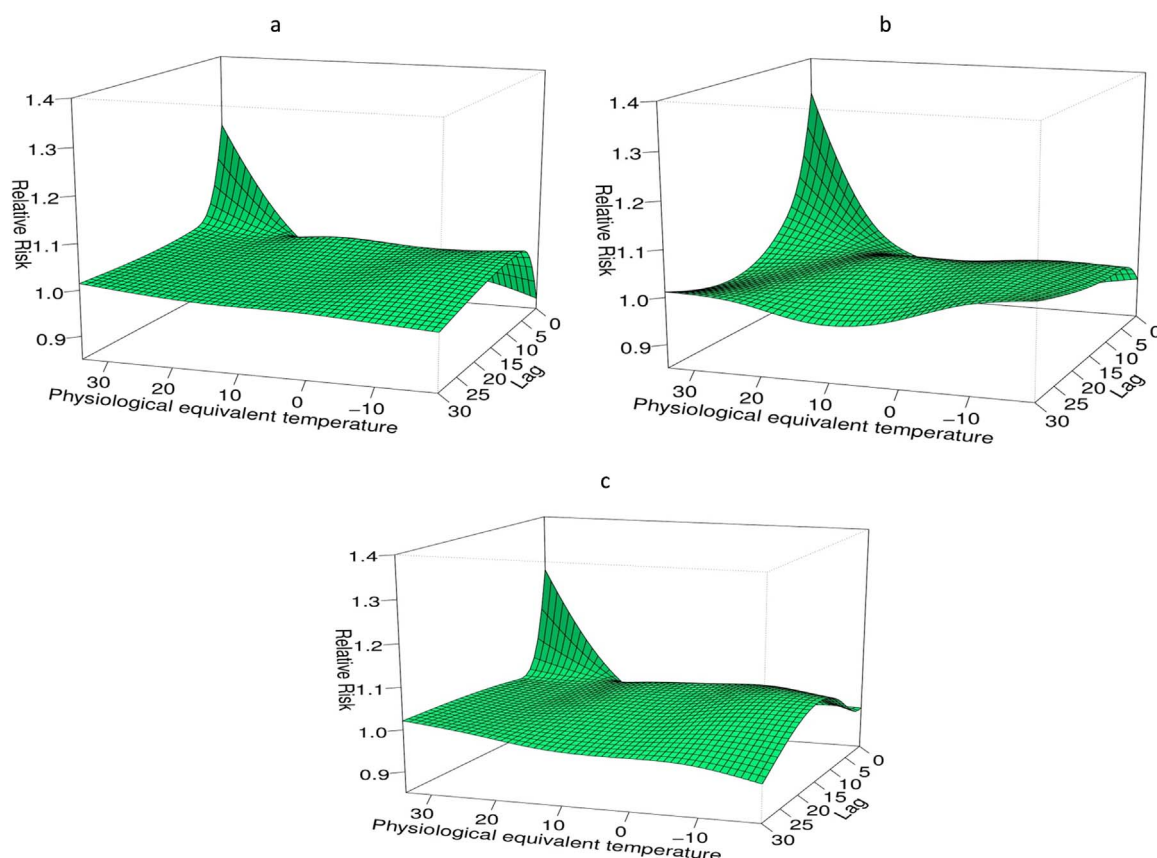


Fig. 1. Relative risks of non-accidental death (a), respiratory death (b) and cardiovascular death by Physiological Equivalent Temperature (PET), using a natural cubic spline-natural cubic spline DLNM with 5 df for PET and 3 df for lag.

et al., 2010). A ‘natural cubic spline–natural cubic spline’ DLNM was used in this study to model the non-linear PET and as well as the lagged effects. Spline knots were set at equally spaced values on the log scale of lags. A maximum lag of 30 days was used to completely capture the overall PET effect. The mean value of PET in the human thermal comfort strata (the average of 17.8 and 27, which is 22.5 °C) (Farajzadeh, 2017) was defined as the baseline PET (centering value) for calculating the relative risks. Akaike information criterion for quasi-Poisson (QAIC) models was used to choose the df (knots) for PET and lags (Gasparrini et al., 2010; Peng et al., 2006). We found that using 5 df for PET and 3 df for lags, produced the best fit in our models. The relative risks for PET and its lags were plotted to show the relationship between PET and mortality. In order to examine the thermal stress caused by heat on cause-specific and age-specific mortality, the relative risk of cause-specific and age-specific mortality associated with high PET (30 °C, 99th percentile of PET) relative to the 75th percentile of PET (19.4 °C) was calculated. In order to examine the thermal stress caused by cold on cause-specific and age-specific mortality, the relative risk of cause-specific and age-specific mortality associated with a low PET (−14.2 °C, 1st percentile of PET) relative to the 25th percentile of PET (−0.8 °C) was also evaluated (Yi and Chan, 2015).

We evaluated the model’s fitness using Q-AIC. Different df (degrees of freedom) were examined for PM<sub>10</sub>, SO<sub>2</sub> and NO<sub>2</sub>, and in various lags from 22 to 30 days, in different DLNM models. Statistical tests were two-sided, and  $p < 0.05$  was considered statistically significant.

We used R software (version 3.4.0; R Development Core Team 2017) to fit all models, with the ‘dlnm’ package (version 2.3.2) to create the DLNM.

### 3. Results

Descriptive statistics of the PET index, types of deaths, air pollutants and mortality in age groups are shown in Table 2.

The 3-dimensional Fig. 1(a, b, c) shows the relation between the PET index (mean daily value) changes and the death types. As it can be observed in Fig. 1(a, b, c), the relation between death and the PET index (mean daily value) is non-linear. At high values of PET and in the initial lags the relative risk of NAD,<sup>1</sup> RD,<sup>2</sup> and CVD<sup>3</sup> increases. At low values of PET, the relative risk of RD had no significant change. But the relative risk of CVD increased in the middle lags (9–19) and the relative risk of NAD decreased in the initial lags (Table 2–Supplementary).

The relation between PET (mean daily value) and mortality, in the first, 5th, 25th, 95th and 99th percentiles of PET compared with the value of 22.5 °C has been shown in Table 3. Since the PET value in the 75th percentile (19.4 °C) was smaller than 22.5 °C, the risk of death was not calculated in the 75th percentile. In NAD, at the 95 and 99th percentiles the cumulative relative risk (CRR) increased in all lags and the highest CRR was in lag 0–30. In RD at the 95 and 99th percentiles, the cumulative relative risk (CRR) increased up to lag 0–13. In cardiovascular deaths, CRR also increased in the 95th and 99th percentiles and this increase was highest in lag 0–30. There were no significant changes in low levels of the PET in RD and CVD; but in NAD, CRR decreased significantly in the 1st, 5th and 25th percentiles in the initial lags.

Table 4 shows the effects of intensified thermal stress (the 1st or 99th percentiles of cold or heat) and cause of death. Intensified cold stress did not have a significant effect on NAD, RD and CVD. But,

<sup>1</sup> Non-Accidental Death.

<sup>2</sup> Respiratory Death.

<sup>3</sup> Cardiovascular Death.

**Table 3**

The cumulative relative risks (mortality in low and high PET values relative to mortality in PET = 22.5 °C) stratified by cause-specific mortality.

Death type	PET value (°C)		Lag 0	Lag 0–2	Lag 0–6	Lag 0–13	Lag 0–20	Lag 0–30
Total	H <sup>a</sup>	30	<b>1.14</b> (1.10–1.19)	<b>1.32</b> (1.23–1.42)	<b>1.42</b> (1.31–1.54)	<b>1.50</b> (1.32–1.68)	<b>1.55</b> (1.31–1.83)	<b>1.67</b> (1.31–2.13)
		27	<b>1.07</b> (1.06–1.10)	<b>1.17</b> (1.13–1.21)	<b>1.21</b> (1.15–1.26)	<b>1.23</b> (1.15–1.31)	<b>1.25</b> (1.14–1.37)	<b>1.31</b> (1.15–1.49)
	L <sup>b</sup>	-0.8	<b>0.90</b> (0.84–0.97)	<b>0.82</b> (0.72–0.94)	<b>0.82</b> (0.68–0.98)	0.85 (0.66–1.10)	0.86 (0.62–1.19)	0.81 (0.53–1.23)
		-9.2	<b>0.91</b> (0.84–0.99)	<b>0.82</b> (0.71–0.96)	<b>0.81</b> (0.67–0.99)	0.91 (0.69–1.20)	0.99 (0.70–1.43)	0.96 (0.59–1.55)
		-14.2	<b>0.89</b> (0.82–0.97)	<b>0.80</b> (0.68–0.94)	<b>0.80</b> (0.65–0.98)	0.90 (0.68–1.19)	0.98 (0.69–1.40)	0.91 (0.57–1.46)
			<b>1.17</b> (1.04–1.33)	<b>1.48</b> (1.19–1.83)	<b>1.84</b> (1.44–2.35)	<b>1.88</b> (1.30–2.72)	1.66 (0.99–2.79)	1.59 (0.76–3.35)
Respiratory	H	30	<b>1.17</b> (1.04–1.33)	<b>1.48</b> (1.19–1.83)	<b>1.84</b> (1.44–2.35)	<b>1.88</b> (1.30–2.72)	1.66 (0.99–2.79)	1.59 (0.76–3.35)
		27	<b>1.08</b> (1.02–1.15)	<b>1.23</b> (1.11–1.37)	<b>1.39</b> (1.22–1.59)	<b>1.40</b> (1.14–1.71)	1.29 (0.97–1.72)	1.27 (0.86–1.89)
	L	-0.8	0.87 (0.69–1.10)	0.73 (0.48–1.14)	0.65 (0.37–1.15)	0.69 (0.31–1.57)	0.82 (0.28–2.37)	0.91 (0.23–3.62)
		-9.2	0.91 (0.70–1.17)	0.81 (0.50–1.31)	0.77 (0.41–1.44)	0.86 (0.34–2.13)	1.07 (0.33–3.53)	1.53 (0.31–7.57)
		-14.2	0.92 (0.70–1.21)	0.84 (0.51–1.38)	0.80 (0.42–1.53)	0.84 (0.34–2.08)	0.99 (0.31–3.20)	1.46 (0.30–7.06)
			<b>1.15</b> (1.08–1.22)	<b>1.33</b> (1.20–1.48)	<b>1.38</b> (1.23–1.56)	<b>1.41</b> (1.18–1.68)	<b>1.48</b> (1.16–1.90)	<b>1.67</b> (1.16–2.40)
Cardiovascular	H	30	<b>1.15</b> (1.08–1.22)	<b>1.33</b> (1.20–1.48)	<b>1.38</b> (1.23–1.56)	<b>1.41</b> (1.18–1.68)	<b>1.48</b> (1.16–1.90)	<b>1.67</b> (1.16–2.40)
		27	<b>1.08</b> (1.05–1.11)	<b>1.17</b> (1.12–1.24)	<b>1.19</b> (1.12–1.27)	<b>1.19</b> (1.08–1.31)	<b>1.21</b> (1.05–1.38)	<b>1.29</b> (1.06–1.56)
	L	-0.8	0.91 (0.82–1.01)	0.84 (0.69–1.02)	0.88 (0.68–1.15)	1.05 (0.73–1.53)	1.18 (0.74–1.89)	1.07 (0.59–1.95)
		-9.2	0.94 (0.84–1.06)	0.89 (0.72–1.11)	0.94 (0.70–1.24)	1.21 (0.81–1.82)	1.54 (0.92–2.58)	1.46 (0.74–2.91)
		-14.2	0.95 (0.84–1.07)	0.88 (0.71–1.11)	0.87 (0.65–1.16)	1.10 (0.74–1.64)	1.42 (0.85–2.36)	1.27 (0.65–2.49)

<sup>a</sup> High PET values.<sup>b</sup> Low PET values.**Table 4**

The cumulative relative risks of mortality in intensified thermal stress stratified by cause-specific mortality.

Cold effect <sup>a</sup>	Lag 0	Lag 0-2	Lag 0-6	Lag 0-13	Lag 0-20	Lag 0-30
NAD	0.99 (0.95–1.03)	0.97 (0.90–1.06)	0.98 (0.89 – 1.08)	1.06 (0.94–1.20)	1.14 (0.98–1.33)	1.24 (0.89 – 1.41)
Respiratory	1.05 (0.93–1.20)	1.14 (0.91 – 1.44)	1.23 (0.92–1.64)	1.22 (0.84 – 1.78)	1.21 (0.75–1.96)	1.61 (0.79–3.28)
Cardiovascular	1.04 (0.98–1.11)	1.05 (0.94 – 1.17)	0.98 (0.86–1.12)	1.04 (0.87 – 1.24)	1.19 (0.96–1.50)	1.18 (0.85–1.64)
Hot effect <sup>b</sup>						
NAD	<b>1.19</b> (1.13–1.24)	<b>1.43</b> (1.32–1.55)	<b>1.54</b> (1.39–1.70)	<b>1.59</b> (1.37–1.85)	<b>1.67</b> (1.35–2.07)	<b>1.85</b> (1.37–2.49)
Respiratory	<b>1.21</b> (1.06–1.39)	<b>1.62</b> (1.26–2.06)	<b>2.13</b> (1.57–2.89)	<b>2.16</b> (1.35–3.46)	1.80 (0.94–3.46)	1.75 (0.70–4.36)
Cardiovascular	<b>1.20</b> (1.13 – 1.29)	<b>1.45</b> (1.29–1.64)	<b>1.51</b> (1.30–1.74)	<b>1.48</b> (1.19–1.86)	<b>1.53</b> (1.12 – 2.08)	<b>1.78</b> (1.15 – 2.77)

<sup>a</sup> The cumulative effects of cold thermal stress on mortality categories, with 1st percentile of pet (–14.2 °C) relative to 25th percentile of pet (–0.8 °C).<sup>b</sup> The cumulative effects of hot thermal stress on mortality categories, with 99th percentile of pet (30 °C) relative to 75th percentile of pet (19.4 °C).**Table 5**

The cumulative relative risks of Non-Accidental mortality in intensified thermal stress stratified by age groups.

Cold effect <sup>a</sup>	Lag 0	Lag 0-2	Lag 0-6	Lag 0-13	Lag 0-20	Lag 0-30
< 65	1.001(0.92–1.08)	0.99(0.86–1.15)	0.97 ( 0.83–1.17)	1.06 ( 0.83–1.34)	1.18 (0.87 – 1.60)	1.25 ( 0.81–1.94)
65–74	1.005 ( 0.92–1.10)	1.03(0.87–1.22)	1.12 (0.92–1.37)	1.29 (0.99 – 1.68)	<b>1.44 (1.03–2.03)</b>	1.62 (0.98 – 2.66)
> =75	0.97 (0.91–1.04)	0.95 (0.85–1.06)	0.94 (0.82–1.07)	0.99 (0.83–1.17)	1.03 (0.83–1.28)	0.93 (0.68–1.28)
Hot effect <sup>b</sup>						
< 65	<b>1.25 (1.15 – 1.37)</b>	<b>1.49 (1.28–1.75)</b>	<b>1.37 (1.13–1.67)</b>	1.33 (0.99–1.79)	1.52( 1.00–2.29)	1.78( 0.99–3.20)
65–74	<b>1.14(1.031.25)</b>	<b>1.30 (1.09 – 1.55)</b>	<b>1.36( 1.09–1.69)</b>	1.34 (0.96–1.85)	1.29 (0.82 – 2.04)	1.28( 0.67–2.44)
> =75	<b>1.18(1.11 – 1.25)</b>	<b>1.45 ( 1.30–1.63)</b>	<b>1.71 ( 1.49 – 1.96)</b>	<b>1.87( 1.51–2.31)</b>	<b>1.93 (1.44–2.59)</b>	<b>2.13 ( 1.41–3.22)</b>

<sup>a</sup> The cumulative effects of cold thermal stress on mortality categories, with 1st percentile of pet (–14.2 °C) relative to 25th percentile of pet (–0.8 °C).<sup>b</sup> The cumulative effects of hot thermal stress on mortality categories, with 99th percentile of pet (30 °C) relative to 75th percentile of pet (19.4 °C).



intensified heat stress increased the CRR of CVD and NAD in all lags especially in the final lags, and also increased the CRR of RD significantly until lag 0–13.

Intensified cold stress in the 65–74 years old group increased the CRR of NAD in the final lags (lag 15–28) (Table 5). However, intensified heat stress increased the CRR of NAD in people under 65 and 65–74 years up to lag 0–6; and in the over 75-year-olds in all lags.

#### 4. Discussion

The present study showed that PET affects the risk of NAD, RD, and CVD. The immediate effect of PET (daily mean) fluctuations on mortality was bigger for high values (99th percentile) versus low values (1st percentile) (Table 3- Supplementary). Also there was a clear trend in higher values of PET; and the CRR<sub>(99th percentile)</sub> of NAD, RD and CVD had a greater value in all lags, while there was no clear pattern in low values of the PET index (Table 3). Probably adaption to the dominant climate of Tabriz (cold weather) and susceptibility and non-adaptation to heat stress is one of causes of these findings.

In low PET, the CRR/RR for RD during lags 0–30 was not significant and low PET did not have an effect on respiratory deaths. But, the CRR/RR for NAD, during initial lags and in low values of PET were significant, and low PET decreased the risk of NAD. While in CVD, RR increased significantly in some middle lags, but all CRR in all lags were insignificant.

Thach et al. conducted a study to investigate the clustering effect of thermal stress and the relation between PET and death in Hong Kong. In their study, 145 small-scale tertiary planning units (TPUs) were studied. Results showed that there was a spatial correlation between the monthly PET index and the standardized death rate; and with each 1 °C increase in PET in the warm season, the percentage of CVD excess risk increased by 4.31% (95% CI 0.12–4.33%); and with 1 °C reduction in PET index in the cold period, the NAD, CVD and RD death risk increased by 3% (95% CI 0.5% to 5.48%), 4.75% (95% CI 1.14–8.36%) and 7.39% (95% CI 4.64–10.10%) respectively (Thach et al., 2015). In the present study (done in Tabriz city with cold weather), cold stress (low values of daily mean PET) decreased the CRR of NAD in the initial lags, but heat stress led to an increase in the CRR of NAD, RD and CVD. While in Thach et al.'s study conducted in Hong-Kong (with subtropical climate) cold stress increased the risk of NAD, CVD and RD.

In the study done by Alfésio et al. to investigate the acute and lagged effects of temperature on cardiovascular and respiratory deaths in the US, cities were categorized into warm (Atlanta, Birmingham, and Houston) and cold cities (Canton, Chicago, Colorado Springs, Detroit, Minneapolis, New Haven, Pittsburgh, and Spokane) based on meteorological variables. In cold cities, heat and cold stresses increased the risk of cardiovascular death, but cold stress did not have a specific effect on respiratory death, while heat stress increased the risk of respiratory death (Braga et al., 2002). In Alfésio et al.'s study in warm cities, cold stress had no significant effect on cardiovascular and respiratory death, but thermal stress increased the risk of death from Chronic Obstructive Pulmonary Disease (COPD) and Myocardial infarction (MI) (Braga et al., 2002). Similar to Alfésio et al.'s results in cold cities, in the present study, heat stress increased the risk of RD and CVD.

Nastos et al. conducted a study to assess the effect of daily temperature, PET and UTCI<sup>4</sup> thermal indicators on daily mortality in 1992–2001 in Athens. The results showed a significant association between PET and mortality; and with each 10 °C decrease in PET the chance of death increased by 7% (Nastos and Matzarakis, 2012). In the present study at low PET levels in the initial lags there was a decreased risk of NAD. In Nastos et al.'s study, by each 10 °C increase in the PET index, the risk of death increased by 3% and this result was consistent with the results of this present study. In Nastos' study a U-shaped

relation was observed between ambient air temperature and PET with NAD.

Farajzadeh and Darand studied the relation between air temperature and cardiovascular, respiratory and stroke deaths in 2002–2005 in Tehran, Iran. Their results showed a significant inverse correlation between mean monthly temperature and cardiovascular, respiratory and stroke deaths and all three deaths increased in cold months of the year. In their study, average monthly temperature was inversely correlated with the total number of deaths ( $r = -0.87$ ), number of cardiovascular deaths ( $r = -0.93$ ), stroke deaths ( $r = -0.89$ ) and respiratory deaths ( $r = -0.86$ ) (Farajzadeh and Darand, 2009). In the present study, there was an insignificant increase in the CRR of CVD and RD in low PET (1st and 5th percentiles) in the final lags, but the CRR of NAD decreased significantly until lag 6. One reason for these different findings is that Iran is a country with enormous diversity in climate (Table 1).

Dadbakhsh et al. investigated the relationship between temperature and respiratory deaths in Shiraz, Iran. After controlling for confounders an inverse relation was found between mean monthly temperature and number of deaths per month and as it became colder, the number of respiratory deaths increased as well (Dadbakhsh et al., 2017). In the present study at lower PET and at final lags a non-significant increase was observed in CRR. Also intensified cold stress resulted in a non-significant increase in CRR of RD in all lags.

Wu et al. performed a study to assess the effects of temperature on NAD in the four subtropical cities of Changsha, Kunming, Guangzhou and Zhuhai in China. Their results showed that cold (1 °C decrease in temperature from the cold threshold in each city) had the highest relative risk in the final lag (lag 27) which was RR = 1.096, 95% CI: 1.075–1.117 in Guangzhou, RR = 1.111, 95% CI: 1.078–1.145 in Zhuhai, RR = 1.061, 95% CI: 1.023–1.099 in Changsha, and RR = 1.044, 95% CI: 1.034–1.055 in Kunming (Wu et al., 2013). In the present study, in low PET, CRR of NAD decreased significantly in initial lags (0–6). Different climate conditions between these two study locations (the Northwest of Iran with cold weather and four subtropical cities in China), is perhaps the main reason for these inconsistent results.

Dadbakhsh et al. assessed the simultaneous and delayed relation between monthly temperature and cardiovascular deaths, in Shiraz, Iran. Their results show there was no significant relation between temperature and cardiovascular deaths which differs from the present study. The probable cause of this difference is probably climate differences. Shiraz has a moderate climate, while Tabriz is a mountainous city with a cold climate.

In the study conducted by Wu the greatest heat effect (1 °C temperature increase from the heat threshold temperature in each city) was observed in Kunming (RR = 1.017, 95% CI: 1.004–1.030), and Zhuhai cities (RR = 1.023, 95% CI: 1.004–1.042) at lag zero. In Guangzhou, at lag 0–20, RR = 1.033, 95% CI: 1.021–1.045 was observed; and in Changsha, at lag 0–20, the RR = 1.020, 95% CI: 1.003–1.037 was observed (Wu et al., 2013), and this is consistent with the results of the present study where there was an increase in NAD risk at high PET. In Wu's study, the effect of cold and hot air was higher in the elderly (over 65 years old). In the present study, intensified heat stress increased the risk of death of NAD in all age groups, especially in the above 75 year olds. Also, intensified cold stress, especially in the final lags, increased the risk of NAD in the 65–75 year old age group.

Muthers et al. compared mortality caused by the heat wave in the summer of 2003 with other times (1970–2007) in Vienna. PET was used as the thermal index in this study. In days with a PET below 29 °C, the mean relative mortality was somewhat lower than the expected level (−1.8%, 95% CI: 2.1 to 1.5%); on days with moderate thermal stress, the mean relative mortality was 0.9% (95% CI 0.4–1.4%); and on days with severe thermal stress it had a significant increase of 13% (95% CI 11.1–14.7%). The results showed that high values of PET index had a significant effect on mortality (Muthers et al., 2010), which is

<sup>4</sup> Universal Thermal Climate Index.

consistent with the results of our study.

Sharafkhani et al. assessed the impact of Diurnal Temperature Range (DTR)<sup>5</sup> fluctuation on mortality in Urmia (Iran) and reported there was a significant relationship between DTR and mortality. There was a significant increase in total (non-accidental), respiratory and cardiovascular deaths in high DTR values; but no significant increase, in low DTR. Their results showed that temperature variation affects the risk of mortality (Sharafkhani et al., 2017), which was consistent with the results of the present study.

Revich et al. conducted a study to investigate the effect of temperature changes on death in Moscow, Russia; and a V-shaped relation was observed between mean daily temperature and mortality. Heat stress (1 °C increase in average daily temperature in the temperature range between 18 and 25 °C) resulted in a significant increase in daily NAD, CVD and RD by 2.8%, 4.7% and 8.7% respectively. Also, cold stress (1 °C reduction in average daily temperature in the temperature range of 18 to –10 °C) resulted in an increase in daily NAD, CVD and RD by 0.49%, 0.78% and 1.5%. The effects of thermal stress (heat and cold) were higher in the elderly (> 75 years) (Revich and Shaposhnikov, 2008). In this study, cold stress increased the risk of death especially in the final lags. In both studies, the effect of heat stress was much greater than that of cold stress and the effect of temperature changes on death was greater in the elderly.

Research shows that the shape and dimensions of the relation between temperature and death depend on several factors such as demographic characteristics and multiple atmospheric parameters (Nastos and Matzarakis, 2012). The threshold temperature after which adverse health effects happen in humans varies in different geographic regions (Armstrong et al., 2011; Khanjani, 2016). For example, the daily maximum temperature threshold in Athens (33 °C) is lower than the daily maximum daily temperature threshold in the city of Seville (41 °C) (Diaz et al., 2002). The temperature threshold (warm and cold) is different based on the PET index classification in different regions of the world; for example in North China (Lai et al., 2014), Europe (Matzarakis and Mayer, 1996) and Iran (Farajzadeh, 2017); temperatures less than –16, 4 and –10.7 are considered as the very cold threshold temperature respectively.

Researchers have reported in cities with warmer temperatures in Europe, coldness has a greater impact on death (Analitis et al., 2008; Healy, 2003). A study conducted by Khanjani et al. in Kerman, Iran; with desert climate also indicated that cold had a greater impact on death; and the risk of cardiovascular and respiratory death increased with decreased temperatures (Khanjani and Bahrampour, 2013).

A limitation of this study was that we did not evaluate the relation for traumatic deaths. A few researchers have reported that high temperature increased the risk of trauma deaths (Ranandeh Kalankesh et al., 2015).

## 5. Conclusion

It seems that physiological adaptability to the dominant climate of the region plays an important role in people's sensitivity to thermal stress. In the cold and mountainous city of Tabriz, heat stress and high PET increases the risk of CVD, NAD and RD; but cold stress (low PET) even decreased the risk of NAD in some circumstances.

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## Conflicts of interest

None

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## Competing interest

The authors declare no Competing interest.

## Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at <http://dx.doi.org/10.1016/j.jtherbio.2017.11.012>.

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<sup>5</sup> Diurnal Temperature Range.

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