Tabari Biomedical Student Research Journal

OPEN ACCESS

ORIGINAL: Association between Smoking and Hypertension: Data from a Cohort Study in West of Iran

Mehdi Moradinazar
Farid Najafi
Ebrahim Shakiba
Mohammad Amin Rezaei

Behavioral Diseases Research Center, Health Institute, Kermanshah University of Medical Sciences, Kermanshah, Iran

ARTICLE INFO

Submitted: 14 Feb 2024
Accepted: 10 Apr 2024
Published: 25 May 2024

Keywords:
Hypertension;
Smoking;
Blood pressure;
Current smoker;
Prevalence;
Iran

Correspondence:
Mohammad Amin Rezaei, Behavioral Diseases Research Center, Health Institute, Kermanshah University of Medical Sciences, Kermanshah, Iran.
.
Email:
rezaei13989898@yahoo.com
ORCID: 0000-0002-1284-5735

Citation:
Moradinazar M, Najafi F, Shakiba E, Rezaei M A. Association between Smoking and Hypertension: Data from a Cohort Study in West of Iran. Tabari Biomed Stu Res J. 2024; 6(2):24-35.

10.22034/6.2.24

ABSTRACT

Introduction: Smoking is important risk factors for many chronic disease and hypertension is main cause of the disability-adjusted life year (DALY) diseases. But the association between smoking as the most common risk factor and hypertension as the most common cardiovascular disease remains unclear. This study investigates the association between smoking and hypertension in the largest population-based study in western Iran.

Material and Methods: This population-based study was based on a recruitment phase of the Ravansar Non-Communicable Disease (RaNCD) cohort study. After inclusion criteria from the total of 10,065 participant's age 35-65years, 6958 participants were assessed.

Results: The lifetime prevalence of smoking was 46.6%, and 8.4% were current smokers. After control of confounding variables smoking after 30 years old, had 4.28 mmHg (-6.66, -1.90) reduction in the SBP than their counterparts. A dose-response model indicated that increasing the duration of smoking and intensity of exposure to SHS increased the SBP in both sexes, but increasing the number of cigarettes smoked per day increased the SBP in women and increasing the age of onset smoking decreased the SBP in women.

Conclusion: Physicians must be conscious of the risk of future hypertension in persistent smokers, passive smokers, and those who have stopped smoking. Stricter disciplinary measures and prevention policies e.g. prohibiting smoking in key public venues are recommended.

Introduction

Hypertension is a serious public health problem across the world. The number of hypertensive people is predicted to enhance from 118 million in 2000 to 214 million in 2025 (1). Hypertension is a well-documented risk factor for global disease burden and its complications annually account for 9.4 million deaths worldwide (2). It contributes to the burden of cardiovascular diseases (CVDs), stroke, and renal failure. It is responsible for 45% of deaths caused by CVDs and 51% of deaths caused by stroke (3).

Cigarette smoking is one of the significant public health challenges and attributes for almost 6.3 million deaths and 6.3% of global DALYs around the world (2). There are nearly one billion smokers worldwide, of

[DOI: 10.22034/6.2.24]

whom 80% live in less developed countries (4). Previous evidence has showed that smoking is a strong risk factor for some chronic diseases, such as cardiovascular events (5), lung cancer (6), kidney disease (7), and stroke (8).

Smoking may directly damage endothelial, consequently, lead to endothelial dysfunction, endothelium-dependent coronary vasodilation imperfection, plaque progression, and increased arterial stiffness (9). Therefore, endothelium damage thought to be effective in the pathophysiology of high blood pressure (BP) (10). Furthermore, studies have found that smoking creates changes in forearm hemodynamics which affects small and large arteries (11). Likewise, cigarette smoking leads to sympathetic activation, oxidative stress, vasoconstriction and increased heart rate (12) that are related to increment in inflammation markers (13), which are associated with hypertension (14).

Although, there is no consensus on the association between smoking and hypertension, several studies presenting a positive (15, 16), and others a reverse relationship (17, 18). Therefore, the effect of cigarette smoking on the development of hypertension, particularly its precise nature, remains unclear.

Given the importance of cigarette smoking, hypertension, and their interaction in determining the risk of chronic diseases such as cardiovascular diseases, we aimed to investigate the association between smoking habits and hypertension in the largest population-based study in western Iran. A better understanding of this association will help health authorities in tailoring health promotion and intervention.

Methods

Study design and population

This population-based study was based on a recruitment phase of the Ravansar Non-Communicable Disease (RaNCD) cohort study in Kermanshah University of Medical Sciences (KUMS). The RaNCD cohort study, as a part of the PERSIAN (Prospective Epidemiological Research Studies in Iran) Cohort that surveyed adults aged 35–65 years in Ravansar. It is located in western Iran close to Iraqi borders. Between November 2014, and February 2017, all participants who met the inclusion criteria were chosen to participate in the study, during which a total number of 10065 willing people signed the written consent form. The design and foundations of the PERSIAN cohort study were detailed here (19, 20).

Inclusion and exclusion criteria

Inclusion criteria in RaNCD cohort study were residency more than one year in that city, people aged 35-65 years who live at least 9 months of the year in the area, persons who were willing to participate and complete the study, people formally consent to participate and were able to communicate with the research team.

In this study, people with history of blood pressure-lowering medications (n=776), renal failure and kidney stones (n=1837), cancer (n=89), liver disease (n=1037), thyroid disease (n=762), and those with cardiovascular disease (n=1702) were excluded to eliminate confounding variables. Finally, out of 10065 participants in the cohort study, 6958 persons including 3774 men and 3184 women, were enrolled in the present study.

Data collection and quality control

In order to ensure that all procedures are carried out in compliance with the PERSIAN Cohort Protocol. For data gathering, participants were invited to the study site. Data were collected by our research team, who were well trained in study protocols for patient entry and data gathering. All completed questionnaires were checked and verified for errors by the quality control team (QC) and quality assurance (QA) before final analysis. The patients' national identification numbers were used to avoid duplicate recruitment.

Definition and measurements

Hypertension, known as high blood pressure,

[DOI: 10.22034/6.2.24]

was defined as SBP ≥140 mmHg and/or DBP≥90 mmHg (21). Each participant in the study has his BP measured twice at the entry to the study. BP was taken from both the left and right arm. Second BP from the right arm was recorded as a reference. Two consecutive readings were reported after the participants had been seated for 10 minutes. The measurements are made by a trained person using sphygmomanometers.

An automatic bioelectric impedance machine was used to assess the anthropometric measurements. Body mass index (BMI) was computed as body weight (kg) divided by height (m2). Participants with BMI <18.5 kg/m2, with 18.5 ≤ BMI ≤ 24.9 kg/m2, with 25.0 ≤ BMI ≤ 29.9 kg/m2 and with BMI ≥ 30 kg/m2 were defined as underweight, normal, overweight, and obese, respectively (22).

Participants with little or no educational literacy were defined as illiterate. Those who completed< 5 years and/or 6–9 years of education were defined as low–educated. The middle-educated and high-educated were considered as those who completed 10–12 years and ≥ 13 years of education, respectively. The wealth index (WI) was used as a representative of economic status. WI was made by applying principal component analysis (PCA) on Information related to infrastructure (e.g. drinking water supply), housing status (e.g. the number of rooms), durable goods (e.g. car), and other welfare facilities (23). Finally, the socio-economic status (SES) index was generated by WI and education levels. Study participants were categorized into 5 SES groups from the poorest (1st) to the richest (5th) quintiles.

We used a 21-item checklist to obtain information about the physical activity. The metabolic equivalent of task (MET) of each activity was calculated. We classified physical activity into three levels including low (24.0 ≤ MET≤ 36.5 hours per week), moderate (36.6≤ MET≤ 44.9 hours per week), and heavy (MET≥ 45 hours per week) (24). The nutritional condition was assessed by the Food Frequency Questionnaire (FFQ) questionnaire. The quality of nutrition was measured using Healthy Eating Index (HEI) – according to the 2015 guideline. Subjects were classified into 5 HEI groups from the unhealthy (1st) to the healthy (5th) quintiles (25).

The national health interview Survey (NHIS) was used to assess smoking conditions. NHIS was determined based on the number of cigarettes (the number of cigarettes smoked each day) and duration of smoking. Participants were categorized into three groups including non-smokers, ex-smokers, and current smokers. Those who stopped smoking cigarettes were defined as ex-smokers. Current smokers referred to persons who smoked at least 100 cigarettes in their lifetime and currently smoking every day or every few days. Those who had not smoked at least 100 cigarettes in their lifetime were defined as nonsmokers. Age of onset smoking was assessed according to the following question: “How old were you when you regularly begun smoking?” the secondhand smoke (SHS) exposures was assessed by the following questions: do any of your family members smoke cigarettes? How many hours per day do you inhale secondhand smoke? Do any smoke cigarettes at your home/ work and etc. when you are present?(26).

Statistical analysis

Data were described using mean ± standard deviation (SD) for quantitative data and frequencies and percentages were reported for qualitative data. Differences between subgroups were assessed using chi-square for categorical variables. The univariate and multiple linear regression analysis were used to assess determinates of SBP and DBP expressed by regression coefficients. Using the backward stepwise elimination method, those variables having no significant effects were deleted. β and 95% confidence intervals (CIs) were calculated.

The dose-response relationship between the numbers of cigarettes smoked per day, age of onset smoking, duration of smoking, and intensity of exposure to SHS with SBP was evaluated after adjustment for confounding factors. A test was considered statistically significant if the probability value (P-value)

[DOI: 10.22034/6.2.24]

was less than 0.05. All analyses were carried out using Stata software (version 14.2) (Stata Corp, College Station, TX, USA)

Ethical approval

The Research Ethics Committee at KUMS approved the study protocol (Ethics No. KUMS.REC.*1394.318*). Also, patients were informed about participating in the study and signed the consent form. Patient data were kept confidential with the access limited to two of researchers and the quality control physician.

Results

Of the 10065 participants in the RaNCD after exclusion criteria 6958 persons, 3774 (54.2%) men and 3184 (45.8%) women met for this study. 3756 participants (54.0%) were aged between 35 and 45 years. Most (90.1%) were married and nearly 38.0% had completed elementary (1-5 year educations). 3303(47.5%) had moderate physical activity (36.6-44.9 hours per week).

The prevalence of hypertension in the non-smokers without passive smoker, non-smokers with passive smoker, current smokers, and ex-smokers was 5.8%, 6.4%, 5.3%, and 11.5%, respectively. The prevalence of hypertension was greater in aged 56-65 years, Illiterate, diabetics, BMI ≥30, and ex-smoker (p-value≤0.05). The prevalence of hypertension in our population was 6.4% (448/6958), nearly 6.8% in men and 6.0% in women (**Table 1)**. Univariate linear regression analysis indicated a significant association between being an ex-smoker and increased SBP. Importantly, in the adjusted analysis after multivariable adjustment for sex, age, marital status, level of education, SES, drink alcohol, BMI, physical activity, diabetes mellitus, and HEI current smokers had a significantly lower SBP comparing with never smokers without passive smokers.

In crude analysis, ex-smokers at the DBP assessment were associated with a 3.08 mmHg (2.23, 3.94) higher DBP compared to never-smoking. In adjusted models, current smoking was associated with a 1.35 mmHg (-2.10, -0.59) lower DBP compared to never-smoking.

The crude analysis showed an association between inhaling SHS 2-5 hours per day and increased SBP, this association remained significant after making adjustments for potential confounding factors. Multivariate linear regression with category of initiation age of smoking demonstrated that the average SBP decreased by smoking initiation at older ages. Those who started to smoke after 30 years old, had 4.28 mmHg (-6.66, -1.90) reduction in the SBP than their counterparts. Also in adjusted models with categorical data there is not association between duration of smoked cigarettes with SBP in men and women (**Table 2)**.

Table 1. Baseline characteristics of participants.

Characteristic		Total	Hypertension		P-value
			Yes	No	
N (%)		6958(100)	448(6.4)	6510(93.6)	
Gender	Male	3774(54.2)	257(6.8)	3517(93.2)	0.170
	Female	3184(45.8)	191(6.0)	2993(94.0)	
Age group	35-45	3756(54.0)	123(3.3)	3633(96.7)	<0.001
	46-55	1955(28.1)	158(8.1)	1797(91.9)	
	56-65	1247(17.9)	167(13.4)	1080(86.6)	
Marital status	Married	6270(90.1)	402(6.4)	5868(93.6)	0.781
	Single	688(9.9)	46(6.7)	642(93.3)	
Educational level	Illiterate	1476(21.2)	160(10.8)	1316(89.2)	<0.001
	1-5 year	2634(37.9)	161(6.1)	2473(93.9)	
	6-9 year	1287(18.5)	64(5.0)	1223(95.0)	
	10-12 year	989(14.2)	34 (3.4)	955(96.6)	
	>13years	572(8.2)	29(5.1)	543(94.9)	

[DOI: 10.22034/6.2.24]

Table 1 Continue					
Socio economic status	Poor(first quantile)	1377(19.8)	89(6.5)	1288(93.5)	0.032
	2 quantile	1385(19.9)	97(7.0)	1288(93.0)	
	3 quantile	1371(19.7)	91(6.6)	1280(93.4)	
	4 quantile	1392(20.0)	104(7.5)	1288(92.5)	
	Rich (5 quantile)	1430(20.6)	67(4.7)	1363(95.3)	
Physical activity (MET hours per week)	24-36.5	1952(28.0)	126(6.5)	1826(93.5)	0.998
	36.6-44.9	3303(47.5)	212(6.4)	3091(93.6)	
	≥45	1703(24.5)	110(6.5)	1593(93.5)	
Drink alcohol	No	6570(94/4)	419(6/4)	6151(93/6)	0.392
	Yes	388(5/6)	29(7/5)	359(92/5)	
Diabetics	No	6565(94.4)	402(6.1)	6163(93.9)	<0.001
	Yes	3915(5.6)	46(11.8)	345(88.2)	
BMI (kg/m²)	Below 18.5	154(2.2)	6(3.9)	148(96.1)	<0.001
	18.5 – 24.9	2152(30.9)	91(4.2)	2061(95.8)	
	25.0 – 29.9	3021(43.4)	182(6.0)	2839(94.0)	
	30.0 and Above	1631(23.4)	169(10.4)	1462(89.6)	
Smoking status	Never smokers without passive smoker	2799(40.2)	162(5.8)	2637(94.2)	<0.001
	Never smokers with passive smoker	2657(13.2)	170(6.4)	2487(93.6)	
	Current smokers	919(8.4)	49(5.3)	870(94.7)	
	Ex-smoker	583(38.2)	67(11.5)	516(88.5)	
Healthy Eating Index	Unhealthy(first quantile)	2149(30.9)	137(6.4)	2012(93.6)	0.326
	2 quantile	1808(26.0)	110(6.1)	1698(93.9)	
	3 quantile	1631(23.5)	98(6.0)	1533(94.0)	
	Healthy (4 quantile)	1359(19.6)	102(7.5)	1257(92.5)	

Table 2. The univariate and multiple linear regression for evaluation of association between SBP and DBP, and smoking by adjusted important predictors.

Variables		Systolic blood pressure		Diastolic blood pressure	
		Crude β (95%CI)	Adjusted* β (95%CI)	Crude β (95%CI)	Adjusted β (95%CI)
Smoking status	Never smokers without passive smoker	Reference	Reference	Reference	Reference
	Never smokers with passive smoker	0.74(-0.08, 1.55)	0.11(-0.65, 0.89)	0.76(0.25, 1.27)	0.39(-0.09,0.88)
	Current smokers	-0.65(-1.80, 0.49)	-3.79(-4.98, -2.60)	0.22(-0.48, 0.93)	-1.35(-2.10, -0.59)
	Ex-smoker	4.69(3.31, 6.07)	-0.43(-1.79, 0.93)	3.08(2.23, 3.94)	0.57(-0.29, 1.43)
Second hand smoke in current smokers	Without Passive smoker	Reference	Reference	Reference	Reference
	<2 hours per day	.09(-0.77, 0.96)	-0.51(-1.33, 0.30)	0.27(-0.26, 0.80)	-0.08(-0.59, 0.43)
	2-5 hours per day	3.79(2.21, 5.36)	2.91(1.43, 4.39)	3.11(2.14, 4.07)	2.56(1.64, 3.49)
	>5 hours per day	0.15(-3.04, 3.34)	-0.63(-3.61, 2.35)	0.95(-1.00,2.90)	0.41(-1.45, 2.28)
Regular age of onset smoke	Never smokers	Reference	Reference	Reference	Reference
	<20 years	-0.96(-2.59, 0.66)	-3.85(-5.55, -2.15)	-0.10(-1.09, 0.89)	-1.51(-2.57, -0.45)
	21-29 years	-0.24(-1.90, 1.41)	-2.90(-4.61, -1.19)	0.14(-0.86, 1.15)	-1.12(-2.19, -0.06)
	>30 years	-1.08(-3.55, 1.37)	-4.28(-6.66, -1.90)	1.13(-0.37, 2.63)	-0.38(-1.87, 1.10)

[DOI: 10.22034/6.2.24]

Table 2 Continue

smoking intensity in current smokers	Never smokers	Reference	Reference	Reference	Reference
	Light	0.53(-1.41, 2.48)	-1.95(-3.86, -0.04)	1.02(-0.16, 2.21)	-0.20(-1.39, 0.99)
	Moderate	-0.73(-3.12, 1.65)	-3.49(-5.82, -1.17)	0.10(-1.35, 1.55)	-1.20(-2.65, 0.25)
	Heavy	-1.25(-2.72, 0.21)	-4.51(-6.09, -2.92)	-0.15(-1.04, 0.74)	-1.68(-2.68, -0.69)
Smoking duration	Never smokers	Reference	Reference	Reference	Reference
	0-4 years	-2.87(-7.12, 1.37)	-5.39(-9.44, -1.35)	1.54(-1.04, 4.13)	0.28(-2.24, 2.81)
	5-14 years	-0.62(-3.47, 2.22)	-2.09(-4.84, 0.65)	0.03(-1.70, 1.77)	-0.81(-2.53, 0.90)
	>15 years	-0.50(-1.74, 0.72)	-3.60(-4.96, -2.24)	0.16(-0.58, 0.91)	-1.29(-2.14, -0.44)

Adjusted by sex, age, marital of status, level of education, socio economic status, drink alcohol, BMI, physical activity, Diabetes mellitus, and Healthy Eating Index.

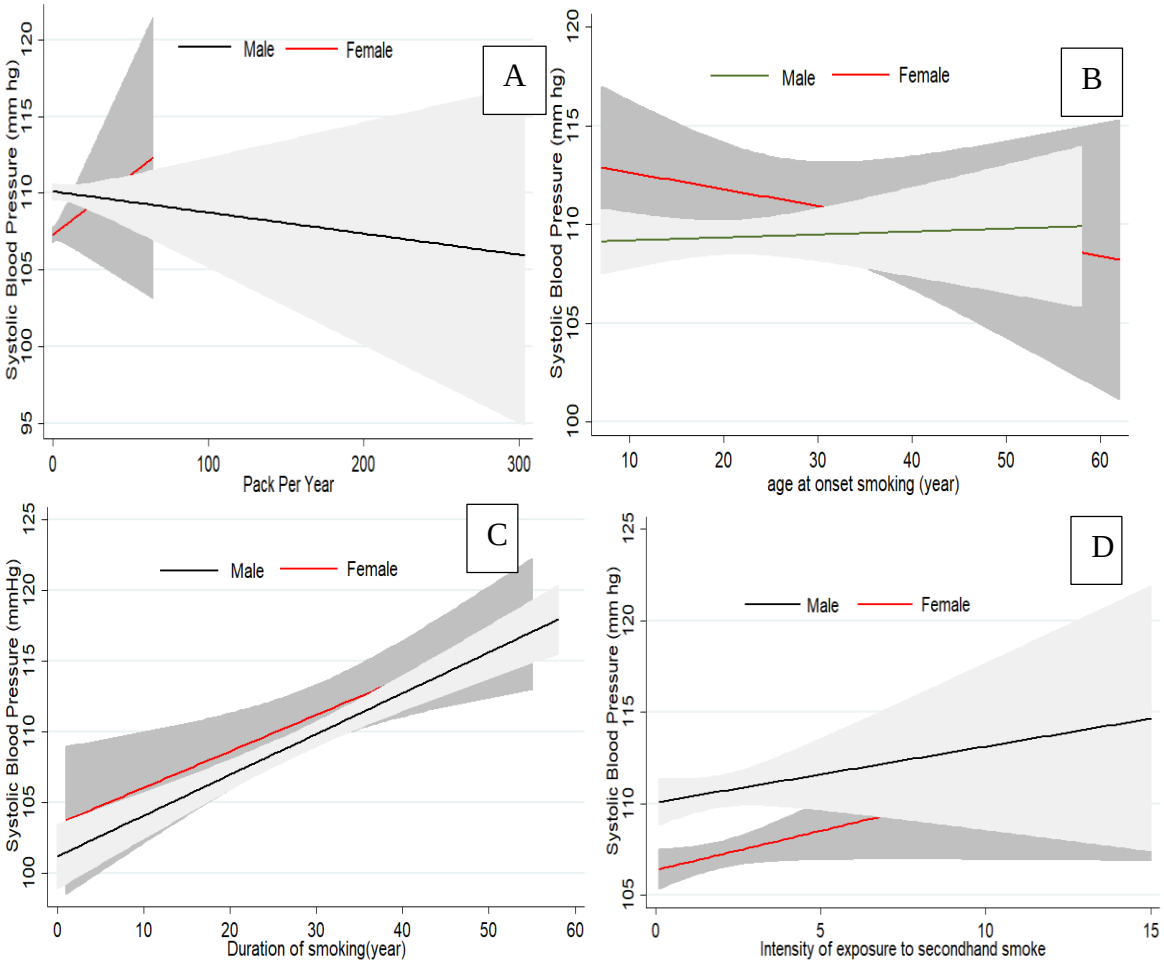

Figure 1. dose-response relationship between the numbers of cigarettes smoked per day (A), age of onset smoking (B), duration of smoking (C), and intensity of exposure to SHS (D) with SBP.

Figure 1 shows the dose-response relationship between the number of cigarettes smoked per day, age of onset smoking, duration of smoking, and intensity of exposure to SHS with SBP. After controlling the effect of confounding factors, with increasing the number of cigarettes smoked per day, the SBP was sharply increased in women. Although, the SBP slightly decreased with increasing the number of cigarettes smoked per day in men **(Fig. 1A)**. Also, the dose-response relationship indicated the role

[DOI: 10.22034/6.2.24]

of age of onset smoking in developing hypertension. So that, with an increase in the age of onset smoking, the SBP decreased in women, but with the increase in the age of onset smoking, the SBP increased with a slow slope **(Fig. 1B).** Furthermore, the SBP linearly increased with increasing the duration of smoking **(Fig. 1C)** and intensity of exposure to SHS **(Fig. 1D)** in both sex groups on the SBP had a steeper slope.

Discussion

Hypertension and smoking are important risk factors for many chronic disease and main causes of DALY. Given the population pyramid of Iran which forecasts the aging of the general population in the not too distant future, health authorities and healthcare providers should pay more attention to hypertension and smoking control. But a consensus has not been gained regarding the relationship between smoking and hypertension.The current study was designed to investigate the association between hypertension and cigarette smoking within a cohort study.

The prevalence of hypertension in our population was 6.4%, nearly 6.8% in men and 6% in women. This finding was similar to the result of a study conducted in Tehran that reported a hypertension prevalence of 5.2% (3.8% vs. 6.6% for men and women) (27). The overall prevalence of hypertension varies in different regions of Iran. In a systematic review published in 2019, in Iran, the overall prevalence of hypertension in the adult population older than 15 years old was 20.4% (28). However, this rate of hypertension prevalence in Ravansar is relatively lower than those reported from other cities of Iran. For example, reported rate is 15.1% vs. 21.4% for men and women in Isfahan (17.5%) (29), 9.0% vs. 14.0% in Mashhad (23.0%) (30), 18.7% vs. 25.8% in Khameneh (22.9%) (31), 17.5% vs. 17.6% in Ahvaz (17.6%) (32), and 37.1% vs. 39.0% in Shahroud (38.2 %) (33). This difference may be due to we excluded patients with renal failure and kidney stones, cancer, liver disease, thyroid disease, cardiovascular disease, and those who were on blood pressure-lowering medications, for the purpose of this study, and we included healthy persons. Therefore, cannot be generalized to our general population. In the main study, the prevalence of hypertension in our population was nearly 16% (34).

We observed that hypertension was more prevalent among people aged 56-65 years, Illiterate, diabetics, BMI ≥30, and ex-smoker. In accordance with our findings, Jamalizadeh and colleagues found that hypertension was more prevalent among the illiterate and 45-70 age group in 2016 (35).

Furthermore, the prevalence of current smoking among our participants aged 35 to 65 was about 8.4%; nearly 12.4% in men and less than 1% in women. Smoking is more popular among men. In Iranian culture, smoking is a stigma for women. Therefore, smoking by women may be underestimated. The prevalence rate of smoking in Ravansar is relatively lower in comparison with some cities of Iran including Bandar Abbas (11.7%) (36), Semnan (12.3%) (37), Hormozgan (19.5%) (38), and Bushehr (11.9%) (39), however, it needs to be noted that the prevalence rate of smoking in Ravansar is higher than one reported on the Birjand (7%) (40). This difference may be due to different study populations, methods, sample sizes, participants ages, and ethnicity. Need to say, Iran is a multicultural and multi-ethnic country. So, there is a significant difference in various geographical locations, various ethnicities, various socioeconomic and cultural statuses, and lifestyles (35).

In our study, the effect of smoking on hypertension was assessed with adjustments for lifestyle, age, BMI, and socio-demographic characteristics. The results of our study indicated that the adujsted mean of SBP and DBP of current smokers was lower than that of non- and ex-smokers. Okubo et al. performed a 5-year follow-up study to illuminate the effects of smoking on hypertension in Japanese men. They found that the adjusted mean of change in SBP and DBP of current smokers was significantly

[DOI: 10.22034/6.2.24]

lower than in non- and ex-smokers. And also, the cumulative incidence of hypertension in smokers was lower than in non- and ex-smokers (41). Savdie et al. carried out a case-control study, after adjustment for age, sex, and obesity, they reported a lower BP in smokers than non-smokers (42). Honda et al. reported a lower BP level among smokers than non-smokers after adjustment for alcohol consumption, smoking habit, and BMI (43). It is supposed that the current smokers are more healthy comparing to ex-smokers. Ex-smokers have been at higher risk of hypertension or other life-threatening diseases and therefore, they abandoned smoking due to doctor's recommendations. On the other hand, it has been found that BP after smoking cessation was increased (44). Lee et al. has done a 4-year follow-up study to assess the effect of smoking cessation on BP. Based on their findings, BP was increased after smoking cessation compared to current smokers and non-smokers (45). The rebound phenomenon and the adaptation process may be a possible description for smokers having lower BP than non- and ex-smokers. According to this assumption, after the first few puffs of a cigarette, BP elevates suddenly and after 1-2 hours revert to pre-smoking levels (46).

Our findings illustrated that the adjusted SBP in light, moderate and heavy smokers was significantly lower than in non-smokers and also, the adjusted DBP in heavy smokers was significantly lower than in non-smokers. In line with our results, Okubo et al found that the adjusted SBP and DBP in light, moderate and heavy smokers were significantly lower than in non- and ex-smokers (47).

We represented that increasing years of smoking was associated with increasing SBP in a dose-response fashion in both men and women. Thuy et al. reported that smoking duration was associated with a higher risk of hypertension in a dose-response model in a sample of Vietnamese men (17). Wenbin et al. showed that there is a dose-response relation between the duration of smoking and hypertension (48). Furthermore, we found that women who consumed more packyears of cigarettes have increased SBP, while the SBP slowly decreased with consuming more packyears of cigarettes by men. Conversely, Thuy et al. showed that men who consumed more packyears of cigarettes had a higher risk of hypertension after adjustment for confounding variables (17). In contrast, Jean-Michel et al reported that the risk of hypertension was associated with the number of cigarettes smoked in French men (49). Women have smoked a maximum of 100 packs per year and men smoked up to 300 packs per year, however, the SBP in women was higher than men. Besides, the physiological and anatomical characteristics of men and women are different. Women are assumed to be inherently more vulnerable, while men have more resilience.

We demonstrated that exposure to SHS was considerably related to higher SBP in a dose-response fashion in both men and women. These results are in agreement with several previous studies. Lei Wu et al. found that the persons who had more than 2 hours of daily passive cigarette smoking, increased mean values of both SBP and DBP (50). The Ohasama et al. conducted a study on Japanese females in 2010, they reported that passive smoking was associated with increased BP (51). Park et al observed that hypertension was more commonly associated with persons who had more than 2 hours of daily passive smoking (52).

The adjusted mean of SBP decreased by smoking initiation at older ages. Those who started to smoke after 30 years old, had a 4.28 mmHg (-6.66, -1.90) reduction in the SBP than their counterparts. However, smoking initiation at a younger age (<30years) was associated with lower DBP. There is no adequate information reported by some studies to compare this result. Morever, the dose-response relationship indicated with an increase in the age of onset smoking, the SBP decreased in women, but with the increase in the age of onset smoking, the SBP increased with a slow slope.

Strengths and limitations of the study

Our study faced several limitations: We used

[DOI: 10.22034/6.2.24]

self-reported data on smoking; such data might be less accurate compared with observation. The nature of the study design (cross-sectional) did not allow further evaluation about the direction of casualty between smoking and hypertension. This study has some strengths for example this was the first population-based study with a large sample done in western Iran. In addition, participants were evaluated by trained and experienced experts. also, people with a history of blood pressure-lowering medications, renal failure, kidney stones, cancer, liver disease, thyroid disease, and those with cardiovascular diseases were excluded to eliminate confounding variables. morever, the dose-response analysis was used to show relationship quantitative.

Conclusion

Current smokers had significantly lower SBP and DBP than their counterparts. smoking duration and exposure to SHS were considerably related to higher SBP in a dose-response modeles in both men and women. Women who consumed more packs per year have increased SBP, while the SBP slowly decreased with consuming more packs per year by men. With increasing the age of onset smoking, the SBP decreased in women, but the SBP has not changed in men. Physicians must be conscious of the risk of future hypertension in persistent smokers, passive smokers, and those who have stopped smoking. Stricter disciplinary measures and prevention policies including prohibiting smoking in key public venues, a ban on smoking in the home, higher taxation of tobacco products are recommended to reduce the burden of chronic diseases either directly or indirectly caused by smoking.

Acknowledgments

The RaNCD is part of the PERSIAN national cohort and we would like to thank Professor Reza Malekzadeh Deputy of Research and Technology at the Ministry of Health and Medical Education of Iran and Director of the PERSIAN cohort and also Dr. Hossein Poustchi Executive Director of the PERSIAN cohort for all their supports during design and running of RaNCD.

Conflicts of interest

Authors have no conflict of interests.

Authors' contributions

All authors were involved in the conception and design, analysis and interpretation of the data, drafting of the manuscript and revising it critically for intellectual content, approved the final version for submission, and agreed to be accountable for all aspects of the work.

Funding

This study was supported by the Ministry of Health and Medical Education of Iran and Kermanshah University of Medical Science (Grant No: 92472).

References

1. Kalhan SC, Prentice AM. Emerging Societies–Coexistence of Childhood Malnutrition and Obesity. In63º Nestlé Nutrition Institute Workshop, Pediatric Program, New Delhi 2008 Mar.
2. Lim SS, Vos T, Flaxman AD, Danaei G, Shibuya K, Adair-Rohani H, et al. A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990–2010: a systematic analysis for the Global Burden of Disease Study 2010. Lancet. 2012;380(9859):2224-60.
3. Organization WH. A global brief on hypertension: silent killer, global public health crisis: World Health Day 2013. World Health Organization; 2013.
4. Organization WH. Tobacco. 2020.
5. Tolstrup JS, Hvidtfeldt UA, Flachs EM, Spiegelman D, Heitmann BL, Bälter K, et al. Smoking and risk of coronary heart disease in younger, middle-aged, and older adults. American Journal of Public Health. 2014;104(1):96-102.

[DOI: 10.22034/6.2.24]

6. Hong S, Mok Y, Jeon C, Jee SH, Samet JM. Tuberculosis, smoking and risk for lung cancer incidence and mortality. International Journal of Cancer. 2016;139 (11):2447-55.
7. Yacoub R, Habib H, Lahdo A, Al Ali R, Varjabedian L, Atalla G, et al. Association between smoking and chronic kidney disease: a case control study. BMC Public Health. 2010;10(1):1-6.
8. Peters SA, Huxley RR, Woodward M. Smoking as a risk factor for stroke in women compared with men: A systematic review and meta-analysis of 81 cohorts, including 3 980 359 individuals and 42 401 strokes. Stroke. 2013;44(10):2821-8.
9. Sharrett AR, Ding J, Criqui MH, Saad MF, Liu K, Polak JF, et al. Smoking, diabetes, and blood cholesterol differ in their associations with subclinical atherosclerosis: the Multiethnic Study of Atherosclerosis (MESA). Atherosclerosis. 2006;186(2):441-7.
10. Ambrose JA, Barua RS. The pathophysiology of cigarette smoking and cardiovascular disease: an update. Journal of the American College of Cardiology. 2004;43(10):1731-7.
11. Berlin I, Cournot A, Renout P, Duchier J, Safar M. Peripheral haemodynamic effects of smoking in habitual smokers. A methodological study. Eur J Clin Pharmacol. 1990;38(1):57-60.
12. Barutcu I, Esen AM, Degirmenci B, Acar M, Kaya D, Turkmen M, et al. Acute cigarette smoking-induced hemodynamic alterations in the common carotid artery a transcranial Doppler study. Circ J . 2004;68(12):1127-31.
13. Bakhru A, Erlinger TP. Smoking cessation and cardiovascular disease risk factors: results from the Third National Health and Nutrition Examination Survey.PLoS Med. 2005;2(6):e160.
14. Sesso HD, Buring JE, Rifai N, Blake GJ, Gaziano JM, Ridker PM. C-reactive protein and the risk of developing hypertension. Jama. 2003;290(22):2945-51.
15. Halperin RO, Michael Gaziano J, Sesso HD. Smoking and the risk of incident hypertension in middle-aged and older men. Am J Hypertens. 2008;21(2):148-52.
16. Dochi M, Sakata K, Oishi M, Tanaka K, Kobayashi E, Suwazono Y. Smoking as an independent risk factor for hypertension: a 14-year longitudinal study in male Japanese workers. Tohoku J Exp Med. 2009; 217(1):37-43.
17. Thuy AB, Blizzard L, Schmidt MD, Luc PH, Granger RH, Dwyer T. The association between smoking and hypertension in a population-based sample of Vietnamese men. Journal of hypertension. 2010;28(2):245-50.
18. Li H, Tong W, Wang A, Lin Z, Zhang Y. Effects of cigarette smoking on blood pressure stratified by BMI in Mongolian population, China. Blood Pressure. 2010;19(2):92-7.
19. Poustchi H, Eghtesad S, Kamangar F, Etemadi A, Keshtkar A-A, Hekmatdoost A, et al. Prospective epidemiological research studies in Iran (the PERSIAN Cohort Study): rationale, objectives, and design. Am J Epidemiol. 2018;187(4):647-55.
20. Eghtesad S, Mohammadi Z, Shayanrad A, Faramarzi E, Joukar F, Hamzeh B, et al. The PERSIAN cohort: providing the evidence needed for healthcare reform. Arch Iran Med. 2017;20(11):691-5.
21. Whelton PK, Carey RM, Aronow WS, Casey DE, Collins KJ, Dennison Himmelfarb C, et al. 2017 ACC/AHA/ AAPA/ABC/ACPM/AGS/APhA/ASH/ASP C/NMA/PCNA guideline for the prevention, detection, evaluation, and management of high blood pressure in adults: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. J Am Coll Cardiol. 2018;71(19):e127-e248.
22. Organization WH. Waist circumference and waist-hip ratio: report of a WHO expert consultation, Geneva, 8-11 December 2008. 2011.
23. Rustein SO, Johnson K. The DHS wealth index. 2004.
24. Karyani AK, Matin BK, Soltani S, Rezaei S, Soofi M, Salimi Y, et al. Socioeconomic gradient in physical activity: findings from the PERSIAN cohort

[DOI: 10.22034/6.2.24]

study.BMC Public Health. 2019;19(1):1-11.
25. Krebs-Smith SM, Pannucci TE, Subar AF, Kirkpatrick SI, Lerman JL, Tooze JA, et al. Update of the healthy eating index: HEI-2015. J Acad Nutr Diet. 2018;118(9): 1591-602.
26. Hamzeh B, Farnia V, Moradinazar M, Pasdar Y, Shakiba E, Najafi F, et al. Pattern of cigarette smoking: intensity, cessation, and age of beginning: evidence from a cohort study in West of Iran. Subst Abuse Treat Prev Policy. 2020;15(1):1-9.
27. Cheraghian B, Asadi-Lari M, Mansournia MA, Majdzadeh R, Mohammad K, Nedjat S, et al. Prevalence and associated factors of self-reported hypertension among Tehran adults in 2011: a population-based study (Urban HEART-2). Med J Islam Repub Iran. 2014;28:105.
28. Afsargharehbagh R, Rezaie-Keikhaie K, Rafiemanesh H, Balouchi A, Bouya S, Dehghan B. Hypertension and pre-hypertension among Iranian adults population: a meta-analysis of prevalence, awareness, treatment, and control. Curr Hypertens Rep. 2019;21(4):27.
29. Pakzad B, Akbari M, Baberi F. Prevalence, awareness, treatment, and control of hypertension in an Isfahan state institution sample. J Tehran Heart Cent. 2018;13(2):65.
30. Ebrahimi M, Heidari-Bakavoli AR, Mazidi M, Moohebati M, Azarpazhooh MR, Nematy M, et al. Prevalence of hypertension, pre-hypertension and undetected hypertension in Mashhad, Iran. Mediterranean Journal of Nutrition and Metabolism. 2016;9(3):213-23.
31. Ghorbani Z, Shamshirgaran SM, Ghaffari S, Sarbakhsh P, Najafipour F, Aminisani N. Hypertension prevalence, awareness, treatment and its correlates among people 35 years and older: Result from pilot phase of the Azar cohort study. J Edu Health Promot. 2018;7.
32. Yazdanpanah L, Shahbazian H, Shahbazian H, Latifi S-M. Prevalence, awareness and risk factors of hypertension in southwest of Iran. J Renal Inj Prev. 2015;4(2):51.
33. Khosravi A, Emamian MH, Shariati M, Hashemi H, Fotouhi A. The prevalence of pre-hypertension and hypertension in an Iranian urban population. High Blood Press Cardiovasc Prev. 2014;21(2):127-35.
34. Rajati F, Hamzeh B, Pasdar Y, Safari R, Moradinazar M, Shakiba E, et al. Prevalence, awareness, treatment, and control of hypertension and their determinants: Results from the first cohort of non-communicable diseases in a Kurdish settlement.Scientific reports. 2019;9(1):1-10.
35. Jamalizadeh A, Kamiab Z, Nadimi AE, Nejadghaderi M, Saeidi A, Porkarami A. Prevalence of smoking and high blood pressure, two major risk factors for non-communicable diseases: the SuRF NCD (surveillance of risk factors of non-communicable disease) report 2012. J Cardiovasc Thorac Res. 2016;8(4):183.
36. Aghamollaei T, Zare S. Pattern of smoking and water pipe in the population over 15 years ofBandar Abbas, a population study. Hormozgan Med J. 2007;11(4):241-6.
37. Ghorbani R, Malek M, Eskandarian R, Rashidy-Pour A. Epidemiology of smoking in an Iranian population (Semnan province): a population-based study. Koomesh. 2012;13(2):Pe247-53.
38. Farshidi H, Aghamolaei T, Soleimani Ahmadi M, Madani AH. Epidemiological study of cigarette smoking among over 15 years old population of Hormozgan in 2014. Journal of Preventive Medicine. 2016;3(2):29-35.
39. Yousefi F, Darabi H, Nabipour I, Assadi M, Vahdat K, Kardeh E, et al. Prevalence of tobacco smoking in Bushehr Province: Comparison of two phases of the Persian Gulf healthy heart study. Iran South Med J. 2014;17(3):487-95.
40. Ziaee M, Hajihosseini M, Sharifzadeh G, Kazemi T, Azarkar G, Saljoughi M. Prevalence of Cigarette Smoking and Related Factors in Birjand, Iran During Year 2014. Mod Care J. 2016;13(2).
41. Okubo Y, Suwazono Y, Kobayashi E, Nogawa K. An association between smoking habits and blood pressure in normotensive Japanese men: a 5-year follow-up study. Drug and alcohol dependence. 2004;73(2):167-74.
42. Savdie E, Grosslight GM, Adena MA.

[DOI: 10.22034/6.2.24]

Relation of alcohol and cigarette consumption to blood pressure and serum creatinine levels. Journal of Chronic Diseases.1984;37(8):617-23.
43. Handa K, Kono S, Ishii H, Shinchi K, Imanishi K, Arakawa K. Relationship of alcohol consumption and smoking to plasma cortisol and blood pressure. J Hum Hypertens. 1994;8(12):891-4.
44. Green MS, Jucha E, Luz Y. Blood pressure in smokers and nonsmokers: epidemiologic findings. Am Heart J. 1986;111(5):932-40.
45. Lee D-H, Ha M-H, Kim J-R, Jacobs Jr DR. Effects of smoking cessation on changes in blood pressure and incidence of hypertension: a 4-year follow-up study. Hypertension. 2001;37(2):194-8.
46. Hansen KW, Pedersen MM, Christiansen JS, Mogensen CE. Night blood pressure and cigarette smoking: disparate association in healthy subjects and diabetic patients. Blood Pressure. 1994;3(6):381-8.
47. Okubo Y, Miyamoto T, Suwazono Y, Kobayashi E, Nogawa K. An association between smoking habits and blood pressure in normotensive Japanese men. J Hum Hypertens. 2002;16(2):91-6.
48. Hu W, Zhang T, Shi J, Qin W, Tong L, Shen Y. Association between cigarette smoking and hypertension in men: a dose response relationship analysis. Zhonghua Xin Xue Guan Bing Za Zhi. 2014;42(9):773-7.
49. Halimi J-M, Giraudeau B, Cacès E, Nivet H, Tichet J. The risk of hypertension in men: direct and indirect effects of chronic smoking. Journal of hypertension. 2002;20(2):187-93.
50. Wu L, Yang S, He Y, Liu M, Wang Y, Wang J, et al. Association between passive smoking and hypertension in Chinese non-smoking elderly women. Hypertens Res. 2017;40(4):399-404.
51. Seki M, Inoue R, Ohkubo T, Kikuya M, Hara A, Metoki H, et al. Association of environmental tobacco smoke exposure with elevated home blood pressure in Japanese women: the Ohasama study. Journal of Hypertension. 2010;28(9):1814-20.
52. Park YS, Lee C-H, Kim Y-I, Ahn CM, Kim JO, Park J-H, et al. Association between secondhand smoke exposure and hypertension in never smokers: a cross-sectional survey using data from Korean National Health and Nutritional Examination Survey V, 2010–2012. BMJ Open. 2018;8(5).

[DOI: 10.22034/6.2.24]