The association between hypertension and different types of dietary carbohydrates

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Background Hypertension is the most prominent and well acknowledged chronic disease in developed countries and is a significant contributor to global mortality. The present study aimed to investigate the association between hypertension and different types of dietary carbohydrates.

Method This cross-sectional study was conducted on 4184 people aged 35–70, including 1239 patients with hypertension and 2945 subjects with normal blood pressure (BP) in Sabzevar, Iran. The dietary intake data were collected through the administration of a semiquantitative Food Frequency Questionnaire. Utilizing Nutritionist IV software, dietary glucose, fructose, simple sugar, carbohydrate, and galactose consumption were evaluated.

Results A direct association was found between dietary glucose and BP (odds ratio: 1.02; 95% CI: 1.01–1.05; $P=0.04$). This association remained significant after adjusting for age. Further adjustments for education, marital status, job, physical activity, and BMI, and additional adjustments for energy intake did not change the results.

Conclusion In summary, the present study identified a significant association between hypertension and dietary intake of glucose. Considering that carbohydrates are an essential part of the diet worldwide, these findings can be valuable in formulating dietary interventions for hypertensive patients. *Cardiovasc Endocrinol Metab* 13: 1–6 Copyright © 2024 The Author(s). Published by Wolters Kluwer Health, Inc.

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Introduction

Hypertension has been recognized as the major chronic disorder in developed countries, a significant contributor to cardiovascular disease (CVD) mortality among modifiable CVD risk factors, and one of the main causes of death worldwide [1,2]. The global prevalence of hypertension was estimated at 34% in 2019 and the prevalence of hypertension among adults had an alarming rise from 594 million in 1975 to 1.13 billion in 2015 [3]. This increase was mostly seen in low-income and middle-income countries [4]. According to the estimates, the prevalence of hypertension is 15–35% and 18–23% in Asia and Iran, respectively [5].

Hypertension may lead to a range of consequences, including myocardial infarction, stroke, and chronic kidney disease, all of which contribute to increased frequency of hospitalization and health care costs [6]. Hypertension arises from a complex interplay of ecological, genetic, and behavioral factors [7], and nutritional behaviors have a significant role in the development of hypertension [8]. A healthy diet has a significant role in preventing and controlling hypertension [9,10].

Asian countries tend to have a larger proportion of energy derived from carbohydrates (approximately 60% of total calories). In contrast, North American and European countries consume less carbohydrates (generally ≤50% of total calories) [11,12]. The evidence of macronutrients regarding the effect on blood pressure (BP) is strong. However, most investigations focus on protein and fat intakes, with dietary carbohydrates receiving less attention [13]. Some investigations have provided evidence of an independent association between total carbohydrate consumption and hypertension or CVD, although with disputed findings. A Chinese national cohort study examined the relationship between carbohydrate quality and new-onset hypertension. The results of the study indicated that the consumption of low-quality carbs, such as refined rice or noodles, was associated with an elevated risk of hypertension. Conversely, the consumption of high-quality carbohydrates, such as whole grains, legumes, or fruits, was shown to be associated with a reduced risk of developing hypertension [14]. Several possible mechanisms have been proposed for the effect of carbohydrates on BP. High intake of low-quality carbohydrates may increase hyperglycemia and oxidative stress, adversely affecting endothelial vasodilation and increasing BP [15]. On the contrary, whole grains, non-starchy vegetables, fruits, and high-quality carbohydrates that are rich in flavonoids and antioxidants have the potential to reduce BP, enhance endothelial function, and minimize oxidative stress. Thus, there may be an association between a restricted consumption of nutritious carbohydrates and a greater risk of hypertension [16,17].

Differences in the results referring to the association between dietary carbohydrates and hypertension could potentially be attributed to variances in the absorbing, metabolism, and impacts of distinct types of dietary carbohydrates. Currently, there is no consensus on the association between different types of carbohydrates and hypertension. For example, according to the findings of a systematic review, a high consumption of fructose in the form of added sugar is independently related to higher BP [18]. Meanwhile, another systematic review suggested that fructose intake was not associated with an increased risk of hypertension [19]. The findings of this study may be especially relevant in Asian nations with higher carbohydrate consumption, such as Iran. So, the purpose of this study was to develop an initial understanding of the possible relationship that exists between hypertension and different types of dietary carbohydrates.

Methods

This cross-sectional study was conducted on 4184 people aged 35–70 including 1239 people with hypertension and 2945 people with normal BP in Sabzevar, Iran. The census sampling method of a geographical region was chosen. As to the guidelines established by the WHO [20], hypertension is characterized by SBP ≥140 mmHg and/or DBP ≥90 mmHg and/or a history of hypertension or currently using antihypertensive drugs. Inclusion criteria consisted of no family history of hypertension, no treatment with drugs affecting BP, and no current use of carbohydrate supplements. Excluding criteria was the inability to collect the required information.

Data collection

The sample was obtained from the Persian cohort research conducted in Sabzevar, Iran, lasting from January 2017 to May 2020. During the phone call, the participants were requested to provide demographic information. The collection of general and medical data was conducted via in-person interviews facilitated by interviewers who had received appropriate training. The assessment of physical activity levels was conducted using a short version of the International Physical Activity Questionnaire [21]. Without shoes and to the nearest 0.1 kg, the body weight of participants wearing lightweight clothes was determined using a calibrated beam scale (Seca 755 mechanical column scale, SECA, Hamburg, Germany). The height of people was measured using a portable stadiometer with an accuracy of 0.1 cm (Seca 204 mobile stadiometers, SECA, Hamburg, Germany). The calculation of the BMI was performed as follows: BMI = weight (kg)/[height (m)^2].

Dietary intake

The data collected from the Food Frequency Questionnaire (FFQ) of the Persian cohort, whose validity and reliability have already been confirmed, were used to assess the dietary intake of the participants. The data from the FFQ was analyzed using the Nutritionist IV software (The Hearst Corporation, San Bruno, California, USA) in terms of the consumption of carbohydrates in the diet, including glucose, fructose, galactose, sucrose, maltose, and lactose. In this assessment, the amount of carbohydrates in each food was calculated at first. Then, it was estimated for food consumed by each person.

Medical and biochemical data

BP was calculated using the mean of three consecutive measures taken after 10 min of seated rest using standard mercury sphygmomanometers. During the examination, participants' blood was drawn after one night of fasting, and their serum was separated and stored at −70 °C. Data related to BP and blood tests, including right SBP (RSBP), right DBP (RDBP), fasting blood sugar (FBS), triglyceride, cholesterol, low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), serum glutamic oxaloacetic transaminase

(SGOT), serum glutamic pyruvic transaminase (SGPT), alkaline phosphatase (ALP), gamma glutamic transferase (GGT), creatinine, and blood urea nitrogen (BUN) were obtained with the coordination of the relevant official.

Statistical analysis

For quantitative data, the independent *t*-test or its non-parametric equivalent (Mann–Whitney) was used, and for qualitative variables, the chi-square test was utilized. To investigate the association between high BP and different types of carbohydrates in the diet, logistic regression method and odds ratio (OR) estimation were used to analyze the chance of developing the disease using the following models: Model 1 was adjusted for age and gender, Model 2 was further adjusted for education, marital status, job, physical activity, and BMI, and model 3 was additionally adjusted for energy intake. All analyses were performed using R Studio software (R studio, Boston, Massachusetts, USA) at a significance level of $P < 0.05$.

Results

Table 1 indicates the characteristics of the participants. In this study, the patients with hypertension were older (53.4 ± 8.3 vs. 47.4 ± 8.35 years, $P < 0.001$) and had higher BMI (29.2 ± 4.7 vs. 27.7 ± 4.7 kg/m^2, $P < 0.001$), RSBP (134.12 ± 0 vs. 106.23 ± 10.0 mmHg, $P < 0.001$), RDBP (82.95 ± 7.6 vs. 67.33 ± 7.0 mmHg, $P < 0.001$), FBS (116.89 ± 49.27 vs. 103.0 ± 35.51 mg/dl, $P < 0.001$), BUN (14.35 ± 3.9 vs. 13.46 ± 3.6 mg/dl, $P < 0.001$), creatinine (1.14 ± 0.28 vs. 1.07 ± 0.18 mg/dl, $P < 0.001$), triglyceride (166.58 ± 121.3 vs. 137.8 ± 92 mg/dl, $P < 0.001$), cholestrol (195.48 ± 42.62 vs. 189.9 ± 39.06 mg/dl, $P < 0.001$), SGOT (20.91 ± 9.59 vs. 19.89 ± 8.63 U/l, $P < 0.001$), SGPT (24.01 ± 14.75 vs. 21.31 ± 15.41 U/l, $P < 0.001$), ALP (234.13 ± 71.13 vs. 217.24 ± 66.04 U/l, $P < 0.001$), LDL-C (111.23 ± 35.3 vs. 109.89 ± 33.02 mg/dl, $P < 0.001$), and GGT (28.85 ± 24.21 vs. 24.09 ± 21.16 U/l, $P < 0.001$) compared with the healthy people. The hypertension group had lower mean physical activity (38.1 ± 7.7 vs. 38.84 ± 7.8, $P \leq 0.008$) and HDL-C (51.11 ± 10.47 vs. 52.87 ± 10.66 mg/dl, $P < 0.001$) than the others.

The dietary intakes of the participants are shown in Table 2. The hypertension group had significantly higher dietary intake of carbohydrates (425.44 ± 144.70 vs. 407.77 ± 135.06 g/day, $P = 0.0001$), energy (2549.93 ± 818.39 vs. 2477.77 ± 770.36 kcal/day, $P = 0.001$), glucose (28.98 ± 17.15 vs. 27.23 ± 16.03 g/day, $P < 0.001$), fructose (33.58 ± 19.33 vs. 31.75 ± 18.37 g/day, $P = 0.001$), maltose (0.53 ± 0.68 vs. 0.51 ± 0.63 g/day, $P = 0.001$), galactose (0.2 ± 0.22 vs. 0.18 ± 0.19 g/day, $P = 0.002$), sucrose (47.39 ± 34.35 vs. 45.79 ± 30.41 g/day, $P = 0.02$), and glucose (143.1 ± 66.76 vs. 137.55 ± 62.85 g/day, $P = 0.002$) than healthy subjects. Also, the patients with hypertension had lower dietary intake of total lipids (64.31 ± 25.06 vs. 64.58 ± 25.06 g/day, $P = 0.01$), starch (12.13 ± 15.28 vs. 12.4 ± 14.05 g/day, $P = 0.01$), lactose (4.17 ± 4.5 vs. 4.34 ± 4.86 g/day, $P = 0.001$), and alcohol (0.02 ± 0.06 vs. 0.03 ± 0.08 g/day, $P = 0.003$) than the healthy subjects.

The association between hypertension and different types of dietary carbohydrates is shown in Table 3. A significant association was observed between hypertension and dietary intake of glucose after adjustment for age and gender (OR: 1.02; 95% CI : 1.00–1.05; $P = 0.04$). The association remained significant after further adjustments for education, marital status, job, biochemical measurements, physical activity, and BMI (Model 2) and additional adjusting for energy and macronutrients intake

Table 1 Characteristics of the participants with normal and high blood pressure

	Normal BP	High BP	*P*
Age (years)	47.46 ± 8.35	53.45 ± 8.3	<0.001
MET (kcal/kg × h)	38.84 ± 7.8	38.1 ± 7.7	0.008
Height (cm)	161.75 ± 9.0	163.32 ± 9.0	<0.001
Weight (kg)	72.51 ± 12.8	78.01 ± 14.0	<0.001
BMI (kg/m)^2	27.76 ± 4.7	29.23 ± 4.7	<0.001
Right SBP (mmHg)	106.23 ± 10.0	134.12 ± 0	<0.001
Right DBP (mmHg)	67.33 ± 7.0	82.95 ± 7.6	<0.001
FBS (mg/dl)	103.0 ± 35.51	116.89 ± 49.27	<0.001
BUN (mg/dl)	13.46 ± 3.6	14.35 ± 3.9	<0.001
Creatinine (μmol/l)	1.07 ± 0.18	1.14 ± 0.28	<0.001
TG (mg/dl)	137.88 ± 92	166.58 ± 121.3	<0.001
Cholesterol (mg/dl)	189.9 ± 39.06	195.48 ± 42.62	<0.001
SGOT (U/l)	19.89 ± 8.63	20.91 ± 9.59	<0.001
SGPT (U/l)	21.31 ± 15.41	24.01 ± 14.75	<0.001
ALP (U/l)	217.24 ± 66.04	234.13 ± 71.13	<0.001
HDL-C (mg/dl)	52.87 ± 10.66	51.11 ± 10.47	<0.001
LDL-C (mg/dl)	109.89 ± 33.02	111.23 ± 35.3	<0.001
GGT (U/l)	24.09 ± 21.16	28.85 ± 24.21	<0.001
Gender (*n*)			
Male	1166 ± 0.39	700 ± 0.56	0.01
Female	1779 ± 0.6	539 ± 0.43	0.1

ALP, alkaline phosphatase; BP, blood pressure; BUN, blood urea nitrogen; FBS, fasting blood sugar; GGT, gamma glutamic transferase; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; MET, metabolic equivalent of task; SGOT, serum glutamic oxaloacetic transaminase; SGPT, serum glutamic pyruvic transaminase; TG, triglyceride.

Table 2 Dietary intakes among the participants with normal and high blood pressure

	Normal BP	High BP	*P*
Protein (g/day)	78.14 ± 25.74	79.98 ± 26.79	0.003
Total lipid fat (g/day)	64.58 ± 25.06	64.31 ± 25.06	0.01
Carbohydrate (g/day)	407.77 ± 135.06	425.44 ± 144.70	0.0001
Energy (kcal/day)	2477.77 ± 770.36	2549.93 ± 818.39	0.001
Starch (g/day)	12.4 ± 14.05	12.13 ± 15.28	0.01
Sucrose (g/day)	45.79 ± 30.41	47.39 ± 34.35	0.02
Glucose – dextrose (g/day)	27.23 ± 16.03	28.98 ± 17.15	<0.001
Fructose (g/day)	31.75 ± 18.37	33.58 ± 19.33	0.001
Lactose (g/day)	4.34 ± 4.86	4.17 ± 4.5	0.001
Maltose (g/day)	0.51 ± 0.63	0.53 ± 0.68	0.001
Alcohol (g/day)	0.03 ± 0.08	0.02 ± 0.06	0.003
Caffeine (mg/day)	190.07 ± 127.62	195.88 ± 126.31	0.1
Total sugar (g/day)	137.55 ± 62.85	143.1 ± 66.76	0.002
Galactose (g/day)	0.18 ± 0.19	0.2 ± 0.22	0.002

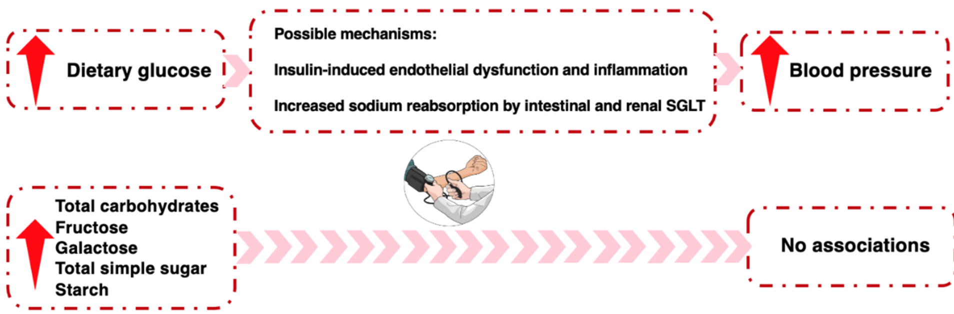
BP, blood pressure.

Table 3 The association of hypertension and different types of dietary carbohydrates

	Total carbohydrates		Glucose		Fructose		Galactose		Starch		Simple sugars	
	OR (95% CI)	*P*	OR (95% CI)	*P*	OR (95% CI)	*P*	OR (95% CI)	*P*	OR (95% CI)	*P*	OR (95% CI)	*P*
Model 1	1.00 (0.99–1.00)	0.25	1.02 (1.01–1.05)	0.04	0.99 (0.96–1.01)	0.38	1.05 (0.66- 1.64)	0.81	0.99 (0.97–1.01)	0.71	0.99 (0.98- 1.01)	0.05
Model 2	1.00 (0.99–1.00)	0.23	1.02 (1.01–1.05)	0.04	0.98 (0.96–1.01)	0.38	1.04 (0.65–1.62)	0.86	0.99 (0.97–1.01)	0.74	0.99 (0.98–1.01)	0.05
Model 3	1.00 (0.99- 1.00)	0.64	1.02 (1.01- 1.05)	0.04	0.98 (0.96–1.01)	0.38	1.04 (0.65–1.62)	0.86	0.99 (0.97–1.01)	0.74	0.99 (0.98–1.01)	0.06

Model 1: adjusted for age and gender; Model 2: further adjustments for education, marital status, job, biochemical measurements, physical activity, and BMI; Model 3: further adjustments for energy and macronutrients intake.
CI, confidence interval; OR, odds ratio.

Fig. 1



The association between carbohydrate and blood pressure.

(Model 3). No association was found between hypertension and dietary intake of total carbohydrates, fructose, galactose, total sugar, and starch (Fig. 1).

Discussion

To our knowledge, this study was the first effort in an Iranian population to evaluate the association between hypertension and different types of dietary carbohydrates. The findings demonstrated a significant association between hypertension and dietary intake of glucose. Following further adjustments for education, marital status, job, physical activity, and BMI, the association remained significant. There were no associations between hypertension and dietary intake of total carbohydrates and other types of carbohydrates.

Previous research on dietary intake of carbohydrates and the risk of hypertension revealed conflicting results. Nguyen and Lustig found that one spoonful of sugar increased BP, which is consistent with our results [22]. A cross-sectional study conducted on the elderly population demonstrated a significant relationship between carbohydrate consumption and both SBP and DBP [23]. In addition, in a systematic review and meta-analysis of prospective cohorts of US men and women, Jayalath *et al.* found that total fructose intake was not associated with an increased risk of hypertension [19]. On the contrary, Brown *et al.* indicated an inverse association between starch intake and BP [24].

Furthermore, Jalal *et al.* reported that high fructose intake, as added sugar, was independently associated with higher BP levels among American adults without a history of hypertension [18]. Zhou *et al.* demonstrated that both high and low percentages of total carbohydrate consumption were associated with a higher risk of new-onset hypertension in a large nationwide cohort of adults in China [25]. The possible difference between our study and other studies may be due to ethnic differences, the sample size, and the research methodology. Some plausible explanations can be offered for the direct association between glucose and the risk of hypertension. For example, the connection between BP and dietary carbohydrate may be influenced by other dietary components. One study in China found that the carbohydrate-to-fiber ratio (CFR) is a crucial factor in cardiometabolic health, and higher CFR was associated with an increased risk of poor BP control in hypertensive patients [26].

Unlike sugar-sweetened beverages and refined grains, healthy carbohydrates such as whole grains, fruits, vegetables, and legumes have a high fiber content linked to appetite suppression, lower body weight, and reduced risk of hypertension [27,28]. For instance, the Dietary Approach to Stop Hypertension, the Mediterranean and vegetarian diets providing more fiber-rich carbohydrates, are considered inversely related to hypertension [29]. However, increased fructose consumption is associated with hyperuricemia and, consequently, renal tubular damage and hypertension; antihypertensive nutritional agents of fruits, such as fiber, potassium, and vitamin C, may improve the action of fructose [30,31].

Even marginal enhancements in BP control at the societal scale may significantly affect CVD prevention. Adopting lifestyle modifications, such as incorporating a balanced and nutritious diet, may serve as a preventive measure for the incidence of new cases of hypertension [32–34]. Neal *et al.* indicated that slight mitigations in BP (ranging from 3 to 6 mmHg SBP and 1–4 mmHg DBP) sustained over an extended period are associated with decreased probabilities of experiencing a stroke, coronary heart disease, cardiovascular incidents, and mortality, with a reduced rate of approximately 20–30% [35]. While it is essential to understand the extent that which alterations in energy consumption and body weight impact the relationship between sugars and cardiometabolic risk, the potential individual and public health advantages resulting from a decrease in added sugar intake are not contingent upon a comprehensive understanding of the underlying mechanism. The difference in the impact of glucose in contrast to other carbohydrates on BP is associated with the process of glucose absorption in conjunction with sodium via sodium-glucose cotransporters in the gut, as well as its subsequent reabsorption in the kidney. Consequently, the increase in glucose consumption results in enhanced salt absorption, thus leading to an overall increase in BP [36]. These transporters do not play a role in fructose transportation, so fructose intake separately does not increase sodium and BP [37]. Moreover, elevated glucose intake from a high glycemic index carbohydrate source can interrupt the glucose-insulin axis by increasing insulin levels, impairing insulin sensitivity and thus may lead to insulin resistance [38,39]. Subsequent insulin-induced endothelial dysfunction, inflammation, and sodium sensitivity may lead to higher BP [40].

The present study has some limitations. First, the design of the present study did not determine a causal relationship. As such, reverse causation remains a suitable alternative explanation for the observed associations between exposure and outcome. Another limitation of the present study is that this evaluation was based on an FFQ tailored to Iranian food consumption patterns. Notably, the dietary information obtained from the FFQ has recall bias and may have underestimation and overestimation of food intake in obese and underweight participants, respectively. However, the questions were conducted by experienced researchers through face-to-face interviews in order to reduce the amount of bias as much as possible. Despite these limitations, the current investigation possesses numerous strengths. The data quality is high because they were acquired from a large sample with an exceptionally high response rate.

The current research effort is the first attempt to investigate the correlation between hypertension and different types of dietary carbohydrates in an Iranian population. In addition, the findings provide guidance for future research efforts to determine the underlying cause of metabolic abnormalities in the Iranian adult population. Further prospective cohort studies applying innovation measurements to assess the quantity, quality, and timing of carbohydrate consumption are necessary considering all of these gaps in knowledge. An adequate follow-up and robust measurement of BP level and circadian pattern are also needed. A more profound comprehension of these associations could shed light on novel lifestyle strategies that can prevent circadian rhythm disruptions that increase the risk of CVD in addition to hypertension. If confirmed in future studies, the obtained results may lead to customized and focused lifestyle interventions that specifically target optimum carbohydrate composition which is essential for sustaining optimal BP.

Conclusion

In conclusion, our results demonstrated a significant association between hypertension and dietary intake of glucose but no associations between hypertension and dietary intake of total carbohydrates, fructose, galactose, total simple sugar, and starch. Although numerous studies have been done to show an association between carbohydrates and hypertension, the effect of carbohydrate consumption on BP is an area ripe for further research. Given that carbohydrates constitute a fundamental component of the dietary intake of Iranian residents, it is prudent to incorporate these observations into the development of logical dietary plans for individuals with hypertension.

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The Ethical Committee of the Research Ethics Committee at Sabzevar University of Medical Sciences in Sabzevar, Iran, provided approval for this study (Code IR.MEDSAB.REC.1400.040). All experimental procedures that involved human subjects were conducted in accordance with the ethical guidelines set forth by the relevant national or institutional research committee, the 1964 Declaration of Helsinki, any subsequent amendments thereby, or comparable standards of ethics. All participants provided written informed consent at baseline.

The datasets used and/or examined in the present investigation can potentially be obtained from the corresponding author upon reasonable requests.

Conflicts of interest

There are no conflicts of interest.

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