European Journal of Nutrition (2019) 58:947–953
https://doi.org/10.1007/s00394-018-1645-x

ORIGINAL CONTRIBUTION

24-h urinary sodium to potassium ratio and its association with obesity in children and adolescents

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Received: 28 April 2017 / Accepted: 20 September 2017 / Published online: 16 March 2018

Abstract

Purpose There are epidemiologic studies indicating a positive correlation between high sodium and low potassium intake and body mass index. Therefore, this study was conducted in a cross-sectional sample of Iranian children and adolescents to evaluate the link between 24-h urinary Na:K ratio and risk of obesity.

Methods In this cross-sectional study, 374 participants aged 11–18 years were included. One 24-h urine sample was collected by each participant to estimate Na:K ratio. Anthropometric measurements were carried out and overweight/obesity was defined as a BMI ≥ 85th percentile and abdominal obesity as a waist:height ratio (WHtR) of more than 0.5.

Results As expected, 24-h urinary Na:K ratio showed significant associations with risk of overweight/obesity. Risk of adiposity assessed by WC and PBF was significantly associated with Na:K ratio after adjusting for SSBs consumption and calorie intake. Urinary Na:K ratio showed significant association with risk of adiposity assessed by WC only in girls in the highest tertile group with OR of 2.71 (95% CI 1.14–6.43), only after the addition of calorie intake. Adiposity assessed by PBF was only associated with Na:K ratio among boys with OR of 4.47 (95% CI 1.44–9.87) and 3.87 (95% CI 1.20–8.48), after adjusting for SSBs consumption and calorie intake, respectively.

Conclusion Our findings suggest that reducing Na and increasing K intake could be used as a useful approach to lower the risk of obesity and associated burden of disease in Iran. However, more studies are warranted.

Keywords Sodium · Potassium · 24-h urine · Overweight · Obesity · Children · Adolescents · Iran

Introduction

Obesity is a major public health challenge worldwide because of its high prevalence both in developed and developing countries. Studies indicate the rising incidence of overweight and obesity and their attributed range of metabolic complications in children and adolescents [1]. Middle-East is one of the regions with the highest prevalence of overweight among preschool children [2] and as one of the countries in the area, Iran also suffers from high prevalence of obesity and related diseases including metabolic syndrome and diabetes in different age groups [3–5]. Therefore, early prevention and control of risk factors related to obesity and related diseases are among important public health priorities in Iran.

In recent years, sodium intake has significantly increased due to the consumption of various types of processed foods with high salt content [6] and, as a result, the mean salt intake in most countries is around 9–12 g/d [7]. On the other hand, there are results indicative of

Electronic supplementary material The online version of this article (https://doi.org/10.1007/s00394-018-1645-x) contains supplementary material, which is available to authorized users.

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inadequate intake of potassium among children [8]. Studies in Iran also indicate that Iranian children consume a large amount of sodium and small amount of potassium [9]. In addition to strong evidence indicating an association between high sodium intake and diseases such as high blood pressure and cardiovascular disease [10], gastric cancer [11], osteoporosis and kidney disease [12], recent studies suggest that there may be an association between sodium intake and obesity epidemic [13]. There are also evidence regarding sodium intake in relation to body weight in children and adolescents independent of energy intake [14–16]. In addition, it has been found that potassium intake slows the harmful effects of sodium. Although this has only been shown with respect to blood pressure [17]. There are also few epidemiologic studies indicating a positive correlation between high sodium and low potassium intake and adiposity measures [18, 19].

The issue here is that, in cases such as high blood pressure, the interaction between high dietary sodium intake and low potassium intake is involved in the pathogenesis of hypertension in a way that the overall effect of high sodium and low potassium diets on blood pressure seems to be more than the effect of each of these factors alone. However, the combined role of dietary sodium and potassium intake in the pathogenesis of other conditions including overweight and obesity especially among children is less known [20]. No population-based study has specifically estimated the effect of Na:K ratio on the risk of obesity among Iranian children using 24-h urine collections as the gold standard method. Therefore, we analyzed data from a cross-sectional sample of Iranian children and adolescents to evaluate the link between 24-h urinary Na:K ratio and risk of obesity.

Materials and methods

Study design and participants

A cross-sectional study including children and adolescents aged 11–18 years living in Isfahan, Iran, was carried out between September 2015 and February 2016. Using random cluster sampling method, participants were selected from 13 schools in four different districts. The main investigators then visited schools and invitations were sent to parents. Parents/students who agreed to participate in the study were asked to complete an informed consent. Participants with any acute or chronic diseases or on any medication or special diet were excluded. From 456 invited participants, 374 of them completed the study (response rate 82%). The study was approved by the Research and Ethics Committee of Isfahan University of Medical Sciences (IR. mui.rec.1394.3/294).

24-h urine collection

24-h urine collection containers were distributed among participants and parents, along with verbal and written instructions on how to complete the procedure. It was emphasized that no change in dietary habits during the day of collection was allowed. Sampling was completed over the weekends from Friday to Saturday during a 24-h period. Each participant was provided with a 2.5-L polypropylene container used for the collection of 24-h urine sample. All participants were instructed to initiate the collection by emptying their bladder, discarding first voiding after getting up on Friday morning and continue the collection till next day morning. To assist with urine collection, an additional 500-ml plastic cup was provided. All participants were asked to keep the containers in cool and dry conditions and samples were immediately transferred to the laboratory on the next day for promp analysis to prevent from microbial degradation. Measured parameters included 24 h urine volume, Na, K and creatinine (Cr). Na and K were estimated using ion selective electrode method and urine Cr was measured by Jaffe reaction method [21]. Completeness of 24-h urine was defined by volume of collected urine $\geq$ 500 ml and 24-h Cr excretion $\geq$ 0.1 mg/ kg body weight [22].

Anthropometric assessment

Body weight was measured using a digital scale with minimal clothing and was recorded to the nearest 100 g. Height was estimated using an upstretched tape without shoes and was registered to the nearest 0.5 cm. Waist circumference (WC) was measured midway between the lowest rib and top of the hip bone to the nearest 0.5 cm. Body mass index (BMI) was calculated as weight in kg divided by height in m^2. Participants were grouped into weight categories (underweight, normal weight, overweight, obese) using the international obesity taskforce BMI reference cut-offs [23]. Abdominal obesity was defined as waist to height ratio (WHtR) of equal or more than 0.5 [24]. Percent body fat (PBF) was measured using Omron BF511 body composition monitor (Omron Corp, Kyoto, Japan) and PBF > 25% for boys and > 35% for girls aged 11 years and older was defined as adiposity risk [25].

Dietary assessment

A validated 168-item self-administered food frequency questionnaire (FFQ) was used to assess usual dietary intake. It consisted of a list of foods and a standard serving size for each [26]. The subjects were asked to report the frequency of consumption of a given serving of each food item during the previous year on a daily, weekly or monthly basis. Portion sizes of consumed foods were converted to grams using household measures [27]. Dietary analysis was performed using Nutritionist IV software (First Databank, Hearst Corp, San Bruno, CA, USA).

Assessment of other variables

Additional information regarding sociodemographic variables including age, sex and parents' education level, household income and past medical history were obtained using a self-administered questionnaire. The Physical Activity Questionnaire (PAQ), a self-administered, 7-day recall instrument with consistently high validity and moderate reliability [28], was used to assess the general levels of physical activity. A summary of physical activity score derived from nine items, each scored on a 5-point scale is provided by this questionnaire. The mean score of these nine items results in the final PAQ activity summary score. A score of 1 indicates low physical activity, a score of 2–4 indicates moderate physical activity and a score of 5 indicates high physical activity.

Statistical analysis

Descriptive statistics (mean values and standard deviations for continuous variables or numbers and percentages for categorical variables) were used to describe participant characteristics. One-way ANOVA and chi-squared tests were used where appropriate. Multiple logistic regression models were used to assess the association between 24 h urinary Na:K excretion and (1) weight category and (2) abdominal obesity. The unadjusted and adjusted models (age, sex, parents' education level, household income, physical activity) are presented. To assess whether the association between 24 h urinary Na:K excretion and adiposity outcome measures was independent of energy and sugar-sweetened beverages intake (sugar-sweetened beverages (SSBs); including carbonated soft drinks, soda, squashes, fruit drinks), additional models were constructed with these covariates. Analyses were completed using SPSS software version 18 (IBM Inc., Chicago, IL, USA), and a P value < 0·05 was considered statistically significant.

Results

Table 1 shows the demographic characteristics of participants and data on urinary excretion and dietary intake across tertiles of Na:K excretion. Of 456 participants who started the 24-h urine collection, 50 samples were not returned. Also, some collections were excluded ($n=32$) due to incompleteness of 24-h collection. Thus, 374 participants had complete and valid urinary samples and were included in the final analysis. Overall, 58.8% ($n=220$) were girls and

Table 1 Baseline characteristics according to tertiles of 24-h urinary sodium to potassium ratio among Iranian children and adolescents aged 11–18 years, Isfahan, Iran

	Tertile of Na:K ratio			
	*T**1 < 1.79	*T*2 1.79–2.49	*T*3 > 2.49	*P****
	Mean (SD)**	Mean (SD)	Mean (SD)	
Participants (*n*)	125	126	123	
Age (year)	14.36 (2.19)	14.49 (2.20)	14.46 (2.09)	0.88
Sex *n* (%)				0.46
Girls	68 (31.0)	76 (34.5)	76 (34.5)	
Boys	57 (37.0)	50 (32.5)	47 (30.5)	
Na:K ratio	1.40 (0.31)	2.13 (0.19)	3.32 (0.83)	< 0.001
Cr excretion (mmol/kg/day)	0.13 (0.09)	0.14 (0.07)	0.13 (0.07)	0.56
Urine output (l/day)	0.97 (0.41)	0.94 (0.38)	0.92 (0.35)	0.04
Energy intake (kcal/day)	1672.96 (301.71)	1706.68 (323.93)	1649.97 (275.12)	0.33
SSBs[1] (g/day)	46.83 (42.24)	46.08 (45.99)	38.31 (37.26)	0.21
Physical activity[2] *n* (%)				0.99
Low	45 (33.1)	46 (33.8)	45 (33.1)	
Moderate	80 (33.6)	80 (33.6)	78 (32.8)	
High	–	–	–	

*Tertile

**Mean (Standard deviation)

***P value (obtained from ANOVA for continuous variables and $\chi 2$ test for categorical variables)

[1]Sugar-sweetened beverages included carbonated soft drinks, soda, squashes and fruit drinks

[2]Physical activity was calculated using PAQ score where score of 1 indicates low physical activity, score of 2–4 indicates moderate physical activity and score of 5 indicates high physical activity

the mean age was 14.4 (2.02) years. As can be seen, no significant differences were found in terms of presented variables across tertiles of Na:K ratio except for urine output. Participants had mostly moderate physical activity level and there were no children and adolescents with high physical activity in our sample.

Based on Table 2, no significant differences were observed regarding body weight across tertiles of Na:K ratio while subjects in the highest tertile were significantly lower in body height. Significant differences were observed only regarding weight categories and abdominal obesity across tertiles of Na:K ratio. The mean BMI of the sample was 20.91 (4.16) kg/m^2 and 18.2% ($n=68$) and 8.6% ($n=32$) of the sample were overweight and obese, respectively. In total, 29.7% ($n=111$) of participants were classified as centrally obese. Based on PBF as another marker of adiposity, 24.9% ($n=93$) of participants had excess body fat.

Multivariate-adjusted odds of overweight/obesity, abdominal obesity and adiposity based on PBF according to tertiles of 24-h urinary Na to K ratio are shown in Table 3. As expected, 24-h urinary Na:K was a strong independent predictor of the risk of obesity. 24-h urinary Na:K ratio showed significant associations with risk of overweight/obesity which remained significant in stratified models. Risk of adiposity assessed by WC and PBF was not significantly associated with Na:K ratio in model 1. However, the addition of SSBs consumption and calorie intake into the multivariate logistic regression (models 2 and 3) created statistically significant differences in the patterns of the association between urinary Na:K ratio and these adiposity measures. In stratified models, urinary Na:K ratio showed significant association with risk of adiposity assessed by WC only in girls in the highest tertile group with OR of 2.71 (95% CI 1.14–6.43), only after the addition of calorie intake. Adiposity assessed by PBF was only associated with Na:K ratio among boys with OR of 4.47 (95% CI 1.44–9.87) and 3.87 (95% CI 1.20–8.48), after adjusting for SSBs consumption and calorie intake, respectively.

Discussion

Among school children, we found that 24-h urinary Na:K was associated with greater risk of overweight/obesity and abdominal obesity. Of note, the association between Na:K and abdominal obesity was not independent of BMI, indicating that the potential effect of Na:K is related to total body weight and is not specific to central fat distribution (data not shown). Our findings clearly show the public health importance of the reduction of Na intake and the increase of K intake for preventing obesity.

To our knowledge, the present study is the first to report the association of Na:K assessed by 24-h urine collection with the risk of obesity in Iran. It has important clinical and public health implications because high Na and low potassium intake and obesity are considered among public health issues in Iranian population [9, 29, 30]. Accordingly, these findings contribute to the existing literature and provide

Table 2 Anthropometric measurements according to tertiles of Na:K ratio among Iranian children and adolescents aged 11–18 years, Isfahan, Iran

	Total	Tertile of Na:K ratio			
		*T**1 < 1.79	*T*2 1.79–2.49	*T*3 > 2.49	*P****
	Mean (SD)**	Mean (SD)	Mean (SD)	Mean (SD)	
Weight (kg)	53.24 (14.22)	53.13 (14.05)	53.43 (14.86)	53.16 (13.82)	0.98
Height (cm)	158.72 (10.58)	159.72 (11.37)	159.66 (10.66)	156.79 (9.43)	0.04
BMI[1] (kg/m^2)	20.91 (4.16)	20.58 (3.10)	20.77 (4.29)	21.40 (4.19)	0.27
WHtR[2]	0.47 (0.06)	0.47 (0.06)	0.47 (0.06)	0.48 (0.05)	0.32
PBF[3] (%)	24.99 (9.65)	24.29 (9.67)	24.49 (9.30)	26.22 (9.93)	0.23
Weight category[4] *n* (%)					0.007
Underweight/normal weight	274 (73.2)	99 (36.1)	97 (35.4)	78 (28.5)	
Overweight	68 (18.2)	13 (19.1)	21 (30.9)	34 (50.0)	
obesity	32 (8.6)	13 (40.6)	8 (25.0)	11 (34.4)	
Abdominal obesity[5] *n* (%)	111 (29.7)	35.0 (31.5)	28 (25.3)	48 (43.2)	0.02
Adiposity by PBF[6] *n* (%)	93 (24.9)	28 (30.1)	26 (28.0)	39 (41.9)	0.10

*Tertile

**Mean (standard deviation)

****P* value (obtained from ANOVA for continuous variables and $\chi 2$ test for categorical variables)

[1]Body mass index, [2]waist to height ratio, [3]percent body fat, [4]underweight was defined as BMI < 5th; normal weight was defined as 5th ≤ BMI < 85th; overweight was defined as 85th ≤ BMI < 95th; obesity was defined as BMI ≥ 95th; [5]abdominal obesity was defined as WHtR > 0.05; [6] adiposity was defined as PBF > 25% for boys and > 35% for girls

Table 3 Odds ratios for overweight/obesity and abdominal obesity according to tertiles of Na:K ratio among Iranian children and adolescents aged 11–18 years, Isfahan, Iran

	Total			Girls			Boys		
	Urinary Na:K ratio			Urinary Na:K ratio			Urinary Na:K ratio		
	*T**1 < 1.79	*T*2 1.79–2.49	*T*3 > 2.49	*T**1 < 1.82	*T*2 1.82–2.57	*T*3 > 2.57	*T**1 < 1.77	*T*2 1.77–2.38	*T*3 > 2.38
		OR (95% CI)	OR (95% CI)		OR (95% CI)	OR (95% CI)		OR (95% CI)	OR (95% CI)
Overweight/obesity**									
Model 1[1]	1	1.11 (0.57–2.12)	2.54(1.36–4.76)	1	1.68 (0.70–4.03)	2.70 (1.16–6.26)	1	0.62 (0.21–1.79)	2.86 (1.05–7.81)
Model 2[2]	1	1.14 (0.58–2.22)	3.01 (1.57–5.76)	1	1.69 (0.69–4.15)	2.94 (1.23–7.02)	1	0.61 (0.20–1.84)	4.09 (1.40–8.94)
Model 3[3]	1	0.86 (0.39–1.91)	4.07 (1.92–8.65)	1	1.52 (0.52–4.45)	5.09 (1.79–9.50)	1	0.29 (0.07–1.17)	4.03 (1.23–8.20)
Abdominal obesity***									
Model 1[1]	1	0.65 (0.35–1.21)	1.63 (0.92–2.88)	1	0.87 (0.38–1.96)	1.94 (0.91–4.14)	1	0.39 (0.14–1.07)	1.29 (0.51–3.30)
Model 2[2]	1	0.65 (0.35–1.22)	1.84 (1.02–3.31)	1	0.84 (0.36–1.94)	2.06 (0.94–4.48)	1	0.37 (0.13–1.07)	1.79 (0.66–4.86)
Model 3[3]	1	0.51 (0.26–1.02)	1.89 (1.02–3.51)	1	0.68 (0.26–1.76)	2.71 (1.14–6.43)	1	0.23 (0.07–1.76)	1.32 (0.48–3.63)
Adiposity based on PBF****									
Model 1[1]	1	0.83 (0.42–1.63)	1.74 (0.92–3.29)	1	0.83 (0.33–2.05)	1.21 (0.51–2.89)	1	0.73 (0.24–2.22)	3.01 (1.04–8.66)
Model 2[2]	1	0.85 (0.42–1.70)	1.99 (1.03–3.85)	1	0.84 (0.33–2.14)	1.28 (0.52–3.15)	1	0.72 (0.22–2.33)	4.47 (1.44–9.87)
Model 3[3]	1	0.75 (035–1.61)	2.22 (1.10–4.46)	1	0.76 (0.26–2.19)	1.65 (0.61–4.44)	1	0.49 (0.14–1.77)	3.87 (1.20–8.48)

*Tertile

**Overweight/obesity defined as BMI ≥ 85th

***Abdominal obesity defined as WHtR > 0.5 cm

****Defined as PBF > 25% for boys and > 35% for girls aged 11 years and older

[1]Adjusted for age, sex, parents' education and household income, sleep hours and physical activity

[2]Additionally adjusted for SSBs (g/day)

[3]Additionally adjusted for energy intake (kcal/day)

new and important information regarding the relationship between Na:K and obesity in a representative sample of the Iranian children and adolescents, and suggest that the reduction of Na intake and increase of K intake should be an important priority for reducing the prevalence of obesity in Iran.

To date, only one cross-sectional study and a multi-ethnic cohort study have attempted to investigate the association between Na:K intake and risk of obesity. Zeng Ge and colleagues [18] reported that the odds of abdominal obesity, but not overweight and obesity, increased significantly with successive Na:K quartiles in Chinese adults aged 18–69 years. Compared with participants in the first quartile of Na:K excretion, the OR (95% CI) of abdominal obesity (WC) and abdominal obesity (WHtR) was 1.35 (1.02, 1.79) and 1.57 (1.18, 2.10) in the fourth quartile, respectively. Similarly, Jain N et al. [19] identified Na:K as an independent predictor of obesity risk. With a three-unit change in urinary Na:K, a statistically significant change of 0.75 (95% CI 0.25, 11.25) in total body percentage fat was documented. Moreover, a study by Shavit L [31] showed significantly higher levels of urinary Na and K in overweight and obese kidney stone formers compared to normal weight participants. This was similarly indicated in another study [32] reporting that urinary excretion of sodium increased with increasing BMI. However, urinary potassium excretion was not associated with BMI. A systematic review [33] also showed that Na to K ratio seems to be a more sensitive index to examine the risk of obesity compared to each factor alone. Although, due to lack of relevant studies, conducting a meta-analysis was not possible and further studies with prospective designs or clinical trials are warranted to assess this association more thoroughly.

Also, this study found that different adiposity measures showed more apparent associations with Na intake in different sexes. Risk of adiposity assessed by WC was significantly associated with dietary Na:K intake only in girls, whereas adiposity risk assessed by PBF was significantly associated with dietary Na:K intake only in boys. Risk of adiposity assessed by BMI was in general consistent in both sexes. Previous studies have not examined sex differences in the associations between NA:K ratio and obesity. Therefore, these differences need to be further explored.

Several mechanisms may explain the relationship between Na:K intake and obesity risk. First, diets high in Na and low in K are often high in energy and, therefore, may promote weight gain. Besides, through the effects on thirst, a salty diet may cause greater consumption of SSBs which are associated with weight gain [14, 34, 35]. However, after adjusting for intake of energy and SSBs, we found that the association between Na:K intake and obesity risk remained significant, suggesting that other mechanisms may be involved. Experimental studies suggest that higher Na intake is associated with increased lipogenic activity and formation of adipocyte tissue. Moreover, in mice, it was seen that the uptake of glucose and its conversion to lipids within adipocytes was higher when their sodium intake increased [36, 37]. The mechanism of dietary K intake in relation to obesity risk is less well understood. It has been suggested that obesity is associated with potassium channel function [38, 39]. Potassium can influence carbohydrate accumulation and glucose homeostasis [40, 41] and has important role in insulin secretion and carbohydrate metabolism [41, 42]. Also, the protective effect of potassium against obesity could be due to high intake of fruits and vegetables which are the main sources of potassium in diet [43]. Furthermore, in this study, subjects in the highest tertile of Na:K ratio were significantly lower in body height (Table 2) with no significant differences in terms of body weight across various tertiles. It could be hypothesized that the increased risk of obesity could be related to decreased body height due to high sodium and low potassium intake. However, this association was not examined in our study and needs further exploration. In summary, more studies are needed to elucidate the exact mechanism of action of dietary potassium in obesity risk reduction.

Potential limitations should be noted. The cross-sectional design precludes us from inferring causal relationship between Na:K intake and risk of obesity. Furthermore, a single 24-h urine collection might have overestimated or underestimated the actual Na and K intake in the study population. Also, factors such as stress or menstruation have been associated with obesity and these were not controlled in our study. Therefore, in future studies, these should be considered. Also, no information on parental overweight/obesity was collected. This could influence the results since parental overweight is an important predictor of the children's body weight. Nevertheless, a major strength of our study includes use of 24-h urine collection as the gold standard method for estimation of Na and K intake. In addition, to the best of our knowledge, this is the first study to show the combined effect of Na and K intake on general and abdominal obesity in a large sample of children and adolescents.

In summary, we report an independent association between the ratio of dietary Na and K intake and obesity risk in a large sample of children and adolescents, even after controlling for important confounding factors. Our findings suggest that reducing Na intake and increasing K intake could be used as a useful approach to reduce the risk of obesity and associated burden of disease in Iran.

Acknowledgements This study was supported by Isfahan University of Medical Sciences and Isfahan Cardiovascular Research Institute. The authors would like to thank all the schools and students/parents who agreed to participate in this study.

Funding None.

Compliance with ethical standards

Conflict of interest The authors declare no conflict of interests.

References

1. Djalalinia S, Kelishadi R, Qorbani M, Peykari N, Kasaeian A, Nasli-Esfahani E, Naderimagham S, Larijani B, Farzadfar F (2016) A systematic review on the prevalence of overweight and obesity, in iranian children and adolescents. Iran J Pediatr 26(3):e2599. https://doi.org/10.5812/ijp.2599
2. de Onis M, Blossner M, Borghi E (2010) Global prevalence and trends of overweight and obesity among preschool children. Am J Clin Nutr 92(5):1257–1264. https://doi.org/10.3945/ajcn.2010.29786
3. Delavari A, Forouzanfar MH, Alikhani S, Sharifian A, Kelishadi R (2009) First nationwide study of the prevalence of the metabolic syndrome and optimal cutoff points of waist circumference in the Middle East: the national survey of risk factors for noncommunicable diseases of Iran. Diabetes Care 32(6):1092–1097. https://doi.org/10.2337/dc08-1800
4. Haghdoost AA, Rezazadeh-Kermani M, Sadghirad B, Baradaran HR (2009) Prevalence of type 2 diabetes in the Islamic Republic of Iran: systematic review and meta-analysis. East Mediterr Health J 15(3):591–599
5. Kelishadi R, Ardalan G, Gheiratmand R, Adeli K, Delavari A, Majdzadeh R (2006) Paediatric metabolic syndrome and associated anthropometric indices: the CASPIAN Study. Acta Paediatr 95(12):1625–1634. https://doi.org/10.1080/08035250600750072
6. He FJ, MacGregor GA (2009) A comprehensive review on salt and health and current experience of worldwide salt reduction programmes. J Hum Hypertens 23(6):363–384
7. Libuda L, Kersting M, Alexy U (2012) Consumption of dietary salt measured by urinary sodium excretion and its association with body weight status in healthy children and adolescents. Public Health Nutr 15(03):433–441

8. El Mallah C, Merhi K, Ghattas H, Shatila D, Francis S, Hlais S, Toufeili I, Obeid O (2016) Elevated urinary Na/K ratio among Lebanese elementary school children is attributable to low K intake. Eur J Nutr. https://doi.org/10.1007/s00394-016-1164-6
9. Kelishadi R, Gheisari A, Zare N, Farajian S, Shariatinejad K (2013) Salt intake and the association with blood pressure in young Iranian children: first report from the middle East and north Africa. Int J Prev Med 4(4):475–483
10. He FJ, MacGregor GA (2007) Salt, blood pressure and cardiovascular disease. Curr Opin Cardiol 22(4):298–305
11. Wiseman M (2008) The second World Cancer Research Fund/American Institute for Cancer Research expert report. Food, nutrition, physical activity, and the prevention of cancer: a global perspective. Proc Nutr Soc 67(03):253–256
12. Cappuccio FP, Kalaitzidis R, Duneclift S, Eastwood JB (2000) Unravelling the links between calcium excretion, salt intake, hypertension, kidney stones and bone metabolism. J Nephrol 13(3):169–177
13. Zhu H, Pollock NK, Kotak I, Gutin B, Wang X, Bhagatwala J, Parikh S, Harshfield GA, Dong Y (2014) Dietary sodium, adiposity, and inflammation in healthy adolescents. Pediatrics 133(3):e635-e642
14. Grimes CA, Riddell LJ, Campbell KJ, Nowson CA (2013) Dietary salt intake, sugar-sweetened beverage consumption, and obesity risk. Pediatrics 131(1):14–21
15. Woodruff SJ, Fryer K, Campbell T, Cole M (2014) Associations among blood pressure, salt consumption and body weight status of students from south-western Ontario. Public Health Nutr 17(05):1114–1119
16. Yoon Y, Oh S (2013) Sodium density and obesity; the Korea National Health and Nutrition Examination Survey 2007–2010. Eur J Clin Nutr 67(2):141–146
17. Rodrigues SL, Baldo MP, Machado RC, Forechi L, Molina MdCB, Mill JG (2014) High potassium intake blunts the effect of elevated sodium intake on blood pressure levels. J Am Soc Hypertens 8(4):232–238
18. Ge Z, Zhang J, Chen X, Yan L, Guo X, Lu Z, Xu A, Ma J (2016) Are 24 h urinary sodium excretion and sodium: potassium independently associated with obesity in Chinese adults? Public Health Nutr 19(06):1074–1080
19. Jain N, Minhajuddin AT, Neeland IJ, Elsayed EF, Vega GL, Hedayati SS (2014) Association of urinary sodium-to-potassium ratio with obesity in a multiethnic cohort. Am J Clin Nutr 99(5):992–998
20. O'Donnell MJ, Yusuf S, Mente A, Gao P, Mann JF, Teo K, McQueen M, Sleight P, Sharma AM, Dans A (2011) Urinary sodium and potassium excretion and risk of cardiovascular events. Jama 306(20):2229–2238
21. Hedayati SS, Minhajuddin AT, Ijaz A, Moe OW, Elsayed EF, Reilly RF, Huang C-L (2012) Association of urinary sodium/potassium ratio with blood pressure: sex and racial differences. Clin J Am Soc Nephrol 7(2):315–322
22. Remer T, Neubert A, Maser-Gluth C (2002) Anthropometry-based reference values for 24-h urinary creatinine excretion during growth and their use in endocrine and nutritional research. Am J Clin Nutr 75(3):561–569
23. Cole TJ, Flegal KM, Nicholls D, Jackson AA (2007) Body mass index cut offs to define thinness in children and adolescents: international survey. BMJ 335(7612):194
24. Garnett S, Baur L, Cowell C (2008) Waist-to-height ratio: a simple option for determining excess central adiposity in young people. Int J Obes 32(6):1028–1030
25. Lee S-K, Kim MK (2016) Relationship of sodium intake with obesity among Korean children and adolescents: Korea National Health and Nutrition Examination Survey. Br J Nutr 115(05):834–841
26. Kelishadi R, Majdzadeh R, Motlagh M-E, Heshmat R, Aminaee T, Ardalan G, Esmaillzadeh A, Azadbakht L, Poursafa P, Movahedian M (2012) Development and evaluation of a questionnaire for assessment of determinants of weight disorders among children and adolescents: The Caspian-IV study. Int J Prev Med 3(10):699
27. Ghaffarpour M, Houshiar-Rad A, Kianfar H (1999) The manual for household measures, cooking yields factors and edible portion of foods. Tehran: Nashre Olume Keshavarzy 7:213
28. Faghihimani Z, Nourian M, Nikkar AH, Farajzadegan Z, Khavariyan N, Ghatrehsamani S, Poursafa P, Kelishadi R (2010) Validation of the child and adolescent international physical activity questionnaires in Iranian children and adolescents. ARYA Atheroscler 5 (4)
29. Fahimi S, Pharoah P (2012) Reducing salt intake in Iran: priorities and challenges. Arch Iran Med 15(2):110–112. http://www.sid.ir/En/Journal/ViewPaper.aspx?ID=243264
30. Rashidi A, Mohammadpour-Ahranjani B, Vafa M, Karandish M (2005) Prevalence of obesity in Iran. Obes Rev 6(3):191–192
31. Shavit L, Ferraro PM, Johri N, Robertson W, Walsh SB, Moochhala S, Unwin R (2015) Effect of being overweight on urinary metabolic risk factors for kidney stone formation. Nephrol Dial Transpl 30(4):607–613. https://doi.org/10.1093/ndt/gfu350
32. Taylor EN, Curhan GC (2006) Body size and 24-h urine composition. Am J Kidney Dis 48(6):905–915. https://doi.org/10.1053/j.ajkd.2006.09.004
33. Cai X, Li X, Fan W, Yu W, Wang S, Li Z, Scott EM, Li X (2016) Potassium and obesity/metabolic syndrome: a systematic review and meta-analysis of the epidemiological evidence. Nutrients 8(4):183
34. Grimes CA, Riddell LJ, Campbell KJ, He FJ, Nowson CA (2016) 24-h urinary sodium excretion is associated with obesity in a cross-sectional sample of Australian schoolchildren. Br J Nutr 115(06):1071–1079
35. He FJ, Marrero NM, MacGregor GA (2008) Salt intake is related to soft drink consumption in children and adolescents a link to obesity? Hypertension (Dallas, Tex: 1979) 51 (3):629–634
36. Fonseca-Alaniz MH, Brito LC, Borges-Silva CN, Takada J, Andreotti S, Lima FB (2007) High dietary sodium intake increases white adipose tissue mass and plasma leptin in rats. Obesity 15(9):2200–2208
37. Fonseca-Alaniz MH, Takada J, Andreotti S, Campos TB, Campaña AB, Borges-Silva CN, Lima FB (2008) High sodium intake enhances insulin-stimulated glucose uptake in rat epididymal adipose tissue. Obesity 16(6):1186–1192
38. Climent B, Simonsen U, Rivera L (2014) Effects of obesity on vascular potassium channels. Curr Vasc Pharmacol 12(3):438–452
39. Crunkhorn S (2013) Metabolic disease: potassium channel blocker prevents obesity. Nat Rev Drug Discov 12(8):577–577
40. Mariosa LSS, Ribeiro-Filho FF, Batista MC, Hirota AH, Borges RL, Ribeiro AB, Zanella MT (2008) Abdominal obesity is associated with potassium depletion and changes in glucose homeostasis during diuretic therapy. J Clin Hypertens 10(6):443–449
41. Yang Y, Jiang H, Wang M, Korpelainen H, Li C (2015) Male poplars have a stronger ability to balance growth and carbohydrate accumulation than do females in response to a short-term potassium deficiency. Physiol Plant 155(4):400–413
42. Ashcroft FM (2005) ATP-sensitive potassium channelopathies: focus on insulin secretion. J Clin Investig 115(8):2047–2058
43. Murakami K, Sasaki S, Uenishi K, for Nutrition JDSS., Group B (2012) The degree of misreporting of the energy-adjusted intake of protein, potassium, and sodium does not differ among under-, acceptable, and over-reporters of energy intake. Nutr Res 32(10):741–750