Original Article

High Salt Intake
Independent Risk Factor for Obesity?

Yuan Ma, Feng J. He, Graham A. MacGregor

Abstract—High salt intake is the major cause of raised blood pressure and accordingly leads to cardiovascular diseases. Recently, it has been shown that high salt intake is associated with an increased risk of obesity through sugar-sweetened beverage consumption. Increasing evidence also suggests a direct link. Our study aimed to determine whether there was a direct association between salt intake and obesity independent of energy intake. We analyzed the data from the rolling cross-sectional study–the UK National Diet and Nutrition Survey 2008/2009 to 2011/2012. We included 458 children (52% boys; age, 10±4 years) and 785 adults (47% men; age, 49±17 years) who had complete 24-hour urine collections. Energy intake was calculated from 4-day diary and misreporting was assessed by Goldberg method. The results showed that salt intake as measured by 24-hour urinary sodium was higher in overweight and obese individuals. A 1-g/d increase in salt intake was associated with an increase in the risk of obesity by 28% (odds ratio, 1.28; 95% confidence interval, 1.12–1.45; P=0.0002) in children and 26% (odds ratio, 1.26; 95% confidence interval, 1.16–1.37; P<0.0001) in adults, after adjusting for age, sex, ethnic group, household income, physical activity, energy intake, and diet misreporting, and in adults with additional adjustment for education, smoking, and alcohol consumption. Higher salt intake was also significantly related to higher body fat mass in both children (P=0.001) and adults (P=0.001) after adjusting for age, sex, ethnic group, and energy intake. These results suggest that salt intake is a potential risk factor for obesity independent of energy intake. **(*Hypertension*. 2015;66:00-00. DOI: 10.1161/HYPERTENSIONAHA.115.05948.)**
● **Online Data Supplement**

Key Words: 24 hour urinary sodium ■ obesity ■ public health ■ salt intake

It is well established that high-salt (1 g salt=0.4 g sodium) intake is the major cause of raised blood pressure and accordingly leads to cardiovascular diseases.[1,2] Recently, several lines of evidence have also shown that high salt intake is associated with an increased risk of obesity. One reason for this association is that high salt intake stimulates thirst and increases fluid intake[3] and thereby increasing sugar-sweetened beverage consumption.[4,5] It has been shown that 1-g/d increase in salt intake is associated with an increase in sugar-sweetened soft drink consumption of 27 g/d in children and adolescents.[4] The association between salt and obesity may also be partially caused by excessive consumption of processed food that is high in both calorie and salt. However, increasing evidence suggests that there may be a direct link between salt intake and obesity independent of total energy intake.[6–11] Among these studies, some used dietary method,[6,9] which is unreliable in estimating salt intake.[12] Among others who measured salt intake by the most accurate method, that is, 24-hour urinary sodium[12] and also accounted for energy intake, few assessed misreporting, especially underreporting of energy intake, which is highly prevalent[13] particularly in overweight and obese individuals.[14] Our study aimed to determine whether there was a direct association between salt intake (assessed by 24-hour urinary sodium, 24hUNa) and obesity independent of energy intake taking into account potential diet misreporting in both children and adults.

Subjects and Methods

A detailed description of the Methods is provided in the Data Supplement. The abridged methods are given below.

We used the data from the National Diet and Nutrition Survey rolling program (NDNS RP) years 1 to 4 (2008/09–2011/12). We obtained the data from the UK Data Service.[15] The NDNS RP was a rolling cross-sectional study aiming to assess the nutritional status of the general UK population aged ≥1.5 years using a 4-day diary. In total, 1982 children and 2174 adults participated in the NDNS RP core survey. Among them, 458 children (age, ≥4 years) and 785 adults had valid weight and height measurement as well as complete 24-hour urine collection mainly verified by para-aminobenzoic acid[16] and were included in our primary analysis. Our secondary analysis included 67 children and 117 adults who had complete 24-hour urine collection and also participated

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From the Center for Environmental and Preventive Medicine, Wolfson Institute of Preventive Medicine, Barts and The London School of Medicine and Dentistry, Queen Mary University of London, London, United Kingdom (Y.M., F.J.H., G.A.M.); and Department of Epidemiology and Biostatistics, School of Public Health, Peking University, Beijing, China (Y.M.).

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Correspondence to Feng J. He, Wolfson Institute of Preventive Medicine, Barts and The London School of Medicine and Dentistry, Queen Mary University of London, Charterhouse Square, London EC1M 6BQ, United Kingdom. E-mail f.he@qmul.ac.uk

in the Doubly Labeled Water (DLW) substudy. DLW[17] is the most accurate method to measure energy expenditure in free-living individuals[18] and can also provide body fat mass and lean mass.

Height and weight were measured in all participants. Waist circumference was measured in individuals aged ≥11 years. Overweight and obesity was defined as body mass index (BMI) ≥85th (overweight)/95th (obese) centile according to the UK90 reference in children[19] and BMI≥25 (overweight)/30(obese) in adults.[20] Central obesity was defined as waist circumference >102 cm for men and >88 cm for women.[20] In children, central obesity was defined as waist circumference at or above the age- and sex-specific 90th centile according to the International Diabetes Federation recommendation.[21]

Dietary misreporting was assessed using 2 methods. First, we compared dietary energy intake (EI_{rep})/basal metabolic rate to the age- and sex-specific Goldberg cutoffs[22] and classified dietary data into either plausible reports or misreports (underreport and overreport were combined as only 2 individuals were identified as overreporters). Second, we used energy expenditure derived from DLW study as a more accurate measure of total energy intake[23] in our secondary analysis.

Statistical Analysis

Our primary analysis was to explore the relationship between salt intake measured by 24hUNa and weight status. General linear models were used to obtain the adjusted mean BMI and waist circumference across the tertiles of salt intake. Logistic models were performed to identify the association of salt intake with the risk of overweight/obese and central obesity. In multivariate models, we adjusted for age, sex, ethnic group, household income, physical activity, energy intake, and misreporting (1=misreporting, 0=plausible report)[24,25] and in adults with additional adjustment of alcohol consumption, smoking, and education level (model-a). Energy intake was replaced with sugar-sweetened beverages in the second model (model-b). Furthermore, to reduce potential bias caused by residual confounding, we calculated the propensity score[26] of salt intake, which is the conditional probability of consuming higher salt intake, based on all confounders in model-a, and sugar-sweetened beverage consumption. Salt intake and the propensity score of higher salt intake were included in model-c. To further control energy intake, we performed a separate analysis replacing salt and energy intake in model-a with the ratio of salt/energy intake (g/2000 kcal; model-d). Logistic regression on central obesity was not performed for children because the sample size of central obesity in children was small.

Our secondary analysis aimed to examine the association between salt intake measured by 24hUNa and body composition (body lean mass and fat mass) measured by DLW. We used general linear models with adjustment for age, sex, ethnic group, and total energy intake. We also performed a separate analysis using the ratio of salt/total energy intake.

We performed several sensitivity analyses to test the robustness of the results. First, we replaced salt intake measured by 24hUNa with salt intake calculated from the 4-day diary. Second, we excluded individuals who potentially misreported their dietary intakes.

We also performed an additional analysis to explore the association between potassium and obesity using the same methods as for salt, the results of which are provided in the Data Supplement.

Results

Table 1 shows the characteristics of the study population according to tertiles of salt intake for children and adults. The mean salt intake as measured by 24hUNa was 5.5±2.7 (SD) g/d in children and 7.6±3.3 g/d in adults. Compared with the participants in the lowest salt intake tertile, those who consumed more salt tended to be men, older children, younger adults, have more energy intake, and have larger BMI and waist circumference. Children who had higher salt intake had slightly less physical exercise.

Association Between Salt Intake Measured by 24hUNa and Continuous Measurement of Weight Status

As illustrated in Figure, both BMI and waist circumference increased from the lowest to the highest tertile of salt intake (both P for trend<0.001) after adjusting for age, sex, ethnic group, household income, physical activity, total energy intake, and misreporting in children. A similar trend was also observed in adults with additional adjustment for alcohol consumption, smoking, and education level.

Association Between Salt Intake Measured by 24hUNa and Obesity Risk

Table 2 shows the relationship between salt intake and weight status. In children, a 1-g/d increase in salt intake was associated with a 28% increase in the risk of overweight or obesity (odds ratio [OR], 1.28; 95% confidence interval [CI], 1.12–1.45) after adjusting for age, sex, ethnic group, household income, physical activity, total energy intake, and misreporting (model-a). The association was almost identical when total energy intake was replaced with the consumption of sugar-sweetened beverages in model-b (OR, 1.28; 95% CI, 1.12–1.47) or replacing all the confounding factors with the propensity score of higher salt intake in model-c (OR, 1.27; 95% CI, 1.11–1.44; Table 2).

In adults, a 1-g/d increase in salt intake was associated with an increase in the risk of overweight or obesity by 26% (OR, 1.26; 95% CI, 1.16–1.37) in model-a. This figure increased to 28% (OR, 1.28; 95% CI, 1.17–1.39) when total energy intake was replaced with the consumption of sugar-sweetened beverages (model-b) but decreased to 21% (OR, 1.21; 95% CI, 1.13–1.31) when replacing all the potential confounding factors with the calculated propensity score of higher salt intake (model-c). Higher salt intake was also associated with an increase in the risk of central obesity by ≈20% in adults in all 3 models (Table 2).

Further analysis showed that a higher salt/energy ratio was also significantly associated with an increased risk of obesity in both children and adults in model-d (Table 2).

Association Between Salt Intake Measured by 24hUNa and Body Composition

As shown in Table 3, a 1-g/d increase in salt intake was associated with an increase of 0.73 kg (P=0.001) and 0.44 kg (P=0.033) in body fat mass and lean mass, respectively, in children after adjusting for age, sex and ethnic group, and energy intake. In adults, a 1-g/d increase in salt intake was associated with an increase of 0.91 kg (P=0.001) and 0.32 kg (P=0.054) in body fat mass and lean mass, respectively. A separate analysis replacing salt and energy intake with the ratio of salt/energy showed a significant association between salt intake and body fat mass (P<0.05), but the association between salt intake and body lean mass disappeared in both children and adults (Table 3).

Association Between Salt Intake Estimated From Dietary Record and Obesity Risk

In total, 1531 children (age ≥4 years) and 1991 adults completed the dietary record and were included in this sensitivity

Table 1. Demographic Characteristics of the Study Population According to Tertiles of Salt Intake Measured by 24-Hour Urinary Sodium Excretion*

	Children				Adults			
	Lower Tertile	Middle Tertile	Upper Tertile	All	Lower Tertile	Middle Tertile	Upper Tertile	All
Salt intake, g/d	3.1±0.8	5.1±0.6	8.5±2.2	5.5±2.7	4.3±1.1	7.2±0.8	11.5±2.4	7.6±3.3
N	152	155	151	458	261	265	259	785
Male, n (%)	75 (49.3)	75 (48.4)	90 (59.6)	240 (52.4)	77 (29.5)	116 (43.8)	178 (68.7)	371 (47.3)
Age, y	8.6±3.9	10.0±3.7	12.4±3.4	10.3±4.0	51.3±16.4	48.9±17.4	47.0±15.7	49.1±16.6
Ethnic group, n (%)								
White	136 (89.5)	133 (85.8)	128(84.8)	397 (86.7)	247 (94.6)	241 (90.9)	242 (93.4)	730 (93.0)
Non-white	16 (10.5)	22 (14.2)	23(15.2)	61 (13.3)	14 (5.4)	24 (9.1)	17(6.6)	55 (7.0)
Household income, n (%)								
<20 000 per y	34 (25.4)	42 (29.8)	41 (30.6)	117 (28.6)	88 (37.9)	77 (33.2)	87 (37.7)	252 (36.3)
20 000–50 000 per y	65 (48.5)	67 (47.5)	51 (38.1)	183 (44.7)	101 (43.5)	101 (43.5)	107 (46.3)	309 (44.5)
>50 000 per y	35 (26.1)	32 (22.7)	42 (31.3)	109 (26.7)	43 (18.5)	54 (23.3)	37 (16.0)	134 (19.3)
Physical activity†	543.0 (420.5–652.9)	488.2 (368.3–615.7)	472.7 (346.7–560.9)	491.8 (378.8–620.9)	0.8 (0.3–1.5)	0.8 (0.3–1.7)	0.9 (0.3–2.4)	0.8 (0.3 to 1.8)
Energy intake, kcal/d‡	1642.3±328.3	1734.2±364.6	2019.2±422.5	1788.5±402.2	2002.9±435.3	2190.5±427.0	2483.0±555.2	2208.9±508.2
Sugar-sweetened beverage, g/d‡	237.5 (104.0–386.8)	187.5 (75.0–361.5)	240.8 (112.5–421.8)	225.0 (100.0–389.5)	121.3 (0–237.5)	112.5 (0–255.0)	106.3 (0–302.5)	113.8 (0–251.3)
Body mass index, kg/m^2	17.7±3.4	18.6±3.4	20.9±3.9	19.1±3.8	26.0±5.2	27.8±5.0	29.4±5.1	27.7±5.3
Waist circumference, cm	70.8±7.6	72.8±9.4	76.7±9.6	74.4±9.4	87.9±13.5	92.7±13.4	98.7±13.6	93.1±14.2
Highest education qualification								
No qualifications	…	…	…	…	39 (15.1)	43 (16.3)	48 (18.6)	130 (16.6)
General Certificate of Secondary Education	…	…	…	…	66 (25.5)	50 (18.9)	55 (21.3)	171 (21.9)
A level or equivalent	…	…	…	…	67 (25.9)	74 (28.0)	69 (26.7)	210 (26.9)
Degree or equivalent	…	…	…	…	67 (25.9)	72 (27.3)	58 (22.5)	197 (25.2)
Other	…	…	…	…	20 (7.7)	25 (9.5)	28 (10.9)	73 (9.3)
Cigarette smoking								
Current smoker	…	…	…	…	49 (18.8)	38 (14.3)	35 (13.5)	122 (15.5)
Ex-regular smoker	…	…	…	…	57 (21.8)	61 (23.0)	74 (28.6)	192 (24.5)
Never regular smoker	…	…	…	…	155 (59.4)	166 (62.6)	150 (57.9)	471 (60.0)
Alcoholic drink in last 12 mo								
≥5 d/wk	…	…	…	…	40 (16.6)	35 (14.3)	23 (9.7)	98 (13.6)
1–4 d/wk	…	…	…	…	124 (51.5)	123 (50.4)	125 (52.5)	372 (51.4)
<1 d/wk	…	…	…	…	77 (32.0)	86 (35.3)	90 (37.8)	253 (35.0)

*Data are shown in the format of mean±SD, median (P25–P75), n (%) unless otherwise specified.
†Mean counts per minute for children aged <16 and hours spent at moderate or vigorous physical activities for adults aged ≥16.
‡Descriptive dietary data were calculated using the diet records assessed as plausible reports.

analysis. As shown in Table 4, there was a consistent significant association between dietary salt intake and weight status after adjusting for the same confounding factors as the primary analysis in model-a, model-b, and model-d in children, and the results were similar to those in the primary analysis. In adults, the corresponding association became weaker than that found in the primary analysis but was still significant in all analyses for model-a and model-b except for the association with overweight or obese which was borderline significant in model-a (P=0.06). These results were not surprising considering that under-reporting was more prevalent in adults (≈56%) compared with children (≈33%). As salt intake estimated from dietary data always had the issue of misreporting, propensity score may further introduce misclassification bias. The analysis using this index (model-c) was therefore not performed.

A separate analysis by excluding individuals who had misreported dietary energy intakes showed consistently significant and similar results as those from the primary analysis (Table 2) but with a wider confidence interval (Table S1 in the Data supplement).

Discussion

Our study was the first to have explored the association between salt intake measured by 24-hour urinary sodium and obesity in a national representative sample of the UK

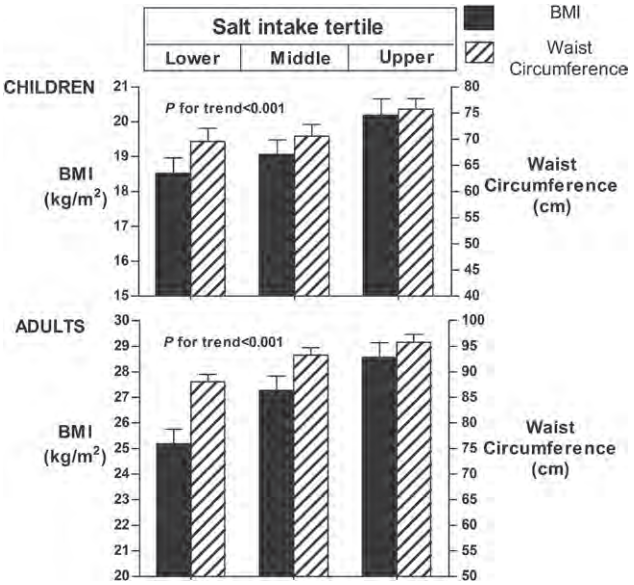

Figure. Adjusted mean body mass index (BMI) and waist circumference according to the tertiles of salt intakes measured by 24-hour urinary sodium excretion.

population. Energy intake was accounted for with critical assessment of diet misreporting. The results showed a consistent significant association between salt intake and various measures of adiposity, including BMI, waist circumference, and body fat mass, after adjusting for potential confounding factors, including total energy intake and sugar-sweetened beverage consumption. These findings suggest a direct association between salt intake and obesity independent of energy intake.

Our findings are consistent with the main body of evidence in this area. Several previous studies showed that salt intake was positively related to weight status[6–11,27,28] and the significant association persisted after adjusting for energy intake[6–11] despite there were methodological problems, such as inaccurate measurement of salt intake[6,9,10] and uncontrolled misreporting of energy intake.

Strengths and Limitations

Our study has several strengths. First, the participants were a national representative sample of the UK population including both children and adults. Second, we used salt intake measured by complete 24-hour urinary sodium excretion mainly verified by para-aminobenzoic acid, which is the most accurate method for assessing salt intake.[12] We further tested the robustness of the results in the sensitivity analysis using salt intake estimated from a 4-day food diary. Third, we assessed the misreporting of dietary energy intake and adjusted for energy intake using different methods. (1) We adjusted for misreporting as a confounder in the primary analysis and further accounted for it by excluding misreports in the sensitivity analysis. (2) We controlled for energy intake by including it as an independent variable or using the ratio of salt/energy intake in different models. These analyses revealed a consistent association between salt intake and obesity independent of energy intake. Finally, we calculated the cutoffs for misreporting using the data derived from the DLW substudy. DLW is the gold-standard method for assessing energy intake,[18] but

Table 2. Association Between Salt Intake Measured by 24-Hour Urinary Sodium Excretion and Weight Status

Weight Status	n (%)	Salt Intake (g/d, SE)	Salt/Energy Intake (g/2000 kcal, SE)	Crude OR		Model-a*		Model-b†		Model-c‡		Model-d§	
				OR (95% CI)	*P* Value	OR (95% CI)	*P* Value	OR (95% CI)	*P* Value	OR (95% CI)	*P* Value	OR (95% CI)	*P* Value
Children (n=458)													
Overweight/obese													
Yes	140 (30.6)	6.4 (0.3)	8.2 (0.5)	1.19 (1.10–1.29)	<0.001	1.28 (1.12–1.45)	<0.001	1.28 (1.12–1.47)	<0.001	1.27 (1.11–1.44)	<0.001	1.13 (1.02–1.24)	0.015
No	318 (69.4)	5.2 (0.1)	6.3 (0.2)	1	…	1	…	1	…	1	…	1	…
Adults (n=785)													
Overweight/obese													
Yes	519 (66.1)	8.4 (0.1)	9.7 (0.2)	1.28 (1.20–1.35)	<0.001	1.26 (1.16–1.37)	<0.001	1.28 (1.17–1.39)	<0.001	1.21 (1.13–1.31)	<0.001	1.19 (1.10–1.27)	<0.001
No	266 (33.9)	6.2 (0.2)	6.8 (0.2)	1	…	1	…	1	…	1	…	1	…
Central obesity													
Yes	314 (40.4)	8.6 (0.2)	10.4 (0.3)	1.16 (1.11–1.21)	<0.001	1.22 (1.14–1.32)	<0.001	1.24 (1.16–1.34)	<0.001	1.18 (1.11–1.26)	<0.001	1.11 (1.06–1.18)	<0.001
No	464 (59.6)	7.0 (0.1)	7.6 (0.2)	1	…	1	…	1	…	1	…	1	…

CI indicates confidence interval; and OR, odds ratio.

*Model-a adjusted for age, sex, ethnic group, household income, physical activity level, energy intake, misreporting for children, and additional confounders, including alcohol consumption, smoking, and education level for adults.

†Model-b replaced energy intake in model-a with sugar-sweetened beverage consumption.

‡Model-c adjusted for the propensity score for higher salt intake.

§Model-d replaced salt and energy intake in model-a with the ratio of salt/energy intake.

Table 3. Association Between Salt Intake Measured by 24-Hour Urinary Sodium Excretion and Body Composition

	Salt Intake, g/d*		Salt/Energy Intake, g/2000 kcal†	
Body Composition	Regression Coefficient	*P* Value	Regression Coefficient	*P* Value
Children (n=67)				
Body fat mass, kg	0.73	0.001	0.53	0.037
Body lean mass, kg	0.44	0.033	0.09	0.767
Adults (n=117)				
Body fat mass, kg	0.91	0.001	1.16	0.003
Body lean mass, kg	0.32	0.054	-0.007	0.984

*Adjusted for age, sex, ethnic group, and energy intake.
†Adjusted for age, sex, and ethnic group.

because of high cost it is hardly used in population-based studies. NDNS RP is one of the few surveys that included DLW method and therefore provided valuable data for us to validate dietary energy intake and to explore the association between salt intake and body composition.

Our study also has several limitations. As a cross-sectional study, the results derived from our study cannot draw a causal relationship between salt intake and obesity. Although our study, in conjunction with other evidence from experimental, cross-sectional as well as prospective cohort studies, indicates that high salt intake is likely to be a contributing factor for obesity, we cannot exclude the possibility that adiposity may predispose people to a higher salt consumption independent of energy intake. Furthermore, 24-hour urine and waist circumference were measured in the second stage of the NDNS RP and there was about 2 to 4 months gap between the first and second stage of the survey. However, because of labor and practical circumstance, this was inevitable in large-scale surveys and the robustness of the results were also tested using salt intake estimated from dietary record which was completed in the same stage as height and weight measurement.

Potential Mechanisms Whereby Salt Is Linked to Obesity

Previous studies have shown that salt intake is associated with obesity through energy intake such as increasing the consumption of sugar-sweetened beverages[4,5] and the coexistence of high salt and energy-dense junk food in diet with poor quality.[29] Our study along with several others suggests that high salt could possibly contribute to obesity independent of energy intake or sugar-sweetened soft drink consumption. The mechanism for such a direct link is not clear. One possible

Table 4. Relationship Between Salt Intake Estimated From 4-Day Food Diary and Weight Status

				Crude OR		Model-a*		Model-b†		Model-d‡	
Weight Status	n (%)	Salt Intake (g/d, SE)	Salt/energy intake (g/2000 kcal, SE)	OR (95% CI)	*P* Value	OR (95% CI)	*P* Value	OR (95% CI)	*P* Value	OR (95% CI)	*P* Value
Children (n=1531)											
Overweight/obese											
Yes	492 (32.1)	5.3 (0.1)	6.5 (0.1)	1.17 (1.10–1.25)	<0.001	1.30 (1.12–1.52)	0.001	1.26 (1.12–1.42)	<0.001	1.22 (1.08–1.37)	0.001
No	1039 (67.9)	5.1 (0.1)	6.2 (0.04)	1		1		1		1	
Adults (n=1991)											
Overweight/obese											
Yes	1266 (63.6)	5.7 (0.1)	6.4 (0.05)	1.13 (1.08–1.19)	<0.001	1.10 (1.00–1.21)	0.062	1.20 (1.11–1.31	<0.001	1.06 (0.97–1.16)	0.173
No	725 (36.4)	5.6 (0.1)	6.2 (0.1)	1		1	…	1	…	1	
Central obesity											
Yes	626 (41.2)	5.6 (0.1)	6.4 (0.1)	1.04 (0.98–1.10)	0.164	1.13 (1.02–1.26)	0.025	1.24 (1.14–1.36)	<0.001	1.08 (0.98–1.19)	0.108
No	894 (58.8)	5.8 (0.1)	6.2 (0.1)	1		1	…	1	…	1	

CI indicates confidence interval; and OR, odds ratio.
*Model-a adjusted for age, sex, ethnic group, household income, physical activity level, energy intake, misreporting for children, and additional confounders, including alcohol consumption, smoking, and education level for adults.
†Model-b replaced energy intake in model-a with sugar-sweetened beverage consumption.
‡Model-d replaced salt and energy intake in model-a with the ratio of salt/energy intake.

mechanism is that if more salt is eaten, this increases the volume of extracellular water which will give rise to a small increase in weight of <1 kg.[30] As shown in several randomized controlled trials, high salt intake could increase body weight in the short term.[31,32] Another mechanism is that salt could directly increase body fat. One experimental study showed that rats in high-salt group had a higher level of plasma leptin concentration as well as excessive accumulation of white adipose fat compared with the rats with lower salt intake.[33] Similar results were observed in epidemiological studies. A cross-sectional study conducted in adolescents revealed a positive relationship between dietary salt intake and subcutaneous abdominal adipose tissue, as well as leptin independent of energy intake.[6] Prospective cohort studies in adolescents[8] and adults[11] showed that baseline salt intake was positively associated with an increase in percentage body fat independent of energy intake. In other words, higher salt intake seems to result in greater deposition of fat suggesting that in some way salt alters body fat metabolism.

Perspectives

Our study using a national representative sample of both children and adults in the UK population showed a significant association between salt intake and various measures of adiposity independent of energy intake or sugar-sweetened beverage consumption. Although the mechanism whereby salt intake is directly related to obesity remains unclear, our findings could potentially have important public health implications. It is well established that a reduction in salt intake lowers blood pressure. Our study, in conjunction with other evidence, demonstrates that salt reduction could also reduce obesity risk. Both decreased blood pressure and obesity will reduce cardiovascular disease.

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Disclosures

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Novelty and Significance

What Is New?

- Our study is the first to have explored the association between salt intake as measured by 24-hour urinary sodium and obesity independent of energy intake with critical assessment of diet misreporting in a national representative sample of the UK population including both children and adults.

What Is Relevant?

- High salt intake is the major cause of raised blood pressure and thereby increases cardiovascular risk.
- Obesity increases blood pressure, type 2 diabetes mellitus, and cardiovascular disease.
- High salt intake is associated with obesity through its effect on increasing the sugar-sweetened beverage consumption. However, it is not known whether there is a direct link between salt intake and obesity.

Summary

Our study showed a consistent significant association between salt intake and various measures of adiposity independent of energy intake.

Our findings could potentially have an important public health implication. A reduction in salt intake could help reduce obesity not only through its effect on reducing sugar-sweetened beverage consumption but may also have a direct effect on lowering obesity risk.

ONLINE SUPPLEMENT

HIGH SALT INTAKE: INDEPENDENT RISK FACTOR FOR OBESITY?

Yuan Ma,[1,2] Feng J He,[1] Graham A MacGregor[1]

[1] Wolfson Institute of Preventive Medicine, Barts and The London School of Medicine & Dentistry, Queen Mary University of London, UK.

[2] Department of Epidemiology and Biostatistics, School of Public Health, Peking University, Beijing, China

Subjects and Methods

Data source and Participants

We used the data from the National Diet and Nutrition Survey rolling programme (NDNS RP) years 1-4(2008/09-2011/12). We obtained the data from the UK Data Service[1]. The NDNS RP was a rolling cross-sectional study originally designed to quantitatively assess the dietary and nutritional status of the general population aged ≥1.5 years living in private households in the UK. The methods of the survey were described in details elsewhere[1], and only methods related to this study were presented here .

The NDNS RP survey selected a core representative UK sample from all four countries and was carried out in two stages. In the first stage, participants were asked to complete a four-day diary, provide demographical and health-related information, and have height and weight measured by nurses. Participants who completed a food diary for at least three days were invited to take further physical measurements including waist circumference and a 24 hour urine collection in the second stage. The elapsed time between these two stages was approximately three months.

In total, 1982 children and 2174 adults participated in the NDNS RP core survey. Among them, 458 children and 785 adults had complete 24h urine collection as well as valid weight and height measurement and were included in our primary analysis. Our secondary analysis included 67 children and 117 adults who had body fat mass measurement. Our sensitivity analysis included 1531 children and 1991 adults who had valid information on salt intake estimated from four-day diary.

Salt intake as measured by 24h urinary sodium excretion

One 24-hour urine collection was made using a standardised protocol in participants aged 4 and over. *Para-aminobenzoic* acid (PABA) recovery, a reliable method of assessing the completeness of collection was used [1, 2]. For participants aged 4-10 and those who did not take PABA, urine collections made between 23-25 hours with no missing collections reported were deemed complete. In total, ≈ 20% urine collection were excluded and among the 1243 urine collections assessed as complete, about 1000 (≈ 80%) were assessed by PABA. Urinary sodium was measured by ion selective electrode on the Siemens Dimension® Xpand clinical chemistry system with the QuikLYTE® module. Salt intake was calculated from 24h urinary sodium excretion.

Indices of anthropometric adiposity

Height and weight were measured for all participants. Waist circumference was measured in individuals aged ≥11 years. Overweight and obesity was defined as body mass index (BMI) ≥ 85th (overweight)/95th (obese) centile according to the UK90 reference in children[3] and BMI≥25 (overweight)/30(obese) in adults[4]. Central obesity was defined as waist circumference greater than 102cm for men and greater than 88cm for women in adults[4]. In children aged between 10 and 17, central obesity was defined as a waist circumference at or above the age-and sex-specific 90th centile according to the International Diabetes Federation (IDF) recommendation [5].

Dietary data collection and assessment

A four-day diary was made under careful supervision. Participants were provided with a diary and asked to keep a record of everything they ate and drank over these four days. For participants aged 11 or younger, a dietary record was kept by a parent. We calculated sugar-sweetened beverage consumption by summing up non-low-calorie soft drinks and fruit juice.

We assessed potential misreporting by comparing dietary energy intake (EI_{rep}) /basal metabolic rate (BMR) to the age- and sex- specific Goldberg cut-offs [6]. EI_{rep} was calculated from diary data and BMR was predicted from age, sex, height and weight using the Schofield's equations[7]. In the Goldberg equation for cut-offs, we used age-and sex-specific physical activity level (PAL) and the between-subject variation in physical activity (CVtp) derived from a double-labelled water(DLW) sub-study in the same population, and we used the average values for variation in basal metabolic rate (CVwb=4%)[6] and within-subject daily variation in energy intake(CVw_{EI}=23%)[6]. We then classified dietary data into either plausible reports or mis-reports (under-report and over-report were combined as only 2 individuals were identified as over-reporters).

Energy expenditure and body composition measured by doubly labelled water

Doubly labelled water (DLW) [8], as the most accurate but expensive method to measure energy expenditure in free-living individuals[9], was used in the NDNS RP in a small but representative sub-population of 165 children and 206 adults, among whom, 67 children and 117 adults had complete 24h urine collection. In the DLW sub-study, participants drank a weighed amount of water labelled with known amounts of the stable isotopes of hydrogen (^2H) and oxygen ($^{18}O2$) based on their body weight. Loss of the two isotopes from body water was assessed by measurement of the rate of decline in concentration of the isotope in samples of the subject's urine, collected during the study period, and measured by isotope ratio mass spectrometry. The difference between the elimination rates of the two isotopes reflected the rate at which CO2 was produced from metabolism. Energy expenditure could then be estimated from the CO2 production. Total body water (kg) could be calculated. Then body fat mass and lean mass was calculated based on the assumption that fat mass has no associated water and lean mass is 73% hydrated using equation 1 and equation 2 as below. A more detailed description of the method can be found in the NDNS report[10].

$$\text{Equation 1: Body Lean mass(kg)} = \frac{\text{Total body water(L)}}{0.73}$$

$$\text{Equation 2: Body fat mass} = \text{Body weight(kg)} - \text{Body lean mass(kg)}$$

Energy expenditure, fat mass (kg) and lean body mass (kg) data derived from DLW study were used to explore the association between salt intake and body composition independent of total energy intake. As total energy expenditure corresponds to energy intake in energy-balanced individuals[11] and is not affected by misreporting, we used energy expenditure rather than the self-reported energy intake as a more accurate estimation of total energy intake in the secondary analysis.

Potential confounding factors

Information on potential confounding factors including age, sex, ethnic group, household income, education level, smoking and alcohol drinking was collected using standardized questionnaires. Physical activity levels were either measured using a physical activity monitor (an ActiGraph) or a self-reported Recent Physical Activity Questionnaire depending on age group. In multivariate regressions, physical activity was entered as a categorical variable according to their respective quintiles.

Statistical analysis

Descriptive statistics including mean ± standard deviation (SD) were reported when variables were normally distributed, otherwise median and interquartile range (25^{th} percentile-75^{th} percentile) were reported.

Our primary analysis was to explore the relationship between salt intake measured by 24-hour urinary sodium and weight status (i.e. BMI, waist circumference as continuous variables and overweight/obese (1=yes, 0=no), central obese (1=yes, 0=no) as categorical variables). General linear models were used to obtain the adjusted mean BMI and waist circumference across the tertiles of salt intake. Logistic models were performed to identify the association of salt intake with the risk of overweight/obese and central obesity. In multivariate models, we adjusted for age, sex, ethnic group, household income, physical activity, energy intake and misreporting(1=misreporting, 0=plausible report) [12,13] and additionally adjusted for alcohol consumption, smoking, and education level in adults in model-a. Energy intake was replaced with sugar-sweetened beverages in model-b. Furthermore, to reduce potential bias caused by residual confounding, we calculated the propensity score [14] of salt intake, which is the conditional probability of consuming higher salt intake, based on all confounders in model-a and sugar-sweetened beverage consumption. Salt intake and the propensity score of higher salt intake were included in model-c. To further control energy intake, we carried out a separate analysis using the ratio of salt to energy intake (g/2000kcal) as an independent variable with adjustment of all other confounding factors (model-d). Logistic regression on central obesity was not performed for children because waist circumference was only measured in individuals aged ≥11 years and the number of children with central obesity was small.

Our secondary analysis aimed to examine the association between salt intake measured by 24-hour urinary sodium excretion and body composition (lean body mass, fat mass) measured by DLW. We used general linear models with adjustment for age, sex, ethnic group, and energy intake. We also carried out a separate analysis using the ratio of salt/energy intake. In this analysis, total energy expenditure measured by DLW was used to estimate energy intake.

We performed several sensitivity analyses to test the robustness of the results. Firstly, we replaced salt intake measured by urinary sodium with salt intake calculated from the four-day diary. Secondly, we performed an analysis that excluded individuals who potentially misreported their dietary intakes (results shown in TableS1 in Data Supplement).

We also performed an analysis to explore the association between potassium and obesity using the same methods as for salt.

Results for the association between potassium and obesity

The results showed that the association between potassium and weight status (obesity, central obesity) was inconsistent and weak, and there was no significant association between potassium and body fat mass after adjusting for confounders including energy intake in all the models.

First, as shown in table S2, potassium intake measured by 24-hour urinary potassium was not significantly associated with obesity in children in the adjusted models but a positive significant association existed for adults. Second, in sensitivity analysis (table S3) using potassium estimated from four-day diary, there was no significant association in all the models for children. The association for adults disappeared in the models adjusted for confounders including energy intake in model-a and model-d) but the association was still significant after adjusting for sugar-sweetened beverage intake in model-b. Finally, as shown in table S4, we further explored the association of potassium intake with body fat using the most accurate measurements, i.e. potassium intake was measured by 24-hour urinary potassium and energy intake was derived from the Doubly Labelled Water method which did not suffer from the issue of dietary misreporting. This analysis showed that there was no significant relationship between potassium intake and body fat in children or adults.

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Table S1. Association between salt intake measured by 24hUNa and weight status (excluding misreported records)

Weight status	N(%)	Misreport (yes/no)	Salt intake (g/d, SE)	Salt / energy (g/1000kcal,SE)	Crude OR		Model-a*		Model-b†		Model-c‡		Model -d§	
					OR	P	OR	P	OR	P	OR	P	OR	P
Overweight/obese in children														
Yes	140 (30.6)	59/81	6.1 (0.3)	6.6 (0.3)	1.18 (1.10-1.30)	0.001	1.31 (1.11-1.55)	0.002	1.33 (1.12-1.58)	0.001	1.32 (1.12-1.55)	0.001	1.22 (1.06-1.40)	0.007
No	318 (69.4)	69/249	5.1 (0.1)	5.8 (0.2)	1		1		1		1		1	
Central obesity in children														
Yes	15 (6.1)	11/4	8.8 (1.5)	8.1 (1.0)	1.43 (0.98-2.10)	0.060	-		-		-		-	
No	231 (93.9)	90/141	6.2 (0.2)	6.3 (0.2)	1		-		-		-		-	
Overweight/obese in adults														
Yes	519 (66.1)	317/202	8.4 (0.2)	7.3 (0.2)	1.24 (1.15-1.34)	<0.001	1.22 (1.09-1.36)	0.001	1.24 (1.11-1.39)	<0.001	1.18 (1.07-1.31)	0.001	1.17 (1.04-1.31)	0.009
No	266 (33.9)	90/176	6.3 (0.2)	6.1 (0.2)	1		1		1		1		1	
Central obesity in adults														
Yes	314 (40.4)	209/105	8.6 (0.3)	7.5 (0.3)	1.15 (1.07-1.23)	<0.001	1.2 (1.08-1.34)	0.001	1.24 (1.11-1.38)	<0.001	1.18 (1.07-1.30)	0.001	1.18 (1.04-1.32)	0.007
No	464 (59.6)	192/272	7.0 (0.2)	6.5 (0.2)	1		1		1		1		1	

*Model-a adjusted for age, gender, ethnic group, household income, physical activity level, energy intake for children and additionally adjusted for alcohol consumption, smoking, education level for adults.

†Model-b replaced energy intake in model-a with sugar sweetened beverage consumption.

‡Model-c adjusted for the propensity score for higher salt intake. §Model-d replaced salt and energy intake in model-a with the ratio of salt/energy intake.

Table S2. Association between potassium(K) intake measured by 24hUK and weight status

Weight status	N (%)	K (mmol/d, SE)	K/energy intake (mmol/2000kcal, SE)	Crude OR		Model-a*		Model-b†		Model-c‡		Model-d§	
				OR	P	OR	P	OR	P	OR	P	OR	P
Children (N=458)													
Overweight/obese													
Yes	140 (30.6)	49.0 (1.7)	62.8 (3.4)	1.013 (1.003-1.024)	0.012	1.015 (0.999-1.031)	0.072	1.015 (0.999-1.032)	0.060	1.010 (0.995-1.025)	0.205	1.006 (0.997- 1.015)	0.201
No	318 (69.4)	44.1 (1.0)	54.1 (1.3)	1		1		1		1		1	
Central obesity													
Yes	15 (6.1)	57.8 (4.4)	65.0 (5.2)	-	-	-	-	-	-	-	-	-	-
No	231 (93.9)	50.3 (1.3)	60.0 (2.2)	-	-	-	-	-	-	-	-	-	-
Adults (N=785)													
Overweight/obese													
Yes	519 (66.1)	76.8 (1.1)	88.5 (1.7)	1.019 (1.012-1.025)	0.009	1.021 (1.011-1.031)	<0.001	1.022 (1.012-1.033)	<0.001	1.016 (1.007-1.025)	<0.001	1.012 (1.003-1.020)	0.005
No	266 (33.9)	65.9 (1.5)	74.0 (1.9)	1		1		1		1		1	
Central obesity													
Yes	314 (40.4)	76.5 (1.4)	92.3 (2.2)	1.008 (1.003-1.014)	0.003	1.019 (1.010-1.028)	<0.001	1.020 (1.011-1.030)	<0.001	1.014 (1.006-1.022)	<0.001	1.007 (1.001-1.013)	0.032
No	464 (59.6)	71.0 (1.2)	77.5 (1.5)	1		1		1		1		1	

24UK=24 hour urinary potassium excretion.

*Model-a adjusted for age, gender, ethnic group, household income, physical activity level, energy intake, misreporting for children and additional confounders including alcohol consumption, smoking, education level for adults.

†Model-b replaced energy intake in model-a with sugar sweetened beverage consumption.

‡Model-c adjusted for the propensity score for higher potassium intake.

§Model-d replaced salt and energy intake in model-a with the ratio of potassium/energy intake.

Table S3. Relationship between potassium(K) intake estimated from 4-day food diary and weight status

Weight status	N(%)	K (mmol/ d, SE)	K/energy intake (mmol/2000 kcal,SE)	Crude model		Model-a*		Model-b†		Model-d§	
				OR	P	OR	P	OR	P	OR	P
Children (N=1531)											
Overweight/obese											
Yes	492 (32.1)	56.2 (0.7)	70.7 (0.7)	0.995 (0.988-1.001)	0.119	0.993 (0.978-1.008)	0.367	1.004 (0.993-1.015)	0.486	0.998 (0.986-1.009)	0.681
No	1039 (67.9)	57.6 (0.5)	70.1 (0.43)	1		1		1		1	
Central obesity‖											
Yes	53 (6.1)	55.8 (2.0)	64.9 (1.9)	0.990 (0.973-1.007)	0.243	-		-		-	
No	813 (93.9)	58.8 (0.6)	68.0 (0.5)	1		-		-		-	
Adults(N=1991)											
Overweight/obese											
Yes	1266 (63.6)	71.7 (0.6)	81.9 (0.5)	1.001 (0.997-1.005)	0.593	1.000 (0.990-1.009)	0.959	1.011 (1.003-1.018)	0.007	0.995 (0.988-1.004)	0.269
No	725 (36.4)	71.1 (0.8)	78.9 (0.7)	1		1		1		1	
Central obesity											
Yes	626 (41.2)	70.7 (0.9)	82.8 (0.8)	0.995 (0.991-1.000)	0.038	0.999 (0.989-1.009)	0.834	1.011 (1.003-1.019)	0.006	0.995 (0.986-1.003)	0.223
No	894 (58.8)	73.1 (0.8)	79.0 (0.6)	1		1		1		1	

* Model-a adjusted for age, gender, ethnic group, household income, physical activity level, energy intake, misreporting for children and additional confounders including alcohol consumption, smoking, education level for adults.

† Model-b replaced energy intake in model-a with sugar sweetened beverage consumption.

§Model-d replaced salt and energy intake in model-a with the ratio of potassium/energy intake.

Table S4. Association between potassium intake measured by 24hUK and body composition

Body composition	Potassium intake (mmol/d)*		Potassium/energy intake (mmol/2000kcal)†	
	Regression coefficient	P value	Regression coefficient	P value
Children(N=67)				
body fat mass, kg	0.01	0.88	-0.03	0.504
body lean mass, kg	0.06	0.08	-0.01	0.832
Adults(N=117)				
body fat mass, kg	0.07	0.07	0.04	0.459
body lean mass, kg	0.04	0.14	-0.04	0.433

24UK=24 hour urinary potassium excretion.

* Adjusted for age, gender, ethnic group and energy intake.

† Adjusted for age, gender and ethnic group.

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Yuan Ma, Feng J. He and Graham A. MacGregor

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