

[ORIGINAL ARTICLE]

Self-reported Slower Eating Is Associated with a Lower Salt Intake: A Population-based Cross-sectional Study

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Abstract:

Objective Evidence suggests that the eating rate is positively associated with the body weight and blood pressure. Furthermore, people who are overweight or obese tend to have higher salt intakes than those of normal weight. To investigate whether or not the eating rate is also associated with the salt intake, a cross-sectional study was conducted using health examination survey data collected in 2014 from 7,941 residents of Sado City, Niigata, Japan.

Methods The eating rates were evaluated using a questionnaire; 11.7% of participants rated themselves as slow eaters, 65.6% as normal eaters, and 22.7% as fast eaters. The salt intake was estimated from sodium and creatinine spot urine measurements using Tanaka's formula. Associations with eating rate were evaluated using multivariate linear regression analyses, with normal eaters as the reference (set at 0).

Results Self-reported eating rates were positively associated with the salt intake after adjustment for age and sex [β coefficient (95% confidence interval) for slow -0.51 (-0.67, -0.35); fast 0.18 (0.05, 0.30)]. Further adjustment for the body mass index showed that slower eaters had lower salt intakes than normal eaters, but there was no marked difference in the salt intake between normal and fast eaters. The association between slower eating and a lower salt intake persisted after further adjustment for comorbidities [slow -0.33 (-0.49, -0.18)].

Conclusion Our results suggest that reducing eating rates may be an effective strategy for reducing dietary salt intake as well as preventing obesity.

Key words: cross-sectional studies, eating behavior, health communication, obesity, salt intake

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Introduction

There is a growing body of evidence to show that eating fast leads to obesity. Many observational studies have shown that the eating rate is positively related to body weight in the general population (1-4) as well as in patients with diabetes (5) or hyperlipidemia (6). Furthermore, interventional studies have shown that slower eating can reduce the food intake (7) and calorie intake (8). These studies suggest that reducing the eating rate may be an effective strategy for preventing obesity.

Obesity has also been associated with a high dietary salt

intake. The salt intake is higher in people who are overweight or obese than in those of normal weight (9-11). Thus, the eating rate may be positively related not only to an increased body weight but also to an increased salt intake, either directly or indirectly through weight gain. To our knowledge, however, no studies have investigated the association between the eating rate and salt intake. If the eating rate was found to be positively associated with the salt intake, even if indirectly through weight gain, then eating slowly may be an effective strategy for reducing dietary salt intake as well as preventing obesity. Because an excess salt intake and obesity increase the blood pressure, eating slowly may also be an effective strategy for preventing hyperten-

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sion.

In this context, we conducted a population-based cross-sectional study to investigate the association between the eating rate and salt intake using data from health examination surveys conducted in a general Japanese population. We hypothesized that the eating rate would be positively related to the salt intake.

Materials and Methods

Study population

We conducted a cross-sectional study of 7,941 adults who attended a health examination in 2014 in Sado City, Niigata, Japan. Details of the health examination survey have been reported elsewhere (12-15). In brief, participants completed a self-administered questionnaire regarding their smoking and drinking habits, medication use, and history of heart disease, stroke, or chronic kidney failure. Trained staff then measured the height, weight, and blood pressure of each participant before collecting serum and spot urine samples. Measurement of urinary sodium and creatinine concentrations in spot urine samples was not mandatory for the health examination but was included in the examination conducted in Sado City. Of the 8,784 participants who underwent a health examination, we excluded 824 with a history of heart disease, stroke, or chronic kidney failure, which can influence the eating rate and salt intake. A further 19 participants were excluded because of missing information, leaving 7,941 participants (3,394 men) for the analysis.

The anonymity of all study participants was maintained, and the study was conducted in compliance with the Declaration of Helsinki and according to Japanese privacy protection laws and ethical guidelines for epidemiological studies published by the Ministry of Education, Science and Culture and the Ministry of Health, Labour and Welfare. The ethics committee of Niigata University Hospital approved the study protocol (IRB No. 2189).

Eating rate

To assess the participants' eating rate, the following question was included in the self-administered questionnaire: 'How fast do you eat?' The responses included 'slow', 'normal', and 'fast'.

Salt intake

The primary outcome measure was the estimated daily salt intake, which we based on the daily urinary sodium excretion, as sodium is mostly excreted into the urine. The daily sodium excretion was estimated from a spot urine sample using the method described by Tanaka et al. (16), as recommended by the Japanese Society of Hypertension (17). The estimated sodium excretion (mEq/day) was multiplied by 0.0585 to convert the values to daily salt intake (g/day) (18).

Covariates

Trained staff measured each participant's height to the nearest 1 mm and weight to the nearest 0.1 kg in a standing position. The body mass index (BMI) was calculated by dividing weight (kg) by the square of height (m). Diabetes mellitus was defined as any use of insulin or oral antidiabetic medication and/or HbA1c $\geq 6.5\%$. Blood pressure was measured according to the recommendations of the Japanese Ministry of Health, Labor, and Welfare (<http://www.mhlw.go.jp/bunya/shakaihoshoh/iryouseido01/info03a.html>) by medical staff using a standard sphygmomanometer or an automated device on the right arm, after the participants had rested for 5 minutes in a seated position. Hypertension was defined as having a systolic blood pressure ≥ 140 mmHg and/or a diastolic blood pressure ≥ 90 mmHg, or taking anti-hypertensive medication. Hypercholesterolemia was defined as use of cholesterol-lowering medication and/or a low-density lipoprotein cholesterol level ≥ 140 mg/dL. Chronic kidney disease was defined as proteinuria in urinalysis and/or a glomerular filtration rate (GFR) ≤ 60 mL/min/1.73 m² (19). Proteinuria was defined as a dipstick urinalysis score of 1+ or greater (equivalent to ≥ 30 mmol/L) because of poor discrimination between negative and trace positive dipstick readings (20). Estimated GFR (eGFR) was calculated using the Japanese equation (21).

Statistical analyses

Participants were divided into three groups according to their self-reported eating speed (slow, normal, or fast). The characteristics of participants for each eating rate category were expressed as the mean [standard deviation (SD)] for continuous variables and percentages for categorical variables. Descriptive statistics for clinical characteristics across categories of self-reported eating speed were compared using the chi-squared test for trends in categorical data and an analysis of variance (ANOVA) for linear trends in continuous variables. Pearson and Spearman correlation coefficients were calculated to evaluate the relationships between variables. A multivariate linear regression analysis was used to estimate changes in the salt intake across eating rate categories, with a normal eating rate as the reference category (set at 0). For model 1, we adjusted for age (continuous, year) and sex. For model 2, we added the BMI (continuous, kg/m²) to model 1. For model 3, we added terms for hypertension (yes/no), diabetes (yes/no), hypercholesterolemia (yes/no), and chronic kidney disease (CKD) (yes/no) to model 2. Multicollinearity was assessed using the variance inflation factor, with a variance inflation factor of >10 considered indicative of serious multicollinearity, and values >4.0 a cause for concern. To assess the robustness of the main results, subgroup analyses according to background characteristics were performed. We included a continuous term for age in the age-stratified subgroup analysis.

All tests were two-tailed, with $p < 0.05$ considered statistically significant. All statistical analyses were performed us-

Table 1. Participant Characteristics according to Self-reported Eating Rate.

Characteristic	Total (n=7,941)	Eating rate			p for trend
		Slow (11.7%)	Normal (65.6%)	Fast (22.7%)	
Estimated salt intake, g/day	9.5 (2.4)	9.0 (2.3)	9.5 (2.3)	9.8 (2.4)	<0.0001
Male, %	42.7	40.5	41.9	46.3	0.001
Age, years	66.9 (13.9)	69.8 (15.4)	67.4 (13.4)	64.0 (14.3)	<0.0001
Body mass index, kg·m ⁻²	22.8 (3.5)	21.7 (3.2)	22.7 (3.4)	23.8 (3.7)	<0.0001
Current smoker, %	12.6	9.9	12.6	13.7	0.03
Daily drinker, %	17.9	14.6	17.9	19.7	<0.001
Systolic BP, mmHg	127 (18)	126 (18)	127 (18)	127 (17)	0.21
Diastolic BP, mmHg	74 (11)	72 (11)	74 (11)	75 (11)	<0.0001
Use of antihypertensive medication, %	36.0	35.6	36.0	36.3	0.75
Hypertension, %	48.8	46.8	49.2	48.9	0.46
LDL cholesterol, mg/dL	115 (28)	112 (28)	115 (27)	117 (28)	<0.0001
HDL cholesterol, mg/dL	56 (15)	58 (15)	56 (14)	55 (15)	<0.0001
Use of cholesterol-lowering medication, %	19.2	16.4	19.3	20.2	0.03
Hypercholesterolemia, %	34.9	30.7	34.9	36.8	0.003
Hemoglobin A1c, %	5.8 (0.6)	5.8 (0.7)	5.8 (0.6)	5.8 (0.7)	0.62
Use of antidiabetic medication, %	6.9	6.5	6.3	8.8	0.003
Diabetes, %	10.3	10.5	9.6	12.3	0.02
Proteinuria, %	4.2	5.5	4.0	4.0	0.13
Creatinine, mg/dL	0.66 (0.21)	0.67 (0.27)	0.66 (0.20)	0.66 (0.18)	0.15
eGFR, mL·min ⁻¹ ·1.73 m ²	76.8 (18.1)	75.1 (19.0)	76.5 (17.9)	78.7 (18.1)	<0.0001
Chronic kidney disease, %	18.3	22.9	18.3	15.9	<0.0001

Definitions of clinical characteristics are provided in the text.

Data represent means (standard deviation) for continuous variables and percentages for categorical variables.

BP: blood pressure, LDL: low-density lipoprotein, HDL: high-density lipoprotein, eGFR: estimated glomerular filtration rate

ing the SPSS statistical package for Windows (Version 18.0; SPSS, Chicago, USA).

Results

The mean (SD) age of the 7,941 participants was 66.9 (13.9) years, and the mean (SD) salt excretion was estimated to be 9.5 (2.4) g per day, with 48.8% of participants classed as hypertensive (Table 1). Regarding the eating rate, 11.7% were self-reported to be slow eaters, 65.6% were normal eaters, and 22.7% were fast eaters. Participants who ate fast were more likely to be male and diabetic. The self-reported eating rate was positively associated with the salt intake (p for trend <0.0001), BMI (p for trend <0.0001), diastolic blood pressure (p for trend <0.0001), low-density lipoprotein cholesterol level (p for trend <0.0001), and eGFR (p for trend <0.0001) and negatively associated with the age (p for trend <0.0001) and high-density lipoprotein cholesterol level (p for trend <0.0001). Systolic blood pressure and the use of antihypertensive medication showed no association with the eating rate, although there were significant differences in the age among the respective categories of each.

In a multivariate linear regression model adjusted for age and sex, the estimated salt intake was positively associated with the eating rate (Table 2). When the model was also adjusted for the BMI, the difference in the estimated salt intake was no longer statistically significant between the fast

and normal eaters but remained significant between the slow and normal eaters. When the model was further adjusted for hypertension, diabetes, hypercholesterolemia, and CKD, the significant difference in the estimated salt intake persisted between the slow and normal eaters. The estimated salt intake of slow eaters was significantly lower than that of normal eaters. When the model was further adjusted for daily drinkers, the association was not changed [β coefficient (95% confidence interval) for slow -0.32 (-0.47, -0.16); fast 0.02 (-0.10, 0.14)].

Figure shows results of the subgroup analyses for which the participants were grouped according to background characteristics. Sex-stratified subgroup analyses revealed that both men and women who ate slower had a significantly lower estimated salt intake than those who ate at a normal speed. Age-stratified subgroup analyses revealed similar associations, except for the group <60 years of age. BMI- or comorbidity-stratified subgroup analyses also revealed similar associations, although in some cases, these did not reach statistical significance, perhaps because of the smaller number of participants in these subgroups.

Discussion

Our study explored the association between the eating rate and dietary salt intake in a general population in Japan. Our results clearly demonstrate that self-reported slower eat-

Table 2. Results of the Multivariate Linear Regression Models for the Association of Estimated Salt Intakes with Self-reported Eating Rate.

Variables in model	Unadjusted	Model 1	Model 2	Model 3
Eating rate				
Slow	-0.58 (-0.74, -0.41)	-0.51 (-0.67, -0.35)	-0.38 (-0.54, -0.22)	-0.33 (-0.49, -0.18)
Normal	0.00	0.00	0.00	0.00
Fast	0.28 (0.15, 0.41)	0.18 (0.05, 0.30)	0.04 (-0.09, 0.16)	0.02 (-0.10, 0.14)
Age		-0.03 (-0.03, -0.02)	-0.02 (-0.03, -0.02)	-0.02 (-0.02, -0.04)
Sex, 1=male and 0=female		0.48 (0.37, 0.58)	0.38 (0.28, 0.48)	0.39 (0.29, 0.49)
Body mass index			0.14 (0.12, 0.15)	0.13 (0.12, 0.15)
Hypertension, 1=yes and 0=no				0.21 (0.10, 0.32)
Diabetes, 1=yes and 0=no				0.23 (0.06, 0.39)
Hypercholesterolemia, 1=yes and 0=no				0.07 (-0.04, 0.18)
Chronic kidney disease, 1=yes and 0=no				-1.32 (-1.45, -1.18)

Data are the β regression coefficient (95% confidence interval), which represent the change in estimated salt intakes from those of the normal eating group.

Model 1: adjusted for age and sex.

Model 2: model 1 further adjusted for body mass index.

Model 3: model 2 further adjusted for hypertension, diabetes mellitus, hypercholesterolemia, and chronic kidney disease.

ing is associated with a lower salt intake, even after controlling for the BMI and other potential confounding factors. To our knowledge, this study is the first to report that slower eating is associated with a lower salt intake. Our findings suggest that eating slowly might be an effective strategy for reducing dietary salt intake as well as preventing obesity and hypertension in the general population.

Although no studies have been published regarding the association between the eating rate and salt intake, some epidemiological studies have shown that the eating rate is related to blood pressure (1-3). A cross-sectional study showed that eating quickly significantly increases the risk of elevated blood pressure in subjects with elevated fasting glucose or diabetes, although this study did not account for obesity status or the BMI (3). Another cross-sectional study in Korean adults showed that fast eating rates significantly increase the odds ratio for elevated blood pressure in men, but this association was no longer significant after adjustment for the BMI (1). A third cross-sectional study of 56,865 participants from a general Japanese population (that used the same self-administered questionnaire to assess eating rate as the present study) found that an association between slow eating and decreased odds of elevated blood pressure remained statistically significant in both men and women after adjustment for the BMI (2). These findings raise the possibility that there may be pathways other than weight gain that underlie the association between slow eating and blood pressure (2). A lower salt intake may be one

such alternative, as our findings show slower eating to be associated with a lower salt intake.

In contrast, the association between fast eating and increased odds of elevated blood pressure was no longer significant after adjustment for the BMI in the previous Japanese cross-sectional study mentioned above (2). Likewise, the association between fast eating and an increased salt intake was also no longer significant after adjustment for the BMI in the present study. These findings suggest that weight gain may be the main pathway underlying the association between fast eating and an elevated blood pressure or excess salt intake. Previous studies have shown that slower eating reduces the food intake (7) and calorie intake (22), thereby resulting in greater satiety after meal completion (22). Faster eating may therefore lead to obesity through the overconsumption of food and energy, which may result in an excess salt intake.

It is also possible that an excess salt intake *per se* may lead to overconsumption of food and energy. Recent studies suggest that the salt intake may increase the obesity risk independent of the energy intake. A 2x2 crossover experimental study of healthy adults demonstrated that the consumption of foods with a higher salt content resulted in an increased food and energy intake independent of the fat content (23). This finding suggests that salt promotes the overconsumption of food and energy. Furthermore, a previous rolling cross-sectional study using a national representative sample of the United Kingdom population revealed a signifi-

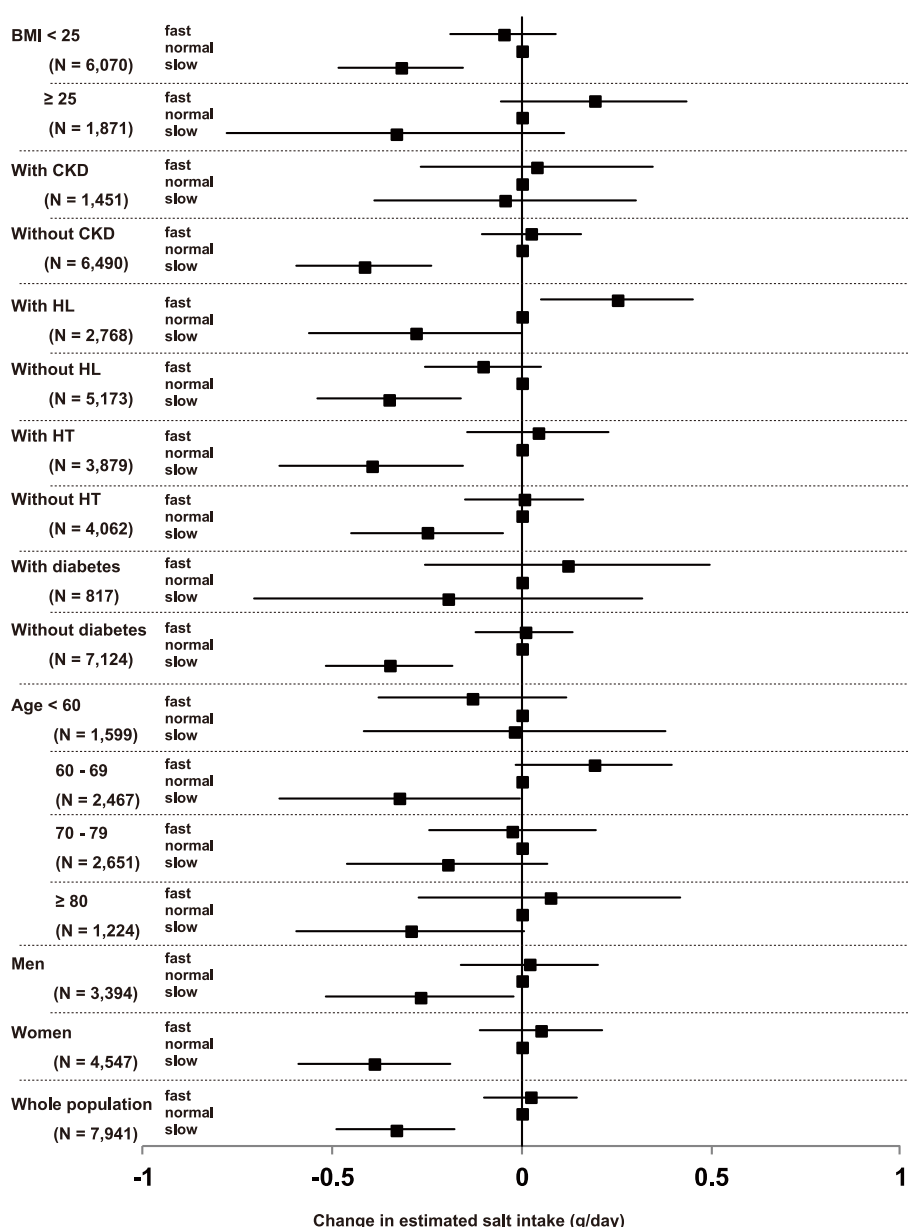


Figure. Subgroup analyses of the association between the self-reported eating rate and estimated salt intake. Data are shown as the means and 95% confidence intervals derived from multivariate linear regression models adjusted for the following covariates (except for variables used to define subgroups in each case): age (years), sex, body mass index (BMI), hypertension (HT; yes/no), diabetes (yes/no), hypercholesterolemia (HL; yes/no), and chronic kidney disease (CKD; yes/no). A self-reported normal eating rate was used as the reference category.

cant association between the salt intake and various measures of adiposity independent of the energy intake or sugar-sweetened beverage consumption (10). In addition, the longitudinal Danish MONICA study showed that the 24-h urinary sodium excretion at baseline was positively associated with the percentage body fat independent of the energy intake (9). These findings show that a high salt intake can result in greater fat deposition, suggesting that salt may affect body fat metabolism. It is therefore possible that an excess salt intake is not only a consequence of obesity but also a potential risk factor as well.

The mechanisms underlying the association between slow

eating and a lower salt intake cannot be determined with certainty based on our data alone, but there are several potential explanations. First, slower eating may lead to a lower salt intake through a reduced food intake, as previous studies have shown that slower eating reduces the food intake and calorie intake and provides greater satiety after meal completion. Second, people who eat slowly may make better food choices, choosing foods with a lower sodium content. However, we did not evaluate the amount or nature of food/energy consumed by participants because this information was not recorded as part of the health examination. Although the mechanisms underlying the association between

slow eating and a lower salt intake are not yet understood, our findings suggest that changing our eating behaviors to reduce the eating rate may prevent an excess salt intake as well as obesity. Because an excess salt intake and obesity are closely associated with elevated blood pressure, eating slowly may also prevent hypertension.

The strengths of our study include its population-based design and large sample size. The relatively homogenous nature of the cohort (all study participants lived on the same small island) helped reduce confounding effects but may limit generalizations. The body height and weight were measured by trained staff, increasing the validity of our study. To our knowledge, this study is the first to report an influence of the eating rate on the salt intake. However, our study also has several limitations. First, the eating rate was self-reported, although self-reported eating rates have been shown to be highly correlated with those reported by a friend or objectively measured (24). Second, the salt intake was estimated from spot urine samples, rather than 24-h urine samples. Spot urine analyses are a less reliable but practical method of such measurements suited to general medical facilities (18). Finally, the cross-sectional study design means that the observed association between the eating rate and estimated salt intake does not necessarily indicate causality.

In conclusion, self-reported slower eating was associated with a lower salt intake in a general population, suggesting that eating slowly may be an effective strategy for reducing the dietary salt intake as well as preventing obesity. Because excess salt intake and obesity cause elevated blood pressure, eating slowly may also be effective for preventing hypertension. Prospective cohort and intervention studies are needed to validate the association between the eating rate and salt intake.

The authors state that they have no Conflict of Interest (COI).

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