



Relationship Between Salt Intake, Salt-Taste Threshold and Blood Pressure in Nigerians

Relation entre l'apport de sel, sel Goût de seuil et la pression artérielle chez les Nigériens

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ABSTRACT

BACKGROUND: Many studies have found an association between sodium intake and blood pressure. Salt taste threshold is thought to be another marker of sodium intake.

OBJECTIVE: This study sought to assess two markers of sodium intake, 24-hour-urinary sodium and salt-taste threshold. We also determined the relationship between these two markers and blood pressure.

METHODS: Salt taste threshold was measured by the ability of the subjects to discern the taste of salt in graded solutions of saline. Twenty-four urinary sodium was measured by flame photometry in a 24-hour urine collection. Other plasma and urine electrolytes and creatinine were measured using standard automated chemistry methods.

RESULTS: There was a significantly higher salt intake measured as 24-hour urinary sodium/mmol of creatinine in the hypertensive group, ($36.6 \pm 20 \text{ mmol/L/mmol creatinine}$) compared with the normotensive group ($14.8 \pm 5.8 \text{ mmol/L/mmol creatinine}$) $p < 0.001$. Urinary potassium was also higher in the hypertensive subjects. When the subjects were grouped into low and high salt taste threshold, the high salt threshold group also had significantly higher 24 hour urinary sodium ($30.3 \pm 5 \text{ mmol/L creatinine}$ vs the low STT urinary sodium of $19.5 \pm 14 p < .05$).

CONCLUSION: Sodium intake measured as 24-hour urinary sodium is increased in subjects with hypertension attesting to sodium intake as a risk factor for the development of high blood pressure. Subjects with high salt taste threshold also have increased urinary sodium excretion which may predispose them to development of hypertension. *WAJM 2011; 30(5): 373–376.*

Keywords: Hypertension, salt-taste threshold, Nigerians, urinary sodium.

RÉSUMÉ

CONTEXTE: De nombreuses études ont trouvé une association entre l'apport en sodium et l'hypertension artérielle. Seuil de goût sel est pensé pour être un autre marqueur de l'apport en sodium.

OBJECTIF: Cette étude visait à évaluer les deux marqueurs de l'apport en sodium, de 24 heures-urinaire de sodium et le sel-goût de seuil. Nous avons également déterminé la relation entre ces deux marqueurs et la pression artérielle.

MÉTHODES: seuil goût de sel a été mesurée par la capacité des sujets à discerner le goût du sel dans les solutions graduées de solution saline. Vingt-quatre de sodium urinaire a été mesurée par photométrie de flamme en un recueil des urines de 24 heures. Autre plasma et l'urine et des électrolytes créatinine ont été mesurés en utilisant des méthodes de chimie automatisés.

RÉSULTATS: On a un apport de sel significativement plus élevée mesurée en 24 heures urinaire de sodium / mmol de créatinine dans le groupe hypertendu, ($36,6 \pm 20 \text{ mmol/L/mmol créatinine}$) par rapport au groupe normotendus ($14,8 \pm 5.8 \text{ mmol/L/mmol créatinine}$) $p < 0,001$. De potassium urinaire était également plus élevé chez les sujets hypertendus. Lorsque les sujets ont été regroupés en seuil de goût sel basse et haute, le groupe sel seuil haut avait aussi significativement plus élevée de sodium urinaire 24 heures ($30,3 \pm 5 \text{ mmol/L de créatinine}$ vs le sodium urinaire basse de la STT ($19,5 \pm 14 p < .05$)).

CONCLUSION: L'apport en sodium mesuré que le sodium urinaire sur 24 heures est augmenté chez les sujets souffrant d'hypertension attestant de l'apport de sodium comme un facteur de risque pour le développement de l'hypertension artérielle. Sujets avec seuil goût sel élevée ont également augmenté excretion urinaire de sodium qui peut les prédisposer à development de l'hypertension. *WAJM 2011; 30(5): 373–376.*

Mots-clés: Hypertension artérielle, le sel-goût de seuil, les Nigériens, urinaire de sodium.

INTRODUCTION

There are reports of an association between salt taste threshold and blood pressure.^{1,2} Salt taste threshold (STT) is the minimum concentration of a salt solution which the taste buds can perceive.^{1,2,3} It has also been shown in previous studies that the taste buds contain taste receptors which express a number of ion channels in their plasma membranes of which the amiloride sensitive sodium channel (ENaC) is one.^{4,5} Sodium appetite depends on salt taste threshold and sodium need for its development, and is said to be a manifestation of sodium intake.^{4,5} A higher threshold reflects a higher salt consumption than normal.^{4,5}

The development of hypertension depends on a complex interaction between genes and environmental factors.³ Since sodium is the main cation in the extra cellular fluid and its source in the diet is sodium chloride, ingestion and absorption of sodium chloride have been reported as major risk factors in the control of blood volume and blood pressure.⁶ Many large scale studies that measured the relationship between long-term salt ingestion and blood pressure did not arrive at conclusive results.⁴⁻⁶ However, the use of controlled diets in which salt intake has been strictly controlled has shown that salt intake is directly associated with the level of arterial blood pressure.⁶

Salt sensitivity is defined as an abnormal blood pressure response to sodium load and salt insensitivity as a normal blood pressure response to sodium load.³ These discrepancies in the relationship between sodium intake, sodium in the extra cellular fluid and urine sodium in different individuals has led to a call for a better method of assessing sodium intake. Several authors have reported that blacks retain more sodium than whites.^{5,7} Pressor responses have been demonstrated in both Nigerian hypertensives and normotensives.⁹

This study sought to determine ways in which sodium intake can be measured, first as 24-hour urinary excretion of sodium and secondly as a measurement of salt-taste threshold. These markers of sodium intake may serve as important indices in the control of blood pressure.

SUBJECTS, MATERIALS, AND METHODS

Forty subjects, 14 men and 26 women, aged 19–70 years, with 18 normotensive and 22 hypertensive, were recruited into this study, performed at the Department of Clinical Pathology, College of Medicine, Lagos, after an informed consent was obtained from them. The subjects were recruited from Idi-Arabia, a suburb located close to the Lagos University Teaching Hospital. The subjects were not on any treatment and also did not know if they were hypertensive or not. They were asked to come for screening to detect hypertension. Hypertension is described in this study as systolic blood pressure greater than 140 mmHg and or diastolic greater than 90 mmHg.

Ethical clearance for the study was obtained from the Research Grants and experimentation ethics committee of the College of Medicine of the University of Lagos.

Exclusion Criteria

Subjects on antihypertensive drugs and subjects with BMI > 30kg/m² were excluded from the study, as were subjects with renal impairment (plasma creatinine level greater than 150µmol/L) and plasma potassium K⁺ greater than 5.5mmol/L

Clinical Procedure

The subjects had their weights and heights measured to the nearest 0.5 kg and 0.5 cm respectively. Blood pressure was measured twice in the sitting position after the subjects had rested for at least 10 minutes, using an Accoson[®] mercury sphygmomanometer. Systolic blood pressure was taken as the point of first appearance of Korotkov sounds and diastolic blood pressure the point of disappearance of the sounds, (Phase V). Two consecutive readings were taken from each subject and the average of these was taken as the mean blood pressure reading. This procedure was repeated on the second visit after 24-hour urine collection was made. The mean blood pressure was taken as the average of the two readings. The subjects were made to complete a questionnaire, which included a family history of hypertension among other questions.

Salt Taste Threshold Measurement

Salt-taste threshold was measured in each subject by the ability to discern the taste of salt in graded solutions of sodium chloride concentration. The salt taste threshold was graded in 5, 15, 30, 60, and 90 mmol/L of salt solution prepared by weighing the amount of sterilised salt and dissolving in distilled and deionised water. Each bottle was coded so as to prevent bias from the examiner and each had a separate pipette to prevent admixture of the different solutions. Three drops of salt solution at room temperature were placed on the tongue of each subject, starting with the lowest concentration, 5mmol/L, and moving up incrementally. The minimum concentration of solution at which the subject was able to discern the taste of salt was taken as the salt taste threshold.^{1,2,11,14,15}

24-hour Urine Collection and Analysis

The subjects were taught the proper collection of urine for the assessment of creatinine clearance. ECA supervised 24-hour urine collection. A four-liter plastic keg was given to each participant; in addition a plastic funnel was supplied to the female participants. They were asked to empty the bladder at 08:00 hours at the start of the collection. All subsequent urine was passed into the keg until the morning of the next day. They were asked to pass the final urine collection into the container at 08:00hours.

Venous blood was taken for measurement of plasma sodium, potassium and creatinine. Plasma and urine concentrations of sodium and potassium were measured with a Corning 4010 flame photometer. Creatinine assays were carried out on plasma and 24-hour urinary samples using the Airone multichannel chemistry autoanalyser which utilises the picric acid method for estimation of plasma creatinine.¹⁶ Urinary clearance of analytes was calculated using standard formula and expressed as volume cleared of analyte (sodium, potassium or creatinine) per (ml/minute).

Statistical Analysis

This was carried out using Microsoft Excel[®] package. Results are presented as mean ± standard error of the mean (SEM) for normally distributed

variables. The student's t-test was used to test for significance of differences in variables. A p value of ≥ 0.05 was considered significant.

RESULTS

There were forty subjects in the study, 14 men and 26 women. Table 1 compares the descriptive variables of participants by blood pressure status. The mean systolic blood-pressure in the normotensive subjects, (n=18) was 116.4 ± 1 mmHg and in the hypertensives, (n=22) was 145 ± 2.6 mmHg. The hypertensive subjects had a significantly higher systolic blood pressure than the normotensive individuals ($p < 0.001$).

The mean salt-taste threshold in the normotensive subjects was 43.3 ± 3.3 mmol/L and in the hypertensive subjects, 41.1 ± 2.9 mmol/L, $p = 0.3$ (Table 2).

Urinary sodium excretion was significantly higher in the hypertensive group, (36.6 ± 3 mmol/L/mmol creatinine), than in the normotensive group, (14.8 ± 0.9 mmol/L/mmol creatinine) $p < 0.001$.

Urinary potassium was also significantly higher in the hypertensive group, (7.3 ± 0.7 mmol/L/mmol creatinine) than in the normotensives group (3.2 ± 0.06 mmol/L/mmol creatinine) $p = 0.003$.

Effect of Salt Threshold

When the results were further divided into those with low salt taste threshold, (STT of 5–30 mmol/L) and those with high salt taste threshold, that is, (STT between 60–90 mmol/L) 22 subjects fell into the low STT group while 15 of the subjects were in the high STT group. A comparison of the variables in the two groups by salt threshold is shown in Table 2.

Mean plasma sodium showed a highly significant difference between the two groups. In the low STT group it measured 135 ± 1.3 mmol/L while in the high STT group it measured 125 ± 1.3 mmol/L, ($p < 0.001$).

Sodium/Potassium ratio in the low STT group recorded a mean of 42.7 ± 0.9 while in the high STT subjects it measured 37.2 ± 0.9 ($p = 0.006$). Urine sodium excretion in the low STT ratio measured 19.5 ± 2 mmol/L/mmol creatinine, while in the high STT ratio it measured 30.3 ± 0.7 mmol/L/mmol creatinine. This was statistically significant. $p = 0.03$.

Table 1: Comparison of Hypertensive and Normotensive Subjects by Study Variables

Variable	Normotensive Group	Hypertensive Group	P
Number	18	22	
Systolic BP (mmHg)	116.4 ± 1.0	145 ± 2.6	0.001
Diastolic BP (mmHg)	77.6 ± 0.8	95 ± 1.4	0.001
Mean age (years)	35.6 ± 2.5	50.6 ± 1.5	0.002
Body mass index (kg/m ²)	23.7 ± 0.4	23.2 ± 0.07	0.01
Plasma sodium (mmol/L)	132 ± 1.2	132 ± 1.7	0.4
Plasma potassium (mmol/L)	7.3 ± 0.7	3.4 ± 0.08	0.1
Creatinine clearance (mls/min)	97.5 ± 4.6	88 ± 2.2	0.1
Salt Taste Threshold (mmol/L)	43 ± 3.3	41 ± 2.9	0.37
Sodium/Potassium ratio	41.5 ± 0.8	40.2 ± 1.1	0.2
Urinary potassium excretion rate (mmol/mmol creatinine)	3.2 ± 0.06	7.3 ± 0.7	0.003
Urinary sodium excretion (mmol/mmol creatinine)	14.8 ± 0.9	36.6 ± 3.0	0.001

Table 2: Comparison of Variables of Subjects by Salt Taste Threshold

Variable	Low Salt Taste Threshold	High Salt Taste Threshold	P
Number	22	15	
Age (years)	45 ± 2.1	44.8 ± 2.4	0.47
Urine Dopamine (μ g/ml)	0.48 ± 0.12	0.71 ± 0.3	0.35
Body Mass Index (kg/m ²)	26.3 ± 0.8	25.6 ± 0.6	0.32
Systolic Blood Pressure (mmHg)	133 ± 3.2	134 ± 4.0	0.34
Diastolic Blood Pressure (mmHg)	88 ± 1.7	86 ± 2.0	0.3
Plasma Sodium (mmol/L)	135 ± 1.3	125 ± 1.3	0.001
Plasma Potassium (mmol/L)	3.2 ± 0.07	3.4 ± 0.01	0.11
Creatinine Clearance (mls/min)	92.5 ± 2.0	99.1 ± 1.2	0.07
Sodium/potassium ratio	42.7 ± 0.9	37.2 ± 0.9	0.006
Urinary Sodium excretion (mmol/mmol creatinine)	19.5 ± 2.0	30.3 ± 0.7	0.03
Urinary Potassium excretion rate (mmol/mmol creatinine)	3.4 ± 0.3	4.8 ± 0.3	0.1

HSTT, high Salt threshold, LST low Salt threshold

DISCUSSION

Urinary Sodium Excretion and Blood Pressure

Urinary sodium excretion was significantly higher in the hypertension group. This finding is in keeping with the general finding of the Inter salt research group which established a link between salt intake as measured by urine sodium excretion and blood pressure.⁶ Urinary potassium excretion was also significantly higher in the hypertensive group. This could be a reflection of increased ENaC activity in the hypertensive patients, as the higher the ENaC activity, the higher the urinary potassium excretion.

There was no difference in salt taste threshold between the normotensive and the hypertensive groups.

Analysis of the normotensive and hypertensive group showed no significant difference in creatinine clearance levels in the two groups. This means that the two groups have similar glomerular filtration rates. This is important when interpreting electrolyte values within two groups, so that low sodium excretion is not attributed to lower glomerular filtration rates.

Salt Taste Threshold and Blood Pressure.

When the subjects were divided into low salt taste threshold and high salt

threshold, urine sodium was significantly higher in the high salt taste threshold group. There was a significantly lower Na/K ratio. Even though not all in this group were clearly hypertensive, the low Na/K ratio suggests probable high renin activity in the high salt taste threshold group and may be an indication that these subjects may become hypertensive in future.¹⁴ Although there was no significant difference in either systolic or diastolic blood pressure in the two groups, the group with high salt threshold has by these findings shown signs of potential development of high blood pressure with time.^{13,14}

Studying salt taste threshold in Nigerians, Obasohan, *et al* (1) found a clear-cut difference between salt taste threshold in normotensive and hypertensive individuals, hypertensives having a higher salt taste threshold. Our findings are not at variance with those of this study as their sample may account for the difference.¹

In our study, the hypertensive group was found to have an increase in urine sodium which is a reflection of increased sodium intake and also an increase in urine potassium which may be a reflection of increased activity of epithelial sodium channels, ENaC. Regrouping the subjects under low salt taste and high salt taste threshold, the high salt taste threshold group was found to have a significantly lower Na/K ratio which is a marker of high renin activity. This marks out the high salt taste threshold group as a potential group for the development of high blood pressure, since not all of them were hypertensive as at the time of this study. However it

should be pointed out that this study was limited by a small sample size. A larger sample size may give a clearer picture.

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REFERENCES

1. Obasohan AO, Ukoh VA, Onyia KA, Isah AO, Salt taste threshold in normotensive and hypertensive Nigerians. *Tropical Cardiology*, 1992; **18**: 183–187.
2. Isezuo SA, Saidu V, Anas S, Tambwal B, Bilbis IS. Comparative analysis of Salt taste perception among diabetics, hypertensives, and diabetic hypertensives. *Nigerian Medical Practitioner* 2008; **53**: 7–10.
3. Rossier BC, Pradervand S, Schild L, Hummler E. Epithelial sodium channels and the control of sodium balance. Interaction between genetic and environmental factors. *Annu Rev Physiol* 2002; **64**: 877–897.
4. Korbmacher C. Proteolytic activation of the epithelial sodium channel (ENaC) in health and disease. *J Med Invest* 2009; **56**: 306–307.
5. Baker EH, Duggal A, Dong Y, Ireson NJ. Amiloride, a specific drug for hypertension in black people with T594 variant? *Hypertension* 2002; **40**: 13–17.
6. Intersalt cooperative research group. Intersalt and intersalt study of electrolyte excretion and blood pressure results for 24 hour urinary sodium and potassium excretion. *Brit Med J*. 1988; **277**: 319–328.
7. Pratt JH, Ambrosius WT, Agarwal R, Eckert GJ, Newman S. *Hypertension* 2002; **40**: 903–908.
8. Sacks FM, Svetkey LP, Vollmer WM, Appel LJ, Bray GA, Harsha D, *et al*. Effects on blood pressure of reduced dietary sodium and the dietary approaches to stop hypertension (DASH) diet. *New Eng J Med*. 2001; **344**: 3–10.
9. Elias SO, Azinge EC, Umoren GA, Jaja SI, Sofola OA. Salt- sensitivity in Normotensive and hypertensive Nigerians. *Nig Qt J Hosp Med*. 2011; **21**: 85–91.
10. Pratt JH, Rebhun JF, Zhou L, Ambrosius WT, Newman S, Gomez-Sanches CE, *et al*. Levels of mineralocorticoids in whites and blacks. *Hypertension* 1999; **34**: 315–319.
11. Conlin PR. Dietary modification and changes in blood pressure. *Current Opinion in Nephrology and Hypertension* 2001; **10**: 359–363.
12. de la Sierra A, Bragulat MD, Larrouse M. Hypertension and salt -sensitivity. Pathophysiology and Clinical implications. CIN2003. <http://www.uninet.edu/cin2003/conf/sierra/sierra.html>.
13. Henkin RI. Salt taste in patients with essential hypertension and with hypertension due to primary hyperaldosteronism. *J of Chronic Diseases* 1974; **27**: 235–244.
14. Lauer RM, Filer LJ, Reiter FA, Clarke WR. Blood Pressure, salt preference, salt threshold and relative weight. *Am J Diseases Children* 1976; **130**: 493–497.
15. Chen J. Sodium sensitivity of blood pressure in a Chinese population. *Curr Hypertens Rep* 2010; **12**: 127–134.
16. Spencer K. Analytical reviews in clinical biochemistry: the estimation of creatinine. *Ann Clin Biochem*. 1986; **23**: 1–25.