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ABSTRACT

Objective: **To determine the salt taste threshold (STT) and salt threshold (STT) and salt intake(SI) in first degree relations of hypertensive and normotensive Nigerians. Hence to determine the relevance of STT in the genesis of hypertension in the Nigerian Africans. The relevance of salt to the development of systemic hypertension continues to attract researchers.**

Design: **A comparative study of STT and salt intake in the first degree offspring of hypertensive and normotensive Nigerians.**

Setting: **University of Benin Teaching Hospital in Benin City, Edo state of Nigeria.**

Subjects: **Fifty three normotensives youths (31 males and 22 females) whose parents were undergoing treatment at the university of Benin Teaching Hospital and 42 age and sex matched normotensive youths (22 males and 16 females) of normotensive parents from similar socio-economic background were recruited for the study.**

Methods: **Salt intake was determined with Corning clinical flame photometer using 24 hour urine sample produced by each participants. STT was determined by a double blind method which employed the forced stimulus drop technique.**

Results: **STT and UNa+ were significantly higher in OH than in ON ($p<0.001$). There was strong positive correlation between STT and UNa+ ($r=0.77$); diastolic blood pressure (DBP) ($r=0.61$); systolic blood pressure (SBP) ($rg=0.54$) and mean arterial pressure (MAP) ($r=0.69$) respectively ($P<0.001$). UNa+ also strongly correlated with DBP ($r=0.59$); MAP $r=0.60$, and SBP ($r=0.36$) respectively $P<0.001$).**

Conclusion: **The study suggests that OH probably consume more sodium than the ON. This tendency to increased sodium (salt) consumption is most likely genetically determined. There is a suggestion that alteration in STT and change in blood pressure tend to occur simultaneously**

INTRODUCTION

The role of salt intake in the development of systemic hypertension has recently been revisited(1). There are experimental, epidemiological and clinical evidence suggesting a causal relationship between salt intake and systemic hypertension(2-4). The tendency of hypertensives to consume more salt has been blamed on elevated STT or higher salt preference in the hypertensive (1-6). Systemic hypertension often runs in families and this familial association could be the result of shared genes or shared environment or a combination of both(3).

Meyer *et al* (7) have demonstrated erythrocyte cation abnormality in one or more members of each consecutive second or third generation of families where either one or two parents, are hypertensive. Abnormality of sodium lithium counter transport has been found to be a highly genetic trait and predictive of hypertension.

There are as yet no studies of STT on pedigree relatives of hypertensives in Nigeria. This study was therefore designed to investigate STT and salt intake in first-degree relations (offsprings) of Nigerian hypertensives in order to ascertain the extent of this problem as genetic familial trait.

MATERIALS AND METHODS

Subjects: The study involved 53 normotensive offsprings of hypertensive Nigerians aged between 15 and 25 years and 42 age and sex matched controls drawn from normotensive offsprings of normotensive Nigerian parents.

The inclusion criteria were as follows:

(i) First degree relation (son or daughter) of hypertensive or normotensive subject.
(ii) Blood pressure $\leq$ 130/80mmHg, in sitting, position.
(iii) Healthy individuals with no concomitant ill health or disability.
(iv) Individuals not on drugs.
(v) Verbal consent of subjects.
(vi) Not a known hypertensive

This study was carried out in the University of Benin Teaching Hospital, Benin City (U.B.T.H.), between the months of July and December 2000. Approval for this study was granted by the Hospital Ethical committee before the study was commenced. The participants included five normotensive youths (31 males and 22 females) of mean age 23.39± 0.10 years, whose parents were undergoing, treatment for systemic hypertension and 42 other normotensive youths (22 males and 16 females) of mean age 23.33± 0.32 years who were children of normotensive parents. For the purpose of this study an offspring of hypertensive (OH) is one whose father or mother or both parents are hypertensive. An offspring of normotensive is one whose father and mother are both normotensive with mean sitting blood pressure of ≥ 130/80. Each participant came from a different family. Systemic hypertension was defined as mean of sitting blood pressure of ≥140/90mmHg, (Korotkov phase V) using standard Accoson Mercury in Glass Sphygmomanometer after resting for 30 minutes at three separate clinic visits. The ON were drawn from families across all social groups similar to those of OH. They included offsprings of normotensive parents: traders, teachers lectures, and hospital workers, all resident in Edo state and its environs. Both parents and offsprings gave their informed consent. Every subject was physically examined and had his or her blood pressure and pulse recorded. A beam balance scale was used to measure their heights and weights. All measurements were done in triplicates and the mean of values was recorded.

Urine collection: On the day of the investigation participants refrained from eating, smoking, alcohol or strenuous exercise for about 30 minutes before being, examined. Each subject was made to produce 24-hour urine collected in a four-liter plastic container. Start and stop of sample collection were done on the collection center. Each 24 hour urine sample was subsequently measured using, a measuring cylinder and recorded. 10 ml aliquot of this sample was then refrigerated at 4°C for subsequent analysis of its sodium and potassium contents using Corning clinical flame photometer 410C. Reference samples were created at the start of the study and were included in each day's analysis to check laboratory variation (2). Every participant also provided a casual (spot) urine specimen for analysis of protein and glucose.

Salt taste threshold: Each participant was made to detect the presence of salt from paired samples of sterile salt solution prepared from analytical sodium chloride. The concentrations were 0mg/dl; 40mg/dl: 80mg/dl; 160m/dl 320mg/dl; 640mg/dl; 1,280mg/dl respectively. The solutions were in stopper containers each with a dropper pipette and were coded (the coding sequence was unknown to both the applicator and the subject). This forced stimulus drop technique was therefore a double blind technique, which does not allow for bias and observer error (6). The applicator only recorded codes that were labeled salty by the subject. Decoding was done only at the end of the testing, of all the subjects for subsequent analysis. Proper identification required that the participant was able to detect salt out of each member of a pair of bottles of the same concentration. All information obtained were fed into the computer and analysed by standard statistical methods. Comparison between the means of two variables was done using the student t-test while Spearman's correlation was used to assess the relationship of two variables. Values of $P<0.05$ were regarded as significant.

RESULTS

Table 1 shows the baseline characteristics of participants. The mean age of OH was 23.39±0.01 years against 23.33±0.32 years for ON. There was no significant difference between the mean age of OH and that of ON or between the mean age for male and female participants ($p\leq0.01$). There was also no significant difference in the means of their body mass index (BMI), heart rate or in the means of their mean arterial pressures (MAP). The two groups however differed in their mean systolic blood pressures (SBP) and mean diastolic blood pressures (DBP) respectively. These were significantly higher in OH than ON (Mean SBP of 118.23±1.38mmHg in OH vs 112.76 ± 2.06mmHg in ON and mean DBP of 75.6±0.74Hg in OH vs 73.63±2.20mmHg in ON ($P<0.01$ and $P<0.05$) respectively.

Table 1

Baseline characteristics of participants, values are given as ± SEM

Groups (No.)	Age (Yrs)	Mean SBP (MmHg)	Mean DBP(MmHg)	Mean MAP(MmHg)	Mean HR (B/M)	Mean BMI(Kg/M^2)
Males (53)	23.13±0.33	118.8±20.79	74.45 ± 0.75	68.66 ± 1.81	73.17 ± 1.39	24.69±0.09
Females (42)	23.10±0.43	117.10±0.75	74.38 ± 1.04	67.66 ± 2.23	72.65 ± 1.20	23.84±0.18
p-value	NS	NS	NS	NS	NS	NS
Males OH (31)	22.96±0.48	122.44±1.04	75.12±1.47	72.36± 1.70	75.22±1.79	24.41±0.18
Females OH(22)	22.81±0.76	121.2± 1.00	75.41 ± 1.58	70.92±1.71	74.50±1.37	24.50±0.02
P-value	NS	NS	NS	NS	NS	NS
Males ON (22)	23.38±0.40	113.79 ±1.16	73.52 ±1.18	64.77 ± 1.74	70.29 ± 2.54	24.45±0.18
Females ON(20)	23.27±0.54	113.33±2.79	73.73 ± 1.21	64.51 ± 1.80	70.70 ± 1.58	24.35±0.25
P-value	NS	NS	NS	NS	NS	NS
All OH (53s)	23.39±0.10	118.23±1.38	75.60 ± 0.74	67.83 ± 1.61	74.34 ± 2.64	23.26±0.71
All ON (42)	23.33±0.32	112.76±2.06	73.63 ± 0.84	63.67 ± 2.20	72.50 ± 1.45	24.20±0.18
P-value	NS	P<0.01	P<0.05	NS	NS	NS

SBP= Systolic Blood Pressure, BMI= Body Mass Index, DBP= Diastolic Blood Pressure MAP= Mean Arterial Pressure, B/m= Beats per minute, NO = Normotensive offspring, HO= Hypertensive offspring, SEM= Standard Error of Mean

Table 2

Mean values of parameters (salt taste threshold, urinary sodium and urinary potassium) in the participants (values are given ±SEM)

Group (No)	STT mg/dl	UNa+ mmol/L	UK+ mmol/L
OH (53)	519.15±34.90	284.25±23.73	35.24±2.86
ON (42)	303.15±26.1	140.60±24.49	19.42±2.54
P-value	P<0.001	P<0.001	P<0.001
All males (53)	437.30±27.78	230.18±9.85	32.33±2.33
All females (42)	390.85±44.85	218.75±24.38	31.48±3.12
P-value	NS	NS	NS
Males OH (31)	521.29±36.06	288.48±27.30	39.34±3.97
Females OH(22)	515±68.9	256.01±33.40	31.42±5.20
P-value	NS	NS	NS
Males ON (22)	320±31.45	132.77±15.2	20.01±2.79
Female ON (20)	265±43.99	152.94±18.22	18.99±3.49
P-value	NS	NS	NS

UNa+ = 24 hour urinary sodium excretion, UK+ = 24 hour urinary potassium excretion, STT = Salt taste threshold

Table 3

Spearman's correlation coefficient between the variables measured in all the subjects

	SBP	DBP	MAP	STT	UNa^+	UK^+	$\frac{UNa}{UK^+}$
SBP	I	-	-	0.54*	0.36*	0.10	0.17
DBP		I	-	0.61*	0.59*	0.34	0.18
MAP			I	0.69*	0.60*	0.08	0.16
STT				1	0.77*	0.24	0.01
UNa^+					I	0.35*	043*
Uk^+						1	0.01
$\frac{UNa}{UK^+}$							1

* = P<0.001

Figure 1

Histogram showing distribution of salt taste threshold among the study population

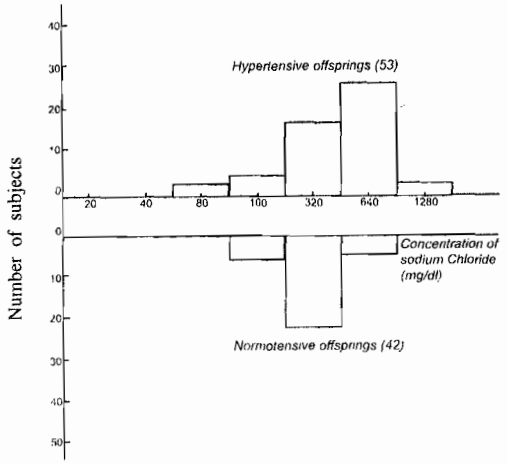

Figure 2

Histogram showing the mean 24 hour urinary sodium (Una+) and urinary potassium (UK) in normotensive offsprings (NO) and hypertensive offsprings (HO)

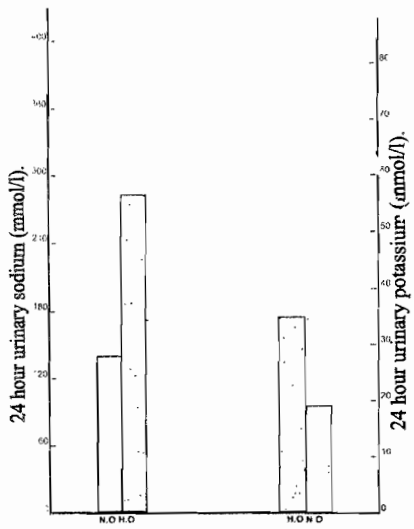

Table 2 shows the means of salt taste threshold, 24-hour urinary sodium and potassium in the participants. Salt taste threshold was significantly higher in the OH than in the ON (P<0.001). Higher urinary sodium UNa^+ and potassium (UK^+) were also found in the OH when compared with ON (P<0.001). In either group there was no significant difference in STT or UNa+ or UK+ between the sexes. Figures 1 and 2 are graphical representations of the STT and urinary electrolytes in the subject population. As can be seen in Figure 1 most ON (63.60%,) had STT of 320mg%, while most of the OH (53.19%) had STT or 640mg%. Table 3 shows the Spearmans correlation coefficient between the variables measured in all the subjects. There was high positive correlation between SBP, DBP, MAP, UNa^+ and STT respectively (P<0.001). A weak correlation was observed between UNa^+ and UK^+ (P<0.001).

DISCUSSION

This study has demonstrated that the offsprings of hypertensives (OH) have higher salt taste threshold than the offsprings of normotensives (ON). Most of the normotensive offsprings (63.60%) had STT of 320mg% while most of the OH (53.19%) had STT of 640mg%. The findings suggest that the tendency of high salt intake in hypertensives runs in the family. Meyer *et al* have demonstrated erythrocyte cation abnormality in one or more members of each consecutive second or third generation families where either one or two parents are hypertensive. Abnormality of sodium-lithium counter transport has been found to be a highly genetic trait and predictive of hypertension(7). The study done by Ikeme *et al* (6) demonstrated higher STT in hypertensive subjects than the normotensive. Obasohan *et al*(4) also demonstrated this in their study. Both studies agree that most normotensives have STT ≤320mg/dl (54.7mmol/L) while most of the hypertensives had STT between 320 to 640mg/dL (109.4mmol/L). This study showed that most OH like the hypertensive subjects in previous studies (4) have STT of 640mg/dl while most ON have STT similar to the normotensive subjects. The implication is that the OH therefore is more likely consume more salt than the ON. Similarly STT correlated positively with MAP, DBP and UNa^+ (Table 3). These findings suggest that alteration in STT or development of high STT probably antedates hypertension, how soon in life this occurs is a question yet to be answered since this study involved only young adults. However, the occurrence of significant difference between SBP and DBP of OH and that of ON (Table 1) suggest that changes in STT and blood pressure may occur simultaneously.

Salt intake as measured by 24-hour urinary sodium excretion was higher in OH than in ON. Several studies have demonstrated positive relationship between high sodium intake and hypertension within population (8,9). Mabadeje *et al*(10) demonstrated high salt intake in the offsprings hypertensives. Since STT measures the affinity for salt, the findings of present study therefore have a lot of implication for the food industry. Perhaps, early introduction of low salt diet to the children may prevent the alteration of STT in the genetically predisposed i.e. in the OH as well as delay the onset of hypertension. Reduction in the salt content in processed food items, which currently form the largest bulk of food items for the younger generation, has therefore become very pertinent.

In conclusion this study has shown that STT is higher in the offspring of Nigerian hypertensives than in the offsprings, of Nigerian normotensives. Early introduction of low salt diet will play a major role in primary prevention of systemic hypertension in the future generation.

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