www.jmscr.igmpublication.org Index Copernicus Value: 79.54 ISSN (e)-2347-176x ISSN (p) 2455-0450 crossref DOI: https://dx.doi.org/10.18535/jmscr/v7i4.105

Jo IGM Publication

Journal Of Medical Science And Clinical Research An Official Publication Of IGM Publication

# Estimation of salt intake assessed by estimation of 24 hour urinary excretion of sodium among pregnant population of Kashmir Valley

Authors

Sakina Akhter<sup>1</sup>, Masood Tanveer<sup>2</sup>, Tanzeela Nazir<sup>3</sup>, Dr Sheikh Mohd Saleem<sup>4</sup> <sup>1</sup>Demonstrator, Department of Physiology, Government Medical College, Srinagar

<sup>2</sup>Professor, Department of Medicine, Government Medical College, Srinagar
 <sup>3</sup>Assistant professor, Department of Obstetrics and Gynecology, Government Medical College, Anantnag
 <sup>4</sup>Demonstrator, Department of Community Medicine, Government Medical College, Srinagar

#### Abstract

**Background:** Hypertension affects approximately 25% of the adult population worldwide, and its prevalence is predicted to increase by 60% by 2025, when a total of 1.56 billion people may be affected. We carried out this study among pregnant women with an aim to determine the dietary salt (sodium) intake patterns of pregnant Kashmiri females and to see its relationship to blood pressure levels as well as to the risk of pre-eclampsia (pregnancy induced hypertension) during the course of pregnancy.

**Methods:** It was a Cohort Study which was conducted from March 2012-March 2013, at Lal Ded Hospital, An Associated hospital of Government Medical College, Srinagar. This study was conducted by the Postgraduate department of Physiology, Government. Medical College Srinagar. The participants enrolled for the study were healthy pregnant females in their first trimester who attended antenatal clinic at Lal Ded Hospital, a 750 bedded maternity hospital associated with Government Medical College, Srinagar.

**Results:** The mean  $\pm$  SD of daily salt intake was 7.07  $\pm$  3.05/day with minimum of 2.70 gm/day and a maximum of 21.01 gm/ day. The mean $\pm$ SD of 24 hour urinary sodium excretion (mmol/24 hours) of normotensive subjects in first trimester was 102.49 $\pm$ 37.1mmol/24 hours, the mean $\pm$ SD of 24 hour urinary sodium excretion in second trimester was 103.55 $\pm$ 30.7mmol/24 hours and the Mean $\pm$ SD of 24 hour urinary sodium excretion in third trimester was 103.60 $\pm$ 30.8 (mmol/24 hours). The Mean 24 hour urinary sodium potassium molar ratio (Na: K) of normotensive subjects was 4.77:1 and that of hypertensive subjects was 8.79:1.The difference in molar ratio was statistically highly significant with P<0.001.

**Conclusion:** The average salt intake in the study participants was higher than recommended. The participants with PIH had increased sodium excretion with increasing during of the pregnancy. Potassium excretion remained constant in our subjects throughout the pregnancy.

Keywords: Cohort Study, PIH, Preeclampsia, Na/K ratio, urinary analysis.

### Introduction

Hypertension affects approximately 25% of the adult population worldwide, and its prevalence is predicted to increase by 60% by 2025, when a total of 1.56 billion people may be affected<sup>(1)</sup>. It is

the major risk factor for cardiovascular disease and is responsible for most deaths worldwide<sup>(2)</sup>. Primary hypertension also known as essential hypertension or idiopathic hypertension, accounts for as many as 95% of all cases of hypertension

<sup>(3)</sup>. Primary hypertension results from interplay of internal derangements (primarily in the kidney) and the external environment. Sodium the main extracellular cation has long been considered the pivotal environmental factor in this disorder. Numerous studies have shown an adverse effect of a surfeit of sodium on arterial blood pressure<sup>(4-7)</sup>. By contrast deficit of potassium the main intracellular cation has a critical role in hypertension and its cardiovascular sequel<sup>(8-10)</sup>. Recent studies as well as classic studies point to interaction of sodium and potassium as compared with an isolated surfeit of sodium or deficit of potassium as a dominant environmental factor in the genesis of primary hypertension and its associated cardiovascular risk.

High blood pressure is a global health problem. Several studies have linked salt intake to the level of blood pressure. Various clinical practice guideline recommendations suggest a restriction in dietary salt intake in hypertensive patients to lower blood pressure. Restriction of dietary salt is widely recommended in the management of hypertension but assessment of individual salt intake has drawn little attention<sup>(11)</sup>. The 2009 Canadian Hypertension Education Programme Recommendation for the management hypertension: Part 2 therapy<sup>(12)</sup>, recommends, restriction of dietary sodium to less than 2300mg (100mmol) / day and 1500mg to 2300mg (65 to 100mmol / day) in hypertension patients. The Working Group for Dietary Salt Reduction of the Japanese Society of Hypertension<sup>(11)</sup> recommends one of the three methods of assessing individual salt intake (a) the measurement of sodium excretion from a 24 hour urine sample or nutritionists analysis of dietary contents, which are reliable but difficult to perform and suitable for facilities specializing in the treatment of hypertension, (b) estimation of sodium excretion from sodium/creatinine ratio in a spot urine is less reliable but more practicable for general medical facilities, (c) estimation using an electronic salt sensor equipped with a calculation formula, though less reliable but can be used by patients

themselves. The Japanese Society for hypertension recommends a salt intake of less than 6gms (102mmol) per day.

An overview of reported studies that used 24 hour urinary excretion to quantify salt intake, shows positive and highly significant correlations between sodium and blood pressure for both men and women and for systolic and diastolic blood pressure. These results are consistent with the INTERSALT findings and those from trials of sodium restriction<sup>(13)</sup>. The measurement of 24 hour urinary sodium excretion is considered the 'gold standard' method for obtaining data of sodium intake in population surveys because of the problems of underestimation of sodium intake based on dietary surveys in most studies<sup>(14)</sup>. Since assessment of salt intake by analyzing dietary patterns presents methodological limitations, and 95% of sodium ingested is excreted in urine, measuring 24 hour urinary sodium excretion is accepted as the most accurate indirect method of determining salt consumption particularly within populations<sup>(15)</sup>. As Kashmir falls in northern region of India and is considered as high salt consuming population mainly for high intake of traditional salt tea in addition to usual diet and there is plausibility of literature on the intake of salt of this particular population. We carried out this study among pregnant women with an aim to determine the dietary salt (sodium) intake patterns of pregnant Kashmiri females and to see its relationship to blood pressure levels as well as to the risk of pre-eclampsia (pregnancy induced hypertension) during the course of pregnancy.

### Methods

Study Design: Cohort Study,

Study Period: March 2012-March 2013,

**Study Site:** Lal Ded Hospital, An Associated hospital of Government Medical College, Srinagar. This study was conducted by the Postgraduate department of Physiology, Government. Medical College Srinagar.

**Study Unit:** The participants enrolled for the study were healthy pregnant females in their first

trimester who attended antenatal clinic at Lal Ded Hospital, a 750 bedded maternity hospital associated with Government Medical College, Srinagar.

**Sample Size:** A total of 274 healthy pregnant women were included in the study.

**Inclusion Criteria:** Normotensive pregnant females in 1<sup>st</sup>trimester.

Exclusion Criteria: Known case of hypertension, gestational diabetes mellitus, women with history of renal diseases, history of steroid intake. Selection of Study participants: Participants who attended Ante natal clinic of the hospital and whose systolic and diastolic blood pressures were found to be <120mm Hg and <80 mmHg respectively, after taking three readings were enrolled for the study after taking informed written consent. A detailed history of all patients was recorded with particular emphasis on the patient's history of blood pressure and cardiovascular disease, history of patient's renal disease and diabetes mellitus, history of any steroid or NSAID intake. A thorough physical examination which included general physical examination, vitals. systemic examination, minimum two blood pressure measurements, weight and BMI were carried out. Baseline investigations carried out in all subjects were hemogram, kidney and liver function test and routine urine examination. The participants were then subjected to 24 hour urine excretion for estimation of sodium and potassium levels. The participants were followed up and the urine sodium and potassium estimation was carried out in  $1^{ist} 2^{nd}$  and  $3^{rd}$  trimester respectively.

**Sample Analysis:** Participants were advised to collect their 24 hour urine in a plastic utensil and bring it to the hospital the following day. Urinary sodium and potassium were determined by Na<sup>+</sup> /  $K^+/C\Gamma^-$ Cc<sub>2</sub> analyzer (Dade Behring Siemens's Biochemistry Analyzer) having multisensor and ion selective (Na<sup>+</sup> /K+ / Cl<sup>-</sup>) electrodes. A reference electrode is also incorporated in the multisensor. This analyzer measures concentration of Na+/ K+ in serum, plasma as well as in urine.

After a diluted sample is positioned in the sensor, Na<sup>+</sup>, K<sup>+</sup> ions establish equilibrium with the electrode surface. A potential is generated proportional to the logarithm of the analytic activity in the sample .The electrode potential generated on a sample is compared to the electrode potential generated on a standard solution ,and the concentration of the desired ions is calculated by use of the Nernst equation.

Estimation of salt intake from 24 hour urinary sodium excretion: The average daily salt intake was then estimated from 24 hour urinary sodium (because 95% of daily dietary sodium intake is excreted in urine) (55) <sup>by</sup> formula (24 hour urinary sodium in mmoles x 17.1 = salt intake in grams/day(66).

**Statistical Analysis:** Data was expressed as Mean±SD and percentage. Nominal data was analysed using chi-square test as applicable. For comparison of groups in case of parametric data two sample t-test, Mann-Whitney-U-test and repeated measures analysis of variance (ANOVA) was used. Statistical package for Social Sciences (SPSS-19) and Microsoft Excel Software was used for analysis.

**Ethical Clearance:** The study had no ethical issue pertaining to animal or human experimentation and was cleared by the Institutional ethical committee.

### Results

The average daily salt intake among study participants is shown in Table 1. The mean  $\pm$  SD of daily salt intake was 7.07  $\pm$  3.05/day with minimum of 2.70 gm/day and a maximum of 21.01 gm/ day. The average salt intake was above the WHO recommendation of daily salt intake (<5gm). Out of all (274) subjects 211 (77%) were consuming salt of more than 5 grams /day and only 63 (23%) were consuming salt  $\leq$  5gram/day (the recommended WHO recommendations).

Table 2 shows the urinary sodium and potassium excretion levels among normotensive and hypertensive participants in  $1^{st}$ ,  $2^{nd}$  and  $3^{rd}$  trimesters of pregnancy. The mean±SD of 24 hour

urinary sodium excretion (mmol/24 hours) of normotensive subjects in first trimester was 102.49±37.1mmol/24 hours, the mean±SD of 24 hour urinary sodium excretion in second trimester 103.55±30.7mmol/24 hours was and the Mean±SD of 24 hour urinary sodium excretion in third trimester was 103.60±30.8 ( mmol/24 hours). The difference in 24 hour urinary sodium excretion of these subjects in different trimesters statistically insignificant with was p=0.615.Moreover, the mean±SD of 24 hour urinary potassium excretion mmol/24 hours of normotensive subjects in first trimester was 21.88±10.; the mean±SD of 24 hour urinary potassium excretion in second trimester was 21.70±8.4 and the mean±SD of these subjects in third trimester was 21.70±8.4.the difference in 24 hour urinary potassium excretion in different trimesters was statistically insignificant with p=0.367.

Table 3 shows the mean  $\pm$ SD of 24 hour urinary sodium mmol/24 hours excretion of hypertensive subjects in first trimester was 182.0±54.93; the mean±SD of 24 hour urinary sodium excretion in second trimester was 196.5±56.93mmol/24 hours and the mean±SD of sodium excretion mmol/day in third trimester was 200.8±57.58.The sodium excretion increased progressively from first to second and second to third trimester and the difference in urinary sodium excretion pattern in three trimesters was statistically highly significant p<0.001 significant. Highest sodium with excretion was seen in third trimester. The mean±SD of 24 hour urinary potassium excretion

mmol/24 hours of hypertensive (PIH) subjects in first trimester was 22.82 $\pm$ 10.35; the mean $\pm$ SD of 24 hour urinary potassium excretion in second trimester was 22.00 $\pm$ 8.4 and the mean $\pm$ SD of these subjects in third trimester was 21.03 $\pm$ 8.4.the difference in 24 hour urinary potassium excretion in different trimesters was statistically insignificant with p=0.077.

Table 4 shows the comparison of average daily salt intake in gm/day among normotensive and hypertensive patients. The mean $\pm$ SD of daily salt intake of normotensive subjects was  $5.99\pm1.87$ gm/day and the Mean $\pm$ SD of daily salt intake of hypertensive subjects was  $11.21\pm3.03$ gm/day. There was a difference of 5.22 grams/day in daily salt intake. The difference between the two groups was statistically highly significant with p<0.001.

The mean $\pm$ SD of daily sodium excretion mmol/24 hours of normotensive subjects of our study was 103.2 $\pm$ 31.19 and the mean $\pm$ SD of hypertensive (PIH) subjects was 193.1 $\pm$ 52.47.There was a difference of 89.9 mmol/24 hours in 24 hour sodium excretion between the two groups.The difference was statistically highly significant with P<0.001 [Table 5].

The Comparison of 24 hour urinary Sodium potassium molar ratio between normotensive and hypertensive subjects is shown in Table 6. The Mean 24 hour urinary sodium potassium molar ratio (Na: K) of normotensive subjects was 4.77:1 and that of hypertensive subjects was 8.79:1.The difference in molar ratio was statistically highly significant with P<0.001.

	Number of subjects	Minimu intake gr		Maximum salt intake gm/day	Mean	SD	Normal WHO daily Recommendation		
Salt intake	274	2.70	)	21.01	7.07	3.05	<5gm/day		
Salt intake grams/day			Number of subjects				Percentage % age		
< 5 gm			63				23.00		
> 5 gm			211				77.00		
Total			274				100.00		

**Table 1:** Average daily salt intake among study participants percentage

Table 2: Urinary Sodium and Potassium excretion levels (mmol/24 hours) among normotensive participants
in different trimesters of pregnancy

Trimester	No.of Participants	(Min; Max)	Mean	SD	SEM	p-value
Sodium						
Trimester-1	217	(44.7;268)	102.49	37.1	2.5	
Trimester-2	217	(57;260)	103.55	30.7	2.1	0.615
Trimester-3	217	(45;264)	103.60	30.8	2.1	
Potassium						
Trimester-1	217	(4.3;59)	21.88	10.2	0.7	
Trimester-2	217	(5.7;47)	21.70	8.4	0.6	0.367
Trimester-3	217	(7.0;52)	21.27	8.4	0.6	

NB: SD=Standard Deviation, SEM= Standard Error of Mean, P-Value <0.05 significant

**Table 3:** Urinary Sodium and Potassium excretion levels (mmol/24 hours) among hypertensive subjects in different trimesters of pregnancy

Trimesters	N0. of Participants	(Min; Max)	Mean	SD	SEM	P value
Sodium						
Trimester-1	57	(41; 367)	182.0	54.93	7.275	
Trimester-2	57	(72.1; 370)	196.5	56.52	7.486	< 0.001*
Trimester-3	57	(80.0; 370)	200.8	57.58	7.626	
Potassium						
Trimester-1	57	(6;52.1)	22.82	10.35	1.371	
Trimester-2	57	(7.8;45)	22.00	10.05	1.331	0.077
Trimester-3	57	(7.2;60)	21.03	10.90	1.444	

NB: SD=Standard Deviation, SEM= Standard Error of Mean, P-Value <0.05 significant

Table 4: Comparison of average daily salt intake gm/day between normotensive and hypertensive subjects

Variable	N	Mean	SD	Mean Diff.	95 % C.	p-Value	
variable	14	Mean	50		Lower Limit	Upper Limit	p-value
Normotensive	217	5.99	1.87	-5.213	-5.846	-4.580	< 0.001
Hypertensive	57	11.21	3.03	-5.215	-3.840	-4.380	< 0.001

**Table 5:** Comparison of Average daily Sodium excretion mmol/24 hours Levels between Normotensive subjects and Hypertensive (PIH) subjects

Variable	Number of	Mean SD		Mean Diff.	95 %	p-Value	
variable	subjects	Mean	50	Mean Diff.	Lower Limit	Upper Limit	p-value
Normotensive	217	103.2	31.19	-89.9	-100.6	-79.2	< 0.001
Hypertensive	57	193.1	52.47	-09.9	-100.0	-19.2	< 0.001

 Table 6: Comparison of 24 hour urinary Sodium potassium molar ratio between normotensive and hypertensive subjects

Study subjects	Sodium Mean±SD	Potassium Mean±SD	Molar Ratio Na : K ratio	p-value
Normotensive	103.2±31.19	21.61±8.4	4.77:1	< 0.001
Hypertensive	193.1±52.47	21.95±56.52	8.79:1	<0.001

### Discussion

Hypertension is a global health problem affecting nearly 25% of population worldwide<sup>(1)</sup>. Hypertension is a major risk factor for cardiovascular disease and is thus responsible for most deaths worldwide<sup>(2)</sup>. Primary hypertension which is responsible for 95% of all cases of hypertension results from interplay of internal derangements and external environmental factors including obesity, sedentary lifestyle, and alcohol. Of the various factors the most important is the consumption of salt<sup>(11)</sup>. Epidemiological, clinical

and animal experiment studies have shown a direct relationship between increased dietary salt (sodium) consumption and blood pressure<sup>(8-10)</sup>. The dietary sodium intake can be assessed by diet surveys but are less accurate. Sodium excretion in 24 hour urine has been known to be the best method to estimate sodium intake<sup>(14)</sup>.

In our study on 300 pregnant females followed in all the three trimesters. The average daily salt intake was 7.07 gm. This goes with the studies by Lopez S et  $al^{(16)}$ .

In our study 26 participants were lost during follow up. Of the remaining 274 participants, 57 (20%) developed hypertension, In India, the incidence of pregnancy induced hypertension is 8-10%. M.V Bhatt<sup>(17)</sup> reported 14.7% incidence of PIH in their study. In our study, the number of Participants with PIH was high. The mean daily salt intake in our study population was 7.07 gm/day in comparison to WHO recommended daily salt intake of < 5gm/day. Seventy seven percent participants in our study exceeded the daily recommendations. This compares to Roso M et al<sup>(16)</sup> who in their study found 88% subjects had salt intake above the recommended levels.

The mean dietary salt intake in those pregnant subjects who developed hypertension was 11.2 gm/day as compared to normotensive pregnant females. The higher intake of salt in the hypertensive group corresponds to the hypertensive effect of sodium and was statistically significant. Our results were similar with Stamler J et al<sup>(18)</sup> who in their study found that salt intake significantly correlated with blood pressure. Catherine et al<sup>(19)</sup>, RA Jan et al<sup>(20)</sup> also found a direct relationship of sodium intake/excretion in perpetuation or causation of hypertension.

In normotensive pregnancies in our study, there was no statistically significant difference in the pattern of urinary sodium excretion in the three trimesters. However, in our study, pregnant participants who developed PIH over the period of time had increased urinary sodium excretion with progression of the pregnancy with highest urinary sodium excretion in 3<sup>rd</sup> trimester. This may

suggest a higher intake of salt as the pregnancy progressed. However this is contrary to the logic that a hypertensive pregnant female would consume more and more salt inspite of being diagnosed hypertensive. This could be possible that pregnant women may consume more salt due to decreased gustatory sensation or due to salt craving under the hormonal effect. Reisberg et al <sup>(21)</sup> in the study found that pregnant women with PIH excrete higher levels of sodium especially in the third trimester due to excess of plasma oxytocin levels.

Potassium intake/excretion is believed to be protective against hypertension. However in our study, there was no difference in urinary potassium excretion between hypertensive and normotensive groups. Also there was no significant difference in the urinary potassium excretion in different trimesters. Thus, potassium excretion was held constant during the entire course of pregnancy. Our finding were consistent with Brown MA et al<sup>(22)</sup> who from their study on 49 primigravidae women suggested that potassium excretion is held constant throughout pregnancy.

In our study the ratio of urinary sodium to potassium was significantly higher in hypertensive group than that of normotensive group. Although this might suggest a protective role of lower urinary sodium to potassium ratio but in our study the higher ratio in hypertensive group was due to higher urinary excretion of sodium in second and third semester in the presence of constant urinary potassium excretion. Thus the ratio is not protective in pregnancy as per our study.

### Conclusion

The average salt intake in the study participants was higher than recommended. The participants with PIH had increased sodium excretion with increasing during of the pregnancy. Potassium excretion remained constant in our subjects throughout the pregnancy.

**Conflict of Interest:** None Declared **Source of Funding:** None

## Acknowledgement

Authors are highly grateful to all the participants who took part in this study. Authors would like to acknowledge the support and technical help Sheikh Mohd offered by Dr. Saleem. Demonstrator from the Department of Community Medicine, Government Medical College in preparation of this manuscript. Authors would like to thank Head of Departments, Department of Obstetrics/Gynecology, **Biochemistry** and Government Medical College, Srinagar for their support and help. All the authors approved the final version of the manuscript before submission.

#### References

- Kearney PM, Whelton M, Reynolds K, Muntner P, Whelton PK, He J. Global burden of hypertension: analysis of worldwide data. Lancet 2002; 360: 1347-60et.
- Ezzati M, Lopez AD, Rodgers A, Vander Hoorn S, Murray CJ. Selected major risk factors and global and regional burden of disease. Lancet 2002; 360: 1347-60.
- Kaplan NM. Primary hypertension: Pathogenesis. In: Kaplan NM, ed. Kaplan's clinical hypertension 9<sup>th</sup> ed. Phledelphia :Lippincot William's and Willkin's,2006:50-121.
- Williams GH, Hollenberg NK. Nonmodulating hypertension: a subset of sodium-sensitive hypertension. Hypertension 1991; 17: Suppl 1
- 5. O'Shaughnessy K M, Karet FE. Salt handling and hypertension. J Clinc Invest 2004;113:1075-81.
- Elliott P. Salt and blood pressure. In: Izzo JL Jr, Black HR, eds. Hypertension primer. 3rd ed. Dallas: American Heart Association/Council on High Blood Pressure Research, 2003: 277-9
- Iwamoto T, Kita S. Hypertension, Na<sup>+</sup>/Ca<sup>2+</sup> exchanger, and Na<sup>+</sup>, K<sup>+</sup>-ATPase. Kidney Int 2006; 69: 2148-54.

- Whelton PK. Potassium and blood pressure. In: Izzo JL Jr, Black HR, eds. Hypertension primer. 3rd ed. Dallas: American Heart Association/Council on High Blood Pressure Research, 2003: 280-
- 9. He FJ, MacGregor GA. Beneficial effects of potassium. BMJ 2001; 323: 497-501.
- Washington, DC: Dietary reference intakes for water, potassium, sodium, chloride, and sulfate. National Academics Press, 2005.
- Yuhei Kawano, Katsuyuki Ando, Hideo Mastuura, Takuya Tsuchihashi et al. Report of working group of dietary salt reduction of Japanese Society of Hypertension: Rationale for salt restriction
- 12. Nadia A. Khan, Brenda Hemmelgarn, Robert J Herman, ChaimM Bell, Jeff L Mahon et al. The 2009 Canadian Hypertension Education Programme, Recommendation for the management of hypertension: Part 2 therapy. Can J Cardiol 2009 May; 25(5): 282-298.
- 13. Elliot P. Observational studies of salt and blood pressure. Hypertension 1991 Jan; 17 (Suppl. 1): 3-8.
- WHO 2007. Reducing salt intake in populations: report of a WHO forum and technical meeting. 5-7 Oct. 2006, Paris France, Geneva: WHO.
- Frost CD, Law MR, Wald NJ. By how much does dietary salt reduction lower blood pressure? II – Analysis of observational data within populations.BMJ 1991; 302(6780): 815-8.
- Rosa M. Ortega, Ana M, Sobaler –Lopez et al. Estimation of salt intake by urinary sodium excretion. British Journal of Nutrition. 2000; 1007.
- 17. Weinberger MH: Salt sensitivity and blood pressure in humans. Hypertension. 2007; 27: 481-90.
- Paul Elliot. INTERSALT: an international study of electrolyte excretion and blood pressure. Results for 24 hour urinary

sodium and potassium excretion. BMJ July 1988; Vol. 29.

- 19. Catherine E Huggens, Sharteen O Reilly, Maree Brinkman Allison, Hodge et al. Relationship of urinary sodium –potassium ratio to blood pressure in older adults in Australia. Med J Aust. 2011;195(3):128-132.
- 20. RA Jan, S Shah, SM Saleem, A Waheed et al. Sodium and potassium excretion in normotensive and hypertensive population in Kashmir. JAPI 2006; 54.
- 21. Reisberg A, Sjoquist M, Lyrenas S, Olsson K."Hormonal changes correlated to excretion of sodium and potassium in urine of pregnant women."Congress of Scavendian Physiological society.1999.
- 22. Tuomilehto Jakko, Karppanen Heikki, Tanskanen Antti, Jorma Tikkanen, Juhani Vuori 'sodium and potassium excretion in a sample of normotensive and hypertensive person in Eastern Finland. Journal of Epedemiology and Community Health.1980,34,174-178.