Asian Pac. J. Health Sci., 2015; 2(3):86-93 e-ISSN: 2349-0659, p-ISSN: 2350-0964

Biochemical parameters-wise hypertension in an Indian community

Maharaj Biswas*, Chanchal Kumar Manna

Endocrinology laboratory, Department of Zoology, Faculty of science, University of Kalyani, Kalyani-741235, Nadia, West Bengal, India

ABSTRACT

Background: Now-a-days, hypertension is a most common cardiovascular disease and it accounts for large proportion of all cardiovascular deaths and disabilities worldwide. The main aim of this study is to show the prevalence and distribution of hypertension according to the various kinds of biochemical parameters within the studied people.

Materials and methods: Here a community-based survey work has been conducted using pre-structured questionnaire for identifying the prevalence and distribution of hypertension in relation to various kinds of factors like sociodemographics, anthropometric and biochemical parameters of blood and urine samples within the Scheduled Caste Community of three selected villages (viz. Chowgachha, Bagula and Priyanagar) of the District Nadia, West Bengal, India. The members of households, who were between 20-70 years of age, were interviewed and examined to collect the data regarding hypertension. Blood and urine samples were also collected by a registered medical practitioner from willing participants for the biochemical study. Data were analyzed using Chi-square test at both 5% and 1% levels of significance.

Results: The biochemical study of blood showed that there was a significant increase of blood pressure with increasing blood glucose, total-cholesterol, triglyceride, LDL-C and Na^+ level. Although the optimal level of HDL-C and K^+ of blood showed the potency of blood pressure regulation. The people those who take additional dietary salt had the greater tendency of blood serum Na^+ as well as blood pressure.

Conclusion: The results of this study clearly indicate that there was a significant role of hyperglycemia, dyslipidemia and high serum Na^+ level in the development of hypertension.

Keywords: Hypertension, Cardiovascular disease, Scheduled Caste Community, Hyperglycemia, Dyslipidemia.

Introduction

Hypertension or abnormally high blood pressure is the leading cause of cardiovascular disease worldwide. It remains silent, being generally asymptomatic during its clinical course. As it is hidden beneath an outwardly asymptomatic appearance, the disease does immense harm to the body in the form of 'Target Organ' damage, hence, it is the 'Silent Killer' [1].

Hypertension and dyslipidemia are widely recognized risk factors for cardiovascular disease [2, 3]. Approximately 40% of the persons with essential hypertension also have hypercholesterolemia. Genetic studies have established a clear association between hypertension and dyslipidemia [4].

Increasing age, body mass index, central obesity and impaired glucose tolerance/diabetes were associated with both hypertension and prehypertension in an affluent north Indian population. Hypertension and type 2 diabetes mellitus also tend to coexist. Hypertension is approximately twice as common in persons without diabetes, and the association is even stronger in African Americans and Mexican Americans. The leading cause of death in patients with type 2 diabetes is coronary heart disease, and diabetes increases the risk for acute myocardial infarction as much as a previous myocardial infarction in a nondiabetic person [5]. Hypertension, insulin resistance, dyslipidemia and obesity often occur concomitantly [6].There was a high prevalence of cardiovascular risk factors like central obesity, elevated LDL-cholesterol, abnormal glucose tolerance and smoking in the general population. Two or more of

**Correspondence*
Dr. Maharaj Biswas
Endocrinology laboratory, Department of Zoology, Faculty of science, University of Kalyani, Kalyani-741235, Nadia, West Bengal, India
E Mail: maharajabiswasbwn@gmail.com

these risk factors were present in a higher proportion of hypertensive and pre-hypertensive, compared to normotensive subjects [7]. Several epidemiological and clinical studies have demonstrated that a low or reduced intake of salt is associated with lower blood pressure [8]. The etiological studies investigating the relation between salt intake and stroke have generally produced positive associations [9, 10]. Studies in animals have shown that a high salt intake significantly increases renal excretion of water due to increased water intake [11-13]. When salt intake was increased there was an increase in plasma sodium and an increase in plasma osmolality, which is known to stimulate thirst and antidiuretic hormone secretion [14]. The biochemical factors which are associated with the development of hypertension within the Scheduled Caste community are not studied elsewhere. To save this community from the harmful effects of hypertension, we estimated the current prevalence and distribution of hypertension according to the different serum biochemical factors within this Scheduled Caste community of the District Nadia, West Bengal, India.

MATERIALS AND METHODS

Population based survey

A cross sectional door-to-door community based survey work was conducted to investigate the prevalence of usual salt-related hypertension within the SC community of three selected villages (viz. Chowgachha, Bagula and Priyanagar) of the district Nadia, West Bengal, India. About 2,453 members of households of study areas were interviewed and detailed information (i.e., age, sex, weight and height for BMI, waist and hip for WHR, HR, blood pressure, physical activity, oil intake, salt intake etc.) regarding hypertension were recorded. Average age of the studied individuals is approximately 41.58±14.25 (range, 20-70 yrs). Data were collected from 10.00 am to 4.00 pm.

Definition criteria and Blood pressure measurement

The seventh report of the Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure (JNC-VII report) has recommended a new classification for adults-18 years or older: Normal: <120/80 mmHg, pre-hypertensive: 120-139/80-90 mmHg and hypertensive: >140/90 mmHg [15]. The blood pressure was measured by the authentic mercury and digital (HEM-7111 and HEM-4030, Omron healthcare, Kyoto, Japan) sphygmomanometer in supine position after taking rest at least for 10 minutes.

Blood and urine biochemical analysis

The biochemical analysis of serum and urine was done in 114 people of the study areas. Out of 114 people, forty three (43) hypertensive and seventy one (71) were normotensive (NTN) people. The studied people took their usual diet and were instructed to collect 24-hour urine sample for the measurement of volume and electrolytes (Na^+, K^+ and Cl^+). Fasting blood sample was also collected for serum electrolytes (Na^+, K^+ and Cl^+) and other biochemical analysis like blood sugar, Hb% and lipid profile regarding hypertension.
The biochemical analysis was done by a semi-automatic biochemical analyzer (Prietest-easy lab, Robonik India Pvt. Ltd., Mumbai, India).

Statistical analysis

Data were expressed as Mean ± SEM or SD, and were analyzed using Chi-square test at both 5% and 1% levels of significance. SPSS 12.0 software for windows was used to analyze the data.

Results

The prevalence and distribution of hypertension according to different kinds of biochemical factors of blood and urine of willing candidates (N=114) during data collection regarding hypertension are depicted in the Table-1. The 26.31% of the total studied sample is known to be hypertension.
From the analyzed data it is found that hypertension is common in those groups who have higher level of blood glucose (blood glucose: <100mg/dl = 20.68% and ≥100 mg/dl = 44.44%), total cholesterol (Total cholesterol: <175mg/dl = 15.25% and ≥175mg/dl = 38.18%), Tg (Tg: <100mg/dl = 10.81% and ≥100 mg/dl = 33.76%), LDL-C (LDL-C: <100mg/dl = 9.09% and ≥100 mg/dl = 33.33%), serum sodium ions (Na^+ : <140mmol/L = 12.72% and ≥140 mmol/L = 38.98%) and urine potassium ions (K^+ : <75mmol/L = 7.69% and ≥75 mmol/L = 36%) level whereas inverse relationship of hypertension occurs in blood HDL-C (HDL-C : <40mg/dl = 39.62% and ≥40 mg/dl = 14.75%), urine Na^+ (Na^+ : <100mmol/L = 44.44% and ≥100 mmol/L = 10%) and serum K^+ (K^+ : <4mmol/L = 48.07% and ≥4 mmol/L = 8.06%) groups. Hypertension is independent with the change of serum VLDL-C, serum Cl^- and urine Cl^- level.
The Chi-square test for the test of independence attribute reveals that hypertension is significant in the

group with higher level of blood glucose (χ^2 =7.716, d.f.=1, p<0.05), total cholesterol (χ^2 =5.99, d.f.=1, p<0.01), Tg (χ^2 =6.791, d.f.=1, p<0.01) LDL-C (χ^2 =7.106, d.f.=1, p<0.05), serum sodium ions (χ^2 =10.119, d.f.=1, p<0.01) and urine potassium ions (χ^2 =10.60, d.f.=1, p<0.01) levels whereas inverse relationship of hypertension occurs in blood HDL-C (χ^2 =5.99, d.f.=1, p<0.01), urine Na^+ (χ^2 =17.389, d.f.=1, p<0.05) and serum K^+ (χ^2 =23.35, d.f.=1, p<0.001 groups. Hypertension is independent with the change of serum VLDL-C (χ^2 =0.305, d.f.=1, p>0.05), serum Cl^- (χ^2 =5.99, d.f.=1, p<0.05) and urine Cl^- (χ^2 =0.174, d.f.=1, p>0.05) levels.

Fig 1: Prevalence of hypertension according to various biochemical parameters of blood and urine within the Scheduled Caste community of the District Nadia, West Bengal, India

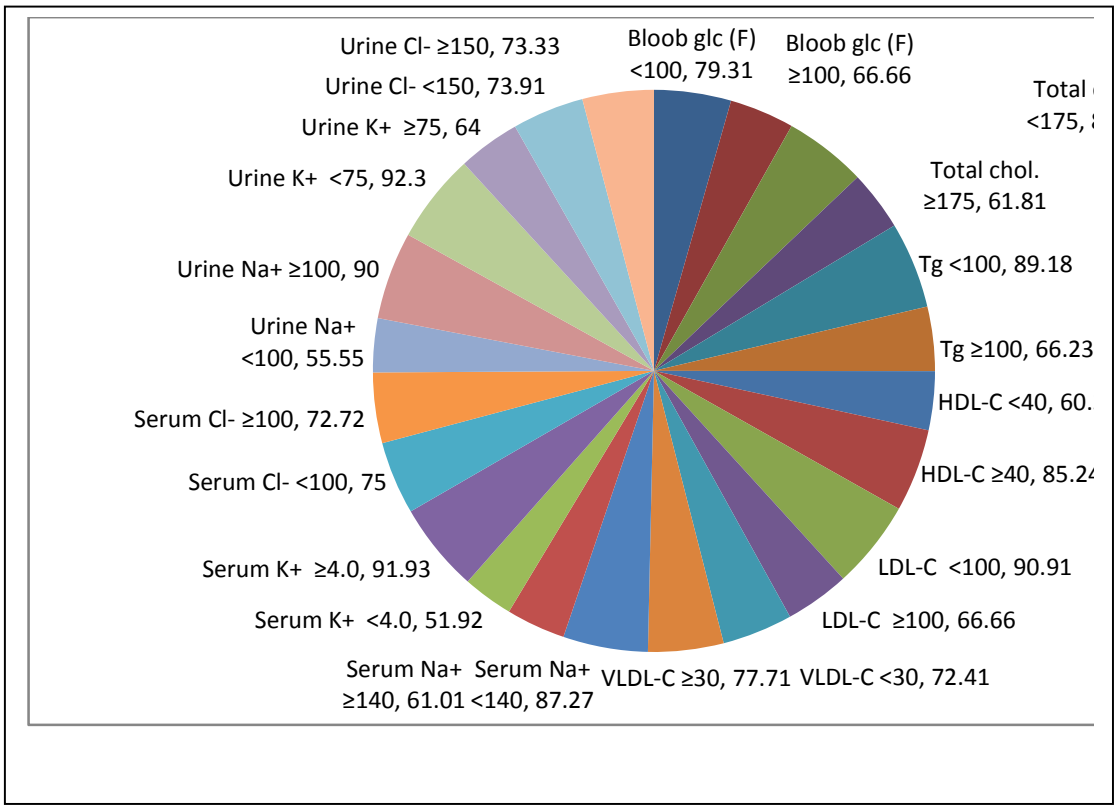

Table 1: Biochemical parameters of blood and urine and prevalence of hypertension within the Scheduled Caste community of the District Nadia, West Bengal, India

Biochemical parameters		Normal range mg/dl or mmol/L		Number	Normotensive (%)	Hypertensive (%)
Total sample studied				**114**	**73.69**	**26.31**
Fasting blood sugar (mg/dl)		70-100	<100	87	79.31	20.68
			≥100	27	66.66	44.44
Lipid	Total cholesterol	<200	<175	59	84.74	15.25

profile	(mg/dl)		≥175	55	61.81	38.18
	Tg (mg/dl)	<150	<100	37	89.18	10.81
			≥100	77	66.23	33.76
	HDL-C (mg/dl)	>35	<40	53	60.37	39.62
			≥40	61	85.24	14.75
	LDL-C (mg/dl)	<100	<100	33	90.91	9.09
			≥100	81	66.66	33.33
	VLDL-C (mg/dl)	<30	<30	87	72.41	27.58
			≥30	27	77.71	22.22
Serum electrolytes	Na^+ (mmol/L)	135-145	<140	55	87.27	12.72
			≥140	59	61.01	38.98
	K^+ (mmol/L)	3.5-5.0	<4.0	52	51.92	48.07
			≥4.0	62	91.93	8.06
	Cl^- (mmol/L)	95-106	<100	48	75	25
			≥100	66	72.72	27.27
Urine Electrolytes	Na^+ (mmol/L)	40-220	<100	54	55.55	44.44
			≥100	60	90	10
	K^+ (mmol/L)	25-125	<75	39	92.30	7.69
			≥75	75	64	36
	Cl^- (mmol/L)	110-250	<150	69	73.91	26.08
			≥150	45	73.33	26.66

Tg : Triglyceride, HDL-C : High density lipoprotein-cholesterol, LDL-C : Low density lipoprotein – cholesterol, VLDL-C : Very low density lipoprotein-cholesterol, Na^+ : Sodium ion, K^+ : Potassium ion, Cl^- : Chloride ion

Table 2: Table indicating blood glucose level wise distribution of hypertension within the Scheduled Caste community of the District Nadia, West Bengal, India

Blood glucose(F)	<100 mg/dl	≥100 mg/dl	Total
Hypertensive	18	12	30
Non-hypertensive	69	15	84
Total	87	27	N=114

χ^2= 5.99, d.f.=1, p<0.05

Table 3: Showing the increased tendency of hypertension with cholesterol within the Scheduled Caste community of the District Nadia, West Bengal, India

Total cholesterol	<175 mg/dl	≥175 mg/dl	Total
Hypertensive	9	21	30

Non-hypertensive	50	34	84
Total	59	55	N=114
χ^2= 7.716, d.f.=1, p<0.01			

Table 4: Triglyceride level and prevalence of hypertension within the Scheduled Caste community of the District Nadia, West Bengal, India

Triglyceride	<100 mg/dl	≥100 mg/dl	Total
Hypertensive	4	26	30
Non-hypertensive	33	51	84
Total	37	77	N=114
χ^2= 6.791, d.f.=1, p<0.01			

Table 5: LDL-C and hypertension distribution within the Scheduled Caste community of the District Nadia, West Bengal, India

LDL-C	<100 mg/dl	≥100 mg/dl	Total
Hypertensive	3	27	30
Non-hypertensive	30	54	84
Total	33	81	N=114
χ^2= 7.106, d.f.=1, p<0.05			

Table 6: HDL-C and prevalence of hypertension within the Scheduled Caste community of the District Nadia, West Bengal, India

HDL-C	<40 mg/dl	≥40 mg/dl	Total
Hypertensive	21	9	30
Non-hypertensive	32	52	84
Total	53	61	N=114
χ^2= 9.045, d.f.=1, p<0.01			

Table 7: VLDL-C and rate of hypertension within the Scheduled Caste community of the District Nadia, West Bengal, India

VLDL-C	<30 mg/dl	≥30 mg/dl	Total
Hypertensive	24	6	30
Non-hypertensive	63	21	84
Total	87	27	N=114
χ^2= 0.305, d.f.=1, Not significant (p>0.05)			

Table 8: Serum sodium and hypertension within the Scheduled Caste community of the District Nadia, West Bengal, India

Serum Na^+	<140 mmol/L	≥140 mmol/L	Total
Hypertensive	7	23	30
Non-hypertensive	48	36	84
Total	55	59	N=114
χ^2= 10.119, d.f.=1, p<0.01			

Table 9: Serum potassium and hypertension within the Scheduled Caste community of the District Nadia, West Bengal, India

Serum K^+	<4 mmol/L	≥4 mmol/L	Total
Hypertensive	25	5	30
Non-hypertensive	27	57	84
Total	52	62	N=114
χ^2= 23.350, d.f.=1, p<0.001			

Table-10: Serum chloride and hypertension within the Scheduled Caste community of the District Nadia, West Bengal, India

Serum Cl^-	<100 mmol/L	≥100 mmol/L	Total
Hypertensive	12	18	30
Non-hypertensive	30	54	84
Total	42	72	N=114
χ^2= 0.174, d.f.=1, Not significant (p>0.05)			

Table 11: Urine sodium and hypertension within the Scheduled Caste community of the District Nadia, West Bengal, India

Urine Na^+	<100 mmol/L	≥100 mmol/L	Total
Hypertensive	24	6	30
Non-hypertensive	30	54	84
Total	54	60	N=114
χ^2= 17.389, d.f.=1, p<0.001			

Table 12: Urine potassium and hypertension within the Scheduled Caste community of the District Nadia, West Bengal, India

Urine K^+	<75 mmol/L	≥75 mmol/L	Total
Hypertensive	3	27	30
Non-hypertensive	36	48	84
Total	39	75	N=114
χ^2= 10.60, d.f.=1, p<0.01			

Table 13: Urine chloride and hypertension within the Scheduled Caste community of the District Nadia, West Bengal, India

Urine Cl^-	<150 mmol/L	≥150 mmol/L	Total
Hypertensive	18	12	30
Non-hypertensive	51	33	84
Total	69	45	N=114
χ^2= 0.004, d.f.=1, Not significant (p>0.05)			

Discussion

Various biochemical factors which are associated for the development of hypertension have been studied in this investigation. The prevalence of hypertension in relation to serum fasting glucose level was increased in this study. The rate of hypertension in the studied people with <100mg/dl and >100mg/dl fasting blood glucose levels were 20.68% and 44.44% respectively. χ^2-test showed the significant association between fasting blood sugar and hypertension. The prevalence of hypertension in patients with type 2 diabetes is up to three times greater than in age and gender matched non diabetic patients [16, 17].The patients with diabetes mellitus have a three to six fold increase in stroke risk, mostly attributable to hypertension, which occurs in 40% to 70% of this population. Tight blood pressure control significantly reduces stroke risk for these individuals [18].Incidence of hypertension was associated with dyslipidemia. The rate of hypertension was gradually increased with increase of serum total cholesterol, triglycerides and LDL-cholesterol levels. On the other hand, prevalence of hypertension was inversely associated with serum HDL-cholesterol level. Several studies were examined prospectively whether serum lipid levels (total-cholesterol, triglycerides, HDL-C and LDL-C) are associated with the subsequent development of hypertension in middle age adults. Higher levels of triglycerides [19-21], higher total-cholesterol 22, 23], and higher level of LDL-C [20, 24] were found to be associated with an increased risk of hypertension. In most studies, serum HDL-C levels showed an independent and inverse association with incident hypertension [19, 20, 22-24]. Both the systolic and diastolic blood pressure is positively correlated with high salt intake. Many other scientists were also worked on the development of salt-related blood pressure [25-28].High salt intake significantly increases renal excretion of water due to increased water intake. This compatible finding was also reported by some other researchers [12, 13, 29, 30]. The development of hypertension in some individuals of high salt intake group is mainly due to poor renal functional capacity to handle/excrete an excess renal volume (Uv) and renal salt or sodium (Us). This is also diagnosed by other workers [31, 32].

Conclusion

From this study it can be concluded that the incidence of hypertension was gradually increased with increase of hyperglycemia, hyperlipidemia and higher salt intake within the Scheduled Caste Community of the District Nadia, West Bengal, India.

Acknowledgements

The authors are grateful to the people of the Scheduled Caste Community of the District Nadia, West Bengal, India, those who provided whole-hearted support for the collection of data regarding hypertension during field study.

References

1. WHO, heart beat (1992). The rhythm of health report on world health day. 7th April 1991. Geneva.
2. Lawes CM, Vander HS, Rodgers A. International society of hypertension. Global burden of blood pressure-related disease, 2001. Lancet. 2008; 371: 1513-8.
3. Di Angelantonia E, Sarwar N, Perry P, Kaptoge S, Ray KK et al. Major lipids, apolipoproteins, and risk of vascular disease. Emerging Risk Factor Collaboration (ERFC). JAMA. 2009; 302: 1993-2000.
4. Selby JV, Newman B, Quiroga J, Christian JC, Austin MA, Fabsitz RR. Concordance for dyslipidemic hypertension in male twins. JAMA. 1991; 265: 2079-84.
5. Haffner SM, Lehto S, RÖnnemaa T, PyÖrälä K, Lakso M. Mortality from coronary heart disease in subjects with type 2 diabetes and in nondiabetic subjects with and without prior myocardial infarction. N Eng J Med. 1998; 339: 229-34
6. Reaven GM Lithell H, Landsberg L. Hypertension and associated metabolic abnormalities- the role of insulin resistance and the sympathoadrenal system. N Eng J Med. 1996; 334: 374-81.
7. Yadav S, Boddula R, Genitta G, Bhatia V, Bansal B, Kongara S, Julka S, Kumar A, Singh HK, Ramesh V, Bhatia E. Prevalence and risk factors of prehypertension and hypertension in an affluent north Indian population. Indian J Med Res. 2007; 128: 712-720.
8. Chobanian AV, Hill M. National heart, lung, and blood institute workshop on sodium and blood pressure. A critical review of current scientific evidence. Hypertension. 2000; 35: 858-863.
9. Xie JX, Sasaki S, Joossens JV, Kesteloot H. The relationship between urinary cations obtained from the INTERSALT study and

cardiovascular mortality. Hum hypertens. 1992; 6: 17-21.
10. Sasaki S, Zhanf X-H, Kesteloot H. Dietary sodium, potassium, saturated fat, alcohol, and stroke mortality. Stroke. 1995; 26: 783-789.
11. Denton DA, Nelson JF, Tarjan E. Water and salt intake in wild rabbits (Oryctolagus cuniculus (L)) following dipsogenic stimuli. J Am Physiol. 1985; 362: 285-301.
12. Kanter GS. Excretion and drinking after salt loading in dogs. Am J Physiol. 1953; 174: 87-94.
13. Di Salvo, NA. Factors which alter drinking responses of dogs to interavenous injections of hypertonic sodium chloride solutions. Am J Physiol. 1955; 180: 139-145.
14. Hladky SB, Rink TJ. Body fluid and kidney physiology, Baltimore. Md: Edward Arnold Publishers Ltd. 1986; 143-177.
15. Chobanian AV, Bakris GL, Black HR, Cushmann WC, Green LA, Izzo JL et al. Seventh report of Joint National Committee on prevention, detection, evaluation and treatment of high blood pressure. Hypertension. 2003; 42: 1206-1252.
16. Bakris GL, Williams M, Dworkin L *et al*. Preserving renal function in adults with hypertension and diabetes: a consensus approach. National kidney foundation hypertension and diabetes executive committee working group. Am J kid disease. 2000; 36: 646-661
17. Sower JR, Williams M, Epstein M, Bakris GL. Hypertension in patients with diabetes. Strategies for drug therapy to reduce complications. Postgrad Med. 2000; 107: 47-54, 60.
18. UKPDS Group 38. Tight blood pressure control and risk of macrovascular and microvascular complications in type 2 diabetes: United Kingdom Prospective Diabetes Study Group 38. BMJ. 1998; 317: 703-713.
19. Haffner SM, Ferrannini E, Hazuda HP, Stern MP. Clustering of cardiovascular risk factors in confirmed prehypertensive individuals. Hypertension. 1992; 20: 38-45.
20. Laaksonen DE, Niskanen L, NyyssÖnen K, Lakka TA, Laukanen JA, Salonen JT. Dyslipidemia as a predictor of hypertension in middle aged men. Eur Heart J. 2008; 29: 2561-8.
21. Dyer AR, Liu K, Walsh M, Kiefe C, Jackobs Jr. DR, Bild DE. Ten year incidence of elevated blood pressure and its predictors: the CARDIA study. Coronary artery risk Development in young adults. J Hum Hypertens. 1999; 13: 13-21.
22. Halperin RO, Sesso HD, Ma J, Buring JE, Stampfer MJ, Gaziano JM. Dyslipidemia and the risk of incident hypertension in men. Hypertension. 2006; 47: 45-50.
23. Sesso HD, Buring JE, Chown MJ, Ridker PM, Gaziano JM. A prospective study of plasma lipid levels and hypertension in women. Arch Intern Med. 2005; 165: 2420-7.
24. de Simone G, Devereux RB, Chinali M, Roman MJ, Best LG, Welty TK et al. Risk factors for arterial hypertension in adults with initial optimal blood pressure: the Strong Heart Study. Hypertension. 2006; 47: 162-7
25. Biswas M, Manna CK. Prevalence of hypertension and sociodemographic factors within the Scheduled Caste community of the district Nadia, West Bengal, India. High Blood Press Cardiovasc Prev. 2011; 18(4): 179-185.
26. MacGregor GA, Sever PS. Salt: overwhelming evidence but still no action: can a consumes be reached with the food industry? BMJ; 1996; 312: 1287-1289
27. Law MR, Frost CD, Wald NJ. By how much does dietary salt reduction lower blood pressure? BMJ. 1991; 302: 811-824.
28. Cutler JA, Follmann D, Allender PS. Randomized trials of sodium reduction: an overview. Am J Clin Nutr. 1997; 65: 643S-651S.
29. Cowley AW, Skeleton MM, Merrill DC, Quillen EW, Switzer SJ. Influence of daily sodium intake on vasopressin secretion and drinking in dogs. Am J Physiol. 1983; 245: R860-R872
30. Fitzsimons JT. The effects of slow infusions of hypertonic solutions on drinking and drinking thresholds in rats. J Physiol. 1963; 167: 344-354.
31. Freis ED. The role of salt in hypertension. Blood Pressure. 1992; 1: 196-200
32. He FJ, Markandu ND, Sagnella GA, MacGregor GA. Effect of salt intake on renal excretion of water in humans. Hypertension. 2001; 38: 317-320.

Source of Support: Nil
Conflict of Interest: None