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Editorial

Salt, Hypertension and Cardiovascular Disease: the Connection

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Hypertension (HT) the most important risk factor of cardiovascular disease (CVD), is a heterogeneous disease; the underlying cause is unknown. Genetic back-ground and environmental influences are both involved. The most important environmental factors are physical inactivity and dietary factors, particularly salt and potassium intake.[1]

There is a relationship between Blood Pressure (BP) and natriuresis which maintains sodium balance and extra cellular fluid volume. An impaired ability of the kidney to excrete sodium, requires an increase in BP to increase natriuresis and correct sodium balance resulting in HT. Much evidence suggests, that in those who develops high BP, there is an underlying defect in the ability of the kidney to excrete salt and that the greater compensatory response required to restore sodium balance is the cause for the increase in BP.[2,3] Epidemiological, migration, intervention, treatment, genetic and animal studies have shown that dietary salt (sodium chloride) plays an important role in BP regulation.[4] The INTERSALT study, the EPIC-Norfolk study and many other studies have shown that BP was higher among subjects with a high sodium intake.[1,5]

The meta-analysis of randomized trials by J E He and G A MacGregor showed a dose response between the change of urine sodium and BP. A reduction of 6g of salt intake resulted in a fall in BP of 7.11/3.88mmHg ($p< 0.001$ for systolic and diastolic) in HT's and 3.57/1.66 mmHg in normotensive's (systolic :$p<0.001$;diastolic: $p<0.05$). This will predict the reduction in stroke deaths by 14% and coronary deaths by 9% in HT's and reduction in stroke and coronary deaths by 6 and 4% in normotensive's respectively.[6]

There is an increase evidence that high salt intake has direct harmful effects on the CV system e.g : an increase Left Ventricular Mass (LVM), stiffness of the conduit arteries thickens and narrows resistance arteries independent to the effect of salt and BP. An increase in sodium concentration directly exerts growth stimulating intracellular signals.

Many studies have shown a positive correlation between urinary sodium excretion and LVM in normotensive's well as in HT's. A reduction in salt intake is associated with a reduction of LVM[2,7-9] at VASCULAR level. High salt intake induce significant structural alterations of arteries, cerebral - as well as renal arteries, independent of BP levels. Salt loading independently has been shown to decrease NO reduction. Salt reduction improves endothelial dependent vasodilatation in normotensive's independent of BP.[10-12]

KIDNEY and salt. High sodium intake was shown to increase glomerular filtration and an increase in intra glomerular pressure. The impact of salt intake on renal function is its effect on albumin excretion. Lowering salt intake in proteinuric

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patients is associated with reduction in proteinurie. Indeed several epidemiologic studies have shown a direct association between salt intake and urinary albumin excretion, independent of BP.[13]

Salt intake and STROKE. The results of a meta analysis from 13 studies with 177.025 participants showed that higher salt intake was associated with a greater risk of stroke and CVD, a 23 % greater risk of stroke for an average difference in sodium intake of 5g/d (one teaspoon).[14] Cook et. al reported the long term effects of salt reduction in CVD, a 25 to 35 % lower incidence of CV events was noted in the reduced salt group.[15] It has been estimated that a 3g/d reduction of dietary salt intake would reduce stroke by 13 % and Ischemic heart disease by 10 %(NHANES I).The average salt intake in most countries around the world is about 9 to 12g/d. The Asian countries more than 12g/d.[3] The World Health Organization recommendations for adults are to reduce salt intake to < 5g/d, the UK and the USA recommendations are < 6g/d. These recommendations will have a major impact on BP and CVD but are not ideal. A reduction to 3g/d will have a much greater effect and should become the long-term target for population salt intake worldwide.[16] Evidence shows that current salt intake contribute to the high prevalence of HT and its consequences.

Public Health efforts to reduce salt intake are justified especially in commercial foods.[17]

References

1. Burnier M, Bochud M, Schmieder R. Update on Hypertension Management: Dietary Sodium Intake and Hypertension. European Society of Hypertension Newsletter. 2009;10: 40.
2. He FJ, Markandu ND, Sagnella GA, de Wardener HE, MacGregor GM. Plasma Sodium. Ignored and underestimated. Hypertension. 2005; 45: 98-102.
3. He FJ, MacGregor GA. Reducing population salt intake worldwide: From evidence to implementation. Prog Cardovasc Dis. 2010; 52: 363-82.
4. He FJ, Marciniak M, Visagie E, et al. Effect of modest salt reduction on blood pressure, urinary albumin, and pulse wave velocity in white, black, and Asian mild hypertensives. Hypertension. 2009; 54: 482-8.
5. Karpanen H, Mervaala E. Sodium Intake and Hypertension. Prog Cardiovasc Dis. 2006; 49: 59-75.
6. He FJ, MacGregor GA. Effect of modest salt reduction on blood pressure : a meta-analysis of randomized trials. Implication for public health. J Human Hypertension. 2002;16: 761-70.
7. Blacher J, Safar ME. Large–artery stiffness, hypertension and cardiovascular risk in older patients. Nat Clin Pract Cardiovasc Med. 2005; 2: 451-5.
8. Frohlich ED. Ventricular remodeling / fibrosis, renal arterial brachytherapy, collagen crosslink breakers, salt, and therapeutic adherence. Curr Op Cardiol. 2004; 19: 323-5.
9. Frohlich ED. The Salt Conundrum: A hypothesis. Hypertension. 2007; 50: 161-6.
10. Li J, White J, Guo L, et al. Salt inactivates Endothelial Nitric Oxide Synthase in endothelial cells. J Nutr. 2009;139: 447-51.
11. Oberleithner H, Riethmuller C, Schiller H, et al. Plasma sodium stiffens vascular endothelium and reduces nitric oxide release. PNAS. 2007; 104: 16281-6.
12. Dickinson KM, Keogh JB, Clifton PM. Effects of low-salt diet on flow-mediated dilation in humans. Am J Clin Nutr. 2009; 89: 485-90.
13. Kimura G, Dohi Y, Fukuda M. Salt sensitivity and circadian rhythm of blood pressure:the keys to connect CKD with Cardiovascular events. Hypertension Research. 2010; 33: 515-20.
14. Strazzulo P, D'Elia L, Kandala NB, Cappucio FP. Salt intake, stroke and cardiovascular disease : meta analysis of prospective studies. BMJ. 2009; 339: 1-9.
15. Cook NR et al. Long term effects of dietary sodium reduction on cardiovascular disease outcomes : observational follow-up of the trials of hypertension prevention (TOHP). BMJ. 2007; 334: 885.
16. He FJ, MacGregor GA. How far should salt intake be reduced? Hypertension. 2003; 42: 1093-9.
17. Ritz E. Salt and Hypertension. Nephrology. 2010; 15: 49-52.