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Sympathetically driven salt-induced hypertension in obesity

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Abstract

High salt induced renal sympathetic nerve activation is the cause of concerned in development of cardiovascular diseases in obese individuals.The exact mechanism between this relationship is not known.However substantial evidences have described the role of high salt on central nervous system through the involvement of brain angiotensin and reactive brain oxygen species in renal sympathoexcitation. Further research is needed to understand the role of high salt induced renal symathoexcitation and the development of hypertension in obesity.This could be helpful in the treatment of salt induced hypertension in obesity.

Keywords: Obesity, Hypertension, Renal sympathetic nerve, High salt.

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Introduction:

Obesity and its deleterious effects on the cardiovascular system have become major health care concerns. Hypertension is the primary cause of cardiovascular morbidity in obese individuals, although the exact mechanisms underlying the relationship between obesity and hypertension are not completely understood. Dietary mediators such as a high-salt diet, which is now common in our civilized society, is the major risk factor for hypertension and various cardiovascular disorders in obese individuals[1]. High salt intake is considered one of the possible factors that mediates the link between hypertension and obesity[2]. Regarding the pathophysiological aspect, reports have suggested the role of increased sympathetic activity in the relationship between salt-induced hypertension and obesity[3, 4]. High sodium intake is known to be a critical factor contributing to increased sympathetic activity, particularly renal sympathetic nerve activity[5,6,7]. In animals, chronic salt intake close to the level present in the human diet since weaning resulted in the development of hypertension and sodium-driven neurogenic mechanisms were considered the possible cause[5]. The pivotal role of sympathetic nerves in obesity-induced hypertension is supported by increased renal tubular sodium reabsorption through the impairment of pressure natriuresis[8]. Furthermore, blood pressure was restored to the normotensive level in dogs with established obesity-induced hypertension through bilateral and partial renal denervation by using catheter-based radiofrequency ablation[8,9,10,11]. Similarly, catheter-based radiofrequency renal denervation reduced blood pressure in obese humans with resistant hypertension[12]. The precise mechanisms underlying the role of high salt intake in sympathetic nerve activation in obese individuals are not completely understood. Several studies have made valuable contributions to understanding the relationship between chronic salt intake and sympathetic activation. Some of them suggest that high salt increases the excitability of presympathetic paraventricular nucleus (PVN) neurons, leading to sympathoexcitation.7 paraventricular nucleus is the area in the brain that contributes to sympathetic activation and salt-sensitive hypertension development. Hence high salt-induced modifications in paraventricular nucleus neuronal excitability can have a profound effect on sympathoexcitation. Recent reports have suggested that high salt intake reduces SK (Ca2+-activated potassium channel) currents, thereby contributing to increased paraventricular nucleus neuronal excitability and sympathetic activation that can act as a precurosor for the development of salt-induced hypertension. Correspondingly salt- induced hypertension in obesity is associated with the generation of brain reactive oxygen species, leading to a sympathoexcitation effect[4].

Additionally, one of the possible regulator of sympathetic nerve activation in salt induced hypertension is the elevation of angiotensin II. Angiotensin II throught its action on the central nervous system pathway could chronically increase renal sympathetic nerve activity thus potentially contributing to the maintenance of hypertension[13]. This fact is supported by the observation that the pharmacological blockade of an angiotensin receptor, angiotensin-converting enzyme inhibitors, reduces blood pressure and attenuates sodium retention to a greater extent in obese experimental animals. The prevalence of obesity-induced hypertension continues to increase due to the intake of a high-salt diet. Although sympathetic nerve activity, particularly renal sympathetic nerve activity, is considered a crucial factor

contributing to the development of hypertension in obese individuals, only few studies have investigated the relationship between salt-induced renal sympathetic nerve activity and hypertension in obese individuals. Additional studies are warranted to understand the role of salt and its effect on the activation of renal sympathetic nerves in obesity-induced hypertension. The results may be helpful in the treatment of salt-induced hypertension in obesity.

References

1. Psaltopoulou, T., Hatzis, G., Papageorgiou, N., Androulakis, E., Briasoulis, A., & Tousoulis, D. (2017). Socioeconomic status and risk factors for cardiovascular disease: impact of dietary mediators. Hellenic Journal of Cardiology, 58(1), 32-42.

2. Landsberg, L., Aronne, L. J., Beilin, L. J., Burke, V., Igel, L. I., Lloyd-Jones, D., & Sowers, J. (2013). Obesity-related hypertension: Pathogenesis, cardiovascular risk, and treatment—A position paper of the The Obesity Society and the American Society of Hypertension. Obesity, 21(1), 8-24.

3. Afsar, B., Sag, A. A., & Kanbay, M. (2017). Should we take renal denervation with a grain of salt?. The Journal of Clinical Hypertension, 19(11), 1134-1136.

4. Ando, K. (2013). Increased salt sensitivity in obese hypertension: role of the sympathetic nervous system. Current hypertension reviews, 9(4), 264-269.

5. Gomes, P. M., Sá, R. W. M., Aguiar, G. L., Paes, M. H. S., Alzamora, A. C., Lima, W. G., ... & Cardoso, L. M. (2017). Chronic high-sodium diet intake after weaning lead to neurogenic hypertension in adult Wistar rats. Scientific reports, 7(1), 5655.

6. Chapp, A. D., Wang, R., Cheng, Z. J., Shan, Z., & Chen, Q. H. (2017). Long-Term High Salt Intake Involves Reduced SK Currents and Increased Excitability of PVN Neurons with Projections to the Rostral Ventrolateral Medulla in Rats. Neural Plasticity, 2017.

7. Larson, R. A., Chapp, A. D., Gui, L., Huber, M. J., Cheng, Z. J., Shan, Z., & Chen, Q. H. (2017). High salt intake augments excitability of PVN neurons in rats: role of the endoplasmic reticulum Ca2+ store. Frontiers in neuroscience, 11, 182.

8. Kawarazaki, W., & Fujita, T. (2016). The role of aldosterone in obesity-related hypertension. American journal of hypertension, 29(4), 415-423.

9. Henegar, J. R., Zhang, Y., Rama, R. D., Hata, C., Hall, M. E., & Hall, J. E. (2014). Catheter-based radiorefrequency renal denervation lowers blood pressure in obese hypertensive dogs. American journal of hypertension, 27(10), 1285-1292.

10. Frame, A. A., Carmichael, C. Y., & Wainford, R. D. (2016). Renal afferents. Current hypertension reports, 18(9), 69.

11. Henegar, J. R., Zhang, Y., Hata, C., Narciso, I., Hall, M. E., & Hall, J. E. (2015). Catheter-based radiofrequency renal denervation: location effects on renal norepinephrine. American journal of hypertension, 28(7), 909-914.

12. Hering, D., Marusic, P., Walton, A. S., Lambert, E. A., Krum, H., Narkiewicz, K., ... & Schlaich, M. P. (2014). Sustained Sympathetic and Blood Pressure Reduction 1 Year After Renal Denervation in Patients With Resistant HypertensionNovelty and Significance. Hypertension, 64(1), 118-124.

13. Guild, S. J., McBryde, F. D., Malpas, S. C., & Barrett, C. J. (2012). High dietary salt and angiotensin II chronically increase renal sympathetic nerve activity: a direct telemetric study. Hypertension, 59(3), 614-620.