ECRONICON OPEN ACCESS

EC CARDIOLOGY
Editorial

The Effect of Urine Sodium/Potassium Ratio on Our Health

Hacı Kemal Erdemli*

Biochemistry Specialist, Düzen Laboratories, Ankara, Turkey

***Corresponding Author:** Hacı Kemal Erdemli, Biochemistry Specialist, Düzen Laboratories, Ankara, Turkey.

Received: January 14, 2023; **Published:** January 16, 2023

Keywords: *Diet; Hypertension; Obesity; Potassium; Sodium; Urine*

Can daily salt intake lead to hypertension?

What are the benefits of measuring the urine sodium/potassium ratio?

Excessive sodium (Na) intake combined with insufficient potassium (K) intake may increase the development of cardiac metabolic disorders. In recent publications, the urinary Na/K ratio is recommended as a reliable index to evaluate the risk of cardiovascular diseases (CVD) and CVD-related death.

What should be the ideal Na/K ratio in the diet remains a matter of debate; A value of ~ 0.5 (2000 mg/day Na and 3500 mg/day K intake) is recommended by the World Health Organization (WHO) and US-Canadian medical authorities. However, a Na/K ratio of < 1.0 has been defined as the best value to balance ideal Na and K intake in reducing CVD and the risk of death [1].

In a study conducted in 2021 [1], the urinary Na/K ratio was low in people who followed a traditional diet, Mediterranean diet and hypertension diet; it was found significantly higher in those have a western-style diet [1].

There are multiple approaches explaining the relationship between salt consumption and the development of arterial hypertension. According to the traditional model, it is associated with increased extracellular fluid volume in the kidney and other tissues and increased blood flow. Increased renal blood flow and glomerular filtration rate increase renal sodium reabsorption. Correspondingly, blood pressure increases, leading to glomerular damage, impaired renal sodium excretion capacity, and finally, a gradual loss of nephron function.

Normally, excessive sodium intake would be expected to reduce aldosterone secretion by inhibiting the renin-angiotensin system. However, it has been suggested that some adipokines may directly stimulate aldosterone release from the adrenals, independent of angiotensin II. It has also been proven that angiotensinogen, angiotensin I and angiotensin II can be produced directly in adipocytes, leading to local aldosterone production.

Another possible factor is the reduced adiponectin levels found in obese individuals. Adiponectin has an important role in the inhibition of renin secretion. Decreased adiponectin leads to increased activity in the renin-angiotensin-aldosterone axis, independent of salt intake. In addition, it is thought that decreased adiponectin levels may contribute to hypertension by causing endothelial dysfunction.

Another factor is vitamin D deficiency. Vitamin D has an inhibitory effect on renin secretion. Vitamin D deficiency directly leads to an increase in renin levels, independent of salt intake. Many observational studies have confirmed the association between vitamin D deficiency and an increased risk of hypertension. However, vitamin D supplementation had no arterial pressure-reducing effect. A more recent observation is that high amounts of salt and glucose in highly processed or fast food affect negatively the renin-angiotensin-aldosterone system regardless of body weight.

According to research results in recent years, aldosterone is not only a hormone that regulates electrolytes and fluid volume, but is an important factor in the development of obesity independent of calorie intake and target organ damage. Excess aldosterone stimulates sodium uptake, induces insulin resistance, leptin resistance, adipose tissue inflammation, and impairs thermogenesis in brown adipose tissue, thereby contributing to the development of obesity and related metabolic abnormalities.

In addition, high sodium intake causes acceleration of adipose tissue deposition. Furthermore, it leads to increased production of cortisol and disruption of the circadian rhythm in obese people. Cortisol, produced in adipose tissue, can stimulate renin production and increase insulin resistance, similar to aldosterone. The processes associated with excess body fat and excessive salt intake leading to the development of hypertension become a vicious circle.

Mineralocorticoids mediate perivascular and interstitial fibrosis, glomerulosclerosis and finally proteinuria by causing podocyte damage in the kidney and creating a proinflammatory response [2].

Hyperinsulinemia and insulin resistance have a similar, additive effect leading directly to kidney damage. Insulin resistance, indeed, reduction in phosphoinositide 3-kinase/protein kinase B signaling causes a reduction in nitric oxide synthesis, leading to impaired tubuleglomerular feedback, subsequent hyperfiltration and sodium retention.

A small increase in plasma sodium also stimulates the thirst center, leading to water uptake and arginine vasopressin secretion, leading to water retention.

These mechanisms restore plasma sodium to its previous level, but also increase extracellular fluid volume. As a result, the blood pressure value rises.

An important link between adipose tissue and the development of arterial hypertension is increased activity of the sympathetic nervous system. The hypersecretion of leptin and insulin also plays a role in the stimulation of the sympathetic nervous system through the proopiomelanocortin-melanocortin 4 receptor in the central nervous system. As a result, peripheral arteriosclerosis increases, kidney damage progresses. In addition, insulin resistance and excessive proinflammatory activity of adipose tissue are responsible for both oxidative stress development and endothelial damage [2].

In a 2021 study by researchers [1], the traditional diet model (vegetables, fruits, sweets and savory snacks, sugary drinks, nuts and seeds, poultry, high-fat dairy products, red meat and beans), the hypertension diet, and the Mediterranean diet model, urinary Na/K ratio was found to be low; in the high-fat Western diet model (dairy products, refined grains, fast foods, hydrogenated and animal fats), a higher urinary Na/K ratio was determined. The concentration of K in the urine was also found to be high in the Japanese diet model, where fish and vegetable consumption was intense. Urine Na/K ratio decreases with the increase of "nuts, seeds, fruits and fish" in the diet.

Although 24-hour urine Na and K measurement is accepted as the gold standard method in the evaluation of dietary intake, it is more preferred because it is easier to collect spot urine samples [3].

Spot urine Na/K ratio can be used as a simple, non-invasive and inexpensive method to monitor diet quality, especially in population-based studies. It is thought to be a useful method for monitoring adherence to dietary recommendations.

To achieve the WHO goals of reduced Na uptake and increased K uptake, it may be good practice to maintain the urinary Na/K ratio < 1. In terms of facilitating follow-up, measurement of Na/K ratio in spot urine will provide convenience [4].

It seems that urine Na/K ratio measurement can be effective in measuring the effectiveness of diets to be applied for the prevention of obesity and hypertension.

Bibliography

1. Mirmiran., *et al*. "Urinary sodium to potassium ratio: a simple and useful indicator of diet quality in population based studies". *European Journal of Medical Research* 26.1 (2021): 3.
2. Wójcik M and Kozioł-Kozakowska A. "Obesity, sodium homeostasis, and arterial hypertension in children and adolescents". *Nutrients* 13.11 (2021): 4032.
3. Toshiyuki Iwahori., *et al*. "Estimating 24-h urinary sodium/potassium ratio from casual ('spot') urinary sodium/potassium ratio: the INTERSALT Study". *International Journal of Epidemiology* 46.5 (2017): 1565-1572.
4. Toshiyuki Iwahori., *et al*. "Urinary sodium-to-potassium ratio and intake of sodium and potassium among men and women from multiethnic general populations: the INTERSALT Study". *Hypertension Research* 42.10 (2019): 1590-1598.

Volume 10 Issue 2 February 2023