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Less sodium and more potassium to reduce cardiovascular risk

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An increase in the dietary consumption of salt is associated with a progressive increase in blood pressure (BP) values, and with an increase in the incidence of cardiovascular disease. Reducing the dietary intake of sodium in the population is a public health goal in many countries around the world. Numerous studies have described a linear relationship between high dietary salt intake and the development of arterial hypertension, as well as a negative association between high potassium intake and BP values. Furthermore, there is evidence that a reduction in salt consumption and an increase in potassium consumption can be associated with a decrease in BP values, improving the general state of health. Therefore, it would be desirable to further improve awareness of the risks associated with an excessive intake of salt and low potassium by maintaining public education campaigns and trying to overcome the numerous obstacles to a process of greater responsibility for people regarding nutrition.

A high dietary intake of sodium is considered one of the main causes of increased blood pressure (BP) values and constitutes one of the most important risk factors linked to diet which favours the onset of cardiovascular events (CV) worldwide.[1,2] The PURE study demonstrated that a 1 g increase in dietary sodium was associated with an increase in mean systolic BP of 2.11 mmHg.[3] The increase was greater in older subjects (2.97 mmHg per 1 g of sodium) and in those who already had a diagnosis of hypertension (2.49 mmHg per 1 g of sodium), while the effect was much smaller or absent in older subjects, <55 years, or with normal BP values. There is a unanimous consensus in stating that the dietary intake of salt should be reduced especially in those populations that have a high consumption, especially in hypertensive and older patients.[4]

At the same time, experimental evidence and observational studies have been collected to demonstrate that the reduction in salt consumption is associated with a reduction in BP and a lower incidence of CV events and death from all causes.[4,5]

In most countries in the world, the consumption of salt far exceeds the amount of 5 g/day (equivalent to 2 g of sodium) and in Asia, the average consumption of salt per day exceeds 10 g/day.

The main international scientific societies and the WHO recommend a reduction in the salt content of the diet as part of a non-pharmacological therapy strategy to reduce BP and prevent CV events,[6] keeping the threshold of the amount of salt below 5 g/day, with no substantial differences between hypertensive patients or normotensive subjects. More recently, it has been reported that the curve of the relationship between sodium intake and the incidence of CV events has a J-shape, whereby a high sodium intake, but also a low one (<6 g of salt/day) can be associated with an increase in CV risk.[3,7–9] Some possible explanations for this phenomenon lie in the inaccurate estimation of dietary sodium consumption with the methods usually used (i.e. the determination of sodium intake on a spot urine sample or on a 24 h urine collection), in the wide daily variability of salt, in the possible influence of concomitant clinical conditions involving the reduction of food consumption (and also of sodium) and which are in any case associated with a high CV risk, but also the possible activation of the sympathetic nervous system and the renin–angiotensin system (RAS), or the possible increase in lipids. This observation has raised a wide discussion on the opportunity to push the reduction of the sodium content in

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the diet to levels <2 g/day.[10] The effect of a high sodium content accumulates over time and this suggests that the benefit deriving from a low sodium diet can be underestimated by observational studies with a short follow-up and at the same time that a dietary intervention should start early, in the paediatric age. It should also be remembered that an increase in the dietary sodium content reduces the effectiveness of some antihypertensive drugs, including diuretics and RAS blockers.

In view of the close relationship between sodium and potassium, it is necessary to evaluate the dietary intake not only of sodium but also of potassium.[11] The major food source of potassium is found in vegetables and fruit and products derived from milk and the use of packaged food products, including bread, promotes sodium dietary excess and potassium deficiency. A diet deficient in potassium can lead to excessive sodium reabsorption via up-regulation of tubular Na/Cl co-transport, resulting in plasma volume expansion and increased BP values. In essence, the enhancement of sodium reabsorption resulting from a diet low in potassium may favour a sodium-sensitivity condition. Some experimental and clinical studies have confirmed a negative correlation between dietary potassium content and BP values. It has also been demonstrated that an increase in the content of potassium in the diet has a favourable effect on various risk factors and on CV diseases[11,12] and for this reason, international guidelines recommend an increase in the regular consumption of fresh fruit and vegetables precisely in order to increase the potassium content in the diet, also in view of the low dietary intake of potassium in the general population. The analysis of several cohorts of healthy adult subjects, in which the 24 h urinary excretion of sodium and potassium was determined, showed that an increase in the daily consumption of salt equal to 1 g of sodium corresponds to an increase equal to 18% of CV events and at the same time an increase in potassium consumption of 1 g is associated with a similar reduction in CV events of 18%.[12]

A recent study conducted in China, partially replacing sodium chloride intake with potassium chloride, demonstrated a clear benefit in restoring a balance between dietary sodium and potassium content.[13] The Salt Substitute and Stroke Study (SSaSS) included 20 995 subjects residing in 600 rural Chinese villages, aged over 60 years or with poor BP control or history of stroke, randomized to take regular salt sodium or a salt substitute (75% sodium chloride and 25% potassium chloride). During the ~5-year follow-up, fewer strokes, major CV events, and death from CV causes were recorded in the potassium chloride group, in the absence of complications such as the development of hyperkalaemia. It should be emphasized that in the SSaSS13 study, the values of sodium and potassium assumed with the diet in basal conditions are very different from those observed in the National Health and Nutrition Examination Survey (NHANES) study in the USA; the Chinese population has a high sodium and low potassium intake (4.3 and 1.4 g/day, respectively) compared with the sodium and potassium content recorded in the USA (3.61 and 2.16 g/day, respectively). In the group of subjects taking the salt substitute containing potassium chloride, a modest reduction in sodium of 8% was observed, while the dietary potassium content increased by 57% (from 1.4 to 2.2 g/day) reaching US levels as reported in the NHANES study. The difference in urinary sodium excretion between the group randomized to use a salt containing potassium chloride and the group taking regular salt in the SSaSS study was lower than expected (−0.35 and −0.79 g/day, respectively).[14] This result could depend on the fact that the sodium consumption was reduced during the study also in the control group, and/or on the possibility that in the group assigned to the intake of the salt substitute with potassium there was, in addition to the use of salt enriched with potassium, the use of regular salt. Although the SSaSS study does not clarify the current controversy regarding the appropriateness of a diet particularly low in sodium (<2 g/day), it demonstrates that the increase in the potassium content, even in the presence of excessive sodium consumption, exceeds what has been observed in the USA or in other European countries is useful in preventing the onset of CV events.[15]

An interesting aspect is related to the sex differences that have been observed with regard to the dietary content of sodium and potassium.[12] A recent meta-analysis including six cohorts of predominantly healthy adults demonstrated that the relative risk reduction for CV disease that corresponds to a 1 g increase in urinary potassium excretion was more pronounced in women than in men. Furthermore, the risk of CV disease related to an increase in urinary excretion of 1 g of sodium was greater in women than in men. These results suggest that although high dietary sodium intake exposes women to a higher risk of CVD, high potassium intake may offer women a higher protective effect. In the EPIC—Norfolk study,[16] a high dietary potassium content was related to a lower risk of CV events, more evident in women than men. In the same study, systolic BP values decreased to a greater extent, even during high sodium consumption (on average 5.8 + 1.0 g, corresponding to 14.5 g of salt per day), in relation to a higher potassium intake only in women and not in men.

Possible explanations for this phenomenon are the known greater sodium sensitivity of women (i.e. greater changes in BP in relation to the dietary sodium content), a possible greater benefit that would derive from a low-sodium diet for women compared with men, and finally, the greater adherence to the diet reported in women.[17]

It is evident that in the population, the identification of subjects most exposed to CV risk in relation to the dietary consumption of sodium and potassium is still inadequate, although some indirect information can be derived from the changes in BP in relation to the diet. In the future, a better definition of the complex interaction between dietary intake of sodium and potassium, BP values, and the incidence of CV events will have to represent one of the aspects to be addressed in the field of precision medicine.

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Data availability

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