



Introduction

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Man needs salt to survive. Although it has become fashionable to claim that the physiological needs of salt in humans are as low as 1 g per day nobody can live without salt. A very low salt intake is incompatible with normal physical activity. It puts the kidney and neuroendocrine regulatory systems at their extremes, so the organism can recover as much of the filtered sodium and chloride as possible.

An association between salt intake and high blood pressure has been suspected since more than a century, based on observations in human subjects and experimental animals. Although findings of several epidemiological studies and some interventions trials were in favor of the hypothesis that high salt intake promotes hypertension many other reports did not confirm this hypothesis ^(1,2), as will be outlined by the subsequent speakers. The political aspect of the ongoing hot debate on the pros and cons of a generalized salt restriction has been elegantly addressed by Taubes, some years ago ⁽³⁾.

In any case, high blood pressure is nothing more than a surrogate marker of outcome. What is really important, is outcome itself. Surprisingly, the medical community has started only recently to examine this issue, namely a possible relationship between salt intake and cardiovascular or all-cause mortality. No prospective randomized controlled trials have been done to address this question. Only observational studies are available, and their results led to contradictory conclusions ⁽⁴⁾.

The solution to the contrasting interpretations of available data may reside in the notion of salt sensitivity ⁽⁵⁾. It differs from person to person. It may even differ for a given person with time. In addition to the inherited genetic background it is also influenced by a variety of environmental factors, including life style and dietary habits. Here are two examples to illustrate the importance of external factors. The first is that of obese adolescents who were found to respond to a high salt intake by a marked increase in blood pressure;

after having lost only some pounds of their excessive body weight, they no longer experienced a blood pressure increase in response to high salt intake. The second example is that of the DASH-2 study where the addition of fruits, vegetables and low-fat dairy products to the standard American diet led to a much better control of blood pressure than a low-salt, but otherwise standard, American diet.

In several countries, heavy efforts are made to reduce salt intake at the population level, including the UK, France and Finland. It would seem more appropriate to avoid overweight and to eat healthy food rich in vegetables and fruits. Concerning the efficacy of measures aimed at inducing life style changes to reduce the incidence of arterial hypertension and improve cardiovascular outcome it is worth mentioning a very recent survey from Finland ⁽⁶⁾. The authors found an increase in body mass index and alcohol intake in the Finnish population from 1982 through 2002, although salt intake decreased. This is a good example of misunderstood messages and misdirected efforts. It would have been more beneficial for these people to lose body weight instead of becoming more obese, and to drink less alcohol instead of becoming more alcoholic, all the more since these two life style factors, unlike salt intake, are long recognized, undisputed risk factors for both hypertension and cardiovascular mortality ⁽⁷⁾.

In conclusion, in our opinion the main focus on salt restriction is erroneous. In addition, although reducing sodium intake has many effects, either by its own or in interaction with other dietary components, we know nothing about their unintended consequences on human health. Current efforts should focus on how to reduce sodium intake and to how much it should be reduced, but on how to achieve a healthy, well balanced diet in the population at large and how to reduce the main, widely recognized culprits of cardiovascular morbidity and mortality.





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