

Opinion

Volume 4 Issue 4 – April 2017
DOI: 10.19080/JOCCT.2017.04.555641

J Cardiol & Cardiovasc Ther

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Appropriate Measures of Sodium Intake Do Not Suggest “The Lower (Sodium Intake) the Better”



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Submission: March 17, 2017; **Published:** April 13, 2017

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Opinion

The pooled analysis of Mente et al. [1] concludes: “Compared with moderate sodium intake, high sodium intake is associated with an increased risk of cardiovascular events and death in hypertensive populations, while the association of low sodium intake with increased risk of cardiovascular events and death is observed in those with or without hypertension. These data suggest that lowering sodium intake is best targeted at populations with hypertension who consume high sodium diets”.

The article “Measurement Matters” [2] challenges potentially biased sodium measurements in the pooled analysis of Mente [1] and cite “a study involving CKD patients that used the average of three nonconsecutive 24-hour urine collections (that) revealed a strong linear association between higher urinary sodium excretion and an increased risk of CVD” [3].

In this prospective cohort study of patients with CKD [3] the exposures were “the cumulative mean of urinary sodium excretion from three 24-hour urinary measurements and calibrated to sex-specific mean 24-hour urinary creatinine excretion”. This method clearly provides a better estimate of the usual dietary sodium intake compared to spot urine collections, which can be a less accurate measure of the intake over a lifetime (as in the cited pooled analysis of Mente [1]).

This study of patients with CKD [3] concludes: “higher urinary sodium excretion was associated with increased risk of CVD”, with a significant linear association.

However, a closer look to the data shows that the nadir of every cardiovascular event was not in the first quartile of calibrated 24-hour urinary sodium excretion (<2894, equivalent to <7,24 grams of salt), but in the second quartile (2894-3649, equivalent to a mean of 8,2 grams of salt). Compared with the

first quartile (1-reference), in the fully adjusted model the second quartiles HRs were: 0.87 (0.69-1.10) for composite CVD, 0.89 (0.68-1.17) for congestive HF, 0.66 (0.45-0.97, significant) for myocardial infarction, 0.93 (0.54-1.61) for stroke [3].

Looking at the actual numbers of the events and of their HRs, the authors’ statement that the statistical analysis “provided no evidence of a nonlinear association (P=0.11) and indicated a significant linear association” [3] becomes irrelevant, and even misleading.

Unfortunately, the insistence about a linear relationship between sodium excretion and CVD can suggest to those who only read the abstract’s conclusions that “the lower (sodium intake), the better for health”. This leads not only to a daily sodium intake lower than 2894-3649mg, associated in these CKD patients with a consistent protective trend from cardiovascular events, but also to more extreme sodium restrictions.

In fact the American Heart Association recommends a limit of 1500mg per day, and the Italian Society of Human Nutrition [4] a limit of 1600mg from 60 years onwards (in 2012 it recommended 1100mg). But the Institute of Medicine’s assessment of the available evidence [5] found that “the evidence on health outcomes is not consistent with efforts that encourage lowering of dietary sodium in the general population to 1500mg/day”.

To resolve such a controversial issue, we need large pragmatic RCTs, which can provide clearer answers about the recommended sodium intake for the population and/or subgroups, evaluating the all-cause mortality, not only cardiovascular events or BP levels.

Pending the results of such RCTs, it would be better to avoid drastic recommendations, more so if the present insufficient evidence is transferred to the whole population, and if there is no hypertension.

References

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DOI: [10.19080/JOCCT.2017.04.555641](https://doi.org/10.19080/JOCCT.2017.04.555641)

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