Editorial Commentary

With a Pinch of Salt
Does Reduced Dietary Sodium Consumption Promote Atherosclerosis?

Annemarieke E. Loot

See related article, pp 98–105

The strong positive correlation of dietary sodium intake with systolic blood pressure and the clear prognostic value of hypertension for cardiovascular disease have instigated nutritional guidelines recommending a reduction of average daily sodium intake to 1500 to 2300 mg/d (corresponding to 4–6 g of NaCl per day; current values for most Western countries lie between 9 and 12 g of NaCl per day).1 That such dietary restrictions effectively lower blood pressure has been clearly shown, and although it has proven much more difficult to show long-term effects of reduced dietary sodium on cardiovascular outcomes, studies in this direction seem to speak in favor of sodium restriction.2 Nevertheless, the rationale for restricting dietary sodium is subject to a highly emotional debate.3 Some “salt skeptics” argue that the rationale for restricting dietary sodium is subject to a highly emotional debate.3 Some “salt skeptics” argue that the rationale for restricting dietary sodium is subject to 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Angiotensinogen

Sodium restriction + Renin - High sodium intake

Angiotensin I

ACE

ACE2, ACE

Angiotensin II

ACE2

Angiotensin-(1-7)

AT1

Vasoconstriction Vascular inflammation Aldosterone release Sodium & fluid retention

NEP

Vasodilation Vasoprotection Blood pressure ↓

Stimulation of the Ang II type 1 (AT1) receptor by Ang II has various local and systemic effects aimed at increasing understanding. In the search for an optimal daily sodium dose, Tikellis et al make a valuable contribution to the discussion. It would be premature to translate the results from this mouse study into clinical practice, but they do underline the need for more substantial data on the impact of dietary changes on cardiovascular outcomes in humans. More information on clinical outcomes, however difficult to acquire, would not in the least also increase the acceptance rate of guidelines among the professionals that should be their advocates.

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**Disclosures**
None.

**References**

highly responsive RAAS. In other words, salt sensitivity indicates RAAS insensitivity and vice versa. Not completely coincidentally, those groups that have a high risk of blood pressure–associated morbidity (eg, individuals with hypertension or diabetes mellitus or blacks) generally have a less responsive RAAS. This is reassuring; it suggests that those who would profit the most from sodium restriction in terms of blood pressure reduction also run the least risk of accelerated atherosclerosis through RAAS activation.

Public health guidelines are subject to continuously increasing understanding. In the search for an optimal daily sodium dose, Tikellis et al make a valuable contribution to the discussion. It would be premature to translate the results from this mouse study into clinical practice, but they do underline the need for more substantial data on the impact of dietary changes on cardiovascular outcomes in humans. More information on clinical outcomes, however difficult to acquire, would not in the least also increase the acceptance rate of guidelines among the professionals that should be their advocates.

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**References**
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