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Research Paper Review

Dietary Sodium Intake and Cardiovascular Mortality: Controversy Resolved?

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ABSTRACT

Universal reduction in sodium intake has long been recommended, largely because of its proven ability to lower blood pressure for some. However, multiple randomized trials have also demonstrated that similar reductions in sodium increase plasma renin activity and aldosterone secretion, insulin resistance, sympathetic nerve activity, serum cholesterol, and triglyceride levels. Thus, the health consequences of reducing sodium cannot be predicted by its impact on any single physiologic characteristic but will reflect the net of conflicting effects. Some 23 observational studies (>360,000 subjects and >26,000 end points) linking sodium intake to cardiovascular outcomes have yielded conflicting results. In subjects with average sodium intakes of less than 4.5 g/day, most have found an inverse association of intake with outcome; in subjects with average intakes greater than 4.5 g/day, most reported direct associations. Finally, in two, a "J-shaped" relation was detected. In addition, three randomized trials have found that heart failure subjects allocated to 1.8 g of sodium have significantly increased morbidity and mortality compared with those at 2.8 g. At the same time, a randomized study in retired Taiwanese men found that allocation to an average intake of 3.8 g improved survival compared with 5.3 g. Taken together, these data provide strong support for a "J-shaped" relation of sodium to cardiovascular outcomes. Sodium intakes above and below the range of 2.5-6.0 g/day are associated with increased cardiovascular risk. This robust body of evidence does not support universal reduction of sodium intake.

ANALYSIS

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Background Information

For half a century, reducing dietary sodium has been recommended to blunt an age-associated rise in blood pressure. This recommendation has become ubiquitous enough to prompt primary healthcare practitioners to recommend sodium reduction as a preventive measure for cardiovascular mortality and morbidity. However, mounting evidence is raising an unexpected uncertainty about the health effects of this recommendation. In this study, the authors attempted to examine the current data linking sodium intake to health outcomes. By doing this, they endeavor to determine whether or not telling patients to minimize sodium intake to decrease the mortality and morbidity of hypertension is a valid piece of advice.

PERTINENT RESULTS

Physiological consequences of sodium reduction

- A multitude of studies have found that a reduction in sodium intake can decrease mean systolic blood pressure by a range of 1-10 mmHg. This effect can occur in any individual, but is greatest in older, afro-Canadian/American hypertensive individuals.
- A recent Cochrane review found that a reduction in daily sodium intake of 2.9g/day (from 4.5g to 1.6g) can produce a reduction in blood pressure. However, longer term studies suggest that the initial fall in average blood pressure attenuates over time (1).
- Other studies have found that a reduction in blood pressure via a decrease in sodium intake can result in an increase in sympathetic nerve activity, serum cholesterol and triglyceride levels, insulin resistance, and the release of plasma renin, aldosterone and adrenalin secretion.

Epidemiologic (observational) evidence of association with cardiovascular disease morbidity and mortality

- Six studies found a direct, independent and significant association between cardiovascular disease (CVD) and increasing sodium. However, an inverse association was observed in 7 separate studies.
- Another two observational studies showed increased risk at both ends of the range, creating a "J-shaped" relationship. One of these studies found safety with a consumption of sodium between 2.3-3.4 g/day, with a sharp increase in risk when sodium consumption fell to lower than 2.0 g/day. There was a more gradual increase in CVD risk with consumption of amounts greater than or equal to 4.6 g/day. The second of these studies found that CVD risk increases significantly when daily sodium intake falls below 3g/day or is consumed in quantities greater than 6 g/day.
- An additional 8 studies found no significant association, mixed or inconsistent findings.

Randomized Controlled Trials (RCTs)

- A 2011 Cochrane review of 7 RCTs concluded that there is not enough evidence to determine whether sodium reduction can yield a meaningful health effect.
- Five of these studies were not designed or powered to determine an effect in mortality or morbidity.
- Some of these 5 studies included in the Cochrane review found a non-significant reduction in

mortality, while 3 others saw few events or found no difference in blood pressure.

- One of the aforementioned 3 RCTs reviewed by Cochrane found a marginally significant 35% reduction in CVD mortality (writer's aside: I am unsure how a reduction this large is insignificant both clinically and statistically). However, the experimental group included in this study received increased dietary potassium, thus making it impossible to know whether the outcome was due to a reduced sodium intake, increased potassium or both in combination.
- The second of the 3 RCTs included heart failure patients allocated to a group who consumed 1.8 g of sodium/day or another group who consumed 2.8 g/day. All other treatments were held constant. Within 6 months of treatment, the 1.8 g/day group had significant increased hospitalization and mortality, compared to the 2.8 g/day group.

CLINICAL APPLICATION & CONCLUSIONS

The current recommendations for sodium intake for all individuals is less than 2.0g/day. Specifically, males who are greater than 50 years of age, afro-Canadian/American and hypertensive should consume less than 1.50 grams of sodium/day.

The current evidence does not support this recommendation. The authors conclude that sodium intake bears a "J-shaped" relationship to CVD outcomes. Observational studies suggest that there is *increased risk at either the high or low end of a wide range of sodium intake. Harm has been found with sodium consumption in excess of 4.6-6g/day or less than 2.0g/day.* These observational studies are weakly supported a few RCTs that in combination report a marginally significant negative relationship between an extremely low sodium intake and CVD, particularly in individuals with heart failure.

The authors also finally state that for those individuals whose sodium intake broadly surrounds 3.5g/day, there is no evidence that supports altering sodium intake will improve health outcomes.

EDITOR'S NOTE: *Sometimes health 'advice' like this proliferates over time because no one takes time to look at the literature and science as a whole. Although more research is required to elucidate the exact relationship between sodium intake and blood pressure (and general health, for that matter), perhaps there are bigger issues to address with our clients regarding their general health – improving nutrition (which, in theory, should reduce sodium intake anyway by reducing processed food consumption), incorporating exercise, stress management, improving sleep hygiene etc…*

STUDY METHODS

The authors of this study searched Medline to find studies elucidating physiological consequences of varying sodium intakes. The search found 7 observational studies published since 2008 and two RCTs dated 2011. The study also included seven RCTs that were included in a 2011 Cochrane Review.

STUDY STRENGTHS / WEAKNESSES

Strengths

- The authors analyzed physiological studies, RCTs and observational studies to find an answer to their research question.
- Their search included articles up to 2011.
- The authors suggested an evidence-informed, physiological mechanism as to why this "J-shaped" relationship may exist.

Weaknesses

- The authors only searched the Medline database – they did not consult any additional databases that may provide more articles.
- Specific search terms, key-words and text-words were not included in this article, which make it difficult to replicate.

Additional References

1. Graudal NA, Hubeck-Graudal T, Jürgens G. Effects of low-sodium diet vs. highsodium diet on blood pressure, renin, aldosterone, catecholamines, cholesterol, and triglyceride (Cochrane Review). American Journal of Hypertension. 2012; 25:1–15.