

Comments, Observations and Rebuttals

A Comment on Severe Headache or Migraine History Is Inversely Correlated With Dietary Sodium Intake: NHANES 1999-2004

The following is a comment about a *Headache* published paper concluding that the authors have provided “first evidence of an inverse relationship between migraine and dietary sodium intake.”¹

Few studies are available on the relationship of dietary sodium intake and migraine, though some research evaluating voltage gated sodium channel mutations suggest that there is a sodium-migraine connection. As early as 1951, a study showed that migraineurs excrete 50% more sodium in their urine than non-migraineurs, suggesting a sodium-migraine connection, albeit explanation was not searched for at the time. While some researchers found opposite findings, many studies conclude that certain types of foods are responsible for migraines and provide explanation either using vasoconstriction and vasodilation as the reason for migraine pain or metabolic syndrome as consequence or cause of migraines. Migraine mechanism is visible in scanners and the associated CD and CSD has been documented widely. It is also known that CSD is caused by a massive depolarization of neurons, caused by the influx of sodium into the cells in large quantity.

Although we firmly support the findings of the noted article, we find the claim that they are the first to find the inverse relation of sodium to migraine questionable. There had been earlier published research showing causes leading up to finding the relationship of how sodium channels are involved in inflammation. The report that the “occurrence of SD [CSD] causes a transient loss of membrane potential, synaptic failure, and block of axonal conduction ... in conditions such as

migraine this ... is believed to be involved in the generation of the headache” clearly associates sodium deficiency with migraine because sodium is a necessary main component of voltage activation. Lack of voltage activation, as a result of not enough sodium, is held responsible for the cause of migraine. In fact, many medicines are based on the understanding that some of the sodium channels of cells are associated with the pain of migraine and thus use a blocking mechanism to prevent pain, albeit not to prevent migraine.

Furthermore, in a study conducted by us in 2014 with 650 volunteer migraineurs, we documented their dietary habits and initial dietary sodium intake. We formulated a specific protocol with increased dietary sodium. The experimental result was published in November 2015² explaining the inverse relationship of dietary sodium and migraine. In this paper, we also reported the specific level of increased sodium to which migraineurs responded positively. Our study shows a definite inverse relation and provides proof of the positive changes in migraine outcome in response to increased dietary sodium aiming at migraine prevention and control.^{2,3} The study also highlights that increased sodium does not help in isolation. Factors that remove sodium from electrolyte, such as glucose, are also greatly involved, providing support and explanation to earlier findings by others that migraine was in some way connected to

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metabolic syndrome.⁴ We found that migraineurs are glucose sensitive. Thus, providing sodium to migraineurs, though may provide temporary relief, is not enough to achieve long term success if simple carbohydrates continue to be consumed beyond the migraineur's individual carbohydrate threshold. We show that a ratio of 2:1.5 potassium to sodium—based on USDA database, 4700 mg potassium and 1500–2400 mg sodium are recommended daily whereas migraineurs need 3525 mg sodium a day as ideal for migraine prevention together with the elimination of simple carbohydrates from the diet.⁵ This we found to be consistently valid for all types of migraineurs regardless of the age of the migraineur.

In conclusion, while we fully support the findings on the inverse relationship of sodium and migraine, we propose that the statement of “first evidence” for such is in error.

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