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Opponent's comments

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Dr Graudal's hostile and scare-mongering article is full of inaccuracies, selected arguments and false statements. He considers the 2013 Institute of Medicine (IOM) report, the remit of which was limited [1], dismissing the positions of the previous IOM report, the World Health Organization, the US Centers for Disease Control and Prevention, the American Heart Association, the British National Institute for Health and Clinical Excellence and many other national health organizations statements that informed the 2011 United Nations resolution and the 2013 World Health Assembly deliberation that the population salt reduction strategy is the second most effective strategy for the prevention of cardiovascular disease (CVD) globally. The presence of a food industry conspiracy biasing research and co-opting unscrupulous opinion leaders to divert attention from salt with surreptitious new theories has been extensively documented over the years [2]. In contrast, the alleged conspiracy of global health organizations in producing a sound piece of public health advice is another fabrication to divert attention again.

Sodium chloride (salt) is not a nutrient. At the current levels added to food, salt is a toxic chemical. Dr Graudal confuses the concepts of *usual/habitual* and *adequate/normal.* If we all smoked, smoking would be normal. If we were to define obesity today, we would have to raise the cut-off points for obesity in many countries. A body mass index of 30 kg/m^2 would not indicate obesity because most people in the population weigh that much. If we were to define the adequate levels of physical activity, we should accept that the normality would be not exercising at all. So it is for salt intake! The *usual/habitual* levels are not *adequate/normal* levels.

Dr Graudal continues to pursue two surreptitious arguments: (i) that the effect of salt reduction on blood pressure (BP) is non-existent and (ii) that salt reduction increases hormones that could be dangerous. His first argument is answered in Figure 1. For the second, he only quotes his meta-analyses including short-term acute studies of salt deprivation. I have already addressed the flaw of his argument and shown that

the meta-analyses published are consistent with each other that there is a beneficial effect on BP. There is no need to remind Dr Graudal that treatment with diuretics reduces stroke mortality and other CVD events due to the decrease in BP, despite a chronic stimulation of the renin–angiotensin–aldosterone system, much greater than that seen with a moderate salt reduction.

There are published positions of the National Heart, Lung and Blood Institute, the Trials of Hypertension Prevention and the Dietary Approaches to Stop Hypertension authors that dismiss the allegations Dr Graudal needlessly uses to win his argument. When referring to the World Action on Salt and Health group, he lumps together all those who do not agree with him (although separate and independent researchers around the world), as if they had been running studies together for 20 years! I would consider their results 'consistent' with each other.

Dr Graudal maintains that population moderate salt reduction kills. This is an irresponsible statement based on a few selected studies (mostly observational) that are flawed for the presence of biases and confounding (see my table). In brief, in the European Project on Genes in Hypertension cohort, the lowest sodium intake tertile was flawed by urine undercollections (urinary creatinine in the first tertile 12 versus 16 mmol in the third tertile) and lower socio-economic status [3]. In the analysis of Ongoing Telmisartan Alone and In Combination with Ramipril Global Endpoint Trial and Telmisartan Randomized Assessment Study in ACE Intolerant Subjects with Cardiovascular Disease studies, participants were old and sick patients on multiple medications (29% on diuretics, but 41% of them in the lowest sodium group) [4] and sodium intake was estimated using inaccurate methods [5]. Finally, the Prospective Urban Rural Epidemiology study [6, 7] is flawed on many grounds. The sodium measurement by single fasting morning urine collection to assess individuals' salt intake is unreliable and biased. The sodium study only included ∼100 000 of the ∼160 000 participants, introducing a self-selection bias, and there were fewer participants from India and more from China, the majority with ill health (hypertension, BP medications, congestive heart disease, and CVD). The lower sodium group (<3 g/day equivalent to <7.5 g salt/day) was unable to discriminate on a 'low' salt intake of <5 g, hence the results are irrelevant to the debate on population salt reduction and targets. Finally, compared with the 'higher' sodium group, the 'lower' sodium group was older, predominantly male, Asian and smokers and had higher low-density lipoprotein cholesterol, a history of CVD, diabetes and medication use, therefore biasing the 'lower' sodium group to older men with ill health, hence the reverse causality risk of dying earlier!

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