Masala

ALL ABOUT SALT, PESS AND SHAKERS

Most people agree that there is a J-shaped curve for salt intake and disease. Too little or too much salt are both harmful. In layperson's terms, less than 1 teaspoon per day and greater than 2 teaspoons per day are both likely to be harmful.

There are many ways to try and curb salt consumption.

One simple way is to use salt substitution. In a trial in rural China,[1] 20 995 patients with either a history of stroke or aged >60 years and with hypertension, were randomized into two simple groups; one group received regular salt while another was given a 25% potassium-enriched salt substitute (PESS). Those who received the salt substitute had lower rates of stroke, cardiovascular events and all-cause mortality than those who received regular salt, with no increase in adverse events with the PESS, over an average follow-up period of 4.7 years.

In the same years, a smaller study was published from India,[2] where 502 patients were randomized to receiving regular salt *v.* a 30% PESS in 7 Indian rural villages and followed up for 3 months. A total of 476 patients completed the follow-up of 3 months. The systolic blood pressure in the PESS group reduced on an average by 4.6 mmHg and the diastolic blood pressure by 1.1 mmHg, with no perceived change in taste between the two products.

The same group that did the rural China study also did a cost-effectiveness analysis[3] and showed that the use of PESS had a 95% probability of being cost-saving and a >99.9% probability of being cost-effective, for the prevention of stroke and for improving quality of life.

Given the burden of hypertension, it makes sense to promote PESS. However, a cursory check of food markets shows that only one major company has a potassium substitute, in two strengths (15% and 30%). Some authors of the rural China study then used the data to do a modelling study in the Indian population[4] and concluded that there would be substantial net benefits, preventing 8%–14% of annual cardiovascular deaths.

One concern is hyperkalaemia, especially in those with chronic kidney disease. This has to be kept in mind at a patient level, but the modelling study suggested that this may not be a problem at the population level.

If using less than two teaspoons of salt per day or a PESS is a challenge, then a recent study[5] has shown that just throwing away your salt-shakers and not adding extra salt to food during meals can save lives. Ma and colleagues[5] studied 176 570 patients from the UK Biobank and found that those who had the least frequency of adding extra salt had lower risks of cardiovascular disease. While there are limitations to the study, because the frequency of adding salt was self-reported and the population studied was only from the UK, the results otherwise make sense and this practice can easily be added to our daily routines and lives.

Sometimes, simple things can make all the difference to our health and lifespan.

SOME MORE VITAMIN D RUBBISH

In an earlier edition of Masala,[6] I wrote about the findings of the D-Health study[7] that found no difference in all-cause mortality or mortality from cardiovascular disease or cancer in those given vitamin D supplements versus those given placebo capsules. And yet, the amount of money being put into vitamin D research just doesn't stop.

A recent study by the EPIC group[8] used Mendelian randomization on a large cohort of over 500 000 people to show a causal relationship between vitamin D levels <16 ng/ml (40 mmol/ml) and mortality. This potentially makes sense, because <20 ng/ml is considered insufficiency and <12 ng/ml, deficient (which is not true of Indian pathology reports, which use higher cut-offs and render a vast number of people between 20 ng/ml and 30 ng/ml, insufficient, when they are actually normal), and a vitamin D level <16 ng/ml could perhaps be associated with increased all-cause mortality.

Except that Mendelian randomization is a new technique that uses genetic variants of a particular biomarker, in this case vitamin D, to define separate population subgroups that may or may not be susceptible to the effects of vitamin D deficiency. This is a complex methodology, understood by very few people (at least four geneticists I reached out to in India confessed they had no clue). It also makes a bunch of assumptions related to the genetic methods and the statistics used. The claim by researchers using Mendelian randomization is that it eliminates confounders and improves causal relationships between problems and outcomes. Unfortunately, the technique itself comes with its own problems.

There were two rebuttal letters[9] published recently in the same journal that challenged these assumptions and the statistical inferences—arguments which the authors accepted.

Their conclusion is that they cannot find a relationship between low vitamin D levels and increased mortality.[9] Unfortunately, unless someone also reads this retraction of the conclusion by the authors (the article itself does not need retraction, because the study is not ethically flawed), the original conclusion will continue to remain in the minds of the people who have read it, either in the journal or in the lay press.

Two other articles[10,11] have used the same UK biobank data to arrive at similar conclusions suggesting that low vitamin D levels are associated with either increased mortality[10] or increased cardiovascular risk.[11] Both these studies have used similar flawed assumptions and statistical analyses and their conclusions also need to be corrected.

It gets even more interesting.

In July 2022, LeBoff and colleagues[12] published a randomized controlled trial that showed that the use of vitamin D supplementation in the general population did not reduce the risk of fractures compared to placebo, a finding that is also in line with the United States Preventive Services Task Force (USPSTF) recommendations. An accompanying editorial[13] has this to say: 'Adding those findings to previous reports from VITAL and other trials showing the lack of an effect for preventing numerous conditions suggests that providers should stop screening for 25-hydroxyvitamin D levels or recommending vitamin D supplements, and people should stop taking vitamin D supplements to prevent major diseases or extend life.'

More tellingly, a recent study from Mongolia by Ganmaa and colleagues[14] in children with low vitamin D levels found that while vitamin D supplementation increased vitamin D levels,

there was no effect on growth, body composition or pubertal development. The authors however gave no explanation for this result.

In effect, people, patients and doctors need to both stop measuring vitamin D levels and giving vitamin D supplements, unless patients are diseased to an extent that they are likely to get vitamin D deficiency, which typically means malnutrition, malabsorption or catabolic conditions such as end-stage malignancies and the like. It is also time researchers started spending their time, energy and effort on other areas and left vitamin D alone, as was the case before the 1990s.

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Notice of Retraction

Guinea-worm (Dracunculus medinensis) infection presenting as a diabetic foot abscess: A case report from Kerala, National Medical Journal of India Vol. 32, No. 1, 2019:22–3.1 DOI: 10.4103/0970-2S8.2721l1 PMID: 31823935

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The article 'Guinea-worm (Dracunculus medinensis) infection presenting as a diabetic foot abscess: A case report from Kerala[1] is being retracted based on a letter received from the Director of the National Centre of Disease Control, New Delhi where the centre has objected to the identification of the guinea-worm by the authors only on the basis of morphological observation, without any microscopic and histopathological examination. The author has agreed to the objection and to the withdrawal of the article. This article is therefore being retracted.

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