HEADS UP **Krishna Chinthapalli**

Dethroning the king of condiments

What is the link between salt, hypertension, and mortality?

Killing an elephant is not easy. The Waliangulu tribesmen in Kenya use an arrow dipped in the poisonous sap of the *Acokanthera* plant. They shoot from a few steps away into an elephant's underbelly so that the active toxin, ouabain, can rapidly enter the bloodstream. Ouabain molecules bind to and inhibit the sodium pump in cardiac muscle. Intracellular sodium thus increases, which stimulates the sodium-calcium exchanger to get rid of sodium and take in calcium instead. The calcium ions make the myocyte contract permanently and can stop a 20 kg heart from beating again.

Ouabain circulates naturally in our own bloodstream too, but instead of stopping the heart it seems to make arteriolar smooth muscle contract and thus raise blood pressure. The theory is that increased dietary salt intake leads to sodium and water retention, which stimulates ouabain, which causes hypertension.[1] But in practice the role of salt has been highly controversial.

In 1988 the *BMJ* published the cross sectional Intersalt study of Kenyan villagers and 51 other populations around the world.[2] In each population a person's urinary sodium excretion (a reliable surrogate for salt intake) correlated with systolic blood pressure. Across populations higher salt intake was linked to a faster increase in blood pressure over time. In 2001 the DASH-sodium study found that reducing salt intake could lower systolic blood pressure by up to 7 mm Hg.[3] In 2007 long term follow-up of 2400 patients in trials of hypertension reduction indicated that education about diet, counselling, and a recommendation to cut dietary salt led to 30% less risk of cardiovascular disease.[4] Graham MacGregor, a former president of the British Hypertension Society, collaborated with Feng He on a meta-analysis in the *BMJ* this summer.[5] This indicated that reducing salt intake by 6 g a day could prevent 35 000 deaths a year in the United Kingdom.

This slow death from salt prompted the UK National Institute for Health and Care Excellence to say that we should be eating less than 3 g of salt a day by 2025. However, in September the Department of Health for England abandoned plans to set a maximum salt level on new food products.

The food industry has lobbied hard against salt reduction. Over four fifths of the salt we eat is in processed foods, a cheap way to improve taste and longevity. Companies have sought to obfuscate the link between salt and hypertension since the 1980s by highlighting negative studies or funding pro-salt research.[6] Their trade lobby, the Salt Institute, says that salt is a "natural food ingredient with no calories" that can help you "live longer, live smarter, and live nutritiously."

Other experts also argue that the evidence cited above (and indeed our meals) should be taken with a pinch of salt. In the same *BMJ* issue as Intersalt, the observational Scottish Heart Health Study found that potassium and alcohol but not sodium affected blood pressure,[7] and an accompanying editorial stated, "Salt has only small importance in hypertension."[8]

Gary Taubes, author of diet books and a science journalist, recently highlighted the role of sugar in obesity in the *BMJ*.[9] In 1998 he wrote a report in *Science* about the politicised debate on salt.[10] He thinks salt has been unfairly maligned but has, to protect his integrity, refused to associate himself with the Salt Institute. Michael Alderman, now editor of the *American Journal of Hypertension*, has disclosed that he was previously a paid consultant to the Salt Institute. From recent studies he concluded that the relation between salt intake and cardiovascular mortality was a J shaped curve, with a salt intake of 5-15 g a day conferring the lowest risk.[11] He also suggested that low salt intake could activate the sympathetic nervous system and increase insulin resistance.

In 2011 Alderman's journal published a Cochrane review of trials of salt reduction interventions, which could not confirm an association with morbidity or mortality.[12] Another Cochrane review of salt intake, blood pressure, and the renin-aldosterone-angiotensin system in the same journal by Niels Graudal and colleagues concluded that low salt diets had no net benefit.[13] This analysis was flawed, say He and Macgregor, because it included short term trials. Graudal, in turn, criticised He and Macgregor's own meta-analysis for concluding that sodium reduction was associated with lower mortality when no direct evidence of a link existed.[14]

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Research: Effect of longer term modest salt reduction on blood pressure (*BMJ* 2013;346:f1325)

This year the US Institute of Medicine convened a panel of experts, bravely including both He and Graudal, to investigate the evidence on behalf of the Centers for Disease Control and Prevention. The panel lamented the quality of recent studies but decided that, though high salt intake was linked to cardiovascular disease, there was no evidence of benefit from reducing salt intake to below 6 g a day and in fact some evidence of harm.

Kenya is the only one of 187 countries with an average salt intake meeting the American Heart Association guideline: less than 3.8 g a day. Towards the end of the 19th century, though, most countries had much lower salt intake, and efficient retention of sodium by the kidneys protected the Waliangulu tribe and others from hyponatraemia.

Meanwhile, in India a crippling British salt tax of over 1000% and a ban on local salt production played a part in millions of deaths from famine, said Florence Nightingale, because of lack of food preservation or salt deficiency from heat exhaustion and diarrhoea. Years later Mohandas Gandhi famously violated the salt tax by picking up a lump of salt off a beach in Gujarat in the early dawn and called salt "the king among condiments." Paradoxically, Gandhi himself had renounced table salt a few years earlier and advised its use only "when necessary as an adjunct." Now, despite hundreds of scientific studies, his ambivalent views are perfectly understandable.

Krishna Chinthapalli is associate editor, *BMJ*
kchinthapalli@bmj.com

Competing interests: None declared.
Provenance and peer review: Commissioned, externally peer reviewed.
References are in the version on bmj.com.
Cite this as: *BMJ* 2013;347:f7192