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Salt reduction lowers cardiovascular risk: meta-analysis of outcome trials

A recent Cochrane Review by Rod Taylor and colleagues, published simultaneously in The Cochrane Library[1] and the *American Journal of Hypertension*[2], stated in the plain language summary that "Cutting down on the amount of salt has no clear benefits in terms of likelihood of dying or experiencing cardiovascular disease".[1] The Cochrane Library's own press release headline included this statement: "Cutting down on salt does not reduce your chance of dying".[3] Both of these statements are incorrect.

The study reported in the paper by Taylor and colleagues is a meta-analysis of randomised trials with follow-up for at least 6 months on the effect of reducing dietary salt on total mortality and cardiovascular mortality and events.[1,2] There were seven trials with 6250 participants (665 deaths). One of these trials in heart failure,[4] in our view, should not have been included because the participants were severely salt and water depleted due to aggressive diuretic therapy (frusemide 250–500 mg twice daily, and spironolactone 25 mg per day) as well as captopril 75–150 mg per day and fluid restriction to 1000 mL per day.[4] While on these treatments, participants were randomly assigned to a reduced salt intake or their usual salt intake.[4] In view of the fact that the dose of diuretics was not adjusted downwards, a lower salt intake is likely to worsen the salt and water depletion and therefore, unsurprisingly, resulted in worse outcomes.

In the remaining six trials, there is a reduction in all clinical outcomes (all-cause mortality, cardiovascular mortality and events) (table), although none of these are statistically significant. This trend of consistent reductions in all clinical outcomes seems to have been overlooked by Taylor and colleagues.[1] The non-significant findings are most likely the result of a lack of statistical power, particularly as Taylor and colleagues analysed the trials for hypertensives and normotensives separately. We have reanalysed the data by combining data for hypertensives and normotensives together. Our results show that there is now a significant reduction in cardiovascular events by 20% ($p<0·05$) (figure) and a non-significant reduction in all-cause mortality (5–7%), despite the small reduction in salt intake of 2·0–2·3 g per day. The results of our reanalysis, contrary to the claims by Taylor and colleagues, support current public health recommendations to reduce salt intake in the whole population.

Taylor and colleagues call for further large long-term randomised trials of salt reduction on clinical outcomes.[1,2] According to their own calculations, at least 2500 cardiovascular events need to be obtained to

	Trials in normotensives (n=3)*	Trials in hypertensives (n=3)*
Reduction in salt intake at end of trial (g per day [95% CI]); duration 6–36 months	2·0 (1·1 to 2·9)	2·3 (1·8 to 2·8)
Fall in blood pressure at end of trial (mm Hg [95%CI]); duration 18–36 months		
Systolic	1·11 (–0·11 to 2·34)	4·14 (2·43 to 5·84)
Diastolic	0·80 (0·23 to 1·37)	3·74 (–0·93 to 8·41)
Difference in all-cause mortality at longest follow-up (95%CI); duration 7 months to 12·7 years	10% reduction (RR 0·90, 0·58 to 1·40)	4% reduction (RR 0·96, 0·83 to 1·11)
Difference in cardiovascular events at longest follow-up (95%CI); duration 7 months to 11·5 years	29% reduction (RR 0·71, 0·42 to 1·20)	16% reduction (RR 0·84, 0·57 to 1·23)
Difference in CVD mortality at longest follow-up (95%CI); duration 7 months to 6 years	..	31% reduction (RR 0·69, 0·45 to 1·05)

RR=relative risk; CVD=cardiovascular disease. * Not all measurements were made in all trials.

Table: **Change in salt intake, blood pressure, and clinical outcomes with results from the meta-analysis by Taylor and colleagues[1] (excluding the trial in heart failure)**

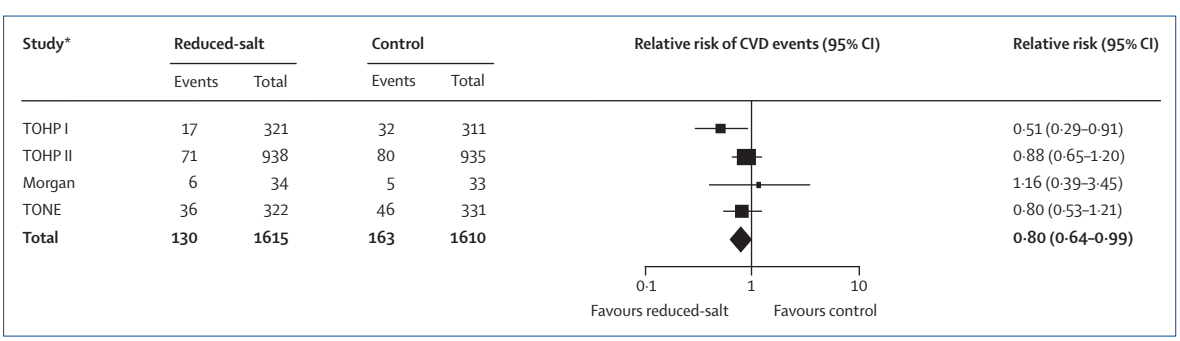

Figure: **Relative risk of cardiovascular disease (CVD) events in our meta-analysis of outcome trials of salt reduction at longest follow-up combining hypertensive and normotensive individuals**

Duration of follow-up ranged from 7 months to 11·5 years. We used fixed effect model with normotensives and hypertensives combined. Heterogeneity χ^2=3·20, df=3 (p=0·36); I^2=6%.Test for overall effect Z=2·02 (p=0·04). TOHP I=Trial of Hypertension Prevention, phase 1. TOHP II=Trial of Hypertension Prevention, phase 2. TONE=Trial of Nonpharmacologic Interventions in Elderly. *Data for individual trials taken from Taylor and colleagues' meta-analysis.[1]

detect a 10% reduction (at 80% power and 5% significance level).[2] This would require randomisation of about 28 000 participants to a low or high salt intake and then maintenance of the two separate diets for at least 5 years. Such a trial is impractical because of logistical and financial constraints, and the ethical issues of putting a group of people on a high salt diet for so many years.

In our view, Taylor and colleagues' Cochrane review and the accompanying press release reflect poorly on the reputation of The Cochrane Library and the authors. The press release and the paper have seriously misled the press and thereby the public—for example, in the UK the *Daily Express* front page headline read "Now salt is safe to eat—Health fascists proved wrong after lecturing us all for years"[5] and there were similar headlines throughout the world.

The totality of evidence, including epidemiological studies, animal studies, randomised trials, and now outcome studies all show the substantial benefits in reducing the average intake of salt.[6–9] Most countries have adopted policies to reduce salt intake by persuading the food industry to reformulate food with less salt, as is occurring successfully in the UK,[10] and also by encouraging people to use less salt in their own cooking and at the table. WHO has recommended salt reduction as one of the top three priority actions to tackle the global non-communicable disease crisis.[11] A reduction in population salt intake will have major beneficial effects on health along with major cost savings in all countries around the world.[6,12,13]

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Will the Decade of Vaccines mean business as usual?

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In 2011, the story of immunisation coverage worldwide hovers between the glass half empty and the glass half full. Anticipated advances in vaccinology during this new Decade of Vaccines will only translate into reductions in global morbidity and mortality from targeted illnesses if fundamental restructuring means that the most marginalised countries (particularly in Africa and southeast Asia) gain access to new and established vaccines. Routine vaccine coverage and the introduction of new vaccines have increased enormously in the past 10 years, with 14·6 million more children receiving the routine diphtheria, tetanus, and pertussis vaccine in 2009 than in 2000.[1] Yet 23 million children younger than 1 year are still missed,[1] particularly those living in the poorest quintile of low-income countries who have not received the primary series of childhood vaccines.[2]

At the World Economic Forum in Davos, Switzerland, in January, 2010, the Bill & Melinda Gates Foundation launched the Decade of Vaccines by pledging US$10 billion to support worldwide efforts to develop and deliver vaccines to the world's poorest children in the next decade.[3] Although this pledge could save the lives of more than 8 million children, this sum will still not reach the potential of vaccines to contribute to the achievement of Millennium Development Goal (MDG) 4—reduce the mortality rate in children younger than 5 years by two-thirds between 1990 and 2015. Partners in the Decade of Vaccines (WHO, UNICEF, the Gates Foundation, and the US National Institute of Allergy and Infectious Diseases) know that there are crucial gaps in policy, resources, advocacy, and research that will need to be addressed if the next 10 years is really to be business unusual for immunisation access.

Although many vaccine strategies target adolescents, adults, and elderly people, the main focus of coverage remains on children younger than 5 years. In 2008, of the nearly 8·8 million deaths in children younger than 5 years worldwide, 68% were caused by infectious diseases, 18% by pneumonia, 15% by diarrhoea, and 8% by malaria.[4] Nearly half of these deaths were in five populous countries: India, Nigeria, Democratic Republic of the Congo, Pakistan, and China.[2] Many of the deaths due to infectious disease can be prevented by the introduction of new and established vaccines, while others, including malaria, tuberculosis, HIV infection, and neglected parasitic diseases, still await the development of effective vaccines. The lag in introduction of life-saving vaccines in low-income countries with high disease burden has been most tragically shown by the *Haemophilus influenzae* type b conjugate vaccine (HibCV).[5] Introduction of this vaccine in low-income countries, where most of the 371 000 yearly deaths from *H influenzae* type b occurred,[6] was started only 12 years after its institution in developed countries. It took another decade before at least 60% of children in low-income countries gained access to the vaccine.[5] This delay in