



18th March 2005

Health Advisory Section (MDP 24)
National Health and Medical Research Council
GPO Box 9848
Canberra, ACT 2601
Australia

Dear Sir/Madam

Submission Nutrient Reference Values

The National Heart Foundation of New Zealand ("the Heart Foundation") welcomes the opportunity to comment on the draft document "Nutrient Reference Values for Australia and New Zealand, including Recommend Dietary Intakes", produced by the National Health and Medical Research Council (NHMRC) in December 2004. This submission specifically focuses on the recommendations to optimise diets for the lowering of chronic disease risk.

- The NHMRC might consider the population goals reported in the recently released WHO Technical Report on Diet, Nutrition and the Prevention of Chronic Diseases to ensure some level of consistency globally [1].
- It is unclear from the document whether this section is making recommendations based on evidence where mortality or morbidity are the primary end-points or whether it gives consideration to evidence using surrogate end-points for chronic disease such as blood pressure, blood lipids, glycaemia, body weight etc. It maybe important to make this distinction as it will determine the breath of research that is presented and the recommendations made e.g. dietary fibre for prevention of coronary heart disease vs. soluble fibre for management of LDL-C [2-5].
- Despite the approach of the NHMRC to include nutrients based on perceived and recorded essentiality, the Heart Foundation believes it prudent to consider the inclusion of nutrients and nutrient-factors also known to be pathogenic for chronic disease to ensure the adoption of a comprehensive series of recommendations.

Experimental and clinical intervention studies have identified energy density, saturated fats (specifically myristic and palmitic), dietary cholesterol, trans-unsaturated fats and excessive alcohol intake as pathogenic for certain chronic diseases [1, 6].

- Nutrient factors that have been associated with protection against chronic diseases or known risk factors for chronic diseases that are not included in these tables are flavonoids and other phytochemicals such as sterol esters, folate, and some minerals including potassium, calcium and magnesium.
- Pg 259, please consider adding to the list of tools for associating specific nutrients with chronic disease the research methods; meta-analysis and systematic review. There are a growing number of these reports presented in peer-reviewed nutrition and public health journals. Several have been released since the IOM documents were first published that you may wish to consider in the background document [2, 7-23].
- Pages 258-283 summarise the evidence for selected nutrients. It should be noted that compared to the traditional approach of risk factor modification that identifies a single food or nutrient component, a growing number of dietary trials are demonstrating greater efficacy for improving surrogate end-points with a more comprehensive approach [24-29]. Optimal prevention may be subtle independent or combined effects of multiple food and nutrient interactions within these diets.
- Given the propensity of the Australian and New Zealand populations to gain weight, the current prevalence of overweight and obesity in children and adults, and the documented associations between even modest weight gain and chronic diseases, the NHMRC might consider including a recommendation on dietary energy intake in Table 2, pg 285.
- Table 2, pg 285; please consider offering guidance on an appropriate level of saturated plus trans-unsaturated fat in the diet.
- Table 2, pg 285; please consider offering guidance on an appropriate level of "free sugar" in the diet or at least appropriate food choices to ensure an adequate intake of dietary fibre, non-starch polysaccharide, whole grains, and/or cereal fibre. Within this, much of the research supporting a protective association between dietary fibre and coronary heart disease is for cereal fibre rather than fruit or vegetable fibre. In the Male Health Professionals' Study, after 6 years, dietary fibre intake was inversely related with CHD death, highest quintile (median, 28.9 g.d⁻¹) versus lowest quintile of intake (median, 12.4 g.d⁻¹) age-adjusted relative risk was 0.45 (95% CI, 0.28 to 0.72), and for total MI was 0.59 (95% CI, 0.46 to 0.76) and after multivariate adjustment 0.64 (95% CI, 0.47 to 0.87) [30]. A 10 g.d⁻¹ increase in dietary fibre intake corresponded with a relative risk of total MI of 0.81(95% CI, 0.70 to 0.93). Cereal fibre was most strongly associated 0.73 (95% CI, 0.56 to 0.94). After simultaneous adjustment for other sources of fibre, only cereal fibre remained significant, and remained so even after further adjustment for β-carotene, folate and vitamin B₆, relative risk 0.73 (95%

CI, 0.57 to 0.94) for each 10 g.d⁻¹ increase. The 12% and 17% reduced risk for vegetable and fruit fibre intakes respectively, lost significance.

In the Nurses' Health Study after 10 years, dietary fibre intake was inversely related with CHD (non-fatal MI and CHD death) [31]. Highest quintile (median, 22.2 g.d⁻¹) versus lowest quintile of intake (median, 11.5 g.d⁻¹) age-adjusted relative risk was 0.53 (95% CI, 0.40 to 0.69, p for trend<0.001), and multivariate relative risk was 0.77 (95% CI, 0.57 to 1.04, p for trend=0.07). Age-adjusted relative risk for nonfatal MI was 0.57 (95% CI, 0.42 to 0.77, p for trend<0.001), and for fatal CHD 0.41 (95% CI, 0.23 to 0.70, p for trend=0.002). A 10 g.d⁻¹ increase in dietary fibre intake corresponded with a multivariate relative risk of CHD of 0.81(95% CI, 0.66 to 0.99), further adjustment for nutrients widened the confidence interval slightly (relative risk, 0.79; 95% CI, 0.59 to 1.07). Only cereal fibre was independently associated with reduced risk. Multivariate risk, after simultaneous adjustment for other sources of fibre and other nutrients, for the highest versus lowest quintiles, was 0.66 (95% CI, 0.49 to 0.88, p for trend<0.001). For each 5 g.d⁻¹ increase in cereal fibre, the multivariate relative risk was 0.63 (95% CI, 0.49 to 0.81). No risk association was found for vegetable or fruit fibre intakes. Mean fibre intakes were relatively low. For lowest versus highest quintiles of intake respectively, total dietary fibre intake was 10.6 g.d⁻¹ and 23.7 g.d⁻¹, cereal fibre 2.8 g.d⁻¹ and 6.0 g.d⁻¹; vegetable fibre 4.0 g.d⁻¹ and 9.2 g.d⁻¹, and fruit fibre 1.6 g.d⁻¹ and 6.0 g.d⁻¹. Soluble fibre intakes were 3.2 g.d⁻¹ and 6.8 g.d⁻¹ and insoluble fibre 7.4 g.d⁻¹ and 16.9 g.d⁻¹, respectively.

Should the differentiation be made in this document or supported through food-based guidance?

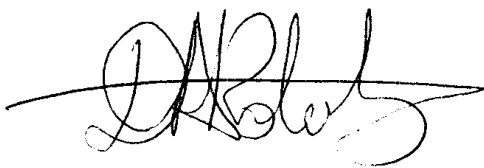
- There is an obvious omission of alcohol from this document and possibly from table 2, pg 285. It is unclear within the document why this omission has occurred. The Heart Foundation recognises that the promotion of the health benefits of alcohol consumption runs the risk of increasing the mean alcohol consumption, and thereby the problems associated with excessive alcohol consumption. Heavy drinking of alcohol is also associated with an increased risk of cardiovascular disease, and there are social risks associated with heavy or binge drinking, particularly among young people. However, the evidence does suggest that a low to moderate intake of alcohol is associated with protection from coronary heart disease [12, 18, 23, 32]. The benefit for the individual will vary according to their underlying absolute risk of a coronary death. Therefore, the group most likely to benefit is older people who have a high absolute risk of a coronary event, and a low risk of injury, cirrhosis and other adverse effects of alcohol. The mortality-related benefits of light to moderate drinking begin to outweigh the risks among men in their 40's and women in their 50's, and continue to increase with age. There is no safe level of alcohol consumption for men under the age of 40 years and pre-menopausal women where the risks of all-cause mortality, outweigh the benefits, even with low levels of alcohol consumption [6].

- Would you please reference the modelling work that is referred to throughout the draft document but specifically in the background evidence supporting the recommendations for the prevention of chronic disease.

Finally, the Heart Foundation would ask that due consideration is given to the communication and implementation of the final recommendations from this report. One aspect that concerns us is the way in which these recommendations could be interpreted by the public and lay-media. For example, one could interpret the finding that as no EAR\RDI or AI was set for carbohydrates that this supports the premise for exclusion of all carbohydrate-containing foods from the diet. This would of course upset many nutrition professionals, add support to the promoters of low-carbohydrate weight reducing diets, create inconsistencies with national dietary guidelines and possibly promote the early development of coronary heart disease due to lack of whole grain, cereal fibre, fruits and vegetables and/or soluble fibre.

Thank you for this opportunity to consider the draft nutrient reference values. Historically, the Heart Foundation has played an important role in promoting evidence-based nutrition for the prevention and management of cardiovascular disease. Our organisation has a keen interest to advocate for the adoption and implementation of evidence-based public health policy by the respective government agencies. We look forward to the next stages in the development process.

Yours sincerely

A handwritten signature in black ink, appearing to read 'DR Roberts', with a long horizontal stroke extending to the right.

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