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Dear Dr. Puska:

Thank you for the opportunity to comment in writing on the draft WHO/FAO report, "Diet, Nutrition and the Prevention of Chronic Diseases." The 60-day extension of the comment period for this very large document was greatly appreciated. Developing general support for food-based scientific and policy positions is often a time-consuming process, but usually the results are worth the wait. Such a process requires participation by a geographically-balanced group of experts and reviewers, since the report is meant to have a global audience, in addition to adequate time to review the draft report.

I am providing these comments on behalf of the International Life Sciences Institute (ILSI), a nonprofit, worldwide foundation established in 1978 to advance the understanding of scientific issues relating to nutrition, food safety, toxicology, risk assessment, and the environment by bringing together scientists from academia, government, industry, and the public sector to solve problems with broad implications for the well-being of the general public. ILSI receives financial support from industry, government, and foundations. Through the ILSI network of 15 branches around the world along with the ILSI Research Foundation and the ILSI Center for Health Promotion, we have considerable expertise related to nutrition, diet and prevention of chronic diseases. As our status as a nongovernmental organization shows, we are proud of our ongoing interaction with WHO and FAO on these and other issues.

We offer the following comments in an effort to improve the science base described in the report, so that effective, sustainable nutrition efforts can be developed to prevent chronic diseases.

Comments related to the overall report:

- The ideas presented in the text are not all held to the same criteria of evidence described as the rule in the various annex documents. There appears to be two

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different evidence scales – one for diet and nutrition studies and another, more relaxed scale for the environmental variables. The hierarchy of evidence is not clearly stated even in the specific annex documents. Thus, statements and conclusions appear that are not proportional to the evidence cited.

As this report is likely to be summarized and much of the methodology lost in the process, it is quite likely that the distinction between types of evidence will be lost on the reader. Furthermore, we believe it is important that the so-called environmental variables be held to the same rigor as the diet and nutrition variables. Failure to do so increases dramatically the risk that the recommendations made will not be effective, sustainable strategies to prevent chronic disease.

For example, in the obesity background paper, the correlation between parental Body Mass Index (BMI) and children's BMI is documented as very strong. So is the home influence on food choice leading to varied diets. No evidence is provided to support the notion that advertising increases obesity. Yet, advertising is listed as a probable risk factor for obesity and parents/home environment is not listed at all.

- Reference is made in several places to the weakness of food intake data collected through questionnaires, but then studies based on data from food frequency questionnaires are held up as the primary evidence for various recommendations.
- Within the evaluation of scientific data, personal viewpoints and/or data from one or two studies often are elevated to the level of international consensus when in fact that is not the case. For example, the background paper on the prevention of dental caries is at odds with all recent international expert consultations, including several organized by FAO and WHO (International Dental Federation Second World Conference on Oral Health Promotion, 1999; FAO/WHO Expert Consultation on Carbohydrate and Human Nutrition, 1998). No scientific justification for the limitation of sugars intake (< 10 percent of calories) is provided. No scientific evidence is provided that implementation of such a limit would reduce dental caries. In fact, data from the WHO Global Oral Health Databank indicate a decline in caries prevalence and severity in countries where sugars intake has remained unchanged or increased.

The international consensus on how best to prevent dental caries is and has been encouraging sound oral hygiene practice, exposure to fluoride through drinking water or toothpaste and sensible eating frequency. There are data to show that these practices result in lowering caries in populations in developing as well as developed countries.

- The report is helpful in describing the full continuum of nutritional problems in the world today – from undernutrition to overnutrition, and acknowledges the importance of micronutrient deficiencies as major public health problems.

However, the recommendations do not carry this point through. There is no recommendation that everyone eat the equivalent of the Recommended Dietary Allowance or its equivalent for all recognized micronutrients, which is a serious omission.

- There is a definite slant in the report to data from the developed world, primarily the United States and Europe. Even the authors are overwhelmingly from these geographic areas. Before assuming that interventions that have been tried, successful or not, in the developed world will work in the developing world, it would seem prudent to have some evidence available. None is presented in this report.

#### Specific Comments on Prevention of Excess Weight Gain and Obesity

- Weight gain and obesity are the result of energy imbalance. Thus, energy expenditure, though not the focus of this report, is a major contributor to prevention of weight gain and lack of activity is a major factor in promoting weight gain. This issue must be adequately addressed.
- Frequency of eating is cited as being inversely correlated with weight, yet snacking is classified as “insufficient evidence”, and a recommendation is made to reduce snacking. This is not logical.
- A recommendation to reduce sugars consumption to < 10 percent of calories appears without any supporting evidence that such an action will prevent excess weight gain or obesity.
- The concept of energy density (calories per gram of food/beverage) is offered as a major contributor to weight gain and obesity and the energy density of beverages is singled out as particularly important. This is another example of a concept that is currently being discussed within the scientific community, but for which there is not a scientific consensus at the moment because adequate data are not yet available.
- A blanket statement is made that all no-fat or reduced fat foods are as energy dense as the full fat form. This will require substantiation beyond the one reference cited (Seidell JC. Prevention of obesity: the role of the food industry. Nutr Metab Cardiovasc Dis 1999;9:45-50). Care must be taken to be sure that the portion sizes being compared are equivalent. Since a gram of fat contains more than twice the caloric density of carbohydrate, it is difficult to understand how removing fat results in a more energy dense product on a gram for gram basis.

#### Specific Comments on Prevention of Type 2 Diabetes

- For Table 1, it is not clear what rationale guided the decisions for putting each factor into a category. There are inconsistencies between the conclusions reached in the text and those expressed in Table 1. For example, maternal diet (protein/carbohydrate ratio) is listed as increasing risk of impaired glucose tolerance conflicting data and uncertain impact on risk for type 2 diabetes. The text states “Two studies on the long-term effects of maternal diet in humans have indicated that the ratio of animal protein to carbohydrate may play a significant role in birth weight (111,112).” This seems to be weak evidence to support the table conclusions. Physical activity should be included as having “convincing” evidence for decreasing risk.
- Clinical trials in type 2 diabetes patients, comparing isocaloric diets high in fat (up to 50 percent energy) and high carbohydrate (up to 60 percent energy) have shown positive effects of the high fat diets on plasma triglycerides and glucose levels. None of these studies are included (Sanz-Paris 1998).
- There is no evidence that monounsaturated fatty acids (MUFA) intake should be at or below a certain level. Clinical trials with high fat diets, including MUFA, have shown positive effects on plasma triglycerides and glucose compared to isocaloric, low fat diets in patients with type 2 diabetes (Sanz-Paris 1998, Garg 1998).
- Here and in the cardiovascular disease paper, there is heavy reliance on the unproven concept of glycemic load. Furthermore, the data used to calculate the glycemic load are food frequency data. Given the growing body of data that demonstrate that a specific food’s glycemic index is altered by the other foods with which it is consumed and how the food was prepared, it is improbable that food frequency data (where there is no knowledge of which foods are eaten together or how each food was prepared) could be used to generate an appropriate glycemic load value. Furthermore, to date, there is no scientific consensus on the usefulness of this value as an indicator of disease risk or for evaluation of a prevention strategy. See Wolever 2001.
- There may well be a difference between the glycemic response of a diet as measured by postprandial insulin, reducing glycosylated hemoglobin levels and diastolic blood pressure and the response to lowering carbohydrate intake. For this reason, the heading in Table 1 should read “Low Glycemic Index Diets”. The same should be done with the table in the obesity chapter.

References:

Garg A. High-monounsaturated-fat diets for patients with diabetes mellitus : a meta-analysis. *Amer J Clin Nutr* 1998;67:577S-582S.

Sanz-Paris A. et al. High-fat versus high carbohydrate enteral formulae: effect on blood glucose, C-peptide, and ketones in patients with type 2 diabetes treated with insulin or sulfonylurea. *Nutrition* 1998;14:840-845.

Wolever T. Carbohydrates and Health – the FAO/WHO Consultation. *Australian J Nutr Dietet* 2001;58 supp 1: S3-S8.

#### Specific Comments on Prevention of Cardiovascular Disease

- WHO and FAO are to be commended for evaluating individual fatty acids in assessing risk for cardiovascular disease. This is a sound reflection of the current state of knowledge.
- Care should be taken to avoid implying that observational data such as that in the Nurses Health Study (Lui et al. Fruit and vegetable intake and risk of cardiovascular disease: the Women's Health Study. *Amer J Clin Nutr* 2000;72:922-8) are intervention data. Such text contributes to the lack of clarity in the hierarchy of evidence used to generate conclusions.
- Recommendations to increase fish consumption should be fully investigated to avoid unintended consequences on aquatic environments worldwide. The statement that more than half of the world's fish catch goes into rendered meal to feed animals is not correct according to FAO statistics. The correct figure is less than one-third. These fish are mostly anchovy, sardines and the unused portions of other commercial fish. Half of meal is fed to aquaculture not to farm animals. These data leave open the question of whether fish meal currently being produced is fit for human consumption and would be culturally acceptable across the world.
- The section on sodium relies to some degree on several meta-analyses that are not considered valid by the experts in this area (ref. 2, 3, 8 and 10). Several other valid meta-analyses were not included (Graudal et al. 1998, Midgley et al. 1996, and Swales 1995).
- The paper also refers to the Dutch neonatal study (ref. 13, 14 and 17). These findings were not confirmed by the recent study in England (Singhal et al. 2001).
- The DASH data are very exciting, but there has been little time to digest the full meaning of the two studies. It is important to note that for both studies the control diet was low in potassium, calcium and magnesium, which would likely induce salt sensitivity and elevate blood pressure as compared to normal Western diets. The subjects selected for the DASH trials were likely salt sensitive already. Thus, the true impact of the DASH approach may be less than reported. It is also important to see how much of the blood pressure reduction was actually achieved by the very low sodium intakes.

As a practical matter, the lowest sodium diet is not one that free-living individuals self-selected. All of the foods used were specially prepared. Is this likely to be a feasible approach in developing countries? The data presented support the benefit of diets high in fruits, vegetables and whole grains, possibly a more effective approach.

- Very little attention is given in this report to the importance of other minerals in modifying the expression of sodium intake on blood pressure. A recent study in China (*J Hypertension* 2001;19:1325-1331) showed that moderate potassium supplementation resulted in a substantial reduction in blood pressure despite the fact that those receiving the supplementation had a significant increase in their dietary intake of sodium compared to the placebo group. It is critical to include all relevant information, not only the data that support one's conclusion.
- The recommendation for restricting dietary sodium intake to approximately 70 mmol or 1.7 grams per day is said to be "not associated with adverse effects." Yet, no data are provided to justify this statement. While there are data that show an adverse biochemical effect, i.e., elevated insulin levels and insulin resistance (Ames 2001), there have been no health outcome intervention studies designed to address either the benefits or the harm of such a large reduction in sodium intake. A particular group at elevated risk may be young people who exercise vigorously in warm climates. It would be prudent to make sure that there are no adverse effects before encouraging adoption of such a recommendation.
- Please see the reference to glycemic load above.
- Evidence cited to support a "probable" ranking for beta carotene supplements is largely based on data from smokers. These individuals do not represent the general population. The recent Heart Protection Study (n= 20,536) found no adverse effects on vascular or non-vascular morbidity or mortality from supplementation with beta carotene, vitamin E and vitamin C (Collins et al. 2002).
- This paper omits large parts of the scientific data from epidemiological, mechanistic and intervention studies available on the relationship between intake of vitamin E as well as other antioxidant vitamins (vitamins C and beta carotene) with the risk reduction of cardiovascular disease (Kaul et al.2001, Pryor 2000, and Witztum 2001). Therefore, the scientific basis for the rating of the evidence is insufficient and the subsequent recommendations are inadequate. The overall scientific evidence strongly suggests a decrease in risk with dietary antioxidants and a possible decreased risk with vitamin E supplements.

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Witztum JL, Steinberg D. The Oxidative Modification Hypothesis of Atherosclerosis: Does It Hold for Humans? *Trends Cardiovasc Med* 2001;11:93-102.

Specific Comments on the Prevention of Dental Caries

- See discussion above under general comments.
- Prevalence data presented are very old and do not present an accurate picture of current caries prevalence.
- This paper omits a large and well-documented body of evidence showing that all fermentable carbohydrates have cariogenic potential (Imfeld 1983, Lingström 2000, Pollard 1993, Van Loveren 2000). Thus, it is not scientifically appropriate to focus only on sugars. Such a focus is in conflict with other recent expert consultation reports – FAO/WHO Expert Consultation on Carbohydrates in Human Nutrition (1998).

- Frequency of consumption of fermentable carbohydrates is a primary risk factor for dental caries, not the amount consumed (König 2000).
- This paper ignores the large body of data that supports proper dental hygiene, including the use of fluoridated toothpaste, and reduction of the frequency of eating as effective public health approaches to preventing dental caries (Miyazaki 1996, Van Loveren and Duggal 2001, Van Loveren 2000, Einarsdottir and Brathall 1996, Gibson and Williams 1999, Duggal et al. 2001, ).
- The description of the dietary risks of dental erosion is misleading. Frequency plus the duration of exposure, the behaviour/contact time with teeth, and the interaction with brushing habits in individuals with a reduced salivary flow increase risk for dental erosion. Continuous contact, together with a low salivary flow, e.g. keeping the fluid longer in the mouth than usual, is more important than the common eating and drinking habits (Ten Cate and Imfeld 1996). To complicate the situation further, it is well known, that acid stimulates salivary flow and thus contributes to a better oral clearance and neutralisation (Imfeld 1983, pp 71-75).

Therefore, the statement in table 9 that only some food items are linked to an increased risk is misleading. Many foods, in addition to soft drinks and juices, are acidic and are potentially erosive. These include fresh whole citrus fruits, wine, yogurt, sauerkraut, salad dressings, and herbal teas to give examples. Data from vegetarians, who usually do not consume carbonated beverages, show this group is at a higher risk for dental erosion. Thus, even individuals eating diets traditionally perceived as healthy are at risk for dental erosion (Ganss C et al. 1999).

The statement that dental erosion is perceived to be increasing is not supported. The International Dental Federation (FDI) issued a consensus statement on erosion in year 2000, which explains the complexities of the problem ([www.fdiworlddental.org](http://www.fdiworlddental.org), FDI 2000, Johansson et al. 2002).

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Ten Cate JM, Imfeld T (eds): Etiology, mechanisms and implications of dental erosion. *Eur. J. Oral Sci* 1996;140:149-244.

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Van Loveren C, Duggal MS. The role of diet in caries prevention. *Internat Dent J* 2001;51:339-406.

#### Specific Comments on the Prevention of Osteoporosis

- The role of vitamin K in the prevention of osteoporosis is underestimated in this report. Human intervention studies and epidemiological data clearly demonstrate that vitamin K can improve bone health.

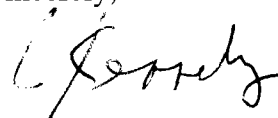
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Finally, we encourage WHO and FAO to strengthen the scientific basis of the report and develop recommendations based on evidence of efficacy and effectiveness. By doing this, we believe WHO and FAO will be able to provide much needed leadership in the important effort to prevent chronic disease worldwide. ILSI is willing to be a part of such an effort based on sound scientific evidence and to help generate such data to support effective sustainable programs.

Sincerely,

A handwritten signature in black ink, appearing to read 'E. Kennedy', written in a cursive style.

Eileen Kennedy, D.Sc.  
Global Executive Director