

## **No need for fish**

The World Health Organisation (WHO) and the Food and Agriculture Organisation (FAO) recently released a draft of a major report on "Diet, nutrition and the prevention of chronic diseases". Comments were invited on the draft.

<http://www.who.int/hpr/nutrition/ExpertConsultationGE.htm>

The Vegan Society responded jointly with the International Vegetarian Union. While we found much to praise in the draft which largely promotes a more plant-based diet, the draft includes one unsubstantiated recommendation that is not consistent with vegetarian diets: to increase the consumption of fish. On behalf of the Vegan Society and the International Vegetarian Union (IVU), Stephen Walsh prepared a critique of the scientific basis for this recommendation assisted by other members of the IVU Science group (IVU-SCI).

This critique exposes:

- the weakness of evidence for a benefit of fish consumption for people meeting the other recommendations in the report, such as reducing saturated fat intake;
- the strong evidence for a benefit from consumption of vegetable fats rich in monounsaturated and omega-3 polyunsaturated fatty acids;
- the evidence that any potential benefit of fish, even in high risk populations, can be overwhelmed by the effect of methyl mercury contamination of fish.

It also emphasises the adverse environmental impact of attempting to increase fish consumption.

Fish has no role in an ideal diet to promote the health of humans, animals and the environment.

**Comments on Diet, nutrition and the prevention of chronic diseases  
(Draft report of the joint WHO/FAO expert consultation)**

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The Vegan Society is an educational charity with the central objective of promoting a vegan diet for the benefit of people, animals and the environment. This encompasses promoting good health within a plant-based diet and representing the vegan view on UK, European and international bodies. The International Vegetarian Union is an umbrella organisation representing vegan and vegetarian societies across the world with the central objective of promoting vegetarianism throughout the world. This encompasses representing the vegetarian cause on appropriate international bodies. Funding of both organisations is by membership subscriptions and donations.

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**Comments**

The draft report on “Diet, nutrition and the prevention of chronic diseases” has much to commend it. As well as being largely scientifically sound in relation to individual health, its overall promotion of an increasingly plant-based diet (more brightly coloured vegetables and fruits, more whole grains and nuts and less saturated fat and cholesterol) will lead to reduced pressures on the environment and less ill treatment of animals and thus contribute to global health in its broadest sense.

We particularly welcome the recommendations to reduce saturated fat intake to less than 7% of calories and trans fats to less than 1%, and to increase consumption of vegetables, fruits, nuts, and whole grains. This provides good guidance for improving any form of diet. The recommendation of 5-8% of calories as n-6 PUFA (omega-6 polyunsaturated fatty acids) and 1-2% as n-3 (omega-3) PUFA is consistent with knowledge on the required balance of n-6 and n-3 to ensure healthful EPA levels in individuals who consume no pre-formed EPA, for whom n-6 PUFA intake (as linoleic acid, LA) should be about 2 to 4 times n-3 PUFA intake (as alpha-linolenic acid, ALNA).

In relation to fish, the report fails to follow the overall logic of making recommendations consistent with global population health. As noted on page 9 of the report:

Encouraging the consumption of fish will further endanger already over-fished oceans. Fish farming, while a positive development with respect to decreasing pressure on wild fish stocks, has also created some (unresolved) problems in terms of feeding and pollution of local environments.

Yet on page 18 the report states (*italics ours*):

The implications of the recommendations would be to increase the consumption of fruits and vegetables, *to increase the consumption of fish* and to alter the types of fats and oils as well as the amount of sugars especially in developed countries.

The recommendation to increase the consumption of fish implies a conflict between individual health and both global health and the religious and ethical beliefs of the large number of vegetarians across the world. We recognise that this recommendation was not made lightly, but consider it to be unjustified by current scientific knowledge. There is a probable beneficial effect of increased fish consumption in medium to high risk populations, but no evidence implying that fish consumption is necessary for optimal individual health.

The evidence indicates that there are alternative means of obtaining the individual health benefit sought which do not conflict with global population health and should therefore be preferentially recommended.

The current balance of evidence is better reflected in “Eat, drink and be healthy” by Walter Willett, produced last year by the Harvard Medical School and the Harvard School of Public Health. In this book, Professor Willett presents an evidence-based dietary pyramid which includes a purely plant-based diet as an option. He recommends that red meat and butter be used no more than occasionally as an essential step towards reducing saturated fat to desirable levels, and suggests that fish, poultry or eggs be consumed *zero* to two times per day. This appropriately reflects the uncertainty as to what constitutes an optimal diet and leaves due latitude for individual choice in the light of cultural, ethical and ecological considerations. As the draft WHO/FAO report appears to disagree with this assessment, we review the relevant scientific evidence below.

Based on both secondary prevention trials and prospective cohort studies, the evidence is compelling for a significant benefit of fish or EPA/DHA supplements in some high risk populations, such as those with high saturated fat intake or cohorts of heart attack survivors. However, as noted in the review by Marckmann and Gronbaek (1999), the evidence is much weaker for a benefit in low risk populations, such as those complying with the report's overall recommendations on saturated fat intake and PUFA intake.

Marckmann and Gronbaek's review does not discuss the results of Pietinen et al. (1997), who found a (marginally significant) 30% increase in risk of coronary death in the highest quintile by n-3 intake from fish compared with the lowest quintile in a multivariate analysis within a prospective study of Finnish male smokers. This observation is supported by Salonen et al. (1995), who found a 100% increase in acute myocardial infarction (AMI) in Eastern Finnish men consuming more than 30 g of fish per day compared with men consuming less than 30g of fish per day. All-cause mortality increased by 0.3% for each gram of fish consumed per day. Salonen et al. attribute these observations to the combination of a strong association of fish intake (particularly local inland fish) with methylmercury levels in hair and urine and a strong association of methyl mercury levels with AMI risk and cardiac death.

Rissanen et al. (2000) shed further light on this by showing that an observed beneficial effect of increased levels of serum DHA on risk of acute coronary events is strongly attenuated in the presence of high levels of mercury in hair. Indeed, their results show no significant difference in risk between the low-mercury low-DHA group and the high-mercury high-DHA group. At the very least, these results require any recommendation for increased fish consumption to be accompanied by a strong warning about the possible counterbalancing impact of methylmercury contamination. More disturbingly, Sorensen et al. (1999) found high prenatal methylmercury exposure to be associated with a 13 mm increase in both systolic and diastolic blood pressure at age 7. Without digressing into the many other ill effects of methylmercury exposure (including impaired neurological function in infants and reduced fertility in adults), it is clear that lifetime exposure to fish containing significant amounts of methylmercury cannot be recommended for the reduction of heart disease risk. This provides strong grounds to question the blanket recommendation of increased fish consumption.

The best evidence for a beneficial effect of fish in medium risk populations comes from the paper by Hu et al. (2002) based on the Nurses Study, which found reduced all-cause mortality of about 25% in the highest category of fish consumption (five or more servings per week) or very long chain n-3 consumption (0.24 % of calories per day). This protective association was attenuated but not eliminated in regular aspirin users. These results should be viewed in the context of an earlier paper on ALNA intake and heart disease in the same cohort, Hu et al. (1999). Median saturated fat intake was 13%, with the lowest quintile consuming 11.4% and the highest 17.4% (considerably higher than the WHO/FAO recommendation of less than 7%). Consumption of alpha-linolenic acid (ALNA) in this cohort ranged from 0.7 g per day in the lowest quintile to 1.36 g per day in the highest quintile (considerably lower than the WHO/FAO recommendations of 2 to 4 g of n-3 PUFA). The highest quintile of ALNA intake showed a similar reduction in fatal coronary heart disease to that found for fish or very long chain n-3 fatty acids (all about 45%). The evidence from this study suggests a benefit from consuming fish five times per week in this medium risk population. This benefit was attenuated by aspirin use and was comparable with the benefit associated with an increase in ALNA intake of 0.66 g per day or about 0.35% of calories. As noted, this is the strongest evidence to date of a benefit of fish in a medium risk population. Nonetheless, it is at least equally supportive of a benefit from ALNA and the baseline characteristics deviate markedly from the overall WHO/FAO recommendations, making extrapolation questionable.

The above results complement the results of the Health Professionals Study, presented by Ascherio et al. (1996). In the Nurses Study the main sources of ALNA were non-hydrogenated vegetable oils. In the Health Professionals Study the main source of ALNA was meat. Unsurprisingly, no beneficial effect of ALNA intake was observed without multivariate analysis adjusting for standard risk factors, fibre and total fat intake. The multivariate analysis indicated a relative risk of coronary heart disease of 0.41 for an increase in ALNA intake of 1% of calories. No association was found with fish intake in this cohort.

Earlier results from the MRFIT cohort presented by Dolecek (1992) showed inverse associations between both ALNA intake and intake of longer chain n-3 fatty acids and all-cause mortality. In both cases there was a reduction of about 30% in all-cause mortality in the upper quintile of intake. A cross-sectional study by Djousse et al. (2001) indicated a 40% reduction in coronary artery disease prevalence for those in the highest quintile of ALNA intake.

The only contrary result from a prospective study is that of Oomen et al. (2001). This study found ALNA intake in the Netherlands to be associated with increased incidence of coronary artery disease. However, the adverse association occurred only for ALNA from sources high in trans fats, which were the main contributors to ALNA consumption in the cohort studied.

The evidence from prospective cohort studies in medium risk populations, such as health professionals in the USA, strongly supports a benefit from ALNA consumption, particularly from non-hydrogenated vegetable oils. Of course, evidence from prospective studies can be overturned by stronger evidence from randomised intervention trials, but two randomised trials have already increased ALNA intake using vegetable oils with remarkably good results.

The spectacularly successful secondary prevention study of de Lorgeril et al. (1994, 1999) included an increase of ALNA consumption of about 0.55% of calories and showed a 65% reduction in cardiac deaths, a 40% reduction in unadjusted all-cause mortality and a 55% reduction in the multivariate-adjusted all-cause mortality. An analysis of serum fatty acids showed that ALNA was the only fatty acid associated with reduced risk after adjustments for standard risk factors, such as blood cholesterol, and for aspirin use. While other factors may have contributed to the reduced risk, the plasma fatty acid analysis points very clearly to a major role for sources of ALNA in the diet. The most likely additional factor to ALNA itself was the displacement of saturated fat by monounsaturated fat, as the ALNA was supplied by rapeseed (canola) oil margarine which displaced 85% of butter and cream use in the experimental group. A recommendation for increased consumption of rapeseed oils and other low cost oils with similar properties, such as mustard oil, would simultaneously increase ALNA and monounsaturated fat and displace saturated fat. The experimental group consumed 47 g per day of fish compared with 40 g per day in the control group, so this is unlikely to have had any effect on the outcome.

These results are corroborated by similar secondary prevention results in India reported by Singh (1997). In this study, 20 g per day of mustard oil, providing 2.9 g per day of ALNA, was provided to the experimental group. A parallel trial was carried out using 1.08 g per day of EPA from fish oil. 11.4% of the fish oil group died, compared with 13.3% of the mustard oil group and 22% of the placebo group. Non-fatal heart attacks were also reduced in both groups. Only the total cardiac event rate in the fish oil group was significantly different from the placebo group, though the results indicate a very similar benefit from both interventions. If the two intervention groups in the Singh study are combined, they show a reduction in mortality of 45% with a confidence interval of 30% to 90% (Bucher, 2002).

Apart from the fish oil trial by Singh, two other trials have reported significant effects of fish or fish oil interventions. Burr et al. (1989) reported a 30% reduction in mortality in an experimental group advised to eat fish. This was based on direct comparison between all groups who received the fish advice and all groups who did not receive this advice, regardless of what other advice the groups received (e.g. reduce fat or increase fibre). Comparison between the no-advice group and the fish-advice-only group indicated a 20% reduction in mortality. A recent report on the GISSI trial (Marchioli et al., 2001) indicated a 15% reduction in mortality when comparing all groups receiving 0.6 g per day of DHA and 0.3 g per day of EPA (derived from fish oil) with all groups not receiving fish oil. Comparison between the group receiving no intervention and the group receiving only fish oil indicated a 21% reduction in mortality. These results are similar to initial reports (GISSI, 1999) showing a 14% and a 20% reduction respectively.

The other intervention in the GISSI trial was Vitamin E. While the vitamin E effect was not statistically significant, it was associated with a 14% reduction in all-cause mortality when vitamin E alone was compared to no treatment. The combined effect of vitamin E and fish oil compared with the placebo group was a 20% reduction in mortality, notably less than the sum of the individual effects, being identical to fish oil alone and just 6% greater than vitamin E alone. This indicates a strong interaction between vitamin E and n-3 supplementation, suggesting that they are acting in part through a common pathway and that one can partially substitute for the other. These trials indicate a reduction in all-cause mortality, in individuals who have already had one heart attack, of 20-30% from fish consumption and 15-20% from fish oil consumption. This compares with a reduction in all-cause mortality of about 50% in the de Lorgeril and Singh trials using rapeseed oil and mustard oil respectively.

Mustard oil and rapeseed (canola) oil have a similar fatty acid profile: mostly monounsaturated, with polyunsaturated fats split about 2:1 between LA and ALNA. Both have variable erucic acid content, and other considerations favour the use of low-erucic-acid varieties. Increased use of these oils is entirely consistent with overall recommendations on fat consumption and presents no major cost, availability or cultural barriers. Flaxseed (linseed), which has a traditional use in many countries, provides a suitable alternative source of ALNA if it is ground shortly before consumption. Storage of the oil requires refrigeration and therefore flaxseed *oil* is a less satisfactory option on a world scale. Similar storage problems apply to fish and fish oil, with purified fish oil being a particularly expensive option. No such problems arise with rapeseed oil or mustard oil or unground flaxseed.

The case for increasing ALNA consumption to at least 1% of calories (in line with the WHO/FAO report's recommendations on overall n-3 consumption) is therefore very strong and can be implemented with minimal environmental burden and cost. In contrast, the case for consumption of fish in the context of a diet otherwise meeting the report's recommendations seems unsubstantiated from the point of view of both environmental feasibility and individual benefit.

## **Recommendations**

The draft report should be modified to acknowledge the lack of evidence that fish or fish oil provides any benefit which cannot be obtained from plant-based ALNA, or perhaps even from antioxidants. All recommendations currently specifically recommending fish should be amended to recommend fish or a plant source of ALNA on an equal basis. This would avoid the current unwarranted and culturally problematic implication that vegetarian diets are second rate for health.

In terms of the quality of evidence for a beneficial effect on cardiovascular health ALNA should be moved from the "probable" list to the "convincing" list since the evidence for ALNA is actually stronger than the evidence for some items already on the "convincing" list, such as potassium and fish.

Extending the provision of low trans fat margarines based on rapeseed oil should be added to the objectives for food policies for countries with a high historical use of butter. This allows the displacement of butter with maximum health benefit without requiring a cultural switch to the use of oils rather than semi-solid fats. The acceptability and effectiveness of this substitution has been demonstrated in the de Lorgeril trial and is the key to the practical implementation of the report's recommendations on fatty acid consumption in many cultures. Regulatory bodies should be encouraged to allow a strong health claim on such products.

A warning about the ill effects of methylmercury contamination of fish should be included in the report.

Finally, for those with a particular need for EPA and DHA due to impaired synthesis of very long chain fatty acids, the report should state that these can be obtained from microbial processes, avoiding cultural, ethical and ecological compromise.

## **References**

Ascherio et al. (1996): Dietary fat and risk of coronary heart disease in men: cohort follow up study in the United States, Alberto Ascherio et al., *British Medical Journal*, 1996; 313: 84-90

Bucher (2002): N-3 polyunsaturated fatty acids in coronary heart disease: a meta-analysis of randomized controlled trials, Heiner C Bucher et al., *American Journal of Medicine*, 2002; 112: 298-304

- Burr et al. (1989): Effects of changes in fat, fish and fibre intakes on death and myocardial reinfarction: Diet And Reinfarction Trial (DART), M L Burr et al., *The Lancet* ii, 1989, 757-761
- De Lorgeril et al. (1994): Mediterranean alpha-linolenic acid-rich diet in secondary prevention of coronary heart disease, Michel de Lorgeril et al., *The Lancet*, 1994; 343: 1454-1459
- De Lorgeril et al. (1999): Mediterranean diet, traditional risk factors, and the rate of cardiovascular complications after myocardial infarction: Final report of the Lyon Diet Heart study, Michel de Lorgeril et al., *Circulation*, 1999; 99: 779-785
- Djousse (2001): Relation between dietary linolenic acid and coronary artery disease in the National Heart, Lung, and Blood Institute Family Heart Study, Luc Djousse et al., *American Journal of Clinical Nutrition*, 2001; 74: 612-619
- Dolecek (1992): Epidemiological evidence of relationships between dietary polyunsaturated fatty acids and mortality in the Multiple Risk Factor Intervention Trial, Therese A Dolecek, *Proceedings of the Society for Experimental Biology and Medicine*, 1992; 200: 177-182
- GISSI (1999): Dietary prevention with n-3 polyunsaturated fatty acids and vitamin E after myocardial infarction: results of the GISSI-Prevenzione trial, GISSI-Prevenzione Investigators, *The Lancet*, 1999; 354: 447-445
- Hu et al. (2002), Fish and omega-3 fatty acid intake and risk of coronary heart disease in women, Frank B Hu et al., *Journal of the American Medical Association*, 2002; 287: 1815-1821
- Hu et al. (1999): Dietary intake of alpha-linolenic acid and risk of fatal ischemic heart disease among women, Frank B Hu et al., *American Journal of Clinical Nutrition*, 1999; 69: 870-877
- Marchioli et al. (2001): Efficacy of n-3 polyunsaturated fatty acids after myocardial infarction, results of the GISSI-Prevenzione trial, Roberto Marchioli et al., *Lipids*, 2001; 36:S119-S126
- Marckmann and Gronbaek (1999): Fish consumption and coronary heart disease mortality. A systematic review of prospective cohort studies, P Marckmann and M Gronbaek, *European Journal of Clinical Nutrition*, 1999; 53: 585-590
- Oomen (2001): Alpha-linolenic acid intake is not beneficially associated with 10-y risk of coronary artery disease incidence: the Zutphen Elderly Study, Claudia M Oomen et al., *American Journal of Clinical Nutrition*, 2001; 74: 457-463
- Pietinen et al. (1997): Intake of fatty acids and risk of coronary heart disease in a cohort of Finnish men, Pirjo Pietinen et al., *American Journal of Epidemiology*, 1997; 145: 876-886
- Rissanen et al. (2000): Fish oil-derived fatty acids, docosahexaenoic acid and docosapentaenoic acid, and the risk of acute coronary events, Tiina Rissanen et al., *Circulation*, 2000; 102: 2677-2679
- Salonen et al. (1995): Intake of mercury from fish, lipid peroxidation, and the risk of myocardial infarction and coronary, cardiovascular, and any death in Eastern Finnish men, Jukka Salonen et al., *Circulation*, 1995; 91: 645-655
- Singh (1997): Randomized, double-blind, placebo-controlled trial of fish oil and mustard oil in patients with suspected acute myocardial infarction: The Indian experiment of infarct survival – 4, Ram B Singh et al., *Cardiovascular Drugs and Therapy*, 1997; 11: 485-491
- Sorensen et al. (1999): Prenatal methylmercury exposure as a cardiovascular risk factor at seven years of age, N Sorensen et al., *Epidemiology*, 1999; 10: 370-375
- Walter Willett, *Eat, drink and be healthy*, 2001, ISBN 0-684-86337-5