COMMENTARY

The importance of omega-3 fatty acids in diet

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One of the most profound multifaceted challenges facing humankind into the next millennium is the need to ensure adequate quantity of but also of high quality food. This is rightly summed up by the German proverb, 'A man is what he eats'. Food provides not only the essential nutrients for life, but also bioactive compounds for prevention of diseases[1,2]. Food contains fatty acids in various forms such as triglycerides, phospholipids, glycolipids and cholesterol esters. There are mainly three types of fatty acids: (i) saturated fatty acids (SFA), (ii) monounsaturated fatty acids (MUFA) and (iii) polyunsaturated fatty acids (PUFA). The first two are synthesized endogenously, but the third one needs to be supplied exogenously. Over the recent years, fat has received a lot of bad publicity. But not all fat is bad; PUFA is the good fat. Modern nutritional theory is focusing on the numerous health benefits of maintaining sufficient levels of fatty acids and in particular, the very long chain PUFA (i.e. C20 and C22) that belong to the omega (ω)-3 family. PUFA have more than one double bond in their hydrocarbon backbone.

Importance of PUFA

Human physiology depends in various ways on PUFA, either as components of membrane phospholipids in specific tissues or as precursors of hormone-like compounds known as eicosanoids (e.g. prostaglandins)[3], which are vital for biological processes in the human body. They have a number of neutraceutical and pharmaceutical applications[4,5]. Eicosapentaenic acid (EPA, 20 : 5 ω3) and docosahexaenoic acid (DHA, 22 : 6 ω3) are the important ω3 PUFA, while arachidonic acid (AA, 20 : 4 ω6), is a vital ω6 PUFA. EPA and DHA are important in treatment of arthrosclerosis, cancer, rheumatoid arthritis, psoriasis and diseases of old age such as Alzheimer's and age-related macular degeneration[6,7]. AA and DHA are of special importance in the brain and blood vessels, and are considered essential for pre- and post-natal brain and retinal development[8]. Eicosanoids such as prostaglandins, prostacyclins and leukotrienes, derived from ω3 PUFA, are also important in new-born and infant development, modulatory vascular resistance and wound-healing[6,9]. PUFA are either directly available as components of the diet or produced from precursors; linoleic acid (LA, C18:2 ω6) and α-linolenic acid (ALA, C18:3 ω3)[10]. Fish oils are the major source of PUFA, and considerable evidence has indicated that ω3 PUFA in fish oils are actually derived via the marine food chain from zooplankton consuming ω3 PUFA-synthesizing microalgae[11]. LA and ALA are predominant in green vegetables and some plant oils. Although some research[12,13] has determined qualitatively that humans can convert the parent ALA to EPA and then to DHA, the most recent consensus is the degree of conversion is 'unreliable and unrestricted'[14].

Dietary aspects

The estimate from studies in Palaeolithic nutrition and modern-day hunter–gatherer populations indicates that humans evolved consuming a diet that was much lower in SFA than is today's diet[15]. Furthermore, the diet contained small and roughly equal amounts of ω6 and ω3 PUFA (ratio of 1–2 : 1)[15]. Recent fossil evidence indicates that the rapid expansion of our species ancestral archaic human brain took place in coastal areas, where aquatic food rich in PUFA, such as algae, molluscs, crustaceans and fish was abundant. Results of the intensive global research for over five decades support the conclusion that diet is the major environmental cause of atherosclerosis and cardiovascular diseases (CVD), especially in genetically susceptible indivivuals[16]. Interestingly, the vegetarian diet has been associated with reduction in many chronic diseases, including CVD[17]. A healthy vegetarian diet is characterized by more frequent consumption of fruits and vegetables, whole grains, legumes and nuts, resulting in higher intake of dietary fibre, antioxidants and phytochemicals compared with a nonvegetarian diet. A high-caloric diet, combined with limited physical activity, contributes to dyslipidaemia, insulin resistance, diabetes and obesity. These in turn increase the risk of CVD. Throughout the Asia-Pacific region CVD is a major cause of mortality, although rates differ from country to country[18]. Over the past few decades, there has been an epidemic of CVD and diabetes underway in India, with no signs of downturn[19]. The rates of CVD have decreased by 60% in the US and increased by 300% in India over the last three decades[20]. This rings alarm bells, as majority of Indians have a vegetarian diet. This increase in CVD is attributed to a form of contaminated vegetarianism[19]. Enas *et al.*[19] have extensively reviewed diets and nutrition for prevention of CVD and diabetes. The results of Bulliyya *et al.*[21] on the South Indian coastal population indicate that people who ate fish regularly appeared to have a better cardiovascular risk profile than did non-fish consumers, which is of public health significance. The association between ω3 fatty acids and CVD was established following the observation that the Greenland Inuit had low mortality from heart disease, despite a diet that is rich in fat[22].

The dangers of current Western diet and the contaminated vegetarian diet, and the benefits of prudent diet need to be disseminated among cardiologists, physicians and the public[20]. As mentioned earlier, the primary sources of these ω3 fatty acids are fish and seafood. For several reasons, our present reliance on marine products for PUFA is highly undesirable. Not all communities have ready access to fish supplies, either for geographic or economic reasons; some people are unable to consume fish owing to allergic reactions; some chose not to eat fish because of a vegetarian lifestyle. This could be overcome by consuming microalgae, which are the primary producers of PUFA. Single-cell oils derived from fermentation of microalgae PUFA have promising biotechnological market both for feed and food, e.g. infant milk formulas with enriched DHA, and hens fed with microalgae to produce 'OMEGA eggs' are already successful ventures in the Western world. Unlike seafood, microalgal oils are cholesterol-free and also might help the body in assimilation of PUFA from other dietary sources. However, at present their

cost of production is high. Crop plants could provide a much cheaper and larger supply, provided they can be genetically engineered to synthesize sufficiently high concentrations of PUFA. In recent years, sequences encoding virtually all enzyme activities involved in microsomal PUFA biosynthesis have been cloned[23]. The recent results[24,25] clearly demonstrate the potential to genetically engineer plants to produce PUFA; several significant hurdles remain to be overcome before plants can become commercially viable sources.

Conclusion

People eat specific foods because of their taste, easy availability and affordability, but are often unaware of the health benefits and risks. According to the references cited in Enas *et al.*[20], dietary modifications remain the cornerstone of both the treatment and prevention of diabetes and CVD, the twin epidemics of the twenty-first century. Compared with medical or surgical interventions, nutritional intervention is a low risk, low-cost and readily available option[26]. Better food habits can help reduce the risk of CVD, diabetes, stroke and death. A healthy eating plan means choosing the right foods to eat, and preparing them in a healthy way. There is increasing evidence that dietary and lifestyle modifications begun in childhood are likely to have benefits later in life[27]. Thus a healthy diet rich in ω3 fatty acids is beneficial for all age groups.

Update

The importance of microalgae as a source of PUFA has been reviewed recently[28]. Also, a recent review on metabolic engineering discusses the assemblage of multigene pathways in plants[29]. The indication of advancement in gene expression and metabolic engineering is further strengthened by Chen *et al.*[30]. In passing, as traditional sources of ω3 fatty acids are diminishing, exploring alternative sources is crucial.

1. Wylie-Rosett, J., *Circulation*, 2002, **105**, 2800–2804.
2. Liu, R. H., *Am. J. Clin. Nutr.* (*Suppl*), 2003, **78**, 517S–529S.
3. Jump, D. B., *J. Biol. Chem.*, 2002, **277**, 8755–8758.
4. Shahidi, F. and Wanasundara, U. N., *Trends Food Sci. Technol.*, 1998, **9**, 230–240.
5. Horrocks, L. A. and Yeo, Y. K., *Pharm. Res.*, 1999, **40**, 211–225.
6. Drevon, C. A., Baksaas, I. and Krokan, H. E. (eds), *Omega-3 Fatty Acids: Metabolism and Biological Effects*, Birkhauser Verlag, Basel, 1993, p. 389.
7. Simopoulos, A. P., Leaf, A. and Salem, Jr. N., *Ann. Nutr. Metab.*, 1999, **43**, 127–130.
8. Crawford, M. A., *Am. J. Clin. Nutr.*, 2000, **71**, 275–284.
9. Nettleton, A. J. (ed.), *Omega-3 Fatty Acids and Health*, Chapman and Hall, New York, 1995, p. 359.
10. Okuyama, H., Kobayashi, T. and Watanabe, S., *Prog. Lipid Res.*, 1996, **35**, 409–457.
11. Yongmanitchai, W. and Ward, O. P., *Process. Biochem.*, **24**, 117–125.
12. Carnielli, V. P., Wattimena, D. J. L., Luijendijk, I. H. T., Boerlage, A., Degenhart, H. J. and Sauer, P. J. J., *Pediatr. Res.*, 1999, **239**, 169–174.
13. Salem, N. Jr., Wegher, B., Mena, P. and Uauy, R., *Proc. Natl. Acad. Sci. USA*, 1996, **93**, 49–54.
14. Gerster, H., *Int. J. Vitam. Nutr. Res.*, 1998, **68**, 159–173.
15. Eaton, S. B. and Konner, M. N., *Engl. J. Med.*, 1996, **334**, 1557–1560.
16. Connor, W. E., *Am. J. Clin. Nutr.*, 1996, **64**, 253–254.
17. Thorogood, M., Roe, L., McPherson, K. and Mann, J. I., *Br. Med. J.*, 1985, **300**, 1271–1301.
18. Khor, G. L., *Asia Pac. J. Clin. Nutr.*, 1997, **6**, 122–142.
19. Enas, E. A., Senthilkumar, H., Chennikkara, H. and Bjurlin, M. A., *Indian Heart J.*, 2003, **55**, 310–338.
20. Enas, E. A., *J. Indian Med. Assoc.*, 2000, **98**, 694–695, 697–702.
21. Bulliyya, G., Reddy, P. C. and Reddanna, P., *Asia Pac. J. Clin. Nutr.*, 1999, **8**, 195–199.
22. Dyerberg, J., Bang, H. O. and Hjorne, N., *Am. J. Clin. Nutr.*, 1975, **28**, 958–966.
23. Napier J. A., Michaelson, L.V. and Stobart, A. K., *Curr. Opin. Plant Biol.*, 1999, **2**, 123–127.
24. Abbadi, A. *et al.*, *The Plant Cell*, 2004, **16**, 2734–2748.
25. Qi, B. *et al.*, *Nature Biotechnol.*, 2004, **22**, 739–749.
26. Mozaffarian, D., Kumanyika, S. K., Lemaitre, R. N., Olson, J. L., Burke, G. L. and Siscovick, D. S., *JAMA*, 2003, **289**, 1659–1666.
27. Deckelbaum, R. J. *et al.*, *Circulation*, 1999, **100**, 450–456.
28. Patil, V. *et al.*, *Curr. Topics Plant Biol.*, 2005 (in press).
29. Kinney, A. J., *Curr. Opin. Biotechnol.*, 2006 (in press).
30. Chen, R. *et al.*, *Plant Sci.*, 2006 (in press).

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