

The role of nutrition and exercise in the prevention and management of cardiovascular disease

PREVENTION AND MANAGEMENT OF CARDIOVASCULAR DISEASE THROUGH NUTRITION AND EXERCISE

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ABSTRACT

Cardiovascular disease, obesity, and type 2 diabetes are rapidly increasing worldwide (WHO, 2003). From a public health perspective, the concern is not only about the sudden increased rate of changes in the pattern of these diseases, but also the way they have started to appear earlier in life (Gortmaker et al, 1993). Furthermore, the shift seems to be much greater in countries of economic transition. In addition, with the change in the population profile and the increased number of elderly, the burden of cardiovascular disease and diabetes would be much greater in the years to come. Hence experts advocate the importance of applying a life course approach to the prevention and control of these diseases (Popkin, 2002 and WHO & FAO, 2003). Nutrition and physical activity are considered as well established major markers for prevention and management of cardiovascular disease, obesity and type 2 diabetes.

This paper focuses on the role of nutrition in relation to the emerging evidence such as inflammation, clotting, oxidative stress, the metabolic syndrome and the experiences of early life in the aetiology of cardiovascular disease. In addition, it will look at the physiological and psychological aspects of exercise in prevention and management of obesity and metabolic syndrome.

Finally, using the current evidence, this paper highlights the best current strategy for prevention and management of cardiovascular disease by examining the opportunities and challenges for implementing „population based“ versus „high risk group“ intervention programmes in relation to cardiovascular disease, obesity and type 2 diabetes.

Key words: Nutrition, Exercise, Coronary heart disease, Prevention

INTRODUCTION

Cardiovascular disease is multi-factorial (WHO 2003). The role of nutrition and exercise on those potentially reversible factors such as obesity particularly increased central body fat, physical inactivity, hypertension, hyperlipidaemia, hyperglycaemia, and type 2 diabetes are well known (Hubert, 1983; Dattilo, 1992; Anderson, 1998; Hu, 2000; WHO, 2000; NAO 2001; WHO & FAO, 2003 and Mann, 2005, Parikh et al, 2005).

While reviewing the role of food and nutrients, this paper concentrates on some of the emerging evidence on protective and risk factors for Coronary heart disease by revisiting the role of dietary fat, complex carbohydrate and glycemic index, fruit and vegetables, and the synergetic impacts of different nutrients in the food basket. The role of physical activity in heart health will focus on its impact on body weight, fat profile, blood pressure and psychological well being.

DIETARY FAT

Data from a number of epidemiological and experimental studies has well established the role of saturated dietary fat in increasing the risk of CHD through influencing low density lipoprotein and elevating cholesterol level. Poly and mono-unsaturated fat on the other hand are known to increase high density lipoprotein, and lower cholesterol level (*Mann 2005*). High density lipoprotein (HDL)-cholesterol which is considered to be protective to heart health, are found in greater quantity in women particularly during pre menopausal stage, physically active people and non obese.

Recent evidence suggest that based on the quantity and type of consumption, both saturated and polyunsaturated fatty acids can have an impact on other lipoprotein fractions (*Stanner 2005*). For example, high intakes of saturated fatty acids appear to be linked with an increase in High Density Lipoprotein (HDL), although the degree of this influence is much less pronounced than the increase in LDL. Also not all saturated fatty acids have the same impacts on lipid profile and increase LDL, for example in metabolic studies stearic acid did not show a cholesterol-raising effect compared with oleic acid. Among the cholesterol-raising saturated fatty acids, myristic acid is believed to be more potent than lauric acid or palmitic acid, although the data are not entirely consistent. Also, as Hu and colleagues (2001) very rightly argue, in a typical diet it is difficult to separate the impact of these individual fatty acids, as they tend to be concentrated on the same food sources such as beef and dietary products, so the collective effect rather than the individual impact of saturated fatty acids should be considered.

Similarly despite the protective effect of n-6 polyunsaturated essential fatty acids in reducing the LDL, high intake of n-6 PUFA (>10% total energy level) could reduce HDL as well as LDL.

The emerging evidence also suggests that changes in the fat composition of the diet not only influence the lipoprotein cholesterol, but also influence other physiological mechanism such as anti inflammatory, antiarrhythmic and antithrombotic effects. For example, increase in the consumption of PUFA in the last two decades, as a result of public awareness about impact of saturated fat on heart disease, resulted in higher consumption of vegetable oil and spread. The changes in the pattern of fat intake not only influenced ratio of n-6 to n3 PUFA, but also raised the following concerns:

a) poly-unsaturates compared to mono-unsaturates are more easily oxidized both during cooking, and food processing, as well as in the body, which has implication for protein and DNA damage, and effects heart through oxidative stress.

b) Trans Fatty Acids which are created through partial hydrogenation of poly-unsaturated fatty acids (PUFA) are even more atherogenic than Saturated Fatty Acids (SFAs), as it changes the plasma lipid profile by elevating LDL-cholesterol similar to SFAs, decreasing HDL-cholesterol, and increasing lipoprotein (a). The low-density lipoproteins appear to be atherogenic especially when the constituent lipid is oxidised (*Stanner, 2005*). Most Trans Fatty Acids from the diet derives from hard margarine, baked food, and deep fried fast food, and the main reason for partial hydrogenation of Poly-unsaturated fatty acids in food industry is to increase the life shelf of PUFs.

In addition, evidence from a number of epidemiological, and human intervention trials suggest that an increase in the ratio of n-6 PUFA to n-3 PUFA may have an adverse health outcome through decreased insulin sensitivity, and increased inflammatory mediators, which in turn may be associated with cardiovascular disease, diabetes and chronic inflammatory disorders (*Giugliano, et al, 2006*). Consumption of fish oil, oily fish, seeds and nuts especially walnuts and butternuts increases the omega 3 poly-unsaturated fatty acid in the diet and maintains the healthy balance of n6-PUFAs to n-3 PUF (*Food Standard Agency, 2005*). It is recommended that the ratio should not exceed 6:1 and the optimal ration should be 2:1

However, despite consistency of evidence in relation to lipoprotein response to manipulation of dietary fatty acids, Mann (2005) suggests that there is considerable individual variation which can not be explained by variation in the compliance of the dietary advice alone, hence the suggestion that the extent of the response to diet is influenced by many different genes.

Furthermore the „French Paradox“ and some prospective studies within Greek population (*Psaltopoulou, 2004, and Trichopoulou, 2005*) did not indicate increased risk of developing CHD despite high consumption of saturated fatty acids, nor demonstrate the protective role of n-6 poly-unsaturated fatty acid. This was suggested to be due to the synergetic effects of other protective factors including high consumption of fruit and vegetables, and red wine with meal, as well other psychosocial factors.

The above evidence therefore suggests that the influence of dietary fatty acids on lipid profile is complex; hence the importance of considering not only the type, amount, and the ratio of dietary fat consumed, but also the intake of other food and nutrients, genetic, socio-cultural and environmental and genetic factors. This approach has been advocated by Ashwell (2005) in her round table model of dietary and obesity factors in the prevention of coronary heart disease. Ashwell's model illustrates the combined role that genetics, and many environmental factors namely diet, obesity, activity and stress play on pathological and physiological risk factors such as adverse blood pressure, homocysteine, lipid oxidation, endothelial function, intravascular infection and inflammation, atherogenic lipid profile, insulin resistance, a pro-coagulant state, platelet aggregation and arrhythmia.

FRUIT AND VEGETABLES

The protective role of fruit and vegetables has been demonstrated through a number of epidemiological, observational studies and randomised control trials (*Eichholzer et al, 2001*). It is now believed that their beneficial effects are mediated through a number of different mechanisms and some of them still unknown and requires further knowledge in this field.

One of their main roles is through dietary antioxidants namely carotene, vitamin C, and E which neutralises free radicals that are capable of causing damage to cells. It is worth noting that data from experimental results indicate the protective effects of dietary antioxidants rather than vitamin supplements. Results from intervention trials, and observational studies on vitamin supplement is in consistent; in fact inverse relationship with vitamin E supplement in smokers has been demonstrated. This reinforces the synergetic effects of other components in fruits and vegetables. For example flavonoids found in onions, citrus fruits, apples, berries, cabbage, red grapes and broccoli are believed to reduce coronary heart disease risk by preventing the oxidation of LDL, reducing clotting, and improving coronary vasodilatation. More recently evidence suggests that lycopene, which is a key component of tomatoes; both fresh & processed, appears to be a potent antioxidant and a cancer growth inhibitor (*Rao, 2002*). Folate found in green leafy vegetables and citrus fruits and to a lesser extent vitamin B2, B6 and B12 are thought to reduce plasma levels of Homocysteine, an amino acid linked with atherosclerotic disease of coronary, cerebral and peripheral vessels (*Ward M, 2001*).

In addition, fruit and vegetables increase the level of potassium which results changes in the ratio of sodium to potassium. Through this mechanism, fruit and vegetables have protective effects on blood pressure and cardiovascular disease. Furthermore, it is possible that fruit and vegetable have other nutrient components which have not yet been identified (*Mann, 2005*).

CARBOHYDRATE AND GLYCIM INDEX

The role of complex carbohydrate, high fiber diet, and in particular the ratio of soluble to insoluble fibre on lipid profile has long been known. Recent evidence, however, suggests that interpretation of the protective role of whole grains due to high fibre contents may be an oversimplification (*Jacobs et al, 2000*), as refined grain foods consumed in large quantity could also increase fiber intake. Jacobs and colleagues (2000), suggest differentiation should therefore be made between nutrient-rich whole grain and plant based foods; legumes, fruit and vegetable and those that are nutrient-poor, due to refining or other processing. Apart from fibre, other biochemicals found in whole grains and plant based foods such as; minerals, vitamins, phenolic compounds and phytoestrogens have additional protective factors (*Slavin, et al, 1997*). This argument does not suggest that dietary fibre itself is not important in heart health as they do play a role in modulating the glycemic index (GI), and lowering serum cholesterol. It only reinforces once again the synergetic impacts of other nutrients within food basket. The glycemic index (GI) is a measure of the blood glucose response to intake of a particular carbohydrate. It offers the potential to rank carbohydrate based of their specific physiological effect on rise and fall of blood glucose concentration. The lower the GI leads to a slower release of blood glucose level. Since hyperglycaemia is a well known risk factor, Low GI food therefore, has a potential for health improvement particularly in relation to insulin resistance, and type 2 diabetes.

Furthermore, recent data from Hu and colleagues (2006) indicate that long term „consumption of high-glycemic index foods may lead to chronically high oxidative stress. A low-GI diet, but not a low-carbohydrate diet, appears to be beneficial in reducing oxidative stress“.

Although the current evidence from intervention studies on the role of a diet low in GI versus high in GI in managing weight loss is not convincing, Pawlak, and colleagues (2002) propose that obese patients should be counselled to follow a low glycemic index diet. The reasons for this suggestion, in addition to high fibre content of most low GI food, may be due to consideration of other beneficial effects of low GI food such as its slow release of insulin and impact on blood glucose concentration, lower oxidative stress which could prevent endothelial damage and activation of coagulation (Lefebvre, 1998). Concerns have been expressed about inconsistency of GI food in classification, a better and consistent classification of GI food, is therefore highly recommended.

NUTRITION EXPERIENCE IN EARLY LIFE

The Barker hypothesis of the fetal origins of degenerative disease has now been extensively researched and proved. It is now believed that the onset of adult chronic disease including blood pressure, type 2 diabetes, insulin resistance, and cardiovascular disease is linked with low birth weights (Barker and Osmond, 1986). Substantial amounts of data suggest that children who are born with low birth weight but become obese in the first year of life run more risk of developing chronic degenerative diseases (Barker et al, 1989, 2002, Fall et al 1995a). These findings strongly indicate that prevention should start even before the child is born, and therefore in order to improve the nation's health attention should be focused on nutritional status on women during preconception and conception stage.

EXERCISE

Exercise plays a major role in the prevention and management of cardiovascular disease through its impact both on physiological and psychological state. Its main physiological influences are on body weight, and body fat. Physical activity promotes fat loss; it has an impact both on the amount of body fat, and fat distribution, whilst preserves or increases lean mass. Higher body lean mass increases energy expenditure and hence contributes to the prevention and management of obesity. A change in the fat distribution reduces central body fat, which could improve insulin resistance, and prevent type 2 diabetes. In addition exercise has a positive impact on blood pressure and lipid profile through decreasing systolic and diastolic blood pressure, increasing HDL-cholesterol, decreasing LDL Cholesterol and triglycerides (DoH 2004a, Stanner, 2005).

Furthermore, regular exercise has psychological benefits and could improve mood state, self esteem, confidence, and prevent depression, anxiety, and stress level (Scully et al, 1998, Hassmen et al, 2000, and Stanner, 2005). The psychological well being from regular exercise could also have an impact on preventing binge drinking, comfort eating, and obesity, as well as promoting stress management. This evidence once again reinforces the combined physiological and psychological impact of exercise on dietary pattern, obesity, in particular central obesity which in turn could prevent and reduce metabolic syndrome, and cardiovascular disease. The current recommendation for adults is at least half an hour of moderate intensity activity on five or more days per week. For children and young people it is recommended at least one hour of moderate intensity physical activity each day (DoH, 2004a).

CURRENT STRATEGY FOR PREVENTION AND MANAGEMENT OF CARDIOVASCULAR DISEASE

In conclusion, evidence from current literature based on epidemiological and observational data, metabolic studies, and randomised control trials suggest that a combination of; smoking cessation, prevention of central obesity, a diet rich in fish, whole grains food, legumes, seeds and nuts, fruit and vegetables, frequent moderate exercise, and the avoidance of stress are still the best current strategy to combat against cardiovascular disease.

Although high risk population groups need special attention, the focus should be on population based intervention programme. This could only be achieved through an integrated approach by those responsible for healthcare, education, transport, sport and recreation, and the production, retailing, labelling & marketing of food (DoH, 2000a).

For encouraging the uptake of exercise, wide ranging and affordable sports and leisure opportunities at local neighbourhood level and reliable public transport should be available (DoH, 2004 b). Investment has to be made in physical education in school, provision of more sport facilities as well as protection of schools' playing fields. Creating a safer environment would encourage outdoor activities especially for young people.

In addition, availability of affordable healthy food, healthier and ethical food production, better food labelling and ethical food marketing, and developing cooking skills especially amongst young people and school children are some examples of the combined efforts on prevention and management of cardiovascular disease through food, nutrition, and physical activity

REFERENCES

1. Anderson, R. E.; Crespo, C. J.; Bartlett, S. J.; Cheskin, L. J.; Pratt, M. (1998) Relationship of physical activity and television watching with bodyweight and level of fatness among children: results from the Third National Health and Nutrition Examination Survey. *Journal of the American Medical Association*, 279:938-942.
2. Ashwell, M. (2005) Prevention of Heart Disease- The search for the Holy Grail, Putting the dietary approach into context. *Proceeding of the 6th National Nutrition & Health Conference*, PP 27, London.
3. Barker, D. J. P, Osmond C (1986) Infant mortality, childhood nutrition, and Ischaemic heart disease in englant and wales. *Lancet*, i,1077-81.
4. Barker, D. J. P, Osmond,C., Winter PDW, Margetts,B., Simmonds, S.J. (1989) Weight in infancy and death from ischaemic heart disease. *Lancet*, ii, 5-80.
5. Barker, D. J. P, Eriks, I.G., Forsen, T, Osmond,C. (2002) Fetal Origins of Adult Disease: Strenght of effects and biological basis. *International Journal of Epidemiology*, 31, 1235-9.
6. Dattilo, A. M. and Kris-Etherton, P. M. (1992) Effects of weight reduction on blood lipids and lipoproteins: a meta analysis. *American Journal of Clinical Nutrition*, 56:320-328.
7. Department of Health (2000 a) National Service Framework for Coronary Heart Disease. The Stationery Office, London.
8. Department of Health (2004 a) At Least Five a Week: Evidence on the impact of physical activity and its relationship to health. Department of Health, London.
9. Department of Health (2004 b) Choosing Health: Making healthier choices easier. The Stationery Office, London.
10. Eichholzer M, Luthy J, Gutzwiller F, Stahelin HB.(2001) The role of folate, antioxidant vitamins and other constituents in fruit and vegetables in the prevention of cardiovascular disease: the epidemiological evidence. *International Journal for Vitamin and Nutrition Research*; 71,1:5-17.
11. Fall, C. H.D., Vijayakumar,M., Barker, D.J., Osmond, C., Duggleby, S. (1995a) weight in infancy and prevalence of coronary heart disease in adult life. *British medical journal*,310, 17-20.
12. Food Standard Agency (2005). Impact of changes in the dietary n-6:n-3 polyunsaturated fatty acid (PUFA) ratio on risk factors for disease. <http://www.foodstandards.gov.uk/science/research/researchinfo/nutritionresearch/dietandcardiovasc/n02programme/n02projilist/n02026/n02026r>. Accessed 17th Oct 06.
13. Giugliano, D.; Ceriello, A. and Esposito, K. (2006), The Effects of Diet on Inflammation: Emphasis on the Metabolic Syndrome *J Am Coll Cardiol*, 2006; 48:677-685.
14. Gortmaker, S. L., Must, A., Perrin, J.M., Sobol, A.M., Dietz, W.H. (1993). Social and economic consequences of overweight in adolescence and young adulthood. *New England Journal of Medicine*, 329:1008-1012.
15. Hassmen P, Koivula N, Uutela A.(2000) . Physical exercise and psychological well-being: a population study in Finland. *Prev Med.*;30(1):17-25.
16. Hubert, H. B.; Feinleib, M.; McNamara, P. M., Castelli, W. P. (1983). Obesity as an independent risk factor for cardiovascular disease: a 26-year follow-up of participants in the Framingham Heart Study. *Circulation*, 67:968-977.
17. Hu, F.B., Manson, J. E., and Willett, W.C. (2001) Types of Dietary Fat and Risk of Coronary Heart Disease: A Critical Review. *Journal of the American College of Nutrition*, Vol. 20, No. 1, 5-19.
18. Hu, F. B.; Rimm, E. B., Stampfer, M.J., Ascherio, A., Spiegelman, D., Willett, W.C. (2000). Prospective study of major dietary patterns and risk of coronary heart disease in man. *American Journal of Clinical Nutrition*, 72:912-921.

19. Hu, y., Block, G., Norkus E.P., Morrow, J.D, Dietrich, M. and Hudes, M. (2006) Relations of glycemic index and glycemic load with plasma oxidative stress markers. *American Journal of Clinical Nutrition*, Vol. 84, No. 1, 70-76.
20. Jacobs, D.R; Pereira, M.A; Meyer, M.A; and Kushi, L.H (2000) Fiber from Whole Grains, but not Refined Grains, Is Inversely Associated with All-Cause Mortality in Older Women: The Iowa Women's Health Study. *Journal of the American College of Nutrition*, Vol. 19, No. 90003, 3265-3305.
21. Lefebvre PJ, Scheen AJ. The postprandial state and risk of cardiovascular disease *Diabet Med* 1998;15:S63-S68.
22. National Audit Office (2001). Tackling obesity in England. Report by the Comptroller and Auditor General HC220, pp. 1-65. The Stationery Office.
23. Mann, J. (2005) Cardiovascular Disease, PP 363-378 in *Human Nutrition*, Geissler,C and Powers, H. (Ed), Elsevier Publishing, London.
24. Parikh, P, McDaniel, M.C., Ashen, M.D. (2005) Diets and cardiovascular disease: an evidence-based assessment. *J Am Coll Cardiol* ;45:1379-87.
25. Psaltopoulou,T., Naska, A., Orfanos, P., Trichopoulos, D., Mountokalakis, T., Trichopoulou, A. (2004) Olive oil, the Mediterranean diet, and arterial blood pressure: the Greek European Prospective Investigation into Cancer and Nutrition (EPIC) study. *American Journal of Clinical Nutrition*, Vol. 80, No. 4, 1012-1018.
26. Pawlak, D.B., Ebbeling, C.D., Ludwig,D.S. (2002) Should Obese patients be counselled to follow a low-glycemic index diet? Yes. *Obesity Reviews*, 3, 325-43.
27. Popkin, B.M. (2002). „An overview on the nutrition transition and its health implications: The Bellagio meeting“. *Public Health Nutrition* 5(1A):93-103.
28. Rao, A.V. (2002) Lycopene, Tomatoes, and the Prevention of Coronary Heart Disease. *Experimental Biology and Medicine* 227:908-913.
29. Scully, D; Kremer, J.; Meade, M.M.; Graham, R, and Dudgeon, K (1998) Physical exercise and psychological well being: a critical review. *British Journal of Sports Medicine*, Vol 32, Issue 2 111-120.
30. Slavin, J., Jacobs, D.R, Marquart, L. (1997) Whole-grain consumption and chronic disease: protective mechanisms. *Nutr Cancer* 27: 14-21.
31. Stanner, S (Ed) (2005) Cardiovascular Disease: Diet, nutrition and Emerging. British Nutrition Foundation, Blackwell, London.
32. Trichopoulou, A., Naska, A., Orfanos, P., Trichopoulos, D. (2005) Mediterranean diet in relation to body mass index and waist-to-hip ratio: the Greek European Prospective Investigation into Cancer and Nutrition Study. *Am J Clin Nutr*;82(5):935-40.
33. Stanner, S. (ed) (2005) Cardiovascular Disease: Diet, Nutrition and Emerging Risk Factors. The report of British Foundation Task Force. Blackwell Publishing, London.
34. Ward,M (2001) Homocysteine, folate, and cardiovascular disease. *International Journal for Vitamin and Nutrition Research*; 71(3):173-8.
35. World Health Organisation (2000). Obesity: preventing and managing the global epidemic.
36. WHO Technical Report Series, 894, Geneva.
37. WHO and FAO (2003) Diet, Nutrition and The Prevention of Chronic Disease. WHO
38. Technical Report Series, 916, Report of a Joint WHO and FAO Expert Consultation, Geneva.