

Review Article

Dietary fat and the prevention of chronic disease

Mark L Wahlqvist

Director, Asia Pacific Health & Nutrition Centre, Monash Asia Institute, Monash University, Melbourne, Australia, Chair, Nutrition Committee, Australian Academy of Science

Chronic diseases are generally taken to include obesity (especially abdominal), diabetes, macrovascular disease (MVD), affecting all medium distributing arteries and the organs they supply, osteoporosis, and various cancers (notably breast, lung, colorectal, pancreatic, prostate and skin) and dementia. Unfortunately, they may not be so chronic, as their consequences for morbidity and mortality may occur early in adult life and proceed rapidly. Since they all, in one way or another, have food, nutritional and other environmental and lifestyle contributions, the term Eco-Nutritional disease may be preferred. Insofar as the nutritional basis of chronic disease is concerned, we may simply speak of nutritionally-related disorders or diseases (NRD). In regard to fat and END or NRD, the key considerations are how diverse the sources are and what it does to energy density (ED) and nutrient density (ND). These are reflected in the 2003 WHO report 9816 on "Diet, Nutrition and the Prevention of Chronic Disease".

Key Words: fat diversity or variety, trans fatty acids, ED (energy density), ND (nutrient density), NRD (nutritionally-related disorders)

Introduction

A recent effort to increase the understanding of the nutritional pathogenesis and preventability of the so-called Chronic Disease has been the WHO Technical Report 916, entitled "Diet, Nutrition and the Preventability of Chronic Diseases". Nutrition is one of a host of lifestyle considerations, including physical activity, social and environmental factors and substance abuse, and these may operate across the whole life-cycle, requiring an integrated approach to prevention. The Report took a rather disease-specific approach, which endeavours to bring this together for optimal general health and longevity.

There is much in the Report about preferred macro- and micro-nutrient intakes in relation to chronic disease. These observations need to be couched in terms of other WHO (and FAO) recommendations about FBDGs (Food Based Dietary Guidelines) which always emphasize the overriding importance of food diversity, as well as energy balance. For the Western Pacific (and elsewhere) diverse sources of macro-nutrients (carbohydrate, fat and protein) are recommended. The 2003 Technical Report also encourages the consumption of nutrient dense (nutritious) food which requires all dietary components including a spectrum of phytochemicals to be considered in relation to health. For fat, it can be said, that the collective position of various recent WHO reports would be:

- (1) To obtain fat from a diversity of sources and obtain the fat relatively unrefined (from seed, nuts, grains, fruits where the source is plant) and from lean or low-fat animal sources).
- (2) Keep the FER (fat energy ratio) between 15 and 35%, the upper end requiring high levels of physical activity.
- (3) Keep trans fatty acid intake to less than 1% of daily-energy intake.

- (4) Have fat as part of a varied diet which provides health protection through its wide range of active components acting synergistically and collectively.

In the preparatory workshop to the WHO Expert Consultation in Vienna, 25-26th August 2001, the value of understanding the eco-nutritional basis of chronic disease was explored. This develops the arguments for food variety and the need to underpin nutritional guidelines with bio-diversity and sustainability. Fat, and especially palm fruit and its oil, can be produced for human consumption in ways that support the WHO position.

Dietary fat

The concept of dietary fat has evolved substantially in recent times. The principal revisions in thinking and practice have been:

- (1) Its contribution to energy density (ED), measured in calories or kilojoules/unit mass, as the principal food component increasing ED and the role of ED in human energy balance.
- (2) The recognition that not all types of fat can have equivalent energy values (Atwater factors) because of the various non-fuel roles of fat (receptor function; cell regulatory eicosonoids depending on fatty acid composition; differential digestion, absorption and handling in the portal circulation depending on triglyceride isomer and chain length of fatty acid).
- (3) The role of companion compounds, notably phytochemicals, in the modulation of fat physiology and

Correspondence address: Professor Mark L. Wahlqvist, Asia Pacific Health & Nutrition Centre, Monash Asia Institute, 8th Floor, Menzies Building, Monash University, Vic 3800, Australia. Tel : 61-3-99058145 ; Fax. 61-3-9905 8146
Email: mark.wahlqvist@adm.monash.edu.au

Accepted 30th June 2005

pathophysiology (eg. lipoprotein oxidation; cholesterol synthesis). On this basis, it is worth differentiating refined from unrefined fat.

- (4) The effects of hydrogenation on the trans fatty acid content of dietary fat
- (5) Its contribution to energy density (ED), measured in calories or kilojoules/unit mass, as the principal food component increasing ED and the role of ED in human energy balance.
- (6) The recognition that not all types of fat can have equivalent energy values (Atwater factors) because of the various non-fuel roles of fat (receptor function; cell regulatory eicosonoids depending on fatty acid composition; differential digestion, absorption and handling in the portal circulation depending on triglyceride isomer and chain length of fatty acid).
- (7) The role of companion compounds, notably phytochemicals, in the modulation of fat physiology and patho-physiology (eg. lipoprotein oxidation; cholesterol synthesis). On this basis, it is worth differentiating refined from unrefined fat.
- (8) The effects of hydrogenation on the trans fatty acid content of dietary fat.
- (9) The differential chemistry and properties of fat which depend on animal or plant source, and whether the animal is ruminant or non-ruminant
- (10) The effects of traditional and novel food processing on fat quality¹ eg. fermentation, cooking, interesterification²
- (11) Plant breeding (eg. canola as a rapeseed cultivar)

Taking these revisions into account, it becomes helpful to categorize fats accordingly by degree of refinement and source (Table 1). It is worth noting that it is more helpful to consider plant sources of fat in regard to whether this is seed (eg. rapeseed, canola, linseed, corn, safflower, sunflower), nut (eg. ground or tree) or fruit (eg. avocado, olive, palm, cocoa, coconut) rather than the undifferentiated descriptor "vegetable oil or fat".

Table 1. Dietary fat

1. Unrefined			
	Plant		Animal
(i)	Seeds	(i)	Fish
(ii)	Nuts	(ii)	Lean meat
(iii)	Fruits	(iii)	Milk and milk products (Cheese and Yoghurt)
	• Avocado		Fermented milk
	• Olive		
	• Palm		
	• Cocoa		
	• Coconut		
and "unrefined" derivatives, eg. Red Palm Oil Virgin Olive Oil			
2. Refined			
	Plant		Animal
	• Oils		• Lard
	• Solid		• Dripping
	• Hydrogenated		• Butter
	• Non-hydrogenated		

The reasons for this are that the basic food commodity is more indicative of the nutritionally relevant characteristics, and with new knowledge, the consumer is more confident about origin and usage.

Chronic disease

The term "Chronic Disease" is a term which has been applied to an increasing array of age-related diseases (Table 2), initially seen in more affluent economies, but which are now affecting to a greater and greater extent the relatively *less socio-economically advantaged peoples* in these and other economies.

Table 2. Burden of chronic disease

The Burden of Chronic Disease is mainly comprised of:	
•	Malnutrition (which may extend from conception to old age)
•	Immuno-deficiency and Recurrent Infection
•	Cardiovascular Disease (CVD)
-	Coronary Heart Disease
-	Cerebrovascular Disease
-	Peripheral Vascular Disease
•	Neoplastic Disease
•	Metabolic, especially cumulative positive energy balance
-	over-fatness with insulin resistance (obesity, impaired fasting glycaemia and diabetes)
•	Loco-motor Disease
-	osteoporosis and fracture
-	sarcopenia (reduced skeletal muscle mass)
-	arthritis
•	CNS Disease
-	mood
-	cognition
-	stroke
•	Dental Disease and Oral Health

Moreover, the descriptor is often qualified by "Non-Communicable" to contrast these diseases with Infectious Diseases which are often acute in onset and short in duration, whether because they are lethal or self-limiting. However, there are now indications that some CDs have *infective components* (eg. obesity, macrovascular disease).³ Or, they are, at least, *inflammatory diseases*. The corollary is that the most chronic of disease complexes can be either PEM (Protein-Energy Malnutrition) and/or *immunodeficiency* with recurrent infection. For these reasons, a more satisfactory nomenclature for chronic disease is required which has to do with aetiology and pathogenesis; as well as with the clustering of these diseases.

The basis of chronic disease

One of the most instructive findings about chronic disease in recent years has been that its origin may be *as early as conception*, or even in the environmental exposures of previous generations.^{3,4} Whereas we attributed some of this phenomenology to genes, now we know that more and more is due to environmentally-altered gene expression, notably maternal exposure, but effects on spermatogenous, through paternal exposure, are also under scrutiny. There is evidence that virtually every CD can be influenced by *dietary factors* at the level of genetic

mutation, gene expression, nutritional pathways, energy balance or factors which confer or decrease resilience and protection against disease.⁵ We have also grossly underestimated how physiological states and their perturbation (eg. the menopause, cognition, mood) can depend on dietary intakes.^{6,7}

Regrettably, *biological adjustment made to cope with undernutrition*, especially in pregnancy, can have life-long and intergenerational effects on health, as nutritional circumstances become less limiting, particularly with the so-called "affluent diet". This has contributed to "transitional health problems" with unexpectedly high burdens of CD in transitional economies⁸ and to the "double burden of disease" where so-called "undernutrition" and "overnutrition" co-exist (eg. protein and micronutrient deficiencies, along with abdominal obesity, diabetes and MVD (macrovascular disease) in the same individual, in families and in communities.⁹ These observations lead to the conclusion that not only the quantity and the availability of food, but *the quality of the human diet at various stages in the life cycle* are critical.¹⁰

Dietary fat has the potential to address food quantity and distribution, but requires more active consideration of how, either as unrefined fat, or as a promoter of nutrient dense (nutritious) food intakes it can serve the quality need. An approach to the latter will be for it to be provided in small quantities combined with nutritious plant or animal derived (especially low cholesterol) food (eg. lentils, or fish). The now established *Science of Food Variety and Health* supports this approach.¹¹⁻¹⁴

An eco-nutritional view of chronic disease

As dietary guidelines emerged in the mid-late 1970s to address the emerging problems of chronic disease, two guidelines stood out as the most consistent and significant in the growing number of national recommendations. They were to encourage breast feeding and to have a *variety of foods*. This in turn spoke to the importance of maternal (and family) health and well-being and to the need for *biodiversity* to underpin the required food variety. With time, more and more evidence has underscored the case for these two recommendations - that to do with the fetal and neonatal origin of disease¹⁵⁻¹⁹ and the case for an eco-nutritional approach to disease prevention.^{20,21}

Put simply, the way we connect with our environment, socially, physically, nutritionally and emotionally, and how sustainable our environment is are *the major determinants of our health*. The best of food cultures are not ones with a narrow range of foods and a staple, but ones where there is food diversity, food adequacy, and regular physical activity, so that we can eat enough without becoming fat, and where food plays a social and festive role.²²⁻²⁵ This understanding is becoming clearer with the growing conjunction between the food, nutritional, health and environmental sciences. It also requires a shared interest in health and economic development by policy makers, the private sector and communities. This contextual and integrated approach is in evidence in the WHO Technical Report 916, "Diet, Nutrition and Chronic Diseases in Context" (Chapter 4, pp 30-53). When embraced, matters like dietary fat and health fall into place.

Dietary fat and chronic disease

The value of the WHO report in relation to the understanding and application of dietary fat science and technology to human health and disease prevention is that it goes beyond the usual analysis of CVD (Cardiovascular Disease) or MVD (Macrovascular Disease) alone, sometimes with obesity, diabetes and cancer as concerns, to a much more *comprehensive health view*. This strengthens the case for a general approach to dietary fat intake because, in aggregate, one can be most confident especially if the story is consistent.

The specific recommendations about fat in the Report are:

- (i) For dietary fat to range between 15 and 30% daily energy intake (DEI)
- (ii) That there be not more than 10% DEI from saturated fat; PUFA (polyunsaturated fatty acid) in the range 6-10% of DEI with n-6 PUFA 5-8% and n-3 PUFA 1-2% DEI; less than 1% of DEI as trans fatty acid; and the remainder from monounsaturated oleic acid.

However, the consistent emphasis throughout the report on food variety requires *that dietary fat also came from a variety of sources*. And since the companion compounds in plant fats, the phytonutrients, have such important health protective roles, dietary fats and oil should be as unrefined as practical.

Dietary fat and FBDGs (Food Based Dietary Guidelines)

WHO and FAO promulgated, in the Cyprus report of 1995²⁵ a Food Based approach to Dietary Guidelines, further developed for various regions.²⁷ Food Based Dietary Guidelines acknowledge the importance of incorporating the best available nutrition science and evidence into identifiable food cultural settings for health advancement. This is now progressing at the community, national and international levels. Such guidelines encourage both food adequacy and the use of quality food and food patterns which are safe and nutritious (generally nutrient-dense and low in energy density). It is highly desirable that recommendations about dietary fat and chronic disease are framed in accordance with these guidelines.

The 2003 WHO Report 916 on "Diet, Nutrition and the Prevention of Chronic Disease"

The report acknowledges both the *dietary energy* (and, therefore, fat) *deficit* for many people world-wide in economically disadvantaged communities, whilst preventing the problem of excess energy (including fat) intake over energy expenditure for growing numbers of people, leading, amongst other factors, to Chronic Disease.

The dilemma is to identify *safe, adequate and health protective ranges of fat intake*, fat quality and food patterns in which such fat can be eaten. Quite reasonable and flexible approaches are formulated in the Report. Over and over again, the report stresses the *importance of regular physical activity* as the best way to ensure that food intake is appropriate to our energy needs and, in turn, *essential nutrient and protective food component (phytonutrient) needs*.

It also argues for *healthy and sustainable environments*. Whilst the report suffers from a disease specific

approach, it endeavours to bring the nutritional analysis to the entire so-called "chronic disease" agenda, since patterns of disease tend to accompany patterns of food intake, physical activity (or inactivity) and substance abuse (alcohol and cigarette smoking in particular). Inevitably, for fat, much is said about *dietary fatty acid patterns*, how saturated monounsaturated or polyunsaturated, how much n-6 and n-3, what chain length, how much trans fatty acid (it is unrealistic to have zero tolerance in a mixed diet and probably not necessary since not all trans are the same or as hazardous).

The concomitant intake of fatty acids, in various esterified forms, with other fats (like cholesterol), other food components and various food is understood to modulate health risk. In particular, in a low fat diet, more cholesterol can be eaten without adverse effect on blood cholesterol. Some of these *cholesterol sources* (like eggs) are highly nutritious and desirable, especially for older people with limited energy expenditure and intake. It is of interest that the excess of haemorrhagic stroke in Asian populations appears related to low dietary cholesterol.²⁸ But blood cholesterol is not the only nutritional pathway to MVD, let alone other health outcomes and so a broader view of diet and cuisine is required to sub-serve optimal health.

The collective WHO position - a distillation

This might be summarized as follows:

- (i) To obtain fat from a diversity of sources and obtain the fat relatively unrefined (from seed, nuts, grains, fruits where the source is plant) and from lean or low-fat animal sources)^{11-14, 27,29}
- (ii) Keep the FER (fat energy ratio) between 15 and 35%, the upper end requiring high levels of physical activity³⁰⁻³²
- (iii) Keep trans fatty acid intake to less than 1% of daily-energy intake^{33,34}
- (iv) Have fat as part of a varied diet which provides health protection through its wide range of active components acting synergistically and collectively¹¹⁻¹⁴

Table 3. Response of Palm Oil Industry to WHO Technical Report Series: 916

- | |
|--|
| 1. Emphasize palm fruit as Reference Point for Palm Oil |
| 2. Promote RPO (Red Palm Oil) and its products |
| 3. Blend various <ul style="list-style-type: none"> • Fatty commodities • Oils • Fats |
| 4. Encourage fat in a varied diet |
| 5. Articulate role of fat in promoting plant food intake |
| 6. Promote non-hydrogenated solid fats (to decrease trans fatty acids) |

An appropriate response for the food industry to the WHO report

Food producers, processors and retailers, severally and collectively, must scrutinize and operationalize WHO Report 916. As an example, the Palm Oil Industry response could be along the lines outlined in Table 3. Industries which respond constructively to the WHO Report will enjoy greater community, governmental and international agency support than those who do not.

References

1. Clugston G, Lupien JR, Savige GS, Winarno FG (eds). Novel Foods in Nutrition Health and Development: Benefits, Risks and Communication. Metung, Australia, 11-14 November 2001. Asia Pac J Clin Nutr 2002; Vol 11 (suppl): S97-S230.
2. Kasai M, Nosaka N, Maki H, Negishi S, Aoyama T, Nakamura M, Suzuki Y, Tsuji H, Uto H, Okazaki M and Kondo K. Effect of dietary medium- and long-chain triacylglycerols (MLCT) on accumulation of body fat in healthy humans. Asia Pac J Clin Nutr 2003; 12 (2): 151-160)
3. Wahlqvist ML. Chronic disease prevention: A life-cycle approach which takes account of the environmental impact and opportunities of food, nutrition and public health policies - the rationale for an eco-nutritional disease nomenclature. Asia Pac J Clin Nutr 2002; 11 (Suppl 9):S759-S762.
4. Barker DJP, Forsen T, Uutela A, Osmond C, Eriksson JG. Size at birth and resilience to effects of poor living conditions in adult life: longitudinal study. BMJ 2001; 323: 1273-1276.
5. World Health Organization (Member of Joint Expert Consultation: ML Wahlqvist). Diet, nutrition and the prevention of chronic diseases. Report of a Joint WHO/FAO Expert Consultation. WHO Technical Report Series 916. Geneva: World Health Organisation, 2003.
6. Wahlqvist ML & Dalais F. Phytoestrogens - the emerging multi faceted plant compounds. [Editorial]. Med J Aust 1997; 167 (3): 119-120.
7. Wilcox G, Wahlqvist ML, Burger HG & Medley G. Oestrogenic effects of plant-derived foods in post-menopausal women. BMJ 1990; 301: 905-906.
8. Popkin BM. Nutrition in transition: the changing global nutrition challenge. Asia Pac J Clin Nutr 2001; 10 (suppl): S13-18.
9. Gillespie S and Haddad L. Attacking the Double Burden of Malnutrition in Asia and the Pacific. Asian Development Bank, Manila, Philippines and International Food Policy Research Institute, Washington DC, 2001.
10. Haveman-Nies A, de Groot L © PGM, Burema J, Amorim Cruz JA, Osler M, van Staveren WA. Dietary quality and lifestyle factors in relation to 10-year mortality in older Europeans. The SENECA study. Am J Epidemiol 2002; 156:962-968.
11. Kant AK, Schatzkin A, Harris TB, Ziegler RG, Block G. Dietary diversity and subsequent mortality in the First National Health and Nutrition Examination Survey Epidemiologic Follow-up Study. Am J Clin Nutr 1993; 57:434-440.
12. Kant AK, Schatzkin A, Ziegler RG. Dietary diversity and subsequent cause-specific mortality in the NHANES I epidemiologic follow-up study. J Am Coll Nutr 1995; 14:233-238.
13. Hodgson JM, Hsu-Hage BH-H, Wahlqvist ML. Food variety as a quantitative descriptor of food intake. Ecology of food and nutrition 1994; 32:137-148.

14. Savige G, Hsu-Hage B and Wahlqvist ML. Food variety as nutritional therapy. *Current Therapeutics*, March 1997; 57-67.
15. Barker DJ. Maternal nutrition and cardiovascular disease. *Nutr Health* 1993; 9 (2): 99-106.
16. Barker DJ, Osmond C, Simmonds SJ, Wield GA. The relation of small head circumference and thinness at birth to death from cardiovascular disease in adult life. *BMJ* 1993; 306 (6875): 422-426.
17. Barker DJ, Martyn CN, Osmond C, Hales CN, Fall CH. Growth in utero and serum cholesterol concentrations in adult life. *BMJ* 1993; 307 (6918): 1524-27.
18. Barker DJ. The intrauterine origins of cardiovascular disease. *Acta Paediatr Suppl* 1993; 82 suppl 391:93-99.
19. Osmond C, Barker DJ, Winter PD, Fall CH, Simmonds SJ. Early growth and death from cardiovascular disease in women. *BMJ* 1993; 307 (6918): 1519-24.
20. Wahlqvist ML, Specht RL. Food variety and biodiversity: Econutrition. *Asia Pac J Clin Nutr* 1998; 7 (3/4):314-31.
21. Wahlqvist ML. Eco-nutritional disease or nutrition and chronic disease. (Foreword). *Asia Pac J Clin Nutr* 2002; 11 (Suppl 9):S753-754.
22. Sho H. History and characteristics of Okinawan longevity food. *Asia Pac J Clin Nutr* 2001; 10(2):159-164.
23. Wahlqvist ML, Worsley A & Lukito W. Evidence-based nutrition and epidemiology in the Asia-Pacific region. *Asia Pac J Clin Nutr* 2001; 10 (2): 72-75.
24. Wahlqvist ML. Nutrition and diabetes in the Asia-Pacific region with reference to cardiovascular disease. *Asia Pac J Clin Nutr* 2001; 10 (2): 90-96.
25. Wahlqvist ML, Yamori Y. Summary and recommendations. The Okinawan Round-table on Nutritional Cardiovascular Disease. *Asia Pac J Clin Nutr* 2001; 10 (2): 172.
26. World Health Organization (Member of Working Party: ML Wahlqvist). Preparation and use of food-based dietary guidelines. Report of a Joint FAO/WHO Consultation (1995: Nicosia, Cyprus). Geneva: World Health Organization, 1998.
27. World Health Organization (Chair, Working Party: ML Wahlqvist). Food-based dietary guidelines for the Western Pacific: Nutrition in transition. World Health Organization, 1997.
28. Eastern Stroke and Coronary heart Disease Collaborative Research Group. Blood pressure, cholesterol, and stroke in eastern Asia. *Lancet* 1998; 352:1801-1807.
29. de Lorgeril M, Salen P, Martin J-L, Monjaud I, Delaye J and Mamelle N. Mediterranean diet, traditional risk factors, and the rate of cardiovascular complications after myocardial infarction. Final report of the Lyon Diet Heart Study. *Circulation* 1999; 99: 779-785.
30. Fries JF. Physical activity, the compression of morbidity, and the health of the elderly. *J Royal Society Med* 1996; 89: 64-68.
31. Gregg EW, Gerzoff RB, Caspersen CJ, Williamson DF, Venkat Narayan KM. Relationship of walking to mortality among US adults with diabetes. *Archives of Internal Medicine* 2003; 163 (12): 1440-1447.
32. Hakim AA, Petrovitch H, Burchfiel CM, et al. Effects of walking on mortality among nonsmoking retired men. *N Engl J Med* 1998; 338:94-99.
33. Salmerón, Hu FB, Manson JE, Stampfer MJ, Colditz GA, Rimm EB and Willett WC. Dietary fat intake and risk of type 2 diabetes in women. *Am J Clin Nutr* 2001; 73 (6): 1019-1026.
34. Oomen CM, Ocke MC, Feskens EJ, van Erp-Baart MA, Kok FJ, Kromhout D. Association between trans fatty acid intake and 10-year risk of coronary heart disease in the Zutphen Elderly Study: a prospective population-based study. *Lancet* 2001; 357 (9258): 746-751.

Dietary fat and the prevention of chronic disease **饮食中脂肪与慢性病的预防**

Mark L Wahlqvist

Director, Asia Pacific Health & Nutrition Centre, Monash Asia Institute, Monash University, Melbourne, Australia, Chair, Nutrition Committee, Australian Academy of Science

慢性病通常包括肥胖(特别是腹部肥胖)、糖尿病、大血管疾病(它会影响到所有中动脉及其所供血的器官)、骨质疏松症、各种癌症(特别是乳腺癌、肺癌、结肠直肠癌、胰腺癌、前列腺癌和皮肤癌)和痴呆。不幸的是,它们可能并不是慢性的,因为它们发病和引起的死亡在成年人的早期就可能出现,而且病情会迅速发展。不管以什么样的方式,它们都和食品、营养和其它环境及生活方式有关,因此提出了环境营养疾病(END)这个术语。就慢性病的营养基础而言,我们可以简单地称之为与营养相关的失调或疾病(NRD)。关于脂肪和END或NRD,需要考虑的关键是脂肪来源的多样性和它对能量密度和营养素密度的作用。这些都体现在世界卫生组织2003年的9816号报告“饮食、营养和慢性病的预防”中。

关键词: 脂肪多样性, 反式脂肪酸, 能量密度, 营养素密度, 营养相关失调