# Clinicians changing individual food habits

Mark L Wahlqvist AO, BMedSc, MDBS (Adelaide), MD (Uppsala), FRACP, FAIFST, FACN, FAFPHM

*International Health and Development Unit, Faculty of Medicine, Monash University, Melbourne, Victoria, Australia*
*Food and Agriculture Organization, Centre of Excellence in Food Safety, Quality and Nutrition, Melbourne, Victoria, Australia*
*Asia Pacific Health and Nutrition Centre, Monash Asia Institute, Monash University, Melbourne, Victoria, Australia*

There is ample evidence from repeat food surveys that people are actually and passively changing their food habits. Understanding the reasons for this are vital to any efforts by clinicians to enable individuals to move in an increasing healthful direction with their food intake, and to dispel the pessimism that often prevails about the ability to make a useful contribution to nutritional status by changing food intake. Current success and failure rates are predicated on inadequate methods and inappropriate outcome measures, rather than an inability to achieve outcomes. Factors that allow for or encourage change are food availability, exposure to new food experiences, food memory, pleasure, eating with peers or companions, health interest and changing constructs and beliefs about food. It is possible to change the health impact of food by non-food means like physical activity, stress management, recreational activities, improved relationships, changing the work environment and through adequate sleep (including siesta). Yet another consideration is that the full consequences of food choice are not appreciated with more and more food–health relationships being defined (e.g. with cataract, macular degeneration, in depression and cognitive function). These various approaches require a management strategy that underpins the field of behavioural therapy. In this approach it is possible to make progress through small but consequential changes, like climbing the stairs, or increasing intake of particular foods like fish or drinking more water; and exploring and contracting ways to do these things.

**Key words: food habits, behavioural change, clinical nutrition, medical practice, cardiovascular disease, cancer, osteoporosis, diabetes, obesity.**

## Introduction

The guidelines for counselling about food and health are:

- Evaluate the need for change with respect for the individual's food culture and beliefs;
- Understand the individual's capacity and interest in change;
- Intensify those changes which would have the greatest health impact at the least cost and risk;
- Prioritize the potential changes and develop a plan for implementation;
- Identify potential worthwhile non-food changes like those to do with relationships, sleep and physical activity, which will make foods more helpful;
- Work with the individual to effect at least one change in the immediate future;
- Have the change recorded by the individual as an index of achievement;
- Arrange for review of successive changes on either a face-to-face or remote basis, in order that the changes may be maintained;
- Allow opportunity for individuals to understand their own progress in relation to the community in which they live and in relation to those who already achieve the best from food–health relationships.

## Food patterns change

Much pessimism often prevails about the ability to make a useful contribution to nutritional status by changing food intakes. As individuals, we know that through our lifespan, we have made actual changes to the way in which we eat, and many of the foods we know and enjoy today were unknown to us in childhood. For many Australians, for example, radical changes in eating are the introduction of yoghurt, polyunsaturated margarines and vegetable oils; in these cases the foods have had huge health significance, albeit some of it requires further evaluation.[1,2]

Documentary evidence of change comes from repeat food surveys, like those first undertaken in Australia in the 1930s and 1940s, and repeated most recently in 1995. The difficulties of such surveys are that they are done infrequently, and the survey methods change from time to time. Market research provides targeted information on a much more frequent basis as a rule, even every few months, although often the scientific rigour with which these surveys are executed leaves room for doubt about the level of accuracy.[3] Food product sales are an increasingly available data source with the scanning of barcodes at the point of purchase, but they may omit fresh and unpackaged produce and they do not take account of urban gardens or the introduction into the home of foods like caught fish, which are outside the commercial food

**Correspondence address:** Professor Mark L Wahlqvist, International Health & Development Unit, PO Box 11A, Monash University, Melbourne, Vic. 3800, Australia.
Tel: 61 3 9905 8145; Fax: 61 3 9905 8146
Email: mark.wahlqvist@med.monash.edu.au

chains. The national perspective of changing food habits is often located in so-called 'disappearance data', which include extensive information about the movement of food at various points in the food chain including production and trade. However, the major problematic assumption with those data is the extent to which the foods are actually eaten.

At the Institute of Horticultural Development, Agriculture, Victoria, Australia, unpublished data are detecting important trends in consumption that are not evident in data sets such as the National Nutrition Survey. These include declines in apple and cruciferous vegetable consumption. From the health point of view, this information is critical given the current understanding of the cardioprotective role of apples,[4] and the bowel cancer protective role of cruciferous vegetables.[5] To the clinician, it would be increasingly important to know of this trend, and also to seek out information from individual patients as part of overall diagnostic and management strategies.

The change in numbers and types of restaurants in urban and regional Australia in the last 20–30 years also gives a very clear indication of shifts in food habits. The proportion of food eaten out grows, but what is eaten out depends on a number of socio-demographic factors, each of which is relevant to a health counsellor.

Further important evidence of extensive food change in recent decades comes from intervention studies at the population level, like the Multiple Risk Factor Intervention Trial (MRFIT) study in North America[6] and in North Karelia in Finland.[7] In both these studies, there was 'contamination' of reference populations beyond the study populations with regard to food intake, which reflects uptake by others who get to know about the potential value of food change.

**Socio-demographic basis of changes in food habit**

There is a host of reasons why the pace of individual change in food habit is quickening (Table 1). The importance of these various socio-demographic factors has been underscored by several investigators in Australia.[8,9]

Of course, these changes can be favourable or unfavourable. They represent, one way or another, opportunities for clinical intervention.

**Success and failure rates**

Current indices of success and failure of recommended food habit change often give little hope to clinicians, but they can be predicated on inadequate methods and inappropriate outcome measures, rather than an inability to achieve appropriate outcomes. Examples of this problem are seen in a comparison of the impact of the American Heart Association guidelines on diet and heart disease through a focus on serum lipids,[10] as opposed to the Lyon Heart Study, which took a whole of diet approach without particular regard to serum lipids and preferred approaches that emphasized fat quality and increased plant food variety[11] rather than saturated fat and cholesterol reductions.

A greater sense of achievement with food habit change can be realized by widening the surrogates for disease endpoints in nutritional intervention beyond the present focus; for example, on serum cholesterol in cardiovascular disease (Table 2).

Indeed, when it is realized that small changes can effect large changes in morbidity and mortality, a sense of much greater optimism enters the role of counselling in clinical practice. A good example of simple dietary change with great consequence is the study of Burr and others in which an increase from zero to three meals of fish a week led to a reduction of all-cause mortality by 30% in two years in those who had suffered an ischaemic event in the past.[15] There are very few interventions of a pharmacological kind that would have this order of outcome, even in a disease-specific way, let alone for all-cause mortality. The study, Dietary Approaches to Stop Hypertension (DASH), which combined changes in dairy product and plant food intake yielded similar major reductions in blood pressure.[1] Bao and coworkers have shown that increasing fish consumption to four meals a week for four months, along with weight reduction, led to a reduction in systolic blood pressure by 6 mmHg and in diastolic blood pressure by 3 mmHg,[16] also changes that are not seen with drugs. In the Lyon Diet Heart Study, patients who maintained the Mediterranean diet for up to four years had a 50% reduction in the rate of cardiac death and non-fatal myocardial infarction.[11]

For the progression from impaired glucose tolerance (IGT) to diabetes, however, so far the impact of increased levels of physical activity have been more impressive or, at least, equivalent to the impact of dietary change.[17,18] What is not acknowledged in these studies, however, is that full advantage has not been taken of the knowledge of low glycaemic foods, whole grain and magnesium-rich foods in deferring progression to diabetes.[19]

Further optimism is supported by the studies of food diversity where favourable health outcomes in populations have been tracked for those who achieve high levels of food

**Table 1.** Reasons for individual change

| |
|---|
| Demographics |
| Ageing |
| Migration |
| Family/household size |
| Work pattern |
| Leisure time activity |
| Gender equity |
| Education |
| Economic status |
| Health interest |
| Secularization |
| Time |
| Mood |

**Table 2.** Surrogates for disease endpoints in nutritional intervention

| | |
|---|---|
| Cardiovascular disease | Arterial wall indices[12] |
| Food variety[22] | |
| Fish[12] | |
| Vitamin B-6 and folate[13] | |
| Osteoporosis | Bone mineral density |
| Cancer | DNA adducts[14] |
| Rectal cell kinetics | |
| Diabetes complications | Glycaemic status/HbA1C |
| Microalbuminuria | |
| Mental health | Mood |
| Dementia | Cognitive function test |

diversity;[20,21] for example, in arterial and metabolic disease indices.[22]

While it is taken for granted that people who are malnourished on account of inadequate food intake will respond to increased food intake, what is less well known is that there are certain foods and nutrients that can very simply change the course of morbidity and mortality in populations, and that may be extrapolated from socio-economically disadvantaged to advantaged populations, in which there remain vulnerable groups. The whole issue of the principal micronutrient deficiencies affecting the world; for example, iron, vitamin A and iodine, in particular, needs to be revisited in advanced economies.[23]

Zinc deficiency is seen in North America, especially amongst Hispanics, and questions have been raised about zinc status in Aboriginal Australians.[24] More recently, a dramatic reduction in childhood pneumonia by 41%, and in diarrhoea by up to a quarter was found by researchers from the Child Health Research Project (CHRP) at Johns Hopkins School of Public Health (Baltimore, MD, USA) and World Health Organization through dietary zinc supplementation.[25] We know that older persons in developed countries are often found to be zinc deficient. In part, this is because of treatments like diuretics which cause zincuria. There is an urgent need to evaluate the extent to which winter respiratory illness and pneumonia could be avoided in older people and, consequently, their admission rates to hospital could be reduced.

The place of increased *n*-3 fatty acids in clinical practices is becoming increasingly well established. But the most pressing reason for assessing the *n*-3 fatty acid intake of patients is the risk of sudden death in those with ischaemic heart disease through ventricular fibrillation.[26] Much evidence points to this risk being reduced by fish intakes of as little as one to three meals a week.[27] For those who are vegetarian or cannot eat fish because of sensitivity reactions, alternative plant sources of *n*-3 fatty acids may be important; these include linseed, rapeseed (sometimes known as Canola), purslane and, to some extent, soy.

Investigators are now modelling traditional food cultures and changes in food cultures, and examining the predictive power of these models in different geographical settings.[28–30] By using models of this kind, and testing their predictability, we can contribute to evidence-based nutrition. Much of the acquired knowledge in food and health is not amenable to double-blind, randomised clinical trials, and those who work in public health and clinical nutrition need the support of other lines of evidence, which modelling and prediction can provide.

The Australian Polyp Prevention Project (APPP), which was a randomised factorial intervention study with colonoscopies done at 0 and 4 years in those who had polyps removed at baseline, demonstrated that a combination of increased dietary fibre (as wheat bran 25 g daily) and reduced percentage fat energy below 25% totally prevented the recurrence of large adenomatous polyps (10 mm or more), which are generally regarded as the precursor of large bowel cancer.[31] This is a finding that deserves much more currency in preventive clinical nutrition practice. However, a wider range of small dietary changes may be more likely to reduce bowel cancer risk.

What can be said, therefore, is that there is an opportunity to make small but significant changes to food intake, which are often less demanding and more appropriately focused on total health than a single pathway to health like serum lipid reduction. Failure to do this will lead to a burgeoning pharmaceutical industry that focuses on single pathways when a more holistic, nutrition and lifestyle approach might yield equivalent, if not superior, overall outcomes.

An example of a more holistic approach is summarized in Table 3.

**Facilitating change in food habits**

Some of the most important factors which allow for or encourage change are listed in Table 4. These can be recruited by clinicians in their food-health plans.

Non-food factors like physical activity, stress management, recreational activity, improved relationships, changing work environment and adequate sleep can allow much more error in food intake without adverse health consequences[33] (Table 5).

An interesting culturally based example of this is the finding that siestas may be protective against ischaemic heart disease in Greeks. But the ability to have a siesta depends very much on where one works in relation to home, questions of social acceptability and provision of places for rest. Few would deny that there is a natural rhythm that makes most of us somewhat drowsy in the early afternoon, and not to respond to this is probably biologically undesirable. When we are sleepy or drowsy, and whether we respond to this state, affects the pattern of eating across the day. Food patterns, in relation to sleep, are poorly studied insofar as health outcomes are concerned. But sleep has been shown to be a key factor in overall well-being.[34]

**Table 3.** Cardioprotective way of eating

| |
|---|
| Food variety |
| Low in ruminant animal (saturated) fat |
| Regular intake of fish or surrogate plant food (linseed, Canola, purslane) |
| $Na^+/K^+$ intake ratio < 1.0 |
| Alcohol in moderation |
| Avoid large meals |

**Table 4.** Factors that allow for or encourage change

| |
|---|
| Food availability |
| Exposure to new food experiences |
| Food memory[32] |
| Pleasure |
| Eating with peers or companions |
| Health interest |
| Changing constructs and beliefs about food |

**Table 5.** Non-food means to change health impact of food

| |
|---|
| Physical activity |
| Stress management |
| Recreational activities |
| Improved relationships |
| Changing work environment |
| Adequate sleep (siesta) |

**Consequences of food choice**

The full consequences of food choice have yet to be appreciated. So far, these are located around the so-called chronic non-communicable diseases (CNCD), which are probably better referred to as eco-nutritional diseases as they represent a problem created by changing environments and especially by the ways in which these influence our food habits. The diseases in question are obesity, especially abdominal obesity, diabetes of the non-insulin dependent type, macrovascular diseases, but sometimes osteoporosis and certain cancers are included in this list. Other emergent health problems related to food choice are shown in Table 6.

**Diagnostic and management strategies**

The first step in enabling healthful changing food habits is to make a nutritional diagnosis.[35] Without a diagnosis the nutrition problem cannot be managed or solved.

All nutritional management strategies require an underlying behavioural approach.[36] With this approach it is possible to make progress through small, but consequential changes, like climbing stairs, or increasing the intake of particular foods like fish or vegetable soups, or drinking more water; and exploring and contracting ways to do these things.

**Guidelines for counselling about food and health**

Guidelines for advising the population about food and health may be summarized as:

- Evaluate the need for change with respect for the individual's food culture and beliefs;
- Understand the individual's capacity and interest in change;
- Intensify those changes that would have the greatest health impact at the least cost and risk;
- Prioritize the potential changes and develop a plan for implementation;
- Identify potential worthwhile non-food changes like those to do with relationships, sleep and physical activity, which will make foods more helpful;
- Work with the individual to effect at least one change in the immediate future;
- Have the change recorded by the individuals as an index of achievement;
- Arrange for review of successive changes on either a face-to-face or remote basis, in order that the changes may be maintained;
- Allow opportunity for individuals to understand their own progress in relation to the community in which they live and in relation to those who already achieve the best from food–health relationships.

**Table 6.** Consequence of food choice

| |
|---|
| Chronic non-communicable disease |
| Mood |
| Cognitive function |
| Immune function |
| Inflammatory disease |
| Musculoskeletal system (MSS) |
| Chronic inflammatory bowel disease (CIBD) |
| Integumentary disease (eczema, psoriasis) |
| Sensory disturbance |
| Taste and smell |
| Vision |
| Sexuality |

**References**


1. Moore TJ, Vollmer WM, Appel LJ, Sacks FM, Svetkey LP, Vogt TM, Conlin PR, Simons-Morton DG, Carter-Edwards L, Harsha DW. Effect of dietary patterns on ambulatory blood pressure: Results from the Dietary Approaches to Stop Hypertension (DASH) Trial. DASH Collaborative Research Group. Hypertension 1999; 34: 472–477.
2. Hodgson JM, Wahlqvist ML, Boxall JA, Balazs NDH. Can linoleic acid contribute to coronary artery disease? Am J Clin Nutr 1993; 58: 228–234.
3. Grant P. What market research can tell us about Australian food habits. In: Truswell AS, Wahlqvist ML, eds. Food habits in Australia, 1st edn. North Balwyn, Vic: Rene Gordon, 1988; 185–200.
4. Knekt P, Jarvinen R, Reunanen A, Maatela J. Flavonoid intake and coronary mortality in Finland: A cohort study. BMJ 1996; 312: 478–481.
5. Lin HJ, Probst-Hensch NM, Louie AD, Kau IH, Witte JS, Ingles SA, Frankl HD, Lee ER, Haile RW. Glutathione transferase null genotype, broccoli, and lower prevalence of colorectal adenomas. Cancer Epidemiol Biomark Prev 1998; 7: 647–652.
6. Gorder DD, Dolecek TA, Coleman GG, Tillotson JL, Brown HB, Lenz-Litzow K, Bartsch GE, Grandits G. Dietary intake in the Multiple Risk Factor Intervention Trial (MRFIT): Nutrient and food group changes over 6 years. J Am Diet Assoc 1986; 86: 744–751.
7. Pietinen P, Nissinen A, Vartiainen E, Tuomilehto A, Uusitalo U, Ketola A, Moisio S, Puska P. Dietary changes in the North Karelia Project (1972–1982). Prev Med 1988; 17: 183–193.
8. Worsley A, Scott V. Consumers' concerns about food and health in Australia and New Zealand. Asia Pac J Clin Nutr 2000; 9: 24–32.
9. Baghurst KI, Baghurst PA, Record SJ. Public perceptions of the role of dietary and other environmental factors in cancer causation or prevention. J Epidemiol Community Health 1992; 46: 120–126.
10. Kris-Etherton PM, Pearson TA, Wan Y, Hargrove RL, Moriarty K, Fishell V, Etherton TD. High-monounsaturated fatty acid diets lower both plasma cholesterol and triacylglycerol concentrations. Am J Clin Nutr 1999; 70: 1009–1015.
11. de Lorgeril M, Salen P, Martin JL, Monjaud I, Delaye J, 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.
12. Wahlqvist ML, Lo CS, Myers KA. Fish intake and arterial wall characteristics in healthy people and diabetic patients. Lancet 1989; 2: 944–946.
13. Vermeulen EGJ, Stehouwer CDA, Twisk JWR, van den Berg M, de Jong SC, Mackaay AJC, van Campen CMC, Visser FC, Jakobs CAJM, Bulterijs EJ, Rauwerda JA. Effect of homocysteine-lowering treatment with folic acid plus vitamin B6 on progression of subclinical atherosclerosis: A randomised, placebo-controlled trial. Lancet 2000; 35: 517–522.
14. Podmore ID, Griffiths HR, Herbert KE, Mistry N, Mistry P, Lunec J. Vitamin C exhibits pro-oxidant properties (letter). Nature 1998; 392: 559.
15. Burr ML, Fehily AM, Gilbert JF, Rogers S, Holliday RM, Sweetnam PM, Elwood PC, Deadman NM. Effects of changes in fat, fish and fibre intakes on death and myocardial reinfarction: Diet and reinfarction trial (DART). Lancet 1989; 2: 757–761.
16. Bao DQ, Mori TA, Burke V, Puddey IB, Beilin LJ. Effects of dietary fish and weight reduction on ambulatory blood pressure in overweight hypertensives. Hypertension 1998; 32: 710–717.
17. Eriksson K-F, Lindgärde F. Prevention of type 2 (non-insulin-dependent) diabetes mellitus by diet and physical exercise. Diabetologia 1991; 34: 891–898.
18. Pan XR, Li GW, Hu YH, Wang JX, Yang WY, An ZX, Hu ZX, Lin J, Xiao JZ, Cao HB, Liu PA, Jiang XG, Jiang YY, Wang JP, Zheng H, Zhang H, Bennett PH, Howard BV. Effects of diet and exercise in preventing NIDDM in people with impaired glucose tolerance. The Da Qing IGT Diabetes Study. Diabetes Care 1997; 20: 537–544.

19. Hu FB, Stampfer MJ, Manson JE, Rimm E, Colditz GA, Rosner BA, Hennekens CH, Willett WC. Dietary fat intake and the risk of coronary heart disease in women. N Engl J Med 1997; 337: 1491–1499.
20. 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.
21. Wahlqvist ML, Hodgson JM, Ng FM, Hage BH-H, Strauss BJG. The role of nutrition in abdominal obesity. Nutr Res 1999; 19: 85–101.
22. Wahlqvist ML, Lo CS, Myers KA. Food variety is associated with less macrovascular disease in those with Type II diabetes and their healthy controls. J Am Coll Nutr 1989; 8: 515–523.
23. Hetzel BS. Iodine deficiency: A global problem. Med J Aust 1996; 165: 28–29.
24. Cheek DB, Spargo RM, Holt AB. Evidence for zinc deficiency in aboriginal settlements in Northwestern Australia. Med J Aust 1981; 1 (Suppl. 2): 4–5.
25. Bhutta ZA, Black RE, Brown KH, Gardner JM, Gore S, Hidayat A, Khatun F, Martorell R, Ninh NX, Penny ME, Roasdo JL, Roy SK, Ruel M, Sazawal S, Shankar A. Prevention of diarrhea and pneumonia by zinc supplementatin in children in developing countries: Pooled analysis of randomized controlled trials. Zinc Investigator's Collaborative Group. J Pediatr 1999; 135: 689–697.
26. Zhang J, Sasaki S, Amano K, Kesteloot H. Fish consumption and mortality from all causes, ischemic heart disease, and stroke: An ecological study. Prev Med 1999; 28: 520–529.
27. Charnock JS. The role of omega-3 PUFA enriched diets in the prevention of ventricular fibrillation. Asia Pac J Clin Nutr 1999; 8: 226–230.
28. Trichopoulou A, Kouris-Blazos A, Wahlqvist ML, Gnardellis C, Lagiou P, Polychronopoulos E, Vassilakou T, Lipworth L, Trichopoulos D. Diet and overall survival of the elderly. BMJ 1995; 311: 1457–1460.
29. Kouris-Blazos A, Gnardellis C, Wahlqvist ML, Trichopoulos D, Lukito W, Trichopoulou A. Are the advantage of the Mediterranean diet transferable to other populations? A cohort study in Melbourne, Australia. Br J Nutr 1999; 82: 57–61.
30. Osler M, Schroll M. Diet and mortality in a cohort of elderly people in a north European community. Int J Epidemiol 1997; 26: 155–159.
31. MacLennan R, Macrae F, Bain C, Battistutta D, Chapuis P, Gratten H, Lambert J, Newland RC, Ngu M, Russell A, Ward M, Wahlqvist ML, The Australian Polyp Prevention Project. Randomized trial of intake of fat, fiber, and beta carotene to prevent colorectal adenomas. J Natl Cancer Inst 1995; 87: 1760–1766.
32. Nishijo H, Ono T. Food memory: Neuronal involvement in food recognition. Asia Pac J Clin Nutr 1992; 1: 3–11.
33. Trichopoulos D, Tzonou A, Christopoulos C, Havatzoglou S, Trichopoulou A. Does a siesta protect from coronary heart disease? (letter). Lancet 1987; 2: 269–270.
34. Briones B, Adams N, Strauss M, Rosenberg C, Whalen C, Carskadon M, Roebuck T, Winters M, Redline S. Relationship between sleepiness and general health status. Sleep 1996; 19: 583–588.
35. Wahlqvist ML, Vobecky JS, eds. Patient problems in clinical nutrition: A manual. London: John Libbey & Co., 1987.
36. Stunkard AJ. Behavioural management of obesity. Med J Aust 1985; 142(Suppl. 7): S13–S20.