IF IT SUCCEEDS . . .

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A fundamental question, and an underlying theme throughout this book, is whether there are more gains than losses for individual and community health through the preventive nutrition approaches at present available. We are indeed likely to be ahead *in general* as far as people's well-being, levels of morbidity and life expectancy are concerned.

Yet there are a number of reasons for caution. That in itself is very encouraging. There are unknowns which need research. Preventive medicine and health care experiments, already carried out on small or large populations, still need evaluation and review. We cannot always be sure of the economic costs and benefits of what we think we know and there is the possibility of altogether new problems emerging, perhaps as a consequence of what we are now doing or in spite of what we are doing.

Linking population and individual health

Well-being

There is evidence from exercise studies that changing life-style towards more exercise can lead to an increased sense of well-being, less mood disorder and better coping skills[1-3]. Food intake patterns have been less well studied as far as well-being is concerned, as opposed to their possible role in avoidance of frank disease. Studies of cognitive function and how it relates to nutrient intake in apparently nutritionally-compromised seem to reflect adequacy of essential micronutrient intakes and therefore by implication to discourage attempts to change the diet (eg through supplementation).[4,5] Nevertheless, psychological and behavioural tests are regarded by toxicologists as sensitive indices of dietary change and are worthy of greater application in the surveillance of nutritionally-related well-being[6].

What is increasingly clear is that social activities and social networks are important measures of well-being and determinants of morbidity and life expectancy[7,8] (Figure 1). Eating is an activity inseparable from overall patterns of home and 'social' life. Thus, a socio-anthropological, rather than a physiological, appreciation of the roles and function of food and eating may reveal more about, and contribute more to, nutritionally-related well-being[9-11] (Figure 2).

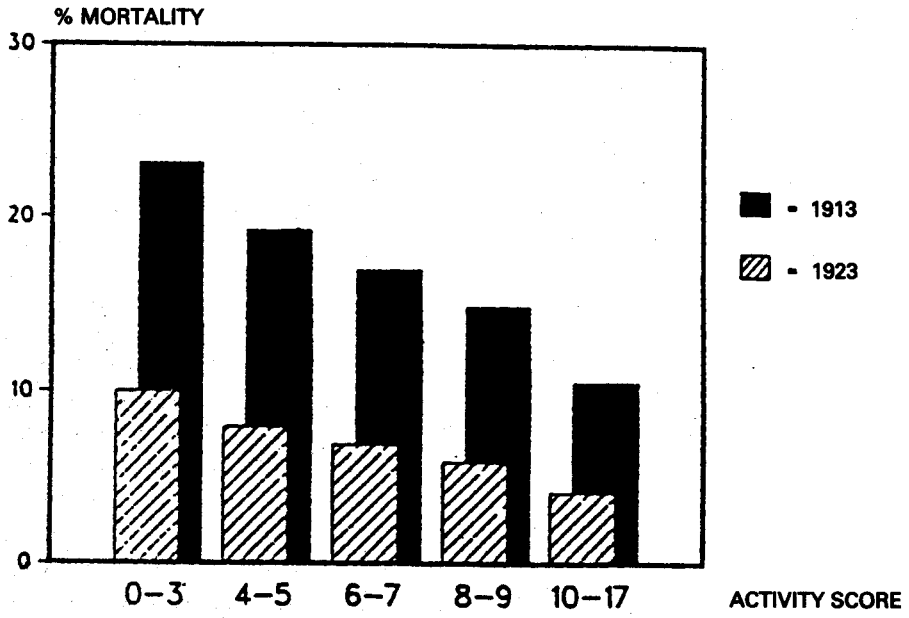


Figure 1. Percent mortality in men born in 1913 and 1923 and grouped according to social-activity score.[8]

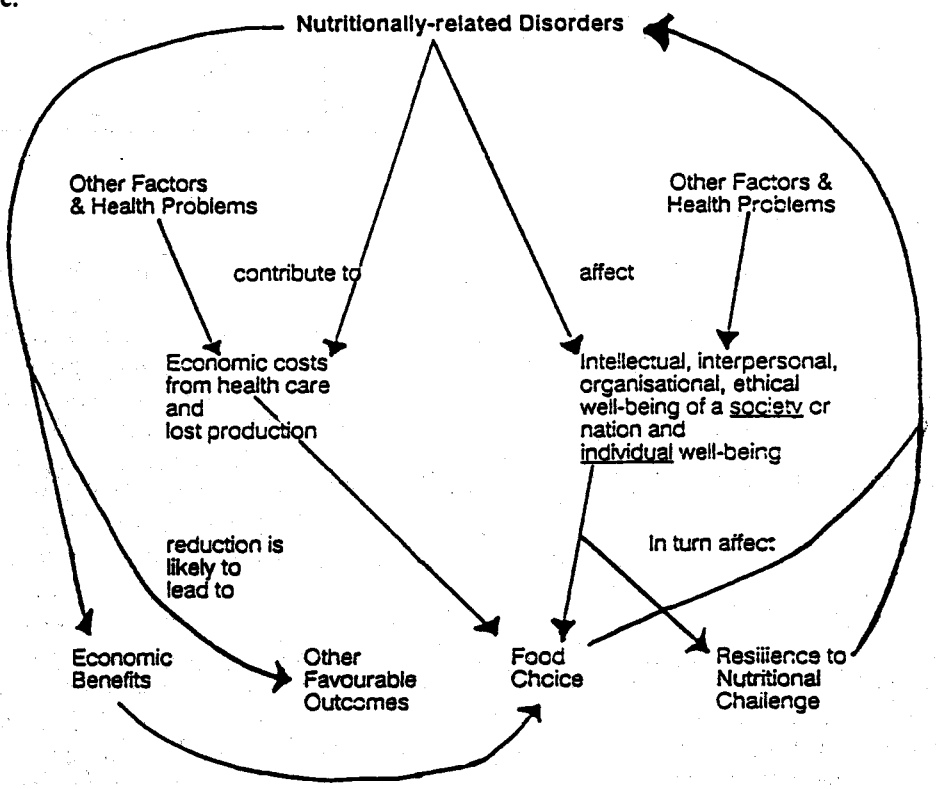


Figure 2. The nutrition-health-economic cycle.

Morbidity

Striking improvements in cardiovascular morbidity and mortality are currently in evidence in some developed countries[12,13]. These are probably partly attributable to nutritional changes, like those in the quality of fat ingested[14], but also to other lifestyle changes such as reduction in cigarette smoking[15-18] and some improvement in level of physical activity[16-18].

However, the problem of obesity is probably growing worldwide, in developing (eg Kenya and China), transitional (Malaysia, Singapore and Thailand) and developed countries[19]. The evidence in Australia during the 1980s, is for a dramatic increase in obesity in capital cities, especially for post-menopausal women[15-18], which is reflected in excessive abdominal fatness as well as total fatness in the 1989 data[20] (Figure 3). Migration to Australia has also compromised fat distribution for both Chinese and Greeks[21] (Figure 4). The problem in Australia at the end of the 1980s equalled that of the USA at the beginning of the 1980s (Table 1). It is not at all clear why this phenomenon has occurred in Australia, but its identification underscores the importance of regular nutritional surveillance of the population.

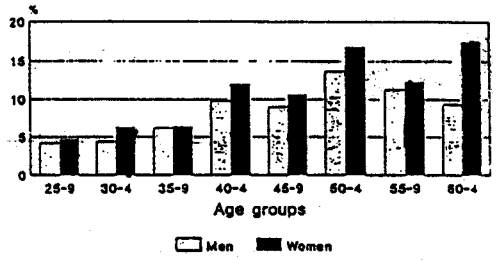
Table 1. Percentage of overweight and obese people in English-speaking countries[25].

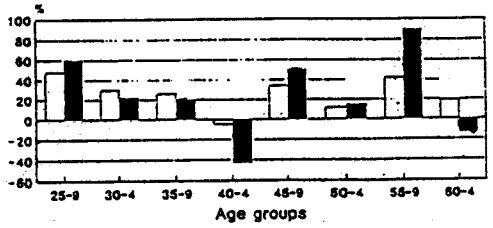
	Age	Overweight (%) M	Overweight (%) F	Obese (%) M	Obese (%) F
Australia	25–64	34	24	7	7
Britain	16–65	34	24	6	8
United States	20–74	31	24	12	12

Overweight = BMI of 25–30 kg/m^2
Obese = BMI above 30 kg/m^2
BMI = body mass index

In Australia the 1980s were characterized by an intensification of public health initiatives and greater focus on food-health issues. They have either been inadequate or, at times, inappropriate. For example, evidence constantly points towards reduced fat intake and increased physical activity as protective against the development of obesity[22,23] and, therefore, it is curious that obesity increased during the 1980s. We must presume that what has been done is still not enough or that some sub-groups in society who are not exercising account for the dominant population increase in obesity: further analysis of individual physical activity and BMI status on a population-wide basis is required. Again, the success of the anti-smoking health lobby in the Australian State of Victoria has been impressive, but the prevalence of obesity in Victoria now exceeds the national average. It could well be that, in part, the reduction in smoking, which must be maintained for multiple health reasons, has exacerbated the obesity problem[24,25]. In that event, more cohesive public health strategies, with less separation of nutrition, exercise, and anti-smoking campaigns, and of these strategies in individual counselling, may be required. An advantage of medical health promotion is that these several areas of public health need can be brought together in terms of the one individual, or of small groups of individuals.

With an aging population, and probable improvements in functional and





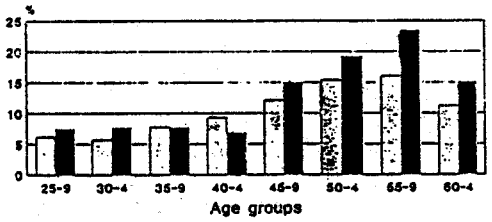


Figure 3. *a:*Obesity (BMI = weight/height^2 >30) estimates for 1983 men and women. *b:* Obesity (BMI = weight/height^2 >30) estimates for 1989 men and women. *c:* Obesity (BMI = weight/height^2 >30) percentage changes from 1983 to 1989 men and women.

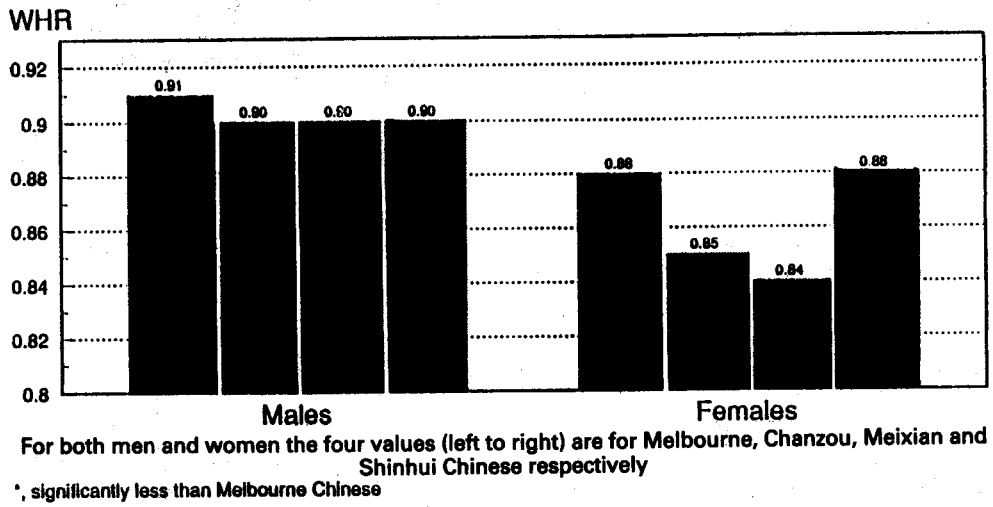


Figure 4. Chinese health study: mean waist to hip ratio by gender by community (Wahlqvist & Hage, 1992).

biological age[25], shifts in age-specific morbidity to later life are likely, and the patterns of morbidity are likely to change (Figure 5, 6). Less cardiovascular morbidity and mortality may be seen in younger individuals and more in older individuals, and it may be progressively replaced with problems such as dementia (with varying ratios of Alzheimer-type and multi-infarct dementia) and osteoporosis-related fractures. Age-related and nutritionally-related immunodeficiency with its sequelae are also likely to be on the increase in absolute terms as the population ages, although it is not clear whether there will be a changing proportion of elderly people affected[12,27].

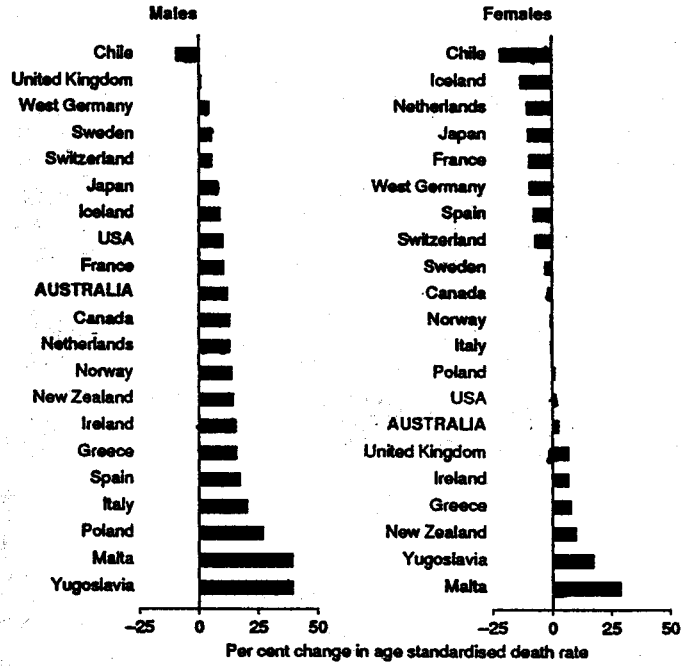


Figure 5. Cancer deaths, changes in rates, selected countries, 1965–1969 to 1980–1984 (Source: World Health Organization 1988).

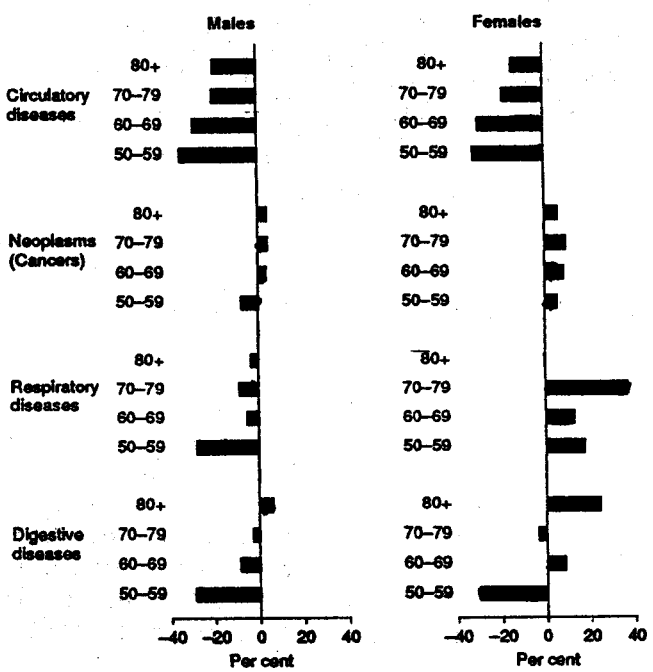


Figure 6. Change in age specific death rates 1981 to 1988 for major causes of death for persons 50 years and over (Source: Australian Institute of Health).

Mortality

The literature on aging generally agrees that the maximum human life span is about 120 years. The best average life expectancies at birth are still increasing, and now exceed 80 years for women. At any one point in time, life expectancy is computed on the basis of composite data at various ages. So it is a statement about what would apply if one were born now and all factors operative currently continued to operate throughout one's life-time. What it does not allow is a consideration of whether various combinations or sequences of risk exposures might lessen or increase with

Table 2. Life expectancy, handicap-free expectancy and disability-free expectancy in years at age 65, and by sex, 1981 and 1988. (Source: Australian Institute of Health)

Health expectancy	*Males*	*Females*	*Total*
Life expectancy(a)			
1981	13.9	18.1	16.0
1988	14.8	18.7	16.8
Change: 1981–1988	+0.9	+0.6	+0.8
Handicap-free expectancy(b)			
1981	9.6	11.4	10.5
1988	8.0	9.6	8.8
Change: 1981–1988	−1.5	−1.8	−1.7
Disability-free expectancy			
1981	7.9	10.1	9.0
1988	6.7	8.6	7.6
Change: 1981–1988	−1.2	−1.5	−1.4

(a) Total life expectancy, all health states.
(b) Free of handicap, ie not limited to any degree in ability to perform tasks relating to self care, mobility, verbal communication, schooling and/or employment.

Table 3. Trends in life expectancy at birth, for different regions (both sexes combined)[a,b].

Region	1950–1955	1980–1985	2020–2025
Northern America	69.0	74.6	79.7
Europe	65.3	73.2	79.1
Oceania	60.8	68.0	75.6
USSR	64.1	67.9	76.7
Latin America	51.2	64.5	72.8
Asia	41.1	59.3	72.8
Africa	38.0	49.9	65.2
Developed countries	65.7	72.3	78.7
Developing countries	41.0	57.6	70.4
World total	45.9	59.6	71.3

[a] In years, medium variant used for projection.
[b] Adapted from *World Population Prospects, 1988* (United Nations publication, Sales No. E.88.XIII.7), reference *12*, by kind permission of the publisher.

Table 4. Elderly population, aged 60 years or more, in millions by region (1950, 1985, and projections for 2025[a]).

Region	Total population 1985	Elderly population					
		1950		1985		2025[b]	
		No.	%	No.	%	No.	%
Europe	492	51	12.9	88	17.8	138	27.0
Northern America	265	20	12.1	43	16.4	88	26.4
USSR	277	16	9.0	37	13.5	72	20.6
Oceania	25	1	11.3	3	12.3	7	18.5
Asia	2834	92	6.7	205	7.2	698	14.3
Latin America	404	9	5.3	27	6.8	97	12.7
Africa	557	12	5.4	27	4.9	101	6.4
Developed countries	1174	95	11.4	189	16.1	343	25.3
Developing countries	3680	106	6.3	243	6.6	858	12.1
World total	4854	201	8.0	432	8.9	1201	14.8

[a] Adapted from *World Population Prospects, 1988* (United Nations publication, Sales No. E.88.XIII.7), reference *12*, by kind permission of the publisher.
[b] Medium variant used for projection.

Table 5. Causes of death in 1980 in developed and developing countries, and world total. Adapted from reference 2.

Causes of death	Percentage of deaths		
	Developed countries	Developing countries	World total
Diseases of the circulatory system	54	19	26
Neoplasms	19	5	8
Infectious and parasitic diseases	8	40	33
Injury and poisoning	6	5	5
Perinatal mortality	2	8	6
All other causes	12	23	21

the passage of time (eg, deprivation in childhood, abundance in later life). An examination of age-specific life expectancies from generation to generation is a partial solution to this problem. What is particularly interesting is that in the last 20 years or so in some countries like Sweden and Australia life expectancy at age 65 has been increasing – apparently a new phenomenon for the human species (Tables 2–5)[12,25].

An understanding of the basis of this phenomenon has considerable potential for preventive nutrition, since this is a period during which there have been major changes in the food supply.

Paradoxically, Sweden, at a time when life expectancies at birth and at age 65 are

increasing, is one country in which coronary mortality has been increasing, albeit from lower levels than most other industrialized countries (Figure 7)[12]. Various reasons have been advanced for this phenomenon including the use of anti-hypertensive medications like thiazides and beta-blockers which can adversely affect coronary risk factors (serum lipids and glucose tolerance). More attention is now being paid to non-pharmacological strategies[30]. As many as half of those previously on anti-hypertensives may be managed by diet and exercise, and moderation in alcohol intake. It is also of interest that the Swedish traditional diet has been high in fish with its now recognized protective role against macrovascular disease[13,29,30], whereas, on account of concerns about the mercury content of fish and organo-mercury poisoning, Sweden has become one of the few countries to have dietary guidelines which discourage fish consumption. The net trade-offs for health in changing Swedish fish consumption may have been unfavourable. The dilemmas in preventive nutrition are real.

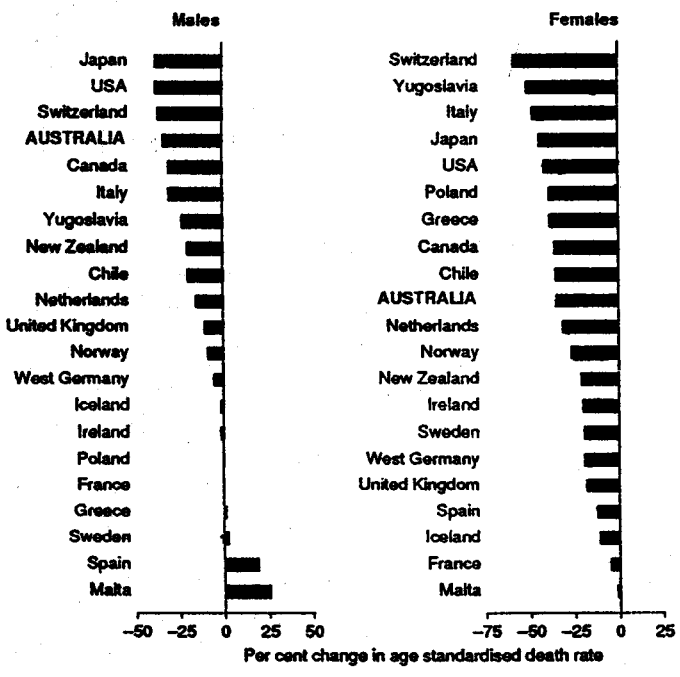


Figure 7. Coronary heart disease deaths, changes in rates, selected countries, 1965–69 to 1980–84 (Source: World Health Organization 1988).

An eye must always be kept on total mortality, as well as on disease-specific mortality. Most pharmacotherapy is directed at disease-specific mortality, such as the reduction of peptic ulcer disease and related haemorrhage and perforation with H^2 receptor antagonists, but what about total mortality? What of emerging evidence to suggest that fish intake may reduce cardiovascular and total mortality as well? Studies in which cholestyramine and gemfibrozil have been used to reduce coronary mortality (Lipid Clinic Study; Helsinki Study) have not seen a reduction in total mortality and, admittedly, were not designed to do so; but a randomized secondary prevention study in Wales in those who had experienced a myocardial infarction,

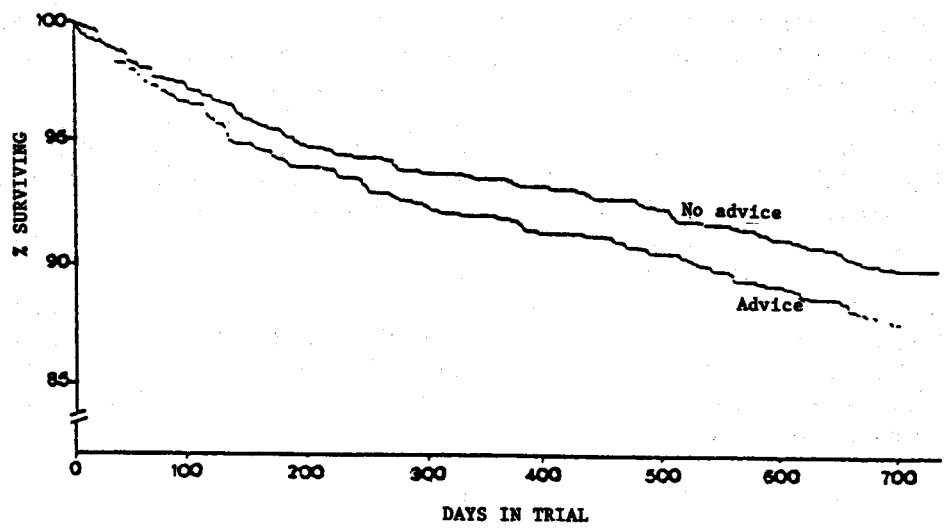


Figure 8. Survival: fish advice[31].

showed in those who increased fatty fish consumption, a 30% reduction in total mortality at 2 years[31] (Figure 8).

Health care systems. What now will emerge?

If we succeed in the prevention of even some of the presently recognized nutrition related health problems, will we inevitably be in better health or will new problems emerge?[32].

Already, the expectations that biomedical science will deliver better and better health status are high. The cost of fulfilling these expectations through the diagnosis and treatment of established disease are likely to be increasingly prohibitive, unless the technology developed can be more and more appropriate, less resource-consuming, and progressively less costly. Much greater cost-effective gains are likely to be made through prevention, which will include attention to nutritional factors, as part of general lifestyle approaches.

There is likely to be nutritional targeting in relation to the expression of certain genetically-based disorders. Food and nutrients can alter gene expression[33]. Thus, medical practice is likely to be engaged in a new kind of nutritional prevention – that which targets a genetically based disorder, which is food and nutrient responsive. In its least subtle form, a disorder like phenlketonuria is an example. Much more subtle are the myriad of genetic lesion which express themselves as various kinds of hyperlipidaemia[34] but which are nutritionally responsive (apo phenotypes; LDL receptor defects). For example, apoE phenotype identifies relative responsiveness to change in saturated fat intake. Where we make particular food or nutrient intake changes, we are likely to disturb other intakes or create new interrelationships (nutrient-nutrient or nutrient-non nutrient). Food is chemically complex. Risk-benefit ratios will need to be considered.

Even greater challenges arise when altogether new diseases, like HIV emerge, perhaps on account of changes in human behaviour or social change. The possibility

that nutritional measures may be preventive in the expression of such disease or in minimizing its sequelae will require consideration.

But probably the greatest challenge to the work of those engaged in preventive nutrition will come from environmental change and the forced changes in the food supply[35] together with major changes in food technology with its increasing ability to create novel foods, food analogues, and so-called 'functional' foods[36]. The possibility of new health problems as a consequence, and the opportunities for using food tailored to genetic disorders, will be considerable.

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