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WHO Technical report Series 916

DIET, NUTRITION AND THE PREVENTION OF CHRONIC DISEASES

ADVANCE FINAL DRAFT COPY (embargoed Monday, March 3, 00.01 GMT)

Report of a
Joint FAO/WHO Expert Consultation

World Health Organization
Geneva 2003

WHO Library Cataloguing-in-Publication Data

Joint WHO/FAO Expert Consultation on Diet, Nutrition and the Prevention of Chronic Diseases (2002 : Geneva, Switzerland)

Diet, nutrition and the prevention of chronic diseases : report of a joint WHO/FAO expert consultation, Geneva, 28 January - 1 February 2002.

(WHO technical report series ; 916)

1.Chronic disease - epidemiology 2.Diet - standards 3.Feeding behavior 4.Energy metabolism 5.Motor activity 6.Cost of illness I.Title II.Series.

ISBN 92 4 120916 X (NLM classification: QU 145)
ISSN 0512-3054

Typeset and printed in Switzerland

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Abbreviations

The following abbreviations are used in this report:

ACC	United Nations Administrative Committee on Coordination
AIDS	Acquired immunodeficiency syndrome
BMI	Body mass index
CARMEN	Carbohydrate Management in European National
CHD	Coronary heart disease
CVD	Cardiovascular disease
DALY	Disability Adjusted Life Year
DASH	Dietary Approaches to Stop Hypertension
DEXA	Dual-Energy X-ray Absorptiometry
DHA	Docosahexaenoic acid
dmf	decade, missing, filled primary (teeth)
DMF	decade, missing, filled permanent (teeth)
dmft	decade, missing, filled primary teeth
DMFT	decade, missing, filled permanent teeth
DONALD	Dortmund Nutritional and Anthropometric Longitudinally Designed Study
ECC	Early Childhood Caries
EPA	Eicosappentaenoic acid
EPIC	European Prospective Investigation into Cancer and Nutrition
ERGOB	European Research Group for Oral Biology
FAO	Food and Agriculture Organization of the United Nations
FAOSTAT	Food and Agricultural Organization of the United Nations Statistical Databases
FER	Fat to energy ratio
GDP	Gross domestic product
GISSI	Gruppo Italiano por lo Studio della Sopravvivenza nell'Infarto Miocardico
GNP	Gross national product
HBP	High blood pressure
HDL	High density lipoprotein
HIV	Human immunodeficiency virus
HFI	Hereditary fructose intolerance
HOPE	Heart Outcomes Prevention Evaluation
IARC	International Agency for Research on Cancer
IDDM	Insulin-dependent diabetes
IGT	Impaired glucose tolerance
IHD	Ischaemic heart disease
IUGR	Intrauterine growth retardation
LDL	Low density lipoprotein
MI	Myocardial infarction
MGRS	Multicentre growth reference study (i.e. the WHO MGRS study)
mRNA	Messenger ribonucleic acid
MSG	Monosodium glutamate
MUFA	Monounsaturated fatty acids
NCD	Noncommunicable disease
NGO	Non-governmental organization
NIDDM	Non-insulin-dependent diabetes mellitus
NSP	Nonstarch polysaccharides
PUFA	Polyunsaturated fatty acids
RCT	Randomised controlled trial
SCN	ACC Sub-committee on Nutrition
SFA	Saturated fatty acid
T1DM	Type 1 diabetes
T2DM	Type 2 diabetes
UN	United Nations
UNICEF	United Nations Children's Fund
VLDL	Very low density lipoprotein
VLDL-C	Very low density lipoprotein cholesterol

WCRF	World Cancer Research Fund
WHO	World Health Organization
WHR	Waist-hip circumference ratio or waist-hip ratio

1. Introduction

A Joint WHO/FAO Expert Consultation on Diet, Nutrition and the Prevention of Chronic Diseases met in Geneva from 28 January to 1 February 2002. The Consultation followed up the work of a WHO Study Group on Diet, Nutrition and Prevention of Noncommunicable Diseases, which had met in 1989 to make recommendations that would help prevent chronic diseases and reduce their impact(*1*). The Consultation recognized that the growing epidemic of chronic disease afflicting both developed and developing countries was related to dietary and lifestyle changes and undertook the task of reviewing the considerable scientific progress that has been made in different areas. For example, there is better epidemiological evidence for determining risk factors in some areas, and the results of a number of new controlled clinical trials are now available. The mechanisms of the chronic disease process are clearer, and interventions have been demonstrated to reduce risk.

In the past decade, rapid expansion of the relevant scientific fields and of the available population-based epidemiological evidence have helped to clarify the role of diet in preventing and controlling morbidity and premature mortality resulting from noncommunicable diseases (NCDs). Some of the specific dietary components that increase the probability of occurrence of these diseases in individuals, and interventions to modify their impact, have also been identified.

Furthermore, rapid changes in diets and lifestyles that have occurred with industrialization, urbanization, economic development and market globalization, have accelerated over the past decade. This is having a significant impact on the health and nutritional status of populations, particularly in developing countries and in countries in transition. While standards of living have improved, food availability has expanded and become more diversified, and access to services has increased, there have also been significant negative consequences in terms of inappropriate dietary patterns, decreased physical activities and increased tobacco use, and a corresponding increase in diet-related chronic diseases, especially among poor people.

Food and food products have become commodities produced and traded in a market that has expanded from an essentially local base to an increasingly global one. Changes in the world food economy have contributed to shifting dietary patterns, for example, increased consumption of energy-dense diets high in fat, particularly saturated fat, and low in unrefined carbohydrates. These patterns are combined with a decline in energy expenditure that is associated with a sedentary lifestyle—motorized transport, labour-saving devices at home, the phasing out of physically demanding manual tasks at work, and leisure time that is preponderantly devoted to physically undemanding pastimes.

Because of these changes in dietary and lifestyle patterns, chronic NCDs—including obesity, diabetes mellitus, cardiovascular disease, hypertension and stroke, and some types of cancer—are increasingly significant causes of disability and premature death in both developing and newly developed countries, placing additional burdens on already overtaxed national health budgets.

The Consultation provided an opportune moment for the World Health Organization (WHO) and the Food and Agriculture Organization of the United Nations (FAO) to draw on the latest scientific evidence available and to update recommendations for action by governments, international agencies and concerned partners in the public and private sectors. The overall aim of these recommendations is to implement more effective and sustainable policies and strategies to deal with the increasing public health challenges related to diet and health.

The Consultation articulated a new platform, not just of dietary and nutrient targets, but of a concept of the human organism's subtle and complex relationship to its environment in relation to chronic diseases. The discussions took into account ecological, societal and behavioural aspects beyond causative mechanisms. The experts looked at diet within the context of the macroeconomic implications of public health recommendations on agriculture, and the global supply and demand for foodstuffs, both fresh and processed. The role of diet in defining the expression of genetic susceptibility to NCDs, the need for responsible and creative partnerships with both traditional and non-traditional partners, and the importance of addressing the whole life course, were all recognized.

Nutrition is coming to the fore as a major modifiable determinant of chronic disease, with scientific evidence increasingly supporting the view that alterations in diet have strong effects, both positive and negative, on health throughout life. Most importantly, dietary adjustments may not only influence present health, but may determine

whether or not an individual will develop such diseases as cancer, cardiovascular disease and diabetes much later in life. However, these concepts have not led to a change in policies or in practice. In many developing countries, food policies remain focused only on undernutrition and are not addressing the prevention of chronic disease.

Although the primary purpose of the Consultation was to examine and develop recommendations for diet and nutrition in the prevention of chronic diseases, the need for sufficient physical activity was also discussed and is therefore emphasized in the report. This emphasis is consistent with the trend to include physical activity as part of diet, nutrition and health. Some relevant aspects include:

- Energy expenditure through physical activity is an important part of the energy balance equation that determines body weight. Decrease in energy expenditure through decreased physical activity is likely to be one of the major factors contributing to the global epidemic of overweight and obesity.
- Physical activity has great influence on body composition—on the amount of fat, muscle and bone tissue.
- Physical activity and nutrients share the same metabolic pathways to a large extent and can interact in various ways that influence the risk and pathogenesis of several chronic diseases.
- Cardiovascular fitness and physical activity have been shown to modulate significantly the effects of overweight and obesity on health, leading to the hypothesis that part of their deleterious effects are caused by lack of physical activity in overweight and obese persons rather than these conditions per se.
- Physical activity and food intake are both specific and mutually interacting behaviours that are and can be influenced partly by the same measures and policies.
- Lack of physical activity is already a global health hazard and is a prevalent and rapidly increasing problem in both developed and developing countries, particularly among poor people in a sprawling metropolis.

In order to achieve the best results in preventing chronic diseases, the strategies and policies that are applied must fully recognize the essential role of diet, nutrition and physical activity.

This report calls for a shift in the conceptual framework and in the strategy for action, placing nutrition—together with the other principal risk factors for chronic disease, namely, tobacco use and alcohol consumption—at the forefront of public health policies and programmes.

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2. Background

2.1 The global burden of chronic diseases

Diet and nutrition are important factors in the promotion and maintenance of good health throughout the entire life course. Their role as determinants of chronic noncommunicable diseases is well established and they therefore occupy a prominent position in prevention activities (*1*).

The latest scientific evidence on the nature and strength of the links between diet and chronic diseases is examined and discussed in detail in the following sections of this report. This section gives an overall view of the current situation and trends in chronic diseases at the global level. The chronic diseases considered in this report are those that are related to diet and nutrition and present the greatest public health burden, either in terms of direct cost to society and government, or in terms of disability adjusted life years (DALYs). These include obesity, diabetes, cardiovascular diseases, cancer, osteoporosis and dental diseases.

The burden of chronic diseases is rapidly increasing worldwide. It has been calculated that, in 2001, chronic diseases contributed approximately 60% of the 56.5 million total reported deaths in the world and approximately 46% of the global burden of disease (*1*). The proportion of the burden of NCDs is expected to increase to 57% by 2020. Almost half of the total chronic disease deaths are attributable to cardiovascular diseases; obesity and diabetes are also showing worrying trends, not only because they already affect a large proportion of the population, but also because they have started to appear earlier in life.

The chronic disease problem is far from being limited to the developed regions of the world. Contrary to widely held beliefs, developing countries are increasingly suffering from high levels of public health problems of chronic diseases. In five out of the six regions of WHO, deaths caused by chronic diseases dominate the mortality statistics (*1*). Indeed, although HIV/AIDS, malaria and tuberculosis, along with other infectious diseases, still predominate in sub-Saharan Africa and will do so for the foreseeable future, on a global basis 79% of all deaths attributable to chronic diseases are already occurring in developing countries (*2*). It is clear that the earlier view of these chronic diseases as "diseases of affluence" is increasingly a misnomer, as they rapidly emerge both in poorer countries and in the poorer population groups in richer countries. This evolution is taking place at an accelerating rate, with the transition occurring at a faster rate in developing countries than in the industrialized regions of the world, which experienced a more gradual evolution in the past century (*3*). This rapid rate of change, together with the increasing burden of disease, is creating a major public health threat which demands immediate and effective action.

It has been projected that, by 2020, chronic diseases will account for almost three-quarters of all deaths, and that 71% of deaths due to ischaemic heart disease (IHD), 75% of deaths due to stroke, and 70% of deaths due to diabetes will occur in developing countries (*4*). The number of people in the developing world with diabetes will increase by more than 2.5-fold, from 84 million in 1995 to 228 million in 2025 (*5*). On a global basis, 60% of the burden of chronic diseases will occur in developing countries. Indeed, cardiovascular diseases are even now more numerous in India and China than in all economically developed countries in the world added together (*2*). As for overweight and obesity, not only has the current prevalence already reached unprecedented levels, but the rate at which it is annually increasing in most developing regions is substantial (*3*). The public health implications of this phenomenon are staggering, and are already becoming apparent.

The rapidity of the transition in developing countries is such that a double burden of disease may often exist. India, for example, at present faces a combination of communicable diseases and chronic diseases, with the burden of chronic diseases just exceeding that of communicable diseases. Projections nevertheless indicate that communicable diseases will still occupy a critically important position up to 2020 (*6*). Another eloquent example is that of obesity, which is becoming a serious problem throughout Asia, Latin America and parts of Africa, despite the widespread presence of undernutrition. In some countries, the prevalence of obesity has doubled or tripled over the past decade.

Chronic diseases are largely preventable diseases. Although more basic research may be needed on some aspects of the mechanisms that link diet to health, the currently available scientific evidence provides a sufficiently strong and plausible basis to justify taking action now. Beyond the appropriate medical treatment for those already affected, the public health approach of primary prevention is considered to be the most cost-effective, affordable and sustainable course of action to cope with the chronic disease epidemic worldwide. The adoption of a common risk-factor

approach to chronic disease prevention is a major development in the thinking behind an integrated health policy. Sometimes chronic diseases are considered communicable at the risk factor level (*7*). Modern dietary patterns and physical activity patterns are risk behaviours that travel across countries and are transferable from one population to another like an infectious disease, affecting disease patterns globally.

While age, sex and genetic susceptibility are non-modifiable, much of the risk associated with age and sex is modifiable. Such risks include behavioural factors (diet, physical inactivity, tobacco use, and alcohol consumption); biological factors (dyslipidemia, hypertension, overweight, and hyperinsulinaemia); and finally societal factors, which include a complex mixture of interacting socioeconomic, cultural and other environmental parameters.

Diet has been known for many years to play a key role as a risk factor for chronic diseases. What is apparent at the global level is that great changes have swept the entire world since the second half of the twentieth century, inducing major modifications in diet, first in industrial regions and more recently in developing countries. Traditional, largely plant-based diets have been swiftly replaced by high-fat, energy-dense diets with a substantial content of animal foods. But diet, while critical to prevention, is just one risk factor. Physical inactivity, now recognized as an increasingly important determinant of health, is the result of a progressive shift of lifestyle towards more sedentary patterns, in developing countries as much as in industrialized ones. Recent data from São Paulo, Brazil, for example, indicate that 70–80 % of the people are remarkably inactive (*8*). The combination of these and other risk factors, such as tobacco use, is likely to have an additive or even a multiplier effect, capable of accelerating the pace at which the chronic disease epidemic is emerging in the developing countries.

The need for action to strengthen control and prevention measures to counter the spread of chronic disease epidemic is now widely recognized by many countries, but the developing countries are lagging behind in implementing such measures. Encouragingly, however, efforts to counteract the rise in chronic diseases are increasingly being assigned a higher priority. This situation is reflected by the growing interest of Member States, the concerned international and bilateral agencies as well as nongovernmental organizations in addressing food and nutrition policy, health promotion, and strategy for the control and prevention of chronic diseases, as well as other related topics such as promoting healthy ageing and tobacco control. The 1992 International Conference on Nutrition specifically identified the need to prevent and control the increasing public health problems of chronic diseases by promoting appropriate diets and healthy lifestyles (9–11). The need to address chronic disease prevention from a broad-based perspective was also recognized by the World Health Assembly in 1998 (12) and again in 1999 (13). In 2000, the World Health Assembly passed a further resolution on the broad basis of the prevention and control of noncommunicable diseases (14), and in 2002 adopted a resolution that urged Member States to collaborate with WHO to develop "…a global strategy on diet, physical activity and health for the prevention and control of noncommunicable diseases, based on evidence and best practices, with special emphasis on an integrated approach…" (15).

Several factors have constrained progress in the prevention of chronic diseases. These include underestimation of the effectiveness of interventions, the belief of there being a long delay in achieving any measurable impact, commercial pressures, institutional inertia and inadequate resources. These aspects need to be taken seriously and combated. One example is provided by Finland. In North Karelia, age-adjusted mortality rates of coronary heart disease dropped dramatically between the early 1970s and 1995 (*16*). Analyses of the three main risk factors (smoking, high blood pressure, raised plasma cholesterol) indicate that diet—operating through lowering plasma cholesterol and blood pressure levels—accounted for the larger part of this substantial decline in cardiovascular disease. The contribution made by medication and treatment (antilipid and hypotensive drugs, surgery) was very small. Rather, the decline was largely achieved through community action and the pressure of consumer demand on the food market. The Finnish and other experience indicates that interventions can be effective, that dietary changes are important, that these changes can be strengthened by public demand, and finally that appreciable changes can take place very rapidly. The experience of the Republic of Korea is also notable since the community has largely maintained its traditional high-vegetable diet despite major social and economic change (*17*). The Republic of Korea has lower rates of chronic diseases and lower than expected level of fat intake and obesity prevalence than other industrialized countries with similar economic development (*18*).

There are several opportunities for new global and national actions, including strengthened interaction and partnerships; regulatory, legislative and fiscal approaches; and more stringent accountability mechanisms.

The broad parameters for a dialogue with the food industries are: less saturated fat; more fruits and vegetables; effective food labelling; and incentives for the marketing and production of healthier products. In working with

advertising, media and entertainment partners, there is a need to stress the importance of clear and unambiguous messages to children and youths. Global "health and nutrition literacy" requires a vast increase in attention and resources.

Many studies show a relationship between health and income, the poorest sections of the population being the most vulnerable. In particular, this increased social disadvantage affects poor people disproportionately in terms of the incidence of chronic diseases, as well as access to treatment. The same disadvantage is reflected in decreased acceptance of health-promoting behaviours amongst the poorest sections of society. Thus, policies need to be pro-poor and appropriately targeted, as poor people are most at risk and have the least power to effect change.

2.2 The double burden of diseases in the developing world

Hunger and malnutrition remain among the most devastating problems facing the majority of the world's poor and needy people, and continue to dominate the health of the world's poorest nations. Nearly 30% of humanity are currently suffering from one or more of the multiple forms of malnutrition (*19*).

The tragic consequences of malnutrition include death, disability, stunted mental and physical growth, and as a result, retarded national socioeconomic development. Some 60% of the 10.9 million deaths each year among children aged under five years in the developing world are associated with malnutrition (*20*). Iodine deficiency is the greatest single preventable cause of brain damage and mental retardation worldwide, and is estimated to affect more than 700 million people, most of them located in the less developed countries (*21*). Over 2000 million people have iron deficiency anaemia (*22*). Vitamin A deficiency remains the single greatest preventable cause of needless childhood blindness and increased risk of premature childhood mortality from infectious diseases, with 250 million children under five years of age suffering from subclinical deficiency (*23*). Intrauterine growth retardation, defined as birth weight below the 10th percentile of the birth-weight-for-gestational-age reference curve, affects 23.8% or approximately 30 million newborns per year, profoundly influencing growth, survival, and physical and mental capacity in childhood (*24*). It also has major public health implications in view of the increased risk of developing diet-related chronic diseases later in life (*25–31*).

Given the rapidity with which traditional diets and lifestyles are changing in many developing countries, it is not surprising that food insecurity and undernutrition persist in the same countries where chronic diseases are emerging as a major epidemic. The global epidemic of obesity, with its attendant comorbidities—heart disease, hypertension, stroke, and diabetes—is not a problem limited to industrialized countries (*32*). Children are in a similar situation; a disturbing increase in the prevalence of overweight among this group has taken place over the past twenty years in developing countries as diverse as India, Mexico, Nigeria and Tunisia (*33*). The increasing prevalence of obesity in developing countries also indicates that physical inactivity is an increasing problem in those countries as well.

In the past, undernutrition and chronic diseases were seen as two totally separate problems, despite being present simultaneously. This dichotomy has obstructed effective action to curb the advancing epidemic of chronic diseases. For example, the prevailing approach of measuring child undernutrition on the basis of the underweight indicator (weight-for-age) can lead to gross underestimation of the presence of obesity in populations that have a high prevalence of stunting. Use of this indicator could lead aid programmes to feed apparently underweight people, with the undesirable outcome of further aggravating obesity. In Latin America, close to 90 million people are beneficiaries of food programmes (*34*) but that group actually comprises only 10 million truly underweight people (after correcting for height). The two facets of nutrition-related problems need to be brought together and treated in the context of the whole spectrum of malnutrition.

2.3 A common approach to diet-related and nutrition-related diseases

The root causes of malnutrition include poverty and inequity. Eliminating these causes requires political and social action of which nutritional programmes can be only one aspect. Sufficient, safe and varied food supplies not only prevent malnutrition but also reduce the risk of chronic diseases. It is well known that nutritional deficiency increases the risk of common infectious diseases, notably of childhood, and vice versa (*35, 36*). There is, therefore, complementarity in terms of public health approaches and public policy priorities, between policies and programmes designed to prevent chronic diseases and those designed to prevent other diet-related and nutrition-related diseases. The double burden of disease is most effectively lifted by a range of integrated policies and programmes. Such a common approach is the key to action in countries where modest public health budgets will inevitably remain mostly devoted to prevention of deficiency and infection. Indeed, there is no country, however

privileged, in which combating deficiency and infection are no longer public health priorities. High-income countries accustomed to programmes designed to prevent chronic diseases can amplify the effectiveness of the programmes by applying them to the prevention of nutritional deficiency and food-related infectious diseases.

Guidelines designed to give equal priority to the prevention of nutritional deficiency and chronic diseases, have already been established for the Latin American region (*37*). Recent recommendations to prevent cancer are reckoned also to reduce the risk of nutritional deficiency and food-related infectious diseases (*38*), and dietary guidelines for the Brazilian population give equal priority to the prevention and control of nutritional deficiency, food-related infectious diseases, and chronic diseases (*39*).

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3. Global and regional food consumption patterns and trends

3.1 Introduction

Promoting healthy diets and lifestyles to reduce the global burden of NCDs requires a multi-sectoral approach, hence the involvement of the various relevant sectors in societies. The agriculture and food sector figures prominently in this enterprise and must be given due importance in any consideration of the promotion of healthy diets for individuals and population groups. Food strategies must not merely be directed at ensuring food security for all, but must also achieve the consumption of adequate quantities of safe and good quality foods that together make up a healthy diet. Any recommendation to that effect will have implications for all components in the food chain. Hence it may be appropriate at this juncture to examine trends in consumption patterns world wide and deliberate on the potential of the food and agriculture sector to meet the demands and challenges posed by this Report.

Economic development is normally accompanied by improvements in a country's food supply and the gradual elimination of dietary deficiencies thus improving the overall nutritional status of the country's population. Furthermore, it also ensues qualitative changes in the production, processing, distribution and marketing of food. Increasing urbanisation will in turn have consequences for the dietary patterns and lifestyles of individuals, not all of which are positive. Changes in diets, patterns of work and leisure, are now already contributing to the causal factors underlying NCDs even in the poorest countries. The pace of this change, often referred to as 'nutrition transition', seems to be accelerating even in the lower- and middle-income countries.

Nutrition transition is characterised by both quantitative and qualitative changes in the diet which are occurring faster now than in the past. The adverse dietary changes include shifts in the structure of the diet towards a higher energy density diet with a greater role for fat and added sugar in foods, greater saturated fat intake mostly from animal sources, reduced intakes of complex carbohydrates and fibre, and reduced fruit and vegetable intakes (1). These dietary changes are compounded by life style changes that reflect reduced physical activity at work and during leisure time (2). At the same time however, poor countries continue to face food shortages and nutrient inadequacies and may not yet demonstrate the features outlined earlier in their diets.

Diets evolve over time, influenced by many factors and complex interactions. Income, prices, individual preferences and beliefs, cultural traditions, as well as geographical, environmental, social and economic factors all interact in a complex manner to shape dietary consumption patterns.

Data on the national availability of the main food commodities give a valuable insight into diets and their evolution over time. The Food and Agricultural Organization's (FAO) produces annual Food Balance Sheets (FBS) which are national data on food availability (for almost all commodities and for nearly all countries). Food balance sheets provide a complete picture of supply (including production, imports, stock changes and exports) and utilization (final demand in the form of food use and industrial non-food use, and intermediate demand such as animal feed and seed use, and waste) by commodity. From such data, average per capita supply of macronutrients, i.e. energy, protein and fats, can be derived for all food commodities. Such average per capita supplies are derived from national data, and may not correspond to actual per capita availability, which is determined by many other factors such as inequality in access to food. Likewise, these data refer to 'average food available for consumption' which, for a number of reasons (e.g. waste at the household level) is not equal to average food intake or average food consumption. In the remainder of this Chapter therefore, the terms 'food consumption' or 'food intake' should be read as 'food available for consumption'.
Actual food availability may vary by region, socioeconomic level and season. Certain difficulties are encountered when estimating trade, production and stock changes on an annual scale. Hence three-year averages are calculated in order to reduce these errors. The FAO statistical database (FAOSTAT), being based on national data, does not provide information on the distribution of food within countries, or within communities and households.

3.2 Developments in the availability of dietary energy

Food consumption expressed in kcals/capita/day is a key variable used for measuring and evaluating the evolution of

the global and regional food situation. A more appropriate term for this variable would be 'national average apparent food consumption' since the data come from national food balance sheets rather than from food consumption surveys. Analysis of FAOSTAT data shows that dietary energy measured in kcals per capita per day has been steadily increasing on a worldwide basis. Availability of calories per capita from the mid-1960s to 1997/99 increased globally by approximately 450 kcal/capita/day and by over 600 kcal/capita/day in developing countries (Table 1). However, this change has not been equal across regions. Per capita supply of calories has remained almost stagnant in sub-Saharan Africa and has recently been showing a decreasing trend in the transition countries. In contrast, the per capita supply of energy has risen dramatically in East Asia (by almost 1000 kcal/capita/day, mainly in China) and in the Near East/North Africa (by over 700 kcal/capita/day).

Table 1

Global and regional per capita food consumption (kcal/capita/day)

	1964/66	**1974/76**	**1984/86**	**1997/99**	**2015**	**2030**
World	2 358	2 435	2 655	2 803	2 940	3 050
Developing countries	2 054	2 152	2 450	2 681	2 850	2 980
Sub-Saharan Africa	2 058	2 079	2 057	2 195	2 360	2 540
Near East/North Africa	2 290	2 591	2 953	3 006	3 090	3 170
Latin America and the Caribbean	2 393	2 546	2 689	2 824	2 980	3 140
South Asia	2 017	1 986	2 205	2 403	2 700	2 900
East Asia	1 957	2 105	2 559	2 921	3 060	3 190
Industrial countries	2 947	3 065	3 206	3 380	3 440	3 500
Transition countries	3 222	3 385	3 379	2 906	3 060	3 180

Source: Reference 3.

In short, it would appear that the world has made significant progress in raising food consumption per person. The increase in the world average consumption would have been higher but for the declines in the transition economies in the 1990s, although it is generally agreed that the declines seen in the transition economies are likely to revert back soon. This growth was accompanied by significant structural changes and diets shifted towards more livestock products, vegetable oils, etc. and away from staples such as roots and tubers (4). Table 1 shows that the current energy intakes (kcal/capita/day) vary from 2681 in developing countries, to 2906 in transition countries and 3380 in industrialized countries. Data shown in Table 2 also suggest that per capita energy supply in transition countries has declined from both animal and vegetable sources while it has increased in the developing and industrialized countries.

Table 2

Vegetable and animal sources of energy in the diet (kcal/capita/day)

	1967/69			1977/79			1987/89			1997/99		
	T	V	A	T	V	A	T	V	A	T	V	A
Developing countries	2059	1898	161	2254	2070	184	2490	2248	242	2681	2344	337
Transition countries	3287	2507	780	3400	2507	893	3396	2455	941	2906	2235	671
Industrialized countries	3003	2132	871	3112	2206	906	3283	2333	950	3380	2437	943

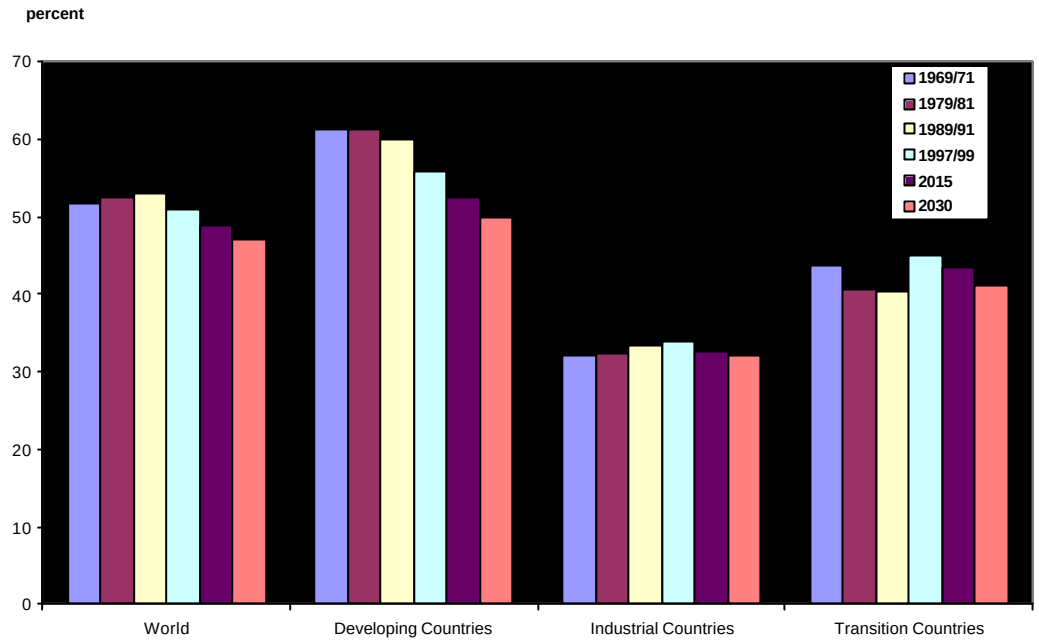
Note: T = total kcal, V = kcal of vegetable origin, A = kcal of animal origin (including fish products).

Source: FAOSTAT.

Similar trends are evident for protein availability which has increased for both developing and industrialized countries, but decreased for transition countries. Although the global supply of protein has been increasing, the distribution of the protein supply is unequal. Per capita supply of vegetable protein is slightly higher in developing countries, while supply of animal protein is three times higher in industrialized countries.

Figure 1

The share of dietary energy derived from cereals

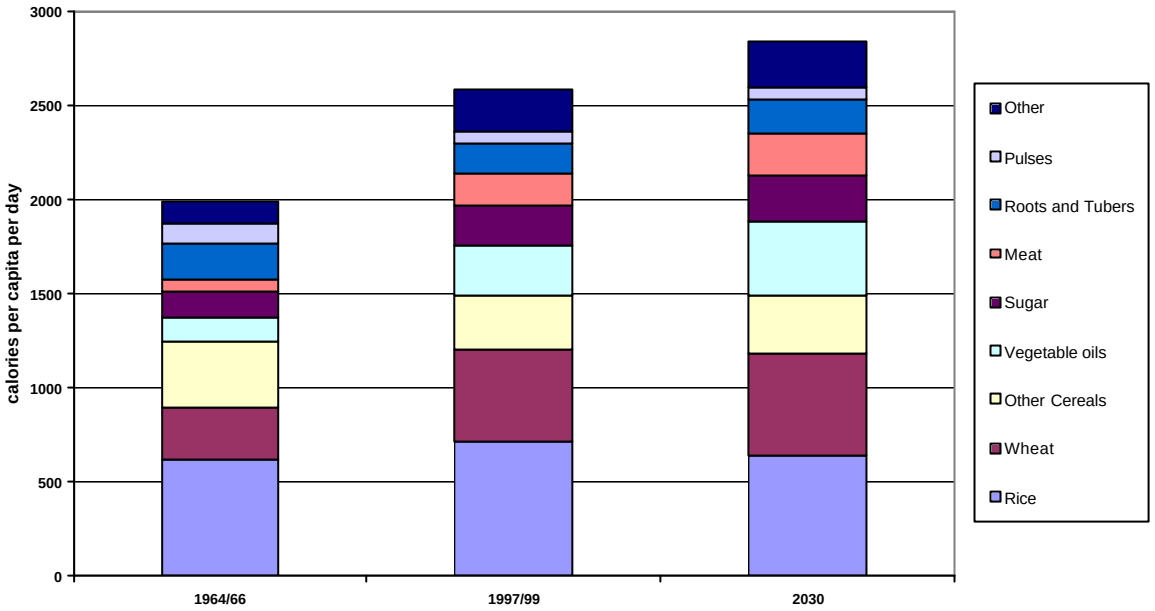


Source: Reference 3

Globally, the share of dietary energy supplied from cereals appears to have remained relatively stable over time, representing about 50 percent of dietary energy supply. Recently however, subtle changes appear to be taking place (Figure 1). A closer analysis of the percent of dietary energy supplied from cereals shows a decrease from 30 percent to 26-27 percent in industrialized countries. An even more rapid shift appears to be taking place in developing countries where the share of energy derived from cereals has fallen from 60 percent to 54 percent in a period of only ten years. Much of this is attributed to cereals, particularly wheat and rice, becoming less preferred foods in middle income countries like Brazil and China, a trend likely to continue over the next 30 years or so. Figure 2 shows the structural changes in the diet of developing countries over the last 3 to 4 decades and FAO's projections to the year 2030 (3).

Figure 2

Calories from major commodities in developing countries



Source: Reference 3

3.3 Availability and changes in dietary fat consumption

The increase in the quantity and quality of the fats consumed in the diet is an important feature of nutrition transition reflected in the national diets of countries. There are large variations across the regions of the world in the amount of total fats (i.e. fats in foods and added fats and oils) available for human consumption. The lowest quantities consumed are recorded in Africa while the highest consumption occurs in parts of North America and Europe. The important point is that there has been a remarkable increase in the intake of dietary fats over the last three decades (Table 3) and that this increase has taken place practically everywhere except in Africa, where consumption levels have stagnated. Per capita supply of fat from animal foods has increased by 14 and 4 grams per capita in developing and industrialized countries respectively, while there has been a decrease of 9 grams per capita in transition countries.

Table 3

Trends in dietary supply of fat (grams per capita per day)

	grams of fat/capita/day				
Region	1967/69	1977/79	1987/89	1997/99	change 1967/69-1997/99
World	52.50	57.15	66.60	73.57	21.07
North Africa	43.97	58.43	65.23	63.93	19.96
Sub-Saharan Africa	41.47	43.03	41.33	44.47	3.00
East and South-East Asia	27.77	32.27	43.90	51.93	24.16
South Asia	29.07	32.37	38.86	45.53	16.46
China	23.23	27.13	47.53	78.50	55.27
Near East	51.20	62.10	73.50	80.63	29.43
Latin America and Caribbean	54.00	64.87	73.40	80.63	26.63
North America	116.90	124.93	138.30	143.00	26.10
European Union	117.20	127.93	142.8	149.03	31.83
Oceania	101.57	101.97	112.83	110.67	9.10
Eastern Europe	90.47	110.53	116.0	104.67	14.20

Source: FAOSTAT.

The increase in dietary fat supply worldwide exceeds the increase in dietary protein supply. The average global supply of fat (grams per capita per day) has increased by 21 grams since 1967/69. This increase in availability (grams per capita per day) has been most pronounced in East Asia, the European Union and the America's. The percentage of energy contributed by the dietary fats exceeds the level of 30 percent in the industrialized regions, but also in nearly all other regions this share is increasing.

The fat to energy ratio (FER) is defined as the share (in percent) of the energy derived from fat in the total supply of energy in kcal. Country specific analysis of FAO data for 1988/90 (5) found a range for the FER of 7- 46 percent. Nineteen countries fell below the minimum recommendation of 15 percent dietary energy supply from fat, with the majority of these countries in sub-Saharan Africa and the remainder in South Asia. Twenty-four countries were above the 35 percent maximum, with the majority of these countries in Western Europe and North America. It is useful to note that limitations of the Food Balance data may contribute to much of this variation in the FER between countries. For instance, in countries like Malaysia with abundant availability of vegetable oils at low prices, Food Balance data may not reflect real consumption at the individual household level.

Rising incomes in the developing world have also meant an increase in the availability and consumption of energy dense high-fat diets. Food balance data can be used to examine the shift in the proportion of energy from fat over time and its relationship to increasing incomes (6). In 1961/63, a diet providing 20 percent energy from fat was

associated only with countries having at least a per capita GNP of US$1475. However, by 1990, even poor countries having a GNP of only US$750 per capita had access to a similar diet comprising 20 percent of energy from fat (both GNP values in 1993 US$). This change was mainly the result of an increase in the consumption of vegetable fats by poor countries, while smaller increases occurred in middle- and high-income countries. By 1990, vegetable fats accounted for a greater proportion of dietary energy than animal fats for countries in the lowest per capita income category. Changes in edible vegetable oil supply, in prices and in consumption equally affected rich and poor countries, although the net impact was relatively much greater in low-income countries. An equally large and important shift in the proportion of energy from added sugar in the diets of lower-income countries was also a feature of the nutrition transition (1).

Examinations of the purchasing habits of people aimed at understanding the relationship between level of education or income, and the different amounts or types of commodities purchased and at different points in time were also revealing. Research conducted in China showed that there have been profound shifts in the purchasing practices with income over time during the last decade. Studies show how extra income in China affects the poor differentially than the rich, enhancing the fat intake of the poor more than the rich (7).

A variable proportion of these fat calories are provided by saturated fatty acids. Only in the two of the most affluent regions (in parts of North America and Europe) is the intake of saturated fat at or above 10 percent of energy intake level. In other less affluent regions, the proportion of dietary energy contributed by the saturated fatty acids is lower, ranging from 5 to 8 percent, and generally not changing much over time. National dietary surveys conducted in some countries confirm these Food Balance Sheets data. The ratio of dietary fat from animal sources is a key indicator since foods from animal sources are high in saturated fat. Data sets used to calculate country specific FERs can also be used to calculate proportions of animal fat in total fat. This analysis indicated that the proportion of animal in total fat was lower than 10 percent in some countries (Sao Tome and Principe, Sierra Leone, Nigeria, Mozambique and Zaire), while it is above 75 percent in some other countries (Mongolia, Denmark, Poland, Finland, Hungary and Uruguay). These findings are not strictly divided along economic lines, as not all of the countries in the high range represent the most affluent countries. Country specific food availability and cultural dietary preferences and norms determine to some extent these patterns.

The types of edible oils used in developing countries are also changing with the increasing use of hardened margarines (rich in trans fatty acids) that do not need to be refrigerated. Palm oil is becoming the main edible oil of the diets of much of South-East Asia and is likely to remain the major source in the coming years. Currently, palm oil consumption is low and the FER ranges between 15 and 18 percent. At this low level of consumption, the saturated fatty acid (SFA) content of the diet reaches only 4 to 8 percent. Potential developments in the edible oil sector affect all stages from plant breeding to processing methods including blending of oils aimed at producing edible oils that have a healthy fatty acid composition.

Olive oil is important edible oil consumed largely in the Mediterranean region. Its production has been driven by rising demand which has increasingly shifted olive cultivation from traditional farms to intensive cultivation. There is some concern that the intensive cultivation of olives may have adverse environmental impacts such as soil erosion and desertification (8). Potential developments in the edible oil sector concern plant breeding and processing methods, including blending of oils aimed at producing edible oils that have a healthy fatty acid composition. At the same time agricultural production methods are being developed to ensure less harmful impacts on the environment.

3.4 Availability and changes in consumption of animal products

There has been an increasing pressure on the livestock sector to meet the growing demand for high value animal protein. The world's livestock sector is growing at an unprecedented rate and the driving force behind this enormous surge is a combination of population growth, rising incomes and urbanisation. There is a strong positive relationship between the level of income and the consumption of animal protein, with the consumption of meat, milk and eggs increasing relatively compared to the consumption of staple foods. Due to the strong decline in prices, developing countries embark on higher meat consumption at much lower GDP levels than industrialised countries could.

Urbanisation is a major driving force influencing global demand for livestock products. Urbanisation stimulates improvements in infrastructure, including cold chains, which permit trade in perishable goods. Compared to the less diversified diets of the rural communities, city dwellers have a varied diet rich in animal proteins and fats and

characterised by higher consumption of meat, poultry, milk and other dairy products. Table 4 shows levels of per capita consumption of livestock products in different regions and country groups.

Table 4
Per capita consumption of livestock products (kg per year)

	Meat			Milk		
Region	1964 /1966	1997 /1999	2030	1964 /1966	1997 /1999	2030
World	24.2	36.4	45.3	73.9	78.1	89.5
Developing countries	10.2	25.5	36.7	28.0	44.6	65.8
Sub-Saharan Africa	9.9	9.4	13.4	28.5	29.1	33.8
Near East & North Africa	11.9	21.2	35.0	68.6	72.3	89.9
Latin America & Caribbean	31.7	53.8	76.6	80.1	110.2	139.8
South Asia	3.9	5.3	11.7	37.0	67.5	106.9
East Asia	8.7	37.7	58.5	3.6	10.0	17.8
Industrialised countries	61.5	88.2	100.1	185.5	212.2	221.0
Transition countries	42.5	46.2	60.7	156.6	159.1	178.7

Source: Reference 4

Diets become richer and more diverse and the high value protein that the livestock sector offers improves the nutrition of the vast majority of the world. Livestock products not only provide high value protein but are also important sources of a wide range of essential micronutrients, in particular minerals like iron, zinc and vitamins such as vitamin A. For the large majority of people in the world, particularly in developing countries, livestock products remain a desired food for nutritional value and taste. However excessive consumption of animal products in some countries and social classes can lead to excessive intakes of fat.

The excessive demand for livestock products is also likely to have an undesirable impact on the environment. There will be more large-scale, industrial production, located close to urban centres, with associated environmental and public health risks. There has been a remarkable increase in the consumption of animal products in countries such as Brazil and China although the levels are still well below the levels of consumption in North American and most other industrialized countries. With increasing numbers of people meat production is projected to increase from 218 million tons in 1997/99 to 376 million tons per year by the year 2030. Attempts have been made to estimate the environmental impact of industrial type livestock production. For instance it has been estimated that the number of people fed in a year per hectare ranges from 22 for potatoes and 19 for rice down to 1 and 2 people respectively for beef and lamb (9). The low energy conversion ratio from feed to meat is another concern since some of the cereal grain food produced is diverted to livestock production. Likewise, land and water requirements for meat production are also are likely to become a major concern with the increasing demand for animal products resulting in more intensive livestock production systems (10).

3.5 Availability and consumption of fish

Despite fluctuations in supply and demand, caused by the changing state of fisheries resources, the economic climate and environmental conditions, fisheries, including aquaculture, remain very important as a source of food, employment and revenue in many countries and communities (11). After the remarkable increase in both marine and inland capture of fish during the 1950s and 60s, since the 1970s world fisheries production has levelled of. This levelling off of the total catch follows the general trend of most of the world's fishing areas, which have apparently

reached their maximum potential for fisheries production, with the majority of stocks being fully exploited. It is therefore very unlikely that substantial increases in total catch will be obtained. In contrast, growth in aquaculture production has shown the opposite tendency. Starting from an insignificant total production, inland and marine aquaculture production has been growing at a remarkable rate and has been substituting for the reduction in ocean catch of fish.

The total food fish supply and hence its consumption has been growing at a rate of 3.6 percent per annum since 1961, while the world's population has been expanding at 1.8 percent per annum. The proteins derived from fish, crustaceans and molluscs account for between 13.8 and 16.5 percent of the animal protein intake of the human population. The average apparent per capita consumption increased from about 9 kg per annum in the early 1960s to 16 kg in 1997. The per capita availability of fish and fishery products has therefore nearly doubled in 40 years, outpacing population growth.

As well as income-related variations, the role of fish in nutrition shows marked continental, regional and national differences. In industrialized countries, where diets generally contain a more diversified range of animal proteins, a rise in per capita provision from 19.7 to 27.7 kg seems to have occurred. This represents a growth rate close to 1 percent per annum. In this group of countries, fish contributed an increasing share of total protein intake until 1989 (accounting for between 6.5 and 8.5 percent), but its importance has gradually declined since then and, in 1997, its percentage contribution was back to the level prevailing in the mid-1980s. In the early 1960s, per capita fish supply in low-income food-deficit countries was, on average, only 30 percent of that of the richest countries. This gap has been gradually reduced however, and in 1997 average fish consumption in these countries was 70 percent of that of the more affluent economies. Despite the relatively low consumption by weight in these countries, the contribution of fish to total animal protein intake is considerable (nearly 20 percent). Over the last four decades, however, the share of fish proteins in animal proteins has slightly declined due to faster growth in the consumption of other animal products.

Currently, two-thirds of the total food fish supply is obtained from capture fisheries in marine and inland waters, while the remaining one-third is derived from aquaculture. The contribution of inland and marine capture fisheries to per capita food supply has stabilized (at 10 to11 kg per capita in the period 1984-1998). Recent increases in per capita availability have, therefore, been obtained from aquaculture production, from both traditional rural aquaculture and intensive commercial aquaculture of high-value species.

Fish contributes up to 180 calories per capita per day, but reaches such high levels only in a few countries where there is a lack of alternative protein foods grown locally or where there is a strong preference for fish (examples are Japan, Iceland and some small island states). More typically, fish provides about 20 to 30 calories per day. Fish proteins are essential and critical in the diet of some densely populated countries, where the total protein intake level may be low, and it is very important in the diets of many other countries. Worldwide, about 1 billion people rely on fish as their main source of animal proteins. Dependence on fish is usually higher in coastal than in inland areas. About 20 percent of the world's population derives at least 20 percent of its animal protein intake from fish and some small island states depend almost exclusively on fish.

Recommending the increased consumption of fish is another area where the feasibility of dietary recommendations needs to be balanced against concerns for sustainability of marine stocks and the potential depletion of this important marine source of high quality nutritious food. Added to this is the concern that a significant proportion of the world fish catch is transformed into fish meal and used as animal feed in industrial type livestock production and thus not available for human consumption.

3.6 Availability and consumption of fruits and vegetables

Consumption of fruits and vegetables plays an important role in providing a diversified and nutritious diet. However, the low consumption of fruits and vegetables in many regions of the developing world is a persistent phenomenon, confirmed by the findings of food consumption surveys. Nationally representative surveys in India (12) indicate a steady level of consumption of only 120-140 g/d, with about another 100 grams coming from roots and tubers and some 40 grams from pulses. This may not be true for urban populations in India with rising incomes and greater access to a diverse and varied diet. In China, on the other hand, a country that is undergoing rapid economic growth and transition, the amount of fruit and vegetables has increased to 369 g/d in 1992.

Only a small and negligible minority of the world's population consumes at present the generally recommended high average intakes of fruits and vegetables. In 1998, only 6 of the 14 WHO regions had an availability of fruit and vegetables equal to or greater than the earlier recommended intakes of 400 g/d. The relatively favourable situation in 1998 appears to have evolved from a markedly less favourable position in previous years, as evidenced by the great increase in vegetable availability recorded between 1990 and 1998 for most of the regions. On the other hand, the availability of fruit generally decreased between 1990 and 1998 in most regions of the world. The increase in urbanisation globally is another challenge. Increasing urbanization will distance people from the primary food production, and influence both the availability and access to a varied and nutritious diet with enough fruits and vegetables to the urban poor. On the other hand, it may facilitate the achievement of other recommendations, as those who can afford it, can have access to a diverse and varied diet. Investment in peri-urban horticulture may provide an opportunity to meet the increasing demand for these healthy foods in the daily diet.

Global trends in production and supply of vegetables indicate that the current production and consumption of vegetables widely varies among regions as illustrated in Table 5. It should be noted that the production of wild and indigenous vegetables is not taken into account in production statistics and might therefore be underestimated in consumption statistics. The average vegetable supply available per person in the world is 102 kg per person per year in 2000, with the highest level in Asia (116 kg), while the lowest level are found in South America (48 kg) and Africa (52 kg). These figures reflect also the large amount of horticultural produce that is consumed on the farm. Table 5 and Figure 3 illustrate the global and regional variations and trends in the availability of vegetables per person over the last decades.

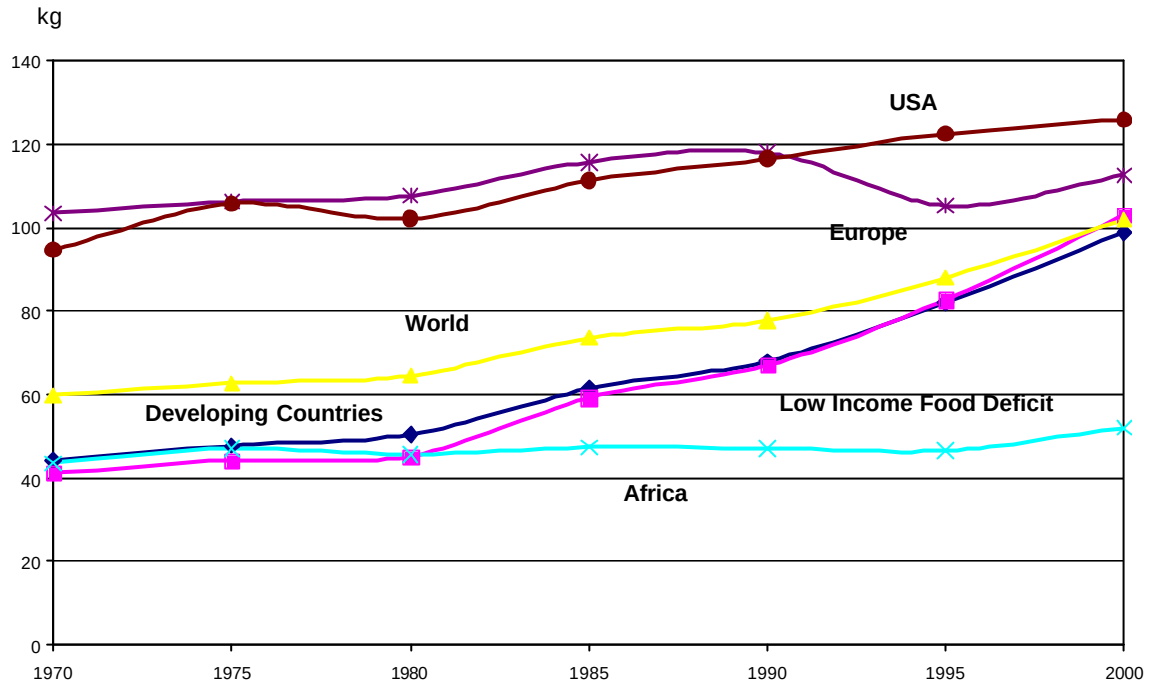
Table 5
Trends in the per capita supply of vegetables

	Supply of vegetables (kg/capita/year)	
	1979	2000
World	66.1	101.9
Developed countries	107.4	112.8
Developing countries	51.1	98.8
Africa	45.4	52.1
North and Central America	88.7	98.3
South America	43.2	47.8
Asia	56.6	116.2
Europe	110.9	112.5
Oceania	71.8	98.7

Source: Reference 13

Figure 3

Trends in the supply of vegetables per capita (by region)



Source: Reference 13

3.7 Future trends in demand, food availability and consumption

In recent years the growth rates of world agricultural production and crop yields have slowed. This has raised fears that the world may not be able to grow enough food and other commodities to ensure that future populations are adequately fed. However, the slowdown has occurred not because of shortages of land or water but rather because demand for agricultural products has also slowed. This is mainly because world population growth rates have been declining since the late 1960s, and fairly high levels of food consumption per person are now being reached in many countries, beyond which further rises will be limited. However it also true that a high share of the world's population remains in poverty and hence lacks the necessary income to translate its needs into effective demand. As a result, the growth in world demand for agricultural products is expected to fall from an average 2.2 percent a year over the past 30 years to 1.5 percent a year for the next 30. In developing countries the slowdown will be more dramatic, from 3.7 percent to 2 percent, partly as a result of China having passed the phase of rapid growth in its demand for food. Global shortages are unlikely, but serious problems already exist at national and local levels and may worsen unless focused efforts are made.

The annual growth rate of world demand for cereals has declined from 2.5 percent a year in the 1970s and 1.9 percent a year in the 1980s to only 1 percent a year in the 1990s. Annual cereal use per person (including animal feeds) peaked in the mid-1980s at 334 kg and has since fallen to 317 kg. The decline is not cause for alarm: it was above all the natural result of slower population growth and shifts in human diets and animal feeds. However, in the 1990s it was accentuated by a number of temporary factors, including serious recessions in the transition countries and some East and Southeast Asian countries.

The growth rate of demand for cereals is expected to rise again to 1.4 percent a year to 2015, slowing to 1.2 percent per year thereafter. In developing countries overall, cereal production is not expected to keep pace with demand. The net cereal deficits of these countries, which amounted to 103 million tonnes or 9 percent of consumption in 1997/99, could rise to 265 million tonnes by 2030, when they will be 14 percent of consumption.

This gap can be bridged by increased surpluses from traditional grain exporters, and by new exports from the transition countries, which are expected to shift from being net importers to being net exporters. Oil crops have seen the fastest increase in area of any crop sector, expanding by 75 million hectares from the mid-1970s until the end of the 1990s, while cereal area fell by 28 million hectares over the same period. Future per capita consumption of oil crops is expected to rise more rapidly than that of cereals. These crops will account for 45 out of every 100 extra kilocalories added to average diets in developing countries between now and 2030.

There are three main sources of growth in crop production: expanding the land area, increasing the frequency with which it is cropped (often through irrigation), and boosting yields. It has been suggested that we may be approaching the ceiling of what is possible for all three sources. A detailed examination of production potentials does not support this view at the global level, although in some countries, and even in whole regions, serious problems already exist and could deepen.

Diets in developing countries are changing as incomes rise. The share of staples, such as cereals, roots and tubers, is declining, while that of meat, dairy products and oil crops is rising. Between 1964/66 and 1997/99, per capita meat consumption in developing countries rose by 150 percent and that of milk and dairy products by 60 percent. By 2030, per capita consumption of livestock products could rise by a further 44 percent. As in the past, poultry consumption will grow fastest. Productivity improvements are likely to be a major source of growth. Milk yields should improve, while breeding and improved management will increase average carcass weights and off-take rates. This will allow increased production with lower growth in animal numbers, and a corresponding slowdown in the growth of environmental damage from grazing or wastes.

In developing countries, demand will grow faster than production, producing a growing trade deficit. In meat products this will rise steeply, from 1.2 million tonnes a year in 1997/99 to 5.9 million tonnes in 2030 (despite growing meat exports from Latin America), while in milk and dairy products the rise will be less steep but still considerable, from 20 million to 39 million tonnes a year. An increasing share of livestock production will probably come from industrial enterprises. In recent years production from this sector has grown twice as fast as that from more traditional mixed farming systems and more than six times faster than from grazing systems.

World fisheries production has kept ahead of population growth over the past three decades. Total fish production almost doubled, from 65 million tonnes in 1970 to 125 million tonnes in 1999, when world average intake of fish, crustaceans and molluscs reached 16.3 kg per person. By 2030, annual fish consumption is likely to rise to some 150 to160 million tonnes, or between 19 and 20 kg per person. This amount is significantly lower than the potential demand, because environmental factors are expected to limit supply. By the turn of the century, three-quarters of ocean fish stocks were over fished, depleted or exploited up to their maximum sustainable yield. Further growth in the marine catch can be only modest. During the 1990s the marine catch leveled out at 80 to 85 million tonnes a year, not far from its maximum sustainable yield.

Aquaculture compensated for this marine slowdown, doubling its share of world fish production during the 1990s. It will continue to grow rapidly, at rates of 5 to 7 percent a year up to 2015. In all sectors of fishing it will be essential to pursue forms of management conducive to sustainable exploitation, especially for resources under common ownership or no ownership.

3.8 Conclusions

There are a number of conclusions that can be drawn from the preceding discussion.

- Most of the information on food consumption has been hitherto obtained from national Food Balance data. In order to understand the relationship between food consumption patterns, diets and the emergence of NCDs, it is crucial that to obtain more reliable information on actual food consumption patterns and changing trends based on representative consumption surveys.

- Then, there is a need to monitor whether the guidelines developed in this report will influence the behaviour of consumers and to what extent consumers will change their diets (and lifestyles) towards more healthy patterns.

- Furthermore, assuming that the new guidelines developed in this report will be adopted, the implications for agriculture, livestock, fisheries and horticulture will have to be assessed and action taken to deal with potential future demands of an increasing and more affluent population. To meet the specified levels of consumption, new strategies may need to be developed.

For example, a realistic approach to the implementation of the recommendation concerning high average intakes of fruits and vegetables, requires that attention be paid to a number of crucial issues such as where would the large quantities needed be produced and how can the infrastructure be developed permitting trade in these perishable products, and would large production of horticultural products be sustainable.

- A number of more novel issues will need to be dealt with, such as: the quality effects on NCDs of intensive production systems, both positive and negative in terms of health, e.g. nitrite in vegetables, heavy metals in irrigation water and manure, pesticide use, but possibly also better dietary quality (e.g. leaner meats in intensive poultry production); the effects of longer food chains, in particular of longer storage and transport routes, such as the higher risk of deterioration (even if most of this may be bacterial and hence not be a factor in chronic diseases), use and misuse of conserving agents and contaminants; and, the effects of changes in varietal composition and diversity of consumption patterns, e.g. the loss of traditional crop varieties and perhaps, even more so, the declining use of foods from 'wild' sources, etc.

- Trade issues need to be considered in the context of improving diet, nutrition and the prevention of chronic diseases. Trade has an important role to play in improving food and nutrition security. On the import side, lower trade barriers reduce domestic food prices, increase the purchasing power of consumers and afford them a greater variety of food products. Freer trade can thus help enhance the availability and affordability of food and contribute to a better-balanced diet. On the export side, access to markets abroad creates new income opportunities for domestic farmers and processors. Developing countries' farmers stand to benefit particularly from the removal of trade barriers for commodities like sugar, fruits and vegetables as well as tropical beverages, which are products for which they have a comparative advantage.

- The impact that agricultural policy, particularly subsidies, has on the structure of production, processing and marketing systems and, ultimately, on the availability of foods that support healthy food consumption patterns should not be overlooked.

All these issues and challenges need to be addressed in a pragmatic and inter-sectoral manner. All sectors in the food chain, from "farm to table", will need to be involved if the food system is to respond to the challenges posed by the need for changes in diets to cope with the burgeoning epidemic of NCDs.

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4. Diet, nutrition and chronic diseases in context

4.1 Introduction

The diets people eat, in all their cultural variety, define to a large extent people's health, growth and development. Risk behaviours, such as tobacco use and physical inactivity, modify the result for better or worse. All this takes place in a social, cultural, political and economic environment that can aggravate the health of populations unless active measures are taken to make the environment a health-promoting one.

Although this report has taken a disease approach for convenience, the Expert Consultation was mindful in all its discussions that diet, nutrition and physical activity do not take place in a vacuum. Since the earlier report (*1*), there have been great advances in basic research, considerable expansion of knowledge, and much community and international experience in the prevention and control of chronic diseases. At the same time, the human genome has been mapped and must now enter any discussion of chronic disease.

Concurrently there has been a return to the concept of the basic life course, of the continuity of human lives from fetus to old age. The influences in the womb work differently from later influences, but clearly have a strong influence on the subsequent manifestation of chronic disease. The known risk factors are now recognized as being amenable to alleviation throughout life, even into old age. The continuity of the life course is seen in the way that both undernutrition and overnutrition (as well as a host of other factors) play a role in the development of chronic disease. The effects of man-made and natural environments (and the interaction between the two) on the development of chronic diseases are increasingly recognized. Such factors are also being recognized as happening further and further "upstream" in the chain of events predisposing humans to chronic disease. All these broadening perceptions not only give a clearer picture of what is happening in the current epidemic of chronic diseases, but also present many opportunities to address them. The identities of those affected are now better recognized: those most disadvantaged in more affluent countries, and—in numerical terms far greater—the populations of the developing and transitional worlds.

There is a continuity in the influences contributing to chronic disease development, and thus also to the opportunities for prevention. These influences include the life course; the microscopic environment of the gene to macroscopic urban and rural environments; the impact of social and political events in one sphere affecting the health and diet of populations far distant; and the way in which already stretched agriculture and oceanic systems will affect the choices available and the recommendations that can be made. For chronic diseases, risks occur at all ages; conversely, all ages are part of the continuum of opportunities for their prevention and control. Both undernutrition and overnutrition are negative influences in terms of disease development, and possibly a combination is even worse; consequently the developing world needs additional targeting. Those with least power need different preventive approaches from the more affluent. Work has to start with the individual risk factors, but, critically, attempts at prevention and health promotion must also take account of the wider social, political and economic environment. Economics, industry, consumer groups and advertising all must be included in the prevention equation.

4.2 Diet, nutrition and the prevention of chronic diseases through the life course

The rapidly increasing burden of chronic diseases is a key determinant of global public health. Already 79% of deaths attributable to chronic diseases are occurring in developing countries, predominantly in middle-aged men (*2*). There is increasing evidence that chronic disease risks begin in fetal life and continue into old age (*3–9*). Adult chronic disease, therefore, reflects cumulative differential lifetime exposures to damaging physical and social environments.

For these reasons a life-course approach that captures both the cumulative risk and the many opportunities to intervene was adopted. While accepting the imperceptible progression from one life stage to the next, five stages were identified for convenience. These are: fetal development and the maternal environment; infancy; childhood and adolescence; adulthood; and ageing and older people.

4.2.1 *Fetal development and the maternal environment*

The four relevant factors in fetal life are: (i) intrauterine growth retardation (IUGR); (ii) premature delivery of a normal growth for gestational age fetus; (iii) overnutrition in utero; and (iv) intergenerational factors. There is considerable evidence, mostly from developed countries, that IUGR is associated with an increased risk of coronary heart disease, stroke, diabetes and raised blood pressure (*9–20*). It may rather be the pattern of growth, i.e. restricted fetal growth followed by very rapid postnatal catch-up growth, that is important in the underlying disease pathways. On the other hand, large size at birth (macrosomia) is also associated with an increased risk of diabetes and cardiovascular disease (*16, 21*). Among the adult population in India, an association was found between impaired glucose tolerance and high ponderal index (i.e. fatness) at birth (*22*). In Pima Indians, a U-shaped relationship to birth weight was found, whereas no such relationship was found amongst Mexican Americans (*21, 23*). Higher birth weight has also been related to an increased risk of breast and other cancers (*24*).

Thus, the evidence suggests that optimal birth weight and length distribution should be considered, not only in terms of immediate morbidity and mortality but also in regard to long-term outcomes such as susceptibility to diet-related chronic disease later in life.

4.2.2 *Infancy*

Retarded growth in infancy can be reflected in failure to gain weight and failure to gain height, while excessive weight or height gain ("crossing the centiles") can also be a factor in later incidence of chronic disease. An association between low growth in early infancy (low weight at 1 year) and an increased risk of coronary heart disease (CHD) has also been described, irrespective of size at birth (*3, 25*). Blood pressure has been found to be highest in those with retarded fetal growth and greater weight gain in infancy (*26*). Short stature, a reflection of socioeconomic deprivation in childhood (*27*), is also associated with an increased risk of CHD and stroke, and to some extent, diabetes (*10, 15, 28–34*). The risk of stroke, and cancer mortality at several sites, including breast, uterus and colon, is increased if shorter children display an accelerated growth in height (*35, 36*).

Breastfeeding

There is increasing evidence that among term and pre-term infants, breastfeeding is associated with significantly lower blood pressure levels in childhood (*37, 38*). Consumption of formula instead of breast milk in infancy has also been shown to increase diastolic and mean arterial blood pressure in later life (*37*). Nevertheless, studies with older cohorts (*22*) and the Dutch study of famine (*39*) have not identified such associations. There is increasingly strong evidence suggesting that a lower risk of developing obesity (*40–43*) may be directly related to length of exclusive breastfeeding although it may not become evident until later in their childhood (*44*). Some of the discrepancy may be explained by socioeconomic and maternal education factors confounding the findings.

Data from most, but not all, observational studies of term infants have generally suggested adverse effects of formula consumption on the other risk factors for cardiovascular disease (as well as blood pressure), but little information is available from controlled clinical trials (*45*). Nevertheless, the weight of current evidence indicates adverse effects of formula milk on cardiovascular disease risk factors; this is consistent with the observations of increased mortality among older adults who were fed formula as infants (*45–47*). The risk for several chronic diseases of childhood and adolescence (type 1 diabetes, coeliac disease, some childhood cancers and inflammatory bowel disease) have also been associated with infant feeding on breast-milk substitutes and short-term breastfeeding (*48*).

There has been great interest in the effect of feeding high-cholesterol in early life. Reiser et al. (*49*) proposed the hypothesis that high cholesterol feeding in early life may serve to regulate cholesterol and lipoprotein metabolism in later life. Animal data in support of this hypothesis are limited, but the idea of a possible metabolic imprinting served to trigger several retrospective and prospective studies comparing cholesterol and lipoprotein metabolism in infants fed human milk and those fed formula. Studies in suckling rats have suggested that the presence of cholesterol in the early diet may serve to define a metabolic pattern for lipoproteins and plasma cholesterol that could be of benefit later in life. The study by Mott, Lewis & McGill (*50*) on differential diets in infant baboons, however, provided evidence to the contrary in terms of benefit. Nevertheless, the observation of modified responses of adult cholesterol production rates, bile cholesterol saturation indices, and bile acid turnover, depending on whether the baboons were fed breast milk or formula, served to attract further interest. It was noted that increased

atherosclerotic lesions associated with increased levels of plasma total cholesterol were related to increased dietary cholesterol in early life. No long-term human morbidity and mortality data supporting this notion have been reported.

Short-term human studies have been in part confounded by diversity in solid food weaning regimens, as well as varied composition of fatty acid components of the early diet. The latter are now known to have an impact on circulating lipoprotein cholesterol species (*51*). Mean plasma total cholesterol by age 4 months in infants fed breast milk reached 180 mg/dl or greater, while cholesterol values in infants fed formula tended to remain under 150 mg/dl. In a study by Carlson, De Voe & Barness (*52*), infants receiving predominantly a linoleic acid-enriched oil blend exhibited a mean cholesterol concentration of approximately 110 mg/dl. A separate group of infants in that study receiving predominantly oleic acid had a mean cholesterol concentration of 133 mg/dl. Moreover, infants who were fed breast milk and oleic acid-enriched formula had relatively higher high-density lipoprotein (HDL) cholesterol and apoproteins A-I and A-II compared to the predominantly linoleic acid-enriched diet group. The ratio of low-density lipoprotein (LDL) cholesterol plus very low density lipoprotein (VLDL) cholesterol to HDL cholesterol was lowest for infants receiving the formula in which oleic acid was predominant. Using a similar oleic acid predominant formula, Darmady, Fosbrooke & Lloyd (*53*) reported a mean value of 149 mg/dl at age 4 months, compared to 196 mg/dl in a parallel breast-fed group. Most of those infants then received an uncontrolled mixed diet and cow milk, with no evident differences in plasma cholesterol levels by 12 months, independent of the type of early feeding they had received. A more recent controlled study (*54*) suggests that the specific fatty acid intake plays a predominant role in determining total and LDL cholesterol. The significance of high dietary cholesterol associated with exclusive human milk feeding during the first 4 months of life has no demonstrated adverse effect. Measurements of serum lipoprotein concentrations and LDL receptor activity in infants suggests that it is the fatty acid content rather than the cholesterol in the diet which regulates cholesterol homeostasis. The regulation of endogenous cholesterol synthesis in infants appear to be regulated in a similar manner to that of adult humans (*55, 56*).

4.2.3 *Childhood and adolescence*

An association between low growth in childhood and an increased risk of CHD has also been described, irrespective of size at birth (*3, 25*). Based only on developed country research at this point, this would support the importance of the role of immediate postnatal factors in shaping disease risk. Growth rates in infants in Bangladesh, most of whom had chronic intrauterine undernourishment and were breastfed, were similar to growth rates of breastfed infants in industrialized countries, but catch-up growth was limited and weight at 12 months was largely a function of weight at birth (*57*).

In a study of 11–12 year old Jamaican children (*26*), blood pressure levels were found to be highest in those with retarded fetal growth and greater weight gain between the ages 7 and 11 years. Similar results were found in India (*58*). Low birth weight Indian babies have been described as having a characteristic poor muscle but high fat preservation, so-called "thin-fat" babies. This phenotype persists throughout the postnatal period and is associated with an increased central adiposity in childhood that is linked to the highest risk of raised blood pressure and disease risk (*59–61*). In most studies, the association between low birth weight and high blood pressure has been found to be particularly strong if adjusted to current body size (i.e. BMI), suggesting the importance of weight gain after birth (*62*).

Relative weight in adulthood and weight gain have also been found to be associated with increased risk of cancer of the breast, colon, rectum, prostate and other sites (*36*). Whether there is an independent effect of childhood weight is difficult to determine, as childhood overweight is usually continued into adulthood. Relative weight in adolescence was significantly associated with colon cancer in one retrospective cohort study (*63*). Frankel, Gunnel & Peters (*64*), in the follow-up to an earlier survey by Boyd Orr in the late 1930s, found that for both sexes, after accounting for the confounding effects of social class, there was a significant positive relationship between childhood energy intake and adult cancer mortality. The recent review by the International Agency for Research on Cancer (IARC) in Lyon, France, concluded that there was clear evidence of a relationship between onset of obesity (both early and later) and cancer risk (*65*).

Short stature (including measures of childhood leg length), a reflection of socioeconomic deprivation in childhood, is associated with an increased risk of CHD and stroke, and to some extent diabetes (*10, 15, 28–34*). Given that short stature, and specifically short leg-length, are particularly sensitive indicators of early socioeconomic

deprivation, their association with later disease very likely reflects an association between early undernutrition and infectious disease load (*27, 66*).

Height serves partly as an indicator of socioeconomic and nutritional status in childhood. As has been seen, poor fetal development and poor growth during childhood have been associated with increased cardiovascular disease risk in adulthood, as have indicators of unfavourable social circumstances in childhood. Conversely, a high calorie intake in childhood may be related to an increased risk of cancer in later life (*64*). Height is inversely associated with mortality among men and women from all causes, including coronary heart disease, stroke and respiratory disease (*67*).

Height has also been used as a proxy for usual childhood energy intake, which is particularly related to body mass and the child's activity. However, it is clearly an imperfect proxy because when protein intake is adequate (energy appears to be important in this regard only in the first 3 months of life), genetics will define adult height (*36*). Protein, particularly animal protein, has been shown to have a selective effect in promoting height growth. It has been suggested that childhood obesity is related to excess protein intake and, of course, overweight or obese children tend to be in the upper precentiles for height. Height has been shown to be related to cancer mortality at several sites, including breast, uterus and colon (*36*). The risk of stroke is increased by accelerated growth in height during childhood (*35*). As accelerated growth has been linked to development of hypertension in adult life, this may be the mechanism (plus an association with low socioeconomic status).

There is a higher prevalence of raised blood pressure with low socioeconomic status in adults (*68–74*), but also in children coming from low socioeconomic backgrounds, although the latter is not always associated with higher blood pressure later in life (*10*). Blood pressure has been found to track from childhood to predict hypertension in adulthood, but with stronger tracking seen in older ages of childhood and in adolescence (*75*).

Higher blood pressure in childhood (in combination with other risk factors) causes target organ and anatomical changes associated with cardiovascular risk, including reduction in artery elasticity, increased ventricular size and mass, haemodynamic increase in cardiac output and peripheral resistance (*10, 76, 77*). High blood pressure in children is strongly associated with obesity, in particular central obesity, and clusters and tracks with an adverse serum lipid profile (especially LDL cholesterol) and glucose intolerance (*76, 78, 79*). There may be some ethnic differences, although these often seem to be explained by differences in body mass index. A retrospective mortality follow-up of a survey of family diet and health in the United Kingdom (1937–1939) identified significant associations between childhood energy intake and mortality from cancer (*64*).

The presence and tracking of high blood pressure in children and adolescents occurs against a background of unhealthy lifestyles, including excessive intakes of total and saturated fats, cholesterol and salt, inadequate intakes of potassium, and reduced physical activity, often accompanied by high levels of television viewing (*10*). In adolescents, habitual alcohol and tobacco use contributes to raised blood pressure (*76, 80*).

There are three critical aspects of adolescence that have an impact on chronic diseases: (i) the development of risk factors during this period; (ii) the tracking of risk factors throughout life; and, in terms of prevention, (iii) the development of healthy or unhealthy habits that tend to stay throughout life, for example physical inactivity because of television viewing. In older children and adolescents, habitual alcohol and tobacco use contribute to raised blood pressure and the development of other risk factors in early life, most of which track into adulthood.

The clustering of risk factor variables occurs as early as childhood and adolescence, and is associated with atherosclerosis in young adulthood and thus risk of later cardiovascular disease (*81, 82*). This clustering has been described as the metabolic—or "syndrome X"—clustering of physiological disturbances associated with insulin resistance, including hyperinsulinaemia, impaired glucose tolerance, hypertension, elevated plasma triglyceride and low HDL cholesterol (*83, 84*). Raised serum cholesterol both in middle age and in early life are known to be associated with an increased risk of disease later on. The Johns Hopkins precursor study showed that serum cholesterol levels in adolescents and young white males were strongly related to subsequent risk of cardiovascular disease mortality and morbidity (*85*).

Although the risk of obesity does not apparently increase in adults who were overweight at 1 and 3 years old, the risk rises steadily thereafter, regardless of parental weight (*86*). Tracking has also been reported in China, where overweight children were 2.8 times as likely to become overweight adolescents; conversely, underweight children were 3.6 times as likely to remain underweight as adolescents (*87*). The study found that parental obesity and

underweight, and the children's initial body mass index, dietary fat intake and family income helped predict tracking and changes. However, in a British prospective cohort study, little tracking from childhood overweight to adulthood obesity was found when using a measure of fatness (percentage body fat for age) that was independent of build (*88*). The authors also found that only children obese at 13 years of age had an increased risk of obesity as adults, and that there was no excess adult health risk from childhood or adolescent overweight. Interestingly, they found that in the thinnest children, the more obese they became as adults, the greater was their subsequent risk of developing chronic diseases.

The real concern about these early manifestations of chronic disease, besides the fact that they are occurring earlier and earlier, is that once they have developed they tend to track in that individual throughout life. On the more positive side, there is also evidence that they can be corrected. Overweight and obesity are, however, notoriously difficult to correct after becoming established, and there is an established risk of overweight during childhood persisting into adolescence and adulthood (*89*). Recent analyses (*90, 91*) showed that the later the weight gain in childhood and adolescence, the greater the persistence. More than 60% of overweight children have at least one additional risk factor for cardiovascular disease, such as raised blood pressure, hyperlipidaemia or hyperinsulinaemia, and more than 20% have two or more risk factors (*89*).

Habits leading to noncommunicable disease development during adolescence

It seems increasingly likely that there are widespread effects of early diet on later body composition, physiology and cognition (*45*). Such observations "provide strong support for the recent shift from defining nutritional needs for prevention of acute deficiency symptoms to long-term prevention of morbidity and mortality" (*45*).

Increased birth weight increases the risk of obesity later, but children with low birth weight tend to remain small into adulthood (*89, 92*). In industrialized countries there have been only modest increases in birth weight so the increased levels of obesity described earlier must reflect environmental changes (*89*).

The "obesogenic" environment appears to be largely directed at the adolescent market, making healthy choice that much more difficult. At the same time, exercise patterns have changed and considerable parts of the day are spent sitting at school, in a factory, or in front of a television or computer. Raised blood pressure, impaired glucose tolerance and dyslipidaemia are associated in children and adolescents with unhealthy lifestyles, such as diets containing excessive intakes of fats (especially saturated), cholesterol and salt, an inadequate intake of fibre and potassium, a lack of exercise, and increased television viewing (*10*). Physical inactivity and smoking have been found independently to predict CHD and stroke in later life.

It is increasingly recognized that unhealthy lifestyles do not just appear in adulthood but drive the early development of obesity, dyslipidaemia, high blood pressure, impaired glucose tolerance and associated disease risk. In many countries, perhaps most typified by the USA, changes in family eating patterns and the consumption of fast foods, pre-prepared meals and carbonated drinks, have taken place over the past 30 years (*89*). Likewise, the amount of physical activity has been greatly reduced both at home and in school, as well as by increasing use of mechanized transport.

4.2.4 ***Adulthood***

The three critical questions relating to adulthood were identified as: (i) to what extent do risk factors continue to be important in the development of chronic diseases; (ii) to what extent will modifying such risk factors make a difference to the emergence of disease; and (iii) what is the role of risk factor reduction and modification in secondary prevention and the treatment of those with disease? Reviewing the evidence in a life-course approach demonstrates the importance of the adult phase of life both in terms of the expression of most chronic disease, as well as a critical time for the preventive reduction of risk factors and for increasing effective treatment (*93*).

The most firmly established associations between cardiovascular disease or diabetes and factors in the lifespan are the ones between those diseases and the major known "adult" risk factors, such as tobacco use, obesity, physical inactivity, cholesterol, high blood pressure and alcohol consumption (*94*). The factors that have been confirmed to lead to an increased risk of CHD, stroke and diabetes are: high blood pressure for CHD or stroke (*95, 96*); high

cholesterol (diet) for CHD (*97, 98*), and tobacco use for CHD (*99*). Other associations are robust and consistent, although they have not necessarily been shown to be reversible (*10*): obesity and physical inactivity for CHD, diabetes and stroke (*100–102*); and heavy or binge drinking for CHD and stroke (*99, 103*). Most of the studies are from developed countries, but supporting evidence from developing countries is beginning to emerge, for example India (*104*).

In developed countries, low socioeconomic status is associated with higher risk of cardiovascular disease and diabetes (*105*). As in the affluent industrialized countries, there appears to be an initial preponderance of cardiovascular disease among the higher socioeconomic groups, for example in China (*98*). It is presumed that the disease will progressively shift to the more disadvantaged sectors of society (*10*). There is some evidence that this is already happening, especially among women in low-income groups, for example in Brazil (*106*) and South Africa (*107*), as well as in countries in economic transition such as Morocco (*108*).

Other factors are continually being recognized or proposed. These include the role of high levels of homocysteine, the related factor of low folate, the role of iron (*109*) and others. From a social sciences perspective, Losier (*110*) has suggested that socioeconomic level is less important than a certain stability in the physical and social environment. In other words, an individual's sense of understanding of his or her environment, coupled with control over the course and setting of his or her own life appears to be the most important determinant of health. Marmot (*111*) and others have demonstrated the impact of the wider environment and societal and individual stress on the development of chronic disease.

4.2.5 *Ageing and older people*

There are three critical aspects relating to chronic diseases in the later part of the life-cycle: (i) most chronic diseases will be manifested in this later stage of life; (ii) there is an absolute benefit for ageing individuals and populations in changing risk factors and adopting health-promoting behaviours such as exercise and healthy diets; and (iii) the need to maximize health by avoiding or delaying preventable disability. Along with the societal and disease transitions, there has been a major demographic shift. Although older people are currently defined as those 60 years of age and above (*112*), this definition of older people has a very different meaning from the middle of the last century, when 60 years of age and above often exceeded the average life expectancy, especially in industrialized countries. Although it is worth remembering that the majority of elderly people will, in fact, be living in the developing world.

Most chronic diseases are present at this period of life—the result of interactions between multiple disease processes as well as more general losses in physiological functions (*113, 114*). Cardiovascular disease peaks at this period, as does type 2 diabetes and some cancers. The main burden of chronic diseases is observed at this stage of life and, therefore, needs to be addressed.

Changing behaviours in the elderly

In the 1970s, it was thought that risks were not significantly increased after certain late ages and, therefore, that there would be no benefit in changing habits, such as dietary habits, after 80 years old (*115*) as there was no epidemiological evidence that changing habits would affect mortality or even health conditions among older people. There was also a feeling that people "earned" some unhealthy behaviours simply because of reaching "old age". Then there was a more active intervention phase, when older people were encouraged to change their diets in ways that were probably overly rigorous for the expected benefit. More recently, older people have been encouraged to eat a healthy diet—as large and as varied as possible while maintaining their weight—and particularly to continue exercise (*113, 116*). Liu et al. (*117*) have reported an observed risk of atherosclerotic disease incidence among older women that was approximately 30% less in women who ate 5–10 servings of fruit and vegetables per day than in those who ate 2–5 servings per day. It seems that, as elderly patients have a higher cardiovascular risk, they are more likely to gain from risk factor modification (*118*).

Although this age group have received relatively little attention as regards primary prevention, the acceleration in decline caused by external factors is generally reversible at any age (*119*). Interventions aimed at supporting the individual and promoting healthier environments will often lead to increased independence in older age.

4.3 Interactions between early and later factors throughout the life course

Low birth weight, followed by subsequent adult obesity, has been shown to impart a particularly high risk of CHD (*120, 121*), as well as diabetes (*18*). Risk of impaired glucose tolerance has been found to be highest in those who had low birth weight, but who subsequently became obese as adults (*18*). A number of recent studies (*12, 13, 25, 59–61, 120*) have demonstrated that there is an increased risk of adult disease when intrauterine growth retardation (IUGR) is followed by rapid catch-up growth in weight and height. Conversely, there is also fairly consistent evidence of higher risk of CHD, stroke, and probably adult onset diabetes with shorter stature (*122, 123*). Further research is needed to define optimal growth in infancy in terms of prevention of chronic disease. A WHO multicentre growth reference study (*124*) currently under way may serve to generate much needed information on this matter.

4.3.1 *Clustering of risk factors*

Impaired glucose tolerance and an adverse lipid profile are seen as early as childhood and adolescence, where they typically appear clustered together with higher blood pressure and relate strongly to obesity, in particular central obesity (*76, 78, 125, 126*). Raised blood pressure, impaired glucose tolerance (IGT) and dyslipidaemia also tend to be clustered in children and adolescents with unhealthy lifestyles and diets, such as those with excessive intakes of saturated fats, cholesterol and salt, and inadequate intake of fibre. Lack of exercise and increased television viewing add to the risk (*10*). In older children and adolescents, habitual alcohol and tobacco use also contribute to raised blood pressure and to the development of other risk factors in early life. Much the same factors continue to act throughout the life course. Such clustering also represents an opportunity to address more than one risk at a time. The clustering of health-related behaviours is also a well-described phenomenon (*127*).

4.3.2 *Intergenerational effects*

Young girls who grow poorly become stunted women and are more likely to give birth to low-birth-weight babies who are then likely to continue the cycle by being stunted in adulthood, and so on (*128*). Maternal birth size is a significant predictor of a child's birth size after controlling for gestational age, sex of the child, socioeconomic status, and maternal age, height, and pre-pregnant weight (*129*). There are clear indications of intergenerational factors in obesity, such as parental obesity, maternal gestational diabetes, and maternal birth weight. Low maternal birth weight is associated with higher blood pressure levels in the offspring, independent of the relation between the offspring's own birth weight and blood pressure (*7*). Unhealthy lifestyles can also have a direct effect on the health of the next generation, for example smoking during pregnancy (*9, 130*).

4.4 Gene–nutrient interactions and genetic susceptibility

There is good evidence that nutrients and physical activity influence gene expression and have shaped the genome over several million years of human evolution. Genes define opportunities for health and susceptibility to disease, while environmental factors determine which susceptible individuals will develop illness. In view of changing socioeconomic conditions in developing countries, such added stress may result in exposure of underlying genetic predisposition to chronic diseases. Gene–nutrient reactions also involve the environment. The dynamics of the relationships are becoming better understood but there is still a long way to go in this, and in other aspects, such as disease prevention and control. Studies continue on the role of nutrients in gene expression; for example, researchers are trying to understand why omega-3 fatty acids suppress or decrease the mRNA of Interleukin, which is elevated in atherosclerosis, arthritis and other autoimmune diseases, whereas the omega-6 fatty acids do not (*131*). Studies on genetic variability to dietary response indicate that specific genotypes raise cholesterol levels more than others. The need for targeted diets for individuals and subgroups to prevent chronic diseases was acknowledged as being part of an overall approach to prevention at the population level. But practical implications of this issue to public health policy have only begun to be addressed. For example, studying the relationship between folate and cardiovascular disease revealed that a common single gene mutation that reduces the activity of an enzyme involved in folate metabolism (MTIIFR) is associated with a moderate (20%) increase in serum homocysteine and higher risk of bothischaemic heart disease and deep vein thrombosis (*132*).

Although humans have evolved being able to feed on a variety of foods and to adapt to them, certain genetic adaptations and limitations have occurred in relation to diet. Understanding the evolutionary aspects of diet and its composition might suggest a diet that would be consistent with the diet to which our genes were programmed to respond. However, the early diet was presumably one which gave evolutionary advantage to reproduction in the early part of life, and so may be less indicative of guidance for healthy eating, in terms of lifelong health and prevention of chronic disease after reproduction has been achieved. Because there are genetic variations among individuals, changes in dietary patterns have a differential impact on a genetically heterogeneous population, although populations with a similar evolutionary background have more similar genotypes. While targeted dietary advice for susceptible populations, subgroups or individuals is desirable, it is not feasible at present for the important chronic diseases considered in this report. Most are polygenic in nature and rapidly escalating rates suggest the importance of environmental change rather than change in genetic susceptibility.

4.5 Intervening throughout life

There is a vast volume of scientific evidence highlighting the importance of applying a life-course approach to the prevention and control of chronic disease. The picture is, however, still not complete, and even sometimes contradictory. From the available evidence, it is possible to state the following:

- Unhealthy diets, physical inactivity and smoking are confirmed risk behaviours.
- The biological risk factors of hypertension, obesity and lipidaemia are firmly established as risk factors for coronary heart disease, stroke and diabetes.
- Nutrients and physical activity influence gene expression and may define susceptibility.
- The major biological and behavioural risk factors emerge and act in early life, and continue to have a negative impact throughout the life course.
- The major biological risk factors can continue to affect the health of the next generation.
- An adequate and appropriate postnatal nutritional environment is important.
- Globally, risk factor trends are rising, especially for obesity, physical inactivity and, in the developing world particularly, smoking.
- Selected interventions are effective but must extend beyond individual risk factors and continue throughout the life course.
- Some preventive interventions early in the life course offer lifelong benefits.
- Improving diets and physical activity in adults and older people will reduce chronic disease risks for death and disability.
- Secondary prevention by diet and physical activity is a complementary strategy in retarding the progression of existing chronic diseases and decreasing mortality and the disease burden from such diseases.

From the above, it is clear that risk factors must be addressed throughout the life course. As well as preventing chronic diseases, there are clearly many other reasons to improve the quality of life of people throughout life. The intention of primary prevention interventions is to move the profile of the whole population in a healthier direction. Small changes in risk factors in the majority who are at moderate risk can have an enormous impact in terms of population-attributable risk of death and disability. By preventing disease in large countries, small reductions in blood pressure, blood cholesterol and so on can reduce health costs by millions of US dollars. It has been demonstrated that improved lifestyles can reduce the risk of progression to diabetes by a striking 58% over 4 years (*133, 134*). Other population studies have shown that up to 80% of cases of coronary heart disease, and up to 90% of cases of type 2 diabetes, could potentially be avoided through changing lifestyle factors, and about one-third of cancers could be avoided by eating healthily, maintaining normal weight, and exercising throughout life (*135–137*).

For interventions to have a lasting effect on the risk factor prevalence and health of societies, it is also essential to change or modify the environment in which these diseases develop. Changes in dietary patterns, the influence of advertising and the globalization of diets, and widespread reduction in physical activity have generally had negative impacts in terms of risk factors, and presumably also in terms of subsequent disease (*138, 139*). Reversing current trends will require a multifaceted public health policy approach.

While it is important to avoid inappropriately applying nutritional guidelines to populations that may differ genetically from those for whom the dietary and risk data were originally determined, to date the information regarding genes or gene combinations is insufficient to define specific dietary recommendations based on a population distribution of specific genetic polymorphisms. Guidelines should try to ensure that the overall benefit of recommendations to the

majority of the population substantially outweighs any potential adverse effects on the rest of the population. For example, population-wide efforts to prevent weight gain may trigger a fear of fatness and, therefore, undernutrition in adolescent girls.

The population nutrient goals recommended by the Joint WHO/FAO Expert Consultation are based on current scientific knowledge and evidence, and are intended to be further adapted and tailored to local or national diets and populations, where presumably the diet has evolved to be appropriate for the culture and local environment.

The goals are intended to reverse or reduce the impact of recent unfavourable changes to the diet which have occurred over the past century and even more recently in many developing countries. Present nutrient intake goals need to reflect the substantial changes in environments from those endured for many ages by humans. For example, the metabolic response to periodic famine and chronic food shortage may no longer represent a selective advantage but rather may increase susceptibility to chronic diseases. An abundant stable food supply is a recent phenomenon; it was not a factor until the advent of the industrial revolution (or the equivalent process in more recently industrialized countries).

A combination of physical activity, food variety and extensive social interaction is the most likely lifestyle profile to optimize health, as reflected in increased longevity and healthy ageing. Some available evidence suggests that, within the time frame of a week, at least 20 and probably as many as 30 biologically distinct types of foods, with the emphasis on plant foods, are required for healthy diets.

The recommendations given in this report consider the wider environment, of which the food supply is a major part (see Chapter 3). The implications of the recommendations would be to increase the consumption of fruits and vegetables, to increase the consumption of fish, and to alter the types of fats and oils, as well as the amount of sugars and starch consumed, especially in developed countries. The current move towards increasing animal protein in diets in countries in economic transition is unlikely to be reversed in countries where there are increased consumer resources, but is unlikely to be conducive to adult health for preventing chronic diseases.

Finally, what success can be expected with developing and updating the scientific basis for national guidelines? The percentage of British adults complying with national dietary guidelines is discouraging, for example, only 2–4% of the population is consuming the recommended level of saturated fat, and 5–25% for fibre. The figures would not be dissimilar for most developed countries, where the majority of people are not aware of what exactly the dietary guidelines suggest. In using the updated and evidence-based recommendations in this report, national governments should aim to produce dietary guidelines that are simple, realistic and food-based. There is an increasing need, recognized at all levels, for the wider implications to be specifically addressed: the implications for agriculture and fisheries, the role of international trade in a globalized world, the impact on countries dependent on primary produce, the effect of macroeconomic policies, and the need for sustainability. The greatest burden of disease will be in the developing world and, in the transitional and industrialized world, amongst the most disadvantaged socioeconomically.

In conclusion, it may be necessary to have three mutually reinforcing strategies that will have different magnitudes of impact over differing time frames. First, with the greatest and most immediate impact, there is the need to address risk factors in adulthood and, increasingly, among older people. Risk-factor behaviours can be modified in these groups and changes will be seen within 3–5 years. With all populations ageing, the sheer numbers and potential cost savings are enormous and realizable. Secondly, societal changes towards health-promoting environments need to be greatly expanded as an integral part of any intervention. Ways to reduce the intake of sugars-sweetened drinks (particularly by children) and high-energy density foods that are micronutrient poor, as well as cigarette smoking and to increase physical activity will have an impact throughout society. Such changes need the active participation of communities, politicians, health systems, town planners and municipalities, as well as the food and leisure industries. Thirdly, the health environment, in which those who are most at risk grow up, needs to change. This is a more targeted and potentially costly approach, but with cost-effective returns even though they are longer term.

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5. Population nutrient intake goals for preventing diet-related chronic diseases

5.1 Overall goals

5.1.1 *Background*

Population nutrient intake goals represent the population average intake that is judged to be consistent with the maintenance of health in a population. Health, in this context, is marked by a low prevalence of diet-related diseases in the population.

Seldom is there a single "best value" for such a goal. Instead, consistent with the concept of a safe range of nutrient intakes for individuals, there is often a range of population averages that would be consistent with the maintenance of health. If existing population averages fall outside this range, or trends in intake suggest that the population average will move outside the range, health concerns are likely to arise. Sometimes there is no lower limit; this implies that there is no evidence that the nutrient is required in the diet and hence low intakes should not give rise to concern. It would be of concern if a large proportion of values were outside the defined goals.

5.1.2 *Strength of evidence*

Ideally the definition of an increased or a decreased risk should be based on a relationship that has been established by multiple randomized controlled trials of interventions on populations that are representative of the target of a recommendation, but this type of evidence is often not available. The recommended dietary/nutrition practice should modify the attributable risk of the undesirable exposure in that population.

The following criteria are used to describe the strength of evidence in this report. They were based on the criteria used by the World Cancer Research Fund (*1*), but were modified by the Expert Consultation to include the results of controlled trials where relevant and available. In addition, consistent evidence on community and environmental factors which lead to behaviour changes and thereby modify risks has been taken into account in categorizing risks. This applies particularly to the complex interaction between environmental factors that affect excess weight gain, a risk factor which the Consultation recognized as contributing to many of the problems being considered.

Convincing evidence: Evidence is based on epidemiological studies showing consistent associations between exposure and disease, with little or no evidence to the contrary. The available evidence is based on a substantial number of studies including prospective observational studies and where relevant, randomized controlled trials of sufficient size, duration and quality showing consistent effects. The association should be biologically plausible.

Probable evidence: Evidence is based on epidemiological studies showing fairly consistent associations between exposure and disease, but where there are perceived shortcomings in the available evidence or some evidence to the contrary, precluding a more definite judgement. Shortcomings in the evidence may be any of the following: insufficient duration of trials (or studies); insufficient trials (or studies) available; inadequate sample sizes; incomplete follow-up. Laboratory evidence is usually supportive. Again, the association should be biologically plausible.

Possible evidence: Evidence is based mainly on the findings from case-control and cross-sectional studies. Insufficient randomized controlled trials, observational studies or non-randomized controlled trials are available. Evidence based on non-epidemiological studies, such as clinical and laboratory investigations, is supportive. More trials are required to support the tentative associations, which should also be biologically plausible.

Insufficient evidence: Evidence is based on the findings of a few studies which are suggestive, but are insufficient to establish an association between exposure and disease. Limited or no

evidence is available from randomized controlled trials. More well-designed research is required to support the tentative associations.

5.1.3 *A summary of population nutrient intake goals*

The population nutrient intake goals for consideration by national and regional bodies establishing dietary recommendations for the prevention of diet-related chronic diseases are presented in Table 5. These recommendations are expressed in numerical terms, rather than as increases or decreases in intakes of specific nutrients, because the desirable change will depend upon existing intakes in the particular population, and could be in either direction.

In Table 5, attention is directed towards the energy-supplying macronutrients. This must not be taken to imply a lack of concern for the other nutrients. Rather, it is a recognition of the fact that previous reports issued by FAO and WHO have provided limited guidance on the meaning of a "balanced diet" described in terms of the proportions of the various energy sources, and that there is an apparent consensus on this aspect of diet in relation to effects on the chronic non-deficiency diseases. This report therefore complements these existing reports on energy and nutrient requirements issued by FAO and WHO (*2*–*4*). In translating these goals into dietary guidelines, due consideration should be given to the process for setting up national dietary guidelines (*5*).

Table 5
Ranges of population dietary intake goals

Dietary factor	Goals
Total fat	15-30% energy
Saturated fatty acids	< 10% energy
Polyunsaturated fatty acids (PUFAs)	6-10% energy
n-6 Polyunsaturated fatty acids (PUFAs)	5-8% energy
n-3 Polyunsaturated fatty acids (PUFAs)	1-2% energy
Trans fatty acids	< 1% energy
Monounsaturated fatty acids (MUFAs)	By difference[a]
Total carbohydrate[b]	55-75% energy
Free sugars[c]	< 10 % energy
Protein	10-15% energy[d]
Cholesterol	< 300 mg/day
Sodium chloride (sodium)[e]	<5 g/day (< 2 g/day)
Fruits and vegetables	≥ 400 g/day
Total dietary fibre Non-starch polysaccharides (NSP)	From foods (see the text under NSP)

[a] This means "total fat – (sats + polyunsaturated fatty acids + trans fatty acids)"

[b] The percentage of total energy available after taking into account that consumed as protein and fat, hence the wide range

[c] The term "free sugars" refers to all monosaccharides and disaccharides added to foods by the manufacturer, cook or consumer, plus sugars naturally present in honey, syrups and fruit juices.

[d] The suggested range should be seen in the light of the Joint WHO/FAO/UNU Expert Consultation on Protein and Amino Acid Requirements in Human Nutrition, held in Geneva from 9 to 16 April 2002 (2).

[e] Salt should be iodized appropriately (6).

Total fat

Highly active groups with diets rich in vegetables, legumes, fruits and whole grain cereals may sustain a total fat intake of up to 35% without the risk of unhealthy weight gain. The recommendations for total fat are formulated to include countries where the usual fat intake is typically above 30% as well as those where the usual intake may be very low, for example less than 15%. Total fat energy of at least 20% is consistent with good health. However, for countries where the usual fat intake is between 15% and 20% of energy, there is no direct evidence for men that raising fat intake to 20% will be beneficial (*7*, *8*). For women of reproductive age at least 20% has been recommended by the Joint FAO/WHO Expert Consultation on Fats and Oils in Human Nutrition that met in 1993 (*3*).

Free sugars

It is recognized that higher intakes of free sugars threaten the nutrient quality of diets by providing significant energy without specific nutrients. The Consultation considered that restriction of free sugars was also likely to contribute to reducing the risk of unhealthy weight gain, noting that:

- Free sugars contribute to the overall energy density of diets.

- Free sugars promote a positive energy balance. Research in human volunteers demonstrates increased total energy intake in acute and short-term studies when the energy density of the diet is increased, whether by free sugars or fat (*9–11*). Diets that are limited in free sugars have been shown to reduce total energy intake and induce weight loss (*12, 13*).

- Drinks that are rich in free sugars increase overall energy intake by reducing appetite control. There is thus less of a compensatory reduction of food intake after the consumption of high sugar drinks than when additional foods of equivalent energy content are provided (*11, 14–16*). A recent randomized trial showed that when soft drinks that are rich in free sugars are consumed there is a higher energy intake and a progressive increase in body weight when compared with energy free drinks that are artificially sweetened (*17*). Children with a high consumption of soft drinks rich in free sugars are more likely to be overweight and to gain excess weight (*16*).

The Consultation recognized that a population goal for free sugars of less than 10% of total energy is controversial. However, the Consultation considered that the studies showing no effect of free sugars on excess weight have limitations. The CARMEN study (Carbohydrate Ratio Management in European National diets) was a multi-centre, randomized trial that tested the effects on body weight and blood lipids in overweight individuals of altering the ratio of fat to carbohydrate, as well as the ratio of simple to complex carbohydrate per se. A greater weight reduction was observed on the high complex carbohydrate diet relative to simple carbohydrates, but did not achieve statistical significance (*18*). Nevertheless, an analysis of weight change and metabolic indices for those with metabolic syndrome revealed a clear benefit of replacing simple by complex carbohydrates (*19*). The Consultation also examined the results of studies that found an inverse relationship between free sugar intakes and total fat intake. Many of these studies are methodologically inappropriate for determining the causes of excess weight gain, since the percentage of calories from fat will decrease as the percentage of calories from carbohydrates increases and vice versa. Furthermore, these analyses do not usually distinguish between free sugars in foods and free sugars in drinks. Thus, these analyses are not good predictors of the responses in energy intake to a selective reduction in free sugar intake.

Non-starch polysaccharides (NSP)

Whole grain cereals, fruits and vegetables are the preferred source of NSP. The best definition of dietary fibre remains to be established, given the potential health benefits of resistant starch. The recommended intake of fruits and vegetables (see below) and consumption of wholegrain foods is likely to provide >20 g/day of NSP (>25 g/day of total dietary fibre).

Fruits and vegetables

The benefit of fruits and vegetables cannot be ascribed to a single or mix of nutrients and bioactive substances. Therefore, this food category was included rather than nutrients. The category of tubers (i.e. potatoes, cassava) should not be included in fruits and vegetables.

Body mass index (BMI)

The goal for body mass index (BMI) included in this report follows the recommendations made by the WHO Expert Consultation on Obesity that met in 1997 (*20*). At the population level, the goal is for an adult median BMI of 21–23 kg/m^2. For individuals, the recommendation is to maintain a BMI in the range 18.5–24.9 kg/m^2 and to avoid a weight gain greater than 5 kg during adult life.

Physical activity

The goal for physical activity focuses on maintaining healthy body weight. The recommendation is for a total of one hour per day on most days of the week of moderate-intensity activity, such as walking. This level of physical activity is needed to maintain a healthy body weight, particularly for people with sedentary occupations. The recommendation is based on calculations of energy balance and on an analysis of the extensive literature on the relationships between body weight and physical activity. This recommendation is also presented elsewhere (*21*). Obviously, this quantitative goal cannot be considered as a single "best value" by analogy with the nutrient intake goals. Furthermore, it differs from the following widely accepted public health recommendation (*22*):

For better health, people of all ages should include a minimum of 30 minutes of physical activity of moderate intensity (such as brisk walking) on most, if not all, days of the week. For most people greater health benefits can be obtained by engaging in physical activity of more vigorous intensity or of longer duration. This cardio respiratory endurance activity should be supplemented with strength-developing exercises at least twice a week for adults in order to improve musculo skeletal health, maintain independence in performing the activities of daily life and reduce the risk of falling.

The difference between the two recommendations results from the difference in their focus. A recent symposium on the dose–response relationships between physical activity and health outcomes found evidence that 30 minutes of moderate activity is sufficient for cardiovascular/metabolic health, but not for all health benefits. Because prevention of obesity is a central health goal, the recommendation of 60 minutes a day of moderate-intensity activity is considered appropriate. Activity of moderate intensity is found to be sufficient to have a preventive effect on most, if not all, cardiovascular and metabolic diseases considered in this report. Higher intensity activity has a greater effect on some, although not all, health outcomes, but is beyond the capacity and motivation of a large majority of the population.

Both recommendations include the idea that the daily activity can be accomplished in several short bouts. It is important to point out that both recommendations apply to people who are otherwise sedentary. Some occupational activities and household chores constitute sufficient daily physical exercise.

In recommending physical activity, potential individual risks as well as benefits need to be assessed. In many regions of the world, especially but not exclusively in rural areas of developing countries, an appreciable proportion of the population is still engaged in physically demanding activities relating to agricultural practices and domestic tasks performed without mechanization or with rudimentary tools. Even children may be required to undertake physically demanding task at very young ages, such as collecting water and firewood and caring for livestock. Similarly, the inhabitants of poor urban sectors may still be required to walk long distances to their jobs, which are usually of a manual nature and often require a high expenditure of energy. Obviously, the recommendation for extra physical activity is not relevant for these sectors of the population.

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5.2 Recommendations for preventing excess weight gain and obesity

5.2.1 *Background*

Almost all countries (high-income and low-income alike) are experiencing an obesity epidemic, although with great variation between and within countries. In low-income countries, obesity is more common in middle-aged women, people of higher socioeconomic status, and those living in urban communities. In more affluent countries, obesity is now common in younger adults and children. It is associated with lower socioeconomic status, especially in women, and the urban-rural differences are diminished or even reversed.

It has been estimated that the direct health care costs of obesity accounted for 6.8% (or US$ 70 billion) and physical inactivity for a further US$ 24 billion in the United States in 1995 and somewhat less, but still a staggering amount, in other industrialized countries (*1*). Indirect costs, far greater than direct costs, include workdays lost, physician visits, disability pensions and premature mortality. Intangible costs such as impaired quality of life are enormous. Because the risk of diabetes, cardiovascular disease and hypertension rise continuously with increasing weight, there is much overlap between the prevention of obesity and the prevention of a variety of chronic diseases, especially type II diabetes. Population education strategies will need a solid base of policy and environment-based changes to be effective in eventually reversing these trends.

5.2.2 *Trends*

The increasing westernization, urbanization and mechanization occurring in most countries around the world is associated with changes in diet towards one of high-fat, high-energy foods and a sedentary lifestyle. In many developing countries undergoing economic transition, as obesity rapidly increases, it often coexists in the same population (or even the same household) with chronic undernutrition. Along with the increase in obesity over the past 30 years, the prevalence of diabetes has increased dramatically (2).

5.2.3 *Diet, physical activity and disease*

Mortality rates increase with increasing degrees of overweight, as measured by BMI. As BMI increases, so too does the proportion of people with one or more comorbid conditions. In one study in the USA (3), over half (53 %) of all deaths in women with a BMI>29 kg/m^2 could be directly attributed to their obesity. Eating behaviours that have been linked to overweight and obesity include snacking/eating frequency, binge-eating patterns, eating out, and (protectively) exclusive breastfeeding. Nutrient factors under investigation include fat, carbohydrate type (including refined carbohydrates such as sugar), the glycaemic index of foods, and fibre. Environmental issues are clearly important, especially as many environments become increasingly “obesogenic” (obesity-promoting).

Physical activity is an important determinant of body weight. In addition, physical activity and physical fitness (which relates to the ability to perform physical activity) are important modifiers of mortality and morbidity related to overweight and obesity. There is firm evidence that moderate to high fitness levels provide substantially reduced risk of cardiovascular disease and all cause mortality and these benefits apply to all BMI levels. Furthermore, high fitness protects against mortality at all BMI levels in men with diabetes. Low cardiovascular fitness is a serious and common comorbidity of obesity, and a sizeable proportion of deaths in overweight and obese populations are probably a result of low levels of cardio-respiratory fitness rather than obesity per se. Fitness is, in turn, influenced strongly by physical activity in addition to genetic factors. These relationships emphasize the role of physical activity in the prevention of overweight and obesity, independently of the effects of physical activity on body weight.

The potential etiological factors related to unhealthy weight gain are shown in Table 6 summarized below.

Table 6

Summary of strength of evidence on factors that might promote or protect against weight gain and obesity[a]

Evidence	Decreases risk	No relationship	Increases risk
Convincing	Regular physical activity High dietary intake of NSP (dietary fibre)[b]		Sedentary lifestyles High intake of energy-dense micronutrient-poor foods[c]
Probable	Home and school environments that support healthy food choices for children[d] Breastfeeding		Heavy marketing of energy-dense foods[d] and fast-food outlets[d] Sugar-sweetened soft drinks and fruit juices Adverse social and economic conditions[d] (in developed countries, especially for women)
Possible	Low glyacemic index foods	Protein content of the diet	Large portion sizes High proportion of food prepared outside the home (developed countries) "Rigid restraint/periodic disinhibition" eating patterns
Insufficient	Increased eating frequency		Alcohol

[a] Strength of evidence: the totality of the evidence was taken into account. The World Cancer Research Fund schema was taken as the starting point but was modified in the following manner: randomized controlled trials were given prominence as the highest ranking study design (randomized controlled trials were not a major source of cancer evidence); associated evidence and expert opinion was also taken into account in relation to environmental determinants (direct trials were usually not available).

[b] Specific amounts will depend on the analytical methodologies used to measure fibre.

[c] Energy-dense and micronutrient-poor foods tend to be processed foods that are high in fat and/or sugar. Low energy-dense (or energy-dilute) foods, such as fruit, legumes, vegetables and whole grain cereals, are high in dietary fibre and water.

[d] Associated evidence and expert opinion included.

5.2.4 **Strength of evidence**

Convincing etiological factors

Regular physical activity (protective) and sedentary lifestyles (causative). There is convincing evidence that regular physical activity is protective against unhealthy weight gain whereas sedentary lifestyles, particularly sedentary occupations and inactive recreation such as watching television, promote it. Most epidemiological studies show smaller risk of weight gain, overweight and obesity among persons who *currently* engage regularly in moderate to large amounts of physical activity (*4*). Studies measuring physical activity at baseline and randomized trials of exercise programmes show more mixed results, probably because of the low adherence to long-term changes. Therefore, it is ongoing physical activity itself rather than previous physical activity or enrolment in an exercise programme that is protective against unhealthy weight gain. The recommendation for individuals to accumulate at least 30 minutes of moderate-intensity physical activity on most days is largely aimed at reducing cardiovascular diseases and overall mortality. The amount needed to prevent unhealthy weight gain is uncertain but is probably significantly greater than this. Preventing weight gain *after* substantial weight loss probably requires about 60–90 minutes per day. Two meetings recommended by consensus that about 45–60 minutes of moderate-intensity physical activity is needed on most days or every day to prevent unhealthy weight gain (*5, 6*). Studies aimed at reducing sedentary behaviours have focused primarily on reducing television viewing in children. Reducing viewing times by about 30 minutes a day in children in the United States appears feasible and is associated with reductions in BMI.

A high intake of dietary non-starch polysaccharides (NSP)/dietary fibre (protective). The nomenclature and definitions for NSP (dietary fibre) have changed with time, and many of the studies available used previous definitions, such as soluble and insoluble fibre. Nevertheless, two recent reviews of randomized trials concluded that the majority of studies showed that a high intake of NSP (dietary fibre) promoted weight loss. Pereira & Ludwig (*7*) found that 12 out of 19 trials showed beneficial objective effects (including weight loss). Howarth Saltzman & Roberts (*8*) found from 11 studies of more than 4 weeks duration and ad libitum eating that the mean weight loss was 1.9 kg over 3.8 months. There were no differences between fibre type or between fibre in food or supplements.

There is convincing evidence that a high intake of energy-dense foods promotes weight gain. In high-income countries (and increasingly in low-income countries) these energy-dense foods are highly processed (low NSP) and micronutrient-poor as well, further diminishing their nutritional value. Energy-dense foods tend to be high in fat (such as butter, oils, fried foods), sugar or starch; while energy-dilute foods have a high water content (such as fruits and vegetables). Several trials have covertly manipulated the fat content and the energy density of diets, and these support the view that so-called 'passive over consumption' of total energy occurs when the energy density of the diet is high and that this is almost always the case in high-fat diets. A meta-analysis of 16 trials of ad libitum high-fat versus low-fat diets of at least 2 months duration suggested that a reduction in fat content by ten percentage points corresponds to about a 1MJ reduction in energy intake and about 3kg in body weight (*9*). At a population level, 3kg equates to about one BMI unit or about 5% difference in obesity prevalence. However, it is difficult to blind such studies and other non-physiological effects may influence these findings (*10*). While energy from fat is no more fattening than the same amount of energy from carbohydrate or protein, diets that are high in fat tend to be energy-dense. An important exception to this is diets based predominantly on energy-dilute foods (eg vegetables, legumes, fruits) but have a reasonably high percent of energy as fat from added oils. The effectiveness over the long term of most dietary strategies for weight loss, including low fat diets, remains uncertain unless accompanied by changes in behaviour affecting physical activity and food habits. These latter changes at a public health level require an environment supportive of healthy food choices and an active life. High quality trials to address these issues are urgently needed. A variety of popular weight-loss diets that restrict food choices may result in reduced energy intake and short term weight loss in individuals but most do not have trial evidence of long-term effectiveness and nutritional adequacy and therefore cannot be recommended for populations.

Probable etiological factors

Home and school environments that promote healthy food and activity choices for children (protective). Despite the obvious importance of the roles that parents and home environments play on children's eating and physical activity behaviours, there is very little hard evidence available in the area. It appears that access and exposure to a range of fruits and vegetables in the home is important for the development of preferences for those foods and that parental knowledge, attitudes and behaviours related to healthy diet and physical activity are important in creating role models (*11*). More data were available for the impact of the school environment on nutrition knowledge, eating patterns and physical activity at school, and sedentary behaviours at home. Some (*12*), but not all, have shown an effect of school-based interventions on obesity prevention. While more research is clearly needed to increase the evidence base in both these areas, supportive home and school environments were rated as a probable etiological influence on obesity.

Heavy marketing of fast food outlets and energy-dense, micronutrient-poor foods and beverages (causative). Part of the consistent, strong relationships between television viewing and obesity in children may relate to the food advertising to which they are exposed (13–15). Fast food restaurants, and foods and beverages that are usually classified under the "eat least" category in dietary guidelines are among the most heavily marketed products, especially on television. Young children are often the target group for the advertising of these products because they have a significant influence on the foods bought by parents (16). The huge investment in marketing fast foods and other "eat least" choices (US$ 11 billion in the United States alone in 1997) was considered a key factor in the increased consumption of food prepared outside the home in general and those energy-dense, micronutrient-poor foods in particular. Young children are unable to distinguish programme content from the persuasive intent of advertisements. The evidence that the heavy marketing of these foods and beverages to young children causes obesity is not unequivocal. Nevertheless, the Consultation considered that there is sufficient indirect evidence to warrant this practice being placed in the "probable" category and thus becoming a potential target for interventions (15–18).

A high intake of sugar-sweetened beverages (causative). Diets that are proportionally low in fat will be proportionally higher in carbohydrate (including a variable amount of sugars) and are associated with protection against unhealthy weight gain, although a high intake of free sugars in beverages probably promotes weight gain. The physiological effects of energy intake on satiation and satiety appear to be quite different for energy in solid foods as opposed to energy in fluids. Possibly because of reduced gastric distension and faster transit times, the energy contained in fluids is not well "detected" by the body and subsequent food intake is poorly adjusted to account for the energy taken in through beverages (*19*). This is supported by data from cross-sectional, longitudinal, and cross-over studies (20–*22*). The high and increasing consumption of sugar-sweetened drinks by children in many countries is of serious concern. It has been estimated that each additional can or glass of sugar-sweetened drink that they consume every day increases the risk of becoming obese by 60 % (*19*). Most of the evidence relates to soda drinks but many fruit drinks and cordials are equally energy-dense and may promote weight gain if drunk in large quantities. Overall, the evidence implicating a high intake of sugar-sweetened drinks in promoting weight gain was considered moderately strong.

Adverse socioeconomic conditions, especially for women in high-income countries (causative). The pattern of the progression of obesity through a population classically starts with middle-aged women in high-income groups but as the epidemic progresses, obesity becomes more common in people (especially women) in lower socioeconomic status groups. The relationship may even be bi-directional, setting up a vicious cycle (i.e. lower socioeconomic status promotes obesity, and obese people are more likely to end up in groups with low socioeconomic status). The mechanisms by which socioeconomic status influences food and activity patterns are probably multiple and need elucidation. However, people living in circumstances of low socioeconomic status may be more at the mercy of the obesogenic environment because their eating and activity behaviours are more likely to be the "default choices" on offer. The evidence for an effect of low socioeconomic status on predisposing people to obesity is consistent (in higher income countries) from cross-sectional and longitudinal studies (23), and was rated as a "probable" cause of increased risk of obesity.

Breastfeeding (protective). Breastfeeding as a protective factor has been examined in at least 20 studies with nearly 40 000 subjects. Five studies (including the two largest) found a protective effect, two found that breastfeeding predicted obesity, and the remainder found no relationships. There are probably multiple effects of confounding in these studies; however, the reduction in the risk of developing obesity observed by the two largest

studies was substantial (20–37%). Promoting breastfeeding has many benefits, and prevention of childhood obesity is probably one of them.

Possible etiological factors

Several other factors were defined as "possible" protective or causative in the etiology of unhealthy weight gain.

Low-glycaemic foods have been proposed as a potential protective factor against weight gain and there are some early studies that support this hypothesis. More clinical trials are, however, needed to establish this with greater certainty.

Large portion sizes are a possible causative factor for unhealthy weight gain (*24)*. The marketing of "supersize" portions, particularly in fast food outlets, is now common practice in many countries. There is some evidence that people poorly estimate portion sizes and that subsequent energy compensation for a large meal is incomplete and therefore is likely to lead to overconsumption.

In many countries, there has been a steady increase in the proportion of food eaten that is prepared outside the home. In the United States, the energy, total fat, saturated fat, cholesterol and sodium content of foods prepared outside the home is significantly higher than that of home-prepared food. People in the United States who tend to eat out have a higher BMI than those who tend to eat at home (*25)*.

Certain psychological parameters of eating patterns may influence the risk of obesity. The "flexible restraint" pattern is associated with lower risk of weight gain, whereas the "rigid restraint / periodic disinhibition" pattern is associated with a higher risk.

Several other factors were also considered but the evidence was not thought to be strong enough to warrant defining them as protective or causative. Studies have not shown consistent associations between alcohol intake and obesity despite the high energy density of the nutrient (7 kcal/g). There are probably many confounding factors that influence the association. While a high eating frequency has been shown in some studies to have a negative relationship with energy intake and weight gain, the types of foods readily available as snack foods are often high in fat and would predispose people to weight gain. The evidence regarding the impact of early nutrition on subsequent obesity is also mixed, with some studies showing relationships for high and low birth weights.

5.2.5 ***General strategies for obesity prevention***

The prevention of obesity in infants and young children should be considered of high priority. For infants and young children, the preventive strategies are: the promotion of exclusive breastfeeding; avoiding the use of added sugars and starches when feeding formula; instructing the mother to accept the child's ability to regulate energy intake rather than feeding until the plate is empty; and assuring the appropriate micronutrient intake needed to promote optimal linear growth. For children and adolescents, prevention of obesity implies the need to: promote an active lifestyle; limit television viewing; promote the intake of fruits and vegetables; restrict the intake of energy-dense, micronutrient-poor foods (e.g. packaged snacks); and restrict the intake of sugar-sweetened soft drinks. Additional measures include modifying the environment to: enhance physical activity in schools and communities, and family interactions (eating family meals); limit the exposure of young children to heavy marketing practices of energy-dense, micronutrient-poor foods; and provide the necessary information and skills to make healthy food choices.

In developing countries, special attention should be given to avoidance of overfeeding stunted population groups. Nutrition programmes designed to control or prevent undernutrition need to assess stature in combination with weight to prevent providing excess energy to children of low weight-for-age but normal weight-for-height. In countries in economic transition, as populations become more sedentary and able to access energy-dense foods, there is a need to maintain the healthy components of traditional diets (high intake of vegetables, fruits, NSP). Education provided to mothers and low socioeconomic status communities that are food insecure should stress that overweight and obesity do not represent good health.

Low-income groups globally and populations in countries in economic transition often replace traditional

micronutrient-rich foods by heavily marketed, sugar-sweetened beverages (i.e. soft drinks) and energy-dense fatty, salty and sugary foods. These trends, coupled with reduced physical activity, are associated with the rising prevalence of obesity. Strategies are needed to improve the quality of diets by increasing consumption of fruits and vegetables, in addition to increasing physical activity, in order to stem the epidemic of obesity and associated diseases.

5.2.6 *Disease-specific recommendations*

Body mass index (BMI)

BMI can be used to estimate, albeit crudely, a population-level measure of overweight and obesity and the risks associated with it. It does not, however, account for the wide variations in obesity between different individuals and populations. The classification of overweight and obesity, according to BMI, is shown in Table 7.

Table 7

Classification of overweight in adults according to BMI (*26*)

Classification	BMI (kg/m^2)	Risk of comorbidities
Underweight	< 18.5	Low (but risk of other clinical problems increased)
Normal range	18.5 - 24.9	Average
Overweight	≥ 25	
Pre-obese	25 - 29.9	Increased
Obese class I	30.0 - 34.9	Moderate
Obese class II	35.0 - 39.9	Severe
Obese class III	≥ 40.0	Very severe

In recent years, different ranges of BMI cut-off points for overweight and obesity have been proposed, in particular for the Asia-Pacific region (*27)*. The data at present available on which to base definitive recommendations are sparse.1 Nevertheless, the consultation considered that, to achieve optimum health, the median BMI for the adult population should be in the range 21–23 kg/m^2, while the goal for individuals should be to maintain BMI in the range 18.5–24.9 kg/m^2.

Waist circumference

Waist circumference is a convenient and simple measurement which is unrelated to height, correlates closely with BMI and the ratio of waist-to-hip circumference and is an approximate index of intra-abdominal fat mass and total body fat. Furthermore, changes in waist circumference reflect changes in risk factors for cardiovascular disease and other forms of chronic diseases, even though the risks seem to vary in different populations. There is an increased risk of metabolic complications for
men with a waist circumference ≥ 102 cm, and women with a waist circumference ≥ 88 cm.

1 At present WHO is undertaking a systematic review of various datasets from the cuntries in the Asia and Pacific region to evaluate and address these issues.

Physical activity

A total of one hour per day of moderate-intensity activity, such as walking on most days of the week, is probably needed to maintain a healthy body weight, particularly for people with sedentary occupations.2

Total energy intake

The fat and water content of foods are the main determinants of the energy density of the diet. A lower consumption of energy-dense (high-fat, high-sugar and high-starch) foods and energy-dense (high free sugar) drinks contributes to a reduction in total energy intake. Conversely, a higher intake of energy-dilute foods (i.e. vegetables and fruits) and foods high in NSP (i.e. whole grain cereals) contributes to a reduction in total energy intake and an improvement in micronutrient intake. It should be noted, however, that very active groups who have diets high in vegetables, legumes, fruits and whole grain cereals, may sustain a total fat intake of up to 35 % without the risk of unhealthy weight gain.

2 See also reference 5.

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5.3 Recommendations for preventing diabetes

5.3.1 *Background*

Type 2 diabetes, formerly known as non-insulin-dependent diabetes (NIDDM), accounts for most cases of diabetes worldwide. Type 2 diabetes develops when the production of insulin is insufficient to overcome the underlying abnormality of increased resistance to its action. The early stages of type 2 diabetes are characterised by overproduction of insulin. Later in the disease process insulin levels may fall as a result of partial failure of the insulin producing β cells of the pancreas. Complications of type 2 diabetes include blindness, kidney failure, foot ulceration which may lead to gangrene and subsequent amputation, and appreciably increased risk of infections, coronary heart disease and stroke. The enormous and escalating economic and social costs of type 2 diabetes make a compelling case for attempts to reduce the risk of developing the condition as well as for energetic management of established disease (*1, 2*).

Lifestyle modification is the cornerstone of treatment as well as of attempts to prevent type 2 diabetes (*3*). The changes required to reduce the risk of developing type 2 diabetes at the population level are, however, unlikely to be achieved without major environmental changes to facilitate appropriate choices by individuals. Diagnostic criteria for the diagnosis of type 2 diabetes and for the earlier stages in the disease process—impaired glucose tolerance and impaired fasting glucose—have recently been revised (*4, 5*). Type 1 diabetes, previously known as insulin-dependent diabetes, occurs much less frequently and is associated with an absolute deficiency of insulin, usually resulting from autoimmune destruction of the β cells of the pancreas. Environmental as well as genetic factors appear to be involved but there is no convincing evidence of lifestyle factors which can be modified to reduce the risk.

5.3.2 *Trends*

Dramatic increases have occurred in both prevalence and incidence of type 2 diabetes globally, but especially in societies in economic transition in much of the newly industrialized world and in developing countries (*1, 6–9*). The current estimated number of approximately 150 million cases worldwide is predicted to double by 2025, the greatest number of cases being expected in China and India. These numbers may represent an underestimate and there are likely to be many undiagnosed cases. Previously a disease of the middle-aged and elderly, type 2 diabetes has recently escalated in all age groups and is now being identified at younger and younger ages, including in adolescence and childhood, especially in high-risk populations. Age-adjusted mortality rates among people with diabetes are 1.5–2.5 times higher than in the general population (*10*). In Caucasian populations, much of the excess is attributable to cardiovascular disease, especially coronary heart disease (*11, 12*); amongst Asian and American Indian populations, renal disease contributes to a considerable extent (*13, 14*); and in some developing nations, infections (*15*) are an important cause of death. It is conceivable that the decline in mortality due to coronary heart disease which has occurred in many affluent societies may be stopped or reversed if rates of type 2 diabetes continue to increase. This may occur if the coronary risk factors associated with diabetes increase to the extent that the risk they mediate outweighs the benefit accrued from improvements in conventional cardiovascular risk factors and the improved care of patients with established cardiovascular disease (*3*).

5.3.3 *Environmental factors and diabetes*

Type 2 diabetes results from an interaction between genetic and environmental factors. The rapidly changing rates, however, suggest a particularly important role for the latter as well as the potential for stemming the tide of the global epidemic of the disease. The most dramatic increases in type 2 diabetes are occurring in societies in which there have been major changes in type of diet consumed, reductions in physical activity, and increases in overweight and obesity. The diets concerned are typically energy-dense, high in saturated fatty acids and depleted in non-starch polysaccharides. In all societies, overweight and obesity are associated with an increased risk of type 2 diabetes, especially when the excess adiposity is centrally distributed. Conventional (BMI) categories may not be an appropriate means of determining the risk of developing type 2 diabetes in individuals of all population groups because of ethnic differences in body composition and because of the importance of the distribution of excess

adiposity. While all lifestyle-related and environmental factors which contribute to excess weight gain may be regarded as contributing to type 2 diabetes, the evidence that individual dietary factors have an effect which is independent of their obesity promoting effect, is inconclusive. Evidence that saturated fatty acids increase risk of type 2 diabetes and that non-starch polysaccharides are protective is more convincing than the evidence for several other nutrients which have been implicated. The presence of maternal diabetes, including gestational diabetes and intrauterine growth retardation, especially when associated with later rapid catch-up growth, appears to increase the risk of subsequently developing diabetes.

5.3.4 *Strength of Evidence*

The association between excessive weight gain, central adiposity and the development of type 2 diabetes is convincing. The association has been repeatedly demonstrated in longitudinal studies in different populations, with a striking gradient of risk apparent with increasing levels of body mass index, adult weight gain, waist circumference or waist-to-hip ratio. Indeed waist circumference or waist-to-hip ratio (reflecting abdominal or visceral adiposity) are more powerful determinants of subsequent risk of type 2 diabetes than BMI (*16–20*). Central adiposity is also an important determinant of insulin resistance, the underlying abnormality in most cases of type 2 diabetes (*20*). Voluntary weight loss improves insulin sensitivity (*21*) and in several randomized controlled trials has been shown to reduce the risk of progression from impaired glucose tolerance to type 2 diabetes (*22, 23*). Longitudinal studies have also clearly indicated that increased physical activity reduces the risk of developing type 2 diabetes regardless of the degree of adiposity (*24–26*). Vigorous exercise (i.e. training to an intensity of 80–90% of age-predicted maximum heart rate for at least 20 minutes, at least five times per week) has the potential to substantially enhance insulin sensitivity (*21*). The minimum intensity and duration of physical activity required to improve insulin sensitivity has not been established.

Offspring of diabetic pregnancies (including gestational diabetes) are often large and heavy at birth, tend to develop obesity in childhood and are at high risk of developing type 2 diabetes at an early age (*27*). Those born to mothers after they have developed diabetes have a threefold higher risk of developing diabetes than those born before (*28*).

In observational epidemiological studies, a high saturated fat intake has been associated with a higher risk of impaired glucose tolerance and higher fasting glucose and insulin levels (*29–32*). Higher proportions of saturated fatty acids in serum lipid or muscle phospholipid have been associated with higher fasting insulin, lower insulin sensitivity and a higher risk of type 2 diabetes (*33–35*). Higher unsaturated fatty acids from vegetable sources and polyunsaturated fatty acids have been associated with a reduced risk of type 2 diabetes (*36, 37*) and lower fasting and 2-hour glucose concentrations (*32, 38*). Furthermore, higher proportions of long chain polyunsaturated fatty acids in skeletal muscle phospholipids have been associated with increased insulin sensitivity (*39*). In human intervention studies, replacement of saturated by unsaturated fatty acids leads to improved glucose tolerance (*40, 41*) and enhanced insulin sensitivity (*42*). Long chain polyunsaturated fatty acids do not, however, appear to confer additional benefit over monounsaturated fatty acids in intervention studies (*42*). Furthermore, when total fat intake is high (greater than 37% total energy), altering the quality of dietary fat appears to have little effect (*42*), a finding which is not surprising given that in observational studies a high intake of total fat has been shown to predict development of impaired glucose tolerance and the progression of impaired glucose tolerance to type 2 diabetes (*29, 43*). A high total fat intake has also been associated with higher fasting insulin concentrations and a lower insulin sensitivity index (*44, 45*). Considered in aggregate these findings are deemed to indicate a probably causal link between saturated fatty acids and type 2 diabetes, and a possible causal association between total fat intake and type 2 diabetes. The two major randomized controlled trials which showed the potential for lifestyle modification to reduce the risk of progression from impaired glucose tolerance to type 2 diabetes included advice to reduce total and saturated fat (*22, 23*), but in both trials it is impossible to disentangle the effects of individual dietary manipulation.

Research relating to the association between NSP and type 2 diabetes is complicated by ambiguity with regard to definitions used (the term dietary fibre and NSP are often incorrectly used interchangeably), different methods of analysis and, consequently, inconsistencies in food composition tables. Observations by Trowell in Uganda more than 30 years ago suggested that the infrequency of diabetes in rural Africa may be the result of a protective effect of substantial amounts of NSP (referred to as dietary fibre) associated with a high consumption of minimally processed or unprocessed carbohydrate and that throughout the world, increasing intakes of highly processed carbohydrate, depleted in NSP, promoted the development of diabetes (*46*). Three cohort studies (the Health Professionals Follow-up Study of men aged 40–75 years, the Nurses Study of women aged 40–65 years, and the

Iowa Women's Health Study in women aged 55–69 years) have shown a protective effect of NSP (dietary fibre) (*47–49*) which was independent of age, BMI, smoking and physical activity (*47–49*). In many controlled experimental studies, high intakes of NSP (dietary fibre) have repeatedly been shown to result in reduced blood glucose and insulin levels in people with type 2 diabetes and impaired glucose tolerance (*50*) and an increased intake of wholegrain cereals, vegetables and fruits (all rich in NSP) was a feature of the diets associated with a reduced risk of progression of impaired glucose tolerance to type 2 diabetes in the two randomized controlled trials previously described (*22, 23*). Thus the evidence for a potential protective effect of NSP (dietary fibre) appears strong. However, the facts that the experimental studies suggest that soluble forms of NSP exert benefit (*50–53*) whereas the prospective cohort studies suggest that cereal-derived insoluble forms are protective (*47, 48*) explain the "probable" rather than "convincing" grading of the level of evidence.

Many foods which are rich in NSP (especially soluble forms), such as pulses, have a low glycaemic index (glycaemic index = glycaemic response to a quantity of food containing a set amount, usually 50 g, of carbohydrate, expressed as a percentage of the glycaemic response following ingestion of a similar quantity of glucose or of carbohydrate in white bread). Other carbohydrate-containing foods (e.g. certain types of pasta), not especially high in NSP, also have a low glycaemic index. Low glycaemic index foods, regardless of their NSP content, are not only associated with a reduced glycaemic response after ingestion when compared with foods of higher glycaemic index, but are also associated with an overall improvement in glycaemic control (as measured by haemoglobin A_1c) in people with diabetes (*54–57*). A low glycaemic index does not, however, per se, confer overall health benefits, since a high fat or fructose content of a food may also result in a reduced glycaemic index and such foods may also be energy-dense. Thus while this property of carbohydrate-containing foods may well influence the risk of developing type 2 diabetes, the evidence is accorded a lower level of strength than the evidence relating to the NSP content. Similarly, the level of evidence for the protective effect of n-3 fatty acids is regarded as "possible" because the epidemiological studies are inconsistent and the experimental data inconclusive. There is insufficient evidence to confirm or refute the suggestions that chromium, magnesium, vitamin E and moderate intakes of alcohol might protect against the development of type 2 diabetes.

A range of studies, mostly in developing countries, suggests that intrauterine growth retardation and low birth weight are associated with subsequent development of insulin resistance (*58*). In those countries where there has been chronic undernutrition, insulin resistance may have been selectively advantageous in terms of surviving famine. In populations where energy intake has increased and lifestyles have become more sedentary, however, insulin resistance and the consequent risk of type 2 diabetes have been enhanced. In particular, rapid postnatal catch-up growth appears to further increase the risk of type 2 diabetes in later life. Appropriate nutrition which may reduce type 2 diabetes risk in this situation involves improved nutrition of young children, promoting linear growth, and preventing energy excess by limiting intake of energy-dense foods, controlling the quality of fat supply and facilitating physical activity. At a population level, fetal growth may remain restricted until maternal height improves. This may take several generations to correct. The prevention of type 2 diabetes in infants and young children may be facilitated by the promotion of exclusive breastfeeding, avoiding overweight and obesity, and promoting optimum linear growth.

Table 8

Summary of strength of evidence on lifestyle factors and risk of developing type 2 diabetes

Evidence	Decreased risk	No relationship	Increased risk
Convincing	Voluntary weight loss in overweight and obese people Physical activity		Overweight and obesity Abdominal obesity Physical inactivity Maternal diabetes[a]
Probable	Non-starch polysaccharides (NSP)		Saturated fats Intrauterine growth retardation
Possible	N-3 fatty acids Low glycaemic index foods Exclusive breastfeeding[b]		Total fat intake Trans fatty acids
Insufficient	Vitamin E Chromium Magnesium Moderate alcohol		Excess alcohol

[a] Includes gestational diabetes.

[b] As a global public health recommendation, infants should be exclusively breastfed for the first six months of life to achieve optimal growth, development and health (*59*).

5.3.5 Disease-specific recommendations

Measures aimed at reducing overweight and obesity, and cardiovascular disease may be expected to reduce the risk of developing type 2 diabetes and its complications. Some measures are particularly relevant to reducing the risk for diabetes. These are listed below. The strength of evidence on lifestyle factors is summarized in Table 8.

Overweight and obesity

- Prevention/treatment of overweight and obesity, particularly in high-risk groups.
- Maintaining an optimum BMI, i.e. at the lower end of the normal range.
- For the adult population, maintaining a mean BMI in the range 21–23 kg/m^2, and avoiding weight gain (>5 kg) in adult life.
- Voluntary weight reduction in overweight or obese individuals with impaired glucose tolerance (although screening for such individuals may not be cost-effective in many countries).

Physical activity

- Practicing an endurance activity at moderate or greater level of intensity (e.g. brisk walking) for one hour or more per day on most days per week.

Fat intake

- Ensuring that saturated fat intake does not exceed 10% of total energy and for high-risk groups, fat intake should be < 7% of total energy.

NSP intake

- Achieving adequate intakes of NSP through regular consumption of wholegrain cereals, legumes, vegetables and fruit. A minimum daily intake of 20 g is recommended.

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5.4 Recommendations for preventing cardiovascular diseases

5.4.1 *Background*

The second half of the twentieth century witnessed major global health transitions, changes that profoundly altered life expectancy and ways of living, but also led to an epidemic of noncommunicable diseases. This epidemic is now also emerging, or accelerating, in most developing countries, even as infections and nutritional deficiencies are receding as leading contributors to death and disability (*1*).

As the developing countries experience rapid health transition, the mismatch between health care needs and resources will be widened, and already scarce resources stretched ever more thinly. Because unbalanced diets, obesity and physical inactivity all contribute to heart disease, addressing these, along with tobacco use, can help to stem the epidemic. A large measure of success has already been demonstrated in many industrialized countries.

5.4.2 *Trends*

Cardiovascular diseases (CVD) are the major contributor to the global burden of disease among the noncommunicable diseases. WHO currently attributes one-third of all global deaths (15.3 million) to CVD, with developing countries, low-income and middle-income countries accounting for 86% of the DALYs attributable to CVD lost worldwide in 1998. The increasing burden of CVD will be borne mostly by developing countries in the next two decades.

5.4.3 *Diet, physical activity and disease*

The "lag-time" effect of risk factors for CVD means that present mortality rates are the effect of previous exposure to behavioural risk factors such as inappropriate nutrition, insufficient physical activity and increased tobacco consumption. Overweight, central obesity, high blood pressure, dyslipidaemia, diabetes and low cardio-respiratory fitness are among the biological factors contributing principally to increased risk. Unhealthy dietary practices include the high consumption of saturated fats, salt and refined carbohydrates, as well as low consumption of fruit and vegetables, and these tend to cluster together.

5.4.4 *Strength of evidence*

Convincing associations for reduced risk include consumption of fruits (including berries) and vegetables, fish and fish oils (eicosappentaenoic acid and docosahexaenoic acid), foods high in linoleic acid and potassium, as well as physical activity and low to moderate alcohol intake. While vitamin E intake appears to have no relationship to risk of CVD, there is convincing evidence that myristic and palmitic acids, trans-fatty acids, high sodium intake, overweight and high alcohol intake contribute to an increase in risk. A "probable" level of evidence demonstrates a decreased risk from α-linolenic acid, oleic acid, NSP, wholegrain cereals, nuts (unsalted), folate, plant sterols and stanols, and no relation for stearic acid. There is a probable increase in risk from dietary cholesterol and unfiltered boiled coffee. Possible associations for reduced risk include intake of flavonoids and consumption of soy products, while possible associations for increased risk include fats rich in lauric acid, beta-carotene supplements and impaired fetal nutrition. The evidence supporting these conclusions is summarized below.

Fatty acids and dietary cholesterol

The relationship between dietary fats and CVD, especially coronary heart disease, has been extensively investigated, with strong and consistent associations emerging from a wide body of evidence accrued from animal experiments, as well as observational studies, clinical trials and metabolic studies conducted in diverse human populations (*2*).

Saturated fatty acids raise total and low-density lipoprotein (LDL) cholesterol, but individual fatty acids have different effects (*3–5*). Myristic and palmitic acids have the greatest effect and are abundant in diets rich in dairy products and meat. Stearic acid has not been shown to elevate blood cholesterol and is rapidly converted to oleic

acid in vivo. The most effective replacement for saturated fatty acids in terms of coronary heart disease outcome are polyunsaturated fatty acids, especially linoleic acid. This agrees with the outcome of large randomized clinical trials, in which replacement of saturated and trans-fatty acids by polyunsaturated vegetable oils effectively lowered coronary heart disease risk (*6*).

Trans-fatty acids are geometrical isomers of unsaturated fatty acids that assume a saturated fatty acid-like configuration. Partial hydrogenation, the process used to create trans-fatty acids, also removes essential fatty acids such as linoleic and alpha-linolenic acid. Metabolic studies have demonstrated that trans-fatty acids render the plasma lipid profile even more atherogenic than saturated fatty acids, by not only elevating LDL cholesterol to similar levels but also by decreasing high-density lipoprotein (HDL) cholesterol (*7*). Several large cohort studes have found that intake of trans-fatty acids increases the risk of coronary heart disease (*8, 9*). Most trans-fatty acids are contributed by industrially hardened oils. Even though trans-fatty acids have been eliminated from retail fats and spreads in large parts of the world, deep-fried fast foods and baked goods are a major and increasing source (*7*).

When substituted for saturated fatty acids in metabolic studies, both monounsaturated fatty acids and n-6 polyunsaturated fatty acids lower plasma total and LDL cholesterol concentrations (*10*). Polyunsaturated fatty acids (PUFAs) are somewhat more effective than monounsaturates. The only nutritionally important monounsaturated fatty acids is oleic acid, which is abundant in olive and canola oils and also in nuts. The most important polyunsaturated fatty acid is linoleic acid, which is abundant especially in soybean and sunflower oils. The most important n-3 PUFAs are eicosappentaenoic acid and docosahexaenoic acid found in fatty fish, and alpha-linolenic acid found in plant foods. The biological effects of n-3 PUFAs are wide ranging, involving lipids and lipoproteins, blood pressure, cardiac function, arterial compliance, endothelial function, vascular reactivity and cardiac electrophysiology, as well as potent anti-platelet and anti-inflammatory effects (*11*). The very long chain n-3 PUFAs (eicosappentaenoic acid and docosahexaenoic acid) powerfully lower serum triglycerides but they raise serum LDL cholesterol. Therefore, their effect on coronary heart disease is probably mediated through pathways other than serum cholesterol. Most of the epidemiological evidence related to n-3 PUFAs is derived from studies of fish consumption in populations or interventions involving fish diets in clinical trials (evidence on fish consumption is discussed further below). Fish oils have been used in the Gruppo Italiano per lo Studio della Sopravvivenza nell'Infarto Miocardico (GISSI) trial among survivors of myocardial infarction (*12*). After 3.5 years of follow-up, the group that received fish oil had a 20% reduction in total mortality, a 30% reduction in cardiovascular death and a 45% decrease in sudden death. Several prospective studies have found an inverse association between the intake of alpha-linolenic acid, high in flaxseed, canola, and soybean oils, and risk of fatal coronary heart disease (*13, 14*).

Cholesterol in the blood and tissues is derived from two sources: diet and endogenous synthesis. Dairy fat and meat are major sources. Egg yolk is particularly rich in cholesterol but unlike dairy and meat does not provide saturated fatty acids. Dietary cholesterol raises plasma cholesterol levels (*15*). Observational evidence on an association of dietary cholesterol intake with CVD is contradictory (*16*). There is no requirement for dietary cholesterol and it is advisable to keep the intake as low as possible (*2*). If intake of dairy fat and meat are controlled then there needs to be no severe restriction of egg yolk intake, although some limitation remains prudent.

Dietary plant sterols, especially sitostanol, reduce serum cholesterol by inhibiting cholesterol absorption (*17*). The cholesterol-lowering effects of plant sterols has also been well documented (*18*) and commercial products made of these compounds are widely available, but their long-term effects remain to be seen.

NSP (dietary fibre)

Dietary fibre is a heterogeneous mixture of polysaccharides and lignin that cannot be degraded by the endogenous enzymes of vertebrate animals. Water-soluble fibres include pectins, gums, mucilages and some hemicelluloses. Insoluble fibres include cellulose and other hemicelluloses. Most fibres reduce plasma total and LDL cholesterol, as reported by several trials (*19*). Several large cohort studies carried out in different countries have reported that a high fibre diet as well as a diet high in wholegrain cereals lowers the risk of coronary heart disease (*20–23*).

Antioxidants, folate, and flavonoids

Even though antioxidants could, in theory, be protective against CVD and there is observational data supporting this theory, controlled trials employing supplements have been disappointing. The Heart Outcomes Prevention Evaluation trial (HOPE), a definitive clinical trial relating vitamin E supplementation to CVD outcomes, revealed no effect of

vitamin E supplementation on myocardial infarction, stroke or death from cardiovascular causes in men or women (*24*). Also, the results of the Heart Protection Study indicated that no significant benefits of daily supplementation of vitamin E, vitamin C and beta-carotene were observed among the high-risk individuals that were studied (*25*). In several studies where dietary vitamin C reduced the risk of coronary heart disease, supplemental vitamin C had little effect. Clinical trial evidence is lacking at present. Observational cohort studies suggest a protective role for carotenoids but the meta-analysis of four randomized trials, in contrast, reported an increased risk of cardiovascular death (*26*).

The relationship of folate to CVD has been mostly explored through its effect on homocysteine, which may be an independent risk factor for coronary heart disease and probably also for stroke. Folic acid is required for the methylation of homocysteine to methionine. Reduced plasma folate has been strongly associated with elevated plasma homocysteine levels and folate supplementation has been demonstrated to decrease those levels (*27*). However, the role of homocysteine as an independent risk factor for CVD has been subject to debate, since several prospective studies have not found this association to be independent of other risk factors (*28, 29*). It has also been suggested that elevation of plasma homocysteine is a consequence and not a cause of atherosclerosis, wherein impaired renal function resulting from atherosclerosis raises plasma homocysteine levels (*30, 31*). Data from the Nurses` Health Study showed that folate and vitamin B6, from diet and supplements, conferred protection against coronary heart disease (*32*). A recently published meta-analysis concluded that a higher intake of folate (0.8 mg folic acid) would reduce the risk of ischaemic heart disease by 16% and stroke by 24% (33).

Flavonoids are polyphenolic compounds that occur in a variety of foods of vegetable origin, such as tea, onions and apples. Data from several prospective studies indicate an inverse association of dietary flavonoids with coronary heart disease (*34, 35*). However, confounding may be a major problem and may explain the conflicting results of observational studies.

Sodium and potassium

High blood pressure is a major risk factor for coronary heart disease and both forms of stroke (ischaemic and haemorrhagic). Of the many risk factors associated with high blood pressure, the dietary exposure most investigated has been daily sodium intake. It has been studied extensively in animal experimental models, in epidemiological studies, controlled clinical trials and in population studies on restricted sodium intake (*36, 37*).

All these data show convincingly that sodium intake is directly associated with blood pressure. An overview of observational data in populations suggested that a difference in sodium intake of 100 mmol/day could be associated with average differences in systolic blood pressure of 5 mmHg at age 15–19 years and 10 mmHg at age 60–69 years (*37*). Diastolic blood pressures are reduced by about half as much, but the association increases with age and magnitude of the initial blood pressure. It was estimated that a universal reduction in dietary intake of sodium by 50 mmol/day would lead to a 50% reduction in the number of people requiring antihypertensive therapy, a 22% reduction in the number of deaths resulting from strokes and a 16% reduction in the number of deaths from coronary heart disease. The first prospective study using 24-hour urine collections for measuring sodium intake, which is the only reliable measure, reported an increased risk of acute coronary events, but not stroke events, by increasing sodium excretion (*38*). The association was strongest among overweight men.

Several clinical intervention trials, conducted to evaluate the effects of dietary salt reduction on blood pressure levels have been systematically reviewed (*39, 40*). Based on an overview of 32 methodologically adequate trials, Cutler, Follmann & Allender (*39*) concluded that a daily reduction of sodium intake by 70–80 mmol was associated with a lowering of blood pressure both in hypertensive and normotensive individuals, with systolic and diastolic blood pressure reductions of 4.8/1.9 mmHg in the former and 2.5/1.1 mmHg in the latter. Clinical trials have also demonstrated the sustainable blood pressure lowering effects of sodium restriction in infancy (*41, 42*), as well as in the elderly in whom it provides a useful non-pharmacological therapy (*43*). The results of a low sodium diet trial (*44*) showed that low sodium diets, with 24-hour sodium excretion levels around 70 mmol, are effective and safe. Two population studies of salt, in China and in Portugal, revealed significant reductions in blood pressure in the intervention group (*45, 46*).

A meta-analysis of randomized controlled trials showed that potassium supplements reduced mean blood pressures (systolic/diastolic) by 1.8/1.0 mmHg in normotensive subjects and 4.4/2.5 mmHg in hypertensive subjects (*47*). Several large cohort studies have found an inverse association between potassium intake and risk of stroke (*48, 49*). While potassium supplements have been shown to have protective effects on blood pressure and cardiovascular

diseases, there is no evidence to suggest that long-term potassium supplements should be administered to reduce the risk for CVD. The recommended levels of fruit and vegetable consumption assure an adequate intake of potassium.

Food items and food groups

While the consumption of fruits and vegetables has been widely believed to promote good health, evidence related to their protective effect against CVD has only been presented in recent years (*50*). Numerous ecological and prospective studies have reported a significant protective association for coronary heart disease and stroke with consumption of fruits and vegetables (*50–53*). The effects of increased fruit and vegetable consumption on blood pressure alone or in combination with a low-fat diet, were assessed in the Dietary Approaches to Stop Hypertension (DASH) trial (*54*). While the combination diet was more effective in lowering blood pressure, the fruit and vegetable diet also lowered blood pressure (by 2.8 mmHg systolic and 1.1 mmHg diastolic) in comparison to the control diet. Such reductions, while seeming modest at the individual level, would result in a substantial reduction in population-wide risk of CVD by shifting the blood pressure distribution.

Most, but not all, population studies have shown that fish consumption in populations is associated with a reduced risk of coronary heart disease. A systematic review concluded that the discrepancy in the studies may be a result of differences in the populations studied, with only high-risk individuals benefiting from increasing their fish consumption (*55*). It was estimated that in high-risk populations, an optimum fish consumption of 40–60 grams per day would lead to approximately a 50% reduction in death from coronary heart disease. In a diet and reinfarction trial, two-year mortality was reduced by 29% in survivors of a first myocardial infarction in persons receiving advice to consume fatty fish at least twice a week (*56*). A recent study based on data from 36 countries, reported that fish consumption is associated with a reduced risk of death from all causes as well as CVD mortality (*57*).

Several large epidemiological studies have demonstrated that frequent consumption of nuts was associated with decreased risk of coronary heart disease (*58, 59*). Most of these studies considered nuts as a group, combining many types of nuts. Nuts are high in unsaturated fatty acids and low in saturated fats, contributing to cholesterol lowering by altering the fatty acid profile of the diet as a whole. However, because of the high energy content of nuts, advice to include them in the diet must be tempered in accordance with the desired energy balance.

Several trials indicate that soy has a beneficial effect on plasma lipids (*60, 61*). A composite analysis of 38 clinical trials found that an average consumption of 47 g of soy protein a day led to a 9% decline in total cholesterol and a 13% decline in LDL cholesterol in subjects free of coronary heart disease (*62*). Soy is rich in isoflavones, compounds that are structurally and functionally similar to estrogen. Several animal experiments suggest that the intake of these isoflavones may provide protection against coronary heart disease, but human data on efficacy and safety are still awaited.

There is convincing evidence that low to moderate alcohol consumption lowers the risk of coronary heart disease. In a systematic review of ecological, case–control and cohort studies in which specific associations were available between risk of coronary heart-disease and consumption of beer, wine and spirits, it was found that all alcoholic drinks are linked with lower risk (*63*). However, other cardiovascular and health risks associated with alcohol do not favour a general recommendation for its use.

Boiled, unfiltered coffee raises total and LDL cholesterol because coffee beans contain a terpenoid lipid called cafestol. The amount of cafestol in the cup depends on the brewing method: it is zero for paper-filtered drip coffee, and high in the unfiltered coffee still used, for example, in Greece, the Middle East and Turkey. Intake of large amounts of such coffee markedly raises serum cholesterol and has been associated with coronary heart disease in Norway (*64*). A shift from unfiltered, boiled coffee to filtered coffee has contributed significantly to the decline in serum cholesterol in Finland (*65*).

Table 9

Summary of strength of evidence on lifestyle factors and risk of developing cardiovascular diseases

Evidence	Decreased risk	No relationship	Increased risk
Convincing	Regular physical activity	Vitamin E supplements	Myristic and palmitic acids
	Linoleic acid		Trans-fatty acids
	Fish and fish oils (eicosappentaenoic acid and docosahexaenoic acid)		High sodium intake
	Vegetables and fruits (including berries)		Overweight
	Potassium		High alcohol intake (for stroke)
	Low to moderate alcohol intake (for coronary heart disease)		
Probable	a-linolenic acid	Stearic acid	Dietary cholesterol
	Oleic acid		Unfiltered boiled coffee
	NSP		
	Wholegrain cereals		
	Nuts (unsalted)		
	Plant sterols/stanols		
	Folate		
Possible	Flavonoids		Fats rich in lauric acid
	Soy products		Impaired fetal nutrition
			Beta-carotene supplements
Insufficient	Calcium		Carbohydrates
	Magnesium		Iron
	Vitamin C		

5.4.5 ***Disease-specific recommendations***

Measures aimed at reducing the risk of CVD are outlined below. The strength of evidence on lifestyle factors is summarized in Table 9.

Fats

Dietary intake of fats, especially the qualitative composition of fats in the diet, strongly influences the risk of cardiovascular diseases such as coronary heart disease and stroke, through effects on blood lipids, thrombosis, blood pressure, arterial (endothelial) function, arrythmogenesis and inflammation.

In developing countries, where energy intake for some population groups may be inadequate, energy expenditure is high and body fat stores are low (BMI < 18.5 kg/m^2). The amount and quality of fat supply has to be considered keeping in mind the need to meet energy requirements. The evidence shows that intake of saturated fatty acids is directly related to cardiovascular risk. The traditional target is to restrict the intake of saturated fatty acids to less than 10% of daily energy intake and less than 7% for high-risk groups. If populations are consuming less than 10%

they should not increase that level of intake. Within these limits, intake of foods rich in myristic and palmitic acids should be replaced by fats with lower content of those fatty acids. Specific sources of saturated fat, such as coconut and palm oil, provide low-cost energy and may be an important source of energy for the poor. Not all saturated fats have similar metabolic effects; those with 12–16 carbons in the fatty acid chain have a greater effect on raising LDL cholesterol. This implies that the fatty acid composition of the fat source should be examined. As populations progress in the nutrition transition and energy excess becomes a potential problem, restricting these fatty acids becomes progressively more relevant to ensuring cardiovascular health.

To promote cardiovascular health, diets should provide a very low intake of trans-fatty acids (hydrogenated oils and fats). This means an intake that is as low as possible. In practice, this implies an intake of less than 1% of daily energy intake. This recommendation is especially relevant in developing countries where low-cost hydrogenated fat is frequently consumed. The potential effect of human consumption of hydrogenated oils of unknown physiological effects (e.g. marine oils) is of great concern.

Diets should provide an adequate intake of polyunsaturated fatty acids (PUFA): 6%–10% of daily energy intake. There should also be an optimal balance of n-6 PUFA and n-3 PUFA 5%–8% and 1%–2% of daily energy intake, respectively.

Intake of oleic acid, a monounsaturated fatty acid, should make up the rest of daily energy intake from fats, with daily total fat intake ranging from 15% up to 30% of daily energy intake. Total fat intake may be based on current levels of population consumption in different regions and modified to take account of age, activity and ideal body weight. Where obesity is prevalent, an intake in the lower part of the range is preferable to achieve a lower energy intake. While there is no evidence directly linking the quantity of daily fat intake to an increased risk of CVD, total fat consumption should be limited to enable the goals of reduced intake of saturated and trans-fatty acids to be met easily in most populations and to avoid the potential problems of undesirable weight gain that may arise from unrestricted fat intake. It should be noted that highly active groups with diets rich in vegetables, legumes, fruits and wholegrain cereals will limit the risk of unhealthy weight gain on a diet comprising a total fat intake of up to 35%.

These dietary goals can be met by limiting the intake of fat from dairy and meat sources, avoiding the use of hydrogenated oils and fats in cooking and manufacture of food products, using appropriate edible vegetable oils in small amounts, and ensuring a regular intake of fish (one to two times per week) or plant sources of a-linolenic acid. Preference should be given to food preparation practices that employ non-frying methods.

Fruits and vegetables

Fruits and vegetables contribute to cardiovascular health through a variety of phyto-nutrients, potassium and fibre. Daily intake of fresh fruit and vegetables (including berries, green leafy and cruciferous vegetables and legumes), in an adequate quantity (400–500 grams per day), is recommended to reduce the risk of coronary heart disease, stroke and high blood pressure.

Sodium

Dietary intake of sodium, from all sources, influences blood pressure levels in populations and should be limited so as to reduce the risk of coronary heart disease and both forms of stroke. Current evidence suggests that an intake of no more than 70 mmol or 1.7 grams of sodium per day is beneficial in reducing blood pressure. The special situation of individuals who may be adversely affected by sodium reduction needs to be kept in mind (pregnant women and non-acclimated people who perform strenuous physical activity in hot environments).

Limitation of dietary sodium intake to meet these goals should be achieved by restricting daily salt (sodium chloride) intake to less than 5 grams per day. This should take into account total sodium intake from all dietary sources, for example additives such as monosodium glutamate and preservatives. Use of potassium-enriched low-sodium substitutes is one way to reduce sodium intake. The need to adjust salt iodization, depending on observed sodium intake and surveillance of iodine status of the population, should be recognized.

Potassium

Adequate dietary intake of potassium lowers blood pressure and is protective against stroke and cardiac arrythmias. Potassium intake should be at a level which will keep the sodium to potassium ratio close to 1.0, i.e. at daily

potassium intake levels of 70–80 mmol per day. This may be achieved through adequate daily consumption of fruits and vegetables.

NSP (dietary fibre)1

Fibre is protective against coronary heart disease and has also been used in diets to lower blood pressure. Adequate intake may be achieved through fruits, vegetables and wholegrain cereals.

Fish

Regular fish consumption (1–2 servings per week) is protective against coronary heart disease and ischaemic stroke and is recommended. The serving should provide an equivalent of 200–500 mg of eicosappentaenoic and docosahexaenoic acid. People who are vegetarians are recommended to ensure adequate intake of plant sources of α-linolenic acid.

Alcohol

Although regular low-to-moderate consumption of alcohol is protective against coronary heart disease, other cardiovascular and health risks associated with alcohol do not favour a general recommendation for its use.

Physical activity

Physical activity is related to the risk of cardiovascular diseases, especially coronary heart disease, in a consistent inverse dose–response fashion when either volume or intensity are used for assessment. These relationships apply to both incidence and mortality rate from all cardiovascular diseases and from coronary heart disease. At present, no consistent dose–response relationship can be found between risk of stroke and physical activity. The lower limits of volume or intensity of the protective dose of physical activity have not been defined with certainty, but the current recommendation of at least 30 minutes of at least moderate-intensity physical activity on most days of the week is considered sufficient. A higher volume or intensity of activity would confer a greater protective effect. The recommended amount of physical activity is sufficient to rise cardio-respiratory fitness to the level that has been shown to be related to decreased risk of cardiovascular disease. Individuals who are unaccustomed to regular exercise or have a high-risk profile for CVD should avoid sudden and high-intensity bursts of physical activity.

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1 Specific amounts will depend on the analytical methodologies used to measure fibre.

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5.5 Recommendations for preventing cancer

5.5.1 *Background*

Cancer is caused by a variety of identified and unidentified factors. The most important established cause of cancer is tobacco. Other important determinants of cancer risk include diet, alcohol and physical activity, infections, hormonal factors and radiation. The relative importance of cancers as a cause of death is increasing, mostly because of the increasing proportion of people who are old, and also in part because of reductions in mortality from some other causes, especially infectious diseases. The incidence of cancers of the lung, colon and rectum, breast and prostate generally increases in parallel with economic development, while the incidence of stomach cancer usually declines with development.

5.5.2 *Trends*

Cancer is now a major cause of mortality throughout the world and, in the developed world, is generally exceeded

only by cardiovascular diseases. An estimated 10 million new cases and over 6 million deaths from cancer occurred in 2000 (*1*). As developing countries become urbanized, patterns of cancer, including those most strongly associated with diet, tend to shift towards those of economically developed countries. Between 2000 and 2020, the total number of cases of cancer in the developing world is predicted to increase by 73% and, in the developed world, to increase by 29%, largely as a result of an increase in the number of old people (*1*).

5.5.3 *Diet, physical activity and cancer*

Dietary factors are estimated to account for approximately 30% of cancers in industrialized countries (*2*), making diet second only to tobacco as a theoretically preventable cause of cancer. This proportion is thought to be about 20% in developing countries (*3*), but may grow according to dietary change, particularly if the importance of other causes, especially infections, declines. Cancer rates change as populations move between countries and adopt different dietary (and other) behaviours, further implicating dietary factors in the etiology of cancer.

Body weight and physical inactivity together are estimated to account for approximately one-fifth to one-third of several of the most common cancers, specifically cancers of the breast (postmenopausal), colon, endometrium, kidney and oesophagus (adenocarcinoma) (4).

5.5.4 *Strength of evidence*

Research to date has uncovered few definite relationships between diet and cancer risk. Dietary factors which convincingly increase risk are overweight/obesity, alcoholic beverages, aflatoxins, and some forms of salting and fermenting fish. Convincing evidence indicates that physical activity decreases the risk of colon cancer. Factors which probably increase risk include preserved meats, salt-preserved foods and salt, and very hot (thermally) drinks and food. Probable protective factors are fruits and vegetables (and physical activity for breast cancer). After tobacco, overweight/obesity appears to be the most important known avoidable cause of cancer.

The role of diet in the etiology of the major cancers

Cancers of the oral cavity, pharynx and oesophagus. In developed countries the main risk factors for cancers of the oral cavity, pharynx and oesophagus are alcohol and tobacco, and up to 75% of such cancers are attributable to these two lifestyle factors (5). Overweight/obesity is an established risk factor specifically for adenocarcinoma (but not squamous cell carcinoma) of the oesophagus (6–8). In developing countries, around 60% of cancers of the oral cavity, pharynx and oesophagus are thought to be a result of micronutrient deficiencies related to a restricted diet that is low in fruit and vegetables and animal products (5, 9). The relative roles of various micronutrients are not yet clear (5, 9). There is also consistent evidence that consuming drinks and foods at a very high temperature increases the risk for these cancers (10). Nasopharyngeal cancer is particularly common in South East Asia (11), and has been clearly associated with a high intake of Chinese-style salted fish, especially during early childhood (12, 13), as well as with infection with the Epstein-Barr virus (2).

Stomach cancer. Until about 20 years ago stomach cancer was the most common cancer in the world, but mortality rates have been falling in all industrialized countries (*14*) and stomach cancer is currently much more common in Asia than in North America or Europe (*11*). Infection with the bacterium Helicobacter pylori is an established risk factor, but not a sufficient cause, for the development of stomach cancer (*15*). Diet is thought to be important in the etiology of this disease; substantial evidence suggests that risk is increased by high intakes of some traditionally preserved salted foods, especially meats and pickles, and with salt per se, and that risk is decreased by high intakes of fruit and vegetables (*16*), perhaps because of their vitamin C content. Further prospective data are needed, in particular to examine whether some of the dietary associations may be partly confounded by Helicobacter pylori infection and whether dietary factors may modify the association of Helicobacter pylori with risk.

Colorectal cancer. Colorectal cancer incidence rates are approximately tenfold higher in developed than in developing countries (*11*), and it has been suggested that diet-related factors may account for up to 80% of the between-country differences in rates (*17*). The best established diet-related risk factor is overweight/obesity (*8*).

Physical activity has been consistently associated with a reduced risk of colon cancer (but not of rectal cancer) (*8, 18*). These factors together, however, do not explain the large variation between populations, and there is almost universal agreement that some aspects of "westernized" diet are a major determinant of risk; there is some evidence that risk is increased by high intakes of meat and fat, and that risk is decreased by high intakes of fruit and vegetables, dietary fibre, folate and calcium, but none of these hypotheses has been firmly established.

International correlation studies show a strong association between per capita consumption of meat and colorectal cancer mortality (*19*), and a recent systematic review concluded that preserved meat is associated with an increased risk for colorectal cancer but that fresh meat is not (*20*), whereas most studies have not observed positive associations with poultry or fish (*9*). Overall, the evidence is not yet conclusive but suggests that high consumption of preserved and red meat probably increases the risk for colorectal cancer.

As with meat, international correlation studies show a strong association between per capita consumption of fat and colorectal cancer mortality (*19*). However, the results of observational studies of fat and colorectal cancer have, overall, not been supportive of an association with fat intake (*9, 21*).

Many case–control studies of colorectal cancer have observed a moderately lower risk in association with high consumption of fruits and vegetables and/or dietary fibre (*22, 23*), but the results of recent large prospective studies have been inconsistent (*24–26*). Furthermore, results from randomized controlled trials have not shown that intervention over a 3–4 year period with supplemental fibre or a diet low in fat and high in fibre and fruit and vegetables can reduce the recurrence of colorectal adenomas (*27–29*). It is possible that some of the inconsistencies are a result of differences between studies in the types of fibre eaten and in the methods for classifying fibre in food tables, or that the association with fruits and vegetables is principally arises from an increase in risk at very low levels of consumption (*30*). At present, the evidence currently available suggests that intake of fruit and vegetables probably reduces the risk for colorectal cancer.

Recent studies have suggested that vitamins and minerals might influence the risk for colorectal cancer. Some prospective studies have suggested that a high intake of folate from diet or vitamin supplements is associated with a reduced risk for colon cancer (*31–33*). Another promising hypothesis is that relatively high intakes of calcium may reduce the risk for colorectal cancer; several observational studies have supported this hypothesis (*9, 34*), and two trials have suggested that supplemental calcium may have a modest protective effect on the recurrence of colorectal adenomas (29, *35*).

Liver cancer. Approximately 75% of cases of liver cancer occur in developing countries, and liver cancer rates vary over 20-fold between countries, being

much higher in sub-Saharan Africa and South East Asia than in North America and Europe and (*11*). The major risk factor for hepatocellular carcinoma, the main type of liver cancer, is chronic infection with hepatitis B, and to a lesser extent, hepatitis C virus (*36*). Ingestion of foods contaminated with the mycotoxin aflatoxin (*13, 37*) is an important risk factor among people in developing countries, together with active hepatitis virus infection. Excessive alcohol consumption is the main diet-related risk factor for liver cancer in industrialized countries, probably via the development of cirrhosis and alcoholic hepatitis (*5*).

Pancreatic Cancer. Cancer of the pancreas is more common in industrialized countries than in developing countries (11, 38). Overweight/obesity possibly increases the risk (9, 39). Some studies have suggested that risk is increased by high intakes of meat, and reduced by high intakes of vegetables, but these data are not consistent (9).

Lung cancer. Lung cancer is the most common cancer in the world (*11*). Heavy

smoking increases the risk by around 30-fold, and smoking causes over 80% of lung cancers in developed countries (*5*). Numerous observational studies have found that lung cancer patients generally report a lower intake of fruits, vegetables and related nutrients (such as β-carotene) than controls (*9, 34*). The only one of these factors to have been tested in controlled trials, namely β-carotene, has, however, failed to produce any benefit when given as a supplement for up to 12 years (*40–42*). The possible effect of diet on lung cancer risk remains controversial, and the apparent protective effect of fruit and vegetables may be largely the result of residual confounding by smoking, since smokers generally consume less fruit and vegetables than non-smokers. In public health terms, the overriding priority for preventing lung cancer is to reduce the prevalence of smoking.

Breast cancer. Breast cancer is the second most common cancer in the world and the most common cancer among women. Incidence rates are about five times higher in industrialized countries than in less developed countries and Japan (11). Much of this international variation is a result of differences in established reproductive risk factors such as age at menarche, parity and age at births, and breastfeeding (43, 44), but differences in dietary habits and physical activity may also contribute. In fact, age at menarche is partly determined by dietary factors, in that restricted dietary intake during childhood and adolescence leads to delayed menarche. Adult height, also, is weakly positively associated with risk, and is partly determined by dietary factors during childhood and adolescence (43). Estradiol and perhaps other hormones play a key role in the etiology of breast cancer (43), and it is possible that any further dietary effects on risk are mediated by hormonal mechanisms.

The only dietary factors which have been established to increase the risk for breast cancer are obesity and alcohol. Obesity increases breast cancer risk in postmenopausal women by around 50%, probably by increasing serum concentrations of free estradiol (*43*). Obesity does not increase risk among premenopausal women, but obesity in premenopausal women is likely to lead to obesity throughout life and therefore to an eventual increase in breast cancer risk. For alcohol, there is now a large amount of data from well-designed studies which consistently shows a small increase in risk with increasing consumption, with about a 10% increase in risk for an average of one alcoholic drink every day (*45*). The mechanism for this association is not known, but may involve increases in estrogen levels (*46*).

The results of studies of other dietary factors including fat, meat, dairy products, fruit and vegetables, fibre and phyto-estrogens are inconsistent (*9, 34, 47, 48*).

Endometrial Cancer. Endometrial cancer risk is about threefold higher in obese women than lean women (*8, 49*), probably because of the effects of obesity on hormone levels (*50*). Some case–control studies have suggested that diets high in fruit and vegetables may reduce risk and that diets high in saturated or total fat may increase risk, but the data are limited (*9*).

Prostate cancer. Prostate cancer incidence rates are strongly affected by diagnostic practices and therefore difficult to interpret, but mortality rates show that death from prostate cancer is about ten times more common in North America and Europe than in Asia (11).

Little is known about the etiology of prostate cancer, although ecological studies suggest that it is positively associated with a "westernized" diet (*19*). The data from prospective studies have not established causal or protective associations for specific nutrients or dietary factors (*9, 34*). Diets high in red meat, dairy products and animal fat have frequently been implicated in the development of prostate cancer, although the data are not entirely consistent (*9, 51–53*). Randomized controlled trials have provided substantial, consistent evidence that supplements of β-carotene do not alter the risk for prostate cancer (*40, 41, 54*) but have suggested that vitamin E (*54*) and selenium (*55*) might have a protective effect. Lycopene, primarily from tomatoes, has been associated with a reduced risk in some observational studies, but the data are not consistent (*56).* Hormones control the growth of the prostate, and diet might affect prostate cancer risk by affecting hormone levels.

Kidney cancer. Overweight/obesity is an established risk factor for cancer of the kidney, and may account for up to 30% of kidney cancers in both men and women (*57).*

Summary of strength of evidence. Table 10 provides a summary of strength of evidence with regard to the role of various risk factors.

Table 10

Summary of strength of evidence on dietary factors, physical activity and the risk of developing cancer

Evidence	Decreased risk	Increased risk
Convincing[a]	Physical activity (colon)	Overweight/obesity (oesophagus, colorectum, breast in postmenopausal women, endometrium, kidney) Alcohol (oral cavity, pharynx, larynx, oesophagus, liver, breast) Aflatoxin (liver) Chinese-style salted fish (nasopharynx)
Probable[a]	Fruit and vegetables (oral cavity, oesophagus, stomach, colorectum[b]) Physical activity (breast)	Preserved meat (colorectum) Salt-preserved foods & salt (stomach) Very hot (thermally) drinks and food (oral cavity, pharynx, oesophagus)
Possible/Insufficient	Fibre, soya, fish, n-3 fatty acids, carotenoids, vitamins B_2, B_6, folate, B_{12}, C, D, E, calcium, zinc, selenium, non-nutrient plant constituents (e.g. allium compounds, flavonoids, isoflavones, lignans)	Animal fats, heterocyclic amines, polycyclic aromatic hydrocarbons, nitrosamines

[a] The "convincing" and "probable" categories in this report correspond to the "sufficient" category of the IARC report on weight control and physical activity (*54*) in terms of the public health and policy implications.
[b] For colorectal cancer, a protective effect of fruit and vegetable intake has been suggested by many case-control studies but has not been supported in several large prospective studies, suggesting that if a benefit does exist it is likely to be modest.

The Consultation recognized the limitation of the data from diet and cancer in terms of representation of the developing world. There are very limited data from Africa, Asia and Latin America; these regions represent two-thirds or more of the world population, and there is an urgent need for epidemiological research on diet and cancer in these regions. The need to evaluate the role of food processing methods, traditional and industrial, was also identified. Microbiological and chemical food contaminants may also contribute to carcinogenicity of diets.

The nutrition transition is accompanied by changes in prevalence of specific cancers. For some cancers, such as stomach cancer, this may be beneficial while for others, such as colorectal and breast cancers, the changes are adverse.

5.5.5 *Disease-specific recommendations*

1. Maintain weight (among adults) such that BMI is in the range of 18.5–24.9kg/m^2, and avoid weight gain (> 5 kg) during adult life (*59*).

2. Maintain regular physical activity. The primary goal should be to perform physical activity on most days of the week. 60 minutes per day of moderate-intensity activity, such as walking, may be needed to maintain healthy body weight in otherwise sedentary people. More vigorous activity, such as fast walking, may give some additional benefits for cancer prevention (*4*).

3. Consumption of alcoholic beverages is not recommended: if consumed, do not exceed two units1 per day.

4. Chinese-style fermented salted fish should only be consumed in moderation, especially during childhood. Overall consumption of salt-preserved foods and salt should be moderate.

1 One unit is equivalent to approximately 10 g of alcohol and is provided by one glass of beer, wine or spirits.

5. Minimize exposure to aflatoxin in foods.

6. Have a diet which includes at least 400 g per day of total fruit and vegetables.

7. Meat: those who are not vegetarian are advised to moderate consumption of preserved meat (e.g. sausages, salami, bacon, ham etc.)1

8. Do not consume foods or drinks when they are at a very hot (scalding hot) temperature.

1 Poultry and fish (except Chinese-style salted fish) have been studied and found not to be associated with increased risk for cancer.

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5.6 Recommendations for preventing dental diseases

5.6.1 *Background*

Oral health is related to diet in many ways, for example, nutritional influences on cranio-facial development, oral cancer and oral infectious diseases. The purpose of this review, however, is to focus on the nutritional aspects of dental diseases. Dental diseases include dental caries, developmental defects of enamel, dental erosion and periodontal disease. Dental diseases are a costly burden to health care services, costing between 5% and 10% of total health care expenditures and exceeding the cost of treating cardiovascular disease, cancer and osteoporosis in industrialized countries (*1*). In low-income countries, the cost of traditional restorative treatment of dental disease would probably exceed the available resources for health care. Health promotion and preventive strategies are clearly more affordable and sustainable.

Despite a low mortality rate associated with dental diseases, they have a detrimental effect on quality of life both in childhood and older age, having an impact on self-esteem, eating ability, nutrition and health. In modern society, a significant role of teeth is to enhance appearance; facial appearance is very important in determining an individual's integration into society, and teeth also play an essential role in speech and communication. Oral diseases are associated with considerable pain, anxiety and impaired social functioning (*2*, *3*). Dental decay may result in tooth loss, which reduces the ability to eat a nutritious diet, the enjoyment of food, the confidence to socialize and quality of life (*4–6*).

5.6.2 *Trends*

The amount of dental decay is measured using the dmf/DMF index representing primary dentition/permanent dentition. This is a count of the number of teeth or surfaces in a person's mouth that are decayed, missing or filled as a result of caries. Dental disease may result in tooth loss and an additional dental status indicator therefore is the proportion of the population who are edentulous (have no natural teeth).

In most low-income countries, the prevalence rate of dental caries is relatively low and more than 90% of caries are untreated. Available reported data (*7*) show that the mean number of decayed, missing or filled permanent teeth (DMFT) at age 12 years in low-income countries is 1.9 as against 3.3 DMFT in middle-income countries and 2.1 DMFT in high-income countries (Table 11).

Data on the level of dental caries in the permanent dentition of 12-year-olds show two distinct trends. First, the fall in the prevalence of dental caries in developed countries, and second the increase in the prevalence of the disease in some developing countries that have increased their consumption of sugars and have not yet been introduced to the presence of adequate amounts of fluoride. Despite the marked decline in dental caries in developed countries over the past 30 years, the prevalence of dental caries remains unacceptably high in many developed countries. Even in countries with low average DMFT scores, a significant proportion of children still have dental caries. There is some indication that the favourable trends in levels of dental caries in permanent teeth have come to a halt (*8*).

Table 11

Trends in dental caries levels of 12-year-olds

Country or area	Mean DMFT[a] per person aged 12 years					
Industrialized	Year	DMFT[a]	Year	DMFT[a]	Year	DMFT[a]
Australia	1956	9.3	1982	2.1	1998	0.8
Finland	1975	7.5	1982	4.0	1997	1.1
Japan	1975	5.9	1993	3.6	1999	2.4
Norway	1940	12.0	1979	4.5	1999	1.5
Romania	1985	5.0	1991	4.3	1996	3.8
Switzerland	1961–1963	9.6	1980	1.7	1996	0.8
United Kingdom	1983	3.1	1993	1.4	1996–1997	1.1
United States of America	1946	7.6	1980	2.6	1998	1.4
Developing						
Chile	1960	2.8	1978	6.6	1996	4.1
Dem. Rep. of Congo	1971	0.1	1982	0.3	1987	0.4–1.1
French Polynesia	1966	6.5	1986	3.2	1994	3.2
Islamic Republic of Iran	1974	2.4	1976	4.9	1995	2.0
Jordan	1962	0.2	1981	2.7	1995	3.3
Mexico	1975	5.3	1991	2.5–5.1	1997	2.5
Morocco	1970	2.6	1980	4.5	1999	2.5
Philippines	1967	1.4	1981	2.9	1998	4.6
Uganda	1966	0.4	1987	0.5	1993	0.4

Source: reference (*7*).

[a] Decayed, missing, filled permanent teeth.

Many developing countries have low DMFT values but a high prevalence of dental caries in the primary dentition. Data on 5-year-old children in Europe suggest that the trend towards reduced prevalence of dental decay has halted (*9–11*). In children aged 5–7 years, average dmft values of below 2.0 have been reported for Denmark, England, Finland, Italy, Netherlands and Norway (*12*). Higher dmft values were reported recently for Belarus (4.7) (*13*), Hungary (4.5) (*14*), Romania (4.3) (*15*) and the Russia Federation(4.7) (*16*).

Being caries-free at age 12 years does not imply being caries-free for life. The mean DMFT in countries of the European Union after 1988 varied between 13.4 and 20.8 at 35–44 years (*17*). The WHO guidelines on oral health state that at age 35–44 years a DMFT of 14 or above is considered high. In most developing countries, the level of caries in adults of this age group is lower, for example 2.1 in China (*18*) or 5.7 in Niger (*19*). Few data are available on the prevalence and severity of root caries in older adults, but with the increasingly ageing population and greater retention of teeth, the problem of root caries is likely to grow as a public health concern in the future.

There has been a reduction over-time in the number of edentulous persons for several industrialised countries (3). However, there is still a large proportion of older adults who are edentulous or partially dentate. As the ageing population increases, tooth loss will continue to affect a sizeable number of persons world wide. Table 2 summarizes the available reported information on the prevalence of edentulousness in old-age populations throughout the world.

Table 12

Prevalence of edentulousness in older people throughout the world

WHO region	Prevalence of edentulousness (%)	Age group (years)
African Region		
Gambia	6	65
Madagascar	25	65–74
Region of The Americas		
USA	26.0	65–69
Canada	58.0	65
Eastern Mediterranean Region		
Egypt	7	65
Lebanon	20	64–75
Saudi Arabia	31–46	65
European Region		
Albania	69	65
Austria	15	65–74
Bosnia and Herzegovina	78	65
Bulgaria	53	65
Denmark	27	65–74
Finland	41	65
Hungary	27	65–74
Iceland	15	65–74
Italy	19	65–74
Lithuania	14	65–74
Poland	25	65–74
Romania	26	65–74
Slovakia	44	65–74
Slovenia	16	65
United Kingdom	46	65
Western Pacific Region		
Cambodia	13.0	65–74
China	11	65–74
Malaysia	57	65
Singapore	21	65
South-East Asian Region		
India	19	65–74
Indonesia	24	65
Sri Lanka	37	65–74
Thailand	16.3	65

Source: reference (*7*).

Dental erosion is a relatively newly recognized dental problem in many countries throughout the world, and is related to diet. There is anecdotal evidence that the prevalence is increasing in industrialized countries, but there are no data over time to indicate patterns of this disease. There are insufficient data available to comment on worldwide trends; in some populations, however, approximately 50% of children are affected (*20*).

5.6.3 *Diet and disease*

Nutritional status affects the teeth pre-eruptively, although this influence is much less important than the post-eruptive local effect of diet (*21*). Deficiencies of vitamins D and A and protein–energy malnutrition have been associated with enamel hypoplasia and salivary gland atrophy (which reduces the mouth's ability to buffer plaque acids). In developing countries, in the absence of dietary sugars, undernutrition is not associated with dental caries. Undernutrition coupled with increased intake of sugars may exacerbate the risk of caries.

There is some evidence to suggest that periodontal disease progresses more rapidly in undernourished populations (*22*); the important role of nutrition in maintaining an adequate host immune response may explain this observation. Apart from severe vitamin C deficiency, which may result in scurvy-related periodontitis, there is little evidence at present for an association between diet and periodontal disease. Current research is investigating the potential role of antioxidant nutrients in periodontal disease. Poor oral hygiene is the most important risk factor in the development of periodontal disease (*21*). Undernutrition exacerbates the severity of oral infections (e.g. acute necrotizing ulcerative gingivitis) and may eventually lead to their evolution into life-threatening diseases such as noma, a dehumanizing oro-facial gangrene (*23*).

Dental caries occur because of demineralization of enamel and dentine by organic acids formed by bacteria in dental plaque through the anaerobic metabolism of sugars derived from the diet (*24*). Organic acids increase the solubility of calcium hydroxyapatite in the dental hard tissues and demineralization occurs. Saliva is saturated with calcium and phosphate at pH 7 which promotes remineralization. If the oral pH remains high enough for sufficient time then complete remineralization of enamel may occur. If the acid challenge is too great, however, demineralization dominates and the enamel becomes more porous until finally a carious lesion forms (*25*). The development of caries requires sugars and the occurrence of bacteria, but is influenced by the susceptibility of the tooth, the bacterial profile, and the quantity and quality of the saliva.

Dietary sugars and dental caries

There is a wealth of evidence from many different types of investigation, including human studies, animal experiments, and experimental studies in vivo and in vitro to show the role of dietary sugars in the etiology of dental caries (*21*). Collectively, data from these studies provide an overall picture of the cariogenic potential of carbohydrates. Sugars are undoubtedly the most important dietary factor in the development of dental caries. Here, the term "sugars" refers to all monosaccharides and disaccharides, while the term "sugar" refers only to sucrose. The term "free sugars" refers to all monosaccharides and disaccharides added to foods by the manufacturer, cook or consumer, plus sugars naturally present in honey, fruit juices and syrups. The term "fermentable carbohydrate" refers to free sugars, glucose polymers, oligosaccharides and highly refined starches; it excludes non-starch polysaccharides and raw starches.

Worldwide epidemiological studies have compared sugar consumption and levels of dental caries at country level. Sreebny (*26, 27*) correlated the dental caries experience (DMFT) of 12-year-olds to sugar supplies data of 47 countries and found a significant correlation (+0.7); 52% of the variation in caries levels was explained by the per capita availability of sugar. In countries with a consumption level of sugar <18 kg per person per year caries experience was consistently < DMFT 3. A later analysis by Woodward and Walker (*28*) did not find an association for developed countries. Sugar availability nevertheless accounted for 28% of the variation in levels of dental caries; 23 out of 26 countries with sugars availability <50 g/day had mean DMFT for 12-year-olds of <3, whereas only

half of the countries with sugar availability above this level had achieved DMFT<3.

Miyazaki & Morimoto (*29*) reported a significant correlation (r= +0.91) between sugar availability in Japan and DMFT at age 12 years between 1957 and 1987. Populations that had reduced sugar availability during the Second World War showed a reduction in dental caries which subsequently increased when the restriction was lifted (*30–32*). Although these data are from before widespread use of fluoride dentifrice, Weaver (*33*) observed a reduction in dental caries between 1943 and 1949 in areas of Northern England with both high and low concentration of fluoride in drinking water.

Isolated communities with a traditional way of life and a consistently low intake of sugars have very low levels of dental caries. As economic levels in such societies rise, the amount of sugar and other fermentable carbohydrates in the diet increases and this is associated with a marked increase in dental caries. Examples of this trend have been reported among the Inuit in Alaska, USA (*34*), as well as in populations in Ethiopia (*35*), Ghana (*36*), Nigeria (*37*), Sudan (*38*),and on the Island of Tristan da Cunha, St. Helena (*39*).

There is evidence to show that many groups of people with high exposure to sugars also have levels of caries higher than the population average. Examples include children with chronic diseases requiring long-term sugar-containing medicines (*40*), and confectionery workers (*41–44*). Likewise, experience of dental caries has seldom been reported in groups of people who have a habitually low intake of sugars, for example children of dentists (*45, 46*) and children in institutions with strict dietary regimens (*47, 48*). A weakness of the data from observations of populations is that changes in intake of sugars are often associated with changes in the intake of refined starches, making it impossible to attribute changes in dental caries solely to changes in the intake of sugars. An exception to this are the data from studies of children with hereditary fructose intolerance (HFI). People with HFI are restricted to a low intake of sugars but do not have a restriction on intake of starch. Studies have shown that people with HFI have a low intake of sugars and a higher than average intake of starch, yet seldom experience caries (*49*).

Human intervention studies are rare, and those that have been reported are now decades old and were conducted in the pre-fluoride era before the strong link between sugars intake and dental caries levels was established. It would not be possible to repeat such studies today because of ethical constraints. The Vipeholm study, conducted in an adult mental institution in Sweden between 1945 and 1953 (*50*), investigated the effects of consuming sugary foods of varying stickiness and at different times throughout the day on the development of caries. It was found that sugar, even when consumed in large amounts, had little effect on caries increment if it was ingested up to a maximum of four times a day at mealtimes only. Increased frequency of consumption of sugar in between meals was, however, associated with a marked increase in dental caries. It was also found that the increase in dental caries activity disappears on withdrawal of sugar-rich foods. Despite the complicated nature of the study the conclusions are valid, although they apply to the pre-fluoride era. The study in Turku was a controlled dietary intervention study carried out on adults in Finland in the 1970s which showed that almost total substitution of sucrose in the diet with xylitol (a non-cariogenic sweetener) resulted in an 85% reduction in dental caries over a 2-year period (*51*).

Numerous cross-sectional epidemiological studies have compared sugars intake with dental caries levels in many countries of the world. Those before the early 1990s have been summarized by Rugg-Gunn (*21*). Nine out of 21 studies that compared amount of sugars consumed to caries increment found significant associations, while the other 12 did not. Moreover, 23 out of 37 studies that investigated the association between frequency of sugars consumption and caries levels found significant relationships, while 14 failed to find an association.

A cross-sectional study in the United States of 2514 people aged 9–29 conducted between 1968 and 1970 found that the dental caries experience of adolescents eating the highest amounts of sugars (upper 15% of the sample) was twice that of those eating the lowest amounts (lower 15% of sample) (*52*). Granath et al. (*53*) showed that intake of sugars was the most important factor associated with caries in the deciduous dentition of pre-school children in Sweden. When the effects of oral hygiene and fluoride were kept constant, the children with a low intake of sugars in between meals had up to 86% less caries than those with high intakes of sugars. Other studies have found fluoride exposure and oral hygiene to be more strongly associated with caries than sugars consumption (*54, 55*). A recent study of in the United Kingdom a representative sample of children aged 4–18 years showed no significant relationship between caries experience and level of intake of free sugars; in the age group 15–18 years, however, the upper band of free sugars consumers were more likely to have decay than the lower band (70% compared with 52%) (*20*).
Numerous cross-sectional studies have shown a relationship between sugars consumption and levels of caries in the

primary and/or permanent dentitions in countries or areas throughout the world, including China (*56*), Denmark (57), Madagascar (*58, 59*), Saudi Arabia (*60*), Sweden (61, *62*), Thailand (*63*), and the United Kingdom (*64*).

When investigating the association between diet and the development of dental caries it is more appropriate to use a longitudinal design in which sugars consumption habits over time are related to changes in dental caries experience. Such studies have shown a significant relationship between caries development and sugars intake (*65–67*). In a comprehensive study of over 400 children in England aged 11–12 years, a small but significant relationship was found between intake of total sugars and caries increment over 2 years ($r = 0.2$) (*67*). The Michigan Study in the USA investigated the relationship between sugars intake and dental caries increment over 3 years in children initially aged 10–15 years (*66*). A weak relationship was found between the amount of dietary sugars and dental caries.

A review addressing whether the relationship between dietary sugars and caries activity is vanishing in countries where the availability of sugars is high and the use of fluoride is extensive concluded that data from longitudinal studies in modern societies that make use of prevention still show a relationship between sugars consumption and caries activity (*68*). Many older studies failed to show a relationship between sugars intake and development of dental caries because they were of poor methodological design, used unsuitable methods of dietary analysis and were of insufficient significance (*68*). Correlations between individuals' sugars consumption and dental caries increments may be weak because of the limited range of sugars intake in the study population. If all people within a population are exposed to the disease risk factor, the relationship between the risk factor and the disease will not be apparent (*69*).

Frequency and amount of sugars consumption. Several studies, including the above mentioned Vipeholm study in Sweden , indicate that caries experience markedly increases when frequency of sugars intake exceeds four times a day (*50, 70–72*). The importance of frequency versus the total amount of sugars is difficult to evaluate as the two variables are hard to distinguish from each other. Data from animal studies have indicated the importance of frequency of sugars intake in the development of dental caries (*73, 74*). Some human studies show that the frequency of sugars intake is an important etiological factor for caries development (*75*). Many studies have related the frequency of intake of sugars or sugars-rich food to caries but have not simultaneously investigated the relationship between amount of sugars consumed and dental caries, therefore no conclusion regarding the relative importance of these two variables can be drawn (*76–78*).

Animal studies have also shown a relationship between amount of sugars consumed and the development of dental caries (*79–82*). Several longitudinal studies in humans also show amount of sugars intake to be more important than frequency (*66, 67, 83, 84*), while Jamel et al. (*85*) found that both the frequency and the amount of sugar intake are important.

The undoubtedly strong correlation between the amount and frequency of sugars consumption has been demonstrated by several investigators in different countries (*67, 86–88*). Therefore, the evidence shows that in terms of caries development both variables are potentially important.

Relative cariogenicity of different sugars and food consistency. The relative acidogenicity of different monosaccharides and disaccharides has been investigated in plaque pH studies, which have shown that lactose is less acidogenic than other sugars (*89*). Animal studies have shown no clear evidence that, with the exception of lactose, the cariogenicity of monosaccharide and disaccharide differs. The above mentioned study in Turku, Finland, showed no difference in caries development between subjects on diets sweetened with sucrose compared with fructose (*51*). Invert sugar (50% fructose + 50% glucose) is less cariogenic than sucrose (*90*).

The adhesiveness or stickiness of a food is not necessarily related to either oral retention time or cariogenic potential. For example, consumption of sugar-containing drinks (i.e. non-sticky) is associated with increased risk of dental caries (*85, 88*).

Potential impact of sugars reduction on other dietary components. It is important to consider the potential impact of a reduction in free sugars on other components of the diet. Simple cross-sectional analysis of dietary data from populations has shown an inverse relationship between the intake of free sugars and the intake of fat (91), suggesting that reducing free sugars might lead to an increase in fat intake. There is, however, a growing body of evidence from studies over time that shows that changes in intake of fat and free sugars are not inversely

related, and that reductions in intake of fat are offset by increases in intakes of starch rather than free sugars (92, 93). Cole-Hamilton et al. (94) found that the intake of both fat and added sugars simultaneously decreased as fibre intake increased. Overall dietary goals that promote increased intake of wholegrain staple foods, fruits and vegetables and a reduced consumption of free sugars will not lead to an increased consumption of fat.

Influence of fluoride. Fluoride undoubtedly protects against dental caries (*95*). The inverse relationship between fluoride in drinking-water and dental caries is well established. Fluoride reduces caries in children by between 20% and 40%, but does not eliminate dental caries. Over 800 controlled trials of the effect of fluoride administration on dental caries have been conducted and show that fluoride is the most effective preventive agent against caries (*95*). Nevertheless, studies indicate that a relationship between sugars intake and caries still exists in the presence of adequate fluoride (*33, 71, 96, 97*). In two major longitudinal studies in children, the observed relationships between sugars intake and development of dental caries remained even after controlling for use of fluoride and oral hygiene practices (*66, 67*). Marthaler (*68*) reviewed the changes in the prevalence of dental caries and concluded that, even when preventive measures such as use of fluoride are employed, a relationship between sugars intake and caries still exists. He also stated that in industrialized countries where there is adequate exposure to fluoride, no further reduction in the prevalence and severity of dental caries will be achieved unless the intake of sugars is reduced. A recent systematic review that investigated the importance of sugars intake in caries etiology in populations exposed to fluoride concluded that: where there is adequate exposure to fluoride, sugars consumption is a moderate risk factor for caries in most people; sugars consumption is likely to be a more powerful indicator for risk of caries in persons who do not have regular exposure to fluoride; with widespread use of fluoride, restricting sugars consumption still has a role to play in the prevention of caries but this role is not as strong as it is without exposure to fluoride. Despite the indisputable preventive role of fluoride, there is no strong evidence of a clear relationship between oral cleanliness and levels of dental caries (*98–100*).

Excess ingestion of fluoride during enamel formation can lead to dental fluorosis. This condition is observed particularly in countries that have high levels of fluoride in water supplies (*95*).

Starches and dental caries

The heterogeneous nature of starch (i.e. degree of refinement, botanical origin, raw or cooked) should be considered when assessing its potential cariogenicity. Epidemiological studies have shown that starch is of low risk to dental caries. People who consume high-starch/low-sugars diets generally have low levels of caries, whereas people who consume low-starch/high-sugars diets have high levels of caries (*39, 48, 49, 51, 67, 101, 102*). In Norway and Japan the intake of starch increased during the Second World War yet the occurrence of caries was reduced. Several types of experiment have shown that raw starch is of low cariogenicity (*103–105*). Cooked starch is about one-third to one-half as cariogenic as sucrose (*106, 107*). Mixtures of starch and sucrose are, however, potentially more cariogenic than starch alone (*108*). Plaque pH studies, using an indwelling oral electrode, have shown starch-containing foods to reduce plaque pH below 5.5, but starches are less acidogenic than sucrose. Plaque pH studies measure acid production from a substrate rather than caries development, and take no account of the protective factors found in some starch-containing foods or the effect of foods on stimulation of salivary flow.

Glucose polymers and pre-biotics are increasingly being added to foods in industrialized countries. Evidence on the cariogenicity of these carbohydrates is sparse and comes from animal studies, plaque pH studies and studies in vitro which suggest that maltodextrins and glucose syrups are cariogenic (*108–110*). Plaque pH studies and experiments in vitro suggest that isomalto-oligosaccharides and gluco-oligosaccharides may be less acidogenic than sucrose (*112–114*). There is, however, evidence that fructo-oligosaccharides are as acidogenic as sucrose (*115, 116*).

Fruit and dental caries

When consumed as part of a mixed human diet there is little evidence to show fruit to be an important factor in the development of dental caries (*67, 117–119*). A number of plaque pH studies have found fruit to be acidogenic, (although less so than sucrose. (*120–122*)). Animal studies have shown that when fruit is consumed in very high frequencies (e.g. 17 times a day) it may induce caries (*123, 124*), but less so than sucrose. In the only epidemiological study in which an association between fruit consumption and DMFT was found (*125*), fruit intakes were very high (e.g. 8 apples or 3 bunches of grapes per day) and the higher DMFT in fruit farm workers compared with grain farm workers arose solely from differences in the numbers of missing teeth.

Dietary factors which protect against dental caries

Some dietary components protect against dental caries. The cariostatic nature of cheese has been demonstrated in several experimental studies (126, 127), human observational studies (67), and intervention studies (128). Cow's milk contains calcium, phosphorus and casein, all of which are thought to inhibit caries. Several studies have shown that the fall in plaque pH following milk consumption is negligible (129, 130). The cariostatic nature of milk has been demonstrated in animal studies (131, 132). Rugg-Gunn et al. (67) found an inverse relationship between the consumption of milk and caries increment in a study of adolescents in England. Wholegrain foods have protective properties; they require more mastication thereby stimulating increased saliva flow. Other foods that are good gustatory and/or mechanical stimulants to salivary flow are peanuts, hard cheeses and chewing gum. Both organic and inorganic phosphates (found in unrefined plant foods) have been found to be cariostatic in animal studies, but studies in humans have produced inconclusive results (133, 134). Both animal studies and experimental investigations in humans show that black tea extract increases plaque fluoride concentration and reduces the cariogenicity of a sugars-rich diet (135, 136).

Breastfeeding and dental caries

In line with the positive health effects of breastfeeding, epidemiological studies have associated breastfeeding with low levels of dental caries (*137, 138*). A few specific case studies have linked prolonged ad libitum and nocturnal breastfeeding to early childhood caries. Breastfeeding has the advantage that it does not necessitate the use of a feeder bottle, which has been associated with early childhood caries. A breastfed infant will also receive milk of a controlled composition to which additional free sugars may not be added. There are no benefits to dental health of feeding using a formula feed.

Dental erosion

Dental erosion is the progressive irreversible loss of dental hard tissue that is chemically etched away from the tooth surface by extrinsic and/or intrinsic acids by a process that does not involve bacteria. Extrinsic dietary acids include citric acid, phosphoric acid, ascorbic acid, malic acid, tartaric acid and carbonic acids found, for example, in fruits and fruit juices, soft drinks and vinegar. Erosion in severe cases leads to total tooth destruction (*139*). Human observational studies have shown an association between dental erosion and the consumption of a number of acidic foods and drinks, including frequent consumption of fruit juice, soft drinks (including sports drinks), pickles (containing vinegar), citrus fruits and berries (*140–144*). Age-related increases in dental erosion have been shown to be greater in those with the highest intake of soft drinks (*20*). Experimental clinical studies have shown that consumption of, or rinsing with, acidic beverages significantly lowers the pH of the oral fluids (*121*). Enamel is softened within one hour of exposure to cola but this may be reversed by exposure to milk or cheese (*145, 146*). Animal studies have shown that fruit and soft drinks caused erosion (*124, 147*), although fruit juices are significantly more destructive than whole fruit (*148, 149*).

5.6.4 *Strength of evidence*

The strength of the evidence linking dietary sugars to the risk for dental caries is in the multiciplicity of the studies rather than the power of any individual study. Strong evidence is provided by the intervention studies (*50, 51*) but the weakness of these studies is that they were conducted in the pre-fluoride era. More recent studies show that an association between sugars intake and dental caries still exists but it is not as strong as in the pre-fluoride era. However, in many developing countries people have not yet been exposed to fluoride.

Cross-sectional studies should be interpreted with caution because dental caries develop over time and therefore simultaneous measurements of disease levels and diet may not give a true reflection of the role of diet in the development of the disease. It is the diet several years earlier that may be responsible for current caries levels. Longitudinal studies (*66, 67*) that have monitored a change in caries and related this to diet factors provide stronger evidence. Such studies have been conducted on populations with an overall high sugars intake but a low inter-individual variation; this may account for the weak associations that have been reported.

The studies that overcome the problem of low variation in consumption of sugars are studies that have monitored dental caries following a marked change in diet, for example those conducted on populations during the Second World War and studies of populations before and following the introduction of sugars into the diet. These have shown clearly that changes in dental caries mirror changes in economic growth and increased consumption of free

sugars. Sometimes changes in sugars consumption were accompanied by an increase in other refined carbohydrates. There are, however, examples where sugars consumption decreased and starch consumption increased yet levels of dental caries declined.

Strong evidence of the relationship between sugar availability and dental caries levels comes from worldwide ecological studies (*26, 28*). The limitations of these studies are that they use data on sugar availability and not actual intake, they do not measure frequency of sugars intake, and they assume that level of intake is equal throughout the population. Also, the values are for sucrose, yet many countries obtain a considerable amount of their total sugars from other sugars. These studies have only considered DMFT of 12-year-olds, not always from a representative sample of the population.

Caution needs to be applied when extrapolating the results of animal studies to humans because of differences in tooth morphology, plaque bacterial ecology, salivary flow and composition, and the form in which the diet is provided (usually powdered form in animal experiments). Nonetheless, animal studies have enabled the effect on caries of defined types, frequencies and amounts of carbohydrates to be studied.

Plaque pH studies measure plaque acid production, but the acidogenicity of a foodstuff cannot be taken as a direct measurement of its cariogenic potential. Plaque pH studies take no account of protective factors in foods, salivary flow and the effects of other components of the diet. Many of the plaque pH studies that show falls in pH below the critical pH of 5.5 with fruits and cooked starchy foods have been conducted using the indwelling electrode technique. This electrode is recognised as being hypersensitive and non-discriminating, tending to give an “all or nothing” response to all carbohydrates (*150)*.

Research has consistently shown that when annual sugar consumption exceeds 15 kg per person per year (40 g per person per day) dental caries increase with increasing sugar intake. When sugar consumption is below 10 kg per person per year (around 27 g per person per day), levels of dental caries are very low (*26, 28, 29, 51, 151–158*). Exposure to fluoride (i.e. where the proportion of fluoride in drinking-water is 0.7–1.0 ppm, or where over 90% of toothpastes available contain fluoride) increases the safe level of sugars consumption.

Tables 13–16 summarize the evidence relating to diet, nutrition and dental diseases.

Table 13

Summary of the strength of the evidence linking diet to dental caries

Evidence	Decreased caries	No relationship	Increased caries
Convincing	Fluoride exposure (local and systematic)	Starch intake (cooked and raw starch foods, such as rice, potatoes and bread; excludes cakes, biscuits and snacks with added sugars)	Amount of free sugars Frequency of free sugars
Probable	Hard cheese Sugars-free chewing gum	Whole fresh fruit	
Possible	Xylitol Milk Dietary fibre		Undernutrition
Insufficient	Whole fresh fruit		Dried fruits

Table 14

Summary of the strength of the evidence linking diet to dental erosion

Evidence	Decreased risk of erosion	No relationship	Increased risk of erosion
Convincing			
Probable			Soft drinks and fruit juices
Possible	Hard cheese		
	Fluoride		
Insufficient			Whole fresh fruit

Table 15

Summary of the strengths and weaknesses of the evidence linking diet to enamel developmental defects

Evidence	Decreased risk	No relationship	Increased risk
Convincing	Vitamin D		Excess fluoride
Probable			Hypocalcaemia

Table 16

Summary of the strength of the evidence linking diet to periodontal disease

Evidence	Decreased risk	*No relationship*	Increased risk
Convincing	Good oral hygiene		Deficiency of vitamin C
Probable			
Possible			Undernutrition
Insufficient	Antioxidant nutrients	Vitamin E supplementation	Sucrose

5.6.5 *Disease-specific recommendations*

It is important that there is a recommended maximum level for consumption of free sugars because when free sugars consumption by a population is low, dental caries levels are low. Population goals enable the oral health risks of populations to be assessed and health promotion goals monitored.

The best available evidence indicates that the level of dental caries is low in countries where the consumption of free sugars is below 15–20 kg per person per year. This is equivalent to a daily intake of 40–55 g and the values equate to 6–10% of energy intake. It is of particular importance that countries which currently have low consumption of free sugars (<15–20 kg per person per year) do not increase consumption levels. For countries with high consumption levels it is recommended that national health authorities and decision makers formulate country-specific and community-specific goals for reduction in the amount of free sugars, aiming towards the recommended maximum of no more than 10% of energy intake.

In addition to population targets given in terms of amount of free sugars, targets for frequency of free sugars consumption are also important. The frequency of consumption of foods and/or drinks containing free sugars should be limited to a maximum of four times per day.

Many countries that are currently undergoing nutrition transition do not have adequate exposure to fluoride. There should be promotion of adequate fluoride exposure via appropriate vehicles, for example affordable toothpaste, water, salt and milk. It is the responsibility of national health authorities to ensure implementation of feasible fluoride programmes for their country. Research into the outcome of alternative community fluoride programmes should be encouraged.

In order to minimize the occurrence of dental erosion, the amount and frequency of intake of soft drinks and juices should be limited. Elimination of undernutrition prevents enamel hypoplasia and the other potential effects of undernutrition on oral health (e.g. salivary gland atrophy, periodontal disease, oral infectious diseases).

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5.7 Recommendations for preventing osteoporosis

5.7.1 *Background*

Osteoporosis is a disease affecting many millions of people around the world. It is characterized by low bone mass and micro-architectural deterioration of bone tissue, leading to bone fragility and a consequent increase in risk of fracture (*1, 2*)

The incidence of vertebral and hip fractures increases exponentially with advancing age (while that of wrist fractures levels off after the age of 60 years) (*3*). Osteoporosis fractures are a major cause of morbidity and disability in older people and, in the case of hip fractures, can lead to premature death. Such fractures impose a considerable economic burden on health services worldwide (*4*).

5.7.2 *Trends*

Worldwide variation in the incidence and prevalence of osteoporosis is difficult to determine because of problems with definition and diagnosis. The most useful way of comparing osteoporosis prevalence between populations is to use fracture rates in older people. However, because osteoporosis is usually not life-threatening, quantitative data from developing countries are scarce. Despite this, the current consensus is that approximately 1.66 million hip fractures occur each year worldwide, that the incidence is set to increase fourfold by 2050 because of the increasing numbers of older people, and that the age-adjusted incidence rates are many times higher in affluent developed countries than in sub-Saharan Africa and Asia (*5–7*).

In countries with a high fracture incidence, rates are greater amongst women (by 3–4 fold). Thus, although widely regarded in these countries as a disease that affects women, 20% of symptomatic spine fractures and 30% of hip fractures occur in men (*8*). In countries where fracture rates are low, men and women are more equally affected (*7, 9–11*). The incidence of vertebral and hip fractures in both sexes increases exponentially with age. Hip-fracture rates are highest in Caucasian women living in temperate climates, are somewhat lower in women from Mediterranean and Asian countries, and are lowest in women in Africa (*9, 10, 12*). Countries in economic transition, such as Hong Kong Special Administrative Region (SAR) of China, have seen significant increases in age-adjusted fracture rates in recent decades, while the rates in industrialized countries appear largely to have reached a plateau (*13, 14*).

5.7.3 *Diet and disease*

Diet appears to have only a moderate relationship to osteoporosis, but calcium and vitamin D are both important, at least in older populations. Calcium is one of the main bone-forming minerals and an appropriate supply to bone is essential at all stages of life. In estimating calcium requirements, most committees have used either a factorial approach, where calculations of skeletal accretion and turnover rates are combined with typical values for calcium absorption and excretion, or a variety of methods based on experimentally-derived balance data (*15, 16*). There has been considerable debate about whether current recommended intakes are adequate to maximize peak bone mass and to minimize bone loss and fracture risk in later life, and the controversies continue (*2, 12, 15–17*).

Vitamin D is obtained either from the diet or by synthesis in the skin under the action of sunlight. Overt vitamin D deficiency causes rickets in children and osteomalacia in adults, conditions where the ratio of mineral to osteoid in bone is reduced. Poor vitamin D status in the elderly, at plasma levels of 25-hydroxyvitamin D above those associated with osteomalacia, has been linked to age-related bone loss and osteoporotic fracture, where the ratio of mineral to osteoid remains normal.

Many other nutrients and dietary factors may be important for long-term bone health and the prevention of osteoporosis. Among the essential nutrients, plausible hypotheses for involvement with skeletal health, based on biochemical and metabolic evidence, can be made for zinc, copper, manganese, boron, vitamin A, vitamin C, vitamin K, the B vitamins, potassium and sodium (*15*). Evidence from physiological and clinical studies is largely lacking, and the data are often difficult to interpret because of potential size-confounding or bone remodelling transient effects.

5.7.4 ***Strength of evidence***

For older people, there is convincing evidence for a reduction in risk in association with sufficient intake of vitamin D and calcium together; and an increase in risk with high consumption of alcohol and low body weight. Evidence suggesting a probable relationship, again in older people, supports a role for calcium and vitamin D separately, but none with fluoride.

Strength of evidence with fracture as outcome

There is considerable geographical variation in the incidence of fractures, and cultural variation in the intakes of nutrients associated with osteoporosis and the clinical outcome of fracture. In Table 17 it is important to note that the level of certainty is based on fracture as the outcome, rather than apparent bone mineral density as measured by dual-energy X-ray absorptiometry or other indirect methods. Since the Consultation addressed health in terms of burden of disease, fractures were considered the more relevant end-point.

Table 17

Summary of the strength of the evidence linking diet to osteoporotic fractures

	Decreased risk	**No relationship**	**Increased risk**
Convincing Older people[a]	Vitamin D Calcium Physical activity		High alcohol intake Low body weight
Probable Older people[a]		Fluoride[b]	
Possible	Fruit and vegetables [c] Moderate alcohol intake Soy products	Phosphorus	High sodium intake Low protein intake (in older people) High Protein intake

[a]In populations with high fracture incidence only. Applies to men and women older than 50–60 years, with a low calcium intake and/or poor vitamin D status.
[b] At levels used to fluoridate water supplies. High fluoride intake causes fluorosis and may also alter bone matrix.
[c] Several components of fruits and vegetables are associated with a decreased risk at levels of intake within the normal range of consumption (e.g. alkalinity, vitamin K, phytoestrogens, potassium, magnesium, boron). Vitamin C deficiency (scurvy) results in osteopenic bone disease.

5.7.5 *Disease-specific recommendations*

In countries with a high fracture incidence, a minimum of 400–500 mg of calcium intake is important to prevent osteoporosis. When consumption of diary products is limited, other sources of calcium such as fish with edible bones, tortillas processed with lime, green vegetables high in calcium (broccoli, kale), legumes and by-products of legumes (e.g tofu) are alternative sources. The interaction between calcium intake and physical activity, sun exposure, and intake of other dietary components (vitamin D, vitamin K, sodium, protein) and protective phytonutrients (soy compounds), needs to be considered before recommending increased calcium intake in countries with low fracture incidence in order to be in line with recommendations for industrialized countries (*18*).

With regard to calcium intakes to prevent osteoporosis, this Consultation referred to the recommendations of the Joint FAO/WHO Expert Consultation on Vitamin and Mineral Requirements in Human Nutrition (*18*) which highlighted the calcium paradox. The paradox (that hip fracture rates are higher in developed countries where calcium intake is higher than in developing countries where calcium intake is lower) clearly calls for an explanation. Recently, the accumulated data indicate the possibility that the adverse effect of protein, in particular animal (but not vegetable) protein, might outweigh the positive effect of calcium intake on calcium balance.

The report of the Joint FAO/WHO Expert Consultation on Vitamin and Mineral Requirements in Human Nutrition made it clear that the recommendations for calcium intakes were based on long-term (90 days) calcium balance data for adults derived from Australia, Canada, the European Union, the United Kingdom and the United States, and were not necessarily applicable to all countries worldwide. The report also acknowledged that evidence was emerging strongly that the requirements for calcium might vary from culture to culture for dietary, genetic, lifestyle and geographical reasons. Therefore, two sets of allowances were recommended: one for countries with low consumption of animal protein, and another based on data from North America and Western Europe.

The following conclusions were reached:

1. There is no case for global, population-based approaches. A case can be made for targeted approaches with respect to calcium and vitamin D in high-risk subgroups of populations with a high fracture incidence.

2. In countries with high osteoporotic fracture incidence, a low calcium intake (below 400–500 mg per day) (*15*) among older men and women is associated with increased fracture risk.

3. In countries with high fracture incidence, increases in dietary vitamin D and calcium in the older populations can decrease fracture risk. Therefore, an adequate vitamin D status should be ensured. If predominantly from dietary sources, for example when sunshine exposure is limited, an intake of 5–10 mg per day is recommended.

4. Although firm evidence is lacking, prudent dietary and some lifestyle recommendations developed in respect of other chronic diseases may prove helpful in terms of fracture risk. These include:

   increase physical activity;
   reduce sodium intake;
   increase consumption of fruit and vegetables;
   maintain a healthy body weight;
   avoid smoking;
   limit alcohol intake.

5. Convincing evidence indicates that physical activity, particularly activity that maintains or increases muscle strength, coordination and balance as important determinants of propensity for falling, is beneficial in prevention of osteoporotic fractures. In addition, regular lifetime weight-bearing activities, especially in modes that include impacts on bones and are done in vigorous fashion, increase peak bone mass in youth and help to maintain bone mass in later life.

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6. Strategic directions and recommendations for policy and research

6.1 Introduction

The principal goal of public health policy is to give people the best chance to enjoy many years of healthy and active life. Public health action to prevent the adverse consequences of inappropriate dietary patterns and physical inactivity is now urgently needed. To this end, the Consultation discussed how nutrient/food intake and physical activity goals could be used by policy-makers to increase the proportion of people who make healthier choices about food and undertake sufficient physical activity to maintain appropriate body weights and adequate health status. This section discusses ways to catalyse the long-term changes that are needed to place people in a better position to make healthy choices about diet and physical activity. Such processes require long-term changes in thinking and action at the individual and societal levels; demand concerted action by national governments, international bodies, civil society and private entities: will need insights and energies contributed by multiple sectors of society.

New scientific information will be essential to permit adjustment both of the policy levers and of the strategic processes to introduce change. This constitutes an important focus for applied research that should yield useful evidence to guide effective interventions.

Three key elements need to be analysed. The first is the range of possible policy principles that would help people achieve and maintain healthy dietary and activity patterns in a simple and rewarding manner. The second is the prerequisites for possible strategies to introduce these policies in different settings. These include the need for leadership, for effective communication of problems and possible solutions, for functioning alliances, and for ways of encouraging enabling environments to facilitate change. The third is the possible strategic actions to promote healthy diets and physical activity.

6.2 Policy principles for the promotion of healthy diets and physical activity[1,2]

The Consultation recommended that different parties consider the following policy principles when developing national strategies to reduce the burden of chronic diseases that are related to diet and physical inactivity.

- Strategies should be *comprehensive* in addressing all major dietary and physical activity risks for chronic diseases together, alongside other risks—such as tobacco use—from a multisectoral perspective.

- Each country should select what will constitute the *optimal mix of actions* that are in accord with national capabilities, laws and economic realities.

- *Governments have a central steering role* in developing strategies, ensuring that actions are implemented and monitoring their impact over the longterm.

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1 During the preparation of this report, by resolution WHA 55.23 (*1*) in May 2002, the World Health Assembly called upon the Director-General to develop a global strategy on diet, physical activity and health (WHA 55.23). The process for developing the WHO global strategy will involve formal consultation with Member States, United Nations agencies, civil society, and the private sector over a period of a year, prior to drafting a proposed global strategy for presentation to the Fifty-seventh World Health Assembly in 2004.

2 Ensuring that people have access to adequate food which is safe and at the same time of appropriate nutritional quality is also important. One of the commitments adopted by the *World Food Summit* convened by FAO in 1996 and reiterated in 2002 at the *World Food Summit: Five Years Later* specifically endorses the implementation of policies aimed at "improving access by all, at all times to sufficient, nutritionally adequate and safe food".

- *The Ministries of Health have a crucial convening role*—bringing together other ministries needed for effective policy design and implementation.

- *Governments need to work together with* the private sector, health professional bodies, consumer groups, academics, the research community, and other nongovernmental bodies if sustained progress is to occur.

- *A life-course perspective* on chronic disease prevention and control is critical. This starts with maternal and child health, nutrition and care practices; it includes school and workplace environments, access to preventive health and primary care, as well as community-based care for the elderly and disabled people.

- Strategies should explicitly address equality and diminish disparities; they should focus on the needs of the *poorest communities and population groups*—this requires a strong role for government. Further, since women generally make decisions about household nutrition, strategies should be *gender* sensitive.

- There are limits to what individual countries can do alone to promote optimal diets and healthy living. Strategies need to draw substantially on existing *international standards* that provide a reference in international trade. Member States may wish to see additional standards that address, for example, marketing of unhealthy food (particularly those high in energy, saturated fat, salt and sugar, and poor in essential nutrients) to children across national boundaries. Countries may also ensure means for ensuring the accessibility of healthier choices (such as fruit and vegetables) to all socioeconomic groups. WHO's international leadership role in pushing forward the agenda on diet, physical activity and health is crucial. FAO also has an important role in this process since it deals with issues on the production, trade, marketing of food and agricultural commodities and in providing guidelines ensuring the safety and nutritional adequacy of food and food products.

6.3 Prerequisites for effective strategies

Drawing on experience with the implementation of local and national strategies for public health matters in different settings, the Expert Consultation concluded that there are a number of prerequisites for success. These include leadership, effective communication, functioning alliances and an enabling environment.

6.3.1 *Leadership for effective action*

Leadership is essential in introducing long-term changes. Within nations, governments have the primary responsibility for providing this leadership. Yet, in some cases leadership may be initiated by civil society organizations prior to government action. There is unlikely to be just one correct path to improved health: each country will need to determine the optimal mix of policies that its particular circumstances fit best. Each country will need to select measures within the reality of its economic and social resources. Within a given country, effective action may call for regional strategies.

More proactive leadership is needed, worldwide, to portray a holistic vision of food and nutritional issues as they affect overall health. Where this leadership has existed, it has been possible to make governments take notice and introduce the necessary changes. The question remains of how to develop and strengthen leadership capacity to reach a critical mass. The WHO collaborating centres in nutrition and the FAO network of centres of excellence are possible routes, although there is a clear need to strengthen existing capabilities.

Governments throughout the world have developed strategies to eradicate malnutrition, traditionally used synonymously with undernutrition. However, the growing problems of nutritional imbalance, overweight and obesity, with their implications for the development of diabetes, cardiovascular problems and other diet-related noncommunicable diseases, are at least as pressing. This applies especially to developing countries undergoing the nutrition transition; such countries bear a double burden of both overnutrition, as well as undernutrition and infectious diseases. Unless there is political commitment to spur governments to achieve results, strategies cannot succeed. Setting population goals for nutrient intake and physical activity is necessary but insufficient. Giving people the best chance to enjoy many years of healthy and active life requires action at the community, family and individual levels.

6.3.2 *Effective communication*

Change can only be initiated through effective communication. The core role of health communication is to bridge the gap between technical experts, policymakers and the general public. The proof of effective communications is its capacity to create awareness, improve knowledge and induce long-term changes in individual and social behaviours, leading to consumption of healthy diets and incorporating physical activity for health. An effective health communication plan seeks to act on the opportunities at all stages of policy formulation and implementation, in order to positively influence public health. Sustained and well-targeted communication will enable consumers to be better informed and make healthier choices. Informed consumers are better able to influence policy-makers; this was learned from work to limit the damage to health from tobacco use. Consumers can serve as advocates or may go on to lobby and influence their societies to bring about changes in supply and access to goods and services that support physical activity and nutritional goals.

The cost to the world of the current and projected epidemic of chronic disease related to diet and physical inactivity dwarfs all other health costs. If society can be mobilized to recognize those costs, policy-makers will eventually start confronting the issue and themselves become advocates of change. Experience shows that politicians can also be influenced by the positions taken by the United Nations agencies, and the messages that they promote. Medical networks have also been found to be effective advocates of change in the presence of a government that is responsive to the health needs of society. Consumer nongovernmental organizations and a wide variety of civil society organizations will also be critical in raising consumer consciousness and supporting the climate for constructive collaboration with the food industry and the private sector.

6.3.3 *Functioning alliances and partnerships*

Change can be accelerated if all groups in favour establish alliances to reach the common objective. Ideally, the effort should include different parties whose actions influence people's options and choices about diet and physical activity. Alliances for action are likely to extend from communities to national and regional levels, involving formal focal points for nutrition within different public, private and voluntary bodies. The involvement of consumers associations is also important to facilitate health and nutrition education. International organizations with nutrition-related mandates, such as FAO and WHO, are expected to encourage the routing of reliable information through these networks. Alliances with other members of the United Nations family are also important—for example, with the United Nations Children's Fund on maternal–child nutrition and life-course approaches to health. Private sector industry with interests in food production, packaging, logistics, retailing and marketing, and other private entities concerned with lifestyles, sports, tourism, recreation and health and life insurance, have a key role to play. Sometimes it is best to work with groups of industries rather than with individual industries that may wish to capitalize on change for their own benefit. All should be invited; those who share the health promotion objective will usually opt to participate in joint activities.

6.3.4 *Enabling environments*

Individual change is more likely to be facilitated and sustained if the macro-environment and micro-environment within which choices are made supports options perceived to be both healthy and rewarding. Food systems, marketing patterns and personal choices should evolve in ways that make it easier for people to live healthier lives, and to choose the kinds of food that bring them the greatest health benefits. An enabling environment encompasses a wide frame of reference, from the environment at school, in the workplace, in the community, to transport policies, urban design policies, and the availability of a healthy diet. Further, it requires supportive legislative, regulatory and fiscal policies to be in place. Unless there is an enabling context, the potential for change will be minimal. The ideal is an environment that not only promotes but also supports and protects healthy living, making it possible, for example, to bicycle or walk to work or school, to buy fresh fruits and vegetables, and eat and work in smoke-free rooms.

The following actions to create enabling environments should be considered:

Supporting the availability and selection of nutrient-dense foods (fruits, vegetables, legumes, whole grains, lean meats and low-fat dairy products).
Within this overall concept, the issue of nutrient-dense foods versus energy-dense/nutrient-poor foods is important as it concerns the balance of providing essential nourishment while maintaining a healthy weight. The quality of the fat and carbohydrate supplied also plays a key role. The following are all important: increasing access—especially of low-income communities—to a supply of nutrient-dense fresh foods; food safety regulations that support this; facilitating access to high-quality diets through food pricing policies; nutrition labels to inform consumers, in particular the appropriate use of health claims. The provision of safe and nutritious food is now recognized not only as a human need but also as a basic right.

Assessing trends in changing consumption patterns and their implications for the food (agriculture, livestock, fisheries and horticulture) economy.
Recommendations, which result in changes in dietary patterns, will have implications for all components in the food economy. Hence it is appropriate to examine trends in consumption patterns world wide and deliberate on the potential of the food and agriculture sector to meet the demands and challenges posed by this Report. All sectors in the food chain, from farm to the table, will have to be involved if the food economy is to respond to the challenges posed by the need for changes in diets to cope with the burgeoning epidemic of NCDs.

Most of the information on food consumption has been hitherto obtained from national Food Balance data. In order to understand better the relationship between food consumption patterns, diets and the emergence of NCDs, it is crucial to obtain more reliable information on actual food consumption patterns and changing trends based on representative consumption surveys.

There is a need to monitor whether the guidelines developed in this Report, and strategies based on them, will influence the behaviour of consumers and to what extent consumers will change their diets (and lifestyles) towards more healthy patterns.

The next step will then be to assess the implications that these guidelines will have for agriculture, livestock, fisheries and horticulture and action taken to deal with potential future demands of an increasing and more affluent population. To meet the specified levels and patterns of consumption, new strategies may need to be developed. This assessment will need to include all stages in the food chain – from production, processing, marketing and consumption.

The implications that these changes in the food economy could have on the sustainability of natural resource use would also need to be taken into account.

Likewise, international trade issues would need to be considered in the context of improving diets. Trade has an important role to play in improving food and nutrition security. Considerations could include the impact of lower trade barriers on the purchasing power of consumers and variety of products available. On the export side, it could involve questions of market access, competitiveness and income opportunities for domestic farmers and processors.

The impact that agricultural policy, particularly subsidies, has on the structure of production, processing and marketing systems and, ultimately, on the availability of foods that support healthy food consumption patterns will need to be examined.

Finally, assessments of the above issues, and more, will certainly have policy implications at both the national and international levels. These implications would need to be taken up in the appropriate forum and considered by the stakeholders concerned.

Sustainable development

Increasing global consumption of intensively produced animal foods, as well as tobacco, is undermining health gains from reducing infectious diseases. For example, the low efficiency of food conversion and high water needs of cattle production poses a significant threat to the world's ability to feed the very poorest people, and also severely threatens many ecosystems. In contrast, increased production and consumption of a more diverse range of plant foods (fruit, vegetables and legumes), ideally closer to consumers' homes, can yield potent health and environmental gains. Agricultural policies often primarily respond to commercial farming concerns, for example farm subsidies for the production of dairy products and cattle, rather than being influenced by health considerations. There is also an apparent disregard for environmental sustainability. Unless this is tackled, there will be a conflict between meeting population nutrient intake goals, and existing structural barriers. An integrated strategy of introducing change, empowering the community and increasing the responsiveness of governments to health concerns is needed. The question of how the world's food supply can be managed so as to sustain the demands made by population-size adjustments in diet is a topic for continued dialogue with multiple partners that has major implications for world trade.

Physical activity

A large proportion of the world's population currently takes an inadequate amount of physical activity to sustain physical and mental health. The massive use of motor cars and other motorized means of saving physical labour contribute to the development of chronic diseases with adverse social, environmental and economic consequences. Cars are also likely contributors to growing urban problems such as traffic congestion and air pollution. Huge cities throughout the world have privileged space for motor vehicles but little space for recreation; major transport problems still remain, with the resulting cost in long travel times to and from work, limiting time for the purchase and preparation of food, and with progressive inequalities in access to the resources needed to support an active life. As occupational expenditure of energy diminishes, urban and workplace planners should ensure that transport and recreation policies promote, support and protect physical activity. For example, urban planning, transportation and building design should give priority to the safety and transit of pedestrians and safe bicycle use.

Traditional diets

Marketing practices commonly displace local or ethnic dietary patterns. Global marketing in particular has wide-ranging effects on both consumer appetite for goods and perceptions of their value. While some traditional diets could benefit from thoughtful modification, research has shown that many are particularly protective of health, and clearly environmentally sustainable. Much can be learned from these.

6.4 Strategic actions for promoting healthy diets and physical activity

The strategies themselves need to reflect local and national realities as well as global determinants of diet and physical activity. They must be based on the evidence on the ways in which people's dietary and physical activity patterns have positive or adverse effects on health. In practice, strategies are likely to include at least some of the following practical actions.

6.4.1 *Surveillance of people's diets, physical activity and related disease burden*

The surveillance system of diet, physical activity and related health problems are essential to enable all interested stakeholders to track progress towards each country's diet-related health targets, and to guide the choice, intensity and timing of measures to accelerate achievement. The data trends required for implementing effective policies need to be specific for age, sex and social group, and indicate changing trends over time.

6.4.2 *Enabling people to make informed choices and take effective action*

Information about fat quality, salt and sugar content, and energy density should be incorporated into nutrition and health promotion messages, and on universal food labelling tailored to different population groups—including disadvantaged population groups—through the wide reach of modern media. The ultimate goal of information and communication strategies is to assure availability and support choice of better quality food, access to physical activity and a better-informed global community.

6.4.3 *Making the best use of standards and legislation*

The Codex Alimentarius—the intergovernmental standard-setting body through which nations agree on standards for food—is currently being reviewed. Its work in the area of nutrition and labelling could be further strengthened to cover diet-related aspects of health. The feasibility of codes of practice in food advertising should also be explored.

6.4.4 *Ensuring that "healthy diet" components are available to all*

As consumers increase their preference for healthy diets, producers and suppliers will wish to orient their products and marketing to respond to this emerging demand. Governments could make it easier for consumers to exercise healthier choices, in accordance with the population nutrient intake recommendations in this report by, for example, promoting the wider availability of nonhydrogenated fat spreads, less processed foods high in trans-fatty acids, the use of vegetable oil for domestic consumers, and an adequate and sustainable supply of fish, fruits, vegetables and nuts in domestic markets.

Consumers consuming meals outside their home should be able to find out nutritional quality in a simple manner so that they can select and consume healthier choices. For example, consumers should be able to ascertain not only the amount of fat or oil in products, but also whether they are high in saturated fat or trans-fatty acids.

6.4.5 *Achieving success through intersectoral initiatives*

Approaches to promoting healthy diets call for comprehensive strategies that cut across many sectors and involve the different groups within countries concerned with food, nutrition, agriculture, education, transport and other relevant policies. They should involve alliances that encourage the effective implementation of national and local strategies for healthy diets and physical activity. They should encourage the adequate production and domestic supply of fruits, vegetables and wholegrain cereals, at affordable prices to all segments of the population, and opportunities for all to access them regularly, and to undertake appropriate levels of physical activity.

6.4.6 *Making the best of health services and the professionals who provide them*

The training of all health professionals (including physicians, nurses, dentists, and nutritionists) should include diet, nutrition and physical activity as key determinants of medical and dental health. The social, economic, cultural and psychological determinants of dietary and physical activity choice should be included as integral elements of public health action. There is an urgent need to develop and strengthen existing training programmes to implement these actions successfully.

6.5 Call to action

There is now a large, convincing body of evidence that dietary patterns and the level of physical activity can not only influence existing health levels, but also determine whether an individual will develop chronic diseases such as cancer, cardiovascular disease and diabetes. These chronic diseases remain the main causes of premature death and disability in industrialized countries and in most developing countries. Developing countries are demonstrably increasingly at risk, as are the poorer populations of industrialized countries.

In communities, districts and countries where widespread, integrated interventions have been implemented, dramatic decreases in risk factors have occurred. Successes have come about where the public has acknowledged that the unnecessary premature deaths that occur in their community are largely preventable and have empowered themselves and their civic representatives to create health-supporting environments. This has been achieved most successfully: by establishing a working relationship between communities and governments; through enabling legislation and local initiatives affecting schools and the workplace; by involving consumers' associations; and by involving food producers and the food-processing industry.

There is also a need for data collection on current and changing trends in food consumption in developing countries including research on what influences people's eating behaviour and physical activity and what can be done to address this, as well as the need, on a continuing basis, to develop strategies to change people's behaviour towards adopting healthy diets and lifestyles, including research on the supply and demand side related to this changing consumer behaviour.

Beyond the rhetoric, this epidemic can be halted—the demand for action must come from those affected. The solution is in our hands.

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Acknowledgements

Special acknowledgement was made by the Consultation to the following individuals, who were instrumental in the preparation and proceedings of the meeting: Dr C. Nishida, Department of Nutrition for Health and Development, WHO, Geneva; Dr P. Puska, Director, Department of Noncommunicable Disease Prevention and Health Promotion, WHO, Geneva; Dr P. Shetty, Chief, Food and Nutrition Division, Rome, Italy; and Dr R. Weisel, Food and Nutrition Division, FAO, Rome, Italy.

The Consultation also expressed deep appreciation to the following individuals, for their contributions to the running of the meeting and finalizing the report: Professor S. Kumanyika, University of Pennsylvania, Philadelphia, PA, USA; Professor J. C. Seidell, Free University of Amsterdam, Amsterdam, the Netherlands; Dr R. Uauy, London School of Hygiene and Tropical Medicine, London, England and Institute of Nutrition of the University of Chile, Santiago, Chile; and Dr Mabel Yap-Deurenberg, Health Promotion Board, Singapore.

The Consultation also thanked the authors of the background papers for the Consultation: Dr N. Allen, University of Oxford, Oxford, England; Dr P. Bennett, National Institute of Diabetes and Digestive and Kidney Diseases, Phoenix, AZ, USA; Professor I. Caterson, University of Sydney, Sydney, Australia; Dr I. Darnton-Hill, Columbia University, New York, NY, USA; Professor P. James, International Obesity Task Force, London, England; Professor M. B. Katan, Wageningen University, Wageningen, Netherlands; Dr T. J. Key, University of Oxford, Oxford, England; Dr J. Lindströmn, National Public Health Institute, Helsinki, Finland; Dr A. Louheranta, National Public Health Institute, Helsinki, Finland; Professor J. Mann, University of Otago, Dunedin, New Zealand; Dr P. Moynihan, University of Newcastle, Newcastle upon Tyne, England; Dr P.E. Petersen, Noncommunicable Disease and Health Promotion, WHO, Geneva, Switzerland; Dr A. Prentice, Medical Research Council Human Nutrition Research, Cambridge, England; Professor K.S. Reddy, All India Institute of Medical Science, New Delhi, India; Dr A. Schatzkin, National Institutes of Health, Bethesda, MD, USA; Dr A.P. Simopoulos, The Centre for Genetics, Nutrition and Health, Washington, DC, USA; Ms E. Spencer, University of Oxford, Oxford, England; Dr N. Steyn, Medical Research Council, Tygerberg, South Africa; Professor B. Swinburn, Deakin University, Melbourne, Australia; Professor N. Temple, Athabasca University, Alberta, Canada; Ms R.Travis, University of Oxford, Oxford, England; Dr J.Tuomilehto, National Public Health Institute, Helsinki, Finland; Dr W. Willett, Harvard School of Public Health, Boston, MA, USA; and Professor P. Zimmet, International Diabetes Institute, Caulfield, Victoria, Australia.

The Consultation also recognized the valuable contributions made by the following individuals, who provided comments on the background documents: Mr G. Boedeker, Economic and Social Department, FAO, Rome, Italy; Professor G.A. Bray, Pennington Biomedical Research Center, Louisiana State University, Baton Rouge, LA, USA; Mr J. Bruinsma, Economic and Social Department, FAO, Rome, Italy;
Dr L.K. Cohen, National Institutes of Health, Bethesda, MD, USA; Professor A. Ferro-Luzzi, National Institute for Food and Nutrition Research, Rome, Italy; Dr R. Francis, Freeman Hospital, Newcastle upon Tyne, England; Dr Ghafoorunissa, Indian Council of Medical Research, New Delhi, India; Dr K. Hardwick, National Institutes of Health, Bethesda, MD, USA; Dr H. King, Department of Management of Noncommunicable Diseases, WHO, Geneva, Switzerland; Dr J. King, University of California, Davis, CA, USA; Dr L. N. Kolonel, University of Hawaii, Manoa, HI, USA; Professor N.S. Levitt, University of Cape Town, Cape Town, South Africa; Dr P. Lingstrom, University of Gothenburg, Gothenburg, Sweden; Professor A. McMichael, Australian National University, Canberra, Australia; Professor S. Moss, Oral Health Promotion Committee, New York, NY, USA; Professor K. O'Dea, Menzies School of Health Research, Alice Springs, Australia; Professor D. O'Mullane, University of Cork, Cork, Ireland; Dr P. Pietinen, National Public Health Institute, Helsinki, Finland; Dr J. Powles, University of Cambridge, Cambridge, England; Dr E. Riboli, International Agency for Research on Cancer, Lyon, France; Dr S. Rösnner, Huddinge University Hospital, Huddinge, Sweden; Professor A. Rugg-Gunn, University of Newcastle Dental School, Newcastle upon Tyne, England; Mr J. Schmidhuber, Economic and Social Department, FAO, Rome, Italy; Professor A. Sheiham, University College London Medical School, London, England; Professor S. Truswell, University of Sydney, Sydney, Australia; Dr S. Tsugane, National Cancer Center Research Institute East, Tsukiji, Tokyo, Japan; Dr A.R.P. Walker, South African Institute for Medical Research, Johannesburg, South Africa; Dr S. Watanabe, Tokyo University of Agriculture, Tokyo, Japan; Dr C. Yajnik, King Edward Memorial Hospital Research Centre, Mumbai, India; and Dr S. Yusaf, McMaster University, Hamilton, Ontario, Canada.

Acknowledgement was made by the Consultation to the following individuals, for their continual guaidance: Dr D. Yach, Executive Director, Noncommunicable Diseases and Mental Health, WHO, Geneva, Switzerland; Dr D. Nabarro, Executive Director, Sustainable Development and Healthy Environments, WHO, Geneva, Switzerland; Mr H. De Haen, Assistant Director-General, Economic and Social Department, FAO, Rome, Italy; Dr G.A. Clugston, Director, Department of Nutrition for Health and Development, WHO, Geneva, Switzerland; and Dr K. Tontisirin,

Director, Food and Nutrition Division, FAO, Rome, Italy.

The Consultation expressed special appreciation to Ms P. Robertson for her valuable contribution to the preparation and running of the meeting, to Mrs A. Haden, Ms C. Allsopp and Mrs A. Rowe for their editorial assistance, and to Mrs R. Imperial Laue, Ms S. Nalty, Ms T. Mutru, Mrs R. Bourne, Mrs A. Manus, Mrs A. Ryan-Röhrich and Ms C. Melin for their assistance in checking, typing and finalizing the manuscript.

Annex

Summary of the strength of evidence for obesity, type 2 diabetes, cardiovascular diseases (CVD), cancer, dental disease and osteoporosis[a]

	Obesity		Type 2 diabetes		CVD		Cancer		Dental disease		Osteoporosis	
Energy and fats												
High intake of energy-dense foods	C	↑↑										
Saturated fatty acids			P	↑	C	↑↑[b]						
Trans-fatty acids					C	↑↑						
Dietary cholesterol					P	↑						
Myristic and palmitic acid					C	↑↑						
Linoleic acid					C	↓↓						
Fish and fish oils (EPA and DHA)[c]					C	↓↓						
Plant sterols and stanols					P	↓						
"-Linolenic acid					P	↓						
Oleic acid					P	↓						
Stearic acid					P	NR						
Nuts (unsalted)					P	↓						
Carbohydrate												
High intake of non-starch polysaccharides (dietary fibre)	C	↓↓	P	↓	P	↓						
Free sugars, frequency and amount									C	↑↑[d]		
Sugar-free chewing gum									P	↓[d]		
Starch[e]									C	NR		
Wholegrain cereals					P	↓						
Vitamins												
Vitamin C deficiency									C	↑↑[f]		
Vitamin D									C	↓↓[g]	C	↓↓[h]
Vitamin E supplements					C	NR						
Folate					P	↓						
Minerals												
High sodium intake					C	↑↑						
Salt-preserved foods and salt							P	↑[i]				
Potassium					C	↓↓						
Calcium											C	↓↓[h]
Fluoride, local									C	↓↓[d]		
Fluoride, systemic									C	↓↓[d]	P	NR[h]
Fluoride, excess									C	↑↑[g]		
Hypocalcaemia									P	↑[g]		
Meat and fish												
Preserved meat							P	↑[j]				
Chinese-style salted fish							C	↑↑[k]				
Fruits (including berries) and vegetables												

	Obesity		Type 2 diabetes		CVD		Cancer		Dental disease		Osteoporosis	
Fruits (including berries) and vegetables	C	↓↓[l]	P	↓[l]	C	↓↓	P	↓[m]				
Whole fresh fruits									P	NR[d]		
Beverages, non-alcoholic												
Sugars-sweetened soft drinks and fruit juices	P	↑							P	↑[n]		
Very hot (thermally) drinks (and food)							P	↑[o]				
Unfiltered boiled coffee					P	↑						
Beverages, alcoholic												
High alcohol intake					C	↑↑[p]	C	↑↑[q]			C	↑↑[h]
Low to moderate alcohol intake					C	↓↓[r]						
Other food-borne												
Aflatoxins							C	↑↑[s]				
Weight and physical activity												
Abdominal obesity			C	↑↑								
Overweight and obesity			C	↑↑	C	↑↑	C	↑↑[t]				
Voluntary weight loss in overweight and obese people			C	↓↓								
Low body weight											C	↑↑[h]
Physical activity, regular	C	↓↓	C	↓↓	C	↓↓	C	↓↓[u]			C	↓↓[h]
							P	↓[v]				
Physical inactivity/sedentary lifestyle	C	↑↑	C	↑↑								
Other factors												
Exclusive breastfeeding	P	↓										
Maternal diabetes			C	↑↑								
Intrauterine growth retardation			P	↑								
Good oral hygiene/absence of plaque									C	↓↓[f]		
Hard cheese									P	↓[d]		
Environmental variables												
Home and school environments that support healthy food choices for children	P	↓										
Heavy marketing of energy-dense foods, and fast food outlets	P	↑										
Adverse social and economic conditions	P	↑										

[a] Only convincing (C) and probable (P) evidence is included in this summary table. The symbols used are as follows:

↑↑: convincing increasing risk; ↑: probable increasing risk;
↓↓: convincing decreasing risk; ↓: probable decreasing risk;
NR: no relationship.

[b] Symbol also shown for myristic and palmitic acid.

[C] EPA = eicosappentaenoic acid;
DHA = docosahexaenoic acid.

[d] For dental caries.
[e] Includes cooked and raw starch foods, such as rice, potatoes and bread. Excludes cakes, biscuits and snacks with added sugar.
[f] For periodontal disease.
[g] For enamel developmental defects.
[h] In populations with high fracture incidence only; applies to men and women more than 50–60 years old.
[i] For stomach cancer
[j] For colorectal cancer.
[k] For nasopharyngeal cancer.
[l] Based on the contributions of fruits and vegetables to non-starch polysaccharides.
[m] For cancer of the oral cavity, oesophagus, stomach and colorectum.
[n] For dental erosion.
[o] For cancer of the oral cavity, pharynx and oesophagus.
[p] For stroke.
[q] For cancer of the oral cavity, pharynx, larynx, oesophagus, liver and breast.
[r] For coronary heart disease.
[s] For liver cancer.
[t] For cancer of the oesophagus, colorectum, breast (in postmenopausal women), endometrium and kidney.
[u] For colorectal cancer.
[v] For breast cancer.

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