Diet, nutrition and the prevention of type 2 diabetes

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Abstract

Objectives: The overall objective of this study was to evaluate and provide evidence and recommendations on current published literature about diet and lifestyle in the prevention of type 2 diabetes.

Design: Epidemiological and experimental studies, focusing on nutritional intervention in the prevention of type 2 diabetes are used to make disease-specific recommendations. Long-term cohort studies are given the most weight as to strength of evidence available.

Setting and subjects: Numerous clinical trials and cohort studies in low, middle and high income countries are evaluated regarding recommendations for dietary prevention of type 2 diabetes. These include, among others, the Finnish Diabetes Prevention Study, US Diabetes Prevention Program, Da Qing Study; Pima Indian Study; Iowa Women's Health Study; and the study of the US Male Physicians.

Results: There is convincing evidence for a decreased risk of diabetes in adults who are physically active and maintain a normal body mass index (BMI) throughout adulthood, and in overweight adults with impaired glucose tolerance who lose weight voluntarily. An increased risk for developing type 2 diabetes is associated with overweight and obesity; abdominal obesity; physical inactivity; and maternal diabetes. It is probable that a high intake of saturated fats and intrauterine growth retardation also contribute to an increased risk, while non-starch polysaccharides are likely to be associated with a decreased risk. From existing evidence it is also possible that omega-3 fatty acids, low glycaemic index foods and exclusive breastfeeding may play a protective role, and that total fat intake and *trans* fatty acids may contribute to the risk. However, insufficient evidence is currently available to provide convincing proof.

Conclusions: Based on the strength of available evidence regarding diet and lifestyle in the prevention of type 2 diabetes, it is recommended that a normal weight status in the lower BMI range (BMI 21–23) and regular physical activity be maintained throughout adulthood; abdominal obesity be prevented; and saturated fat intake be less than 7% of the total energy intake.

Type 2 diabetes, formerly known as non-insulin-dependent diabetes (NIDDM), accounts for most cases of diabetes mellitus worldwide. It is estimated that in 2000 there were approximately 150 million individuals with the disease and that this number is likely to double by 2025[1]. Type 2 diabetes is the fourth or fifth leading cause of death in most developed countries and there is growing evidence that it has reached epidemic proportions in many developing and newly industrialised countries[2]. The lowest rates of type 2 diabetes are found in rural communities where people retain traditional lifestyles[2].

Dramatic changes in the prevalence or incidence of type 2 diabetes have been observed in communities where there have been major changes in the type of diet consumed, from a traditional indigenous diet to a typical 'Western' diet, e.g. Pima Indians in Arizona, Micronesians in Nauru and Aborigines in Australia[3−5]. Changing disease rates are almost certainly explained by changes in several dietary factors as well as by changes in other lifestyle related factors, notably a reduction in physical activity.

Classification and diagnosis

As undiagnosed diabetes represents an important fraction of the population with the disease, most epidemiological studies are undertaken by testing all subjects in the population of interest. Without systematic testing an incomplete and potentially misleading picture of the frequency or distribution of the disease is obtained.

Furthermore, because of the differences in criteria, comparisons of rates between recent and earlier studies should be made with caution.

The 1985 internationally accepted World Health Organisation (WHO) criteria for the diagnosis and classification of diabetes led to much greater uniformity of methods used in epidemiological studies[6]. Recently, the diagnostic criteria have been revised, first by the American Diabetes Association[7] and then by the WHO[8], thus complicating comparisons of studies that are analysed according to the new criteria and those with the earlier WHO criteria.

The revised diagnostic criteria accord greater importance to the fasting plasma glucose (FPG) concentration as a criterion for diagnosis. The FPG value considered diagnostic of diabetes has been lowered to ≥ 126 mg/dL (≥ 7.0 mmol/L)[7,8] from the former value of 140 mg/dL (7.8 mmol/L) and over. The category of impaired glucose tolerance (IGT) was retained at 2-hr post-load glucose levels from 140 mg/dL (7.8 mmol/L) to less than 200 mg/dL (11.1 mmol/L). An additional category, impaired fasting glycaemia (IFG), was introduced to categorise individuals who have FPG levels that are above normal but fall short of the new diagnostic FPG level for diabetes, i.e. FPG 110 mg/dL (6.1 mmol/L) to 125 mg/dL (<7.0 mmol/L). It is now apparent that only a minority of individuals with IGT have IFG, and conversely, only a minority of those with IFG have IGT[9,10].

The change in diagnostic criteria has resulted in some individuals being reclassified as having diabetes, i.e. individuals with FPG from 126 mg to 139 mg/dL and with post-load 2-hr glucose values of <200 mg/dL, thereby resulting in an increase in prevalence[9]. Furthermore, some recent papers have reported the prevalence of diabetes based solely on fasting glucose values, thereby ignoring individuals with abnormal 2-hr values based on oral glucose tolerance testing. Such a strategy leads to rates that are appreciably lower than in studies where glucose tolerance tests are done.

Epidemiology

Prevalence and incidence of type 2 diabetes

In recent decades, dramatic increases in the prevalence and incidence of type 2 diabetes have occurred in many parts of the world especially in the newly industrialised and developing countries[11] (Fig. 1). Indeed, the majority of cases of type 2 diabetes in the future will occur in developing countries with India and China having more cases than any other country in the world[1].

In the United States, the most complete information on the prevalence of type 2 diabetes has been obtained from the US National Health Examination Surveys[12,13]. Surveys carried out in adults aged 20–74 years in representative samples of the US population show that the prevalence differs considerably in different ethnic groups[13,14] (Fig. 1).
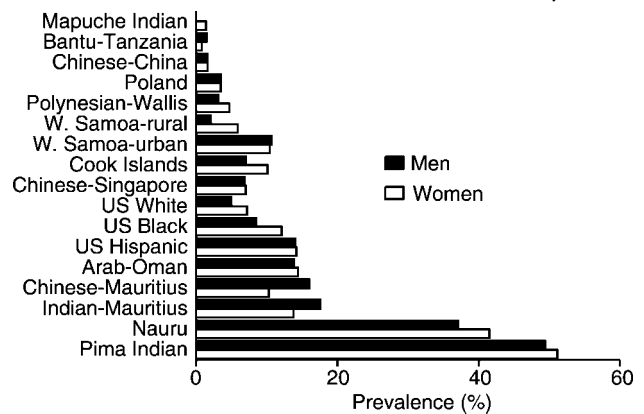
Fig. 1 Prevalence of diabetes according to 1980 WHO criteria in men and women aged 30–64 years in different countries and various ethnic groups (adapted from King and Rewers[11])

The prevalence in Hispanic Americans, particularly Mexican Americans, is higher than in the white or black populations and African Americans have greater prevalence than whites[15]. Native American populations have prevalence rates of type 2 diabetes which are even higher than those of Hispanic and African Americans, although the prevalence does vary from one native American group to another[16,17].

Data from the health examination surveys (NHANES III) conducted between 1988 and 1994 showed that 5.1% of US adults aged 20 years and older had previously diagnosed diabetes[12]. The prevalence of undiagnosed diabetes, based only on FPG levels of ≥ 126 mg/dL, was 2.7%, whereas with glucose tolerance tests and using the 1985 WHO criteria was 6.3%[9]. The total prevalence was 7.8% based on diagnosed diabetes and FPG levels only, or 11.4% when based on the 1985 WHO criteria, rates appreciably higher than seen in earlier surveys. Among persons aged 40–74 years the prevalence (based on the fasting criteria) had increased from 8.9% in 1976–1980 to 12.3% in 1980–1994—a 38% increase over the course of a decade[12]. Data from the Behavioural Risk Factor Surveys, carried out on representative samples of US adults, 18 years and older, indicated an increase of diagnosed diabetes between 1991 and 1999 from 4.1 to 6.0% in men, and from 5.6 to 7.6% in women, an increase of approximately 40% in less than a decade[18,19].

Turning to the situation in developing populations, in African Sub-Saharan countries, formerly, the disease either was absent, or was very low in occurrence. Even as late as 1987, in a rural village in Togo, West Africa, none of a large series of subjects examined had diabetes[20]. In South Africa, formerly, the disease was rare in rural areas[21], moreover, even in urban centres the proportion affected was low, 1.1%[22]. However, in strong contrast, in present day city populations, as in Durban in 1993, the crude incidence rate in Africans is actually higher than that in the local white population, namely 6.7 versus 4.5%[23].

Increases in type 2 diabetes have been observed in many other populations in the past half-century. For example, among the Pima Indians a 40% increase in the prevalence occurred between 1967 and 1977, primarily due to an increase in the incidence (the rate of development in new cases) of the disease[17].

Incidence studies using standardised glucose tolerance tests have been performed in the Pima Indians of Arizona[24,25] and among Micronesians in the central Pacific island of Nauru[26], populations with very high incidence rates. Among Pima Indians, the age-specific incidence and aged-adjusted incidence rates of diabetes have increased over the course of two decades[25,27] whereas in Nauru, the incidence may now be falling.

Mortality

Type 2 diabetes is associated with excess mortality mainly attributable to the vascular complications of the disease. In Caucasian populations, much of the excess is attributable to cardiovascular disease, especially ischaemic heart disease[28-30], but in others such as Asian and American Indian populations renal disease contributes to a considerable extent[31,32]. In some developing nations, an important component of the excess is due to infections[33].

Age-adjusted mortality rates among persons with diabetes are 1.5–2.5 times higher than in the general population[28,30] but the excess is greater in younger age groups and diminishes at older ages. The excess mortality leads to a decreased life expectancy among those with type 2 diabetes[32,34]. The extent of the reduction of life expectancy is dependent on the age of onset of the disease, but averages approximately 10 years in countries such as the United States. Mortality in women is generally lower than in men, but the relative risk of death in women with diabetes is increased so that the absolute rates approach those seen in men with type 2 diabetes[35,36].

Increased mortality in patients with type 2 diabetes is seen mainly among those with complications. Risk factors include proteinuria and retinal disease, and the classical risk factors for heart disease. Hyperlipidaemia, hypertension and smoking each contribute disproportionately to death rates among those with type 2 diabetes[37]. Mortality rates also increase with increasing duration of the disease[30,32].

Metabolic changes during development of type 2 diabetes: insulin resistance and impaired glucose homeostasis

Resistance to the action of insulin is the underlying abnormality in most people who develop type 2 diabetes[38,39] (Fig. 2). Insulin resistance results from an interaction between genetic and environmental factors. The genetic factors are not clearly understood and it is also uncertain whether some environmental factors are more important than others. A wide range of lifestyle related
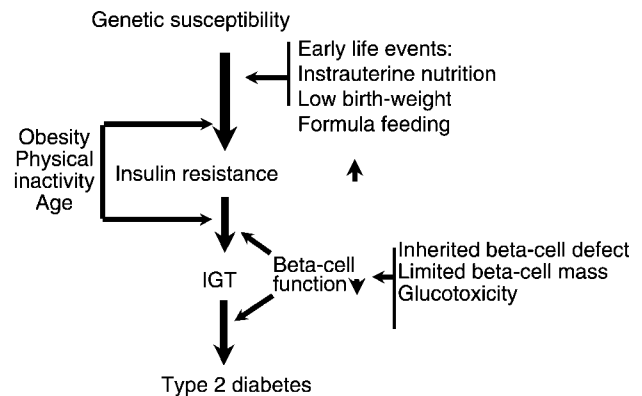
Fig. 2 Stages and risk factors in the development of type 2 diabetes

factors has been implicated, ranging from early life events to physical inactivity, several dietary attributes and subsequent development of overweight and obesity. These factors may be associated both with the development of insulin resistance as well as with progression from insulin resistance to states of impaired glucose metabolism (IFG, IGT) and eventually type 2 diabetes. The initial reaction of the β cells is to increase output of insulin in order to overcome the insulin resistance and so to maintain normal blood glucose levels.

Fasting insulin concentrations in subjects with normal glucose tolerance represent a surrogate marker of insulin resistance and numerous longitudinal studies have shown that higher fasting insulin levels (or other indices of insulin resistance) predict the development of type 2 diabetes. Unless insulin resistance is reversed, hypersecretion of insulin is usually insufficient to maintain normoglycaemia indefinitely and progression to IGT and IFG follows[39-42]. These early abnormalities of glucose metabolism usually, progress further to type 2 diabetes[43,44]. While raised levels of insulin are invariably associated with insulin resistance, the insulin secretion declines at a variable rate as the disease progresses through IGT to type 2 diabetes. This occurs either because of an inherent defect of the β cell or because of glucotoxicity, whereby the β cell is damaged as blood glucose levels rise[45]. The extent to which this sequence of events can be halted or reversed is considered in Section 8.

IGT or type 2 diabetes often coexist with other clinical and metabolic abnormalities which are associated with insulin resistance and central obesity. Hypertension, raised levels of triglyceride, reduced levels of high density lipoprotein, hyperuricaemia and raised levels of plasminogen activator inhibitor-1 are some of the constellation of features which along with hyperglycaemia of varying severity constitute the 'syndrome' now known as the metabolic syndrome or insulin resistance syndrome[46-50]. These features all contribute to the increased risk of cardiovascular disease associated with IGT and type 2 diabetes and may coexist together with hyperinsulinaemia before abnormalities of blood glucose are detectable.

Irreversible risk factors

The following section deals with important risk factors for the development of type 2 diabetes, which are based on inherent genetic or developmental factors, which are not modifiable by dietary and/or other lifestyle changes.

Race/ethnicity

The prevalence of type 2 varies considerably among populations of different ethnic origins living in apparently similar environments[11]. For example, in Singapore the frequency of diabetes in 1992 was 8.5–7.7% in Chinese men and women aged 18–69 compared with 13.3 and 12.3%, respectively, among the Asian Indians and Malays[51]. High prevalence rates of diabetes have also been found among Asian Indians compared with the indigenous populations in the United Kingdom, Fiji, South Africa and in the Caribbean[52–54]. Considerable differences in the prevalence of diabetes have also been described among the multi-ethnic populations of Hawaii and New Zealand, where the Native Hawaiians and Maori populations, both of Polynesian origin, have higher prevalences than other ethnic groups[55,56]. While environmental factors undoubtedly account for some of these differences, they are likely also to reflect inherent ethnic differences in susceptibility to the disease.

Familial aggregation

The empirical risk of having type 2 diabetes is increased 2 to 6-fold if a parent or sibling has the disease[57]. Consequently, a positive family history is a practical, albeit a crude way, of estimating if an individual is likely to have inherited susceptibility to the disease. On the other hand, familial aggregation may occur for non-genetic reasons. Family members often share a similar environment, particularly as children and in adolescence, thus familial aggregation alone is not definitive evidence of genetic determinants. Furthermore, with a disease as frequent as type 2 diabetes two or more family members may well have the disease by chance alone.

Genetic factors

A higher degree of concordance for type 2 diabetes in identical twins than in dizygotic twins provides strong evidence that genetic factors are important in determining susceptibility[58,59]. However, the fact that not all monozygotic twins are concordant for the disease confirms the importance of environmental factors.

Further evidence of the importance of genetic factors as predisposing factors for type 2 diabetes comes from studies of admixed populations. Differences in prevalence among persons of mixed racial background from that in parent populations with notably different prevalence of the disease are indicative of the importance of genetic determinants. Such relationships have been described among Nauruans and Pima Indians where full-heritage members of these groups have significantly higher rates of diabetes than those of mixed heritage[60,61]. Similarly, among the Mexican American population of San Antonio, the prevalence of type 2 diabetes is related to the degree of American Indian admixture, with higher rates associated with greater proportions of American Indian genes[62].

Much research activity has centred on attempts to unravel the genes, which confer susceptibility to type 2 diabetes, a number of genes are likely to be involved. At present, it is impossible to quantify the relative contributions of genetic and environmental factors.

Age and gender

The prevalence and incidence of type 2 diabetes vary to some extent between the sexes from one population to another, but these differences are relatively small and appear to be accounted for by differences in other risk factors such as obesity and physical activity.

The prevalence of type 2 diabetes increases with age although the patterns of incidence vary considerably. In high incidence populations, the prevalence may increase markedly in the younger adult years (e.g. 20–35 years of age), whereas in others the incidence and prevalence increase mainly in older individuals (e.g. 55–74 years of age). In most populations, a decrease in prevalence is seen in the oldest age groups (e.g. 75+ years) because of higher mortality rates in those with the disease.

Type 2 diabetes in relatively affluent societies usually develops in the middle to older age groups. In developing countries, however, because of the younger age distribution of the population, many cases occur in young and middle aged adults. In Caucasian populations in the United States and Europe, the prevalence of type 2 diabetes increases with age at least into the seventies[63].

Type 2 diabetes was formerly considered as a disease of adults. In recent years, however, there have been many reports of its occurrence in childhood and adolescence[64–66]. As in adults, the disease in children is frequently asymptomatic and is detected mainly by screening. In Japan, a national programme for screening school children has been in place since 1992 and the numbers recognised to have type 2 diabetes have increased progressively so that the prevalence and incidence of type 2 diabetes greatly exceed those of type 1 diabetes[67]. In American Indian children and adolescents, type 2 diabetes was first described among the Pima Indians[68] and the prevalence has increased steadily over the past 30 years[64]. Reports of type 2 diabetes in this age group have appeared from many ethnic groups in recent years including other Native American tribes, Mexican Americans, African Americans, Chinese, Polynesians, Asian Indians and Arabs from the Gulf States. It appears that among Caucasians, type 2 diabetes is still seen relatively infrequently in children and young adults.

Modifiable risk factors

This section comprises a review on risk factors related to dietary and other lifestyle factors. These factors have been shown to have an increased or a decreased risk for the development of type 2 diabetes and can be modified by lifestyle changes.

Obesity

Obesity is a frequent concomitant of type 2 diabetes, and in many longitudinal studies has been shown to be a powerful predictor of its development[25,69]. Obesity has increased rapidly in many populations in recent years[70-72] because of an interaction between genetic and environmental factors. These include: metabolic characteristics[73-75]; physical inactivity[74,76-78]; habitual energy intake in relation to expenditure[76]; and macronutrient composition of the diet[78-84].

This increase in obesity has been accompanied by an increasing prevalence of type 2 diabetes[19]. Since obesity is such a strong predictor of diabetes incidence, it appears that the rapid increases in the prevalence of type 2 diabetes seen in many populations in recent decades are almost certainly related to increasing obesity. Data from the Nurses' Health Study suggest that the lowest risk of diabetes occurs in individuals who have a body mass index (BMI) <21, with increasing prevalence seen as obesity levels increase[69].

Only a limited number of studies have measured age and sex specific incidence rates for type 2 diabetes in relation to obesity. Such rates vary considerably according to other risk factors. There are large differences in age-specific incidence rates according to BMI in the Pima Indians[25]. Those with higher BMI have much higher incidence rates of type 2 diabetes at earlier ages than those with lower BMI among whom the incidence rises in the older age groups. In non-obese individuals, the incidence of type 2 diabetes is low even in populations such as the Pima Indians where the overall risk of the disease is very high. The relationship of incidence of type 2 diabetes to obesity also varies with other risk factors. For example, in the Pima Indians the incidence rises much more steeply with BMI in those whose parents have diabetes than in these who do not. This relationship indicates an interaction between risk factors.

Several studies indicate that waist circumference or waist-to-hip ratio may be a better indicator of the risk of developing diabetes than BMI[85-87]. Such data suggest that the distribution of body fat is an important determinant of risk as these measures reflect abdominal or visceral obesity. In Japanese American men, for example, the intra-abdominal fat, as measured from CAT scans, was the best anthropometric predictor of diabetes incidence[86].

Given the importance of central adiposity as a determinant of diabetes risk it is necessary to consider whether the usually quoted 'normal range' for BMI $(18.5-24.9 \, kg/m^2)$ is appropriate for all populations. It might be appropriate to also suggest an appropriate range for some measure of the distribution of body fat (e.g. waist circumference, waist/hip ratio). The Nurses Health Study suggests that for populations of European descent risk of type 2 diabetes increases even within the normal BMI range and that a BMI of $21 \, kg/m^2$ might be an optimum level[69]. However, for a given BMI, several (perhaps all) populations of Asian descent appear to have an appreciably greater proportion of body fat than that of Europeans. It seems conceivable, therefore, that a lower BMI might be desirable[88]. In the absence of definitive data from prospective studies in these countries, at present it may be appropriate to similarly suggest an optimum level towards the lower end of the normal range. On the other hand, people of Pacific descent (Polynesians) have a relatively high proportion of lean body mass compared with Europeans for any given BMI. Therefore, a higher BMI cut off may be acceptable. However, the particularly high risk of type 2 diabetes and other co-morbidities of obesity in these populations may negate this apparently beneficial anthropometric attribute. Again, in the absence of appropriate prospective studies, it may be wise to suggest that their BMI should not exceed the conventional normal range. Because there are fewer data available concerning waist circumference or waist/hip ratio in different populations, it is appropriate to continue to use the WHO recommended BMI range $(18.5-24.9 \, kg/m^2)$ and population mean of $21 \, kg/m^2$ [88].

Physical inactivity

Numerous studies have indicated the importance of physical inactivity in the development of type 2 diabetes[89-92]. Indeed, in most studies its relative importance may be underestimated because of imprecision in measurement. In the Nurses' Health Study, women who reported exercising vigorously had an age-adjusted incidence rate of self-reported clinically diagnosed diabetes that was two-thirds as high as that of women who exercised less frequently[89]. The deleterious effect of low levels of physical activity is seen particularly among those subjects who have other risk factors such as high BMI, hypertension or parental diabetes. Similarly, among male physicians, the incidence of self reported diabetes was negatively related to the frequency of vigorous exercise and the strength of this relationship was greater in those with higher BMI[90]. For equivalent degrees of obesity, more physically active subjects have a lower incidence of the disease[92].

Recommendations with regard to physical activity as a preventative measure for developing type 2 diabetes are still difficult to quantify. Currently, guidelines propose moderate physical activity on at least 5 days per week and do not specify heart rate targets However, more recent evidence suggests that vigorous exercise is required to improve insulin sensitivity[93]. A study by McAuley et al.[93]

showed that insulin sensitivity improved in normoglycae-mic insulin-resistant adults who undertook vigorous exercise and not in those who complied with current moderate exercise programmes. The vigorous exercise programme required participants to train five times a week for at least 20 min per session at an intensity of 80–90% of age-predicted maximum heart rate[93].

Fat: quantity and quality

Both the amount and quality of dietary fat may modify glucose tolerance and insulin sensitivity[94–96]. A high fat content in the diet may result in deterioration of glucose tolerance by several mechanisms including decreased binding of insulin to its receptors, impaired glucose transport, reduced proportion of glycogen synthase and accumulation of stored triglycerides in skeletal muscle[97–101]. The fatty acid composition of the diet, in turn, affects tissue phospholipid composition, which may relate to insulin action by altering membrane fluidity and insulin signalling[94].

Amount of fat consumed
In experimental animals, all high-fat diets (with the exception of *n*-3 fatty acids) have been shown to result in insulin resistance relative to high carbohydrate diets[94,99,100,102,103]. The data from epidemiological and human intervention studies are less consistent. In two cross-sectional studies, total fat intake was higher in glucose intolerant and type 2 diabetic subjects[104] and in subjects with recurrent gestational diabetes mellitus[105] compared with normoglycaemic controls. Furthermore, a high fat intake has been shown to predict development of IGT in a group of healthy subjects[106] and progression from IGT to type 2 diabetes in a group of subjects with IGT[107]. High total fat intake has also been associated with higher fasting insulin concentrations[108,109] and a lower insulin sensitivity index[110]. On the other hand, there are several studies, which show no association between diabetes risk and total fat intake[111–119]. Few human intervention studies have examined the effects of high fat, low carbohydrate diets on diabetes risk, and the results have been inconsistent[120–127].

Nature of dietary fat
As previously mentioned, in animal experiments saturated, monounsaturated and polyunsaturated fats, excluding *n*-3 fatty acids, have caused insulin resistance when fed as high-fat diets[94,99,100,102,103]. In epidemiological studies, a high saturated fat intake has been associated with higher risk of IGT[106,128] and higher fasting glucose[129,130] and insulin levels[108,131]. Higher proportions of saturated fatty acids in serum lipids/muscle phospholipids have been associated with higher fasting insulin levels[132], lower insulin sensitivity[133] and higher risk of developing type 2 diabetes[134]. Higher vegetable fat (unsaturated fat) and PUFA intake have in turn been associated with a lower risk of type 2 diabetes[115,118,119], as well as lower fasting and

2-hr glucose concentrations[130,135]. Furthermore, higher proportions of long-chain polyunsaturated fatty acids in skeletal muscle phospholipids have been associated with better insulin sensitivity in humans[136,137]. With respect to monounsaturated fatty acids, the epidemiological data are inconsistent. Some studies indicate that a high intake of monounsaturated fatty acids may be detrimental in terms of increasing diabetes risk[106,138]. However, it should be noted that in a typical 'Western' diet the monounsaturated fatty acids are not derived from vegetable oils but to a large extent coexist with saturated fat in sources such as meat and milk products. Therefore, the detrimental effects reported[106,138] may be due to the effects of saturated fatty acids in these food sources[106].

In two short-term human intervention studies, replacement of a considerable portion of saturated fat by unsaturated fat improved glucose tolerance in young healthy women[139] and in middle-aged glucose-intolerant hyperlipidaemic subjects[140]. A longer-term study by Vessby *et al*.[141] confirmed that a substitution of mono-unsaturated fat for saturated fat significantly improved insulin sensitivity in healthy subjects after a 3-month dietary period. An interesting interaction between total fat intake and fatty acid composition of dietary fat was reported: the favourable effect of substituting monounsa-turated fat for saturated fat was lost in individuals consuming more than 37% of energy as fat[141]. There are only a few studies in which the effects of single fatty acids on glucose and insulin metabolism have been examined. In two short-term studies with single saturated fatty acids—lauric, palmitic and stearic acids—no effect on glucose and insulin metabolism was found when these saturated fatty acids were compared with an equivalent energy exchange with monoenes[142,143].

In the light of present knowledge regarding the relationships between type 2 diabetes and nature and quantity of dietary fat, as well as the absence of definitive data regarding precise percentage of fat to total energy, it seems reasonable to suggest that quantitative recommen-dations should follow those suggested for reduction of cardiovascular risk.

The role of trans fatty acids
Data on the effects of *trans* fatty acids on glucose metabolism are sparse. A report from the Nurses Health Study[118] suggests a positive association between *trans* fatty acid intake and risk of type 2 diabetes. In mouse islet cells *trans* fatty acids potentiate insulin secretion compared with *cis*-isomers[144]. Two human experiments have been reported. In patients with type 2 diabetes, a 6-week diet high in *trans* fatty acids (20% of energy) increased post-prandial c-peptide and insulin responses compared with a diet high in *cis*-monounsaturated fatty acids[145]. In healthy young women, consumption of a 4-week diet with 5% of energy from *trans* fatty acids did not change insulin sensitivity compared to an oleic acid enriched diet[146].

The role of *n*-3 fatty acids

In rodent studies, *n*-3 fatty acids ameliorate insulin resistance induced by high-fat feeding [95,100,103]. In some epidemiological studies, an inverse association between fish intake and risk of IGT has been observed [106,114]. Most intervention studies aimed at investigating the effect of fish oil on insulin sensitivity have been done in patients with type 2 diabetes and have been negative [147−151]. A negative result has, however, also been reported in healthy subjects [141].

Carbohydrates: quantity and quality

Total carbohydrates

As mentioned earlier, some controversy surrounds the optimal ratio of carbohydrate-to-fat in the diet with respect to the prevention of chronic diseases, including type 2 diabetes. The main question is whether high intakes of either carbohydrate or fat are deleterious over time and will predispose individuals to diabetes [152].

Due to a paucity of controlled trials on which to base recommendations for macronutrients we have to rely on the available evidence from epidemiological studies. Causal inferences thus need to be made with the recognition that the probability of confounding variables may exist in the different populations [73]. There are marked differences in various countries of the world with respect to the fat-to-carbohydrate ratios consumed by different populations [153]. A significant positive association has been shown between dietary fat consumption and the proportion of the population who are overweight. This supports the notion of a reduced fat (increased carbohydrate) intake as a preventative measure for chronic diseases [154]. There is some evidence that a high carbohydrate intake decreases the prevalence of diabetes [104,155]. However, numerous studies have reported that an increased intake of carbohydrates can reduce HDL levels and raise fasting plasma triacylglycerol concentrations [156−160]. These are mainly short-term, isocalortic, metabolic studies rather than long-term *ad lib* studies where weight is allowed to decrease on the lower fat content diets.

Two cohort studies [116,117−161] and a recent review [162] did not find an association between total carbohydrate and diabetes risk. It is known that a high carbohydrate intake increases the requirement for insulin secretion in order to maintain glucose homeostasis [163]. Insulin secretion by beta-cells is glucose sensitive and a high intake of carbohydrate in relation to energy intake, produces higher post-prandial insulin levels. It is possible that repeated stimulation of a high insulin output by a high carbohydrate diet could speed up an age-related decline in insulin secretion and lead to an earlier onset of type 2 diabetes [152,164]. The quality as well as the quantity of carbohydrate may hasten this response [152]. The most recent American dietary guidelines recommend intake of a variety of grain products (including whole grains) equating to six or more servings a day [165]. The FAO/WHO recommend that carbohydrate in the diet should comprise at least 55% of total energy intake in 'normal' healthy individuals [166]. There is, however, no specific carbohydrate guideline, which is aimed at the prevention of type 2 diabetes. Therefore, a wide range of carbohydrate intakes may be acceptable in terms of achieving a low risk of type 2 diabetes with type and source of carbohydrate being more important than quantity.

Dietary fibre and the glycaemic index

Dietary fibre in this context is composed of non-starch polysaccharides (NSPs), plus lignin, oligosaccharides and resistant starch. Resistant starch is that portion of starch that resists digestion, passes into the lower intestine, and ferments there. Dietary fibre found in foods consists of cellulose, hemicelluloses, pectins, hydrocolloids, resistant starches and resistant oligosaccharides [166]. Dietary fibre is one of the factors that influences post-prandial glucose and insulin responses. Other factors include the macronutrient composition of meals, processing, cooking and other characteristics of the carbohydrates [167−170]. Since there are considerable differences in the physiological responses to different forms of carbohydrate, the term glycaemic index (GI) was coined in 1981 [171]. It is defined as the glycaemic response elicited by a 50 g carbohydrate portion of a food expressed as a percentage of that elicited by a 50 g portion of a reference food (glucose or white bread). GI is a measure of the post-prandial glucose response after carbohydrate consumption. Low GI foods have lower 2-hr areas under the glucose curve than the reference food, while high GI foods have higher areas.

The effects of the various components of dietary fibre have been implicated in the prevention and management of a range of diseases, including type 2 diabetes, as early as the seventies [172,173]. Cross-sectional studies suggest that lack of dietary fibre may be a causative factor in type 2 diabetes and have shown an inverse relationship between fibre intake and blood insulin levels [110,174]; implying that fibre improves insulin sensitivity. Three large cohort studies, the Health Professionals Follow-up study carried out on men aged 40−75 [117], the Nurses Study carried out on women aged 40−65 [116], and the Iowa Women's Health Study carried out on women aged 55−69 years [161] have studied the effects of fibre and glycaemic load on risk of developing diabetes. All three studies clearly showed that a relatively low intake of dietary fibre significantly increased the risk of type 2 diabetes. The association was found to be strong for cereal fibre, a rich source of insoluble fibre, but much weaker for sources of soluble fibre [117−161,175,176]. The protective association in the extreme quintiles revealed a risk ratio of 0.64−0.72, after correcting for related variables such as age, BMI, smoking and physical activity. Two of the studies reported that glycaemic load is associated with risk of diabetes [116,117]. They showed an increased relative the risk of type 2

diabetes of 2.2 in women and 2.1 in men, with a combination of low cereal fibre intake and a high glycaemic load. However, the third cohort study did not detect a risk associated with GI[117]. Studies carried out in people with diabetes shed further light on the possible roles of dietary fibre and GI. Many studies have shown that glycaemic control is improved and the total LDL cholesterol reduced on relatively high carbohydrate, low fat diets including naturally occurring fibre-rich foods compared with relatively low carbohydrate, higher fat diets. A similar benefit has also been shown when comparing diets with similar carbohydrate:fat ratios, but with the experimental diet being appreciably higher in fibre. Feeding supplements of dietary fibre for several weeks has also been shown to lower both post-prandial glycaemia, insulin levels[177–179] and an overall improve-ment in glycaemic control as measured by HbA_1. This is seen both in normal subjects and in those with type 2 diabetes. Of considerable interest is the fact that in the dietary intervention studies, soluble forms of dietary fibre, regardless of whether it was taken as a supplement or in food, had an appreciably greater beneficial effect than was the case for insoluble, largely cereal derived forms of dietary fibre. Numerous studies have illustrated beneficial effects of a diet comprising foods with a low GI in type 2 diabetics[180–183]. Furthermore, some studies have found that foods with a high GI increase fasting triacylglycerol concentrations, even when the amount of carbohydrate is kept constant[184,185].

Recently, investigators have also considered the possibility that factors other than fibre that are contained in cereals and legumes may influence the risk for chronic diseases[186]. Some of these include micronutrients such as selenium and vitamin E, antioxidants, phytochemicals, isoflavins and lignans. Since many of these factors occur together in cereals it is difficult to determine the precise benefits of each. While the benefits of the 'whole' grains have been demonstrated to reduce risk of CHD in women in the Nurses Health Study[187] and in the Iowa Women's Health Study[188], the effects of whole grains on the reduction of type 2 diabetes risk remains to be explored.

In general dietary recommendations, the recommended fibre intake in terms of NSPs has been set at an average intake of 18 g (range 12–24 g) of NSP per day for adults in the UK[189] and at 25 g fibre in the USA[165]. Good sources of NSPs (>4 g per portion) are: legumes, pulses, brussels sprouts, whole wheat bread, rye bread, wholemeal pasta, bran cereals and wholegrain cereals[190]. However, the wide range of studies quoted here suggest that an appreciably larger quantity is required to reduce the risk of diabetes or improve glycaemic control in those with the disease. Since there is no clear indication regarding the precise quantity of dietary fibre to protect against diabetes, or which type of dietary fibre confers the greatest benefit, it may be most appropriate to emphasise appropriate carbohydrate sources rather than to specify precise quantities.

Micronutrients

Vitamin E

Studies in patients with type 1 diabetes have revealed an increased level of oxidative stress[191] and evidence that vitamin E may help prevent this[192]. However, little is known concerning the relationship between vitamin E intake and the development of type 2 diabetes.

Two cohort studies conducted in Finland examined the relationship between the blood level of vitamin E and the risk of type 2 diabetes[193,194]. One study reported that a low plasma level of this anti-oxidant vitamin is associated with a 3.9-fold elevated risk of developing the disease[193]. The association was independent of various possible con-founding factors. A nested case–control study carried out within a cohort study reported that subjects with a high serum vitamin E level had a 39% lower risk of diabetes compared to those with a low level of vitamin E[194]. However, in contrast to the previous study[193] this association disappeared when the risk ratio was adjusted for various coronary heart disease risk factors. This suggests that a high level of vitamin E may be merely a marker for a healthy lifestyle.

While the relationship between vitamin E and the risk of diabetes should be further investigated, there is insuffi-cient evidence that an increased intake of this nutrient will prevent the disease.

Magnesium

Three large American cohort studies have reported a strong negative association between intake of magnesium and risk of type 2 diabetes. This was seen in the Health Professionals Follow-up Study carried out on men aged 40–75[117], the Nurses Study carried out on women aged 40–65[116], and the Iowa Women's Health Study carried out on women aged 55–69[161]. In each case the protective association in the extreme quintiles revealed a risk ratio of about 0.7, after correcting for related variables such as age, BMI, smoking and physical activity. While the association was attenuated after adjusting for cereal fibre, an important source of magnesium, the protective association remained strong and statistically significant. In contrast to these reports, no association was seen between mag-nesium intake and risk of type 2 diabetes in the ARIC study, a cohort study on men and women aged 45–64[195]. However, this study was considerably smaller than the other three and, therefore, had much less statistical power. In the absence of any evidence regarding mechanisms of action, it seems inappropriate to offer recommendations regarding intake.

Chromium

The relationship between chromium and glucose metabolism has been under investigation since the late 1950s[196–198]. Anderson *et al.*[199] reported that subjects with mildly impaired IGT showed an improvement in

glucose tolerance and a lower level of blood insulin after receiving chromium supplementation. This was not seen in subjects with normal glucose tolerance. The subjects had been consuming a low intake of chromium. This study suggests that when the intake of chromium is low, some people develop glucose intolerance, which can be corrected by chromium supplementation. The lowering in blood insulin level indicates that the chromium improves tissue sensitivity to insulin (i.e. helped reverse insulin insensitivity). Comparable observations have been reported from studies in rats[200–202]. A study undertaken with adults who already have type 2 diabetes showed improved glycaemic control with chromium supplements, compared with placebo[203].

Alcohol intake

Several studies have suggested that moderate alcohol intake is associated with a reduced incidence of type 2 diabetes. Among women in the Nurses Health Study, there was a reduced incidence of diabetes in women who consumed alcohol compared with those who did not. There was a strong inverse relation between alcohol consumption and body weight, which could explain much of the apparent protective effect of alcohol consumption[204]. Among 20,000 male physicians, those consuming more than 2–4 drinks per week had a lower incidence of type 2 diabetes in the subsequent 12 years compared with non-drinkers, relationships that persisted after adjustment of BMI and other diabetes risk factors[205,206].

These apparent male–female differences were examined among 12,000 45–64 year old participants in the Atherosclerosis Risk in Communities Study (ARIC)[207]. After adjustment for other diabetes risk factors men consuming more than 21 drinks per week had a significant increase in the incidence of diabetes, whereas no significant association with alcohol intake was found among the women. The apparent inconsistencies in the results of these studies preclude clear recommendations regarding alcohol in the prevention of diabetes.

Intrauterine environment

There has been much recent interest in the extent to which intrauterine environment may influence the subsequent risk of developing diabetes and other diseases. Gestational diabetes, which is a strong risk factor for development of type 2 diabetes, is also considered here because of its association with overweight in pregnancy and possible intrauterine factors, which may play a role in the offspring.

Gestational diabetes

Gestational diabetes (diabetes first recognised during pregnancy) is more frequent among women from subgroups of the population who have a high risk of type 2 diabetes, e.g. older, overweight or obese women, certain ethnic groups. In some cases, gestational diabetes represents diabetes that was present, but undiagnosed before pregnancy, whereas in others it develops during pregnancy, most frequently towards the end of the second trimester. It is in this latter group, that following delivery glucose tolerance is likely to become normal, but such women carry a high risk for developing diabetes subsequently[208,209].

The intrauterine environment influences the risk of developing type 2 diabetes. Offspring of diabetic pregnancies are often large and heavy at birth; they tend to develop obesity in childhood and are at high risk of developing type 2 diabetes at an early age[210]. Such individuals have lower insulin secretion than similarly aged offspring of non-diabetic pregnancies[211].

A substantial part of the excess risk of diabetes in the offspring of diabetic pregnancies appears to be the result of exposure to the diabetic intrauterine environment. Among offspring born to mothers before and after the development of type 2 diabetes, those born after the mother developed diabetes, have a 3-fold higher risk of developing diabetes than those born before[212]. Thus, the enhanced risk among the offspring from diabetic pregnancies among such women appears to be the result of intrauterine programming that has long-term effects on the offspring in later life. The early appearance of type 2 diabetes in female offspring increases the likelihood that their offspring in turn will be exposed to a diabetic intrauterine environment, leading to an increased prevalence of diabetes in subsequent generations.

Intrauterine growth retardation and low birth weight

Evidence that type 2 diabetes may be programmed during foetal development comes from numerous studies on adults[213–221]. These showed an association between birth weight and abnormal glucose–insulin metabolism in later life. The prevalence of type 2 diabetes and IGT in adults was found to decrease from 40% by those who weighed <2.54 kg to 14% among those who weighed >4.31 kg at birth[213]. In Pima Indians, this relationship was found to be U-shaped, with a high prevalence of diabetes also found in those who had a birth weight >4.5 kg[216]. Additionally, the highest prevalence of type 2 diabetes was found in people who were small at birth and obese as adults[213,218]. Low birth weight is also associated with other traits that are associated with the development of diabetes including increased blood pressure, elevated triglycerides and lower HDL concentrations—all characteristic of the insulin resistance or metabolic syndrome[222].

Studies in many populations indicate that low-birth weight babies have an increased risk of developing type 2 diabetes in adult life[213,223–226]. This relationship was first described by Hales and Barker[227] who suggested that low birth weight due to nutritional deprivation *in utero* resulted in reduced beta-cell mass. They suggested that the

relationship might represent a 'thrifty phenotype'—an acquired rather than inherited defect—which was expressed as type 2 diabetes when those with the genotype were exposed to a more affluent nutritional environment[222].

It appears that foetal adaptations take place during times of undernutrition. Some organs and systems may be permanently altered or programmed to develop certain diseases later in life. Examples of this are the outcomes regarding adults who were *in utero* during the Dutch famine. They were about 200 g lighter at birth than those who were not exposed to the famine. As adults they show a reduced glucose tolerance and evidence of insulin resistance[228]. Birth weight serves as a marker for foetal nutrition and growth[229]. There is evidence to suggest that thin babies are more likely to develop type 2 diabetes[214], as are short fat babies[230].

It is important to appreciate that intrauterine growth retardation may not necessarily be due to deficiencies in maternal nutrition. A cohort study undertaken in Portsmouth on 693 nulliparous white women with singleton pregnancies showed that placental and infant birth weights were not associated with the intake of energy or any macronutrient intakes early or later in pregnancy[231]. The researchers suggest that among relatively well-nourished women in industrialised countries, maternal nutrition may only have a marginal effect on infant and placental size.

Animal studies have shown that a protein or energy deficient maternal diet can result in offspring having abnormal proportions at birth[221]. Two studies on the long-term effects of maternal diet in humans have indicated that the ratio of animal protein to carbohydrate may play a significant role in birth weight outcome[232,233].

Thus, while intrauterine growth retardation would seem to be well established as a risk factor for the later development of diabetes, the precise role of maternal diet is less well established.

Breastfeeding
Early feeding may also play a role in subsequent development of type 2 diabetes in later life[234–236]. Bennett *et al.*[234] found that the prevalence of diabetes was about 50% lower in Pima Indians who had been exclusively breastfed for the first 2 months of life. The mechanism for the protective effect of breastfeeding remains to be established and further confirmatory evidence is required.

Other risk factors

Several other risk factors have been related to the development of diabetes. These include several inflammatory markers (e.g. interleukin-6, C-reactive protein, other cytokines and acute phase reactants)[237–240] and variation in levels of sex hormones[241–244] (e.g. low levels of sex hormone binding globulin in women, low testosterone levels in men and women with high androgen levels).

Lifestyle modifications and risk reduction

Three recent studies have examined the potential of lifestyle intervention programmes in reducing progression of IGT to diabetes. Two randomised controlled trials have demonstrated that weight loss achieved by an increase in physical activity and dietary change including reduction in total and saturated fat and increased dietary fibre can reduce the incidence of diabetes[175,176]. Other studies, which did not involve individual randomisation, provide confirmatory evidence[245,246].

The aim of the Finnish Diabetes Prevention Study (FDPS)[175] was to assess the efficacy of an intensive diet-exercise programme on prevention of type 2 diabetes in 522 adults with IGT. Overweight subjects were randomised either to an intervention group or to a control group. The intervention group received individual counselling with respect to diet, weight loss and physical activity. There was a significant difference in weight loss between the two groups after 1 year (4.2 versus 0.8 kg) and after 2 years (3.5 versus 0.8 kg) of intervention. The cumulative incidence of diabetes was 11% in the intervention group and 23% in the control group. During the trial the risk of diabetes was reduced by 58% ($P < 0.001$) in the intervention group.

A similar intervention programme was undertaken in the United States on a larger sample of more than 3000 overweight adults with IGT[176]. Participants were representative of a variety of national groups resident in the United States. This study – the US Diabetes Prevention Program (DPP) – included an additional arm whereby some participants were randomised to the drug metformin. About 29% of the DPP control group developed diabetes during the follow-up of 3 years. By contrast 22% of the metformin arm and 14% of the diet and exercise group developed diabetes. Participants in the diet and exercise group achieved a 7% weight loss in the first year and generally sustained a 5% total loss for the duration of the study.

In both studies, the estimated risk reduction was about 58%. While lifestyle intervention studies such as the FDPS[154] and the DPP[176] have shown that quite modest changes can reduce the progression from IGT to diabetes by 50–60%, it has not been clear as to whether it will be possible to translate these findings to larger cohorts or to maintain these lifestyle changes in the longer term.

The Da Qing Study[245] was undertaken over a longer intervention period (6 years), than the FDPS and the DPP. The Da Qing Study differed from the FDPS and the DPP in that participants with IGT were randomised by intervention rather than as individuals, into a control group or into one of three interventions: diet only, exercise only, or diet and exercise, in order to test the effectiveness of diet and

exercise separately. The cumulative incidence of diabetes after 6 years was 67.7% in the control group, 43.8% in the diet group, 41.1% in the exercise group and 46% in the diet plus exercise group. All the groups differed significantly from the control group ($p < 0.05$). Proportional hazards analyses adjusted for differences in baseline BMI and fasting glucose, were associated with 31% reduction in risk of developing diabetes with diet alone; 46% reduction in risk with exercise alone and 42% reduction in risk with diet and exercise.

These three studies (among others) provide evidence that risk of developing type 2 diabetes can be reduced by changes in lifestyle by adults who are at high risk for the disease. Although the average amount of weight lost was relatively small, the difference between the incidence of diabetes in the intervention groups and the control groups was substantial. The latter finding emphasises the importance of even a small reduction of weight loss in conjunction with increase in physical activity in the prevention of diabetes. In addition, some intervention studies have demonstrated benefits in terms of several cardiovascular risk factors in addition to improved glucose tolerance[175,246,247].

While these trials are of enormous importance, it is nevertheless equally important to appreciate that even in the intensive intervention groups an appreciable proportion goes on to develop type 2 diabetes and that the number of individuals progressing to diabetes increases with time. It is not clear whether this is due to inability to sustain the necessary intensive lifestyle interventions or whether this is due to deteriorating β cell function. It is well established that the increased insulin secretion evident prior to the development of hyperglycaemia starts to reduce even during the phase of IGT and declines progressively as the disease process continues, regardless of treatment. Thus, the greatest hope of truly 'preventing' type 2 diabetes probably lies not in identifying those with IGT but rather in implementing lifestyle intervention programmes in populations at large (especially those at high risk of type 2) or in individuals at the stage of insulin resistance.

Recommendations and policy implications

The enormous economic, social and personal cost of type 2 diabetes make a compelling case for prevention. In recent years, there has been much new evidence demonstrating the potentially preventable nature of type 2 diabetes, particularly by the implementation of lifestyle measures such as weight control and exercise. In view of this and the devastating health impact of the disease it seems prudent that primary prevention should be a major priority. Tables 1 and 2 summarise the current evidence and recommendations made with respect to the development of type 2 diabetes.

Since the 1970s there have been numerous health promotion projects that have attempted to encourage people to lead a healthier lifestyle. These have been carried out variously in the community, in doctors' offices or at the worksite. While some have achieved success, more often their impact on the lifestyle of the intended target audience has been rather limited. It seems likely that even a modest change in diet and exercise habits can be of considerable importance when extrapolated to the population.

In order to achieve maximum benefit from lifestyle interventions changes in government policies and legislation will be needed in addition to individual and community-based programmes[248]. This would include mandating more nutrition education in schools, banning the advertising of unhealthy products and subsidising healthy foods at the expense of less appropriate foods.

Despite much research on nutritional factors in the aetiology of type 2 diabetes, the risk associated with several individual nutrients is not entirely clear. Thus, evidence-based advice to governments and public health authorities emphasises the role of weight reduction in the overweight and obese and an increase in physical activity. This approach can also be expected to have a positive effect for prevention of other major non-communicable diseases such as cardiovascular disease and hypertension.

Many developing countries now report the onset of type 2 diabetes at an increasingly young age. This trend towards younger age of onset implies a huge additional burden to the individuals and society and necessitates a lifetime approach to prevention.

In making recommendations to governments regarding the prevention of type 2 diabetes, priority should be given to the following:

1. *Promotion and evaluation* of 'healthy' lifestyle programmes, which focus on the following aspects:

    - Prevention and early treatment of overweight and obesity, particularly in high risk groups;
    - Consumption of a nutrient-dense diet, which is low in fat, particularly saturated fat, and free sugars and high in NSPs.
    - An active lifestyle, which includes regular physical activity of at least 1 hr/day, and vigorous activity, which is required to reduce the risk of developing type 2 diabetes.
    - Moderate alcohol intake and cessation of cigarette smoking.
    - Demonstration projects in developing and developed countries since it is recognised that there is a paucity of data on the efficacy of community-based lifestyle programmes.

Table 1 Summary of level 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†
Probable	NSPs*	–	Saturated fats Intrauterine growth retardation (IUGR)
Possible	*n*-3 Fatty acids Low glycaemic index foods Exclusive breastfeeding‡	–	Total fat intake *Trans* fatty acids
Insufficient	Vitamin E Chromium Magnesium Moderate alcohol	–	Excess alcohol

* NSP – Non-starch polysaccharide.
** Overweight: BMI $\geq$ 25 kg/m^2, obesity: BMI $\geq$ 30 kg/m^2.
*** Waist circumference: men $\geq$ 102 cm, women $\geq$ 88 cm.
† This includes gestational diabetes.
‡ 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.

Table 2 Summary of recommendations on diet and prevention of type 2 diabetes

Risk factors	Recommendations
1. Overweight/ obesity	Prevention/early treatment of overweight and obesity, particularly in high risk groups Avoid adult weight gain of $>$ 5 kg The optimum BMI for individuals is at the lower end of the normal range An optimum mean BMI for a population is in the range of 21–23 kg/m^2
2. Physical inactivity	Increase physical activity (moderate or greater level of intensity) to at least 1 hr every day for most days of the week Vigorous activity is required to reduce the risk of developing type 2 diabetes
3. Fat intake	Saturated fat intake should not exceed 7% of total energy intake in high risk groups Total fat intake should not exceed 30% of total energy intake
4. NSPs	Adequate intakes of NSPs can be achieved through whole grain cereals, legumes, vegetables and fruit
5. Further research required on diet and type 2 diabetes	Effect of maternal diet on infant birth weight and subsequent growth and development Long-term effects of early stunting Glycaemic index of different foods (and in combination) *n*-3 Fatty acids as protective factors in decreasing risk of type 2 diabetes *Trans* fatty acids as possible risk factors for developing type 2 diabetes Benefits of exclusive breastfeeding in reducing risk type 2 diabetes

NSP – Non-starch polysaccharide.

2. *Early identification of subjects at risk of developing type 2 diabetes*

- Identifying subjects at high risk of hypertension, diabetes and heart disease;
- Screening for gestational diabetes;
- Optimal maternal nutrition and weight maintenance.

3. *Healthy lifestyle programmes/interventions* should focus on a *life course* perspective and not on a specific age group or developmental stage.

4. *Legislative action* will be necessary to promote a healthier lifestyle for all populations. All governments and their employees in the social, health, nutritional, economic and welfare arenas should move forward on an integrated approach to the prevention of the disease and not only in treating the symptoms. There is compelling evidence for the benefits likely to accrue from policies and programmes aimed at reducing rates of overweight and obesity, at increasing physical activity and at reducing intake of saturated fatty acids. Individual countries, based on their national and individual food preferences, should develop dietary recommendations that are more specific in this regard.

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