

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 characterized by overproduction of insulin. As the disease progresses, 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 the established disease (1, 2).

Lifestyle modification is the cornerstone of both treatment and 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. 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 a role for lifestyle factors which can be modified to reduce the risk.

5.3.2 *Trends*

Although increases in both the prevalence and incidence of type 2 diabetes have occurred globally, they have been especially dramatic in societies in economic transition in much of the newly industrialized world and in developing countries (1, 6–9). Worldwide, the number of cases of diabetes is currently estimated to be around 150 million. This number is predicted to double by 2025, with 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 in younger and younger age groups, including adolescents and children, 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 mortality is attributable to cardiovascular disease, especially coronary heart disease (11, 12); amongst Asian and American Indian populations, renal disease is a major contributor (13, 14), whereas in some developing nations, infections are an important cause of death (15). It is conceivable that the decline in mortality due to coronary heart disease which has occurred in many affluent societies may be halted or even 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 ***Diet, physical activity and diabetes***

Type 2 diabetes results from an interaction between genetic and environmental factors. The rapidly changing incidence rates, however, suggest a particularly important role for the latter as well as a 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 the 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 NSP.

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 NSP 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 BMI, 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 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 three-fold 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% of 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 probable 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 randomized controlled trials which showed a 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 intake and type 2 diabetes is complicated by ambiguity with regard to the 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 in the diet (referred to as dietary fibre) associated with a high consumption of minimally-processed or unprocessed carbohydrate. The author also hypothesized that throughout the world, increasing intakes of highly-processed carbohydrate, depleted in NSP, had promoted the development of diabetes (46). Three cohort studies (the Health Professionals Follow-up Study of men aged 40–75 years, the Nurses' Health 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. 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). Moreover 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 fact that the experimental studies suggest that soluble forms of NSP exert benefit (50–53) whereas the prospective cohort studies suggest that it is the cereal-derived insoluble forms that are protective (47, 48) explain the “probable” rather “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.¹ Other carbohydrate-containing foods (e.g. certain types of pasta), which are 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 results of 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 number of studies, mostly in developing countries, have suggested 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 strategies which may help to reduce type 2 diabetes risk in this situation include improving the 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. The strength of evidence on lifestyle factors is summarized in Table 9.

¹ The glycaemic index is calculated as the 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.

Table 9

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	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

¹ NSP, non-starch polysaccharides.

^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 are likely to also 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:

- 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, this means maintaining a mean BMI in the range 21–23 kg/m² 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).
- Practising 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.
- 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.
- Achieving adequate intakes of NSP through regular consumption of wholegrain cereals, legumes, fruits and vegetables. A minimum daily intake of 20 g is recommended.

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