1: Epidemiology and prevention of type 2 diabetes and the metabolic syndrome

Jonathan E Shaw and Donald J Chisholm

National prevention programs are needed, but GPs can contribute through screening and lifestyle advice

ABSTRACT

- The prevalence of type 2 diabetes in Australia has doubled over the past 20 years; more than 7% of Australian adults now have diabetes.
- An additional 16% of Australian adults have lesser abnormalities of glucose tolerance.
- Insulin resistance and increased cardiovascular risk occur in both these groups (the metabolic syndrome).
- 50% of cases of type 2 diabetes are undiagnosed; screening is indicated in everyone aged over 55 and in younger people with risk factors such as obesity, hypertension, family history or certain ethnic backgrounds.
- Dietary modification and increased physical activity have been shown to dramatically reduce the incidence of type 2 diabetes in those at high risk.
- General practitioners should target individuals at high risk, but this needs to be reinforced by community-wide preventive action.

MJA 2003; 179: 379–383

THE DRAMATIC WORLDWIDE increase in the prevalence of type 2 diabetes is posing a massive health problem in both developed and developing countries.[1] Interestingly, in developed countries, lower socioeconomic groups are most affected, while, in developing countries, the reverse applies.[2] The magnitude of the healthcare problem of type 2 diabetes results not just from the disease itself but also from its association with obesity and cardiovascular risk factors, particularly dyslipidaemia and hypertension.[1] Indeed, type 2 diabetes has now been recognised as one manifestation of the "metabolic syndrome", a condition characterised by insulin resistance and associated with a range of cardiovascular risk factors.

In this article, we will discuss the epidemiology of type 2 diabetes and the metabolic syndrome, as well as screening, diagnosis and prevention.

Definitions

Type 2 diabetes is a complex metabolic disorder characterised by hyperglycaemia and associated with a relative deficiency of insulin secretion, along with a reduced response of target tissues to insulin (insulin resistance). Its metabolic and clinical features are heterogeneous; people with type 2 diabetes range from those of normal weight or underweight with a predominant deficiency of insulin secretion (in whom slowly evolving type 1 diabetes should be considered) to the more common obese person with substantial insulin resistance.

The genetic determinants of type 2 diabetes are still poorly defined, except in the few people with an early-onset, dominantly inherited form of the disorder (maturity-onset diabetes of the young), in whom specific genetic mutations have been identified (eg, of the glucokinase gene).[3]

The metabolic syndrome started as a concept rather than a diagnosis,[4] when it was realised that insulin resistance is associated with a variety of cardiovascular risk factors, including central adiposity, glucose intolerance, dyslipidaemia and hypertension. The World Health Organization subsequently provided a definition that can be used for individual diagnosis (Box 1).[5] As insulin resistance is difficult to determine in routine practice, diagnosis of the metabolic syndrome in people without diabetes requires demonstration of impaired glucose tolerance (IGT), together with two of the following: hypertension, obesity, dyslipidaemia (hypertriglyceridaemia or low level of high-density lipoprotein cholesterol), or microalbuminuria.

The increase in cardiovascular risk associated with IGT is not as great as that associated with diabetes itself, but nevertheless remains substantial.[1]

Epidemiology

It is calculated that worldwide there are now 150 million people with diabetes, and that this number will rise to 300 million by 2025.[1] In Australia, the AusDiab study reported in 2000 that 7.4% of the population aged 25 or over had diabetes (type 2 in 90%), and that about 50% were undiagnosed.[6] Prevalence increases progressively with age, so that more than 20% of the population aged over 60 have type 2 diabetes.[6] The prevalence of type 2 diabetes has more than doubled in Australia since 1981, and the total number of

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1: Definition of the metabolic syndrome[1]

Metabolic syndrome is defined by the World Health Organization as

At least one of	*Plus at least two of*
■ Type 2 diabetes	■ Hypertension (BP ⩾ 140/90 mmHg)
■ Impaired glucose tolerance	■ Obesity (BMI ⩾ 30 kg/m^2, or waist–hip ratio > 0.90 for men, > 0.85 for women)
■ Insulin resistance	■ Hypertriglyceridaemia (⩾ 1.7 mmol/L) or low serum HDL level (< 0.9 mmol/L for men, < 1.0 mmol/L for women)
	■ Microalbuminuria (albumin creatinine ratio > 2.5 mg/mmol for men, > 3.5 mg/mmol for women)

BP = blood pressure. BMI = body mass index. HDL = high-density lipoprotein.

cases has increased threefold.[6] As the prevalence of type 1 diabetes is low in Asian, Indian, Middle Eastern and African populations, type 2 diabetes would constitute well over 90% of diabetes cases worldwide.

Precise figures for the prevalence of the metabolic syndrome are not generally available, but, in Australia, the AusDiab study showed a 16% prevalence of impaired glucose metabolism (IGT or impaired fasting glucose [IFG]).[6] This suggests that, in a developed country, for every person with type 2 diabetes probably at least two more have the metabolic syndrome. Moreover, the AusDiab data, which are probably representative of most developed countries, demonstrate that various cardiovascular risk factors, including hypertension and dyslipidaemia, become progressively worse with progression from normal glucose tolerance to IGT/IFG to diabetes (Box 2). In addition, polycystic ovary syndrome, which is increasing in frequency with increasing adiposity in the community, is strongly associated with insulin resistance and glucose intolerance[7] (as will be discussed later in this series).

There are major ethnic differences in susceptibility to type 2 diabetes, which are probably largely genetically determined; people of Micronesian, Polynesian, Aboriginal and Torres Strait Islander, Indian or Chinese background are at substantially increased risk.[1]

While there is good evidence for a strong genetic contribution to both obesity and diabetes, the increase in these conditions in both developed and developing countries appears to be due to a changing balance between energy intake and energy expenditure through physical activity.[1] Much is written about the unhealthy Australian diet, but, although we may have substituted hamburgers and chips for our parents' lamb chops and sausages, the total calorie intake and macronutrient composition have changed little over the past 50 years. However, our physical activity levels have probably diminished by half![8]

The tendency for the increased prevalence of type 2 diabetes to be concentrated in lower socioeconomic groups in developed countries and higher socioeconomic groups in developing countries[2] probably reflects the adoption of a "healthier" lifestyle by better educated people in developed countries, while it is generally the affluent in developing countries who enjoy a high calorie intake and low level of physical activity.

Disorders and medications that increase risk

Some medical disorders can cause secondary diabetes (eg, pancreatitis and acromegaly). In addition, a number of medications increase the risk of type 2 diabetes,[3,5,9] the most important being glucocorticoids, which both enhance liver glucose output and increase insulin resistance. Other commonly used drugs that may impair glucose tolerance include thiazide diuretics and β-blockers, but their quantitative importance in generating diabetes is probably fairly small.

Two relatively new types of pharmacotherapy have been recognised to have considerable diabetogenic effects. In HIV therapy, a syndrome of lipodystrophy with hyperlipidaemia, insulin resistance and increased risk of impaired glucose tolerance or diabetes is very common. The mechanisms involved are poorly understood and may relate to an interaction between the anti-HIV drugs and HIV infection itself, with protease inhibitors seeming to play a dominant role. The condition is associated with a loss of peripheral fat, but an increase in visceral fat, the latter being closely associated with insulin resistance and dyslipidaemia.[10]

Secondly, the use of antipsychotic drugs, both new and old, has been associated with an increased incidence of weight gain and type 2 diabetes.[11] The mechanisms involved are still poorly understood, and the effects on insulin resistance and insulin secretion are not well documented. The incidence of diabetes seems out of proportion to the weight gained, and additional effects on pancreatic β-cell function are strongly suggested. This area clearly requires further good clinical research studies.

Consequences of diabetes and the metabolic syndrome

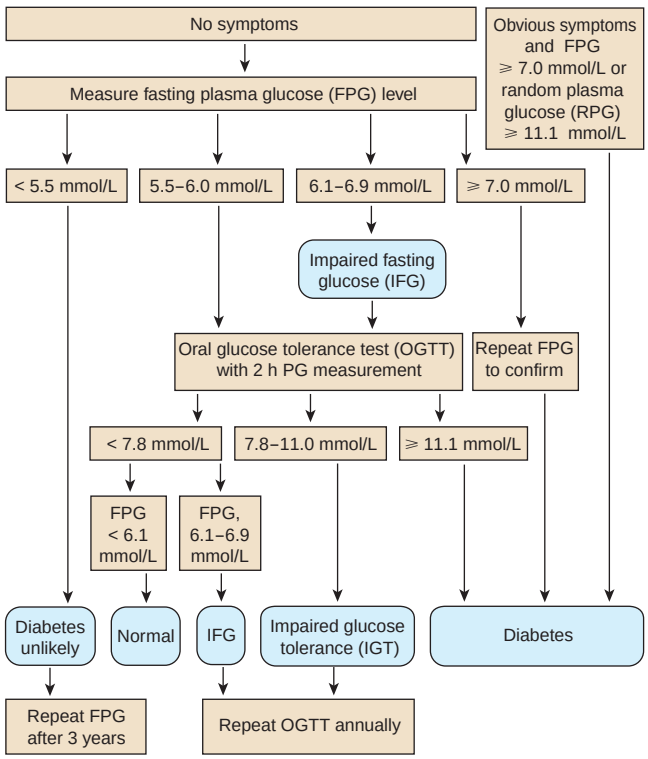
The long-term complications of diabetes, including macro- and microvascular disease and neuropathies, will be discussed in detail in a subsequent article. Briefly, by far the greatest cause of morbidity and mortality in type 2 diabetes is cardiovascular disease, and even the "pre-diabetic" state of IGT carries a substantial risk of this disease.[1,12] In fact, recent evidence suggests that the other features of the metabolic syndrome are important in determining cardiovascular risk independent of the degree of glucose intolerance.[13] Clearly, the cardiovascular "clock" starts ticking

2: Prevalence of cardiovascular risk factors according to glucose tolerance status*

Risk factor	Normal glucose tolerance	Impaired fasting glucose	Impaired glucose tolerance	Diabetes (type 1 or 2)
Obesity (BMI ⩾ 30 kg/m^2)	16.2%	29.9%	31.4%	46.2%
Hypertension (BP ⩾ 140/90 mmHg)	21.1%	44.4%	51.1%	68.6%
LDL ⩾ 3.5 mmol/L	47.3%	69.9%	62.1%	63.8%
HDL < 1.0 mmol/L	14.9%	27.1%	22.5%	39.1%
Triglycerides ⩾ 2.0 mmol/L	19.6%	39.9%	38.8%	56.7%

BMI = body mass index. BP = blood pressure. LDL = low-density lipoprotein. HDL = high-density lipoprotein.
* Data from the AusDiab study.[6] Data are not age or sex adjusted.

3: Diagnosis and screening for type 2 diabetes and impaired glucose metabolism*



*All glucose measurements are for venous plasma glucose.

many years before the development and diagnosis of type 2 diabetes.

Diagnosis and screening for type 2 diabetes

As 50% of cases of type 2 diabetes are currently undiagnosed, enhanced detection and diagnosis are critical. This is an important objective of the Australian Department of Health and Ageing National Integrated Diabetes Program (NIDP), which aims to improve management of diabetes in general practice.[14]

Most patients with obvious symptoms, such as thirst or polyuria, will have plasma glucose levels ⩾ 7.0 mmol/L (fasting) or ⩾ 11.1 mmol/L (random), which establishes the diagnosis. In this situation, an oral glucose tolerance test (OGTT) is not necessary and is, in fact, inappropriate.

However, as a large proportion of people with type 2 diabetes have no symptoms, screening is necessary. Screening may also detect IFG and IGT, with the potential for preventing progression to type 2 diabetes and other adverse effects.

How should screening be done?

While more data on the cost effectiveness of different methods of screening are needed, the current consensus is that the appropriate initial test is measurement of fasting plasma glucose (FPG) level. While measurement of random plasma glucose level may be more convenient, it has limited ability to correctly classify individuals. Measurement of glycosylated haemoglobin (HbA_{1c}) is theoretically an attractive alternative. However, as standardising the different assay methods remains an issue, cut-off levels for screening and diagnosis are not available.

If FPG level on initial screening is in the range 5.5–6.9 mmol/L, it should be followed up with an OGTT (Box 3).[14,15] If FPG level is < 5.5 mmol/L, FPG testing should be repeated every 3 years. If either IFG or IGT is diagnosed (FPG level of 6.1–6.9 mmol/L or 2 h plasma glucose level after OGTT of 7.8–11.0 mmol/L, respectively), testing for diabetes by OGTT should be repeated annually. About 25%–35% of people with IFG also have IGT, and vice versa, but many people have one condition without the other. While those with both IFG and IGT are at greatest risk of progressing to diabetes, those with one but not the other still have an increased risk of both future diabetes and cardiovascular disease.

Who should be screened?

Screening should be offered to people with any one of:[13]

- age > 55 years (or 35 years if Aboriginal or Torres Strait Islander, Pacific Islander, Indian or Chinese);
- known IGT or IFG;
- ischaemic cardiovascular disease;
- prior gestational diabetes; or
- polycystic ovary syndrome combined with obesity.

Screening should also be offered to people aged 45–55 years with any one of the following:

- obesity (body mass index ⩾ 30 kg/m^2);
- a first-degree relative with type 2 diabetes; or
- hypertension.

Prevention of type 2 diabetes

Are lifestyle changes effective?

Epidemiological data suggest that lifestyle changes involving increased physical activity and reduced energy intake will at least partially prevent type 2 diabetes. It is only very recently that prospective intervention studies have clearly confirmed the efficacy of such measures[16,17] (Box 4). The American[16] and Finnish[17] prevention studies illustrate the benefits in a developed country. Participants in both studies were people who already had IGT and were overweight or obese (thus a very high-risk group). With a mean follow-up of about 3 years, the two studies had remarkably concordant outcomes, with a 58% reduction in incidence of type 2 diabetes, resulting from a reduction in energy intake targeted to achieve weight reductions of 7% and 5%, respectively, and an exercise regimen targeted to achieve moderate levels of exercise for 150 and 210 minutes per week, respectively. Of course, adherence to the recommendations was not perfect in either study, but it was impressive that, among participants in the Finnish study who adhered to all components of the recommended regimen, not a single case of type 2

diabetes appeared during 4 years.[17] In a developing country, China, the Da Qing study also showed substantial preventive benefit of exercise, diet, or a combination of the two.[18]

The interventions in the American and Finnish studies were very resource intensive. The resources required to apply them to a whole population would be impossibly expensive. The challenge now is to find practical measures to achieve increased physical activity and reduced calorie intake in much larger population groups at reasonable cost.

4: Major intervention studies to reduce the incidence of type 2 diabetes

Study	Participants, mean duration	Participant characteristics	Incidence of diabetes
Diabetes Prevention Program (USA)[16]	3234, 2.8 years	IGT Mean age, 50.6 years Mean BMI, 34 kg/m^2	*Control:* 11.0% pa *Lifestyle:** 58% reduction *Metformin:* 31% reduction
Finnish study[17]	522, 3.2 years	IGT Mean age, 55 years Mean BMI, 31 kg/m^2	*Control:* 9.8% pa *Lifestyle:*† 58% reduction
Da Qing IGT and Diabetes Study[18]	577, 6 years	IGT Mean age, 45 years Mean BMI, 26 kg/m^2	*Control:* 13.3% pa *Diet:*‡ 33% reduction *Exercise:*§ 47% reduction *Diet plus exercise:* 38% reduction
STOP-NIDDM Acarbose Study[19]	1429, 3.3 years	IGT Mean age, 54 years Mean BMI, 31 kg/m^2	*Placebo:* 12.7% pa *Acarbose:* 25% reduction

IGT = impaired glucose tolerance. BMI = body mass index. pa = per annum.
* At least 7% weight loss and 150 min physical activity per week.
† At least 5% weight loss and 210 min physical activity per week.
‡ Target BMI of 23 kg/m^2.
§ Increase exercise by at least 1 unit per day (eg, extra 30 min of slow walking or 5 min of swimming).

Can drugs prevent type 2 diabetes?

Drug prevention of type 2 diabetes is, and likely will remain for some time, controversial. However, by analogy with lipid-lowering therapy to prevent cardiovascular disease, drug therapy may well become a major plank of preventive therapy for type 2 diabetes in the future. In the American Diabetes Prevention Program, the metformin arm (without lifestyle intervention) showed a 31% reduction in incidence of type 2 diabetes,[16] while a study of the α-glucosidase inhibitor acarbose showed a 25% reduction.[19]

Results of ongoing trials of the efficacy of the thiazolidinediones ("glitazones") in preventing type 2 diabetes are awaited with great interest.[20] These drugs are insulin-sensitising agents, and an early study with troglitazone (now withdrawn because of liver toxicity) showed very positive results.[21] In addition, some prospective clinical trials of the cardiovascular benefits of statins and angiotensin-converting enzyme (ACE) inhibitors have unexpectedly shown a modest reduction in incidence of diabetes,[22,23] but emerging data from other studies are inconsistent.

Further study is needed to determine the place of pharmacotherapy in diabetes prevention,[20] and whether it is less than additive, additive, or more than additive (synergistic), to the effects of lifestyle modification.

How should we apply this knowledge?

For the community: There is a pressing need for a national preventive program to combat obesity, diabetes and related comorbidities. While healthy eating and physical activity programs already exist, they do not seem be achieving wide penetration in the community. Recently, a National Obesity Taskforce was established between the Commonwealth, states and territories.[24] It is hoped this will result in substantial coordinated activity covering a broad spectrum of measures to facilitate increased physical activity, reduce energy intake and ensure a better mix of macronutrients (particularly reduced saturated fat intake). Actions that could be considered range from providing better bicycle paths and safer play areas for children, to healthcare insurance rebates for demonstrated participation in physical activity programs, and, in the food area, further enhancement of food labelling, reduced television advertising of food to children, and a tax on high-fat foods. The cost of an effective program might be great, but, if we do nothing, the resulting healthcare costs from the massive increase in obesity, diabetes and comorbidities will surely be greater.

For targeted risk groups: Several categories of people are at substantially increased risk of type 2 diabetes. Particular ethnic groups at high risk might be targeted for preventive information and action through ethnic organisations, while Indigenous people could be targeted through Aboriginal and Torres Strait Islander health facilities. In addition, there is the potential to channel preventive measures to the siblings and children of people with type 2 diabetes through the register of the National Diabetes Services Scheme, although privacy legislation may be an obstacle. About 90% of people with diagnosed diabetes in Australia are registered with this outstanding scheme, run through Diabetes Australia, whereby the federal government subsidises the supply of injection consumables and testing strips.[25] Follow-up preventive measures for women with gestational diabetes, who are at very high long-term risk of diabetes, are also important.

For individuals: General practitioners, physicians specialising in diabetes, and hospital and community diabetes services could target "at-risk" individuals in a variety of ways. However, GPs have a paramount role, as they see a constant stream of individuals at high risk because of a family history of diabetes, hypertension, obesity, cardiovascular disease and other conditions. Instruction on preventive measures can be delivered in a variety of ways, including the *Smoking, nutrition, alcohol and physical activity (SNAP) framework for general practice*.[26] Appropriate screening of at-risk individuals will identify those with IFG or IGT. Impart-

5: Case report — preventing type 2 diabetes

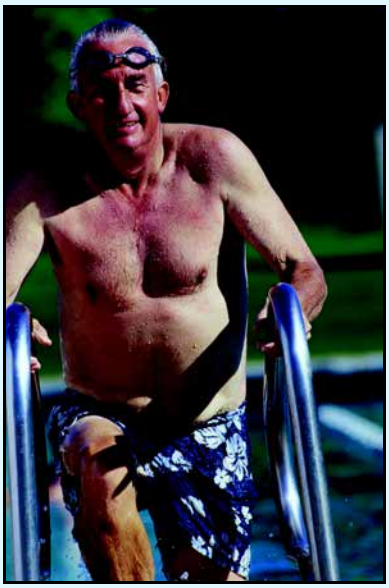
Presentation: A 52-year-old man presented to his general practitioner wanting a "cholesterol check". He mentioned that his mother had "mature-onset" diabetes and asked if he should also have a check for this. He had no symptoms, did not smoke and had no significant past illnesses. He did little physical activity.

Examination: He was 176 cm tall and 89 kg (body mass index, 29 kg/m^2, equating to overweight), with waist measurement, 107 cm, waist/hip ratio, 1.2 (reference range [RR] for male, ≤ 0.9]), and blood pressure, 150/90 mm Hg (RR, < 140/90 mmHg). Physical examination showed no other abnormalities.

Investigations: Measurement of serum lipid levels showed total cholesterol, 4.9 mmol/L (RR, < 5.5 mmol/L), high density lipoprotein [HDL], 0.9 mmol/L (RR, ≥ 1.0–2.0 mmol/L), and triglycerides, 2.7 mmol/L (RR, < 2.0 mmol/L). Fasting plasma glucose (FPG) level was at the upper end of the reference range at 5.9 mmol/L (RR, < 6.1 mmol/L), A glucose tolerance test was therefore indicated. This showed an FPG level of 5.8 mmol/L (normal) and a 2 h PG level of 9.0 mmol/L (indicating impaired glucose tolerance).

Diagnosis: The patient was diagnosed with metabolic syndrome on the basis of impaired glucose tolerance, central obesity, hypertension and dyslipidaemia (increased triglyceride level and low HDL level).

Management: After discussion of the risk of developing type 2 diabetes, and the substantially increased risk of cardiovascular disease, the patient agreed to modify his lifestyle. He and his wife saw a dietitian, who emphasised reducing calories and saturated fat. He purchased an exercise bike and either rode this, walked briskly or swam laps at the local swimming pool for 30 minutes each morning before breakfast.



Course: After 6 months, he had lost only 3 kg weight, but waist circumference was reduced by 6 cm. Blood pressure was 130/75 mm Hg; FPG, 5.4 mmol/L; total cholesterol, 4.2 mmol/L; HDL, 1.2 mmol/L; and triglycerides, 1.5 mmol/L. He felt generally better and more energetic and indicated his willingness to continue the diet and exercise regimen. His GP encouraged this, but also warned him that discontinuing the program would put him at high risk of developing type 2 diabetes, hypertension and cardiovascular disease. The GP also recommended that, in any case, he should have an annual glucose tolerance test, and check of blood pressure and fasting lipid levels.

- *FPG sceening was indicated based on the patient's age, hypertension and family history of type 2 diabetes.*
- *As the FPG level was between 5.5 and 6.9 mmol/L, an oral glucose tolerance test was indicated.*

ing the knowledge to patients that they have a "pre-diabetic state" may be the spur they need to take preventive action (see *Case report*, Box 5).

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(Received 12 Jun 2003, accepted 18 Aug 2003) ❑