# Clinical presentations, diagnosis and prevention of diabetes

## PCDS Free Care Labore Core CCPD Module 6 Second edition

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## Philip Evans

Researchers, public health physicians and frontline clinicians, including GPs, are increasingly convinced that we are entering an epidemic (if not a pandemic) of diabetes mellitus. Rates of diabetes prevalence are increasing across the world, particularly in developing countries, and an increasing number of people are being diagnosed in primary care. This article explores the causes and diagnosis of type 2 diabetes, discussing risk factors, and management and prevention strategies for both type 2 diabetes and pre-diabetes in primary care.

The number of patients with diabetes mellitus continues to escalate dramatically. Rates of diabetes are increasing across the world. In 2007 it was estimated that 2.45 million people in the UK population had diabetes (Yorkshire & Humber Public Health Observatory, 2007). In 2012 this has risen to 2.9 million people, and it is predicted that by 2025 there will be 5 million people in the UK with diabetes (Diabetes UK, 2012). Worldwide there were thought to be 366 million people with diabetes in 2011 – 90% of patients with diabetes have

type 2 diabetes (Whiting et al, 2011). The commonest cause of death in diabetes remains cardiovascular disease, and it accounts for 44% of all deaths in people with type 1 diabetes and 52% of deaths in people with type 2 diabetes (Morrish et al, 2001). Life expectancy is shortened considerably by both types of diabetes – more than 20 years for type 1 diabetes and up to 10 years for type 2 (Department of Health [DH], 2001).

Allied to this increase in prevalence of type 2 diabetes is the growing number of people with intermediate or borderline

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#### Learning objectives

After reading this article, the participant should be able to:

- 1. Describe the various forms of diabetes and how they differ.
- 2. Explain the process of diagnosing diabetes.
- 3. Define the concept and reasoning behind the term "pre-diabetes".
- Outline the evidence that exists regarding interventions to prevent or delay the onset of diabetes.

#### Key words

- Diagnosis
- Pre-diabetes
- Prevention

Philip Evans is a GP in Exeter, Senior Clinical Research Fellow, University of Exeter Medical School, and Co-director of the South West Peninsula Diabetes Local Research Network. "Diabetes mellitus is 'a group of metabolic diseases characterised by hyperglycaemia resulting from defects in insulin secretion, action or both'." hyperglycaemia (often known as prediabetes). This condition carries a raised cardiovascular risk (Tabák et al, 2012) and the challenge to primary care still remains that of early diagnosis, effective intervention and, if possible, prevention of both of these disorders.

Recent changes in diagnosis will soon have a major impact on primary care, but the major issues remain: how can we define diabetes and pre-diabetes and how can we prevent people developing these potentially life-threatening conditions and their complications?

#### What is diabetes?

It is recognised that chronically raised blood glucose (hyperglycaemia) has numerous implications for the health of the individual. Diabetes mellitus is "a group of metabolic diseases characterised by hyperglycaemia resulting from defects in insulin secretion, action or both". This definition by the American Diabetes Association (ADA; 2009) illustrates the fact that diabetes is a syndrome with multiple causes. The vast majority of people with diabetes fall into two main groups, i.e. type 1 and type 2 (ADA, 2009).

Type 1 diabetes is caused by an absolute deficiency of insulin, thought to be due to auto-immune destruction of pancreatic islet cells. Type 1 diabetes accounts for between 5 and 10% of all cases, and is often seen in younger people. Type 2 diabetes, however, is far more common (90% of all cases) and is usually diagnosed in people over 45 years of age who are often obese or physically inactive. It is rapidly increasing in prevalence and is the driver for the current diabetes epidemic. It is strongly dependent on ethnicity and is more common in South Asian or Afro-Caribbean populations. In these populations in the UK, people may develop type 2 diabetes at an earlier age and at a lower BMI. Unlike type 1 diabetes, type 2 diabetes is characterised by a relative insulin deficiency and is often associated with insulin resistance and features of the

so-called metabolic syndrome (an increase in waist circumference and raised blood pressure, low high-density lipoprotein cholesterol, raised plasma triglycerides or a raised blood glucose; Alberti et al, 2005).

Type 2 diabetes usually develops after a long prodromal period of several years of gradually increasing glucose levels (Harris et al, 1992) and most people pass through a period of pre-diabetes before their hyperglycaemia reaches the diabetes threshold. Research published from the Whitehall II prospective study shows that people diagnosed with type 2 diabetes had a slow increase in their blood glucose levels over the 13 years of the study but then their blood glucose levels rose rapidly in the 2 or 3 years preceding the diagnosis (Tabák et al, 2009). A recent study by the Cambridge team, following up their Ely study, suggested that this lead time for diagnosing diabetes has shortened from the 9-12 years suggested in the original US study in 1992 (Harris et al, 1992) to 3.3 years in 2012 (Rahman et al, 2012). This may of course be due to greater screening and awareness of diabetes on the part of primary care teams.

Diabetes is often asymptomatic until glucose levels rise. Whatever the cause of the hyperglycaemia, however, the symptoms of diabetes include polyuria, urinary frequency and polydipsia (often waking up needing a drink in the middle of the night), all caused by an osmotic diuresis due to glycosuria. Other symptoms are weight loss (sometimes dramatic), tiredness, blurred vision and susceptibility to infections such as vaginal or penile candidiasis. Long-term complications can be disabling, even fatal, and include neuropathy, retinopathy, cardiovascular disease, sexual dysfunction and a significant impact on the individual's quality of life and social functioning. However, even at diagnosis, around 25% of people may already have complications (UK Prospective Diabetes Study [UKPDS] Group, 1998). It has also been noted at diagnosis that by that stage nearly half of the patients' insulin secretion had been lost (UKPDS Group, 1995), indicating that

the progressive loss of insulin secretory reserve underpins the progression of diabetes with time, and, hence, the onset of symptoms.

#### Rarer causes of diabetes

Type 2 diabetes is generally considered to be a polygenic disorder. Monogenic, as opposed to polygenic, causes of diabetes are seen less frequently (1-2% of all cases) but nevertheless can present to GPs. For example, it is thought that each GP practice has at least one patient whose diabetes is due to maturity-onset diabetes of the young (MODY), although this is unlikely to have been recognised as such. MODY is a monogenic autosomal dominant condition often causing hyperglycaemia in people before their 20s and hence is likely to be diagnosed as either type 1 or early type 2. The chromosomal defects and functional deficiencies have now been determined. The commonest form involves a mutation in one of the liver transcription factors known as hepatocyte nuclear factor (HNF)-1 alpha. Treatment options in these people are often dependent on the patient's genetic sub-type (e.g. the use of low-dose sulphonylureas in people with HNF-1 alpha; Murphy et al, 2008). A very practical and educational website is www.diabetesgenes.org run by Professor Hattersley's team in Exeter.

Latent autoimmune diabetes of adulthood (LADA) is a variant of diabetes that, like MODY, is receiving more attention of late. It is relatively common and has been estimated to constitute up to 12% of people initially diagnosed with type 2 (Naik et al, 2009). It is frequently misdiagnosed but should be considered in people who do not fit the typical picture of type 2 diabetes (Appel et al, 2009). LADA is an autoimmune disorder that often presents with chronic hyperglycaemia and rarely diabetic ketoacidosis. People may be only marginally overweight and lack the other classical features of insulin resistance. Other autoimmune conditions such as thyrotoxicosis or coeliac disease are often seen in the same patient. However these people have progressive hyperglycaemia not responding to treatment and hence people with LADA will frequently need insulin treatment within several years of diagnosis as the remaining beta cells fail. In LADA, anti-pancreatic antibodies including antiglutamic acid decarboxylase (GAD) are often positive. LADA, rather confusingly, is also known as type 1.5 diabetes as it has some characteristics of both types of diabetes! If you need advice on this type of patient your local diabetologist would be happy to help.

#### Coding of diabetes

With the changes in the Quality and Outcomes Framework (QOF) in 2006 there has been a steady demand for effective and accurate coding of type 1 and type 2 diabetes in general practices. In the light of the diagnostic issues described above, GPs need to be clear what type of diabetes the patient has, in order to optimise treatment and prevent complications. For example MODY can often be diagnosed as type 1 diabetes (yet insulin is often not needed); type 1 diabetes can occur in older people as highlighted in the *BMJ* recently (Lasserson et al, 2012), and type 2 diabetes is now seen in younger people.

A recent Working Group commission by NHS Diabetes and the Royal College of General Practitioners (RCGP) produced an excellent report in 2011 (NHS Diabetes and RCGP, 2011). This followed on from a systematic review which investigated incorrect coding and classification of diabetes (Stone et al, 2010). The Working Group identified three common failings: Misdiagnosis (the patient doesn't actually have diabetes); Misclassification (the patient is coded as having the wrong type of diabetes); and Miscoding (when the wrong computer code is used). The Group agreed that accurate coding was a complex and exacting task but offered a simple algorithm support classification: http://www. to diabetes.nhs.uk/information and data/ classification\_of\_diabetes\_/. It also has made available six MIQUEST queries that

#### Page points

- GPs need to be clear what type of diabetes the patient has, in order to optimise treatment and prevent complications.
- 2. Maturity-onset diabetes of the young (MODY) can often be diagnosed as type 1 diabetes (though insulin is often not needed).
- 3. Type 1 diabetes can occur in older people.
- 4. Type 2 diabetes is now seen in younger people.

"Diabetes can and should be diagnosed in primary care without specialist referral unless the patient's condition is potentially life-threatening, such as diabetic ketoacidosis, or his or her hyperglycaemia is severe and needs immediate insulin treatment." practices can download and use to look for evidence of misdiagnosis or misclassification in their patients. These are well worth doing as an audit in your practice and sharing with your staff or putting in your revalidation portfolio. The associated British Medical Journal editorial (Farmer and Fox, 2011) is also helpful.

#### **Diagnosing diabetes**

Diabetes can and should be diagnosed in primary care without specialist referral unless the patient's condition is potentially lifethreatening, such as diabetic ketoacidosis (DKA), or his or her hyperglycaemia is severe and needs immediate insulin treatment.

Until recently, the diagnosis of diabetes or pre-diabetes was based upon blood glucose estimations. The World Health Organization International (WHO) and Diabetes Federation (IDF; 2006) and the ADA (2009) recommended that the diagnosis of diabetes (and pre-diabetes states) was based on a blood glucose measurement that was random, fasting or after a glucose load (oral glucose tolerance test [OGTT]). Traditionally the OGTT was promoted as the gold standard for the diagnosis of diabetes and has been used extensively in epidemiological studies. However, the recommended use of repeated fasting plasma glucose estimations, which were cheap and more convenient for both doctor and patient, meant that GPs in the UK moved away from the OGTT.

However, in 2011 the WHO recommended the use of  $HbA_{1c}$  as a diagnostic test for diabetes (WHO, 2011). It recommended that a level of  $\geq 48$  mmol/mol (6.5%) was the cut-off for diagnosing diabetes and this guidance was reiterated in UK-wide guidance via a recent consensus statement (John et al, 2012). HbA<sub>1c</sub> is known to reflect elevated levels of blood sugar (hyperglycaemia) over the preceding 2–3 months and an analysis of a venous blood sample in an accredited laboratory using quality assurance tests was recommended. Point-of-care HbA<sub>1c</sub> tests are not recommended for diagnosis unless their performance can match that of other laboratory methods. However  $HbA_{1c}$ , which does not need a fasting test, is far more practical than either fasting glucose tests or an OGTT and may well promote more widespread screening for diabetes. It is generally accepted that  $HbA_{1c}$  testing identifies fewer people with diabetes than glucose tests (fasting glucose or OGTT; Inzucchi, 2012), although many people consider an OGTT to be not feasible in a primary care setting due to the need for fasting, waiting and double appointments.

John et al (2012) recommended that an HbA<sub>1c</sub> level of  $\geq$ 48 mmol/mol (6.5%) should be used to diagnose diabetes in most situations but in people without diabetes symptoms, a repeat HbA<sub>1c</sub> in the same laboratory within 2 weeks was recommended. In people symptomatic of hyperglycaemia with relatively slow onset of symptoms, a single result would suffice. However, there are some clinical situations when HbA1c should not be used for diagnosis (Table 1). Perhaps the most important situation is not to use HbA1c when considering a diagnosis of type 1 diabetes. There are also difficulties in using HbA<sub>1c</sub> in people with haemoglobinopathies,

#### Table 1. When HbA<sub>1c</sub> must not be used as the sole test to diagnose diabetes (John et al, 2012).

As HbA<sub>1c</sub> reflects glycaemia over the preceding 2-3 months, it may not be raised if blood glucose levels have risen rapidly. Examples of instances where HbA<sub>1c</sub> should not be used as the sole test are:

- ALL symptomatic children and young people
- Symptoms suggesting type 1 diabetes at any age
- Diabetes symptoms of short duration
- People at high risk of diabetes who are acutely ill
- When the individual is taking medication that may cause a rapid rise in glucose levels, e.g. corticosteroids or antipsychotics
- Acute pancreatic damage/pancreatic surgery

anaemia or disorders causing an altered red cell lifespan. The report also concludes that a value less than 48 mmol/mol (6.5%) does not exclude diabetes diagnosed on glucose tests. The expert group also recommended that those people with an HbA<sub>1c</sub> of 42-47 mmol/mol (6.0-6.4%) should be considered to be at high risk and the equivalent of impaired fasting glucose (IFG) or impaired glucose tolerance (IGT; i.e. pre-diabetes). A value under 42 mmol/mol (6.0%) was considered to be "normal". However, unlike in the UK, the ADA suggested that pre-diabetes should include people with an HbA1c of 37-47 mmol/mol (5.7-6.4%). This group they have termed increased glycated haemoglobin (IGH).

The NHS Health Check programme in the UK advocates the same use of  $HbA_{1c}$  with a cut-off of  $\geq$ 48 mmol/mol (6.5%) as diagnostic of diabetes (NHS Health Check Programme, 2009) as does the recently published NICE guidance (NICE, 2012).

It should be noted that any of these glucose or  $HbA_{1c}$  cut-offs for the development of diabetes are in effect arbitrary thresholds along the continuum of hyperglycaemia as they are considered to be the level above which diabetic retinopathy (a specific diabetes-related microvascular complication) is more prevalent.

The introduction of diagnosis based on  $HbA_{1c}$  now means that diabetes can be diagnosed in four ways (see *Table 2*). There has been considerable discussion in the international diabetes community about this change (Bonora and Tuomilehto, 2011). The situation remains rather complex and potentially confusing to those of us working in primary care. How these changes will impact on primary care and the prevalence of diabetes in the UK is not known at this time and will need to be watched carefully.

#### Defining pre-diabetes

Another area which is still much debated is the diagnosis of the intermediate hyperglycaemic states collectively known as pre-diabetes. All these conditions have in common the fact that blood glucose levels or  $HbA_{1c}$  are raised, yet are not above the threshold that is diagnostic of type 2 diabetes. The two most important features of pre-diabetes in primary care are the increased risk of cardiovascular disease (CVD), which is two to three times that of normoglycaemic individuals (Coutinho et al, 1999), and the increased risk of progression to type 2 diabetes. Hence the potential for prevention of both diabetes and CVD in this high-risk group.

The term pre-diabetes has been considered by some as being potentially misleading, as a large proportion of people with prediabetes do not progress to diabetes. Other terms such as non-diabetic hyperglycaemia (NDH), intermediate hyperglycaemia (IH) and impaired glucose regulation (IGR) are therefore gaining in popularity. The RCGP guidelines recently suggested the term "Non-Diabetic Hyperglycaemia" as its preferred term and included IGT, IFG and gestational diabetes in this group (NHS Diabetes and RCGP, 2011).

Both IFG and IGT, for example, are increasingly prevalent. It is estimated that 5.1% of the UK population aged 20–79 may have IGT (IDF, 2003). Pre-diabetes carries an increased risk of progression to type 2

#### Page points

- 1. The term pre-diabetes has been considered by some as being potentially misleading.
- 2. Terms such as nondiabetic hyperglycaemia, intermediate hyperglycaemia and impaired glucose regulation are gaining in popularity.
- 3. The Royal College of General Practitioners guidelines recently suggested the term "nondiabetic hyperglycaemia" as its preferred term.

Table 2. Recommendations for the diagnostic criteria for diabet	es
and intermediate hyperglycaemia.	

Measure	Diabetes	IGR
Fasting plasma glucose	≥7.0 mmol/L	6.1–6.9 mmol/L
2-hour glucose post-OGTT	≥11.1 mmol/L	≥7.8 mmol/L and <11.1 mmol/L
Random glucose in presence of symptoms	≥11.1 mmol/L	NA
HbA <sub>1c</sub>	≥48 mmol/mol (6.5%)	42–47 mmol/mol (6.0–6.4%)

IGR=impaired glucose regulation; NA=not applicable; OGTT=oral glucose tolerance test.

"At least 70% of people with pre-diabetes will eventually progress to frank diabetes and it is estimated that by the year 2030, 470 million people globally will have pre-diabetes." diabetes although this can vary dependent on ethnicity and other factors (Unwin et al, 2002). It is widely accepted that people with these conditions are at greater risk of both type 2 diabetes and cardiovascular disease (Coutinho et al, 1999) and interventions designed to prevent diabetes have in the main been targeted at this group. The ADA recently concluded that at least 70% of people with pre-diabetes will eventually progress to frank diabetes and it is estimated that by the year 2030, 470 million people globally will have pre-diabetes (Tabák et al, 2012).

#### Education of people with pre-diabetes

Our previous work in developing a pragmatic screening programme using the GP database identified a large proportion of people with pre-diabetes (Greaves et al, 2004). Studies had previously shown that people and health professionals alike were confused about the implications of the diagnosis (Wylie et al, 2002; Whitford et al, 2003; Williams et al, 2004). We therefore developed an educational package for people with prediabetes and their healthcare professionals. This package, known as WAKEUP (Ways

#### Box 1. Case study.

#### Narrative

Mrs C, aged 72, presented to her GP with essential hypertension in 2005. This was well controlled with an angiotensin-converting enzyme inhibitor and she had regular annual fasting blood glucose measurements as part of her hypertensive care. Recently, her GP has used HbA<sub>1c</sub> results in a non-fasting state to assess her risk of diabetes.

In 2011, at her annual review, her HbA<sub>1c</sub> level was 46 mmol/mol (6.4%). The diagnosis of pre-diabetes was made and Mrs C was started on a statin (her total serum cholesterol was 6.5 mmol/L) and advice was given about weight loss and exercise. She was referred to an exercise-on-prescription scheme locally and has managed to lose 5 kg ( $\geq$ 5% of her weight). Subsequent HbA<sub>1c</sub> measurements have fallen and her most recent level was 43 mmol/mol (6.1%).

#### Discussion

This case illustrates the importance of addressing cardiovascular risk in the context of pre-diabetes. It demonstrates the need for regular  $HbA_{1c}$  measurements to track glycaemia in an individual patient. It also shows that hyperglycaemia can improve with time and inexorable progression to type 2 diabetes is not always seen.

of Addressing Knowledge Education and Understanding in Pre-diabetes), was found to be acceptable to both patients and healthcare professionals (Evans et al, 2007). Even in 2012, this is still one of the few resources available to give to people with pre-diabetes.

#### Primary prevention of diabetes

As the transition from normoglycaemia through impaired glucose regulation to type 2 diabetes takes several years it is logical to try to intervene and aim to prevent or delay the onset of diabetes before its onset. This can be at a population level or at the level of the individual patient. The best evidence exists in high-risk people with IGT or IFG.

There is now substantial evidence from large-scale randomised trials in various populations across the world that progression to diabetes can be prevented or delayed in high-risk groups both by behavioural (Tuomilehto et al, 2001; Knowler et al, 2002; Ramachandran et al, 2006) and by pharmacological interventions (Chiasson et al, 2002; Knowler et al, 2002; Torgerson et al, 2004; Gerstein et al, 2006). *Box 1* gives a case study highlighting some common problems encountered in primary care.

#### Lifestyle

A meta-analysis has shown that lifestyle interventions can produce a 50% relative risk reduction in the incidence of type 2 diabetes at 1 year (Yamaoka and Tango, 2005). Typically these interventions are in high-risk individuals, such as those with pre-diabetes (usually IGT), and interventions are targeted at halting or slowing beta-cell dysfunction and hence incident type 2 diabetes. The majority of behavioural interventions are intensive and designed to increase an individual's physical activity levels and encourage weight loss and dietary change. Relatively modest changes in lifestyle such as a 5% reduction in weight or an increase in moderate physical activity to 4 hours a week can have important benefits in reducing the risk of diabetes. It was also noted that the beneficial effects observed in the Finnish Diabetes Prevention Study persisted when the people were followed up a median of 3 years after the intervention had finished (Lindström et al, 2006) and up to 10 years in the case of the Diabetes Prevention Program (DPP) in the US (DPP Research Group, 2009). Also, lifestyle change has other general benefits for the patient. Hence lifestyle interventions are the cornerstone of the treatment of prediabetes (Tabák et al, 2012).

#### Drugs

As well as lifestyle interventions, drugs have also been shown to reduce progression to type 2 diabetes. These include metformin (Knowler et al, 2002; Ramachandran et al, 2006), acarbose (Chiasson et al, 2002), orlistat (Torgerson et al, 2004) as well as troglitazone (Azen et al, 1998) and rosiglitazone (Gerstein et al, 2006) - the latter two of which have now both been withdrawn. A meta-analysis (Gillies et al, 2008) showed that drug interventions were both less effective and less cost-effective than lifestyle. There is also much debate about whether these drugs simply mask progression to diabetes by lowering blood glucose, which then rises in the subsequent wash-out period once treatment has finished. On balance, however, it is generally recognised that diabetes prevention through lifestyle or drugs is cost-effective and should be actively promoted in clinical practice (Gillies et al, 2008). However, it was only in 2012 that NICE offered formal guidance on

#### Box 2. Useful resources.

preventing type 2 diabetes (NICE, 2012).

#### NICE: Preventing type 2 diabetes

Although population screening for diabetes is not thought to be appropriate (Wareham and Griffin, 2001), targeted or selective screening for both diabetes and pre-diabetes is now considered to be both effective and cost-effective (Waugh et al, 2007). NICE guidance (NICE, 2012) does not recommend a national screening programme for diabetes; instead it recommends a twostage process of risk identification and screening. It was designed to run alongside the NHS Health Check programme (NHS Health Check Programme, 2009) and complements its previous advice on population interventions to prevent diabetes (NICE, 2011).

Initially people at high risk of diabetes will be identified using a stepped approach conducted in surgeries, pharmacies or other primary care venues. Firstly a validated risk assessment score is recommended and, secondly, a blood test (glucose or  $HbA_{1c}$ ) is taken. Those at high risk are then provided with a quality-assured, evidence-based, intensive lifestyle-change programme as outlined above.

Other screening methods such as opportunistic screening by GPs and their teams as described in our practice (Evans et al, 2008) could provide a complementary screening system but have tended to be overlooked in the recent guidance, despite the

#### Page points

- 1. As well as lifestyle interventions, drugs have also been shown to reduce progression to type 2 diabetes.
- 2. These include metformin, acarbose, orlistat, troglitazone and rosiglitazone (the latter two of which have now both been withdrawn).
- 3. On balance, it is generally recognised that diabetes prevention through lifestyle or drug treatment is cost-effective and should be actively promoted in clinical practice.
- 4. NICE offered formal guidance on preventing type 2 diabetes in 2012.

- NICE public health guidance 35: Preventing type 2 diabetes: population and community interventions (NICE, 2011)
- NICE public health guidance 38. Preventing type 2 diabetes: risk identification and interventions for individuals at high risk (NICE, 2012)
- WAKEUP materials website: http://www.pcmd.ac.uk/pms/research/wakeup.php
- American Diabetes Association clinical standards 2012 (www.diabetes.org)
- IMAGE website (www.image-project.eu)
- For information on coding see www.diabetes.nhs.uk/information\_and\_data/classification\_of\_diabetes\_/
- For an informative overview of the diagnosis of diabetes see Inzucchi's review (Inzucchi, 2012)

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"NICE has suggested that other community settings such as optometrists, pharmacists and dental surgeries would be appropriate places for diabetes screening within their recommendations, although initiatives involving non-generalpractice screening are in the majority at a pilot stage." cost of a new diagnosis being £377 (Pereira Gray et al, 2012).

Validated risk assessment tools suggested include the FINDRISC and Leicester questionnaires (Lindström and Tuomilehto, 2003; Gray et al, 2010), which are both patient-completed tools and can be done online. GPs can also identify high-risk people from their practice computers using the QDScore (Hippisley Cox et al, 2009) or the Cambridge score (Griffin et al, 2000). Interestingly NICE recommends the use of metformin in treating prediabetes, although it is not licensed for this indication.

In the NHS Health Checks programme, (NHS Health Check Programme, 2009), which is now up and running, all people aged 40–74 who are not on a vascular disease or diabetes register will be called in for a face-to-face check and assessment of their broader vascular risk. Those who are overweight or obese or have a raised blood pressure will also be screened for diabetes along the lines that NICE recommends.

The initial pilot for the NHS Health Check programme was promising in many respects (Goyder et al, 2008). However, it has been acknowledged that uptake amongst primary care trusts in England has been variable. NICE has also suggested that other community settings such as optometrists, pharmacists and dental surgeries would be appropriate places for diabetes screening within their recommendations, although initiatives involving non-general-practice screening are in the majority at a pilot stage.

Despite all these initiatives, the challenge still remains for primary care to prevent both type 2 diabetes and its complications.

- Azen SP, Peters RK, Berkowitz K et al (1998) TRIPOD (TRoglitazone In the Prevention Of Diabetes): a randomized, placebo-controlled trial of troglitazone in women with prior gestational diabetes mellitus. *Control Clin Trials* **19**: 217–31
- Bonora E, Tuomilehto J (2011) The pros and cons of diagnosing diabetes with A<sub>1c</sub>. *Diabetes Care* **34**: s184–s216
- Chiasson JL, Josse RG, Gomis R et al (2002) Acarbose for prevention of type 2 diabetes mellitus: the STOP-NIDDM randomised trial. *Lancet* **359**: 2072–7
- Coutinho M, Gerstein HC, Wang Y, Yusuf S (1999) The relationship between glucose and incident cardiovascular events. A metaregression analysis of published data from 20 studies of 95,783 individuals followed for 12.4 years. *Diabetes Care* **22**: 233–40
- Department of Health (2001) National Service Framework for Diabetes: Standards. DH, London. Available at http://bit.ly/NDAmort (accessed 26.11.12)
- Diabetes Prevention Program Research Group (2009) 10year follow-up of diabetes incidence and weight loss in the Diabetes Prevention Program Outcome Study. *Lancet* **374**: 1677–86
- Diabetes UK (2012) Diabetes in the UK 2012 (April 2012): Key statistics on diabetes. Diabetes UK, London. Available at: http://www.diabetes.org.uk/Professionals/ Publications-reports-and-resources/Reports-statisticsand-case-studies/Reports/Diabetes-in-the-UK-2012/ (accessed 26.11.12)
- Evans PH, Greaves C, Winder R et al (2007) Development of an educational 'toolkit' for health professionals and their patients with pre-diabetes: The WAKEUP study (Ways of Addressing Knowledge Education and Understanding in Pre-diabetes). *Diabet Med* **24**: 770–7
- Evans P, Langley P, Gray DP (2008) Diagnosing type 2 diabetes before patients complain of diabetic symptomsclinical opportunistic screening in a single general practice. *Fam Pract* **25**: 376–381
- Farmer A, Fox R (2011) Diagnosis, classification, and treatment of diabetes. *BMJ* **342**: 3319
- Gerstein HC, Yusuf S, Bosch J et al (2006) Effect of rosiglitazone on the frequency of diabetes in patients with impaired glucose tolerance or impaired fasting glucose: a randomised controlled trial. *Lancet* **368**: 1096–105
- Gillies CL, Lambert PC, Abrams KR et al (2008) Different strategies for screening and prevention of type 2 diabetes in adults: cost effectiveness analysis. *BMJ* **336**: 1180-5
- Goyder E, Wild S, Fischbacher C et al (2008) Evaluating the impact of a national pilot screening programme for type 2 diabetes in deprived areas of England. *Fam Pract* **25**:370–5
- Gray LJ, Taub NA, Khunti K et al (2010) The Leicester Risk Assessment score for detecting undiagnosed type 2 diabetes and impaired glucose regulation for use in a multiethnic UK setting. *Diabet Med* **27**: 887–95
- Greaves CJ, Stead JW, Hattersley A et al (2004) A simple pragmatic system for detecting new cases of type 2 diabetes and impaired fasting glycaemia in primary care. *Fam Pract* **21**: 57–62
- Griffin SJ, Little PS, Hales CN et al (2000) Diabetes risk score: towards earlier detection of type 2 diabetes in general practice. *Diabetes Metab Res Rev* 16: 164–71
- Harris MI, Klein R, Wellborn TA, Knuiman MW (1992) Onset of NIDDM occurs at least 4–7 years before clinical diagnosis. *Diabetes Care* 15: 815–19

Alberti KG, Zimmet P, Shaw J (2005) IDF Epidemiology Task Force Consensus Group. The metabolic syndrome – a new worldwide definition. *Lancet* **366**: 1059–62

American Diabetes Association (2009) Diagnosis and classification of diabetes mellitus. *Diabetes Care* **32**: S62–7

Appel SJ, Wadas TM, Rosenthal RS et al (2009) Latent autoimmune diabetes of adulthood (LADA): an often misdiagnosed type of diabetes mellitus. *J Am Acad Nurse Pract* **21**:156–9

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- Hippisley-Cox J, Coupland C, Robson J et al (2009) Predicting risk of type 2 diabetes in England and Wales: prospective derivation and validation of QDScore. *BMJ* **338**: b880
- International Diabetes Federation (2003) *Diabetes Atlas*. 2nd ed. IDF, Brussels, Belgium
- Inzucchi SE (2012) Diagnosis of diabetes. N Engl J Med 367: 542-50
- John WG, Hillson R, Alberti SG (2012) Use of haemoglobin A<sub>1c</sub> (HbA<sub>1c</sub>) in the diagnosis of diabetes mellitus. The implementation of World Health Organization (WHO) guidance 2011. Practical Diabetes 29: 12–12a
- Knowler WC, Barrett-Connor E, Fowler SE et al (2002) Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. *N Engl J Med* **346**: 393–403
- Lasserson D, Rox R, Farmer A (2012) Late onset type 1 diabetes. *BMJ* 344: e2827
- Lindström J, Tuomilehto J (2003) The Diabetes Risk Score: a practical tool to predict type 2 diabetes risk. *Diabetes Care* **26**: 725–731
- Lindström J, Ilanne-Parikka P, Peltonen M et al (2006) Sustained reduction in the incidence of type 2 diabetes by lifestyle intervention: follow-up of the Finnish Diabetes Prevention Study. *Lancet* **368**: 1673–9
- Morrish NJ, Wang SL, Stevens LK et al (2001) Mortality and causes of death in the WHO multinational study of vascular disease in diabetes. *Diabetologia* 44: s14–s21
- Murphy R, Ellard S, Hattersley AT (2008) Clinical implications of a molecular genetic classification of monogenic beta-cell diabetes. *Nat Clin Pract Endocrinol Metab* 4: 200–13
- Naik RG, Brooks-Worrell BM, Palmer JP (2009) Latent autoimmune diabetes in adults. J Clin Endocrinol Metab 94:4635–44
- NHS Diabetes, Royal College of General Practitioners (2011) *Coding Classification and Diagnosis of Diabetes*. Available at: http://www.diabetes.nhs.uk/information\_ and\_data/classification\_of\_diabetes\_/ (accessed 26.11.12)
- NHS Health Check Programme (2009) NHS Health Check: Vascular Risk Assessment and Management Best Practice Guidance. Department of Health, London
- NICE (2011) Public health guidance 35: Preventing type 2 diabetes: population and community interventions. NICE, London
- NICE (2012). Public health guidance 38: Prevention of type 2 diabetes-risk identification and interventions for individual at high risk. NICE, London
- Pereira Gray D, Evans PH, Wright C et al (2012) The cost of diagnosing Type 2 Diabetes Mellitus by clinical opportunistic screening in general practice. *Diabet Med* **29**:863–68
- Rahman M, Simmons RK, Hennings SH et al (2012) How much does screening bring forward the diagnosis of type 2 diabetes and reduce complications? Twelve year follow-up of the Ely cohort. *Diabetologia* **55**:1651-9
- Ramachandran A, Snehalatha C, Mary S et al (2006) The Indian Diabetes Prevention Programme shows that lifestyle modification and metformin prevent type 2 diabetes in Asian Indian subjects with impaired glucose tolerance (IDPP-1). *Diabetologia* **49**: 289–97
- Stone MA, Camosso-Stefinovic J, Wilkinson J et al (2010) Incorrect and incomplete coding and classification of diabetes: a systematic review. *Diabet Med* **27**: 491–7

- Tabák AG, Herder C, Rathmann W et al (2012) Prediabetes: a high–risk state for diabetes development. *Lancet* **379**: 2279–90
- Tabák AG, Jokela M, Akbaraly TN et al (2009) Trajectories of glycaemia, insulin sensitivity, and insulin secretion before diagnosis of type 2 diabetes: an analysis from the Whitehall II study. *Lancet* **373**: 2215–21
- Torgerson JS, Hauptman J, Boldrin MN, Sjöström L (2004) XENical in the prevention of diabetes in obese subjects (XENDOS) study: a randomized study of orlistat as an adjunct to lifestyle changes for the prevention of type 2 diabetes in obese patients. *Diabetes Care* **27**: 155–61
- Tuomilehto J, Lindström J, Eriksson JG et al (2001) Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. *N Engl J Med* **344**: 1343–50
- UK Prospective Diabetes Study (UKPDS) Group (1995) Overview of 6 years' therapy of type II diabetes: a progressive disease. *Diabetes* **44**:1249–58
- UK Prospective Diabetes Study (UKPDS) Group (1998) Intensive blood-glucose control with sulphonylureas or insulin compared with conventional treatment and risk of complications in patients with type 2 diabetes (UKPDS 33). *Lancet* **352**: 837–53
- Unwin N, Shaw J, Zimmet P, Alberti KG (2002) Impaired glucose tolerance and impaired fasting glycaemia: the current status on definition and intervention. *Diabet Med* **19**: 708–23
- Wareham NJ, Griffin SJ (2001) Should we screen for type 2 diabetes? Evaluation against National Screening Committee criteria. *BMJ* 322: 986–8
- Waugh N, Scotland G, McNamee P et al (2007) Screening for type 2 diabetes: literature review and economic modelling. *Health Technol Assess* **11**: iii–iv, ix–xi, 1–125
- Whitford DL, Lamont SS, Crosland A (2003) Screening for type 2 diabetes: is it worthwhile? Views of general practitioners and practice nurses. *Diabet Med* 20: 155–8
- Whiting DR, Guariguata L, Weil C, Shaw J (2011) IDF Diabetes Atlas: global estimates of the prevalence of diabetes for 2011 and 2030. *Diabetes Res Clin Pract* 94: 311–21
- Williams R, Rapport F, Elwyn G et al (2004) The prevention of type 2 diabetes: general practitioner and practice nurse opinions. *Br J Gen Pract* 54: 531–5
- World Health Organization (2011) Use of Glycated Haemoglobin ( $HbA_{1c}$ ) in the Diagnosis of Diabetes Mellitus. Abbreviated Report of a WHO Consultation. WHO, Geneva. Available at: http://bit.ly/seLcYT (accessed 26.11.12)
- World Health Organization, International Diabetes Federation (2006) Definition and Diagnosis of Diabetes Mellitus and Intermediate Hyperglycaemia. Report of a WHO/IDF Consultation. WHO, Geneva
- Wylie G, Hungin AP, Neely J (2002) Impaired glucose tolerance: qualitative and quantitative study of general practitioners' knowledge and perceptions. *BMJ* **324**: 1190
- Yamaoka K, Tango T (2005) Efficacy of lifestyle education to prevent type 2 diabetes: a meta-analysis of randomized controlled trials. *Diabetes Care* **28**: 2780–6
- Yorkshire and Humber Public Health Observatory (2007) Diabetes Key Facts Supplement 2007. YHPHO, York

### Online CPD activity

#### Visit www.diabetesonthenet.com/cpd to record your answers and gain a certificate of participation

Participants should read the preceding article before answering the multiple choice questions below. There is ONE correct answer to each question. After submitting your answers online, you will be immediately notified of your score. A pass mark of 70% is required to obtain a certificate of successful participation; however, it is possible to take the test a maximum of three times. A short explanation of the correct answer is provided. Before accessing your certificate, you will be given the opportunity to evaluate the activity and reflect on the module, stating how you will use what you have learnt in practice. The new CPD centre keeps a record of your CPD activities and provides the option to add items to an action plan, which will help you to collate evidence for your annual appraisal.

- 1. In which ONE of the following situations would it be the MOST appropriate to use HbA<sub>1c</sub> as a sole diagnostic test for diabetes?
  - A. A 14-year-old boy with weight loss and excessive thirst
  - B. A 25-year-old man with polyuria, recurrent intertrigo and ketonuria
  - C. A 47-year-old Asian man who is admitted to hospital with an acute coronary syndrome
  - D. A 52-year-old obese man who has felt lethargic for the past 3 months. He has raised blood pressure and hyperlipidaemia
  - E. A 75-year-old woman who is being treated with high-dose prednisolone. She is worried that she is at risk of diabetes
- 2. Life expectancy for people with type 1 diabetes is approximately HOW MANY years SHORTER than for people without diabetes? Select ONE option only.
  - A. 5
  - B. 10
  - C. 15
  - D. 20
  - E. 30

#### 3. Which SINGLE ONE of the following is NOT associated with insulin resistance? Select ONE option only.

- A. Increased waist circumference
- B. Raised blood pressure
- C. Raised glucose
- D. Raised high-density lipoprotein cholesterol
- E. Raised triglycerides

- 4. According to UK Prospective
   Diabetes Study figures, approximately
   HOW MANY people with diabetes
   have already got complications at
   diagnosis? Select ONE option only.
  - A. 10%
  - B. 25%
  - C. 33%
  - D. 50%
  - E. 66%
- 5. Monogenic causes account for approximately WHAT percentage of people with type 2 diabetes? Select ONE option only.
  - A. 1
  - B. 5
  - C. 10
  - D. 20
  - E. 33
- 6. Which ONE of the following diseases is MOST likely to be associated with the latent autoimmune diabetes of adulthood (LADA) variant of diabetes?
  - A. Coeliac disease
  - B. Colorectal cancer
  - C. Generalised osteoarthritis
  - C. Psoriasis
  - E. Pulmonary fibrosis
- 7. In the UK, which ONE of the following ethnic groups has the HIGHEST risk of developing type 2 diabetes?
  - A. African-Caribbean
  - B. Chinese
  - C. Irish
  - D. Polish
  - E. Russian

- 8. According to WHO recommendations, what is the THRESHOLD level of HbA<sub>1c</sub> (mmol/mol) ABOVE which the diagnosis of diabetes should be made? Select ONE option only.
  - A. 30
  - B. 36
  - C. 42
  - D. 48
  - E. 54
- 9. Which ONE of the following is the MOST appropriate statement regarding HbA<sub>1c</sub> testing to diagnose type 2 diabetes?
  - A. Fewer people are identified compared with using fasting glucose
  - B. More people are identified compared with using fasting glucose
  - C. More people are identified compared with using the oral glucose tolerance test (OGTT)
  - D. The same number of people are identified compared with using fasting glucose
  - E. The same number of people are identified compared with using the OGTT
- 10. According to recent studies, which ONE of the following is the MOST appropriate statement regarding the PREVENTION of the progression of pre-diabetes to diabetes?
  - A. Drug interventions are clinically as effective as lifestyle interventions
  - B. Drug interventions are clinically more effective than lifestyle interventions
  - C. Lifestyle interventions are as costeffective as drug interventions
  - D. Lifestyle interventions are more costeffective than drug interventions