Type 2 diabetes in childhood: epidemiological and clinical aspects

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**Background:** The global obesity epidemic has raised concerns about the risk of a tide of Type 2 diabetes (T2DM) in childhood. This paper aims to review the recent data on the epidemiology of this problem as well as the clinical concerns.

**Sources of data:** A literature search was performed on Medline, and articles about childhood T2DM, in English and published from 2000 to 2008, were reviewed.

**Areas of agreement:** A review of 16 paediatric studies suggest that although T2DM is now more widely reported in childhood, the numbers are still reasonably small although the data do suggest that ethnicity is an important risk factor.

**Areas of controversy:** Although there are emerging data on what appears to be a significant risk of both microvascular and macrovascular complications in youth onset T2DM, the most appropriate management remains unclear. Currently, adult guidelines for management of T2DM are being extrapolated to the adolescent population with T2DM.

**Growing points:** Studies are currently underway to examine the pharmacological management of childhood T2DM.

**Areas timely for developing research:** More data are necessary on the exact prevalence of T2DM among a variety of populations to provide a greater understanding of the risk factors for T2DM and provide indications for screening. In the meantime, great emphasis needs to be placed on obesity prevention if we are to protect the health of future generations of children.

**Keywords:** child/type 2 diabetes
Introduction

Type 2 diabetes mellitus (T2DM) has long been considered a disease of adult middle age, and patients were often overweight or obese, presented incidentally and had a variety of associated cardiovascular disease (CVD) risk factors. Paediatricians, on the other hand, saw only young, slim children with a rapid onset of osmotic symptoms who needed insulin treatment immediately without which they would become seriously ill. Over the last 20 years, the clinical presentation of diabetes in childhood has become much more complicated, and young people are increasingly recognised to have non-type 1 diabetes. Advances in genetic testing have identified a number of young people with maturity onset diabetes of the young (MODY) which can present as insulin- dependent or -independent diabetes and secondary forms of diabetes such as cystic fibrosis related diabetes (CFRDM) are more common. T2DM does appear to present more frequently in childhood causing both diagnostic and management dilemmas. This paper aims to review the epidemiology and risk factors for T2DM in childhood.

Type 1 or type 2?

The diagnosis of diabetes is based upon a combination of symptoms and an abnormal response to an oral glucose challenge. Diagnostic cut-offs define a group of people who are at risk of the long-term complications of diabetes in both macrovascular and microvascular terms and who therefore need treatment. The same definitions of diabetes for adults are also used to diagnose diabetes in childhood, yet there are a number of unresolved issues:

1. The data upon which abnormalities are defined are based upon an adult population and there do not appear to be any data that have examined the normal responses to an oral glucose challenge in a large cohort of children of different ages and stages of development.
2. The correct ‘dose’ of glucose during a challenge for children is unknown.
3. Glucose metabolism varies throughout childhood and adolescents go through a period of insulin resistance going through puberty, which is transient. The significance of transient glucose intolerance during young life to later adult health is not clear.
4. While the presence of raised markers of CVD risk is well described during childhood, symptomatic CVD is extremely rare. The implications of an abnormal glucose tolerance test on either current or future cardiovascular risk is not known.
Nevertheless, the oral glucose tolerance test is used in the diagnosis of childhood diabetes where diagnostic uncertainty lies but the classification may not be clear.1–3

Historically, the diagnosis of Type 1 diabetes (T1DM) has depended on a classic clinical presentation of polyuria, polydipsia and weight loss in a child who is slim and ketotic. Yet, with the growing prevalence of obesity, more children with T1DM can be overweight at presentation and young people with T2DM may have lost a considerable amount of weight prior to diagnosis and have ketonuria. The inclusion of other laboratory analyses has been considered, including C-peptide concentrations and antibody tests. Many of these are expensive and none of them reliably distinguish one form of diabetes from another (although genetic testing is invaluable in the diagnosis of MODY). A consensus report from the International Diabetes Federation has suggested that a combination of clinical characteristics and laboratory investigations can be used to differentiate between T1DM and T2DM and hence influence treatment (Table 1).3 Often, the correct diagnosis can become more obvious with time.

### Epidemiology

Given that the correct clinical classification is difficult, it is not surprising that studies of the epidemiology of T2DM in childhood are sparse. There are few population-based prevalence studies with more being either clinic-based studies of groups of children with problems of excess weight or reviews of local or national diabetes registers.
The difficulties with studying groups of young people attending clinical services due to weight concerns are that this is likely to be an extremely non-heterogeneous, and unrepresentative, group of young people. Certainly, in secondary care, children with severe obesity are likely to be over-represented and it is well described that many people from black and minority ethnic groups, who often have the highest risk of T2DM and CVD, are less likely to access healthcare for a variety of reasons. It is, therefore, difficult to predict which child may be at greatest risk of T2DM (discussed later).

Table 2 summarizes some of the larger studies that are apparent in the literature since 2000.

**Population-based studies**

The most recent study comes from Germany where a cross-sectional survey of around 700 school leavers (mean age, 15.5 years) was performed using a fasting plasma glucose. Among this group of predominantly European students, a prevalence rate of glucose intolerance, either impaired fasting glucose (IFG), impaired glucose tolerance (IGT) or T2DM, was 2.5%. The largest study has been performed in Japan where over 7 million young people have undergone urine screening and those with persistent glycosuria underwent blood glucose testing. Over a 20-year period from 1976 to 1997, T2DM increased 10-fold in young children from 6 to 12 years of age and doubled among adolescents from 7.3 to 13.9/100 000. Yet, more recent data suggest that this tide of T2DM may be slowing down, which may be related to a decrease in the prevalence of obesity among young children in Japan. Another study of 3 million Taiwanese students has shown a striking difference between the prevalence of undiagnosed diabetes between boys 9.0-fold/100 000 and girls 15.3/100 000. A follow-up study of these students after 3 years revealed that 54% of the total had T2DM, 10% had T1DM and 20% were no longer diabetic at all. Interestingly, a study of over 1600 Turkish children showed no undiagnosed cases of diabetes. Yet, a study from North America showed a striking tendency to T2DM among ethnic minority groups. This latter observational study found that the majority of diabetes in prepubertal children was classified as T1DM. In young people aged 10–19 years, the prevalence of T2DM varied from 6% in the non-Hispanic whites to 76% among the American Indians. Finally, data from the Third National Health and Nutrition Examination Survey which included measuring glucose concentrations on almost 3000 adolescents aged 12–19 years showed that 0.4% had diabetes: 31% of these had T2DM and 1.8% had impaired fasting glycaemia.
Table 2: Studies of the epidemiology of Type 2 diabetes in childhood.

<table>
<thead>
<tr>
<th>Country</th>
<th>Study Period</th>
<th>Population</th>
<th>Age (years)</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Germany (Herder et al.⁴)</td>
<td>2007</td>
<td>721 school leavers. Fasting glucose</td>
<td>15.5 mean 2.5% IFG, IGT, T2DM</td>
<td></td>
</tr>
<tr>
<td>Japan (Urakami et al.⁶)</td>
<td>1975–2000</td>
<td>School based urine glucose screening programme</td>
<td>&lt;20 years</td>
<td>0.18% prevalence of diabetes overall. 0–9 years: &gt; 80% had Type 1 diabetes 10–19 years: 6% (non-Hispanic white) to 76% (American Indian) had Type 2 diabetes 1.96% IGT, none had Type 2 diabetes</td>
</tr>
<tr>
<td>US (SEARCH study⁹)</td>
<td>2001</td>
<td>Population-based observational six centre study</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Turkey (Uckun-Kitapci et al.⁸)</td>
<td>2004</td>
<td>Population-based study of 1647 adolescents. Fasting plasma glucose.</td>
<td>6–18 years</td>
<td>Prevalence of undiagnosed diabetes (all types) 9.0/100 000 boys and 15.3/100 000 girls 0.4% had diabetes—31% of these had Type 2 diabetes and 1.8% had IGT</td>
</tr>
<tr>
<td>Taiwan (Wei et al.⁷)</td>
<td>1999</td>
<td>3 million students: fasting blood glucose if persistent glycosuria</td>
<td></td>
<td></td>
</tr>
<tr>
<td>US (Fagot-Campagna et al.¹⁰)</td>
<td>1988–1994</td>
<td>2867 adolescents as part of third NHANES</td>
<td>12–19</td>
<td></td>
</tr>
<tr>
<td>UK (Haines et al.¹¹)</td>
<td>2004–2005</td>
<td>Monthly reporting of cases by paediatricians</td>
<td>&lt;17</td>
<td>67 cases reported</td>
</tr>
<tr>
<td>Argentina (Mazza et al.¹²)</td>
<td>2005</td>
<td>Clinic population—427 obese children</td>
<td>10.7 ± 3.5 years</td>
<td>7% IGT, 1.6% T2DM</td>
</tr>
<tr>
<td>Germany (Wabitsch et al.¹³)</td>
<td>2004</td>
<td>520 obese children from weight management clinic—OGTT</td>
<td>14.0 ± 2.0 years</td>
<td>1.5% T2DM, IFG in 3.7%, IGT in 2.1%</td>
</tr>
<tr>
<td>Australia, New South Wales (Craig et al.¹⁶)</td>
<td>2001–2006</td>
<td>Australasian Paediatric Endocrine Group NSM Diabetes Register</td>
<td>&lt;19 years</td>
<td>11% of incident cases of diabetes. Annual incidence of 2.5/100 000 person years 43 patients with T2DM with average annual increase 27%</td>
</tr>
<tr>
<td>Western Australia (McMahon et al.⁴⁹)</td>
<td>1990–2002</td>
<td>Prospectively recorded diabetes database</td>
<td>&lt;17 years</td>
<td></td>
</tr>
<tr>
<td>Austria (Rami et al.¹⁵)</td>
<td>1999–2001</td>
<td>Nationwide diabetes register</td>
<td>&lt;15</td>
<td>8 cases of T2DM. 0.25/100 000 incidence Annual prevalence 0.7/100 T1DM and 1.3/1000 T2DM. 53% of prevalent cases and 70% of incident cases 18 cases of T2DM. incidence of T1DM 1.4/100 000 vs. 0.1/100 000 T2DM</td>
</tr>
<tr>
<td>Hong Kong (Huen et al.¹⁴)</td>
<td>1984–1996</td>
<td>Register of all diabetes cases in Hong Kong</td>
<td>&lt;15</td>
<td></td>
</tr>
</tbody>
</table>
Case series

The only data apparent from the UK are the data collected through the British Paediatric Surveillance Unit (BPSU). Monthly reporting of new cases of T2DM during 2004–05 showed 67 new cases reported.\textsuperscript{11} This will only include those cases that have presented to paediatricians, and no population based data are available from the UK.

Clinic based

A number of clinicians have examined the prevalence of T2DM and IGT within their clinic populations of obese children. Most of these studies have examined only small numbers of patients who are also unlikely to be representative of the general population. Two larger studies have emerged recently. One study from Argentina of over 400 obese children showed a prevalence rate of 1.6\% for T2DM and 7\% for IGT.\textsuperscript{12} Another study from Germany of 500 obese children and adolescents showed a similar rate of impaired glucose metabolism: 1.5\% had T2DM and 2.1\% had IGT.\textsuperscript{13}

Diabetes registers

A few studies have examined the numbers of children with T2DM through their regional or national diabetes registers. From these studies it would appear that the total number of cases is quite small: 18 cases in Hong Kong from 1984 to 1996, 8 cases in Austria from 1999 to 2001.\textsuperscript{14,15} However, a recent study from New South Wales suggests that, even though the total numbers are low, this can represent a significant proportion of all children with diabetes.\textsuperscript{16} Children with T2DM represent 11\% of incident cases of diabetes now in this part of Australia.

Risk factors

As has been highlighted above, there are very few robust data looking at the prevalence of T2DM among groups of healthy children in the community and so there are no studies that have been able to provide risk factors for T2DM within communities. However, a number of studies have examined the clinical characteristics of children who have been diagnosed with T2DM by either paediatric diabetologists or by paediatricians running weight management clinics. From these studies, a number of common themes emerge.
Ethnicity

The earliest reports of children with T2DM came from studies of diabetes in Pima Indians. The great majority of children with T2DM come from an ethnic minority background. In North America, the at risk groups appear to be Hispanics and Native Americans. In 128 Australian children with T2DM, 29% were white Australian, 22% were Indigenous Australians, 22% Asian, 12% North African or Middle eastern and 10% Maori or Polynesian. In the UK, these children are predominantly from Black and South Asian backgrounds; in the recent case study report, the prevalence rates were 3.9/100 000 Black, 1.25/100 000 South Asians compared to 0.35/100 000 White.

Globally, it seems likely that the most at risk group are Indo-Asians. Indo-Asians who have migrated to western societies as well as those living in urban areas of the Indian subcontinent have a greatly higher prevalence of T2DM and CVD compared with Europeans. In the UK, the rate of CVD among Pakistani men is 60–70% higher than the general population. South Asians have a tendency to central adiposity despite similar body mass indices to Caucasians—a pattern of obesity that is strongly linked to diabetes risk. Data from Professor Yajnik’s group in Pune suggest that this process originates in childhood. Indian babies are usually lighter at birth; yet, their fat mass is preserved and there is a tendency to truncal or central adiposity even during intrauterine development. Higher umbilical cord insulin concentrations have been demonstrated, suggesting that an insulin-resistant phenotype is present at birth. By 8 years of age, these lower birth weight Indian babies have abnormalities in systolic blood pressure, fasting plasma insulin, subscapular/triceps skinfold ratios and total LDL cholesterol concentrations. Data from the UK suggest that these abnormalities also exist in South Asian children whose parents have migrated to the UK. In this study, higher mean insulin, triglyceride and fibrinogen concentrations were found in healthy 8–11-year-old children from ethnic minorities, mainly Pakistani Muslims, than matched Caucasian children.

Insulin resistance and hence diabetes risk is related to changes in adipose tissue deposition. Studies using magnetic resonance spectroscopy have found that there is an excessive hepatic fat accumulation in Caucasian and Hispanic adolescents compared with African Americans. In addition, obese Hispanic adolescents have greater intramyocellular lipid content than both Caucasians and African Americans, of comparable weight, age and gender. These data do suggest that there are striking ethnic differences in the amount of lipid accumulated in skeletal muscle and liver which occur independently of the degree of obesity.
Puberty

The majority of children with T2DM are pubertal at the time of presentation. T2DM is also more common in girls than boys. This is most likely to be due to the transient insulin resistance during puberty in which a 30% reduction in insulin action compared with prepubertal children or adults has been reported. Interestingly, some obese adolescents have been shown to have temporary deteriorations in glucose tolerance. A study from the Yale weight management clinic performed oral glucose tolerance tests (OGTTs) in 117 obese children and adolescents at presentation and then 2 years later. Thirty-three subjects had IGT at baseline. At 2 years, 8 subjects had developed T2DM and 15 subjects had reverted to normal glucose tolerance. The implications for management and follow-up of these young people during this dynamic phase of life are not clear.

Family history

The great majority of children with T2DM have a strong family history of T2DM. Recent UK data suggest that 84% of children diagnosed with T2DM have a family history. Data from a community cohort study from Mexico of over 300 adolescents showed that family history of T2DM was independently associated with hyperinsulinaemia in the offspring. The reason for this can be multifactorial. Families will share the same dietary habits as well as approaches to physical activity. However, data from adult studies suggest that defects in insulin sensitivity do also appear to be inherited.

Obesity

The recent increases in the prevalence of childhood T2DM are felt to be linked to the increase in childhood obesity. Prevalence rates have been rising and recent data from the UK show that between 1995 and 2003, the prevalence of obesity among children aged 2–10 rose from 9.9 to 13.7%. Obesity prevalence in the UK varies according to socio-demographic groups and different levels of area deprivation. Family history is also important among children with obesity: 19.8% of children living in households where both parents were either overweight or obese were themselves obese compared with 6.7% of children living in households where neither parents were overweight or obese and 8.4% of children living in households where one of the two parents was overweight or obese. The prevalence data in the UK mirror those seen across the globe: for a recent review of this see Wang et al.
increasing problem of childhood obesity, more and more children are presenting to healthcare for advice on management—some of these will already have some of the many consequences of excess weight but it can be very difficult to ascertain which children may need further investigation and medical management. Per cent body fat and central fat is related to defects in insulin-stimulated glucose uptake and reductions in lipid oxidation even in young children with short duration of obesity. The great majority, up to 85%, of children with T2DM are obese or overweight at presentation.

Criteria for investigating for T2DM

Although data are not available, the presence of excess weight gain in an adolescent from an ethnic background with a family history of T2DM is likely to present the highest risk. Recommendations from the American Diabetes Association suggest that children who are overweight (BMI, 85th percentile for age and sex) and who have any two of the following risk factors:

- Family history of T2DM in first- or second-degree relative
- Race/ethnicity (American Indian, African American, Hispanic, Asian/Pacific Islander)
- Signs of insulin resistance or conditions associated with insulin resistance (acanthosis nigricans, hypertension, dyslipidaemia, polycystic ovarian syndrome)

should be tested for T2DM. It is recommended that testing takes the form of fasting plasma glucose, commences at the age of 10 years and should be repeated every 2 years. It is not clear how long-testing should continue.

Complications

The main reason for looking for T2DM in childhood is that the available data suggest that T2DM does appear to be a ‘severe’ form of childhood diabetes (for a comprehensive review of the subject, see Pinhas-Hamiel et al. 31).

Microvascular complications

In young people with T2DM, the presence of complications at the time of diagnosis is not unusual. Studies of Pima Indians suggest that 22% of children have evidence of microalbuminuria at presentation.
In addition, the rate of progression seems to be faster than in young people with T1DM. A study from Korea comparing young people with T1DM and T2DM found that overall control, as judged by HbA1c, was inadequate in both groups.\(^{33}\) Although disease duration was shorter in children with T2DM, persistent microalbuminuria was found in 18% compared with 11% of children with T1DM. Retinopathy may also be present at the time of diagnosis. There are few studies but the available data suggest that, although the prevalence of retinopathy is lower among patients with T2DM versus those with T1DM, the disease duration is often strikingly shorter.\(^{34}\) This may reflect the delay in the diagnosis of T2DM, even in young people, as symptoms may be subtle in the early course of the disorder.

**CVD risk**

Hypertension is reported at 10–32% of adolescents with T2DM and is eight times more frequent in adolescents with T2DM than in young people with T1DM at diagnosis.\(^{35}\) A variety of lipid abnormalities have also been described. A large multicentre study from North America comparing children with T1DM (\(n = 1963\)) versus a group with T2DM (\(n = 283\)) showed that, of those with T2DM, 33% had elevated total cholesterol, 24% had elevated LDL-cholesterol, 29% had high triglyceride concentrations and 44% had low concentrations of HDL-cholesterol.\(^{36}\) Interestingly, only 1% of children were receiving any pharmacological treatment for their dyslipidaemia.

**Management**

As already mentioned, the majority of young people with T2DM will be obese at presentation. Although there are no data to suggest that there would be resolution of T2DM with weight loss, there are a few data in childhood which suggest that insulin sensitivity may be improved with weight loss through diet and physical activity. There are three main aspects to the management of T2DM in childhood: weight management, management of glucose intolerance and management of complications.

**Weight management**

It is beyond the scope of this article to summarize the effective interventions for childhood obesity. There have been a large number of
studies that have examined a wide variety of interventions in a wide variety of clinical settings. These articles have examined lifestyle changes, pharmacological interventions, as well as the very difficult issue of the place of bariatric surgery in childhood weight management. There are few data which have examined the benefit of any lifestyle modification on clinical parameters other than weight change but, given the lifelong nature of T2DM, it does seem appropriate to provide ‘healthy living’ advice to all families of children with T2DM.

Glucose management

There are very few studies which have examined interventions for management of T2DM in childhood. The most recent consensus statement from the American Diabetes Association suggests that diet and exercise should be the initial approach to treatment although they acknowledge that the majority of patients will require pharmacological intervention. They have stated that successful treatment with lifestyle would be suggested by a cessation of excessive weight gain while maintaining normal linear growth, near-normal fasting glucose values and a glycosylated haemoglobin value less than 7%. If lifestyle interventions are unsuccessful, then metformin should be tried. Only one study is apparent in the literature that has examined the effectiveness of metformin in children with T2DM. In a placebo controlled study, Jones et al. found that metformin is useful in managing childhood T2DM. There were around 40 children in each study arm. Owing to the difficulties in finding suitable numbers of patients for the study, there were some discrepancies in the two groups at baseline with respect to metabolic control: the proportion of subjects with fasting glucose concentrations >11.1 mmol/l and with glycosylated haemoglobin concentrations >8% was higher in the control group compared with the treatment group. However, those children in the treatment arm had a significant reduction in HbA1c during the 16-week study with a difference from placebo of $-1.2 \pm 0.2\%$. No serious adverse events occurred related to taking metformin although 25% of children on metformin experienced abdominal pain.

If, after a period of 3–6 months, glycaemic control is not improving, then additional therapy needs to be considered (Table 3). There are no data regarding the most appropriate management algorithm, but a number of agents have been tried including sulphonylureas, meglitinides and insulin although these obviously carry the side effects of increasing weight gain. Owing to the lack of useful data regarding diabetes management in these complicated young people, a large multicentre study is about to start sponsored by the National Institutes of
Health. The study will have three study arms: metformin alone versus metformin and rosiglitazone versus metformin and an intensive lifestyle programme. It is anticipated that 800 adolescents will be recruited and studied for between 2 and 6 years.43

The management of IGT in childhood also remains controversial. Data from adult studies suggest that lifestyle interventions using both dietary approaches as well as increased physical activity are effective in delaying the progression to T2DM.44,45 In addition, metformin has also been shown to be beneficial.44 No such data are available in paediatrics although studies are being planned which will provide information on the best management and natural history of the progression of this obesity-related complication. Until data are available, it seems reasonable to advise lifestyle changes to improve BMI in obese children with IGT.

### Management of metabolic complications

A number of children will present with other features of the metabolic syndrome such as dyslipidaemia and hypertension. One of the concerns

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**Table 3** Glycemic control measurements at baseline and last double-blind visit.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Metformin</th>
<th>Placebo</th>
<th>Difference (metformin–placebo)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline mean FPG (mmol/l)</td>
<td>9.0 ± 2.7</td>
<td>10.7 ± 2.7</td>
<td></td>
</tr>
<tr>
<td>Last double-blind visit mean FPG (mmol/l)</td>
<td>7.0 ± 2.2</td>
<td>11.5 ± 4.5</td>
<td></td>
</tr>
<tr>
<td>Adjusted mean × FPG change from baseline (mmol/l)</td>
<td>−2.4 ± 0.5</td>
<td>1.2 ± 0.5</td>
<td>−3.6 ± 0.8</td>
</tr>
<tr>
<td>95% CI</td>
<td>−3.5 to 1.3</td>
<td>0.1–2.3</td>
<td>5.1 to −2.0</td>
</tr>
<tr>
<td>( \rho ^\dagger )</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline mean HbA1c (%)</td>
<td>8.2 ± 1.3</td>
<td>8.9 ± 1.4</td>
<td></td>
</tr>
<tr>
<td>Last double-blind visit mean HbA1c (%)</td>
<td>7.2 ± 1.2</td>
<td>8.9 ± 1.6</td>
<td></td>
</tr>
<tr>
<td>Adjusted mean* HbA1c (%)</td>
<td>7.5 ± 0.2</td>
<td>8.6 ± 0.2</td>
<td>−1.2 ± 0.2</td>
</tr>
<tr>
<td>95% CI</td>
<td>(7.2–7.8)</td>
<td>8.3–9.0</td>
<td>−1.6 to −0.7</td>
</tr>
<tr>
<td>( \rho ^\ddagger )</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Mean adjusted for baseline FPG or for baseline HbA1c.
†\( \rho \) value is based on an ANCOVA, comparing metformin to placebo using baseline FPG as the covariate and treatment as the main effect.
‡Significance level \( \rho \), 0.03355, where the testwise critical value was adjusted for an 8-week interim analysis of FPG, to preserve an overall \( \alpha \) level of ≤0.05 using the O’Brien–Fleming method with an \( \alpha \) level of 0.025 at the interim analysis.
§\( \rho \)-value is based on an ANCOVA, comparing metformin to placebo using baseline HbA1c as the covariate and treatment as the main effect.

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when managing children is that there are emerging data that suggest that some of these features are not consistent findings and can disappear, or appear, over a relatively short period of time. A recent study examined the prevalence of the features of the metabolic syndrome in a group of 1000 adolescents and then re-examined these children 3 years later. The authors found that, using a variety of definitions of the metabolic syndrome including both American Heart Association and International Diabetes Federation guidelines for diagnosis of the metabolic syndrome in adults, 50% of adolescents who were initially diagnosed as having metabolic syndrome lost their diagnosis at 3 years and almost 5% developed the new diagnosis during that time. This has significant implications when considering pharmacological interventions which can have significant side effects, and the authors felt, quite reasonably, that the emphasis should be very much on lifestyle interventions in the first instance. The emphasis may need to be different for children who already have T2DM, but the data on the clinical benefits of therapeutic interventions in these young people are currently not available.

There are guidelines for management of dyslipidaemia in childhood and these have recently been revised by the American Heart Association and published in the form of a care recommendation. These guidelines are for any child with dyslipidaemia not just those with T2DM, but do suggest a stratified approach, again highlighting the importance of lifestyle changes before pharmacological treatment although the evidence for use of lipid-lowering agents in childhood dyslipidaemia is discussed in this document.

The management of hypertension is also challenging in this group of young people, many of whom are obese. It can be difficult to obtain robust measures of blood pressure in obese individuals and this has implications both for diagnosis and management. It may also seem prudent to investigate for underlying causes of hypertension in childhood before contemplating intervention. The National High Blood Pressure Education Program Working Group provides a useful guideline on the management of paediatric hypertension and provides centiles for diagnosing hypertension in childhood, which also considers the height of the child: many obese children are taller than average and so may expect to have higher blood pressure readings than children of their age who are around the middle centiles for height.

### The case for screening

Childhood obesity is reaching epidemic proportions in many parts of the developed and developing worlds. There are almost no robust
longitudinal data which can accurately define the natural history of the metabolic complications, including T2DM, of childhood obesity. As already mentioned, some studies of selected clinical groups do suggest that changes in metabolic variables may also wax and wane over time—puberty being a time of greatest metabolic turbulence. It is, therefore, difficult to know how best to manage CVD risk in childhood obesity. In addition, most paediatricians are unfamiliar with the management of these metabolic complications which have typically been restricted to adults from middle age. No one would want to increase the risk of morbidity of a child by the imprudent use of pharmaceutical agents especially in young girls who may get pregnant.

It is essential that further studies are performed to examine the optimal management of these complications and should be designed as randomized placebo controlled studies to assess the effect of time on these variables. Examining the effect of obesity management per se, with special emphasis on lifestyle approaches, on metabolic risk is essential as the lifetime risk of taking oral hypoglycaemics or statins from childhood may never be realized.

Summary

Although the total numbers of young people with T2DM do not seem great from these studies, the prevalence does appear to be increasing. We still have little perspective on the potential scale of the problem and a view to the future particularly in light of the emerging data on diabetes-related complications. Further population-based studies are needed, which will examine the prevalence and risk factors for T2DM among children and adolescents. With this information, it may be possible to provide more targeted diabetes prevention programmes to those most at risk.

References


