A global epidemic of paediatric obesity occurred in recent years, and prevalence of obesity is continuing to rise. In the developed world obesity is now the most common disease of childhood and adolescence. Paediatric obesity is not a cosmetic issue, being associated with a significant burden of ill health both for obese children and for adults who were obese as children. Health professionals tend to underestimate the impact of paediatric obesity, and lack the skills, knowledge, and time to treat it effectively. This short review aims to summarise recent systematic reviews on the origins, consequences, prevention, and treatment of paediatric obesity.

DEFINITIONS AND DIAGNOSIS

Obesity is an excess of body fat: a level of body fat that is harmful. This “definition” is expressed in terms of body fat content, but in clinical practice and epidemiology body fat content can rarely be measured with acceptable precision and accuracy. So simpler alternative definitions are required. There is a good deal of evidence that subjective assessment of obesity is inadequate, and that of the objective approaches the body mass index (BMI; weight in kg divided by height^2 in m^2) is the best available option. The basis for a definition (in epidemiology) or diagnosis (in clinical practice) is therefore the BMI, but as the BMI changes with age and differs between the sexes, it must be interpreted using centile charts that describe population reference data (figs 1 and 2), or by calculation of a standard deviation or (Z score or SD score) relative to population reference data.

Children and adolescents with a high BMI centile (such as BMI >98th centile on the UK 1990 charts) are highly likely to be excessively fat—that is, this obesity definition has high diagnostic specificity (low false positive rate). Such definitions have a moderate sensitivity—that is, modest false negative rate. Such definitions are also clinically meaningful: they identify children and adolescents at high risk of the comorbidities of obesity.

In the UK, the best evidence suggests that overweight and obesity should be defined as BMI >85th and >95th centiles in research and epidemiology. In clinical practice the UK charts only provide 91st and 98th centiles and for pragmatic reasons these should be the basis of our definitions of overweight and obesity respectively. A number of other countries now have BMI population reference data and centile charts. Where such “national” reference data are available, they should be used to define/diagnose overweight and obesity. Where national data are unavailable there is a choice of using either reference data/charts from another nation (such as the USA whose charts are available from the Centers for Disease Control and Prevention) or use of newer “international” definitions of overweight and obesity.

The international approach attempts to link adult BMI based definitions of overweight (BMI>25) and obesity (BMI>30) to paediatric definitions by providing age and sex specific “equivalent” BMIs in childhood and adolescence. For ease of international comparisons these international definitions are practical. However, four recent studies have compared the diagnostic ability of the international definitions with that of the more traditional national definitions based on national BMI centiles.

In all four cases diagnostic ability of the national approach was greatly superior, and so use of the international approach should be considered with caution.

The scale of the obesity epidemic is also heavily dependent on the definition used. Using data from the same survey, prevalence of obesity can vary twofold to sevenfold depending on whether national reference data or the international approach is taken. Further discussion on the merits of national compared with international approaches is beyond the scope of this
review, but detailed arguments can be found elsewhere.21-22

PREVALENCE

As prevalence of obesity depends heavily on the definition used and on a number of other factors (for example, the age and representativeness of the sample, or survey, the timing of the study or survey, whether or not height and weight are measured or self-reported), summarising recent prevalence estimates around the world is problematic. Nevertheless, in almost all countries to date that have reported on prevalence it is continuing to increase, and dramatic increases have been typical, showing rapid environmental and lifestyle changes in recent years (fig 3). For the developed world, estimates from 2000 onward using national definitions (such as BMI ≥95th centile) suggest that 10%–23% of the paediatric population was obese.19-20 23-32 In England in 2003 for example, 28% of 2–11 year olds were overweight or obese (BMI ≥85th centile), up from 22% in 1995, while 14% were obese (BMI ≥95th centile), up from 10% in 1995.33

In the developing world, prevalence of overweight and obesity has also increased dramatically in recent years.34-37 A few exceptions have been described, notably in sub-Saharan Africa and much of the former Soviet Union (where extreme economic hardship has limited the epidemic). Using the international definitions at least 10% of school age children are overweight or obese worldwide,4 and this is highest in the Americas (32%), then Europe (20%), and the Middle East (16%).

In the developed world prevalence of paediatric obesity is generally as common in boys as girls. The picture with respect to sex differences in prevalence in the developing world is more complex and difficult to predict.4-7 In the developed world paediatric obesity is generally more common in children and adolescents from families of lower socioeconomic status,4 11 but the magnitude of these socioeconomic differences in prevalence is quite limited. In the developing world the picture is again more complex: higher socioeconomic status has usually been associated with higher risk of paediatric obesity, although as the epidemic progresses in the developing world lower socioeconomic status may become more of a risk for obesity.6 7

In the USA youth prevalence of obesity is much higher in some ethnic minorities than in the general population.7 Evidence from outside the USA is limited at present but suggests that some ethnic minority groups may also be at higher risk. Again, reasons for differences are complex and not entirely clear at present.

HAS THE SCALE OF THE OBESITY EPIDEMIC BEEN UNDERESTIMATED?

Secular trends in BMI may underestimate the scale of the epidemic. Secular trends in fat distribution (as shown by changes in waist circumference) in England have been even more pronounced than secular trends in BMI,38 showing a more intra-abdominal or central pattern of fat distribution in modern children. This evidence also suggests that the cardiovascular and metabolic consequences of the epidemic may have been underestimated using trend data obtained from BMI. In addition, in adults the BMI as a definition of obesity suffers from the weakness that it tends to under-diagnose obesity in non-white populations.39 40 Evidence from paediatric studies is limited at present but it seems probable that prevalence of obesity in South Asian, Chinese, and possibly Hispanic and African children will be underestimated if criteria based on white populations are applied to them without any attempt to take account of these real biological differences between populations.21 Certain populations seem to have, for any given BMI, higher body fat content and a more central fat distribution (the pattern of fat distribution most likely to be associated with cardiovascular and metabolic derangements).35 36 In addition, these same populations may be at higher risk of some of the comorbidities of obesity, notably diabetes.

CONSEQUENCES—WHY DOES PAEDIATRIC OBESITY MATTER?

A detailed description of the many and varied adverse consequences of paediatric obesity would be beyond the scope of this review. A recent systematic review of evidence from the developed world concluded that paediatric obesity was an important health burden in childhood/adolescence and in adulthood. Box 1 gives a summary of the principal health and other adverse effects. In the developed world the most widespread adverse effects in childhood are probably psychosocial.13 From a public health perspective the important concern is the impact of child and adolescent obesity on cardiovascular and metabolic health.16-18 It is becoming increasingly clear that the obesity epidemic is likely to herald an important public health burden in future from diabetes and cardiovascular disease,16-18 and evidence on its economic impact is also emerging.37 38 In developing countries the combination of fetal undernutrition followed by neonatal infant overnutrition seems to be particularly likely to be associated with later obesity and metabolic/cardiovascular morbidity.39-40

PERCEPTIONS OF OBESITY AMONG HEALTH PROFESSIONALS AND FAMILIES

Despite the large body of high quality evidence that paediatric obesity does matter in both the short and long term, there is also a great deal of evidence, summarised by Baur,41 that it is not perceived as being clinically important by many health professionals, by patients, and by their families (parents). In addition, among those families who are concerned about obesity in their children, the failure of health professionals to treat obesity seriously is probably a common cause of tension.42 There is commonly a large gap between the hard evidence and public perceptions of paediatric obesity. Bridging this gap is likely to be necessary if clinical management is to be effective, and if clinical and public health responses to the epidemic are going to succeed. This will require greater efforts in medical education, and education of the wider population as to the impact of paediatric obesity.

CAUSES OF THE PAEDIATRIC OBESITY EPIDEMIC

In its simplest terms obesity is caused by a chronic positive energy balance—that is, an excess of energy (food) input over output. The magnitude or rate of this energy imbalance can be surprisingly small (<100 kcal/day in many cases), but must be sustained for a long period for a non-obese child to become obese. Researchers have made a number of attempts to test whether obesity is caused by low energy expenditure (low physical activity) or high energy intake. Most of these attempts at physiological explanations for the epidemic of paediatric obesity have been inconclusive, for several reasons. “Pre-obese” people are not readily identifiable, yet are crucial to understanding the development of obesity. The small degree of daily energy imbalance required to produce obesity may preclude identification of abnormalities in either energy intake or limited expenditure that cause susceptibility to obesity, because of the limited accuracy and precision of measurement of these variables.43 44 Measurement of dietary energy intake in particular is imprecise and prone to biases, particularly in older children and adolescents where substantial underreporting of energy (food) intake is common.45 46 Measurement of total energy expenditure with the doubly labelled water method is accurate and precise.47
However, it is also expensive, and the availability of the isotope $^{18}$O necessary for doubly labelled water measurements has been limited. Studies to date that have tested the hypothesis that a low total energy expenditure predisposes children and adolescents to obesity or excess fat gain have therefore been few in number, rather small, and inconclusive.11

It is increasingly being appreciated that the paradigm of identifying an energy imbalance (in intake or expenditure) as a "cause" of obesity might be limited.43 45 The underlying causes of energy imbalance—that is, behaviours or risk factors, may not only be more readily measurable than energy intake and expenditure, but may also be more useful in that they represent possible behavioural targets for any future preventive interventions.45

One historical difficulty with this alternative—more epidemiological—approach to studying the causes of obesity has been that systematic reviews have reported that older epidemiological studies of "risk factors" for childhood and adolescent obesity were usually flawed.46 Many of these older

---

Figure 1  Body mass index centile charts for UK boys, for diagnosis and monitoring of overweight and obesity. (Available from Child Growth Foundation, 2 Mayfield Avenue, London W4 1PW; or from Harlow Printing (http://www.harlowprinting.co.uk).)
epidemiological studies were underpowered and had serious limitations in design (many cross sectional studies that used simple univariable analysis). More recent epidemiological studies have been more likely to identify causes of obesity because they have been larger and have adopted longitudinal study designs with multivariable analysis (for example, to control for the confounding effects of socioeconomic status). These recent studies have identified a surprisingly large number of potential risk factors for the development of paediatric obesity, reviewed elsewhere.

From these candidate risk factors for obesity we can apply "decision rules" to identify which are the most promising targets for interventions aimed at paediatric obesity prevention. Whitaker has suggested that candidate behaviours should meet certain criteria before they are selected as the basis of interventions. Firstly, the behaviour(s) targeted should be causally related to the development of obesity—this requires reasonable evidence from energy balance studies or epidemiology, combined with biological plausibility. Secondly, the intervention should do
Box 1 Principal consequences of paediatric obesity*

<table>
<thead>
<tr>
<th>In childhood and adolescence</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Psychological ill health</td>
<td></td>
</tr>
<tr>
<td>Cardiovascular risk factors</td>
<td></td>
</tr>
<tr>
<td>Asthma</td>
<td></td>
</tr>
<tr>
<td>Chronic inflammation</td>
<td></td>
</tr>
<tr>
<td>Diabetes (probably type I and II)</td>
<td></td>
</tr>
<tr>
<td>Orthopaedic abnormalities</td>
<td></td>
</tr>
<tr>
<td>Liver disease</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>In adulthood</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Persistence of obesity</td>
<td></td>
</tr>
<tr>
<td>Cardiovascular risk factors</td>
<td></td>
</tr>
<tr>
<td>Adverse socioeconomic outcomes†</td>
<td></td>
</tr>
<tr>
<td>Premature mortality</td>
<td></td>
</tr>
</tbody>
</table>

*From systematic review and critical appraisal.†Long term social and economic disadvantage pronounced in women, less obvious in men.

Of all the candidate behaviours or risk factors those that are most likely to meet these Whitaker criteria at present are as follows: formula feeding during infancy (breast feeding provides modest protection against later obesity40–51); consumption of sugar sweetened drinks (energy consumed in dissolved sugar may not be adequately "recognised" and therefore compensated for by reductions in later energy intake); excessive television viewing (which may either reduce energy expenditure or increase energy intake, or both52–55); low physical activity.54–55 These four behaviours should therefore be regarded as the highest priorities for interventions aimed at prevention of paediatric obesity.

EVIDENCE ON PREVENTIVE INTERVENTIONS

The evidence on interventions aimed at preventing paediatric obesity has been reviewed systematically by a number of authors40–44 in recent years. The conclusions of these reviews have been remarkably consistent, in part because of the use of objective methods for literature searching and assessing study quality, and in part because of the limited number of high quality, medium-long term, randomised controlled trials (RCTs). Most RCTs to date have been of limited quality methodologically, short term (often lasting six months or less, which probably biases outcomes in favour of the intervention), and many have tested interventions that are sufficiently complex or specialised that they are unlikely to be generalisable.40–44 A further concern is that even fewer of the RCTs to date have reported on successful interventions.

To date the only high quality RCT that has tested an intervention that is likely to be successful and generalisable is the Planet Health intervention (box 2) in schools in the Boston area of the USA.52 This comparatively large trial (1295 participants, mean age 11 at study entry) used a complex intervention over two school years. The intervention consisted of changes to the school curriculum (such as improved physical education), changes to school meal provision, targeted reductions in television viewing, and promotion of walking to/from school.52 The intervention was successful (in girls, not in boys) in that risk of becoming obese was significantly reduced (adjusted odds ratio, AOR 0.47, 95% CI 0.24 to 0.93), and there was a significant remission of existing obesity in those who were obese at the start of the trial (AOR 2.16, 95% CI 1.07 to 4.35). The benefits of the intervention were attributable largely to reductions in television viewing.52 Reduced television viewing is one of our most promising strategies for obesity prevention because it is a behaviour that seems to be fairly modifiable, and because reduced time spent watching television probably reduces food intake and/or increases energy expenditure modestly (by increasing physical activity).52–55

The Planet Health trial is a model of obesity prevention intervention, and is particularly promising because its authors have subsequently tackled issues of generalisability and sustainability of the intervention (for example, by producing a handbook for schools that describes how to implement a Planet Health approach into school curricula). The Planet Health investigators also have promising data on the economic benefits of the intervention.55 Nevertheless, the Planet Health approach may not be generalisable to all settings and there is an urgent need to assess the success of other interventions in other populations. Despite the scale and impact of the obesity epidemic there are in fact comparatively few preventive interventions being

Box 2 Planet Health: a school based intervention for obesity prevention*

<table>
<thead>
<tr>
<th>The intervention</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Modified school meals</td>
<td></td>
</tr>
<tr>
<td>Increased walking to/from school</td>
<td></td>
</tr>
<tr>
<td>Reduced television viewing</td>
<td></td>
</tr>
<tr>
<td>Changed physical education</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Outcomes</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Reduced obesity risk†</td>
<td></td>
</tr>
<tr>
<td>Curriculum modifications sustainable and cost effective</td>
<td></td>
</tr>
<tr>
<td>Curriculum modifications generalisable</td>
<td></td>
</tr>
</tbody>
</table>

*For further information see Gortmaker et al.52 †In girls, not boys.52
tested in RCT, and most of these have focused on minority
groups of adolescents in the USA. For other population
groups in the developed world (for example, young children)
there is an even greater dearth of evidence. There are few if
any trials aimed at testing preventive interventions underway
in the developing world and this is another important
research gap that must be tackled urgently.

It has been argued that one of the reasons for the failure of
many previous preventive interventions is that by targeting
behaviour modification at the “micro” level (that is, individual
children, their families, or schools) they are unable to have an impact on the many other influences on
weight status that determine the environment at the
“macro” level, such as economic or transport policies. Successful obesity prevention may therefore require a more
macro-environmental approach (in addition to or as an
alternative to the micro level behavioural changes that have
been tested in interventions to date). Taking such an
approach would require political/economic action on a scale
that has not been attempted by governments yet, to “detoxify” the wider “obesogenic environment”. Some
governments may be at least considering actions of this
kind. For example, the recent UK Parliament Health Select
Committee Inquiry into Obesity listed a large number of
political and economic options for tackling the obesity
epidemic that would operate at the macro or societal level.

There is little doubt that governmental and policy issues
such as transport and food pricing have had an important
impact on the obesity epidemic. The evidence from the
developing world has shown that there are a variety of
features of societies in economic or nutritional “transition”
that commonly underpin dramatic increases in obesity
prevalence, and these are discussed in detail elsewhere. Even in the developed world apparently basic economic and
political decisions such as regulations on food pricing may
have been important “drivers” of the paediatric obesity
epidemic.

PREVENTION OR TREATMENT?
The adage that “prevention is better than cure” is commonly
repeated, but it is worth examining the implications of this
statement for childhood obesity prevention and manage-
ment. Large—and increasing—numbers of obese children and
adolescents suffering from the comorbidities of obesity are
now seeking treatment across the world. Given the high
prevalence and adverse consequences of paediatric obesity it
would seem inappropriate not to attempt to treat it. In
addition, treatment of those children and adolescents who
are already obese is a form of secondary prevention, because
successful treatment can prevent adult obesity. In the
modern world most preadolescent obese children will remain
obese (and so become obese adults), and at least 70% of
obese adolescents will remain obese into adulthood. This
evidence is an argument for greater efforts at both
prevention and treatment.

From a public health perspective obesity in itself may be
less of a problem than the pervasive nature of some of the
health behaviours that cause it. For example, high levels of
sedentary behaviour and low levels of habitual physical
activity may be typical of modern children, and these
behaviours may be established at an early age, well before
school entry. This provides an argument for the promotion of
lifestyle modification across the paediatric population, not
just in those currently defined as overweight and obese. How
this might be achieved is unclear and not without difficulties.

EVIDENCE ON TREATMENT INTERVENTIONS
The evidence on interventions aimed at treating childhood
and adolescent obesity has been reviewed systematically in
recent years, and critically appraised, by a number of
authors. Many of the weaknesses in the evidence on
prevention, listed above, are common to the obesity treat-
ment literature. There have been comparatively few treat-
ment RCTs to date, most have been rated as being of low
methodological quality and so prone to bias, most have been
comparatively short term (biasing the trial in favour of
intervention), and most have tested interventions that are
unlikely to be generalisable. However, the literature on
treatment has provided some guidance as to how to treat
paediatric obesity (box 3), and some “best bets” in treatment
are clear from the treatment RCTs and expert committee/
consensus reports. The most promising results have been
obtained using the pioneering multidisciplinary and intensive
treatment approach of Epstein, and making this approach
more generalisable is a promising strategy for treatment.

Treatment should probably be limited to motivated
families, where the child (and preferably the family) perceive
the child’s obesity as a problem, and seem to be motivated to
attempt lifestyle changes. The development of obesity is the
result of a long term positive energy balance and treating it
requires maintenance of a zero or slightly negative energy
balance over a long period (months or years). From a
theoretical point of view our treatments should probably
continue for longer than is traditionally the case, and should
entail more frequent and longer consultations with patients,
and these recommendations are backed by some empirical
evidence. In children (although possibly not in adoles-
cents), treatment success is most probable if the approach
taken is to treat the whole family, rather than just focusing
treatment on the obese child. Treatment must entail dietary
changes, and the “traffic light” approach of Epstein, or
modified versions of it seem to be practical and promising
dietary modification strategies. In brief, this entails children
and their families being taught to group foods into three
categories: red (high energy density foods, for strictly limited
consumption), amber (medium energy density foods, for
limited consumption, for example, to mealtimes only), and
green (foods of low energy density, to be consumed freely as
substitutes for red and amber foods).

Treatment should not focus solely on diet, and must aim to
reduce sedentary behaviour (particularly television viewing),
and/or increase physical activity. Targeting a reduction in
sedentary behaviour, perhaps by introducing limits to
television viewing time such as two hours/day (including
media use such as computer games and internet use), is one of
the most promising elements of treatment. As with
obesity prevention interventions, reductions in television
viewing may be associated with reductions in energy intake
and/or with modest increases in physical activity and energy
expenditure. Directly promoting increases in physical activity
may also be helpful, although it is unclear whether the focus
should be on promoting structured aerobic exercise and/or

---

**Box 3 Treatment guidance**

- Treat motivated patients and families
- Modify diet, perhaps using a regimen such as the “traffic light diet”
- Encourage reduced television viewing and media use
- Encourage increased physical activity
- Treat the family, not the child
- Encourage families to self monitor their lifestyle
- Provide more time for consultations with families, and more consultations

www.postgradmedj.com

---

*www.postgradmedj.com*
lifestyle physical activity. Obese children and adolescents generally have limited exercise tolerance and may be more amenable to increases in lifestyle physical activity (such as walking to school).

Recent systematic reviews have concluded that several alternative approaches to treatment, which have been the source of great interest, (specific dietary modifications, for example, modifying the glycaemic load of the diet; residential treatment; pharmacotherapy; surgery) are not currently based on high quality evidence. Again, there is a need for more high quality and long term RCTs to test the evidence for these approaches. The most recent evidence suggests that many of these newer approaches are promising. For example, glycaemic load modification holds out hope of changing appetite regulation and may eventually be a highly practical dietary strategy; residential treatments can produce very favourable outcomes at least in the short-medium term (including noticeable improvements in self esteem and psychosocial wellbeing); recent trials of pharmacotherapy in severely obese adolescents suggest that as an adjunct to lifestyle modification this may be beneficial; surgery is promising for the more severely obese adolescents and/or those with serious comorbidity. Given this high degree of promise and the scale of the obesity epidemic it is a matter of some urgency that these trials are funded and carried out.

Audits of traditional dietetic and paediatric approaches to treatment of paediatric obesity are generally disappointing. For example, in a five year audit of 254 patients at the Sick Children’s Hospital in Edinburgh, mostly referred from primary care, 52% of the patients failed to attend any of the appointments made for them. In the remaining 48% of patients who attended at least one of the three appointments made, weight maintenance over six months (an aim of management) was achieved in only 22% (equivalent to about 12% of all those referred to the clinic). Audit results like these are probably not specific to particular clinic, but reflect a wider failure of treatment. They have been interpreted as being unsupportive of any efforts at treatment, but it may be that current treatment approaches are inadequate. Traditional approaches to treatment are probably not sufficiently evidence based, may be insufficiently client centred (see below), and/or insufficiently intense.

A degree of resistance to lifestyle changes must be expected even from patients who seem motivated to change lifestyle. Making and sustaining lifestyle changes is extremely difficult and is usually without any short term benefit to children or their families. Analogies to obesity treatment can be made to other chronic diseases of childhood such as cystic fibrosis.

Key points
- Obesity is now extremely prevalent in children and adolescents across the globe, and prevalence is still increasing.
- Paediatric obesity is best diagnosed using the BMI centile (using centile charts) or standard deviation score.
- Obesity has serious adverse consequences, both for the obese child and for the adult who was obese as a child.
- Most health professionals are unaware of the clinical consequences of paediatric obesity.
- Evidence on the prevention and treatment of paediatric obesity is limited, but useful guidance on best evidence is now widely available.

Novel “behavioural” approaches to management of cystic fibrosis are more promising in achieving adherence to treatment than more traditional medical/dietary management, and these may provide useful lessons for treatment of other chronic childhood diseases such as obesity.

EVIDENCE ON MANAGEMENT STRATEGIES
Recent systematic reviews and critical appraisal exercise have concluded that the evidence on approaches to management is limited, consisting largely of expert committee and consensus reports. Nevertheless, these reports provide very useful guidance on basic issues such as: who to treat; when to refer from primary care; what treatment should aim for.

Treatment should be reserved for families with obese patients (defined objectively using the BMI centile or Z score as described above) who seem to be motivated to change their lifestyle.

Referral from primary care should be considered when there is a possibility that there may be an underlying pathological cause of obesity, for example an endocrine cause or genetic syndrome. Such causes of obesity are extremely rare and in the vast majority of paediatric patients the origin of their obesity will lie in their lifestyle. These patients referral to secondary care will therefore entail ruling out any underlying pathology. An underlying pathological cause should be suspected in very young (pre-school) children who are severely obese (this may suggest an underlying genetic cause such as monogenic obesity), and in obese children who are short for their age (most obese children are comparatively tall and short stature may suggest a syndromic cause). Referral from primary care will also be necessary where a comorbid condition requires investigation or management (for example, hypertension, dyslipidaemia, metabolic syndrome, sleep apnoea, liver disease, type 2 diabetes).

The principal aim of treatment should be, for most patients, maintenance (not loss) of body weight. The underlying aim of treatment is sustainable lifestyle change: permanent changes to diet, sedentary behaviour, and physical activity. Maintenance of weight is difficult to achieve in the long term but is complemented by the fact that, so long as they are still growing, patients will “grow into their weight” to a degree. For more severely obese patients, and patients where comorbidities may be improved by weight loss (for example, patients with sleep apnoea or type 2 diabetes),

Key references
then modest weight loss (usually no more than 0.5 kg/month) should be the aim, and a more intensive approach to treatment would be justified. 

It is possible that greater clinical and public health efforts should be directed at children and adolescents who are overweight rather than obese (for example, those above the 85th but below the 95th centile for BMI), but the evidence on treatment strategies for such patients is even more limited. At present most advice is simply to monitor such children as they are probably at high risk of progression to obesity. 

CONCLUSIONS
Paediatric obesity has increased considerably in prevalence across much of the world in recent years and prevalence continues to increase. Obesity in childhood and adolescence is not a cosmetic issue, but has important impacts on short and long term health that are not widely appreciated by patients, their families, or health professionals. The evidence base on strategies for prevention and treatment is limited at present, and is virtually non-existent for the developing world. There is an urgent need for greater research on improved approaches to prevention and treatment across the world. Clinical and public health responses to the epidemic have been overtaken by the speed and scale of the increase in obesity, and the recent emergence of evidence on co-morbidities. Large scale societal actions aimed at making the environment less “obesogenic” are probably necessary if the projected increases in prevalence of paediatric obesity are to be avoided. Despite weaknesses in the evidence base, useful guidance on prevention and treatment are now widely available that are based on systematic review and critical appraisal. Some existing treatment and prevention strategies are promising models on which to build future responses to this important crisis in public health.

MUL TIPLE CHOICE QUESTIONS (TRUE (T)/FALSE (F); ANSWERS AT THE END OF THE REFERENCES)
1. Obesity should be diagnosed by
   (A) “Eyeballing” the child
   (B) Measuring body weight
   (C) Comparing weight and height
   (D) Calculating BMI and plotting on a centile chart
   (E) Measuring waist circumference

2. The following are recognised complications of paediatric obesity
   (A) Liver disease
   (B) Asthma
   (C) Constipation
   (D) Insulin resistance
   (E) Chronic inflammation

3. Obesity in children in the developed world is
   (A) More common in girls than boys.
   (B) More common in boys than girls.
   (C) More common in families of lower socioeconomic status.
   (D) More common in some ethnic minority groups.
   (E) More common in children of obese parents.

4. The following behaviours are well established risk factors for childhood obesity
   (A) Rapid growth in infancy
   (B) Formula feeding in infancy
   (C) Television viewing in childhood
   (D) Low fruit and vegetable consumption in childhood
   (E) High soft drink consumption in childhood

5. Treatment of obese children should usually aim to
   (A) Achieve maintenance of body weight
   (B) Achieve loss of body weight
   (C) Change diet of patients
   (D) Change television viewing of patients
   (E) Focus solely on the patient, not the family

6. Obese patients should be referred from primary care when
   (A) They are severely obese and pre-school age
   (B) They are short for their age
   (C) They are tall for their age
   (D) A comorbid condition is suspected
   (E) A pathological cause is suspected

7. What percentage of obese adolescents will become obese adults?
   (A) More than 90%
   (B) Less than 10%
   (C) 30%–50%
   (D) 70%–80%
   (E) 20%–30%

Funding: none. Conflicts of interest: none declared.

REFERENCES