

The Bristol Online Obesity Screening Tool: experience of using a screening tool for assessing obese children in primary care

Sarah E. Owen¹, Deborah J. Sharp² and Julian P.H. Shield³

¹Academic GP Registrar, Academic Unit of Primary Health Care, School of Social and Community Medicine, University of Bristol, Bristol, UK

²Professor of Primary Health Care, Academic Unit of Primary Health Care, School of Social and Community Medicine, University of Bristol, Bristol, UK

³Professor of Diabetes and Metabolic Endocrinology, Bristol Royal Hospital for Children, School of Clinical Sciences, University of Bristol, Bristol, UK

The incidence of childhood obesity is rising in the United Kingdom and this has far-reaching and serious consequences both for the physical and psychological well-being of the child, as well as significant financial implications for the health service. General practitioners (GPs) play a central role in identifying and assessing such children and directing them to the best services. While most cases of obesity are simply due to an imbalance in calorie intake and expenditure, children do need to be formally assessed to ensure that red flags are not missed, which might signify an important underlying aetiology, co-morbidity or complication. To date, there have not been tools available to guide a GP through this assessment. In this paper, we present and explain the thinking behind a tool, which was developed for use by GPs from Bristol as part of a trial to assess the transferability of a childhood obesity clinic into primary care. We look at the evidence base behind the guidelines and then assess the appropriateness and safety of the 152 referrals made using this tool. We believe that this screening tool would enable over 85% of obese children to seek their initial weight management in primary care. Additional evaluation is needed in different regions to ensure effectiveness, sensitivity and specificity of this new tool.

Key words: childhood obesity; primary care

Received 24 December 2010; accepted 17 March 2011; first published online 22 June 2011

Introduction

A total of 25% of 10–11-year-old children in England are overweight or obese (Dinsdale and Ridler, 2010). This has far-reaching physical and psychological consequences for the child (Rudolf, 2004; Sabin *et al.*, 2006), as well as financial implications for the National Health Service (NHS) and society in the future (Foresight, 2007).

While primary care has often been identified as a potential setting for managing simple childhood obesity (National Institute for Health and Clinical Excellence (NICE), 2006; Scottish Intercollegiate Guidelines Network (SIGN), 2010), there is a lack of evidence-based guidelines to help general practitioners (GPs) assess the obese child.

The ‘Bristol Obesity Online Screening Tool’ (BOOST – see Appendix 1) is an evidence-based tool that was developed in 2006 for use in a National Institute for Health Research (NIHR)-funded pilot trial ‘Evaluating the transferability of a successful, hospital-based, childhood obesity clinic to primary care: a pilot study (Hamilton-Shield, 2006). It was

Correspondence to: Dr Sarah E. Owen MA (Cantab), MBBS MRCP, GP Retainer, Pembroke Road Surgery, 111 Pembroke Road, Bristol BS8, UK. Email: Sehills76@aol.com

designed for GPs to facilitate their assessment of obese children and to ensure that 'red flags' were not missed. These 'red flags' were to highlight either secondary causes of obesity or serious comorbidities, necessitating specialist referral as opposed to recruitment into a practice nurse-led, primary care obesity clinic. It was completed in electronic format in GP surgeries and then downloaded for forwarding to the consultant paediatrician.

This paper reviews the evidence base to these guidelines and then assesses the appropriateness and safety of the 152 GP referrals made using BOOST during the care of childhood obesity (COCO) trial.

Evidence base and development of BOOST

BOOST provides a link to the 'Health for all Children' website (see Health for All Children, 2004), which gives clinicians a tool to calculate the child's body mass index (BMI) percentile, specific for age and sex. Childhood obesity, unlike in adults, cannot be based simply on a BMI score, as body composition varies between girls and boys and changes during childhood with respect to the proportion of fat and lean body mass (SIGN, 2010). The 1990 Body Mass Index Reference Curves for the UK (Cole *et al.*, 1995) have therefore been traditionally used to define childhood obesity (Shield and Summerbell, 2009; SIGN, 2010). Research studies tend to define obesity as above the 95th percentile as does the National Child Measurement Programme, whereas routine clinical situations recommend the 98th percentile, as used in our study (Rudolf, 2004; NICE, 2006; SIGN, 2010). This is the level at which screening for co-morbidities or a secondary cause of obesity is recommended.

While the majority of childhood obesity can be ascribed to an imbalance between energy intake and expenditure (SIGN, 2010), the questions posed by BOOST aim to systematically review the child in order to exclude 'red flags'.

Rare, but important, genetic causes of obesity exist (Shield and Summerbell, 2009; Bochukova *et al.*, 2010), including monogenic disorders such as melanocortin-4 receptor, leptin and its receptor gene mutations affecting the neuroendocrine regulation of satiety and eating. In significantly obese

childhood cohorts, these mutations may account for up to 6% of cases (Farooqi and O'Rahilly, 2006). In addition, there are multiple syndromic causes, the most familiar being the Prader-Willi syndrome (incidence estimated at 1:52 000; Whittington *et al.*, 2001). Recently, chromosomal copy number variants have been identified in association with profound obesity and autism (Bochukova *et al.*, 2010). Dysmorphic features, learning difficulties or significant sensory or motor developmental delay should alert clinicians to the possibility of these underlying conditions, which are likely to be more frequently diagnosed as our understanding of childhood obesity improves.

BOOST screens for endocrine causes of obesity, including hypothyroidism, Cushing's syndrome and growth hormone deficiency. These children tend to present with a weight that is disproportionate to their height. Other features and examination findings that might highlight these conditions include striae, truncal obesity and a general change in appearance (Cushing's syndrome) and either delayed or precocious puberty, alerting a clinician to a number of possibilities including underlying hypothalamo-pituitary, adrenal, chromosomal or central nervous system abnormalities. Overweight and obese children-initial assessment in primary care. Map of Medicine http://eng.mapofmedicine.com/evidence/map/obesity_in_children1.html.

BOOST provides a web link to the Tanner definitions regarding pubertal development. While this specific link is no longer available, there are many alternative online resources available (eg, for boys <http://bjsm.bmj.com/content/44/7/476/F5.large.jpg> and for girls <http://bjsm.bmj.com/content/44/7/476/F4.large.jpg>) and BOOST provides a summary reference table regarding pubertal stages. Assessing puberty status can be a relatively subjective process, particularly in boys who lack the easily defined menarche stage. This is further complicated by the suggestion that increased adiposity might be linked to earlier onset of puberty (Lynn Ahmed *et al.*, 2009). However, if a clinician had any suspicion of abnormal pubertal development, we recommended referral to secondary care.

As the incidence of childhood obesity rises, so does the incidence of type 2 diabetes, suggesting a causal relationship (Alberti *et al.*, 2004). BOOST screens for type 2 diabetes using both a urine dip as well as posing questions relating to possible symptoms and a family history. This multifactorial

approach reflects findings from a Bristol study of 126 obese children, which identified that children with impaired glucose tolerance were more likely to have a parental history of type 2 diabetes (Sabin *et al.*, 2006).

BOOST seeks to highlight obesity co-morbidities warranting a specialist referral. These include sleep apnoea and benign intracranial hypertension. Benign intracranial hypertension has an incidence up to 19/100 000/year in the high-risk group of obese women in the reproductive age range (Acheson, 2006), while identification in children and teenagers is less common. Interestingly, while in the adult population they tend to be female and obese, in children the relationship is less clear. One meta-analysis observes that in younger children the sex distribution is equal without any relationship to obesity. Adolescents showed the more typical pattern – with a propensity towards sufferers being overweight females (Genizi *et al.*, 2007).

Adolescent children, similarly to adults, show an increased risk of having obstructive sleep apnoea syndrome with increasing weight (Kohler *et al.*, 2009). Furthermore, at any level of sleep apnoea, obese children are more likely to experience excessive daytime sleepiness as compared with non-obese children (Gozal and Kheirandish-Gozal, 2009). This has obvious consequences for the physical, cognitive and mental well-being of the teenager.

BOOST also screens for overt eating disorders, which would need specialist review with the child and adolescent psychiatric team before an obesity clinic intervention. Similarly, obesity suspected to be secondary to iatrogenic causes such as anti-convulsants needs to be discussed with relevant specialists, to see whether adjustments to medication can be made. For example, unlike many of the other anti-convulsants used in childhood, topiramate has a weight-reducing effect in many patients (Sanker, 2004).

The only examinations required by BOOST are the urine dipstick analysis and a blood pressure measurement. Completed questionnaires were sent to a specialist paediatrician who used the age- and sex-specific blood pressure reference charts to determine whether there were concerns about hypertensive disease. It is known that obese children are likely to have higher levels of both systolic and diastolic blood pressure (Ribeiro *et al.*, 2003), but those with true hypertensive levels need to be referred to secondary care.

Assessment of referrals made using BOOST

Subjects and methods

Study recruitment was undertaken in Bristol between April 2008 and June 2009. A total of 152 referrals were made to the study from 50 general practices across the greater Bristol area using BOOST. The target children were those aged five to sixteen years with a BMI > 98th percentile. Before study recruitment initiation, three open meetings were held with GPs and other stakeholders to identify key requirements for primary care clinics and the referral tool to be used in the study.

During the study, all 152 referrals from general practice using BOOST were reviewed by a consultant paediatrician and the appropriateness of referral and likelihood of complicated obesity were assessed.

Results

Of the 152 referrals, two were rejected due to the children being outside the study age range. Thirty-two (21%) cases were 'red flagged' on scrutiny of the BOOST form. The most frequent reasons for red flagging were a history of parental type 2 diabetes (6%); potential endocrine disorders such as relative short stature for weight or concerns about pubertal development (6%); and children with a potential genetic disorder or associated learning difficulties (6%). The identification of potential co-morbidities (proteinuria/possible idiopathic intracranial hypertension and systemic hypertension) or overt eating disorders was less frequent causes for flagging (see Table 1).

Of those children identified as having a parent with type 2 diabetes, none had glycosuria reported from testing in general practice, although in two cases no details were provided on the form. None of these children had silent type 2 diabetes, as confirmed by oral glucose tolerance tests (OGTTs) conducted at the hospital.

In those in whom endocrine disorders were queried, no overt pathology requiring treatment was identified, although one female had adrenarache clinically and one boy had constitutional delay in growth and puberty that did not require a specific intervention.

Table 1 Causes for red flagging by BOOST

Red flag identified	Number of cases (% of cohort)
Parental type 2 diabetes	9 (6)
Possible endocrine disorder (short for weight or pubertal issues)	9 (6)
Possible genetic disorder or learning difficulties	9 (6)
Identified co-morbidity	2 (1)
Overt eating disorder	2 (1)
Iatrogenic cause	1 (<1)
Total	32 (21)

BOOST = Bristol Obesity Online Screening Tool.

Only 76% of children had their blood pressure recorded on the BOOST form, while 70% had a test for glycosuria or had a note that blood glucose was normal (2 cases). The GPs indicated on four forms that the family had declined these tests: in two cases for both blood pressure and urine testing and in one case each for blood pressure or urine. The majority of questionnaires provided no reason for absent data. In nearly half of the cases in which tests were missing, both urine analysis and blood pressure were omitted, while blood pressure was reported alone over twice as often as urine analysis.

Discussion

The Bristol Online Obesity Screening Tool was designed for two main purposes: to provide an accurate and user-friendly tool for GPs to rapidly assess a child's BMI percentile to diagnose obesity and allow referral into our study, and also to ensure that those children requiring secondary care assessment were easily identified and given hospital appointments. Primary care has been identified as a potential setting for managing childhood obesity (NICE, 2006; Reddy, 2006), but GPs feel they have neither the skills to assess obese children nor effective interventions to refer patients to (Turner *et al.*, 2009).

On the basis of our review of 152 referral forms, it would appear that the vast majority of children are suitable for interventions in primary care without recourse to secondary care assessment for underlying pathology. In reality, those cases 'red-flagged' for parental type 2 diabetes could be

effectively screened by routine questioning and testing for glycosuria. Within this study and more importantly, within the childhood obesity clinic in Bristol, no case of silent diabetes has ever been identified from routine oral glucose tolerance tests ($n > 300$, paper in preparation). Adolescents with type 2 diabetes present with similar symptoms to children with type 1 or adults with type 2 diabetes: polyuria, polydipsia, weight loss and in some cases ketonuria (Haines *et al.*, 2007) and testing for glycosuria plus questions regarding possible symptoms should pick up those needing urgent referral. Therefore, while a screening tool should explicitly consider type 2 diabetes in obese children, especially in the context of parental diabetes, the need for secondary care referral and an OGTT is unlikely to be necessary with absent symptoms and a negative urine screen. While the tool seems to accurately direct appropriate children to the attention of secondary care, this study was not designed to examine whether other children might have benefited from a secondary care opinion, but were 'missed'.

The tool was designed for use with children aged above five years. If used for younger children, it would be necessary to add a prompt about extreme weight gain in infancy or early years, alerting the clinician to the need for referral to exclude possible genetic causes of obesity (SIGN, 2010). We believe that our screening tool would enable >85% of obese children to receive their initial weight management safely in primary care. However, this figure takes into account that both blood pressure and a urine analysis are mandatory components of the primary care assessment, which was not the case in approximately 25% of the referrals in this study. GPs do not typically assess a child's blood pressure and so reference tables to interpret readings would need to be available (these are accessible online: www.rcpch.ac.uk/doc.aspx?id_Resource=1764). Furthermore, we suggest that a useful modification to the questionnaire would be to highlight the need for these tests to be performed before referral under a new heading in the form. In the current format, this mandatory component might simply go unnoticed by the referring doctor at the end of the consultation.

As the prevalence of childhood obesity continues to rise, with potentially serious physical, psychological and economic sequelae, it is vital to equip the GP with tools to assess obese children.

We believe that BOOST is a useful adjunct to the sparse resources available in primary care, enabling GPs to rapidly calculate BMI, allowing comparison with national percentiles and ensures that those patients requiring secondary care assessment can be identified with ease and safety. Further evaluation is now needed in different centres to ensure the effectiveness, sensitivity and specificity of this new tool.

Acknowledgement

This paper presents independent research commissioned by the NIHR under its Research for Patient Benefit Programme (Reference no. PB-PG-0706-10090). The views expressed are those of the author(s) and not necessarily those of the NHS, the NIHR or the Department of Health.

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Appendix 1: Bristol Obesity Screening Tool

Referral form for GPs: ‘Care of Childhood Obesity Clinic’, Bristol Royal Hospital for Children

This form needs to accompany referrals to the obesity clinic before a patient can be given an appointment

Criteria for referral: Age 5-16 years; BMI>98th percentile for age and sex*

This can be calculated using BMI centile charts or following the link:

<http://www.health-for-all-children.co.uk/pro.epl?SHOP=HFAC4&DO=USERPAGE&PAGE=CALCULATOR>

If you are not using this form in an electronic format, you can access the above link by typing the following abbreviated URL into your web browser: <http://tinyurl.com/2xtmt9>

** If child does not meet COCO referral criteria (BMI below 98th centile) and is 7–13 years of age, please direct parent to the **Bristol MEND** weight management programme, details can be found at <http://www.bristol.gov.uk/mend> or give parent local MEND phone number ☎ 0117 922 3656*

Name of patient					
Weight		Height		BMI percentile	
Date of birth		School year		Sex	
Address					
Postcode		Telephone			

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Please complete **all** of the following questions and take **blood pressure (see note over page)** and perform a **urine analysis**:

<p>1. Are you concerned that your patient might have a genetic cause for their obesity? If so, please include details in a separate referral letter.</p>	<p>Yes</p>	<p>No</p>
<p>Indicators might include:</p> <ul style="list-style-type: none"> • Learning difficulties • Visual and hearing difficulties • Obvious dysmorphic features 		
<p>2. Do you suspect an associated endocrine disorder? If so, please include details in a separate referral letter.</p>	<p>Yes</p>	<p>No</p>
<p>Indicators might include:</p> <ul style="list-style-type: none"> • Recent onset of excessive weight gain in a child previously of normal height. • Weight and height are disproportionate e.g. short (height less than 50th percentile) and obese. In simple nutritional obesity children are heavy and tall. • Features suggestive of Cushing's syndrome (all or some) facial plethora; striae on abdomen; thighs and breasts; moon face; hirsutism; truncal obesity; and poor linear growth. • Features of delayed or precocious puberty? See details over page. 		
<p>3. Is there an increased likelihood of type 2 diabetes? If so, please see below and include details in a separate referral letter.</p>	<p>Yes</p>	<p>No</p>
<p>Indicators might include:</p> <ul style="list-style-type: none"> • Type 2 diabetes in either parent. • Symptoms of polyuria and polydipsia (requires urgent referral by fax/phone to Dr Shield or to his colleagues at Bristol Royal Hospital for Children). 		
<p>4. Are you concerned that your patient might have one of the rare obesity co-morbidities? If so, please follow the advice below. Please also include details of this in a separate referral letter if still eligible to refer.</p>	<p>Yes</p>	<p>No</p>
<p>Indicators might include:</p> <ul style="list-style-type: none"> • Symptoms suggestive of sleep apnoea, such as snoring and daytime somnolence. <u>They will need concurrent referral to the sleep clinic.</u> • Severe headaches that make you concerned about benign intracranial hypertension. <u>They will need referral to a neurologist to rule out benign intracranial hypertension prior to referral to the childhood obesity clinic.</u> 		

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<p>5. Are you concerned that your patient has an overt eating disorder?</p> <p>Your referral to the childhood obesity clinic will need to be delayed until the patient has had a thorough assessment from child and adolescent mental health services.</p>	Yes	No
<p>Indicators might include: History of binge eating or use of laxatives</p>		
<p>6. Is an iatrogenic cause for the obesity possible? E.g. cranial surgery in the past, certain anticonvulsants.</p>	Yes	No
<p>It would be worth discussing this possibility with the relevant hospital specialist to elicit whether the childhood obesity clinic is appropriate. Please include details in a separate referral letter</p>		

Test	Result	Test	Glucose present	
Blood pressure (cuff size: if child < 55 kg use child cuff, 55–110 kg, use small adult cuff, if >110 kg, use a large adult cuff)		Urine analysis: glucose	Yes	No

Tips for assessing if child in early/ delayed puberty (reference question 2)

1. The following link might help- providing line drawings of the tanner stages of puberty:

<http://www.springerlink.com/content/u458217667341960/fulltext.pdf>

2. See advice in the table below

Sex	Pointers in examination and history
Girl	<p>Precocious:</p> <p>Girls less than 8 years with evidence of breast buds (palpable small swelling directly under the nipple) or pubic hair (initially might be lightly coloured and long). Pubic hair alone may indicate pubarche but this still requires investigation in the children's hospital</p>
Girl	<p>Delayed:</p> <p>Girls aged 13 years without evidence of breast buds or pubic hair growth</p>
Boy	<p>Precocious:</p> <p>Boys less than 9 years with evidence of testicular development (testes > 3mlS volume) or pubic hair growth (initially might be lightly coloured, long and at the base of the penis)</p>
Boy	<p>Delayed:</p> <p>Boys aged 15 years without evidence of testicular development or pubic hair growth</p>

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