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Measuring growth and obesity across childhood and adolescence

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The dramatic rise in childhood obesity has driven the demand for tools better able to assess and define obesity and risk for related co-morbidities. In addition, the early life origins of non-communicable diseases including type 2 diabetes are associated with subtle alterations in growth and body composition, including total and regional body fatness, limb/trunk length and skeletal muscle mass (SMM). Consequently improved tools based on national reference data, which capture these body components must be developed as the limitations of BMI as a measure of overweight and obesity and associated cardiometabolic risk are now recognised. Furthermore, waist circumference as a measure of abdominal fatness in children is now endorsed by the International Diabetes Federation and National Institute for Clinical and Health Excellence for diagnostic and monitoring purposes. The present paper aims to review the research on growth-related variations in body composition and proportions, together with how national references for percentage body fat, SMM and leg/trunk length have been developed. Where collection of these measures is not possible, alternative proxy measures including thigh and hip circumferences are suggested. Finally, body ratios including the waist:height and muscle:fat ratios are highlighted as potential measures of cardiometabolic disease risk. In conclusion, a collection of national references for individual body measures have been produced against which children and youths can be assessed. Collectively, they have the capacity to build a better picture of an individual’s phenotype, which represents their risk for cardiometabolic disease beyond that of the capability of BMI.

**Children: Growth: Obesity: BMI: Waist: Skeletal muscle: Centile charts:
Cardiometabolic risk**

Within the disciplines of child health and nutrition, growth measurement has historically performed important clinical and public health functions, being an indicator of feeding and environmental circumstances of infants and children. As a result, tools have been developed to identify and monitor children and infants displaying poor growth, failure to thrive and malnutrition. These tools, mainly in the form of growth charts and reference data are still very much in use today, with continuing improvements in their diagnostic ability⁽¹⁾.

However, in the 21st century, a completely different nutritional and physical environment exists, whereby in countries such as Ireland and the UK, there is less of a problem with poor growth and under nutrition, but now a near epidemic of overweight and obesity. Thus, our focus has had to switch to measuring what could be termed ‘overgrowth’ in the form of excess body fat. Consequently, the tools needed to measure obesity have also had to adapt and improve to meet the requirements for population surveillance and clinical identification.

Abbreviations: BIA, bioelectrical impedance analysis; FM, fat mass; HC, hip circumference; IOTF, International Obesity Task Force; MFR, muscle:fat ratio; NICE, National Institute for Clinical and Health Excellence; SDS, sd score; SMM, skeletal muscle mass; SMMa, appendicular SMM; TC, thigh circumference; WC, waist circumference.

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Despite this shift in focus, the requirements for assessment tools remain unchanged, in that techniques should be simple, quick and inexpensive to perform, yet still meet the demands for accuracy, reproducibility, sensitivity and specificity. There is a need to avoid the misclassification of individuals and for the measure to be strongly related to morbidity or risk for later disease especially cardiometabolic disease. Indeed the leading causes of deaths in England and Wales in men aged 35 and above, and in women aged 65 and above is attributed to CHD and in the 35–64 years age range in women it is breast cancer⁽²⁾. Both of these non-communicable diseases have strong links with body composition especially excess body fat, obesity and body fat distribution.

Early growth, obesity and chronic disease risk

There is an additional dimension to measuring growth and obesity since it is now recognised that growth patterns and nutritional experience during fetal development and across infancy and childhood are associated with later risk for components of the metabolic syndrome, including type 2 diabetes, dyslipidaemia, non-alcoholic steatohepatitis (fatty liver) and hypertension. Much of this evidence has stemmed from the original epidemiological studies of Professor David Barker and his team where this pioneering research demonstrated a link between low birth weight and low weight at the age of 1 and later risk for hypertension, type 2 diabetes and CHD⁽³⁾. As an alternative to the thrifty genotype hypothesis for risk for type 2 diabetes, the thrifty phenotype hypothesis stemmed from the Barker studies. This hypothesis suggests that type 2 diabetes risk could arise, in part, through a non-genetic, vertical transmission from mother to offspring, whereby metabolic adaptation occurs during fetal life and early infancy. Here, it is proposed that a sensing of a nutritionally poor environment *in utero* triggers the fetus to adapt its metabolism in a direction better able to aid survival and withstand a similar nutritional environment post-natally. However, a conflict between this altered setting and exposure to an environment where food energy availability is abundant and demands for physical activity are low could set the individual on a life course trajectory to type 2 diabetes and cardiometabolic disease⁽⁴⁾.

Body composition, body dimensions and proportionality

With respect to patterns of physical growth, such an adaptation can also be reflected in subtle changes in body composition, including body fat distribution and body proportions and dimensions, which cannot always be easily identified and quantified by some assessment tools with poor sensitivity. Over the past 20 years, however, this initial hypothesis has become refined, with one key newer suggestion being that a lower birth weight coupled with a rapid post-natal growth (or rapid post-natal growth alone) promotes the accumulation of

excess visceral adipose tissue mass and its associated risk for obesity-related morbidity⁽⁵⁾. Thus, when assessing and characterising growth and obesity, the tools should be able to capture both elements while ensuring they are also able to associate these measurements to the phenotype associated with cardiometabolic disease risk. Up until very recently, the accurate characterisation of this phenotype has been constrained by the lack of sensitive and accurate assessment tools. For example, infant and child growth can proceed at different rates, depending largely on the post-natal diet and environment, and can be quantified using weight-for-age and height-for-age charts.

BMI and BMI centile charts

The BMI and BMI charts based on reference data are used to classify individuals across childhood and adolescence that are underweight, of a healthy weight, overweight and obese. These classifications can be statistically based, typically with the 91st and 98th centiles used to define overweight and obese children, and the 2nd centile to define underweight⁽⁶⁾. This form of classification has been improved through the development of the International Obesity Task Force (IOTF) cut-offs, where the centile curves defining overweight and obesity pass through the adult BMI cut-offs of 25 and 30 at age 18 years⁽⁷⁾. However, such assessment tools give no indication of the partitioning of dietary energy and nitrogen across growth into fat- and fat-free tissues; a division that can determine later disease risk. Furthermore, looking more closely at what lies behind BMI indicates that this measurement reflects a composite of a number of aspects of body composition and proportion, which individually can reflect variations in patterns of growth and disease risk. For example, within the BMI the following components of body structure and composition are amalgamated: Skeletal (bone) mass; Skeletal muscle mass (SMM); Organ mass; Adipose tissue mass; Body fat distribution; Limb length; Trunk length; Hydration of the fat-free mass; Stature.

Some of these components are known to have reciprocal effects on disease risk. For example (see later for more details), skeletal muscle and adipose tissue have opposing effects on insulin sensitivity and energy disposal and a deficit in muscle mass coupled with an excess of adipose mass could balance out each other with respect to body weight, with the possibility of leading the BMI to classify a child within the healthy range. Thus, the BMI is limited at an individual level as it correlates with both fat mass and fat-free mass, it has a low sensitivity, i.e. it fails to identify relatively large numbers of children with excess fat and it can underestimate obesity prevalence by the creation of false negatives. Furthermore, the BMI gives no indication of body fat distribution. This is important as an upper body or abdominal distribution of body fat carries a greater risk for cardiometabolic disease due to the accumulation of excess visceral adipose tissue, fat tissue with greater lipolytic activity drained by the portal system and with



increased release of NEFA. This raised fatty acid release is considered a key contributor to decreased insulin sensitivity. These limitations of BMI are widely acknowledged but largely ignored. Nevertheless in the UK, the formal guidelines on obesity assessment produced by the National Institute for Clinical and Health Excellence (NICE) recommend the use of BMI as a practical estimate in children, even though it concedes that it should be interpreted with caution as it is not a direct measure of adiposity⁽⁸⁾. Thus, in child obesity surveillance such as the National Child Measurement Programme where BMI is the assessment tool used, prevalence figures can be under-reported, especially in areas of the country, for example, where a large proportion of children from South Asian backgrounds reside⁽⁹⁾. This stems from the observation that the South Asian phenotype is characterised by a higher fat mass with a more abdominal distribution of body fat and a lower SMM compared with white Europeans of the same BMI for age. Consequently, the WHO has now formally lowered the BMI thresholds for defining overweight (BMI=23) and obesity (BMI=25) in South Asian adults⁽¹⁰⁾. These lowered thresholds should also translate into lower BMI centile cut-offs to define overweight and obesity in South Asian childhood populations. However, a review of the evidence up to 2010 led to the conclusion that there was insufficient evidence to support separate BMI definitions for obesity in children and adolescents from South Asian ethnic groups in the UK⁽¹¹⁾. With advancing technological ability to characterise further the body composition of children and youths, together with evidence arising out of clinical referrals for type 2 diabetes diagnoses in children and youths from this ethnic background, it may well be timely to revisit this position, especially as one of the aims of developing assessment tools is to make them easily understandable and as user friendly as possible. A lack of harmonisation between child/adolescent and adult systems illustrates a major limitation with the current separate systems. For example upon reaching age 18, an individual of South Asian origin with a BMI of 25 would be classified using the IOTF cut-off as borderline healthy weight/overweight before the 18th birthday; but then switching to the adult system at age 18, the individual would now be classified as obese without any change in their BMI. Such are the limitations of BMI that its use in children and youths has now been brought to the attention of the general public through profiling of the issue within the media, with one prominent child obesity researcher claiming 'it's just not a sufficiently sensitive measure'⁽¹²⁾. This statement mirrors similar observations in adults where it has been shown that in the USA, the prevalence of obesity is drastically underestimated when BMI is used compared with the more sophisticated technology of dual energy X-ray absorptiometry⁽¹³⁾.

Abdominal adiposity and waist circumference centile charts

The recognition that abdominal adiposity and visceral fat relate to cardiometabolic disease risk in children

and youths dates back to the mid-1990s. Prior to that time, it was assumed that abdominal obesity was a phenomenon of adulthood, especially the accumulation of excess visceral fat. However, the correlation of waist circumference (WC) with visceral fat accumulation in children has been demonstrated⁽¹⁴⁾ and several studies beginning around the mid-1990s demonstrated that WC was a sensitive marker for several components of cardiometabolic disease. WC has been shown to be independently related to an adverse atherogenic lipoprotein profile in 12–14 year olds⁽¹⁵⁾ and an adverse (raised) fasting insulin concentration in 5–17 year olds⁽¹⁶⁾. More recent studies have extended the relationship between WC and cardiometabolic disease components in children to include non-alcoholic fatty liver disease (non-alcoholic steatohepatitis)⁽¹⁷⁾, elevated blood pressure⁽¹⁸⁾, markers of inflammation⁽¹⁹⁾ and a clustering of components of the metabolic syndrome⁽²⁰⁾. Such observations have led to a demand for national references for WC in children and youths to be produced, which could be used in both clinical and epidemiological contexts. The UK WC charts were produced in 2001⁽²¹⁾ followed by a global expansion of equivalent charts in more than twenty countries, with some of the most recent additions including Malaysia, Germany and Poland^(22–24). Despite this expansion in awareness and utility of WC in obesity and cardiometabolic disease risk assessment, there remains a reluctance to fully embrace this assessment tool by some formal authorities. For example, NICE obesity guidelines state that WC is not recommended as a routine measure in children and youths but concedes that it may be used to give additional information on risk of developing long-term health problems, preferring instead to support the continued use of BMI⁽⁸⁾. Paradoxically, the recent NICE clinical guidelines on psychosis and schizophrenia in children and young people (CG155), recommend the monitoring of WC and hip circumference (HC) and use of WC and HC centile charts for patients receiving anti-psychotic drugs, due to the known effect of this type of pharmacotherapy on weight gain⁽²⁵⁾. More encouragingly, the International Diabetes Federation supports the measurement of abdominal obesity as the 'sine qua non' for the diagnosis of metabolic syndrome in children and adolescents. It bases this statement on the data relating WC with components of the metabolic syndrome in children and the unequivocal evidence of the dangers of abdominal obesity in adulthood. The International Diabetes Federation states the WC>90th centile cut-off as the diagnostic criterion as several studies have shown that these children are more likely to have multiple risk factors than those with a WC below this level⁽²⁶⁾. A further potential improvement to the 90th WC centile cut-off has been the recent development of centiles for UK children which pass through the adult WC cut-offs at age 18 which relate to 'increased risk' (94cm men, 80cm women) and 'high risk' (102cm men, 88cm women)⁽²⁷⁾. Such centiles are the equivalent to the IOTF BMI cut-offs and research is currently being conducted to evaluate the effectiveness of these new centiles.

Body fatness, body fat centile charts and bioimpedance technology

Up to this point, both BMI and WC are used as proxy measures of total and abdominal adiposity. The ideal assessment tool should directly assess adiposity particularly since pathology is driven, in part by excess adipose mass. However, until recently it has been difficult to quantify fat mass outside the laboratory but improvements in bioelectrical impedance technology and its incorporation into step-on scales have taken the assessment of fat and lean masses into the clinical and community settings. Although slightly less accurate compared with the more sophisticated laboratory techniques, they offer practical advantages in being simple to use, with a relatively low cost. Thus in 2006, the first body fat centile charts based on bioelectrical impedance analysis (BIA) technology were produced for the UK child and youth population⁽²⁸⁾. These charts clearly illustrate the gender differences in total body fat accumulation as well as the pattern of accumulation across childhood and adolescence; differences that are obscured in BMI charts. Indeed, the shape of the curves matches the expected changes in body fat patterning across human growth and clearly illustrates the pubertal influences in boys and girls. These charts show, for example, that at age 18 girls have proportionately 60% more body fat than boys, with the median per cent values being 24.6 and 15.4%, respectively. Furthermore, to define useful cut-offs for clinically and epidemiologically purposes that broadly agree with the IOTF BMI cut-offs, the 85th and 95th centiles provided a reasonably close approximation to the IOTF overweight and obese boundaries, with the 2nd centile chosen to approximate the IOTF underweight equivalent. Thus these body fat boundaries define underfat, normal, overfat and obese. We are now seeing body fat centile charts emerging in other paediatric populations, for example, in Hong Kong and Turkish children^(29,30), suggesting the wider recognition of the importance of body fat assessment. One additional advantage of body fat charts over BMI charts is the reduction in the misclassification of children who can be categorised as healthy by the BMI despite having excess body fat. Similarly, large framed or overly muscled children and youths can be classed as overweight or obese by BMI but normal or healthy when based on body fat. Indeed, we have used these body fat charts to demonstrate the extent of misclassification by BMI and in a sample of 1088 children aged between 5 and 13 years, 31% of individuals were declared false negatives (i.e. excess body fat but healthy BMI) and only 4% were rated as false positives (normal fat but overweight BMI)⁽³¹⁾. To emphasise, it is the 'hidden' overfat children within the healthy BMI range, which creates the under estimation of true overweight and obesity prevalence in surveillance systems such as the National Child Measurement Programme.

Components of stature, obesity and chronic disease risk

Overall stature and the height component of BMI can be divided into two simple components: trunk length and

leg length. The ratio of these two elements (expressed as the leg length:height ratio) varies with age across childhood and adolescence as well as varying between the sexes and ethnic groups. Mean leg length:height ratio increases between ages 5 and the pubertal stage, at which point leg length:height ratio declines, illustrating the relative growth of the limbs and trunk at different stages of child development⁽³²⁾. Proportionality is now considered an additional dimension to how body size and composition can influence chronic disease risk. For example, components of cardiometabolic disease including type 2 diabetes, hypertension, and non-alcoholic steatohepatitis as well as overall CHD risk are associated with having both absolute and relatively shorter legs as well as shorter overall stature^(33,34). Leg growth appears to display a degree of plasticity in early life, with a suggestion that a certain proportion of leg growth can be sacrificed in favour of the growth of essential organs such as the brain (whose impaired growth would have far greater negative consequences) when pre- and early post-natal energy and nutrient availability may be compromised. This plasticity of growth in response to nutritional experience comprises a key element of the thrifty phenotype hypothesis⁽³⁵⁾. However, caution must be exercised when relating relative leg length to prior early nutrition as it has been shown through nutritional supplementation trials in undernourished populations, that any increased growth in height as a result of supplementation was due almost entirely to increases in trunk length growth. However, the authors concluded that it may be that the timing of dietary intervention is crucial to influencing leg growth *v.* trunk growth⁽³⁶⁾. Nevertheless, we have recently demonstrated that overweight and obesity in children and adolescents (assessed by either BMI SD score (SDS) or WC SDS) is associated with having relatively shorter legs (as illustrated by the leg length:height ratio)⁽³²⁾. The corollary of this is that overweight and obese children tend to have a greater relative trunk length, therefore suggesting a greater potential for higher truncal fat accumulation. Thus, altered limb and trunk proportions appear to be linked to measures of overweight and obesity, again illustrating that overweight/obesity, at least in part, can be a consequence of altered patterns of growth⁽³⁷⁾. Thus, more epidemiological studies of are incorporating measures of leg and trunk length into study design and analyses, supported by the availability of national references for leg length and sitting height in children and adolescents⁽³⁸⁾.

Child and adolescent height for age and body fatness

Aside from the alterations in the components of stature and proportionality, overweight and obese children (defined by their BMI SDS) also tend to be taller for their age compared with non-overweight children, with this observation even being seen in early childhood^(32,39). Thus, being tall for age could be considered an early marker of risk for overweight and obesity. It had been initially assumed that this height for age-overweight/obese relationship was simply a characteristic of using

BMI as the proxy measure for overweight/obesity, with BMI tending to increase with increasing height and that BMI preferentially classified taller children as overweight or obese. However, this height-for-age-increased adiposity phenomenon has recently been confirmed in a small sample (280 children aged 7–12) in whom, body composition was measured using dual energy X-ray absorptiometry, a laboratory-based reference technique that quantifies tissue masses⁽⁴⁰⁾. Using the simpler field-based method of BIA, conducted in the community on a larger sample (n 2298) of children and covering a wider age range (4.5–14 years) we have confirmed the findings of the laboratory-based study that children who are taller for their age also tend to have higher body fat levels for their age⁽⁴¹⁾. In this study, children were divided into quartiles of height SDS, and mean percentage body fat increased from 18.2% in the first quartile, to 21% in the fourth quartile. In addition, prevalence of overfat/obesity increased from 9.4% in the first quartile of height-for-age, to 29.7% in the fourth quartile of height-for-age. This height-for-age pattern of prevalence was mirrored when BMI was used as the measure of overweight/obesity as well as when WC is used as the measure of central overweight/obesity. It is possible that excess weight and fat gain in childhood is driving additional gains in height and suggests that if energy intake is more than sufficient to promote a rapid linear growth, the excess may be stored as fat, resulting in a positive relation between height and adiposity⁽³⁹⁾. This drive in height growth is not a universal observation however, as it must be remembered that overweight and obese children were present (albeit to a lower extent) in the lower quartiles of height-for-age and in their cases diet was not driving height gain. However, caution must be exercised when attempting to relate height for age (cross-sectional) with growth rates (longitudinal) as genetics (parental height) is the major determinant of height growth with environment (diet) playing a minor part. In addition, rapid linear growth might also lead to advanced skeletal maturation⁽⁴²⁾. Advances in skeletal age in overweight or obese individuals appear to precede increases in height, starting in mid-childhood, suggesting that factors involved with physical maturation are aetiologically linked with overweight risk⁽⁴³⁾. Thus, childhood height could be used to identify more accurately children who may be likely to become overweight or obese in later life, a suggestion previously proposed by others, where height measurement could not only improve the identification of obese children but that special attention be paid to children who have a high BMI and are also taller for their age^(43–45).

Socio-economic status, height-for-age and obesity

Risk for overweight and obesity is also strongly related to socio-economic status, with children from a lower income background demonstrating a higher prevalence compared with children from a higher income background. When examining socio-economic status influences on obesity prevalence, one overlooked

contributory factor is the shorter height-for-age in children from a lower income background compared with those from a higher income background. Thus, at similar body weights across income groups, BMI would be greater in children from a lower income background. This shorter stature effect on BMI would be compounded when body weight-for-age is higher compared with those from a higher income background. However, as discussed earlier, overweight and obese children tend to be taller for age compared with children who are of a healthy weight. Thus, for overweight and obese children from a low-income background, there can be opposing influences on their height-for-age. Employing BIA to quantify adiposity, overfat/obese children from a low-income background have been shown to be significantly taller than their healthy weight peers, but significantly shorter than overfat/obese children from a higher income background⁽⁴⁶⁾. Similar income group differences in height and hence BMI have also been observed in a Canadian population, indicating this phenomenon to be reproducible across population groups⁽⁴⁷⁾. Clearly, shorter height is a second (and likely a more significant) contributor to the greater BMI, BMI z -score and overweight/obesity prevalence in lower income children. These studies illustrate how height-for-age measurement can reveal potential growth-related issues which can impact on adiposity and risk for overweight/obesity.

Skeletal muscle, cardiometabolic disease and muscle mass centile charts

Up to now, development of growth/body composition assessment charts has focused on measures of adiposity, especially as this tissue is a key driver of risk for cardiometabolic disease. However, skeletal muscle, which has been largely overlooked in epidemiological and laboratory studies, is gaining greater recognition as an independent marker of metabolic health, with muscle strength for example, being positively related to greater insulin sensitivity⁽⁴⁸⁾ and low muscle fitness associated with greater metabolic risk⁽⁴⁹⁾. Indeed, skeletal muscle is a major site of insulin-mediated glucose disposal and therefore an important regulator of whole-body glucose homeostasis. Beyond metabolic health, sarcopenia (age-related loss of muscle mass) is emerging as an important clinical and public health issue. Sarcopenia is the major cause of decreased mobility and function in old age, with quality of life diminishing due to a loss of independent living. A key to delaying age-related SMM loss is the optimal buildup of muscle across childhood and adolescence and the maintenance of this mass throughout adulthood⁽⁵⁰⁾, a pattern that has long been recognised for maintaining bone mass and delaying the risk for osteoporosis. Thus for clinical and epidemiological purposes, it has become important to define and identify healthy (and by definition unhealthy) levels of SMM in children and youths. However, due to the difficulty in measuring SMM outside of the laboratory, there has been a lack of age- and gender-specific normative data. Using the same BIA technology used to develop the

UK body fat reference charts, (which has the ability to produce regional or segmental body composition), we have been able to quantify the SMM in the arms and legs of children and youths (defined as appendicular skeletal muscle mass (SMMa)) which acts as a good proxy for whole-body SMM and to generate centile curves for SMMa⁽⁵¹⁾. Three formats of SMMa curves have been constructed, which can be used to support different purposes, namely absolute SMMa (kg), SMMa expressed as a percentage of total body mass (%SMMa) and SMMa as a percentage of fat-free mass (SMMa/fat-free mass (kg) × 100). In addition, centile curves for %fat-free mass have been generated and reflect the reciprocal of %fat mass (FM). Thus, users now have the opportunity to assess body composition in children and youths against a reference population with greater detail compared with equivalent BMI charts. These charts can allow the tracking of SMMa in a clinical setting similar to the way height and weight charts are currently used. The reference data can also be used for epidemiological purposes. SMMa charts have also been produced in a Canadian population⁽⁵²⁾ and it is likely that we shall see them appearing for different global child and youth populations in due course. However, where BIA facilities for quantifying SMMa are not available or appropriate, alternative and simpler prediction tools need to be developed.

Thigh and hip circumference measurement

There is potential for thigh circumference (TC) to act as a proxy for leg SMM. Approximately 75% of whole-body SMM is found within the arms and legs, with the group of muscles in the upper leg constituting a major fraction of total leg muscle mass. A recent observation in adults revealed that a smaller TC, which most likely reflects a low thigh muscle mass is associated with the development of cardiovascular morbidity and early mortality⁽⁵³⁾. With this in mind, reference data for TC across childhood and adolescence could serve a useful purpose and the first set of TC charts for the UK paediatric population have been developed⁽⁵⁴⁾. For completion, HC centile charts have also been developed in the UK⁽⁵⁵⁾, which should prove valuable in the future, especially as NICE guidelines on psychosis and schizophrenia in children and young people (CG155) also recommend the measurement of HC together with WC in patients receiving anti-psychotic pharmacotherapy⁽²⁵⁾.

Muscle:fat and waist:height ratios

Now that both FM and SMMa can be quantified with reasonable accuracy in girls and boys across the age spectrum, this has facilitated the development of a new potential measure of cardiometabolic risk, the muscle:fat ratio (MFR). This measure, originally proposed by Professor Andrew Prentice at the London School of Hygiene and Tropical Medicine is based on the rationale that FM and SMM have opposing effects on insulin sensitivity and energy disposal, with the balance between them more accurately able to determine cardiometabolic

risk beyond the ability of BMI. An individual having both a low SMM and high FM would have a MFR towards the low end of the MFR spectrum and may present a risk for metabolic disease greater than having excess FM alone. We now have generated reference data on the MFR distribution in boys and girls across childhood and adolescence⁽⁵¹⁾. In our population, the mean MFR in boys equals 1.99 (SD 0.60) with a range between 0.69 and 4.84. The equivalent values in girls are 1.38 (SD 0.40), range 0.49–3.22. In addition, MFR is significantly and positively age related in boys and negatively related in girls. MFR also decreases with increase in BMI z-score. An MFR of 1.25 in boys, 1.10 in younger girls and 0.80 in older girls can be used to define the lower limit of normal (mean minus 2 SD). It should however be noted that since the gluteofemoral adipose mass has been shown to be protective for metabolic health, the relationships between MFR and metabolic disease risk may be non-linear^(56,57) and replacing the total body fat with abdominal body fat in this ratio could further improve this measure. However, this proposition also needs to be systematically evaluated, although it is interesting to note that a ratio of SMM to visceral fat area ratio has been shown to be associated with metabolic syndrome and arterial stiffness in a Korean elderly population⁽⁵⁸⁾. This MFR follows a similar principle of the more established waist:height ratio, which has been shown to be effective across all ages and ethnicities as a screening tool for the prediction of cardiovascular diseases and type 2 diabetes, with a waist:height ratio of 0.50 performing as a suitable global boundary value⁽⁵⁹⁾.

Conclusions

Given the importance of cardiometabolic disease as a major global public health and clinical condition, a need has arisen for better tools to monitor individual growth patterns, evaluate at-risk body composition and identify those most at risk of developing the metabolic components of the disease. A suite of individual measures, with national references, against which children and youths can be assessed have now been produced. Used collectively, they have the capacity to build a better picture of an individual's phenotype, which represents their risk for cardiometabolic disease beyond that of the capability of BMI. In the immediate future, most likely some of these tools will find greatest utilisation in a research capacity where they can be better evaluated and refined. In time, the associations of these body measures with components of the metabolic syndrome should be better defined through evaluation of their sensitivity, specificity and validity. Through wider use and awareness and their incorporation into clinical practice, they should be able to stimulate support by policy makers at national and international levels.

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Conflicts of Interest

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H. D. Mc. solely contributed to the writing of this manuscript.

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