Using Body Composition Assessment to Evaluate the Double Burden of Malnutrition

Jonathan C.K. Wells

Childhood Nutrition Research Centre, UCL Great Ormond Street Institute of Child Health, London, UK

Abstract

Background: Populations in low- and middle-income countries are increasingly experiencing a double burden of malnutrition (DBM), incorporating both persistent levels of child undernutrition and rising prevalence of overweight/obesity at later ages. A growing number of individuals experience both components of the DBM through the life-course, thereby accumulating high susceptibility to noncommunicable disease (NCD). *Summary:* Measurements of body composition may prove valuable for assessing NCD risk at the level of the individual. The capacity-load model provides a simple conceptual framework for integrating data on different components of body composition to predict NCD risk. Poor growth in early life, indexed by becoming wasted or stunted, constrains the development of lean mass components such as muscle and organ mass, each of which contribute to the metabolic capacity for homeostasis. Catch-up weight gain in early life, or the development of excess weight from childhood onwards, is associated with elevated adiposity, especially abdominal adiposity, which challenges cardio-metabolic homeostasis and elevates NCD risk. *Key Messages:* A variety of techniques are now available for the measurement of body composition, helping research the association of the DBM with NCD risk. Reference charts allow raw data to be converted to age- and sex-specific z-scores, aiding interpretation.

© International Atomic Energy Agency 2019
Published by S. Karger AG, Basel

Introduction

There is a need to improve monitoring of noncommunicable disease (NCD) risk within and between populations and to assess the efficacy of public health interventions. This is particularly important in the context of the double burden of malnutrition (DBM), where widespread undernutrition in early life co-exists with overweight and obesity in older children, adolescents and adults.

NCD risk has a complex aetiology and is shaped by different types of physiological responses manifesting in different periods of the life-course. Beyond genotype and the effects of adult lifestyle, patterns of nutrition and growth in early life also predict NCD risk. Initial studies focused on the elevated risks associated with low birth weight, widely interpreted as the consequences of foetal "malnu-

Jonathan Wells
Childhood Nutrition Research Centre
UCL Great Ormond Street Institute of Child Health
30 Guilford Street, London WC1N 1EH (UK)
E-Mail Jonathan.Wells@ucl.ac.uk

trition" [1]. Subsequent research indicated a more complex epidemiology, whereby both poor and rapid infant growth predict elevated NCD risk [2, 3].

Body Composition and NCD Risk

Several physiological mechanisms mediate the effects of early environmental stimuli and stresses on NCD risk, including growth patterns, epigenetic modifications, telomere attrition, the development of the gut microbiota and the programming of hormonal axes. These mechanisms are important in the peri-conceptional period and through foetal life and infancy, a period now known as the "first thousand days" [4]. Recognizing this complexity, what simple measurements can be used to monitor the cumulative development of NCD risk? Despite much emphasis on nutrition, it is notable that patterns of early growth are strong markers of NCD risk, along with nutritional status and body composition in adulthood [2, 3]. What is required is a broad framework that can integrate these generic risk markers and their responses to ecological factors.

The first conceptual model of NCD risk was the thrifty phenotype hypothesis, which proposed that malnourished foetuses protected the brain by reducing the growth of other organs and tissues [1]. Individuals adjusting in this way would then have greater susceptibility to the harmful effects of obesity and rich diets in later life. Thus, much attention has been directed to the interactive effects of 2 extremes, low birth weight and adult obesity [1]. However, more detailed analyses revealed dose-response associations between early growth, adult body mass index (BMI), and NCD risk. Broadly, birth weight scales inversely, and adult BMI positively, with adult NCD risk, though at very high birth weight, and very low adult BMI, NCD risk increases as well [5].

Building on these insights, the capacity-load model predicts that NCD risk can be modelled as a consequence of interactive associations between 2 broad traits, metabolic capacity and metabolic load. Metabolic capacity relates to numerous components of organ size and function that have long-term implications for the capacity for homoeostasis [5, 6]. These traits scale closely with patterns of foetal and infant growth. Metabolic load refers to a variety of factors that challenge homeostasis, including components of lifestyle (e.g., dietary glycaemic load, sedentary behaviour, smoking, infectious disease) as well as the level of body fat and its distribution. NCD risk is then predicted to be greatest among those who develop a high metabolic load, but also developed poor metabolic capacity in early life [5, 6].

In the context of the DBM, several anthropometric markers of malnutrition or growth retardation can be reconsidered as markers of metabolic capacity. Beyond the high-risk group of low birth weight, those who became wasted and/or stunted in early life have elevated NCD risk at later ages [7–10]. For example, short adult stature is associated with increased risk of type 2 diabetes, hypertension and cardiovascular disease, and short leg length appears to be especially important in this context [11, 12].

Body composition data may provide further information on metabolic capacity and load [13]. A simple 2-component model partitions body mass into lean mass (used synonymously here with fat-free mass) and fat mass. This model can be enhanced by evaluating the regional anatomical distribution of both tissues. For example, lean tissue incorporates skeletal muscle, important for glucose clearance, as well as the vital organs that perform specific homeostatic functions. Likewise, the metabolic characteristics of adipose tissue vary according to its anatomical location. Subcutaneous fat, in particular gluteofemoral fat, has low metabolic activity and may even be beneficial for homeostasis. In contrast, visceral abdominal adipose tissue emits diverse cytokines that may contribute inflammatory effects and elevate metabolic load [14].

Given these organ-/tissue-specific metabolic effects, it may seem surprising that some studies have failed to show that direct measurement of body fatness predicts NCD risk better than a much simpler marker, BMI [15]. However, it is important to note that BMI is not merely a proxy for adiposity. Calculated as weight/height^2, BMI incorporates information both on capacity (height) and on load (weight). A limitation of BMI for assessing the DBM is that any measurement error of height generates an auto-correlation between underestimation of stature and over-estimation of BMI, increasing the likelihood that individuals will be categorized as both stunted and overweight [16]. BMI is also problematic for comparing NCD risk across ethnic groups, due to variability in physique.

The capacity-load model provides a theoretical framework for predicting how each component of growth and body composition may impact NCD risk. Markers of early growth and lean mass are broadly expected to correlate inversely with risk, whereas markers of catch-up growth and adiposity are expected to correlate positively. Table 1 sets out this rationale on an organ- and tissue-specific basis. Measurement of body composition at any given time point may therefore provide information on both capacity and load, as may markers of lean tissue function such as grip strength [13].

Table 1. Associations of growth and body composition components with the risk of NCD

Trait	Type of association
Growth	
Total stature	Taller stature is associated with lower risk, mediated by traits such as lean mass, skeletal muscle mass and organ size
Leg length	Leg length is associated with lower risk [12, 26], mediated by traits such as lean mass, skeletal muscle mass and organ size, as well as markers of organ function [27]
Trunk length	Trunk length is associated with higher risk [12, 26]. Possible mediating factors include its link with catch-up growth and hence adiposity, and physiological markers such as telomere attrition
Lean mass	
Total lean mass	Greater lean mass is generally associated with lower risk, mediated by traits such as skeletal muscle mass and organ size. However, large lean mass has been associated with higher blood pressure [26]
Skeletal muscle mass	Greater muscle mass is associated with lower risk, mediated by traits such as greater capacity for glucose uptake
Organ mass	Greater organ mass appears to be associated with lower risk. However, it remains unclear how much this is driven by direct benefits of larger organs [19], versus smaller organs marking growth retardation in early life [1]
Adiposity	
Total fat mass	Greater fat mass is associated with higher risk, mediated by association with central abdominal fat
Peripheral adiposity	Associations of peripheral fat with risk are mixed and may relate in part to correlations with visceral fat. Greater peripheral fat may directly increase some risk markers, such as blood pressure, however, greater gluteofemoral fat may improve insulin sensitivity [28]
Central abdominal adiposity	Greater abdominal fat mass is associated with higher risk, mediated by the production of inflammatory cytokines [14]

NCD, noncommunicable disease.

In terms of the DBM, several studies have linked stunting with reduced lean mass, muscle mass and organ mass at later ages [8, 9]. Whether stunting inherently predisposes to obesity and elevated metabolic load is less certain. Some studies have linked stunting with impaired fat oxidation and elevated central fat [e.g., 7], whereas others report long-term deficits in both fat and lean [e.g., 8]. These differences might be mediated by variability in the composition of the diet. A study from Cambodia showed that infant undernutrition is associated in the short term with the relative preservation of fat at the expense of lean mass [9]. A longer-term follow-up of survivors of severe undernutrition in Malawi found several markers of depleted metabolic capacity (shorter leg length, lower lean mass, reduced grip strength), but less evidence of elevated adiposity or metabolic load [10]. Most likely, these individuals will remain more susceptible to NCD risk if they gain excess weight in later life, relative to their peers who escaped early undernutrition.

Body composition data may be particularly valuable in infancy, which appears to represent a key critical window in the aetiology of NCD risk. Different studies have linked both poor and rapid infant growth with adult NCD risk [2, 17]. This can be interpreted as poor growth constraining the development of metabolic capacity and rapid growth elevating metabolic load [6]. Using repeat measurements of body composition through the first 6 months after birth in an Ethiopian birth cohort, latent class analysis identified 4 different patterns of fat accretion and 2 different patterns of lean accretion [18]. Further work will explore how these different tissue accretion patterns relate to long-term NCD risk. Few studies have yet explored the specific association of organ phenotype with NCD risk. A longitudinal study of children in Nepal found that birth weight, leg length and kidney length were all inversely associated with systolic blood pressure, whereas trunk length, fat mass and kidney anterior–posterior diameter were all positively associated [19]. These data broadly support the predictions in Table 1, though it remains unclear why "wider" kidneys are associated with high blood pressure.
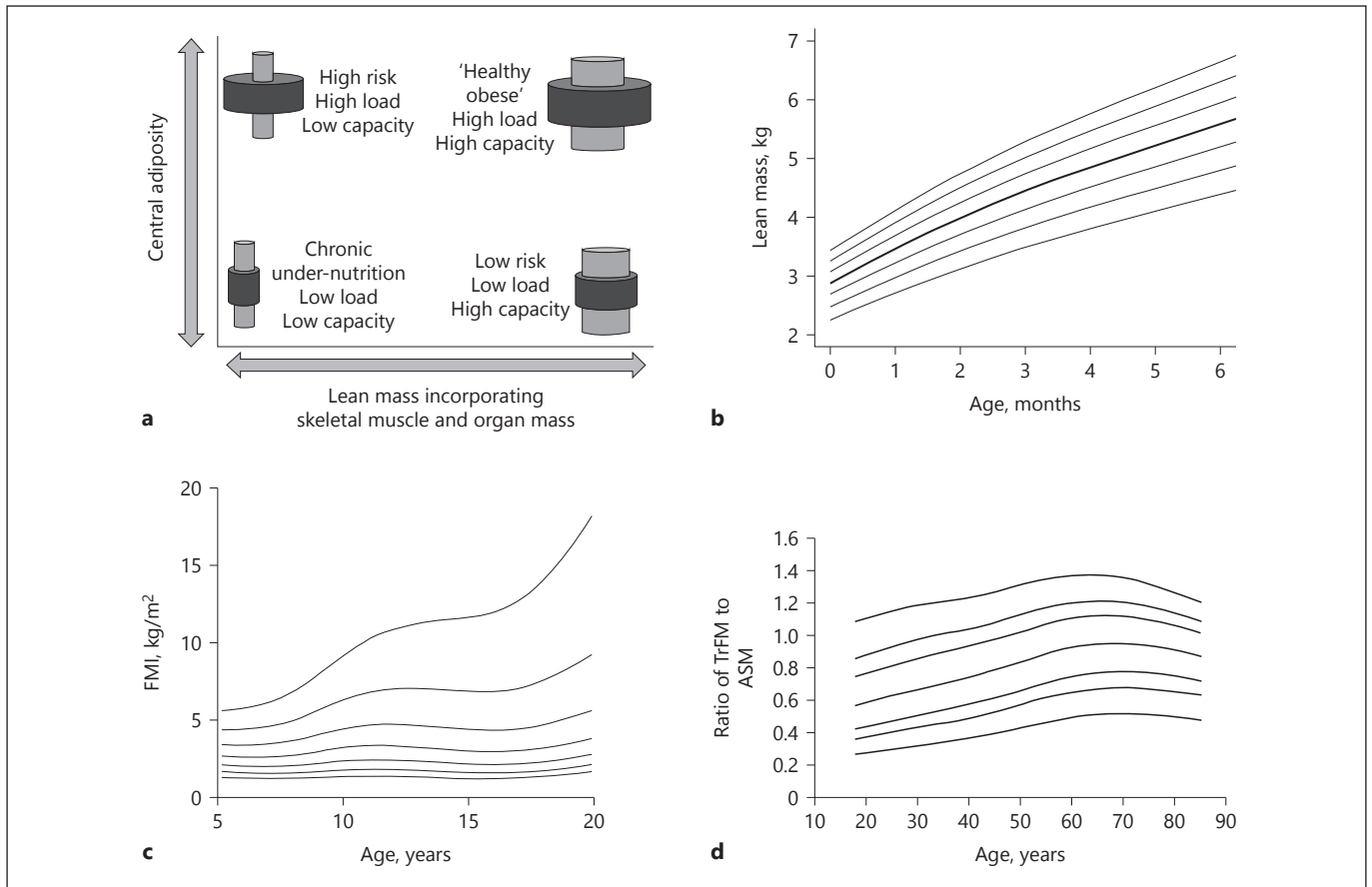
Fig. 1. Body composition growth charts for the assessment of NCD risk in the context of the DBM. **a** Schematic diagram showing how variability in central fat mass and lean mass interact in shaping cardio-metabolic health. **b** Growth chart for lean mass over the first 6 months of life in males from Jimma, Ethiopia (reproduced with permission from Andersen et al. [24]). **c** Growth chart for to-tal fat mass index between 5 and 20 years in UK females (reproduced with permission from Wells et al. [25]). **d** Chart for the ratio of TrFM to ASM in US women over adult life to index the degree of sarcopenic obesity (reproduced with permission from Siervo et al. [22]). TrFM, trunk fat mass; ASM, appendicular skeletal muscle mass; FMI, fat mass index.

Beyond basic anthropometry, a variety of techniques are now available for body composition assessment, even in younger age groups [20]. These include skinfold thicknesses at various anatomical locations to assess subcutaneous adiposity, and several 2-component techniques that differentiate fat and lean mass. The options include isotope dilution for the quantification of total body water, from which lean mass can be predicted by adjusting for hydration, or air-displacement plethysmography, which estimates the ratio of fat to lean in body weight using Archimedes principle. Bio-electrical impedance analysis can be used either to predict total body water directly and hence lean and fat mass, or in abstract "vector analysis", which can help differentiate cell mass from hydration status. Dual-energy X-ray absorptiometry uses the differential attenuation of X-rays to differentiate bone, lean soft tissue and

fat and can provide both whole- and regional-body composition data, while magnetic resonance imaging can provide sophisticated data on the anatomical distribution of adipose and adipose-free lean tissue, though is unsuitable for the younger paediatric population other than neonates.

Once obtained, however, body composition data like anthropometric data are difficult to interpret in absolute terms due to the wide range of variability. This problem can be resolved by developing "body composition growth charts", allowing all data to be converted to age- and sex-specific z-scores [21]. Such reference charts are emerging not only for adults, but also infants and children [22–25]. Reference charts may address either absolute tissue masses or for size-adjusted variables (e.g., fat mass index, lean mass index, each expressed in the same units as the BMI) [23–25]. Body composition charts can also address the

relationship between metabolic capacity and load. For example, sarcopenic obesity represents a state indicative of a DBM within individuals. Finally, new charts have been developed to express the ratio of abdominal fat to skeletal muscle mass [22]. Future charts might plot a z-score for central fat (metabolic load) against a z-score for lean mass (metabolic capacity) to help visualize metabolic risk. Figure 1 highlights these different approaches.

Conclusion

Information on body composition may be obtained indirectly from anthropometry or directly from a wide range of measurement techniques [13, 20] and analysed relative to reference data to adjust for age and sex variability. Such measurements are increasingly being incorporated in research addressing the health impact of the DBM [7–10, 18, 19]. Data from different life-course periods or different traits may potentially be combined in "clustered z-scores" to provide more composite indices of NCD risk [13].

Author Contributions

J.W. conceptualized the overall design of the manuscript and contributed to writing of the final manuscript and approved the final draft.

References

1 Hales CN, Barker DJ. Type 2 (non-insulin-dependent) diabetes mellitus: the thrifty phenotype hypothesis. Diabetologia. 1992 Jul; 35(7):595–601.

2 Hales CN, Barker DJ, Clark PM, Cox LJ, Fall C, Osmond C, et al. Fetal and infant growth and impaired glucose tolerance at age 64. BMJ. 1991 Oct;303(6809):1019–22.

3 Bhargava SK, Sachdev HS, Fall CH, Osmond C, Lakshmy R, Barker DJ, et al. Relation of serial changes in childhood body-mass index to impaired glucose tolerance in young adulthood. N Engl J Med. 2004 Feb;350(9):865–75.

4 Cunha AJ, Leite ÁJ, Almeida IS. The pediatrician's role in the first thousand days of the child: the pursuit of healthy nutrition and development. J Pediatr (Rio J). 2015 Nov-Dec; 91(6 Suppl 1):S44–51.

5 Wells JC. The capacity-load model of non-communicable disease risk: understanding the effects of child malnutrition, ethnicity and the social determinants of health. Eur J Clin Nutr. 2018 May;72(5):688–97.

6 Wells JC. The thrifty phenotype: an adaptation in growth or metabolism? Am J Hum Biol. 2011 Jan-Feb;23(1):65–75.

7 Hoffman DJ, Sawaya AL, Verreschi I, Tucker KL, Roberts SB. Why are nutritionally stunted children at increased risk of obesity? Studies of metabolic rate and fat oxidation in shantytown children from São Paulo, Brazil. Am J Clin Nutr. 2000 Sep;72(3):702–7.

8 Wells JC, Devakumar D, Manandhar DS, Saville N, Chaube SS, Costello A, et al. Associations of stunting at 2 years with body composition and blood pressure at 8 years of age: longitudinal cohort analysis from lowland Nepal. Eur J Clin Nutr. 2019 Feb;73(2):302–10.

9 Skau JK, Grenov B, Chamnan C, Mary C, Wieringa FT, Dijkhuizen MA, et al. Stunting, wasting and breast-feeding as correlates of body composition in Cambodian children at 6 and 15 months of age. Br J Nutr. 2019 Mar; 121(6):688–98.

10 Lelijveld N, Seal A, Wells JC, Kirkby J, Opondo C, Chimwezi E, et al. Chronic disease outcomes after severe acute malnutrition in Malawian children (ChroSAM): a cohort study. Lancet Glob Health. 2016 Sep;4(9):e654–62.

11 Paajanen TA, Oksala NK, Kuukasjärvi P, Karhunen PJ. Short stature is associated with coronary heart disease: a systematic review of the literature and a meta-analysis. Eur Heart J. 2010 Jul;31(14):1802–9.

12 Lawlor DA, Ebrahim S, Davey Smith G. The association between components of adult height and Type II diabetes and insulin resistance: British Women's Heart and Health Study. Diabetologia. 2002 Aug;45(8):1097–106.

13 Wells JC, Shirley MK. Body composition and the monitoring of non-communicable chronic disease risk. Glob Health Epidemiol Genom. 2016 Oct;1:e18.

14 Wells JC. Ethnic variability in adiposity and cardiovascular risk: the variable disease selection hypothesis. Int J Epidemiol. 2009 Feb; 38(1):63–71.

15 Kuper H, Taylor A, Krishna KV, Ben-Shlomo Y, Gupta R, Kulkarni B, et al. Is vulnerability to cardiometabolic disease in Indians mediated by abdominal adiposity or higher body adiposity. BMC Public Health. 2014 Dec; 14(1):1239.

16 Timæus IM. Stunting and obesity in childhood: a reassessment using longitudinal data from South Africa. Int J Epidemiol. 2012 Jun; 41(3):764–72.

17 Skilton MR, Sullivan TR, Ayer JG, Garden FL, Harmer JA, Leeder SR, et al. Weight gain in infancy is associated with carotid extra-medial thickness in later childhood. Atherosclerosis. 2014 Apr;233(2):370–4.

18 Andersen GS, Wibaek R, Kaestel P, Girma T, Admassu B, Abera M, et al. Body Composition Growth Patterns in Early Infancy: A Latent Class Trajectory Analysis of the Ethiopian iABC Birth Cohort. Obesity (Silver Spring). 2018 Jul;26(7): 1225–33.

19 Wells JC, Devakumar D, Grijalva-Eternod CS, Manandhar DS, Costello A, Osrin D. Blood pressure and the capacity-load model in 8-year-old children from Nepal: testing the contributions of kidney size and intergenerational effects. Am J Hum Biol. 2016 Jul;28(4): 555–65.

20 Wells JC, Fewtrell MS. Measuring body composition. Arch Dis Child. 2006 Jul;91(7):612–7.

21 Wells JC. Toward body composition reference data for infants, children, and adolescents. Adv Nutr. 2014 May;5(3):320S–9S.

22 Siervo M, Prado CM, Mire E, Broyles S, Wells JC, Heymsfield S, et al. Body composition indices of a load-capacity model: gender- and BMI-specific reference curves. Public Health Nutr. 2015 May;18(7):1245–54.

23 Wells JC, Williams JE, Chomtho S, Darch T, Grijalva-Eternod C, Kennedy K, et al. Body-composition reference data for simple and reference techniques and a 4-component model: a new UK reference child. Am J Clin Nutr. 2012 Dec;96(6):1316–26.

24 Andersen GS, Girma T, Wells JC, Kæstel P, Leventi M, Hother AL, et al. Body composition from birth to 6 mo of age in Ethiopian infants: reference data obtained by air-displacement plethysmography. Am J Clin Nutr. 2013 Oct;98(4):885–94.

25 Wells JCK, Davies PSW, Fewtrell MS, Cole TJ. Body composition reference charts for UK infants and children aged 6 weeks to 5 years based on measurement of total body water by isotope dilution. Eur J Clin Nutr. 2019, Epub ahead of print.

26 Montagnese C, Nutile T, Marphatia AA, Grijalva-Eternod CS, Siervo M, Ciullo M, et al. Body composition, leg length and blood pressure in a rural Italian population: a test of the capacity-load model. Nutr Metab Cardiovasc Dis. 2014 Nov;24(11):1204–12.

27 Fraser A, Ebrahim S, Davey Smith G, Lawlor DA. The associations between height components (leg and trunk length) and adult levels of liver enzymes. J Epidemiol Community Health. 2008 Jan;62(1):48–53.

28 Snijder MB, Visser M, Dekker JM, Goodpaster BH, Harris TB, Kritchevsky SB, et al.; Health ABC Study. Low subcutaneous thigh fat is a risk factor for unfavourable glucose and lipid levels, independently of high abdominal fat. The Health ABC Study. Diabetologia. 2005 Feb;48(2):301–8.