International Journal of Epidemiology, 2014, 672–674
doi: 10.1093/ije/dyu060
Advance Access Publication Date: 1 April 2014

Commentary: The paradox of body mass index in obesity assessment: not a good index of adiposity, but not a bad index of cardio-metabolic risk

Jonathan CK Wells

Childhood Nutrition Research Centre, UCL Institute of Child Health, 30 Guilford Street, London WC1N 1EH, UK. E-mail: Jonathan.Wells@ucl.ac.uk

Among the technologies currently used to describe and investigate human obesity are magnetic resonance imaging, computed tomography, ultrasound, dual-energy X-ray absorptiometry and electron microscopy. With these techniques we can discern different anatomical depots of fat, the deposition of lipid around organs and in arteries, the distribution of lipid droplets in muscle tissue, and intracellular lipid.

Notwithstanding all this technology, however, the routine categorization of obesity remains based on a very simple statistical construct that was first proposed by Adolphe Quetelet in 1832.[1] Quetelet was fascinated by the population distribution of traits, especially that the Gaussian or bell-shaped curve applied not only to the phenomena of nature but also to human characteristics. However, when he began to collect data on body weight (WT) and height (HT), he encountered difficulties in demonstrating the expected normal distribution:

> If man increased equally in all dimensions, his weight at different ages would be as the cube of his height. Now, this is not what we really observe. The increase of weight is slower, except during the first year after birth; then the proportion we have just pointed out is pretty regularly observed. But after this period, and until near the age of puberty, weight increases nearly as the square of the height … which naturally leads to this conclusion … that the transverse growth of man is less than the vertical.

By the start of the 20th century, the issue of adjusting for height when assessing weight had come to the attention of the life insurance industry, which was already aware that life span was associated with relative body weight.[2] But by this time several different approaches had been proposed, and no convincing argument in favour of any one index had yet been proposed. Alongside Quetelet's WT/HT^2, others had suggested[3] WT/HT^3, W/H and $HT/WT^{0.33}$.

In a classic paper published in 1972, Keys and colleagues set out to resolve this dilemma,[3] using the following criteria. First, the best index should be one that maximized its correlation with the numerator WT while minimizing its correlation with the denominator HT. They approached this by secondly considering the correlation of the index with measurements of adiposity, as the physiological penalties associated with high body weight were already being attributed to high body fat content. Using these two criteria, Keys and colleagues identified WT/HT^2 as the optimum obesity index, and proposed that it be known as the 'body mass index' or BMI. They also considered it ideal for all human populations.[3]

In the following decade, BMI rapidly became adopted as the primary obesity index. In adults, cut-offs of $25\,kg/m^2$ and $30\,kg/m^2$ were proposed to classify overweight and obesity,[4] and these remain widely used in many populations, though lower cut-offs have been proposed for Asian populations.[5] 'International' BMI growth charts were also developed for children using data from several countries, with overweight and obesity defined using age-specific cut-offs that converged on the adult values described above.[6] Obesity was now a condition defined on relatively robust statistical, rather than direct physiological, criteria. On this basis, it was possible for

© The Author 2014. Published by Oxford University Press on behalf of the International Epidemiological Association

This is an Open Access article distributed under the terms of the Creative Commons Attribution Non-Commercial License (http://creativecommons.org/licenses/by-nc/3.0/), which permits non-commercial re-use, distribution, and reproduction in any medium, provided the original work is properly cited. For commercial re-use, please contact journals.permissions@oup.com

Downloaded from http://ije.oxfordjournals.org/ at UCL Library Services on July 24, 2014

epidemiologists to observe global trends in obesity prevalence, to investigate the association of obesity and cardio-metabolic risk and to identify obesity risk factors.

It was only after BMI had already become established as the primary obesity index that the accurate assessment of human body composition became possible in large samples. By this time, the health risks of obesity were increasingly attributed to excess adiposity, in particular central abdominal adiposity surrounding the viscera.[7] Peripheral adiposity, in the gluteo-femoral region, not only appears much less harmful, but may even be protective against chronic diseases.[8] Give these complex associations between different adipose tissue depots and metabolic health, it seemed logical to revisit the question of the utility of BMI as an index of adiposity—in other words, to re-examine the physiological rather than the statistical basis of BMI.

The conventional approach to this issue involved exploring the association between BMI and the proportion of fat in weight, or % fat. In any dataset, it was clear that as % fat rose, so on average did BMI. On this basis, BMI seemed an adequate index of adiposity. But aside from this correlation, several other facts have become evident. First,[9] just as Quetelet observed, infancy and puberty are periods during which BMI is not independent of height, and WT is proportional to HT^3. Second, BMI correlates strongly with other components of weight, such as lean mass and bone mass. Third, both in the normal range of weight and among those who are obese, individuals can differ widely in their body composition even if they have the same BMI value.[10] In other words, BMI is poor at discriminating the ratio of fat to lean tissue within body weight, as shown in Figure 1.

On this basis, the utility of BMI as the primary index of obesity may seem questionable, and a number of studies were conducted to test the hypothesis that more specific indices of adiposity would improve the categorization of cardio-metabolic risk. Surprisingly, the results were mixed; for example, whereas some studies reported waist girth to have a stronger association than BMI with chronic disease risk,[11] other studies found the two variables to be similarly predictive.[12]

Paradoxically, it seems that the various limitations of BMI as a specific index of adiposity may also be its strengths as a composite index of cardio-metabolic risk. In children and adolescents, tall height and lean mass have been shown to be associated with cardio-metabolic risk, independently of the effects of adiposity,[13,14] hence part of the 'risk' categorized by BMI appears not to derive from adiposity. In adulthood, it is short stature that correlates with cardio-metabolic risk, independently of adiposity.[15] The contribution of elevated lean mass to cardio-metabolic risk in adults remains little explored, but unpublished data

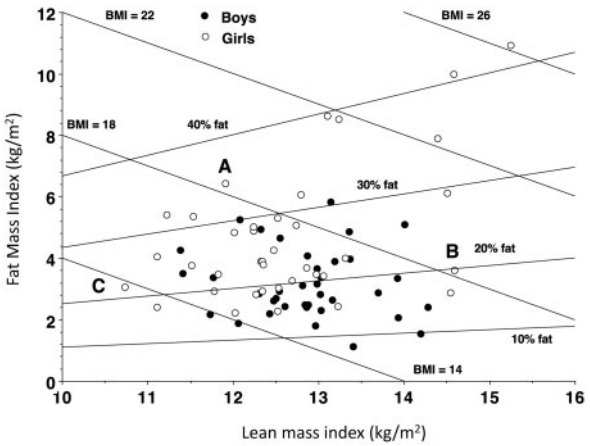

Figure 1. Hattori chart for children aged 8 years, measured by isotope dilution. Lean mass index (x-axis) and fat mass index (y-axis) sum to give body mass index (BMI). The arrows identify three boys: A and B have similar BMI but contrasting adiposity; whereas B and C have similar percentage fat despite contrasting BMI. Reproduced with permission.[16]

from south Italy (Montagnese, Marphatia, Wells and Ciullo) indicate that lean mass is positively associated with blood pressure, independently of height and adiposity, in adult men but not women.

Obesity remains widely conceptualized as a condition of excess body fat, but we should revisit this assumption; for many aspects of cardio-metabolic risk, it may be useful to think of BMI as a composite index of risk, before we look for specific effects of organs, tissues and their regional distribution. BMI reflects variability in each of adiposity, lean mass and height, and each may contribute to cardio-metabolic risk. This generates the paradox that whereas dividing weight by the square of height is a very simplistic way of assessing nutritional status, it may still generate a valuable index of cardio-metabolic risk. In turn, the role of measuring body composition in more detail may be less about improving the categorization of risk, and more about understanding how that risk is generated through the life course.[14]

Conflict of interest: None declared.

References

1. Quetelet A. *A Treatise on Man and the Development of his Faculties*. 1842. Reprinted New York: Burt Franklin, 1968.
2. Eknoyan G. Adolphe Quetelet (1796-1874) – the average man and indices of obesity. *Nephrol Dial Transplant* 2008;**23**:47–51.
3. Keys A, Fidanza F, Karvonen MJ, Kimura N, Taylor HL. Indices of relative weight and obesity. *J Chronic Dis* 1972;**25**:329–43. Reprinted *Int J Epidemiol* 2014; doi:10.1093/ije/dyu058.
4. Garrow JS, Webster J. Quetelet's index (W/H2) as a measure of fatness. *Int J Obes* 1985;**9**:147–53.

Downloaded from http://ije.oxfordjournals.org/ at UCL Library Services on July 24, 2014

5. World Health Organization Expert Consultation. Appropriate body-mass index for Asian populations and its implications for policy and intervention strategies. *Lancet* 2004;**363**:157–63.
6. Cole TJ, Bellizzi MC, Flegal KM, Dietz WH. Establishing a standard definition for child overweight and obesity worldwide: international survey. *BMJ* 2000;**320**:1240.
7. Fox CS, Massaro JM, Hoffmann U *et al*. Abdominal visceral and subcutaneous adipose tissue compartments: association with metabolic risk factors in the Framingham Heart Study. *Circulation* 2007;**116**:39–48.
8. Snijder MB, Visser M, Dekker JM *et al*. 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;**48**:301–08.
9. Cole TJ. Weight/height^p compared to weight/height^2 for assessing adiposity in childhood: influence of age and bone age on p during puberty. *Ann Hum Biol* 1986;**13**:433–51.
10. Wells JC, Fewtrell MS, Williams JE, Haroun D, Lawson MS, Cole TJ. Body composition in normal weight, overweight and obese children: matched case-control analyses of total and regional tissue masses, and body composition trends in relation to relative weight. *IntJ Obes Relat Metab Disord* 2006;**30**:1506–13.
11. Yusuf S, Hawken S, Ounpuu S *et al*. Obesity and the risk of myocardial infarction in 27,000 participants from 52 countries: a case-control study. *Lancet* 2005;**366**:1640–49.
12. Taylor AE, Ebrahim S, Ben-Shlomo Y *et al*. Comparison of the associations of body mass index and measures of central adiposity and fat mass with coronary heart disease, diabetes, and all-cause mortality: a study using data from 4 UK cohorts. *Am J Clin Nutr* 2010;**91**:547–56.
13. Grijalva-Eternod CS, Lawlor DA, Wells JC. Testing a capacity-load model for hypertension: disentangling early and late growth effects on childhood blood pressure in a prospective birth cohort. *Public Library of Science One* 2013;**8**:e56078.
14. Wells JC, Cole TJ. Height, adiposity and hormonal cardiovascular risk markers in childhood: how to partition the associations? Epub *IntJ Obes Relat Metab Disord* 2014. doi: 10.1038/ijo.2014.31.
15. The Emerging Risk Factors Collaboration. Adult height and the risk of cause-specific death and vascular morbidity in 1 million people: individual participant meta-analysis. *Int J Epidemiol* 2011:**41**:1419–33.
16. Wells JC, Chomtho S, Fewtrell MS. Programming of body composition by early growth and nutrition. *Proc Nutr Soc* 2007;**66**:423–34.

Downloaded from http://ije.oxfordjournals.org/ at UCL Library Services on July 24, 2014