



A requiem for BMI in the clinical setting

Maria Cristina Gonzalez^{a,b}, Maria Isabel T.D. Correia^c, and Steven B. Heymsfield^b

Purpose of review

Quetelet reported in the nineteenth century that body weight varies across adults with the square of height. Quetelet's index, now known as BMI, is accepted by most health organizations as a first-level measure of body fat and as a screening tool for diagnosing excess adiposity. Modern imaging methods now, however, indicate that BMI has limited predictive value for estimating body fat and lean mass at the individual level. The use of BMI as a measure of body composition in the clinical setting should therefore be challenged.

Recent findings

Recent studies enrolling cancer and surgical patients reported discrepant outcomes when BMI was used as a body composition surrogate. Sarcopenia, loss of muscle mass and function, which affects the elderly and those with chronic and acute diseases, is not accurately diagnosed with BMI. The distribution of adipose tissue is not characterized by BMI, specific measures of which have greater predictive value for metabolic impairments and clinical outcomes.

Summary

BMI, as the traditional tool for assessing malnutrition and obesity, is not appropriate to accurately differentiate between important body weight components and therefore should not be used for making clinically important decisions at the individual patient level.

Keywords

BMI, body composition, malnutrition, obesity, sarcopenia

INTRODUCTION

L.O.V.E, a 19-year-old Olympic gymnast, is admitted to the emergency department with acute appendicitis. She undergoes a laparoscopic appendectomy but on the subsequent 2 days presents several vomiting episodes and abdominal distension. She undergoes nutritional screening, as demanded by the hospital quality protocol, using the malnutrition universal screening tool (MUST). She is classified at a high risk of malnutrition based on her current BMI of 17.7 kg/m², although she has had no loss of body weight and her current disease has not yet impacted the 'acute disease effect score'. Despite the latter, but due to her lean body composition, which placed her at risk according to the used screening tool and her vomiting, she is prescribed parenteral nutrition. As a consequence of subclavian vein access, she develops a 30% right pneumothorax, which is then drained.

S.E.E., a 79-year-old man with perforated diverticulitis and peritonitis, is admitted to the ICU after a right colectomy and a colostomy. He develops severe abdominal distension with vomiting on the subsequent 3 days. As a routine protocol, he

undergoes nutritional screening, using the same above instrument (MUST). He is classified at a low risk of malnutrition because his current BMI is 32.2 kg/m², he has not lost weight and has not been eating only in the last 4 days. Therefore, he should have reassessment within a week based on his screening score. However, the professional who undertakes the screening is well trained and decides to carry out a full nutritional assessment that further indicates this patient to be severely malnourished.

The two above clinical cases are examples of how tools that rely on BMI [1] might mislead to

^aPost-graduate Program in Health and Behavior, Catholic University of Pelotas, Pelotas, RS, Brazil, ^bPennington Biomedical Research Center, Louisiana State University, Baton Rouge, Louisiana, USA and ^cDepartment of Surgery, Federal University of Minas Gerais Medical School, Belo Horizonte, MG, Brazil

Correspondence to Maria Cristina Gonzalez, MD, PhD, Post-graduate in Health and Behavior, Catholic University of Pelotas, R. Gonçalves Chaves, 377 Room 411, Pelotas CEP 96015-560 RS, Brazil. Tel: +55 53 99982 1328; e-mail: cristinagbs@hotmail.com

Curr Opin Clin Nutr Metab Care 2017, 20:000–000

DOI:10.1097/MCO.0000000000000395

KEY POINTS

- BMI has a good correlation with % body fat at the population level, but the predictive value at the individual level is very limited.
- BMI use in clinical practice may jeopardize the nutritional diagnosis, in particular of malnutrition.
- BMI less than 30 kg/m² does not exclude the presence of metabolic risks associated with the excess of adiposity.
- BMI at least 30 kg/m² does not exclude the presence of low muscle mass (sarcopenic obesity), mainly in some clinical situations as in elderly, cancer, chronic diseases and critically ill patients.

equivocal situations that place patients at increased risks, although they do not consider this variable alone. L.O.V.E. was prescribed unnecessary parenteral nutrition, while S.E.E., if not further nutritionally assessed, would have been treated later than recommended. It is indisputable that BMI has been valuable ever since its development. However, in current clinical practice, particularly when the obesity pandemic is mounting worldwide [2], relying on BMI alone or tools to screen or assess the nutritional status of individuals may be misleading and does not help the diagnosis of a hidden burden in the hospital setting [3,4]. Furthermore, as an instrument of body composition assessment, BMI lacks the capacity to provide adequate individual diagnoses because of its nonspecific characteristics, which will be discussed in detail in the next sections.

BACKGROUND

BMI resulted from the initial studies carried out during the mid-nineteenth century by Lambert Adolphe Jacques Quetelet, the renowned Belgian astronomer, mathematician and sociologist. He reported that body weight (W) across adults varied with the square of height (Ht). This observation, repeated later by others [5], is recognized as Quetelet’s rule: $W \propto Ht^2$. According to Quetelet’s Rule, W/Ht^2 is a shape index that is independent of height. Later, workers in the early twentieth century named the shape index, W/Ht^2 , Quetelet’s Index. Several such indices were developed during this time period as a means of describing individual and group health and nutritional status.

More than one century following Quetelet’s seminal observation, Keys *et al.* [6] at the University of Minnesota reported that the shape index W/Ht^2 showed the highest correlation with adiposity among the factors evaluated, measured as the sum of skinfolds and %fat by underwater weighing. The authors renamed Quetelet’s index as BMI, and this taxonomy remains in place today.

BMI today is taken by most major health organizations as the first-level measure of adiposity. As a screening tool, BMI is relatively easy to calculate from measured or self-reported weight and height. Cost is low and there are no individual health risks posed by measuring weight and height. BMI weight guidelines are simple and clear.

At the population level, BMI is highly correlated with %fat. As an example, Fig. 1 presents a scatter plot of BMI against %fat for men (left) and women (right) who participated in the National Health and

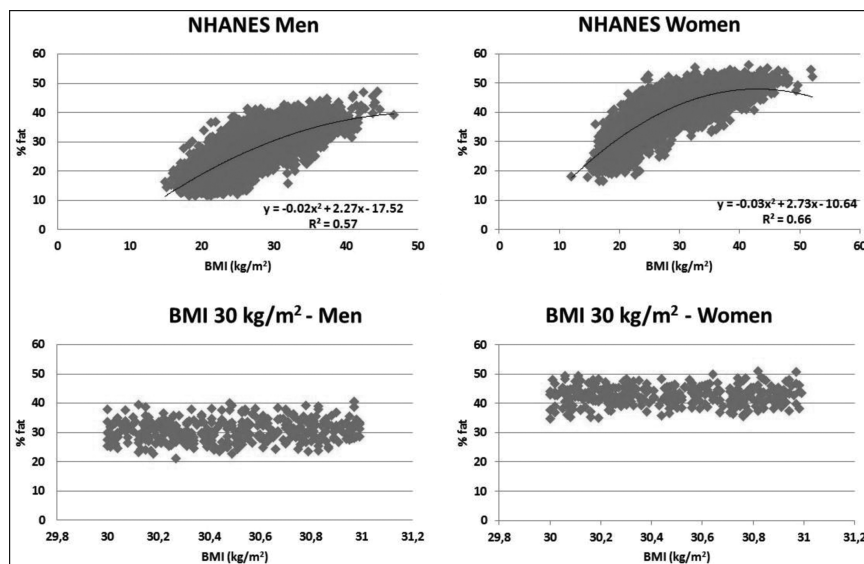


FIGURE 1. %fat varies from 21.2 to 40.5% for men (lower left) and from 34.7 to 50.9% for women (lower right). This observation shows that BMI has a limited predictive value at the individual level.

Nutrition Examination Survey (NHANES 1999–2004) [5^{*}]. Body fat was measured by dual-energy X-ray absorptiometry (DXA). In this example, the correlation between BMI and %fat has an R^2 of 0.57 for men and 0.66 for women, both $P < 0.001$. Note that the relation between BMI and %fat is curvilinear and that this function can be linearized by using inverse BMI ($1/\text{BMI}$) as the predictor variable for %fat.

Despite the good correlation between BMI and %fat at the population level, variability is large at any given BMI level. For example, at BMI 30 kg/m^2 for the NHANES individuals plotted in Fig. 1, %fat varies from 21.2 to 40.5% for men (lower left panel) and from 34.7 to 50.9% for women (lower right panel). This observation shows that BMI has a limited predictive value at the individual level.

What factors account for this variability? First, %fat at any given BMI level is greater in old people than it is in young people [5^{*}]. This observation reflects the loss of skeletal muscle and other lean tissues that are typical in most adults with ageing. Second, the NHANES data set includes three main ethnic groups, non-Hispanic white, non-Hispanic black and Mexican American. For the same BMI and age, predicted %fat is smaller for non-Hispanic black individuals and larger for Mexican American individuals than it is for non-Hispanic white individuals [5^{*}]. These variations, reported for other race/ethnic groups, reflect differences in body shape and composition. A third factor influencing BMI–adiposity relations is physical activity levels. High levels of exercise, particularly weight training, increase skeletal muscle mass and through that mechanism (after controlling for body weight) reduce body fat. Thus, active exercisers have a lower %fat for the same BMI than sedentary adults. After controlling for these three BMI–adiposity effects, other likely factors include smoking, genetics and other mechanisms.

BMI AND MALNUTRITION

BMI is frequently used by various investigators and societies to determine the nutritional status and prognosis of patients, particularly in the cancer and surgical populations [7–20]. A recent study carried out in German geriatric trauma centres showed that 50% of the institutions relied on BMI to diagnose malnutrition [21]. Thus, one could argue that if there are data pointing to such nutritional predictive capacity of BMI, why should we be speculative about its role in the clinical setting?

Chris Anderson [22] has raised attention to an important aspect we should address when analysing data: “Scientists are trained to recognize that

correlation is not causation, that no conclusions should be drawn simply on the basis of correlation between X and Y (it could just be a coincidence). Instead, you must understand the underlying mechanisms that connect the two. Once you have a model, you can connect the data sets with confidence. Data without a model is just noise.’ Furthermore, in the modern era of petabytes, computers and statistics have made data interpretation easier but not necessarily more clinically fit, especially for individuals such as those presented herein, and caution should be the tone when interpreting them. In these regards, health data without clinical reasoning may jeopardize patients’ outcomes and treatments.

The Adjuvant Colon Cancer End Points database analysis of 25 291 patients showed that men with stage II or III disease who were either underweight or obese had overall survival, disease-free survival and time to recurrence results worse than those with an intermediate BMI and when compared with women with low BMI [23]. Renfro *et al.* [18] showed that low and high BMI (a U-curve shape) was associated with an increased risk of progression and death among colon cancer patients. Further, men with low BMI presented with poorer survival than women. With respect to these data, they may indicate that men with low BMI lost more weight than women and thus had a poorer nutritional status.

Another study encompassing surgical colorectal cancer patients and using multivariate analysis indicated that low BMI and stage III and IV disease were identified as independent prognostic factors for decreased survival. In addition, patients with low BMI presented with more severe postoperative complications and poorer prognoses. On the contrary, a higher risk of mild postoperative complications was seen in the high-BMI patients who paradoxically exhibited ‘superior’ survival outcomes compared with the normal-BMI patients. The authors attribute the findings to the ‘obesity paradox’ [10]. In this case, an interplay between advanced disease and loss of muscle mass as well as adiposity may explain the results, which have been well demonstrated to occur in cachexia syndrome. In the latter condition, a significant depletion of muscle mass is related to the inflammatory status triggered by the tumor, as well as, the production of tumor-related factors such as the proteolysis-inducing factor [24].

Among 106 resected pancreatic cancer individuals, length of hospital stay was associated with low BMI and serum albumin. In addition, low concentrations of albumin, node positivity and poor differentiation were associated with worse overall survival on multivariate analysis [25]. Pancreatic cancer patients are usually severely malnourished

at the time of diagnosis [26], with only 20% of them presenting resectable disease. This might explain why the results from Gavriilidou *et al.* [25] indicated that node positivity and poor tumour differentiation together with low albumin (all of them potentially associated with the aggressivity of the tumour) negatively impacted outcome variables.

On the contrary, Allard *et al.* [27], in a recent Canadian cohort study of hospitalized patients, did not show any association between low BMI and length of hospital stay, while those patients diagnosed as malnourished by subjective global assessment (SGA) or with low hand grip strength had an increased hospitalization time, after controlling for demographic, socioeconomic and disease-related factors and treatment.

In breast cancer patients at the beginning of chemotherapy, only 3.2% of the women were classified as being low weight according to BMI, but when assessed by SGA, 19.2% of them were malnourished [28]. Among Swedish elders, the prevalence of undernutrition in men and women was 3.9 and 8.6%, respectively, when assessed by knee height and demispans equations, compared with 2.4 and 5.4% by standard BMI. There was a more pronounced discrepancy for all women aged 85+ years (16.5 vs. 10% by standard BMI). The authors attribute their results to the inaccurate height estimation among the elderly [25].

In summary, the diagnosis of malnutrition, a highly prevalent syndrome that negatively impacts patients' morbidity, mortality, length of hospital stay and costs [29,30], may be jeopardized if BMI is used alone as previously discussed.

BMI LESS THAN 30 kg/m²: CAN WE IDENTIFY HARDY LOOKING AT LAUREL?

BMI is the main tool used to define obesity, although its limitation is well known [31]. On the basis of BMI, we have seen the dramatic increase of global obesity prevalence, from 105 million in 1975 to 641 million in 2014. If overweight is included, approximately 40% of the world's population and two out three Americans have BMI more than 25 kg/m² [32].

The data from NHANES 2005–2012 showed that 30% of individuals with normal BMI were cardiometabolically unhealthy and more than 74 million individuals would have a misclassification of their cardiometabolic health according to BMI categories [33]. These findings prove that the use of BMI as the general indicator of obesity (excessive fatness) is not suitable to diagnose, prevent and treat obesity-related conditions in clinical practice [34].

Table 1. Techniques of body composition according to ability to assess body fat distribution, precision and accuracy and availability in clinical practice

Body composition technique	Assessment of body fat distribution	Accuracy and precision	Use in clinical practice
Weight and BMI	No	+	++++
Circumferences (waist and hip)	Yes	+	++++
Skinfold thicknesses	Yes	++	++++
Bioelectrical impedance analysis	No	++	+++
Dual energy X-ray absorptiometry	No	+++	++
Computerized tomography	Yes	++++	++
MRI	Yes	++++	+

Adapted with permission from [35].

Although BMI is the most used tool to assess the excess of adiposity, not only is the amount of fat a determinant of risk associated with obesity but also fat distribution, the central or android distribution being associated with a higher risk than the peripheral or gynoid distribution. This abnormal fat distribution may be the reason explaining the higher metabolic risk found in individuals with normal weight [35]. The use of other measurements, such as waist circumference (WC), skin fold thickness, waist-to-hip ratio and waist-to-height ratio, can help to identify this anomalous distribution in clinical practice. Table 1 summarizes body composition techniques that can be used alone or in combination with BMI to increase its ability to identify body fatness.

BMI shows no good agreement in the assessment of obesity when compared with these methods. In a sample of cancer patients, it was shown that when obesity was defined by excess of fat mass using bioelectrical impedance analysis, BMI had 56% false-negative results in individuals classified as nonobese (BMI < 30) but with a high fat mass index [36]. Kjær *et al.* [37] showed that there was only a fair agreement between WC and BMI in the assessment of obesity in a national sample of Norwegians. In a cohort of patients undergoing coronary artery bypass grafting, patients in the upper WC quartile in each BMI category showed an increased risk of adverse effects after cardiac surgery when compared with the lower three WC quartiles, indicating that the risk associated with a higher adiposity can be found at any BMI [38].

Body composition techniques have shown that metabolic impairments can be found in any BMI

Table 2. Obesity classification, based in body mass index, body fat amount and distribution, risk for diseases and metabolic impairments

	Body fat	Risk for cardiometabolic disease	Metabolic impairments
Normal-weight obesity (NWO) BMI 18.5–24.9 kg/m ²	↑ ↑ body fat ↑ ↑ VAT/SAT ratio	Increased	Absent
Metabolically obese normal weight (MONW) or thin outside fat inside (TOFI) BMI 18.5–24.9 kg/m ²	↑ ↑ body fat ↑ ↑ VAT/SAT ratio	Increased	Present
Metabolically healthy obese (MHO) BMI ≥ 30 kg/m ²	↑ ↑ body fat Normal distribution	Absent	Absent
Metabolically unhealthy obese (MUO) BMI ≥ 30 kg/m ²	↑ ↑ body fat ↑ ↑ VAT/SAT ratio	Increased	Present

VAT/SAT, visceral adipose tissue/subcutaneous adipose tissue.
Adapted with permission from [35[■],39[■]].

category. On the basis of the presence of metabolic impairments and the amount and distribution of body fat and BMI, there are four different phenotypes to define obese individuals (Table 2). It can be seen that two of them have normal weight (BMI 18.5–24.9 kg/m²), and the difference between normal-weight obesity (NOW) and metabolic obese normal weight (MONW) is the metabolic impairment present in the last one, probably caused by the excessive visceral fat. Because of this special characteristic, the MONW is called ‘thin on the outside fat on the inside’ (TOFI). The metabolic healthy obese (MHO) is a new concept based on the findings that 15–45% of obese individuals show no signs of metabolic impairment, despite having a higher body fat mass [35[■],39[■]].

In conclusion, a BMI less than 30 kg/m² does not exclude the metabolic risks associated with an excess of adiposity. At any BMI, even with normal weight, an unhealthy metabolic profile may be present, and BMI alone is not enough for its identification. Other techniques of body composition assessment should be implemented in our clinical practice to better identify them.

BMI AT LEAST 30 kg/m²: CAN WE SEE LAUREL INSIDE HARDY?

There are several clinical situations in which skeletal muscle loss can occur in obese individuals, and simultaneously, there could be adipose tissue accretion, generating the condition known as ‘sarcopenic obesity’ or ‘sarcobesity’ [31,40[■]]. Sarcopenic obesity can also be called the ‘hidden’ sarcopenia because the underlying muscle loss is generally undetected when assessed by BMI alone. This condition is found not only as a consequence of age-related changes in

body composition but also in situations in which the combination of inactivity, inflammation and anorexia can determine the increase of fat mass and loss of skeletal muscle and dysfunction as in chronic diseases [41].

Sarcopenic obesity in ageing

As a reflection of the obesity pandemic, the prevalence of geriatric obesity is also increasing, and more than one-third of U.S. adults older than 60 years have a BMI at least 30 kg/m², reaching 40.8% in the range of 65–74 years [42]. The combination of inactivity, the ageing process and nutritional impairments leads to a higher risk of development of sarcopenic obesity in the elderly. This age-related muscle mass loss can be further exacerbated with weight reduction therapies alone [43]. Another risky situation is the obese old patient admitted at the hospital. The combination of bed rest, inadequate protein ingestion and a catabolic state can lead to a loss of almost 95 g/day of muscle mass [44]. In none of these situations can BMI identify the presence of sarcopenic obesity. The importance of its early diagnosis is that, as in other clinical situations, sarcopenic obesity seems to result in a synergistic negative effect in functional impairment [43]. For this reason, it is very important to perform sarcopenia screening and diagnosis in these older obese patients using the recommended criteria alongside BMI assessment [41] to prevent the development of sarcopenic obesity and promote early interventions.

Apart from the elderly obese, a large proportion of obese patients can be affected by several clinical conditions associated with the development of sarcopenia, such as those with cancer, in the ICU or with chronic diseases.

Sarcopenic obesity in cancer

During cancer investigation/treatment and its progression, abdominal computed tomography (CT) scans can be conveniently used to assess body composition as an opportunistic tool, and this approach has shown that obesity (BMI $\geq 30 \text{ kg/m}^2$) is frequently associated with skeletal muscle depletion and that BMI leads to a misclassification of these patients [45].

Although several tools can be used to assess sarcopenia (defined only as low muscle mass), there is no consensus about the definition of obesity itself (Table 3). For this reason, the prevalence of sarcopenic obesity can vary if based on BMI only (15–36% in patients with BMI $> 25 \text{ kg/m}^2$) or on excess of body fat assessed by other body composition tools (1–29%) [46^{□□}]. CT is an opportunistic body composition tool in cancer patients, as muscle mass and adipose tissue and its distribution can be easily estimated from a scan at the third lumbar vertebra [47^{□□}].

Sarcopenic obesity in cancer is associated with a decrease in functionality and negative clinical outcomes, such as higher mortality, dose-limiting toxicity associated with chemotherapy, worse post-operative outcomes and longer hospitalization [36,46^{□□},48^{□□}]. Lou *et al.* [49] showed that the presence of sarcopenia, defined as low muscle mass, low strength and low performance, can increase six times the risk of postoperative complications in gastric cancer patients.

Kazemi-Bajestani *et al.* [50^{□□}] reviewed studies that used CT to assess muscle and fat mass in cancer patients and tried to explain their relationship with negative outcomes (chemotherapy toxicity, post-surgery complications and survival). The studies reviewed showed that muscle depletion was seen

in patients with any BMI. The adverse outcomes were significantly related to muscle depletion, and sarcopenic obesity had a strong association with poor survival. This review indicated the superiority of CT for the assessment of muscle mass and fat in cancer patients, instead of only weight and BMI.

Sarcopenic obesity in ICU and chronic diseases

One-third of critically ill patients are obese [51[□]], and these patients undergo all of the other determinants of skeletal muscle loss: systemic inflammation, hypermetabolism and catabolism, inactivity and insufficient protein intake. Segaran *et al.* [52] showed that a high BMI did not prevent muscle loss in the ICU, and muscle loss was approximately 1.6% per day. The combination of a high prevalence of obesity and muscle loss can be the cause for the development of sarcopenic obesity in these patients, and again, the high BMI would mask its identification. Although the negative effects of sarcopenic obesity are not well known in ICU patients, obese patients should have a complete nutritional assessment and sarcopenia screening as any other patient, and they should receive special attention regarding an adequate amount of protein and energy intake.

Sarcopenic obesity in the ICU can be even more hidden if the patient is older. Ageing is the primary determinant factor for the development of sarcopenia, and patients 65 years and older were responsible for more than half of the occupancy of ICU days in the United States in 2000 [53]. Sarcopenic obesity in the elderly can be found at any BMI because of the simultaneous changes in fat and muscle during the ageing process. Therefore, this condition should be considered independently of BMI, so that preventive measures, such as early mobility and protein supplements, can be delivered to avoid the appearance of sarcopenia-related disability.

Body composition tools are not routinely available for the ICU population, and BMI does not give all the information to identify sarcopenic obesity and its relationship with outcomes. Simple measurements, such as mid-arm muscle circumference, assessment of fat loss and muscle wasting by using the SGA seem to be better predictors of mortality than BMI alone [54,55[□]]. Ultrasound and abdominal CT scans acquired during routine care are new emerging tools for body composition assessment in ICU patients, but further validation of these techniques in this population is still needed.

Sarcopenic obesity is also prevalent in other chronic diseases besides cancer and ICU patients. Joppa *et al.* [56[□]] showed that the prevalence of sarcopenic obesity is 2.5 times higher in chronic

Table 3. Methods used in the assessment of obesity and sarcopenia for sarcopenic obesity diagnosis

	Method	Assessment
Obesity (excess of body fat)	Anthropometry	BMI
	Abdominal CT scan	% body fat or visceral fat area
	DXA or BIA	Fat mass index
Sarcopenia (low muscle mass)	Abdominal CT scan	Skeletal muscle index
	DXA	Appendicular skeletal muscle index
	BIA	Fat-free mass index

BIA, Bioelectrical impedance analysis; CT, computerized tomography; DXA, dual-energy X-ray absorptiometry. Adapted with permission from [46^{□□}].

obstructive pulmonary disease patients than in controls (10 vs. 4%, respectively), and sarcopenic obesity has a negative impact on physical performance. It is noteworthy that the mean BMI for sarcopenic obese men and women was 27.9 and 23.7 kg/m², respectively, giving no clue about body composition abnormalities. In patients with cirrhosis assessed by CT, sarcopenic obesity was present in 20% of the sample, and it was independently associated with a worse survival [57[¶]]. The mean BMI of sarcopenic obese patients was 30 ± 0.5 kg/m², not significantly different from patients with no muscular abnormalities, showing again the uselessness of BMI in this situation.

CONCLUSION

BMI, a traditional tool used for the screening and assessment of malnutrition, obesity and healthy body composition among populations, is not appropriate to differentiate between components of body weight, and it does not appear to be a reliable marker of adiposity at the individual level. Simple questions about weight loss, inadequate food intake or loss of appetite, independently of a BMI value, is a better means of screening for and/or assessing malnutrition. The use of new body composition tools may help identify metabolic risk by assessing body fat and its distribution as well as the loss of muscle mass associated with malnutrition and sarcopenia.

Acknowledgements

None.

Financial support and sponsorship

None.

Conflicts of interest

There are no conflicts of interest.

REFERENCES AND RECOMMENDED READING

Papers of particular interest, published within the annual period of review, have been highlighted as:

- of special interest
- of outstanding interest

1. van Bokhorst-de van der Schueren MA, Guaitoli PR, Jansma EP, *et al.* Nutrition screening tools: does one size fit all? A systematic review of screening tools for the hospital setting. *Clin Nutr* 2014; 33:39–58.
2. World Health Organization. Obesity and overweight. Fact sheet N 311 2006. <http://www.who.int/mediacentre/factsheets/fs311/en/>. [Accessed 4 October 2017]
3. Correia MIT. Addressing the hidden burden of malnutrition for hospitalized patients. *J Am Med Dir Assoc* 2017. [Epub ahead of print]
4. Soeters P, Bozzetti F, Cynober L, *et al.* Defining malnutrition: a plea to rethink. *Clin Nutr* 2017; 36:896–901.
5. Heymsfield SB, Peterson CM, Thomas DM, *et al.* Why are there race/ethnic differences in adult body mass index-adiposity relationships? A quantitative critical review. *Obes Rev* 2016; 17:262–275.
6. Keys A, Fidanza F, Karvonen MJ, *et al.* Indices of relative weight and obesity. *J Chronic Dis* 1972; 25:329–343.
7. Acharya M, Harling L, Moscarelli M, *et al.* Influence of body mass index on outcomes after minimal-access aortic valve replacement through a J-shaped partial upper sternotomy. *J Cardiothorac Surg* 2016; 11:74.
8. Balakrishnan VS. Low BMI linked to worse colorectal cancer outcomes. *Lancet Oncol* 2015; 16:e593.
9. Cederholm T, Bosaeus I, Barazzoni R, *et al.* Diagnostic criteria for malnutrition – an ESPEN consensus statement. *Clin Nutr* 2015; 34:335–340.
10. Chen HN, Chen XZ, Zhang WH, *et al.* The impact of body mass index on the surgical outcomes of patients with gastric cancer: a 10-year, single-institution cohort study. *Medicine (Baltimore)* 2015; 94:e1769.
11. Hendifar A, Osipov A, Khanuja J, *et al.* Influence of body mass index and albumin on perioperative morbidity and clinical outcomes in resected pancreatic adenocarcinoma. *PLoS One* 2016; 11:e0152172.
12. Jensen GL. Malnutrition and inflammation: 'burning down the house' inflammation as an adaptive physiologic response versus self-destruction? *JPEN J Parenter Enteral Nutr* 2015; 39:56–62.
13. Jensen GL, Compher C, Sullivan DH, *et al.* Recognizing malnutrition in adults: definitions and characteristics, screening, assessment, and team approach. *JPEN J Parenter Enteral Nutr* 2013; 37:802–807.
14. Jun DH, Kim BJ, Park JH, *et al.* Preoperative body mass index may determine the prognosis of advanced gastric cancer. *Nutr Cancer* 2016; 68:1295–1300.
15. Kroenke CH, Neugebauer R, Meyerhardt J, *et al.* analysis of body mass index and mortality in patients with colorectal cancer using causal diagrams. *JAMA Oncol* 2016; 2:1137–1145.
16. Lee YL, Li WC, Tsai TH, *et al.* Body mass index and cholesterol level predict surgical outcome in patients with hepatocellular carcinoma in Taiwan: a cohort study. *Oncotarget* 2016; 7:22948–22959.
17. Nakagawa T, Toyazaki T, Chiba N, *et al.* Prognostic value of body mass index and change in body weight in postoperative outcomes of lung cancer surgery. *Interact Cardiovasc Thorac Surg* 2016; 23:560–566.
18. Renfro LA, Loupakis F, Adams RA, *et al.* Body mass index is prognostic in metastatic colorectal cancer: pooled analysis of patients from first-line clinical trials in the ARCAD database. *J Clin Oncol* 2016; 34:144–150.
19. Wada T, Kunisaki C, Ono HA, *et al.* Implications of BMI for the prognosis of gastric cancer among the Japanese population. *Dig Surg* 2015; 32:480–486.
20. Wu N, Zhu Y, Kadel D, *et al.* The prognostic influence of body mass index, resting energy expenditure and fasting blood glucose on postoperative patients with esophageal cancer. *BMC Gastroenterol* 2016; 16:142.
21. Eschbach D, Kirchbichler T, Oberkircher L, *et al.* Management of malnutrition in geriatric trauma patients: results of a nationwide survey. *Eur J Trauma Emerg Surg* 2016; 42:553–558.
22. Anderson C. The end of theory: the data deluge makes the scientific method obsolete. *Wired Magazine* 2008; 16:16–17.
23. Sinicrope FA, Foster NR, Yothers G, *et al.* Body mass index at diagnosis and survival among colon cancer patients enrolled in clinical trials of adjuvant chemotherapy. *Cancer* 2013; 119:1528–1536.
24. Ryan AM, Power DG, Daly L, *et al.* Cancer-associated malnutrition, cachexia and sarcopenia: the skeleton in the hospital closet 40 years later. *Proc Nutr Soc* 2016; 75:199–211.
25. Gavrilidou NN, Pihlsgård M, Elmståhl S. High degree of BMI misclassification of malnutrition among Swedish elderly population: age-adjusted height estimation using knee height and demispan. *Eur J Clin Nutr* 2015; 69:565–571.
26. Gilliland TM, Villafane-Ferriol N, Shah KP, *et al.* Nutritional and metabolic derangements in pancreatic cancer and pancreatic resection. *Nutrients* 2017; 9:243.
27. Allard JP, Keller H, Jeejeebhoy KN, *et al.* Malnutrition at hospital admission: contributors and effect on length of stay: a prospective cohort study from the Canadian Malnutrition Task Force. *JPEN J Parenter Enteral Nutr* 2016; 40:487–497.
28. Bering T, Mauricio SF, Silva J, *et al.* Nutritional and metabolic status of breast cancer women. *Nutr Hosp* 2015; 31:751–758.
29. Brito PA, de Vasconcelos Generoso S, Correia MITD. Prevalence of pressure ulcers in hospitals in Brazil and association with nutritional status: a multicenter, cross-sectional study. *Nutrition* 2013; 29:646–649.
30. Correia MI, Perman MI, Waitzberg DL. Hospital malnutrition in Latin America: a systematic review. *Clin Nutr* 2016; Jul 19. pii: S0261-5614(16)30160-1. doi: 10.1016/j.clnu.2016.06.025. [Epub ahead of print]
31. Laurel & Hardy: the official website. <http://www.laurel-and-hardy.com>. [Accessed 4 December 2017]
32. Maffetone PB, Rivera-Dominguez I, Laursen PB. Overfat and underfat: new terms and definitions long overdue. *Front Public Health* 2016; 4:279. The authors discussed the BMI limitations as a measure of obesity, proposed new terms based in the amount of adipose tissue (overfat/underfat) and discuss the different phenotypes of overfat, independently of weight or BMI.
33. Tomiyama A, Hunger J, Nguyen-Cuu J, *et al.* Misclassification of cardiometabolic health when using body mass index categories in NHANES 2005–2012. *Int J Obes (Lond)* 2016; 40:883–886. This study showed how cardiometabolic health can be misclassified based only in BMI, using data from NHANES 2005–2012. Using BMI categories, almost 75 million US adults would be classified in a erroneous cardiometabolic status.

This review presents a critical evaluation of the differences found in the relations between BMI and adiposity according to different races and ethnicities and across adult age span.

34. St-Onge MP. Are normal-weight Americans over-fat? *Obesity* 2010; 18:2067–2068.
35. De Lorenzo A, Soldati L, Sarlo F, *et al.* New obesity classification criteria as a tool for bariatric surgery indication. *World J Gastroenterol* 2016; 22:681–703. This is an excellent review, wherein the authors discussed the problems of using BMI to identify obesity due to the four phenotypes of obesity. A new term 'adiposopathy' is suggested, to highlight the pathogenic role of adipose tissue. They also discuss the body composition methods and the importance of identifying the excess of body fat as a risk factor of adiposopathy.
36. Gonzalez MC, Pastore CA, Orlandi SP, *et al.* Obesity paradox in cancer: new insights provided by body composition. *Am J Clin Nutr* 2014; 99:999–1005.
37. Kjær I, Kolle E, Hansen B, *et al.* Obesity prevalence in Norwegian adults assessed by body mass index, waist circumference and fat mass percentage. *Clin Obes* 2015; 5:211–218.
38. Chassé M, Mathieu P, Voisine P, *et al.* The underestimated belly factor: waist circumference is linked to significant morbidity following isolated coronary artery bypass grafting. *Can J Cardiol* 2016; 32:327–335.
39. Müller MJ, Braun W, Enderle J, *et al.* Beyond BMI: conceptual issues related to overweight and obese patients. *Obes Facts* 2016; 9:193–205. This is a critical review about the use of BMI as a measure of adiposity and its limitations. The authors also reviewed all the body composition tools, obese phenotypes and suggested the concept of functional body composition.
40. Andreoli A, Garaci F, Cafarelli FP, *et al.* Body composition in clinical practice. *Eur J Radiol* 2016; 85:1461–1468. This is an excellent review about all the body composition methods used in clinical practice, and the implications of body composition assessment.
41. Biolo G, Cederholm T, Muscaritoli M. Muscle contractile and metabolic dysfunction is a common feature of sarcopenia of aging and chronic diseases: from sarcopenic obesity to cachexia. *Clin Nutr* 2014; 33:737–748.
42. Starr KNP, Bales CW. Excessive body weight in older adults. *Clin Geriatr Med* 2015; 31:311–326.
43. Weinheimer EM, Sands LP, Campbell WW. A systematic review of the separate and combined effects of energy restriction and exercise on fat-free mass in middle-aged and older adults: implications for sarcopenic obesity. *Nutr Rev* 2010; 68:375–388.
44. Paddon-Jones D, Leidy H. Dietary protein and muscle in older persons. *Curr Opin Clin Nutr Metab Care* 2014; 17:5–11.
45. Martin L, Birdsell L, MacDonald N, *et al.* Cancer cachexia in the age of obesity: skeletal muscle depletion is a powerful prognostic factor, independent of body mass index. *J Clin Oncol* 2013; 31:1539–1547.
46. Carneiro IP, Mazurak VC, Prado CM. Clinical implications of sarcopenic obesity in cancer. *Curr Oncol Rep* 2016; 18:62. This review included 14 studies and the authors discussed important aspects of sarcopenic obesity, from its definition to its implications in cancer treatment. The authors highlighted the need of a well defined diagnostic criteria and better knowledge of their nutritional need.
47. Prado C, Cushen S, Orsso C, *et al.* Sarcopenia and cachexia in the era of obesity: clinical and nutritional impact. *Proc Nutr Soc* 2016; 75:188–198. The authors discussed how computerized tomography increased the knowledge about the body composition in cancer patients, and how variable body composition can be in these patients. They also showed the clinical implications of this abnormal body composition in the several steps of cancer treatment.
48. Peterson SJ, Mozer M. Differentiating sarcopenia and cachexia among patients with cancer. *Nutr Clin Pract* 2017; 32:30–39. The difference between sarcopenia and cachexia, two wasting conditions present in cancer patients, is discussed in this review. Their cause, diagnostic criteria, prevalence and treatment are compared to help their recognition in patients with cancer.
49. Lou N, Chi C-H, Chen X-D, *et al.* Sarcopenia in overweight and obese patients is a predictive factor for postoperative complication in gastric cancer: a prospective study. *Eur J Surg Oncol* 2017; 43:188–195.
50. Kazemi-Bajestani SMR, Mazurak VC, Baracos V. Computed tomography-defined muscle and fat wasting are associated with cancer clinical outcomes. *Semin Cell Dev Biol* 2016; 54:2–10. This review showed how computerized tomography can help the identification of muscle and fat-wasting in cancer patients and the relationship between these features and clinical outcomes. The authors showed the results from 53 studies, including 9138 patients and once more the association between muscle loss and adverse outcomes is confirmed.
51. Patel JJ, Rosenthal MD, Miller KR, *et al.* The critical care obesity paradox and implications for nutrition support. *Curr Gastroenterol Rep* 2016; 18:1–8. The authors discussed several aspects of obesity and the challenges associated with obese patient and critical illness. The obesity paradox found in these patients is discussed, mainly the pitfalls of using only BMI to define obesity in these patients.
52. Segaran E, Wandrag L, Stotz M, *et al.* Does body mass index impact on muscle wasting and recovery following critical illness? A pilot feasibility observational study. *J Hum Nutr Diet* 2017; 30:227–235.
53. Hanna JS. Sarcopenia and critical illness a deadly combination in the elderly. *J Parenter Enteral Nutr* 2015; 39:273–281.
54. Fontes D, de Vasconcelos Generoso S, Correia MITD. Subjective global assessment: a reliable nutritional assessment tool to predict outcomes in critically ill patients. *Clin Nutr* 2014; 33:291–295.
55. Simpson F, Doig GS. Bedside nutrition evaluation and physical assessment techniques in critical illness. *Curr Opin Crit Care* 2016; 22:303–307. The authors showed an update in tools used to assess lean and fat mass at bedside in an ICU, highlighting the new techniques, ultrasound and abdominal CT scans.
56. Joppa P, Tkacova R, Franssen FM, *et al.* Sarcopenic obesity, functional outcomes, and systemic inflammation in patients with chronic obstructive pulmonary disease. *J Am Med Dir Assoc* 2016; 17:712–718. By using data from a large longitudinal study with COPD patients (ECLIPSE), the authors showed that sarcopenia (low muscle mass, low force and low performance) associated with obesity was associated with the worse physical performance and higher inflammatory burden.
57. Montano-Loza AJ, Angulo P, Meza-Junco J, *et al.* Sarcopenic obesity and myosteatosis are associated with higher mortality in patients with cirrhosis. *J Cachexia Sarcopenia Muscle* 2016; 7:126–135. In this longitudinal study, 678 cirrhotic patients who had their body composition analysed by using abdominal computerized tomography, the authors showed that patients with sarcopenia, sarcopenic obesity and myosteatosis had the worse survival.