Use of Body Fatness Cutoff Points

To the Editor: In the July 2010 editorial of *Mayo Clinic Proceedings*, although correctly making the argument that "BMI [body mass index; calculated as weight in kilograms divided by height in meters squared] does not reflect true body fatness," Lavie et al[1] refer to the "National Institutes of Health [NIH] criterion standards" for percent body fat (BF) as greater than 25% in men and greater than 35% in women. The reference provided for this statement is a pamphlet[2] for the general public, issued by the Weight Information Network (WIN), an NIH initiative to provide "science-based information on weight control, obesity, physical activity, and related nutritional issues." Although I did not find any mention of body fat (BF) cutoff points in that publication, an earlier version[3] did state that "Most health care providers agree that men with more than 25 percent body fat and women with more than 30 percent body fat are considered obese." Note the discrepancy in cutoff for women between the editorial and the pamphlet.

The discrepancy in cutoff for women aside, assuming that the earlier version is the intended citation, it is a bit of a stretch to elevate an unreferenced statement from a WIN pamphlet to an "NIH criterion standard." Moreover, as one of the authors of the editorial recently stated, "Unfortunately, neither the World Health Organization nor any major scientific society involved in the study of obesity has defined a normal value for BF%."[4]

Regarding BMI, the World Health Organization and NIH cutoff point of 25 was chosen because, in most epidemiological studies, mortality in both men and women begins to increase above this value,[5] ie, there is evidence of a threshold effect. By contrast, there is little if any evidence to support that cutoff points of 25% in men and 35% (or 30%) in women are the optimal values for BF-based risk stratification. In the absence of a substantial body of literature characterizing the sex-specific relation between a continuum of BF percent values and morbidity and mortality, as well as potential moderating effects of age and race, the choice of BF percent cutoff points in research or clinical practice remains a highly subjective decision.

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In reply: We appreciate Dr Snitker's interest in our recent editorial[1] and his insightful comments regarding body fat (BF) and obesity. Additionally, we are aware of his clinical and research efforts in the area of obesity in children at the University of Maryland School of Medicine. Dr Snitker is correct that currently there is no definitive cutoff for percent BF in defining overweightness or obesity in men or women. In our efforts to simplify the message for readers, we referenced an easily accessible National Institutes of Health (NIH) publication[2] that we thought was representative. Generally, we have referenced a major source from the World Health Organization (WHO)[3] as opposed to this simple NIH Web site in our research publications from Ochsner Clinic[4] and Mayo Clinic.[5,6] However, we agree with Snitker that, regardless of the reference, there is no criterion standard for defining overweightness or obesity by the BF method.

We previously demonstrated in a cross-sectional design of 13,601 participants (age, 20-80 years; 48% men) from the Third National Health and Nutrition Examination Survey (NHANES III)[6] that the mean ± SD body mass index (BMI; calculated as weight in kilograms divided by height in meters squared) in men was 26.6±4.6 and the mean ± SD percent BF was 24.8%±6.0%. Corresponding values in women were 27.6±6.4 and 36.7%±7.4%, respectively. In 6171 participants in NHANES III who had a BMI in the reference range (18.5-24.9), the highest tertile of BF was greater than 23.1% in men and greater than 33.3% in women (labeled as *normal weight obesity*).[7] In this cohort with normal weight obesity defined by elevated BF, the prevalence of metabolic syndrome was 4 times higher than in those with low BF, and these individuals had a higher prevalence of dyslipidemia (men and women) and of hypertension (men) and a 2.2-fold increased risk of cardiovascular (CV) mortality (women) compared with those with low BF. These data suggest that this level of BF is associated with adverse CV risk and prognosis in primary prevention.

In secondary prevention, having increased BF (>25% in men and >35% in women) appears to be associated with a protective effect in patients with coronary heart disease (CHD).[4] In fact, in patients with CHD[4,8] and in those with heart failure,[9,10] a higher BF was an independent predictor of event-free survival because of the *obesity paradox*, which we discussed in our editorial.[1] Oreopoulos et al[11] in their heart

failure study used a Gallager classification of BF based on age, sex, and race and classified patients as underweight, normal, overweight, and obese.[12] In preliminary data from our CHD population (n=581) using this Gallager classification, we have found the highest mortality in the underweight and lowest mortality in the overweight, who also had significantly lower mortality than the "normal BF" group during a 3-year follow-up (A.D.S, C.J.L, and R.V.M., unpublished observations, May 1, 2010). The obese group had intermediate mortality, which was significantly lower than the underweight and trended lower than the normal BF group but did not reach statistical significance.

Therefore, current research suggests that the obesity cutoff points of BF are in the 23%-25% range in men and 33%-35% range in women (or 30% in women from the NIH,[2] as Dr Snitker stated in his letter), which are associated with increased CV risk in primary prevention and reduced risk in patients with established CV disease (obesity paradox). However, we agree that additional research is needed to clearly define optimal BF in patients of both sexes and of various ages, races, and ethnic groups, as well as disease states. Clearly, major organizations such as the WHO, NIH, and major obesity societies should attempt to establish such cutoff points for BF, as was done years ago with BMI.

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Does Vitamin D Have a Role in Reducing the Risk of Peripheral Artery Disease?

To the Editor: In their otherwise excellent review, Olin and Sealove[1] did not consider the role of vitamin D status in the development of peripheral artery disease (PAD).

Vitamin D deficiency is highly prevalent in the United States and worldwide. In particular, a recent study reported that vitamin D levels were independently associated with PAD among 4839 participants of the National Health and Nutrition Examination Survey 2001 to 2004.[2] For each decrease of 10 ng/mL in the 25-hydroxyvitamin D level, the multivariable-adjusted prevalence ratio of PAD was 1.35 (95% confidence interval, 1.15-1.59).[2] Furthermore, racial differences in vitamin D concentrations could explain nearly one-third of the excess risk of PAD in black adults, above and beyond differences in established and novel risk factors for cardiovascular disease.[3]

Several mechanisms may explain the association of vitamin D deficiency with PAD. Low vitamin D status is associated with obesity, diabetes, and hypertension, all of which increase the risk of PAD. However, the inverse relationship between vitamin D status and PAD remained after adjustment for these risk factors, suggesting additional explanatory mechanisms.[3]

Vitamin D receptors have a broad distribution that includes vascular smooth cells, macrophages, and lymphocytes. Directly or indirectly, 1,25-dihydroxyvitamin D (the active form of vitamin D) regulates the expression of a number of proteins relevant to the arterial wall, such as vascular endothelial growth factor, matrix metalloproteinase type 9, myosin, elastin, and type I collagen.[4]