

“Obesity Paradox” or a Better Nutritional Status?

To the Editor:

We read with great interest the recent article by Uretsky et al¹ about obesity’s impact on survival in patients with cardiovascular disease. However, some aspects of the study are worthy of comment.

First, the study has serious limitations, such as the short-term follow-up and the younger age of obese patients compared with normal-weight patients. We suggest that the consequences of obesity on mortality could be delayed and not observed in short-term studies. When long-term outcome is evaluated, it should take into account all classical risk factors related to obesity. Eilat-Adar et al² reported a purposeful weight reduction in obese patients with cardiovascular disease, with a lower long-term incidence of cardiovascular events, despite the “obesity paradox.”

Second, the authors discussed the cardioprotective role of soluble TNF α “antagonist” receptors produced by adipose tissue.³ However, if adipose tissue releases these inhibitors, it also produces pro-inflammatory cytokines and inflammatory mediators, as recently observed by our group.⁴ In addition, intentional weight loss reduced the production of pro-inflammatory cytokines by adipose tissue and increased the expression of anti-inflammatory cytokines (ie, IL-10).⁵

Third, body mass index (BMI) is not a direct measure of body composition and does not reflect the body muscle mass content, reduced in patients with chronic diseases.⁶ Subjects with a well-represented muscle mass show a lower cardiovascular mortality.⁷

In conclusion, we suggest that an association between higher BMI and better survival in patients with cardiovascular disease should be re-evaluated in concert with nutritional status assessment. Higher BMI may confer a survival advantage if muscle mass is well represented. We recom-

mend easy and noninvasive body composition methods (such as bioelectrical impedance analysis) to more accurately evaluate the relationship between BMI and mortality.

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