Letter by Malavazos et al Regarding Article, “Sweetened Beverage Consumption, Incident Coronary Heart Disease, and Biomarkers of Risk in Men”

To the Editor:

We read with great interest the paper entitled “Sweetened Beverage Consumption, Incident Coronary Heart Disease, and Biomarkers of Risk in Men” by de Koning et al.1 As stated by the authors,1 the consumption of sugar sweetened beverages (SSB), but not artificially sweetened beverages, was associated with a significantly increased risk of coronary heart disease and some adverse changes in blood lipids and inflammatory adipocytokines.

In the study by de Koning et al1 the association between SSB consumption and the increased risk of coronary heart disease remained significant also after adjusting for a number of possible confounders, including overall diet quality and body mass index (BMI). However, BMI values were similar in subjects from all SSB consumption quartiles.1 Indeed, BMI is a useful index of weight excess in population-based and clinical studies, but it does not distinguish between fat and fat-free mass or between visceral and subcutaneous fat. Rather than BMI, waist circumference and waist-to-hip ratio, as expression of visceral adipose tissue (VAT) accumulation, are more reliable parameters to determine and quantify body fat distribution. Therefore, some clinical guidelines recommend waist circumference measurement as a tool providing additional and more precise information regarding cardiovascular risk.2 We are suggesting that subjects with different SSB consumption habits and similar BMI may also have a different adipose tissue distribution, which may be quantified by commonly used surrogates of visceral adiposity.

Lately, as postulated by Stanhope and colleagues,3 high fructose consumption, the major sweetener in SSB, increases both de novo lipogenesis and insulin resistance in the liver. The consequent lower activation of lipoprotein lipase in subcutaneous adipose tissue increases availability of triglycerides and their uptake into VAT.3 This dietary fructose–visceral fat relationship has also been reported in a recent article by Pollock et al,4 who conclude that greater fructose consumption is associated with multiple markers known to increase the risk for cardiovascular disease, such as blood lipids and inflammatory adipocytokines, suggesting that these relationships are dependent on visceral obesity.

As a matter of fact, in the study by de Koning et al,1 SSB consumption was also associated with increased levels of several circulating inflammatory factors.

 Nowadays, the distribution assessment of adipose tissue is notable because increased VAT is linked to a high risk of cardiovascular disease resulting from a low-grade inflammation state: increased VAT becomes infiltrated with macrophages, and a variety of proinflammatory adipocytokines, such as tumor necrosis factor α, interleukin 6, and monocyte chemoattractant protein-1, are overexpressed.5

In this context, we suggest that VAT, rather than BMI, should be considered as a concurrent factor in the association between SSB consumption and increased risk of coronary heart disease. According to this, it would be newsworthy to perform further adjustments for baseline and follow-up changes in fat distribution indexes, and not only for BMI, which may scarcely take into account the herein suggested relationship. Taken together, these findings prompt that information about body fat distribution, such as waist circumference, may provide important insights into metabolic and cardiovascular disease risk.

Disclosures

None.

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References

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