To the Editor:

I read with interest the recent report by Shamsuzzaman et al of elevated C-reactive protein (CRP) in patients with obstructive sleep apnea (OSA). Although the patients and control subjects were matched for body mass index (BMI), 36±4 kg/m² versus 34±4 kg/m², all were very obese (upper normal BMI=25 kg/m²).

Several recent studies have indicated that CRP levels are elevated in obese patients. Adiposity, in particular visceral adipose tissue, is a key promoter of low-grade chronic inflammation. The central distribution of body fat as evaluated by waist circumference is a stronger predictor of CRP levels than total adiposity as evaluated by BMI.

Thus, it would be of interest to know the differences in the fat distribution between the Mayo Clinic patients with OSA and the control subjects. Shinohara et al reported a relatively higher waist circumference and a greater amount of abdominal visceral fat accumulation in obese patients with OSA. Therefore, one should not compare apples with oranges; perhaps, I should say, one should not compare (android obesity) with pears (gynecoid obesity).

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Response

We appreciate Dr Cheng’s interest in our work. There is emerging evidence of a number of variables that may influence C-reactive protein (CRP) levels, including, as Dr Cheng points out, obesity, visceral adiposity, and sex. Patients with increased abdominal visceral fat may indeed have a higher prevalence of obstructive sleep apnea (OSA). It is important to consider whether higher levels of CRP in subjects with visceral obesity may be a consequence of the visceral obesity or the sleep apnea. We believe that both conditions may conceivably be implicated. Prior studies linking visceral obesity to CRP levels have not excluded the effects of the presence of OSA.

In our study, patients with OSA had body mass indexes (BMIs) and percent body fat similar to controls. Although waist-to-hip ratio was higher in patients with OSA than controls (0.998 versus 0.905, P=0.0002), multivariate analysis showed that the association between elevated CRP and OSA remained statistically significant after adjustment for age, sex, BMI, and waist-to-hip ratios (F=8.07, P=0.008). By contrast, after adjusting for age, sex, BMI, and apnea hypopnea index, there was no significant association between waist-to-hip ratio and CRP (F=3.1, P=0.09). These findings speak to the broader concept of including the possible confounding effects of occult sleep apnea in studies addressing the pathophysiological mechanisms linking obesity and fat distribution to cardiovascular disease.

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Could Elevated C-Reactive Protein in Patients With Obstructive Sleep Apnea Be Due to Obesity per se?
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