metabolic effects (4). The relationships between OSA treatment and subsequent obesity levels are complex. For example, in a 6-month randomized controlled trial conducted by Quan and colleagues, treatment with CPAP resulted in weight gain (5). They speculated that CPAP induced a state of positive energy balance that resulted in weight gain and recommended that patients with obesity and OSA undergoing CPAP treatment should engage in increased physical activity and reduction in caloric intake to lose weight. Weight loss is effective in patients with mild-to-moderate OSA (6), and excess deposition of fat tissue in upper airways should also be controlled for improvement in OSA. ■

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Tomoyuki Kawada, M.D., Ph.D.*
Nippon Medical School
Tokyo, Japan

*Corresponding author (e-mail: kawada@nms.ac.jp).

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Reply to Kawada

From the Authors:

We thank Dr. Kawada for his interest in our study "Effect of Weight Loss on Upper Airway Anatomy and the Apnea–Hypopnea Index: The Importance of Tongue Fat" (1).

Dr. Kawada's first concern about our study was related to a finding in a study by Sutherland and colleagues (2). This study showed that improvements in the apnea–hypopnea index (AHI) with weight loss were associated with reductions in upper airway length and visceral abdominal fat. However, in our study (1), analyses showed that reductions in tongue fat were the primary mediator connecting weight loss to a reduction in AHI, not reductions in abdominal fat volumes. In our study (1), reductions in abdominal fat were correlated with reductions in AHI (Table 3 from Reference 1), similar to the data in Sutherland and colleagues' study (2). However, a mediation analysis (which was not performed in Sutherland and colleagues' study) (2) found that percentage reduction in tongue fat volume was a significant mediator between percentage change in weight and AHI, accounting for approximately 30% of the overall effect of weight loss. A percentage change in airway size or abdominal fat volumes did not mediate this relationship (Table 4 from Reference 1). Thus, we believe our data agree with Dr. Kawada's premise that reductions in abdominal fat are strongly correlated with changes in AHI, but the key mediator of this change is reduction in tongue fat, not reductions in abdominal fat.

Dr. Kawada's second concern relates to the bidirectional relationship between sleep apnea and obesity. We agree with Dr. Kawada that not only is obesity the primary risk factor for sleep apnea, but also sleep apnea and treatment with continuous positive airway pressure can promote weight gain (3, 4). Thus, patients may benefit from additional treatment options aimed at weight loss or, based on the results from our study, reducing fat in the tongue. As we do not know how different treatment modalities (including continuous positive airway pressure, oral appliances, hypoglossal nerve stimulation, surgery, or upper airway exercises) affect tongue fat, this is an important area of research. Other studies have shown that exercise can lead to weight loss and specific upper airway exercises (which have been shown to decrease AHI) may reduce tongue fat (5–7). We believe that this is a fruitful area for additional study. ■

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Richard J. Schwab, M.D.*
Brendan T. Keenan, M.S.
University of Pennsylvania
Philadelphia, Pennsylvania

David B. Sarwer, Ph.D.
College of Public Health at Temple University
Philadelphia, Pennsylvania

Allan I. Pack, M.B. Ch.B., Ph.D.
University of Pennsylvania
Philadelphia, Pennsylvania

*Corresponding author (e-mail: rschwab@mail.med.upenn.edu).

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