To Salt or Not to Salt? Is That a Question in Obstructive Sleep Apnea?

Takatoshi Kasai, M.D., Ph.D.[1,2,3], and Yüksel Peker, M.D., Ph.D.[4,5,6,7*]

[1]Department of Cardiovascular Medicine and [2]Cardiovascular Respiratory Sleep Medicine, Juntendo University Graduate School of Medicine, Tokyo, Japan; [3]Sleep and Sleep Disordered Breathing Center, Juntendo University Hospital, Tokyo, Japan; [4]Department of Pulmonary Medicine, Koc University School of Medicine, Istanbul, Turkey; [5]Department of Molecular and Clinical Medicine/Cardiology, Sahlgrenska Academy, University of Gothenburg, Gothenburg, Sweden; [6]Department of Clinical Sciences, Respiratory Medicine and Allergology, Faculty of Medicine, Lund University, Lund, Sweden; and [7]Division of Pulmonary, Allergy, and Critical Care Medicine, University of Pittsburgh School of Medicine, Pittsburgh, Pennsylvania

ORCID IDs: 0000-0001-5747-7668 (T.K.); 0000-0001-9067-6538 (Y.P.).

Obstructive sleep apnea (OSA) is a common chronic condition (1) that is observed in 10–20% of the general population (2). Although continuous positive airway pressure (CPAP) has been established as an effective first-line therapy for moderate to severe OSA, adherence to CPAP is challenging in many cases, especially among asymptomatic or minimally symptomatic patients with OSA with concomitant cardiovascular diseases (3). Therefore, more research regarding the pathophysiological mechanisms involved in the occurrence of OSA as well as effective alternative approaches that can alleviate the severity of the sleep-related breathing disorder (i.e., apnea–hypopnea Index [AHI]) is needed.

A key pathological feature of OSA is the repetitive complete or partial collapse of the upper airway during sleep, resulting in apneas or hypopneas as well as in repetitive arousals and oxygen desaturations (1). Obesity is an important and well-recognized risk factor for OSA. However, 60% of patients with OSA are not obese (4), suggesting that factors other than obesity contribute to the pathogenesis of OSA. Compared with the general population, OSA is more prevalent in adults with several conditions/diseases in which fluid retention plays a central pathophysiological role, such as resistant or refractory hypertension, kidney disease, and heart failure (HF) (4). These raise the possibility that fluid retention may increase the risk for OSA given that some of the excess fluid retained in the legs during the day may accumulate in the neck while lying down at night and contribute to the narrowing of the upper airway (4). Thus, it seems likely that high sodium intake, which induces fluid retention, may play a role in the pathogenesis of OSA. In fact, increased sodium intake has been directly related to the severity of OSA in patients with resistant hypertension and hyperaldosteronism and in those with HF (5, 6). These findings were further supported by a recent study in which an intensified diuretic therapy promoted a modest but significant reduction in the AHI in proportion to the degree of the overnight rostral fluid shift attenuation in patients with uncontrolled hypertension (7). Moreover, in another study in which men with severe OSA were randomly allocated into diuretics or sodium-restricted diet or placebo, the group with the sodium-restricted diet showed greater reduction in the AHI compared with the groups on diuretics or placebo (8). To date, whether the relationship between the dietary sodium intake and the severity of OSA exists in general populations has been uncertain. The question is important because the modulation of sodium intake may be an alternative therapeutic strategy to reduce the risk for occurrence of OSA as well as to reduce the severity of OSA in patients who are poorly adherent to the CPAP. Moreover, lowering the sodium intake has long been a focus of cardiovascular risk reduction (9), and the question is crucial even in the context of minimizing the cardiovascular consequences of OSA regardless of the CPAP adherence.

In this issue of *AnnalsATS*, Giatti and colleagues (pp. 502–510) provide valuable insights into the relationship between dietary sodium intake and OSA (10). The authors analyzed data regarding the dietary salt intake, which was assessed by urinary sodium excretion or by the food frequency questionnaire, and the AHI by using a home sleep apnea testing (HSAT) device from a large community-based population in the ELSA-Brasil (Brazilian Longitudinal Study of Adult Health) study. They found that there were significant associations between the AHI and salt intake as well as urinary sodium excretion. After adjustment for confounding factors, the relationships were no longer meaningful in the entire population. However, the associations remained significant in the multivariate models in the subgroup of patients with hypertension. Thus, these findings suggest that the relationship between the high sodium/salt intake and severity of OSA cannot be generalizable to the entire community-based sample and that the role of dietary sodium in the pathogenesis of OSA may be limited to cases with concomitant hypertension. These results are basically in line with the results from previous studies investigating the relationship between rostral fluid shift and OSA in otherwise healthy nonobese men (11), patients with HF (12), patients with chronic kidney disease (13), and patients with resistant hypertension (14). Given that the relationship between rostral fluid shift and OSA was more obvious in men than in women (15), the greater proportion of men

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in the hypertensive subgroup compared with that in the normotensive subgroup may have contributed to the significant relationship between sodium/salt intake and the OSA severity among the patients with concomitant hypertension in the current study.

As also acknowledged by the authors, there are several limitations that should be considered in the interpretation of the results in this study. First, in patients with hypertension, sodium/salt intake based on urinary sodium excretion could be biased by the use of diuretics (16) and possibly by the dosage of the drug. Of note, the association was not significant in the subgroup of patients with hypertension without medication. However, the use of diuretics was adjusted for in the multivariate model in the entire hypertensive population, suggesting that this effect can be considered as minimum. Second, because patients with HF and chronic kidney disease were included, there might be some patients who had central sleep apnea. However, the HSAT system used in the current study is reliable for distinguishing between obstructive and central events, and subjects with dominantly central sleep apnea were not included in the study. On the other hand, the limitation of the HSAT in overestimating or underestimating the AHI because of the lack of information regarding the total sleep time may have led to misclassification of the cases as OSA or non-OSA on the basis of the AHI cutoff value chosen in the study. Third, no detailed information regarding the use of aldosterone blockers was provided. Aldosterone blockers can reduce AHI (4, 7), possibly through the increase in the urinary sodium excretion. Finally, this is a cross-sectional study and does not give insights into the causality issue between the sodium intake and the development or worsening of OSA. Though the study was not designed for the exploration of the mechanisms involved in the relationship between the salt intake/sodium excretion and the severity of OSA, the question of whether the urinary sodium excretion is a compensatory mechanism to lower the blood pressure (mainly by the activation of atrial natriuretic peptide during intrathoracic pressure swings) remains unanswered.

Despite these limitations, Giatti and colleagues should be commended for this work. They have provided novel findings regarding the relationship between the dietary sodium intake and severity of OSA in a large general population of 1,946 adults. The lack of a significant association between sodium and OSA in an otherwise healthy general population suggests that the role of dietary sodium in the pathogenesis of OSA seems to be limited to the cases with hypertension. How these findings relate to the role of dietary sodium restriction in patients with hypertension and OSA in the cardiovascular prevention models (9) as well as how these results would have implications in other subgroups, such as patients with diabetes mellitus (17), will require additional studies. Notwithstanding, focusing on the dietary sodium restriction would be a prudent intervention, as these data suggest, in patients with hypertension and OSA. This may have a particular importance for the individuals with OSA who are poorly adherent to CPAP treatment. ■

Author disclosures are available with the text of this article at www.atsjournals.org.

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