



■ Editorial

Obesity, Sarcopenia, and Smoking: Landscape in the Mist

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Smoking and obesity are both major public health problems. However, there is no consensus regarding their relationship. Numerous epidemiological studies have shown an inverse relationship between smoking status and body weight, suggesting that quitting smoking can lead to weight gain.^{1,2)} This is mainly because nicotine is both an appetite suppressant and metabolic stimulant. However, some studies have reported that smoking status is not significantly associated with body mass index, even though an association with central obesity was reported.^{3,4)} Hence, although smokers tend to have a lower body mass index than non-smokers, smoking may favor abdominal body fat accumulation. Various mechanisms of action, such as those of cortisol, sex hormones, insulin resistance, and chronic inflammation, may account for the effect of smoking on central obesity.

Sarcopenia is the degenerative loss of skeletal muscle mass and strength with aging. Considering the body fat increase with older age, several studies evaluated the relationship between sarcopenia and smoking. Although smoking may promote skeletal muscle protein breakdown due to oxidative stress,⁵⁾ the results of recently published studies raise some doubts. In a meta-analysis, smoking was found to be associated with an increased risk of sarcopenia.⁶⁾ However, only few studies showed a significant association, and there was a high heterogeneity among individual studies.

In a study from the current issue, Jo et al.⁷⁾ investigated the association between cigarette smoking and sarcopenia by analyzing data from the Korea National Health and Nutrition Examination Survey (KNHANES). In this study, current smoking was associated with sarcopenia among men. Although smoking may increase the risk of sarcopenia, there remain some

uncertainties. First, a significant association was only observed among men, which the authors attributed to the effect of hormonal differences. However, misclassification of data should also be considered. In a previous KNHANES study, females exhibited a higher level of false self-responses, which resulted in the underestimation of the female smoking rate.⁸⁾ Second, a consistent dose-response effect was not observed in this study. The obese male group showed the highest risk of sarcopenia in the heavy smoker, while there was no such a dose-response relationship in the non-obese group. So far, there are very few studies on smoking and sarcopenia, and it is not possible to make definitive conclusions even after including the results of the present study. Therefore, long-term prospective research is needed.

CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

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