



Editorial

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Adipokine Profiles and Metabolic Health

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The epidemic of obesity is a major health problem that leads to medical and socioeconomic burdens on society. Although body mass index (BMI) has been the most commonly used measure to determine the degree of obesity, it does not necessarily reflect the degree of adiposity. Recent observations show that some subpopulations exhibit unexpected metabolic profiles that deviate from the typical dose-response relationship between BMI and metabolic disturbances [1-3]. Individuals with impaired insulin sensitivity and increased levels of visceral adiposity, despite being nonobese, are defined as metabolically obese but normal weight or metabolically unhealthy nonobese (MUHNO) [1,2]. This subgroup is also characterized by a more atherogenic lipid profile, increased arterial stiffness and carotid atherosclerosis, and higher levels of blood pressure, oxidative stress, and vascular inflammation compared with metabolically healthy normal weight (MHNW) people [4-6]. Another subset of individuals with a lower degree of insulin resistance and favorable metabolic profiles, despite being obese, has been defined as metabolically healthy obese (MHO) [1,3]. Because the notion that the clinical outcomes of individuals differ according to their metabolic health status has been supported by several large-scale prospective studies [7-9], interest in this emerging concept has been growing.

In this issue, Lee et al. [10] measured several serum adipokine levels in nondiabetic Korean subjects and showed that the levels of tumor necrosis factor-α (TNF-α) and adipocyte fatty acid binding protein were significantly higher in the MUHNO

group compared with their metabolically healthy counterparts. However, the difference between MHO subjects and their metabolically unhealthy counterparts was not significant. The authors also showed that the levels of these adipokines were significantly correlated with several parameters that are usually used to define metabolic health [10]. Because insulin resistance and excess adiposity are considered core pathophysiologies of metabolic unhealthiness, it could be easily assumed that adipokines might have a role in the pathogenic mechanism or could be influenced by metabolic health status. Although prospective studies to define the cause-and-effect relationship are lacking, previous studies have also investigated the crosssectional relationship between various adipokines and metabolic health. Similar to the results of Lee et al. [10], individuals with the MUHNO phenotype were known to have higher circulating levels of leptin, TNF-α, and interleukin 6 (IL-6) and lower levels of adiponectin compared with the MHNW group [11-13]. Several reports also demonstrated lower levels of TNF-α, IL-6, plasminogen activator inhibitor-1, programulin, retinol-binding protein-4, and chemerin and higher levels of adiponectin in individuals with the MHO phenotype compared with metabolically unhealthy obese subjects [13-15].

Because there is no consensus on how to define metabolic health status, the clinical characteristics, metabolic profiles and outcomes can be largely affected by the diagnostic criteria used [8,16]. Although some conflicting results for adipokine levels also exist, current data support a close relationship to

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metabolic health status. Therefore, various adipokines might be used as important contributors to the identification or characterization of metabolic health status. Importantly, advancing our knowledge of the molecular indicators of metabolic health would lead to better risk management and prevention of metabolic obesity-related diseases [17].

CONFLICTS OF INTEREST

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

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