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Response

Association between Serum Dipeptidyl Peptidase-4 Concentration and Obesity-Related Factors in Health Screen Examinees (J Obes Metab Syndr 2017;26:188-96)

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Dipeptidyl peptidase-4 (DPP-4), a serine protease, degrades peptides containing alanine or proline residues and amino-terminal residues of proteins.[1] It is an aminopeptidase found in most tissues of the body, including the liver, lung, kidney, intestine, lymph nodes, and endothelial cells, and resides on cell membranes, where it exerts its effect. Physiological effects of DPP-4 can be broadly divided into enzymatic effects such as the inhibition of incretins and non-enzymatic effects associated with immune regulation, behavioral response, and inflammation depending on the substance on which DPP-4 acts.[2,3] Since Lamers et al.[4] reported DPP-4 as novel adipokine, many studies have shown serum DPP-4 level or activity to be associated with obesity, diabetes mellitus, and fatty liver.[5,6] Our previous study showed serum DPP-4 concentration was positively correlated with lean body mass, total cholesterol, and creatinine and was elevated in the obese group compared to the normal weight group, as reported in *Journal of Obesity & Metabolic Syndrome*.[7] It is an honor to reply to a Letter to the Editor with great comments on our study. Thus, we kindly respond to the issues raised in the Letter.

Our studies evaluated whether serum DPP-4 concentration is associated with obesity and obesity-related factors such as glucose, lipid profile, and body fat using blood test and bio-impedance analysis. Even though our study showed that serum DPP-4 level was higher in the obese group, these studies indirectly reflect the relationship between DPP-4 and obesity. Therefore, there is a limitation to understanding the mechanism and role of DPP-4 in adipose tissue. Recently, some studies have supported the association between DPP-4 and obesity. Lamer et al.[4] showed higher release of DPP-4 in fully differentiated adipocytes than in preadipocytes, and that direct addition of DPP-4 to fat and skeletal muscles impaired insulin signaling, suggesting that DPP-4 is an adipokine and might have direct effects on adipose tissue. To assess the tissue sources of circulating DPP-4, Sell et al.[8] assessed DPP-4 expression and release in the adipose tissue of lean and obese patients. Their results showed that DPP-4 expression was correlated with body mass index and was higher in visceral adipose tissue (VAT) than subcutaneous adipose tissue (SAT). It was also increased in VAT of lean patients with impaired glucose tolerance. In addition, DPP-4 release is higher in VAT than in SAT in both lean and obese patients. Moreover, Stengel et al.[9]

showed that DPP-4 protein level in the plasma was higher in obese patients and correlated with body mass index. These studies suggest that adipose tissue could be a primary source of higher serum DPP-4 or activity. However, the exact mechanisms of DPP-4 on adipose tissue are not known yet. Therefore, further studies are needed to elucidate the functional role of DPP-4 and its contribution to the incretin system. Finally, we thank you for the Letter and the opportunity to respond. We hope that *Journal of Obesity & Metabolic Syndrome* will continue to prosper in the future.

CONFLICTS OF INTEREST

The authors declare no conflict of interest.

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