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Letter to the Editor

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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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Incretin is a hormone that is secreted from the enteroendocrine cells in response to food ingestion and stimulates pancreatic β-cells to produce insulin, which leads to postprandial glucose stabilization. Glucagon-like peptide-1 (GLP-1) and glucose-dependent insulinotropic polypeptide are two principal incretin hormones that are secreted from L-cells in the ileum and K-cells in the duodenum and jejunum, respectively. Intact incretin, a biologically active form of incretin, is degraded by the enzyme dipeptidyl peptidase-4 (DPP-4) to an inactive metabolite shortly after its secretion. Several reports describe that serum DPP-4 activity is increased in obesity[1] and type 2 diabetes[2], and this could be an explanation for the usefulness of the inhibitors.

Lee et al.[3] investigated the associations between serum DPP-4 concentration and obesity-related factors in healthy people. They reported that serum DPP-4 concentration was associated with obesity-related factors and was higher in the obese group than in the normal body mass index group. It has been reported that serum DPP-4 activity was closely correlated to serum DPP-4 concentration.[4,5] Therefore, the study results are not much different from previous findings that serum DPP-4 activity was higher in obese people.[5,6]

DPP-4 is widely distributed in tissues, such as lung, spleen, liver, kidney, intestines, endothelial cells, bone marrow, and blood cells, and is also present in serum as a soluble form.[7] Some studies show that a large proportion of intact GLP-1 secreted from intestinal L cells is rapidly degraded by DPP-4 in the capillaries supplying the cells[8], and less than 5% of intact GLP-1 finally reaches systemic circulation.[9] Accordingly, local tissue DPP-4 activity may be more important than plasma DPP-4 activity in determining blood intact incretin level. Lee et al.[3], however, measured serum DPP-4 concentration, not tissue activity. It is also not known whether local tissue DPP-4 activity is regulated in parallel with serum DPP-4.[10] Therefore, local tissue DPP-4 activity should be measured for research on incretin metabolism *in vivo*. To do this, various methods of measuring tissue DPP-4 activity must be developed.

The study by Lee et al.[3], using health examination data from a single center, reconfirmed that obesity is associated with higher levels of serum DPP-4. However, serum DPP-4 concentration or activity plays a minor role in incretin metabolism *in vivo*. If tissue DPP-4 activity is measured, the effect of obesity or increasing body fat on incretin metabolism will be known. Future studies measuring local tissue DPP-4 activity in obese people are necessary for

pathophysiologic study between obesity and incretin metabolism.

CONFLICTS OF INTEREST

The author declares no conflict of interest.

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