



Association of Vaspin with Metabolic Syndrome: The Pivotal Role of Insulin Resistance (*Diabetes Metab J* 2014;38:143-9)

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We read with interest Dr. Choi's comments on our article titled "Association of Vaspin with Metabolic Syndrome: The Pivotal Role of Insulin Resistance" published in *Diabetes & Metabolism Journal* [1].

Vaspin is a novel adipokine linking adipocytes and components of metabolic syndrome, although the exact role of vaspin remains unclear [2,3]. In our study, we found that central obesity, raised triglyceride, and raised fasting blood glucose are linked to higher vaspin concentrations; meanwhile, the relationship abolished after controlling for homeostasis model assessment of insulin resistance and high sensitivity C-reactive protein revealing the leading role of insulin resistance and chronic inflammation in this regard.

Choi et al.'s reported a significant relationship between vaspin concentrations and metabolic syndrome component including obesity and raised triglyceride. However, they did not find any connection between raised fasting plasma glucose and vaspin, which is evident in our series of patients. Discrepancies observed between the two studies likely reflect the differences in glycemic status of the patients enrolled. Choi et al. included patients from a routine health examination center (Seoul National University Bundang Hospital, Seoul, Korea) whereas our sample comprised patients visited at a diabetes clinic of a teaching hospital (Valiasr Hospital, Tehran, Iran). The proportion of patients with diabetes in the Korean sample was low (16.0%). In our sample a significantly larger proportion of pa-

tients were diagnosed with type 2 diabetes (59.3%) and another 9.7% had impaired fasting glucose. We suggest the more prominent association observed between insulin resistance/chronic inflammation and vaspin is due to prolonged hyperglycemia and insulin insensitivity.

It is hypothesized that the principal event for the development of metabolic syndrome is the deposition of fat in adipose tissue, liver, muscles, and pancreas in the face of impaired triglyceride/cholesterol metabolism and central obesity. This process in turn triggers the development of insulin resistance through various pathways including oxidative stress and chronic inflammation which, in a proportion of patients, lead to type 2 diabetes mellitus [4,5]. Chronic inflammation in turn could impair insulin signaling pathways in liver and other organs [6]. Vaspin is an adipocytokine involved in early as well as late stages of metabolic syndrome. In this view, results of the present study complement Choi et al. findings by showing the unflagging contribution of vaspin to the pathogenesis of metabolic syndrome. Further investigating the mechanisms by which vaspin promotes metabolic syndrome and resultant atherosclerosis is a focus of future research. The authors would like to thank Dr. Choi for thoughtful comments which put the findings of our study in perspective and postulate possible hypotheses for future research.

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CONFLICTS OF INTEREST

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

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