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Reply to Aydin et al: "To what extent is it right to measure serum vaspin, obestatin, and apelin-36 levels without a protease inhibitor in nonalcoholic fatty liver disease?"

Reply:

We thank Dr Aydin and colleagues [1] for their interest in our article [2] and their questions about the lack of aprotinin pretreatment before measuring serum peptide levels (and obestatin in particular) in patients with nonalcoholic fatty liver disease (NAFLD). However, we respectfully disagree with their comments. First, pretreatment of biological samples with aprotinin is generally required for the extraction of peptides from plasma or tissue homogenates [3]. However, only serum samples were used in our study. Second, the use of aprotinin was not specifically recommended by the manufacturer of the obestatin immunoassay kit used in our study (Yanaihara Institute, Fujinomiya-SHI Shizuoka, Japan). Third, Dr Aydin and coworkers claim that the increase of caspase levels associated with the development of nonalcoholic steatohepatitis could have confounded our results on serum peptide levels. This shortcoming, however, is common to all studies focusing on peptide hormones in patients with NAFLD. Unfortunately, this important issue cannot be simply overcome by the use of aprotinin, as there is no evidence to indicate that aprotinin is capable of inhibiting caspases. In addition, we are unaware of previous studies showing that caspase activated in nonalcoholic steatohepatitis can specifically break down apelin-36, vaspin, and obestatin. Finally, the serum levels of apelin-36, vaspin, and obestatin found in our study were largely in line with those reported in previous studies [4-10]. It is also noteworthy that Gutierrez-Grobe et al [4] have failed to find significant differences in serum obestatin between patients with NAFLD and healthy controls.

From these considerations, we conclude that our data on serum levels of apelin-36, vaspin, and obestatin in NAFLD are valid and potentially generalizable.

Yusuf Yilmaz
Department of Gastroenterology, Department Marmara University School of Medicine, Altunizade, Istanbul 34662, Turkey
Institute of Gastroenterology, Marmara University Maltepe, 34840, Istanbul, Turkey

Fatih Eren
Institute of Gastroenterology, Marmara University Maltepe, 34840, Istanbul, Turkey

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