

# Insulin like growth factor-1 and lipoprotein metabolism in stable angina patients

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We read the article: "Association between insulin like growth factor-1 and lipoprotein metabolism in stable angina patients on statin therapy: a pilot study" by Burchardt et al. [1] with interest. The authors concluded that owing to association with products of lipid oxidation, oxidised proteins, and high values of Lp(a); IGF-1 and IGFBP3 levels could be useful indicators of atherosclerosis progression. We believe that these findings should act as a guide for further studies.

IGF-1 plays an important role in the cell protection of multiple systems, where its signal transduction helps to preserve tissues from hypoxia, ischaemia and oxidative stress. IGF-1 and its binding proteins (IGFBP) have association with hypertensive patients with left ventricular hypertrophy and arteriosclerosis, especially coronary artery sclerosis [2, 3]. IGF is produced mostly by the liver and transported by their own binding proteins (IGBP) in the circulation. Seven different IGFBPs have been reported to date, with IGF-1 being the most abundant. The effects of the-

se proteins not only bind IGFs, but also have receptors in tissues. An increase in IGF-1 levels in patient with a chronic kidney disease (CKD) is shown [4]. IGF levels have been affected in different ways in CKD. It would be better if it could be clearly stated that CKD and liver diseases are excluded from the study.

**Conflict of interests:** none declared

## References

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## Author's response

I would like to thank Demirkol et al. for their interest and their comments on our article [1]. As to their remarks, of course we agree on the impact of both — kidney and liver — on IGF-1 and IGF-1 plasma levels. However, the exclusion criteria from the study were provided in the quoted article [2]. For the sake of thoroughness, we would like to emphasize that patients with documented liver disease or its dysfunction (ALT, AST > ULN) were excluded from the study. Also excluded were patients with chronic kidney disease or their dysfunction qualified when GFR was < 60 mL/min. We must admit that the method of assessing GFR by the MDRD rule used by us has its own limitations. Secondly, GFR 60 mL/min was a priori assumed as the cut-off point for kidney disease. Thus, patients with kidney disease in stage I by Kidney Diseases Outcome Quality Initiative (without GFR limitation) or

in stage II (90 < GFR > 60 mL/min) could be included in the study, which is another limitation of our protocol.

**Conflict of interest:** none declared

## References

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