

Letters to the Editor

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Response to Letter by Pilz et al

Response:

While our work passed through the peer-review process for publication in *Stroke*, Pilz et al published data demonstrating an inverse and independent correlation between adiponectin levels and carotid intima-media thickness (IMT).¹ In this work, the authors compared 140 obese juveniles (mean age 13.5±4.4 years) with 100 age-matched, healthy, normal-weight controls and demonstrated a significant negative correlation between serum adiponectin levels and carotid IMT, even after controlling for common cardiovascular risk factors. The authors do not report the numbers of males and females, respectively, in their study population. In our study we investigated the association between plasma adiponectin levels and sonographic phenotypes of subclinical atherosclerosis, which may represent different stages of disease as well as common and distinct determinants.² Thus, our work focused not only on the association between adiponectin levels and IMT, but we investigated the association of this biomarker with different stages of atherosclerosis. Furthermore, our study population represents a healthy, middle-aged, white population with a mean body mass index of 26.7 and, therefore, differs substantially from the population of Pilz et al. Our results demonstrate an inverse correlation between adiponectin levels and IMT, thus confirming the observation that early stages of atherosclerosis, as defined by an increased IMT, are associated with hypoadiponectinemia. In addition, our results failed to demonstrate an association between adiponectin levels and advanced stages of atherosclerosis as represented by the presence of plaques. Earlier studies demonstrated a stronger impact of adiponectin on atherosclerosis and clinical end points almost exclusively in men.^{3–5} These data are supported by our results, which demonstrate a stronger impact of hypoadiponectinemia on IMT in men. The lack of an association between adiponectin levels and the risk of coronary artery disease in women, as demonstrated recently,⁶ is consistent with these data. The results of Lindsay and colleagues⁷ suggest that adiponectin is not an independent risk factor for coronary artery disease and that its role is more important in insulin resistance and diabetes. These results were found in a diabetic and obese population of volunteers of American Indian heritage, which differs substantially from our study population, thus suggesting that differences in the results between studies may be explained by underlying differences in the study populations. Furthermore, the cross-sectional design of our study warrants cautious interpretation of the results, and we agree with Pilz and colleagues that further investigations are necessary to substantiate the definitive diagnostic or therapeutic impact of adiponectin in atherosclerosis.

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