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Letter to the Editor

Oligomerization status and post-translational modification of adiponectin: A possible association between adiponectin and risk of coronary artery disease

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Dear Editor

We have read the article by Au Yeung et al. [1] with great interest. In their opinion, adiponectin is unlikely to be a cause of coronary artery disease (CAD) and factors that drive adiponectin may be overlooked causes of CAD, based on a bi-directional Mendelian randomization analysis. This study brings our attention to the exact role of adiponectin in initiation and progression of atherosclerotic-related metabolic diseases.

However, we have some reservations about the paper. First, the study predicted adiponectin levels mainly based on different single nucleotide polymorphisms (SNP), but adiponectin in vivo actually exists in trimeric and hexameric forms and high-molecular-weight oligomeric complexes [2] that exert different biological effects in metabolism modulation and might not be directly reflected by the SNP prediction. Second, the biological effects of adiponectin not only depend on its plasma level, but also on different post-translational modifications, including O-glycosylation, sialylation, and hydroxylation of such secreted cytokine [3]. Third, in addition to the SNP of adiponectin, activities of other genes such as lysyl hydroxylase 3 and DNA methyltransferase 1 also play a key role in the biological effects of such multifunctional adipokines [4,5], which were not included in the authors' consideration.

In summary, although the above mentioned remain to be considered and included in future investigations, this study failed to find a strong association between adiponectin and risk of cardiovascular disease, which inspired further exploration for hidden etiological factors.

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Conflict of interest

The authors declare that they have no conflict of interest.

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