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Letter to the Editor

Response to letter of He et al.: Oligomerization status and post-translational modification of adiponectin: A possible association between adiponectin and risk of coronary artery disease

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Dear Dr. Liang,

We thank you for your interest in our paper [1], and would like to respond to the points you raised regarding the role of adiponectin in coronary artery disease (CAD) [2].

The use of single nucleotide polymorphisms (SNPs) from various gene regions is an advantage for Mendelian randomization since this enables the use of sensitivity analyses, such as MR Egger, which are more robust to violation of the Mendelian randomization assumptions. We also chose SNPs from genome wide association studies [3], which reduces the likelihood of false positives compared to selecting SNPs from specific gene regions.

We agree our study may not be best suited to examining the cardioprotective effects of different forms of adiponectin. However, we are uncertain as to how likely these are to explain the discrepancies between Mendelian randomization and observational studies given the lack of relevant evidence from human experimental studies. It is noted that similar arguments were put forward when Mendelian randomization studies did not suggest a causal role of high density lipoprotein cholesterol in CAD, although the findings from Mendelian randomization were consistent with randomized controlled trials [4].

Considering existing evidence from Mendelian randomization studies [1,5], the relation of adiponectin with CAD risk in observational studies is likely confounded. Whilst we recognize studying adiponectin in various forms may be etiologically revealing, the lack of genetic validation by Mendelian randomization suggests efforts should be redirected to the identification of other hidden etiologic factors that drive both adiponectin and CAD, and could be targeted to mitigate CAD burden.

Conflict of interests

The authors report no relationships that could be construed as a conflict of interest.

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