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Letter to the Editor

Response to letter on "The role of remote ischemic perconditioning beyond myocardial infarction size reduction"

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We read with great interest Dr. Zheng's comments [1] on our study of investigating the anti-remodelling effects of remote ischemic perconditioning (RIPerc) [2]. Accordingly, we found that RIPerc markedly decreased inflammation and improved left ventricle hemodynamic function in association with the enhancement of NRG-1 levels and ErbB3 expression in the myocardial tissue. As Dr. Zheng describes, how the signalling mechanism by RIPerc acts on NRG-1 and ErbB receptors is still unclear. Nevertheless, to investigate the causal role of NRG-1 in cardioprotection from RIPerc, some limitations have to be considered. First, in animal models, homozygous germline deletion of NRG-1, erbB2 or erbB4 results in mortality during early embryonic period due to failure of proper cardiac ventricular and endothelial deletion of NRG-1 *per se* markedly increased myocardial infarct size in mice and subsequent leads to worse outcome [3]. However, these studies highlight and emphasize the importance to target endothelial cells (primary source of NRG-1) integrity and function in post-infarct myocardial remodelling. In line with this, a previous study demonstrated the association between microvascular dysfunction in the infarct-border region and changes in LV volume [4]. Reflect comments on the time and mode of RIPerc performed, in our study we use a "single" application of RIPerc. In contrast, Wei et al. [5] described that protection from adverse LV remodelling was obtained by RIPerc and additionally "repeated" RIPostC. Even though, both studies demonstrated similar levels of cardioprotection by remote ischemic conditioning, further preclinical and clinical studies are requested to optimize the remote conditioning algorithms.

Conflict of interest

None declared.

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