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Letter to the Editor

Response to letter on "Post-translational modifications: Novel mechanism to clarify the cardioprotective effects of remote ischemic conditioning by Tang and Yang"

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We thank Dr. Tang and Dr. Yang for his thoughtful comments [1] regarding our recent study [2] demonstrating that remote ischemic perconditioning (RIPerc) enhanced the activity of NRG-1 promoter and subsequent improved cardiac function. Accordingly, a recent clinical trial demonstrated that cardioprotection by combined intrahospital RIPerc and postconditioning in addition to primary PCI have favourable effect on the rate of new congestive heart failure [3]. However, the mechanisms underlying the long-term beneficial effects of RIC after MI are largely unknown. We found that [2], activation of the NRG1 promoter, through tri-methylation of lysine 4 in histone H3 (H3K4me3), is considered to be a novel mechanism for the cardioprotective effects of RIPerc. We thank for Dr. Tang and Dr. Yang [1] for pointing out that post-translational modifications (PTMs) may be novel underlying mechanisms that mediated the cardioprotective effects of RIC. Indeed, recent studies are process of shedding light on the roles played by non-coding RNAs, nucleic acid methylation and histone/chromatin modifications in specific pathologies of the heart. While it is clear that alterations of the chromatin state and DNA methylation patterns are involved in the response of cardiac cells to ischemic injury [4], epigenetic approaches to limiting infarct size is still restricted to preclinical models, where e.g. histone deacetylase inhibition has proven to be a promising strategy in various cardiac related dysfunction e.g. hypertrophy, fibrosis and contractility [5]. Nevertheless, further studies are warranted to confirm that PTMs play a casual role in the cardioprotective effects of RIC.

Declaration of Competing Interest

None declared.

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