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Letter to the Editor

# Post-translational modifications: Novel mechanism to clarify the cardioprotective effects of remote ischemic conditioning

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Remote ischemic conditioning (RIC), either by preconditioning or postconditioning, could protect against ischemia/reperfusion (I/R) injuries. However, the underlying mechanisms remain to be clarified. Recently, Pilz P et al. demonstrated that the methylation of NRG1, which may be mediated by H3K4me3, could attenuate the decreased NRG1 and contribute to the anti-remodeling and anti-inflammatory effects of RIC in rat I/R model [1]. These results suggest that post-translational modifications (PTMs) may be novel underlying mechanisms that mediated the cardioprotective effects of RIC.

PTMs refer to the enzymatic modification of proteins after translation, which play a major role in protein folding, stability, conformation, localization and contribute to the regulation of cellular processes. There are evidence showed that PTMs, including phosphorylation, methylation, acetylation, deacetylation, glycosylation, were involved in I/R injuries [2,3]. However, whether PTMs mediated the cardioprotective effects of RIC remains unclear. To clarify the function of PTMs during RIC, several key points need to be answered. First, the comprehensive and dynamic changes of PTMs during I/R injuries. Second, which kind of PTMs is regulated by RIC. Third, the main targets, in addition to NRG1, regulated by PTMs after RIC. Fourth, whether manipulating PTMs, either by genetic or pharmacological approaches, could reverse the cardioprotective effects of RIC. For this study [1], it will be interesting to see whether blocking or depleting H3K4me3 could attenuate the anti-remodeling effects of RIC.

Though the study [1] was very interesting and indicative, more studies are required to confirm that PTMs do participate in the cardioprotective effects of RIC.

## Conflict of interest

None.

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