



## Remote Ischemic Conditioning Influences Mitochondrial Dynamics

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Titre Remote Ischemic Conditioning Influences Mitochondrial Dynamics

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Résumé en anglais Remote ischemic preconditioning (RIPC) has emerged as an attractive strategy to protect the heart against ischemia-reperfusion (I/R) injury. The mechanisms by which remote ischemic conditioning (RIC) is protective are to date unknown, yet a well-accepted theory holds that the mitochondria play a central role. Mitochondria are dynamic organelles that undergo fusion and fission. Interventions that decrease mitochondrial fission or increase mitochondrial fusion have been associated with reduced I/R injury. However, whether RIPC influences mitochondrial dynamics or not has yet to be ascertained. We sought to determine the role played by mitochondrial dynamics in RIPC-induced cardioprotection. Male adult rats exposed in vivo to myocardial I/R were assigned to one of two groups, either undergoing 40 min of myocardial ischemia followed by 120 min of reperfusion (MI group) or four 5-min cycles of limb ischemia interspersed by 5 min of limb reperfusion, immediately prior to myocardial ischemia and 120 min of reperfusion (MI+RIPC group). After reperfusion, infarct size was assessed and myocardial tissue was analyzed by Western blot and electron microscopy. RIPC induced smaller infarct size (-28%), increased mitochondrial fusion protein OPA1, and preserved mitochondrial morphology. These findings suggest that mitochondrial dynamics play a role in the mechanisms of RIPC-induced cardioprotection.

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