



## Role of hypoxia inducible factor-1 $\alpha$ in remote limb ischemic preconditioning.

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Résumé en anglais	<p>Remote ischemic preconditioning (RIPC) has emerged as a feasible and attractive therapeutic procedure for heart protection against ischemia/reperfusion (I/R) injury. However, its molecular mechanisms remain poorly understood. Hypoxia inducible factor-1<math>\alpha</math> (HIF-1<math>\alpha</math>) is a transcription factor that plays a key role in the cellular adaptation to hypoxia and ischemia. This study's aim was to test whether RIPC-induced cardioprotection requires HIF-1<math>\alpha</math> upregulation to be effective. In the first study, wild-type mice and mice heterozygous for HIF1a (gene encoding the HIF-1<math>\alpha</math> protein) were subjected to RIPC immediately before myocardial infarction (MI). RIPC resulted in a robust HIF-1<math>\alpha</math> activation in the limb and acute cardioprotection in wild-type mice. RIPC-induced cardioprotection was preserved in heterozygous mice, despite the low HIF-1<math>\alpha</math> expression in their limbs. In the second study, the role of HIF-1<math>\alpha</math> in RIPC was evaluated using cadmium (Cd), a pharmacological HIF-1<math>\alpha</math> inhibitor. Rats were subjected to MI (MI group) or to RIPC immediately prior to MI (R-MI group). Cd was injected 180 min before RIPC (Cd-R-MI group). RIPC induced robust HIF-1<math>\alpha</math> activation in rat limbs and significantly reduced infarct size (IS). Despite Cd's inhibition of HIF-1<math>\alpha</math> activation, RIPC-induced cardioprotection was preserved in the Cd-R-MI group. RIPC applied immediately prior to MI increased HIF-1<math>\alpha</math> expression and attenuated IS in rats and wild-type mice. However, RIPC-induced cardioprotection was preserved in partially HIF1a-deficient mice and in rats pretreated with Cd. When considered together, these results suggest that HIF-1<math>\alpha</math> upregulation is unnecessary in acute RIPC.</p>

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