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Role of adiponectin in ischemic postconditioning mediated cardioprotection in diabetes

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Hearts of diabetes are prone to ischemia/reperfusion(IR) injury. Ischemic postconditioning (IPostC) achieved by brief episodes of IR, given at the onset of prolonged ischemia, protects the myocardium. Unfortunately, IPostC cardioprotection is absent in diabetes and the mechanism remains unclear. Adiponectin(APN) confers cardioprotection in non-diabetes by increasing nitric oxide(NO) (the major mediator of IPostC cardioprotection), which needs the participation of adiponectin receptor-1(AdipoR1). However, in diabetes, APN is decreased, resulting in reduced NO bioavailability. We hypothesized that impaired APN production/signaling is responsible for the loss of IPostC cardioprotection in diabetes. Control (C) and Streptozotocin-induced diabetic(D) rats were subjected to 30 minutes coronary artery ligation followed by 2 hours reperfusion, without or with IPostC achieved by 3 episodes of 10s reperfusion and 10s re-occlusion preceding full reperfusion. Rats were injected with APN(1X109 pfu) 7 days before inducing IR. Cardiac APN expression was upregulated by IPostC both in C and D, but the interaction of AdipoR1 with caveolin-3(Cav3), a molecular known to interact with eNOS to facilitate NO production, was reduced in D. Post-ischemic myocardial infarction was higher in D relative to C,accompanied with significant reductions of myocardial p-eNOS and NO and end systolic pressure volume relation. All these changes in D were reverted by IPostC in the presence but not in the absence of APN. We conclude that disruption of AdipoR1 with Cav3 may responsible for the loss of IPostC cardioprotectin in diabetes and APN restores diabetic heart sensitivity to IPostC by enhancing myocardial eNOS signaling.

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