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Ecrg4 attenuates apoptosis of cardiomyocytes to ischemia/reperfusion through STAT3 signaling pathway

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Esophageal Cancer Related Gene-4 (ECRG4), a newly characterized tumor suppressor gene, is constitutively expressed in cardiomyocytes and conduction systems. However, the roles of Ecrg4 in heart remain to be explored. Previously, we showed that Ecrg4 functions as a 'sentinel' molecule maintaining cardiac homeostasis and loss of Ecrg4 contributes to atrial fibrillation. In this report, we show that cardiac ischemia inhibits the expression of Ecrg4 at both mRNA and protein levels in an acute myocardial infarction mouse model, which was validated in CoCl2-induced hypoxia of neonatal cardiomyocytes. Bioinformatics analysis identified a canonical hypoxia response element (GCGTG) at -304 to -300 in the promoter region, which was confirmed to be responsible for hypoxia-induced down-regulation of ECRG4 expression. Forced expression of Ecrg4 inhibited CoCl2-induced apoptosis of neonatal cardiomyocytes. Furthermore, hypoxia activated STAT3 in vivo, and blockade of the activation of STAT3 abolished CoCl2-induced apoptosis in neonatal cardiomyocytes. Given that Ecrg4 is down-regulated in tumor, the discovery that Ecrg4-mediates hypoxia-induced cardiomyocyte apoptosis suggests that Ecrg4 may be the long-sought molecule accounting for the increased incidence of cardiovascular events in tumor patients.