Effects on E4BP4-Induced IGF1R Activation in H9c2 Cardiomyoblast cells

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Abstract

Hypoxia and hypoxia in combination with ischemia lead to overproduction of nitric oxide (NO), a potent free radical molecule. Capase-3 activity and fragmentation of nuclear DNA in the hypoxic newborn piglet brain are NO mediated. The nuclear factor IL-3(NFIL3 also called E4BP4) is a member of the mammalian basic leucine zipper (bZIP) transcription factor superfamily. E4BP4, is up-regulated by the interleukin-3 (IL-3) signaling pathway and plays an important role in the anti-apoptotic response of IL-3. E4BP4 overexpression enhance the cardiac survival signaling by activating IGF1R/p-Akt pathway and inhibited the apoptotic proteins in cardiomyocyte. In the previous study found that the up-regulation of IGF2 and IGF2R genes is essential for angiotensin II (ANGII)-induced cell apoptosis and correlates with the promotion of cardiomyocytes apoptosis in hypertensive rat hearts. Here we show that E4BP4 down-regulates IGF2/mannose 6-phosphate receptor (IGF2R) signal pathway through Gαq/Calcineurin in H9c2 cardiomyoblast cells.