# E4BP4 Inhibits AngII-Induced Apoptosis by Activating the PI3K-Akt Pathway and Promoting Calcium Uptake in H9c2 Cardiomyoblasts

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The bZIP transcription factor E4BP4 is a survival factor in pro-B lymphocytes. In addition, E4BP4 expression has been shown to be elevated in hearts of spontaneously hypertensive rats (SHR). We tested the effect of E4BP4 on AngII-induced cardiomyocyte apoptosis. Cardiomyoblast cells (H9c2) that had been engineered to overexpress E4BP4 were exposed to AngII. The results of the TUNEL and DNA fragmentation assays revealed that E4BP4 attenuated the degree of AngII-induced apoptosis. Furthermore, Western blot and RT-PCR analysis showed that E4BP4 inhibited AngII-induced IGF-II mRNA expression and cleavage of caspase-3 through the PI3K-Akt pathway. In addition, E4BP4 improved calcium reuptake into the sacroplasmic reticulum via down-regulating PP2A and by up-regulating the phosphorylation of PKA and PLB. Our findings indicate that E4BP4 functions as a survival factor in cardiomyoblasts by inhibiting IGF-II transcription and by regulating calcium cycling.