ARC'14
مؤتمر مؤسسة قطر السنوي للبحوث
QATAR FOUNDATION ANNUAL RESEARCH CONFERENCE
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Heme Oxygenase Isoform 1 Regresses Cardiomyocyte Hypertrophy Through Regressing Sodium Proton Exchanger Isoform 1 Activity

10.5339/qfarc.2014.HBSP1112

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Abstract

Background: Pathological cardiac hypertrophy is a worldwide problem and an independent risk factor that predisposes the heart to failure. Enhanced activity or expression of the sodium proton exchanger isoform 1 (NHE1) has been implicated in conditions of cardiac hypertrophy. Induction of cGMP has previously been demonstrated to reduce NHE1 activity and expression, which could be through the expression of heme oxygenase isoform 1 (HO-1), a stress-induced enzyme that shows cardioprotective properties. In our study, we aimed to investigate the role of inducing HO-1 in a cardiac hypertrophy model that expresses active NHE1 to determine whether HO-1 could protect against NHE1 induced cardiomyocyte hypertrophy.

Methods: H9c2 cardiomyocytes were infected with the active form of the NHE1 adenovirus in the presence and absence of protoporphyrin (CoPP). Which was used to induce HO-1. Protein and mRNA expression of HO-1 were invested in H9c2 cardiomyocytes in the presence and absence of the expression of the active form of the NHE1 adenovirus. The effects of HO-1 induction on NHE1 protein expression and cardiomyocyte hypertrophic markers were measured respectively by western blotting and analyzing the cell surface area of H9c2.

Results: Our results showed a significant decrease in HO-1 mRNA expression in cardiomyocytes expressing active NHE1 (74.84 ± 9.19 % vs. 100 % normal NHE1 expression, $p<0.05$). However, we did not see any changes in NHE1 protein expression following HO-1 induction. A trend towards decrease in cardiomyocyte hypertrophy was observed in H9c2 cardiomyoblasts infected with the active form of NHE1 following stimulation with HO-1 (NHE1, 154.93 ± 14.87 % vs. NHE1 + CoPP, 109 ± 16.44 %).

Conclusion: In our model, HO-1 maybe a useful means to reduce NHE1 induced cardiomyocyte hypertrophy, although the mechanism by which it does that requires further investigation.