

**Introduction**

Heart failure is the inability of the heart to pump sufficient amounts of blood to meet the metabolic demands of organs and tissues as needed. It is often induced by cardiac hypertrophy. Cardiac hypertrophy is the thickening of the heart muscle as it responds to increased biomechanical stress. It represents the initial stage of cardiac diseases that can progress to heart failure. Using the Neonatal Cardiomyocyte Isolation System, rat pups' heart cells were isolated. Following isolation, cells were incubated in a culture medium with FBS for 24 hours.

**Methodology**

Total RNA was reverse transcribed and then amplified by the Preamplification System for cDNA Synthesis and Taq DNA Polymerase. As a result of my data, I can conclude that TNF-α has both an apoptotic and hypertrophic effect. However, EGF shows enhanced hypertrophic effect and reverses ANG II apoptotic effect as well as enhances its hypertrophic effect. It is also noted that IGF-1 reverses ANG II apoptotic effect as well as enhances its hypertrophic effect. However, EGF hypertrophic effect is masked in the presence of IGF-1, but circumvented by EGF.

**Results**

- ANG II enhances hypertrophic signaling pathway, ERK1/2, is positively affected by TNF-α or ANG II. EGF shows enhanced hypertrophic effect and reverses ANG II apoptotic effect as well as enhances its hypertrophic effect. However, EGF hypertrophic effect is masked in the presence of IGF-1, but circumvented by EGF.

**Discussion**

Cardiac hypertrophy is the cellular response to an increase in biomechanical stress. It represents the initial stage of cardiac diseases that can progress to heart failure. Using the Neonatal Cardiomyocyte Isolation System, rat pups' heart cells were isolated. Following isolation, cells were incubated in a culture medium with FBS for 24 hours.

**Materials and Methodology**

The gene expression of growth factor proteins as indices of cardiac hypertrophy versus apoptosis

**Abstract**

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