

عنوان مقاله:

LOXL₂ silencing suppresses angiotensin II-induced cardiac hypertrophy through the EMT process and TGF- β /Smad 3 /NF- κ B pathway

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خلاصه مقاله:

Objective(s): Atrial fibrillation (AF) is a common arrhythmia with atrial myocyte hypertrophy linked with stroke, heart failure, and increased mortality. Lysyl oxidase-like 2 (LOXL₂) involves the cross-linking of collagen in the extracellular matrix (ECM). In the present study, we investigated the roles and underlying mechanisms of LOXL₂ on cardiomyocyte hypertrophy. **Materials and Methods:** The expression of LOXL₂ mRNA and protein were detected in angiotensin II (Ang II) treated rat cardiomyocytes H9c2 by RT-qPCR and western blot. Small interfering RNA (siRNA) mediated LOXL₂ gene silencing was used to evaluate cardiac hypertrophy and related markers. Also, the protein expression of EMT markers and Smad 3 /NF- κ B pathway was determined by western blot. **Results:** Ang II significantly increased mRNA and protein expressions of LOXL₂ and increased mRNA levels of myocardial hypertrophy markers, including ANP, BNP, and β -MHC in H9c2 cells. Silencing of LOXL₂ significantly suppressed Ang II-induced hypertrophy and reversed the increase in ANP, BNP, and β -MHC mRNA levels. Also, EMT markers' expressions, as evidenced by increased E-cadherin and decreased vimentin, α -smooth muscle actin (α -SMA), fibroblast-specific protein (FSP), and collagen 1A1. Mechanistically, we found that LOXL₂ silencing suppressed protein expressions of TGF- β 1, p-Smad 3 , and p-NF- κ B in Ang II-stimulated H9c2 cells. LOXL₂ silencing also attenuated Ang II-induced increased expression and content of proinflammatory cytokines IL-1 β (H) and TNF- α . **Conclusion:** Our data speculated that LOXL₂ might be a potential contributing factor to Ang II-induced cardiac hypertrophy, and TGF- β 1/Smad 3 /NF- κ B is involved in a signal axis and

کلمات کلیدی:

Angiotensin II, Atrial fibrillation, Epithelial-mesenchymal - transition, Hypertrophy, LOXL₂ protein

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