

عنوان مقاله:

Angiotensin II signalling pathways in Cardiac Fibrosis

محل انتشار:

چهارمین همایش بین المللی زیست شناسی و علوم زمین (سال: 1400)

تعداد صفحات اصل مقاله: 3

نویسندگان:

Samane Sadat Hosseiny - Department of Biology, Faculty of Sciences, Ferdowsi University of Mashhad, Mashhad, Iran

Zeinab Neshati - Novel Diagnostics and Therapeutics, Research Group, Institute of Biotechnology, Ferdowsi University of Mashhad, Mashhad, Iran

خلاصه مقاله:

Cardiac fibrosis is the main cause of nearly all forms of heart failure, such as hypertensive heart disease, myocardial infarction, pulmonary arterial hypertension and dilated cardiomyopathy. The fibroblast cells play a main role in tissue fibrosis through the establishment of transforming growth factor-beta, excessive collagen secretion and other extracellular matrix proteins, which create pathologic scars. Angiotensin II (Ang II) is a mediator directly resulting in cardiac fibrosis via the induction of immediate early genes, such as alpha-smooth muscle actin, collagen types I and III, matrix metalloproteinase-) and tissue inhibitor of matrix metalloproteinase peptidase. Ang II leads to cardiac fibrosis by affecting many signaling pathways, such as nuclear factorerythroid Y related factor Y, peroxisome proliferatoractivated receptor-gamma and phosphatidylinositol \(\mathbb{P}\)-kinase/proteinkinase B. Several techniques, including cellular direct reprogramming and molecular targeting (e.g., such as noncoding RNAs and epigenetic modifiers) are novel therapeutic options targeting fibrosis. The current data show that the inhibition of Ang II-stimulating drugs is probably one of the best ways to prevent cardiac fibrosis

کلمات کلیدی:

Angiotensin II; Cardiac fibrosis; Fibroblast cells

لینک ثابت مقاله در پایگاه سیویلیکا:

https://civilica.com/doc/1435430

