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Renin-Angiotensin Aldosterone System in the Pathogenesis of Fibrosis

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DESCRIPTION

Fibrosis occurs in the heart, kidneys, liver, skin, or other body organs in response to injury or maladaptive repair processes, leading to decreased overall function and ultimately organ failure. The renin-angiotensin-aldosterone system interacts with the potent profibrotic pathway of transforming growth factor-β to mediate fibrosis in many cell and tissue types. The renin-angiotensin-aldosterone system consists of both classical and alternative signalling pathways that act to either potentiate or antagonize fibrotic signalling mechanisms. This review describes the role of the renin-angiotensin-aldosterone system in the pathogenesis of fibrosis, especially in the liver, heart, kidney and skin, and provides an overview of the current literature related to renin-angiotensin-aldosterone system interactions. A system involving transforming growth focusing factor β signalling. And encourage animal and clinical studies investigating the effects of synthetic drugs. Aldosterone-based angiotensin-based signalling is also being explored as a means of treating fibrosis.

The extracellular matrix is a key component of organ structure and function and is typically dynamically regulated by fibroblasts. In pathological conditions, abnormal extracellular matrix accumulation or fibrosis is induced by pathological stimuli and associated with liver, lung, kidney, and heart organ dysfunction. During the fibrosis process, complex molecular mechanisms such as the renin-angiotensin-aldosterone system and transforming growth factor-beta signalling play critical roles in regulating fibroblast activation and extracellular matrix deposition. In addition to resident fibroblasts, other cell types derived from epithelial/endothelial-mesenchymal junctions, circulating fibrocytes or pericytes have also been reported to contribute to fibrosis. Detailed studies of the molecular and cellular mechanisms of fibrosis can contribute to future treatments of organ failure.

The renin-angiotensin framework plays a basic part within the movement of renal fibrosis. Angiotensin II sort 1 receptor (AT1R) has a place to the B family of the G protein-coupled receptor family. B-Arrestins are known as negative controllers of G protein-coupled receptors. As of late, β-arrestins have been found to direct numerous intracellular signaling pathways autonomous of G proteins. In this think about we explored the part of β-arrestins in directing extracellular lattice union in renal fibrosis. The rodent kidney fibroblast cell line was treated with the β-arrestin one-sided agonist [1-sar, 4, 8-ile] angiotensin, which does not start AT1R-G protein signalling. The cells were transfected with recombinant adenoviruses communicating β-arrestin-2 quality or small-interfering RNA (sirna) focusing on β-arrestin-2. The one-sided ureteral hindrance demonstrates was utilized. MRNA and protein levels of β-arrestin-2, not β-arrestin-1, were altogether upregulated within the ureteral hindrance kidney tissues. SII actuated the tight authoritative of β-arrestin-2 with AT1R. SII expanded the blend of collagen I and fibronectin in NRK-49F, which were nullified when pretreated with candesartan. Transfection of sirna focusing on β-arrestin-2 diminished the impacts of SII on extracellular framework amalgamation. Overexpression of β-arrestin-2 upgraded SII-stimulated ECM union. SII actuated ERK1/2 phosphorylation in NRK-49F. Transfection of sirna focusing on β-arrestin-2 repressed ERK phosphorylation. Overexpression of β-arrestin-2 expanded ERK1/2 phosphorylation. Our ponder to begin with appeared that AT1R-β-arrestin-2 pathway signalling plays an critical part in renal fibrosis, in spite of the fact that it was already accepted that the AT1R-G protein pathway plays a major part. Focusing on β-arrestin-2 may be a potential restorative specialist for renal fibrosis.

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CONFLICT OF INTEREST

The author's declared that they have no conflict of interest.

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