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Mir-351-5p contributes to the establishment of a pro-inflammatory environment in the H9c2 cell line by repressing PTEN expression.

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Abstract

The activated renin-angiotensin-aldosterone system modulates several metabolic pathways that contribute to left ventricular hypertrophy and heart failure. In this metabolic system, angiotensin II modulates heart morphophysiological changes triggered by a series of inflammatory and pro-inflammatory responses; however, the fine tuning associated with the control of this biochemical pathway remains unknown. Here, we investigated elements involved in the post-transcriptional regulation of the pro-inflammatory environment in the H9c2 cardiac cell line, focusing on miRNA elements that modulate PTEN expression. A cellular model of investigation was established and the miR-315-5p was identified as a novel element targeting PTEN in this cardiac cell line, thereby controlling the protein level. This interconnected pathway contributes to the control of the pro-inflammatory environment in Ang II-treated cells.

KEYWORDS: Angiotensin II; Cell culture; PTEN; Post-transcriptional regulation; miRNA

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