Study the effects of tanshinone II A on angiotensin II-induced rat thoracic aortic vascular smooth muscle cell proliferation

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Abstract: Recent evidence suggests that angiotensin II (Ang II) plays an important part in atherosclerosis and cardiovascular disease. Ang II, a potent vasoconstrictor, its role in stimulating abnormal proliferation of vascular smooth muscle cells (VSMCs). Ang II-mediated signal events likely to be important in VSMCs are the increase of cellular reactive oxygen species (ROS), the following phosphorylation of extracellular signal-regulated kinases (ERK) and expression of endothelin-1 (ET-1). As widely studies, tanshinone IIA (Tan IIA) extracted from Salvia miltiorrhizais, a popular medicinal herb used in traditional Chinese medicine, exhibits a variety of cardiovascular activities, including vasorelaxation, and cardioprotective and anti-atherosclerosis effects. However, the effects of TanIIA on VSMCs are not well understood. Therefore, the present study, we used rat thoracic aortic vascular smooth muscle cell line A7r5 to evaluate the effect and investigate mechanisms mediating Ang II-stimulated VSMC proliferation. Prior to the cells were preincubation of Tan IIA and then treated with Ang II to determine the cell viability, ROS production, ERK phosphorylation, and ET-1 expression, were measured by 3-(4,5-dimethylthiazol-2-yl)- 2,5-diphenyltetrazolium bromide (MTT) assay, flow cytometry, western blotting assay and reverse transcription polymerase chain reaction (RT-PCR), respectively. Our results demonstrate that TanIIA significantly reduced Ang II-induced cell proliferation. In addition, Tan II A also significantly suppressed the production of ROS, ERK phosphorylation and ET-1 expression, all of which were decreased by the treatment with Tan IIA. In conclusion, our results suggest that Tan IIA significantly suppresses Ang II-induced VSMCs proliferation in part through the inhibition of ROS production, ERK phosphorylation and ET-1 production.