

Study on Thyroxine Preventing Coronary Atherosclerosis through TGFBR1 Signaling Pathway

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ABSTRACT To explore the mechanism of Thyroxine inhibiting Human Aortic Vascular Smooth Muscle Cell (HASMC) proliferation, migration and vascular intimal hyperplasia through the transforming growth factor β receptor 1(TGFBR1). 3-(4,5-dimethylthiazol-2-yl)-5-(3-carboxymethoxyphenyl)-2-(4-sulfophenyl)-2H-tetrazolium (MTS), 5-ethynyl-2'-deoxyuridine (EdU) staining, Transwell, cell scratch test and Western blotting were performed sequentially to detect the effects of Thyroxine on HASMC and the TGFBR1 signaling pathway. A cell model of TGFBR1 overexpression was constructed to further explore the mechanism of TGFBR1 in Thyroxine inhibiting the activation of HASMC. Thyroxine can inhibit HASMC proliferation and migration, and the activation of TGFBR1 signaling pathway, both in a concentration-dependent manner. Following the overexpression of TGFBR1, the effect of Thyroxine on inhibiting HASMC proliferation, migration and activation of TGFBR1 signaling pathway was partially inhibited. Thyroxine can inhibit the proliferation, migration and vascular intimal hyperplasia of HASMC by inhibiting the activation of TGFBR1 signaling pathway, thereby playing a role in preventing and treating vascular restenosis and related diseases.