題名:Magnolol Depresses Urotensin-II-Induced Cell Proliferation in Rat Cardiac Fibroblasts.

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摘要:1. Accumulating evidence suggests that oxidative stress plays a key role in the development of cardiac fibrosis. Urotensin-II (U-II) has been reported to play an important role in cardiac remodelling and fibrosis. Recently, we demonstrated the involvement of reactive oxygen species (ROS) production in U-II-induced cardiac fibroblast proliferation. Magnolol is an anti-oxidant compound extracted from the cortices of Magnolia officinalis. Thus, it is feasible that magnolol may attenuate cardiac fibroblast proliferation by inhibiting ROS production. Therefore, the aims of the present study were to determine whether magnolol alters U-II-induced cell proliferation and to identify the putative underlying signalling pathways in rat cardiac fibroblasts. 2. Cultured rat cardiac fibroblasts were pretreated with magnolol (1, 3 and 10 micromol/L) for 30 min, followed by exposure to U-II (30 nmol/L) for 24 h, after which cell proliferation and endothelin-1 (ET-1) protein secretion was examined. The effects of magnolol on U-II-induced ROS formation and extracellular signal-regulated kinase (ERK) phosphorylation were examined to elucidate the intracellular mechanisms by which magnolol affects cell proliferation and ET-1 expression. 3. Urotensin-II (30 nmol/L) stimulated cell proliferation, ET-1 protein secretion and ERK phosphorylation, all of which were inhibited by magnolol (10 micromol/L). Pretreatment of cardiac fibroblasts with N-acetylcysteine (5 mmol/L) for 30 min prior to exposure

to U-II resulted in inhibition of U-II increased ROS formation. Similar effects were observed with 10 micromol/L magnolol. 4. In conclusion, the results suggest that magnolol inhibits cardiac fibroblast proliferation by interfering with ROS generation. Thus, the present study provides important new insights into the molecular pathways involved, which may contribute to our understanding of the effects of magnolol on the cardiovascular system.