

GW23-e0630

**HIGH DENSITY LIPOPROTEIN INHIBITS MECHANICAL STRESS-INDUCED CARDIAC HYPERTROPHY THROUGH DOWNREGULATION OF ANGIOTENSIN II TYPE 1 RECEPTOR**

doi:10.1136/heartjnl-2012-302920a.106

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**Objectives** This study is designated to investigate whether HDL inhibits cardiac hypertrophy induced by mechanical stress and whether HDL inhibits AT1 receptor.

**Methods** Ten-week old male mice were subjected to transverse aortic constriction for 2 weeks and were monitored for changes in cardiac structure and function by echocardiography. Hearts were collected 2 weeks after surgery for molecular and histological analyses. In addition, cultured cardiac myocytes were exposed to stretch. myocytes were collected 48 h after stretch for molecular and histological analyses.

**Results** Mechanical stresses for 2 weeks and for 48 h at in vivo and in vitro levels, respectively, resulted in marked cardiac hypertrophic responses including increased protein synthesis, enlarged sizes of cardiomyocytes and hearts, upregulated phosphorylation levels of protein kinases and reprogrammed expression of specific genes, all of which were significantly attenuated by the treatment with HDL. Furthermore, mechanical stress induced upregulation of AT1 receptor expression either in cultured cardiomyocytes or in hearts of mice and HDL significantly suppressed the upregulation of AT1 receptor.

**Conclusions** Our results suggest that HDL inhibited mechanical stress-induced cardiac hypertrophy through downregulation of AT1 receptor expression.