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OXIDISED LOW-DENSITY LIPOPROTEIN INCREASES MECHANICAL STRESS -INDUCED CARDIAC HYPERTROPHY

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Objective This study is designed to investigate whether ox-LDL increases cardiac hypertrophy induced by mechanical stress.

Methods ApoE-deficient (ApoE KO) mice of eight to 10 weeks were divided into three groups, respectively: Sham-operated group (Sham), transverse aorta constriction group (TAC) and transverse aorta constriction plus high fat diet group (HFD+TAC). After two weeks of TAC, cardiac hypertrophy was confirmed by echocardiography and special genes and protein associated with cardiac hypertrophy. We also stretched cardiac myocytes cultured in the presences of ox-LDL and assessed for development of hypertrophy. cardiomyocytes

were cultured and divided into three groups: control group (Control), stretch group (Stretch) and ox-LDL plus stretch group (ox-LDL+Stretch).

Results Mechanical stresses for two weeks and for 24 h at in vivo and in vitro levels, respectively, significantly induced upregulation of Angiotensin II receptor type 1 (AT1) expression and hypertrophic responses, such as increases in cardiomyocytes size and specific gene expressions. Compared with Sham group, all the values of cardiac hypertrophic index for TAC group increased significantly. Compared with TAC group, High fat diet can significantly increase cardiac hypertrophy and significantly increase the upregulation of AT1 receptor induced by pressure overload. In vitro levels, ox-LDL significantly increased cardiac hypertrophy and AT1 receptor expression induced by mechanical stress.

Conclusions Ox-LDL can increase mechanical stress-induced cardiac hypertrophy, and the underlying mechanisms may be that ox-LDL increases the expression of AT1 receptor.