Yuanjie Mao, Transl Med 2013, 3:2
http://dx.doi.org/10.4172/2161-1025.S1.009

OMICS Group Conferences
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2nd International Conference on
Translational & Personalized Medicine
August 05-07, 2013 Holiday Inn Chicago-North Shore, IL, USA

Endogenous ghrelin attenuates cardiac hypertrophy in pressure-overloaded mice through cholinergic anti-inflammatory pathway

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Cardiac hypertrophy, resulting from pressure overload, is the main risk factor of sudden death and heart failure. Ghrelin has been demonstrated to have beneficent effects on cardiac remodeling and heart failure. We hypothesized that it could attenuate cardiac hypertrophy and aimed to clarify the mechanism in this study. Pressure overload was introduced by transverse aortic constriction in ghrelin knockout mice and their wild-type littermates. At 12 weeks later, knockout mice were shown to have significant cardiac hypertrophy and remodeling indicated by greater heart weight/tibial length and augmented left ventricular anterior and posterior wall thicknesses than wild-type mice. Meanwhile, plasma interleukin-1β and interleukin-6 levels were increased and parasympathetic nervous activity represented by high frequency in heart rate variability analysis was decreased in ghrelin knockout mice. Nicotine, administrated orally in knockout mice to activate the cholinergic anti-inflammatory pathway, attenuated the cardiac hypertrophy and remodeling as well as suppressed the elevation of cytokines. In conclusion, endogenous ghrelin plays a beneficial role in cardiac hypertrophy after pressure overload, and it is likely through activation of cholinergic anti-inflammatory pathway.

Biography

Yuanjie Mao has completed his MD and Ph.D. from Peking University Health Science Center, and now undertaking the postdoctoral studies in National Cerebral and Cardiovascular Center, Japan. He has published 5 papers in reputed journals.

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