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HINDLIMB UNLOADING AND RELOADING OF MICE LEAD TO DIFFERENT REMODELING OF LEFT VENTRICLE AND RIGHT VENTRICLE

Abstract

Physiological adaptations to microgravity involve alterations in cardiovascular, neuromuscular, and neuroendocrine systems. These adaptations result in cardiac remodeling and orthostatic hypotension. However, the response of Left ventricular (LV) and right ventricular (RV) following hindlimb unloading and reloading is not clear and the underlying mechanism remains to be disclosed. The purpose of this paper was to identify the different changes of LV and RV following hindlimb unloading and reloading and explore the key molecular regulator involved in this process. In mice, hindlimb unloading (HU) was used to simulate the effects of microgravity induced heart remodeling. Three group mice were subjected to HU by tail suspension for 28 days. After that, two groups recovered for 7 and 14 days, respectively (HU-28d, n=10; HR-7d, n=10; HR-14d, n=10), the control group (n = 10) were treated equally, with the exception of tail suspension. In left ventricle, echocardiography results revealed cardiac enlargement and decreased contractility in HU group. Compared with control, the HU group mice showed higher LVIDs (systolic left ventricular internal diameter) and LV Vols (systolic left ventricular volume), lower LV-EF (left ventricular ejection fraction) and LV-FS (left ventricular fractional shortening). However, mice recovered 7 days after HU (HR-7d) showed much higher LVIDs and LV Vols than both control and HU group. Mice recovered 14 days after being reloaded returned to the normal state. However, in right ventricle, RV-EF and RV-FS didn't recover until being reloaded for 14 days. Histological analysis and cardiac remodeling gene expression showed hindlimb unloading induced left and right ventricular remodeling. Western blot demonstrated that pathological remodeling signals, such as, HDAC4, ERK1/2 and LC3-, were activated following HU and recovered following HR in both LV and RV, and physiological remodeling signal, AMPK, was inhibited in both LV and RV following HU, but only restored in LV following HR for 14 days. These results indicate that simulated microgravity leads to cardiac remodeling, and the remodeling could be recovered, but in the early stage of recovering, cardiac remodeling may be more intensified because of increased pressure load. Moreover, compared with LV, RV is more sensitive to HU and more difficult to restore.