The declined phosphorylation of Hsp27 in rat cardiac muscle after simulated microgravity induced by hindlimb unloading

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Many studies have shown that simulated microgravity induced by hindlimb unloading can decrease the contractility of rat cardiac muscle, however, the mechanisms responsible for which remain unclear. Actin polymerization, which can be regulated by Hsp27, has important role in the transmission of stress force during the contraction of cardiac muscle. In this study, western blot analysis was used to detect the expression of Hsp27 and phosphorylated Hsp27, FAK and phosphorylated FAK, P38 MAPK and phosphorylated P38 MAPK in rat cardiac muscle after 14d hindlimb unloading. The results showed that the phosphorylation levels of both Hsp27 and P38 MAPK were declined significantly, which may decrease actin polymerization and inhibit the transmission of stress force during the contraction of rat cardiac muscle after hindlimb unloading. However, the phosphorylation level of FAK was not declined significantly in cardiac muscle. The results suggested that the declined phosphorylation level of Hsp27, which may be ascribable to the decline of contractility of rat cardiac muscle after 14d hindlimb unloading, may be induced by the declined phosphorylation level of P38 MAPK, but not phosphorylation level of FAK.