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CALPAIN ACTIVATION CONTRIBUTES TO ADULT MOUSE CARDIOMYOCYTES INJURY INDUCED BY H_2O_2

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Background Although restoration of blood flow after ischemia is essential for cardiomyocytes salvation, reperfusion itself exacerbates myocardial injury. Considerable reactive

oxygen species (ROS) are produced by the myocardium and infiltrating inflammatory cells, during the early stage in this process. The exact mechanism for calpain activation by ROS in myocardial is unknown.

Aim We aim to investigate how calpain activity contributes to ROS introduced cells injury.

Methods Adult mice cardiomyocytes were isolated and cultured. The cardiomyocytes were divided into the following groups: control; the H_2O_2 group, cardiomyocytes exposed to MEM with 50 μM H_2O_2 for 6 h; and the PD+H_2O_2 group, cardiomyocytes was treated with MEM containing 25 μM PD150606 (inhibitor of calpain) for 1 h before H_2O_2 treatment. 2-hydroxyethidium (2-OHE), superoxide-specific product, was detected by HPLC. The survival of cardiomyocytes was counted and the activity of calpain, and the activity of caspse3, and the release of cytochrome c were determined in the three groups.

Results The survival of the cells significantly decreased in H_2O_2 treated compared to normal cultured cardiomyocytes ($p<0.05$). The activation of calpain and caspase-3, and cytoplasmic DNA fragments, cytochrome c and 2-OHE concentrations were significantly increased in H_2O_2 treated cardiomyocytes compare to normal group ($p<0.05$). These effects of H_2O_2 on cardiomyocytes were deduced by calpain inhibitor PD150606 ($p<0.05$).

Conclusion ROS may play an important role by inducing calpain activation in the H_2O_2-induced injury of adult cardiac myocytes.