[gw22-e0636] CALPAIN ACTIVATION CONTRIBUTES TO ADULT MOUSE CARDIOMYOCYTES INJURY INDUCED BY H₂O₂

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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$_2$O$_2$ group, cardiomyocytes exposed to MEM with 50 μM H$_2$O$_2$ for 6 h; and the PD+H$_2$O$_2$ group, cardiomyocytes was treated with MEM containing 25 μM PD150606 (inhibitor of calpain) for 1 h before H$_2$O$_2$ 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 caspase3, and the release of cytochrome c were determined in the three groups.

**Results** The survival of the cells significantly decreased in H$_2$O$_2$ 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$_2$O$_2$ treated cardiomyocytes compare to normal group (p<0.05). These effects of H$_2$O$_2$ 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$_2$O$_2$-induced injury of adult cardiac myocytes.