1-O-D1-4 Oral Sessions

2,5-Dimethylcelecoxib attenuates cardiac remodeling after cryoinjury-induced myocardial infarction by suppressing the fibroblast-myofibroblast transformation

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Secretion of extracellular matrix by myofibroblasts, transform from fibroblasts and express alpha smooth muscle actin (aSMA), lead to cardiac fibrosis and progress cardiac remodeling. We previously reported the 2,5-dimethylcelecoxib (DM-C) attenuated cardiac remodeling in different type of cardiac hypertrophy models. However, as the effect of DM-C on myofibroblasts transformation and cardiac remodeling after myocardial infarction (MI) are still unclear, we tried to clarify these points. In *in vitro* study, dermal fibroblasts from SD rat were used for analysis of myofibroblast transformation. In *in vivo* study, cryoinjury-induced MI (CMI) mouse model was used. In vitro studies showed that DM-C significantly suppressed the expression of aSMA and the phosphorylation levels of Akt, GSK-3 b and Smad2/3, all of them were elevated by TGF-b treatment. In vivo studies showed that the expression of aSMA and fibrosis area were decreased in DM-C group compared with control group. Further, evaluation of the cardiac function revealed that DM-C-treatment alleviated the reduction of the ejection fraction. These results suggest that DM-C attenuates cardiac remodeling after CMI by inhibiting fibrosis via suppression of the myofibroblast transformation. Therefore, DM-C has potential as the novel drug for treatment of cardiac remodeling after MI.