

The Inhibition of RNF20 Causes Inflammation in the Model of Myocardial Infarction through the NLRP3 Inflammasome Activation

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ABSTRACT This study aimed to examine the function and its possible molecular biological mechanisms of Ring Finger Protein 20 (RNF20) in the model of myocardial infarction (MI). Patients with MI infarction were collected and analysed. C57BL/6J mice or H9c2 cell were induced using the left anterior descending coronary artery or hydrogen peroxide and Lipopolysaccharides. The serum mRNA expression of RNF20 levels in patients with MI or in the mice with MI was down regulated. RNF20 gene reduced inflammation *in vitro* model of MI. Knock-out of RNF20 causes inflammation and MI in the mice model. RNF20 suppressed NLRP3 inflammasome by Vitamin D Receptor. RNF20 interacted with Vitamin D Receptor to reduce degradation of Vitamin D Receptor. Vitamin D Receptor interacted with NLRP3 protein. The inhibition of RNF20 causes inflammation in the model of MI through NLRP3 activation by Vitamin D Receptor, and might be a potential treatment target for the MI -related diseases.