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Letter to the Editor

MALAT1/miR-144/Brg1: A potential regulated axis of inflammation in myocardial ischemia-reperfusion injury

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Dear Editor,

Zhang et al. recently reported that ablation of brahma related gene 1 (Brg1) in the endothelium suppressed neutrophil infiltration and down-regulated the levels of pro-inflammatory mediators in the heart following ischemia-reperfusion (IR) injury [1]. This finding implies that Brg1 is an effective therapeutic target for improving inflammation in myocardial IR injury. However, the mechanism by which Brg1 is regulated in myocardial IR injury is still unclear.

Recently, Li et al. suggested that miR-144-3p contributes to oxygen-glucose deprivation and reoxygenation (OGD/R)-induced neuronal injury in vitro through negatively regulating Brg1 signaling [2]. This finding implies that miR-144-3p can negatively regulate Brg1 and miR-144/Brg1 axis maybe a potential regulated axis of inflammation in myocardial IR injury. Long non-coding RNAs (LncRNAs) are highly expressed in the cardiomyocytes and regulate their physiological and pathophysiological processes [3]. However, the potential role of LncRNAs in myocardial IR injury remains largely unknown. Recently, It has been confirmed that the expression of lncRNA metastasis-associated lung adenocarcinoma transcript 1 (MALAT1) is significantly increased in the myocardium of patients with myocardial infarction and is closely associated with IR injury [4]. Interestingly, the constructed luciferase assay verified that MALAT1 was a target of miR-144-3p and MALAT1 can inhibit the function of miR-144 [5]. This means that the MALAT1/miR-144-3p axis may play a key role in myocardial IR injury.

Therefore, it is speculated that MALAT1/miR-144/Brg1 axis is a potential regulated axis of inflammation in myocardial I/R injury. However, this speculation needs to be further proved by experimental evidence.

Conflict of interest

The authors report no relationships that could be construed as a conflict of interest.

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