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Letter to the Editor

MALAT1/miR-204/LC3-II: A potential regulated axis of autophagy in myocardial ischemia-reperfusion injury

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Dear Editor,

Yu et al. recently reported that lncRNA metastasis-associated lung adenocarcinoma transcript 1 (MALAT1) may increase cardiomyocyte autophagy and myocardial injury during ischemia-reperfusion (I/R) by negatively regulating microRNA-204 (miR-204) expression [1]. This finding implies that MALAT1/miR-204 axis may act as a novel therapeutic strategy for I/R-induced autophagy.

It has been confirmed that the expression of MALAT1 is significantly increased in the myocardium of patients with myocardial infarction and is closely associated with the I/R-induced autophagy and I/R injury [2]. However, the regulatory mechanisms involved are not clear. Autophagy is a homeostatic, tightly regulated process that provides organelle quality control and generates intracellular nutrients from lysosomal processing of cellular structures [3]. Its overactivation can lead to self-digestion and subsequent cell death, thus playing an important role in a variety of myocardial I/R injury. A lot of literatures have confirmed that MALAT1 can sponge miR-204 and inhibit the function of miR-204, which confirm the guess of Yu et al. again [4]. Interestingly, Xiao et al. confirmed that miR-204 plays an important role in regulating autophagy through LC3-II protein during myocardial I/R [5]. This means that the miR-204/LC3-II axis plays a key role in myocardial I/R injury.

Therefore, it is speculated that MALAT1/miR-204/LC3-II axis is a potential regulated axis of autophagy 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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