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Letter to the Editor

LncRNA NEAT1 aggravates diabetic myocardial ischemia-reperfusion injury through regulating PINK1 by targeting miR-27b

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Dear Editor,

We have recently read the report by Li et al. that inhibition of PTEN-induced putative kinase 1 (PINK1)/Parkin pathway activation is an effective treatment to improve myocardial ischemia/reperfusion (MI/R) injury [1]. However, the specific mechanism by which PINK1 expression is regulated in MI/R injury has not yet been elucidated.

Recently, Kim et al. revealed that miR-27b can negatively regulate PINK1 expression and promote autophagic clearance of damaged mitochondria [2]. This implies that promoting PINK1 expression by inhibiting miR-27b is likely to be a potential mechanism for improving MI/R injury. In addition, long non-coding RNA (lncRNA)-nuclear-enriched abundant transcript (NEAT1) is highly expressed in the diabetic cardiomyocytes and regulating their physiological and pathophysiological processes [3]. However, the potential role of NEAT1 in diabetic MI/R injury remains largely unknown. Recently, it has been confirmed that elevated NEAT1 expression aggravates MI/R injury via activation of apoptosis and autophagy in diabetic rats [3]. Interestingly, Wang et al. confirmed that lncRNA NEAT1 can sponge miR-27b and inhibit the function of miR-27b, which was validated by dual-luciferase reporter assay and RNA immunoprecipitation experiments [4]. Thence, NEAT1/miR-27b/PINK1 axis is probably the key mechanism for regulating diabetic MI/R injury.

Based on the mutual corroboration between the above conclusions, we speculated that lncRNA NEAT1 may aggravates diabetic MI/R injury through regulating PINK1 via targeting miR-27b. However, this speculation needs to be further verified by experimental evidence.

Conflict of interest

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

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