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Letter to the Editor

LncRNA MALAT1 aggravates inflammation response through regulating PTGS2 by targeting miR-26b in myocardial ischemia-reperfusion injury

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Dear Editor,

Recently, Ge et al. reported that microRNA-26b (miR-26b) inhibits prostaglandin-endoperoxide synthase 2 (PTGS2, also called as COX-2) to activate the MAPK pathway, so as to reduce inflammatory response and improve myocardial remodeling in mice with myocardial infarction (MI) [1], which has attracted our attention. Although it has demonstrated that miR-26b may play a key role in regulating the inflammatory response in myocardial ischemia/reperfusion (MI/R) injury, the specific mechanisms regulating miR-26b expression have not yet been elucidated.

Long non-coding RNA (lncRNA)-metastasis-associated lung adenocarcinoma transcript 1 (MALAT1) is significantly increased in the myocardium of patients with MI [2] and regulates cardiovascular physiology and pathological processes [3]. In addition, it has shown that lncRNAs can act as ceRNAs by binding to consensus MREs (miRNA response element), thereby reducing the level of miRNA available for target mRNA. Recently, Li et al. revealed that MALAT1 can act as ceRNAs by binding to consensus MREs of miR-26b and reduce the level of miR-26b available for target mRNA [4]. This implies that MALAT1/miR-26b/PTGS2 axis is probably an important mechanism for regulating inflammation in MI/R injury.

Based on the mutual corroboration between the above conclusions, we speculated that lncRNA MALAT1 may aggravate inflammation response through regulating PTGS2 by targeting miR-26b in MI/R injury. 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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References

[1] Z.W. Ge, X.L. Zhu, B.C. Wang, J.L. Hu, J.J. Sun, S. Wang, et al., MicroRNA-26b relieves inflammatory response and myocardial remodeling of mice with myocardial infarction by suppression of MAPK pathway through binding to PTGS2, Int. J. Cardiol. 280 (2019) 152–159.
[2] M.H. Bao, V. Szeto, B.B. Yang, S.Z. Zhu, H.S. Sun, Z.P. Feng, Long non-coding RNAs in ischemic stroke, Cell Death Dis. 9 (2018) 281.
[3] M. Ma, J. Hui, Q.Y. Zhang, Y. Zhu, Y. He, X.J. Liu, Long non-coding RNA nuclear-enriched abundant transcript 1 inhibition blunts myocardial ischemia reperfusion injury via autophagic flux arrest and apoptosis in streptozotocin-induced diabetic rats, Atherosclerosis 277 (2018) 113–122.
[4] Z. Li, J. Li, N. Tang, Long noncoding RNA Malat1 is a potent autophagy inducer protecting brain microvascular endothelial cells against oxygen-glucose deprivation/reoxygenation-induced injury by sponging miR-26b and upregulating ULK2 expression, Neuroscience 354 (2017) 1–10.

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