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Letter to the Editor

TUG1/miR-421/PINK1: A potential mechanism for treating myocardial ischemia-reperfusion injury

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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]. This suggests that inhibition of PINK1 expression is an important mechanism for treating MI/R injury. However, the mechanism for regulating PINK1 expression in MI/R injury has not yet been elucidated.

Recently, Wang et al. revealed that miR-421 promotes cardiomyocytes apoptosis and myocardial infarction by suppressing PINK1 translation [2], which reminds that miR-421/PINK1 axis is likely to be an effective mechanism for treating MI/R injury. Interestingly, emerging evidence shows that long non-coding RNA (lncRNA) is abundantly expressed in cardiomyocytes and plays an important regulatory role in MI/R injury [3]. Wu et al. showed that overexpression of TUG1 aggravated MI/R and TUG1 may be an effective diagnostic marker and therapeutic target for MI/R [4]. In addition, lncRNAs can act as ceRNAs by binding to consensus miRNA response element, thereby reducing the level of miRNA available for target mRNA. Li et al. revealed that TUG1 competitively inhibited the expression of miR-421 and abolished the inhibitory effect of miR-421 on expression of target mRNA [5]. Thence, TUG1/miR-421/PINK1 axis is probably the key mechanism for regulating MI/R injury.

According to the conclusions of the above articles and the interconnection between them, we speculated that lncRNA TUG1 may exacerbate MI/R injury through regulating PINK1 by targeting miR-421. 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] F. Li, X. Fan, Y. Zhang, Y. Zhang, X. Ma, J. Kou, et al., Inhibition of myosin IIA-actin interaction prevents ischemia/reperfusion induced cardiomyocytes apoptosis through modulating PINK1/Parkin pathway and mitochondrial fission, Int. J. Cardiol. 271 (2018) 211–218.
[2] K. Wang, L.Y. Zhou, J.X. Wang, Y. Wang, T. Sun, B. Zhao, et al., E2F1-dependent miR-421 regulates mitochondrial fragmentation and myocardial infarction by targeting Pink1, Nat. Commun. 6 (2015) 7619.
[3] C.Y. Liu, Y.H. Zhang, R.B. Li, L.Y. Zhou, T. An, R.C. Zhang, et al., LncRNA CAIF inhibits autophagy and attenuates myocardial infarction by blocking p53-mediated myocardin transcription, Nat. Commun. 9 (2018) 29.
[4] Z. Wu, S. Zhao, C. Li, C. Liu, LncRNA TUG1 serves an important role in hypoxia-induced myocardial cell injury by regulating the miR-145-5p-Binp3 axis, Mol. Med. Rep. 17 (2) (2017) 2422–2430.
[5] G. Li, H. Song, L. Chen, W. Yang, K. Nan, P. Lu, TUG1 promotes lens epithelial cell apoptosis by regulating miR-421/caspase-3 axis in age-related cataract, Exp. Cell Res. 356 (2017) 20–27.

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