International Journal of Cardiology 278 (2019) 224

Contents lists available at ScienceDirect

International Journal of Cardiology

journal homepage: www.elsevier.com/locate/ijcard

Letter to the Editor

LncRNA H19 ameliorates myocardial ischemia-reperfusion injury by targeting miR-22-3P

Bo-fang Zhang, Jing Chen *, Hong Jiang *

Department of Cardiology, Renmin Hospital of Wuhan University, Cardiovascular Research Institute of Wuhan University, Wuhan, 430060, Hubei Province, China

ARTICLE INFO

Article history:
Received 3 November 2018
Accepted 6 November 2018

Keywords:
LncRNA H19
miR-22-3P
Myocardial ischemia-reperfusion injury

Dear Editor:

We have recently read the report by Boven et al. that "Serially measured circulating miR-22-3p is a biomarker for adverse clinical outcome in patients with chronic heart failure: The Bio-SHiFT study" [1]. This clinical study implied that miR-22-3p was a potential diagnostic biomarker for patients with chronic heart failure. Here, we speculate that miR-22-3p may play a crucial role in LncRNA H19 mediated cardioprotective efficiency during myocardial ischemia-reperfusion injury (MIRI).

The LncRNA H19 locates on human chromosome 11, which is abundantly existed in embryonic tissues, but mainly expressed in skeletal muscle and heart after birth. Recently, Wang et al. demonstrated that LncRNA H19 could protect cardiomyocytes from programmed necrosis induced by hypoxia through regulating miR-103/107 [2]. Besides, Yu et al. indicated LncRNA H19 alleviated apoptosis by targeting miR-29b during the pathological process of acute myocardial infarction [3]. Therefore, it is reasonable to speculate LncRNA H19 might be a novel therapeutic option for the treatment of ischemic myocardial damage via interacting with pivotal miRNAs. Moreover, both bioinformatics software and prospective experiment have confirmed that LncRNA H19 possesses a miR-22-3p binding site at the 3′UTR and acts as a competing endogenous RNA to suppress the activity of miR-22-3p [4]. Of note, miR-22-3p has been affirmed to aggravate apoptosis caused by MIRI via accelerating mitochondrial oxidative stress [5].

In summary, LncRNA H19/miR-22-3p axis might be a potential regulated signaling pathway of apoptosis in MIRI. However, more systematic researches still need to be performed to provide ample evidence for the speculation.

Conflict of interest statement

None.

Acknowledgement

This work was supported by the National Natural Science Foundation of China (No. 81570331),

References

[1] N. van Boven, K.M. Akkerhuis, S.S. Anroedh, et al., Serially measured circulating miR-22-3p is a biomarker for adverse clinical outcome in patients with chronic heart failure: the bio-SHiFT study, Int. J. Cardiol. 235 (2017) 124–132.
[2] J.X. Wang, X.J. Zhang, Q. Li, et al., MicroRNA-103/107 regulate programmed necrosis and myocardial ischemia/reperfusion injury through targeting FADD, Circ. Res. 117 (4) (2015) 352–363.
[3] B.Y. Yu, B. Dong, LncRNA H19 regulates cardiomyocyte apoptosis and acute myocardial infarction by targeting miR-29b, Int. J. Cardiol. 271 (2018) 25.
[4] X. Wang, M. Zou, J. Li, et al., LncRNA H19 targets miR-22 to modulate H2 O2 -induced deregulation in nucleus pulposus cell senescence, proliferation, and ECM synthesis through Wnt signaling, J. Cell. Biochem. 119 (6) (2018) 4990–5002.
[5] J.K. Du, B.H. Cong, Q. Yu, et al., Upregulation of microRNA-22 contributes to myocardial ischemia-reperfusion injury by interfering with the mitochondrial function, Free Radic. Biol. Med. 96 (2016) 406–417.

* Corresponding authors at: Department of Cardiology, Renmin Hospital of Wuhan University, 238 Jiefang Road, Wuchang, Wuhan 430060, China.
E-mail addresses: chenjing821111@163.com (J. Chen), hong-jiang@whu.edu.cn (H. Jiang).

https://doi.org/10.1016/j.ijcard.2018.11.017
0167-5273/