Letter to the Editor

LncRNA H19 regulates cardiomyocyte apoptosis and acute myocardial infarction by targeting miR-29b

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Dear Editor:

We have recently read the report by Grabmaier et al. [1] concerning “Diagnostic and prognostic value of miR-1 and miR-29b on adverse ventricular remodeling after acute myocardial infarction - The SITAGRAMI-miR analysis”. This clinical study provided insights into the potential of miR-29b as a diagnostic biomarker for patients with acute myocardial infarction (AMI). However, very little is known about the molecular mechanism by which miR-29b is regulated in this scenario.

The lncRNA H19, a 2.3 kb lncRNA transcribed from the H19 gene, is highly expressed in the developing embryo, but its expression is significantly downregulated after birth, except in cardiac and skeletal muscle. A recent study demonstrated that H19 might play a protective role in H2O2-induced H9c2 cell necrosis through inhibiting miR-103/107 [2]. More recently, Gong et al. [3] found that H19 could alleviate hypoxia-induced myocardial cell injury by sponging miR-139. These findings suggest the possible involvement of H19 in ischemic heart diseases (including AMI) via targeting miRNAs. It is worth to note that H19 can also interact with miR-29b through directly binding to the 3’UTR and function as a competing endogenous RNA to suppress miR-29b action [4]. More notably, miR-29b has been identified to participate in the regulation of cardiomyocyte apoptosis upon H2O2 treatment [5].

In summary, we speculate that lncRNA H19 may protect cardiomyocytes against AMI via anti-apoptosis by targeting miR-29b. However, more systematic research needs to be performed to explore the underlying rules that modulate the interaction between lncRNA H19 and miR-29b in this context.

Conflict of interest

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

References


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