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Editorial

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The Role of Long Non-Coding RNA (LncRNA) in Acute Myocardial Infarction: Novel Strategy for the Treatment of Acute Myocardial Infarction

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▸ See the article "LncRNA AC005332.7 Inhibited Ferroptosis to Alleviate Acute Myocardial Infarction Through Regulating miR-331-3p/CCND2 Axis" in volume 53 on page 151.

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Acute myocardial infarction (AMI) refers to fatal symptom of myocardial ischemia caused by coronary artery occlusion and it further promotes myocardial necrosis. Therefore, patients' quality of life is extremely destroyed and even loses their life. Currently, ischemia-reperfusion therapy can better improve the injury and necrosis of cardiomyocytes. In Korea, it has been reported that the characteristics of patients with AMI and its disease patterns have gradually changed, and revascularization practices have also improved dramatically.[1] However, the treatment of ischemia-reperfusion is very time-limited, and it usually causes secondary damage to the myocardium, which limit the application of these methods in AMI. Moreover, the mechanism of cardiomyocyte injury caused by AMI is extremely diverse and complicated. Hence, it is essential to study the biological mechanism of cardiomyocyte injury caused AMI and recently, various methods to investigate the mechanism of cardiovascular diseases are developed including single-cell RNA sequencing.[2]

AMI contributes to a large number of myocardial cell death, which further leads to cardiac dysfunction and serious consequences for patients. Programmed cell death including apoptosis and necroptosis plays a pivotal role in various biological processes such as development, immune response and tumorigenesis.[3] Ferroptosis was recently categorized in non-apoptotic cell death. It is processed by the oxidative modification of phospholipid membranes via an iron-dependent manner. Ferroptosis-inducing factors can directly or indirectly affect glutathione peroxidase through different pathways, resulting in a decrease in antioxidant capacity and accumulation of lipid reactive oxygen species in cells, ultimately leading to oxidative cell death. Although the physiological function of ferroptosis is poorly understood, it is closely related to the pathophysiological processes of many diseases, such as tumors, nervous system diseases, kidney injury, blood diseases, and ischemia-reperfusion injury.[4] At present, ferroptosis has been reported to play an indispensable role in the development of AMI, establishing a positive correlation between ferroptosis and AMI.[5] However, the detailed mechanism of ferroptosis in AMI has not been investigated.

Conflict of Interest
The author has no financial conflicts of interest.

Data Sharing Statement
The data generated in this study are available from the corresponding author upon reasonable request.

Long non-coding RNAs (LncRNAs) are defined as RNAs longer than 200 nucleotides in length. These are not translated into functional proteins. Some of them have microRNA (miRNA)-complementary sites and they regulate gene expression through competitive endogenous RNAs or absorb miRNAs like sponges. Thereby they can reduce miRNA availability to target mRNAs.[6] They mediate physiological and pathological processes of various diseases including AMI.

Dai et al.[7] reported that LncRNA, AC005332.7 suppressed ferroptosis and alleviated AMI. In this study, they found that LncRNA (AC005332.7) reduced the level of miR-331-3p like the sponge absorbs. They also found the novel target of miR-331-3p, CCND2 (Cyclin D2), which is known as the crucial factor in regulating biological functions of cardiomyocytes.[8] CCND2 overexpression could reduce infarct size and alleviate ischemia-induced cardiomyocyte injury.[9] Reduction of miR-331-3p increased the level of CCND2 to promote cell viability and suppress ferroptosis, thereby alleviating AMI.[7]

Therefore, they suggested new strategies for the treatment of AMI with regulating LncRNAs.

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