Peer Review File

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Reviewer A:

Thanks for the invitation to review the manuscript "Long non-coding RNA SENCR alleviates H/R-induced cardiomyocyte apoptosis and inflammatory response by sponging miR-1" that revealed the clinical significance and the function of SENCR in AMI patients and H/R-induced cardiomyocyte apoptosis. The experiments of this study are well-designed, but some details need to be completed as follows:

1. In the description of PCR method, the reaction condition should be specified.

Reply: Thanks for your suggestion. We have added the reaction condition of PCR in our revised manuscript.

Changes in the text: Page 4 Line 88-89. The reaction conditions were 95°C for 10 min, 40 cycles of 95°C for 10 s and 60°C for 45 s, then 95°C for 15 s, 60°C for 1min, 95°C for 15 s and 60°C for 15 s.

2. The description of ELISA was too simple, it should be expanded to make the readers clear.

Reply: Thanks for your suggestion. We have supplemented the measurement of the absorbance in our revised manuscript.

Changes in the text: Page 4 Line 93

3. The specific use of statistical analysis methods should be clarify and the post-hoc test should be stated.

Reply: Thanks for your suggestion. The description of the statistical analysis methods has been completed in our revised manuscript.

Changes in the text: Page 5 Line115. One-way ANOVA followed by the Turkey post-hoc.

Reviewer B:

a) The number of recruited patients should be added in Figure 1.

Reply: Thanks for your suggestion. We have added the number of AMI patients and healthy volunteers in our Figure 1.

Changes in the text: Revised Figure 1.

b) In the mechanism experiments, the binding sites between miR-1 and SENCR were showed in the Figures. However, it is necessary to show the sequence of SENCR MUT to declaim SENCR MUT did not bind with miR-1.

Reply: Thanks for your suggestion. We have added the sequences of SENCR MUT in Figure 3.

Changes in the text: Revised Figure 3.

Reviewer C:

It is an interesting and meaningful study that investigated the role of SENCR in cardiomyocyte apoptosis. It was found that SENCE was downregulated in AMI patients and was negatively correlated with the cTnI and CK-MB of patients. In H/R induced

cell apoptosis, SENCR showed notably reversed effect to protect cardiomyocyte from H/R induced injury by sponging miR-1. These results provided evidence for the protective effect of SENCR on cell apoptosis induced by H/R and declaimed its potential mechanism. It is relatively well-writing and logistically constructed, and the topic was consistent with Cardiovascular Diagnosis and Therapy. I give approval for publication to this article.

Reply: Thanks for your comments. We have revised our manuscript according to reviewers' suggestions. We are glad to receive your further comments and suggestions on our revised manuscript. Thanks for your help and attention again.