

S100A9 as a Key Myocardial Injury Factor Interacting with ATP5 Exacerbates Mitochondrial Dysfunction and Oxidative Stress in Sepsis-Induced Cardiomyopathy [Corrigendum]

Pei H, Qu J, Chen J, Zhao G, Lu Z. *J Inflamm Res.* 2024;17:4483–4503.

The authors advised that Figure 8A on page 4496 has an error in the merge image of S100A9^{KD}-lps group. The correct Figure 8 is as follows.

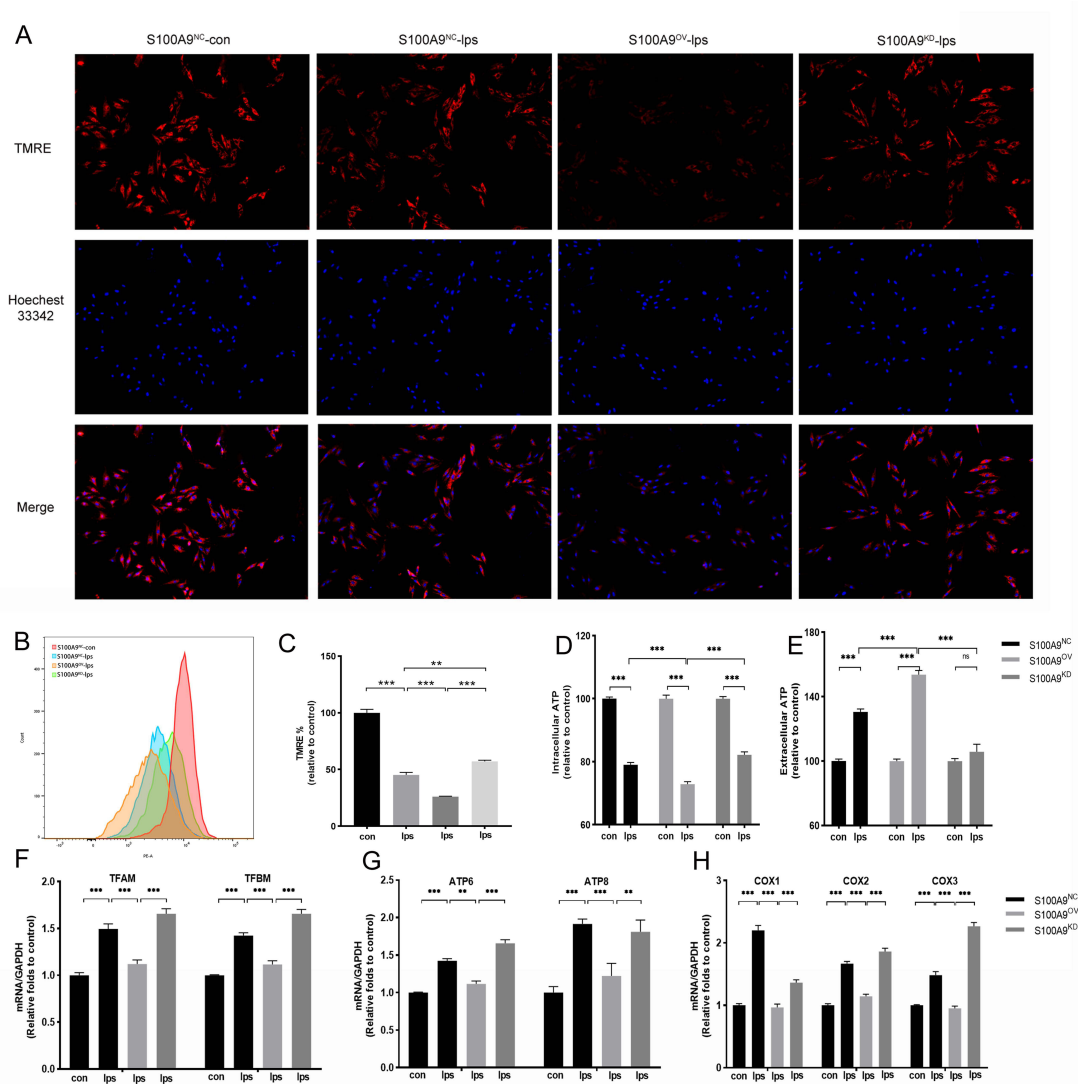


Figure 8 S100A9 induces mitochondrial damage and energy metabolism impairment. (A) MMP fluorescence staining by TMRE probe (red) and Hoechst 33342 (blue). (B and C) Flow cytometry detection of TMRE fluorescence intensity in H9C2 cells. (D and E) Detection of intracellular and extracellular ATP content by fluorescence luminescence method. (F–H) Mitochondrial mRNA transcript levels of TFAM, TFBM, ATP6, ATP8, COX1, COX2 and COX3 by qPCR. The above statistical results were compared with the corresponding control group. *P<0.05. **P<0.01, ***P<0.001. **Abbreviations:** Con, Control; LPS, Lipopolysaccharides; NC, negative control; OV, Overexpression of S100A9 gene; KD, Knockdown of S100A9 gene; TMRE, tetramethylrhodamine, specifically recognize mitochondrial membrane potentials; MMP, mitochondrial membrane potential.

The authors apologize for this error and advise it does not change the results and conclusion for this paper.

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