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CORRIGENDUM

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.

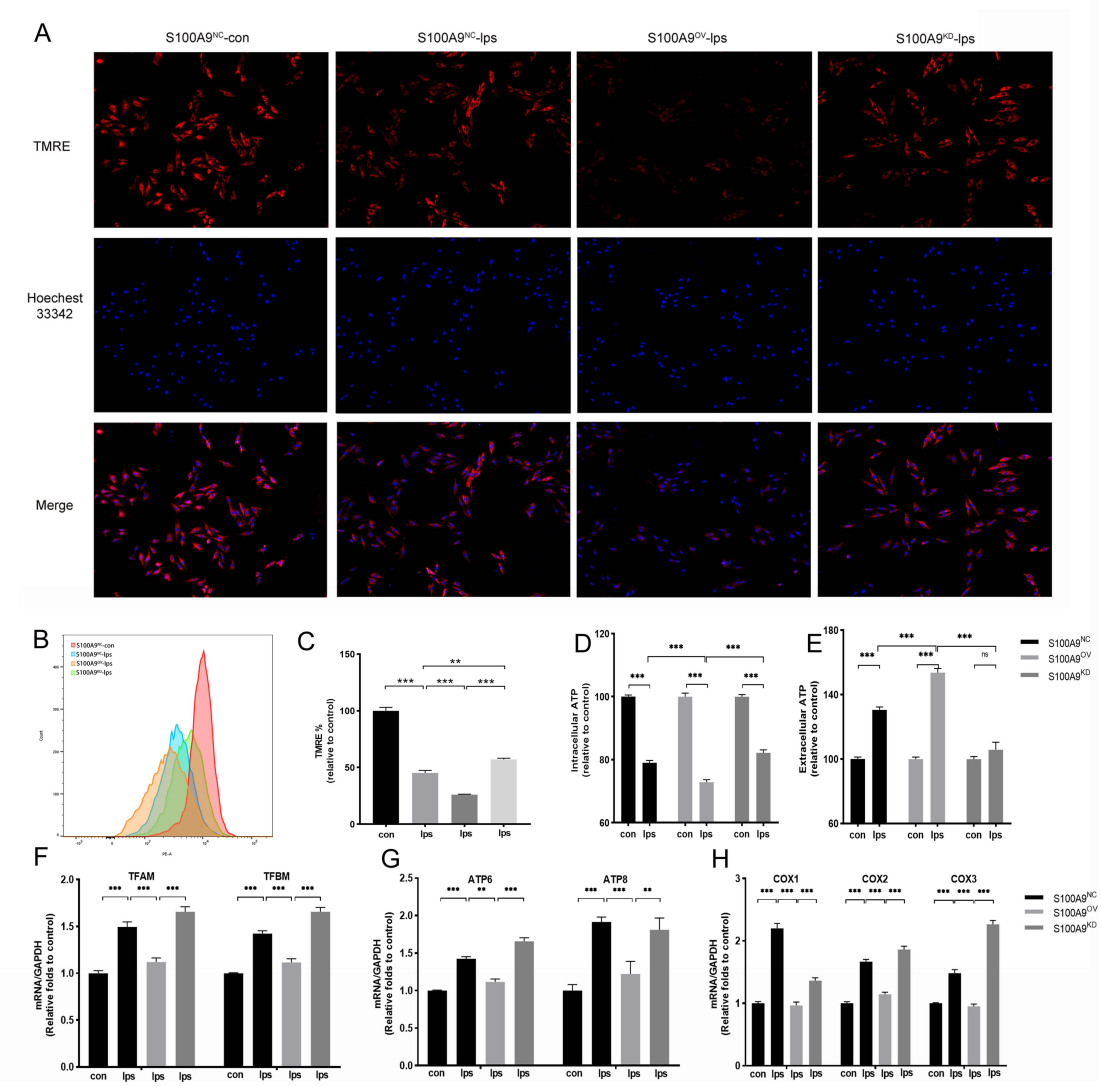

Figure 8 S100A9 induces mitochondrial damage and energy metabolism impairment. (**A**) MMP fluorescence staining by TMRE probe (red) and Hoechst 33342 (blue). (**B** and **C**) Flow cytometry detection of TMRE fluorescence intensity in H9C2 cells. (**D** and **E**) Detection of intracellular and extracellular 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.

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

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