CORRECTION



Correction: Zafirlukast ameliorates lipopolysaccharide and bleomycin-induced lung inflammation in mice

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Following publication of the original article [1], the authors identified an error in Fig. 1A (H&E lung) and Fig. 4. The correct figures are given below.

The original article has been corrected.

The online version of the original article can be found at https://doi.org/10.1186/s12890-024-03273-6.

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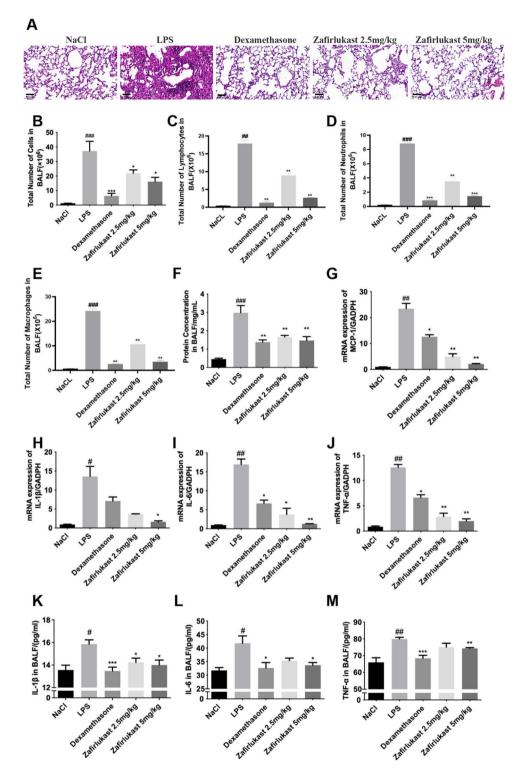


Fig. 1 Zafirlukast attenuates acute lung injury induced by LPS in mice. A H&E staining of lung tissue segments (Scale: 50μ m) and the BALF cell of mice (Scale: 50μ m). B Total cell counts in BALF of mice. C-E Number of inflammatory cells, lymphocytes, neutrophils, and macrophages. F Protein concentration in BALF of mice. G–J MCP-1, IL–1 β , IL-6 and TNF- α mRNA expression in lung tissues of mice. K-M Measure the protein level of inflammatory factors IL–1 β , IL-6 and TNF- α in BALF using ELISA. The data which presented in this study are expressed as mean ± SD (*n*=3). the statistical analysis revealed significant differences when compared with the control group, denoted as #P < 0.05, #P < 0.01, ##P < 0.001, *P < 0.05, **P < 0.01, ***P < 0.001 as compared with model group

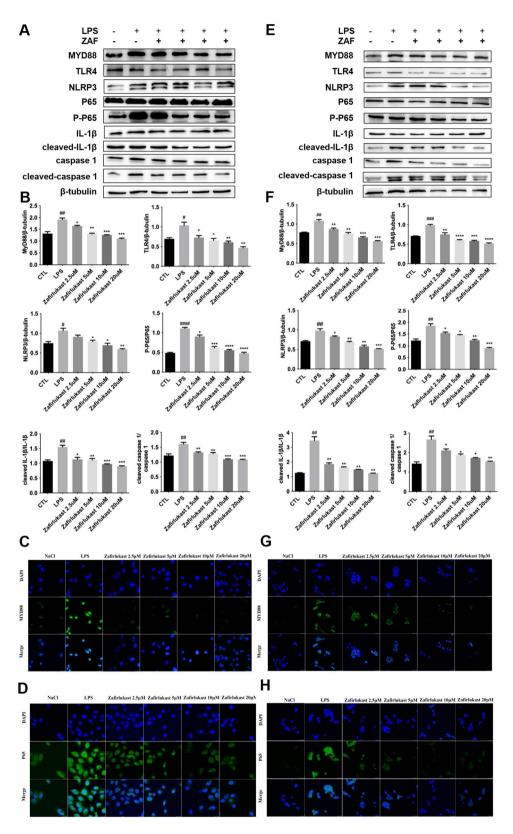


Fig. 4 (See legend on next page.)

(See figure on previous page.)

Fig. 4 Zafirlukast effectively inhibits the LPS-induced inflammation of epithelial cells through TLR4/NF-KB/NLRP3 pathway in vitro. (**A-B**) A549 cells were subjected to different concentration of LPS or Zafirlukast (2.5 μ M, 5 μ M, 10 μ M, 20 μ M) for a duration of 24 h, Western blotting was then performed for assessing the protein expression levels of MyD88, TLR4, P65, P-P65 as well as inflammasome pathway-related protein NLRP3, caspase1, Cleaved-caspase1, IL-1 β and Cleaved-IL-1 β in the cells (**A**). The quantification of optical density was also determined (**B**). (**C-D**) Additionally, the activation of MYD88 (**C**) and P-P65 (**D**) by immunofluorescence in A549 cells subjected to LPS or Zafirlukast (2.5 μ M, 5 μ M, 10 μ M, 20 μ M) for 24 h (Scale: 25 μ m). (**E-F**) MLE-12 cells were subjected to LPS or Zafirlukast (2.5 μ M, 5 μ M, 10 μ M, 20 μ M) for 24 h, and the Western blotting was used to assess the MyD88, TLR4, P65, P-P65 and the inflammasome pathway related protein NLRP3, caspase1, Cleaved-caspase1, IL-1 β and Cleaved-IL-1 β protein expression levels in the cells (**E**). The optical density was quantified and presented (**F**). (**G-H**) MLE-12 cells were subjected to LPS or Zafirlukast (2.5 μ M, 5 μ M, 10 μ M, 20 μ M) for 24 h, and the activation of MYD88 (**G**) and P-P65 (**H**) was assessed through immunofluorescence (Scale: 25 μ m). Data are shown as mean ± SD (n=3). Statistical significance was denoted as follows: #P<0.05, ##P<0.001, ****P<0.001 compared with control group, *P<0.05, **P<0.01, ****P<0.001, ****P<0.001 compared with model group

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References

1. Xue T, Zhang Q, Zhang T, et al. Zafirlukast ameliorates lipopolysaccharide and bleomycin-induced lung inflammation in mice. BMC Pulm Med. 2024;24:456. https://doi.org/10.1186/s12890-024-03273-6.