

CORRECTION

Open Access



Correction: L-Arginine, as an essential amino acid, is a potential substitute for treating COPD via regulation of ROS/NLRP3/NF- κ B signaling pathway

Chunhua Ma^{1,2†}, Kexi Liao^{3†}, Jing Wang^{4†}, Tao Li^{1*} and Liangming Liu^{1*}

Correction: *Cell & Bioscience* (2023) 13:152
<https://doi.org/10.1186/s13578-023-00994-9>

In this article [1], the wrong figures appeared as Figs. 3 and 7; the figures should have appeared as shown below.

[†]Chunhua Ma, Kexi Liao and Jing Wang contributed equally.

The original article can be found online at <https://doi.org/10.1186/s13578-023-00994-9>.

*Correspondence:

Tao Li
lt200132@tmmu.edu.cn

Liangming Liu
lmliu62@163.com

¹ State Key Laboratory of Trauma, Burns and Combined Injury, Shock and Transfusion Research, Department of Army Medical Center, Army Medical University, Chongqing 400042, People's Republic of China

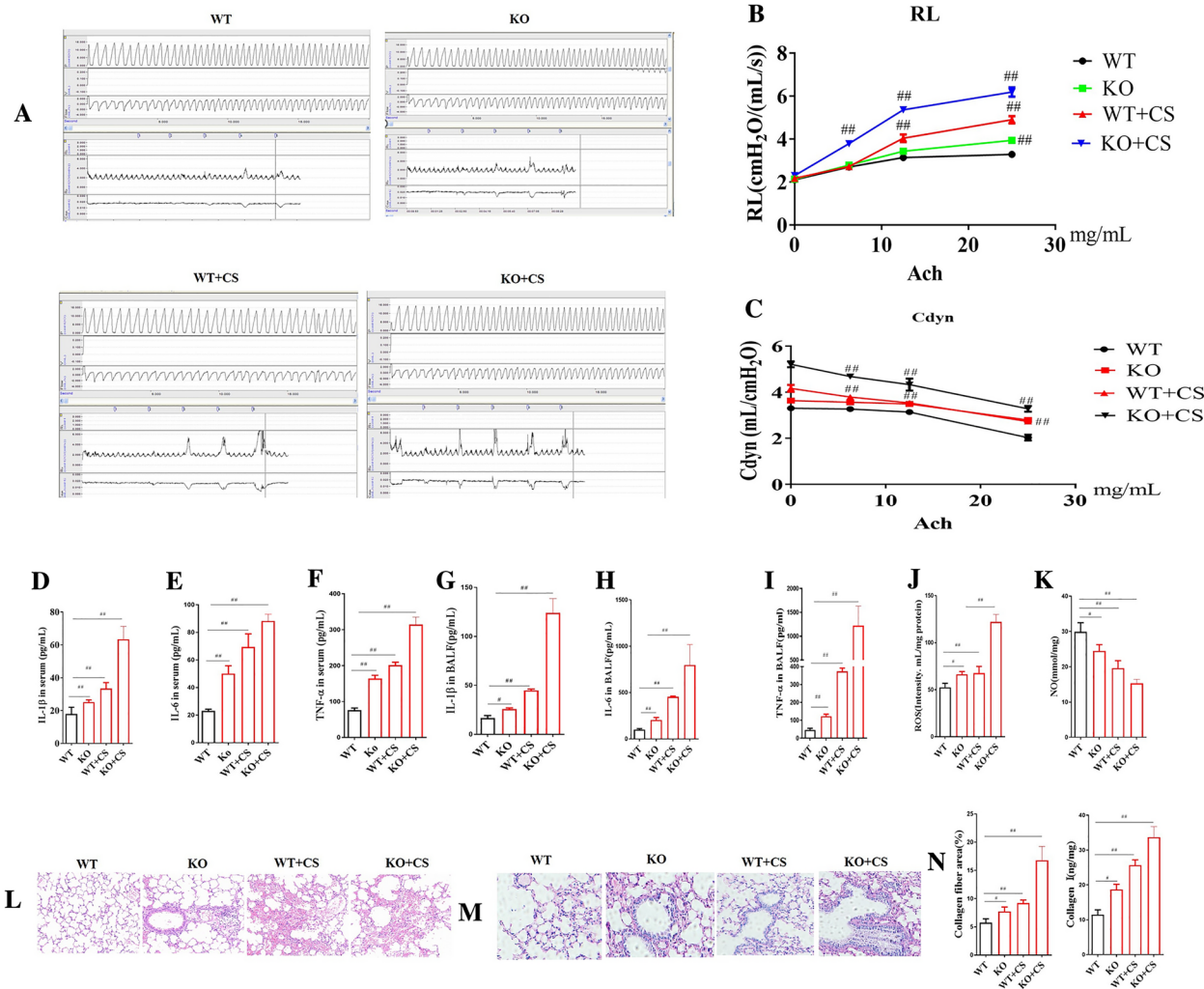
² The Affiliated Nanjing Hospital of Nanjing University of Chinese Medicine, Nanjing 210001, China

³ Institute of Hepatobiliary Surgery, First Affiliated Hospital, Army Medical University, Shapingba District, Gaotanyan Road 30, Chongqing 400038, China

⁴ School of Biology and Food Engineering, Institute of Pharmaceutical Biotechnology, Suzhou University, Anhui, China



Incorrect Fig. 3



Correct Fig. 3

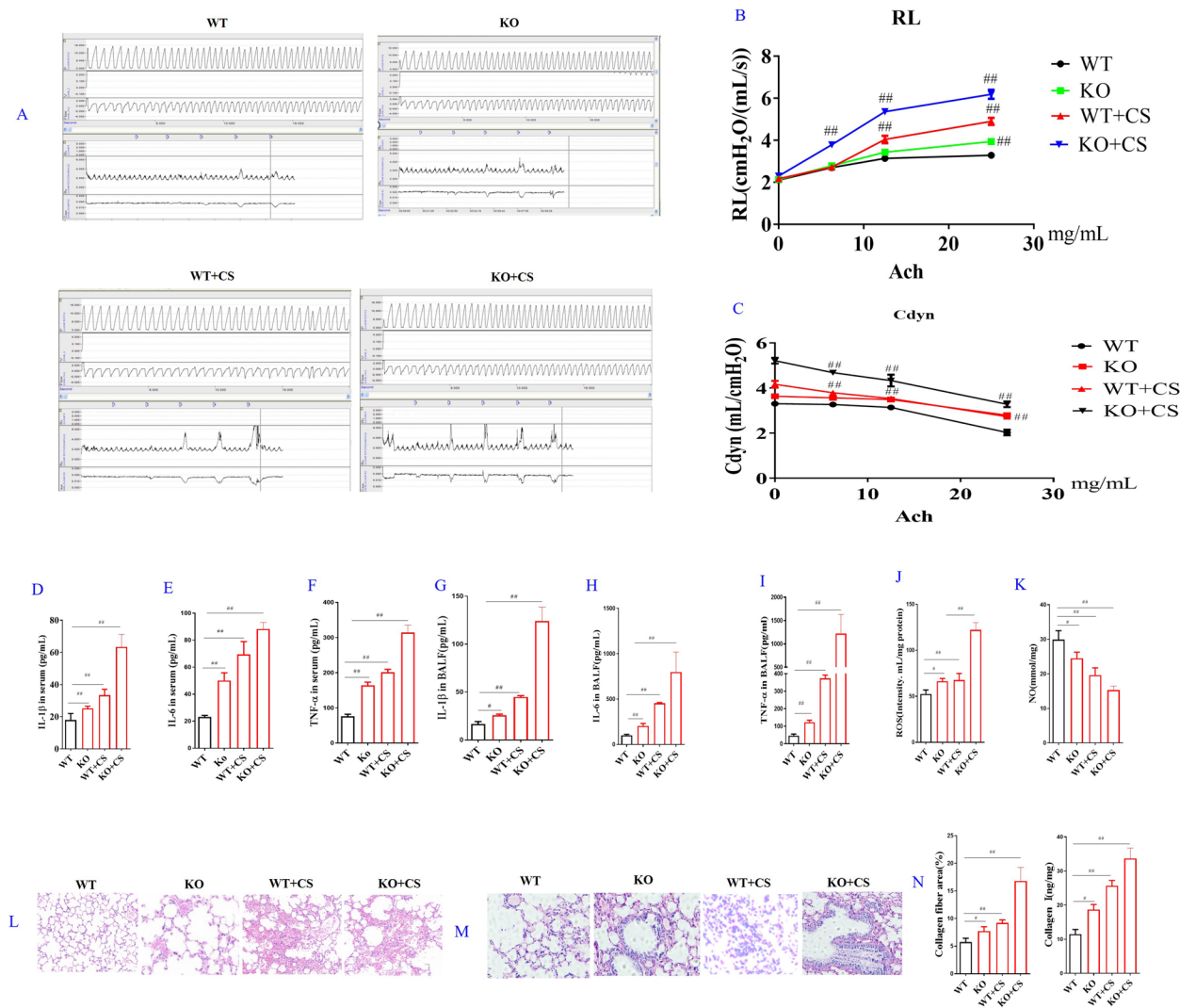
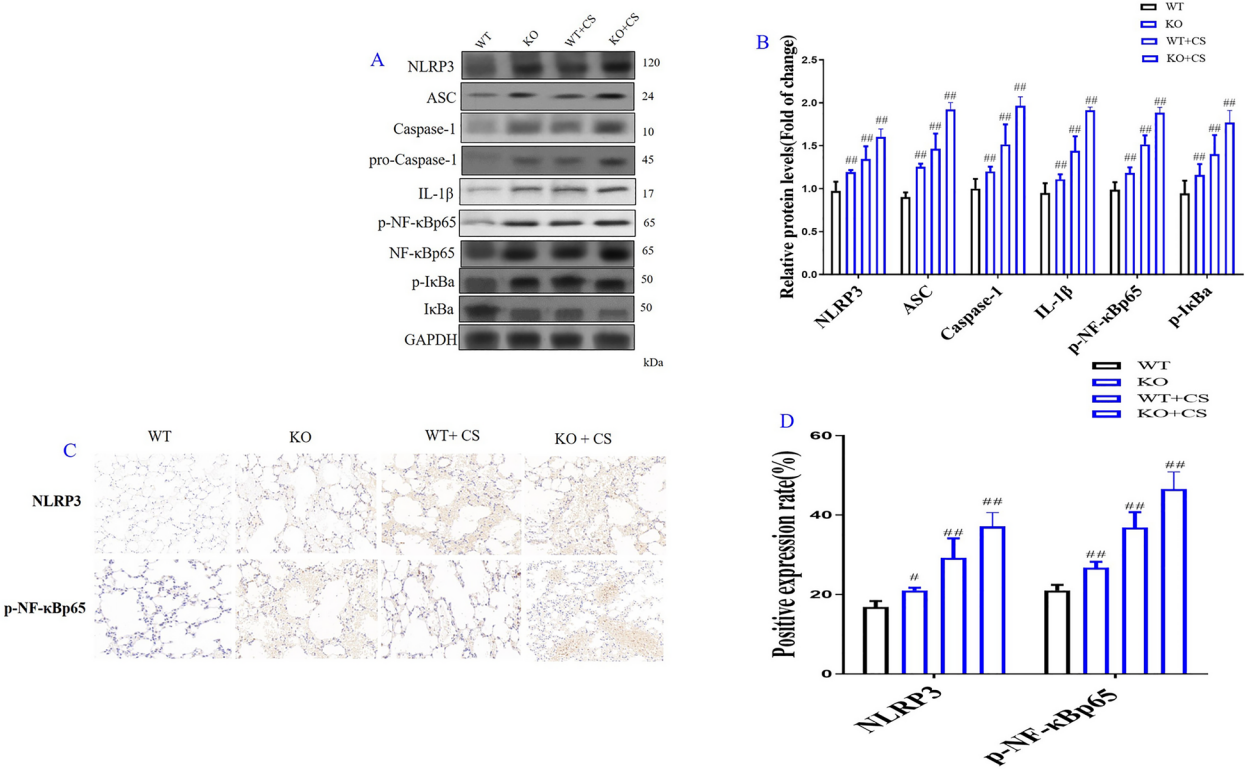


Fig. 3 Effects of l-arginine (LA) on KO COPD mice. Airway reaction (**A**). The percentage changes of the resistance of lung (RL) (**B**) and lung dynamic compliance (Cdyn) (**C**) in WT and KO COPD mice. Serum cytokines: The contents of interleukin-1β (IL-1β) (**D**), interleukin-6 (IL-6) (**E**), tumor necrosis factor-α (TNF-α) (**F**). BALF cytokines: the contents of Interleukin-1β (IL-1β) (**G**), interleukin-6 (IL-6) (**H**), tumor necrosis factor-α (TNF-α) (**I**). Reactive oxygen species (ROS) (**J**) and nitric oxide (NO) (**K**) contents in lung tissues. Pathological changes (HE staining) and Masson staining of lung in COPD rats: HE staining of lung in COPD rats (×200) (**L**), Masson staining of lung in COPD rats (×200) (**M**), Collagen quantification of Masson staining and collagen I contents of lung in COPD KO mice (**N**). (n = 10). All data were presented as mean ± SD. Compared with WT mice: ##P < 0.01

Incorrect Fig. 7



Correct Fig. 7

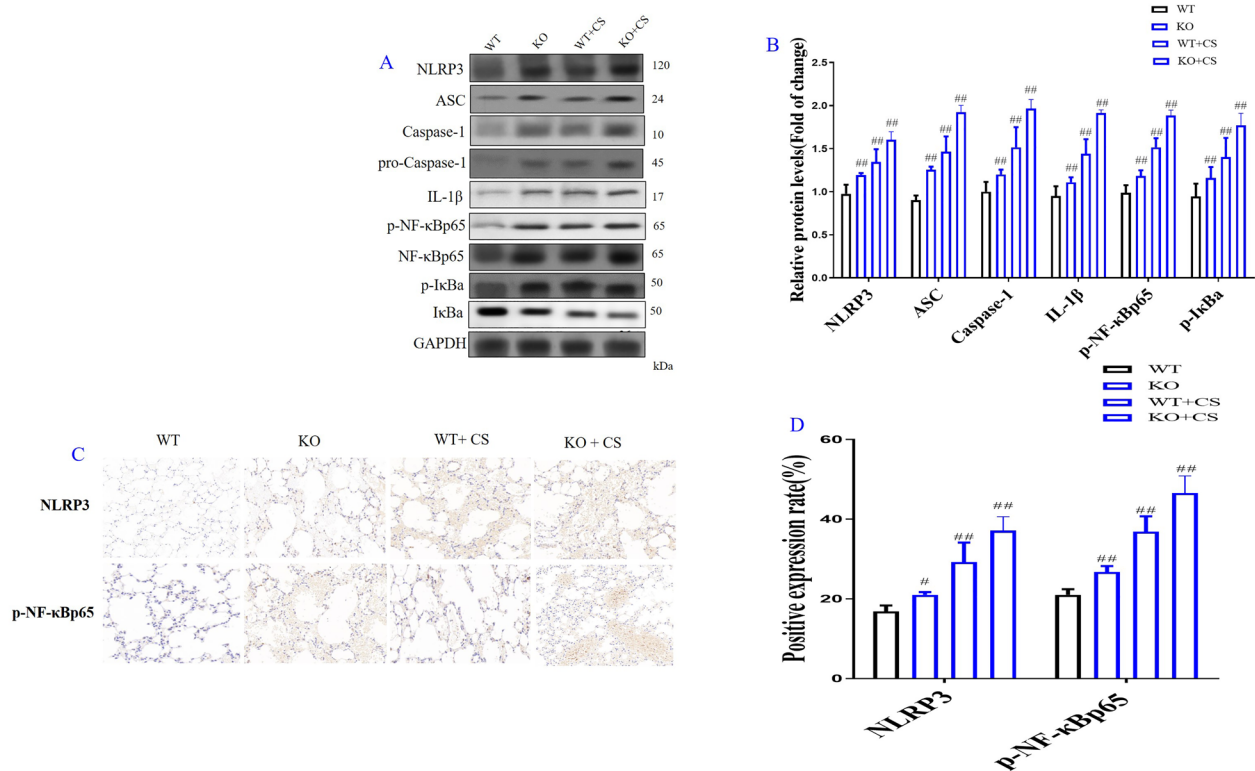


Fig. 7 Role of l-arginine (LA) mediated ROS/NLRP3/NF-κB signaling pathway in cigarette smoke extract (CSE)-induced primary bronchial epithelial cell (BEC) injury and molecular docking of LA and NLRP3. Western blot of ROS/NLRP3/NF-κB signaling pathway in CSE-induced BECs (**A**), Quantification of ROS/NLRP3/NF-κB signaling pathway in CSE-induced BECs (**B**). The expression levels of NLRP3 (**C**) and p-NF-κBp65 (**D**) in CSE-induced BECs by immunofluorescence (× 100). (n = 3). Molecular docking result of LA and NLRP3 (**E**): The binding energy predicted by Autodock is −5.79 kcal/mol for LA-NLRP3 (The binding energy predicted by Autodock < −6.00 is considered to be high degree of integration). All data were presented as mean ± SD. Compared with control: ##P < 0.01

Accepted: 24 February 2025
Published online: 17 March 2025

Reference

- Ma C, Liao K, Wang J, Li T, Liu L. L-Arginine, as an essential amino acid, is a potential substitute for treating COPD via regulation of ROS/NLRP3/NF-κB signaling pathway. *Cell Biosci.* 2023;13:152. <https://doi.org/10.1186/s13578-023-00994-9>.

Publisher's Note

Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.