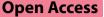
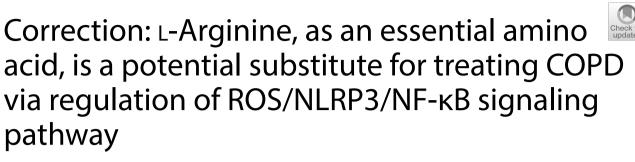
CORRECTION





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

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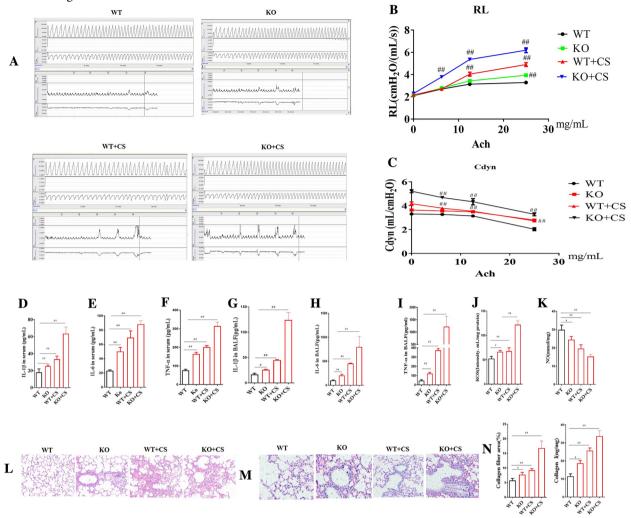
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Incorrect Fig. 3



Correct Fig. 3

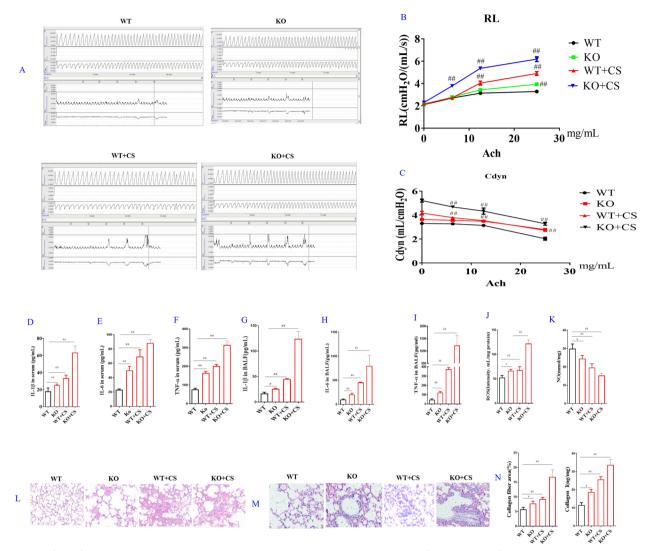
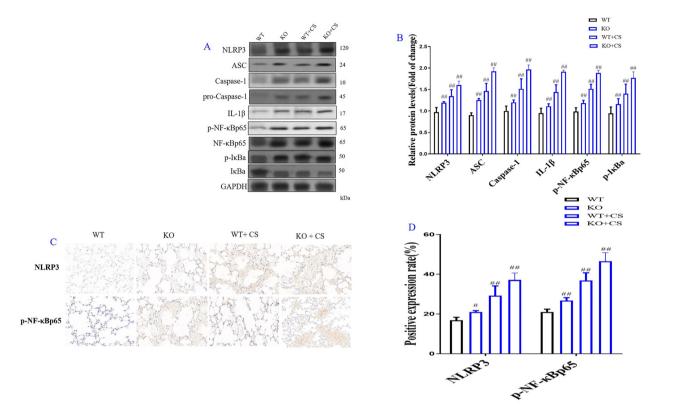


Fig. 3 Effects of I-arginine (LA) on KO COPD mice. Airway reaction (**A**). The percentage changes of the resistance of lung (RL) (**B**) and lung dynamic compliance (Cdyn) (**C**) in WT and KO COPD mice. Serum cytokines: The contents of interleukin-1 β (IL-1 β) (**D**), interleukin-6 (IL-6) (**E**), tumor necrosis factor- α (TNF- α) (**F**). BALF cytokines: the contents of Interleukin-1 β (IL-1 β) (**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



Correct Fig. 7

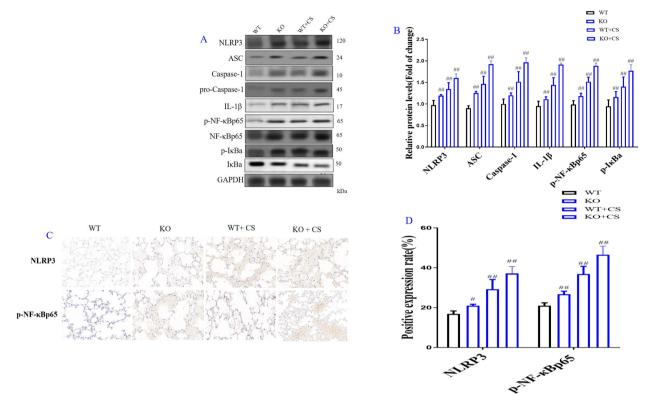


Fig. 7 Role of I-arginine (LA) mediated ROS/NLRP3/NF- κ B signaling pathway in cigarette smoke extract (CSE)-induced primary bronchial epithelial cell (BEC) injury and molecular docking of LA and NLRP3. Western blot of ROS/NLRP3/NF- κ B signaling pathway in CES-induced BECs (**A**), Quantification of ROS/NLRP3/NF- κ B signaling pathway in CES-induced BECs (**B**). The expression levels of NLRP3 (**C**) and p-NF- κ Bp65 (**D**) in CES-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

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Reference

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