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Author Correction: Angiotensin-converting enzyme 2 prevents lipopolysaccharide-induced rat acute lung injury via suppressing the ERK1/2 and NF-κB signaling pathways

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Correction to: *Scientific Reports* https://doi.org/10.1038/srep27911, published online 15 June 2016

This Article contains errors.

For Fig. 6C, the lanes shown for JNK are incorrect. The correct Fig. 6 and accompanying legend appear below.

In addition, full blots for the data presented were omitted in the original Article. They have been included in the attached Supplementary Information file.

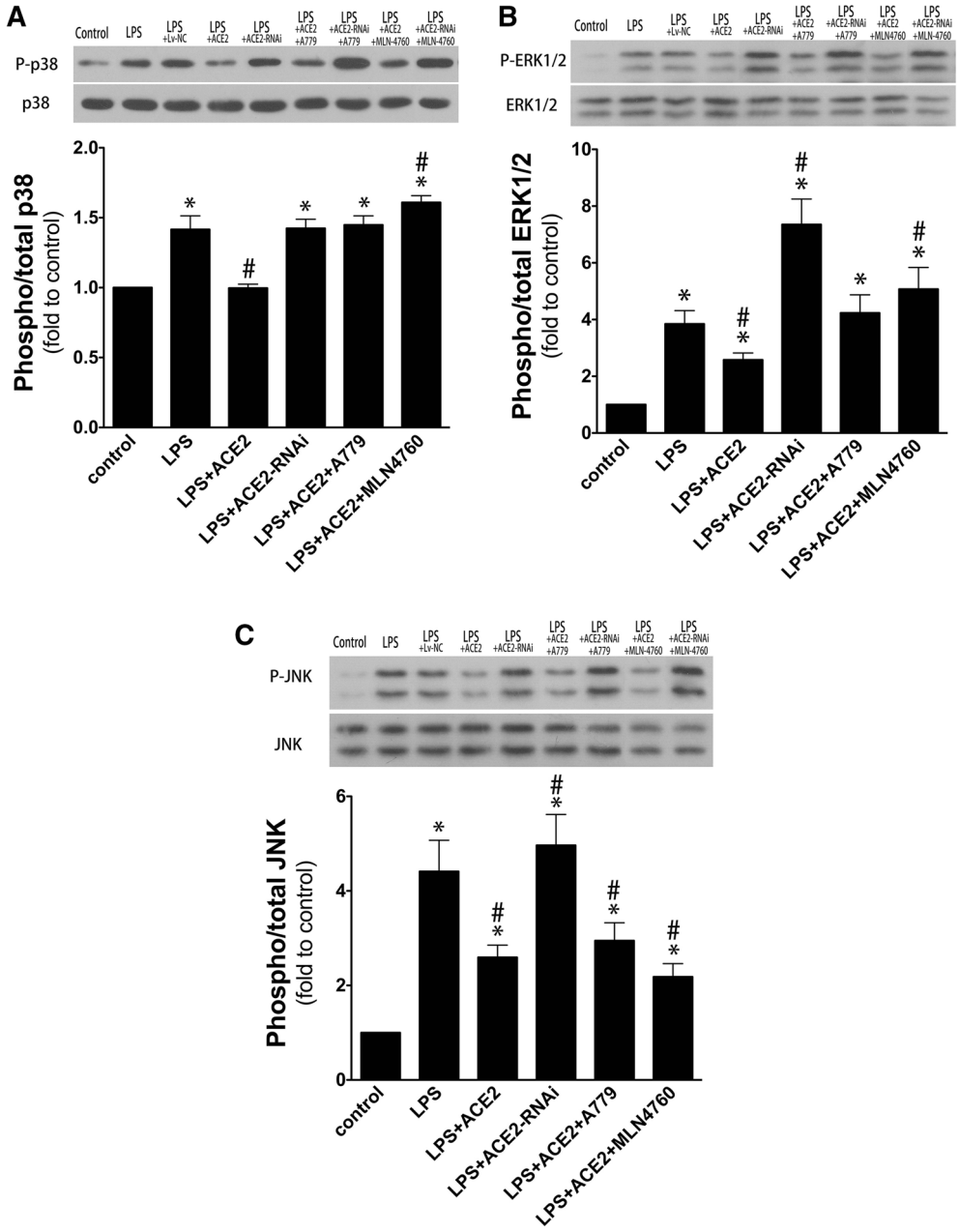

Figure 6. Effects of ACE2 and different treatments on the phosphorylation of MAPKs in lung tissue. LPS exposure caused a marked increase of the phosphorylation levels of p38 MAPK (**A**), ERK1/2 (**B**) and JNK (**C**), which was significantly suppressed by ACE2 overexpression in rat lung. Pretreatment with A779 or MLN-4760 completely abolished the inhibitory effects of ACE2 overexpression on LPS-induced p38 MAPK and ERK1/2 phosphorylation, but did not affect the level of JNK phosphorylation. ACE2 RNAi in rat lung significantly enhanced the LPS-induced ERK1/2 and JNK phosphorylation but did not change p38 MAPK phosphorylation. Data are represented as mean ± SD. $^{*}p < 0.05$, versus control group; $^{\#}p < 0.05$, versus LPS group; $^{\$}p < 0.05$, versus ACE2 group (n = 6, per group).

Additional information

Supplementary Information The online version contains supplementary material available at https://doi.org/10.1038/s41598-022-09404-5.