

Roles of peroxiredoxin II in the regulation of proinflammatory responses to LPS and protection against endotoxin-induced lethal shock
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The authors regret that an error appeared in the third paragraph under the subheading “Prx II negatively modulates NF- κ B activation and the phosphorylation of MAPK pathway kinases in response to LPS.” In the sentence, “lower” should have read “higher.” The corrected sentence appears below:

Consistent with the findings in Fig. 3 C, primary BMDMs from Prx II-deficient mice showed higher activation of MAPKs, which included JNK, ERK 1/2, and p38, than the primary BMDMs from WT mice, although the kinetics of activation during LPS treatment were similar for cells for the two types of mice (Fig. 3 D).

In addition, the rule below “+ LPS” in Fig. 4 A should have extended over lane 2. The corrected figure is shown below.

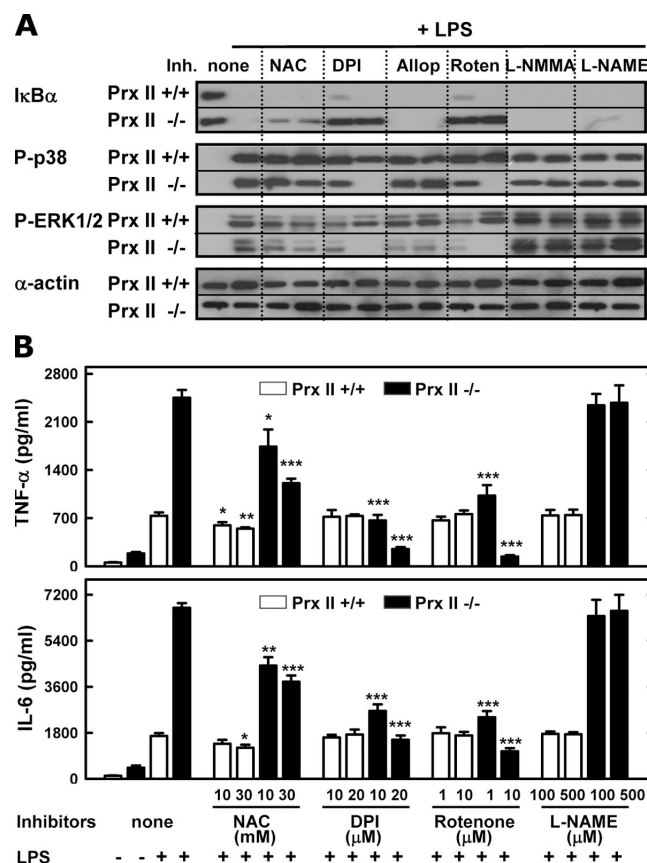


Figure 4. Endogenous ROS induced by NADPH oxidase is specifically involved in LPS signaling and proinflammatory responses in Prx II-deficient cells. (A) After preincubation for 30 min with 10 and 30 mM NAC, 10 and 20 μ M DPI, 10 and 100 μ M allopurinol, 1 and 10 μ M rotenone, 100 and 500 μ M L-NMMA, or 100 and 500 μ M L-NAME, BMDMs were stimulated with 1 μ g/ml LPS for 30 min. The cells were harvested and subjected to Western blot analysis for I κ B α , phosphorylated ERK 1/2, and p38 MAPK. The same blots were washed and blotted for β -actin as the loading control. Data shown are representative of three independent experiments that gave similar results. (B) The experimental conditions followed the pattern outlined in A. Culture supernatants were harvested after stimulation with LPS for 18 h, and the TNF- α and IL-6 expression levels were measured by ELISA. Data shown are the mean \pm SD of three experiments. *, $P < 0.05$; **, $P < 0.01$; and ***, $P < 0.001$ compared with WT cell cultures stimulated with LPS. MC, media control.