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CXCL1 Contributes to Host Defense in Polymicrobial Sepsis via Modulating T Cell and Neutrophil Functions

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In the original publication, Figure 3 had an inadvertently duplicated, misplaced GAPDH blot. The spleen 6hr GAPDH blot in Figure 3A was used incorrectly as the lung 24hr GAPDH blot in Figure 3D. Since this paper was published more than six years ago, the authors no longer had access to the original blots or samples. Therefore, the authors repeated the experiment at least twice and ran all samples and controls together on one blot. The figure legend was correct as published and is shown below.

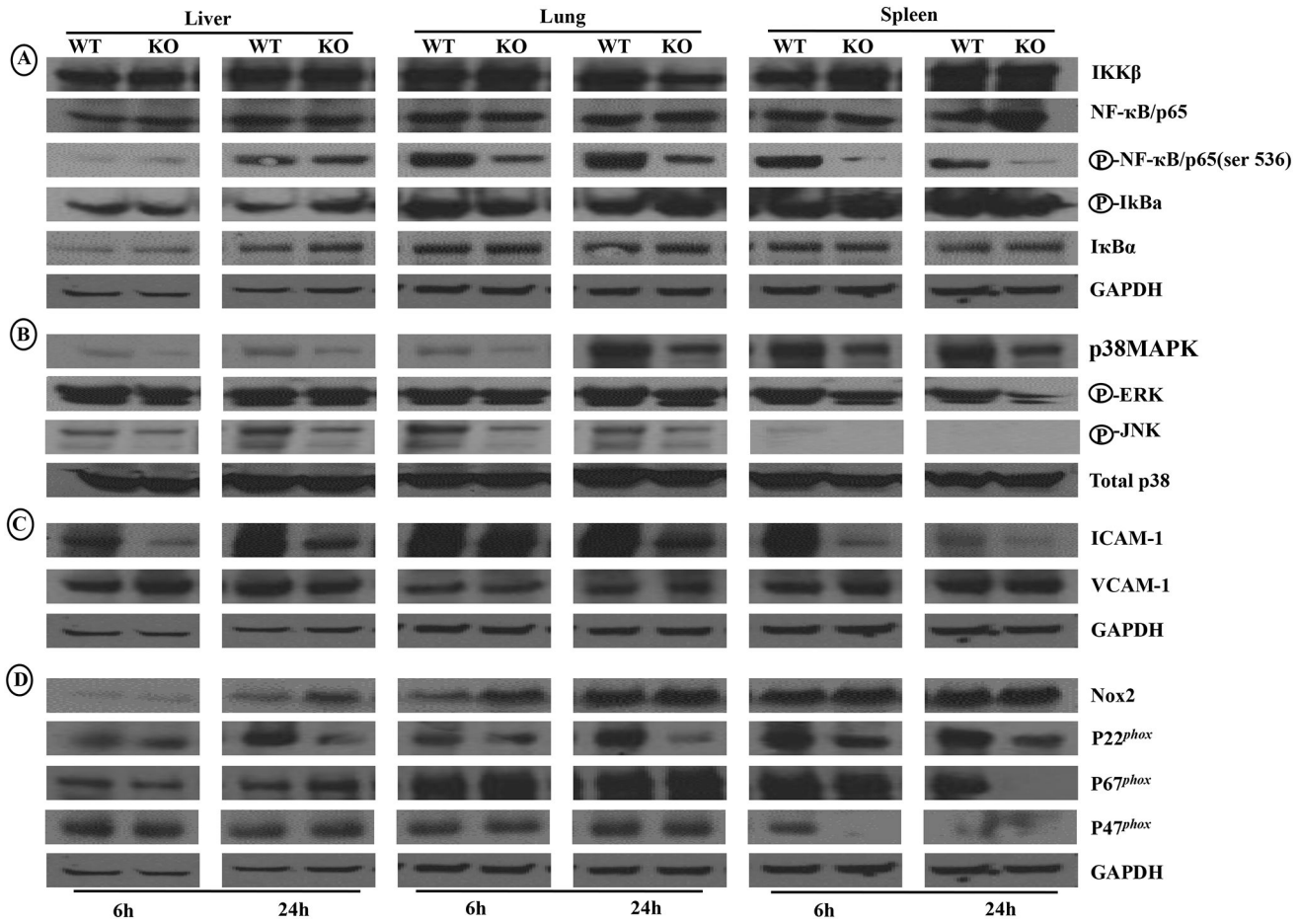

Figure 3. Activation of NF-κB, MAPK, and NADPH oxidase, and expression of adhesion molecule ICAM-1 were impaired in *cxcl1*$^{-/-}$ mice following PMS

A, CXCL1 is essential for the activation of NF-κB during PMS induction. Phosphorylation of NF-κB/p65(ser 536), and IκBα in the homogenates from liver, lung and spleen kidney from *cxcl1*$^{-/-}$ as compared to WT mice. **B**, Attenuated activation of MAPKs in the organs of *cxcl1*$^{-/-}$ mice. Phosphorylated p38-MAPK, ERK, and JNK in the homogenates from different organs from *cxcl1*$^{-/-}$ and WT mice were probed with their respective antibodies. **C**, Expression of adhesion molecule ICAM-1 but not VCAM-1 is reduced in *cxcl1*$^{-/-}$ mice. The expression level of ICAM-1 and VCAM-1 were determined in the organs of *cxcl1*$^{-/-}$ and WT control mice following PMS for time points 6 and 24 h. **D**, The activation of NADPH oxidase is impaired in *cxcl1*$^{-/-}$ mice. The expression levels of Nox2, p22^{phox}, p67^{phox}, and p47^{phox} were determined in the homogenates of organs of *cxcl1*$^{-/-}$ and WT control mice by immunoblotting at 6 and 24 h post-PMS. GAPDH or total p38 expression levels were assessed in all samples as internal loading control and the blots are representative of 2 independent experiments with similar results (A, B, C, and D). (n=4–6/group).