

Supporting Information

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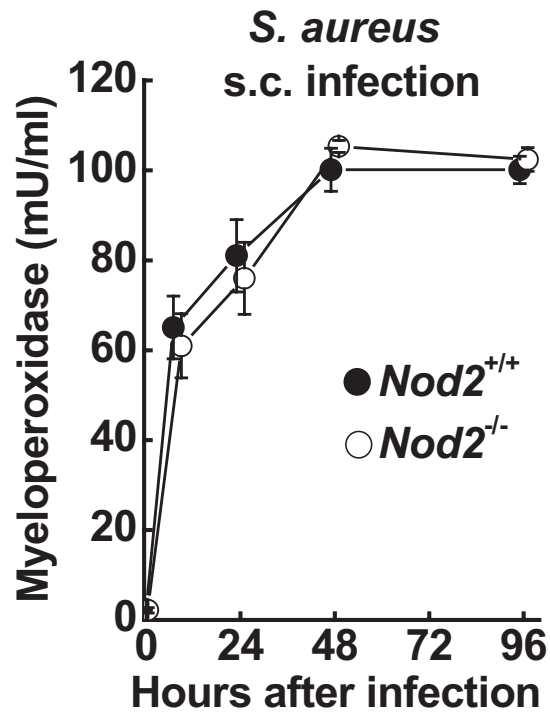


Fig. S1. Cutaneous neutrophil recruitment in *S. aureus*-infected *Nod2*^{-/-} and WT mice. *Nod2*^{-/-} mice (open circles) and WT mice (filled circles) were infected s.c. with 5×10^7 cfu of WT *S. aureus*. At the indicated times, myeloperoxidase activity was determined in skin homogenates. Data are mean (SE); $n \geq 4$ /group.

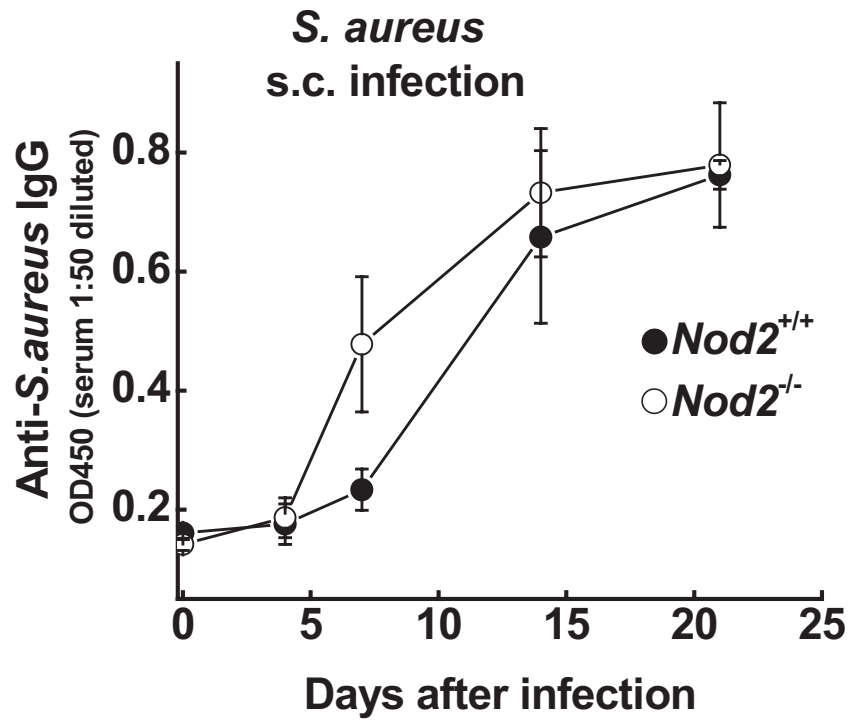
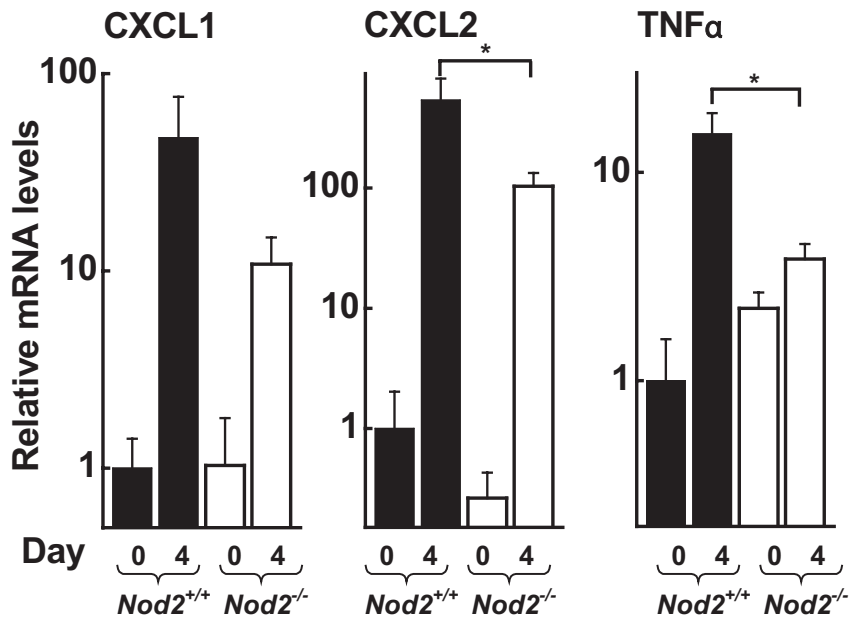


Fig. S2. NOD2 independence of IgG response to *S. aureus*. *Nod2*^{-/-} (open circles) and WT (filled circles) mice were infected s.c. with 5×10^7 cfu of WT *S. aureus*, and specific IgG titers against the bacteria were determined at the indicated times. Data are mean (SD); $n = 4$ /group.

A



B

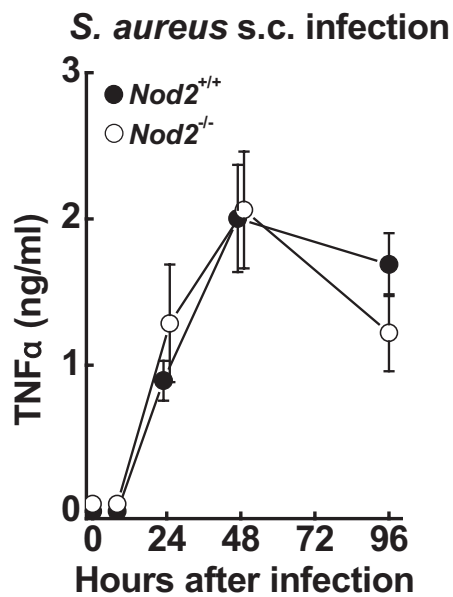


Fig. S3. Cytokine response in skin homogenates. (A) *Nod2*^{-/-} mice (open bars) and WT mice (closed bars) were infected s.c. with 5×10^7 cfu of WT *S. aureus*. Cytokine mRNA levels (normalized to GAPDH) in the infected skin were determined by real-time PCR and are expressed relative to the levels in uninfected WT mice ($n = 5$ mice/group). (B) TNFα levels were assayed by ELISA in skin homogenates of *Nod2*^{-/-} mice (open circles) and WT mice (filled circles). Data are mean (SE); $n \geq 4$ /group.

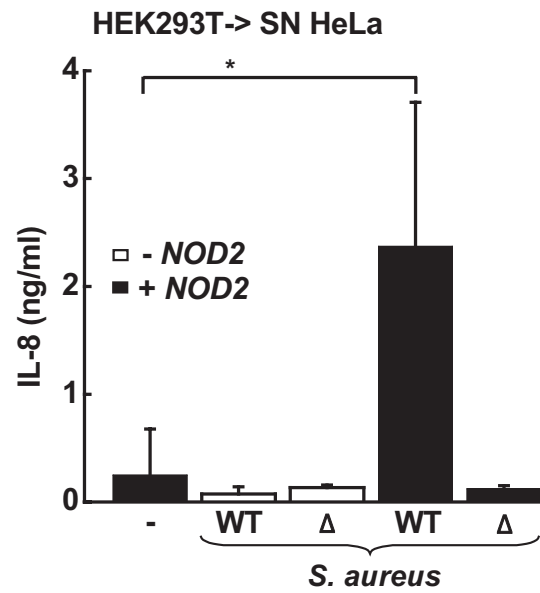


Fig. S4. *S. aureus*-induced release of bioactive IL-1. IL-1-responsive HeLa cells were incubated for 8 h with supernatants from NOD2-transfected HEK293T cells, which were incubated for 8 h with WT or α -hemolysin-deficient (Δ) *S. aureus* at a MOI of 5, as described in Fig. 5A. IL-1-dependent IL-8 secretion into the supernatants was determined by ELISA. Data are mean (SD); *, $P < 0.05$.