

CORRECTION

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Correction to: CCL3 contributes to secondary damage after spinal cord injury

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Following publication of the original article [1], the authors noticed that there was a covered up area which seems to be hiding something found in Fig. 7a. Presented here is the corrected Fig. 7. The original article has been corrected.

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1. Pelisch N, Rosas Almanza J, Stehlik KE, et al. CCL3 contributes to secondary damage after spinal cord injury. *J Neuroinflammation*. 2020;17:362 <https://doi.org/10.1186/s12974-020-02037-3>.

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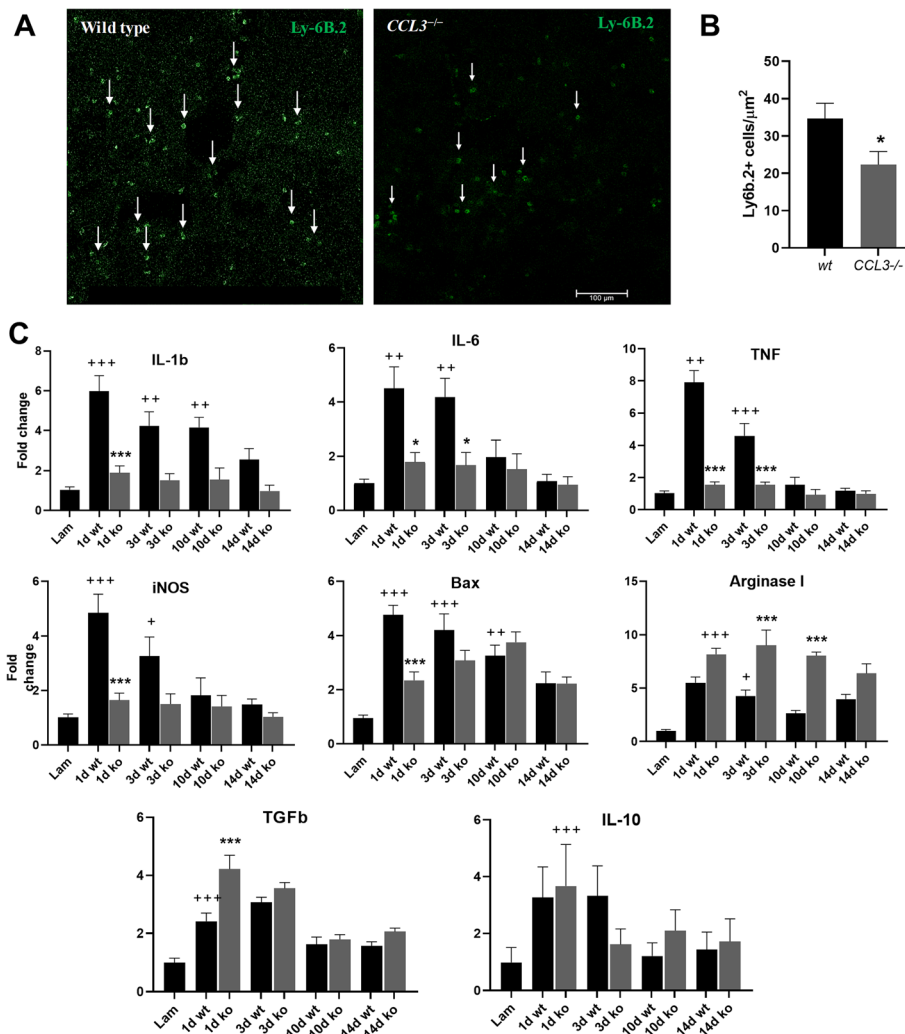


Fig. 7 Inflammatory response in *CCL3*^{-/-} mice is reduced after SCI. **a** Representative images of Ly-6B.2-positive neutrophils in wild-type and *CCL3*^{-/-} SCI tissue at the lesion epicenter at day 1 after injury. Scale bar = 100 μm. Arrows indicate Ly-6B.2-positive profiles. **b** Quantitative analysis of neutrophil recruitment shows a significant reduction of neutrophils at the lesion epicenter of *CCL3*^{-/-} mice. *n* = 3/group. * = *p* value < 0.05. **c** Expression levels of the pro-inflammatory cytokines *il-1b*, *il-6*, *tnf*, *inos*, and the apoptotic marker *bax* were significantly increased at different time points in wild-type mice compared to laminectomy controls. *CCL3*^{-/-} mice, however, showed significantly lower expression levels compared to wild-type. *Arginase-1* and *TGFb*, which can indicate anti-inflammatory properties, were significantly upregulated in *CCL3*^{-/-} mice in relation to wild-type mice. *IL-10* was upregulated compared to the laminectomy control but did not differ between genotypes. *n* = 6 wild-type, 5 *CCL3*^{-/-} mice. + = *p* value < 0.05, ++ = *p* value < 0.01, +++ = *p* value < 0.001 (wild-type compared to laminectomy control), * < *p* value 0.05, *** < *p* value 0.0001 (*CCL3*^{-/-} compared to wild-type, same day)