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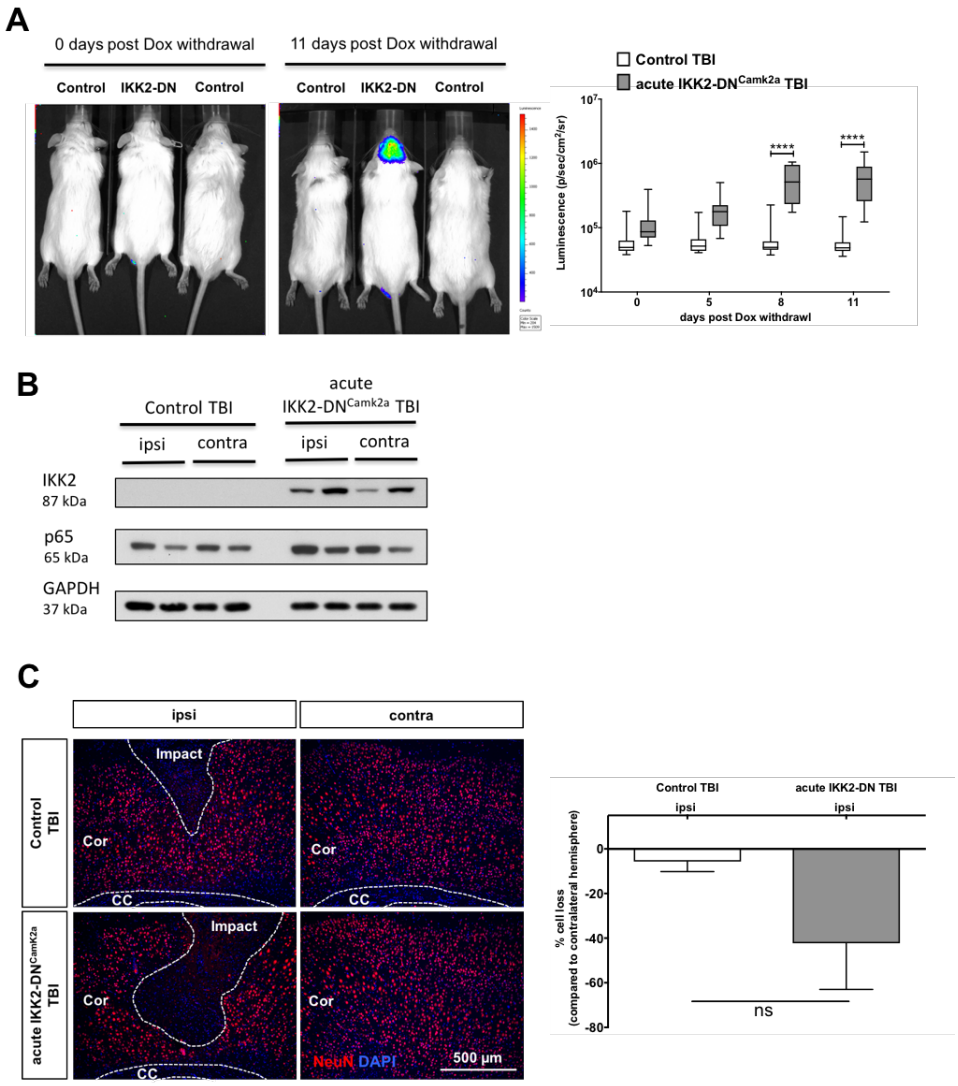
Supplementary Figure 8



Characterization of the acute IKK2-DN^{Camk2a} animal model.
(A) *In vivo* luciferase measurement of acute IKK2-DN^{Camk2a} mice indicates forebrain-restricted IKK2-DN transgene expression from 8 days after Dox removal. Diagram shows luciferase activity (p/s/cm2/sr, mean ± SEM), statistical analysis: 2-way-ANOVA followed by Bonferroni's post test (n=10-14) ****$p < 0.0001$.
(B) IKK2-DN transgene expression was monitored by immunoblot in the injured (ipsi) and uninjured (contra) hemisphere of control and acute IKK2-DN^{Camk2a} mice 6h after head trauma. p65 levels were unchanged between the two animal groups. GAPDH is used as loading control (n=2).
(C) Neuronal cell loss in the cortex of acute IKK2-DN^{Camk2a} mice 3d after CHI. Immunofluorescent staining and quantification of NeuN^{+} cells reveal a tendency for enhanced neuronal cell loss in the ipsilateral cortex of acute IKK2-DN^{Camk2a} mice compared to control animals. Percentage of neuronal cell loss was calculated as the ratio of neuronal cell from the injured (ipsilateral) hemisphere compared to the number of neurons in the uninjured (contralateral) hemisphere. Scale bar 500 µm. Impact: TBI impact area, Cor: Cortex, CC: Corpus Callosum. ns: not significant ($p>0.05$) according to 1-way ANOVA with Bonferroni correction (n=3-4).