Homozygous *Smpd1* deficiency aggravates brain ischemia/ reperfusion injury by mechanisms involving polymorphonuclear neutrophils, whereas heterozygous *Smpd1* deficiency protects against mild focal cerebral ischemia
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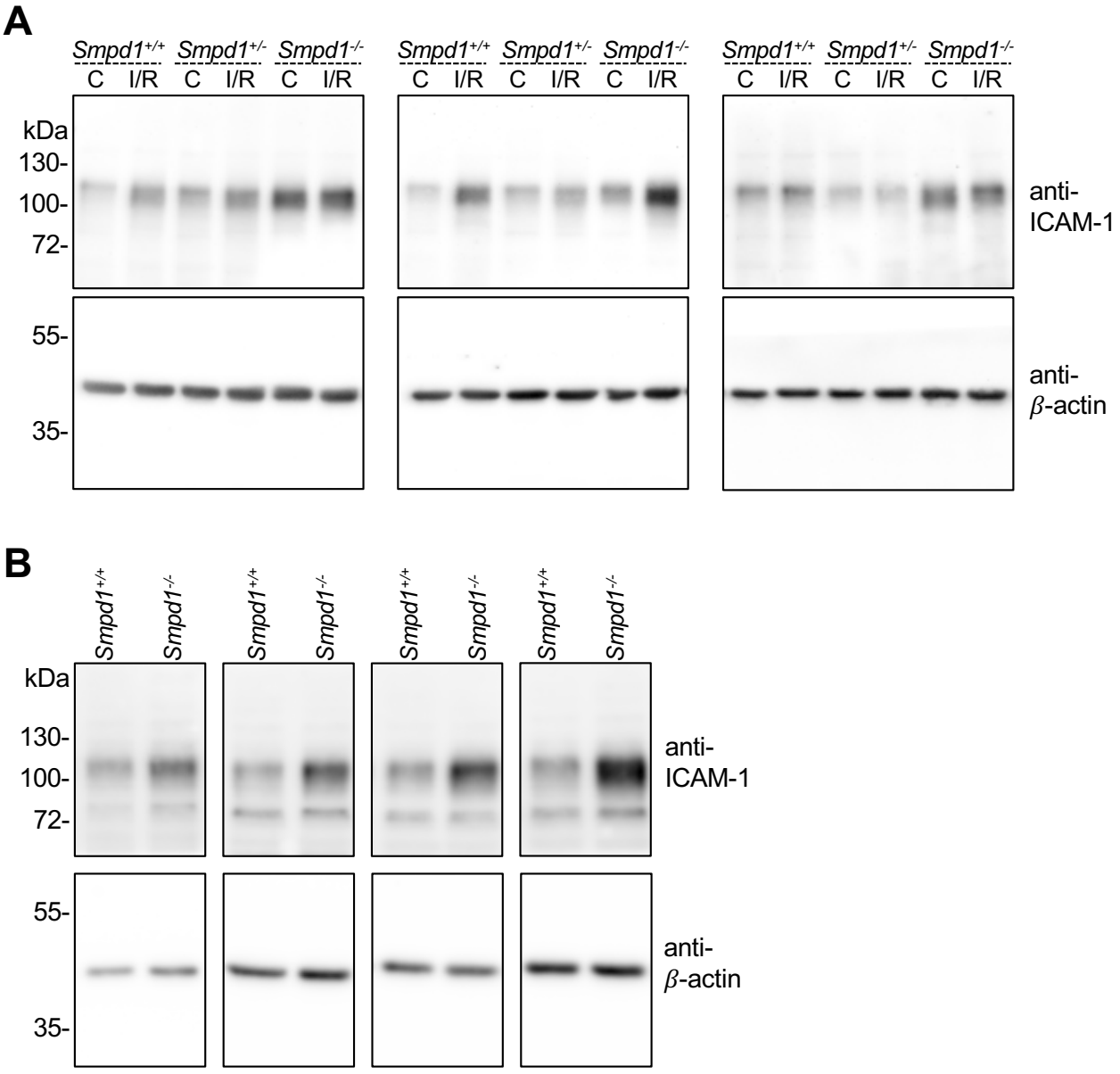
Supplementary Figure 5



Supplementary Figure 5. Homozygous, but not heterozygous *Smpd1* deficiency increases ICAM-1 abundance on cerebral microvessels. Complete Western blot membranes exhibiting ICAM-1 protein abundance **(A)** in the contralateral non-ischemic striatum (C) and the reperfused ischemic striatum (I/R) of 8-week-old male *Smpd1*$^{+/+}$, *Smpd1*$^{+/-}$ or *Smpd1*$^{-/-}$ mice, which were exposed to 30 minutes of MCAO followed by animal sacrifice 24 hours after reperfusion and **(B)** in the striatum of 8-week-old male naïve *Smpd1*$^{+/+}$, *Smpd1*$^{+/-}$ or *Smpd1*$^{-/-}$ mice which had not been exposed to experimental interventions or anesthesia. β-actin blots, which were used as loading controls, are also shown. For the semiquantitative analysis of Western blots see **Figure 5** of the main manuscript.