

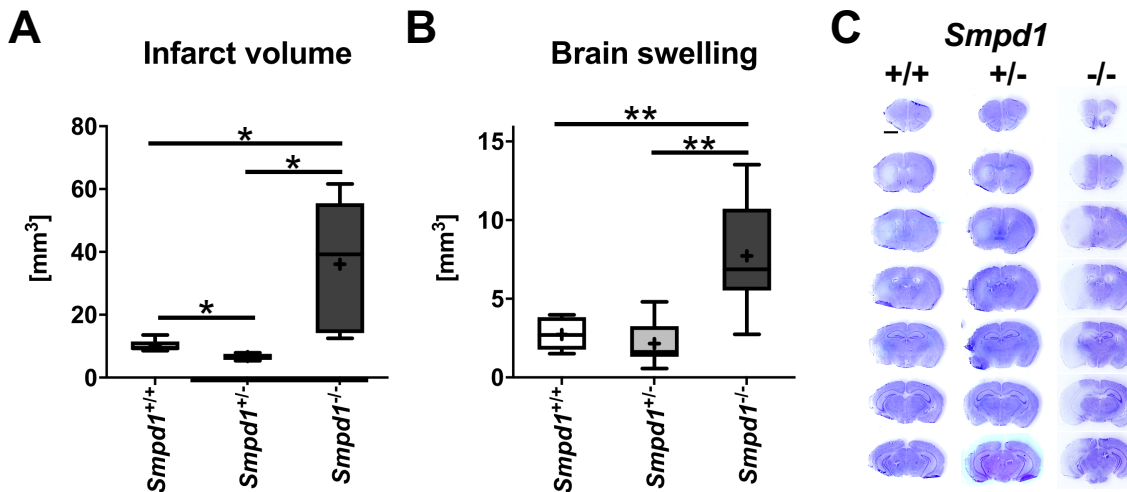
Homozygous *Smpd1* deficiency aggravates brain ischemia/ reperfusion injury by mechanisms involving polymorphonuclear neutrophils, whereas heterozygous *Smpd1* deficiency protects against mild focal cerebral ischemia

Basic research in Cardiology

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Supplementary Figure 4



Supplementary Figure 4. Homozygous *Smpd1* deficiency exacerbates I/R injury independent of animal age, whereas heterozygous *Smpd1* deficiency protects against focal cerebral ischemia. (A) Infarct volume and (B) brain swelling evaluated on cresyl violet-stained brain sections of 12-week-old male *Smpd1*^{+/+}, *Smpd1*^{+/-} or *Smpd1*^{-/-} mice, which were exposed to 30 minutes of MCAO followed by animal sacrifice 24 hours after reperfusion. Representative cresyl violet-stained brain sections are shown in (C). This data set confirms the observations of **Figure 3** in an independent cohort of mice. Data were analyzed by oneway ANOVA, followed by Bonferroni-corrected posthoc t-tests. Data are box plots with medians (line)/ means (plus) ± IQRs with minimum and maximum data as whiskers. *p<0.05, **p<0.01 (n=6-10 animals per group). Scale bar, 2 mm.