

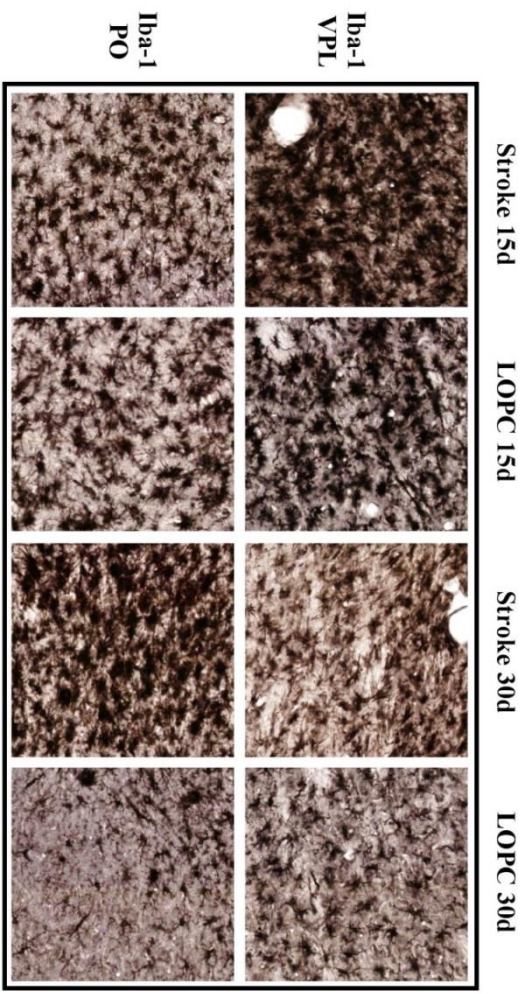
Low Oxygen Post Conditioning prevents thalamic secondary neuronal loss caused by excitotoxicity after cortical stroke

Authors:

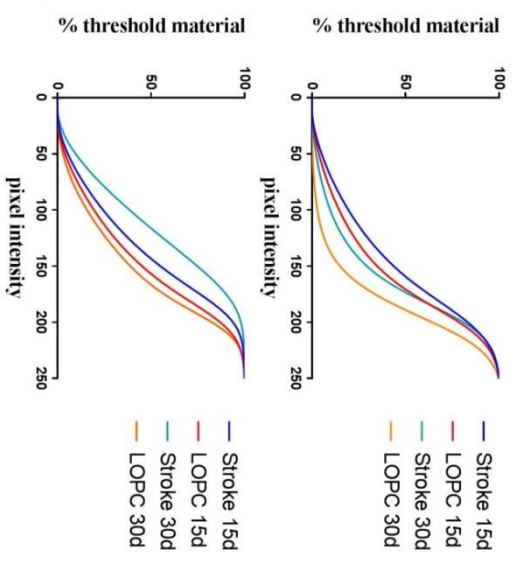
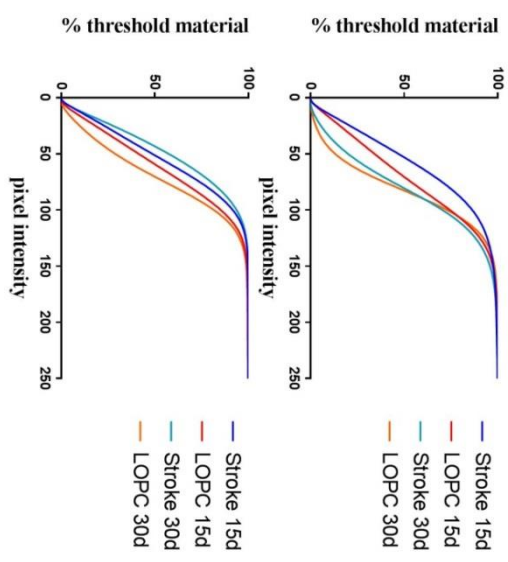
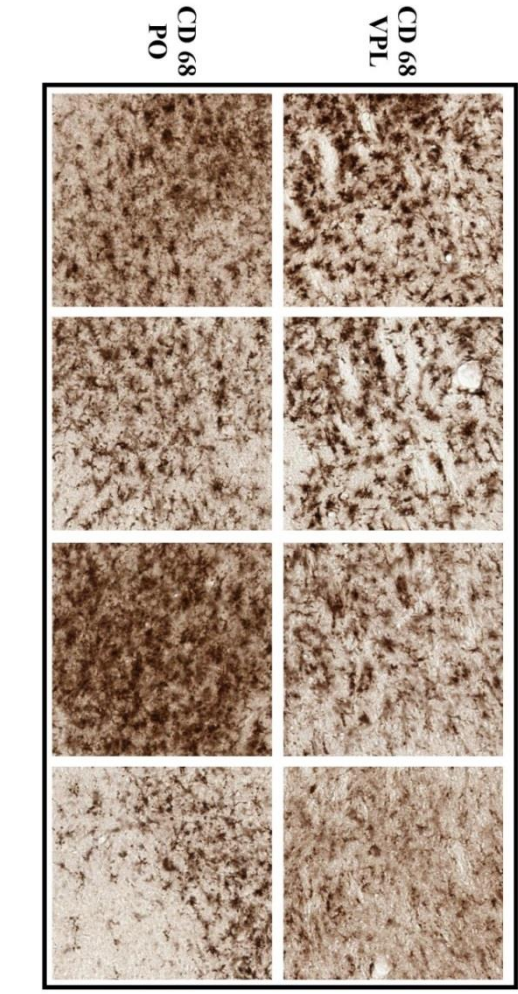
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Supplementary Information

A

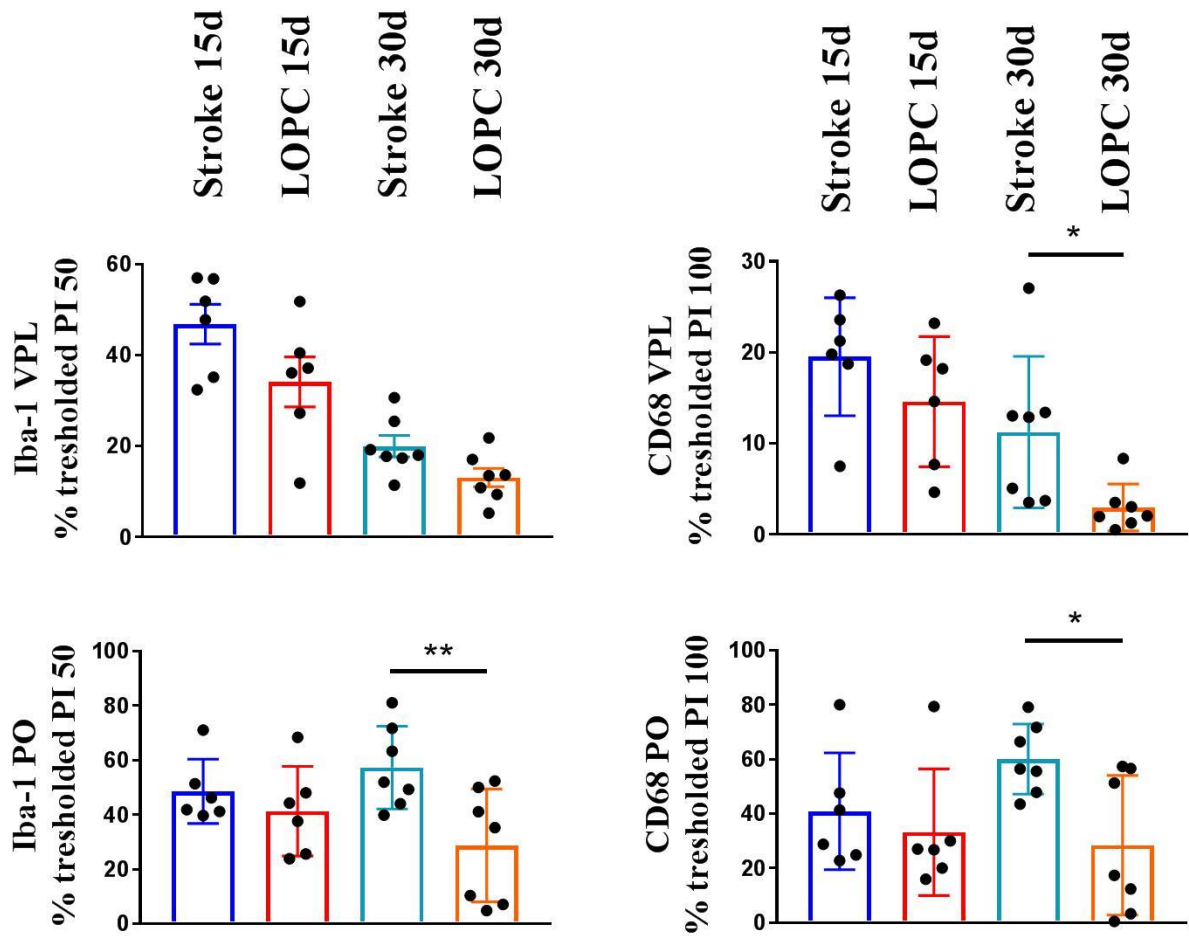


B



Supp Fig 1. Quantification of immunolabelling for Iba1 and CD68 in the PO and VPL.

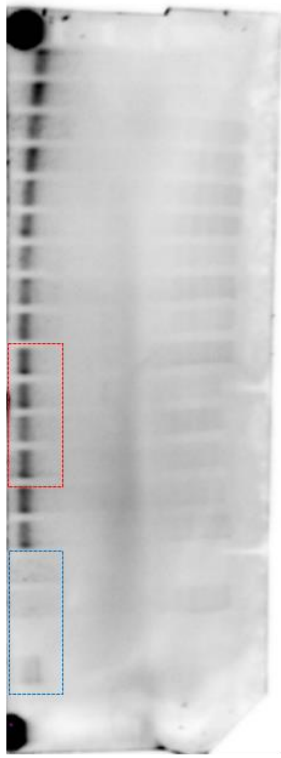
Cumulative thresholding analysis of the intensity of immunolabelling confirms general decrease in the levels of microglia activation at 30 days after LOPC compared to stroke alone.



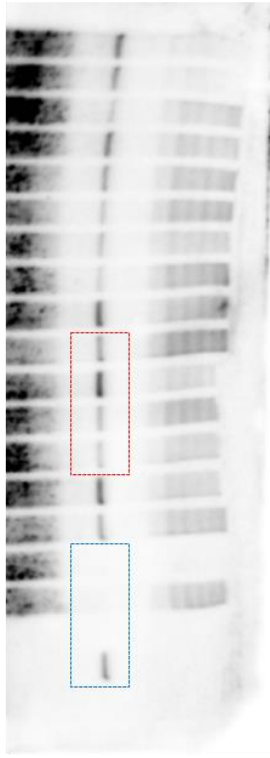
Supp Fig 2. Specific quantification of cumulative thresholding. Analysis of the thresholded material of Iba-1 and CD68 staining in the VPL and PO. The optimal pixel intensity (PI) was assessed manually.



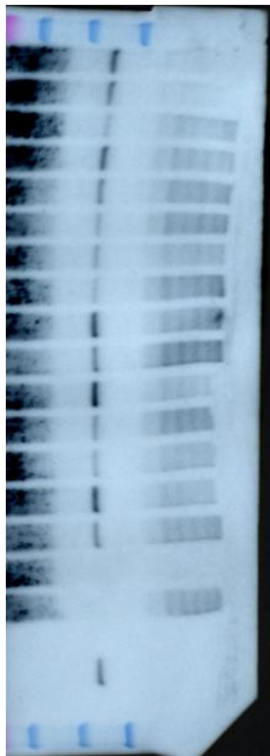
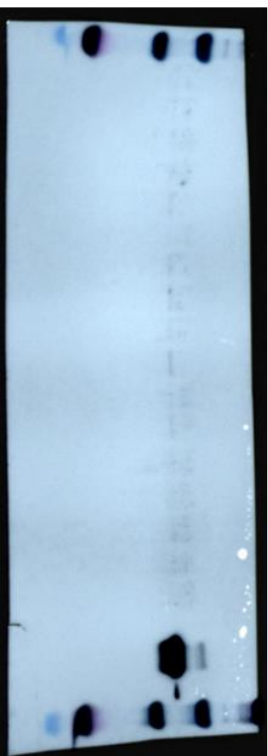
B-ACTIN



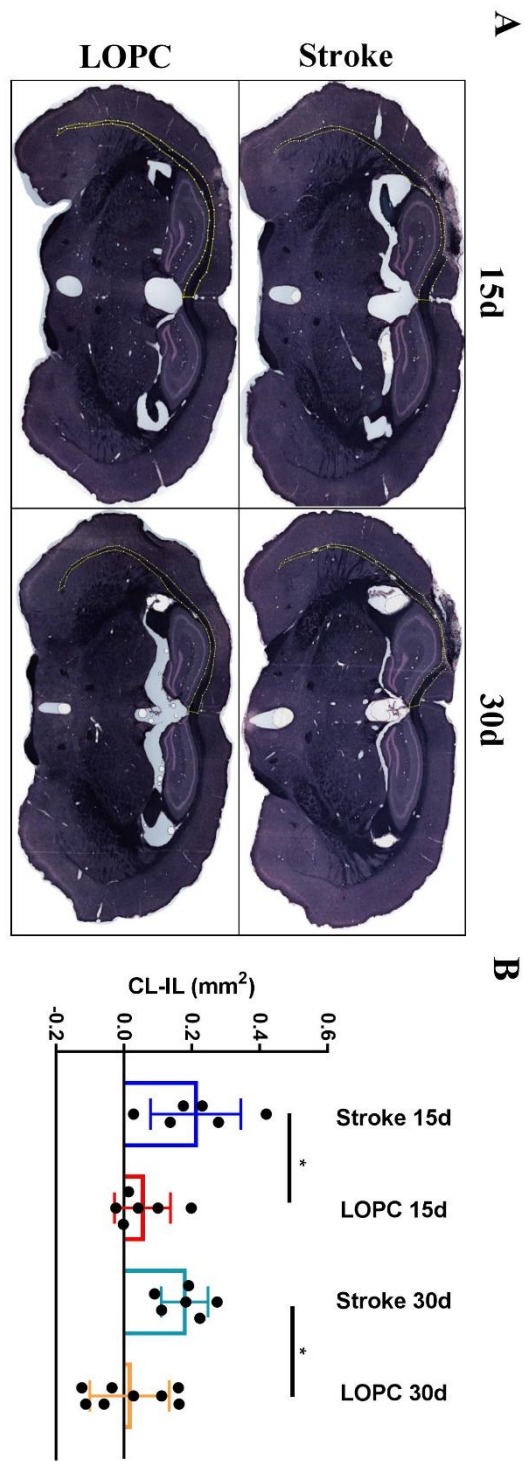
PSD-95



N2B



Supp Fig 3. CO-IP original western blot. Picture of the original western blot cropped for Fig. 5c and overlay with white light for molecular weight marker (Bio Rad, Cat# 161-0374) visualization. Loading order: Stroke1, Stroke2, LOPC1, LOPC2, Stroke3, Stroke4, LOPC 3, LOPC4, Stroke5, Stroke6, Stroke7, LOPC5, LOPC6, LOPC7, LOPC8, -Ab, -Lys, empty lane, 1 μ g Stroke thalamus lysate. Red box highlights the left part of panel Fig 5c, blue box the right panel of Fig 5c.



Supp Fig.4 LOPC ameliorates white matter loss. (A) Sudan Black B staining at Bregma levels -1.5. Yellow line represents the corpus callosum traced using ImageJ. (B) Quantification of corpus callosum area measured as a difference between the contralateral (CL) and ipsilateral (IL) hemisphere revealed a significant decrease in white matter structural loss at both 15 and 30 days post-stroke. Results are shown as the mean \pm SD. * $p < 0.05$, Mann Whitney U test.