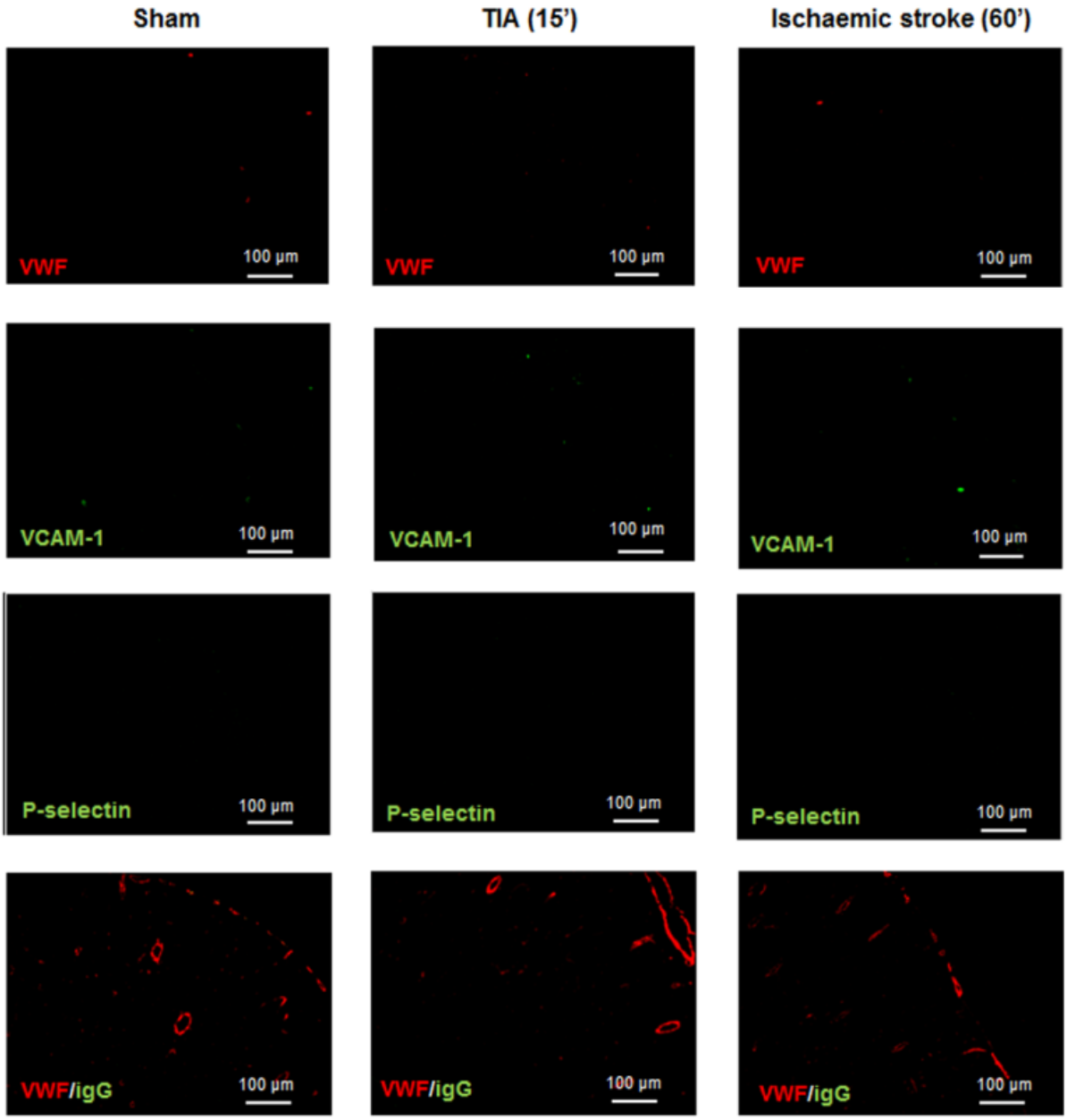
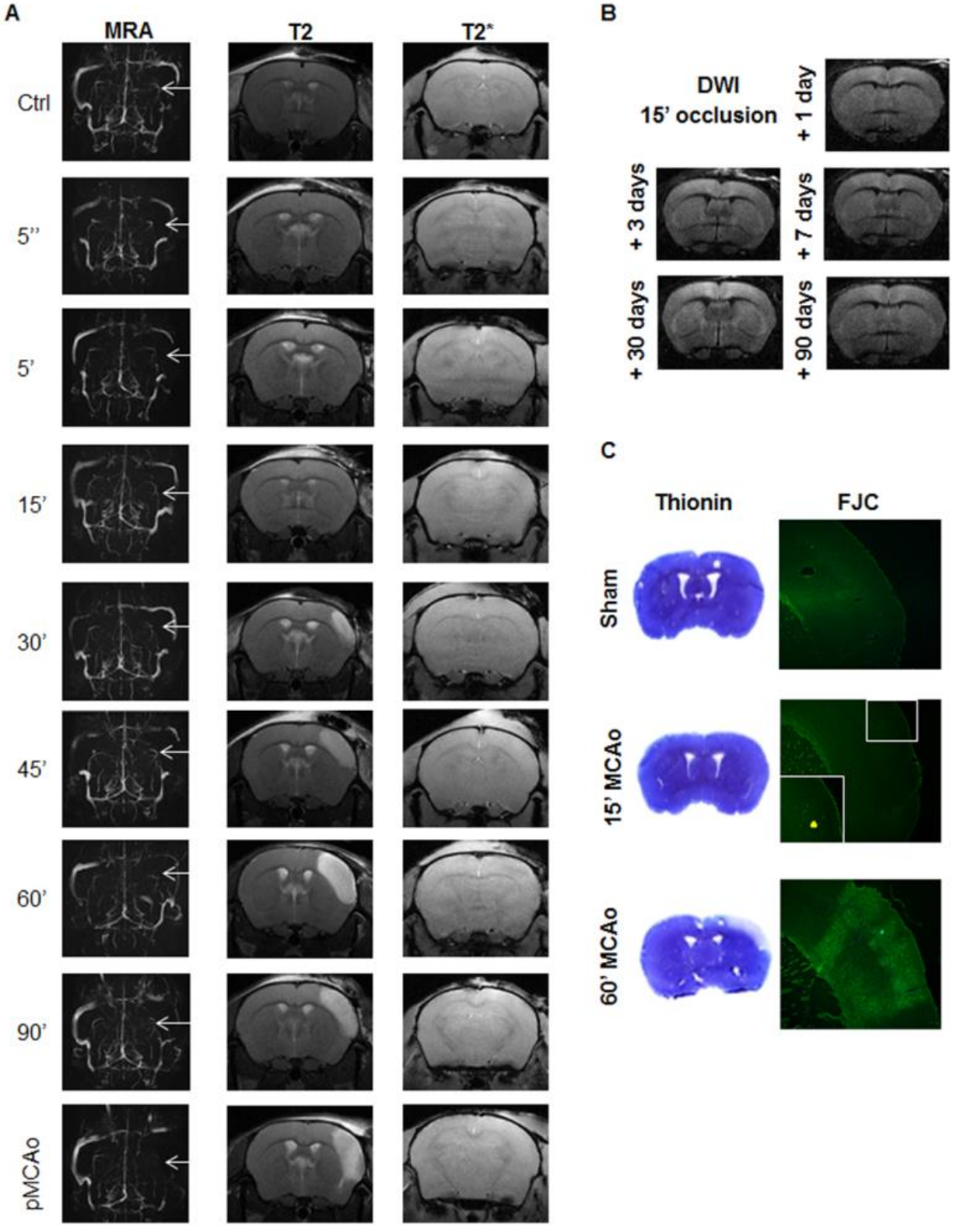


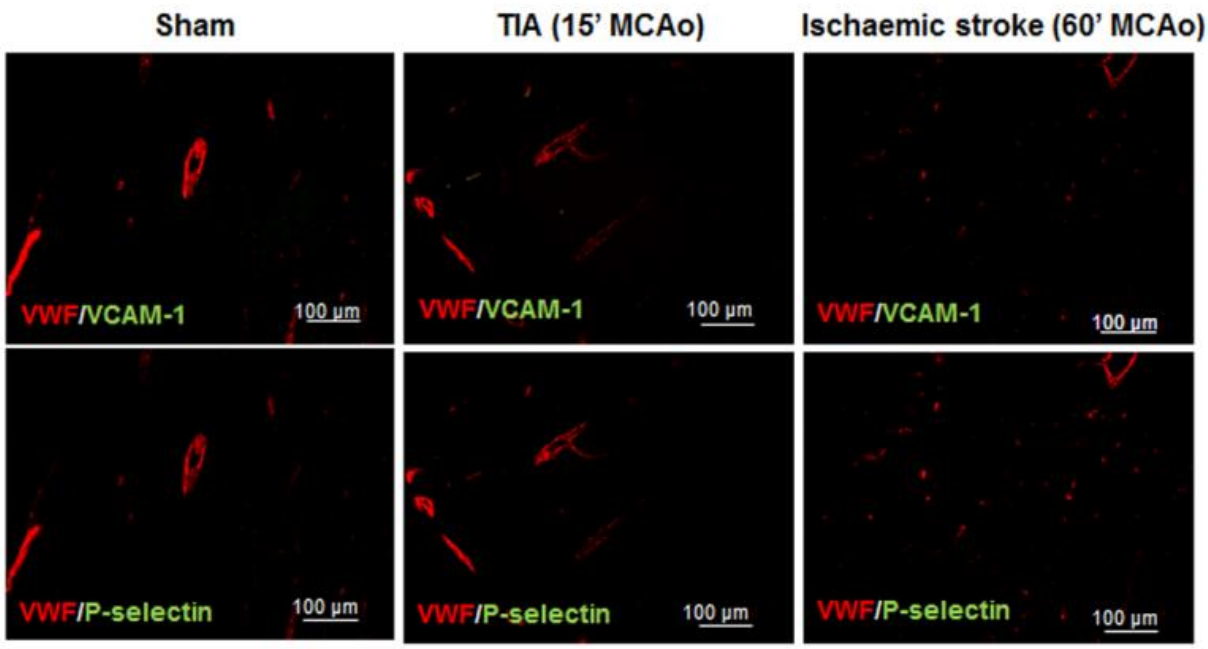
Supplementary Fig. 1. Controls of immunohistological studies. Representative immunohistological images of slices stained with secondary antibodies only (used to detect antibodies against VWF, P-selectin, VCAM-1) and antibodies against VWF and IgG in the cortex of sham, TIA and stroke mice (n = 3).

duration of compression	rCBV during compression (% of baseline)
sham	100
5''	25.2 ± 3.3
5'	21.1 ± 2.6
15'	27.9 ± 2.8
30'	27.2 ± 3.0
45'	22.7 ± 3.8
60'	28.3 ± 3.1
90'	25.8 ± 3.6
pMCAo	21.4 ± 2.8

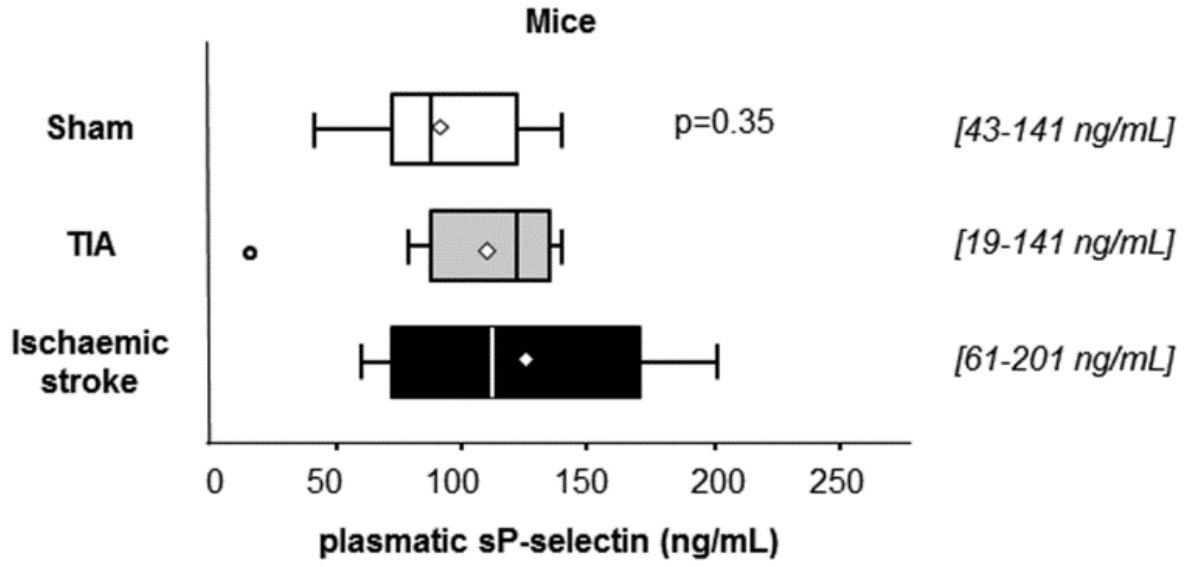
Supplementary Fig. 2. Vascular characterization of a preclinical mouse model corresponding to the tissue-based definition of TIA in humans: compression-induced MCA occlusion leads to transient focal brain ischemia. Quantifications of residual CBV during compression for all groups, mean+/− SEM; n = 10 per group.


A
MRA
T2
T2*
Ctrl
5''
5'
15'
30'
45'
60'
90'
pMCAo
B
DWI
15' occlusion
+ 1 day
+ 3 days
+ 7 days
+ 30 days
+ 90 days
C
Thionin
FJC
Sham
15' MCAo
60' MCAo

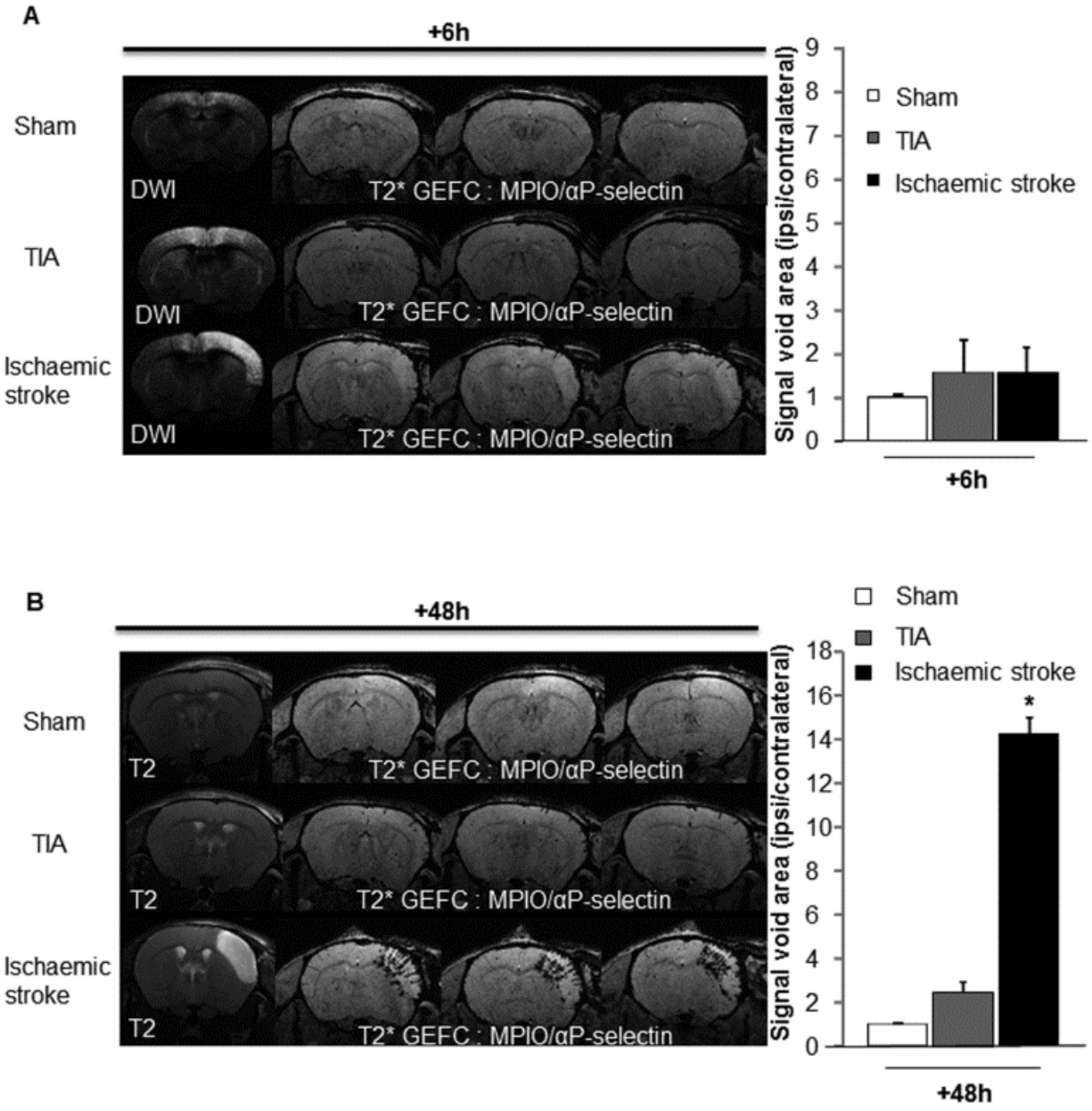
Supplementary Fig. 3. **Radiological characterization of a preclinical mouse model corresponding to the tissue-based definition of TIA in humans: a threshold of duration of MCA occlusion distinguishes TIA from ischemic stroke. (A)** Representative images of MRA, T2 and T2* acquired 24h after MCAo of different durations (0/sham, 15 or 60min, and permanent occlusion). Haemorrhagic transformation was never observed. MCA remained occluded only in the pMCAo (white arrow). (**B**) Representative images (n = 3 animals per delay) of longitudinal DWI acquired after 15 min MCAo. No lesion was observed even after 3 months. (**C**) Left: Coronal brain sections (20 µm) stained with thionin 24 hours after surgery in Sham, TIA (15min MCAo) and ischaemic stroke (60min MCAo) mice. An ischaemic lesion was only detected in ischaemic stroke animals (mean lesion volume: 16.95±1.68 mm^3; n = 5). Right: Fluorojade C (FJC) staining of suffering neurons: representative images showing minor stainings (yellow arrowhead) in sham and TIA animals vs widespread staining in ischaemic stroke animals at 24 hours (corresponding counting revealed a mean total number of labeled neurons of 79, 254 and 4989 in sham, TIA and ischaemic stroke mice, respectively; n = 3).



Supplementary Fig. 4. **Immunohistological detection of adhesion molecules in the contralateral cortex.** Representative immunohistological images of slices stained with antibodies against VWF, P-selectin, VCAM-1 in the contralateral cortex of sham, TIA and stroke mice (corresponding to Fig 2B; $n = 3$ per condition).



Supplementary Fig. 5. **Circulating levels of sP-selectin fail to discriminate TIA from non stroke.** Plasmatic levels of sP-selectin were quantified by ELISA in mice. In sham, TIA or stroke mice, none of the groups could be distinguished by their levels of sP-selectin (n = 10 per group; P = 0.35).



Supplementary Fig. 6. **High resolution molecular MRI of P-selectin 6 and 48 hours after surgery.** Representative DWI, T2 images and molecular imaging of P-selectin and corresponding quantifications of signal void areas (ipsi/contralateral MCA territory; n = 5 per group; * $P < 0.05$ versus sham) 6h (**A**) and 48h (**B**) post-surgery in sham, TIA and ischaemic stroke mice.