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Deficient hippocampal neurogenesis and cortical angiogenesis in the chronic stages of ischemic stroke

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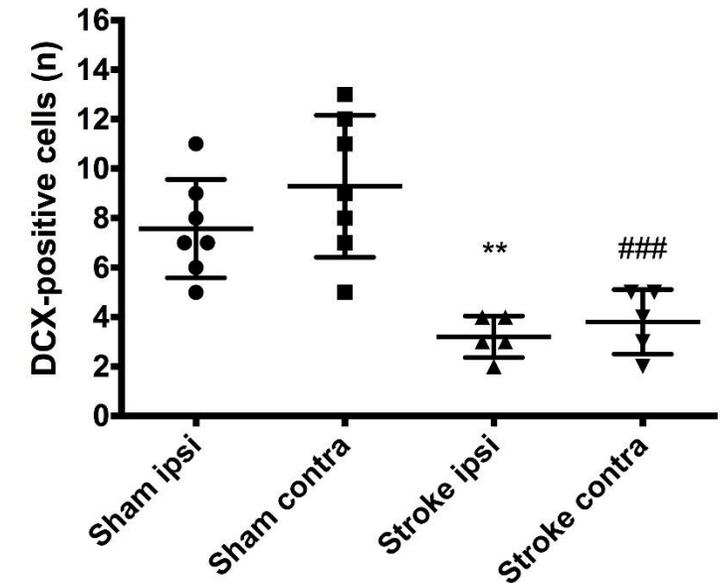
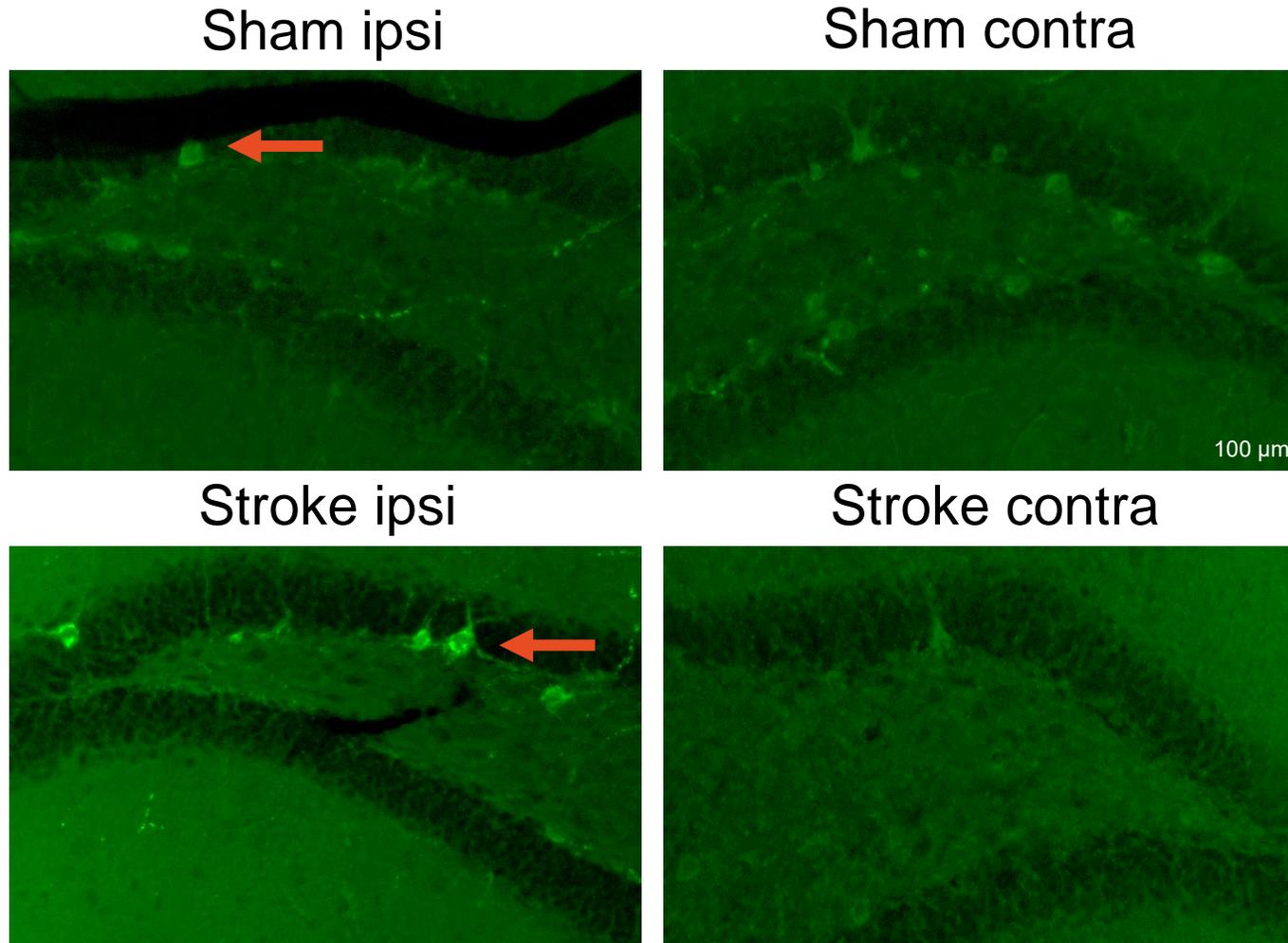
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Background. Ischemic stroke is one the most common causes of death and disability worldwide. It is caused by the obstruction of cerebral blood vessels and insufficient cerebral blood flow, resulting in cell injury and death in the lesion area. In the chronic stages of ischemic stroke, generation of new neurons (neurogenesis) and blood vessels (angiogenesis) around the ischemic lesion occur as a part of the recovery process (Rajkovic et al., 2018). Nevertheless, these two processes have not yet been extensively studied in ischemic stroke model animals.

Aim. The objective of this study was to assess whether the number of doublecortin (DCX)-positive cells in the hippocampus and the expression of vascular endothelium growth factor (VEGF) in the cortex are altered two months after the ischemic stroke induction in model mice.

Methods. An endovascular filament-induced middle cerebral artery occlusion (fMCAo, 60 min) was performed in male C57BL/6 mice (18-21 g, 10-12 w.o.). Two months after the surgery animals were euthanized and the number of DCX-positive cells and the density of VEGF were determined immunohistochemically in coronal brain slices. Statistical analysis was used to compare the measured values within group (ipsilateral or ischemized side vs. contralateral or non-ischemized side) and between groups (Sham vs. Stroke).

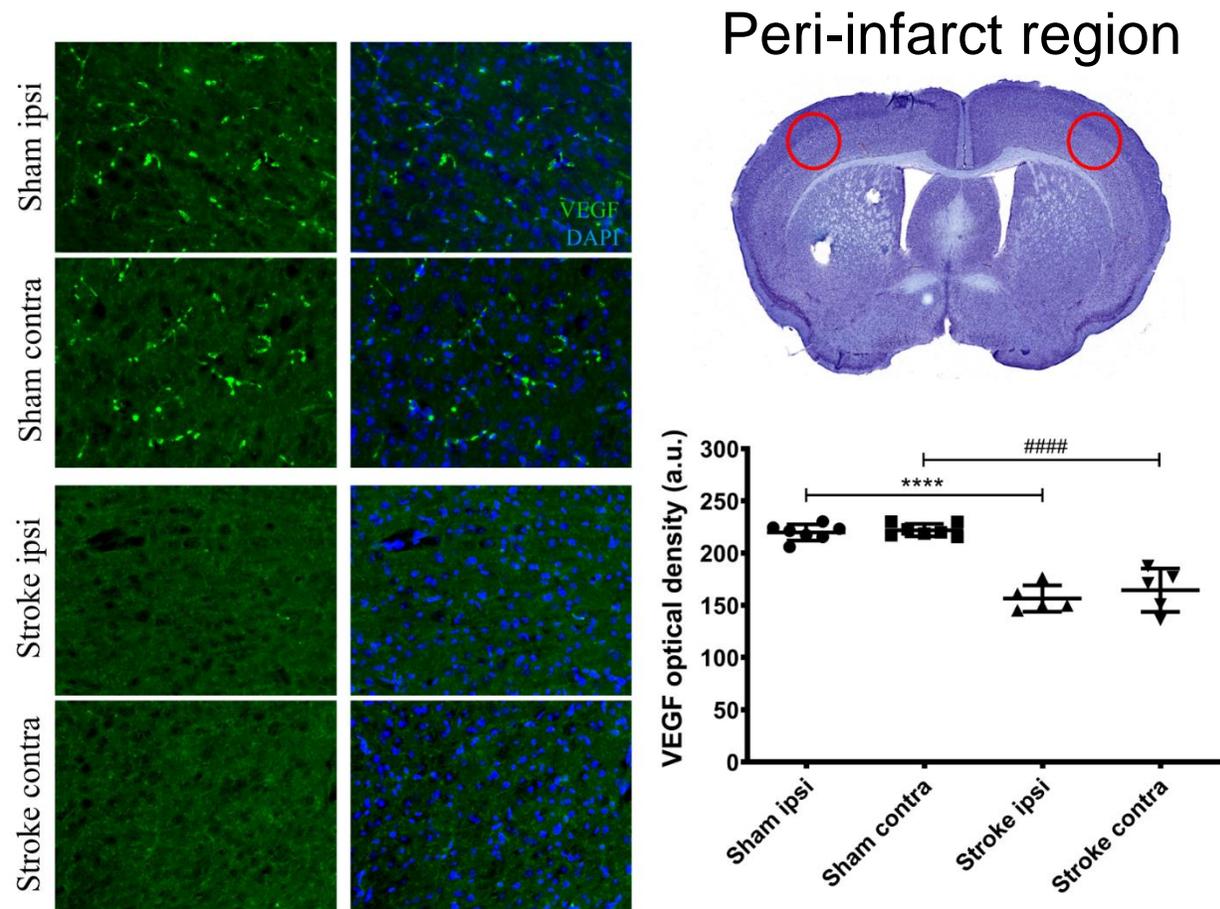
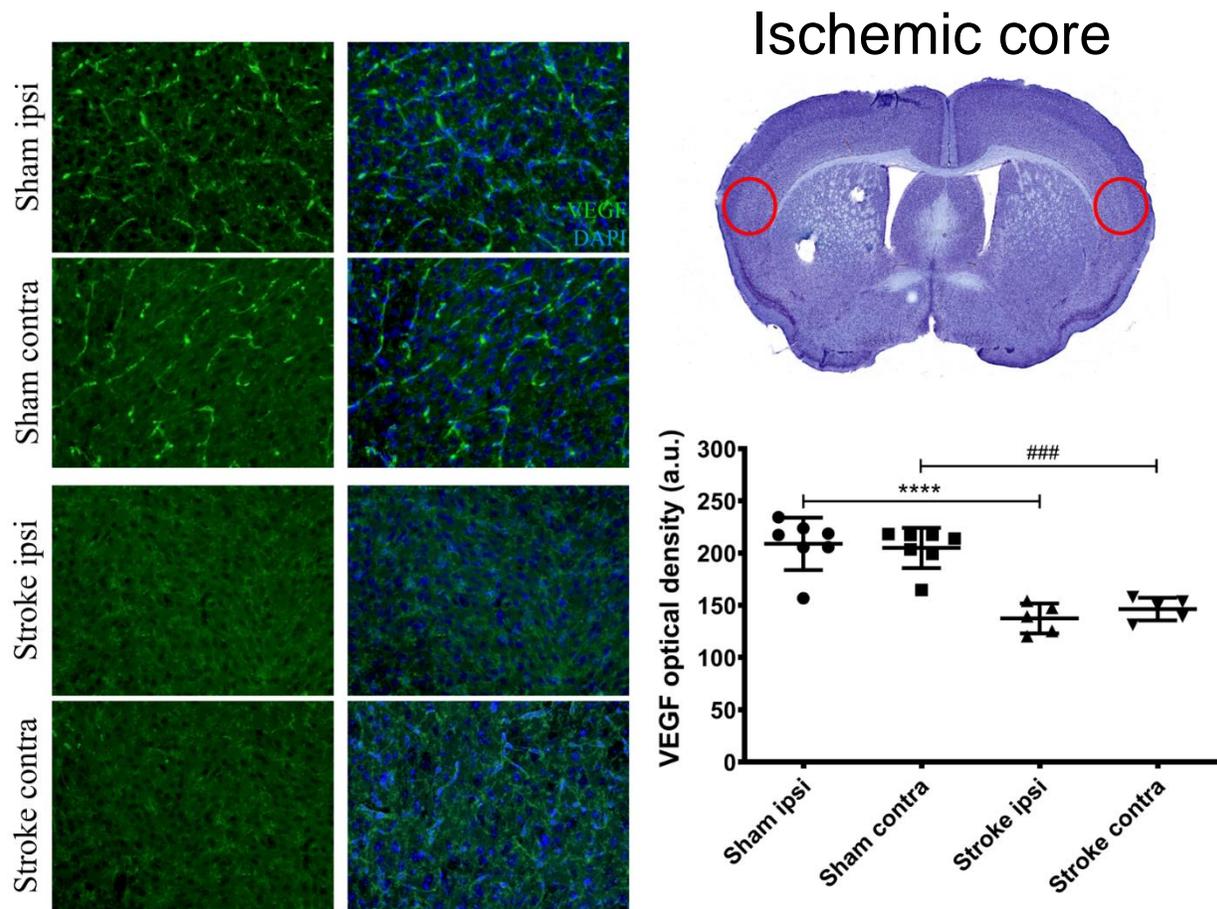
Markedly lower DCX+ cell number in the hippocampus 2 months after stroke induced by fMCAo



** $P < 0.01$ versus Sham ipsi

$P < 0.001$ versus Sham contra

Decreased VEGF density after stroke induced by fMCAo



**** $P < 0.0001$ versus Sham ipsi

$P < 0.001$ versus Sham contra

**** $P < 0.0001$ versus Sham ipsi

$P < 0.0001$ versus Sham contra

Conclusions and acknowledgements

Conclusions. Obtained preliminary data show that two months after fMCAo induction, neurogenesis and angiogenesis are suppressed in both sides of the male mice brain – ipsi- and contralateral. Moreover, angiogenesis is suppressed both in the ischemic core and in the peri-infarct region of the brain. Further research of neurogenesis and angiogenesis in other regions of the ischemic mice brain is imperative.

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