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Increased microglial activation in the chronic stages of experimental ischemic stroke

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Background. Neuroinflammation is a hallmark of many neurological deficits, including ischemic stroke, the top three morbidity cause worldwide. The central role in neuroinflammation, on the other hand, is played by microglia, resident immune cells of the brain (Zhang, 2019). Microglial activation, or microgliosis, is a complex process that can be both neuroprotective and neurotoxic in the ischemic brain. Therefore, investigating the rate of microgliosis in different stages of ischemic stroke is crucial to understand its role in the disease progression.

Aim. The aim of this study was to detect whether microglial activation and changes in microglial phenotype take place in ischemic stroke model mice in the chronic stages after stroke induction.

Methods. Endovascular filament-induced middle cerebral artery occlusion (fMCAo) in naïve male C57BL/6 mice (18-21g) for 60 min (n=9). Brief filament insertion in sham controls (n=5). Two months after fMCAo, immunohistochemistry was done to analyze the cells positive for ionized calcium-binding adapter molecule-1 (Iba-1). Microglial phenotype was assessed based on the cellular shape. One-way ANOVA followed by Holm-Šidák post hoc test within groups (ischemized versus non-ischemized brain hemisphere) and between groups (sham controls versus fMCAo mice).

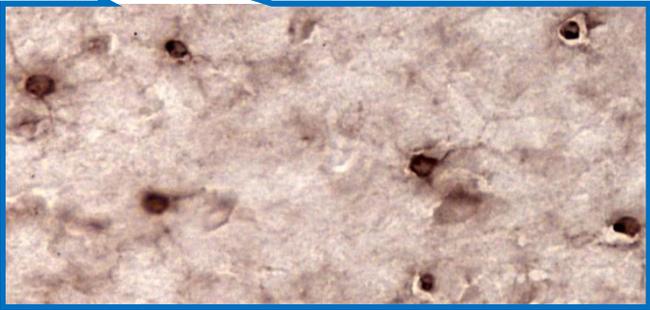
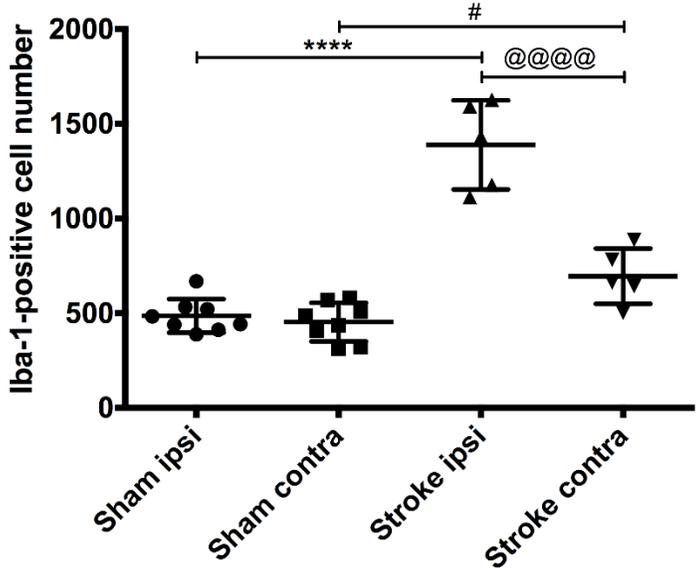
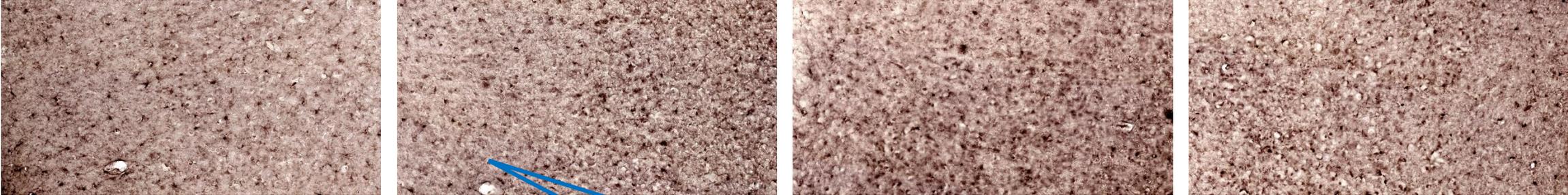
Iba-1-positive cell number is increased in the selected ischemic core region 2 months after stroke

Sham ipsi

Sham contra

Stroke ipsi

Stroke contra



**** $P < 0.0001$ versus Sham ipsi
 # $P < 0.05$ versus Sham contra
 @@@@ $P < 0.0001$ versus Stroke ipsi

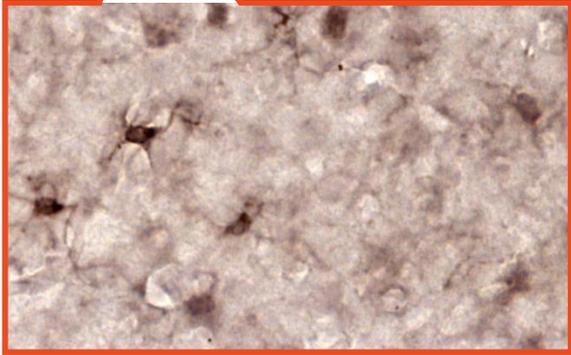
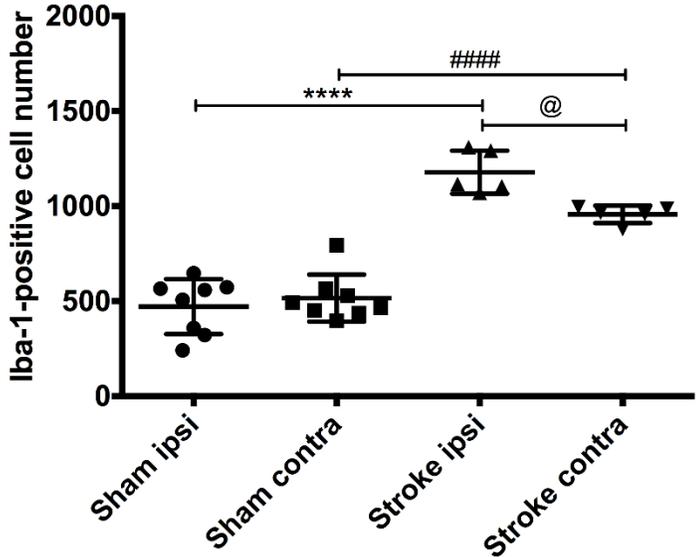
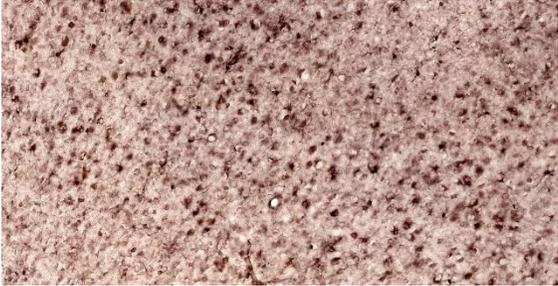
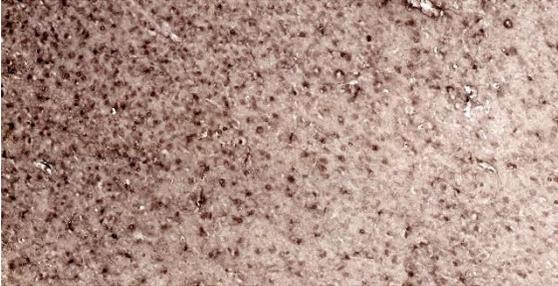
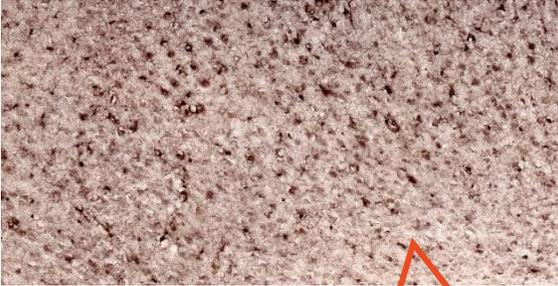
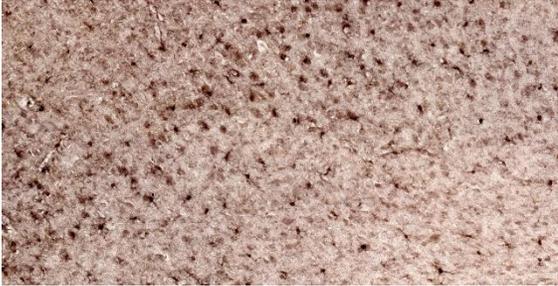
Iba-1-positive cell number is increased in the selected ischemic peri-infarct region 2 months after stroke

Sham ipsi

Sham contra

Stroke ipsi

Stroke contra



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

$P < 0.0001$ versus Sham contra

Conclusions & acknowledgements

Conclusions. These preliminary results show that ischemic stroke produces chronic microglial activation in the infarct area two months after fMCAo, yet microglial phenotype does not seem to be affected. The observed changes can be associated with the length of fMCAo induction.

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