**Response of glial cells in an *in vitro* model of stroke.**

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**Introduction.** Glial cells comprise a large volume of the brain. Originally described as ‘nerve-glue’ (Virchow, 1858), more recent studies indicate glia are fundamental to support brain function. Indeed, a network of glia often surrounds neurons, the electrically-active nerve cells that ultimately execute brain functions. Glia play key roles in brain development, regulate neurotransmitter, ion and nutrient levels, and also mediate repair and remodelling; thus, these cells are critical for supporting neuron activity. However, our understanding of glial function is still limited, and in particular, it remains to be established whether certain brain disorders affect glial cell physiology. We aimed to characterise early-phase (acute) electrophysiological and chemical responses evoked in glial cells during brain ischemia, a condition involving the depletion of oxygen and glucose, as occurring during a stroke.

**Methods.** Brain slices containing the Substantia Nigra (SN) or CA1 hippocampal brain regions were obtained from rats, and submerged in artificial cerebrospinal fluid (ACSF). Ischemia was modelled by switching perfusion to ACSF deprived of oxygen and glucose (OGD, 10 min). Cell membrane potentials were recorded using sharp intracellular microelectrodes. Following recordings, slices were fixed and processed for immunohistochemistry with glial cell markers (GFAP, S100β).

**Results.** Glial cells in the SN and CA1 hippocampal brain regions developed a cell membrane potential depolarization during OGD. Hippocampal glia repolarized after oxygen and glucose were restored (during reperfusion), however, cells in the SN continued to depolarize during this period. A loss of GFAP immunoreactivity was also observed after OGD in the SN, but not the hippocampus.

**Conclusions.** Our *in vitro* model of ischemia suggests that glial cells in the SN are particularly sensitive to ischemia-reperfusion. This unexpected high sensitivity should be investigated further, as the withdrawal of glial support may impair the activity of Substantia Nigra neurons following even a short period of brainstem ischemia, and lead to ‘vascular forms’ of parkinsonism.