

New research reveals brain's protection mechanism during stroke

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Neuroscientists have identified a natural protection mechanism in some of the brain's nerve cells during the onset of stroke. The findings, published today in the *Journal of Neuroscience*, could be used to develop treatments to protect other nerve cell types responsible for speech and movement.

Stroke — the third largest cause of death in the UK — causes disruption to the blood supply to the <u>brain</u>, depriving nerve cells of oxygen and nutrients. This leads to the death of nerve cells and the consequent loss of the brain's cognitive functions such as speech and movement. However, not all nerve cells are equally susceptible to stroke-induced damage.

The research, led by Dr Jack Mellor from the University of Bristol, examined two types of nerve cell in a part of the brain called the hippocampus — the region linked to memory and navigation. One of these cell types, the CA1 cell, is highly susceptible to damage after stroke whereas the other, the CA3 cell, is much more resistant despite many other similarities between the two cell types.

Dr Mellor, Senior Lecturer in the University's School of Physiology and Pharmacology, said: "We hope that if we can understand why some nerve cells are resistant to stroke damage we may be able to develop strategies to protect those cells that are sensitive."

The researchers found that the CA3 cells possess a mechanism for



reducing their susceptibility during, and immediately after, a laboratorybased model for stroke. This mechanism involved making the CA3 cells less sensitive to the neurotransmitter glutamate, which is released in large quantities during stroke, by removing glutamate receptor proteins from the surface of these cells.

The removal of glutamate receptors was triggered by adenosine A3 receptors that are activated by very high levels of the neurotransmitter adenosine found only during stroke conditions. Interestingly, CA1 cells that are susceptible to stroke damage did not have adenosine A3 receptors and did not respond to the stroke model by removing surface glutamate receptors. The findings reveal that CA3 cells possess a mechanism for neuronal protection during stroke.

Dr Mellor added: "Historically, stroke has been very difficult to treat because of its unpredictability and the need to administer drugs within minutes of the onset of a <u>stroke</u>. These problems will not be overcome by our research but our findings do reveal a natural protection mechanism in some <u>nerve cells</u>, which may be useful in developing treatments to protect other nerve <u>cell types</u>."

More information: The research, entitled 'Oxygen/glucose Deprivation Induces a Reduction in Synaptic AMPA Receptors on Hippocampal CA3 Neurons Mediated by mGluR1 and adenosine A3 Receptors', is published in the *Journal of Neuroscience*.

Provided by University of Bristol

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