

# New understanding of brain chemistry could prevent brain damage after injury

May 15 2011

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A protective molecule has been identified in the brain which, if used artificially, may prevent brain damage from the likes of stroke, head injury and Alzheimer's.

By looking at what happens in the brain after an [injury](#), new research has finally ended speculation over whether a key molecule, 'KCC2' causes [brain cell death](#) after an injury or prevents it. The finding, published today (16th May 2011) in The [Journal of Physiology](#) now opens the door to the development of artificial forms of the compound which could provide 'neuroprotection' to those who have suffered a [brain injury](#) – to prevent further damage.

Lead author of the research Dr Igor Medina from the Universite de la Mediterranee said: "Neuron damage can result from acute events such as [stroke](#), epilepsy or head injury or by chronic degeneration found in Alzheimer's and Parkinson's.

"When brain tissue is damaged, cells often continue to die after the initial stimulus has stopped. So it is important to find a way of stopping this cascade of cell death."

KCC2 is known as a 'neuronal membrane transporter' and plays a valuable role in regulating brain cell growth and their connections with other neurons. Previous research has shown that the level of KCC2 drops drastically after the brain has been injured, but it was unknown whether this drop was causing the damage to the cells, or was decreasing because

of it.

"The destiny of neurons in a damaged brain depends on a tiny equilibrium between pro-survival and pro-death signals. We wanted to know what KCC2 was signalling for – was it killing neurons or protecting them after an injury? Our study has found that KCC2 actually rescues the damaged cells."

The team studied damaged neurons from the hippocampus region of the brain, an area responsible for attention, spatial memory and navigation. They removed KCC2 altogether from damaged cells and found they died. But when they artificially increased the levels of KCC2 (by stimulating its expression using gene therapy), they found the damaged cells were protected from further damage, and death.

Dr Medina continued: "The death of neurons in the brain can be triggered by an imbalance of oxygen – known as oxidative damage, or where cells are incorrectly instructed to die by a neurotransmitter – a process known as excitotoxicity. KCC2 protects against both. It's really encouraging that we have identified a means of potentially protecting the [brain](#) from these common conditions."

Now the protective function of KCC2 is known, scientists can look at ways to maintain its levels in the brains of injured patients and prevent the cascade of damage that occurs. This could be achieved pharmaceutically, to naturally increase the levels of KCC2, or with gene therapy to introduce artificial KCC2.

"Neuroprotective agents that may stem from this research would benefit the victims of car crashes, stroke and those suffering with epilepsy, Parkinson's and Alzheimer's – it's a major focus for further studies," concluded Dr Medina.

**More information:** Knocking down of the KCC2 in rat hippocampal neurons increases intracellular chloride concentration and compromises neuronal survival. Christophe Pellegrino, Olena Gubkina, Michael Schaefer, H'el'ene Becq, Anastasia Ludwig, Marat Mukhtarov, Ilona Chudotvorova, Severine Corby, Yuriy Salyha, Sergey Salozhin, Piotr Bregestovski and Igor Medina. *J Physiol* 589, 2475-2496.

Provided by Wiley

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