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Scientists develop world's most advanced drug to protect the brain after a stroke

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Scientists at the Toronto Western Research Institute (TWRI), Krembil Neuroscience Center, have developed a drug that protects the brain against the damaging effects of a stroke in a lab setting. This drug has been in development for a few years. At this point, it has reached the most advanced stage of development among drugs created to reduce the brain's vulnerability to stroke damage (termed a "neuroprotectant"). Over 1000 attempts to develop such drugs by scientists worldwide have failed to be translated to a stage where they can be used in humans, leaving a major unmet need for stroke treatment. The drug developed by the TWRI team is the first to achieve a neuroprotective effect in the complex brain of primates, in settings that simulate those of human strokes. ischemic stroke.

The study, "Treatment of Stroke with a PSD95 inhibitor in the Gyrencephalic Primate Brain", published online today in *Nature*, shows how the drug, called a "PSD95 inhibitor" prevents brain cell death and preserves brain function when administered after a stroke has occurred.

"We are closer to having a treatment for stroke than we have ever been before," said Dr. Michael Tymianski, TWRI Senior Scientist and the study's lead author. "Stroke is the leading cause of death and disability worldwide and we believe that we now have a way to dramatically reduce its damaging effects."

During a stroke, regions of the brain are deprived of blood and oxygen. This causes a complex sequence of chemical reactions in the brain,

which can result in neurological impairment or death. The PSD95 inhibitor published by the Toronto team acts to protect the brain by preventing the occurrence of these neurotoxic reactions.

The study used cynomolgus macaques, which bear genetic, anatomic and behaviour similarities to humans, as an ideal model to determine if this therapy would be beneficial in patients.

Animals that were treated with the PSD95 inhibitor after a stroke had greatly reduced brain damage and this translated to a preservation of neurological function. These improvements were observed in several scenarios that simulated human strokes. Specifically, when the treatment was given either early, or even at 3 hours, after the stroke onset, the animals exhibited remarkable recoveries. Benefits were also observed when the drug therapy was combined with conventional therapies (aimed at re-opening blocked arteries to the brain). Beneficial effects were observed even in a time window when conventional therapies on their own no longer have an effect.

"There is hope that this new drug could be used in conjunction with other treatments, such as thrombolytic agents or other means to restore blood flow to the brain, in order to further reduce the impact of stroke on patients," said Dr. Tymianski. "These findings are extremely exciting and our next step is to confirm these results in a clinical trial."

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