Antidepressants as treatment immediately following a stroke?

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A study at the Buck Institute for Age Research suggests a new strategy for the treatment of stroke. Research in rodents shows the growth of new neurons, also known as neurogenesis, lessens the severity of stroke and dramatically improves function following a stroke. The research suggests that drugs shown to promote neurogenesis in rodents could have benefits for human stroke victims and that those drugs-which include antidepressants and mood stabilizers such as lithium-may be suitable for study in human clinical trials. Results of the research appear the week of April 12 in the online edition of the *Proceedings of the National Academy of Sciences*.

"What this study shows more convincingly than in the past is that the production of new neurons after stroke is beneficial in rodents," said Buck faculty member and senior author David Greenberg, MD, PhD. "Assuming that neurogenesis is also beneficial in humans, drugs approved by the FDA for other purposes and already shown to promote new neuron growth in rodents might be worth studying as a potential treatment for stroke in humans. For example, antidepressants are often used to treat post-stroke depression, but their potential for improving outcome from stroke itself is less certain."

Previous research by the same group at the Buck Institute, which includes Drs. Kunlin Jin, Xiaomei Wang, Lin Xie and Xiao Mao, showed that the brain attempts to heal itself following stroke by growing new neurons, but it has not been shown clearly that those new neurons improve function.

About 795,000 Americans suffer a stroke each year. Stroke is the third leading cause of death in the U.S. and is the leading cause of serious long-term disability in this country. Treatments for stroke are limited. Clot busting drugs, which have to be given within hours of the stroke, have been of great benefit to a small number of patients, but stroke is not usually diagnosed in time for them to be used.

The Buck Institute study, which did not involve screening any of the existing drugs that support neurogenesis, compared stroke size and recovery in mice who were genetically altered and treated to either grow or not grow new neurons prior to stroke. Greenberg says strokes were about 30 percent larger in the animals that did not grow new neurons; the rodents that did grow new neurons showed dramatic improvement in motor function following the stroke. The exact mechanism by which the new neurons improve outcome is unknown.

Greenberg says future research at the Buck will likely involve testing drugs that stimulate neurogenesis at various dosages and treatment times to see if they improve outcome following stroke in rodents. Building on the Institute's collaborative approach to research involving other age-related disorders, Greenberg says its also likely that the impact of the growth of new neurons will be examined in animal models of Alzheimer's, Parkinson's and Huntington's disease.

Although the possibility of using existing drugs for the treatment of stroke is one that may excite patients and patient advocates, Greenberg urges caution. He says those suffering from stroke should not treat themselves, even with FDA-approved drugs, without medical advice. "Everything has potential side effects," said Greenberg. "Even taking something as seemingly innocuous as an antidepressant carries the possibility of making someone worse. These drugs need to be tested in a controlled clinical setting."