

Substance naturally found in humans is effective in fighting brain damage from stroke

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Micrograph showing cortical pseudolaminar necrosis, a finding seen in strokes on medical imaging and at autopsy. H&E-LFB stain. Credit: Nephron/Wikipedia

A molecular substance that occurs naturally in humans and rats was found to "substantially reduce" brain damage after an acute stroke and contribute to a better recovery, according to a newly released animal study by researchers at Henry Ford Hospital.



The study, published online before print in *Stroke*, the journal of the American Heart Association, was the first ever to show that the peptide AcSDKP provides neurological protection when administered one to four hours after the onset of an <u>ischemic stroke</u>.

This type of a stroke occurs when an artery to the brain is blocked by a blood clot, cutting off oxygen and killing brain tissue with crippling or fatal results.

"Stroke is a leading cause of death and disability worldwide," said Li Zhang, M.D., a researcher at Henry Ford and lead author of the study. "Our data showed that treatment of acute stroke with AcSDKP alone or in combination with tPA substantially reduced neurovascular damage and improved neurological outcome."

Commonly called a "clot-buster," tPA, or <u>tissue plasminogen activator</u>, is the only FDA-approved treatment for acute stroke.

However, tPA must be given shortly after the onset of stroke to provide the best results. It also has the potential to cause a <u>brain hemorrhage</u>.

The Henry Ford study found that this narrow "therapeutic window" is extended for up to four hours after stroke and the therapeutic benefit of tPA is amplified when tPA is combined with AcSDKP. Further, the researchers discovered that AcSDKP alone is an effective treatment if given up to one hour after the brain attack.

The researchers tested the actions of both substances on laboratory rats in which acute stroke had been induced. It was already known that the peptide AcSDKP provides anti-inflammatory effects and helps protect the heart when used to treat a variety of cardiovascular diseases. The Henry Ford scientists reasoned that the peptide may have similar neurological benefits.



Significantly, they found that AcSDKP can readily cross the so-called "blood brain barrier" that blocks other neuroprotective substances.

A battery of behavioral tests was given to the lab rats both before and after stroke was induced to measure the effects of AcSDKP administered alone one hour after onset and combined with tPA four hours after stroke.

Besides finding that both methods "robustly" decreased neurological damage associated with <u>stroke</u>, they did so without increasing the incidence of <u>brain</u> hemorrhage or the formation of additional blood clots.

"With the increased use of clot-busting therapy in patients with <u>acute</u> <u>stroke</u>, both the safety and effectiveness of the combined treatment shown in our study should encourage the development of clinical trials of AcSDKP with tPA," Dr. Zhang says.

More information: <u>stroke.ahajournals.org/content ...</u> <u>004399.full.pdf+html</u>

Provided by Henry Ford Health System

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