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GDF10 molecule identified as a key player in repair mechanisms after stroke

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Looking at brain tissue from mice, monkeys and humans, scientists have found that a molecule known as growth and differentiation factor 10 (GDF10) is a key player in repair mechanisms following stroke. The findings suggest that GDF10 may be a potential therapy for recovery after stroke. The study, published in *Nature Neuroscience*, was supported by the National Institute of Neurological Disorders and Stroke (NINDS), part of the National Institutes of Health.

"These findings help to elucidate the mechanisms of repair following stroke. Identifying this key protein further advances our knowledge of how the brain heals itself from the devastating effects of stroke, and may help to develop new therapeutic strategies to promote recovery," said Francesca Bosetti, Ph.D., stroke program director at NINDS.

Stroke can occur when a brain blood vessel becomes blocked, preventing nearby tissue from getting essential nutrients. When brain tissue is deprived of oxygen and nutrients, it begins to die. Once this occurs, repair mechanisms, such as axonal sprouting, are activated as the brain attempts to overcome the damage. During axonal sprouting, healthy neurons send out new projections ("sprouts") that re-establish some of the connections lost or damaged during the stroke and form new ones, resulting in partial recovery. Before this study, it was unknown what triggered axonal sprouting.

Previous studies suggested that GDF10 was involved in the early stages of axonal sprouting, but its exact role in the process was unclear. S. Thomas Carmichael, M.D., Ph.D., and his colleagues at the David Geffen School of Medicine at the University of California Los Angeles took a closer look at GDF10 to identify how it may contribute to axonal sprouting.

Examining animal models of stroke as well as human autopsy tissue, Dr. Carmichael's team found that GDF10 was activated very early after stroke. Then, using rodent and human neurons in a dish, the researchers tested the effect of GDF10 on the length of axons, the neuronal projections that carry messages between brain cells. They discovered that GDF10 stimulated axonal growth and increased the length of the axons.

"We found that GDF10 caused many different neurons in a dish to grow, including human neurons that were derived from stem cells," said Dr. Carmichael.

His group also found that GDF10 may be important for functional recovery after stroke. They treated mouse models of stroke with GDF10 and had the animals perform various motor tasks to test recovery. The results suggested that increasing levels of GDF10 were associated with significantly faster recovery after stroke. When the researchers blocked GDF10, the animals did not perform as well on the motor tasks, suggesting the repair mechanisms were impaired--and that the natural levels of GDF10 in the brain represent a signal for recovery.

"We were surprised by how consistently GDF10 caused new connections to form across all of the levels of analysis. We looked at rodent cortical neurons and human neurons in dish as well as in live animals. It's a demanding gauntlet to run, but the effects of GDF10 held up in all of the levels that we tested," said Dr. Carmichael.

It has been widely believed that mechanisms of brain repair are similar to those that occur during development. Dr. Carmichael's team conducted comprehensive analyses to compare the effects of GDF10 on genes related to stroke repair with genes involved in development and learning and memory, processes that result in connections forming between neurons.

Surprisingly, there was little similarity. The findings revealed that GDF10 affected entirely different genes following stroke than those involved in development or learning and memory.

"We found that regeneration is a unique program in the brain that occurs after injury. It is not simply Development 2.0, using the same mechanisms that take place when the nervous system is forming," said Dr. Carmichael.

More research is necessary to determine whether GDF10 can be a potential treatment for stroke recovery.

Source:

NIH/National Institute of Neurological Disorders and Stroke
