Novel Small Molecule Treatment for Hemorrhagic Stroke

Published date: Feb. 15, 2018

Technology description

Researchers at the University of New Mexico have developed a pharmaceutical treatment utilizing small molecules to inhibit mechanisms of brain damage from intracerebral hemorrhage (ICH)/hemorrhagic stroke (HS).
The invention identifies a zinc specific chelator capable of inhibiting zinc insertion into protoporphyrin, greatly decreasing intracerebral hemorrhage (ICH)-induced endogenous zinc protoporphyrin (ZnPP) production. ZnPP accumulation is prevalent in surrounding brain tissue, following ICH-induced brain damage; therefore, since the chelator can be readily inhibited by small molecules, this may provide a treatment target for ICH therapy.

Background

Stroke is a leading cause of death and adult disability in the world. The second most prevalent type of stroke, known as intracerebral hemorrhage (ICH) and hemorrhagic stroke (HS), has the poorest prognosis of all stroke subtypes and is responsible for about 40 percent of all stroke deaths. The lysis of red blood cells, hemin release, and overload of iron are recognized as the primary causes of ICH-induced brain damage. There are currently no treatments available for ICH, as the cellular mechanisms behind ICH-induced brain damage are only partially understood. However, recent discoveries have emerged exploiting the underlying relationship between the accumulation of zinc and brain damage.

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Application area

Associated zinc accumulation to cell death
Potential target for reducing brain damage following intracerebral hemorrhage
Identified the mechanistic link between ZnPP and ICH-induced brain damage, leading to further translational studies
Hypoxia, zinc, and blood are three indispensable factors in ZnPP generation following ICH

Institution

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