Researchers help find new therapeutic target for treating traumatic brain injury

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(Medical Xpress)—A research team including members of the Department of Bioengineering in the University of Pennsylvania School of Engineering and Applied Science has discovered that drug intervention to reduce intercellular signaling between astrocytes following traumatic brain injury reduces cognitive deficits and damage.

David F. Meaney led the study alongside members of his lab and researchers from Penn's Perelman School of Medicine, Columbia University, Rutgers University and Tufts University.

It was published in the journal *Brain*.

Traumatic brain injury will be the third highest cause of death and disability in the world by 2020, according to researchers. Existing therapies, which focus on the early changes that occur within neurons of the brain after injury, may interfere with the brain's processing of information through its connected networks.

The collaborative group of investigators discovered that intercellular signaling between astrocytes, the star-shaped glial cells in the brain and spinal cord, plays a prominent role in cell death after brain injury. The team found that a single injury to the brain can trigger widespread signaling through the astrocyte network, which can adversely affect the communication among neurons in the network.

"We were initially very surprised that the effect of astrocyte signaling on

neuronal communication was so profound after injury," Meaney said.

By studying how these changes traveled through the astrocytes in the brain and in cell culture, the team discovered one specific type of signaling that could reduce the response dramatically. After screening several drug candidates, the team showed that one type of drug could improve cognitive recovery following a single traumatic brain injury.

"We are very excited at the promise of this new direction for treating traumatic injury," Meaney said, "because it may represent a different angle of attack for treating a complicated disease."

More information: brain.oxfordjournals.org/content/136/1/65.long

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