Improving functional recovery of the brain and heart after traumatic brain injury

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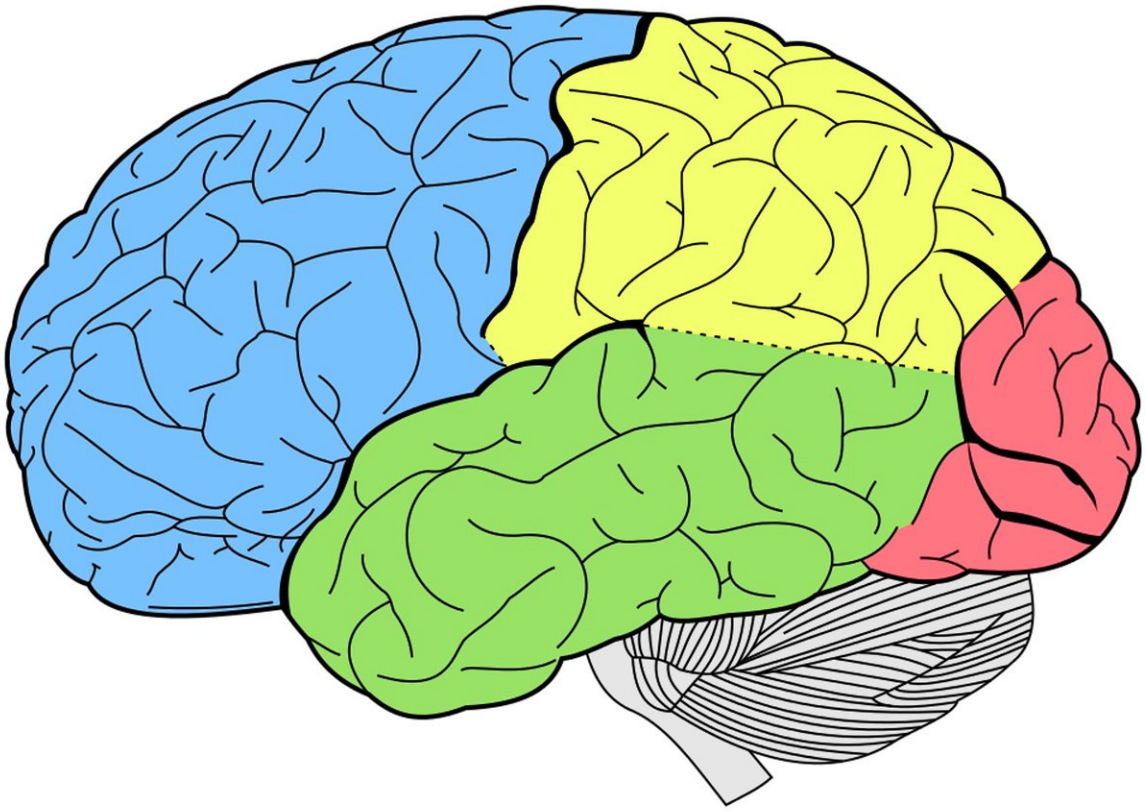

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A new study in the *Journal of Neurotrauma* has shown that Angiotensin II type 2 receptor (AT2R) activation improves neurological and cardiac

deficiencies caused by traumatic brain injury (TBI) in mice. AT2R activation is known to exert protective roles in the brain and heart.

TBI of any severity is associated with a high risk of developing long-term neurological and cardiovascular complications.

Based on previous studies, which have reported that activation of AT2R in stroke confers neuroprotection, Rongcai Jiang, Ph.D., from Tianjin Medical University General Hospital, and co-authors hypothesized that AT2R may have an impact on both the brain and heart after TBI.

The results indicated that treatment with an AT2R agonist (C21) 24 hours after TBI alleviated blood-brain barrier leakage and brain swelling, inhibited the expression of proinflammatory cytokines in the brain and heart, and benefited cardiac function.

"Delayed administration of C21 improves neurological and cardiac recovery after TBI," stated the investigators. "Our findings suggest that activation of AT2R may serve as a feasible strategy to benefit the long-term prognosis of TBI patients."

"This is an impressive preclinical study. The authors did a lot of things right from a transparency and rigor perspective: They began administering the therapeutic at a clinically realistic time point- 24 hours after injury. They randomly assigned mice to groups and evaluated them in a blinded fashion. They accounted for all the animals. I'm excited to see where this line of research leads," says David L. Brody, MD, Ph.D., Editor-in-Chief of *Journal of Neurotrauma*.

More information: Yu Qian et al, Delayed administration of an angiotensin II type 2 receptor agonist promotes functional recovery of the brain and heart after traumatic brain injury, *Journal of Neurotrauma* (2024). DOI: 10.1089/neu.2023.0375

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