

# One type of brain cell might hold key to inflammation after head injury

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By eliminating one type of immune cell in the brain, researchers were able to erase any evidence of inflammation following traumatic brain injury, according to a new study from The Ohio State University.

"We used a drug to wipe out [cells](#) called microglia in mice that had experienced [brain injury](#), and the inflammation that is a hallmark of traumatic [brain injury](#) vanished," said Kristina Witcher, the Ohio State graduate student who led the study, which was presented Nov. 7 in San Diego at the annual Society for Neuroscience meeting.

Finding potential targets for treatment of serious brain injury is a major goal of neuroscience because there are currently no known approved medications to treat it, Witcher said. Furthermore, understanding cellular-level changes associated with sports-related concussion and other brain injuries could give health care providers better scientific support for post-injury recommendations, such as how long an athlete should stay off the field, she said.

The study was designed to mimic the type of traumatic brain injury a person would experience after hitting his or her head with enough force to briefly lose consciousness.

"Chronic inflammation with brain injury is harmful, and in this study we were able to eliminate that inflammatory response of the immune system by targeting just one specific cell type," said Jonathan Godbout, the study's senior author and assistant director for basic science at Ohio

State's Institute for Behavioral Medicine Research.

"Now, we have a specific cell to aim for when looking at potential interventions to decrease the harm caused by concussions," Godbout said.

Though other cell types, including those that make up blood vessels, have been previously implicated in the inflammation following serious head injury, this study offers detailed proof that immune cells called microglia play a key role, said Godbout, a professor of neuroscience who is part of Ohio State Wexner Medical Center's Neurological Institute.

The drug used in the study to eliminate the microglia from the mouse brain isn't likely a potential treatment for brain injury in humans because it would cause too much damage to other vital functions of these cells, which make up about 10 to 15 percent of all brain cells, he said.

"We don't know the long-term effects of eliminating these [immune cells](#), but we are doing more physiological, biochemical and behavioral analysis to get to the bottom of that question," Godbout said.

The research team also is seeking more details about the inflammatory response at different periods of time after injury.

"You have to understand the changing nature of what's happening in these cells in order to better determine where and when to intervene," Witcher said.

Previous efforts to treat [traumatic brain injury](#) with anti-inflammatories have been unsuccessful in humans, Witcher said, highlighting the need for neuroscientists to explore novel treatment approaches.

The research also uncovered an anomaly in the microglia cells in the

brain after injury: They were elongated.

"For now, we don't really know what that structure means and whether it has any functional significance, but those are questions we'd like to explore," Witcher said.

She and Godbout also said they're interested in understanding if some of the cells are "good guys" and others are "bad guys."

"It's possible that some promote inflammation and others work against it, maybe even by keeping neurons alive," Godbout said.

Provided by The Ohio State University

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