

Kansas City University Review Highlights Astrocytes as Key Drivers of Chronic Traumatic Encephalopathy

Scoping review suggests astrocytic dysfunction may precede neuronal degeneration in chronic traumatic encephalopathy

CHINA, July 16, 2026 /EINPresswire.com/ -- Chronic traumatic encephalopathy (CTE) is a progressive neurodegenerative disease linked to repetitive head impacts and traumatic brain injuries. It has been most commonly identified in contact-sport athletes, military personnel, and others exposed to repeated brain trauma. Traditionally, researchers have viewed CTE primarily as a neuron-centered disease characterized by the accumulation of abnormal tau proteins in the brain. However, a new scoping review from Kansas City University School of Medicine, USA, suggests that another type of brain cell, astrocytes, may play a much larger role in the disease process than previously recognized.

Now, a review led by Dr. Kameron Hahn and his team, examined 40 studies spanning postmortem human brain analyses, experimental models, molecular investigations, and biomarker research. This review was published in Volume 12 of the Chinese Neurosurgical Journal on May 21, 2026. Together, these studies provide growing evidence that astrocytes may be key contributors to both the initiation and progression of CTE, potentially reshaping how researchers understand and approach the disease. Astrocytes are star-shaped glial cells that perform a variety of critical functions in the brain. They help maintain the blood-brain barrier, regulate communication between neurons, support metabolic processes, and participate in the removal of waste products from brain tissue. "We studied the role of astrocytes in the pathogenesis of CTE, and to what extent astrocytic mechanisms contribute to disease initiation, propagation, and clinical manifestation relative to neuronal pathology," says Dr. Hahn.

The researchers identified four major themes that consistently emerged across the literature. These included interface-specific astrogliosis, disruption of aquaporin-4-mediated waste-clearance pathways, astrocytic degeneration associated with impaired glutamate regulation, and chronic neuroinflammation driven by interactions between astrocytes and microglia. Collectively, these mechanisms may contribute to the development of the characteristic pathological features observed in CTE. One of the review's most significant observations is that astrocytic abnormalities often appear early in the disease process. Several studies demonstrated that astrocytes become activated in brain regions that experience the greatest mechanical stress from repetitive head impacts, particularly around blood vessels and within the depths of cortical

sulci. These findings suggest that astrocytes may not simply react to existing damage but could actively influence the cascade of events that eventually leads to widespread neurodegeneration.

The review also highlights the role of astrocytes in maintaining the brain's glymphatic system, a network responsible for clearing metabolic waste and potentially harmful proteins from brain tissue. Astrocytes regulate this process through specialized water channels known as aquaporin-4. When these channels become disrupted following repeated brain injury, the brain's ability to remove toxic proteins may be compromised. This dysfunction could contribute to the accumulation of hyperphosphorylated tau, a hallmark feature of CTE pathology. Another important finding centers on neuroinflammation. Astrocytes communicate closely with microglia, the brain's resident immune cells. Evidence reviewed by the authors suggests that repeated injury may trigger a persistent inflammatory response involving both cell types. Over time, this chronic inflammatory environment may accelerate tissue damage and contribute to cognitive, behavioral, and neurological decline.

The review also explores the potential clinical implications of astrocyte-related biomarkers. In particular, glial fibrillary acidic protein (GFAP), a protein released during astrocytic injury, has emerged as a promising candidate for monitoring neuroglial damage. Although no biomarker currently provides a definitive diagnosis of CTE in living individuals, astrocyte-derived markers may eventually become part of multimodal diagnostic approaches aimed at identifying individuals at risk before irreversible brain damage occurs. According to the authors, the evidence supports a broader understanding of CTE as a disorder involving dysfunction of the neuroglial and neurovascular systems rather than a disease driven solely by neuronal pathology. This shift in perspective could have important implications for future research, helping scientists identify new therapeutic targets and improve strategies for diagnosis and prevention.

By placing astrocytes at the center of disease pathogenesis, the review challenges long-held assumptions about CTE and opens new avenues for investigating how repetitive brain trauma leads to chronic neurological decline. "The findings may ultimately help guide the development of earlier diagnostic tools and more effective interventions for individuals affected by repetitive head injuries," concludes Dr. Hahn.

Reference

Title of original paper: Astrocytic contributions to the pathogenesis of chronic traumatic encephalopathy: a scoping review

Journal: Chinese Neurosurgical Journal

DOI: <https://doi.org/10.1186/s41016-026-00434-w>

About Kansas City University, USA

Kansas City University (KCU) is one of the oldest and largest health sciences universities in the United States, dedicated to educating future leaders in medicine, biosciences, dental medicine, and health professions. Founded in 1916, KCU is recognized for its innovative, student-centered

approach to healthcare education and research. With campuses in Kansas City and Joplin, Missouri, the university trains physicians and healthcare professionals committed to improving patient care and advancing medical knowledge. Through academic excellence, research, and community engagement, KCU continues to contribute to healthcare innovation and workforce development nationwide.

Website: <https://www.kansascity.edu/>

About Dr. Kameron Hahn from Kansas City University School of Medicine, USA

Dr. Kameron Hahn is a medical student at Kansas City University, Joplin, Missouri, USA, where he is pursuing his Doctor of Osteopathic Medicine (DO) degree. His academic and research interests include neuroscience, neurodegenerative diseases, chronic traumatic encephalopathy (CTE), translational medicine, and biomedical sciences. Hahn has contributed to multiple peer-reviewed scholarly publications, including reviews focused on neurodegenerative disorders and oncology-related research. He was recognized as a 2025 Barry Goldwater Scholar, one of the United States' most prestigious undergraduate awards for students pursuing research careers in science and medicine. His work aims to advance understanding of disease mechanisms and improve future diagnostic and therapeutic strategies.

Funding information

This research did not receive any specific grant from funding agencies in the public, commercial, or non-profit sectors

Dr. Kameron Hahn

Kansas City University School of Medicine, Joplin, MO, USA

+861059978478 ext.

[email us here](#)

This press release can be viewed online at: <https://www.einpresswire.com/article/926878308>

EIN Presswire's priority is source transparency. We do not allow opaque clients, and our editors try to be careful about weeding out false and misleading content. As a user, if you see something we have missed, please do bring it to our attention. Your help is welcome. EIN Presswire, Everyone's Internet News Presswire™, tries to define some of the boundaries that are reasonable in today's world. Please see our Editorial Guidelines for more information.

© 1995-2026 Newsmatics Inc. All Right Reserved.