Study identifies distinct origin of ADHD in children with history of brain injury

A study in Biological Psychiatry compares the genetic and neural contributions to attention-deficit/hyperactivity disorder in youth with or without a history of traumatic brain injury

Philadelphia, August 13, 2018 – According to a study in Biological Psychiatry, physical brain injury in children contributes to the development of attention-deficit/hyperactivity disorder (ADHD), distinct from genetic risk for the disorder.

In the study, youth who reported ADHD symptoms and had a history of mild traumatic brain injury (TBI), such as a concussion, did not have an increased genetic risk for the disorder. This is in contrast to developmental ADHD, which is caused in part by the small cumulative effects of multiple ADHD-related genes.

“This article suggests that there are at least two forms of ADHD. One that is an expression of a risk inherited within families and the other which develops after traumatic brain injury,” said John Krystal, MD, Editor of Biological Psychiatry. “The latter is of particular interest in light of the growing evidence that contact sports and combat are associated with higher rates of traumatic brain injury than we previously recognized,” he added.

“Mild traumatic brain injury (which includes concussion) is very common in adolescents; epidemiological data show that approximately 1 in 5 report a previous mild traumatic brain injury,” said senior author Anne Wheeler, PhD, of SickKids Research Institute and University of Toronto, Canada. This rate is alarming considering that as many as 50 percent of children develop ADHD symptoms soon after the injury. Although symptoms resolve over time in most children, others convert to a diagnosis of ADHD.

In the study, first author Sonja Stojanovski, a doctoral student in Dr. Wheeler’s laboratory, and colleagues compared the origins of ADHD symptoms in 418 youth with a history of TBI and 3,193 with no TBI, all of whom were between 8 and 22 years old. Genetic risk score was associated with increased ADHD symptom severity, but only in youth without TBI. There was no association in those with TBI, meaning that genetic predisposition does not appear to make children more vulnerable to developing ADHD after brain injury.

The researchers also looked for hallmark abnormalities in brain structure associated with the disorder. The association between volumes of ADHD-related brain structures and ADHD symptom severity was similar between the two groups. However, an analysis of the connections bridging the two brain hemispheres revealed opposite relationships with ADHD symptoms between the groups. The structural findings indicate the presence of both similar and distinct neural mechanisms that cause ADHD after TBI.
“When thinking about treating youth with ADHD it is important to understand what the underlying causes are and how they may differ from person to person to move towards a personalized medicine approach,” said Dr. Wheeler. According to the findings of this study, this personalized approach may be based on whether a patient has experienced TBI.

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Notes for editors

Copies of this paper are available to credentialed journalists upon request; please contact Rhiannon Bugno at Biol.Psych@UTSouthwestern.edu or +1 214 648 0880. Journalists wishing to interview the authors may contact Anne Wheeler at anne.wheeler@sickkids.ca or +1 416 813 7654 ext 309585.

The authors’ affiliations and disclosures of financial and conflicts of interests are available in the article.

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