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Review Article

What Are the Long-Term Effects of Concussions on Autistic Youth?

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ABSTRACT

Concussions, a type of mild traumatic brain injury (mTBI), are infamous for their instant effects of dizziness, fatigue, and mental confusion. While it is known that concussions can cause lasting neurological effects, including impacts on cognitive function, behavior, and focus, the specific long-term effects on the brain, particularly among autistic youth, are not yet fully understood. Autistic youth may experience these effects differently from neurotypical populations, necessitating specialized care. Inherent differences in the neuropsychology and neurological structure of autistic individuals mean that diverse parts of the brain will be affected resulting in various behaviors. This review paper answers the following questions: What regions of the brain do concussions affect the most in neurotypical populations? How is the development of each of these regions affected by concussions in neurotypical populations? Are these trajectories different for autistic youth? By evaluating findings from existing studies, this paper enhances science's current understanding of the interplay between concussions and autism among the developing population.

Keywords: Concussions; Autistic Youth; autism; mean diffusivity; white matter; Fractional anisotropy

INTRODUCTION

Every year, an estimated 1.1 to 1.9 million sports and recreation-related concussions occur in children under 18, yet the drive to succeed keeps many young athletes pushing forward (1). For those aspiring to greatness in competitive sports, the threat of concussions could potentially derail both their athletic dreams and academic success. 1 out of 5 high school athletes involved in a contact sport will get a concussion sometime this year. Concussions in youth can seriously deter future athletic performance and impair cognitive function. Research on the effects of concussions on *autistic* youth can encourage more neurodivergent youth to get involved with the sports they love, help doctors identify concussion symptoms unique to autistic youth, and help clinical scientists develop specialized treatment plans. Examination of autistic youth in sports raises awareness and advocates for those with special needs in the sports domain as well as increases inclusion and participation.

WHAT IS A CONCUSSION?

Concussions are a type of mild traumatic brain injury (mTBI) induced by the head receiving a hard hit. The brain can bounce or twist in the skull which may result in a stretch of brain cells, a change in consciousness, or damage to the brain tissue. Some immediate symptoms include slurred speech and dizziness, with longer-term effects including problems with memory and concentration, chronic headaches, and an increased risk for neurological conditions. These immediate

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symptoms are caused by a chain reaction in the brain. This includes nerve cells getting overexcited, changes in brain chemicals, shifts in how the brain uses energy and blood, and problems with how brain cells send signals to each other (2). Concussions are particularly detrimental to developing populations, such as adolescents and children, because they take longer to recover than adults and often experience more severe effects. These effects can include greater difficulties with gait and balance control, compounded by the fact that their brains are still undergoing significant maturation (3). Additionally, young people are more susceptible to contracting subsequent concussions with less force and a longer recovery period (4).

WHAT PARTS OF THE BRAIN DO CONCUSSIONS CHIEFLY AFFECT NEUROTYPICAL POPULATIONS AND WHAT ARE THE FUNCTIONS OF THESE REGIONS?

Concussions chiefly affect the white matter in the frontal lobe (such as the prefrontal cortex), temporal lobe (such as the hippocampus), and midbrain. White matter (WM) is a network of nerve fibers that allows for the exchange of information between different areas of gray matter. It is found in the subcortical tissues of the brain, beneath gray matter, and contains axons (extensions of neurons). The prefrontal cortex within the frontal lobe focuses on voluntary movements and governs higher executive functions such as cognitive control. The temporal lobe dictates one's primary senses, especially auditory processing and visual perception (5). In general, there is a degradation of white matter integrity throughout the brain due to concussion (6). White matter diffusivity can be measured by fractional anisotropy, mean diffusivity (MD), and radial diffusivity (Figure 1).

FA is a measurement between 0 and 1 that shows how directionally water moves through white matter in the brain. A value of 0 means water diffuses equally in all directions (isotropic), while a value of 1 means water moves entirely in one direction along a single pathway (highly directional). A study found a decrease in the FA in the right temporal subcortical white matter, specifically the corpus callosum (the WM connecting the two hemispheres of the brain) and parietal and frontal subcortical white matter, as a result of concussion (8). Another study showed a significant reduction of the FA of WM in a cluster (group of neurons) on the superior corona radiata. Other clusters formed in areas dictating visuospatial information and relaying information from the thalamus (9).

An important marker of an acute concussion is a change in mean diffusivity MD, which is how freely water molecules move in brain tissue. As a result of a concussion, MD increased primarily in bilateral longitudinal fasciculi and left-lateralized superior corona radiata, areas that process visual information, especially object recognition (10). In this study, global functional connectivity revealed distinct clusters of neurons in some regions involved in visual processing. Additionally, clusters of neurons in the bilateral postcentral gyri, supplementary motor area, and paracentral lobule, all pertaining to sensorimotor functions, were identified. Young children's post-concussion are prone to developing malfunctions in these very brain regions, leading to cognitive declines down the line. In one study, teenagers aged 11-15 experienced diminished cerebral blood flow days after concussion. After a few weeks this subsided, and after a month over half the children were back to normal values (11).

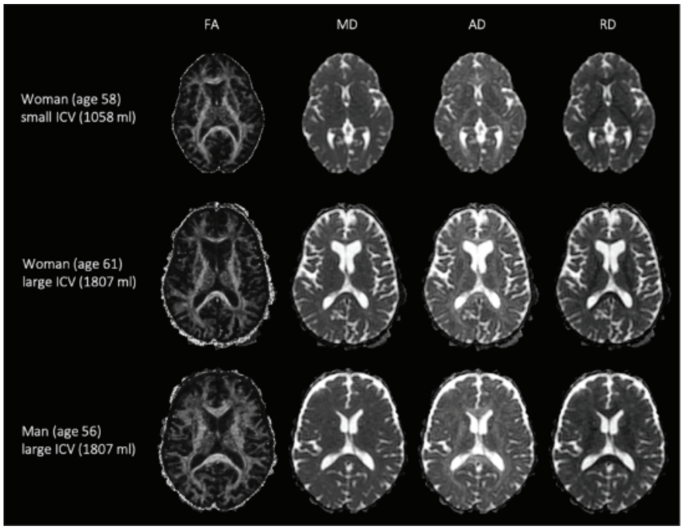

Figure 1. Diffusion tensor imaging (DTI) scans from three individuals showing measurements of white matter integrity across four metrics: fractional anisotropy (FA), mean diffusivity (MD), axial diffusivity (AD), and radial diffusivity (RD). This image highlights structural differences in white matter microstructure, particularly in the frontal and temporal lobes—regions commonly impacted by concussion. FA reflects fiber organization, while MD, AD, and RD provide insight into tissue density, axonal integrity, and myelin health. Such imaging tools are critical for identifying white matter degradation following mTBI (7).

WHAT ARE LONG-TERM BEHAVIORAL DAMAGES DUE TO CONCUSSIONS IN DEVELOPING POPULATIONS?

The collection of symptoms observed after concussions is categorized as post-concussion syndrome (PCS). One is said to have PCS if they possess at least three of the following symptoms within a month of the injury: headaches/fatigue, emotional instability, difficulty concentrating, increased alcohol intolerance, or insomnia. PCS lasts for an average of a few weeks, however, 15% of people still experience PCS up to a year after the concussion. In such cases, the most persistent symptoms seem to be headaches and emotional instability. PCS may lead to depression and anxiety long term. Experiences of these symptoms may be due to the metabolic alterations' concussion triggers, including reduced glucose metabolism, enhanced lipid peroxidation (free oxidation of polyunsaturated fats), disordered neurotransmitter secretion, and imbalanced trace element synthesis. These metabolic changes are typically present after 24 hours but have disappeared before the three-month mark (12).

Concussion also significantly impacts children and adolescents. For instance, children between 6 and 12 had weaker cognitive skills, such as verbal memory tests, compared to the rest of the kids in their age group, observed by using a Test for Attentional Performance battery. The Test of Attentional Performance for Children (KiTAP) assesses attentional performance, specifically cognitive speed, distractibility, and lapses of attention. A child's behavioral symptoms both have to do with the intensity of the concussion and their pre-existing cognitive abilities. A study on an eight-year-old girl found lasting electrophysiological deficits up to a year after her injury. While the girl's cognitive impairments resolved within 22 weeks, it was the electrophysiological deficits within the cortical regions that lasted for a year, demonstrating the lasting neurobiological impact of concussion among youth. However, the effects of concussion among youth are so diverse that children with innate higher cognitive abilities report less severe cognitive effects as they have different chemicals to make up for tissue damage. Interestingly, while some PCS symptoms in youth and adults are comparable, other symptoms may be more exacerbated for younger people. A study found that high school football players take longer to recover from concussions than college football players. Younger people typically have more severe cognitive sequelae and exhibit greater memory deficits than adults.

The most plausible explanation for increased vulnerability in younger brains is that the frontal lobe continues to develop well into the mid-twenties, making it more susceptible to injury (13). In immature brains, post-concussion damage often triggers elevated levels of apoptosis, a process where neurons that would otherwise integrate into functional circuits are lost. This disruption can impair critical cognitive functions such as decision-making, emotional regulation, and impulse control, which are governed by the still-developing prefrontal cortex. Additionally, while younger brains exhibit greater neuroplasticity, the ability to reorganize and form new neural connections, this adaptability is a double-edged sword. In neurotypical brains, plasticity allows for efficient recovery and learning. However, in autistic individuals, plasticity is often altered or dysregulated, potentially contributing to heightened sensitivity to sensory input and atypical synaptic pruning. This can lead to either hyperconnectivity or underconnectivity in brain networks, affecting communication and social behavior.

The sensitive periods of childhood and adolescence, times when the brain is especially responsive to environmental input, are thus crucial. Experiences during these windows can shape long-term brain structure and function. For instance, a lack of supportive social interaction or exposure to chronic stress during these periods can disrupt the development of emotional regulation and social cognition. In contrast, well-timed interventions and enriched environments can help reinforce adaptive pathways. Improperly timed or negative environmental influences during these sensitive periods can result in persistent cognitive and behavioral impairment, underscoring how deeply early experiences imprint on the developing brain (14).

WHAT ARE THE DEVELOPMENTAL AND STRUCTURAL BRAIN DIFFERENCES IN INDIVIDUALS WITH AUTISM COMPARED TO NEUROTYPICAL INDIVIDUALS?

Most people with autism spectrum disorder (ASD) have less brain tissue in their cerebellum (15), a region of the brain dictating language, attention, movement, and muscle control. Also, individuals with ASD have an enlarged amygdala, which regulates autonomic and endocrine functions. The width of gray matter, cortical thickness, is generally higher in those with ASD. However, the orbitofrontal cortex and posterior cingulum have a smaller surface area in autistic individuals (16). Tract-based spatial statistics (software utilizing tensor-fitting techniques to analyze diffusion data) showed regions of significantly reduced FA, increased MD, and increased radial diffusivity (a metric that quantifies the extent of water diffusion across the axonal fibers rather than along them) in subjects with ASD compared to neurotypical subjects. Further, those with ASD often exhibit degeneration of the corpus callosum (17). The nerve fibers in WM stretch to connect different regions of the brain, and it is proposed that these disruptions in these corpus callosum fibers lead to decreased connectivity in the brain and an increase in autistic tendencies.

An autistic individual's brain volume is, on average, larger than that of a neurotypical person due to rapid cortical expansion during infancy and early childhood. Additionally, an excess of cerebrospinal fluid often gives the brain an enlarged appearance. This difference is especially notable in the frontal and temporal lobes during early development—regions critical for social behavior, language processing, and executive function (18). As a result, the atypical growth in these areas is thought to contribute to common behavioral characteristics in autism, such as difficulties with social communication, language delays, and challenges with flexible thinking. The cerebral hemispheres remain enlarged into adulthood, suggesting these structural differences may have lasting effects on cognition and behavior. In addition, a study showed that the FA trajectories for 12 fiber tracts differed significantly between infants with and without ASD. Those with ASD had higher values until 6 months old, and soon after, their values started changing slower than those of neurotypical children. By 24 months, the FA values were lower in those with ASD, therefore demonstrating that ASD infants consistently display atypical characteristics of water diffusion in the brain across development (19). Similarly, another study examined children between 2 and 4 years old and confirmed that the long fibers of neurons of autistic youth are structured differently from the neurotypical children in the same age group. The research team used diffusion-weighted imaging (MRI method measuring diffusion of water molecules) to track the movement of water along white matter. It was found that the measure of water increases with age amongst neurotypical children, but it decreases with age amongst children with ASD (20). A study in 2019 concluded that adolescents and adults with ASD have decreased FA compared to neurotypicals along numerous fiber tracts, while younger children with ASD around the ages of 30-40 months have increased levels of white matter FA. These neurodivergent preschoolers had increased measures of FA in eight clusters including regions of the genu, body, and splenium of the corpus callosum, inferior frontal-occipital fasciculi, inferior and superior longitudinal fasciculus, middle and superior cerebellar peduncles, and corticospinal tract, which are all parts of the brain that connect other regions (21). The WM in those with ASD is more diffused in areas relating to behavior and emotion, including the hypothalamus and hippocampus. Taken together, ASD significantly affects FA values (and thus, water flow in the brain) across key developmental periods.

UNIQUE VULNERABILITIES AND ASSESSMENT CHALLENGES

Autism and pediatric traumatic brain injury (TBI) share several overlapping features, including sensory sensitivities, gastrointestinal issues, learning difficulties, and seizures. These shared symptoms are thought to arise from common biological mechanisms such as neuroinflammation, microglial activation, disrupted connectivity across brain regions, and imbalances in excitatory and inhibitory neural signaling. For individuals with autism, these systems are often already altered as part of their neurodevelopmental profile, so a concussion may further destabilize circuits that are already operating differently. This can result in a more pronounced or prolonged response to brain injury, with symptoms that may not follow the typical progression seen in neurotypical individuals.

Assessment is another area where concussions present unique challenges in autistic populations. Standardized tools like the ImPACT test, which evaluate visual motor speed, reaction time, and symptom reporting, are normed on neurotypical individuals. However, studies have shown that autistic athletes tend to score lower on certain components of these tests, such as visual motor speed, even before experiencing a concussion (22). This makes it difficult to interpret post-injury results, since it's unclear whether score changes reflect the concussion itself or the individual's baseline. Without autism-specific normative data, there is a risk of over- or under-diagnosing concussion severity, which can affect treatment and return-to-activity decisions. Additionally, because many autistic individuals process sensory and motor information differently and may already experience difficulties with coordination and planning, concussions can further strain systems that are already taxed, potentially lengthening recovery time and making symptom management more complex (23).

These biological and functional differences underscore the importance of developing concussion protocols that are tailored to autistic individuals. Recovery plans should take into account the pre-existing variability in motor skills, processing speed, and cognitive function, rather than applying a one-size-fits-all framework (24). Clinicians should be cautious in interpreting symptom severity and duration, recognizing that behaviors such as slower reaction times or greater sensitivity to light and sound may be part of an individual's baseline rather than new post-injury symptoms. Ultimately, a more personalized approach to both assessment and rehabilitation will help ensure that autistic individuals receive accurate diagnoses and effective care, minimizing the risk of prolonged recovery or mismanagement due to misunderstanding their unique neurological profile (25).

DISCUSSION

Concussions have a profound effect on white matter, specifically the FA and MD of it. Concussions typically decrease FA in regions like the corpus callosum and superior corona radiata, which are critical for interhemispheric communication and visuospatial processing, while MD tends to increase in tracts such as the bilateral longitudinal fasciculi, indicating disrupted fiber integrity and potential demyelination. These changes suggest less restricted water diffusion in white matter tracts and increased extra-neurite water volume (the amount of water in the brain tissue outside of the neurites), reflecting disrupted fiber pathways and possible long-term neuroinflammation.

Multiple studies agree that FA reductions and MD increases are reliable markers of concussion-related microstructural damage. For example, Narayana and Drottar both observed FA decreases in the superior corona radiata, while Churchill noted concurrent MD increases in the same regions (7, 9, 10). These converging results suggest a consistent vulnerability of sensorimotor and visual processing areas following mTBI. However, some variation exists in the specific tracts affected, with Maugans focusing more on cerebral blood flow disruptions rather than diffusivity markers, implying that additional physiological changes may also play a role, especially in early stages post-injury (8).

These water volume effects might be even more pronounced in youth with ASD, who often show naturally lower FA and higher MD during critical developmental periods. Studies such as Wolff and Andrews report atypical diffusion patterns in white matter tracts among autistic children, even in the absence of brain injury (19, 15). This raises an important point of contrast: while neurotypical youth exhibit clear post-injury changes in FA and MD, autistic youth may begin with altered baselines, making it difficult to distinguish injury effects from neurodevelopmental traits.

Furthermore, while several studies agree that adolescents and young adults are the cohort most vulnerable to sport-related concussions due to ongoing brain maturation, there is less consensus on the timeline and extent of recovery. Maugans found cerebral blood flow returned to baseline within a month in most children, but Boutin reported that some neurophysiological impairments could persist for up to a year (8, 21). This discrepancy suggests that while physical symptoms may resolve quickly, underlying neurobiological changes might linger undetected, particularly in more vulnerable populations.

Given these concerns, there is a strong case for clinical and research efforts to address this intersection more directly. Clinically, early screening for concussion symptoms in autistic youth should include neuroimaging, cognitive, sensory, and emotional markers, recognizing that presentations may differ from neurotypical individuals. Consensus is growing around the need for autism-specific concussion assessment norms, especially for tools like the ImPACT test. Post-concussion care protocols should be adapted to account for sensory sensitivities, communication differences, and slower recovery trajectories common in ASD.

From a research perspective, there is agreement on the value of longitudinal studies, but few currently track both neuroimaging and behavioral outcomes in ASD youth post-concussion. A key research gap lies in differentiating concussion-related white matter changes from autism-related white matter abnormalities, particularly since both involve disrupted connectivity in frontal and temporal regions. Developing tailored return-to-learn and return-to-play guidelines for this population could reduce long-term harm and support more equitable participation in sports and physical activity.

While there is a growing body of evidence pointing to consistent white matter vulnerabilities and behavioral impairments following concussion, the interpretation of these changes becomes more complex in autistic populations. More work is needed to reconcile baseline differences with post-injury outcomes and to develop interventions that are both inclusive and effective.

Continued research into the long-term effects of concussions on autistic youth holds the promise of uncovering targeted interventions and protective strategies that can support healthier neurodevelopment and improve the quality of life of autistic youth.

CONFLICTS OF INTERESTS

The author declares that there are no conflicts of interest regarding the publication of this article.

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