A Review on the Role of Inflammation in Attention-Deficit/Hyperactivity Disorder

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Abstract

Attention-deficit/hyperactivity disorder (ADHD) is a prevalent neurodevelopmental condition that impairs quality of life in social, academic, and occupational contexts for both children and adults. Although a strong neurobiological basis has been demonstrated, the pathophysiology of ADHD is still poorly understood. Among the proposed mechanisms are glial activation, neuronal damage and degeneration, increased oxidative stress, reduced neurotrophic support, altered neurotransmitter metabolism, and blood-brain barrier disruption. In this way, a potential role of inflammation has been increasingly researched. However, evidence for the involvement of inflammation in ADHD is still scarce and comes mainly from (1) observational studies showing a strong comorbidity of ADHD with inflammatory and autoimmune disorders; (2) studies evaluating serum inflammatory markers; and (3) genetic studies. A co-occurrence of ADHD with inflammatory disorders has been demonstrated in a large number of subjects, suggesting a range of underlying mechanisms such as an altered immune response, common genetics, and environmental links. The evaluation of serum inflammatory markers has provided mixed results, likely due to the small sample sizes and high heterogeneity between biomarkers. However, there is evidence that increased inflammation during the early development may be a risk factor for ADHD symptoms. Although genetic studies have demonstrated a potential role for inflammation in this disorder, there is no clear evidence. To sum up, inflammation may be an important mechanism in ADHD pathophysiology, but more studies are still needed for a more precise conclusion.