

Oxidative stress in attention deficit hyperactivity disorder patients

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Attention Deficit Hyperactivity Disorder (ADHD) is a mental disorder diagnosed by the symptoms of inattention, hyperactivity and impulsivity when the extent of symptoms impairs the child's ability to function. As with many psychiatric disorders, the underlying pathaetiological basis of ADHD remains unknown. Oxidative stress resulting in cell damage, mainly through the production of reactive oxygen species (ROS), has been implicated in various psychiatric disorders. Byproducts of lipid peroxidation as Malondialdehyde (MDA), transition metals as zinc, and levels of Non-Estrified Fatty Acids (NEFA) can be used as indirect biochemical indicators of oxidative stress. Many findings suggest that lipid peroxidation, in particular, may play a central role in the pathophysiology of different mental disorders including ADHD. Aim of the work: To elucidate the possible role of oxidative stress in ADHD patients compared with normal controls through assessing different biochemical markers of disrupted oxidative metabolism. Methods: The study recruited twenty children diagnosed with ADHD using DSM-IV criteria and sixteen matched healthy controls. Both were subjected to comprehensive psychiatric evaluation and the Wisconsin Card Sorting Test to assess cognitive set shifting, planning and conceptualisation. Patients with ADHD were further categorized by Conner's Parent Rating Scale- Revised to recognise the predominant deficit associated with the disorder. Levels of Malondialdehyde (MDA), Zinc, and Non-Estrified Fatty Acids (NEFA) were assessed in both groups. Laboratorial assay for Malonaldehyde was done using spectrophotometric method, while Non Esteified Fatty Acid and Zinc Levels were measured colorimetrically. Results: The current study revealed evidence of increased lipid oxidation with evidence of disturbed lipid peroxidation. The increased malonaldehyde level compared with control (94.1 ± 32.2 , 39 ± 21.9 $p < 0.001$) was correlated to attention /hyperactivity predominantly inattentive. Additionally zinc levels were decreased compared to control (69 ± 16.9 , 95.5 ± 8.8 $p < 0.001$); but the difference was not statistically correlated with any ADHD subtypes. In conclusion, the increased malonaldehyde level as a lipid peroxidation marker and the decreased zinc status and their correlation with the inattention profile are in support of the oxidative stress theory in the pathogenesis of ADHD. This may point to the potential implication of antioxidants as a treatment option for ADHD. Larger scale studies and additional research examining the levels of other non-enzymatic antioxidants as well as the enzymatic antioxidants in children with ADHD are required to verify this possible association.