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# Mitochondrial abnormalities and oxidative stress in Autism: Impact of genetic and environmental factors

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## ABSTRACT

Autism is a neurodevelopmental disorder associated with social deficits and behavioral abnormalities. According to the Centers for Disease Control and Prevention (CDC), 1 in 36 children is affected with autism in the United States. Accumulating evidence suggests that oxidative stress and mitochondrial dysfunction may provide a link between susceptibility genes and pre- and post-natal environmental risk factors in the pathophysiology of autism. The free radicals, i.e., reactive oxygen species (ROS) are generated endogenously during oxidative metabolism and energy (ATP) production by mitochondria. We compared the status of ROS-mediated oxidative damage, glutathione antioxidant status, monoamine oxidase A (MAOA) activity, as well as mitochondrial functions assessed by studying expression and activities of mitochondrial electron transport chain (ETC) complexes and pyruvate dehydrogenase (PDH), as well as mitochondrial biogenesis in postmortem brain tissue samples from the cerebellum and frontal, temporal, parietal and occipital cortices of autistic subjects and age-matched normal subjects. In the cerebellum, frontal cortex, and temporal cortex of subjects with autism, the oxidation of lipid, protein and DNA was increased, glutathione antioxidant defense was impaired, and mitochondrial ETC complexes (I, III and V) and PDH were decreased as compared with age-matched controls. On the other hand, parietal and occipital cortices were unaffected in autism. In addition, MAOA activity was lower that can explain elevated levels of related neurotransmitters such as serotonin in autism. The mitochondrial biogenesis was impaired in the brain of subjects with autism. The high energy demand of the developing brain, thus, may trigger a cascade of structural and functional changes leading to the autistic phenotype if mitochondrial functions are impaired and the energy need of the brain is not fulfilled. In this presentation, the potential role of genetic and environmental factors (such as bisphenol A) in increasing the vulnerability to oxidative stress and mitochondrial abnormalities in autism will also be discussed.