Children with autism are far more likely to have deficits in their ability to produce cellular energy than are typically developing children, reported School of Veterinary Medicine researchers November 30 in the *Journal of the American Medical Association*.

Cecilia Giulivi, professor in the Department of Molecular Biosciences, and fellow researchers found that cumulative damage and oxidative stress in mitochondria, the cell’s energy producer, could influence both the onset and severity of autism, suggesting a strong link between autism and mitochondrial defects.

Deficiencies in the ability to fuel brain neurons might lead to some of the cognitive impairments associated with autism. Mitochondria are the primary source of energy production in cells.

**Multiple Abnormalities**

Giulivi and her colleagues recruited 10 previously identified autistic children aged two to five, and 10 age-matched typically developing children from similar backgrounds.

Mitochondria from children with autism consumed far less oxygen than mitochondria from the group of control children, a sign of lowered mitochondrial activity.

Reduced mitochondrial enzyme function proved widespread among the autistic children.

Levels of pyruvate, the raw material mitochondria transform into cellular energy, also were elevated in the blood plasma of autistic children, suggesting that their mitochondria are unable to process pyruvate fast enough to keep up with the demand for energy.

Hydrogen peroxide levels in autistic children were twice as high as in normal children. As a result, cells of children with autism were exposed to higher oxidative stress.

Mitochondria often respond to oxidative stress by making extra copies of their own DNA. The researchers found higher mtDNA copy numbers in the lymphocytes of half of the children with autism. These children carried equally high numbers of mtDNA sets in their granulocytes, another type of immune cell, demonstrating that these effects were not limited to a specific cell type.

**Earlier Diagnosis?**

“We took a snapshot of the mitochondrial dysfunction when the children were two to five years old. Whether this happened before they were born or after, this study can’t tell us,” Giulivi said. “However, the research furthers the understanding of autism on several fronts and may, if replicated, be used to help physicians diagnose the problem earlier.”

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