Autism, Mitochondrial Disease, & Cognitive Fatigue

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Autism Spectrum Disorder (ASD) Overview

- Challenges with developing relationships, social & communication skills (speech, non-verbal communication), repetitive behaviors.
- Typically identified no later than 3 years of age.
- Many types (spectrum), wide range of cognitive and interpersonal strengths & weaknesses.
- Mitochondrial defects and markers appear in a small subset of patients.
- Co-occurring GI problems, seizure disorders, ADHD, anxiety, obsessions, and phobias. Some ASD children have had regression and some regressive encephalopathy.
- Many different combinations of genetic and environmental factors.
- Current treatments mostly intense behavioral with adjunct medication.
The brain has one of the highest energy expenditures/ATP requirements of any organ. It is particularly vulnerable to mitochondrial dysfunction, and high metabolic areas are most sensitive to such problems, potentially disrupting normal development and functioning of the brain.
Lactate/Pyruvate Ratio and Processing Speed
Sustained Attention

3 above typical range, 5 below

16 above typical range, 2 below

RT

Trials
Understanding the causes and nature of reported “cognitive fatigue”

A “time-related deterioration in the ability to perform certain mental tasks” (De Luca, 2005).

Dysfunction in the striato-thalamo-cortical loop connecting the neo-striatum with the prefrontal cortex (Chaudhuri & Behan, 2004)
Hypothesis: The inability to utilize delivered O2 causes a decrease in cerebral metabolic rate of oxygen (CMRO2), resulting in an up-regulation of local blood flow. The increased blood flow, and decreased CMRO2 results in a greater BOLD response, suggesting some evidence of cognitive fatigue.
Mito and ASD Dimensions and Arterial Spin Labeling Blood Flow Changes Over Time
Opposite relationship b/t ASD and Non-ASD in DLPFC CBF and AD; possible energy crisis?
Thanks to all the Participating Families and Children

Funding from: DOD Idea Award Autism Research Program W81 XWH-10-1-0547
Foundation for Mitochondrial Medicine
GSU Psychology Research Leverage Funds

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