Research proposes common link between autism, diabetes

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A review of the genetic and biochemical abnormalities associated with autism reveals a possible link between the widely diagnosed neurological disorder and Type 2 diabetes, another medical disorder on the rise in recent decades.

"It appears that both Type 2 diabetes and autism have a common underlying mechanism -- impaired glucose tolerance and hyperinsulinemia," said Rice University biochemist Michael Stern, author of the opinion paper, which appears online in this month's issue of *Frontiers in Cellular Endocrinology*.

Hyperinsulinemia, often a precursor to insulin resistance, is a condition characterized by excess levels of insulin in the bloodstream. Insulin resistance is often associated with both obesity and Type 2 diabetes.

"It will be very easy for clinicians to test my hypothesis," said Stern, professor of biochemistry and cell biology at Rice. "They could do this by putting autistic children on low-carbohydrate diets that minimize insulin secretion and see if their symptoms improve."

Stern said the new finding also suggests that glucose tolerance in pregnant women may need to be addressed more seriously than it is now.

Stern said he first realized there could be a common link between Type 2 diabetes and autism a few years ago, but he assumed someone else had already thought of the idea.

Stern's lab, which is located at Rice's BioScience Research Collaborative, specializes in investigating the genetic interactions associated with genetic diseases like neurofibromatosis, a disorder in which patients are several times more likely to be afflicted with autism and autism spectrum disorders (ASD) like Asperger's syndrome.

Autism and ASD are neurological disorders that have a strong but poorly understood genetic basis. The U.S. Centers for Disease Control and Prevention estimates that about nine out of 1,000 U.S. children are diagnosed with ASD.

Stern said at least four genes associated with increased frequency in autism are known to produce proteins that play key roles in a biochemical pathway known as PI3K/Tor. Stern said he had been studying a form of abnormal function in the synapses of fruit flies that was remarkably similar to abnormalities observed in rats and mice with defects in a different pathway known as mGluR-mediated long-term depression.

"I had also spent a lot of time thinking about insulin signaling because another project in my lab is an endocrinology project in which we're studying how key proteins involved in insulin signaling affect the timing of metamorphosis in fruit flies," Stern said.

From his studies in both areas, Stern knew two things: PI3K/Tor was the major pathway for insulin signals within cells, and insulin could affect synapses in a remarkably similar way to the mGluR defects associated with autism.

"When I read that the incidence of autism was increasing, and combined that with the fact that the incidence of Type 2 diabetes is also increasing, it seemed reasonable that each increase could have the same ultimate cause -- the increase in hyperinsulinemia in the general population,"

Stern said. "I didn't do anything with this notion for a few years because it seemed so obvious that I figured everyone already knew this hypothesis, or had tested it and found it was not true."

Stern said he changed his mind a few months ago when a health care consulting firm asked him to provide input about autism.

"In preparing for this interview, I discovered that gestational diabetes was the most important identified maternal risk factor for autism, but that 'no known mechanism could account for this,'" Stern recalled. "When I read this, I was speechless. That's when I realized that this was not obvious to others in the field, so I decided to write this up with the hope that clinicians might become aware of this and treat their patients accordingly."

In writing the article, Stern said he learned that the role of insulin in cognitive function is becoming more widely accepted.

"I was checking to see if insulin was known to affect synaptic function, and I learned that the nasal application of insulin is already being tested to see if it is beneficial for both Alzheimer's and schizophrenia."

Stern said he also found preliminary studies that indicated that low-carb diets were therapeutic for some individuals with autism and ASD.

"Based on what's already in the literature, insulin needs to be taken seriously as a causative element in autism," Stern said. "I hope that clinicians will take the next step and put this to a rigorous test and determine how to best use this information to benefit patients."

More information: The opinion article is available at: www.frontiersin.org/cellular_e … endo.2011.00054/full

Provided by Rice University