Study finds potential solution for feeding, swallowing difficulties in children with autism

February 19 2014

Collaborative research out of the George Washington University (GW) reveals new information on the pathogenesis of feeding and swallowing difficulties often found in children with neurodevelopmental disorders, including autism and intellectual disability. Using an animal model of DiGeorge/22q11 Deletion Syndrome, a genetic disorder that causes autism and intellectual disability, the GW group found clear signs of
early feeding and swallowing disruption, and underlying changes in brain
development. The research, featured on the cover of Disease Models &
Mechanisms, may even lead to a cure for these difficulties—known as
pediatric dysphagia.

"We found that the same mechanisms causing neurodevelopmental
disorders are disrupting development in parts of the nervous system that
control swallowing and feeding," said Anthony-Samuel LaMantia, Ph.D.,
professor of pharmacology and physiology at the GW School of
Medicine and Health Sciences (SMHS) and director of the GW Institute
for Neuroscience. "Cranial nerves, which control food intake and
swallowing, aren't developing correctly, which likely contributes to mis-
coordination. This is good news—this is something we can fix."

Up to 80 percent of children with developmental disorders have
difficulty ingesting, chewing, or swallowing food, leading to food
aspiration, choking, or life-threatening respiratory infections. Despite its
high co-incidence with developmental disorders, little was previously
known about pediatric dysphagia.

"A lot of children with pediatric dysphagia tend to be sicker from birth
onward. Making the health of these kids as stable as possible from birth
onward would allow clinicians to pick up on developmental signs sooner,
which are often masked by more immediate problems like having ear or
respiratory infections, not sleeping or not gaining weight," said
LaMantia. "The physiological stress caused by the complications of
dysphagia early on likely exacerbates the fundamental behavior issues
that will emerge later. A happy, healthy baby is often able to focus on
observing and gathering information to drive important experience
dependent changes in the brain. A sick baby has less time to do so,
possibly making cognitive outcomes even worse."

These findings were a collaborative effort between LaMantia, and Sally
Moody, Ph.D., professor of anatomy and regenerative biology at SMHS, with important contributions from Beverly Karpinski, a research scientist who works jointly with LaMantia and Moody; Thomas Maynard, Ph.D., associate research professor of pharmacology and physiology at SMHS and director of the GW Institute for Neuroscience Biomarkers Core; and Irene Zohn, Ph.D. associate professor of pediatrics and pharmacology and physiology and Investigator in the Center for Neuroscience Research at Children's National Medical Center.

LaMantia's lab had been working on issues surrounding disrupted development from DiGeorge/22q11Deletion Syndrome and Moody's lab had, over the course of her career, been working on issues specific to cranial nerve neurons and how they relate to the development of peripheral neurons and cranial facial targets. The combined expertise led to this discovery and will lead to future collaborations.

**More information:** The study, titled "Dysphagia and Disrupted Cranial Nerve Development in a Mouse Model of DiGeorge (22q11) Deletion Syndrome," is available at dmm.biologists.org/content/7/2/245.abstract.

Provided by George Washington University


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