

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

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The research was featured on the cover of *Disease Models & Mechanisms*. Credit: *Disease Models & Mechanisms* 

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 <u>neurodevelopmental</u> <u>disorders</u> 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 miscoordination. 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 <u>dmm.biologists.org/content/7/2/245.abstract</u>.

## Provided by George Washington University

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