



Paths to Autism: One or Many?

Answers from a new study in Biological Psychiatry

Philadelphia, PA, July 19, 2016 – A new [report](#) in [Biological Psychiatry](#) reports that brain alterations in infants at risk for autism may be widespread and affect multiple systems, in contrast to the widely held assumption of impairment specifically in social brain networks.

Autism is diagnosed based on impairments in social and communication behaviors. These symptoms tend to emerge in the second year of life, but identifying abnormalities in early infancy could help researchers understand how autism develops and potentially allow clinicians to predict the disorder before it emerges.

Attempts to identify precursors have primarily focused on social behaviors, based on the assumption that abnormalities in social brain networks arise early in life and compound throughout development. But Dr. Mayada Elsabbagh from McGill University in Canada, and Dr. Mark Johnson, from Birkbeck, University of London, suggest that recent studies do not support the idea of a singular pathway in the development of autism.

In their synthesis of studies examining infants at risk for autism, Elsabbagh and Johnson highlight behavioral research providing evidence for general abnormalities during the first year of life. These include delayed motor maturation, higher level of perceptual sensitivity, and poor attention flexibility. The authors also highlight brain imaging studies that provide evidence for widespread alterations throughout brain networks, rather than focal deficits in social networks.

The behavioral and imaging studies challenge the assumption of early social network abnormalities that persist throughout development and lead to emergence of the disorder.

“Our review reveals little support for localized deficits in social brain network systems within the first year of life,” said Elsabbagh. “It instead favors the view that atypical development involving perceptual, attentional, motor, and social systems precede emerging autism and lead to overt behavioral symptoms by the second year.”

The review suggests that focusing on a single deficit may not be sufficient to identify early warning signs and will likely adjust how researchers conceptualize the disorder.

“There has been a concerted effort to identify the final common neural pathways underlying symptoms and deficits for psychiatric disorders,” said Dr. John Krystal, Editor of *Biological Psychiatry*. “Yet the perspective shared by Elsabbagh and Johnson suggests that there are widespread disturbances in brain development in autism spectrum disorder and that the prominent social deficits either reflect the fact that circuits underlying social behaviors are among the many circuits affected or that some functional deficits are emergent properties of multiple affected circuits.”

Notes for editors

The article is "Autism and the Social Brain: The First-Year Puzzle," by Mayada Elsabbagh and Mark H. Johnson (doi: [10.1016/j.biopsych.2016.02.019](https://doi.org/10.1016/j.biopsych.2016.02.019)). It appears in *Biological Psychiatry*, volume 80, issue 2 (2016), published by [Elsevier](#).

Copies of this paper are available to credentialed journalists upon request; please contact Rhiannon Bugno at +1 214 648 0880 or biol.psych@utsouthwestern.edu. Journalists wishing to interview the authors may contact Mayada Elsabbagh at mayada.elsabbagh@mcgill.ca.

The authors' affiliations, and disclosures of financial and conflicts of interests are available in the article.

John H. Krystal, M.D., is Chairman of the Department of Psychiatry at the Yale University School of Medicine, Chief of Psychiatry at Yale-New Haven Hospital, and a research psychiatrist at the VA Connecticut Healthcare System. His disclosures of financial and conflicts of interests are available [here](#).

About *Biological Psychiatry*

Biological Psychiatry is the official journal of the [Society of Biological Psychiatry](#), whose purpose is to promote excellence in scientific research and education in fields that investigate the nature, causes, mechanisms and treatments of disorders of thought, emotion, or behavior. In accord with this mission, this peer-reviewed, rapid-publication, international journal publishes both basic and clinical contributions from all disciplines and research areas relevant to the pathophysiology and treatment of major psychiatric disorders.

The journal publishes novel results of original research which represent an important new lead or significant impact on the field, particularly those addressing genetic and environmental risk factors, neural circuitry and neurochemistry, and important new therapeutic approaches. Reviews and commentaries that focus on topics of current research and interest are also encouraged.

Biological Psychiatry is one of the most selective and highly cited journals in the field of psychiatric neuroscience. It is ranked 5th out of 140 Psychiatry titles and 11th out of 256 Neurosciences titles in the Journal Citations Reports® published by Thomson Reuters. The 2015 Impact Factor score for *Biological Psychiatry* is 11.212.

About Elsevier

[Elsevier](#) is a world-leading provider of information solutions that enhance the performance of science, health, and technology professionals, empowering them to make better decisions, deliver better care, and sometimes make groundbreaking discoveries that advance the boundaries of knowledge and human progress. Elsevier provides web-based, digital solutions — among them [ScienceDirect](#), [Scopus](#), [Elsevier Research Intelligence](#) and [ClinicalKey](#) — and publishes over 2,500 journals, including *The Lancet* and *Cell*, and more than 35,000 book titles, including a number of iconic reference works. Elsevier is part of [RELX Group](#), a world-leading provider of information and analytics for professional and business customers across industries. www.elsevier.com

Media contact

Rhiannon Bugno
Editorial Office, *Biological Psychiatry*

+1 214 648 0880

biol.psych@utsouthwestern.edu