UNIVERSITÉ DE GENÈVE

PRESS RELEASE

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Autism: the neural origin of the social bound

UNIGE scientists identified a brain circuit where lies the origin the social difficulties experienced by people with autism spectrum disorders.

From birth, human survival depends on the ability to engage with others. This ability, which is essential for development, seems to be impaired very early on in children with autism spectrum disorders (ASD), who show limited interest in social stimuli from their first year of life. To understand the neurobiological basis of this phenomenon, scientists at the University of Geneva (UNIGE) combined data from clinical and animal research. They identified a defect in a communication pathway between two brain structures that prevents rapid redirection of attention, a key mechanism for decoding social interactions. These results, published in the journal *Molecular Psychiatry*, pave the way for better prediction of development and more targeted interventions.

It is currently estimated that one child in 36 develops an autistic disorder, of whom a third is at risk of cognitive impairment. “In the children who show a delay, the cognitive difficulties are the consequence of a lack of understanding of social interactions,” explains Camilla Bellone, Associate Professor in the Department of Basic Neurosciences at the UNIGE Faculty of Medicine, and co-last author of the study. “We learn through interaction with others. As young children with ASD are less oriented towards social cues very early on, they are less likely to develop the tools that enable them to navigate the social world and learn.” While the consequences of this lack of social interest on development are well known, the neurobiological causes are much less so.

Studying brain networks with mouse models

At the UNIGE Faculty of Medicine, the Synapsy Centre for Research in Neuroscience for Mental Health brings together neuroscientists and psychiatrists in a joint network. This sharing of expertise has led to a major discovery for understanding the very essence of social interaction: the ability to maintain a social interaction depends on the speed with which attention can be shifted from one stimulus to another.

High resolution pictures

“In mice lacking the Shank3 gene – the most common single gene cause of ASD found in humans - we observe orientation deficit towards other mice, which reflects the alterations in social interactions already described in children with ASD. These mice therefore represent a good model for the study of ASD,” explains Marie Schaer, Associate Professor in the Department of Psychiatry at the UNIGE Faculty of Medicine and co-last author of the study.

Impaired neuronal synchronisation

In previous research, Camilla Bellone's team identified a neuronal communication pathway whose role is to send information between the superior colliculus, a brain structure linked to orientation, and particularly to social orientation, and the ventral tegmental area, linked to the reward system. "This time, we were able to show in our mouse model of ASD that a lack of neural synchronisation in the superior colliculus altered the exchange of communication between the two cerebral areas, resulting in defects in the orientation and social behaviour of individuals". These experiments were carried out in vivo using miniaturised microscopes that enable the monitoring of neural activity in moving animals. They were conducted by Alessandro Contestabile, co-first author of the study and a post-doctoral researcher in Camilla Bellone's laboratory.

A specific protocol for ASD children

To confirm this hypothesis in humans, Nada Kojovic, a researcher in Marie Schaer's team and co-first author of the study, developed an original protocol for obtaining brain MRIs without sedation in children aged 2 to 5 years. "It is obviously impossible to ask such young children to remain motionless in the MRI scanner for the 30 minutes needed to obtain the images," she explains. "We therefore developed a habituation protocol, fitted out the MRI room and worked closely with the families to provide optimum conditions for the child to fall asleep, which worked very well for over 90% of the children for whom we obtained very good quality MRI images."

The two teams observed that the circuit changes identified in mice were identical in the children. Furthermore, the level of connectivity in this circuit makes it possible to predict their cognitive development in the following year. While it is not yet possible to intervene directly on this brain network, this discovery provides a guide to behavioural interventions, in particular to reinforce children's ability to redirect their attention from one thing to another rapidly from an early age. An intensive treatment method developed in the United States and used in Geneva which require 20 hours a week for 2 years, has already proved its worth. With early intervention, the children gain an average of 20 IQ points, and 75% of them can go on to attend ordinary school.

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contact

Marie Schaer
Associate Professor
Department of Psychiatry
Faculty of Medicine
UNIGE
+41 22 379 12 30
marie.schaer@unige.ch

Camilla Bellone
Associate Professor
Department of Basic Neuroscience
Faculty of Medicine
UNIGE
+41 22 379 54 33
camilla.bellone@unige.ch

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UNIVERSITÉ DE GENÈVE
Communication Department
24 rue du Général-Dufour
CH-1211 Geneva 4

Tel. +41 22 379 77 17

media@unige.ch
www.unige.ch