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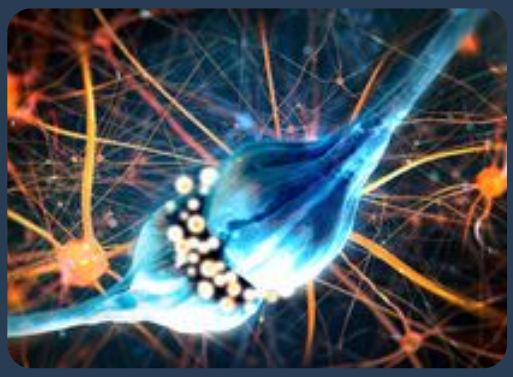
Therapeutic Target for Neurodevelopmental Disorders

Modulation of the mitochondrial transporter, Aralar, governs GABA level and social behaviour

Request an introduction



Reference: IDF 11-19

Source: solvod, https://stock.adobe.com/uk/252082985, stock.adobe.com

IP Status

Patent application submitted

Seeking

Licensing, Development partner, University spin out

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Background

Social impairment is frequently associated with mitochondrial dysfunction and altered neurotransmission. Although mitochondrial function is crucial for brain homeostasis, this has not yet been exploited to develop therapies for neurodevelopmental disorders.

Tech Overview

Using CYFIP1 deficiency, a key interactor in Fragile Mental Retardation with a drosophila model, researchers have demonstrated alterations in mitochondrial metabolism that causes observed social behavior deficits.

Increased mitochondrial activity causes GABA sequestration in the mitochondria, reducing GABAergic signaling and resulting in social deficits.

Pharmacological and genetic manipulations of mitochondrial activity or GABA signaling corrects the observed abnormalities. This could be done by modulating the newly discovered mitochondrial transport, Aralar, that sequesters GABA upon increased mitochondrial activity.

Further Details:

- https://linkinghub.elsevier.com/retrieve/pii/S009286742030221X (Cell, 2020).

Stage of Development

Technology Readiness Level (TRL): 3.

Benefits

This newly discovered GABA transporter has not yet been exploited as a target for drug discovery to treat social impairment. There is tremendous potential for drug screening using this target.

Applications

Therapeutic target for neurodevelopmental disorders such as:

- Autism spectrum disorder
- Schizophrenia
- Fragile X syndrome

Opportunity

PACTT offers to grant exclusive or non-exclusive license to industrial partners able to develop and commercialize the technology.

Patents

- WO2021176098

Learn more about this opportunity

About IN-PART

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