Short Communication
Volume 11:10, 2022

Journal of Trauma & Treatment

ISSN: 2167-1222 Open Access

A Short Communication on Neurodevelopmental Disorders

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Introduction

Numerous neurodevelopmental illnesses, including Rett Syndrome, schizophrenia, and Autism Spectrum Disorders (ASDs), have endosomal trafficking disruption as a common molecular route (RTT). Within endosomal organelles such the Golgi, early endosomes, recycling endosomes, and lysosomes, endosomal trafficking controls the transit of vesicles from the donor membrane to the acceptor membrane. By GTPases and their activating proteins, coat proteins like Adaptor Protein 3 (AP3), the biogenesis of lysosome related organelles complex 1 (BLOC-1), and SNARE complexes that are particular to that organelle, trafficking within the endosomal pathway is controlled at the organelle. Endosomal trafficking is essential for correct dendritic spine expansion, synaptic development, and receptor recycling within of a neuron [1].

Description

Due to abnormal central nervous system development, neurodevelopmental diseases affect learning, emotion, cognition, communication, and communication. Speech, learning and memory, sensory processing, mobility, loss of efficient hand usage, social anxiety, sleep difficulties, and seizures are all symptoms of RTT, a neurodevelopmental condition. RTT was formerly classified as an ASD (DSM IV) due to common characteristics [2-4]. Language treatment can help with nonverbal communication, and physical and occupational therapy can enhance movement, balance, and intentional hand use. Some RTT symptoms, including as breathing, sleep, GI, and cardiac issues, can be treated with medication. However, because we still don't fully comprehend how RTT affects brain function during development, there is still a gap in our ability to significantly improve the quality of life for patients with RTT.

One of the earliest studies to show that RTT was brought on by MECP2 mutations. The MECP2 gene has since been linked to more than 60 mutations that cause RTT, the majority of which occur de novo at CpG dinucleotides. The particular cellular and molecular mechanisms underlying the synaptic abnormalities seen in RTT are still poorly understood, despite the identification of mutations. Short PJ, et al. [5] examined the transcripts changed in the hypothalamus of MeCp2 defective mice in order to better understand the function of MeCP2. They discovered that Mecp2 acts as a transcriptional activator or repressor in the hypothalamus to control the expression of approximately 2,000 genes. Endosomal trafficking mRNAs, such as the Arf GAP, Arfgap, coat and coat-associated proteins COG, Gga2, Adaptor Protein 1 (AP-1), Adaptor Protein 2 (AP-2) subunits, palladin, and cappuccino (subunits of the BLOC-1 complex), were among the 2,000 mRNAs that were changed. Studies in mouse models have also indicated a potential role of endosomal trafficking in RTT pathophysiology, in addition to the mRNA evidence that suggests that endosomal trafficking may be disrupted in RTT. The absence of synaptic plasticity in Mecp2-deficient neurons has been shown to be caused by low amounts of EEA1, an early endosome producer that docks, fuses, and recycles vesicles [6].

Similar to RTT, SZ is a neurodevelopmental condition that is characterised by speech, learning, and memory deficits as well as sensory processing, disordered or aberrant motor movement, social processing, sleep abnormalities, and seizures. Apathy, depression, disordered thinking, and hallucinations are further symptoms of SZ. Endosomal proteins, particularly BLOC-1 subunits, have also been linked to SZ in genome-wide association studies of patients [7]. The DTNBP1 gene is one of the most important genes linked to SZ risk, despite the fact that there is no one genetic factor that causes SZ. Dysbindin, an octameric protein subunit of BLOC-1, is encoded by the DTNBP1 gene. Palladin, Snapin, Cappuccino, Muted, Blos-1, and Dysbindin make up this octameric complex. In the post-mortem tissue of SZ patients, dysbindin levels in the hippocampus have been found to be lower [8].

The recruitment of the coat and coat-associated proteins from the cytosol, which starts the curving of the donor membrane to form a vesicle, is what drives the production of endosomal vesicles. An early endosome protein called BLOC-1 is connected with the endosomal coat. It is in charge of moving vesicles to the lysosome from the endosome or, in the case of neurons, synaptic vesicles destined for the axon terminal [2]. Endosomal trafficking may have a role in SZ, as shown by the failure of BLOC-1 dependent cargo to reach the axon terminal in mice lacking certain BLOC-1 subunits. We examined the protein expression of important indicators as well as endosomal dynamics in models of RTT and SZ in order to better comprehend the role of endosomal trafficking in neurodevelopmental disorders.

Conclusion

Each neurodevelopmental illness had unique sub-endosomal compartment abnormalities, which correspond to potential sites of phenotypic changes or phenotypic severity.

Acknowledgement

Not applicable.

Conflict of Interest

There is no conflict of interest by the author.

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Received: 02 October, 2022, Manuscript No. JTM-22-83674; **Editor assigned:** 04 October, 2022, PreQC No. P-83674; **Reviewed:** 17 October, 2022, QC No. Q-83674; **Revised:** 21 October, 2022, Manuscript No. R-83674; **Published:** 28 October, 2022, DOI: 10.37421/2167-1222.2022.11.532

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How to cite this article: Dang, Jing and Yo Tia. "A Short Communication on Neurodevelopmental Disorders." J Trauma Treat 11 (2022): 532.