Proceedings of *Autism Challenges and Solutions XII International Research and Practice Conference*, ABU-DHABI UAE, 27-30th April 2024. Published and Indexed by Emirates Scholar Center for Research and Studies.

Zinc Deficiency and Autism - From cellular mechanisms to clinical studies - an update

Andreas M. Grabrucker
andreas.grabrucker@ul.ie
Cellular Neurobiology and Neuro-Nanotechnology lab, Dept. of Biological Sciences, University of Limerick, Limerick, Ireland

ARTICLE INFO

Published on 7th of June 2024.
Doi: 10.54878/r4dyxe73

KEYWORDS

Zinc, Zinc Deficiency, Neurodevelopmental Disorders, Gene Expression

HOW TO CITE

Zinc Deficiency and Autism – From cellular mechanisms to clinical studies – an update. (2024). *Autism Challenges and Solutions*, *2*(1). https://doi.org/10.54878/r4dyxe73

ABSTRACT

Zinc is a versatile element crucial in various biological processes, including gene expression, cell division, and enzymatic reactions. In recent years, zinc has gained increasing attention as a potential regulator of healthy brain development. Zinc deficiency, in turn, was linked to neurodevelopmental disorders like autism spectrum disorder (ASD). Our studies showed that genetic and environmental factors, such as prenatal zinc deficiency, seem to converge on a neurobiological process determining the characteristic ASD-linked behaviors. One critical convergence point is synaptic vesicle release and recycling. This process is influenced by gut-brain signaling. We could show that compromised gastrointestinal barrier tightness (leaky gut) is zinc status-dependent and that pro-inflammatory processes in the gut trigger neuroinflammation. This talk summarizes our most recent findings on how zinc deficiency contributes to abnormal brain function via the microbiota-gut-brain axis mechanistically at cellular and organ levels. In particular, astrocyte activation by zinc deficiency is a novel finding that will be highlighted. Further, new data from recent clinical trials using zinc supplementation in pregnant women will show that findings from cell and animal models can be translated into human studies, with the effects of zinc supplementation on microbiota and inflammatory status in participants. Thus, for the first time, we can span the bridge from the cellular functions of zinc to its effects on human health in the context of ASD.