The Autism Neurological Disorders Caused by Food Allergy

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Statement of the problem: It was observed by our studies that autism often establishes itself as a disease in normal patients with adequate psychomotor development and without previous neurological conditions, but with FA preceding the neurological deficits. Primary inflammation and infections of the CNS cause inflammation in neurons and have the potential to make the CNS the "homing" site or target organ, attracting the circulating lymphocytes and immunoglobulins involved in the process of FA. The clinical manifestations of FA in the CNS may, therefore, cause the individual to present within the autism spectrum disorder, clinically varying according to the affected area and the extent of allergic aggression towards this system.

Material and Methods: We included in this study 132 patients with previous diagnosis of ASD, attended at our unit of outpatient clinic of Food Allergy and Autism at Santa Casa da Misericordia do Rio de Janeiro. The diagnosis of FA was made through the score obtained from the anamnesis of each patient. The inflammation of the CNS and glutamate as a neurotransmitter in the brain was detected by studies with MR with spectroscopy and diffusion. We collect information by reviewing the medical records, characterizing this study as a retrospective cross-sectional study. The Ethics and Research Committee approved this research project under number CAAE 66813917.0.0000.5283. The Free and Informed Consent Term is in accordance with resolution number 466 of December 12, 2012, of National Health Council, on research involving human beings.

Findings: The presence of neurons with inflammation in the CNS in patients with FA favors homing of the immune disturbance to local neurons. This causes neurological disorders favoring the development of autism secondary to food allergy. These mechanisms include multilevel pathways in the gut–brain axis contributing to alterations in behavior and cognition. Recent studies have shown that pathogenetic factors and pathophysiological mechanisms can link ASD and GI disturbances through intestinal inflammation and dysregulation of the gut microbiome.

Conclusion: We hypothesized that FA is one of the foregoing factors in patients who develop ASD, if they suffer from inflammation of the central nervous system (CNS). This inflammatory injury may turn neurons the target organ or the FA homing site once the brain-gut connection is established by different mechanisms.

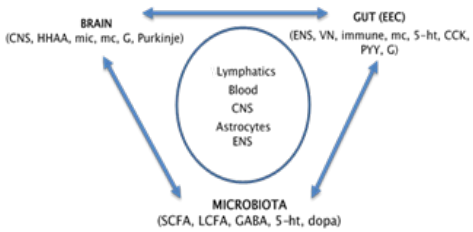

Figure: The brain-gut-microbiome axis

BRAIN: CNS- central nervous system; HHAA- hypothalamus-hypophysis-adrenal-axis; mic- microglia; mc- mast cell; G- glutamate; Purkinje. GUT: ENS- enteric nervous system; VN- vagal nerve; immune- immune system; mc- microglia; 5-ht- serotonin. EEC- enteroendocrine cells: CCH-cholecystokinin; PYY- peptide YY. MICROBIOTA: SCFA- short chain fatty acid; LCFA- long chain fatty acid; GABA- gamma amino butyric acid; 5-ht-serotonin; dopa- dopamine.

Speaker Biography

Aderbal Sabra is a member of the Brazilian Academy of Medicine. He is the professor of Pediatric Gastroenterology, Food Allergy, and Autism at Santa Casa da Misericordia do Rio de Janeiro, Brazil. He is the author of the book Food Allergy, which is currently in its Fourth Edition. In the past five years, his primary research efforts and publications were related to the association of autism neurological disorders caused by food allergy.

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