IJAE
Vol. 126, n. 1 (Supplement): 274, 2022
ITALIAN JOURNAL OF ANATOMY AND EMBRYOLOGY

Melatonin supplementation in Autistic BTBR T^{+}Itpr3^{tf}/J Mice: goblet cells and sodium glucose transporter as new targets in autism spectrum disorder

Caterina Franco[1], Riccardo Guarneri[1], Francesca Bonomini[1,2,3], Rita Rezzani[1,2,3]

[1]Anatomy and Physiopathology Division, Department of Clinical and Experimental Sciences, University of Brescia, 25123 Brescia, Italy
[2] Interdipartimental University Center of Research "Adaption and Regeneration of Tissues and Organs-(ARTO)", University of Brescia, 25123 Brescia, Italy
[3] Italian Society of Orofacial Pain (SISDO), 25123 Brescia, Italy

Autism spectrum disorder (ASD) identifies a neurodevelopmental syndrome characterized by a complex etiology. It primarily affects the brain, but actually 5 to 80 % of the autistic patients suffer from gastrointestinal (GI) symptoms and the existence of a link between GI diseases and ASD has already been demonstrated. In this sense, fundamental are the morphological alterations that affect the GI tract of autistic patients and the imbalance between the physiological condition and the inflammatory/stressed one [1]. Moreover, also the goblet cell's (GC) content and the expression of sodium glucose transporter (Sglt-1 and -3) undergo changes in mouse model of autism [2]. Starting from this background, the aim of this work was to evaluate the role of melatonin's (MT) supplementation in the gut of BTBR mice. These mice are considered a good ASD-like model because they present behavioral and psychological alterations like those observed in patients with ASD. On the other side these mice showed also the typical autistic metabolic pattern, showing insulin resistance, diabetes- induced nephropathy and phenylketonuria [3].

Considering the important role of MT as antioxidant and considering the role that GC and Sglt-1 and -3 have in ASD pathogenesis, we want to understand whether restoring the oxidative balance in the autistic patient can also have an impact on the gut's morphology and functionality [4].

Gut morphology and GC's content has been evaluated by histological staining, inflammation processes have been expressed considering some markers, such as SOD and CAT and Sglt-1 and -3 expression has been studied by immunohistochemical and quantitative analyses.

Our data showed that: 1) GI tract from autistic mice presented an alteration in GC's content and in the number of inflammatory cells; 2) the morphological changes are linked to functional alterations, represented by the different Sglt's expression; 3) MT supplementation could work on the stressed background restoring an oxidative balance and improving the clinical GI manifestations.

Despite these starting results, further experimental researches are needed to better evaluate the molecular mechanisms involved in GI diseases in order to protect the gut morphology and function reducing the number and severity of comorbidities that autistic patients often present.

References

[1] Vellingiri, B.; Aishwarya, S.Y.; Benita Jancy, S.; Sriram Abhishek, G.; Winster Suresh Babu, H.; Vijayakumar, P.; Narayanasamy, A.; Mariappan, S.; Sangeetha, R.; Valsala Gopalakrishnan, A.; Parthasarathi, R.; Iyer, M. An anxious relationship between Autism Spectrum Disorder and Gut Microbiota: A tangled chemistry? *J Clin Neurosci*. **2022**; 99:169-189.