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SESSION VI
PERIPHERAL SYSTEM AND GUT-BRAIN AXIS

TARGETING GUT-BRAIN AXIS IN ALZHEIMER'S DISEASE

D'Antongiovanni V[1], Segnani C[1], Pierucci C[1], Di Salvo C[2], D'Amati A[3], Errede M[3], Virgintino D[3], Antonioli L[2], Pellegrini C[1], Bernardini N[1]

[1]Unit of Histology and Medical Embryology, Department of Clinical and Experimental Medicine, University of Pisa; [2]Unit of Pharmacology and Pharmacovigilance, Department of Clinical and Experimental Medicine, University of Pisa; [3]Department of Basic Medical Sciences, Neuroscience, and Sensory Organs, University of Bari School of Medicine, Bari, Italy

Several studies highlighted the relevant role of microbiota-gut-brain (MGB) axis in the pathophysiology of Alzheimer's disease (AD) and related intestinal symptoms. In this context, the modulation of gut microbiota is emerging as a suitable additional therapeutical option, targeting MGB axis, to halt or slow down the cognitive impairment and intestinal symptoms associated with AD. The aim of the present study was to evaluate the putative beneficial effect of mixture of probiotics (MP) in counteracting central and peripheral morphological and functional alterations in a spontaneous murine model of AD. Senescence-accelerated mouse prone 8 (SAMP8) mice (4 months old) and control SAMR1 strain were treated orally with MP $1X10^9$ CFU/mouse/day or placebo for two months (up to 6 months of aged) to evaluate the effects of probiotics during the earliest stages of AD before the full development of brain pathology. Cognitive functions and *in vitro* colonic motility were assessed. Then, the following parameters were evaluated by immunohistochemistry and ELISA techniques: 1) brain and colonic interleukin (IL)-1β levels; 2) alterations of the intestinal epithelial barrier (plasma LBP levels, acid/neutral mucin ratio and claudin-1). SAMP8 mice showed cognitive impairment and colonic dysmotility as well as an increase in brain and colonic IL-1β. In addition, SAMP8 animals displayed an increase in plasma LBP levels and intestinal acid mucins along with a reduction in intestinal claudin-1, as compared with SAMR1 mice. Intake of MP counteracted cognitive impairment, colonic dysmotility, the increase in central and peripheral IL-1β levels in SAMP8 mice. MP also restored colonic acid mucins and reduced plasma LBP levels.
In conclusion, the MP alleviates cognitive decline and restores colonic motility, by preventing gut barrier impairments and decreasing gut and brain inflammation in AD mice in the prodromal phases of the disease, via MGB axis. Therefore, dietary supplementation with MP can represent a useful therapeutic approach to counteract the morphological and functional alterations underlying neurodegeneration and intestinal dysfunction associated with AD.

MORPHOLOGICAL MODULATION OF COLONIC ENTERIC PLEXI IN MICE MODEL OF COLITIS AND THE POSSIBLE EFFECTS OF BACTERIAL STRAIN SUPPLEMENTATION

Bellitto V[1], Gabrielli MG[2], Martinelli I[1], Roy P[1], Salvesi C[2], Silvi S[2], Galosi L[2], Miceli C[2], Tayebati SK[1], Tomassoni D[2]

[1]School of Medicine and Health Products, University of Camerino; [2]School of Biosciences and Veterinary Medicine, University of Camerino, Italy

Inflammatory bowel diseases (IBD) are gastrointestinal disorders associated with altered intestinal permeability, which causes a degeneration of the enteric plexi of the enteric nervous system (ENS). Treatments for IBD show poor efficacy. Many studies have identified probiotic supplementation as a possible method for alleviating clinical symptoms. The potential properties of Pediococcus acidilactici 46A (Pa) were evaluated on a murine model of Dextran sulfate sodium (DSS)-induced colitis as models of chemically induced IBD. Colitis was induced in 8-week-old mice, administering 2.5% (w/v) DSS in drinking water for 7 days. Pa was supplemented orally (1×108 CFU daily) for 10 days before DSS administration. General conditions, body weight loss, stool characteristics, and occult blood were monitored to evaluate the clinical progression of colitis. Histological damage, neurodegeneration, and pro-inflammatory cytokines expression were detected on proximal and distal colon sections and the histological index scoring was evaluated. Pa in the pretreated mice was able to reduce the colitis severity while not affecting weight loss, compared to the DSS group. Defects of enteric glial cells (EGCs) function and localization, barrier integrity dysfunction, and immune cell infiltrations were observed in colitis-induced groups and a positive improvement was evidenced in Pa-supplemented groups. Morphological modification of neurons of the myenteric plexus was assessed by evaluating HuC/D pan-neuronal marker, then colonic nitrergic and cholinergic pathways were focused, and a neurodegeneration was appreciated in DSS-mice. These results demonstrate that Pa seems to counteract colonic mucosal degeneration and neuronal alteration. However, further studies are needed to demonstrate the use of specific bacterial strains to manage intestinal disorders and correlated ENS modulation in IBD.

PERIPHERAL NERVE STIMULATION PRESERVES PERIPHERAL NERVE INJURY INDUCED MORPHOLOGICAL CHANGES IN THE TRIGEMINAL SYSTEM

Korai SA[1], Panetsos F[2], Papa M[1], Cirillo G[1]

[1]Division of Human Anatomy, Neuronal Morphology Networks & Systems Biology Lab, Department of Mental and Physical Health and Preventive Medicine, University of Campania "Luigi Vanvitelli, Naples, Italy; [2]Neurocomputing & Neurorobotics Research Group, Universidad Complutense de Madrid, Spain

Peripheral nerve injury (PNI) triggers a complex cascade of neurobiological events within the somatosensory system, the full extent of which remains to be fully elucidated. Previous studies have primarily focused on specific components of the ascending system, with limited attention given to comprehensively exploring the entire somatosensory system and understanding the morpho-