



MNS
MNS2023 Tunisia
Carthage, 14-18 October 2023

**9th Conference of the Mediterranean
Neuroscience Society**



PROGRAMME

MNS2023, Carthage, Tunisia, 14th - 18th October

The neuroprotective effects of pituitary adenylate cyclase-activating polypeptide(PACAP) have been shown in numerous in vitro and in vivo models of Parkinson's disease(PD) supporting the theory that PACAP could have an important role in the pathomechanism of the disorder affecting mostly older patients. Earlier studies found changes in PACAP levels in neurological disorders, therefore, the aim of our study was to examine PACAP in plasma samples of PD patients. Peptide levels were measured with ELISA and correlated with clinical parameters e.g. age, stage of the disorder based on the Hoehn and Yahr (HY) scale, subtype of the disease, treatment and specific scores measuring motor and non-motor symptoms, such as Movement Disorder Society-Unified Parkinson's Disease Rating Scale(MDS-UPDRS), Epworth sleepiness scale(ESS), Parkinson's disease sleep scale(PDSS-2) and Beck-Depression Inventory(BDI). Our results showed significantly decreased PACAP levels in PD patients without deep brain stimulation (DBS) therapy and in akinetic-rigid subtype, additionally we also described further decrease in the HY stage 3 and 4. Elevated PACAP levels were found in patients with DBS. There were no significant correlations in PACAP level with MDS-UPDRS, type of pharmacological treatment, PDSS-2 sleepiness and depression (BDI) scales, but we found increased PACAP level in patients with more severe sleepiness problems based on the ESS scale. Based on these results we suggest that following the alterations of PACAP with other frequently used clinical biomarkers in PD patients might improve strategic planning of further therapeutic interventions and help to provide a clearer prognosis regarding the future perspective of the disease.

P44 A new potential therapeutic target in inflammatory retinopathy

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Retinal inflammation may lead to visual impairment, even blindness in most severe cases. Pituitary adenylate cyclase activating polypeptide (PACAP) is a neuropeptide, which has strong neuroprotective and general cytoprotective effects. It can also be found in the eye and it is involved in several ocular processes. Three receptors can be distinguished, however the specific PAC1 receptor plays the key role in its protective mechanisms. Our aim is to investigate the effectiveness of a specific, exogenous PAC1 receptor agonist agent, maxadilan, in inflammatory retinopathy. Inflammation was induced by bacterial lipopolysaccharide in mice. Maxadilan was administered by intravitreal injection. Optical coherence tomography was used to follow the changes in thickness of all retinal layers. Change of ganglion cell

number was evaluated after toluidine blue staining. Electroretinography provided functional information. Expression level of forty different types of cytokines was also analyzed. Our data show that maxadilan is able to prevent the decrease of the outer nuclear layer, outer plexiform layer, inner nuclear layer, inner plexiform layer and the photoreceptor layer. In addition, it improves the functional outcome. Significant ganglion cell degeneration was observed in inflamed group. However, ganglion cell number remained similar to control group after maxadilan treatment. Based on our results, PAC1 receptor-mediated signaling pathways significantly influence the level of several cytokines and chemokines. The specific, exogenous PAC1 receptor agonist maxadilan prevents the morphological and functional damage in inflammatory retinopathy. Based on our results PAC1 receptor is a new possible therapeutic target in this disease.

P45 Octadecaneuropeptide promotes the migration of astrocyte via ODN metabotropic receptor and calcium signaling pathway

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Background: Astrocytes specifically synthesize and release endopeptides, a family of regulatory peptides, including the octadecaneuropeptide (ODN). ODN promotes proliferation and prevents oxidative damage induced cell death apoptosis on both neurons and astrocytes. However, little is known regarding the effects of ODN on neuronal cell migration. Migration is an essential characteristic of cells that occurs during many physiological and pathological processes. The purpose of the present study was to investigate the potential effect of ODN in induction of astrocytes migration, which is critical for formation of neurons during development and maintaining brain homeostasis

Materials/Methods To investigate the effect of ODN on cell migration, we used the wound healing technique. Then the wound edges were remicrographed 48 after treatment. Images of migratory cells from the scratched boundary were observed at 48h with different concentrations of ODN and using an inverted microscope. We measured astrocyte cells viability following injury with the fluorescein diacetate (FDA) and the thiazolyl blue tetrazolium blue (MTT) Assay with different concentrations of ODN. We therefore investigated the effect of ODN on intracellular Ca²⁺ levels. Finally, we tried to provide a new insight into the effect of ODN on mTOR gene expression for cell migration through RT-qPCR (luna universal One –step RT-qPCR kit) and data were corrected using the reference gene GAPDH.

Results: In the wound healing assay, the representative photomicrograph images of cultured astrocytes showed that administration of graded concentrations of ODN (10⁻¹⁶ to 10⁻⁸ M)