INTERNATIONAL SYMPOSIUM OF EXPERTS 2020

Under the patronage of
Her Highness Sheikha Madiyah Dalmook Bin Juma Al Maktoum

BOOK OF ABSTRACTS

REGENERATIVE MEDICINE and AGEING

February 01- 02 / 2020
Dubai, UAE

PIAS the negative regulator of STAT3. Here, we show for the first time that PIM serine/threonine kinases, also bind and phosphorylate TRIM8, resulting in increased stabilization of the ubiquitin ligase activity. Since SOCS-1 is functionally phosphorylated and also stabilized by PIMs, co-expression of a heterotrimeric TRIM8/SOCS-1/PIM complex promotes partial destabilization of the complexes and pursues degradation of the SOCS-1 protein. Additionally, co-expression of SOCS-1/TRIM8/PIM kinase complexes in 293T cells mitigates the repression of an interferon-γ-mediated signaling in responsive cells in vivo. These data add new partners to the complex network of protein-protein interactions that regulate SOCS-1 function and modulate the cytokine biological response.

BIOPEPTIDES AS MASTER REGULATORS OF SIGNAL TRANSDUCTION PATHWAYS INVOLVED IN THE PROLIFERATION AND POLARIZATION OF A MACROPHAGE CELLULAR MODEL DERIVED FROM THP-1 CELL LINE.

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In this study, we have evaluated the effects of 5 different biopeptides as regulators of inflammatory and proliferative effects on a model of monocytes and macrophages derived from the THP-1 cell line. The peptide bioregulators utilized in this project are specific and unique compounds naturally derived from specific organs and characterized by Prof. Khavinson since 1973. During 1973 - 2013 Prof. Khavinson extracted from various organs over 20 complexes of physiologically active peptides, (di-, tri-, tetrapeptides), all being covered by patents in many countries. It has been extensively demonstrated that injection and/or oral administration of such peptides are beneficial in vivo for a variety of physiological functions including modulation of the immune system as well as anti-inflammatory and anti-oxidant effects on cells of different origin.

On this project, we used 5 different synthetic and natural peptide preparations, namely Epitalon (pineal gland), Vilon (dipeptide), Thymogen (dipeptide), Thymalin (calf thymus extract) and Chonluten (tripeptide). We tested their effects in vitro by incubating monocyte/macrophages with different concentrations of compounds at a proper concentration in a time course manner.

We evidentiated that all the biopeptides increase the speeding of cell division modulating cell cycle progression and attenuate an inflammatory related cytokine profile induced by LPS-treated cells. In addition effects shown by biopeptides on the polarization and M1/M2 transaction of target macrophage cells will be analysed and discussed.

PURINERGIC SIGNALLING IN AGEING-RELATED NEURODEGENERATIVE DISEASES

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Physiological extracellular ATP levels remain low; however, following injury and stress by stressed or dying cells, extracellular nucleotide concentrations drastically increase. Once released, ATP induces stimulation of ionotropic P2X (P2X1-P2X7) and metabotropic P2Y (P2Y1-P2Y14) receptors and P1 adenosine receptors following hydrolysis of ATP into adenosine. Aberrant purinergic signaling participates in pathological conditions, including inflammatory diseases and cell death. Among P2 receptors, the P2X7 subtype is very much studied due to its cytotoxic properties and capability of forming a membrane pore. P2X7 and further purinergic receptors, including P2Y2 and P2Y6 receptors, regulate neurogenesis, inflammatory processes and microglial status during ageing, proceeding neurodegeneration. Further, P2X7 and other P2 purinergic receptors regulate neurogenesis and neural plasticity, which decline during ageing. The P2X7 receptor is believed to be involved in neurodegenerative diseases, and its inhibition presents a promising strategy for prevention of neuroinflammation and neuronal cell death. Deletion of P2X7 receptor expression recovered animals from ageing-related cognitive decline processes. Further, it is suggested that P2X7 receptor activity could prevent endogenous NSC from mobilization and differentiation, such as studied in neurosphere models obtained from P2X7(+/+, wildtype) and P2X7(-/-, knock-out) mice. An animal model of Parkinson’s disease was used to further address this question. For this purpose, unilateral hemisphere lesions of the nigrostriatal pathway of adult male Sprague-Dawley rats were induced by stereotactic injection of 6-hydroxydopamine (6-OHDA). One week after lesion, the animals presented rotational behavior when challenged with apomorphine.