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BIOGRAPHY

Irene Athanassakis is a Professor of Immunology at University of Crete, Greece. She obtained her PhD in Immunology from the Medical School of the University of Alberta, Canada. She has published 114 papers and book reviews and has given more than 70 invited talks in international meetings and has 180 abstracts in congresses with H-index 21; 154 co-authors; RG score=36.98 and more than 1500 citations. She is an active reviewer for 19 international journals.

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SOLUBLE MHC CLASS II MOLECULES IN IMMUNE REGULATION AGAINST AUTOIMMUNITY

The involvement of major histocompatibility complex (MHC) antigens in the regulation of immune response has been well defined over the years. During the intracellular trafficking, from synthesis to antigen loading and transport to the cell membrane MHC antigens find a way to be excreted by the cells, since they can be found as soluble MHC class I (sMHCI) and class II (sMHCII) molecules in all body fluids. Although secretion mechanisms have not been sufficiently studied, sMHC molecules have been shown to display important immunoregulatory properties. Concentrating on sMHCII molecules, latest findings indicate that these are loaded with self-peptides and play an important role in tolerance maintenance. Antigen-specific tolerogenic stimulation has been shown to increase serum sMHCII levels as compared to the corresponding immunogenic stimulus in mice *in vitro* as well as *in vivo*. Serum isolated syngeneic sMHCII proteins were shown to stimulate spleen cell proliferation, their major target being the CD4-positive cell population. At the physiological level, sMHCII proteins were shown to suppress not only an antigen-specific but also antigen-non-specific immune activation, correlated to increase of CD25 and CTLA-4 and decrease CD28 expression on naive CD4-positive cells, decreased IL-2 and increased IL-10 production. In addition, these molecules inhibited phosphorylation of ZAP-70 and especially LAT proteins in the downstream pathways of TCR activation signalling. Taking advantage of the above properties of sMHCII, these were applied on experimental mouse models of systemic lupus erythematosus (SLE) as well as autoimmune hepatitis (AIH) and it was shown that syngeneic or allogeneic sMHCII proteins could alleviate SLE and AIH symptoms in experimental mouse models *in vitro* as well as *in vivo*, introducing thus the ability of sMHCII proteins to suppress specific autoantigen responses, opening new areas of research and offering novel therapeutic approaches to SLE and AIH with expanding features to other autoimmune diseases.

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Rina Aharoni is working as a Senior Research Staff Scientist in the Department of Immunology at The Weizmann Institute of Science, Israel. She completed her BSc in Biology at Hebrew University, Israel. She completed her MSc and PhD in Life Sciences from the Weizmann Institute of Science, Israel and Postdoctoral Research from Stanford University, USA. Her area of research interests are in neuroimmunology, autoimmunity, pathology and therapy of multiple sclerosis (MS) and its model experimental autoimmune encephalomyelitis (EAE), immunomodulation, neuroprotection and repair processes in the central nervous system and inflammatory bowel diseases (IBD). She has published more than 70 papers and reviews on these subjects. She is the Editorial Board Member of 20 journals.

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THE STORY OF GLATIRAMER ACETATE (COPAXONE) IN THE TREATMENT OF MULTIPLE SCLEROSIS-THE POTENTIAL FOR NEUROPROTECTION BY IMMUNOMODULATORY TREATMENT

Multiple sclerosis (MS) is currently recognized as complex diseases in which inflammatory autoimmune reactivity in the central nervous system (CNS) results in demyelination, axonal and neuronal pathology. Treatment strategies thus aim to reduce the detrimental inflammation and induce neuroprotective repair processes. The synthetic copolymer Copaxone (glatiramer acetate, GA), an approved drug for the treatment of MS, is the first and so far the only therapeutic agent to have a copolymer as its active ingredient. Using the animal model of MS -experimental autoimmune encephalomyelitis (EAE), the mechanism of action of GA was elucidated. These studies indicated that GA treatment generates immunomodulatory shift from the inflammatory towards the anti-inflammatory pathways, such as Th2-cells that cross the blood brain barrier (BBB) and secrete in situ anti-inflammatory cytokines, as well as T-regulatory cells (Tregs) that suppress the disease. The consequences of GA treatment on the CNS injury inflicted by the disease were studied using immunohistochemistry, electron microscopy and magnetic resonance imaging. These analyses revealed reduced demyelination and neuro-axonal damages, as well as neuroprotective repair processes such as neurotrophic factors secretion, remyelination and neurogenesis. These combined findings indicate that immunomodulatory treatment can counteract the neurodegenerative disease course, supporting linkage between immunomodulation, neuroprotection and therapeutic activity in the CNS.