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BIOGRAPHY

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.