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## The story of glatiramer acetate (copaxone) in the treatment of multiple sclerosis - the potential for neuroprotection by immunomodulatory treatment

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Multiple sclerosis (MS) is currently recognized as complex diseases in which inflammatory 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.

### Recent Publications:

1. R Eilam, M Segal, R Malach, M Sela, R Arnon and R Aharoni (2018) Astrocyte disruption of neurovascular communication is linked to cortical damage in an animal model of multiple sclerosis *Glia* 66:1098-1117.
2. R Aharoni, C Rosen, E Shezen, D Bar-Lev, O Golan, Y Reisner, M Sela, R Arnon (2016) Assessing remyelination - metabolic labeling of myelin in an animal model of multiple sclerosis. *Journal of Neuroimmunology*. 301, 7-11.
3. R Aharoni (2015) Remyelination in multiple sclerosis: realizing a long standing challenge. *Expert Review of Neurotherapeutics*. 22(41), 1-4.
4. R Aharoni (2015) Animal models of multiple sclerosis: imperfect but imperative *Journal of Multiple Sclerosis* 2(4), 2-4.
5. RAharoni(2014) Immunomodulation, neuroprotection and remyelination - The fundamental therapeutic effects of glatiramer acetate: A critical review. *Journal of Autoimmunity*. 54, 81-92.

### Biography

Rina Aharoni is currently a Senior Staff Scientist in the Department of Immunology, Weizmann Institute of Science, Israel. She has completed her BSc in Biology at Hebrew University, Jerusalem, Israel and MSc and PhD in Life Sciences at Weizmann Institute of Science, Rehovot, Israel. She did Postdoctoral Research at Stanford University, USA. Her main research interests include "Neuro-immunology, 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, inflammatory bowel diseases (IBD)". She has published more than 60 papers and is also an Editorial Board Member of 20 journals.

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