

Η νέα μας δημοσίευση, σε συνεργασία με την Dr Lesley Probert από το Ινστιτούτο Pasteur, με ανοσογενετικά, νευροανοσολογικά και παθολογοανατομικά δεδομένα σε ένα μοντέλο ποντικού για ΣκΠ, επαγόμενου από λεμφοκύτταρα περιφερικού αίματος ασθενών με ΣκΠ !

Μετά πέντε χρόνια εργασίας και συνεργασίας, τα αποτελέσματα μας δικαίωσαν και μας δίνουν την δυνατότητα για περαιτέρω χρήση των αποτελεσμάτων, στην χορήγηση ειδικών πεπτιδίων/”εμβολίων” που χρησιμοποιήθηκαν και στην δημοσίευσή μας, σε ασθενείς πλέον, σε κλινικές μελέτες, σε μελλοντικό χρόνο.

Ευχαριστούμε την εκλεκτή συνεργάτιδα και υπεύθυνη της μελέτης, Dr Lesley Probert, αλλά και την εκλεκτή ομάδα των συνεργατών της!



OPEN ACCESS

**Edited by:**  
Bruno Stanekoff,  
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**Specialty section:**  
This article was submitted to  
Multiple Sclerosis and  
Neuroimmunology,  
a section of the journal  
Frontiers in Immunology

**Received:** 23 June 2020  
**Accepted:** 12 October 2020  
**Published:** 19 November 2020

**Citation:**  
Dagkonaki A, Avloniti M,  
Evangelidou M, Papazian I,  
Tsevelaki V, Lampros F, Tsalkis T,  
Jensen LT, Möbius W, Ruhwedel T,  
Androutsou M-E, Matsoukas J,  
Anagnostouli M, Lassmann H and  
Probert L (2020) Mannan-MOG35-55  
Reverses Experimental Autoimmune  
Encephalomyelitis, Inducing a  
Peripheral Type 2 Myeloid Response,  
Reducing CNS Inflammation,  
and Preserving Axons in  
Spinal Cord Lesions.  
*Front. Immunol.* 11:575451.  
doi: 10.3389/fimmu.2020.575451

## Mannan-MOG35-55 Reverses Experimental Autoimmune Encephalomyelitis, Inducing a Peripheral Type 2 Myeloid Response, Reducing CNS Inflammation, and Preserving Axons in Spinal Cord Lesions

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CNS autoantigens conjugated to oxidized mannan (OM) induce antigen-specific T cell tolerance and protect mice against autoimmune encephalomyelitis (EAE). To investigate whether OM-peptides treat EAE initiated by human MHC class II molecules, we administered OM-conjugated murine myelin oligodendrocyte glycoprotein peptide 35-55 (OM-MOG) to humanized HLA-DR2b transgenic mice (DR2b.Ab<sup>h</sup>), which are susceptible to MOG-EAE. OM-MOG protected DR2b.Ab<sup>h</sup> mice against MOG-EAE by both prophylactic and therapeutic applications. OM-MOG reversed clinical symptoms, reduced spinal cord inflammation, demyelination, and neuronal damage in DR2b.Ab<sup>h</sup> mice, while preserving axons within lesions and inducing the expression of genes associated with myelin (Mbp) and neuron (Snap25) recovery in B6 mice. OM-MOG-induced tolerance was peptide-specific, not affecting PLP178-191-induced EAE or polyclonal T cell proliferation responses. OM-MOG-induced immune tolerance involved rapid induction of PD-L1- and IL-10-producing myeloid cells, increased expression of Chi3l3 (Ym1) in secondary lymphoid organs and characteristics of anergy in MOG-specific CD4<sup>+</sup> T cells. The results show that OM-MOG treats MOG-EAE in a peptide-specific manner, across mouse/human MHC class II barriers, through induction of a peripheral type 2 myeloid cell response and T cell anergy, and suggest that OM-peptides might be

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