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Modulation of MS-like disease by regulatory CD11c+CD11b+GR1+ myeloid-derived dendritic cells induced by antigen-specific therapy with synthetic multi epitope protein

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It is now widely accepted that the pathogenic autoimmunity in MS, as well as in other organ-specific autoimmune diseases, can bedirected against several target antigens. We recently showed that a concomitant "multi-epitope-targeting" approach, using a specifically designed artificial multi-epitope protein (Y-MSPc) is required foreffective antigen-based immune-specific therapy of organ-specificautoimmune diseases associated with complex and dynamic pathogenic autoimmunity, such as MSY-MSPc was superior to peptide(s) in concomitantly downregulating pathogenic T-cells reactivity against multiple myelin antigens/epitopes, via the induction of effective and longer lasting peripheral regulatory mechanisms (cytokine shift, anergy, and Foxp3+ CTLA4+ regulatory T-cells . Moreover, we identified a tolerogenic myeloid DC subset in the CNS and spleen of EAE mice playing an important role in immunoregulation process in EAE, following Y-MSPc treatment. Our results demonstrate that immune tolerance induced by Y-MSPc is associated by increase of $CD11c^{+}CD11b^{+}Gr1$ myeloid derived dendritic cells in the CNS and spleen. These myeloid DCs exhibited immunoregulatory characteristics, including increased production of IL-4, IL-10 and TGF-b but reduced IL-12. Furthermore, $CD11c^{+}CD11b^{+}Gr1$ DCs were also capable of inhibiting the proliferation of PLP139-151-specific T cells in vitro and significantly suppressed ongoing EAE upon adaptive transfer. In addition, adaptive transfer of $CD11c^{+}CD11b^{+}Gr1$ DCs derived from YMSPc treated mice to EAE induced mice resulted in remarkably upregulation of CD4FOXP3 regulatory cells. Taken together, these findings suggest that i.v. administration of the multi epitope protein (Y-MSPc) results in maintaining peripheral tolerance and reduce EAE incidence by an increase in tolerogenic $CD11c^{+}CD11b^{+}Gr1$.

Biography

Nathali Kaushansky, PhD, is a staff scientist in the Neurobiology Department at the Weizmann institute of Science. She has received her BSc in Chemistry and MSc in Biomedical Engineering from the Technion – Israel Institute of Technology, Israel. She completed her PhD studies and post-doctoral training in the Immunology Department in The Weizmann Institute of Science.

In the last 10 years she work has focused on characterization of autoimmune T- and B-cells against myelin and neuronal target antigensin Multiple Sclerosis (MS). Her study aims to establish a highly specific "multi – targeting" immunemodulatory approach via co-antagonizing of most known and potentially pathogenic T-cell autoreactivities in MS. The primary goals of her recent research were1: studying mechanisms underlying immunomodulation by this multi epitope targeting agent (specificallydesigned artificial multi-epitope protein (Y-MSPc)-recently published) and 2: studying immunogenetics of susceptibility to MS, mainly at defining HLA-DR2-related epitopes of the different most important myelin target antigens in MS.

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