glatiramer binds to MHC class II, it is not processed inside DCs with the subsequent formation of an intracellular complex with MCH class II, prior to insertion of MCH class II into the plasma membrane, as is the case with other antigens. According to Dr Masha Hareli, glatiramer "binds to MHC class II with no processing and displaces the bound autoantigens, e.g., MBP, from the binding site" (79,80).

Second, according to Arnon and Aharoni (81), glatiramer treatment of experimental autoimmune encephalomyelitis (EAE) mice provokes the generation of glatiramer-specific $CD4^+$ T cells. Consequentially, these T cells migrate across the blood–brain barrier and into the CNS, where they accumulate and express antiinflammatory cytokines, that is, Th2-type cytokines. Once in the brain, the glatiramer-specific $CD4^+$ T cells also may stimulate microglia and astrocytes to express Th2-type cytokines, thus further dampening the inflammatory environment in the brain.

MHC class II includes two polypeptide chains, the alpha chain and beta chain, which form a dimer (82). MHC class II resides in a subcellular structure, that is, an endosome, located inside DCs. Peptides derived from extracellular antigens are loaded on to MHC class II, and then the endosome fuses with the plasma membrane, resulting in the peptide-located MHC class II being located in the plasma membrane, where the MHC class II positions the peptide so that it can be presented in the immune synapse. The endosome is called "MHC class II-containing compartment (MIIC)" (83,84,85). Polypeptides, can bind directly to MHC class II that resides on the surface of the DC. This capability results from the fact that the ends of the binding groove on MHC class II are open and allow the polypeptide to extend well beyond the binding groove while, in contrast, with MHC class I, the ends of the binding groove are closed (86,87).

[79]Kind response from Dr Masha Hareli, in e-mail of May 1, 2015.

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