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Deimination of the myelin basic protein decelerates its proteasome-mediated metabolism

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Abstract

© 2016, Pleiades Publishing, Ltd.Deimination of myelin basic protein (MBP) by peptidylarginine deiminase (PAD) prevents its binding to the proteasome and decelerates its degradation by the proteasome in mammalian cells. Potential anticancer drug tetrazole analogue of chloramidine 2, at concentrations greater than 1 μM inhibits the enzymatic activity of PAD in vitro. The observed acceleration of proteasome hydrolysis of MBP to antigenic peptides in the presence of PAD inhibitor may increase the efficiency of lesion of the central nervous system by cytotoxic lymphocytes in multiple sclerosis. We therefore suggest that clinical trials and the introduction of PAD inhibitors in clinical practice for the treatment of malignant neoplasms should be performed only after a careful analysis of their potential effect on the induction of autoimmune neurodegeneration processes.

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