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Antibody-proteases as novel biomarkers and highly informative molecular tools to predict and to prevent demyelination

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Proteolytic Abs (*Ab-proteases*) are multivalent immunoglobulins (Igs) endowed with a capacity to degrade the antigenic (Ag) substrate. Anti-MBP autoAbs from MS patients and mice with EAE exhibited specific proteolytic cleavage of MBP The activity of the Ab-proteases markedly differs between: *(i)* MS patients and healthy controls; *(ii)* different clinical MS courses; *(iii)* EDSS scales of demyelination to correlate with the disability of MS patients *to predict* transformation prior to changes of the clinical course, i.e., changing of a remitting type (moderate one) into the secondary progradient type (aggressive one) prior to changing in a pattern of the clinical manifestations.

The *sequence-specificity* of Ab-proteases demonstrates five sites of preferential proteolysis to be located within MBP. Those sites are located within the immunodominant regions; and two of them falling inside the sequence covering a *81-103* peptide segment and its *82-98* subsegment as well, with the highest encephalitogenic properties both to act as a specific inducer of EAE and to be attacked by the Ab-proteases very often in MS patients with the most severe (progradient) clinical courses. Sites localized within 43-68 and 146-170 whilst being less immunogenic happened to be EAE inducers very rare but were shown to be attacked by Ab-proteases very often in MS patients with moderate (remission-type) type. The activity of Ab-proteases was first registered at the *subclinical* stages 1-2 years prior to the clinical illness. About 24% of the direct MS-related relatives were seropositive for low-active Ab-proteases from which 38% established were demonstrating a stable growth of the activity. Moreover, we see also low-active Ab-proteases (to target 43-68 and 146-170 sites) in persons at MS-related risks (at the subclinical stages), and primary MRT manifestations observed were coincided with the mid-level activity. And registration in the evolution of highly immunogenic Ab-proteases to attack 81-103 and 82-98 sites predominantly would illustrate either risks of transformation of subclinical stages into clinical ones, or risks of exacerbations to develop. Further studies on Ab-mediated proteolysis may provide a supplementary tool for predicting demyelination.

Biography

Sergey Suchkov graduated from Astrakhan State Medical University and was awarded with MD. In 1985, Suchkov obtained his Ph.D. He is the Ph.D. student of the I.M. Sechenov Moscow Medical Academy and Institute of Medical Enzymology, USSR Academy of Medical Sciences, Moscow, Russia. In 2001, Suchkov finished the PostDoc Research Fellowship Program and maintained his Doctor Degree at the National Institute of Immunology, Russia. From 1987 through 1989, Dr. Suchkov was a senior Researcher, Lab of Developmental Immunology, Koltzov Institute of Developmental Biology, USSR Academy of Sciences to deal to developmental immunology. From 1989 through 1995, Dr. Suchkov was being a Head of the Lab of Clinical Immunology and Im-munobiotechnology, Helmholtz Eye Research Institute in Moscow. From 1995 through 2004, Dr. Suchkov was being a Chairman of the Department for Clinical Immunology, Moscow Clinical Research Institute (MONIKI) and the Immunologist-in-Chief of the Moscow Regional Ministry of Health. At present, Dr. Sergey Suchkov, MD, Ph.D., is Professor in Immunology, Department of Pathology, School for Pharmacy, I.M. Sechenov First Moscow State Medical University, Dean of the Department (Faculty) of The PPPM Development, and the First Vice-President of the University of World Business, Politics and Law and Secretary General, United Cultural Convention (UCC), Cambridge, UK.

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