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Postinfectious Autoimmune Syndrome (PIFAS) as a pathogenic and predictive factor to trigger chronic diseases of infectious origin (CDIO)

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Immune responses triggered by *microbial* Ags can be ignored at the autoagression suppression by tools of immune surveillance, because autoreactive T- and B-cells are able to survive for several reasons, including *molecular mimicry* phenomenon. The outcome of the phenomenon is implemented during auto-Ag epitopes recognition to result manifestations of so-called *postinfectious autoimmune syndrome* (*PIFAS*), a new combinatorial biomarker demonstrating immune-mediated disorders.

Development of PIFAS is featured with a progression of chronic relapsing diseases of infectious origin to cover models of the syndrome-like immunopathology as applicable to chronic pyelonephritis, myocardites, and obstructive bronchitis.

In view of the *structural homology* immune response caused by a microbial pathogen to balance between two categories of epitopes (self-epitopes and microbial epitopes) is being developed (even in the absence of the pathogen) through both autoreactive T-cells and auto-Abs on account of existing autoepitopes possessing mimicking properties in point of primary microbial (triggering) Ag epitope. In fact the identification of such pathogen is restricted by some difficulties, because it is practically impossible to detect autoaggressive changes before the elimination of inducing pathogen from the patient. Thus, for autoimmune myocarditis to make a bridging link with the infection is established for two-thirds of all patients, and transformation of primary (infectious) phase into PIFAS is initiated by mimicking epitopes of, for instance, Coxsackievirus (CVB3) and/or Herpesviridae (CMV), herewith presence of cardiomyo-sineautoreactive CTLs (CM-autoreactive CTLs) and anti-CM auto-Abs, damaging myocardium to release sequestered autoAgs and to facilitate the induction and/or development of PIFAS is required.

We can stress that a strictly fixed tandem of two categories of mutually mimicking epitopes (microbial and self-epitopes) is actively implicated in the pathogenesis of PIFAS.The rational approach for identifying PISAS at the subclinical stages could involve ELISA, PCR and flow cytometry as a TRIO to predict PIFAS development from both standpoints of epitope immunogenicity and related auto-Abs and TCR avidity.

The identification of the primary pathogen or associate (regardless of its area) is an important part of the protocol being used, for what we apply immune technologies combined with molecular diagnostics (*blot hybridization* and *PCR-detection*) in microbial gene pools being screened for.

An application of transgenic models to suit the aims and objectives of clinical practice will give an opportunity to reveal the sequence of events between induction and progressing of PIFAS and will allow to pre-select specific targets to be utilized as targets for therapeutic effects to control induction and progression of PIFAS and thus chronization of the disease to prevent the latter in time.

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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