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## Post-infectious autoimmune syndrome (PIFAS) as a combinatorial biomarker to monitor autoimmune myocarditis and managing chronification of the latter

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Immune reactions triggered by microbial antigens (Ags) can be ignored by tools of immune surveillance (apoptosis or immune suppression), and auto-reactive T- and B-cells can survive for molecular mimicry phenomenon based on the activation of auto-reactive lymphocytes by cross-reactive epitopes of the pathogen. The outcome of the event would result in manifestations of a so-called post-infectious autoimmune syndrome (PIFAS), a new combinatorial biomarker illustrating immune-mediated disorders including latent ones. Wherein the evaluation of triggering role of infection in the pathogenesis of PIFAS is often difficult since the time for provoking the disease to be transformed into PIFAS may begin prior specific manifestations of two-thirds of all the patients, and transformation of primary or infectious phase into PIFAS is initiated by mimicking epitopes of, for instance, CVB3 and/or CMV, herewith presence of auto-reactive CTLs and anti-CM auto-Abs, to release sequestered auto-Ags and to facilitate the induction and/or development of PIFAS is required. Despite a vast armamentarium of approaches to assess PIFAS, there are still no obvious clinical and laboratory criteria to get the syndrome validated. An application of transgenic models to suit the objectives of clinical practice will give an opportunity to reveal the sequence of events between induction and progressing of PIFAS and allow to control induction and progression of PIFAS and thus chronification of the disease to prevent the latter in time.

## Biography

Bazyleva Ekaterina Vladlenovna completed her Graduation from German School in Moscow in 2014. Currently, she is a student of Medical University of Varna in Bulgaria and pursuing her PhD in Department for Personalized and Precision Medicine at University of World Politics and Law, Moscow, Russia.

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