8th International Congress on Autoimmunity

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Final Program

Novel Diagnostic Tools & New Therapeutic Avenues in Autoimmune Diseases

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Parallel Session

Please note that the most updated version of the program and the abstracts is available on the congress website.
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TETANUS TOXOID RELATED APS MODEL HAS DIFFERENT MANIFESTATIONS IN C57BL/6 AND BALB/C MICE

L. Dimitrijević[1], I. Živković[1], M. Stojanović[1], V. Petrušić[1], S. Živančević-Simonović[2]

[1]Institute of Virology, Vaccines and Sera Torlak, Belgrade, [2]Department of Pathophisiology, Faculty of Medicine, Kragujevac, Serbia

Previously described manifestations of antiphospholipid syndrome (APS) in experimental mice generally include decrease in fecundity and/or fertility. Here we report on two different responses of BALB/c and C57BL/6 mice to TTd hyperimmunization and APS induction, with both molecular mimicry and polyclonal cell activation being included. Successful induction of APS in these two different non-autoimmune prone mice strains was achieved by tetanus toxoid (TTd) hyperimmunization using different adjuvants (glycerol or aluminium hydroxide), and different adjuvant pretreatments (glycerol or CFA). BALB/c mice responded primarily by generating pathological anti-b_2GPI Abs and fetal resorptions as a consequence of strong T cell activation. In contrast, C57BL/6 mice responded primarily by generating a strong polyclonal Ab response and fecundity lowering. Here we present that in BALB/c mice fetal resorption coincided with glycerol and CFA pretreatments, while in C57BL/6 mice fecundity lowering was most obvious in mice immunized with TTd in aluminium hydroxide irrespective of adjuvant pretreatment. Both molecular mimicry and polyclonal B cell activation takes part in APS induction, with molecular mimicry effects being dominant in BALB/c mice, and polyclonal cell activation being dominant in C57BL/6 mice.

Our model of antiphospholipid syndrome (APS) differs from those previously described as it is generated through tetanus toxoid (TTd) hyperimmunization.

Apparently, multifactorial etiopathogenesis of APS is stressed in our model as it is shown to involve responses of both the adaptive and the innate immunity, being supported by the genetic background and triggered by environmental factors.