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# Exploring new targeted therapeutic agents for SLE

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Interferon regulatory factor 5 (IRF5) polymorphisms are strongly associate with an increased risk of systemic lupus erythematosus (SLE). IRF5 plays an essential role in pathogenesis in experimental lupus mouse model through type I IFN production dependent and independent ways. The critical role of TLR7 in acceleration of SLE was already established. Thus, the molecules involved in TLR7 and IRF5 signaling pathway are potential therapeutic targets for lupus. In present study, we firstly set up luciferase immunoprecipitation system (LIPS) assay for detecting anti-U1A autoantibody in culture supernatant of B cells stimulated by TLR7 ligand, R848. An IFN beta promoter reporter system was used to monitor type I IFN production. Our results showed MyD88 homodimerization inhibitory peptide could significantly inhibit human anti-U1A autoantibody production from B cells stimulated by R848 in vitro. The kaposi's sarcoma-associated herpesvirus encoded vIRF3 and IRF5 variant 12 (IRF5-V12) could significantly inhibit type I IFN production stimulated by R848. Moreover, the central 70 amino acids of vIRF3 and the first 100 amino acids of IRF5-V12 are sufficient for suppressing R848 stimulated type I IFN production. The role of vIRF3 and IRF5-V12 in suppressing anti-U1A autoantibodies production stimulated by R848 was under investigation. These results indicate that MyD88 inhibitor and IRF5 inhibitors are promising therapeutic agents for SLE

## Biography

Shuhong Han has completed her Ph.D at the age of 29 years from Peking University, Health Science Centor, Beijing, China and postdoctoral studies from University of Florida College of Medicine. She is an assistant scientist in the Division of Rheumatology and Clinical Immunology at Department of Medicine in University of Florida. She has published 14 papers and was membership in Clinical Immunology Society ,American Society of Hematology and American Diabetes Association

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