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# New prospects of human C4 complement component system in recognizing diagnostics and analyses of autoimmune diseases

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**Aim:** The aim of this study was to summarize our data on human complement C4 subcomponent system involving in co-functioning between complement and other human organism innate protective elements against both infectious and autoimmunity diseases.

**Methods:** Components of the patient serum complement (CPSC) were registered by quantitative immunochemical methods in microplates (variants of functional analyses of isotypes C4A and C4B including their simultaneous evaluation, and C1-inhibitor upon supramolecular assembling on well bottom) and on the blot (preliminary isoelectrofocusing of patient sera in the plate of polyacrylamide gel was performed to separate isotypes C4A and C4B complexes including sub-isotypes diagnostic forms). Rabbit and goat polyclonal antibodies against purified human complement components were used. Activity of antibodies conjugated peroxidase bound to the CPSC was detected in the presence of TMB (for analyses in microplates) or chemiluminescent substrate BioWest (Pierce) in a real time.

**Results:** 1. Sera of patients possessing autoimmune diseases were characterized on the blot by appearance of complexed (covalently aggregated) C4B and C4A in more acidic region (pI 4.0-4.7) compared to that for free isotypes. Functional abilities of isotypes were confirmed by analyses in microplate. Absolute amounts of isotypes and their subisotypes as well as ratio of isotypes characterized prognostic-diagnostic patient groups of autoimmune diseases (SLE, antiphospholipid syndrome, rheumatoid arthritis). Appearance and relative intensities of the system of aggregated isotypes and sub-isotypes of C4 indicated the presence of disease, its initiation, reached phase of disease and disease character. 2. Similar localization on the blot for the complex C4B and C1-inhibitor of patients was registered.

**Conclusions:** Results indicate possible co-functioning C4B and C1-inhibitor in protection complement network upon development of autoimmune diseases. New mechanisms of cascade protection involving new combinations of CPSC may be revealed. Results open new practical possibilities in deeper (at the level of C4 sub-isotypes) diagnostics of early, progressive and chronic autoimmune diseases.

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