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Systemic lupus erythematosus (SLE) is a systemic autoimmune disease with symptoms ranging from skin related problems to more severe cardiovascular effects. The heterogenous representation of the disease might be the reason for the lack of efficient treatment. The authors hypothesise that subgroups of SLE can be characterised by different biochemical pathways and that specific biomarkers along these pathways can be identified.

In this study, the authors have utilised the Karolinska lupus cohort that consists of 320 SLE patients and 320 age-matched controls. Two main subgroups were defined: One group was defined as having SSA and SSB antibodies and a negative lupus anticoagulant test (LAC), that is, a 'Sjögren-like' group. The other group was defined as being negative for SSA and SSB antibodies but positive in the LAC test. According to previous studies these patients are at increased risk for cardiovascular events as compared to the 'Sjörgren-like' group. A pilot study was designed and EDTA-plasma from selected patients in these two groups and controls were analysed using a proteomic and metabolomic approach. Pathway analysis was then performed on the obtained data.

The pilot study showed that it was possible to differentiate the two subgroups of SLE based on the proteomic profile. From the proteins found to be significantly different between the groups, several proteins known to be involved in SLE were detected, for example, Apolipoprotein A1 and complement factor 3. In addition, proteins that to our knowledge have not been reported earlier to correlate with SLE, for example, Apolipoprotein M, were detected and are subject for further investigations. Apolipoprotein E was one of the proteins that was found to be significantly different between the two subgroups of SLE and will be investigated in the entire cohort. Preliminary data from metabolomics demonstrate that it is possible to separate patients from controls and the authors found for example that tryptophan levels were lower in SLE patients. Pathway analyses of proteomics and metabolomics data strongly predict that the changes in SLE patients compared to controls are associated with inflammation and immunity related pathways.

This project will provide new knowledge about SLE taking several complex systems into account simultaneously. Using selected biomarkers it will be possible to identify more homogenous patient populations for clinical trials and thereby increase the efficacy. The systems biology approach is likely to identify pathways that may lead to better understanding of the disease, identification of novel drug targets and biomarkers supporting improved diagnosis of SLE.

27 SYSTEMS BIOLOGY OF SLE: BIOCHEMICAL CHARACTERISATION OF SUBGROUPS WITHIN SLE FOR IMPROVED DIAGNOSIS AND TREATMENT

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