

Abstracts

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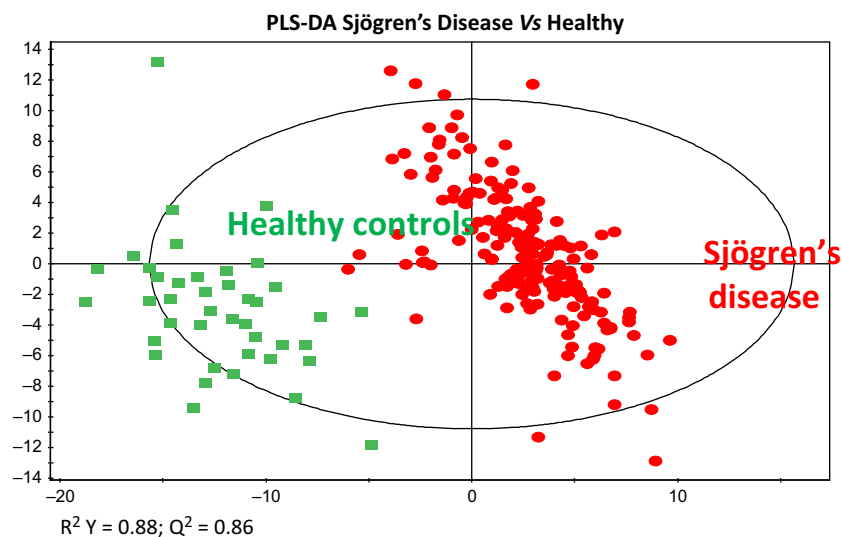


Figure 1: PLS-DA scores plot based on NMR spectra of blood samples obtained from Sjögren's Disease patients (red dots) and healthy controls (green boxes)

Conclusion: This first high-throughput analysis of metabolic pathways disclosed a specific metabolomic signature of pSS allowing discriminating all patients with pSS from controls. This new and very potent means of metabolic analysis may help to increase our knowledge on the pathogenesis of pSS, identify biomarkers, and new therapeutic targets.

S3.7

Fatigue in Primary Sjögren's Syndrome; A Proteomic Study of Cerebrospinal Fluid

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Background: Fatigue is a frequent and often disabling phenomenon that occurs in patients with chronic immunological diseases, such as primary Sjögren's syndrome (pSS). The biological mechanisms that cause fatigue are largely unknown and hypotheses are conflicting. An important task is to uncover the pathophysiology and signaling pathways that lead to fatigue. The aim of this study was to discover cerebrospinal fluid (CSF) proteins potentially involved in the generation and regulation of

fatigue using a liquid chromatography mass spectrometry (LC-MS/MS)-based proteomic approach.

Methods: From a cohort of 55 pSS patients that underwent lumbar puncture for research purposes, CSF samples from 10 subjects with high and 10 with low fatigue (measured by a fatigue visual analogue scale, fVAS) were selected for this study. To minimize abundant proteins masking the detection of the less abundant proteins, 14 high abundance proteins (HAPs) were depleted from the CSF samples prior to proteomic analysis. The depleted CSF samples were then proteolytically digested and analyzed by LC-MS/MS (LTQ Orbitrap). The resulting protein profiles from patients with high and low fatigue were compared by multivariate statistical analysis.

Results: A total of 1016 proteins were identified in the CSF samples. Supervised partial least square discrimination analysis (PLS-DA) showed that pSS patients with low and high fatigue can be separated based on their CSF protein profiles, and a set of 7 proteins were selected as the most promising discriminatory proteins. An unsupervised principal component analysis (PCA) was then performed to confirm that the majority of subjects with low versus high fatigue could be separated based on the reduced dataset, which included the 7 selected proteins only.

Conclusion: Seven proteins in the HAP depleted CSF proteome were identified which enables the discrimination between pSS patients with low or high fatigue. These proteins may have central roles in regulating fatigue.