expression of a set of TRIM genes between pSS patients and healthy controls. TRIM5, TRIM6, TRIM14, TRIM19, TRIM22 and TRIM25 were upregulated in the patients while TRIM18 and TRIM68 were downregulated. Of certain interest is TRIM5, a well-characterised retroviral restriction factor that acts on the viral capsid to destabilise it. Single nucleotide polymorphisms (SNPs) in the TRIM5 gene have been correlated to multiple sclerosis, indicating its possible importance in autoimmune disease. TRIM22 has also been identified as a retroviral restriction factor acting on HIV-1. TRIM68, also known as SS56, is an autoantigen in SLE and pSS. TRIM25 acts on the pattern recognition receptor RIG-1 after viral infection leading to an increased production of IFNα. TRIM25 is overexpressed in breast and ovarian cancers.

Conclusions Our findings show that several TRIM genes are differentially expressed in patients with pSS compared to controls. TRIM genes are important for the proper regulation of immune responses suggesting that the identified TRIMs might contribute to the pathology of pSS.

21 TRIM GENES ARE PART OF THE INTERFERON SIGNATURE OBSERVED IN PATIENTS WITH PRIMARY SJÖGREN'S SYNDROME

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Background and objectives Autoimmune disease can arise when the immune system is overactive in response to a trigger. This results in an overproduction of proinflammatory cytokines and type I interferons (IFNs). The latter is associated with the pathology of several systemic autoimmune diseases such as systemic lupus erythematosus (SLE) and primary Sjögren's syndrome (pSS). In this study the authors wanted to identify interferon signature genes that contribute to the pathogenesis of pSS. To do so, the authors did gene expression profiling on patients with pSS. The authors identified several tripartite-motif (TRIM) genes that were overexpressed in patients and that are candidates for IFN signature effector genes. The human TRIM family consists of approximately 70 genes, many of which are important infection-restricting factors or immune regulators. Interestingly, altered expression of TRIMs has previously been reported in several autoimmune diseases.

Materials and methods PBMCs from 14 female SSA positive and untreated patients with pSS and 18 age and gender matched healthy controls without any immunological disease were collected. mRNA was prepared using the QIAGEN RNeasy Plus Mini Kit and a gene expression array was performed using the Human Exon 1.0 ST chip from Affymetrix. CEL files were preprocessed using the RMA algorithm as implemented in Affymetrix Power Tools-1.12.0.

Results A strong upregulation of IFN induced genes in PBMCs from patients with pSS compared with PBMCs from healthy controls was observed. Data also revealed differential