LONG NON-CODING RNA, LINC00487 EXPRESSION IS UPREGULATED IN B CELLS AND CORRELATES TO DISEASE ACTIVITY IN PATIENTS WITH PRIMARY SJÖGREN’S SYNDROME

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Background: Primary Sjögren’s syndrome (pSS) is a systemic autoimmune disease characterized by interferon signature [1] and exocrine gland dysfunction which leads to dryness of the eyes and mouth [2]. B cells are considered to play an important role in the pathogenesis of pSS. Dysregulation of B cells can lead to production of anti-Sjögren’s syndrome-related antigen A (anti-SSA) autoantibodies.

Objectives: We investigated the differential gene expression of peripheral B cell subsets to reveal the precise role of B cells in the pathogenesis of pSS.

Methods: We enrolled pSS patients (n=6) and healthy controls (HC) (n=6), with matching for age and sex. Peripheral B cells acquired from the participants were separated by cell sorting into 4 subsets: CD38 IgD+, CD38+ IgD+, CD38high IgD+ and CD38± IgD-. Total RNA was extracted and gene expression was measured using the Human Genome U133 Plus 2.0 Array (Affymetrix). The data were bioinformatically analyzed with reference to the corresponding clinical information.

Results: Using principal component and clustering analyses, we found that transcript expression patterns depended on cell type rather than clinical condition (pSS or HC). Interferon signaling was the most upregulated pathway in many of B cell subsets of pSS patients. As well as HLA and interferon signature genes, LINC00487 was upregulated significantly in all B cell subsets. Its fold changes in pSS patients. As well as HLA and interferon signature genes, LINC00487 expression patterns depended on cell type rather than clinical condition.

Conclusion: Using principal component and clustering analyses, we found that transcript expression patterns depended on cell type rather than clinical condition (pSS or HC). Interferon signaling was the most upregulated pathway in many of B cell subsets of pSS patients. As well as HLA and interferon signature genes, LINC00487 was upregulated significantly in all B cell subsets. Its fold changes in pSS patients. As well as HLA and interferon signature genes, LINC00487 expression patterns depended on cell type rather than clinical condition.

REFERENCES

ESSDAI; EULAR Sjögren’s Syndrome Disease Activity Index


AB0173 CLINICAL SIGNIFICANCE OF THE URINARY CD11c+ MYELOID CELLS IN THE PATIENTS WITH LUPUS NEPHRITIS

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Background: It is known that various immune cell populations play important pathogenic roles in lupus nephritis (LN). Kidney-infiltrating myeloid cells are responsible for the pathology in murine model for LN. However, the characteristics of myeloid cells in patients with LN have not previously been investigated because of difficulties of access to affected tissue. Recent studies have reported that the urine has become an alternative substitute for kidney tissue to study renal pathology.

Objectives: Therefore, we investigated the characteristics and functions of urinary myeloid cells in patients with LN.

Methods: Within viable CD45+ cells, the frequency and absolute numbers of myeloid cells in the urine were examined after exclusion of lymphoid cells by expression of CD3, CD19 and CD56. Expression of phenotypic markers and...