ILC1(Lin−IL7R+ NKp46+ ILCs), ILC2(Lin−IL7R+CRTH2+ILCs) and ILC3(Lin−IL7R +CRTH2−CD117+ILCs)[1]. Little research on ILC in the pathogenesis of Primary Sjögren’s Syndrome (primary SS)

**Objectives:** Our purpose is to explore the function and role of ILC in the pathogenesis of Primary Sjögren’s Syndrome and its correlations with clinical markers.

**Methods:** 20 patients with pSS and 15 age-matched healthy non-immune-related diseases controls were enrolled. The frequency of ILCs, B cells, CD4+ T and CD8+ T cells from PBMCs was detected by flow cytometry. Analysis of the subsets of ILCs in each group which compared with B cells and T cell subsets respectively and correlation with clinical serologic markers. Analyze the levels of IL-4, IL-9, IL-33, IL-72 and IFN-γ in each group by ELISA.

**Results:** Compared with the control group, the percentage of ILC was decreased significantly in primary SS (P<0.0001); Meanwhile ILC1 was significantly increased in primary SS (P=0.0001), ILC2 was decreased significantly in primary SS (P=0.00009) and ILC3 has no significant difference in the primary SS (P=0.05).

The frequency of ILCs in all patients positively correlated with antinuclear antibody titer (ANA(D)) (r=0.295, P=0.0133), moreover, the frequency of ILC2 in primary SS was positively correlated with B cells (r=0.3896; P=0.05), and the serum IgG was negatively correlated with ILC2 of all patients (r=-0.2091, P=0.0427). Compared with Healthy control group, the level of IL-22 was significant higher in primary SS (P=0.0003), however, the levels of IL-4, IL-9, IL-33 and IFN-γ were not significant different with healthy control group (P>0.05).

**Conclusion:** The frequency of ILCs is related to ANA(D) of primary SS patients and ILCs play a critical role in the pathogenesis of primary SS. Its function and mechanism are worth further exploration.

**REFERENCE**


**Disclosure of Interests:** None declared

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**AB0049C**

**INFLAMMASOME DRIVES RELEASE OF MITOCHONDRIAL DNA ENCLOSED IN EXTRACELLULAR MEMBRANE VESICLES AND PROPAGATION OF INFLAMMATION IN BEHÇET’S DISEASE**

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**Background:** It has been reported that mitochondrial DNA (mtDNA) is released into the cytosol by mitochondrial stress and induces pro-inflammatory cytokine production via inflammasome and intracellular DNA sensors. Also, mtDNA in the extracellular space is known to result in sterile inflammation. However, the molecular mechanism of mtDNA release and its pathological significance in autoimmune diseases (ADs) has not been elucidated.

**Objectives:** To clarify the molecular mechanism of mtDNA release and its pathological significance in ADs.

**Methods:** We collected the serum from various AD patients and analyzed the levels of mtDNA in serum. We digested mtDNA in serum by DNase treatment and measured the level of mtDNA in serum and synovial fluid during flare-ups. We digested mtDNA in serum by DNase treatment and found that serum mtDNA levels were significantly high in patients with ADs.

**Results:** We first measured the levels of mtDNA in serum of the various ADs by quantitative PCR and found that serum mtDNA levels were significantly high in BD. Interestingly, mtDNA in BD serum could not be digested by DNase treatment and was detected in the extracellular membrane vesicles (EMVs) purified by ultra-centrifugation. Since EMVs are known to deliver various molecules from one cell to another, we stimulated monocytic cells with BD-derived EMVs and found that these EMVs could induce IL-1β production in an NLRP3 inflammasome-dependent manner. We then studied the mechanism of secretion of mtDNA in EMVs and found that both human primary monocyte and monocyte-like cell line containing mtDNA-containing EMVs after stimulation with ATP or LPS. Further, BD-derived monocytes secreted more abundant mtDNA in EMVs than monocytes derived from healthy donors. Additionally, the inhibition of caspase-1 activity reduced the secretion of mtDNA in EMVs.

**Conclusion:** We revealed a novel mechanism of inflammation propagation involving inflammasome and mtDNA; activated inflammasome releases mtDNA-containing EMVs and subsequently leads to mtDNA-induced inflammation via NLRP3 inflammasome. Such inflammatory mechanism may contribute to the exacerbation of inflammation in BD.

**Disclosure of Interests:** None declared

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**Cytokines and inflammatory mediators**

**AB0050**

**DETECTION OF SERUM AND SYNOVIAL FLUID LEVELS OF VISFATIN DURING FLARE-UPS AND REMISSION OF PRIMARY OSTEOARTHRITIS OF THE KNEES**

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**Background:** OA is the most common form of joint disease and a major contributor of disability in older people. OA is a chronic joint disease characterized by cartilage breakdown, bone remodeling, osteophyte development and synovial inflammation [1]. Adipose tissue expresses and secretes a large number of proteins that often share functional and structural properties of cytokines and are there classified as adipokines [2]. These include leptin, adiponectin, resistin, visfatin, and others. These factors are associated with inflammation and immune response. Visfatin is an adipokine identified in 2004 and was identified first as Pre B cell Colony Enhancing Factor [3]. Visfatin is a potent inducer of PGE2 release in both human and immortal mouse articular chondrocytes, as a result of increased messenger prostanflad E synthase and decreased 15 Prostaglandin dehydrogenase synthases [4].

**Objectives:** The aim of the study was to measure the level of visfatin in serum and synovial fluid from patients with knee osteoarthritis in flare-up and after they enter in remission.

**Methods:** To achieve the target of our study 20 patients with OA of the knee in flare-up were selected from out-patients clinic. The patients were followed up every two weeks after the first setting until they entered into remission. Twenty normal controls age, sex and body mass index (BMI) matched were recruited. In the first setting all patients had their sera, and synovial fluid measured for Visfatin, in the second setting sera and synovial fluid (if any) was drawn for visfatin measurement. Measurement of Visfatin by (ELISA) for quantitative determination of human visfatin in biological fluids.

**Results:** Visfatin was elevated both systemically and locally in the patients in flare-up and after they enter in remission.

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**REFERENCE**


SEMAPHORIN 3A: A POSSIBLE MARKER FOR DISEASE ACTIVITY IN PATIENTS WITH RHEUMATOID ARTHRITIS

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Background: Sema 3A is concerned in the pathogenesis of many autoimmune diseases because it is involved in regulation of immune responses and maintenance of self-tolerance. Regulatory T cells play an important role in maintaining immunological self-tolerance by suppressing autoreactive T cells. Sema3A promotes regulatory T cells by enhancing IL-10 production.

Objectives: The current study aimed at testing the possible role of Semaphorin 3A (Sema 3A) in activity and in remission in rheumatoid arthritis patients and to assess whether this level correlates with interleukin 10 (IL-10) level.

Methods: Sixty Egyptian patients with rheumatoid arthritis (RA) were divided into three groups according to modified Disease Activity Score (DAS28), RA in high activity (group II), RA in moderate activity (group III), and RA in remission (group IV). Serum levels of Sema 3A and IL-10 were measured and correlated with ESR, CRP, Rheumatoid factor, DAS28 and Health Assessment Questionnaire (HAQ).

Results: Serum levels of Sema 3A were significantly lower in high activity (55.7±25.5 pg/ml) compared to high activity one) and the control groups (76.5±23.6 pg/ml, p < 0.001 respectively linked to cartilage degradation biomarkers in osteoarthritis. Rheumatol Int 2012; 32:985–90.


SEMAPHORIN 3A: POSSIBLE MARKER FOR DISEASE ACTIVITY IN PATIENTS WITH RHEUMATOID ARTHRITIS

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Disclosure of Interests: IRINA GUSEVA1

Conclusion: reduced serum level of Sema 3A was found to be correlated with disease activity (group IV, n=20) and compared with 20 normal individuals (group I). Serum levels of Sema 3A and IL-10 were measured and correlated with ESR, CRP, Rheumatoid factor, DAS28 and Health Assessment Questionnaire (HAQ).

Sixty Egyptian patients with rheumatoid arthritis (RA) were divided into three groups according to modified Disease Activity Score (DAS28), RA in high activity (group II, n=20), RA in moderate activity (group III, n=20) and RA in remission (group IV, n=20) and compared with 20 normal individuals (group I). Serum levels of Sema 3A and IL-10 were measured and correlated with ESR, CRP, Rheumatoid factor, DAS28 and Health Assessment Questionnaire (HAQ).

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