Background: Cellular senescence is a state of proliferation arrest of cells. The persistence and accumulation of senescent cells has been implicated in the pathogenesis of age-related diseases like rheumatoid arthritis (RA). Clinical disease exacerbation is preceded by loss of immune tolerance and autoimmunity. Lymph node stromal cells (LNSC) are important regulators of this tolerance. Therefore, senescent LNSC may affect tolerance and the development of systemic autoimmunity.

Objectives: To determine the extent of cellular senescence of LNSC during early phases of systemic autoimmunity.

Methods: We included individuals with arthralgia without any evidence of arthritis who were positive for IgM rheumatoid factor (IgM-RF) and/or anti-citrullinated protein antibodies (ACPA; RA-risk group), early arthritis patients (ACR/EULAR 2010 criteria; disease duration < 1 year) and seronegative healthy controls. All study subjects underwent ultrasound-guided inguinal lymph node biopsy. LNSC were isolated and cultured from freshly collected lymph node needle biopsies and passages 0–9 were used for experiments. Cellular senescence was assessed by measuring cell size, granularity, and reactivity frequencies between the groups.

Results: Our data revealed four antigens associated with the ACPA status for early RA and provide a rationale for investigating the consequence of senescent LNSC on immune cell responses.

Disclosure of Interests: None declared.

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