Systemic Lupus Erythematosus (SLE) has immune dysregulation, with lymphopenia being one of the most frequent clinical findings, namely the CD4 T cells. It has been associated not only to higher risk of infections, but also to disease activity and risk of flares.

The authors pretended to describe the prevalence of lymphopenia in SLE in a major centre of Immunology in Portugal. A retrospective analysis on the SLE patients was performed, with a longitudinal description of the subpopulations of lymphocytes, relating it with disease activity, organ involved, therapeutics and major infections.

The sample had 48 patients, mainly constituted by females (85.4%), median age of 43 y. 62.5% had lymphopenia in the diagnosis. At the time of the most recent peripheral blood flow cytometry 66.7% of the sample had lymphopenia, with different values of cytopenia according to lymphocytes subpopulations – T CD4: 77.4%; T CD8: 75%; B: 83.3%; NK: 91.7%. Severe T CD4 lymphopenia (below 200 uL) was present in 8.3% of the sample. 18.8% had severe flares (SLEDAI Index) and 90% of these had low T CD4 counts (below 700 uL). The majority of patients with T CD4 under 200 uL were on severe flare. Higher frequency of corticosteroids and immunosuppressors, namely cyclophosphamide (12.5%) was observed on the patients. 17 patients of the sample had sequential cytometry analysis and a correlation between lymphopenia and activity has not observed. 17 cumulative infections were described, the majority (70.6%) with lymphopenia. Although opportunistic infections (pulmonary aspergillosis, PML due to JC virus) were mainly seen on patients with T CD4 under 200 uL it was not mandatory this condition on this sample – cryptococcal meningitis was described on a patient with 300 uL T CD4.

Lymphopenia was present in the majority of active lupus and T CD4 seems to correlate with severe flare. Lymphopenia seems to be a bystander on the evolution of the disease. Despite rare, unpredictable infections can appear on patients with T CD4 counts superior to 200 uL.

**Purpose** Systemic lupus erythematosus (SLE) is associated with dyslipidemia and increased cardiovascular risk. The SLE pattern is characterised by high plasma levels of low-density lipoproteins (LDL) and triglycerides and low levels of high-density lipoproteins (HDL). HDL is a complex plasma lipoprotein that is recognised for its protective role in atherosclerotic disease. It consists of an outer layer of lipids and apolipoproteins, with apolipoproteinA-1 (apoA-1) constituting 70% of the protein content, a triglyceride and cholesterol ester-rich core, and several enzymes. In addition to its antiatherogenic properties, mainly associated with reverse cholesterol transport from vessels, HDL has also anti-inflammatory properties that are not clearly understood. This work aims to show the effect of HDL on T lymphocyte proliferation.

**Methods** Peripheral blood mononuclear cells (PBMCs) were isolated from 7 SLE patients, with at least 4 SLICC/ACR classification criteria and normal serum lipid profiles, and 3 healthy donors. PBMCs were cultured with and without HDL (at the concentrations of 50, 300 and 600 μg/mL) before CD3 and CD28 stimulation. After 4 days in culture, T cell proliferation was measured by flow cytometry through Ki-67 staining. Regulator T cells (Tregs) phenotyping (CD4 +CD25+CD27−FoxP3+) was performed. The expression of the cholesterol transporter ABCA1 in T lymphocytes was also measured by flow cytometry.

**Results** HDL decreased T cell proliferation in a dose-dependent manner, with the biggest effect obtained with the physiologic concentration of 600 μg/mL. The inhibition of T cell proliferation was more pronounced in SLE patients than in the healthy donors. SLE patients tend to have higher baseline T cell proliferation measured by ki-67 expression. There were no differences in the prevalence of Tregs among patients and healthy donors. The expression of ABCA1 on the surface of T lymphocytes was similar between groups.

**Conclusions** This study is the first demonstration of a regulatory effect of HDL on the adaptive immune system of SLE patients. Here we show that HDL can decrease T cell proliferation, which is not correlated with the expression of the ABCA1, the main cholesterol transporter to ApoA-1. We expect to further elucidate HDL effects on the immune system in future studies.