Background The course and evolution of the SLE depends on the affected organ, adherence to treatment and accumulated organ damage, but ethnicity plays a fundamental role, leading to worse results in the non-Caucasian population. Our goal was to describe the demographic, clinical and serological characteristics of a cohort of patients diagnosed with SLE, treated at a private center in the City of Cali, Colombia.
Methods Descriptive analytical study of a cohort of patients older than 18 years, diagnosed with Systemic Lupus Erythematosus who met the SLICC 2012 classification criteria and who were treated at a Private Center in the City of Cali, Colombia between January 2016, and January 2023.

Categorical variables were expressed in percentages and 95% confidence intervals (95% CI). Continuous variables were expressed as means with standard deviations (SD) and medians with interquartile ranges (IQR).
Results 848 patients were included. The mean age of the patients attended was 48.4 years (SD: 15.34). 92.2% correspond to the female gender. The median follow-up (time between the first and last visit) was 24.9 months (IQR: 10.0–38.28). The ANA titer that we found most frequently was 1/1280 in 21.77% of the cases followed by 1/640 in 18.08% and 1/160 in 17.34%. The anti-DNA was positive in 42.61% of the cases. Regarding antiphospholipid antibodies, the presence of IgG-anticardiolipin (ACL) was positive in 21.37%, IgM-ACL positive in 18.92%, IgG-B2GP (Beta 2 glycoprotein) in 10% and IgM-B2GP in 5.45% of the cases. Lupus anticoagulant was positive in 5.88% of the cases. Regarding disease activity, most patients (53.47%) presented a SLEDAI of 0 (remission), 32.04% a SLEDAI between 1 and 4 (low activity), 12.86% between 5 and 10 (moderate activity) and only 1.63% a SLEDAI greater than 10 (high activity).
Conclusions The results are similar to the data published in different national and international studies.

6. SLE etiology & pathogenesis

LP-082 EFFECTS OF ROSUVASTATIN FOR THE TREATMENT IN LUPUS-PRONE MICE

[1]Wook-Young Baek*, [2]Sang-Won Lee, [1]Seung-Ju Kim, [1,2]Chang-Hee Suh. *[1]Department of Molecular Science and Technology, Ajou University, Republic of Korea; [2]Department of Rheumatology, Ajou University, Republic of Korea*

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Background Systemic lupus erythematosus (SLE) is a chronic autoimmune disease characterized by the production of autoantibodies to various nuclear antigens and high serum cholesterol levels. 3-hydroxy-3-methylglutaryl-coenzyme A (HMG-CoA) reductase inhibitors has been shown to have exhibited anti-inflammatory effects in several clinical trials. We conducted this study to evaluate the effect of rosuvastatin on inflammatory responses in MRL/lpr mice, a representative model of human SLE.
Methods MRL/lpr mice were intraperitoneally injected with rosuvastatin (10 mg/kg, n = 4) or vehicle [2% dimethyl sulfoxide (DMSO), n = 4] five times a week from 13 to 17 weeks of age. The serum levels of low-density lipoprotein (LDL) cholesterol and autoantibodies were measured, as well as the urine levels of albumin. Renal tissues were stained with HE and PAS for histopathological analysis. Concentrations of key inflammatory cytokines in serum were measured, and messenger RNA (mRNA) levels in target organs (kidney, spleen, and lymph nodes) were evaluated.
Results Rosuvastatin treatment significantly decreased serum LDL-cholesterol concentration in MRL/lpr mice. However, the treatment of rosuvastatin did not improve clinical manifestations nor the titers of autoantibody. In addition, serum inflammatory cytokines and proteinuria were not changed with treatment of rosuvastatin. Furthermore, histopathological analysis of the kidneys did not improve with rosuvastatin treatment. When assessing the expression of mRNA, the treatment with rosuvastatin decreased tumor necrosis alpha in spleen and kidney tissue, and interleukin-17 concentration in the kidney and lymph node of MRL/lpr mice.
Conclusions The treatment of rosuvastatin is insufficient to improve disease activity of SLE, although it can decrease inflammatory cytokines in the lymphoid organs and kidneys of MRL/lpr mice.

LP-083 PHORBOL ESTER ACTIVATES HUMAN MESENCHYMAL STEM CELLS TO INHIBIT B CELLS AND AMELIORATE LUPUS SYMPTOMS IN MRL.FASLPR MICE

[1,2]Hong Kyung Lee*, [1]Sang-Bae Han. *[1]College of Pharmacy, Chungbuk National University, Cheongju, Chungbuk 28160, Republic of Korea; [2]Bioengineering Institute, Corestem Inc., Gyeonggi 13486, Republic of Korea*

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Description Rationale: Systemic lupus erythematosus (SLE) is a multi-organ autoimmune disease characterized by autoantibody production by hyper-activated B cells. Although mesenchymal stem cells (MSCs) ameliorate lupus symptoms by inhibiting T cells, whether they inhibit B cells has been controversial. Here we address this issue and reveal how to prime MSCs to inhibit B cells and improve the efficacy of MSCs in SLE.
Methods We examined the effect of MSCs on purified B cells in vitro and the therapeutic efficacy of MSCs in lupus-prone MRL.Faslpr mice. We screened chemicals for their ability to activate MSCs to inhibit B cells.
Results Mouse bone marrow-derived MSCs inhibited mouse B cells in a CXCL12-dependent manner, whereas human bone marrow-derived MSCs (hMSCs) did not inhibit human B (hB) cells. We used a chemical approach to overcome this hurdle and found that phorbol myristate acetate (PMA), phorbol 12,13-dibutyrate, and ingenol-3-angelate rendered hMSCs capable of inhibiting IgM production by hB cells. As to the mechanism, PMA-primed hMSCs attracted hB cells in a CXCL10-dependent manner and induced hB cell apoptosis in a PD-L1-dependent manner. Finally, we showed that PMA-primed hMSCs were better than naïve hMSCs at ameliorating SLE progression in MRL.Faslpr mice.
Conclusions Conclusion: Taken together, our data demonstrate that phorbol esters might be good tool compounds to activate MSCs to inhibit B cells and suggest that our chemical approach might allow to improve the therapeutic efficacy of hMSCs in SLE.