Background: Anti-ribosomal P protein autoantibodies (anti-P) are found in 10% of all patients with lupus1 and their pathogenic role is supported by clinical association as expected for these kinds of enzymes.

Results: NMDAR. In addition, NSPA contains an APC-10 domain belonging to E3 ubiquitin ligases and a cotransfection assay in HEK293 cells demonstrated NSPA ubiquitylation as expected for these kinds of enzymes.

Conclusions: All together, these results suggest that NSPA is an ubiquitin ligase that having PTPN4 as a substrate regulates the tyrosine phosphorylation status and consequently the function of NMDAR at the synaptic region.

References:

Acknowledgements: Financed by CONICYT Basal grant# PFB12/2007 and FONDECYT grant #1160513.

Disclosure of Interest: None declared


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Disclosure of Interest: None declared


Results: LN patients had a lower percentage of CD19+ cells than controls (9.2% vs 10.6%; p=0.01) as well as a lower percentage of memory unswitched cells CD27+IgD+ (10.7% vs 15.3%; p<0.001) while patients had an higher percentage of plasmablasts and double negative memory cells CD27-IgD- (respectively 5.9% vs 1%; p<0.001% and 10.9% vs 4.1%; p<0.01).

No significant differences regardless B cells subsets were found between early LN patients and long ones as well as between LN patients at the onset and LN patients during renal flare. We found a correlation between an higher disease activity (assessed with SLEDAI 2K) and lower percentage of memory B cells IgD- CD27+ (p=0.02). Double negative B cells CD27-IgD- tended to be correlated with an higher disease activity. Of interest the correlation between persistent proteinuria detected during the follow-up and a lower percentage of plasmablasts at the baseline (p=0.01).
THE RELATIONSHIP BETWEEN THE DIFFERENT TYPES OF CELL DEATH IN SYSTEMIC CONNECTIVE TISSUE DISEASES

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OBJECTIVES: To study the basic mechanism of cell death (autophagy, apoptosis and necrosis) typical of patients with rheumatoid arthritis (RA), systemic lupus erythematosus (SLE) and systemic scleroderma (SSD). During systemic connective tissue diseases and especially in SLE, a close relationship between various types of cellular death is observed.

METHODS: 7 SLE-, 10 RA-, 10 SSD patients' sera and 5 donors' sera were studied. The level of Ca ions was registered by the method of atomic absorption spectrometry. Adenosine monophosphate-activated protein kinase (AMPK) was estimated by the Western blotting method. The activity of ATP-ase was measured spectrophotometrically. In order to find out the level of p53 protein, the immune-enzyme Human p53 Platinum ELISA method was employed. The quantity of hemoprotein (Cyt c) and the level of 8-hydroxy-2-deoxyguanosine (8-OH-dG) was measured by employing the immune-enzyme method.

RESULTS: Assessing the functional activity of AMPK is a specific marker and a strategic biopower regulator of autophagy, as well as a specific indicator of red-ox strategic biopower regulator of autophagy. In systemic connective tissue diseases, the oxidative stress is matched by urinary calcium and a decrease in the level of calcium in the blood. It is a biological marker of the free-radical damage of the DNA.

CONCLUSIONS: Chaperone-mediated induction of the immune response by autophagy, an evolutionary enshrinled only in mammals, perhaps, is the central link in the pathogenesis of systemic connective tissue diseases.

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Disclosure of Interest: None declared