Methods: Twenty- two individuals with APS with or without other associated autoimmune disease (20 females, two males, median age 55 (range 18–70) years) had blood samples taken before and 12 weeks after starting HCQ 200mg. Plasma was stored at -80°C and thawed to measure TF using Imubind TF kit (Invitech Ltd, Cambridgeshire, UK). The assay was performed according to the manufacturer's instructions. Patient characteristics are outlined in table 1. Statistical analysis was performed using SPSS Version 22. For continuous normally distributed data a two-tailed student's paired t-test was performed. A value of p=0.05 was considered as significant. There are no previous data in this area, so we were unable to do a power calculation to work out study size. Our study is therefore a pilot study.
Results: Soluble TF levels were above our normal range (40–300 pg/ml) prior to the commencement of HCQ and were significantly reduced (pre level mean (SD) 401.8 (152.8) pg/ml versus post 300.9 (108) pg/ml (p=0.010).

Table 1: Demographic data

Variables	APS (N=22)
Age: median (range)	47 (18-69)
Sex (female:male)	20:02
Ethnicity (white:asian:black)	(20:1:1)
aPL subtype	
LA* positive	19
IgG or IgM aCL positive (>99th centile)	5
IgG or IgM anti-Beta2GPI antibody positive (>99th centile)	0
aCL and anti-Beta2GPI antibody positive (>99th centile)	0
aCL antibody and LA* positive (>99th centile)	3
Anti-Beta2GPI antibody and LA* positive (>99th centile)	0
aPL complication	
Thrombosic APS	
Previous arterial thrombosis	10
Previous venous thrombosis	7
Previous arterial and venous thromboses	4
Obstetric APS	
Previous recurrent 1st trimester pregnancy loss	2
Previous pre-eclampsia	1
Previous intrauterine growth restriction	1
Previous stillbirth	3
Treatment	
Warfarin	14
Heparin	0
Aspirin	3
Autoimmune profile	
Antinuclear antibodies (ANA)	10
Extractable nuclear antibodies (ENA)	2
Double stranded DNA antibodies (dsDNA)	1

Key: aCL - anticardiolipin; anti-Beta2GPI - anti-Beta2glycoproteinI; LA - lupus anticoagulant. *LA detected by either DRVVT, dilute aPTT or TSVT

Conclusions: There was a significant reduction in soluble TF levels in this patient cohort of patients with apL and APS after commencing HCQ. Our previous work has shown that HCQ has not affected complement turnover, VEGF levels, thromboelastometry findings or CRP levels. Our findings of a reduction of soluble TF levels in aPL positive patients after the commencement of HCQ maybe a key mechanism by which HCQ reduces thrombotic risk. Further studies of a larger patient cohort are required to confirm our observation.
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AB0148 INDUCTION OF HO-1 EXPRESSION IN MONOCYTES MIGHT PREVENT KIDNEY DAMAGE IN LUPUS NEPHRITIS (LN)

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Background: Systemic lupus erythematous (SLE), is an autoimmune disease characterized by autoantibody synthesis and inflammation. During disease course, up to 70% of SLE patients will develop LN. Emerging evidence has demonstrated that infiltrating monocytes and macrophages are associated with LN pathogenesis. We have previously demonstrated that HO-1, a haem-degrading enzyme with anti-inflammatory properties is decreased in peripheral monocytes of SLE patients. Therefore, we decided to explore the contribution of HO-1 expression to LN pathogenesis.
Objectives: To explore the role of HO-1 in modulating innate immunity through a cytoprotective effect in monocytes of LN nephritis patients. Accordingly, we examined the expression of HO-1 in circulating monocytes, and the effect of HO-1 induction in reactive oxygen species (ROS) production and the phagocytic activity of monocytes from peripheral blood of LN patients and healthy controls (HC).
Methods: SLE patients with proliferative LN confirmed by renal biopsy (Class III, IV or V ISN/RPS) were recruited at Hospital Clinico of PUC. All individuals signed an informed consent form. Monocytes were purified from peripheral blood mononuclear cells (PBMCs) of LN patients and HC using pan-monocytes MACS kit. Subpopulations of monocytes and HO-1 expression were measured by FACS. ROS was determined using CellRox Kit. The phagocytic ability of monocytes was assessed by FACS and the total phagocytosis was calculated as the percentage of cells with engulfed beads.
Results: We found that monocytes from LN patients show significant differences when compared to HC in all the parameters analyzed. The percentage of CD16+ inflammatory monocytes was higher in LN patients (6.72±0.98%) compared to HC (4.07±0.48%) ($p<0.05$). HO-1 protein expression is decreased in circulating LN monocytes (4789±911 vs 1572±481, p=0.005). Baseline levels of ROS are elevated in LN monocytes with similar values that the ones found in monocytes from HC treated with a ROS inducer (HC: 3509±584; HC+TBHP: 8436±1909; LN: 8355±1714). Furthermore, phagocytic activity is increased in LN monocytes (77.97±3.31%) compared to HC (39.63±2.75%). Moreover, our preliminary data indicate that HO-1 induction, using cobalt protoporphyrin (CoPP), leads to downregulation of ROS production in LN (~60%) and HC (~40%) leaving both in similar levels of ROS production. In addition, phagocytic activity is also decreased in LN and HC monocytes in the presence of CoPP (~30%).
Conclusions: Decreased HO-1 expression in circulating monocytes of LN patients leads to higher ROS production and phagocitic activity. ROS level and phagocytosis are reduced when we induce HO-1 expression with CoPP. We propose that HO-1 induction might exert a cytoprotective role in LN by regulating innate immunity. FONDECYT N° 1150173.
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AB0149 PROLACTIN AND AUTOPHAGY IN SYSTEMIC LUPUS ERYTHEMATOSUS: CLINICAL SIGNIFICANCE OF CORRELATION BETWEEN PRL-R+ (RECEPTOR), CD19+, ATG14+, AND CD25+ EXPRESSION ON B AND T REGULATORY CELLS

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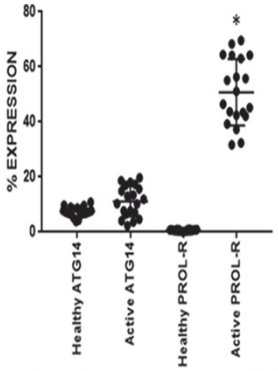
Background: Systemic Lupus Erythematosus (SLE) is a prototype of autoinmune diseases with excessive anti-nuclear autoantibodies production. B cells activation with immune complex formation is the main characteristic of SLE with abnormalities in immune cells, dysregulation of apoptosis, and defects in the clearance of apoptotic materials. Autophagy, a highly conserved protein degradation pathway, is essential for removing protein aggregates and misfolded proteins in cells and its defects contributes to SLE pathogenesis. On the other hand, multiple evidences in humans and experimental models suggest that prolactin (PRL) is associated with active SLE and participates in the immune dysregulation, and one of the mechanisms of PRL action is the inhibition of apoptosis.
Objectives: Analyze the relationship between PRL receptors (PRL-R) on B cells and markers of autophagy on T regulatory cells and the association, if any, with clinical characteristics of SLE.



Expresssion of PRL-R and ATG14 in active SLE patients and healthy controls. *p=0.026 Dunn´s multiple comparisons test.