Background and aims Liver X receptors (LXRs) are originally identified as ligand-dependent transcriptional activators and induce target genes involved in lipid metabolism. Also, LXRs have emerged as important regulators of inflammatory gene expression in several diseases. We previously reported that induction of target genes involved in lipid metabolism. Also, LXRs identified as ligand-dependent transcriptional activators and 

Methods Macrophages were obtained after 72 hour of culture of human monocytes (U937 and THP-1) supplemented with PMA (80 nM). Cells were transfected with LXRα promoter constructs. Supernatants were evaluated by enzyme-linked immunosorbent assay for proinflammatory cytokines. Also, peripheral blood mononuclear cells (PBMCs)-derived macrophages from SLE patients were evaluated for proinflammatory cytokines according to genotypes of LXRα –1830 T>C.

Results The expression of LXRα is increased in human monocyte-derived macrophages. Proinflammatory cytokines, such as IL-1β and TNF-α, are decreased in expression of LXRα. Production of proinflammatory cytokines are different according to expression of genotypes of LXRα –1830 T>C. Especially, expression of LXRα is decreased and proinflammatory cytokines are increased in TC type of LXRα –1830 T>C compared to TT type. These data are consistent in human PBMC-derived macrophages from SLE patients according to genotypes. Increased expression of proinflammatory cytokines is related to TLR7 and TLR9 expression with LXRα.

Conclusions These data suggest that expression of LXRα according to genotypes of LXRα –1830 T>C may contribute to the inflammatory response by induction of inflammatory cytokines in SLE.

Background and aims Increased levels of MMP-9 were reported in serum samples of SLE patients versus healthy controls, with the suggestion that MMP-9 plays a negative role in SLE. As a contrast, others demonstrated an inverse correlation between the levels of MMP-9 and anti-dsDNA in serum of SLE patients, the latter being a marker of disease severity. The double knock-out mice model B6(lpr/lpr)MMP-9-/- mice shows reduced survival with extreme lymphadenopathy and splenomegaly, high lymphoproliferation, increased autoantibody (aAb) production and pronounced autoimmune tissue injury comparing with B6(lpr/lpr)MMP-9-/- mice. These data supports our suggestion that MMP-9 plays an important role in the clearance of auto-antigens (aAg).

Methods Our present goal is to analyse if MMP-9 plays a role in immune complex (IC) clearance.

Biochemical and molecular techniques Results Our data suggested that MMP-9 degrades aAg, coupled to immunoglobulins in IC, but the efficiency of cleavage depends of the nature of the IC. Our preclinical data in the B6(lpr/lpr) lupus animal model were validated with samples from SLE patients.

Conclusions All these data may be interpreted in the way that MMP-9 acts as a protective factor in the development of the disease. Consequently, a profound study of the role of MMP-9 in SLE will generate new and interesting data about pathophysiology and progression of the disease and allow us to develop new effective treatment options.