neutrophil elastase (NE), proteinase 3 (PR3), and cathepsin G (CG) and myeloperoxidase (MPO) expression by qRT-PCR. NE−/PR3−/CG− triple knockout mice and in vitro depletion of neutrophils approaches were performed to determine the role of NSPs and neutrophils in estrogen-mediated inflammatory responses. The splenic neutrophil and NSPs expression in lupus-prone mice and estrogen-treated activity with regards to the increase of neutrophils and NSPs in Overall, we demonstrated a remarkable common-Other than neutrophils) and LPS-induced IFNg and MCP-1 tion of splenic neutrophils in vitro.

Results Although oestrosten reduced total splenocytes number, it markedly increased the splenic neutrophil numbers, NSPs and MPO expression in B6 mice (Figure 1). Splenic neutrophils, NSPs and MPO were also significantly increased in MRL-lpr, B6-lpr and NZB/WF1 mice (Figure 2). Despite of the critical role of NSPs and neutrophils in inflammation, dep-ition of NSPs in vivo did neither affect oestrogen’s ability to increase in splenic neutrophils nor the induction of inflammatory mediators from ex vivo activated splenocytes, and depletion of splenic neutrophils in vivo had also no obvious effect on NSPs expression (due to the increase of NSPs in cells other than neutrophils) and LPS-induced IFNg and MCP-1 (Figure 3).

Conclusions Overall, we demonstrated a remarkable common-ality with regards to the increase of neutrophils and NSPs in the spleens of autoimmune-prone mice and estrogen-treated B6 mice.

113 EFFECT OF MICROPARTICLES DERIVED FROM PATIENTS WITH LUPUS ERYTHEMATOSUS SYSTEMIC (SLE) ON MODULATION OF MICRONAS 146A AND 126, IN A MONOCYTE CELL LINE

1G Vasquez, 1L Camarona-Perez*, 2CH Munoz-Vahos, 3AL Venegas-Garcia, 4M Rojas,
1Universidad de Antioquia, Grupo de Immunologia Celular e Imunogenetica GICICG, Instituto de Investigaciones Medicas- Facultad de Medicina-, Medellin, Colombia; 2Hospital Universitario San Vicente Fundacion, Section Reumatologia, Medellin, Colombia

Background and aims Important miRNAs are involved in the modulation of immune functions and can be found in the intra- and extracellular environments, in circulation attached to RNA-binding molecules or packed in form of microparticles (MP). Given this, MP could have an important role in inter-cellular communication, modulation and expression of miR-NAs in their target cells.

To establish whether MP of SLE patients can modulate the expression of miRNAs (miRNA 126, miRNA 146a) and their target molecule (interferon response factor 5, IRF5) in the monocytic cell line U937.

Methods MP obtained from serum of SLE and other autoin-mune diseases (OAD) patients, and healthy controls were used as stimulus in the cell line U937 to evaluate their effect over: 1) The expression of membrane markers through flow cytometry, 2) content of miRNAs 126 and 146a through PCR and 3) expression of their target molecule, IRF5 by Western Blot.

Results We observed, a decrease in HLA-DR, CD18, CD119 and an increase of IL-6 in U937 stimulated with MP from healthy controls, patients with active and inactive SLE, as well as patients with OAD. Additionally, a positive effect over the expression of miR126 and a negative effect over the expres-sion of miR146a were observed. IRF5 as a target of miRNA146, did not change after MP treatment independent of MP origin.

Conclusions Our results suggested that MPs may have a regulatory effects, inducing a decreased expression of membrane molecules and miRNAs-146 levels, without effect in IRF5. In addition, MP increased levels of cytokines and miRNA-126, latter is related with the demethylation.

114 N-ACETYL-L-CYSTEINE (NAC) CONTROLS OSTEOCLASTOGENESIS THROUGH REGULATING TH17 DIFFERENTIATION AND RANKL PRODUCTION IN RHEUMATOID ARTHRITIS

1HR Kim*, 2BM Kim, 3KA Lee, 4SH Lee, 5KW Kim, 1Konkuk University Hospital, Rheumatology, Seoul, Republic of Korea; 2Seoul St. Mary’s Hospital- College of Medicine- The Catholic University, Convergent Research Consortium for Immunologic disease, Seoul, Republic of Korea

Background and aims This study aimed to determine the regulatory role of N-Acetyl-L-cysteine (NAC), an antioxidant, in IL-17-induced osteoclast differentiation in rheumatoid arthritis (RA).

Methods After RA synovial fibroblasts were stimulated by IL-17, the expression and production of RANKL was determined by real-time PCR and ELISA. Human peripheral blood monocytes were cultured with M-CSF, IL-17, RANKL, and/or various concentrations of NAC, followed by counting of the cells for tartrate-resistant acid phosphate activity to determine osteoclast formation. Osteoclastogenesis was also determined after cocultures of IL-17-stimulated RA synovial fibroblasts, Th17 cells and various concentrations of NAC with monocytes. After human peripheral CD4+ T cells were cultured with NAC under Th17 condition, IL-17, IFN-g, IL-4, Foxp3, RANKL and IL-2 expression and production was determined by flow cytometry or ELISA.

Results When RA synovial fibroblasts were stimulated by IL-17, IL-17 stimulated the production of RANKL, and NAC reduced the IL-17-induced RANKL production in a dose-dependent manner. NAC decreased IL-17-activated phosphorylation of mTOR, JNK and IkB. When human peripheral blood CD14+ monocytes were cultured with M-CSF and IL-17 or RANKL, osteoclasts were differentiated, and NAC reduced the osteoclastogenesis. After human peripheral CD4+ T cells were co-cultured with IL-17-pretreated RA synovial fibroblasts or Th17 cells, NAC reduced their osteoclastogene-sis. Under Th17 polarising condition, NAC decreased Th17 cell differentiation and IL-17 and RANKL production.

Conclusions NAC inhibits the IL-17-induced RANKL production in RA synovial fibroblasts and IL-17-induced osteoclast differentiation. NAC also reduced Th17 polarisation. NAC could be a supplementary therapeutic option for inflammatory and bony destructive processes in RA.

115 QUANTITATIVE AND FUNCTIONAL EVALUATION OF PLASMA MICROPARTICLES IN SYSTEMIC LUPUS ERYTHEMATOSUS

1A Lateef*, 2L Shaffe, 3GK Gill, 3P Cheung, 4YC Lim, 1National University Hospital, Medicine, Singapore, Singapore; 2Yang Loo Lin School of Medicine- National University of Singapore, Medicine, Singapore, Singapore; 3National University of Singapore, Physiology, Singapore, Singapore; 4National University of Singapore, Pathology, Singapore, Singapore

Background and aims Plasma microparticles (MPs) have been implicated in the pathogenesis of lupus, especially in the induction of inflammatory and immune responses. We aimed to determine the concentration of MPs in plasma and their effect on cell activation.

Methods We measured the concentration of MPs in plasma from patients with systemic lupus erythematosus (SLE) and healthy controls. We also evaluated the effect of MPs on the activation of monocytes, lymphocytes, and T cells using flow cytometry and cytokine production.

Results We found a significant increase in the concentration of MPs in plasma from SLE patients compared to healthy controls. MPs were also shown to induce the activation of monocytes, lymphocytes, and T cells, as indicated by an increase in the expression of activation markers and cytokine production.

Conclusions Our results suggest that MPs may play a significant role in the pathogenesis of lupus by inducing inflammation and immunological responses. Further studies are needed to understand the mechanisms by which MPs contribute to the disease process.

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