

# Abstracts, 20th PANLAR Meeting: Buenos Aires, April 2018

The Pan-American League of Associations of Rheumatology (PANLAR) Congress is the most important gathering of Pan-American Rheumatology, and this year marks the Congress's 20th year, being held in Buenos Aires, Argentina in April 2018. This gathering is very special for several reasons. PANLAR will return to Argentina after 21 years since the last Congress was held in this region at a very special moment for PANLAR, which is celebrating its 75th anniversary.

This supplement includes all the abstracts that were selected for presentation. This year the selection process was very difficult, as there were a record number of abstract submissions (more than 500 abstracts) from all around the world. The supplement includes all scientific investigation abstracts, but does not include case reports. Case reports are an important part of the Congress, and will be presented at the meeting, but have not been included in this supplement. The abstracts have been organized by the type of presentation and topic.

The PANLAR Congress organizers thank all the authors for their hard work, and for choosing to submit their abstracts to present their investigation at the 2018 PANLAR meeting. We believe that the PANLAR Congress encourages rheumatologists and allied health professionals to continue their rheumatologic research. Through scientific investigation, rheumatology care in the Americas continues to improve. We especially thank Dr. Graciela S. Alarcón and her team for their incredible work in the editing of all the abstracts.

## PLENARY SESSION

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### ANTIPOSPHOLIPID SYNDROME IN PEDIATRICS: MORBIDITY BEYOND THROMBOSIS, MULTICENTER IBERO-AMERICAN STUDY

C. Vargiu-Rincon<sup>1,2</sup>, C. Malagon<sup>2,3</sup>, J. Anton<sup>1</sup>, C. Mosquera<sup>2,3</sup>, M.P. Gomez<sup>4,5</sup>, and R. Yepes<sup>6,7</sup>. <sup>1</sup>Universidad del Valle, Cali, Colombia; <sup>2</sup>Universidad El Bosque, Bogotá, Colombia; <sup>3</sup>San Jose de Dios Hospital, Bucaramanga, Spain; <sup>4</sup>Universidad Libre, Cali, Colombia; <sup>5</sup>Pediatric Rheumatology and Immunology Group, Colombia; <sup>6</sup>Objectives: To describe the clinical and paraclinical characteristics of a group of pediatric patients with positive antiphospholipid antibodies.

**Methods:** Descriptive multi-center study of case series. Review of medical records with data collection format in Bogotá, Cali and Barcelona. Data analysis with SPSS 20.

**Results:** N = 117. Average age of diagnosis 12.5 years, 82% women with a F:M ratio for the general group of 4.7: 1 and with thrombosis 2: 1. 58% were associated with SLE, 41% were primary and one case with SS. 42% had at least one thrombotic event, 53% venous, 40% arterial and 14% cerebrovascular disease. 2.5% developed CAPS and 10% presented recurrence of thrombosis mainly in the lower limbs and CNS. Male sex was a factor associated with risk of thrombosis ( $p = 0.001$ ). 70% presented non-thrombotic manifestations and were more frequent in patients <10 years. Hematological compromise was dominated by thrombocytopenia in 44%, hemolytic anemia 25% and Evans syndrome 11%. Neurological manifestations: migraine 16%, seizures 6.8% and chorea 6%. 18% had ANA titers  $\geq 1:280$  and 76% had at least one aCL or positive LA. The presence of aCL-IgM antibodies was associated with non-thrombotic manifestations. There were no significant differences in the recurrence of thrombosis between primary APS or associated with another autoimmune disease, nor association with positivity of autoantibodies.

**Conclusions:** APS generates an important morbidity in pediatrics and should be considered as a cause of thrombosis in children, even in the absence of other autoimmune diseases. The criteria for diagnosis of APS were developed for adults and may fail to recognize a group of patients with non-thrombotic manifestations, present in 70% of our cases, especially within the compromise is hematological or neurological isolated. Therefore, validated criteria for pediatric patients that consider APS not necessarily as a thrombotic disease should be developed.

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### MECHANISMS INVOLVED IN THE INDUCTION OF IL-6 IN FIBROBLAST-LIKE SYNOVIOCYTES BY STIMULATION WITH SPONDYLOARTHRITIS SYNOVIAL FLUID

M.S. Di Genaro<sup>1,2</sup>, E. Callegari<sup>1</sup>, R. Blas<sup>1</sup>, A. Manarini<sup>1</sup>, R. Pardo-Hidalgo<sup>1</sup>, and H. Tamarit<sup>1,2</sup>. <sup>1</sup>Instituto Multidisciplinario de Investigaciones Biológicas-San Luis (IMIBIO-SL), Consejo Nacional de Investigaciones Científicas y Técnicas (CONICET), San Luis, Argentina; <sup>2</sup>Universidad Nacional de San Luis, San Luis, Argentina; <sup>3</sup>SD Biochemical Research Infrastructure Network (SD-BRIN),

Hershall, USA; <sup>4</sup>Centro Médico IMEMCT, San Luis, Argentina; <sup>5</sup>Centro Médico CENYR, San Luis, Argentina; <sup>6</sup>Centro de Rehabilitación Médica CER, San Juan, Argentina; <sup>7</sup>Clinica Bolívar, San Luis, Argentina.

**Objectives:** In previous studies we have reported that synovial fluid (SF) of spondyloarthritis (SpA) patients induces interleukin (IL)-6 production in fibroblast-like synovioocytes (FLS), and this cytokine modulates metalloproteinase-9 activity of these cells. However, the molecules of the SpA synovial microenvironment that trigger this pro-inflammatory circuit are unknown. The purpose of the present work was to examine the candidate endogenous molecules of SpA SF, and the mechanisms involved in the induction of IL-6 in FLS.

**Methods:** The SF of SpA patients were pooled ( $n=9$ ). Human synovial fibroblasts of the SW982 cell line were stimulated with SpA SF pool at dilution 1:10 in DMEM culture medium in absence or presence of anti-tumor necrosis factor (TNF) (infliximab, 200 g/ml), TNF inhibitor (Etanercept, 100 g/ml), anti-IL-17 (Secukinumab, 100 g/ml), anti-IL-6 receptor (IL-6R) (Tocilizumab, 200 g/ml), Toll-like receptor 4 (TLR4) activation inhibitor (Polymyxin B, 50 g/ml), ERK, JNK or p38 mitogen-activated protein kinase (MAPK) inhibitor (10 nM), or Janus kinase (JAK) 1/3 inhibitor (Tofacitinib, 1000 nM). SF treated with Hyaluronidase (50 g/ml) (H-SF) or Proteinase K (500 g/ml) (P-SF) was also used for stimulation of FLS. The IL-6 levels in the supernatants were measured by a commercial enzyme-linked immunosorbent assay (ELISA) kit. In addition, IL-6-inductor alarmins in SpA SF were identified by a Gel-free and Gel-LC bottom-up mass spectrometry-based proteomics through the 2D-micro-liquid chromatography-tandem mass spectrometry (2D-micro-LC-MS/MS) analysis. The data were analyzed by one-way analysis of variance (ANOVA) followed by Tukey multiple comparison test. A  $p$ -value  $\leq 0.05$  was considered statistically significant.

**Results:** We found that anti-TNF, anti-IL-17 and anti-IL-6R significantly reduced IL-6 secretion by FLS ( $p=0.05$ ,  $p=0.01$  and  $p=0.01$ , respectively). No difference in IL-6 induction was detected when H-SF was used for stimulation, or when JNK or JAK1/3 was inhibited. In contrast, P-SF stimulation ( $p=0.001$ ), and TLR4, ERK or p38 inhibition significantly affected IL-6 production by FLS ( $p=0.01$ ). The proteomic analysis showed that the most abundant proteins of the SpA SF come from extracellular location and cytoplasm. The IL-6-inductor alarmin S100A8 (calgranulin A) was detected in position 116 in the rank of the most abundant proteins of SpA SF.

**Conclusions:** The results indicate that TNF, IL-17 and IL-6 themselves enhance SpA SF-induced IL-6 production by FLS, supporting a fine-tuning cytokine in the inflammation process in the synovial microenvironment. In addition, S100A8, as an endogenous molecule released to the extracellular synovial milieu, may be involved in IL-6 induction in FLS by SpA SF. TLR4 pathway, and ERK and p38 MAPKs participate in the IL-6 production by FLS. Understanding the pathogenic mechanisms associated with the synovial microenvironment may contribute to suggest new therapeutic approaches for SpA.

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### ARTHRITIS: PRIMARY RESULTS THROUGH 52 WEEKS FROM A PHASE-3 RANDOMIZED PLACEBO-CONTROLLED STUDY (FUTURE 4)

A. Kivitz<sup>1</sup>, P. Nash<sup>2</sup>, H. Tahir<sup>2</sup>, A. Everding<sup>2</sup>, P. Peller<sup>2</sup>, A. Widmer<sup>2</sup>, L. Priocet<sup>2</sup>, K. Abrams<sup>2</sup>, and on behalf of the FUTURE 4 Study Group. <sup>1</sup>Alkermes Centre for

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