with idiopathic ataxia, but recent studies have indicated that they are also found in systemic lupus erythematosus (SLE). The goals of this study were to determine the frequency of anti-MPP-1 in a local SLE cohort and then identify demographic, clinical, and serologic correlations.

Methods Patients fulfilling the American College of Rheumatology (ACR) or Systemic Lupus International Collaborating Clinics (SLICC) Classification Criteria for SLE were enrolled in the SouThern Alberta Registry for Lupus Erythematosus (STARLET) cohort (Calgary, Canada). Demographic, clinical information (disease activity – SLEDAI-2K; damage – SLICC/ACR DI), and sera were collected at time of enrollment. Antibodies to MPP-1 were determined by an addressable laser bead immunoassay (ALBIA) utilizing an *in vitro* expressed MPP-1 cDNA construct inserted into a GFP vector (Clontech Laboratories Inc., Saint-Germain-en-Laye, France). ALBIA results were expressed as median fluorescence units (MFU) and a dilution of $\geq 1:500$ MFU was considered highly positive. Univariable analysis was performed to determine associations between the prevalence of high positive anti-MPP-1 and demographic (age, sex, race/ethnicity), clinical features (SLEDAI-2K and SLICC/ACR DI total scores and subscales and neurological subscale of the ACR and SLICC Classification Criteria), medications, other autoantibodies (anti-dsDNA, extractable nuclear antigens, and anti-phospholipid antibodies).

Results One hundred and forty SLE patients were included; 89.3% were female with a mean age of 47.3 years (SD 16.3) and disease duration of 13.9 years (SD 11.6). The prevalence of high titre anti-MPP-1 was 15.0% (21/140) respectively. Univariable analysis demonstrated that high anti-MPP-1 positivity was associated with a higher total SLEDAI-2K score (Odds Ratio (OR), 1.1 [95% CI 1.0 to 1.3]), particularly with the serositis (OR 3.0, [95% CI 1.4 to 6.6]) and immunological subscales (OR 2.0, [95% CI 1.4 to 2.9]). High anti-MPP-1 positivity was also associated with anti-dsDNA (OR 5.5 [95% CI 1.8 to 16.6]), anti-SSA/Ro60 (OR 3.1 [95% CI 1.0 to 8.9]) and anti-phosphatidylserine/prothrombin complex (aPS/PT)-IgG (OR 3.6 [95% CI 1.1 to 11.5]).

Conclusions High titer anti-MPP-1 antibodies were common in this SLE cohort (15.0%) and may be associated with greater clinical and serologic SLE disease activity. A larger study is currently underway to more clearly delineate its role as a biomarker in SLE.

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AA-05 B CELL INTRINSIC IFN β IS ASSOCIATED WITH AUTOANTIBODIES AND ACTIVE RENAL DISEASE IN SLE

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Background Dysregulated responses to type I interferons (IFNs) is a hallmark of autoreactive B cell development in SLE patients. High sera levels of type I IFN protein were recently shown to occur in the absence of increased circulating pDCs and in the absence of increased pDC IFNs, suggesting the likelihood of other important sources of type I IFN that may act on B cells. The present study determined the cellular

source of IFN β and its association with disease activities in SLE.

Methods Peripheral blood mononuclear cells (PBMCs) were obtained from 31 SLE patients meeting ACR 1997 revised criteria for SLE and 9 healthy controls. Intracellular IFN β was determined using flow cytometry with FITC-anti-IFN β mAb. Comprehensive clinical data was recorded for each SLE subject and the clinical data and laboratory analysis of B-cell intracellular IFN β expression were collected in a double-blind fashion.

Results IFN β was detected in various cell types including CD4 T cells, B cells and pDCs in PBMCs of SLE patients. Endogenous IFN β in B cells was significantly higher than endogenous IFN β in CD4 T cells and were equivalent to that seen in pDCs. Within B cells, there was a significant increase in endogenous IFN β in all B cell subpopulations of SLE patients compared to healthy controls. The most significant increase was found in the CD27⁺IgD⁻ memory subpopulation. B-cell endogenous IFN β was not a result of B-cell uptake of exogenous IFN β as coculture of SLE B cells with HEK293 reporter cells resulted in induction of interferon stimulatory genes as determined by the secreted alkaline phosphatase assay. This was further blocked by an anti-IFN β neutralization antibody. Interestingly B-cell endogenous IFN β was highly correlated with clinical disease including renal disease and autoantibodies including anti-dsDNA, anti-Sm and anti-SSA. Strikingly, the highest correlation of IFN β with clinical manifestations was observed in African-American patients. B-cell IFN β expression was significantly correlated with CD19^{lo}CD38^{hi}CD27⁺ plasma cell formation.

Conclusion Intracellular IFN β production by B cells is a novel and important B-cell intrinsic factor that may be essential for B-cell development into autoantibody producing B cells. The present work suggests a need for future human lupus studies into type I IFN dysregulation that pioneer beyond the view of pDC produced IFN α . These results also provided a mechanistic basis for development of more effective therapies to target the high-affinity IFN β or the enhanceosome components that promote its induction in a subgroup of lupus patients.

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Big Data Analyses

BD-01 E-GENES IDENTIFIED VIA TRANSANCESTRAL SNP MAPPING AND GENE EXPRESSION ANALYSIS REVEAL NOVEL TARGETED THERAPIES FOR AFRICAN-AMERICAN AND EUROPEAN-AMERICAN SLE PATIENTS

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Background Systemic lupus erythematosus (SLE) in African-Americans (AA) is more prevalent, more severe and associated