expression revealed suppression in the relative fold change of IL-6 by 23%, IL-8 by 30%, TNF- α by 11%, and IFN- γ by 21% with Tai Chi.

Conclusions: These data suggest that moderate exercise and stress management can have potent immunoregulatory effects on the chronic, systemic inflammation associated with SLE and identify daily Tai Chi exercise as a viable adjunct therapy to complement current pharmacological interventions.

References:

[1] Wang C, et al. A randomized trial of tai chi for fibromyalgia. *The New England journal of medicine* 2010, 363(8): 743–754.

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AB0152 DISEASE ACTIVITY AND DAMAGE SCORES CORRELATED DIFFERENTLY WITH THE PATIENT REPORTED OUTCOMES IN PATIENTS WITH PRIMARY SJÖGREN'S SYNDROME COMPARED TO LUPUS ASSOCIATED WITH SECONDARY SJÖGREN'S SYNDROME

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Background: Previous studies showed a poor correlation between EULAR Sjögren's Syndrome Patient Reported Index (ESSPRI) and EULAR Sjögren's Syndrome Disease Activity Index (ESSDAI) in patients with Sjögren's syndrome (SS) (1). There are no previous studies correlating these scores with the SS Disease Damage Index (SSDDI) or assessing comparatively patients with primary (pSS) and secondary SS associated with lupus (SLE/SS).

Objectives: We aimed to assess how well scores assessing disease activity, damage and patient reported outcomes correlate with each other in patients with pSS compared to SLE/SS.

Methods: We conducted a cross-sectional study of 55 patients with pSS and 15 SLE/SS. Detailed clinical and laboratory data, along with patient reported outcomes (ESSPRI) and disease activity and damage scores (ESSDAI, BILAG and SSDDI) were collected. We compared the clinical and laboratory parameters of the two patient groups (pSS and SLE/SS) and assessed the correlations between maximum ESSDAI score since diagnosis and ESSDAI, ESSPRI, SSDDI and BILAG scores at the time of assessment.

Results: The table below shows comparatively the differences between the pSS and SLE/SS patient groups.

Table 1

Demographic	pSS (n=55)	SLE/SS (n=15)	Significance
Sex n (female/male %)	55 (100/0)	15 (100/0)	
Ethnicity n (C/A/MN/O)	43/6/3/3	9/1/5/0	P=0.4571
Age mean (range)	58 (28–84)	56 (25–78)	P=0.5923
Disease duration mean (range)	12 (2–34)	23 (3–40)	P=**0.005
Anti-Ro+ % (n)	76 (42)	73 (11)	P=0.7273
Anti-La+ % (n)	49 (27)	27 (4)	P=0.0565
ESSPRI median [IQR]	5 [3–7]	4 [2–6]	P=0.3382
ESSDAI median [IQR]	2 [0–4]	2 [1–4]	P=0.8884
Maximum ESSDAI [IQR]	4 [2–6]	3 [2–6]	P=0.9519
SSDDI median [IQR]	1 [1–2]	1 [0–2]	P=0.1992
Global BILAG median [IQR]		0 [0–2]	
Treatment			
Hydroxychloroquine (%)	65	40	P=0.0775
Methotrexate (%)	5	33	P=***0.0092

Unpaired T test performed; **p<0.01, n = total number, % percentage, IQR = interquartile range, ethnicity: C = Caucasian, A = Asian, MN = African Caribbean, O = Chinese, anti-Ro = anti-Sjögren's-syndrome-related antigen A, anti-La = anti-Sjögren's-syndrome-related antigen B.

We found significant correlations of SSDDI score with disease duration and maximum ESSDAI score in patients with pSS ($r=0.27$, $p=0.05$ and $r=0.67$, $p=0.0001$, respectively). In SLE/SS patients, ESSPRI scores correlated with both BILAG and maximum ESSDAI score ($r=0.55$, $p=0.03$ and $r=0.7$, $p=0.02$, respectively). The SSDDI score correlated with the disease duration only in pSS patients ($r=0.27$, $p=0.05$).

Conclusions: Our study showed that there was no similar correlation between various disease scores in patients with pSS compared to SLE/SS patients. If the patient reported outcomes correlated with the disease activity (ESSDAI and BILAG) in SLE/SS patients, this correlation was not seen in pSS patients, in which the significant correlations were found only between damage scores, highest disease activity score and disease duration.

References:

[1] Responsiveness of disease activity indices ESSPRI and ESSDAI in patients with primary Sjögren's syndrome treated with rituximab, *Ann Rheum Dis* 2012;71:.

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AB0153 DELETION OF RECEPTOR FOR ADVANCED GLYCATION END PRODUCTS (RAGE) DOES NEITHER AFFECT AUTO-ANTIBODY PRODUCTION NOR DEVELOPMENT OF RENAL DISEASE IN PRISTANE-INDUCED LUPUS

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Background: Systemic lupus erythematosus (SLE) is characterized by autoantibodies to diverse autoantigens, especially to nuclear components such as double-stranded DNA (dsDNA) or nucleosomes. Still, it remains unclear why poorly immunogenic molecules such as dsDNA and nucleosomes become targets of humoral autoimmunity in SLE. Increased signaling via pattern-recognition receptors (PRRs) through pathogen-associated molecular patterns (PAMPs) or endogenous damage-associated molecular patterns (DAMPs) released from damaged or stressed host cells and tissues, may be critically involved in the break of tolerance against such nuclear antigens. One important PRR in this context may be RAGE. Among many others, RAGE recognizes High mobility group box 1 (HMGB1). HMGB1 is a DNA-binding nuclear protein that has been found at elevated levels in patients with SLE and other autoimmune diseases; likewise perpetuation of RAGE signaling sustains inflammation and leads to the establishment of chronic inflammatory disorders.

Objectives: We therefore examined, using the Pristane-induced SLE model, if increased RAGE signaling may be involved in the break of tolerance against nuclear antigens and contributes to chronic inflammation.

Methods: To that end, WT and RAGE-/- animals were injected intra-peritoneally with a single dose of pristane. Disease manifestations were determined after 7 months and included the determination of proteinuria and renal pathology by evaluating the glomerular cellularity and matrix on H&E stained paraffin-embedded kidney sections and glomerular depositions of IgG and C3c. In addition to that we checked for auto-antibody production at several time points during disease development and immune cell distribution, differentiation and phenotype in inflamed kidneys and spleen.

Results: Apart from a slight decrease in GL7^{hi}Fas^{hi} germinal center B cells and B220⁺CD21^{low}CD23^{hi} follicular B cells in RAGE-/- animals, we did not detect differences in auto-antibody secretion or disease manifestations between RAGE-/- and WT mice.

Conclusions: Our data contrast with recently published data showing that a deletion of RAGE exacerbated lupus nephritis and lymphoproliferation in a different SLE model (B6-MRL Fas lpr/lj). Therefore, we are currently looking into the effects of RAGE deletion in additional models of auto-antibody-mediated immune disease.

Disclosure of Interest: None declared

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AB0154 CIRCULATING CPG DNA PROMOTER FRAGMENTS IN SLE ACTIVATE INTRARENAL TLR9 SIGNALING AND ACCELERATE RENAL INFLAMMATION AND FIBROGENESIS

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Background: In systemic lupus erythematosus (SLE), lupus nephritis (LN) is associated with chronic inflammation and perpetuated fibroblast activation, both determined by epigenetic mechanisms involving aberrant CpG DNA promoter methylation. During SLE progression, global methylation patterns are commonly lost. These CpG DNA promoter methylation patterns are not limited to the kidney, circulating CpG-rich DNA is also detectable in the blood allowing for biomonitoring ("liquid biopsy"). However, little is known about its specific contribution to determining disease progression. In the kidney, CpG-rich DNA activates TLR9 signaling mechanisms involved in inflammation and fibrogenesis. Based on these observations, we hypothesized that CpG-rich DNA promoter fragments potentially accelerate renal inflammation and fibrogenesis in SLE-associated LN.

Objectives: To analyze the role of circulating CpG-rich DNA on endothelial TLR9 signalling and the effect of experimental modification of oligodinucleotides on kidney inflammation in the Pristane-induced murine model of SLE.

Methods: We isolated circulating CpG-rich DNA from blood samples in a cohort of SLE patients. Then, we tested how these DNA promoter fragments influenced the LN phenotype in a TMPD ("pristane")-induced mouse model. Further, we investigated how this renal response could be influenced by the administration of either human or synthetic methylated/unmethylated CpG-rich DNA oligodinucleotides. Additionally, the effects of the administration of circulating CpG-rich DNA fragments on TLR9-signalling was analyzed in endothelial cell cultures.

Results: We show that circulating CpG-rich DNA promoter fragments are detectable in SLE patients' blood. Furthermore, SLE-associated LN is associated with accumulation of unmethylated CpG-rich DNA promoter fragments, implicating a mechanistic connection. These observations were further corroborated in a rodent model of TMPD-induced SLE where administration of CpG-rich DNA (isolated from LN patients or synthetic unmethylated CpG-rich DNA oligonucleotides) worsened the renal phenotype in terms of inflammation and fibrogenesis.

Causal contribution of TLR9 was further confirmed in *Tlr9*^{-/-} knockout mice with protection from renal inflammation and kidney fibrosis after administration of unmethylated CpG-rich DNA promoter oligonucleotides. TLR9-mediated intrarenal