Conclusions: The Portuguese version of the ASQoL performed well, demonstrating good psychometric properties for use in clinical studies and trials of patients with AS. The lack of significance in the analysis by self-perceived disease severity may be due to the relatively small sample size.

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AB1162 RAPID3 SCORE CAN PREDICT DISEASE ACTIVITY IN PRIMARY SJÖGREN'S SYNDROME

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Background: Sjögren's syndrome (SS) is a chronic autoimmune disease that causes salivary and lacrimal gland dysfunction, resulting in oral and ocular dryness. The European League Against Rheumatism (EULAR) SS disease activity index (ESSDAI) is a systemic disease activity index measuring disease activity in patients with SS. The ESSDAI includes 12 domains. EULAR SS patient-reported index (ESSPRI) is used to evaluate dryness, fatigue, and pain symptoms, and their impact on the disease. Routine Assessment of Patient Index Data 3 (RAPID3) is used to evaluate disease activity in patients with rheumatoid arthritis which is another inflammatory disorder.

Objectives: This study aims to evaluate whether RAPID3 is useful in primary SS.

Methods: 30 patients with primary SS were enrolled in the study. ESSDAI, ESSPRI and RAPID3 scores were recorded. Chi-square, Mann Whitney U test and Pearson correlation analysis were performed for the statistical analysis.

Results: Demographically and clinical data were shown in the Table-1. Mean ESSDAI, ESSPRI and RAPID3 scores were 3.8±3.6, 5.8±1.7, and 14.8±5.2, respectively. RAPID3 scores were positively correlated ESSPRI ($r=0.669$, $p<0.001$). In addition, when we set the cut-off value to 12 on the RAPID3 score (>12 accepted as active, and ≤ 12 accepted as inactive), ESSPRI score was significantly higher in active patients (6.4±1.4 vs. 4.1±1.4, p=0.002). However, there was no relationship between RAPID3 and ESSDAI scores.

Schirmer test was positively correlated with tear break up time (BUT) ($r=0.573$, $p=0.007$). Lissamine green score was negatively correlated with Schirmer test and BUT ($r=-0.484$, $p=0.007$, and $r=-0.507$, $p=0.004$, respectively). Despite there was high compliance among these three scales evaluating eye involvement, these scales did not appear to correlate with the ESSDAI, ESSPRI, and RAPID3 scores that assess global disease activity. The mean age was significantly higher in patients with Schirmer test ≤ 5 mm compared to the patients with >5 mm (55.6±6.9 vs. 47.6±8.5 years, p=0.044).

Table 1. Demographics and clinical variables

	SS (n=30)
Mean age, years	51.0±8.7
Disease duration, years	6.3±4.6
Sex, % females	100
WBC, $10^3/\mu l$	5.9±1.8
Hemoglobin, g/dl	13.3±1.5
ESR, mm/h	19.5±16.4
CRP, mg/dl	7.2±13.5
ANA positivity, %	83.3
Anti-Ro positivity, %	65.5
Anti-La positivity, %	46.2
HAQ	32.4±4.9
Schirmer test, mm	11.4±6.4
BUT, sec	3.2±1.8
Lissamine green score	2.2±1.1

SS; Sjögren's syndrome, WBC; white blood cell count, ESR; erythrocyte sedimentation rate, CRP; C-reactive protein, ANA; anti-nuclear antibody, HAQ; health assessment questionnaire, BUT; tear break up time.

Conclusions: In SS, it is not simple to detect disease activity. Comorbid psychosomatic diseases affect the set detecting global disease activity. On the other hand, the activity of glandular involvement and global disease activity are not with compliance. Therefore, new and easy tools are necessary in primary SS. In our study, RAPID3 score is corelated with ESSPRI. This result suggests that RAPID3 is useful to detect disease activity in primary SS.

Disclosure of Interest: None declared

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AB1163 ANTIBODIES BINDING SYNTHETIC OLIGONUCLEOTIDES DISTINGUISH LUPUS FROM RHEUMATOID ARTHRITIS, SCLERODERMA AND SJOGREN'S SYNDROME

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Background: The SLE-key® RuleOut iCHIP® antigen microarray-based test rules out a diagnosis of SLE with a sensitivity of 94%[1].

Objectives: Here we report the use of the iCHIP® platform and a set of synthetic oligonucleotide antigens to distinguish between SLE subjects and those with a diagnosis of Rheumatoid Arthritis (RA), Scleroderma (SSc), Sjogren's syndrome (SS), or healthy individuals (HC).

Methods: We examined IgM and IgG antibody binding to 22 synthetic oligonucleotides (44 features) in the sera of HC subjects (N=40); SLE (N=30); SSc (N=40); SS (N=20); or RA (N=30) patients. Univariate analysis (FDR adjusted p-values) was used to determine the ability of each feature to separate between SLE and the different classes of subjects.

Results: Table 1 shows that multiple oligonucleotides successfully distinguished SLE patients from all other groups. All significant features were IgG antibodies, except for 1 IgM. Table 2 shows the impact of single nucleotide change on autoantibody binding. PolyG (G17) separates SLE from all but SS. T1G16 separates SLE from HC subjects, while G16T1 gave no significant separation. The addition of a G to the 5' and 3' end of T16 enhanced IgG antibody binding and improved separation between SLE and other autoimmune diseases with at least 10-fold improved significance as compared to T20. PolyG sequence length impacts the ability of the oligonucleotides to separate between SLE and the other groups (Fig. 1A). Unexpectedly, sequences either shorter or longer than G14 were effective in separating SLE from HC, RA, and SSc, while G14 was not effective. Furthermore, none of the polyG homopolymers could separate SLE from SS. Sequences rich in C or T were more effective at separating between SLE and SS patients (Fig. 1B).

Table 1

SLE Compared to:	Number of significant oligonucleotides Minimal FDR corrected p value: 0.003–4E-6	
	IgG	IgM
HC	17	0
RA	10	0
SSc	14	1
SS	2	0

Table 2

Class	Oligo	HC Vs SLE	RA Vs SLE	SSc Vs SLE	SS Vs SLE
PolyG ± 3' or 5' T	G17	0.04	**0.0006**	**0.007**	NS
	T1G16	0.03	NS	NS	NS
	G16T1	NS	NS	NS	NS
PolyT ± 3' & 5' G	GT16G	**0.000004**	**0.008**	**0.0001**	**0.003**
	T20	**0.001**	0.03	**0.007**	0.04

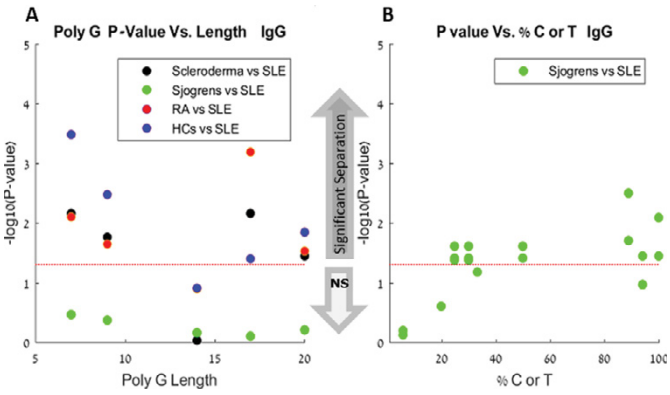

Figure 1

Conclusions:

- Autoantibody binding to oligonucleotides can be used to differentiate SLE from other autoimmune conditions and healthy subjects.
- The structural basis for the differences in binding of antibodies from disease sera to the various oligonucleotides is not yet understood, but may be due to immunologically unique conformations and secondary structures of oligonucleotides of defined length and sequence.
- SSc can be differentiated from SLE based on particular antibody binding to epitopes of oligonucleotides containing C and T.
- RA can be differentiated from SLE more significantly than the other autoimmune conditions.