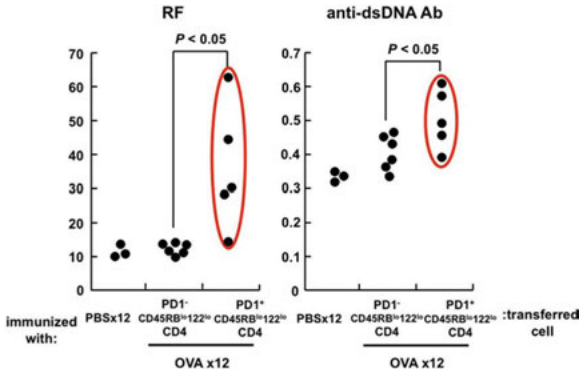
cells were adoptively transferred into naïve recipients. Autoantibodies in sera of recipient mice were measured 2 weeks after cell transfer.
Results: Under microarray analysis, we found that gene expression of PD-1 was increased x2 in the CD45RB^{lo}122^{lo} CD4 subset. Simultaneously, surface expression of PD-1 protein was also significantly increased in this subset. Adoptive cell transfer of PD-1^{+}CD45RB^{lo}122^{lo} CD4 T cells from 12x immunized mice significantly increased both RF and anti-dsDNA antibody in the naïve recipients.



Conclusions: The *ai*CD4 T cell that induces SLE belongs to PD-1^{+}CD45RB^{lo}122^{lo} CD4 subpopulation.
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Scleroderma, myositis and related syndromes – etiology, pathogenesis and animal models

AB0193 ANTIBODIES TO HNRNP B1 (RA33) IN PATIENTS WITH SYSTEMIC SCLEROSIS

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Background: Systemic sclerosis (SSc) is an autoimmune disease characterized by the occurrence of a wide range of autoantibodies (Aab) which can predict distinct clinical features of SSc. Aab to hnRNP A2 and its alternatively spliced variants B1 (anti-hnRNP B1) and B2 are generally referred to as anti-RA33 and have been extensively studied in rheumatoid arthritis (RA) [1]. However, the prevalence of anti-RA33 Aab in other autoimmune diseases, such as scleroderma, mixed connective tissue disease and systemic vasculitides, is far from determined [2–4].
Objectives: To compare the frequency of anti-hnRNP B1 with other SSc-specific Aab and to determine an association thereof with the clinical phenotype in patients with SSc
Methods: We studied Aab prevalence in 64 patients with SSc, 29 with diffuse cutaneous SSc and 26 with limited cutaneous disease as well as 9 patients with overlap syndrome. Diagnosis of SSc in all patients was based on 1980 ACR (ARA) criteria and the disease phenotype was analyzed according to LeRoy's classification criteria [7]. Serum samples from clinically healthy blood donors (n=174) were used as a control group. Skin involvement and disease severity was measured with Rodnan's skin score and Valentini activity index. Vascular involvement was accessed by measurement of pulse wave velocity (PWV) and augmentation index (AI) with applanation tonometry (SphygmoCor system, AtCor Medical Pty Ltd., Sydney, Australia). Anti-hnRNP B1 IgG was assessed by an ELISA employing recombinant human hnRNP B1 expressed in E.coli (in.vent DIAGNOSTICA GmbH, Germany). SSc-specific Aab were measured with line immunoassay (Euroimmun AG, Germany) according to the manufacturer's instructions.
Results: Anti-hnRNP B1 were found in 18.5% (12/64) of SSc patients and in 1.1% (2/174) of controls ($p<0.001$). Median concentrations were also significantly higher in SSc patients ($p<0.001$). There were no significant associations of anti-hnRNP B1 level with any other particular SSc-specific Aab, clinical form of the SSc, degree of skin involvement, and clinical activity. We found that anti-hnRNP B1 antibodies directly correlated with the presence ($r=0.33$ $p=0.009$) of arterial hypertension and vessel-wall stiffness parameters like PWV ($r=0.39$, $p=0.004$) and AI ($r=0.35$, $p<0.001$).
Conclusions: Anti-hnRNP B1 (RA33) is an independent serological marker in SSc and is associated with vascular involvement.
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AB0194 LL-37: A NEW MARKER FOR INTERSTITIAL LUNG DISEASE (ILD) IN SYSTEMIC SCLEROSIS (SSC)?

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Background: Fibrosis of the skin and visceral organs is the hallmark of SSc and ILD is the leading cause of SSc related morbidity and mortality [1]. LL-37 peptide is the only cathelicidin of the human antimicrobial peptide family with antimicrobial effects and an immunomodulatory activity [2]. LL-37 was shown to decrease with age [3]. Recent data defined its anti-fibrotic effects on dermal fibroblasts and anti-apoptotic effects on SSc dermal fibroblasts [4].
Objectives: To investigate the association between SSc related ILD and circulating levels of LL-37.
Methods: SSc patients and healthy controls aged 18-80, without signs or symptoms of systemic infection. Clinical data, autoantibody panel and internal organ assessment results (echocardiogram, esophageal manometry, chest HRCT, Lung Function Tests, Capillaroscopy). The pulmonary involvement was defined as SSc related ILD appearance on HRCT scans like ground-glass, reticular and

Table 1. LL37 and age in the study population groups

	A) SSc with ILD (n=30)	B) SSc without ILD (n=28)	C) controls (n=28)	p (A vs B)	p (A vs C)	p (B vs C)
AGE (mean± SD)	64.2 ± 12.3	67.4 ± 9.2	45.8 ± 15.0	0.327	**<0.01***	**<0.001***
LL37 (ng/ml)	1.3575 (0.16-1341.69)	4.6175 (0.39-22922.95)	5.53 (0.29-22658.50)	**0.035***	**0.009***	0.812*

Table 2. LL37 and clinical, laboratory, instrumental data in SSc patients.

	SSc (n=58)		Association with LL37 levels
AGE (mean±SD)	65.7 ± 10.9		0.203
Sex, male	7	(12.1 %)	0.471
LcSSc vs DcSSc	45 vs 13		0.351
ACA	35	(60.3%)	0.499
SCL70	18	(31.0%)	0.507
ESR (mm/hr)	27.5	(2-69)	0.532
Increased CRP	14	(25.8%)	0.928
VCP Early (n)	11	(21%)	0.073
VCP Active (n)	26	(46.3%)	0.063
VCP Late	17	(30.7%)	0.665
Patten VCP (n) (Early vs Active vs Late)	11 vs 26 vs 17		0.229
ILD on HRCT (n)	30	(51.7%)	**0.035***
Lower esopageal sphincter pressure <15mmHg on esophageal manomwetry	37	(63.8%)	0.365
DU (N)	28	(48.3%)	0.403
FVC (% pred, median)	111.5	(67-182)	0.430
DLCO (% pred, median)	74	(25-114)	0.626
mRSS (median)	6	(1-35)	0.955
PAPs (median, mmHg)	26	(19-51)	0.212
LVEF (median,%)	63	(28-72)	0.562

Table 3. LL37 and different ILD pattern in SSc patients.

	SSc with ILD pattern (n=30)		p
HRCT reticular	12	(40%)	0.837
HRCT ground glass	15	(50%)	0.951
HRCT honeycombing	3	(10%)	0.467