Abstract PS2:26 Table 1 Immunoglobulins autoantibodies and pro-inflammatory cytokines in SLE-SS, SLE-noSS and population controls

	Controls N=322 median (IQR) or N(%)	SLE-SS N= 117 median (IQR) or N(%)	SLE-noSS N=387 median (IQR) or N(%)	p-value SLE-SS vs. SLE-noSS
IgA total g/L	2.1 (1.5-2.8)	2.9 (1.8-4.3)	2.7 (1.9-3.6)	0.38
IgG total g/L	10.9 (9.5-12.2)	14.5 (10.4-18.3)	12.4(9.8-15.8)	0.009
IgM total g/L	1.1 (0.8-1.6)	1.0 (0.5-1.6)	0.9 (0.6-1.5)	0.89
anti-dsDNA % positive (+)	5(1.6)	36(31.3)	154(41)	0.06
anti-Ro52 % +	3 (0.9)	56(47.9)	84(21.8)	<0.0001
anti-Ro60 % +	5 (1.6)	69(59)	137(35.9)	<0.0001
anti-La/SSB % +	10 (3.1)	44(37.6)	69(18)	<0.0001
anti-Sm % +	1 (0.3)	19(16.2)	75(19.5)	0.42
anti-RNP 68 % +	0 (0)	11(9.4)	40(10.4)	0.74
Rf IgG % +	10/261(3.8)	17/80(21.2)	35/259(13.5)	0.09
Rf IgM % +	14/283(4.9)	32/83(38.6)	56/281(19.9)	0.0005
Rf IgA % +	12/282(12.4)	34/74(45.9)	75/267(28.0)	0.004
TNF-α pg/mL	2.3(2.0-2.8)	4.9 (3.6-7.1)	4.4 (3.0-6.0)	0.008
IL-6 pg/mL	0.5 (0.4-0.7)	1.5 (0.8-3.0)	1.1 (0.6-2.0)	0.009
MCP-4 pg/mL	55.8 (40.8-80.5)	94.9 (66.9-131.3)	74.7 (52.4-120.0)	0.019
MIP-1 β pg/mL	43.7 (33.4-56.4)	81.1 ((54.8-123.6)	68.9 (50.3-105.1)	0.021
IL12/IL-23p40 pg/mL	131.2 (99.8-179.5)	211.3 (141.4-363.8)	177.1 (119.6274.5)	0.032
IP-10 pg/mL	351.9 (259.2 -476.4)	808 (536-1911)	726 (440-1471)	0.036

PS2:27 ANTIBODIES TO CARBAMYLATED VIMENTIN IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS ARE ASSOCIATED WITH RENAL INVOLVENMENT

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Vimentin is a cytoskeletal protein expressed by mesenchymal cells, including endothelial and renal tubular cells. Antibodies to vimentin were described in 10%–53% of patients with Systemic Lupus Erythematosus (SLE). Vimentin has been proposed as a target of the *in situ* immune response in lupus nephritis. Post-translational modifications increase the immunogenicity of vimentin, as demonstrated by the detection of anti-modified-vimentin antibodies in rheumatoid arthritis. Carbamylation is a non-enzymatic post-translational modification (addition of a cyanate group on lysine and arginine residues), which has been linked to NETosis. The role of carbamylated vimentin (Car-Vim) as an antigenic target in SLE has not been evaluated yet.

Aim of the study was to assess the prevalence of anti-Car-VIm and to investigate any association with clinical and serological features in SLE patients.

We enrolled SLE diagnosed according to 1997 ACR criteria. Clinical features, autoantibodies profile and disease activity – according to SLEDAI 2K – were collected. Patients' sera were tested for anti- Car-Vim by a home-made enzyme-linked immunoassay. Data were expressed as mean ±standard deviation or median (interquartile range) when appropriate. Mann-Whitney and Chi square test were applied to investigate differences in anti-carbamylated vimentin prevalence and serum levels. P value<0.05 was considered statistically significant.

We enrolled 109 SLE patients (102F:7M, mean age 39.4 ±12.6 years, mean disease duration 10.5±9.5 years, mean SLEDAI 2K 5±5.5). Table 1 summarises the main clinical and serological features. Overall, 30/109 patients (27.5%) were positive for anti-Car-Vim. The prevalence of anti-Car-Vim was significantly higher in patients with lupus nephritis (18/44) compared to those without (12/66) (41.8% vs 18.2%, p=0.006); moreover, anti-Car-Vim serum levels were significantly higher in patients with lupus nephritis [2561 (1783) OD] compared to those without [1970 (1123) OD; p=0.0178]. No difference was found in prevalence or titre of anti-Car-Vim in presence/absence of other clinical or serological manifestations. No correlation between anti-Car-Vim serum levels and SLEDAI 2K was found.

Higher prevalence and serum levels of anti-carbamylated vimentin antibodies in patients with lupus nephritis confirm the role of vimentin as a target of the immune response in glomerulonephritis and suggest their possible role as a biomarker of kidney involvement in SLE.

Abstract PS2:27 Table 1 Clinical and serological feature of the patients at the time of enrolment

Clinica/seroligical feature	N(%)
Artrhritis	15 (13.8)
Skin involvement	16 (14.7)
Lupus nephritis	43 (39.4)
CNS lupus	7 (6.4)
Serositis	3 (2.7)
Hematological disorders	21 (19.3)
Anti-dsDNA +	39/74 (52.7)
Low complement levels	35/61 (57.4)

PS2:28 AUTOANTIBODY PROFILING IN PROSTVAC AND IPILIMUMAB TREATED PROSTATE CANCER PATIENTS REVEALS POTENTIAL BIOMARKERS OF IMMUNE-RELATED ADVERSE EVENTS

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Purpose Autoantibodies (AAB) targeting self-antigens can be found in two clinically and immunologically opposing diseases, autoimmune diseases and cancer. While in autoimmune diseases, the immune system is hyperactivated against self-antigens, many tumours suppress the anti-tumour immune response. The therapeutic cancer vaccines PSA-Tricom (Prostvac) is designed to generate an antigen-specific tumour response in metastatic castration-resistant prostate cancer (mCRPC), which is in phase 3 testing. To further augment the immune response, combination therapies of Prostvac with ipilimumab are currently tested in clinical studies. Ipilimumab is an antibody that blocks the immune checkpoint molecule cytotoxic T-lymphocyte-associated antigen 4 (CTLA-4). However,