their parental cells and tissues, and could serve as markers of pathology. Particularly in SLE, MPs are potential biomarkers and triggers of autoimmunity. Recent studies have demonstrated increased of plasmatic EMPs in patients with SLE active disease and their reduction after treatment.

The aim of this study was to investigate levels of EMPs in a cohort of SLE patients with and without renal involvement compared to healthy controls.

MPs were isolated from plasma and urine and characterised by flow cytometry using AnnessinV (a probe that binds to the exposed phosphatidilserine – PS) and antibodies against surface markers endothelial cells (CD31 + CD41-).

Sixty SLE patients and 29 HC were studied. Twenty-eight patients had renal involvement.

The total number of plasmatic MPs was lower in SLE patients than HC (p=0.001).

In contrast there was no significant difference EMPs between the two groups. When the patients were divided according to renal involvement, the patients with active-LN (A-LN) showed lower plasmatic EMPs in comparison to inactive LN (I-LN) (p=0.01), while the patients with I-LN had higher EMPs than HC (p=0.002). There was no significant difference of total urinary MPs between SLE patients and HC. Urinary EMPs were higher in SLE and in LN patients than in HC.

The results of the present study show increased EMPs in patients with LN in remission. Circulating-EMP have been detected in patients with vasculitis and associated with disease activation. According to our results, plasmatic EMPs are higher in inactive-LN patients than in HC. These results may suggest a potential role of EMP as a biomarker of LN.

**Introduction**

Systemic lupus erythematosus (SLE) is a systemic autoimmune disease with various clinical manifestations and serological markers. In this study, we analysed nine polyamine profiles of plasma from patients with SLE and healthy controls, and the relationship between the polyamine profiles and disease activity.

**Methods**

The alterations of the polyamine metabolome in the plasma of 44 patients with SLE and fever were investigated using gas chromatography mass spectrometry in selected ion monitoring mode using N-ethoxycarbonyl/N-pentafluoropropionyl derivatives, and compared with those of 43 healthy controls.

**Results**

Patients with SLE and healthy controls showed differences in five of nine polyamine metabolome profiles. Among five polyamines changed levels, four polyamines, namely N1-acetylcadaverine, spermidine, N1-acetyl spermidine, and spermine, were dramatically decreased. However, the level of cadaverine was increased in patients with SLE. In the partial correlation with polyamine profiles and disease activity markers of SLE, several disease activity markers and nutritional markers were correlated with cadaverine, spermidine, and N8-acetyl spermidine.

**Conclusion**

Thus, our results provide a comprehensive understanding of relationship between that polyamine metabolomes and disease activity markers in patients with SLE and fever.
Abstract PS2:26 Table 1 Immunoglobulins autoantibodies and pro-inflammatory cytokines in SLE-SS, SLE-noSS and population controls

<table>
<thead>
<tr>
<th></th>
<th>Controls N=101</th>
<th>SLE-SS N=117</th>
<th>SLE-noSS N=187</th>
<th>p-value SLE-SS vs. SLE-noSS</th>
</tr>
</thead>
<tbody>
<tr>
<td>lgA total g/L</td>
<td>2.1 (1.7-3.8)</td>
<td>2.7 (1.4-3.3)</td>
<td>2.7 (1.7-3.0)</td>
<td>0.18</td>
</tr>
<tr>
<td>lgC total g/L</td>
<td>16.9 (9.0-22.2)</td>
<td>14.7 (10.4-19.3)</td>
<td>12.4 (8.3-15.6)</td>
<td>0.009</td>
</tr>
<tr>
<td>lgM total g/L</td>
<td>1.1 (0.8-1.6)</td>
<td>1.0 (0.8-1.6)</td>
<td>0.9 (0.6-1.5)</td>
<td>0.19</td>
</tr>
</tbody>
</table>

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 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

<table>
<thead>
<tr>
<th>Clinical/serological feature</th>
<th>N (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arthritis</td>
<td>15 (13.8)</td>
</tr>
<tr>
<td>Skin involvement</td>
<td>16 (14.7)</td>
</tr>
<tr>
<td>Lupus nephritis</td>
<td>43 (39.4)</td>
</tr>
<tr>
<td>CNS lupus</td>
<td>7 (6.4)</td>
</tr>
<tr>
<td>Serositis</td>
<td>3 (2.7)</td>
</tr>
<tr>
<td>Hematological disorders</td>
<td>21 (19.3)</td>
</tr>
<tr>
<td>Anti-dsDNA +</td>
<td>39/74 (52.7)</td>
</tr>
<tr>
<td>Low complement levels</td>
<td>35/61 (57.4)</td>
</tr>
</tbody>
</table>