with nonspecific LBP without SpA-features or MRI findings suggestive of axSpA. Diagnosis of axSpA was based on multidisciplinary team conference consensus after 3.5 years of follow-up (3). Plasma levels of 10 lectin pathway proteins (MBL, CL-L1, H-ficolin, M-ficolin, MASP-1, MASP-2, MASP-3, Map44, and MAP19) were measured by immunosassays developed in-house.

**Results:** Patient characteristics are shown in Table 1. Plasma levels of lectin pathway proteins L-ficolin, M-ficolin and CL-L1 differed significantly in the patient groups (p = 0.03). L-ficolin and M-ficolin were elevated in axSpA-patients compared with patients with SpA-features without axSpA and nonspecific LBP patients (Figure 1). CL-L1 was elevated in axSpA-patients and patients with SpA-features without axSpA compared with nonspecific LBP patients (Figure 1). No significant differences were observed for MBL, H-ficolin, MASP-1, MASP-2, MASP-3, Map44, and MAP19. L-ficolin levels correlated with CRP in axSpA-patients (Spearman’s rho=0.58, p=0.004). M-ficolin levels correlated weakly with CRP in nonspecific LBP patients (Spearman’s rho=0.36, p=0.003). Lectin pathway proteins did not correlate with disease activity (ASDAS).

**Table 1. Patient characteristics**

<table>
<thead>
<tr>
<th></th>
<th>axSpA</th>
<th>Not axSpA</th>
<th>Non-specific low back pain</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Median age, years (range)</td>
<td>32 (19-40)</td>
<td>31 (19-41)</td>
<td>32 (18-39)</td>
<td>0.75</td>
</tr>
<tr>
<td>Males, n (%)</td>
<td>10 (43)</td>
<td>9 (42)</td>
<td>10 (41)</td>
<td>0.01</td>
</tr>
<tr>
<td>MBL-B27 positive, n (%)</td>
<td>17 (74)</td>
<td>15 (69)</td>
<td>14 (62)</td>
<td>0.01</td>
</tr>
<tr>
<td>Inflammatory back pain, n (%)</td>
<td>18 (78)</td>
<td>16 (77)</td>
<td>19 (70)</td>
<td>0.11</td>
</tr>
<tr>
<td>Good response to NSAID</td>
<td>14 (61)</td>
<td>12 (55)</td>
<td>16 (70)</td>
<td>0.97</td>
</tr>
<tr>
<td>Socrallotin levels</td>
<td>22 (96)</td>
<td>21 (93)</td>
<td>21 (88)</td>
<td>0.52</td>
</tr>
<tr>
<td>n (%)</td>
<td>3 (13)</td>
<td>7 (13)</td>
<td>3 (13)</td>
<td></td>
</tr>
<tr>
<td>ASDAS (range)</td>
<td>2.5 (12-37)</td>
<td>2.3 (0-8.3)</td>
<td>2.3 (0.8-3.8)</td>
<td></td>
</tr>
</tbody>
</table>

*compared by Kruskal-Wallis test. *compared by Mann Whitney U test.

**Conclusion:** L-ficolin and M-ficolin are increased in patients with axSpA when compared with relevant control cohorts of patients with LBP or with SpA-features without axSpA. Our findings support a potential pathogenic role for complement in axSpA, however, further studies are needed to elucidate the diagnostic potential of the specific complement proteins.

**REFERENCES:**


**Disclosure of Interests:** None declared

**DOI:** 10.1136/annrheumdis-2022-eular.3846

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**Figure 1.** Plasma levels of L-ficolin, M-ficolin and CL-L1.

**Background:** In systemic lupus erythematosus (SLE), the complement system (CS) is activated and thought to occur primarily through the classical pathway (CP) (1). Traditionally, when diagnosing SLE or assessing disease activity, measurement of low C3 or C4 are used as proxies for complement activation (2). However, measurement of C3 and C4 does not differentiate which complement pathway initiated the activation (i.e., the lectin pathway (LP), the CP, or the alternative pathway) (1, 3).

C1-esterase inhibitor (C1inh) is one of the key regulators of the CS. C1inh is the exclusive inhibitor of the active CP enzymes C1r and C1s (4), and the major inhibitor of active LP enzymes MASP-1 and MASP-2 (5). A possible way of assessing complement activation through a specific pathway, is by measuring activated enzymes complexed with C1inh in plasma, as these complexes only exist after complement enzyme activation.

**Objectives:** Our aim was to investigate and unravel LP and CP complement activation in SLE, by measuring the protein complexes MASP1/C1inh (LP specific activation) and C1r/C1inh (CP specific activation). Furthermore, we aimed to investigate whether there is an association between complement activity, disease activity (SLEDAI) and disease manifestations (lupus nephritis (LN)).

**Methods:** A cross sectional cohort of 150 patients with SLE fulfilling the 1997 ACR classification criteria for SLE were included from the outpatient clinic at the department of Rheumatology, Aarhus University Hospital (AUH), Denmark. Disease manifestations and disease activity using SLEDAI score was assessed at inclusion. Fifty healthy individuals included at the Blood Bank, AUH, were used as controls. Both C1s/C1inh and MASP1/C1inh complexes were measured in all samples using two newly developed sandwich ELISAs (C1s/C1inh: cat#: HK399; MASP1/C1inh: Cat#:3001, Hyclut Biotech, Uden, The Netherlands). EDTA-samples from both SLE patients and controls were measured in duplicates.

**Results:** When comparing SLE patients to controls, we observed a difference in complement activation through the LP, where a lower mean MASP1/C1inh plasma concentration was observed (p<0.01). C1s/C1inh concentrations were significantly increased in active SLE patients (SLEDAI >6) when compared to SLE patients with low disease activity (SLEDAI <6, p<0.01) and correlated with SLEDAI score (r=0.285, p<0.01). C1s/C1inh concentrations were increased in SLE patients with active LN compared to non-active LN, however this was not statistically significant (p=0.09).

No differences in MASP1/C1inh plasma concentrations were observed between active SLE patients and patients with low disease activity (p=0.11), nor did we observe a significant correlation with disease activity (r=0.12, p=0.13). In active LN, plasma concentrations of MASP1/C1inh were significantly elevated compared to non-active LN (p<0.02).

**Conclusion:** Our data suggest that the CP and the LP is activated in SLE. CS is generally activated in active SLE disease, whereas activation of the LP might be more specific to particular disease manifestations like LN. Our findings warrant further research into activation of the specific pathways in relation to specific disease manifestations in SLE.

**REFERENCES:**


**Disclosure of Interests:** None declared

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