Table 1. Autoinflammatory variants in AS, PsA, RA and Ps

<table>
<thead>
<tr>
<th>Disease</th>
<th>No. of Patients</th>
<th>No. of Autoinflammatory Variants</th>
<th>Average No. of Autoinflammatory Variants per patient</th>
</tr>
</thead>
<tbody>
<tr>
<td>AS</td>
<td>1264</td>
<td>937</td>
<td>0.741</td>
</tr>
<tr>
<td>PsA</td>
<td>886</td>
<td>780</td>
<td>0.880</td>
</tr>
<tr>
<td>RA</td>
<td>5361</td>
<td>2081</td>
<td>0.388</td>
</tr>
<tr>
<td>Ps</td>
<td>5567</td>
<td>2015</td>
<td>0.362</td>
</tr>
</tbody>
</table>

Acknowledgements: NIL

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Keywords: Spondyloarthritids, Animal Models

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Background: The complement factor H-related protein-5 (FHR-5), a member of the human factor H protein family, enhances complement activation. The influence of complement activation on bone and joint was recognized in bone fracture healing, arthritis, and osteomyelitis. Recently, FHR-5 has been linked to eye, kidney, infection, cancer and autoimmune diseases. FHR-5 was also significantly up-regulated in proteomic analysis of serum and synovial fluid for ankylosing spondylitis (AS).

Objectives: We aimed to evaluate whether FHR-5 exacerbates bone inflammation and ectopic formation of AS.

Methods: The study included 65 patients with AS and 25 healthy controls (HC). Collected sera were divided into three groups according to HC, two AS groups (low CRP and high CRP) based on the CRP 0.8. Human TNF, IL6, IL-17, IL-23, and FHR-5 in these three groups were measured with ELISA and human FHR-5 levels were significantly correlated with proinflammatory cytokines, IL-17A, and IL-23 in AS group compared to HC group. Basal FHR-5 expression was upregulated in AS-osteoprogenitors compared to control cells. Also, treatment with FHR-5 remarkably induced bone mineralization status of AS-osteoprogenitors during osteogenic differentiation accompanied by MMP13 expression.

Conclusion: We provide the first evidence demonstrating that FHR-5 can exacerbate pathological bone formation of AS. Therapeutic modulation of FHR-5 could be promising for future treatment of AS.


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Keywords: Enthesitis, Cytokines and chemokines, Targeted synthetic drugs

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POS0048

LIPOLYSACCHARIDE ACTIVATED ENTHESEAL MYELOID CELL INHIBITION WITH UPADACITINIB PARADOXICALLY INCREASES MYELOID PRO-INFLAMMATORY CYTOKINES INCLUDING TNF AND IL-23 BY RESTRAINING AN IL-10 NEGATIVE FEEDBACK-BUT T-CELL TNFA AND IL-17 IS STRONGLY BLOCKED

Keywords: Enthesitis, Cytokines and chemokines, Targeted synthetic drugs