Table 1. Adjusted \( R^2 \) of the models and indices. \( R^2 \)/m: adjusted \( R^2 \)/of the models; \( R^2 \)/l: adjusted \( R^2 \) of the index included in the model; LR: Likelihood-Ratio test comparing HUPI models vs other indices.

<table>
<thead>
<tr>
<th>OUTCOME</th>
<th>INDEX</th>
<th>ACT-RAY</th>
<th>LR</th>
<th>PEARL</th>
<th>LR</th>
</tr>
</thead>
<tbody>
<tr>
<td>RADIOPHIC PROGRESSION</td>
<td>HUPI</td>
<td>0.025</td>
<td>0.024</td>
<td>ref</td>
<td>0.110</td>
</tr>
<tr>
<td></td>
<td>DAS28</td>
<td>0.031</td>
<td>0.030</td>
<td>&lt;0.000</td>
<td>0.102</td>
</tr>
<tr>
<td></td>
<td>SDAI</td>
<td>0.051</td>
<td>0.050</td>
<td>&lt;0.000</td>
<td>0.109</td>
</tr>
<tr>
<td>HAG</td>
<td>HUPI</td>
<td>0.353</td>
<td>0.323</td>
<td>ref</td>
<td>0.477</td>
</tr>
<tr>
<td></td>
<td>DAS28</td>
<td>0.329</td>
<td>0.296</td>
<td>&lt;0.000</td>
<td>0.472</td>
</tr>
<tr>
<td></td>
<td>SDAI</td>
<td>0.334</td>
<td>0.303</td>
<td>&lt;0.000</td>
<td>0.486</td>
</tr>
<tr>
<td>IL6</td>
<td>HUPI</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>0.212</td>
</tr>
<tr>
<td></td>
<td>DAS28</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>0.204</td>
</tr>
<tr>
<td></td>
<td>SDAI</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>0.201</td>
</tr>
</tbody>
</table>

**Figure 1.** Box plots comparing the adjusted \( R^2 \) of each index estimated in models for HAQ in ACT-RAY.

**Figure 2.** Curves for comparison of predicted serum IL6 using fractional polynomials for HUPI and DAS28.

**Conclusion:** Although all indices explained the outcomes’ variability similarly, HUPI did it better than DAS28 and SDAI for almost all outcomes except for \( \Delta \) Genant and HAQ in PEARL.

**References:**

**Disclosure of Interests:** Sebastian C Rodriguez-Garcia Speakers bureau: Novartis Farmaceutica, S.A., Merck Sharp & Doyme España, S.A., Sanofi Aventis, UCB Pharma, Nuria Montes: None declared, José Ivorra: None declared, Ana Triguero-Martinez: None declared, Luis Rodriguez Rodriguez: None declared, Isabel Castrejon: None declared, Loretto Carmona Grant/research support from: Novartis Farmaceutica, SA, Pfizer, S.L.U., Merck Sharp & Doyme España, S.A., Roche Farma, S.A, Sanofi Aventis, AbbVie Spain, S.L.U., and Laboratorios Gebro Pharma, SA (All through institution), Isidoro Gonzalez-Alvaro Grant/research support from: Roche Laboratories, Consultant of: Lilly, Sanofi, Paid instructor for: Lilly, Speakers bureau: Abbvie, MSD, Roche, Lilly

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**Table AB1253**

**EVALUATION OF CYSTEINE-RICH 61 IN RHEUMATOID ARTHRITIS AS A DIAGNOSTIC MARKER AND PREDICTOR OF ATHEROSCLEROSIS**


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**Background:** Matricellular protein Cysteine-rich protein 61 (Cyr61) is involved in chronic inflammatory disorders like rheumatoid arthritis (RA) and atherosclerosis.

**Objectives:** This study aimed to assess the value of serum Cyr61 in diagnosis of rheumatoid arthritis, evaluating its correlation with disease activity and its relation to atherosclerosis.

**Methods:** Cross-sectional study included 105 RA patients classified into active and inactive groups according to disease activity score (DAS28) with 50 healthy age and gender-matched controls. Full clinical and laboratory assessment was done including enzyme-linked immunosorbent assay (ELISA) measurement of Cyr61. Bilateral assessment of carotid intima-media thickness (CIMT) was done using high-resolution-ultrasonography. Comparison of Cyr61 between RA patients and controls, correlation between Cyr61 and disease activity and CIMT were analyzed with appropriate statistical analyses.

**Results:** Significant elevation of Cyr61 in RA patients compared to controls (235.62±6.25 vs. 73.11±18.2) respectively. The cut off value of Cyr61 was 99.25 pg/ml, with area under the curve (AUC) =0.995, P <0.001, 98 % sensitivity and 95% specificity. Cyr61 was inversely correlated with DAS28 and its components in RA patients (r= 0.92, P= 0.94) (p<0.001). There was a significant positive correlation between Cyr61 levels and CIMT in inactive and active RA patients (r=0.88, r=0.47) respectively.

**Conclusion:** Serum Cyr61 as a potential diagnostic biomarker in RA is inversely correlated with disease activity. High Cyr61 in RA is a risk factor for atherosclerosis. Disruption of serum Cyr61 is engaged in the pathogenesis of both rheumatoid arthritis and atherosclerosis which is a clue for a future treatment strategy of RA.

**References:**


**Disclosure of Interests:** None declared

**DOI:** 10.1136/annrheumdis-2020-eular.752

**Table AB1254**

**PHENOTYPING OF MULTIPLE BIOFLUIDS FOR LIQUID BIOMARKERS FOR DIAGNOSTICS AND PERSONALIZED MEDICINE OF RHEUMATOID ARTHRITIS, SPONDYLOARTHRITIS AND OSTEOARTHRITIS**


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**3Aalborg University Hospital,**

**4Menoufi University,**

**5Aalborg, Denmark**

**Background:** Inflammatory and autoimmune diseases include multifactual path-omechanisms and systemic responses. The etiology of the joint diseases rheuma-toid arthritis (RA), spondyloarthritis (SpA) in relation to osteoarthritis (OA) remain incomplete and establishing the correct diagnose remains nontrivial. Advances in high-throughput molecular technologies have increased investigations into the
utility of transcriptomic, proteomic and high-density protein arrays approaches as diagnostic tools and companion diagnostics for precision medicine. To increase our understanding of the molecular pathogenesis, we extracted synovial fluid from the joints from multiple patient groups and characterized the protein composition in relation to plasma. Basic blood tests include inflammatory markers and autoantibodies, however, now analysis speed and robustness allow more readily clinical insight biofluids.

Objective: We present recent Omics concepts and studies investigating inflammatory state and treatment outcome in different biofluids from plasma to synovial fluid accessing causatives leading to inflammation and pain. Additionally, the aim was to investigate in any proteomic findings in synovial fluid can be correlated to proteomic changes in patient plasma and can be used as biomarkers for treatment effect.

Methods: Plasma and synovial fluid were investigated in multiple pathologies before and after treatment in patients (biologics; MTX; intraarticular gold). Deep proteomics, PTM and EV profiling were accomplished using quantitative proteomics approaches using quantitative mass spectrometry-based analysis by DIA/PASEF followed by deep datamining. All biological samples were digested according to a Filter Aided Sample Preparation (FASP) protocol before analysis with tandem mass spectrometry (MS/MS). PTM profiling were evaluated by 4D CCS based feature finding.

Results: Mass spectrometry based profiling allowed quantitative profiling of up to 480 proteins in matched synovial fluid and plasma. Complementary analysis by Olink proteomics, cytokine profiling and cell-free DNA. Multiple acute inflammatory proteins were more abundant in the RA synovial fluid, including proteins originating from neutrophil granulocytes, whereas SpA patients had a higher concentration of haptoglobin. Complementary analysis by Olink immunoassay identified significantly downregulated inflammation markers out of 96 tested in relation to antiinflammatory treatment.

Conclusion: Discovery of biomarkers and/or inflammatory signatures through integration of multi-omic data allowed stratify patients for improved treatment and prognosis. Firstly, our data using next generation proteomics approaches alleviates many pitfalls of missing values and poor proteome coverage including unbiased PTM profiling without enrichment strategies.

Disclosure of Interests: None declared.

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The sample size calculation was based on a prespecified equivalence margin of ±0.11 HAQ-DI points (i.e. 50% of half of the minimal important difference of 0.22 points) yielding a power of 99.2% for 60 enrolled patients. There was a wash-out period of 1-2 days between the two device registrations to minimise the potential carryover effect. A paired t-test was used to calculate the mean HAQ-DI score for the two devices and the difference in HAQ-DI score with a 95% confidence interval (CI). A Bland-Altman plot was used to assess limits of agreement (LoA).

Results: 60 patients (20 with RA, 20 with PsA and 20 with axSpA) were randomised of whom 51.7% were male. Mean age was 53.7 years (range 22-77) and mean disease duration was 12.5 years (range 1-34.8). Mean HAQ-DI was 0.608 (95% CI 0.437; 0.779) for the DANBIO app and 0.614 (95% CI 0.446; 0.783) for the touchscreen (Table 1). Agreement between scores obtained with the two devices is illustrated with Bland-Altman plots in figure 2A and 2B. The paired mean difference of HAQ-DI between the two devices was 0.006 (95% CI -0.042; 0.030); thus the 95% confidence interval for the mean difference was within the prespecified equivalence margin of ±0.11 HAQ-DI points.

Table 1. HAQ-DI scores, difference and LoA for the two devices.

<table>
<thead>
<tr>
<th>App</th>
<th>mean (SD)</th>
<th>Touchscreen</th>
<th>mean (SD)</th>
<th>Difference, mean (95% CI)</th>
<th>LoA, Missing values</th>
</tr>
</thead>
<tbody>
<tr>
<td>HAQ-DI (0-3)</td>
<td>0.608 (0.656)</td>
<td>0.614 (0.646)</td>
<td>-0.006 (-0.042; 0.030)</td>
<td>-0.277; 0.264</td>
<td></td>
</tr>
</tbody>
</table>

Conclusion: The current study showed no statistical or clinically important difference in HAQ-DI measurement captured by a smartphone app or outpatient touchscreen. Therefore, we feel confident that the two devices perform similarly enough to be used interchangeably in patients with inflammatory arthritis.