against targets not tested for in clinic. In this study, we aimed at extending the detection of autoantibodies by including all cytoplasmic aaRS in the analysis of patients with IIM. We hypothesized the existence of new potential autoantigens within the 20S protein subunit.

Methods: The presence of anti-aaRS autoantibodies was determined using a multiplex suspension bead array assay on 242 IIM patients from the Karolinska University Hospital myositis cohort. A panel of 186 recombinant constructs, representing 57 proteins that included full-length or partial sequence overlaps between constructs of all cytoplasmic aaRS as well as other myositis related proteins, were coupled to magnetic color-coded beads and each plasma sample was tested against the complete antigen panel.

Results: By the use of this multiplex method we identified patients with autoantibodies against many of the tested aaRS. Autoantibodies binding to HisRS have previously been shown to bind with higher reactivity to the WHEP domain of HisRS and this was also confirmed in this study. We confirmed reactivity against three of the other aaRS tested for in the clinic (PL-12, PL-7, and EJ). In addition, we identified patients positive for anti-Zo, -KS and -HA, autoantibodies usually not screened for in routine. Finally, our data indicates that there are autoantibodies binding to other aaRS than the previously known eight autoantigens, which will be presented.

Conclusion: In this study, we could detect autoantibodies in plasma from patients with IIM, both against the most common aaRS autoantigens, but also against other aaRS that are usually not tested for in clinic. We conclude that it is important to continue the studies of anti-aaRS autoantibodies, and their correlation to clinical manifestations, and in the long run also include more aaRS autoantigens in clinical practice.

References:

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SAT0290
THE INS AND OUTS OF EPIDERMAL DYSFUNCTION IN SYSTEMIC SCLEROSIS (SSC): RESULTS FROM A NOVEL TISSUE ENGINEERED EPIDERMAL EQUIVALENT FROM SSc KERATINOCYTES
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Background: Skin fibrosis is a hallmark of systemic sclerosis (SSc). It is commonly accepted that vascular damage, immune system activation and abnormal fibroblasts-to-myofibroblasts differentiation are pathological capital features. Nevertheless, recent evidence portrays a potential role of the epidermis in the pathogenesis of SSc skin fibrosis. This new angle on skin fibrosis pathogenesis is particularly attractive as the epidermis is an easy to access therapeutic target.

Objectives: Hence we investigated the Myostatin: Follistatin system in the serum as a reflection of early senescence in myositis as compared with healthy and diseased controls.

Methods: Patients with inflammatory myositis (ACR/EULAR criteria) presenting to the wards and outpatient clinic between December 2017 to August 2019 were recruited. Those with active infection, pregnancy, renal dysfunction or chronic infections were excluded. Serum Myostatin, Follistatin and their ratio were estimated in sera using ELISA (R&D systems, USA). Juvenile myositis and young adults (18-40 years) were subsequently analyzed separately. Non-parametric tests were used for paired and unpaired analysis. Results expressed as median.

Results: 95 myositis (8 Juvenile myositis, 26 DM, 10 PM, 29 Overlap, 2 NAM 1 CAM and 19 ASS) patients (23 Male and 72 Female) with median age 38 (24.5-46.0) years and disease duration 0.9 (2.3-5.1) years were included. Serum Myostatin was lower in IIM than in healthy control (HC) (153.5 vs. 243.6 p<0.0001, Fig 1A) but higher in IIM as compared with disease controls (153.5 vs 86.1, p=0.0174 Fig 1B). Serum Myostatin was comparable between juvenile and adult myositis and in the various subsets of adult myositis (Fig 1 C and D). Myostatin levels were higher in active as compared with inactive myositis in young adults (211.7 vs. 158.9, p=0.0149, Figure 1E). Serum Myostatin correlated with height (r 0.3, p=0.003) and weight (r 0.2, p=0.047) but not BM1 or muscle enzymes.

SAT0289
HIGH SERUM MYOSTATIN LEVELS SUGGEST ACCELERATED MUSCLE SENESCENCE IN ACTIVE IDIOPATHIC INFLAMMATORY MYOSITIS
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Background: Inflammation is the forerunner to fibrosis and premature ageing in various systemic diseases. Hence it seems plausible that idiopathic inflammatory myopathies (IIM) may exhibit accelerated senescence too.

Objectives: Hence we investigated the Myostatin: Follistatin system in the serum as a reflection of early senescence in myositis as compared with healthy and diseased controls.

Methods: Patients with inflammatory myositis (ACR/EULAR criteria) presenting to the wards and outpatient clinic between December 2017 to August 2019 were recruited. Those with active infection, pregnancy, renal dysfunction or chronic kidney disease were excluded. Patient from and disease variables, activity and damage were assessed using standard IIMQ score set measures. Patients in inception cohort were additionally followed up at 1 and 6 months. Myostatin and Follistatin were estimated in sera using ELISA (R&D systems, USA). Juvenile myositis and young adults (18-40 years) were subsequently analyzed separately. Non-parametric tests were used for paired and unpaired analysis. Results expressed as median.

Results: 95 myositis (8 Juvenile myositis, 26 DM, 10 PM, 29 Overlap, 2 NAM 1 CAM and 19 ASS) patients (23 Male and 72 Female) with median age 38 (24.5-46.0) years and disease duration 0.9 (2.3-5.1) years were included. Serum Myostatin was lower in IIM than in healthy control (HC) (153.5 vs. 243.6 p<0.0001, Fig 1A) but higher in IIM as compared with disease controls (153.5 vs 86.1, p=0.0174 Fig 1B). Serum Myostatin was comparable between juvenile and adult myositis and in the various subsets of adult myositis (Fig 1 C and D). Myostatin levels were higher in active as compared with inactive myositis in young adults (211.7 vs. 158.9, p=0.0149, Figure 1E). Serum Myostatin correlated with height (r 0.3, p=0.003) and weight (r 0.2, p=0.047) but not BM1 or muscle enzymes.