A209 SERUM LEVELS OF IFN- α DO NOT CORRELATE WITH DISEASE ACTIVITY IN PATIENTS WITH DERMATOMYOSITIS/POLYMYOSITIS

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Introduction Recent studies show an important role of type I interferon (IFN) in the proinflammatory process leading to disease manifestations in muscle tissue in patients with polymyositis (PM) and dermatomyositis (DM).

Aim To investigate the relationship of soluble IFN- α serum levels with clinical and laboratory characteristics in patients with PM and DM.

Methods Serum samples of 43 patients with DM (n=24) and PM (n=19) (32 females/11 males) were selected from a cohort of 81 PM/DM cases with the preference for those with anti-Jo-1 antibodies (n=26) and those with muscle MRI performed (n=22). IFN- α levels were measured using bead-based assay (human IFN- α FlowCytomix Simplex, Bender MedSystems, sensitivity 2.2 pg/ml). Clinical activities were assessed using the MYOACT tool. Degree of muscle oedema on STIR muscle MRI was used to measure local disease activity and evaluated using 10 cm visual analogue scale. Patients were either untreated (n=19), treated with prednisone at doses ≤ 20 mg (n=15), or >20 mg per day (n=9) at the time of blood collection. Healthy subjects (n=25) and patients with viral infection (n=6) were used as controls.

Results Significantly lower levels of IFN- α were found in sera of myositis patients in comparison with controls. No difference was noted between patients who were untreated, or treated with low or high prednisone. IFN- α levels were significantly higher in anti-Jo-1 positive patients (median 117.8

pg/ml, range 70–378) in comparison with anti-Jo1 negative (median 93.4 pg/ml, range 0–199) ($p=0.05$). There were no significant differences in IFN- α levels between PM and DM patients. Significant negative correlation was found between IFN- α and the intensity of MRI signal. None of the clinical or laboratory parameters as assessed by MYOACT showed any correlation with IFN- α levels, with the exception of the tendency to higher IFN- α levels ($p=0.064$) in the presence of interstitial lung disease.

Conclusion Serum levels of IFN- α in patients with PM and DM were lower compared to controls. This is not a consequence of the treatment since the levels of IFN- α were not different in patients tested before or during therapy. Serum levels of IFN- α do not seem to be an indicator of clinical activity; the lower the serum level the more severe the muscle oedema, as demonstrated by the intensity parameter on MRI. This could indicate rather local than systemic effect of IFN- α . Alternatively, other type I interferons and not the IFN- α may be responsible for the type I IFN signature that is characteristic for many DM and PM patients.