Methods Cynomolgus monkeys were randomly assigned to three treatment groups (saline, etanercept, and anti-TNF α Mab) by body weight and each group contained two male and two female monkeys. Abscess formation was induced with subcutaneous inoculation of *Staphylococcus aureus*.

Results Intravenous administration of anti-TNF α Mab on days -7, -3, 1, and 4 delayed the onset and reduced the incidence and the severity of *S. aureus*-induced abscess formation as compared with the saline-treated group at both 10⁹ and 10¹⁰ CFU/ml of bacterial inocula given on day 1. In contrast, no improvement in subcutaneous abscess formation as measured by swelling scores was observed in the receptor construct-treated animals as compared with the saline-treated animals when delivered with the same regimen at the same doses as anti-TNF α Mab. Rather, treatment with receptor construct resulted in a modest increase in the incidence and severity of abscess formation with inocula of 10⁹ and 10¹⁰ CFU/ml.

Conclusion Our results provide the initial evidence that a specific anti-TNF α monoclonal antibody therapy administered at 3 mg/kg provides a beneficial effect against *S. aureus*-induced infection as shown in this non-human primate model of abscess formation.

THU0042 **IN VIVO PRO-INFLAMMATORY AND ANTI-INFLAMMATORY CYTOKINES LEVELS IN PATIENTS WITH RHEUMATOID ARTHRITIS**

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Background Pro-inflammatory and anti-inflammatory cytokines play important roles in the rheumatoid activity process.

Objectives This study aims to demonstrate the cytokine profiles in rheumatoid arthritis (RA) patients with active and inactive disease process.

Methods Fifty-three Chinese rheumatoid arthritis patients (8M:45F) and 38 healthy individuals (19M:19F) were enrolled into the study. RA patients were classified as having active disease or in remission according to clinical and serological criteria (morning stiffness, joint pain, joint tenderness, joint swelling, ESR, CRP). At venepuncture, the patients and healthy controls did not have any active infective process. All except 3 patients (in remission) were on SAARDs. Sera obtained in the morning were assayed for the following cytokines: TNF α , IFN γ , TGF β , IL-2, IL-4, IL-6, IL-8, IL-10, IL-12 and for rheumatoid factor (RF) by the ELISA method. HLA-DRB1*04 typing was performed using PCR-RFLP.

Results Forty-two patients had active disease and 11 patients were in remission. Rheumatoid factor was detected in 28 patients and HLA-DR*0405 allele was significantly elevated in RA patients ($p < 0.001$). TNF α , IL-6 and IL-8 were significantly elevated in RA patients when compared to controls ($p < 0.01$, $p < 0.001$, $p < 0.018$, respectively). Patients with RF positivity were noted to have much higher levels of these 3 pro-inflammatory cytokines. There was however no association with HLA-DR*0405. The anti-inflammatory TGF β was noted to be significantly lower in active patients when compared to inactive patients or controls ($p < 0.05$).

Conclusion In conclusion, the serum pro-inflammatory cytokines were significantly elevated in RA patients irrespective of their clinical disease activity while serum TGF β was significantly

depressed in active RA patients and normalises when in remission. The exact role that TGF β plays in RA is yet to be fully elucidated.

THU0043 **THE EXPRESSION OF CYCLOOXYGENASE, LIPOXYGENASE AND NEUROPEPTIDES IN MUSCLE TISSUE OF PATIENTS WITH POLYMYOSITIS AND DERMATOMYOSITIS**

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Background

Objectives To investigate pathogenesis of inflammation in muscles of patients with idiopathic inflammatory myopathies (IIM) – dermatomyositis (DM), polymyositis (PM) by studying expression of cox, lox and neuropeptides, using *in situ* hybridization and immunohistochemistry.

Methods Muscle tissue samples were obtained by needle biopsy from affected and non-affected sites distinguished by MRI-STIR technique. The second biopsy was performed after treatment. For studying cox, lox we used *in situ* hybridization with mRNA probes – antisense cox1, 2 and lox 5. Neuropeptides, such as substance P (SP), calcitonin gene related peptide (CGRP), vasoactive intestinal peptide (VIP) and neuropeptide Y (NPY), were evaluated by using immunohistochemistry.

Results 11 patients with DM or PM (9 female, 2 male) were examined. The expression of all investigated molecules (cox1, cox2, lox5, CGRP, SP, VIP, NPY) was detected on all muscle tissue. mRNA expression of cox and lox was increased in affected tissue (detectable expression was on 47 sections of 53–89%) in comparison with non-affected (40 sections of 64 $\pm$ 63%) and tissue after treatment. Expressing cells were muscle cells, inflammatory cells and vessels. Cox1 expression was found mainly on inflammatory cells and vessels, cox2 on muscle cells, inflammatory cells and vessels, lox5 mainly on muscle cells. Expression of neuropeptides was detected mainly on vessels, not on nerve cells. SP and CGRP were expressed in all tissues, VIP and NPY mainly in affected tissue. CGRP was expressed on smaller and thicker vessels in non-affected tissue and on bigger and thinner vessels in affected tissue, SP on smaller and thicker vessels in affected tissue. VIP and NPY staining was weak.

Conclusion Expression of all investigated molecules was observed in all muscle tissue samples from IIM patients. This expression was increased in affected tissues compared with non-affected and tissue after treatment, which indicates the role of cox, lox and neuropeptides in pathogenesis of IIM. Expression of neuropeptides was detected on vessels, not on nerve cells that suggests activation of vascular system in inflamed tissue.

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THU0044 **METHOTREXATE INHIBITS EX-VIVO IFN-GAMMA PRODUCTION IN WHOLE BLOOD OF RHEUMATOID ARTHRITIS PATIENTS**

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