detected in 50% of ACPA-positive RA, 20% of ACPA-negative RA, and in 37% of non-RA controls ($p<0.0001$). Notably, this antibody response was citrulline-specific, as the antibody response against the arginine-containing control peptide RPP3 was significantly lower in all subsets ($p<0.0001$). Among 55 mAbs from gingival tissue, 14 (25%) unique clones were CPP3-reactive, of which 4 showed cross-reactivity with RPP3. Interestingly, 4 out of 14 (29%) CPP3-reactive clones also bound citrullinated peptides derived from human a-enolase, filaggrin and histone 4, demonstrating cross-reactivity between a bacterial epitope and human epitopes on a monoclonal level.

Conclusions This study shows that a substantial proportion of systemically healthy individuals possess ACPAs directed against *P.gingivalis*, and these ACPAs also bind epitopes on human proteins. Based on our data, we propose that the ACPA response may be triggered by *P.gingivalis* via an antibody response against CPP3, which cross-reacts with human citrullinated proteins by mechanisms of molecular mimicry.

Disclosure of Interest None declared.

P043 INFECTION WITH CITRULLINATING PORPHYROMONAS GINGIVALIS CAN INDUCE AUTOIMMUNITY TO HUMAN RIBOSOMAL PROTEINS

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Career situation of first and presenting author Young investigator.

Introduction Porphyromonas gingivalis (P g.) is involved in triggering self-reactive immune responses when citrullinating bacterial or human proteins. However, first evidence to link anti-ribosomal T and B cells responses to rheumatoid arthritis (RA) has been published but the mechanism is still not clear.[1] Infection based autoimmunity induced by citrullination of human proteins with P g. peptidyl arginine deiminase from RA patient (RA-PPAD) and crossreactivity binding induced by P g. was investigated using affinity purified RA patient antibodies and a monoclonal antibodies to cit-RA-PPAD.

Objectives Antibodies to RA-PPAD isolated from an RA patients (RA-PPAD) was first time linked to target specific citrullinated ribosomal proteins.

Methods Screening of RA sera was conducted on 37.830 unique human proteins on protein marcoarrays (http://www.engine-gmbh.de) with 30 RA sera. The autoantibody response to 840 different proteins was recorded and bioinformatically evaluated. Protein arrays were citrullinated with PAD2,4,rabbit PAD and RA-PPAD. Sera and affinity purified antibodies were used to detect reactivity to 840 autoantigens.

Results A human protein macroarray consisting of 840 indentified autoantigens from RA patients was modified by human PAD2 and PAD4, rabbit PAD, and RA-PPAD form P g..Using cit specific monoclonal antibodies we identified the ribosomal proteins (RP), RPL18a, RPS27a, modified by PAD2, RPL18a and MRPS11 modifies by Pad4, and RPL7L1 modified by rabbit PAD specifically targeted. In addition 6 RA patient sera and 3 osteoarthritis (OA) control sera were used to identify the citrullinated RA-PPAD specific modified autoantigens not targeted when modified by human PAD2 or PAD4 or rabbit Pad. We identified the RA-PPAD citrullinated ribosomal Proteins RPL3, RPL21, RPS24, RPL9, RPL15, RPS24.RPS3a, MRPL28 specifically targeted by RA patients. This identifies ribosomal proteins as major specific RA-PPAD citrullination targets. Moreover, affinity purified antibodies bound to native and citrullinated RA-PPAD from 6 RA patient sera and 3 OA patient sera were tested for crossreactvity on citrullinated human proteinarray. Antibodies to citrullinated ribosomal proteins MRPS11, RPL21, RPS3a, RPL18a, RPS27a, MRPL28 were detected in the RA group but not in the OA control group.

Conclusions Failure of *Porphyromonas gingivalis* clearance in RA patients leads to infection induced enzymatic mimicry based autoreactivity targeting evolutionary conserved human ribosomal proteins. Autoimmunity to ubiquitous self-antigens may trigger localized tissue damage in RA.

REFERENCE

1. Ito Y, *et al*. Detection of T cell responses to a ubiquitous cellular protein in autoimmune disease. *Science* 2014;**346**:363–368.

Disclosure of Interest None declared.

P044 EFFECT OF SPECIALIZED 6-MONTH ADL TRAINING WITH SUBSEQUENT A 6-MONTH FOLLOW-UP PERIOD IN PATIENTS WITH MYOSITIS – PRELIMINARY DATA

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Career situation of first and presenting author Student for a master or a PhD.

Introduction Idiopathic inflammatory myopathies (IIM) are accompanied by muscular weakness caused by inflammation and immune changes in affected muscles, resulting in reduced quality of life. The aim of our study was to determine the effect of ADL training on muscle strength and endurance and quality of life of IIM patients.

Objectives The study included a total of 50 IIM patients who fulfilled the Bohan and Peter 1975 criteria and had skeletal muscle involvement. 27 patients were recruited into the intervention group (IG) and 23 patients into the control group (CG). Both groups received an educational material for home exercise, but only the IG underwent a 6 month intervention with a subsequent 6 month follow-up period.

Methods Patients were assessed by a physician and a physiotherapist blinded to intervention at months 0, 3, 6, and 12, and parameters evaluating quality of life, muscle strength and endurance were recorded. Patients also filled out PRO (patient reported outcomes) questionnaires and provided blood for routine laboratory analysis and bio-banking. Data analysis was performed between groups and within the group.

Results Compared to the observed statistically significant deterioration in the CG over the intervention period of 0–6 months, we found a statistically significant improvement in the IG in objectively assessed strength and endurance of muscles as well as in subjectively assessed functional abilities