comparison of studies difficult. In particular, there are limited data describing a standardised scanning method and standardised definitions of US gland pathologies. Even if we obtained truth validity, discrimination validity (reliability) is not yet validated. Therefore, although SG-US is an upcoming modality in rheumatology, it is not yet ready to make diagnostic or therapeutic decisions regarding salivary glands in daily routine practice. It should still be considered to be a research tool, until we are able to demonstrate acceptable intra- and interobserver reliabilities. Preliminary work addressing these issues has been done so far by the USpSS study group consisting of both clinicians and ultrasonographers.^{6,7}

More recently, a SG-US OMERACT task force group composed of international clinicians and ultrasonographers was created with the aim of developing a standardised scanning method using standardised definitions of gland pathology. A consensual Delphi process about definitions and scoring using B mode modality of SG-US was used and the intra and inter reliabilities between 18 sonographers showed good to excellent reliabilities (Light's kappa=0.81, Light's kappa=0.71 respectively) (article in preparation). Currently, there is an unmet need concerning the use of SGUS in the monitoring of patients. Even if it seems that structural damage are stable over time.⁸ Another possible way to study SG in the disease course is to use Doppler. Doppler waveform analysis was found useful for detecting blood flow abnormalities in SG of patients with pSS compared to controls.⁹ Given the importance of parotid and submandibular glands involvement in pSS, we believe that the vascularisation and B mode sonography should be evaluated routinely. A new US technique measuring the elasticity of SG parenchyma using elastography has recently emerged and could be implemented in the evaluation of SGUS pSS patients.¹⁰⁻¹² Some authors showed that stiffness of SG parenchyma was increased compared to healthy controls and suggested to adjunct this procedure to gray-scale ultrasonography for the clinical diagnosis of pSS. The last important challenge is to know if imaging techniques are capable to replace minor salivary glands biopsy. A recent study¹³ has shown good agreement between SGUS and parotid gland and moderate with labial glands.

In conclusion, US has nearly completed the 3 pillars of the OMERACT process (truth validity, discrimination validity and feasibility). The use of different imaging technique and particularly ultrasonography should be educated and it is now of importance to train the rheumatologists to this technique as proposed by The US EULAR courses.

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THURSDAY, 14 JUNE 2018

How monogenetic autoinflammatory diseases help to understand and treat rheumatic diseases

SP0051 CLINICAL PRESENTATION OF AUTOINFLAMMATORY DISEASES IN CHILDREN AND ADULTS: HOW NOT TO MISS THESE PATIENTS

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Autoinflammatory diseases are diseases of the innate immune system, characterised by attacks of inflammation. The inflammatory attacks can manifest in a variety of forms; fever, is frequent, the skin and musculoskeletal system is often affected. Acute phase reactants are increased in almost all of the patients. The autoinflammatory diseases can be classified according to their leading manifestations such as: those with periodic fevers (and other various features), those with pyogenic

features, those with psoriasis, those with features of interferopathies and those with vasculopathy. With this large range of features these diseases are in the differential of many common diseases. Genetic testing is confirmatory but may not be widely available.

The differentiating features of especially the common autoinflammatory diseases will be reviewed.

Disclosure of Interest: S. Ozen Consultant for: Novartis, Speakers bureau: SOBI

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THURSDAY, 14 JUNE 2018

Joint EULAR – EFIS session: I've got a B in my bonnet

SP0052 POSTTRANSLATIONAL MODIFICATION OF AUTOANTIBODIES IN RA

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Rheumatoid Arthritis (RA) is a common autoimmune disease in which autoantibodies serve as interesting biomarkers. In a large proportion of the patients several types of autoantibodies are present that target post-translationally modified proteins. Next to antibody responses against citrullinated proteins (ACPA) we now also describe antibody responses against carbamylated proteins (anti-CarP) in RA. The use of these autoantibodies in the prediction, diagnosis and prognosis will be discussed, as well as the clues, obtained in animal models, as to how such an antibody response may be initiated.

In addition the findings that antibodies themselves are also post-translationally modified and the functional consequences of such modifications will be highlighted both regarding carbamylation of IgG and modifications of ACPA IgG.

Disclosure of Interest: None declared

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SP0053 MEMORY PLASMA CELLS IN CHRONIC INFLAMMATORY DISEASES – A ROADBLOCK TO TOLERANCE INDUCTION

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Regeneration of tolerance is the Holy Grail of treating inflammatory rheumatic diseases. Obviously neither physiological nor therapeutic immunosuppression does achieve this for most patients. What are the roadblocks for therapies inducing therapy-free remission? We have identified memory plasma cells secreting pathogenic (auto)antibodies as one such roadblock. Such cells are often generated already before disease becomes clinically apparent. They persist over time in inflamed tissues and in the bone marrow, in niches which protect them from environmental stress, including conventional therapies. By constitutive secretion of pathogenic antibodies they fuel relapse and refractoriness. Any therapy aiming at induction of therapy-free remission will have to remove this roadblock. Recently we and others have developed therapeutic concepts to ablate memory plasma cells, based on a molecular understanding of their lifestyle. Such therapies, however, have two major inherent problems:¹ they also ablate protective humoral immunity, increasing risk of infection, and ² the pathogenic memory plasma cells can be regenerated, if tolerance is not regenerated. To address the latter point, ablation of plasma cells has to be combined with tolerance-inducing therapeutic options, and until then, patients have to be protected passively by IV-Ig. For selective targeting of pathogenic memory plasma cells, differences in the lifestyle between protective and pathogenic plasma cells could be exploited, but few have been identified so far. We have now developed a novel technology to target plasma cells according to the specificity of the antibodies they secrete. Plasma cells are labelled *in vivo* with a given antigen, and those plasma cells secreting specific antibodies are selectively killed by their own antibodies. We have now demonstrated feasibility and efficacy of this therapeutic approach in a preclinical model. Apart from the therapeutic perspective, this technology also offers a unique option to identify pathogenic plasma cell specificities in experimental and clinical rheumatology.

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