However, the approach based on interfering with TNF exceeds simple neutralisation of inflammatory cytokine and, possibly, this is why the prediction of response remains elusive. High costs of the treatment and irreversible tissue damage in non-responders have forced multiple attempts to predict response to anti TNF treatment using clinical, laboratory and molecular markers. There are several issues related to this type of studies that may include genetically heterogeneous groups of patients, different types of anti-TNF treatment, multiple types of response measures and are typically underpowered. Nowadays, an enormous amount of genetic, epigenetic and genomic data revived new expectations and stimulates producing of models for prediction of anti TNF response. We performed several studies based on different omics data in genetically homogeneous Swedish population that raises importance of disease subgrouping and interference of environmental factors on prediction values.

Disclosure of Interest: None declared

DOI: 10.1136/annrheumdis-2018-eular.7745

SATURDAY, 16 JUNE 2018

How do you sleep?

SP0170 RE-EVALUATE LIFE WHEN BROKEN SLEEP HAS A NEGATIVE EFFECT ON INFLAMMATORY ARTHRITIS

B.A. Esbensen. Rigshospitalet – Glostrup, Copecare, Centre for Rheumatology and Spine Diseases and Research Unit, Glostrup, Denmark

Despite improved possibilities for early diagnosis and medical treatment rheumatoid arthritis (RA) still causes stiffness and swelling in the joints. Poor sleep, chronic pain, fatigue, reduced physical function, depression and reduced quality of life are consequences of these symptoms and the inflammation is.¹⁻³ About 60%–80% of patients with RA report poor sleep compared to 10%–30% in the background population.⁴⁻⁶ Patients with RA indicate sleep as one of the most important parameters evaluating their medical treatment with anti-rheumatics, which indicate the impact sleep can have on physical and mental well-being.⁷ Common meanings of good and bad sleep in a healthy population is characterised by subjective satisfaction, appropriate timing, adequate duration, high efficiency, and sustained alertness during waking hours.⁸ Thereby, sleep health is seen as a multidimensional pattern of sleep-wake-fulness, adapted to individual, social, and environmental demands, that promotes physical and mental well-being. Initially, this lecture will focus on “state of the art” regarding sleep and inflammatory joint diseases. Also different suggestions of non-pharmacological treatments targeting improved sleep quality will be presented.

Subsequently, experiences from a nurse-led sleep outpatient clinic which was established in 2016 in the Department of Rheumatology and Spine diseases, Rigshospitalet – Glostrup, will be presented focusing on how the clinic is organised and how specialised nurses are meeting patients with poor sleep with non-pharmacological interventions. We have systematically collected data about the patients sleep when included in the clinic for treatment and when discharged from the clinic. We will present results from these patients.

REFERENCES:

- [1] Uhlig, T., et al., *Rheumatoid arthritis is milder in the new millennium: health status in patients with rheumatoid arthritis 1994–2004*. Ann Rheum Dis, 2008. **67**(12): p. 1710–5.
- [2] Hewlett, S., et al., *‘I’m hurting, I want to kill myself’: rheumatoid arthritis flare is more than a high joint count—an international patient perspective on flare where medical help is sought*. Rheumatology (Oxford), 2012. **51**(1): p. 69–76.
- [3] Carr, A., et al., *Rheumatology outcomes: the patient’s perspective*. J Rheumatol, 2003. **30**(4): p. 880–3.
- [4] Loppenthin, K., et al., *Sleep quality and correlates of poor sleep in patients with rheumatoid arthritis*. Clin Rheumatol, 2015. **34**(12): p. 2029–39.
- [5] Goes, A.C.J., et al., *Rheumatoid arthritis and sleep quality*. Rev Bras Reumatol Engl Ed, 2017. **57**(4): p. 294–298.
- [6] Abbasi, M., Z. Yazdi, and N. Rezaie, *Sleep disturbances in patients with rheumatoid arthritis*. Niger J Med, 2013. **22**(3): p. 181–6.
- [7] Kirwan, J.R., et al., *Incorporating the patient perspective into outcome assessment in rheumatoid arthritis—progress at OMERACT 7*. J Rheumatol, 2005. **32**(11): p. 2250–6.
- [8] Dickerson, S.S., K.J. K.J. Klingman, and C.R. C.R. Jungquist, *Common meanings of good and bad sleep in a healthy population sample*. Sleep Health, 2016. **2**(3): p. 253–259.

Disclosure of Interest: None declared

DOI: 10.1136/annrheumdis-2018-eular.7675

SP0171 SLEEP DISTURBANCES IN PRIMARY SJÖGREN'S SYNDROME: EVIDENCE FROM THE LITERATURE, PATIENT SLEEP DIARIES AND A QUALITATIVE FOCUS GROUP STUDY

K.L. Hackett^{1,2,3}. ¹Department of Social Work, Education and Community Wellbeing, Northumbria University; ²CRESTA Fatigue Clinic, Newcastle upon Tyne NHS Foundation Trust; ³Musculoskeletal Research Group, Institute of Cellular Medicine, Newcastle University, Newcastle upon Tyne, UK

Primary Sjögren's syndrome is a systemic autoimmune disease which targets secretory glands resulting in dryness. Extra-glandular features include fatigue, pain and sleep disturbances. There are few studies exploring the specific sleep disturbances experienced by PSS patients; the impact of these disturbances or the potential acceptability of interventions to address some of these problems. In this talk, I will present work which begins to map the landscape of sleep disturbances in PSS:

Firstly, a systematic review of the literature explores sleep disturbances in PSS patients and identifies particular sleep symptoms which are problematic in these patients.¹

Secondly, I will explore the relationship between daytime sleepiness (hyper-somnolence) and other clinical parameters in patients recruited to the UK Primary Sjögren's Syndrome Registry.

Thirdly, I will report on sleep diary data from 30 patients attending a multidisciplinary fatigue clinic in the North East of England.

Finally, I will present findings from focus groups conducted with PSS patients and their partners. In this qualitative study, we explored the impact of sleep disturbances on patients and their families and potential acceptability of a non-pharmacological intervention (cognitive behavioural therapy for insomnia) to address specific sleep disturbances.²

REFERENCES:

- [1] Hackett KL, Gotts ZM, Ellis J, Deary V, Rapley T, Ng W-F, Newton JL, Deane KHO. An investigation into the prevalence of sleep disturbances in primary Sjögren's syndrome: a systematic review of the literature. Rheumatology (Oxford). 2017;56(4):570–80.
- [2] Hackett KL, Deary V, Deane KHO, Newton JL, Ng W-F, Rapley T. Experience of sleep disruption in primary Sjögren's syndrome: A focus group study. British Journal of Occupational Therapy. 2018;0(0):0308022617745006.

Disclosure of Interest: None declared

DOI: 10.1136/annrheumdis-2018-eular.7724

SP0172 WHAT EFFECT DOES EXERCISE HAVE ON SLEEP IN RMD?

S.G. McKenna. Discipline of Physiotherapy, School of Allied Health, University of Limerick, Limerick, Ireland

Circadian rhythms are physical, mental and behavioural changes that follow a daily cycle. Sleep is an essential aspect in maintaining the body's circadian rhythm and maintain health-related quality of life (HQoL) therefore, sleep disturbances can have a detrimental impact on same. The Outcome Measures in Rheumatology (OMERACT) has identified sleep as one of the key outcomes important to RMD patients.¹ Patients with various immune-mediated inflammatory diseases, including rheumatoid arthritis, have reported disturbed sleep and reduced sleep duration, further adding to their disease burden.^{2, 3} It has been well established that being physically active and taking regular exercise are important for those who have been diagnosed with RMD's.⁴ Exercise has been identified as an important part of the nonpharmacological management of poor sleep duration and in improving sleep quality however, in a 2013 Cochrane review that examined exercise and fatigue in RA, it was noted by the authors that sleep quality was yet to be examined in this population.⁵ Moreover, it is known that in general exercise improves mood state, which can also be an additional factor in improving sleep duration and sleep quality.⁶

Exercise offers a potentially attractive alternative or adjuvant treatment for those people with RMD who have sleep issues. The position stand from the American College of Sports Medicine (ACSM) regarding exercise for those with chronic conditions is categorised by cardio-respiratory exercise, resistance exercise, flexibility exercise and neuro-motor exercise. This presentation will consider the evidence regarding the effect exercise has on sleep in general and how the examination of a participants exercise habits prior to any study might be important, as it may have an impact on its effectiveness. A key message from the talk will be the presentation of evidence of exercise programmes in people with RMD, according to the Frequency, Intensity, Time and Type (FITT) principle will be presented.⁸

REFERENCES:

- [1] Kirwan JR, Boonen A, Harrison MJ, Hewlett SE, Wells GA, Singh JA, Furst DE, Dworkin RH. OMERACT 10 Patient Perspective Virtual Campus: Valuing health; measuring outcomes in rheumatoid arthritis fatigue, RA sleep, arthroplasty, and systemic sclerosis; and clinical significance of changes in health. *The Journal of Rheumatology* 2011;38:1728–34.
- [2] Graff LA, Walker JR, Russell AS, Bissonnette R, Bernstein CN. Fatigue and quality of sleep in patients with immune-mediated inflammatory disease. *The Journal of Rheumatology Supplement* 2011;88:36–42.
- [3] McKenna S, Donnelly A, Fraser A, Kennedy N. Sleep and physical activity: a survey of people with inflammatory arthritis and their engagement by health professionals in rheumatology in Ireland. *Disability and Rehabilitation* 2017;1–7.
- [4] Hurkmans E, van der Giesen FJ, Vliet Vlieland TPM, Schoones J, Van den Ende ECHM. Dynamic exercise programs (aerobic capacity and/or muscle strength training) in patients with rheumatoid arthritis. *The Cochrane Library* 2009.
- [5] Nicassio PM, Ormseth SR, Kay M, Custodio M, Irwin MR, Olmstead R, Weisman MH. The contribution of pain and depression to self-reported sleep disturbance in patients with rheumatoid arthritis. *Pain* 2012;153:107–12.
- [6] Cramp F, Hewlett S, Almeida C, Kirwan JR, Choy EHS, Chalder T, Pollock J, Christensen R. Non-pharmacological interventions for fatigue in rheumatoid arthritis. *The Cochrane Library* 2013.
- [7] Moore G, Durstine JL, Painter P, American College of Sports M. ACSM's Exercise Management for Persons With Chronic Diseases and Disabilities, 4E. *Human Kinetics* 2016.
- [8] McKenna S, Donnelly A, Fraser A, Comber L, Kennedy N. Does exercise impact on sleep for people who have rheumatoid arthritis? A systematic review. *Rheumatology International* 2017;37:963–74.

Disclosure of Interest: None declared

DOI: 10.1136/annrheumdis-2018-eular.7716

SATURDAY, 16 JUNE 2018

Stratifying connective tissue diseases

SP0173 STRATIFYING PRIMARY SJÖGREN'S SYNDROME PATIENTS

W.-F. Ng. *Institute of Cellular Medicine, Newcastle University, Newcastle Upon Tyne, UK*

The clinical manifestations of primary Sjögren's syndrome (PSS) vary considerably between individual patients. The pathogenesis of PSS is not fully understood and although many aberrant biological pathways have been identified, the relationships between these pathways and clinical manifestations remain unclear. Furthermore, there is substantial heterogeneity in long-term outcomes and in health economic cost among patients with PSS. Stratification of PSS into more homogeneous subsets will facilitate better understanding of the pathogenesis of the disease, development of more cost-effective management strategies and accelerate therapeutic development programmes of targeted therapies in PSS. The establishment of several large cohorts of clinically well characterised PSS cohorts with associated biobanked materials has provided a rich resource for stratified medicine research in PSS. The growing number of datasets from clinical trials in PSS provide an excellent opportunity to evaluate the potential impact of stratified approach to therapy development in PSS.

In this presentation, different approaches to identify clinically and biologically meaningful PSS subsets will be considered. In addition, the key challenges and potential future directions in Stratified Medicine in PSS will be discussed.

Disclosure of Interest: W.-F. Ng Grant/research support from: ElectroCore, Resolve Therapeutics, Nascient, Consultant for: GlaxoSmithKline, Novartis, Abbvie, MedImmune

DOI: 10.1136/annrheumdis-2018-eular.7701

SP0174 THE VALUE OF TYPE I INTERFERON SIGNATURE TO STRATIFY PATIENTS

L. Rönnblom. *Uppsala University, Uppsala, Sweden*

Since the first reports 2003 of an increased expression of type I interferon (IFN) regulated genes in cells from patients with SLE, a number of studies have demonstrated that many systemic inflammatory autoimmune diseases display this over-expression of type I IFN regulated genes, now known as an IFN signature. This is

true for a majority of patients with SLE, but also large proportion of patients with primary Sjögren's syndrome, myositis, systemic sclerosis and rheumatoid arthritis (RA). The so called type I interferonopathies is a group of rare Mendelian diseases with a very strong IFN signature. Other autoimmune diseases, such as type I diabetes, also display an IFN signature. Type I IFN seems to be most important for inducing the IFN signature, but it's clear that type II and type III IFNs can contribute to the IFN signature. Besides the expression of type I IFN regulated genes, recent studies have shown that patients with SLE and other autoimmune disease, have epigenetic changes in IFN regulated gene, which are hypomethylated. Thus, there are strong evidences that type I IFN system activation is a key event in many autoimmune diseases. Can the IFN signature help us to stratify patients in order to predict risk for major organ manifestations, disease outcome or response to treatment? Early studies showed that patients with active disease and more severe disease manifestations usually have a more prominent IFN signature, which still is true. However, recent studies have revealed that the IFN signature needs to be combined with a broader analysis of expressed genes. For instance, a plasmablast signature seems to correlate best with SLE disease activity and a neutrophil signature associates with lupus nephritis. For treatment stratification, it's perhaps not surprising that SLE patients with a high IFN signature have a better treatment response to type I IFN inhibition, compared to IFN low patients. At least when type I IFN receptor inhibition has been used to downregulated the type I IFN response. In RA, the IFN signature at baseline predict a poor response to TNF-alpha treatment as well as nonresponse to B-cell depleting therapy. In conclusion, there are several indications that the type I IFN signature is a valuable biomarker for patient stratification. However, we are just at the brink of the understanding how to apply this signature in clinical practice.

Disclosure of Interest: L. Rönnblom Grant/research support from: AstraZeneca, Consultant for: UCB, AstraZeneca, Speakers bureau: Biogen

DOI: 10.1136/annrheumdis-2018-eular.7782

SP0175 HETEROGENEITY OF MYOSITIS AND THERAPEUTIC CONSEQUENCES

J. Vencovsky. *Institute of Rheumatology, Prague, Czech Republic*

Idiopathic inflammatory myopathies, or myositis, are a heterogeneous group of chronic inflammatory disorders affecting striated muscle leading to muscle weakness and impaired function. In addition, other organs are variably involved including the skin, lungs, heart, joints, and the gastrointestinal tract contributing to morbidity and mortality and to low health-related quality of life. Treatment decisions are often guided by the most prominently affected organ. The best recognised disease subsets are polymyositis (PM), dermatomyositis (DM), juvenile PM and DM, sporadic inclusion body myositis (sIBM), and immune-mediated necrotizing myopathy (IMNM). Muscle specific autoantibodies and muscle associated autoantibodies are frequently found in different forms of myositis and collectively they can be detected in approximately 80% adults and 60% children with myositis. They can identify homogeneous subsets, and have both diagnostic and prognostic value. The worst functional outcomes in terms of muscle involvement are often seen in patients with sIBM and with anti-SRP antibodies. sIBM patients are notoriously non-responsive to any treatment. Patients with anti-SRP display necrotizing myopathy in the muscle biopsy and treatment effect is usually low. In some patients an improvement or at least stabilisation can be achieved with the use of rituximab. The other patients with IMNM are those with anti-HMGCR antibodies and with statin medication history. Here the response to glucocorticoids (GC) and immunosuppressives (IS) is mostly good, although some patients require treatment with intravenous immunoglobulins (IVIg) to which they respond very well even without concomitant GC. Patients with antisynthetase syndrome are characterised by the presence of interstitial lung disease, which may be in some cases the prevailing manifestation of the disease with only mild or none muscle weakness. Treatment is often successful with calcineurin inhibitors, mycophenolate mofetil (MMF), cyclophosphamide, or rituximab in these patients. Subgroup of patients with anti-NXP2 antibodies is characterised with strong association with calcinosis, both in children and adults, and the treatment represents a difficult problem. The effect was reported with the use of calcium channel blockers, warfarin, bisphosphonates, sodium thiosulfate, aluminium hydroxide, probenecid, colchicine as well as IVIGs, rituximab, abatacept, infliximab, and thalidomide. In any case, patients need to be treated aggressively if calcinosis progresses. Muscle disease in dermatomyositis is often responsive but the treatment of cutaneous manifestations may occasionally prove difficult. Antimalarials are usually prescribed first, sometimes in combination with systemic treatment using IVIGs, tacrolimus, MMF, or other IS. Patients with anti-MDA5 antibodies often develop clinically amyopathic or hypomyopathic DM, but at the same time their ILD can be rapidly progressive. Aggressive treatment with high dose GC and IS needs to be started early to increase the chance for survival. A proportion of patients with myositis manifests high type I interferon signature and the clinical trial suggests that these are the patients who benefit most from the treatment with interferon type I inhibition. In the future careful considerations based on the disease subsets,