Although the literature on the outcomes of total hip and knee arthroplasty (THA and TKA) is vast, relatively little is known about physical activity levels of OA patients undergoing surgery.
This presentations summarizes the state-of-the-art literature on the physical (in) activity level of patients with an indication for TKA or THA and its determinants, and the changes of physical (in)activity levels after surgery. These results are presented taking into account the activity levels of the general population. The presentation will end with an informal quiz on physcial (in)activity in THA and TKA patients.

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[2] Mills K, et al. Physiotherapy. 2019 MAR;105(1):35-45.
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Disclosure of Interests: None declared
DOI: 10.1136/annrheumdis-2019-eular.8482

SP0051 **CASE 2 DISCUSSANT: DISCUSSION OF HOW A LARGE PROPORTION OF PEOPLE DO NOT BECOME ACTIVE AFTER JOINT REPLACEMENT DESPITE ADEQUATE PAIN RELIEF, REASONS, NEGATIVE CONSEQUENCES OF REDUCED ACTIVITY AND POSSIBLE APPROACHES**

Thea Vliet Vlieland. *Leiden University Medical Center, Orthopaedics, Rehabilitation and Physiotherapy, 2300 RC Leiden, Netherlands*

The literature on the effect of total hip and knee arthroplasty (THA and TKA) on the amount of physical activity is scanty, with various studies suggesting that physical activity levels do not increase after surgery. These results raise several questions, such as: Are patients who will undergo or underwent THA or TKA more or less physically active than the general population? And, if not, should they become more physically active, what are barriers and facilitators for physical activity levels, what would be the potential benefits? Who are the stakeholders that should be involved in the promotion of physical activity in this patient group? These, and other questions will be highlighted in an interactive discussion with the attendees of the session.

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[1] Pellegrini CA, et al. Disabil Rehabil. 2018;40:2004-2010.
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[3] Hodges A, et al. Clin Rehabil. 2018;32:1271-1283.

Disclosure of Interests: None declared
DOI: 10.1136/annrheumdis-2019-eular.8618

THURSDAY, 13 JUNE 2019

13:30:00 – 15:00:00

My joints hurt and I'm overwhelmingly tired – fatigue in rheumatoid arthritis

SP0052 **FATIGUE IN RHEUMATOID ARTHRITIS: WHAT IT IS AND HOW TO ASSESS IT?**

José Antonio P. da Silva. *Centro Hospitalar e Universitário de Coimbra, Rheumatology, Coimbra, Portugal*

Background: Fatigue is a frequent and important symptom in rheumatoid arthritis (RA), and it is associated with significantly reduced health-related quality of life, thus contributing to the impact of disease upon patients' lives.
Objectives: To collect, summarise and interpret available evidence on the nature of fatigue and the best ways to assess it in Rheumatoid Arthritis
Methods: A systematic literature research was performed in trying to (i) to synthesize the role of fatigue in the global impact of rheumatoid arthritis; (ii) describe validated instruments and their psychometric properties as measures of fatigue among patients with RA; and finally (iii) propose a clinically meaningful, valid and feasible process to measure fatigue in clinical practice.
Results: Fatigue has a major relevance in the overall burden of disease in RA. Several instruments have been validated o measure it, but no consensus has yet been reached to recommend a "gold-standard".
Conclusion: Although fatigue is recognized by the rheumatology community as an important consequence of RA and a major gap in its current management of the disease, it has not been easy to measure and grasp. The problem seems to reside in the multidimensional causality and subjetive nature of this phenomenon, which may warrant dedicated measures and interventions.
Disclosure of Interests: None declared
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SP0053 **HOW TO PREVENT AND MANAGE FATIGUE IN PATIENTS WITH RA**

James Galloway. *King's College London, Inflammation Biology, London, United Kingdom*

Background: Fatigue is, similar to pain, a subjective symptom that occurs in many different diseases, particularly in patients with RA. Indeed, RA patients consistently report fatigue as one of the most disabling features of the disease (second only to pain). Fatigue is multifactorial in origin and reflects a complex construct of psychological, biochemical and physiological processes.
Objectives: If we are to effectively support our patients with RA, we must address fatigue. This requires understanding of which of our interventions work. At the end of the talk you should be familiar with how you can help manage fatigue, know which non-pharmacological interventions have a robust evidence and be aware of the impact of our different mode of action targeted RA therapies upon fatigue. For most our patients, a multimodal approach is needed.
Methods: This talk will present a narrative review of the evidence base around fatigue interventions in RA.
Disclosure of Interests: James Galloway Consultant for: Pfizer Inc
DOI: 10.1136/annrheumdis-2019-eular.8483

THURSDAY, 13 JUNE 2019

13:30:00 – 15:00:00

Molecular fingerprinting

SP0054 **DECONVOLUTION OF THE IMMUNE RESPONSE**

Lars Rogge. *Institut Pasteur, Immunoregulation Unit, Immunology Department, 75015 Paris, France*

Background: Anti-TNF therapy has revolutionized treatment of many chronic inflammatory diseases, including rheumatoid arthritis, Crohn's disease and spondyloarthritis (SpA). However, clinical efficacy of TNF-inhibitors (TNFi) is limited by a high rate of non-responsiveness (30-40%) both in SpA and other diseases, which exposes a substantial fraction of patients to important side-effects without clinical benefit. Despite TNFi having been used extensively for many years, it is still not possible to determine which patients will respond to TNFi before treatment initiation.
Objectives: In this study, we have tested the hypothesis that the functional analysis of immune responses of patients before and after anti-TNF therapy may not only improve our understanding of the molecular mechanisms of TNF-blocker activity, but also identify correlates of therapeutic responses in SpA patients.
Methods: To facilitate the potential translation of our findings into a clinical setting, we have used standardized whole-blood stimulation assays ("TruCulture" assays; Duffy et al., 2014), and have minimized sources of pre-analytical variability, implementing a highly sensitive and robust pipeline to assess immune functions in patients.
Results: We show that anti-TNF therapy induces profound changes in patients' immune responses, affecting predominantly several innate immune pathways. Of note, all these effects could be measured in stimulated, but not in resting immune cells, and were already detectable early, after a single TNFi injection. Modular transcriptional repertoire analysis revealed that TNFi affect immune responses via multiple mechanisms, such as directing monocyte/macrophage polarization and the inhibition of a TNF- and IL-1-dependent feed-forward loop of NF-kB activation.
On the other hand, the action of anti-TNF treatment was surprisingly selective, in that it did not affect the IL-6/Th17 or Th1 arm of the patients' immune response. These findings have important implications for therapy, given the recent approval of anti-IL-17 antibodies as an alternative agent for the treatment of several chronic inflammatory diseases.
To investigate the concept that the immune status of a patient will define their response to TNFi treatment, we have used machine-learning algorithms to identify, in whole-blood stimulation assays, immunological transcripts that correlate with clinical efficacy of TNFi. We found that high expression, before treatment initiation, of molecules associated with leukocyte invasion/migration and inflammatory processes predisposes to favorable outcome of anti-TNF therapy, while high-level expression of cytotoxic molecules was associated with poor therapeutic responses to TNF-blockers.
Conclusion: These findings suggest that SpA patients whose immune response is characterized by strong, NF-kB-mediated inflammation are more likely to benefit