monitoring of treatment response, understanding mechanisms of disease, and facilitating patient understanding of the condition will be explored.

Disclosure of Interest: N. Dalbeth Consultant for: Kowa, Horizon

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SP0069 IMPROVING ADHERENCE IN GOUT THERAPY

A. Abhishek. *Academic Rheumatology, The University of Nottingham, Nottingham, UK*

Gout is the commonest inflammatory arthritis and affects between 2.5% and 3.9% of the adult population in the western world, with even higher prevalence estimates reported elsewhere. It is the only form of arthritis that can be "cured" with simple, inexpensive pharmacologic urate lowering treatment. Such medicines offer the potential for "cure" to most people with gout. However, despite this, gout patients have the lowest adherence to long-term pharmacologic treatment among people with different rheumatic diseases. The reasons for this are multifactorial but include misconceptions about the disease and its treatment among patients, physicians and the wider community.

The long-term management of gout is overseen by primary care physicians in most countries. Recent studies have explored alternate models of gout management and care, such as by community pharmacists and nurses. Such interventions have associated with a high uptake of urate lowering treatment and excellent persistence with treatment for up to five years.

This lecture will summarise the key barriers to the uptake of and adherence with urate lowering treatment. Recent randomised controlled trials and pilot studies examining the role of nurses and pharmacists in the management of gout using a treat-to-target approach will be discussed. New data about the mechanism by which such interventions may promote adherence with urate lowering treatment will be presented.

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SP0070 WHAT ARE GOUT GUIDELINES GOOD FOR ?

T. Bardin. *Rheumatology, hôpital Lariboisière, Paris, France*

Guidelines aim to help physicians to make decisions in daily practice for an individual patient with a given condition. Guidelines should therefore be clear, easily readable and understandable by all physicians involved in disease care. They should also be as simple and brief as possible to be easily memorised by physicians, and this is a real challenge when guidelines aim at a full coverage of gout management. Most importantly, they cannot be taken as strict rules with legal implications. The final decisions concerning an individual patient remain in the hands of the responsible physician.

Guideline methodology has improved over years but still varies across recently published works. Ad hoc committees regularly involve all specialties involved in the disease care, and in the case of gout, general practitioners, who take care of most gout patients are now included but their number vary. Patients are not always included, despite being the final target of guidelines. Conflicts of interest of participating physicians are taken into account to a varying extent. The guideline development process involves an evaluation of all evidence available at the time of writing. Treatment impact on outcome, and assessment of drug benefit ratios are unanimously considered as important in the elaboration of guidelines. Factors such as drug pricing, availability and local regulatory agency recommendations, for instance about reimbursement, also matter, as guidelines should be practical in order to help physicians, but are rarely taken into account in guidelines.

There are several levels of evidence and the best ones, such as randomised placebo-controlled trials (RCT) or RCT meta-analyses are not available in all aspects of gout management. A large part of the published guidelines on the management of gout therefore relies on expert opinion, which remains fragile and may vary from one group to the other.

The numerous guidelines presently available for gout management exhibit more or less striking differences, and this diversity does not help guidelines to reach their goal. In this regard, the major differences observed between the simple and short guidelines issued by the American College of Physicians and the usually more complicated recommendations from the Rheumatology societies are the most detrimental. Pursuing therapeutic research on gout management and improving guideline methodology appear as sine qua non conditions to reach consensus and improve gout management.

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THURSDAY, 14 JUNE 2018

"Why does BMI matter?"

SP0071 IS OBESITY A RISK FOR RA?

T. Uhlig. *Rheumatology, Diakonhjemmet Hospital, Oslo, Norway*

According to the World Health Organisation (WHO) guidelines, individuals with a BMI of $>30 \text{ kg/m}^2$ are classified as obese. Obesity is on a global scale rising in prevalence and is a recognised as a cause of chronic disability. As is threatens public health, obesity has attracted significant attention in the general population, especially due to its association with significant metabolic and cardiovascular complications.

In research on rheumatoid arthritis (RA) BMI is frequently reported as a demographic variable, but obesity has not been thoroughly studied. A link between obesity and RA is plausible, as in adipose tissue biologic mechanisms of inflammation exist and may be linked to chronic systemic inflammation.

Obesity has in performed studies been considered a controversial risk factor for RA. While several studies have examined the potential influence of obesity on the development of RA, the results have been inconsistent.

While a few studies from the United States have found no or only modest association between obesity and the risk of RA, some European studies found significant risks for an association between RA and obesity. The prevalence of obesity has risen sharply in recent years which may increase our ability to better study possible associations between obesity and RA in the future. Existing evidence may thus point toward a slightly increased of obesity for the risk of RA.

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SP0072 DOES OBESITY INFLUENCE RA OUTCOMES ?

P.D. Kiely. *Rheumatology, St Georges University Hospitals NHS Foundation Trust, London, UK*

The prevalence of obesity at diagnosis of rheumatoid arthritis (RA) has doubled in N. European and N. American populations from ~14% ten years ago to ~30% now. Several prospective studies of patients with early RA, treated with csDMARD regimes, have found an association of baseline obesity with worse short and long term outcomes including DAS28 remission, low DAS, HAQ, pain assessed by VAS and SF36 physical and mental components. The most recently published cohorts report O.R. of obese RA patients at baseline achieving DAS28 remission of 0.71 (0.55–0.93) at 2 years (versus non obese) and 0.53 (0.39–0.71) at 3 years (versus normal BMI). A variety of composite responses (DAS28, CDAI, SDAI, EULAR) to TNF inhibitors, whether given in fixed dose s.c. or weight dosed i.v. formulations are significantly worse in obese RA patients from trial and registry data. In contrast the DAS28 response to Rituximab in RA patients is not affected by BMI, and ACR responses in Abatacept and Tocilizumab treated RA patients, whilst numerically less good in obese patients, are not disadvantaged by fixed s.c. dosing in comparison to i.v. dosing adjusted by weight.

Obesity is an increasingly prevalent co-morbidity and is associated with worse outcomes, including DAS28 remission, HAQ and pain scores.

Disclosure of Interest: P. Kiely Speakers bureau: BMS, Pfizer, Roche

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THURSDAY, 14 JUNE 2018

Musculoskeletal pain; feeding the opioid epidemic_

SP0073 OPIOID PRESCRIBING: WHAT'S THE PROBLEM?

B.H. Smith^{1,2}, on behalf of Scottish Pain Research Community (SPaRC). ¹Pain Service, NHS Tayside; ²Division of Population Health Sciences, University of Dundee, Dundee, UK

Opioids have been used for at least 7,000 years in the treatment of acute pain, to aid sleep and to promote euphoria. Their short-term benefits are well-recognised, but longer-term benefits are less well understood and associated harms well-known. For pain lasting beyond the acute period, the prescribed use of strong opioids was, until recently, mainly restricted to cancer and palliative care, when life expectancy is limited. More recently, however, we have seen rapidly increasing use of both strong and weak opioids for chronic non-malignant pain. This has particularly been in North America where an "opioid epidemic" is apparent, but similar patterns are emerging across the world, including Europe.

The commonest problems with long-term use of opioids include constipation, nausea and sedation. Other, perhaps more serious problems include hyperalgesia,

endocrine and immune dysfunction, mood disorders, tolerance, addiction and death. Our systematic review estimated an incidence of iatrogenic misuse or addiction of ~4% among all people prescribed an opioid. The other problem is with the lack of high quality evidence to support their effectiveness in the long-term relief of pain, as we will hear later. These concerns have prompted the International Association for the Study of Pain to issue a recent carefully-worded statement on the use of opioids in chronic pain, advising caution and close monitoring. This presentation will summarise these issues, and present evidence of the increasing use of opioids in recent years, focusing on Europe and particularly Scotland, where prescribing rates rose 65% in a 10 year period. I will present evidence of both good and potentially harmful prescribing, evidence that most prescribing is "appropriate" in that it is associated with severe chronic pain, and despite this, that a very large proportion of people with chronic pain are not prescribed any opioid. Opioids may still have a role in chronic non-malignant pain, but the goals of their use need to be increased function and improved quality of life, and we need careful selection of patients and evaluation of outcomes.

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SP0074 ARE ALL OPIOIDS THE SAME?

C. Stein. *Anesthesiology and Intensive Care Medicine, Charité – Freie Universität Berlin, Berlin, Germany*

Indiscriminate activation of opioid receptors provides pain relief but also severe central and intestinal side effects such as respiratory depression, sedation, addiction, nausea and constipation. Novel strategies to avoid adverse effects include the design of "biased agonists" and of environment-sensitive ligands. Exploiting pathological (rather than physiological) conformation dynamics of opioid receptor-ligand interactions might yield ligands without adverse actions. By computer simulations at low pH, a hallmark of injured tissue, we designed an agonist that, due to its low pKa, selectively activates peripheral mu-opioid receptors in injured/inflamed tissue, i.e. at the source of pain generation. Unlike conventional opioids, this agonist showed pH-sensitive binding, G-protein subunit dissociation, and cAMP inhibition *in vitro*. It produced injury-restricted analgesia in rats with different types of inflammatory pain without exhibiting respiratory depression, sedation, constipation or addiction potential.

Disclosure of Interest: None declared

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What is lupus – syndrome or different entities?_____

SP0075 AUTOANTIBODIES – NOT ALWAYS WHAT THEY SEEM

P.L. Meroni. *Immunorheumatology Research Laboratory, IRCCS Istituto Auxologico Italiano, 20095 Cusano Milanino, Milan, Italy*

Autoantibodies (autoAbs) are the most popular and used biomarkers for systemic rheumatic diseases (SRD). AutoAbs are becoming more and more important for: i) classification (including identification of disease subtypes), ii) diagnosis, iii) prognosis, iv) monitoring activity/organ-tissue damages as well as v) response to therapy. Moreover, autoAbs have been recently used as inclusion criteria in clinical trials and their presence is a formal requisite for prescribing some new drugs. The advent of new techniques, as well as the increasing number of autoimmune diagnostic laboratories, raise the issues of assay variability and reproducibility. In addition, the new techniques need to be validated versus the standard ones in order to determine and possibly improve the correct interpretation of their results. Harmonisation of the assays for autoAbs is becoming increasingly urgent and the availability of suitable reference material for calibration and quality control is emerging as a valuable tool for increasing assay reliability. Several international committees are joining their efforts in order to improve harmonisation and at the same time to avoid misdiagnosis, unnecessary repetition of tests and ultimately to optimise patient care and costs. On the other hand, the correct interpretation of the results depends on the technique used for detecting a given autoantibody and on its own characteristics (Meroni et al. *Nat Rev Rheumatol*. 10:35–43; 2014). The best example is the screening tests for antinuclear antibodies (ANA). Indirect immunofluorescence (IF) ANA testing has been suggested to be the golden

standard technique for ANA screening by the American College Rheumatology task force in 2010 (Meroni and Schur *Ann Rheum Dis*. 69:1420–2; 2010). While the sensitivity of IF ANA is high, its specificity is low as well as its post-test probability. So, the strength of a positive ANA should be evaluated in the clinical context to avoid mistakes (Mahler et al *J Immunol Res*. 2014:315179 2014). In addition, the isolated ANA positivity with specific staining pattern (i.e. dense fine speckled 79 – DFS70) does not support the diagnosis of a SARD and should not be taken into consideration for both classification and inclusion criteria (Mahler et al. *Autoimmun Rev*. 15:198–01; 2016). IF offered advantages in comparison with the screening solid phase assays (SPA) available at the moment of the ACR position paper; however the improvement of the solid phase assays in the last years changed the situation in a substantial way. Advantages and disadvantages of the two techniques have been reviewed and discussed by several groups and none of the two tests appears to satisfy completely our needs. However, the combination of IF and SPA that include the main nuclear (cytoplasmic) antigens diagnostic for SARD, has been reported to display higher specificity and post-test probability than the use of the single tests (Bossuyt et al *Ann Rheum Dis*. 73:e10; 2014). New tests that employ a panel of autoantigens specific for a given subset of SARD (e.g. lupus-like, scleroderma, myositis, anti-phospholipid syndrome) are now available or are going to be launched soon increasing their specificity/post-test probability in a significant manner. For example, the combination of IF and SPA for ANA screening could decrease the risk of "false positive or negative" results, while the use of one screening assay and the new ANA profiles in the context of a specific clinical setting might increase the diagnostic power. In conclusion, our strategy to autoAb testing is changing and we should take advantage of the combination of the new serological tools for better understanding the meaning of a given positive result. The combination of the new tests will be pivotal for addressing the need for a *Personalised Medicine* in rheumatology.

Disclosure of Interest: P. L. Meroni Speakers bureau: Pfizer, Abbvie, INOVA Diagnostics

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Genetics, epigenetics and disease: is it all in the genes?_____

SP0076 GENOMIC DISSECTION OF IMMUNE-MEDIATED DISEASE: PROGNOSIS, VASCULITIS AND EOSINOPHILS

K.G. Smith. *Department of Medicine, University of Cambridge, Cambridge, UK*

Modern medicine has developed by classifying disease into defined diagnostic categories on clinical grounds. Even within a specific clinical diagnostic category, however, the features of a disease and the clinical course it takes can vary greatly between individuals. I will examine two examples of how application of genomic technologies to patient cohorts has provided insight into disease behaviour.

1. A distinct biology underlying immune-mediated disease prognosis?

This presentation will explore transcriptome data that defines a clinically useful prognostic biomarker in IBD, but also addresses the specific immunological mechanisms driving long-term outcome many immune-mediated diseases (including SLE and ANCA-associated vasculitis (AAV)), and the genetics that underpins this. Evidence will be presented suggesting that the biology underlying long-term disease outcome, or prognosis, is distinct from that driving specific diagnosis, and represent an under-investigated but clinically relevant aspect of disease pathogenesis.

2. Patient stratification in ANCA-associated vasculitis: GWAS highlights new disease entities.

AAV has been defined using pathological criteria into granulomatosis with polyangiitis (GPA, formerly Wegener's), microscopic polyangiitis (MPA) and eosinophilic granulomatosis with polyangiitis (EGPA, or Churg-Strauss syndrome). GWAS studies performed by the European Vasculitis Genetics Consortium have revealed 4 genetically distinct syndromes within AAV – which are aligned with autoantibody specificity, and better align with clinical phenotype, patient outcome and treatment response than the traditional clinically-defined syndromes. GWAS can therefore be a tool for enhancing patient stratification, and revealing new insights into disease susceptibility and perhaps therapy.

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