than 40 years underwent uricemia testing (HumaSens). Gout was defined by a validated algorithm (1). Hyperuricemia was defined by capillary level equivalent to plasma uric acid level (PUA) >6 mg/dl (2) and/or urate lowering drug (ULD) prescription.
Results: 1.144 participants (mean age 37.7 years; 50.4% men) were included. Prevalence of gout in the entire redressed sample was 3.3% (95% CI 2.2–4.9). The prevalence was 4.1% (1.8–8.9), 2.6% (1.4–4.7), 6.7% (2.5–16.8), and 1.9% (0.5–6.6) for Europeans, Melanesians, Polynesians and other ethnicities, respectively. After adjustment for age and sex, Polynesians showed higher risk for gout than Europeans (adjusted odds ratio [aOR] 4.57 [95% CI 1.3–16.7]). Prevalence of hyperuricemia, determined in 658 participants, was 67.0% (95% CI 61.9–71.6). Prevalence of hyperuricemia was greater for Polynesians (aOR 9.17 [3.2–26.4]), Melanesians (aOR 4.02 [2.2–7.2]) and other ethnicities (aOR 2.18 [1.1–4.5]) than Europeans. On univariate analysis, factors associated with gout were hyperuricemia, male gender, age, BMI, waist circumference, renal failure, hypertension, diabetes, history of major episodes of depression and cancer but not dietary factors or physical activity, despite a consistent association with BMI. Among gout and non-gout patients, 45.9% and 0.7% were receiving ULT. Overall, 29.6% of patients receiving ULD had proper control of PUA levels (≤6 mg/dl).
Conclusions: As compared with Europe, in New Caledonia, the prevalence of gout and hyperuricemia was high, including in patients with European descent, as was previously reported for New Zealand (3). The prevalence of gout and hyperuricemia differed by ethnicity. For Melanesians, the prevalence of hyperuricemia was higher but risk of gout similar to that for Europeans, so factors (e.g., genetics) other than those involved in hyperuricemia may intervene in the risk of gout.
References:
[1] Richette et al. Ann Rheum Dis 2015;74:1684–90.
[2] Fabre et al ACR meeting, Washington 2016.
[3] Winnard et al Rheumatology 2012;51:901–9.
Disclosure of Interest: None declared
DOI: 10.1136/annrheumdis-2017-eular.3409

THU0464 A GENOME-WIDE ASSOCIATION STUDY OF GOUT IN PEOPLE OF EUROPEAN ANCESTRY

T.R. Merriman[1], M. Cadzow[1], M. Merriman[1], A. Phipps-Green[1], R. Topless[1], A. Abhishek[2], M. Andres[3], L. Bradbury[4], R. Buchanan[5], K. Cremin[6], E. de Guzman[4], J. de Zoysa[7], M. Doherty[2], C. Hill[8], T. Huizinga[9], T. Jansen[10], M. Janssen[11], L. Joosten[12], F. Kurreeman[9], S. Lester[8], F. Liote[13], D. Macartney-Coxson[14], H. Matsuo[15], G. McCarthy[16], S. McCormick[1], R. Murphy[7], K. Pavelka[17], F. Perez-Ruiz[18], J. Puig[19], T. Radstake[20], P. Riches[21], M. Rischmueller[8], E. Roddy[22], M. Smith[23], E. Stahl[24], B. Stiburkova[17], R. Stubbs[25], A.-K. Tausche[26], R. Torres[19], R. Walker[1], K. Yamamoto[27], M. Brown[4], H. Choi[28], N. Dalbeth[7], A. So[29], L. Stamp[1], T. Flynn[1]. *[1]University of Otago, Dunedin, New Zealand; [2]University of Nottingham, Nottingham, United Kingdom; [3]Universidad Miguel Hernández, Alicante, Spain; [4]Queensland University of Technology, Brisbane; [5]University of Melbourne, Melbourne; [6]University of Queensland, Brisbane, Australia; [7]University of Auckland, Auckland, New Zealand; [8]Queen Elizabeth Hospital, Adelaide, Australia; [9]Leiden University Medical Center, Leiden; [10]VieCuri Medical Centre, Venlo; [11]Rijnstate Hospital, Arnhem; [12]Radboud University Medical Center, Nijmegen, Netherlands; [13]Hospital Lariboisière, Paris, France; [14]Environmental and Scientific Research, Wellington, New Zealand; [15]National Defense Medical College, Saitama, Japan; [16]University College Dublin, Dublin, Ireland; [17]institute of Rheumatology, Prague, Czech Republic; [18]Hospital de Cruces, Vizcaya; [19]Hospital Universitario la Paz, Madrid, Spain; [20]University Medical Centre Utrecht, Utrecht, Netherlands; [21]University of Edinburgh, Edinburgh; [22]Keele University, Newcastle, United Kingdom; [23]Flinders Medical Centre and Repatriation Hospita, Adelaide, Australia; [24]Mt Sinai School of Medicine, New York, United States; [25]P3 Research Ltd, Wellington, New Zealand; [26]University of Dresden, Dresden, Germany; [27]Kurume University, Kurume, Japan; [28]Harvard Medical School, Boston, United States; [29]University of Lausanne, Lausanne, Switzerland*

Background: Gout progresses through three stages: hyperuricemia, deposition of monosodium urate (MSU) crystals, and innate immune system response to MSU crystals. Genome wide association studies (GWAS) have provided insight into the molecular control of progression to hyperuricemia. However, less is known about the progression from hyperuricemia to gout.
Objectives: To conduct a GWAS for gout (where an immune response to MSU crystals has occurred) using 5,835 cases - the largest GWAS of gout to date.
Methods: The GWAS comprised 3 data sets: NZ/Eurogout (2,365 clinically-ascertained cases; 1,485 controls), the Health Professionals Follow-Up (HPFS) and Nurses' Health Studies (NHS) (1,038 cases, self-ascertained using ACR criteria; 1,095 controls), and UK Biobank (2,432 cases, ascertained by self-report of gout, hospital records, and/or use of urate-lowering therapy; 102,989 controls). The NZ/Eurogout samples were genotyped using the Illumina CoreExome v24 bead chip array (547,644 markers), the HPFS/NHS samples using the Illumina OmniExpress v12 bead chip array (730,525 markers), and the UK Biobank samples using an Affymetrix Axiom array (820,967 markers). UK Biobank genotypes had been imputed to ~73.3M SNPs. Neither the NZ/Eurogout nor NHS/HPFS genotype sets were imputed. Markers common to all three data sets (279,939) were associated with gout (adjusted for sex, age) within each data set separately using PLINK 1.9. An inverse-variance weighted meta-analysis was done with meta v4.4 in R.
Results: There were seven loci with genome-wide significant ($P<5x10^{-8}$) evidence for association with gout: *SLC2A9* (OR=1.67), *ABCG2*(OR=1.72), *GCKR* (OR=1.24), *SLC17A1-A4* (OR=1.20), *SLC22A12* (OR=1.21), *PDZK1* (OR=1.14), *TRIM46* (OR=1.18).
Conclusions: All seven loci have been previously associated with serum urate levels in GWAS. Our data emphasise the relative importance of genetic control of serum urate, compared to the genetic control of MSU crystal formation or the innate immune response, in determining gout.
Disclosure of Interest: None declared
DOI: 10.1136/annrheumdis-2017-eular.5634

THU0465 CALCIUM PYROPHOSPHATE DEPOSITION DISEASE AND OSTEOARTHRITIS: TWO FACES OF THE SAME MEDAL? AN ULTRASONOGRAPHIC AND MICROSCOPY STUDY

V. Picerno[1], A. Scanu[2], A. Adinolfi[1], C.A. Scirè[3], B. Frediani[1], L. Punzi[2], G. Filippou[1]. *[1]University of Siena, Siena; [2]University of Padua, Padua; [3]University of Ferrara, Ferrara, Italy*

Background: Calcium pyrophosphate deposition disease (CPPD) and Osteoarthritis (OA) are frequently associated and CPPD with OA is recognized as a clinical subtype [1]. However, the differences in pathogenetic, microscopic and clinical aspects between the two diseases are not clear and how CPPD and OA could affect each other is still a matter of debate.
Objectives: To assess the differences between CPPD and OA in terms of anatomic alterations of the joint, evaluated with US, and characteristics of the synovial fluid (SF) of knees affected by CPPD and/or OA.
Methods: consecutive patients reaching the outpatient clinic for the presence of knee pain and with any amount of joint effusion were eligible for the study. Patients with diagnosis or suspicion of chronic inflammatory rheumatic conditions were excluded. All enrolled patients underwent US of the knee for the assessment of joint effusion (JE), synovial hypertrophy (SH), synovial power Doppler (PD), femuro-tibial osteophytes (FTO) and alterations of the femoral hyaline cartilage (FHC), using a semiquantitative score (0: normal to 3: severe alteration) and finally a US guided aspiration of the SF. SF was examined by optical and polarized light microscopy to determine total and differential white blood cell (WBC) counts, and for crystal identification. The concentration of the main inorganic ions involved in CPP crystal formation ($P_2O_7^{4-}$, PO_4^{3-}, Ca^{2+} and Mg^{2+}) was assessed by fluorometric and colorimetric assays. CPP crystal detection was also used for the classification of patients. Further, in CPPD patients, a count of the CPP crystals detectable in a single slide was carried out. Depending on the variables, chi-square, Mann Whitney and Spearman Ro tests were used for statistical analysis.
Results: 49 patients (28 women), mean age 70.29 yo (SD±10.93) were enrolled in the study; 23 subjects presented OA and 26 CPPD (23.07% acute arthritis, 77.6% CPPD with OA). At US, a statistically significant difference between CPPD and OA was found only for the grade of effusion, being more abundant in CPPD patients. On the contrary, no differences were found regarding SH, PD, FTO, FHC. SF analysis showed that CPPD patients presented a higher volume of SF, a higher total WBC count with a higher polymorphonuclear (PMN) cells percentage and lower monocytes percentage than patients with OA. Further, both total cell count and PMN percentage were positively correlated with the number of crystals in the SF. On the other hand, no statistically significant differences were found in the content of inorganic ions between the two groups.
Conclusions: According to these results, patients with CPPD and OA present some distinct features, mainly regarding the characteristics of the SF, compared to patients with OA alone. These differences may reflect different underlying pathogenetic pathways for the two diseases. Surprisingly, the concentration of inorganic ions in the two populations was similar.Further studies are necessary in order to better understand the link between CPPD and OA and the role of ions concentration in the SF for the formation of crystals.
References:
[1] Zhang W et al. European League Against Rheumatism reccomandations for calcium pyrophosphate deposition. Pat I: terminology and diagnosis. Ann Rheum Dis 2011; 70: 563–570.
Disclosure of Interest: None declared
DOI: 10.1136/annrheumdis-2017-eular.6606

THU0467 SAFETY AND EFFICACY OF FEBUXOSTAT IN ADVANCED CKD PATIENTS WITH HYPERURICEMIA

Y.-J. Oh, E.S. Park, S.S. Ahn, K.Y. Lee, S.-J. Byun, J.Y. Pyo, S.M. Jung, S.-W. Lee, Y.-B. Park, J.J. Song. *Internal medicine, Yonsei University College of Medicine, Seoul, Korea, Republic Of*

Background: In chronic kidney disease (CKD) patients, hyperuricemia is a common finding and might be one of modifiable risk factors for renal progression. However, dosing adjustments and increased risk of serious side effects of uric acid lowering agents in patients with reduced renal function lead to undercorrection of hyperuricemia, especially in patients with advanced CKD. Febuxostat is highly

effective and well-tolerated to treat hyperuricemia in CKD patients. Although several evidences demonstrated the usefulness of febuxostat in hyperuricemic CKD patients, clinical studies aimed at the CKD patients with inappropriately controlled hyperuricemia by allopurinol have been relatively lacking.
Objectives: The study objective is to evaluate the safety and efficacy of febuxostat in patients, who had CKD with severe renal impairment and did not meet with the target uric acid levels using allopurinol.
Methods: Data were collected from 168 patients who had CKD with more than stage 3b and changed from allopurinol to febuxostat due to uncontrolled hyperuricemia between 2005 and 2014 at Yonsei University Medical Center. Uric acid and creatinine were analyzed at baseline and during the first 6 and 12 months after conversion of febuxostat. Estimated glomerular filtration rate was calculated using the formula of MDRD equation. The patients were defined as a well-controlled state when the uric acid values of the study subjects reached within 6.0 mg/dL.
Results: The mean age was 60.7±14.6 years, and 129 patients (76.8%) were male. The number of patients was 25 (14.9%) in CKD stage 3b, 75 (44.6%) in stage 4, 8 (4.8%) in stage 5, 38 (22.8%) in patients treated with maintenance dialysis, 22 (13.1%) in patients underwent kidney transplantation. The mean estimated GFR (eGFR) and uric acid levels at baseline was 23.1±17.3 ml/min/1.73m^2 and 8.3±2.4 mg/dL, respectively. Most of the patients was treated with 40 or 80mg of febuxostat during the study period. The mean uric acid levels at 6- and 12-month after febuxostat treatment were significantly reduced compared to uric acid levels at baseline (5.2±2.1 mg/dL at 6-month and 4.9±2.2 mg/dL at 12-month, $p<0.001$, respectively). More than 70% of study subjects reached to the target of uric acid levels less than 6mg/dL at 6- and 12-months after treatment of febuxostat [122 (72.6%) patients at 6-month and 133 (79.2%) patients]. The creatinine levels at baseline and 6-month were comparable (3.42±2.03 vs. 3.38±2.16 mg/dL at baseline and 6-month, p=0.61), meanwhile, the creatinine levels were significantly increased after 12-month compared to those at baseline (3.69±2.46 mg/dL, $p<0.01$). Abnormality of liver function test was observed in only one patient during the follow up period. None of the patients did not discontinue drug due to adverse events.
Conclusions: Present study demonstrated that substantial hyperuricemic CKD patients treated with febuxostat were achieved the target of uric acid levels without adverse events. Febuxostat is an effective and safe uric acid lowering drug in allopurinol-intolerant patients with advanced CKD.
Disclosure of Interest: None declared
DOI: 10.1136/annrheumdis-2017-eular.2925

THURSDAY, 15 JUNE 2017

Fibromyalgia

THU0468 DIAGNOSTIC EXPERIENCE OF PATIENTS WITH FIBROMYALGIA – A SYSTEMATIC SYNTHESIS OF QUALITATIVE STUDIES

A.M. Mengshoel[1], J. Sim[2], B. Ahlsen[1], S. Madden[3]. *[1]Institute of Health and Society, University of Oslo, Oslo, Norway; [2]Institute for Primary Care and Health Sciences, Keele University, Staffordshire; [3]Faculty of Medical Science, Anglia Ruskin University, Chelmsford and Cambridge, United Kingdom*

Background: In recent years, the evidence-based practice (EBP) movement has become embraced by clinicians and scientists world-wide. A guiding principle of the EBP is that the best evidence available should inform clinical practice. At the same time, patients' experiences should be taken into consideration in the clinical disicion-making. Over the years, the diagnosis of fibromyalgia (FM) has been subject to numerous debates among scientists and clinicians. Despite that a diagnosis may lead to a biographical disruption, be a starting point for patients to make sense of their illness, and shape expectations for the future, the diagnostic debates have not been appreciably informed by how patients themselves perceive the diagnosis.
Objectives: To examine how individuals experience the process and consequences of receiving a diagnosis of fibromyalgia
Methods: A systematic literature search of qualitative studies up to May 2016 was performed. A systematic search was carried out in Medline (*n*=562), PsychInfo (n=430), Cinahl, AMED (*n*=95), and Social Science Citation Index (*n*=486) up to May 2016, supplemented by items from the authors' knowledge of the literature. Duplicates, quantitative studies, studies addressing chronic pain with no specification of diagnosis or including patients with other diagnoses, editorials, reviews, conference reports and dissertations were excluded. After this reading 93 qualitative studies of patients' experience were read to identify whether they included information about patients' diagnostic experience. Twenty-six papers, one book and a book chapter were included. Information about diagnostic experiences were extracted and subjected to an interpretive analysis in accordance with principles of meta-ethnography.
Results: Years were normally spent consulting specialists in an attempt to confirm the reality of symptoms and make sense of the illness. In this process, the reality of the illness was questioned. Great relief was expressed at finally achieving the FMS diagnosis. However, relief waned when therapies proved ineffective. Health professionals and others again questioned whether individuals were genuinely ill, that the illness had a psychological nature, and whether they were doing their best to recover. For the patients, the diagnosis did not provide a meaningful explanation of their suffering and had limited power to legitimate illness. The patients felt blamed for their failure to recover which meant their personal credibility and moral identity were put at stake.
Conclusions: The FMS diagnosis has limitations in validating and making sense of patients' illness experiences and in providing social legitimation of their illness. Social relationships are strained during the diagnostic process and in the course of ineffective therapies.
Disclosure of Interest: None declared
DOI: 10.1136/annrheumdis-2017-eular.5035

THU0469 COMPARATIVE EFFECTIVENESS OF TAI CHI VERSUS AEROBIC EXERCISE FOR FIBROMYALGIA: A RANDOMIZED CONTROLLED TRIAL

C. Wang[1], C. Schmid[2], R. Fielding[3], W. Harvey[1], K. Reid[3], L.L. Price[1], J. Driban[1], R. Kalish[1], R. Rones[4], T. McAlindon[1]. *[1]Tufts Medical Center, Boston; [2]Brown University School of Public Health, Providence; [3]Tufts University; [4]Center for Mind-Body Therapies, Boston, United States*

Background: Fibromyalgia is a complex disorder with strong psychological and pain components. Tai Chi is an integrated mind-body approach that enhances both physical and mental health and has great potential to treat fibromyalgia (1–2).
Objectives: We aimed to investigate whether Tai Chi is more effective with longer lasting effects than aerobic exercise.
Methods: We conducted a 52-week, single-blind, randomized trial of Tai Chi vs. aerobic exercise for fibromyalgia (ACR 1990 and 2010 diagnostic criteria). Participants were randomized to 1 of 4 Tai Chi interventions: 12 or 24 weeks of Tai Chi once or twice per week, or aerobic exercise held twice per week for 24 weeks. The primary endpoint was change in the Revised Fibromyalgia Impact Questionnaire (FIQR) score at 24 weeks. Secondary endpoints included change in patient's global assessment, the Hospital Anxiety and Depression scale (HADS), Sleep Quality Index (PSQI), arthritis self-efficacy scale (ASES-8), and quality of life. The comparative efficacy of the five groups was determined using longitudinal models based on the intent-to-treat principal.
Results: The mean age of subjects was 52 years (SD 12), mean years of body pain of 9 years (SD 8), 92% were women, and 61% were white. Treatment groups did not differ in baseline outcome expectation. The average of all 4 Tai Chi groups, compared to aerobic exercise, showed significant improvements in FIQR scores, patient's global, anxiety, and self-efficacy. All other outcomes favored Tai Chi over aerobic exercise (Table 1). The Tai Chi treatment with the same dosage as the aerobic group demonstrated an even larger effect for FIQR and for most other outcomes. The benefit of Tai Chi was consistent across instructors.
Conclusions: Tai Chi is more effective than aerobic exercise and can be considered as an important therapeutic option for patients with fibromyalgia.
References:
[1] Jones KD. Recommendations for resistance training in patients with fibromyalgia. Arthritis Res Ther 2015;17:258.
[2] Wang C, Schmid CH, Rones R, et al. A randomized trial of tai chi for fibromyalgia. N Engl J Med 2010;363:743–54.
Disclosure of Interest: None declared
DOI: 10.1136/annrheumdis-2017-eular.3484

Abstract THU0469 – Table 1. Between-Group Differences in Outcomes at Week 24

Variable	Aerobic Exercise vs Tai Chi groups combined		Tai Chi 12-week vs 24-week		Tai Chi 1x/week vs 2x/week		Aerobic Exercise 2x24 Weeks vs Tai Chi 2x24 Weeks	
	Mean (95% CI)	P-Value	Mean (95% CI)	P-Value	Mean (95% CI)	P-Value	Mean (95% CI)	P-value
FIQR	5.5 (0.6, 10.4)	**0.03**	9.6 (2.6, 16.6)	**0.007**	4.5 (-2.5, 11.4)	0.21	16.2 (8.7, 23.6)	**<0.0001**
Patient's Global	0.9 (0.3, 1.4)	**0.005**	0.7 (-0.2, 1.5)	0.12	0.4 (-0.4, 1.2)	0.35	1.6 (0.7, 2.5)	**0.0006**
HADS Depression	0.7 (-0.3, 1.6)	0.16	1.4 (0.1, 2.6)	**0.04**	0.7 (-0.6, 1.9)	0.31	2.1 (0.5, 3.7)	**0.01**
HADS Anxiety	1.2 (0.3, 2.1)	**0.006**	0.4 (-0.8, 1.6)	0.55	-0.2 (-1.4, 1.0)	0.74	2.1 (0.6, 3.6)	**0.008**
ASES	1.0 (0.5, 1.6)	**0.0004**	0.5 (-0.3, 1.3)	0.23	0.1 (-0.7, 0.9)	0.73	1.5 (0.6, 2.5)	**0.002**
PSQI	0.3 (-0.6. 1.3)	0.49	1.0 (-0.4, 2.3)	0.16	0.3 (-1.0, 1.7)	0.62	1.0 (-0.6, 2.5)	0.22
SF-36 MCS	2.5 (-0.1, 5.0)	0.06	4.4 (0.8, 8.1)	**0.02**	-0.4 (-4.0, 3.2)	0.83	6.2 (1.9, 10.6)	**0.006**
SF-36 PCS	0.3 (-1.7, 2.2)	0.79	2.4 (-0.3, 5.1)	0.09	1.2 (-1.5, 3.9)	0.38	2.0 (-1.3, 5.3)	0.24

Positive scores indicate improved outcome in second listed group. Boldface indicates statistically significant differences between groups.