actual improvement at one month for back pain ($r = 0.758$; $P = 0.001$), improvement in ADLs ($r = 0.709$; $P = 0.001$) and to a lesser extent for walking ability ($r = 0.452$; $P = 0.045$).

There was no correlation between severity of back pain at baseline and the actual improvement in back pain at one month suggesting that the degree of improvement depended more on the individual's expectation of benefit rather than the severity of symptoms/ signs.

Conclusions: This study shows that facet joint injections reduce disability as measured by the Roland Morris disability questionnaire. Additionally, outcomes after facet joint injection correlated more with the patients' expectations rather than the severity of back pain at baseline. Patients with very low expectations of benefit from this procedure tended to have poor outcomes. This is relevant when selecting patients for facet joint injection.

Disclosure statement: All authors have declared no conflicts of interest.

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Rheumatoid Arthritis: Clinical Aspects

322. THE EFFECT OF BIOLOGICS ON CARDIOVASCULAR DISEASE IN PATIENTS WITH RHEUMATOID ARTHRITIS: A SYSTEMATIC LITERATURE REVIEW

Sarah L. Westlake[1,2], Alexandra N. Colebatch[2], Janis Baird[3], Patrick Kiely[4], Mark Quinn[5], Ernest Choy[6], Andrew J. Ostor[7] and Christopher J. Edwards[2]
[1]*Department of Rheumatology, Poole Hospital NHS Foundation Trust, Poole, UK;* [2]*Department of Rheumatology, Southampton University Hospitals NHS Trust, Southampton, UK;* [3]*MRC Epidemiology Resource Centre, University of Southampton, Southampton, UK;* [4]*Department of Rheumatology, St George's Healthcare NHS Trust, London, UK;* [5]*Department of Rheumatology, York Hospital/ Hull York Medical School, York, UK;* [6]*Department of Rheumatology, King's College London, London, UK;* [7]*Department of Rheumatology, Cambridge University Hospitals NHS Foundation Trust, Cambridge, UK*

Background: Patients with RA have an increased prevalence of cardiovascular disease (CVD). This is due to increased traditional risk factors and the effect of chronic inflammation. TNF antagonists are potent suppressors of inflammation and may reduce the risk of CVD.

We performed a systematic literature review to determine whether TNF antagonists affect the risk of clinical CVD events in RA.

Methods: We searched Medline, Embase, Cochrane database, DARE, HTA and Science Citation Index from 1980-2008. Papers were included if they assessed the relationship between the use of TNF antagonists and clinical CVD outcomes in RA. All articles were assessed for study quality.

Results: 1840 abstracts were identified. Two reviewers independently assessed each title and abstract. 20 studies fulfilled the inclusion criteria: 1 RCT, 11 cohorts, 7 case-controls and 1 cross-sectional study(Table to be included in a poster). 7 studies considered all CVD events, 4 demonstrating a significant decreased CVD risk and 3 no change in risk. 7 studies assessed the association of TNF antagonists and MI; 2 demonstrated a significant decreased risk and 5 no difference. The study with the lowest risk of bias, showed a significantly lower risk of MI in TNF responders compared with non-responders. 3 studies considered stroke and TNF antagonists, two demonstrated no change in risk and 1 a reduced risk after 6 months of treatment. 6 studies considered heart failure (HF): 1 demonstrated a significantly increased risk of HF in elderly RA patients, 3 no difference in risk and 2 a significantly decreased risk of HF. In the study with the lowest risk of bias, a non-significant increased risk of HF was considered to be offset by the efficacy of TNF antagonists.

Conclusions: For all CVD events there may be a decreased risk associated with use of TNF antagonists. For specific events, e.g. MI and HF the effect is less clear. Atherosclerosis is an inflammatory process, TNF antagonists would be expected to reduce the risk of CVD by decreasing the burden of systemic inflammation. There are several reasons this may not be apparent. Firstly, TNF antagonists may have adverse effects on traditional risk factors (lipids, etc.). Secondly MTX reduces the risk of CVD events, it is commonly used in combination with TNF antagonists and in some of the studies was used by controls. TNF antagonists may therefore have no additional benefit to MTX. In contrast to MTX, TNF antagonists are often used late in the disease when significant irreversible damage has occurred. Finally the effect on CVD may depend on response to treatment as some of the studies demonstrated a lower risk of CVD events in responders compared with non-responders. To determine the true effect of TNF antagonists on CVD risk future studies should publish data on CVD risk factors and clinical events, this is particularly important in studies of early disease when the burden of inflammation has not been realised.

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323. QUALITY OF LIFE PREDICTORS OF PATIENTS WITH RHEUMATOID ARTHRITIS

Beata Jankowska[1], Izabella Uchmanowicz[1], Jacek Polanski[2] and Krzysztof Dudek[3]
[1]*Faculty of Health Sciences, Medical University, Wroclaw, Poland;* [2]*Atos Healthcare, Sheffield, UK;* [3]*Institute of Machines Design and Operation, Technical University, Wroclaw, Poland*

Background: Although rheumatoid arthritis is not a curable disease and gradually leads to patient's immobility there could be an improvement in QoL through ameliorating well-being and getting back their social functioning which seems to be a strategic therapeutic goal. Identifying factors which have significant impact on QoL of RA patients might be important information when choosing the most beneficial treatment for the patient.

Methods: This study examines the factors which influences quality of life (QoL) of patients with rheumatoid arthritis (RA). Design: The following QoL predictors were taken into account: socio-demographical, clinical - disease duration and severity, smoking, morning stiffness duration, BMI, DAS 28, treatment and co-morbidity. Sample: The study was held with 60 RA patients (including 42 females) between 23 and 79 years of age ($= 52.1$, S.D. $= 16.3$) hospitalized in Rheumatology Unit of Academic Clinical Hospital in Wroclaw between November 2007 and March 2008. They were diagnosed with RA based on 1987 year criteria of American College of Rheumatology. The mean illness duration for 33.3% of patients in the study group was up to 5 years since having been diagnosed, 36.7% from 6 to 10 years and 21.7% with RA over 11 years. For clinical estimation of patients activity, the Disease Activity Score (DAS 28) and Visual Analogue Scale (VAS) for pain were used. The QoL was assessed using key areas of self-reported health status of Health Assessment Questionnaire (HAQ) for validation of functional disability and WHOQOL-BREF questionnaire.

Results: The study ($n = 60$) showed statistical significance ($P < 0.05$) between HAQ score and patients' age ($r = 0.638$), marital status ($r = 0.548$), BMI ($r = 0.503$), DAS 28 ($r = 0.375$) and illness duration ($r = 0.376$). There was also statistically significant dependence ($P < 0.05$) found between the results of WHOQOL-BREF questionnaire and age in psychological ($r = 0.362$), social ($r = 0.416$) and environmental ($r = 0.304$) domain as well as BMI in physical ($r = 0.270$), psychological ($r = 0.408$) and social ($r = 0.371$) area and a correlation with patients education in all four WHOQOL domains: physical($r = 0.296$), psychological ($r = 0.283$), social ($r = 0.425$) and environmental ($r = 0.289$). Other significant correlation between WHOQOL outcome and the illness duration was found in psychological ($r = 0.254$) and social ($r = 0.316$) domain.

Conclusions: The socio-demographic factors influencing QoL of RA patients include: education, marital status, age and living conditions. The clinical factors impinging on life quality are DAS 28, BMI > 30, disease duration. The lowest results RA patients obtain in social area.

Exploring all the factors impinging on RA patients' QoL appears to be very useful and important whilst defining the best treatment methods which will correlate with a better quality of life of patients and result in its improvement.

Disclosure statement: All authors have declared no conflicts of interest.

324. IS PERIPHERAL VASCULAR DISEASE INCREASED IN PATIENTS WITH RHEUMATOID ARTHRITIS COMPARED WITH THE GENERAL POPULATION?

Resmy Suresh and Natalie Horwood
Rheumatology, Mayday University Hospital, Croydon, UK

Background: The presence of peripheral vascular disease (PVD) is an independent marker for coronary artery disease. Even though the prevalence of coronary and cerebrovascular disease in patients with rheumatoid arthritis (RA) is well established, the risk of development of PVD in this patients group is not clearly known. RA predisposes to premature atherosclerosis driven by underlying chronic inflammation and this could lead to significant peripheral vascular disease. Early recognition and effective management of this problem can improve the patient quality of life and possible long term outcome.

The aim of systematic review is to establish whether there is an increased risk for peripheral vascular disease in RA patients

Methods: Medical subject headings (MESH) terms used in the Medline database search included rheumatoid arthritis, peripheral vascular disease, peripheral arterial disease, ankle brachial pressure index, intima-media thickness and related mesh terms.

Results: Out of 801 articles retrieved, 21 were deemed suitable for full text review and of these only 6 papers were of direct relevance. No systematic reviews were found.

Among the four cross sectional studies three of them showed statistically significant ankle brachial pressure index (ABPI) in RA patients and the fourth one showed significant increase in femoral and carotid arterial wall thickness. Similarly two cohort studies showed significant increase in peripheral vascular disease using ABPI in all two and femoral angiogram in one. (Table)

Conclusions: There is an increased risk of PVD in RA in comparison with matched controls with a more than two fold increase in relative risk. Hence an increased risk of cardiovascular disease. The aetiology of this accelerated atherosclerosis is most likely multifactorial. There may, however be a significant contribution by systemic inflammation. In future, more research needs to be done on RA patients to establish the evidence of improvement in long term outcomes by early detection and management of PVD and by controlling inflammation.

Author	Type of study	Numer of patients/ controls	Type of intervension	P value
Kumeda et al. 2002	Cross sectional	138/94	Ultrasound-femoral and carotid artery intimal thickness	0.0124
Alkaabi et al. 2003	Cross sectional	40/40	ABPI	<0.007
Rincon et al. 2005	Cohort study	234/102	ABPI	$=0.06$
Liang et al. 2006	cohort study	609 RA	ABPI or angiogram	<0.012
Han et al. 2006	Cross sectlonal comparatative study	714RA/ 690PsA/ 720 AS	ABPI	<0.01
Joseph et al. 2002	Cross sectional	30/30	ABPI	$=0.01$

Disclosure statement: All authors have declared no conflicts of interest.

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325. ENDOTHELIAL FUNCTION ASSOCIATES WITH CLASSICAL CARDIOVASCULAR DISEASE RISK BUT NOT DISEASE RELATED INFLAMMATION IN RHEUMATOID ARTHRITIS

Aamer Sandoo[1,2], Jet Veldhuijzen van Zanten[1,2], Jacqueline P. Smith[2], Douglas Carroll[1], Tracey E. Toms[2] and George D. Kitas[2,1]
[1]*School of Sport and Exercise Sciences, University of Birmingham, Birmingham, UK;* [2]*Department of Rheumatology, Dudley Group of Hospitals NHS Trust, Dudley, UK*

Background: Patients with rheumatoid arthritis (RA) have an increased risk for cardiovascular disease (CVD). At present, the exact mechanism for this is unknown, but RA-disease related inflammation has been postulated to affect the vasculature and as such contribute to CVD risk. However, not all studies in RA patients report associations between endothelial function and inflammation. In addition, most studies have restricted their vascular assessments to a single vascular bed (i.e., either micro- or macrovascular beds). We explored the associations between microvascular and macrovascular endothelial function with classical CVD risk and disease-related inflammation in patients with RA.

Methods: 99 RA patients (72 females, 56 ± 12 years, Body Mass Index: 30 ± 6 kg/m^2) underwent assessments of microvascular function (Laser Doppler Imaging with iontophoresis of acetylcholine (ACh) and sodium-nitroprusside (SNP)) and macrovascular function (flow-mediated dilatation (FMD) and glyceryl-trinitrate-mediated dilatation (GTN)). Arterial stiffness was quantified by the augmentation index (AIx) using pulse wave analysis (PWA). Framingham risk score (FRS), Systematic Coronary Risk Evaluation for total cholesterol (TC SCORE) and HDL cholesterol (TC:HDL SCORE) were used to estimate CVD risk. Disease activity was assessed with DAS28, erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP). Linear regression was performed with disease-specific parameters or CVD risk scores entered as independent variables and each vascular parameter entered as the dependent variable separately.

Results: ESR, CRP and DAS28 were not associated with any measure of endothelial function. FRS and TC SCORE were inversely associated with ACh% ($\beta = -0.26$, $t = 20.46$, $P = 0.01$, $R^2 = 0.068$ and $\beta = -0.26$, $t = 2.09$, $P = 0.04$, $R^2 = 0.068$ respectively).

Conclusions: CVD risk scores were associated with microvascular but not macrovascular endothelial function. No associations were found between disease activity and endothelial function. These findings suggest that classical CVD risk might have a larger impact on vascular function than inflammation in patients with RA.

Inflammation, global CVD risk and endothelial function

Study Variables	RA Patients
ESR (mmh)	23 ± 22
CRP (mg/l)	12 ± 19
DAS28	3.6 ± 1.3
FRS	7 ± 5
TC SCORE	2 ± 2
TC:HDL SCORE	1 ± 2
Increase in Perfusion (ACh %)	308 ± 235
Increase in Perfusion (SNP %)	303 ± 200
FMD (%)	9 ± 6
GTN (%)	23 ± 9
AIx (%)	32 ± 9

Mean $\pm$ standard deviation

Disclosure statement: All authors have declared no conflicts of interest.

326. ANTI-CCP TITRE PREDICTS ONSET OF INFLAMMATORY POLYARTHRITIS IN PATIENTS PRESENTING WITH NON-INFLAMMATORY ARTHRALGIA

Sarang Chitale[1,2], Cristina Estrach[1,2], Robert Thompson[2], Sangita Sathyamurthy[2] and Nicola Goodson[1,2]
[1]*School of Clinical Sciences, University of Liverpool, Liverpool, UK;* [2]*Rheumatology, University Hospital Aintree, Liverpool, UK*

Background: Anti-cyclic citrullinated peptide antibodies (anti-CCP) are highly specific for rheumatoid arthritis and often predate disease onset. Anti-CCP titre is shown to be significantly increased at disease onset but its role in predicting the onset of inflammatory polyarthritis (IP) is not clear.

Aim: To explore whether titre of anti-CCP predicts development of IP in anti-CCP positive arthralgia patients.

Methods: Patients with small joint arthralgia (but no clinical or ultrasonographic evidence of IP) and positive anti-CCP antibodies (defined as titre > 7 U/ml) were identified from their initial attendance at an early arthritis clinic between Jul 06-Sep 09. All had symptom duration of less than 12 months. Patients were followed up until they either developed IP or the end of the study period 10/11/09. Diagnosis of IP was based on clinical and ultrasound evidence of IP in rheumatoid distribution requiring initiation of disease modifying anti-rheumatic therapy (DMARDs). Cox proportional hazards modelling was used to explore whether high baseline anti-CCP titre (defined as > 600 U/ml) predicts the development of IP. Multivariate models adjusting for symptom duration, smoking status, gender and age were generated.

Results: 38 patients with arthralgia and positive anti-CCP antibody were identified. 61% were female and 79% were rheumatoid factor (RF) positive at baseline. Median follow-up time was 8 months [IQR 3,

24]. 12 (32%) patients developed IP during the follow-up period and 6 of these had high anti-CCP titres (median titre 420 U/ml [IQR 100, > 600]). Of arthralgia patients who did not progress to IP, 7 had high titres of anti-CCP, (median titre 158 U/ml, IQR [50, > 600]).

Univariate analysis demonstrated a 3 fold increased risk for development of IP in patients with high titre anti-CCP compared with patients with lower titres (Hazards Ratio (HR) 3.42, 95% CI [1.03, 11.3]). Multivariate analysis adjusting for age, gender and smoking status and symptom duration revealed an 8 fold increased risk for development of IP in patients with high titre CCP than those with lower titre (HR 8.27, 95% CI [1.49, 45.9]). Kaplan-Meier survival curves demonstrated that patients with high titre anti-CCP developed IP rapidly with 50% developing IP within 6–7 months and 80% within the first year, after their initial presentation. Proportional hazards assumption remained true.

Conclusions: Titre of anti-CCP is an important predictor of progression to IP in patients with anti-CCP positive arthralgia. Those with anti-CCP titres> 600 should be closely followed up in rheumatology clinics to promptly identify the onset of IP and allow rapid initiation of DMARDs

Disclosure statement: All authors have declared no conflicts of interest.

327. DO THE NEW 2009 METABOLIC SYNDROME JOINT CONSENSUS CRITERIA OVER DIAGNOSE THE METABOLIC SYNDROME IN RHEUMATOID ARTHRITIS?

Tracey E. Toms[1,2], Vasileios F. Panoulas[1], Karen M. Douglas[1] and George D. Kitas[1,2]
[1]*Rheumatology, Dudley Group of Hospitals NHS Foundation Trust, Dudley, UK;* [2]*arc Epidemiology Unit, Manchester University, Manchester, UK*

Background: The metabolic syndrome (MetS) may contribute to the excess cardiovascular burden observed in rheumatoid arthritis (RA). However, the number of definitions (WHO, EGIR, IDF, NCEP2001, NCEP2004) of the MetS has resulted in a degree of confusion regarding the prevalence and clinical associations of the condition. In RA, we have previously shown that the prevalence of the MetS ranges from 12.1% to 45.3% accordnig to the definition used, with EGIR reporting the lowest and IDF reporting the highest prevalance. Earlier this year, a joint consensus statement recommended modified MetS criteria based on an amalgamation of the 5 established definitions. In this study, we aimed to 1) assess the prevalence of the metabolic syndrome in RA according to the 2009 joint consensus criteria (MetS2009) 2)identify factors that predict the Mets2009 in RA

Methods: 400 well-characterised RA patients fulfilling the American College of Rheumatology criteria were studied cross-sectionally. The prevalence of the MetS was assessed according to the 2009 joint consensus criteria (Table). A logistic regression model was used to identify factors predicting the MetS2009.

Results: The prevalence of the MetS2009 was 45.6%. The prevalence of the MetS2009 increased until the age of 70 and then declined. factors found to independently predict the MetS2009 were older age ($P < 0.001$), lower methotrexate use ($P = 0.014$)and higher health assessment questionnaire scores ($P = 0.018$)

Conclusions: The MetS2009 consensus criteria identify a large percentage of RA patients as having the metabolic syndrome. the prevalence observed with the MetS2009 criteria is comparable to the highest prevalence rate seen with the IDF criteria (45.6% *vs* 45.3%). this observation could imply either that the MetS2009 criteria over diagnose the MetS in RA or conversley that they are the most sensitive method. Further work is required to test the validity and reliability of the MetS2009 criteria in RA.

The 2009 Metabolic syndrome criteria (3 out of 5 required for disgnosis)

Waist circumference >=94 cm in men or >=80 cm in women
TG>=1.7 mmol/l or on lipid lowering therapy
HDL<1.0 mmol/l in men or <1.3 mmol/l in women, or on lipid lowering therapy
fasting glucose >=5.5 mmol/l or on anti-diabetic medication
Systolic BP>=130 mmHg and/or diastolic BP>=85 mmHg, or on anti-hypertensive medication

BP = Blood pressure, HDL: high density lipoproteins, TG: triglycerides

Disclosure statement: All authors have declared no conflicts of interest.

328. ETHNICITY AND RHEUMATOID ARTHRITIS: A SYSTEMATIC REVIEW

Hanan S. Abozaid[1,2], Nihal A. Fathi[2], David L. Scott[1] and Sophia Steer[1]
[1]*Academic Rheumatology, King's College London, London, UK;* [2]*Rheumatology, Sohag University, Sohag, Egypt*

Background: Several patient-related factors impact on the clinical phenotype and outcome of rheumatoid arthritis (RA). The effects of gender and deprivation are well known. The effects of ethnicity have received less attention. Differences in the clinical expression of disease between different ethnic populations may reflect different environmental, social or genetic factors or all. Understanding these factors will be useful in elucidating basic disease mechanisms and may change management. We systematically reviewed studies examining the effects of ethnicity in RA to define the impact of this key factor.

Methods: We systematically searched electronic databases and published bibliographies (up to March 2009) to identify cohort, case control and cross sectional studies which compared two or more ethnic populations of adults with RA and evaluated one or more standard outcomes. These spanned clinical (joint counts, extra articular features); laboratory (ESR/CRP and rheumatoid factor), functional (HAQ) and radiological (erosions) assessments. Two authors independently selected studies; extracted data and assessed study quality, results were presented separately by each outcome using the Cochrane software RevMan 5.

Results: From 2246 citations we reviewed 30 potentially relevant studies. 17 met our entry criteria and were included; they enrolled 10,002 patients. 11 studies compared African American/African Caribbean (AA/AC) patients with White/European (WE) populations, including 6 comparisons within single centres. 3 studies compared Asian with WE populations and the remaining studies compared diverse populations, including different areas of Europe, different Jewish populations and Arab WE populations.

The 6 studies comparing AA/AC patients with WE populations within single centres showed significant differences between groups. HAQ scores were lower in WE (standardised mean difference 0.30 (95% CI 0.29, 0.31), though there was significant heterogeneity between studies ($P = 0.01$). Other clinical outcomes, including swollen and tender joint counts and ESR, showed some differences but the findings were inconsistent across studies. There were no consistent differences in rheumatoid factor positivity, erosive disease and extra-articular features such as nodules. We identified several important confounding factors including differences in socioeconomic status and access to treatments.

Conclusions: There is evidence that the impact of RA is worse in patients from some ethnic backgrounds, particularly AA/AC patients, with higher HAQ scores in these patients. We consider ethnicity is a potentially important factor in the outcome of RA. However, it is uncertain whether this reflects differences in the clinical phenotype, which might result from genetic differences or is a consequence of socioeconomic factors and relative deprivation.

Disclosure statement: All authors have declared no conflicts of interest.

329. THE RISK OF SERIOUS INFECTIONS IN PATIENTS RECEIVING ANAKINRA FOR RHEUMATOID ARTHRITIS (RA): RESULTS FROM THE BSR BIOLOGICS REGISTER (BSRBR)

James Galloway[1], Will Dixon[1], Louise Mercer[1], Kath Watson[1], Lunt Mark[1], Kimme Hyrich[1], BSRBR Control Centre Consortium[2], Deborah Symmons[1], on behalf of the BSR Biologics Register[1]
[1]*arc Epidemiology Unit, University of Manchester, Manchester, UK;* [2]*British Society of Rheumatology, London, UK*

Background: A recent Cochrane review of trials of ANA (an interleukin-1 receptor antagonist) in RA identified no significant difference in serious infection (SI) rates compared with placebo. Our aim was to explore the influence of ANA on SI rate using data from the BSRBR, a prospective cohort study set up in 2001 to monitor the safety of biologic therapies in the UK.

Methods: Consecutive RA patients treated with ANA were recruited between October 2001 and May 2008 by the BSRBR and were followed 6 monthly via consultant and patient questionnaires until December 2008, end of follow up or death. A comparison cohort with active RA on disease modifying anti-rheumatic drugs (DMARD) was recruited and followed up in the same way. SI was defined as an infection requiring hospitalization or intravenous antibiotics or resulting in death. Incident cases of SI were identified from follow-up questionnaires and verified via medical records. SI was attributed to ANA if it was diagnosed while on drug or within 30 days of the last dose. Event rates in the ANA and DMARD cohorts were compared using Cox proportional hazard ratio estimates adjusted for age, gender, disease severity, prior joint replacement, co-morbidity, year of entry into the study and steroid use.

Results: 111 patients received ANA as their initial biologic. Patients prescribed ANA were younger, had more severe disease, longer disease duration and higher exposure to steroids than the comparison cohort (Table). Drug survival on ANA was short (median survival on drug 5 months (IQR 3–12)) and 84% of patients switched biologic agent during follow up. In total 15 SI occurred in 10 patients on ANA, with the most common sites being respiratory and skin / soft tissue. 3 ANA treated patients died from SI. The crude rate of first SI was 92/1000 pyrs (95% CI 44, 170) in the ANA cohort compared with 34/1000

pyrs (30, 38) in the DMARD cohort. The unadjusted hazard ratio (HR) for first SI was 2.8 (1.5, 5.3) in the ANA cohort. After adjusting for confounding factors the HR was 2.5 (1.0, 6.4).
Conclusions: The rate of SI in the ANA cohort appears higher than in the DMARD cohort; however, the number of events in the ANA cohort were small and there may be residual confounding.

No of patients ever received the drug	Control cohort n = 3515	Anakinra cohort n = 111
Person years follow up (pyrs)	9062	108
Age (mean / SD.)	60 (12)	56 (10)*
Gender (% female)	75	72*
Disease duration years median (IQR)	6	13*
DAS mean (sd)	5.1 (1.3)	6.4 (1.0)*
HAQ mean (sd)	1.5 (0.8)	2.0 (0.6)*
Steroid use %	23	50*
Total serious infections (n)	290	10
HR (unadjusted) (95% CI)	Ref	2.8 (1.5, 5.3)
Age and gender adjusted (95% CI)	Ref	3.7 (1.9, 7.0)
Fully adjusted (95% CI)	Ref	2.5 (1.0, 6.4)

*P values < 0.001

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330. THE ROLE OF MUSCULOSKELETAL ULTRASOUND IN POTENTIAL INFLAMMATORY ARTHRITIS: A COMPARISON OF ULTRASOUND FINDINGS WITH SUBSEQUENT DIAGNOSIS AND TREATMENT

G. Hirsch and R. Klocke
Department of Rheumatology, Dudley Group of Hospitals, Dudley, UK

Background: Musculoskeletal ultrasound (MUS) provides a more sensitive method of detecting synovitis than clinical examination. It may provide a useful diagnostic test in the early diagnosis of inflammatory arthritis (IA), that does not yet meet diagnostic criteria otherwise, but this remains to be proven. We have compared retrospectively the sonographic probability of IA with subsequent clinical diagnosis and treatment decisions.
Methods: We screened stored reports of MUS examinations made by one clinician (RK) between August 2007 and June 2009. Studies of the hands and/or wrist were selected that had been requested to investigate for potential inflammatory arthritis (IA). 69 studies were selected for analysis. These included patients newly referred and patients under follow-up for conditions previously felt to be non-inflammatory, whose symptom pattern had altered. 50/69 were female, 8/69 rheumatoid factor (RhF) positive and 5/69 anti-CCP antibody (ACPAb) positive. Median age was 52. Median time from scan to last follow-up was 6 months (0-20 months). Scan reports were reviewed and the probability of IA indicated was categorized as high, low or intermediate probability by a second clinician (GH), according to MUS findings and any opinion made in the report. Categorization was made without knowledge of the patient's subsequent diagnosis or medication. Data were recovered for each patient, including the date of last follow-up, working diagnosis at last follow-up, medication and RhF and ACPAb status.
Results: 19/69 (23%) patients were deemed to have high probability of IA on MUS. Of these, 11/19 (58%) were negative for both RhF and ACPAb, 3/19 (16%) were positive for both ACPAb and RhF. 18/ 19 patients with high probability on MUS were described as having IA on follow-up and 16/ 19 were on DMARD at follow-up. 7/19 commenced DMARD at or soon after MUS. 41/69 (59%) patients were deemed to have low probability of IA on MUS. Of these, 3/41 were described as having IA at follow-up and in 1/41 cases, diagnosis was not clear. Of these patients, 2/3 were negative for both RhF and ACPAb. 2/41 deemed low probability of IA on MUS were on DMARD at follow-up.
Conclusions: Our observations suggest that MUS may identify most cases with and without IA correctly amongst those patients who are clinically suspected to have IA, but do not yet reach a clinical diagnosis.The data further suggests that patients with negative RhF and ACPAb might benefit most from the inclusion of MUS in the diagnostic process.

US probability for IA *vs* subsequent diagnosis, treatment and serological parameters

US prob. for IA (n)	High 19 (27.5)	Int. 9 (13)	Low 41 (59.5)
RhF+(n)	5	1	3
ACPAb+ (n)	4	1	0
IA at follow-up (n)	18	2	3
DMARD at follow-up (n)	16	1	2

See text for further information regarding serological status.

Disclosure statement: All authors have declared no conflicts of interest.

331. DO TITRES OF RHEUMATOID FACTOR AND ANTI-CCP ANTIBODY PREDICT FOR THE PRESENCE OF INTERSTITIAL LUNG DISEASE IN RHEUMATOID ARTHRITIS?

Elizabeth Toberty, Elizabeth Coulson, Vadivelu Saravanan, Carol Heycock, Martin Rynne, Jennifer Hamilton and Clive Kelly
Rheumatology, Queen Elizabeth Hospital, Gateshead, UK

Background: Rheumatoid Factor (RF) and anti-citrullinated peptide (anti-CCP) are used to aid the diagnosis of Rheumatoid Arthritis (RA). Several observers have reported RF and anti-CCP positive patients develop more severe, persistent disease requiring aggressive therapy. One study also found that anti-CCP positivity may be associated with extra-articular manifestations of RA including interstitial lung disease (RA-ILD). Since RA-ILD leads to a significant increase in mortality, these antibodies may have prognostic value and may aid in identifying such patients early. We assessed the presence and titres of RF and anti-CCP in a group of patients with RA-ILD and age and gender matched controls with RA only. We then compared the frequency and titres of RF and antiCCP between the two patient groups.
Methods: All patients on the hospital RA database were searched for the presence of systemic disease and those with HRCT-proven RA-ILD were identified. Their notes were examined to confirm the diagnosis and each individual was matched for age and gender to 3 other RA patients with no clinical, physiological or radiological evidence of ILD. Patient demographics were obtained from notes and autoantibody levels from the laboratory reporting system. The number of patients in whom the assays were available was recorded, together with titres and the data compared between the two groups. We assumed RF titres of less than 1:80 and anti-CCP levels < 5 to be clinically insignificant.
Results: We identified 23 RA-ILD patients (12 male, 11 female) and 69 RA only case controls (36 male, 33 female). RF results were available in 100% of patients with RA-ILD and in 94% of RA controls. Anti-CCP results were available in 40% of both groups. Positive RF was found in 68% RA-ILD and 28% RA only patients [$P = 0.005$]. Anti-CCP was found in 92% RA-ILD patients and 17% RA only patients [$P = 0.01$] where results were available. Median titres of RF (1/320 *vs* 1/120) and anti-CCP (88 *vs* 14) were significantly greater in those with RA-ILD compared with RA only controls [$P < 0.05$ for both]
Conclusions: RF and CCP antibodies appear to be useful markers of systemic disease in RA and are strongly linked to the presence of ILD. CCP outperforms RF in terms of both specificity and sensitivity for RA-ILD and elevated titres of both carry a high positive predicted value for the presence of ILD. CCP should be measured in all patients with RA. This may prove valuable in prospectively identifying patients with RA-ILD needing specific therapeutic intervention.
Disclosure statement: All authors have declared no conflicts of interest.

332. EFFICACY AND SAFETY OF INTRAVENOUS IRON IN RHEUMATOID ARTHRITIS

Rachel Tsang, Elizabeth Coulson, Vadivelu Saravanan, Carol Heycock, Jennifer Hamilton and Clive Kelly
Rheumatology, Queen Elizabeth Hospital, Gateshead, UK

Background: Anaemia is common in rheumatoid arthritis (RA) and most often reflects disease activity or iron deficiency. Iron deficiency anaemia (IDA) is usually treated using oral supplements but these are not always effective. Parenteral iron supplements have been used for decades but have a reputation for inducing unwanted side effects. Intravenous (IV) iron, in particular, was reported as causing acute flares in RA and fell out of favour as a consequence two decades ago. However, newer formulations of IV iron have been available for some time and the efficacy and safety of these have not been previously studied in RA.
Methods: All RA patients receiving IV iron for correction of IDA over two years were included in this study. All had previously failed to tolerate or respond to oral iron. Haemoglobin (Hb) and erythrocyte sedimentation rate (ESR) levels pre and at 1, 2 and 3 months post infusion were measured and documented. Medical notes were reviewed for any infusion reactions or evidence of disease flare in the three months post infusion. In addition, patients were asked whether they noticed any change in their disease activity or fatigue over this time by telephone questionnaire.
Results: 42 RA patients received IV iron, mainly in the form of Cosmofer (iron dextran,) over a 24 month period. They accounted for

22% of all patients requiring parenteral iron therapy over this time. Their mean age was 70 (44-92 years). The mean pre infusion Hb was 9.9 mg/dl (7.2–11.6). This improved to 11.3 at one month and the benefit was maintained at two (mean Hb 11.2) and three months (mean Hb 11.5) post treatment. This improvement was statistically significant at each month ($P < 0.001$). Mean ESR prior to treatment was 38 mm/h (7-108 mm/h) and remained stable at 1 month, 2 months and 3 months after treatment (33 mm/h, 38.6 mm/h and 33.3 mm/h respectively). The change in haemoglobin was independent of the baseline ESR, being identical at 1.6 gm over 3 months in those patients with a normal ESR at baseline and in those with an elevated baseline ESR. 71% of patients reported improvement in fatigue and none reported deterioration in their disease activity over the ensuing 3 months.

Conclusions: IV iron did not cause significant flares in disease activity in patients with RA on conventional treatment regimes. This approach was effective in correcting IDA and was sustained at three months. This does not appear to be due to an effect of IV iron on RA disease activity. Modern preparations of IV iron may be less immunogenic than those used previouslly and present drug regimes may help prevent any disease flares following therapy. Investigations of the cause of IDA still need to be undertaken. We recommend that IV iron is considered for all RA patients with IDA who fail to respond to or to tolerate oral iron supplements.

Disclosure statement: All authors have declared no conflicts of interest.

333. FIVE-YEAR OUTCOME OF EARLY ARTHRITIS CLINIC: A NICE EFFECT ON THE PATIENTS' OUTCOME

Yasser El Miedany and Deborah Palmer
Rheumatology, Darent Valley Hospital, Dartford, UK

Background: The current paradigm for early arthritis suggests that the inflammatory process is at its peak in the first few years, leading to erosive joint damage and functional disability. It is therefore clear that halting the damage early may have significant long-term benefits. The latest NICE guidelines (2009) supported the recent trend of early diagnosis and management of the condition. An early arthritis clinic has been set up at Darent Valley Hospital, Dartford, for the past 5 years.

Objective: To assess the outcome as measured by health status, disease activity and radiographic progression in a cohort of patients with persistent early inflammatory arthritis monitored for 5 years in a specialized early arthritis clinic.

Methods: Patients presenting with early arthritic symptoms and disease duration < 6 months were assessed regularly every 3 months in the first year, then every 6 months in a specialized early arthritis clinic. Disease modifying antirheumatic drug (DMARD) therapy was initiated once the diagnosis is established. Initially, methotrexate monotherapy was the first option. A second DMARD was added in cases where the arthritis remained persistent. This changed to combination therapy as the first option following the recent NICE guidelines (2009). Biologic therapy was commenced in cases of 2 DMARD failure. Disease process was assessed by clinical, radiological (X-ray and US) and laboratory measures of disease activity. Evaluation of disease course was carried out using DAS-28.

Results: 131 patients with early arthritis (mean disease duration $4.4 + 1.46$ months) were followed in the clinic. Baseline (prior to treatment) DAS-28 mean score was $5.05 + 0.17$. MTX monotherapy (mean dose $22.5 + 2.4$, mean treatment duration $8.4 + 0.62$ months) failed in 64.9% (85/131) to achieve clinical remission in disease activity and a second DMARD was added (SZP in 42 patients and leflunomide in 43 patients) At the end of the second year of combined DMARD therapy 35/42 (83.3%) of the MTX/sulfasalazine group did not achieve clinical remission, similarly 33/43 (76.7%) in the MTX/leflunomide group did not achieve remission. There was significant ($P < 0.01$) radiographic progression in the 3 groups. Mean time to start biologic therapy was $14.3 + 1.8$ months. This was cut down by the latest NICE guidelines to $6.4 + 0.52$ months. By the end of year 5, 72/131(55%) of the patients were on biologic therapy. Mean DAS-28 among biologic therapy group was $3.0 + 0.15$, whereas in the DMARD group was $3.9 + 0.46$.

Conclusions: Disease activity is high early in the disease course. NICE guidelines (2009) had a positive impact on the management of patients with early arthritis. In concordance with NICE guidelines, it is advisable to commence DMARD therapy in combination early in the disease course. Early referral to a rheumatologist for definitive diagnosis and early DMARD treatment has improved the long-term outcome of RA.

Disclosure statement: All authors have declared no conflicts of interest.

334. RATES AND CAUSE OF DEATH IN RHEUMATOID PATIENTS: ARE PATTERNS CHANGING?

David Collins, Tracy Arnold, Maria Juarez, Rosemary Waller, Lyn Williamson and Elizabeth Price
Rheumatology, Great Western Hospital, Swindon, UK

Background: Current figures suggest an excess mortality in patients with Rheumatoid Arthritis (RA) with reported standardized mortality ratios (SMR) varying between 1.27 and 2.70. It is not clear whether survival has improved over recent years. Cause specific mortality reveals excess deaths predominantly being due to infection, cardiovascular disease and respiratory disease. The majority of studies reported predate the use of biological therapies and the trend for using disease-modifying drugs (DMARDS) in combination. We have reviewed our own recent mortality figures.

Methods: In our practice all patients receiving immunomodualtory drugs (including hydroxychloroquine) from a single primary care trust are managed on a prescribing and monitoring database (DAWN). Between 2004 and 2009, 1654 patients were recorded on the database, 157 had moved away leaving data available on 1496 patients.

Results: 91 deaths were recorded (6.08%; 36 Male, 55 Female) giving a SMR of 1.19 (95% CI 0.97-1.46 ns). Mean age at death was 71.3 years; (range 41–98 years). A predicted mean lifespan of 82.1 years was derived from age sex and regional tables (P 0.00028).

76 deaths were in RA patients (30 Male, 46 Female; mean age 73.1; range 50-98), SMR 1.32 (0.58-1.51 ns). Disease duration varied from 0.1 to 53 years (mean 9.5, median 5.0 years). 3 deaths occurred out of hospital and to date the cause remains unknown. Of the remaining 73 the main causes of death were cancer 21 (28.7%), cardiovascular 20 (26.3%), sepsis 15 (19.7%), trauma 6 (7.89%) and COPD 5 (6.6%). Of the cancer deaths lung (8), bowel (4), breast (2) and melanoma (2) were the majority. No lymphoma/leukaemia deaths occurred. Of the 15 sepsis deaths chest (8) and abdominal (5) predominated. 38 patients had been on corticosteroid therapy in the 5 years preceding death and 24 were on treatment at the time of death -no particular trends were noted. 11 patients were known to be diabetic, including 5 in the 15 sepsis deaths. 17 patents were not on DMARDS immediately prior to death, 48 were on monotherapy with methotrexate predominating, 10 were on 2-drug combination and 3 on 3-drug combination. No significant association between particular DMARDS or combinations and age or cause of death was noted.

Conclusions: Compared with published figures, our SMRs for all deaths and RA deaths are favourable, but age at death remains premature by approximately 10 years.

Previous reports have highlighted infection, respiratory and cardiovascular deaths above cancer and within cancer a preponderance of haematological malignancy.

Our own figures reveal cancer and cardiovascular deaths to be most frequent and no haematological malignancy, with infection being third most common. This is too small and localized a study to state confidently a change in pattern of RA outcome, but our results suggest a larger population study is required.

Disclosure statement: All authors have declared no conflicts of interest.

335. LESSONS LEARNED FROM QUANTIFYING BONE MARROW OEDEMA IN THE RHEUMATOID CERVICAL SPINE

Ravi Suppiah[1,2], Anthony Doyle[3], Raylynne Rai[4], Nicola Dalbeth[2,5], Maria Lobo[2], Jeurgen Braun[7] and Fiona McQueen[2,6]
[1]*Rheumatology, Nuffield Orthopaedic Centre, Oxford, UK;* [2]*Rheumatology, Auckland District Health Board, Auckland, New Zealand;* [3]*Radiology, Auckland District Health Board, Auckland, New Zealand;* [4]*Radiology, Counties Manukau District Health Board, Manukau, New Zealand;* [5]*Medicine, University of Auckland, Auckland, New Zealand;* [6]*Molecular Medicine and Pathology, University of Auckland, Auckland, New Zealand;* [7]*Rheumazentrum Ruhrgebiet, Ruhr-University Bochum, Herne, Germany*

Background: Magnetic resonance imaging (MRI) is an accepted modality for imaging the peripheral joints of patients with RA. In this setting the presence of MRI bone marrow oedema (BMO) predicts future erosions at that particular site. The risk of developing erosions in the peripheral joints can be attenuated by treatment with TNF inhibitors with recent evidence also showing that aggressive treatment of RA reduces the risk of accumulating damage in the cervical spine. If it were possible to determine which patients were more likely to have progressive cervical spine damage, this information could be incorporated into clinical decision-making regarding who should receive these expensive new therapies.

The aims of this study were to (1) to test the reliability and feasibility of a new scoring system to quantify BMO, synovitis and erosions in the cervical spine and (2) to determine whether neck pain or other markers of rheumatoid disease activity correlated with MRI BMO of the cervical spine.
Methods: Thirty patients with RA (50% with neck pain) and a DAS28 $>$ 3.2 had an MRI scan of their cervical spine. STIR, VIBE and T1w post gad sequences were used to quantify BMO. The MRI scans were scored for total BMO, synovitis and erosions using a new scoring method developed by the authors and assessed for reliability, feasibility and correlation between neck pain and clinical markers of RA disease activity.
Results: Overall pain scores (mean VAS 39.9 *vs*. 17.8, $P < 0.01$) and 68 tender joint counts (mean 25.0 *vs*. 13.7, $P = 0.03$) were higher in patients with neck pain; otherwise there were no significant differences in disease activity scores or disease duration between the groups. BMO was present in 14/30 patients; 9/14 (64%) had atlanto axial BMO, 10/14 (71%) had sub axial BMO and 5/14 (36%) had both. Inter-observer reliability for total BMO score was moderate (ICC = 0.51). The ICC improved to 0.67 if only the vertebral bodies and dens were considered. Spearman's rho for the correlation between total BMO score and the total erosion score was 0.70 ($P < 0.01$). There was a strong correlation between the total erosion score and total synovitis score (Spearman's rho = 0.88, $P < 0.01$) and also for synovitis at the same site as erosion (Spearman's rho = 0.60, $P < 0.01$). BMO, synovitis, or erosions in the cervical spine as determined by MRI did not correlate with neck pain or clinical measures of RA disease activity.
Conclusions: Current RA disease activity scores do not capture disease activity in the cervical spine. An MRI score that quantifies disease activity in the cervical spine may provide this information and predict future damage to this area. We propose a new simple method of quantifying disease activity in the cervical spine by scoring BMO, erosions and synoivitis based on the lessons learned from this study.
Disclosure statement: All authors have declared no conflicts of interest.

336. THE RELATIONSHIP BETWEEN ANTI-CYCLIC CITRULLINATED PEPTIDE ANTIBODIES AND CLINICAL PHENOTYPE IN VERY EARLY RHEUMATOID ARTHRITIS

Zaeem Cader, Andrew Filer, Christopher D. Buckley and Karim Raza
Rheumatology Research Group, Division of Immunity and Infection, University of Birmingham, Birmingham, UK

Background: Anti-cyclic citrullinated peptide (anti-CCP) antibodies are highly specific for RA. However, they are not detectable in all patients. This raises the possibility that distinct mechanisms exist for the pathogenesis of anti-CCP positive and negative disease. Indeed, anti-CCP positive patients show both genetic and environmental associations not present in anti-CCP negative RA. Furthermore, in established RA, synovial pathology differs between these subsets of disease and anti-CCP positive patients have poorer long-term outcomes. The aim of this study is to establish whether the clinical phenotype of anti-CCP positive and negative disease are distinct at the earliest clinically apparent phase of disease.
Methods: Patients were recruited from the Rapid Access Early Inflammatory Arthritis Clinic, Sandwell and West Birmingham Hospitals NHS Trust. Participants were included in the current study if they presented within 3 months of symptom onset and fulfilled 1987 ACR criteria for RA at some point during a systematic 18 month follow-up schedule. Data were collected on patient demographics, joint symptoms and tender ($n = 68$) and swollen ($n = 66$) joint counts. CRP, ESR, rheumatoid factor and anti-CCP2 status were measured.
Results: 92 patients were included (48 anti-CCP positive at inclusion; 50 female; median age 61 years [IQR: 49-70]; median symptom duration 47 days [IQR: 35-72]). The anti-CCP positive and negative groups were comparable in terms of age, gender, ethnicity, presence / duration of morning stiffness, symmetry, involvement of 3 or more joint areas and hand joint involvement at baseline. The only ACR classification criterion that differed between the groups was that the anti-CCP positive patients were more likely to be seropositive for rheumatoid factor (83.3% *vs*. 11.4%, $P < 0.01$). However there was no difference in the mean time taken to develop 4 or more ACR criteria and reach a diagnosis of RA (157.5 days *vs*. 130.7 days from symptom onset, $P > 0.05$) in those who did not fulfil classification criteria at baseline. There were also no significant differences in the total tender or swollen joint counts or levels of inflammatory markers. Furthermore there was no significant difference in the pattern of joint involvement on examination, except for an increased prevalence of knee joint swelling in anti-CCP positive patients (42.9% *vs*. 22.2%, $P = 0.03$).
Conclusions: Patients with and without anti-CCP antibodies present similarly, even at a very early stage of clinically apparent disease that eventually develops into RA. There is an emerging consensus that the presence of this antibody defines a distinct disease subset with specific genetic and environmental associations. Further work is needed to determine whether the shared clinical phenotype we report conceals differences at a pathological level with consequent therapeutic implications.
Disclosure statement: All authors have declared no conflicts of interest.

337. WORSENING LDL: HDL ATHEROGENIC INDEX DESPITE IMPROVEMENTS IN INFLAMMATORY POLYARTHRITIS PARAMETERS IN THE FIRST 2 YEARS OF DISEASE: RESULTS FROM THE NORFOLK ARTHRITIS REGISTER (NOAR)

Hoda Mirjafari[1], Tracey Farragher[1,4], Suzanne M. Verstappen[1], Diane Bunn[3], Valentine Charlton-Menys[2], Tarnya Marshall[3], Deborah P. Symmons[1] and Ian N. Bruce[1]
[1]*arc Epidemiology Unit, The University of Manchester, Manchester, UK;* [2]*Cardiovascular Research Group, The University of Manchester, Manchester, UK;* [3]*Norfolk Arthritis Register, Norfolk and Norwich University Hospital, Norfolk, UK;* [4]*Centre for Medical Statistics and Health Evaluation, The University of Liverpool, Liverpool, UK*

Background: The excess cardiovascular disease (CVD) in patients with inflammatory polyarthritis (IP) may be caused, in part, by traditional CVD risk factors such as dyslipidaemia. Inflammation is associated with a reduction in low density lipoprotein (LDL) and a greater reduction in high density lipoprotein (HDL) levels leading to an unfavourable atherogenic index (AI). In this study, we investigated whether changes in disease activity were associated with changes in AI in the early stages of IP.
Methods: Consecutive patients aged 18-65 years, with early IP (≥ 2 joints swollen for ≥ 4 weeks) within 24 months of symptom onset, were recruited as part of a primary-care-based inception cohort. Each patient was assessed at baseline and after 2 years. At both time points the patient completed a HAQ. A research nurse administered a standardised questionnaire including therapy and examined the joints. Fasting blood was taken for CRP and lipid level analysis including total cholesterol, HDL and LDL. AI was calculated as LDL divided by HDL. The 10-year Framingham risk score was calculated. The differences between baseline and 2 year data were examined using the paired t-test. Linear regression was carried out to assess the association between the changes in parameters after 2 years.
Results: 223 patients had completed two years follow-up by October 2009. 151 (68%) were female with a median (IQR) age at baseline of 50 (40-57) years and median (IQR) symptom duration of 7 (5–11) months. Median (IQR) DAS 28_{CRP} at baseline was 3.8 (3.0–4.8). 12 patients (5%) were on a statin therapy, 51 (23%) were smokers and 20 (9%) had a 10-year Framingham risk score of over 20%.

There was a significant reduction in the mean HAQ score and 'both swollen and tender' joint count after 2 years accompanied by a significant increase in the mean AI and fall in HDL (Table). There was a significant association between the reduction in 'both swollen and tender joint count' and increasing AI [coefficient (95% CI); -1.11 (-1.94, -0.29)]. There was not a statistically significant association between a reduction in HAQ and increase in AI [coefficient (95% CI); -0.03 (-0.16, 0.09)].
Conclusions: In this inception cohort of patients with early IP followed over a 2-year period, a successful reduction in IP disease parameters was accompanied by an unfavourable change in AI. Assessment and management of the dyslipidaemia associated with IP may require attention to the individual lipid parameters, independently of managing IP disease severity.

Variable	Baseline Mean (S.D.)	After 2 years Mean (S.D.)	Mean difference (95% CI)
HAQ score	0.95 (0.73)	0.81 (0.74)	-0.13 (-0.22, -0.04)
Swollen and tender joint count	3.73 (5.84)	2.00 (4.19)	-1.73 (-2.52, -0.94)
Atherogenic index	2.38 (1.00)	2.60 (0.95)	0.22 (0.11, 0.32)
LDL	3.22 (0.93)	3.31 (0.06)	0.09 (-0.02, 0.19)
HDL	1.47 (0.44)	1.36 (0.36)	-0.11 (-0.15, -0.08)
Total cholesterol	5.34 (1.07)	5.30 (1.00)	-0.04 (-0.15, 0.08)

Disclosure statement: All authors have declared no conflicts of interest.

338. PROTEINASE-ACTIVATED RECEPTOR-2 INFLUENCES TLR-4-MEDIATED INFLAMMATORY RESPONSES IN MACROPHAGES

Rachael Steven[1], Anne Crilly[1], John C. Lockhart[1], William R. Ferrell[2] and Iain B. McInnes[3]
[1]*School of Science, University of the West of Scotland, Paisley, UK;* [2]*Integrative Biology, University of Glasgow, Glasgow, UK;* [3]*Immunity, Infection and Inflammation, University of Glasgow, Glasgow, UK*

Background: Proteinase activated receptor-2 (PAR-2) is a cell surface receptor activated by proteolytic cleavage, revealing a tethered ligand which binds to extracellular loop 2 resulting in intracellular signalling. Activation of PAR-2 has been shown to result in the enhanced secretion of pro-inflammatory chemokines and cytokines (1) whilst inhibiting it reduces synovial generation of TNFα (2). It has recently been suggested that PAR-2 activation may act in synergy with responses to toll like receptor 4 (TLR-4) agonists (3). This study investigated the ability of PAR-2 antagonism to alter responses to LPS via TLR-4 in macrophages.
Methods: Human CD14 +cells were isolated from buffy coats. These cells were matured in M-CSF for six days before treatment with LPS alone (100 ng/ml) or LPS plus a PAR-2 antagonist (ENMD-1068, 200 μM). Cells were stimulated for 24 h and the supernatants analysed by ELISA for TNFα secretion. In parallel murine studies, bone marrow derived cells matured to a macrophage-like phenotype from wild-type and PAR-2 deficient mice were stimulated with LPS for 24 h and supernatants analysed for TNFα.
Results: Inhibition of PAR-2 in CD14+ cells resulted in reduced response to LPS (880 ± 762 pg/ml; mean ± S.D., n = 5) as assessed by secretion of TNFα, compared with vehicle treated cells (1362 ± 1061 pg/ml). Overall, a 35% decrease in TNFα secretion was observed when cells were cultured with the PAR-2 antagonist ($P = 0.04$ paired t-test). Trypan blue staining showed that cell viability was unaffected by the antagonist. Similar studies conducted in PAR-2 null mice also showed that bone marrow derived macrophages produced less TNFα in response to LPS (204 ± 50 pg/ml) compared with their wild-type littermates (1418 ± 75 pg/ml, $P = 0.002$, n = 4).
Conclusions: Collectively, these data suggest that PAR-2 may interact synergistically with TLR-4, since inhibition of PAR-2 activation led to a blunted LPS response in macrophages. Our data suggest an important role for PAR-2 in innate inflammatory responses.
Disclosure statement: All authors have declared no conflicts of interest.
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339. CHANGES IN ADVANCED GLYCATION ENDPRODUCTS IN SYNOVIAL FLUID IN EARLY RESOLVING ARTHRITIS AND EARLY AND ESTABLISHED RHEUMATOID ARTHRITIS

Usman Ahmed[1], Naila Rabbani[1], Andrew Filer[2], Richard Watts[3], Karim Raza[2] and Paul Thornalley[1]
[1]*Warwick Medical School, University of Warwick, Coventry, UK;* [2]*College of Medical and Dental Sciences, University of Birmingham, Birmingham, UK;* [3]*Department of Rheumatology, Ipswich Hospital NHS Trust, Ipswich, UK*

Background: Proteins damaged by AGEs often exhibit functional impairment and undergo cellular proteolysis. Such protein modification can result from chronic persistent inflammation by associated decrease in the enzymatic defence against glycation. The aim of this study was to assess if advanced glycation endproducts (AGEs) residues in synovial fluid proteins were increased in established rheumatoid arthritis (RA) compared with early RA and resolving inflammatory arthritis (non-RA).
Methods: Patients were recruited from the Rheumatology clinic at Ipswich Hospital (established RA) and the rapid access early synovitis clinic at Sandwell and West Birmingham Hospitals NHS Trust (early RA and non-RA). The age (years; mean ± S.D.) of subjects was (mean ± S.D.): non-RA 37 ± 11 ($n = 10$, 2 female), established RA 65 ± 20 ($n = 8$; 6 female), early RA 65 ± 11 ($n = 10$; 6 female). Patients with early RA and non-RA had a symptom duration of 9 ± 5 weeks. The duration of established RA was 9 ± 7 years. The concentrations of AGE residues were assayed by stable isotopic dilution analysis liquid chromatography with tandem mass spectrometric detection (LC-MS/MS) in exhaustive enzymatic digests of synovial fluid protein. Significance of difference between study groups was assessed by Kruskal-Wallis and Mann-Whitney U test.
Results: Synovial protein contents of methylglyoxal-derived hydroimidazolone (MG-H1) and 3-deoxyglucosone-derived hydroimidazolone (3DG-H), major arginine-derived AGEs, and Nε-carboxymethyllysine (CML), Nε-carboxyethyl-lysine (CEL), major lysine-derived AGEs, are reported (mmol/mol amino acid modified) (Table).

AGE content of synovial protein of nonRA and eRA was similar. CML, MG-H1 and 3DG-H were increased in established RA compared with early RA ($P < 0.001$).
Conclusions: Major quantitative AGEs, MG-H1, 3DG-H and CML were increased in synovial proteins of patients with established RA. Further work is necessary to determine the impact of this protein modification on disease processes operating in the established rheumatoid joint.

	Patient group		
AGE	nonRA	earlyRA	established RA
MG-H1	0.250 (0.009 - 0.428)	0.271 (0.173 - 0.481)	0.976 (0.631 - 2.53)***,ooo
3DG-H	0.060 (0.001 - 0.096)	0.044 (0.022 - 0.080)	0.914 (0.499 - 1.20)***,ooo
CML	0.031 (0.019 - 0.982)	0.034 (0.021 - 0.056)	0.274 (0.156 - 1.21)*,ooo
CEL	0.034 (0.020 - 0.054)	0.029 (0.010 - 0.079)	0.029 (0.022 - 0.078)

Data are median (range). * and ***, $P < 0.5$ and $P < 0.001$ with respect to nonRA; ooo, $P < 0.001$ with respect to eRA.

Disclosure statement: All authors have declared no conflicts of interest.

340. MOVING FROM MONOTHERAPY TO TRIPLE THERAPY AS INITIAL THERAPY FOR EARLY RA: RESULTS FROM AN INCEPTION COHORT ON TREATMENT TRENDS FROM 2002 TO 2009

Elena Nikiphorou, Adam Young, Patrick Kiely, David Walsh and Richard Williams
ERAN, c/o St Albans City Hospital, St Albans, UK

Background: Remission has been the ultimate goal in the treatment of RA. In recent years treatment trends have changed with the introduction of early intensive combination and biologic therapies. There is evidence that with intensive treatment, disease activity has improved. In this study, we investigate the treatment trends and changes in DAS28 scores over a period of 7 years, 2002-2009 in an early RA network.
Methods: All patients with early RA (less than 2 years) and prior to initiation of DMARDs, were enrolled in an early RA inception cohort at 21 UK centres ($n = 1122$). Patients were assessed at diagnosis, 6 months and yearly, using standardised measures. We examined the time in months from onset of symptoms to initiation of therapy and the use of initial DMARDs and steroids according to year of recruitment. We also included frequency of remission as defined by DAS28 (<2.6) at 1 year. The mean age at diagnosis was 56.8 years, 68% were females, 60% were rheumatoid factor positive and 29% had erosions at baseline.
Results: The median time from onset of symptoms to first outpatient visit was 6 months, but varied from 7 months in 2002 to 4.5 months in 2009 (Table). The main delay was symptom onset to GP referral (median 4–6 months). The median time from first outpatients to first DMARD ranged from 0 to 1 months. 1042 (93%) patients received DMARD therapy ± steroids. Overall, monotherapy was used in 34%, monotherapy plus steroids in 52% and the remainder had combination therapy (two concurrent drugs) or triple therapy (methotrexate, sulphasalazine and hydroxychloroquine). The Table shows the trend towards triple therapy rather than monotherapy and more use of steroids over time. Sulphasalazine was initially the treatment of choice as monotherapy, but was later replaced by methotrexate. Increased remission rates were seen over time, 32% of patients achieving remission by 2008, compared with 22% in 2003. Details of actual timing and type of DMARDs, steroid dosages and change in DAS28 will be displayed graphically.

Conclusions: In recent years, more intensive initial treatment has been used in this cohort, with an increasing proportion of patients started on combination or triple therapy. A greater number of patients achieved remission over the same period of time.

Recruitment year	Total	2002	2003	2004	2005	2006	2007	2008	2009
n=	1122	51	109	178	232	207	150	137	58
Onset to OPD *		7.0	7.0	6.0	6.0	7.0	5.5	6.0	4.5
OPD to 1st DMARD *		0	1.0	1.0	1.0	1.0	0	1.0	0
Monotherapy †	34	45	42	40	39	32	30	25	19
Monotherapy + steroids †	52	45	51	54	52	54	54	55	40
Combination therapy †	2	0	3	1	1	1	1	2	2
Combination therapy + steroids †	5	9	3	4	6	8	4	3	4
Triple therapy †	3	2	0	1	0	1	3	6	19
Triple therapy + steroids †	4	0	1	0	2	4	8	8	15

*median in months †% of year

Disclosure statement: All authors have declared no conflicts of interest.

341. THE ROSE ANGINA QUESTIONNAIRE DOES NOT IDENTIFY THOSE PATIENTS WITH INFLAMMATORY POLYARTHRITIS WHO ARE AT GREATEST RISK OF PREMATURE CARDIOVASCULAR MORTALITY

Muhammad Iskandar[1], Tracey Farragher[1], Diane Bunn[2] and Deborah Symmons[1]
[1]*arc Epidemiology Unit, University of Manchester, Manchester, UK;* [2]*Norfolk Arthritis Register, University of East Anglia, Norwich, UK*

Background: The Rose Angina Questionnaire (RAQ) was developed for use in epidemiological studies to identify individuals who are likely to be suffering from angina. It has been shown to predict all cause and cardiovascular (CVD) mortality in the general population. Its value in patients with inflammatory polyarthritis (IP) and its subset rheumatoid arthritis (RA) has not been established. The aim of this study was to investigate whether a positive RAQ predicts cardiovascular mortality amongst patients with IP.
Methods: 2,013 patients registered with the Norfolk Arthritis Register (NOAR), a primary care based inception cohort of patients with recent onset IP, completed at least one RAQ between 2001 and 2006 as part of their annual follow-up. They were followed from the time they completed their first RAQ until 31 December 2008. These annual assessments also included a co-morbidity questionnaire and joint examination. Patients completed a HAQ at each annual assessment. All patients were flagged for mortality with the Office for National Statistics. Cox proportional hazard models were used to estimate the risk of mortality associated with a positive RAQ adjusted for age and gender.
Results: The cohort comprised 678 men and 1,355 women. Median age at symptom onset was 54 years (IQR 42.6, 65.6). 903 (44.4%) satisfied the ACR criteria for RA at baseline and 689 (33.9%) were rheumatoid factor positive. The median symptom duration at completion of the first RAQ was 47 months (IQR 10.4, 121.4). 176 patients (8.7%) were RAQ positive at this timepoint. A positive RAQ was associated, cross-sectionally, with significantly higher swollen and tender joint counts, higher DAS-28 scores, higher HAQ scores and a self-reported history of hypertension, angina and myocardial infarction. Compared with the general population of Norfolk, the SMR for CVD was increased in both RAQ positive (SMR 2.45; 95% CI 1.27, 4.29) and RAQ negative patients (SMR 2.18; 95% CI 1.77, 2.65). The risk of CVD mortality was no higher in patients with a positive RAQ than in those with a negative RAQ (hazard ratio 0.9; 95% CI 0.49, 1.73 adjusted for age and gender).
Conclusions: IP patients with a positive RAQ are no more likely to experience premature CVD mortality than RAQ negative patients. The problem is related to high prevalence of false negative RAQs: not to a high prevalence of false positives. CVD is more likely to be clinically silent in patients with IP and RA than in the general population.
Disclosure statement: All authors have declared no conflicts of interest.

342. FURTHER ANALYSIS OF HELPLESSNESS MEASUREMENT IN INFLAMMATORY ARTHRITIS/ SPONDYLOARTHRITIS: THE DEVELOPMENT OF THE MODIFIED RHEUMATOLOGY ATTITUDE INDEX

Yasser El Miedany[1], Maha El Gaafary[2] and Deborah Palmer[1]
[1]*Rheumatology, Darent Valley Hospital, Dartford, UK;* [2]*Community, Environmental and Occupational Medicine, Ain Shams University, Cairo, Egypt*

Background: We assessed validity, reliability and sensitivity to change of a new questionnaire for assessment of emotional deficit and sense of helplessness among patients with rheumatic diseases.
Methods: Using Rasch analysis and 17 questions item pool (derived from interviews with patients suffering from inflammatory arthritis and spondyloarthritis); content analysis and semi structured group discussion, the modified rheumatology attitude index (mRAI) was developed including: 10-items scale. The mRAI is scored using the numeric VAS on 0–10 scale. The newly developed scale was studied in 241 RA patients, 211 psoriatic arthritis, 134 patients with IBD associated arthritis and 104 ankylosing spondylitis (AS) patients. Construct validity was assessed by correlating the score of the questionnaire to arthritic parameters of disease activity namely, the joint count (both tender and swollen), pain score, patient and physician Global assessment, fatigue score, HAQ, duration of morning stiffness, DAS-28 score and the newly developed combined inflammatory arthritis questionnaire for functional impairment (CIAQ-FI) and quality of life scores (CIAQ-QoL). In AS, the mRAI score was correlated with BASFI, BASDAI, BASG and the newly developed ASDAS. Test-retest reliability at 2 weeks time was assessed. Sensitivity to change after 3-months of biologic therapy was also assessed (this included 161 patients with inflammatory arthritis and 84 patients with AS/spondyloarthritis). All patients included in the study were asked to rate the questionnaire comprehensibility.
Results: The mRAI (total score 0–10) showed accepted validity as it correlated significantly ($P < 0.01$) with clinical parameters of disease activity, DAS-28 score, CRP as well as both CIAQ-FI and CIAQ-QoL. Similar significant correlation was found with AS parameters of disease activity ($P < 0.01$). The mRAI was also reliable (Cronbach's alpha 0.89-0.92 in arthritis patients and 0.88–0.91 in AS patients). In addition the mRAI was sensitive to change. A significant correlation was observed in percentage of change and effect size of the mRAI and changes in disease activity parameters as well as DAS-28 score ($P < 0.001$). Also significant correlation ($P < 0.001$) was seen on assessment of the effect size of BASG, BASFI, BASDAI as well as ASDAS. The mRAI questionnaire showed also a high degree of comprehensibility (9.3).
Conclusions: The mRAI is a valuable tool which is reliable and valid for assessment of self-helplessness among patients with inflammatory arthritis as well as AS. One potential use of the mRAI is to identify patient subgroups that, more likely, would benefit from psychological counseling and patient education. The mRAI is well accepted by patients, sensitive to change, easy to administer and score and more feasible to monitor the impact of management.
Disclosure statement: All authors have declared no conflicts of interest.

343. UTILITY OF ULTRASOUND JOINT COUNTS AS PREDICTORS OF OUTCOME IN PATIENTS WITH VERY EARLY ARTHRITIS

Andrew Filer[1], Paola de Pablo[1], Gina Allen[4], Peter Nightingale[2], Alison Jordan[3], Paresh Jobanputra[3], Chris Buckley[1] and Karim Raza[1]
[1]*Rheumatology Research Group, University of Birmingham, Birmingham, UK;* [2]*Wolfson Research Laboratory, University Hospital Birmingham Foundation Trust, Birmingham, UK;* [3]*Department of Rheumatology, University Hospital Birmingham Foundation Trust, Birmingham, UK;* [4]*Green Templeton College, University of Oxford, Oxford, UK*

Background: Early aggressive therapy improves outcomes in RA and data suggest that the first 3 months of disease may represent a therapeutic window of opportunity. It is therefore important to accurately predict the development of RA in patients during this window. Musculoskeletal ultrasound (MSUS) is more sensitive than clinical assessment in the detection of joint swelling and more sensitive than conventional radiography in the detection of erosive disease. We therefore evaluated the contribution of MSUS joint assessment as a potential predictor of outcome in patients with very early disease.
Methods: We prospectively recruited participants with clinically apparent synovitis of at least one joint and inflammatory joint symptoms of ≤ 3 months. We collected baseline clinical information (66 tender and swollen joint counts, DAS28 score), serological data and conventional radiography of hands and feet. Within 24 h of clinical evaluation, a blinded ultrasonographer systematically assessed 50 joints, grading greyscale synovitis (GS) and Power Doppler (PD) using four point semi quantitative scales and recorded the presence of erosions (Siemens Antares scanner and multifrequency linear array transducers). Participants were followed prospectively for 18 months.
Results: 58 participants were included with a mean age of 56 (18–83). Of these, 16 had resolving disease, 13 developed non-RA persistent disease and 29 developed very early rheumatoid arthritis (VERA) of

which 14 were ACPA positive. Overall, MSUS detected significantly more joint involvement than clinical examination in all regions. Amongst VERA participants, detection of PIP, MCP and shoulder involvement was not significantly increased by Greyscale MSUS assessment. However clinically silent involvement of wrist ($P < 0.01$), elbow ($P < 0.01$), knee ($P < 0.01$), ankle ($P < 0.01$) and MTP ($P < 0.001$) regions was identified significantly more often by MSUS. MSUS detected significantly more erosions (in 24 joints of 10 patients, one ACPA negative) than radiographs, which revealed only one wrist erosion. The most sensitive predictors for RA were clinical involvement of MCP joints and hand arthritis. The greatest specificity was shown by ACPA positivity, clinical polyarthritis, clinical involvement and symmetry of MTP joints and the following MSUS variables: MCP joints, PIP joints, wrist symmetry and MTP power doppler involvement and symmetry. Regarding predictors of persistence, variables with the greatest PPV were clinical and MSUS MCP, wrist and MTP involvement and symmetry and MSUS PIP involvement. Predictive models for the development of RA and persistence of disease are presented.

Conclusions: This prospective study shows that MSUS evaluation of multiple joints significantly increases detection of joint involvement in all regions and all outcome groups. Selection of appropriate variables can improve the ability of predictive models to identify those patients requiring treatment.

Disclosure statement: All authors have declared no conflicts of interest.

344. A SUPRA-DISTRICT AUDIT OF THE MANAGEMENT OF RHEUMATOID ARTHRITIS IN ADULTS (2009 NICE GUIDANCE)

Ruth Gordon[1,2] and Neil Snowden[1,2]
[1]*Rheumatology and Clinical Audit, Pennine Acute NHS Trust, North Manchester General Hospital, Manchester, UK;* [2]*Greater Manchester Rheumatology Supradistrict Audit Group, Greater Manchester, UK*

Background: Recently published NICE guidance (2009) gives a framework for the management of RA. The National Audit Office RA Census (2009) suggests that there are wide variations in the level of care available to people with RA across the UK, with shortfalls in many areas. We audited performance against key NICE standards across Greater Manchester.

Methods: We used retrospective case note review and a patient survey to focus on the key priorities for implementation of the 2009 NICE Guidance for Management of RA. We developed audit proformas (approved by the NICE Audit department), covering the following areas:

1) Referral for specialist treatment
2) The multidisciplinary team
3) Disease modifying drugs
4) Monitoring of disease activity.

Rheumatology services based at 10 hospital and community sites across Greater Manchester were asked to collect data on up to 60 outpatients with inflammatory arthritis. One hundred patient surveys per site were distributed at random to patients with inflammatory arthritis.

Results: Data was collected from 337 sets of case notes and patients completed and returned 331 (33%) of surveys distributed. Results are presented from patients with a clinical diagnosis of RA only.

According to the patient survey, most (95%) patients were satisfied with most aspects of their care. However, there were delays in access: patients waited on average 54 days for their first appointment with a specialist. Access to multidisciplinary and community support were both poor, with 37% patients not being offered access to any multidisciplinary or community support.

Although NICE recommends combination therapy with DMARDs, the vast majority (90%) of patients on all sites were receiving monotherapy. NICE recommend that in recent onset active RA, key components of disease activity should be measured monthly to inform decisions about treatment. Use of validated disease activity scores was documented in less than 50% cases and very few sites offered review at monthly intervals for patients with early disease (5% patients with early disease were seen monthly). Significant variations were noted in the management of RA between trusts.

Conclusions: Although patients are generally satisfied with their rheumatological management, most local rheumatology services have difficulty providing a service that is compliant with the NICE guidelines. The service shortfall is reflected in terms of delays in access, sub-optimal MDT support and difficulty providing detailed and frequent patient assessment. This data should inform resource allocation for rheumatology services.

Disclosure statement: All authors have declared no conflicts of interest.

345. DAS ESR AND DAS CRP: THE CLINICAL IMPLICATIONS OF SWITCHING SCORES

Catherine Gwynne[1], Nick Amos[2] and Jeremy Camilleri[1]
[1]*Rheumatology, University Hospital of Wales, Cardiff, UK;* [2]*Cardiff University, Cardiff, UK*

Background: The Disease Activity Score with 28 joints (DAS 28) using Erythrocyte Sedimentation Rate (ESR) for Rheumatoid Arthritis (RA) has been extensively validated for clinical practice and clinical trials. A DAS calculation using CRP was shown to be comparable to DAS ESR at a group level in an initial validation study. Subsequent studies however suggest that DAS CRP may underestimate disease activity.

The National Institute for Clinical Excellence (NICE) have adopted the DAS score to guide treatment decisions for biologic therapies: treatment is recommended if DAS 28 is greater than 5.1 on 2 occasions 1 month apart; adequate response to treatment is defined as an improvement in DAS 28 of 1.2 or more. Use of either ESR or CRP is not specified. In our institution DAS ESR is generally utilized. If an ESR is not available, CRP has been substituted.

This study aims to assess the correlation between DAS CRP and DAS ESR in our population and establish if there are clinical implications of switching between the scores.

Methods: 80 RA patients were selected from the University Hospital of Wales database, all of whom were treated with anti-TNF therapy. All had both DAS ESR and DAS CRP scores and a pre-treatment DAS ESR > 5.1. DAS ESR and DAS CRP correlation will be analysed by linear regression. We will analyse the impact of substituting the DAS CRP score.

Results: A significant correlation was found between DAS ESR and DAS CRP scores (R 2 = 0.90). However, pre treatment DAS score was significantly higher in the DAS ESR group (mean 6.59 *vs* 6.22, $P < 0.01$). If DAS CRP was substituted at the initial assessment, 5 patients (6.25%) would not have met NICE criteria to commence treatment (Mean DAS ESR 5.59, DAS CRP 4.90).

There was significant correlation between the change in DAS ESR and DAS CRP (R^2 = 0.84) following treatment. The Table shows the number of patients achieving an improvement of 1.2 or greater. Scores are similar if either DAS ESR or DAS CRP were used (responders 75% and 77%, respectively).

The most significant differences are noted when DAS CRP is substituted for either the pre or post treatment score (responders 71% and 85%, respectively). This infers that in these groups, patients would potentially be under- or over-treated.

Conclusions: Absolute values and change in DAS ESR and DAS CRP values correlate well on a group level, however, on an individual level values may vary. In our group, mean DAS CRP was significantly lower than DAS ESR and may underestimate disease activity. Interchanging DAS ESR and DAS CRP may lead to the over- or under-treatment of patients. It is strongly advised therefore to adhere to either ESR or CRP calculations. Further large scale validation of DAS CRP and treatment threshold values is needed.

Pre Treatment Score	Post Treatment Score	Number with pre treatment DAS > 5.1	Number (%) achieved response > 1.2	Number (%) did not achieve response > 1.2
DAS ESR	DAS ESR	80	60 (75)	20 (25)
DAS CRP	DAS CRP	75	58 (77)	17 (23)
DAS ESR	DAS CRP	80	68 (85)	12 (15)
DAS CRP	DAS ESR	75	53 (71)	22 (29)

Disclosure statement: All authors have declared no conflicts of interest.

346. INCORPORATING PATIENT REPORTED OUTCOME MEASURES IN CLINICAL PRACTICE: DEVELOPMENT AND VALIDATION OF A PROMS QUESTIONNAIRE FOR INFLAMMATORY ARTHRITIS

Yasser El Miedany[1], Maha El Gaafary[2], Sally Youssef[1] and Deborah Palmer[1]
[1]*Darent Valley Hospital, Rheumatology, Dartford, UK;* [2]*Community, Environmental and Occupational Medicine, Ain Shams University, Cairo, Egypt*

Background: Rheumatology is embarking on a fundamental redesign of rheumatic disease care. It became mandatory not only to recognize disease activity core set data, but also the risks for other co-morbidities associated with inflammatory arthritis. Measurement of patient reported outcomes have become critical in both standard clinical practice and long term observational studies.

We assessed validity, reliability and responsiveness to change of a patient self-reported questionnaire, which can assess construct outcome measures of patients with inflammatory arthritis.
Methods: 462 patients with inflammatory arthritis were included in this work. The questionnaire was developed by integrating information obtained from patients suffering from inflammatory arthritis based on the Rasch model for ordered response options. The questionnaire includes assessment for functional disability, quality of life, VAS for joint pain, global status, fatigue, duration of morning stiffness, review of the systems, falls and cardiovascular risks, the modified rheumatology attitudes Index (mRAI) and self-reported joint pain.
Results: The questionnaire was reliable as demonstrated by a high-standardized alpha (0.891-0.992). The questionnaire items correlated significantly ($P < 0.01$) with clinical parameters of disease activity. RA patient reported tender joints correlated significantly with the physician's scores (0.842). Changes in functional disability, quality of life as well as mRAI scores showed significant ($P < 0.01$) variation with diseases activity status. The PROMs questionnaire showed also a high degree of comprehensibility (9.4).
Conclusions: Integrating patient reported outcome measures into standard clinical practice is feasible and applicable. This version of multidimensional questionnaire was found to be valid and reliable. It provides informative quantitative measure for the disease activity core set data and in the mean time, facilitates assessing the patient's health-related quality of life measure, cardiovascular and falls risks on individual basis.
Disclosure statement: All authors have declared no conflicts of interest.

347. IS DLCO A USEFUL SCREENING TOOL IN DETECTING INTERSTITIAL LUNG DISEASE IN RA PATIENTS COMMENCING METHOTREXATE?

Suzy Silburn[1], Tom Pullar[1], Kumar Vinod[1] and Tom Fardon[2]
[1]*Rheumatology Dept, Ninewells Hospital, Dundee, UK;* [2]*Dept of Chest Medicine, Ninewells Hospital, Dundee, UK*

Background: Methotrexate (MTX) pneumonitis is a rare but potentially fatal complication of treatment, thought to occur more frequently in those with pre-existing interstitial lung disease (ILD). Pulmonary function tests (PFTs) are performed in all our rheumatoid arthritis (RA) patients within 2 months of commencing MTX. Those with a transfer factor (DLCO) of $<70\%$ predicted go on to have a high resolution CT (HRCT) chest if no other cause is identified, as suggested by Saravanan et al. in their review article.
Methods: Pulmonary function test results and patient data for all RA patients starting MTX in past 1 year were retrospectively reviewed ($n = 76$).
Results: Of 76 patients, 32 had a DLCO of $<70\%$ predicted. Of these 21 went on to HRCT chest. In only 3 of these patients was ILD detected. Each of these patients had at least one of the following chest X-ray (CXR) suggestive of ILD, audible chest crackles, exertional dyspnoea. Seven of those with a DLCO $<70\%$ predicted had an obstructive pattern on spirometry and 5 went on to have HRCT, which found changes only of obstuctive airways disease. The remaining 11 did not have HRCT for a variety of reasons including methotrexate already stopped due to side effects, clinical decision it was unnecessary and reason unknown. Follow up is between 2 and 12 months and no patients commenced on methotrexate have developed pneumonitis to date.
Conclusions: HRCT chest exposes the patient to more than 12 times the radiation dose of a plain CXR and ideally should be limited to those in whom the index of suspicion is high. In our patients DLCO was poorly specific for ILD.

The BSR guidelines suggest performing CXR and considering PFTs and HRCT. A CXR combined with symptoms of breathlessness and chest crackles on examination would have detected ILD in all our patients. We now question whether we should reserve PFTS for those patients with dyspnoea, abnormal examination findings or abnormal CXR. If performed, DLCO should be correlated with symptoms, examination findings, spirometry and CXR prior to considering HRCT.
Disclosure statement: All authors have declared no conflicts of interest.

348. A SYSTEMATIC REVIEW OF THE IMPACT OF RHEUMATOID ARTHRITIS AND ITS TREATMENTS ON QUALITY OF LIFE

Ian C. Scott[1], Gabrielle Kingsley[1] and David L. Scott[2]
[1]*Rheumatology Department, University Hospital of Lewisham, London, UK;* [2]*Rheumatology Department, Kings College Hospital, London, UK*

Background: RA impacts detrimentally on Quality of Life (QoL). In the absence of a cure improving QoL is a major focus of care. Whilst biologic therapies improve overall QoL, the extent of these benefits on individual health components have not been fully evaluated. The SF-36 is a commonly used generic QoL assessment tool. It assesses health in 8 different domains, which can be compiled into physical and mental component summary scores. It is reliable and valid in RA, correlating with disease specific measures like HAQ.

As the effects of RA on QoL have not previously been methodically evaluated we undertook a systematic review of this topic using the SF-36. Our first aim was to evaluate QoL in observational studies and RCTs of patients with RA using the SF-36 in comparison to patients with other long-term disorders and normal populations. Our second aim was to establish the impact of treatment with DMARDs and biologic therapies on QoL in RCTs of patients with RA assessed using the SF-36.
Methods: We searched MEDLINE and EMBASE from 1992 to 2009. We included observational studies, randomized controlled trials (RCTs) or experimental studies without randomization with QoL assessed in RA using SF-36. Studies had ≥ 50 patients per observational study/treatment arm and reported mean data. SF-36 was used as it is reliable in RA and allows comparisons between different patient populations. Studies of normal people and other common long-term disorders were included as comparators.
Results: 17 observational studies and 9 RCTs were included, evaluating 5090 and 4019 patients respectively. RA (in the pre-treatment RCT cohort) significantly reduced SF-36 scores in all domains compared with the normal population and other long-term disorders (Table). Unlike other chronic diseases RA impacted significantly on both physical and mental health. Low scores correlated with high disease activity. Meta-analysis showed biologics improved both physical and mental component summary scores with significant weighted mean differences of 4.97 (95% CI 3.46–6.49) and 3.25 (95% CI 1.20–4.84), respectively.
Conclusions: RA is unique in that it affects physical, social and mental health universally. Active disease is associated with low QoL. Its effects on mental health are comparable to that of major depression. Although treatment with biologics improves QoL these therapeutic effects are better for physical as opposed to mental health. An increased focus of care on the psychological impact of RA is required.
Disclosure statement: All authors have declared no conflicts of interest.

349. INCIDENCE AND PREDICTIVE VALUE OF THE CHARLSON CO MORBIDITY INDEX FOR OUTCOMES IN RA: RESULTS FROM AN INCEPTION COHORT

Gouri Koduri[1], Sam Norton[2], Adam Young[1], Nigel Cox[1], Peter Prouse[1], Josh Dixey[1] and Peter Williams[1]
[1]*ERAS Rheumatology, ERAS c/o St Albans City Hospital, St Albans, UK;* [2]*Centre for Lifespan and Chronic Illness Research, University of Hertfordshire, Hatfield, UK*

Background: RA is a chronic systemic inflammatory disease associated with premature mortality. Co-morbidity is an important

Cohort	Cases	Physical function	Role physical	Bodily pain	General health	Vitality	Social functioning	Role emotional	Mental health
RA observational	5090	47	34	44	46	46	65	56	66
RA RCT	2773	29	32	32	36	39	35	36	41
Normal population	2042	84	81	80	72	64	87	88	77
Knee osteoarthritis	547	68	61	63	60	59	76	80	72
Ankylosing spondylitis	314	71	44	44	51	43	70	66	70
Ischaemic heart disease	268	65	36	72	70	69	77	62	68
Depression	502	72	44	59	53	40	57	39	46
Diabetes	555	74	55	72	53	54	81	70	73

predictor of health outcomes whether measured by quality of life, functional disability, increase in hospitalization, psychological distress or mortality and has an impact on the use of health care resources. There are few validated methods to measure co-morbidity. The Charlson index is a weighted count of 17 co-morbid conditions, validated to predict 1 year mortality. The aim of the study is to determine the impact of co morbidity using Charlson scores on RA outcomes.

Methods: Consecutive patients with RA were recruited from 9 outpatient clinics in England to an inception cohort. Standard clinical and laboratory assessments were made at baseline and yearly. Type of employment, reasons for work loss and major co morbidities were recorded yearly. Date and cause of death were provided and coded (ICD10) by the Office of National Statistics.

Baseline clinical features were used as predictors in the incidence models. Cox regression was employed to generate crude, age-sex adjusted and fully adjusted hazard ratios (HR) for the presence of baseline Charlson co morbidity and outcomes. Mixed effects models with random intercepts and slopes were employed to examine the effect of baseline Charlson co morbidity on disability (HAQ).

Results: 1460 patients were recruited to the study, 66% females, mean age of onset of RA 55 yrs. 573 deaths occurred over maximum 23yrs follow up (mean 8.6 yrs). Prevalence of Charlson index >=1 was 17.5 % at baseline and increased to 53.7% by 15 years, with an annualized incident rate of 6.1%.

Using Cox regression analysis, baseline predictors for the 15-year incidence of Charlson co morbidity were older age (HR 1.04, 95% CI 1.03–1.05), past/current smokers (HR 1.28, 95% CI 1.02–1.60) and worse functional grade (HR 2.08, 95% CI 1.04–1.05). Charlson scores at baseline predicted mortality and work disability (Table). In addition, presence of Charlson comorbidity at baseline was associated with a worse baseline HAQ ($b = 0.04$, $P < 0.05$) and an increased rate of decline in HAQ scores over the course of follow-up ($b = 0.02$, $P < 0.05$).

Conclusions: This study reports the substantial presence of co-morbidities in RA, which have a significant impact on premature mortality, functional and work disability. Older age, smoking and baseline worse function predicted future Charlson co-morbidity. This data suggest that outcomes in RA may need to be adjusted for co-morbidities to fully reflect the course of the condition. In addition to routine therapy, better prevention strategies and early recognition of co existent disease are all important for better care.

Mortality	HR: Crude	Age/Sex adjusted	Fully adjusted
Charlson > 1	1.75 (1.42-2.16)	1.20 (0.97-1.49)	1.14 (0.78-1.67)
Charlson > 2	2.29 (1.59-3.28)	1.53 (1.06-2.19)	1.78 (1.01-3.13)
Work Disability (Charlson > 1)	1.72 (1.16-2.54)	1.53 (1.02-2.30)	1.89 (1.07-3.33)

Disclosure statement: All authors have declared no conflicts of interest.

350. CORRELATION BETWEEN PHYSICAL ACTIVITY IN PATIENTS WITH INFLAMMATORY ARTHRITIS AND BELIEFS ABOUT AEROBIC EXERCISE: A PILOT STUDY

Natasha Jones, Ravi Suppiah and Julia Newton
Sport and Exercise Medicine, Nuffield Orthopaedic Centre, Oxford, UK

Background: Patients with inflammatory arthritis have an increased morbidity and mortality from cardiovascular disease. Department of health recommendations are for > 150 min/week of aerobic activity. Regular aerobic exercise significantly reduces the risk of CV events in the general population. Current ACR guidelines recommend that regular aerobic exercise should be introduced as part of routine care for rheumatoid arthritis.

Our aims included:

1. To survey the ideas and perceptions about aerobic exercise in adult patients with RA, adolescents with JIA and parents of children with JIA.
2. To quantify the level of activity undertaken by these patients
3. To identify any correlation between the outcome expectations and perceptions about exercise with self reported physical activity levels.

Methods: 108 patients sequentially attending general rheumatology clinics.

A questionnaire containing 5 questions concerning exercise perceptions, fears and beliefs was given to adults ($n = 84$), adolescents ($n = 9$) and parents of children ($n = 19$) with inflammatory arthritis. This was answered on a 4 point Likert scale -2 (least positive) to +2 (most positive).

The same patients and parents completed internationally validated physical activity questionnaires (IPAQ, PAQ-A and PAQ-C)

Patients were also asked how many minutes per week of physical activity they think would be most beneficial to them.

Physical activity levels and positivity of outcome expectation were calculated for each group. Outcome expectation was then correlated with physical activity levels using the Spearman correlation.

Results: On a scale of -10 (least positive) to +10 (most positive), the mean outcome expectation among adults was 1.65, among adolescents was 5.1 and among children was 7. 51% of adults felt that exercise would damage their joints. 35% of adults felt that aerobic exercise would damage their health. 41% of adults felt that through aerobic exercise their fitness level would improve and they would feel better. 82% of adults agreed that aerobic exercise was good for their heart. Exercise levels were generally low with only 25% of adults scoring high on IPAQ. Exercise levels correlated significantly with outcome expectations in adults ($P = 0.01$). The correlation in adolescents and children was not significant ($P = 0.089$). Views about ideal exercise dose fell below department of health guidelines in all three groups.

Conclusions: Children and adolescents have higher outcome expectations of aerobic exercise than adults. Outcome expectation of aerobic exercise in adults correlates significantly with self reported physical activity level. Adults with inflammatory arthritis are generally poorly informed about the benefits of aerobic exercise. These findings suggest that improved education may improve physical activity levels in patients with inflammatory arthritis.

Disclosure statement: All authors have declared no conflicts of interest.

351. PROTECTING THEIR HEART: CARDIOVASCULAR RISK ASSESSMENT IN PATIENTS WITH RHEUMATOID ARTHRITIS

Anna E. Litwic and Joanna M. Ledingham
Rheumatology Department, Queen Alexandra Hospital, Portsmouth, UK

Background: Coronary heart disease is the most common cause of death in patients with rheumatoid arthritis (RA). British Society for Rheumatology's guidelines state the importance of recognition by health care providers that patients with RA are at high risk of cardiovascular disease (CVD) and that all patients with RA should have cardiovascular risk (CVR) screening on a minimum of an annual basis. A previous departmental audit in 2005 highlighted that our RA patients were not reliably having CVR assessments and action agreed from this audit was that the department would notify general practitioners (GPs) of the increased CVR, the need to undertake, as a minimum, an annual assessment of CVR and the need to take into account the increased risk when deciding on thresholds for targeted interventions in RA patients. The aim of our study was to assess how reliably this message was being sent to GPs and whether this had had impact on CVR assessments in our RA patients.

Methods: 100 consecutive patients attending our department with RA in 2009 were identified. Letters generated to their GPs in the preceding 12 months were analysed and the presence or absence of information supplied on the increased CVR associated with RA, the need for annual screening and the need to bear in mind the increased risk when deciding on thresholds for targeted interventions was noted. Blood results were also analysed for record of any tests relevant to CVR assessment (lipids and glucose).

Results: 70% of the 100 patients studied were women (average age 58 years) and 30% were men (average age 65 years). Information about the increased risk of CVD, the need for annual review of CVR and the need to take into account the increased CVR when deciding on thresholds for targeted interventions in RA was present in letters to GPs in 66% of all patients (76% of women and 43% of men; 70% of patients aged 40–75, 60% aged > 75 and 44% aged < 40 years). Lipid levels were measured in 72% of patients. For 60% of the 28 patients in whom lipids had not been checked in the preceding 12 months the patient's GP had been informed of the need for CVR assessment and had been asked to assess these. Information about the presence or absence of diabetes was available in 71% of patients (8% diabetic, 63% not diabetic, 29% not documented). Body weight and blood pressure were documented in all patients but information on other risk factors was less reliably identified within rheumatology records. Comparison with the 2005 audit data showed an increase in lipid and diabetes checks from 5%–72% and 14–71% of patients, respectively.

Conclusions: Screening of CVR in RA patients within our department has significantly improved since our last audit in 2005 but there remains room for improvement. The department plans to provide more information to patients directly, although patient education

programmes and through specific information leaflets, with the aim of informing patients and encouraging them to discuss their individual CVR with their GPs.
Disclosure statement: All authors have declared no conflicts of interest.

352. PHYSICAL ACTIVITY MIGHT COUNTERACT THE ADVERSE EFFECTS OF SMOKING CESSATION ON BODY WEIGHT AND COMPOSITION IN PATIENTS WITH RHEUMATOID ARTHRITIS

Antonios Stavropoulos-Kalinoglou[1,2], Giorgos S. Metsios[1,3], Vasileios F. Panoulas[1], Yiannis Koutedakis[3] and George D. Kitas[1]
[1]*Rheumatology, Dudley Group of Hospitals, Dudley, UK;* [2]*Schoold of Sport and Exercise Science, University of Birmingham, Birmingham, UK;* [3]*School of Sports, Performing Arts and Leisure, University of Wolverhampton, Walsall, UK*

Background: Smoking is an unhealthy habit and its cessation can improve several health parameters. In rheumatoid arthritis (RA), smoking has been implicated in the pathogenesis of the disease and could increase RA activity, making smoking cessation crucial in such patients. However, in the general population smoking cessation usually leads to significant weight gains. In RA, obesity is already highly prevalent and it associates with worse overall health and quality of life. Thus any smoking cessation regimes for RA patients should address potential adverse effects on body weight and composition. This study aims to identify associations of smoking and its cessation with body weight and composition of RA patients and also whether habitual physical activity could influence these associations.
Methods: A total of 392 patients (290 females) with RA were assessed for body mass index (BMI), body fat (BF), fat-free mass (FFM), waist circumference and habitual physical activity (PA) as well as smoking history (current smoker; ex-smoker; never-smoker) and intensity (pack-years). Results were standardized for disease activity and severity.
Results: Current smokers had significantly lower BMI compared with ex-smokers ($P < 0.05$) and never-smokers ($P < 0.05$). Similarly, the BF of current smokers was lower compared with that of ex-smokers ($P < 0.05$) and never-smokers ($P < 0.05$). Current smokers had a significantly smaller waist circumference compared with ex-smokers ($P < 0.05$) only. Following adjustments for age, disease duration and HAQ score, smoking remained a significant predictor for BMI ($P < 0.001$), BF ($P < 0.05$) and waist circumference ($P < 0.05$). Pack-years were inversely correlated with BF ($r = -0.46$; $P < 0.001$).

Following grouping of ex-smokers into three groups based on their PA, those in the lowest PA group had a significantly higher prevalence of obesity compared with those in the highest PA group ($P < 0.001$). Most significantly, ex-smokers in the highest PA groups had a similar prevalence of obesity to current smokers.
Conclusions: Cigarette smoking associates with a reduced BMI and BF in patients with RA. Smoking cessation appears to associate with increased BMI, BF and waist circumference in these patients. However, increased levels of physical activity seem to positively affect body weight and composition of patients who stop smoking. Given the numerous adverse effects of smoking on general health and RA, patients should be actively advised against it. However, smoking-cessation regimens in RA may need to include more general lifestyle counselling particularly about weight control. Increasing physical activity could prove to be an effective such intervention.
Disclosure statement: All authors have declared no conflicts of interest.

353. EVALUATION OF THE CLINICAL UTILITY OF ANTI-CCP ANTIBODY TEST IN DAILY CLINICAL PRACTICE

Jagdish Ramachandran Nair and Devesh Mewar
Rheumatology, Royal Liverpool University hospital, Liverpool, UK

Background: Anti-CCP antibodies are important markers for diagnosis and prognosis in RA. They may predict the development into RA in undifferentiated arthritis and are a marker of erosive disease.Their sensitivity is 50–75% and specificity 90–95%. Early diagnosis of RA is important as early treatment with DMARDs will induce remission and prevent functional impairment.

Most studies have examined well defined groups of patients in controlled settings. However the use of anti CCP antibodies in decision making as part of usual clinical practice has not been well studied and is of considerable interest. Evidence-based recommendations on its use are relatively scarce. Recently published NICE guidance on the management of RA recommends anti-CCP testing only in individuals who are RF seronegative or when extra prognostic information is required. Against this background, we evaluated the use of this test in the management of our patients in a teaching hospital. Our aim was to examine positive anti-CCP antibody tests at a University teaching hospital in the North-West of UK and to establish whether a positive test resulted in a change in diagnosis and management decision.
Methods: All requests for anti-CCP antibodies to the department of Immunology between Jan 2008 and May 2009 were evaluated.

Case notes and outpatient/GP letters were reviewed to establish reasons for carrying out the test, initial diagnosis, final diagnosis and the clinical outcome of a positive test.
Results: The number of anti-CCP antibody tests done between Jan 2008 and May 2009 were 510. Positive test results were obtained in 175 (35%). The positive results from various specialties included Rheumatology ($n = 164/469$, 35%), GP ($n = 0/10$, 0%) and other medical specialties ($n = 11/31$, 35%). 150 positive test results were analysed. The initial diagnosis considered included possible RA ($n = 45$), definite RA ($n = 101$), gout ($n = 1$), psoriasis ($n = 02$) and OA ($n = 1$). New referrals were 53 (35%) and a diagnostic uncertainty on initial interview was noted in 19 (35%) of the new patients. The test was used for assessment of disease status and prognostication in the remaining 131 cases. X-rays confirmed erosions in 83 (55%). RA was the final diagnosis in 133 cases? RA in 12 and definitely not RA in 2 cases. DMARDs were started in 32 (21%), drugs continued in 86 (57%), discontinued in 2 (1.3%) and surveillance was continued in 17 (11%) cases.
Conclusions: The anti-CCP antibody test is a useful test for diagnosis and prognostication.It is especially useful in new cases where there is some diagnostic uncertainty.

In our series of 19 patients in this category, we were able to initiate DMARD treatment 7 cases, stop drugs in 2 cases and maintain surveillance in others without drug treatment. This test influences clinical decision making in appropriate clinical circumstances. Our recommendation is that the anti-CCP antibody test should be used in appropriate clinical settings when there is a diagnostic uncertainty.
Disclosure statement: All authors have declared no conflicts of interest.

354. WHAT FACTORS PREDICT PNEUMONIA IN PATIENTS WITH RHEUMATOID ARTHRITIS?

Kin S. Long, Elizabeth Coulson, Vadivelu Saravanan, Carol Heycock, Jennifer Hamilton and Clive Kelly
QEH, Gateshead, UK

Background: Pneumonia occurs twice as often in rheumatoid arthritis (RA) as in age-matched controls and has a higher case fatality rate in RA. We have previously shown that this can be significantly reduced by a package of measures including pneumococcal vaccination. Recently, concern has been raised over the potential attentuation of benefit of vaccination by methotrexate (MTX). The present project was designed to assess the uptake of pneumococcal vaccination (pneumovax) among patients with RA and to compare the effectiveness of vaccination in preventing pneumonia in RA patients receiving MTX with a control group of RA patients who were prescribed disease modifying drugs (DMARDs) other than MTX.
Methods: We identified 53 consecutive outpatients with RA treated with oral MTX and defined their demographic details, treatment, smoking status, pneumococcal vaccination status and past medical history by questionnaire. We identified 53 MTX naive RA case controls with the same duration of RA and DAS scores, treated with other DMARDs and collated the equivalent data for them. We then examined all patients' medical notes and contacted their GPs to confirm details of treatment and date of any pneumovax administration. Patients were then telephoned to ensure these details were correct before data was compared by Students t test. Wilcoxon rank test was used to compare those with and without a history of pneumonia.
Results: Mean age was 67 years and mean duration of RA was 14 years in each group. Combined data revealed a mean DAS score of 3.8 (range 1.2–5.9) and that 60% were never smokers. 81% had been received pneumovax. 19% had COPD and 9% were on long term oral steroids. There were no differences between groups with regard to any of these variables. In total, 11 RA patients (10%) had developed pneumonia over the preceding 10 years and 4 of these were on MTX at that time [NS]. Among these 11, never having pneumovax (7) [$P = 0.03$], having COPD (6) [$P = 0.04$] and being on long-term steroids (5) [$P = 0.03$] all significantly predicted for pneumonia. Among the 4 who had been vaccinated, mean interval between vaccination and infection was 6 years.
Conclusions: This study showed no evidence that MTX reduced the effectiveness of pneumovax in preventing pneumonia in RA. Lack of vaccination was a major risk factor for pneumonia and time interval

since last pneumococcal vaccination may also be important. The use of oral steroids and presence of underlying lung disease also increased the risk of infection. We suggest that all RA patients receive pneumovax independent of DMARD therapy.
Disclosure statement: All authors have declared no conflicts of interest.

355. RHEUMATOID ARTHRITIS *VS* PSORIATIC ARTHRITIS: CLINICAL AND US ASSESSMENT OF THE WRIST AND HAND JOINTS

Yasser El Miedany, Sally Youssef and Deborah Palmer
Rheumatology, Darent Valley Hospital, Dartford, UK

Background: Though both rheumatoid arthritis (RA) and psoriatic arthritis (PsA) share synovitis and bone erosions as the common pathological feature. Enthesitis and tendinitis are particularly frequent and considered a classic PsA manifestation.

We assessed the ultrasonographic (US) features in the wrist and hand joints among RA in comparison to PsA patients and its correlation to clinical joint examination as reported by the specialist nurse as well as patient self reported tender joints.
Methods: Prior to clinical assessment each patient completed a patient reported outcome measure questionnaire whilst waiting for assessment in the rheumatology clinic. Clinical assessment was carried out by the specialist nurse and findings were recorded in a proforma. US examination of both wrist and hand joints was carried out by the same rheumatologist who was blinded to the patients' symptoms or clinical findings; using Esaote, Mylab25 (Italy) with a linear probe operating at 18 MHz. US examination included radiocarpal, intercarpal, MCP, PIP and DIP joints as well as flexor and extensor tendons of both hands. Correlations between both the patients' as well as physician reported outcome measures and US changes were carried out. Intra-observer agreement for US examination was assessed using Kappa statistics.
Results: In total, 57 patients were included in this work. This involved 30 RA patients (mean age 51.7 + 6.8 years, disease duration 10.4 + 7.3 years) and 27 PsA patients (mean age 50.4 + 7.4 years, disease duration 11.1 + 6.5 years). US examination revealed synovitis in the wrist joint in 15/30 (50%) and 14/30 (46.7%) RA and PsA patients, respectively. Synovitis of the small joints of the hands was prevalent in 19/30 (63.3%) and 17/30 (56.7%) RA and PsA patients. DIP joint was affected in 8/30 (26.7%) of the PsA patients, whereas dacylitis was prevalent in 4/30 (13.3%) of PsA patients. Bone erosions were present in 12/30 (40%) and 11/30 (36.7%) RA and PsA patients, respectively. Tendon involvement was prevalent in 10/30 (33.3%) and 14/30 (46.7%) in RA and PsA patients respectively. US changes were significantly correlated with both the patient's ($r = 0.847$) and the rheumatologist ($r = 0.715$) reported joint tenderness. There was discrepancy between the rheumatologist and the US findings (enhanced vascularity on Power Doppler assessment) in only 2 wrist joints. The intra-observer agreement between the US examinations were good ($k = 0.91$)
Conclusions: DIP joint affection and tendinopathy are US changes that are more prevalent in PsA patients. There was no significant difference observed on assessment of the wrist/MCP/PIP joints among both RA and PsA patients. Patient's self reported joint tenderness should be considered before deciding which joints to be scanned. Disagreement between the clinical assessment and the US findings is less likely.
Disclosure statement: All authors have declared no conflicts of interest.