spondylarthropathies (group 2), 22 patients with more than 3 years duration of rheumatoid arthritis (group 3) and 20 patients with arthralgia (group 4).

Results The total outlined synovial volumes in the finger joints before Gadodiamide injection and the number of bone erosions at baseline MRI were highly predictive of bone erosions one year later in patients with rheumatoid arthritis (group 1 and 3). The number of bones with erosions and oedema varied significantly between the patient groups. The strongest individual predictor of bone erosions at one year follow up was bone marrow oedema, if present at the wrist at baseline. Bone erosions on baseline MRI were in few cases reversible at follow up MRI.

Conclusion The total synovial volume in the finger joints before contrast, the number of bone erosions and presence of bone oedema in the wrist at baseline seems to be predictive for the number of bone erosions 1 year later and may be used in screening. The importance of very early bone changes on MRI and the importance of these findings reversibility remain to be clarified.

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OP0012 DOES HELICOBACTER INFECTION INCREASE THE RISK OF NSAID INDUCED PEPTIC ULCERATION IN PATIENTS WITH RHEUMATOID ARTHRITIS?

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Background Patients with rheumatoid arthritis (RA) frequently develop dyspepsia due to peptic ulceration. There has been conflicting data published on the interactive roles of non steroidal anti inflammatory drugs (NSAIDs) and colonisation of the gastric antrum with *Helicobacter pylori*.

Objectives We wished to assess the prevalence of peptic ulceration in dyspeptic RA patients and matched controls. We intended to calculate the relative risks (RR) of NSAID consumption and *Helicobacter* infection, both singly and in combination.

Methods We endoscoped 100 dyspeptic patients with RA and 50 age and sex matched controls with dyspepsia but no evidence of RA. We recorded all medication taken over the preceding 6 months and assessed the presence of *Helicobacter* using a CLO test at endoscopy.

Results Endoscopic evidence of peptic ulceration was found in 39% of RA patients and 12% of controls. ($p < 0.01$). Multiple ulcers were significantly commoner in the RA patients (15% v 2%). NSAIDs were used by 60% of RA patients and 22% controls ($p < 0.01$). *Helicobacter* was found in 41% of RA patients and 36% of controls (NS). The consumption of NSAIDs conferred a RR of 5.2, while the presence of *Helicobacter* gave an RR of 2.0 in RA patients. The RR for the combination of NSAID consumption and *Helicobacter* infection was 12.3.

Conclusion RA patients have more major and more multiple pathology at endoscopy than age, sex and symptom matched control subjects. This is mainly due to their increased NSAID consumption but is also contributed to by the presence of *Helicobacter* infection.

OP0013 PREVALENCE, CLINICAL EXPRESSION AND CAUSES OF ISCHAEMIC HEART DISEASE IN RHEUMATOID ARTHRITIS

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Background Cardiovascular mortality is increased in Rheumatoid Arthritis (RA). This may be due to early development and increased prevalence of Ischaemic Heart Disease (IHD).

Objectives We aimed to assess the prevalence, clinical presentation and risk factors of IHD in RA compared to matched controls.

Methods Prospective, random recruitment of 104 hospital outpatients: 67 RA, 37 osteoarthritis controls. IHD detection: Rose questionnaire, ECG, adenosine-stressed myocardial perfusion SPECT imaging. Assessments: demographics, body mass index (BMI), disease and drug history, Health Assessment Questionnaire (HAQ), current and cumulative inflammatory burden (ESR, CRP), Joint Count, Rheumatoid Factor, von Willebrand Factor (vWF), angiotensin converting enzyme (ACE), lipids, fibrinogen, homocysteine, H. pylori, CMV and C. pneumoniae titers.

Results RA and controls were matched for all classical IHD risk factors (age, sex, family history, smoking, hypertension, lipids, diabetes, BMI) and NSAID use. Despite this, IHD in RA was almost twice as prevalent (49%) as in controls (27%) [$p = 0.03$] in both sexes, clinically silent in 52% vs 20% [$p = 0.003$], high risk in 60% vs 70% and present a decade earlier in RA. ESR, CRP, fibrinogen, vWF, ACE were significantly higher in RA [$p < 0.001$]. By logistic regression, age, male sex, RA and BMI independently predicted IHD in the study population. Within the RA group, patients with IHD had higher age, male sex, blood pressure, triglycerides, BMI, H. pylori titers and steroid use than those without IHD [$p < 0.05$]; RA duration, activity, seropositivity or functional ability were not significantly different. Age, male sex and BMI were the only independent predictors of IHD in RA.

Conclusion IHD is highly prevalent, clinically silent, occurs early and may be the main cause of increased cardiovascular death in RA. Within a hospital population, RA is an independent risk factor for IHD, with clinical and outcome characteristics similar to type II diabetes. In RA, classical IHD risk factors and systemic inflammation may both play a role. Further research may elucidate the mechanisms involved and help design risk reduction strategies specific to RA.

OP0014 THE ACUTE PHASE RESPONSE DOES NOT FULLY EXPLAIN DYSLIPIDEMIA AND INSULIN RESISTANCE IN INFLAMMATORY ARTHRITIS

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Background An increased frequency of insulin resistance (IR) and dyslipidemia has been identified in inflammatory arthritis (IA). These metabolic disturbances may be attributable to disease activity and therefore were implicated in the excess cardiovascular disease mortality as reported in IA.

Objectives In the present study we evaluated insulin sensitivity and lipid metabolism, as well as the potential role of the acute

phase response as a predictor of these metabolic disturbances in IA.

Methods Eighty seven patients (38 rheumatoid arthritis, 29 spondyloarthropathy, 20 undifferentiated inflammatory arthritis) and 30 age-, gender- and race matched healthy controls (CN) were enrolled. Patients who had taken glucocorticoids during the previous 2 months or were taking agents known to affect insulin sensitivity or lipid metabolism were excluded. The body mass index (kg/m^2) was calculated in each subject. The erythrocyte sedimentation rate (ESR), plasma glucose, serum insulin and total cholesterol (chol), LDL-chol, HDL-chol and triglycerides were determined on fasting blood samples. IR was estimated by the homeostasis model assessment (HOMA).

Results The mean (SD) BMI (kg/m^2) was 26.5 (4.2) in the patients and 22.9 (2.8) in the CN ($p < 0.001$). The mean (SD) HOMA ($\text{uU.mmol}/\text{ml.l}$) was 1.9 (1.3) in patients as compared to 1.1 (0.5) in CN ($p = 0.001$). The HDL-chol was lower ($p = 0.002$) and the total chol/HDL-chol was higher ($p < 0.001$) in patients as compared to CN. Only after controlling for the BMI in addition to the ESR, was the HOMA no longer different between patients and CN. The ESR and HOMA, but not the BMI, significantly predicted ($p < 0.05$) high chol/HDL-chol ratios. However, after controlling for these variables, this lipid parameter remained different in patients as compared to CN. Also, IA was associated with a positive family history for premature coronary arter disease ($p < 0.03$).

Conclusion Metabolic disturbances were identified in the majority of our patients and should therefore be addressed in the management of IA. IR could be attributed to the acute phase response and excess weight. However, dyslipidemia was associated with IA independent of the ESR, BMI and IR. Dyslipidemia may constitute a risk factor for IA.

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OP0015 THE DISEASE COURSE OF PATIENTS WITH RHEUMATOID ARTHRITIS IN REMISSION; A PROSPECTIVE 2 YEAR FOLLOW-UP STUDY

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Background

Objectives To assess the prevalence of persistent remission and factors associated with persistent remission in a large cohort of RA patients in remission.

Methods RA patients in remission according to the ACR/Prins criteria were evaluated every 3 months for disease activity during 2 years. Factors measured at baseline and possibly associated with persistent remission were evaluated.

Results Of the 180 RA patients included 93 (52%) experienced persistent remission. Those patients had significantly ($p < 0.05$) less tender joints, lower Ritchie score, lower VAS score, lower serum IgM-RF and IgA-RF levels, lower HAQ score at baseline when compared to patients suffering an exacerbation. DMARD use was not associated with persistent remission. In a multivariate analysis, the following variables were independently associated with persistent remission: DAS < 1.6 (odds ratio with 95%

CI: 3.7, 1.5–9.5) and VAS < 12 (1.1, 1.0–1.2). The area under the curve of the DAS < 1.6 in the first year was associated with remission in the second year (9.9, 4.1–24).

Conclusion Persistent remission occurs frequently and is associated with a low disease activity score (DAS), in particular with a low cumulative DAS. The use of a cumulative DAS may be a tool in taking therapeutic decisions.

OP0016 OSTEOCLAST NUMBERS IN JOINTS OF RATS WITH ADJUVANT ARTHRITIS ARE DECREASED BY TREATMENT WITH IL-1RA AND/OR PEG STNF-RI

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Background Adjuvant arthritis (AdA) induced in Lewis rats by injection of mycobacteria in oil is characterised by marked inflammation and skeletal damage. Recent work by our group using the AdA model has shown that skeletal destruction is mediated by the osteoclast differentiation factor osteoprotegerin (OPG) ligand,¹ and that inhibition of bone loss is associated with anti-inflammatory efficacy.²

Objectives The current study was performed to assess whether anti-inflammatory molecules act to spare bone by regulating intra-lesional numbers of osteoclasts.

Methods AdA was induced in male Lewis rats ($n = 6/\text{group}$) on Day 0, and hind paw swelling developed on Day 9. Animals were treated for 7 days (Days 9–15) and then necropsied (Day 16). Therapy consisted of interleukin-1 receptor antagonist (IL-1ra) or pegylated soluble tumour necrosis factor receptor type I (PEG sTNF-RI), administered alone or in all possible combinations, at the following doses: IL-1ra, 0.2, 1.0 or 5.0 mg/kg/hr (by s.c. infusion); PEG sTNF-RI, 0.25, 1.0 or 4.0 mg/kg/day (s.c bolus). Osteoclasts were detected by an indirect immunohistochemical method for cathepsin K. Inflammation, bone erosion and osteoclast numbers were scored blindly using tiered, semi-quantitative grading scales.

Results Dose-dependent reductions in inflammation, bone erosion and osteoclast scores were observed for high doses of both anti-cytokine therapies, whether given alone or in combination.

Conclusion These data indicate that anti-inflammatory doses of IL-1ra and PEG sTNF-RI protect the structural integrity of arthritic joints by regulating osteoclast numbers.

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OP0017 DEPRESSION INCREASES THE RISK OF MORTALITY IN RHEUMATOID ARTHRITIS

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Background Whether depression makes RA worse is unclear because active RA can itself cause depression. The issue is even less clear when cross-sectional studies are performed, for there is no way to determine the direction of causality, and patient self-reports are also influenced by depression. Longitudinal