RA exhibited an AUC of glucose and GIP greater than controls and a slower C-Peptide response time (75 vs. 30 minutes, p=0.029).
Conclusions: Incretins-insulin axis is altered in patients with RA compared to controls.
Disclosure of Interest: None declared
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SAT0096 MYOCARDIAL FUNCTION IMPROVES IN RHEUMATOID ARTHRITIS PATIENTS TREATED ACTIVELY A MAGNETIC RESONANCE FOLLOW-UP STUDY

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Background: Rheumatoid arthritis (RA) patients are susceptible to development of heart failure (HF). Increased HF risk is not explained by increased prevalence of coronary heart disease (CHD) or traditional cardiovascular (CV) risk factors. Chronic inflammation is suggested to play an important role. In parallel with others (1, 2), we observed RA patients with active disease to have myocardial dysfunction and local myocardial late gadolinium enhancement (LGE) indicative of fibrosis or inflammation on cardiac magnetic resonance (cMR) (3).
Objectives: In our patients (3), we here studied the effects of disease modifying anti-rheumatic drugs (DMARDs) on the myocardium over one-year period.
Methods: Fifty-eight female patients with active RA (<70 years) and 22 fibromyalgia (FM) patients underwent cardiac magnetic resonance (cMR). Two RA groups existed: patients with untreated active early RA (ERA) starting conventional synthetic DMARDs (csDMARDs) or biological DMARDs (bDMARDs) and patients with chronic RA (CRA) who had inadequate response to csDMARDs and were candidates for bDMARDs. Patients with CHD, diabetes and smoking were excluded. CMR was performed to analyze LGE and ventricular function before and after one-year DMARD therapy
Results: Of 30 ERA patients, each started csDMARDs (77% as combination), two started also bDMARD. Of 28 CRA patients, each started bDMARD (one monotherapy).

Table 1. Patient charasteristics at baseline

	RA patients	FM patients	p-value
Age, years; mean ± SD	49±14	54±12	0.112
Rheumatoid factor positivity; n (%)	48 (84)		
Anti-citrullinated peptide antibody positivity; n (%)	51 (90)		
Extra-articular features; n (%)	19 (33)		
Erosions on radiographs; n (%)	26 (47)		
Body mass index, kg/m^2; mean±SD	25±4	27±5	0.012
Mean blood pressure, mmHg; mean±SD	113±17	114±15	0.814
Glycosylated hemoglobin A1C, mmol/mol; mean±SD	5.4±0.3	5.6±0.3	0.037
Low density lipoprotein, mmol/l; mean±SD	3.0±0.8	3.4±0.8	0.025

In RA patients, biventricular systo-diastolic function of the heart was impaired compared to FM (Table 2). Over the study-period, myocardial function improved (Table 2) and DAS28-CRP declined (3.5±1.1 vs 2.3±1.0; p<0.001). Only RA patients had LGE, with no improvement over time (67%).

Table 2. Cardiac magnetic resonance findings in RA and FM patients

RA patients	Baseline mean±SD	Follow-up mean±SD	p-value	FM vs RA patients at baseline	p-value
LV EF%	59±4	59±5	0.477	61±7	0.085
LV ESV, ml/m^2	34±6	33±8	0.449	29±8	0.011
LV EDV, ml/m^2	82±11	81±11	0.645	74±11	0.010
LV TPFR, ms	472±99	445±106	0.035	–	–
RV EF%	59±6	60±6	0.065	61±7	0.314
RV ESV, ml/m^2	34±9	32±8	0.009	29±7	0.043
RV EDV, ml/m^2	81±12	79±11	0.034	73±9	0.006

LV = left ventricle, RV = right ventricle, ESV = end-systolic volume, EDV = end-diastolic volume, EF = ejection fraction, TPFR = time to peak filling rate.

Conclusions: Myocardial function was impaired in RA patients with active RA compared to FM controls, although the latter group had worse classical CV risk factor profile. After one-year DMARD-treatment targeting to remission, myocardial function improved in parallel with decreasing RA activity. Inflammation seems to be deleterious to the myocardium. Tight control of RA activity may improve myocardial function.
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SAT0097 FACTORS ASSOCIATED WITH ATHEROSCLEROSIS PROGRESSION IN PATIENTS WITH LOW-ACTIVE RHEUMATOID ARTHRITIS

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Background: Longitudinal studies of the carotid intima-media thickness (IMT) change in RA suggested a role for inflammation in atherosclerosis progression. However, data on well controlled joint disease are scarce since most studies enrolled patients with very high disease activity.
Objectives: To estimate atherosclerosis progression and identify influencing factors in a cohort of longstanding and well controlled RA patients.
Methods: One hundred nine RA patients (females 80%, age 59±12 years, disease duration 15.6±10.6 years, mean Framingham 10-year CV disease risk score 16±12%) without previous cardiovascular (CV) events underwent carotid ultrasound (CUS) examination at baseline and after a mean time of 1.1±0.3 years. Atheromatous plaques and intima-media thickness (IMT) were assessed. Data on CV risk factors, inflammation markers, medications, and RA characteristics were collected.
Results: Overall, we observed a significant increase of IMT (0.03±0.10 mm, p=0.005) and plaques (+ 8%, p=0.035). The IMT progression rate was 0.027 mm/year (95% CI 0.007 - 0.046). Disease activity (DAS28-CRP) remained stable (2.68±1.01 vs 2.79±1.33, p=0.45). Anti-rheumatic, cardiovascular medications and the number of CV risk factors were substantially unchanged. In models of regression analysis sex, age, dyslipidemia, hypertension and use of corticosteroids were independently associated with the increase of IMT, whereas there were no confounding from use of biological therapies, seropositivity or disease duration. Patients with active disease (DAS28-CRP ≥2.6) had a significant increase in IMT (0.04±0.11 mm, p=0.009). Conversely, there was not a significant progression of patients in remission, who had also a lower prevalence of hypertension (40% vs 64%, p=0.027), dyslipidemia (43% vs 58%, p=0.044), and use of corticosteroids (37% vs 63%, p=0.007) and were receiving more frequently methotrexate (60% vs 40%, p=0.027).
Conclusions: In patients with established and controlled RA, the progression of atherosclerosis is mainly driven by traditional CV risk factors than disease activity. In addition, a remission state is associated with a lower prevalence of CV risk factors, which in turn could account for a slower progression of atherosclerosis in these patients. This study provides evidence that even in RA patients who achieve good disease control the treatment of CV risk factors should be optimized.
Disclosure of Interest: None declared
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SAT0098 THE ASSOCIATION BETWEEN WORK DISABILITY AND MENTAL HEALTH IN RHEUMATOID ARTHRITIS

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Background: Rheumatoid arthritis (RA) is characterised by joint inflammation, resulting in functional impairment. Consequently, it has long been recognised that work disability is common in RA. However, despite significant pharmacological advances in disease control, little is known about contemporary impact upon work. Depression has emerged in recent years as a key comorbidity in RA. In the general population, depression is strongly correlated with work disability
Objectives: To establish the extent to which depression associates with work ability in RA in a cross sectional study.
Methods: Our hospital routinely collects data via an electronic screening interface, which incorporates a series of validated questionnaires, which patients complete via an iPad while waiting for their appointment. The dataset is linked to the clinical record. For this study, cross sectional data were extracted on adults with RA. Question 2 of the Work and Social Adjustment Scale ("Because of my [RA] my ability to work is impaired") was used as a measure or work disability (scale 0–8). Mental health status was collected using the Patient Health Questionnaire-2 (PHQ), with a score≥3 considered a positive screen for depression.

Table 1

Characteristic	Whole Cohort N=385	PHQ2 score <3 n=263	PHQ2 score ≥3 n=122	p value
Age, mean (SD)	54 (15)	54 (15)	55 (14)	0.768
Female, n (%)	305 (79)	207 (79)	98 (80)	0.681
Seropositive, n (%)	261 (73)*	175 (72)*	86 (76)*	0.705
Disease duration, mean (SD)	6.6 (8.0)	6.0 (7.0)	8.0 (10.0)	0.118
DAS28, mean (SD)	4.0 (1.7)	3.6 (1.6)	4.7 (1.6)	<0.001
HAQ, mean (SD)	1.3 (0.9)	1.0 (0.8)	1.9 (0.7)	<0.001
Work ability, mean (SD)**	3.1 (2.9)	2.1 (2.4)	5.1 (2.6)	<0.001
Univariate regression				
Beta coefficient (95% CI)		Ref	2.94 (2.40 to 3.50)	<0.001
Multivariate regression***			1.40 (0.84 to 1.96)	<0.001

*Missing data on serostatus in 28 subjects. **Work ability on scale 0–8 from WSAS scale. ***Adjusted for age, gender, disease duration, HAQ and DAS score.