immune status between regressive and persistent LPDs developed under MTX administration.

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Acknowledgements: None.

Disclosure of Interest: S. Saito: None declared, K. Suzuki: None declared, K. Yamaoka Speakers bureau: Pfizer, Chugai Pharma, Mitsubishi-Tanabe Pharma, Takeda Industrial Pharma, GlaxoSmithKline, Nippon Shinyaku, Eli Lilly, Janssen Pharma, Eisai Pharma, Astellas Pharma and Actelion Pharmaceuticals, K. Amano: None declared, M. Tokuhira Speakers bureau: Eisai Co., T. Takeuchi Grant/research support from: Astellas Pharma, Bristol–Myers K.K., Chugai Pharmaceutical Co, Ltd., Daiichi Sankyo Co., Ltd., Eisai Co.,Ltd., Mitsubishi Tanabe Pharma Co., Pfizer Japan Inc., Santen Pharmaceutical Co., Ltd., Takeda Pharmaceutical Co.,Ltd., Teijin Pharma Ltd., AbbVie GK, 2,Astra Zeneca K.K., Eli Lilly Japan K.K., Novartis Pharma K.K.,

DOI: 10.1136/annrheumdis-2017-eular.4662

FRI0169 GLOMERULAR FILTRATION RATE IS LOW IN RHEUMATOID ARTHRITIS COMPARED TO HEALTHY POPULATION: ESSENTIAL ROLE OF INFLAMMATION

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Background: Rheumatoid Arthritis is associated with subclinical renal impairment which contributes to increase mortality and morbidity. The role of inflammation on kidney function in inflammatory arthritis is not well studied.

Objectives: To investigate the associations between estimated glomerular filtration rates (eGFR), traditional cardiovascular risk factors, and markers of inflammation in rheumatoid arthritis compared to healthy controls.

Methods: RA patient were recruited through a specialized rheumatology clinic at the Ministry of Health and Prevention of UAE, from January 2013 to January 2016. Healthy subjects recruited from the community through brochure advertisement. The Modification of Diet in Renal Disease Study (MDRD) formula was used to calculate the eGFR. ttest was used to compare the laboratory values and renal function parameters between two groups. Linear regression analysis used to look for the correlation between the eGFR and each of the traditional cardiovascular risk factors and inflammatory markers

Results: 98 RA patients and 82 controls were recruited. None of the patients has history of diabetes, atherosclerosis or renal impairment. The mean age for the total participants was 49±13 years (Min16 –Max 82). The mean eGFR of the inflammatory arthritis patients was 118±30 ml/min (range 60–227) and 128±37 ml/min (range 62–286) for the controls. Patients and control had no significant difference in Systolic and diastolic blood pressure.

Inflammatory arthritis patients had lower GFR, albumin ($P<0.001$), and total protein (p=0.03) levels, and had higher Erythrocyte Sedimentation Rate (ESR) ($P<0.001$), C-reactive protein (CRP) ($P<0.001$), and uric acid level (p=0.01),

Negative linear relationships were found as follows:

Among RA patients and controls: There was a negative linear relationship between GFR and each of the age of the participants; ($p<0.001$, CI: -1.24, -0.40 for the patients and p=0.01, CI: -1.82, -0.26 for the controls), and the systolic blood pressure; (p=0.04, CI: -0.61 for the patients and, -.00 and p=0.022, CI: -0.61, -0.05 for the controls).

Among RA patiens: The GFR had a negative linear relationship with the age of the participants, age at RA onset (p=0.002, CI: -1.18, -0.29), diastolic blood pressure (p=-2.14, CI: -1.24, -0.05), ESR level (p=0.04, CI: -0.24,-0.01), C-reactive protein; CRP level (p=0.02, CI: -0.47, -0.04), uric acid level ($p<0.001$, CI -0.18, -0.05), and total protein (p=0.01, CI: -0.91, -0.16). There was a positive linear relationship between eGFR and albumin level (p=0.03, p=0.14, 2.35),

Conclusions: In RA non-traditional cardiovascular risk factors such as inflammatory markers are associated with sub-clinical presence of renal injury. Our data indicate that in RA, inflammation is involved in the early stages of impaired kidney function. Whether anti-inflammatory therapies are effective in slowing down the deterioration of kidney function in the arthritis diseases remain to be established.

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Disclosure of Interest: None declared

DOI: 10.1136/annrheumdis-2017-eular.1658

FRI0170 CARDIOVASCULAR RISK FACTORS AND DISEASE CHARACTERISTICS ARE CONSISTENTLY ASSOCIATED WITH ARTERIAL STIFFNESS IN RHEUMATOID ARTHRITIS

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Background: In the non-rheumatoid arthritis (RA) population, arterial stiffness contributes to cardiovascular disease risk beyond brachial blood pressure and other established cardiovascular risk factors. The increased cardiovascular disease risk in RA is now well documented. In this regard, how RA impacts on arterial stiffness remains uncertain.

Objectives: The aim of the present study was to identify potential determinants of comprehensively assessed arterial stiffness in a relatively large group of ethnically diverse patients with RA.

Methods: Relationships of traditional cardiovascular risk factors and RA characteristics with 9 arterial stiffness markers including central systolic and pulse pressure, pulse wave velocity, augmentation index, forward and reflected wave pressure, reflection magnitude, brachial-to-aortic pulse pressure amplification (a marker of reduced wave reflection) and peripheral pulse pressure were identified in multivariable backward regression models among 177 (118 white, 32 Asian, 22 black, 5 mixed ancestry) patients without established cardiovascular disease.

Results: Recorded characteristics explained 37% (pulse wave velocity) to 71% (reflected wave pressure) of the variability in arterial stiffness. RA duration (partial r=0.17, p=0.04), rheumatoid factor status (partial r=-0.19 to 0.20, p=0.01 to 0.03), leukocyte counts (partial r=0.16 to 0.19, p=0.02 to 0.05) and total cholesterol (-0.18 to 0.26, p=0.00 to 0.03) were associated with enhanced central systolic blood pressure or/and wave reflection markers. C-reactive protein (partial r=-0.24, -0.17 and -0.20, respectively, $p\leq0.05$) was paradoxically related to reduced central pulse pressure, pulse wave velocity and forward wave pressure, and body mass index (partial r=-0.39 to 0.42, p=0.00 to 0.02) and insulin resistance (partial r=-0.21 to -0.20, p=0.00 to 0.01) to reduced wave reflection and peripheral pulse pressure. Exercise (partial r=0.19, p=0.02) and alcohol (partial r=-0.27, p=0.00) consumption were associated with increased pulse pressure amplification and decreased peripheral pulse pressure, respectively. Tumour necrosis factor-α inhibition (partial r=-0.25, p=0.00) was related to reduced pulse wave velocity and tetracycline use (partial r=-0.20, p=0.02) to reduced peripheral pulse pressure.

Conclusions: Traditional cardiovascular risk factors and disease characteristics are consistently associated with vascular hemodynamic alterations in RA. The role of arterial stiffness in cardiovascular disease risk in RA needs further study.

Disclosure of Interest: None declared

DOI: 10.1136/annrheumdis-2017-eular.4277

FRI0171 OBESITY CONTRIBUTES TO SUBOPTIMAL PHYSICAL FUNCTION IN RHEUMATOID ARTHRITIS

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Background: Aggressive, early treatment of RA with new therapeutic agents has dramatically improved the management of RA. However, many studies have failed to show greater improvements in function or disability reduction between targeted control vs. less-aggressive care. The prevalence of obesity is increasing dramatically globally, and may be even higher in RA patients.[1]

Objectives: Because obesity is also a risk factor for disability, we evaluated the extent to which excess weight may independently contribute to poorer physical function (PF) in RA.

Methods: RA patients enrolled in an observational study at an academic Inflammatory Arthritis Clinic in Baltimore USA completed the patient global, pain VAS, and PROMIS measures assessing PF, pain, fatigue, sleep, and depression. RA clinical indicators were also collected at the visit. Outcomes were compared in obese and non-obese patients using t-tests and chi-square. Multiple regression was used to evaluate the effects of pain, fatigue, and BMI on PF, after controlling for age and disease activity.

Results: Participants were mostly female (82%) and white (83%) with mean (SD) age of 55 (13) years; 24% had $\leq$ high school; RA duration 12 (9). Mean CDAI was 8.1 (8.1). Most were in CDAI remission (n=57; 32%) or LDA (n=64; 36%); 40 (23%) were in MDA and 16 (9%) in HDA. 49 (28%) were classified as normal weight (BMI 18.5–24.9), 46 (26%) were overweight (BMI 25–29.9), and 82 (46%) were obese (BMI$\geq$30). Men had a significantly higher mean BMI than women (33.6 [8.4] vs. 29.5 [7.2], p=0.006).

As compared to non-obese participants, obese participants had a significantly ($p<.05$) higher CDAI (6.1 [6.9] vs. 10.4 [8.9]; p=.000, respectively) and worse PF, pain, fatigue, sleep, and depression (mean differences -5.0, -4.6, 4.6, 3.8, 2.9, 3.6, and -4.7, respectively). In regression analyses, pain, fatigue, and BMI (but not sleep or depression) were inversely related to PF, after controlling for age and disease activity. In the final model, pain, fatigue and BMI were significantly and inversely related to PF (β=-.39, -.24, and -.153, respectively) after controlling for age and disease activity ($F(5, 170) = 55.5$, p=.000, adjusted r^2=.61).

Table. Independent Contributors of Function in RA.

	Coefficient		Std		Collinearity	
	B	SE	Beta	Sig.	Tol	VIF
Pain	-.386	.064	-.405	.000	.500	2.001
Fatigue	-.240	.060	-.266	.000	.509	1.965
BMI	-.153	.058	-.128	.010	.932	1.073
Adjusted for age and CDAI.						

Conclusions: Our results suggest that excess weight also contributes to poorer PF in addition to pain and fatigue. As the prevalence of obesity continues to