or remission. Rescue rates were 7.3% for bari 4mg, 17.1% for bari 2mg. Most rescued patients could regain LDA or remission. Dose reduction was associated with a lower rate of non-serious infections; rates of SAEs and AEs leading to discontinuation were similar across groups.

Conclusions: These data indicate that 4mg once daily was the most efficacious dose of bari for pts with RA; increases in disease activity were consistently seen with dose reduction from 4mg to 2mg in well-controlled pts. However, after step-down most pts could maintain LDA or remission, or recapture control with return to 4mg if needed. Attempted dose taper may be a reasonable consideration for some pts following induction of sustained disease control with bari 4mg.

Disclosure of Interest: T. Takeuchi Consultant for: Pfizer Japan, Astra Zeneca KK, Eli Lilly Japan KK, Novartis Pharma KK, Daiichi Sankyo Ltd, Nipponkayaku Ltd, Janssen Pharma KK, Merck Serono Ltd, Takeda Pharma Ltd, Mitsubishi Tanabe Pharma, Astellas Pharma, Abbvie GK, Bristol-Myers KK, Asahi Kasei Medical KK, Speakers bureau: Celtrion, Nipponkayaku Ltd, Pfizer Japan, UCB Japan, Daiichi Sankyo Ltd, Takeda Pharma Ltd, Chugai Pharma Ltd, Abbvie GK, Bristol-Myers KK, Eisai Co Ltd, Mitsubishi Tanabe Pharma, Janssen Pharma KK, Astellas Pharma, M. Genovese Grant/research support from: Eli Lilly and Company, Abbvie, Astellas, Galapagos, Pfizer, Vertex, Consultant for: Eli Lilly and Company, Abbvie, Astellas, Galapagos, Pfizer, Vertex, B. Haraoui Grant/research support from: Amgen, BMS, UCB, Consultant for: Abbvie, Amgen, BMS, Celgene, Janssen, Eli Lilly and Company, Pfizer, Roche, UCB, Speakers bureau: Pfizer, UCB, L. Xie Employee of: Eli Lilly and Company, R. Klar Employee of: Quintiles IMS Holdings, Inc., A. Luisa Pinto Correia Employee of: Eli Lilly and Company, T. Rooney Shareholder of: Eli Lilly and Company, Employee of: Eli Lilly and Company, S. Otawa Employee of: Eli Lilly and Company, J. Smolen Grant/research support from: from Abbvie, Janssen, Eli Lilly and Company, MSD, Pfizer, Roche, Consultant for: Abbvie, Amgen, Astra-Zeneca, Astro, BMS, Celgene, Celltrion, Chugai, Gilead, Glaxo, ILTOO, Janssen, Eli Lilly and Company, Medimmune, MSD, Novartis-Sandoz, Pfizer, Roche, Samsung, Sanofi, UCB, Speakers bureau: Abbvie, Amgen, Astra-Zeneca, Astro, BMS, Celgene, Celltrion, Chugai, Gilead, Glaxo, ILTOO, Janssen, Eli Lilly and Company, Medimmune, MSD, Novartis-Sandoz, Pfizer, Roche, Samsung, Sanofi, UCB

DOI: 10.1136/annrheumdis-2017-eular.1073

SAT0073 MIR-186-5P TARGETING IL-33 GENE AS BIOMARKER TO PREDICT SUBCLINICAL ATHEROSCLEROSIS IN PATIENTS WITH EARLY RHEUMATOID ARTHRITIS

T.H. Cheng[1], Q. Shang[1], W.Y. Mak[1], M. Li[1], K.Y. Kwok[2], I.C.W. Yim[3], E.K.M. Li[1], P.C.H. Wong[4], V.W.N. Lao[5], H.T. Pang[5], L.S. Tam[1]. *[1]Department of Medicine and Therapeutics, the Chinese University of Hong Kong; [2]Department of Medicine, Queen Elizabeth Hospital; [3]Department of Medicine, Tseung Kwan O Hospital; [4]Department of Medicine and Therapeutics, Prince of Wales Hospital; [5]Department of Medicine and Geriatrics, Kwong Wah Hosptial, Hong Kong, Hong Kong*

Background: Patients with rheumatoid arthritis (RA) die prematurely compared with the general population, primarily because of cardiovascular diseases (CVD). Interleukin-33 (IL-33) is a member of the IL-1 cytokine family which was important in the pathogenesis of RA and development of CVD. Blood IL-33 protein was not detectable in most subjects, even in RA patients. Thus, the usability of IL-33 as a biomarker for CVD is limited. MicroRNAs (miRNAs) are small non-coding RNAs that function as post-transcriptional regulators of gene expression.

Objectives: This study was to ascertain if dysregulated miRNAs targeting *IL-33* gene in early RA (ERA) patients were associated with subclinical atherosclerosis in ERA patients.

Methods: 76 ERA patients were recruited in this cross-sessional study. Potential miRNAs binding to 3'UTR of the *IL-33* gene were predicted by miRanda (www.microRNA.org). 10 miRNAs with highest possibility targeting functional sites of *IL-33* gene were quantified in cell free plasma samples using a $2^{\Delta Ct}$ method. Caenorhabditis elegans miR-39 (cel-miR-39) was used as spike-in control. The results were then log transferd. Carotid plaque (CP) was measured and identified at bilateral common carotid artery, bulb, and proximal internal carotid artery using a high-resolution B mode ultrasound. Receiver-operating characteristic curve (ROC) analysis was performed to determine the discriminating power of the miRNA for the presence of CP.

Results: CPs were identified in 26/76 (34%) subjects (CP+ group). Subjects in the CP+ group were older [58±10 vs 48±11 years old, p=0.001], predominantly male [48 (42.3%) vs 43 (14.0%), p=0.006], with a higher C-reactive protein (CRP) level [24.9±25.0 vs 11.8±13.7 mg/dL, p=0.018] and higher cardiovascular risk [Framingham risk score (FRS): 12.8±11.6 vs 5.7±6.8, p=0.008] (Table 1). All miRNAs were detected in >80% of subjects in both group. Plasma level of miR-186–5p in the CP+ group was significantly higher than that in the CP- group [log miRNA: 3.28±3.21 vs 2.58±1.13 p=0.008]. It was still significant after adjusting age, sex, plasma CRP and FRS (p=0.030) (Table 1). Using multivariate logistic regression, miRNA-186–5p was an independent predictor of the presence of carotid plaque (OR: 1.919, 95% CI=1.096–3.361, p=0.023) after adjustment of FRS and CRP level. [Area under the ROC (AUC) 0.66, 95% CI: 0.60–0.80 p=0.024].

Conclusions: miR-186–5p was an independent predictor for presence of subclinical atherosclerosis and may serve as a novel biomarker for risk stratification in ERA patients with mild to moderate cardiovascular risk.

Table 1 - Characteristics of patients with and without carotid plaque

	Carotid Plaque						p	p*
	Absence (n=50)			Presence (n=26)				
Age	48	±	11	58	±	10	0.001	
Gender, male	43		14.0%	15		42.3%	0.006	
CRP, mg/dL	11.8	±	13.7	24.9	±	25.0	0.018	
Franmingham Risk Score	5.7	±	6.8	12.8	±	11.6	0.008	
log miRNA								
miR_9_5p	0.39	±	0.99	0.26	±	0.36	0.809	0.225
miR_382_5p	0.87	±	0.88	0.94	±	0.82	0.809	0.730
miR_377_3p	0.69	±	0.61	0.94	±	0.83	0.227	0.550
miR_590_3p	0.10	±	0.20	0.33	±	0.61	0.684	0.222
miR_499a_5p	0.11	±	0.17	0.19	±	0.29	0.809	0.406
miR_145_5p	2.43	±	1.05	3.30	±	1.82	0.227	0.091
miR_542_3p	0.08	±	0.27	0.08	±	0.14	0.373	0.886
miR_186_5p	2.58	±	1.13	3.28	±	1.21	0.008	0.030
miR_214_3p	0.41	±	0.66	0.61	±	0.65	0.091	0.085
miR_496	0.14	±	0.32	0.17	±	0.25	0.809	0.546

**Multivariate analysis adjusted with age, sex, plasma CRP and FRS*

Disclosure of Interest: None declared

DOI: 10.1136/annrheumdis-2017-eular.2840

SAT0074 PERSISTENCE WITH METFORMIN TREATMENT AND ONSET OF RHEUMATOID ARTHRITIS

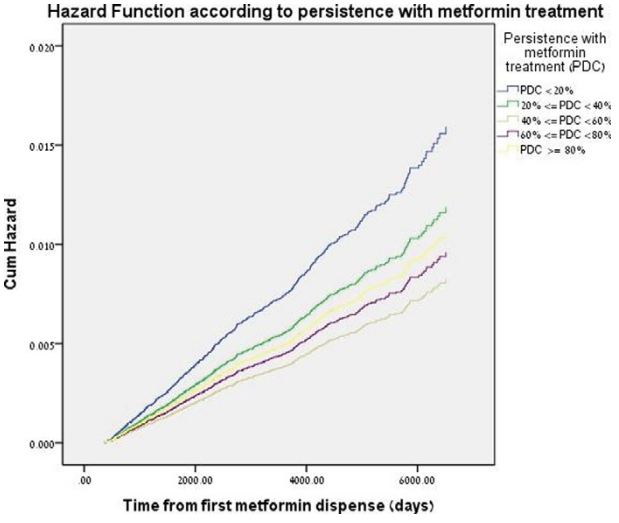
M.E. Naffaa[1], V. Rosenberg[2], G. Chodick[2,3], V. Shalev[2,3], S. Tiosano[3,4], H. Amital[3,4]. *[1]Rheumatology unit, Galilee Medical Center, Nahariya; [2]Maccabi Healthcare Services; [3]Sackler Faculty of Medicine, Tel-Aviv University, Tel-Aviv; [4]Internal Medicine 'B', Sheba Medical Center, Ramat-Gan, Israel*

Background: Several studies have suggested that metformin, an oral hypoglycemic agent, possess an anti-inflammatory property and may have a role in the treatment of rheumatoid arthritis (RA)[1,2], but little is known on its preventive effects.

Objectives: To examine the association between persistence with metformin and the onset of RA.

Methods: Using the computerized medical database of a large health organization in Israel (Maccabi Healthcare Services, MHS) we have identified incident RA cases among new users of metformin between 1998 and 2014. Included were patients aged 18 or above with one year of follow up before and after the therapy initiation. RA was defined according to physician diagnoses. Participants were followed until the earliest of the following dates: onset of RA, leaving MHS, death, end of follow up (1.1.2016). Persistence with metformin was assessed by calculating the mean proportion of follow-up days covered (PDC) with metformin during the study period.

Results: A total of 113,749 eligible patients were included. During the study follow up period (794,386 person-years) we identified 600 incident cases (incidence rate of 75 cases per 100,000 PY). The incidence of RA in women (111 per 100,000 PY) was higher compared to men (42 per 100,000 PY). In a multivariable model, persistence with metformin (PDC≥80%) was associated with lower risk of RA (hazard ratio (HR) 0.66; 95% confidence interval (CI) 0.53–0.82) compared to non-persistent participants (PDC<20%). Figure 1 shows the hazard function according to persistence with metformin treatment. Similar risk reduction was observed among men but did not reach statistical significance (HR=0.85; 95% CI 0.54–1.32).



Conclusions: In the present study, we observed an association between high persistence to metformin therapy and reduced risk of developing RA in women.