Acknowledgements: We would like to thank all patients and rheumatologists contributing to the SCQM registry, as well as the entire SCQM staff. A list of rheumatology offices and hospitals which contribute to the SCQM registry can be found at <http://www.scqm.ch/institutions>. The SCQM is financially supported by pharmaceutical industries and donors. A list of financial supporters can be found at <http://www.scqm.ch/sponsors>.

Disclosure of Interests: Enriqueta Vallejo-Yagüe: None declared, Theresa Burkard: None declared, Burkhard Moeller Speakers bureau: AbbVie, Bristol Myers, Eli Lilly, Janssen, Pfizer, Roche, Novartis, Merck, Axel Finckh Speakers bureau: Pfizer, Eli-Lilly, Paid instructor for: Pfizer, Eli-Lilly, Consultant of: AbbVie, AB2Bio, BMS, Gilead, Pfizer, Viatris, Grant/research support from: Pfizer, BMS, Novartis, Andrea Michelle Burden: None declared

DOI: 10.1136/annrheumdis-2021-eular.1348

POS0213 20 YEAR FOLLOW-UP OF CARDIOVASCULAR EVENT RISK IN RHEUMATOID ARTHRITIS COMPARED TO DIABETES

R. Raadsen¹, R. Agca^{1,2}, A. Voskuyl², M. Boers³, W. Lems^{1,2}, M. Nurmohamed^{1,2}, ¹Amsterdam Rheumatology and Immunology Center, location Reade, Rheumatology, Amsterdam, Netherlands; ²Amsterdam Rheumatology and Immunology Center, location VU University Medical Center, Rheumatology, Amsterdam, Netherlands; ³Amsterdam University Medical Center, Location VU University Medical Center, Biostatistics and Epidemiology, Amsterdam, Netherlands

Background: Patients with rheumatoid arthritis have an increased risk for developing cardiovascular diseases (CVD) compared to the general population, similar to the CVD risk in patients with diabetes mellitus. However, there are no controlled studies investigating the incidence of cardiovascular (CV) events in RA patients with follow up of more than 20 years.

Objectives: The objectives of the current study were to investigate the incidence rates of CV events in a long-term follow up cohort of RA patients, and to compare these to a similar cohort representing the general population, ie. The Hoorn study.

Methods: The CARRÉ study is an ongoing prospective cohort study, which started in 2001, investigating CV mortality and morbidity in 353 randomly selected patients with RA. Primary endpoints, i.e. verified medical history of coronary, cerebral or peripheral arterial disease, were determined at baseline, and after three, ten, fifteen and twenty years of follow up. Patients were censored at the date of an experienced CV event or their death. Incidence density rates per 100 patient years were calculated. Data were compared to results from the Hoorn study, a Dutch cohort study of glucose metabolism and other CV risk factors that began in 1989. All 2,484 participants were subject to an extensive and repeated CV screening program similar to that used in the CARRÉ study.

Results: After 20 years of follow up 118 patients (33%) developed at least one CV event in the Carré group. Mean (SD) follow up time was 11 (6) years with a total of 3,500 years at risk and an incidence rate of 3.4 per 100 patient-years; this is slightly up from the figure reported at 15 years, i.e. 3.2 per 100 patient-years. A CV event-free survival curve is shown in figure 1. After 30 years of follow up, 295 participants of the Hoorn study had developed a CV event, during a mean follow up time 20 (8) years. Total time at risk was 50,000 years, with an incidence rate of 0.6 CV events per 100 patient years.

Conclusion: In our cohort the incidence rate of CV events in RA patients has remained consistently high when compared with the general population, despite better control of RA inflammation in recent years. This again confirms the need for timely CVD-risk screening and management.

REFERENCES:

- Agca R, Hopman L, Laan KJC, van Halm VP, Peters MJL, Smulders YM, et al. Cardiovascular Event Risk in Rheumatoid Arthritis Compared with Type 2 Diabetes: A 15-year Longitudinal Study. *J Rheumatol*. 2020;47(3):316-24.

Figure 1. Survival curve of participants with rheumatoid arthritis. RA = rheumatoid arthritis

Disclosure of Interests: None declared

DOI: 10.1136/annrheumdis-2021-eular.1744

POS0214 ASSOCIATION BETWEEN C-REACTIVE PROTEIN AND 10-YEAR RISK OF CARDIOVASCULAR DISEASE IN RHEUMATOID ARTHRITIS USING THE ERS-RA SCORE: A CROSS-SECTIONAL ANALYSIS OF THE CORDIS COHORT

G.L. Erre¹, F. Cacciapaglia², G. Sakellariou³, A. Manfredi⁴, E. Bartoloni Bocci⁵, O. Viapiana⁶, M. Fornaro², M. Dessi⁷, A. A. Mangoni⁸, B. L. Palermo⁹, E. Gremese¹⁰, G. Cafaro⁵, V. Nucera¹¹, C. Vacchi¹², F. R. Spinelli¹³, F. Atzeni¹¹, M. Piga¹⁴ on behalf of "Cardiovascular Obesity and Rheumatic Disease Study (CORDIS)" Group of the Italian Society of Rheumatology (SIR). ¹University and AOU of Sassari, Department of Medical, Surgical and Experimental Sciences, Sassari, Italy; ²University and AOU, Policlinico of Bari, Department of Emergency and Organs Transplantation, Bari, Italy; ³University of Pavia, Istituti Clinici Scientifici Maugeri IRCCS Pavia, Pavia, Italy; ⁴Azienda Ospedaliera Universitaria Policlinico di Modena, Unit of Rheumatology, Modena, Italy; ⁵University of Perugia, Department of Medicine and Surgery, Perugia, Italy; ⁶University of Verona, Dipartimento di Medicina, Verona, Italy; ⁷University of Cagliari, Department of Medical Sciences and Public Health, Cagliari, Italy; ⁸Flinders University, College of Medicine and Public Health, Adelaide, Australia; ⁹University of Pavia, IRCCS San Matteo, Pavia, Italy; ¹⁰Policlinico Gemelli, Università Cattolica del Sacro Cuore, Dipartimento di Scienze Mediche e Chirurgiche, Roma, Italy; ¹¹University of Messina, Dipartimento di Medicina Clinica e Sperimentale, Messina, Italy; ¹²University of Modena and Reggio Emilia, Clinical and Experimental Medicine PhD Program, Modena, Italy; ¹³Università La Sapienza, Dipartimento di Scienze Cliniche Internistiche, Anestesiologiche e Cardiovascolari, Roma, Italy; ¹⁴University of Cagliari, Department of Medical Sciences and Public Health, Monserrato, Italy

Background: Rheumatoid arthritis (RA) is associated with an increased risk of atherosclerotic cardiovascular disease (CVD). The Expanded Cardiovascular Risk Prediction Score for Rheumatoid Arthritis (ERS-RA) estimates the 10-year risk of myocardial infarction, stroke or CVD-related death based on conventional and RA-specific (clinical disease activity index, CDAI, disease duration, glucocorticoid use) risk factors (1).

Objectives: We evaluated the associations between ERS-RA 10-year risk of CVD, high-sensitivity C-reactive protein (hs-CRP) concentrations, and pharmacological treatment in 1,251 RA patients collected by the "Cardiovascular Obesity and Rheumatic Disease Study (CORDIS)" group of the Italian Society of Rheumatology (SIR).

Methods: We assessed independent associations between ERS-RA risk score and each relevant variable using multivariate regression (ENTER approach; listwise deletion analysis). Given the relatively high number of missing hs-CRP data (n=385), regression analysis was also performed using multiple imputation (10 sets, Stata 16.1). Regression models were not adjusted for independent variables included in the ERS-RA score.

Results: Among 1,251 RA patients [mean (SD) age 60.4(9.3), range (40-80) years; 78% female; mean (SD) disease duration, 11.6(8) years; mean (SD) CDAI, 9(9); mean (SD) HAQ, 0.77(0.7); mean (SD) hs-CRP, 6.8(12) mg/L] the estimated 10-year CVD risk was 11.6(0.9) % [mean (SD)]. Regarding treatment, 539(43%) received glucocorticoids, 676(54%) a biological or targeted synthetic disease-modifying anti-rheumatic drug (b/tsDMARD) (n missing=1), and 885(81%) at least one conventional synthetic DMARD (csDMARD). Ninety-three (7.4%) patients did not receive any treatment. After adjusting for the use of b/tsDMARD and csDMARD, hs-CRP concentrations were significantly associated with 10-year risk of CVD both in standard multiple regression (n=865; coefficient=0.005 for each 10 mg/L hs-CRP increment, 95% confidence interval (0.000-0.100), p=0.043) and after multiple imputation (n=1,251; coefficient=0.005 for each 10 mg/L hs-CRP increment, 95% confidence interval (0.000-0.114), p=0.035) (Table 1). This corresponds to an increase of 10-year CV risk of 1% for every 20 mg/L increase in hs-CRP concentrations.

Conclusion: In a large cohort of RA patients, we observed a significant, positive, and independent association between hs-CRP concentrations and 10-year CV risk estimated by ERS-RA. The cross-sectional design of the study did not allow to establish a cause-effect relationship between hs-CRP and CV risk. Given that conventional CV risk factors and inflammation-related variables are accounted for in the ERS-RA risk score, other, unexplored, mechanisms may underlie the observed association between hs-CRP and CV risk.

REFERENCES:

- Solomon, D. H., et al. "Derivation and internal validation of an expanded cardiovascular risk prediction score for rheumatoid arthritis: a Consortium of Rheumatology Researchers of North America Registry Study." *Arthritis & rheumatology* 67.8 (2015): 1995-2003.

Table 1. Multiple regression models

	Model 1 n= 865		Model 2 n= 1, 251	
ERS-RA score	Coefficient	95% CI, <i>p</i>	Coefficient	95% CI, <i>p</i>
hs-CRP, every 10 mg/L increment	0.005	0.000 to 0.100, 0.043	0.005	0.000 to 0.011, 0.035
b/tsDMARD use	-0.002	-0.005 to 0.001, 0.199	-0.000	-0.002 to 0.002, 0.963
csDMARD use	0.002	-0.003 to 0.007, 0.394	0.002	-0.002 to 0.006, 0.371
Prob >F, model with only CRP	0.03		0.03	
Prob >F, full model	0.07		0.08	

A multiple linear regression (ENTER method) was performed for the dependent variable ERS-RA score using a listwise deletion analysis (Model 1) and a multiple imputation analysis (Model 2).

Disclosure of Interests: None declared

DOI: 10.1136/annrheumdis-2021-eular.2643

POS0215

DIRECT AND CONDITIONAL EFFECTS OF EPICARDIAL ADIPOSE TISSUE VOLUME ON CORONARY PLAQUE PROGRESSION IN RHEUMATOID ARTHRITIS

G. Karpouzas¹, S. Ormseth¹, E. Hernandez², M. Budoff². ¹The Lundquist Institute, Rheumatology, Torrance, United States of America; ²The Lundquist Institute, Cardiology, Torrance, United States of America

Background: Epicardial adipose tissue volume (EATv) predicts coronary atherosclerosis presence, progression and cardiovascular event risk in general patients^{1,2}. Our group recently reported that EATv associated with greater sub-clinical coronary plaque burden, non-calcified plaque presence and vulnerable plaque characteristics in patients with rheumatoid arthritis (RA). The relationship was stronger in RA patients with lower disease duration, no traditional cardiac risk factors, and who were not obese.

Objectives: To evaluate the predictive value of EATv on long-term coronary atherosclerosis development, and moderators of the association between EATv and plaque formation.

Methods: This single-center observational cohort study included 100 patients without symptoms or diagnosis of cardiovascular disease who underwent computed tomography angiography for evaluation of EATv and coronary atherosclerosis at baseline and repeat assessments 6.9±0.3 years later to evaluate plaque progression. New plaque formation in segments without plaque at baseline was the main outcome. Robust multivariable logistic regression evaluated the effect of high versus low EATv (based on median) on likelihood of new plaque formation, accounting for clustering of segments within patients. Potential moderator effects of prespecified predictors were also assessed.

Results: High EATv (>107 cm³) predicted new plaque formation in segments without baseline plaque (OR 2.77 [95% CI 1.43-5.37], *p*= 0.003); however, significance was lost in the multivariable model. Importantly, high EATv associated with formation of higher-risk non- and partially calcified plaque after adjusting for Framingham D'Agostino risk score, obesity, segment location, time-averaged CRP, duration of bDMARD and statin treatment and cumulative prednisone dose (adjusted OR 2.57 [95% CI 1.02-6.48], *p*= 0.045). RA duration (<10 versus >10 years), cardiac risk factor burden (≤1 versus >1), and presence of mixed/calcified plaque in other coronary segments at baseline, and statin exposure (≤1 versus >1 year, based on median) moderated the effect of EATv on all new plaque formation (all *p* for interaction ≤ 0.021). Specifically, high EATv predicted new plaque formation in patients with RA duration <10 years (adjusted OR 5.75 [95% CI 1.77-18.67]), those with ≤1 cardiac risk factors (adjusted OR 3.40 [95% CI 1.46-7.90]), those without calcification at baseline (adjusted OR 2.65 [95% CI 1.11-6.31]) and those with statin treatment <1 year (adjusted OR 3.33 [95% CI 1.13-9.77]). This was not the case for patients with RA >10 years, ≥ 2 cardiac risk factors, calcification at baseline and statin treatment >1 year (figure 1).

Conclusion: High baseline EATv independently predicted future higher-risk non-calcified and mixed coronary plaque in RA. Moreover, it conditionally promoted new plaque formation overall in patients with earlier disease, low cardiac risk factor burden, who had little or no atherosclerosis at baseline and who had limited exposure to statin therapy. These findings indicate the need for a larger prospective evaluation of the role of EATv as a biomarker of coronary atherosclerosis development in RA.

REFERENCES:

- [1] Hwang I-C et al. *J Atheroscler Thromb* 2017;24:262–74. 2. Ding J et al. *Am J Clin Nutr* 2009;90:499–504.

*All models adjusted for proximal segment location, time-averaged CRP, cumulative prednisone dose, and total methotrexate dose.
†Additionally adjusted for Framingham risk score, body mass index, and statin duration.
‡Additionally adjusted for age, gender, body mass index, and statin duration.
§Additionally adjusted for Framingham risk score and statin duration.
¶Additionally adjusted for Framingham risk score and body mass index.

Figure 1. Moderators of influence of EATv on new coronary plaque formation

Disclosure of Interests: George Karpouzas Speakers bureau: Sanofi/ Genzyme/ Regeneron, Consultant of: Sanofi/ Genzyme/ Regeneron, Grant/research support from: Pfizer, Sarah Ormseth: None declared, Elizabeth Hernandez: None declared, Matthew Budoff Consultant of: Pfizer

DOI: 10.1136/annrheumdis-2021-eular.3347

POS0216

GREATER HIGH-DENSITY LIPOPROTEIN LEVELS OVER TIME ARE LINKED TO DECREASED CORONARY PLAQUE FORMATION AND REGRESSION AND STABILIZATION OF HIGH-RISK LESIONS IN RHEUMATOID ARTHRITIS

G. Karpouzas^{1,2}, S. Ormseth¹, E. Hernandez¹, M. Budoff³. ¹The Lundquist Institute, Rheumatology, Torrance, United States of America; ²Harbor-UCLA Medical Center, Rheumatology, Torrance, United States of America; ³The Lundquist Institute, Cardiology, Torrance, United States of America

Background: The relationship between serum lipoproteins and cardiovascular disease risk in rheumatoid arthritis (RA) is complex¹. Their levels and function may vary based on disease activity and medication use. Beneficial effects on high-density lipoprotein (HDL-C) levels, structure and behavior, in response to treatment have been described. However, the impact of HDL-C levels over time on coronary atherosclerosis progression in RA is unknown.

Objectives: We here evaluated the influence of HDL-C levels over time on long-term coronary plaque formation and progression in patients with RA.

Methods: One hundred one RA patients without symptoms or history of cardiovascular disease who participated in a computed tomography angiography study of coronary atherosclerosis had repeat assessments after 6.9±0.3 years to evaluate plaque progression. Clinical, laboratory and medication data were recorded at baseline and regular outpatient follow-up visits thereafter. Time-averaged HDL-C was calculated for each patient using available consecutive HDL measurements between baseline and follow-up. Robust logistic regression assessed the association between time-averaged HDL-C and likelihood of new plaque formation in segments without plaque at baseline, and transition of prevalent mixed plaque to calcified plaque. Robust multinomial logistic regression evaluated the effect of time-averaged HDL-C on likelihood of new non-calcified, mixed or calcified plaque formation in segments without plaque (compared to remaining without plaque), and non-calcified plaque regression or transition to mixed or calcified plaque at follow-up (compared to remaining non-calcified). All models accounted for clustering of coronary segments within patients and adjusted for Framingham D'Agostino risk score, proximal segment location, time-averaged CRP, cumulative prednisone dose, bDMARD duration, statin duration, waist-to-height ratio, and time-averaged triglycerides.

Results: Participants were mostly female (n=87, 86.1%), with a mean ± standard deviation (SD) age of 51.5±10.3 years and time-averaged HDL-C of 51.7±13.9. Ninety-seven new plaques formed in segments without plaque at baseline; 20 were noncalcified, 21 were mixed, and 56 were calcified. Time-averaged HDL-C