only in the RA population. In RA patients, treatment regimen and Disease Activity Score 28 were recorded.

Results: There were 413 participants (208 RA patients and 205 in the Vo group; mean age, 58 years) enrolled in the study, 349 of whom were female. In RA patients (mean disease duration, 13 years), bone density was significantly lower ($p<0.001$, repeated-measures ANOVA) and Hcy ($p<0.0001$, repeated-measures ANOVA) was higher in comparison with the Vo group during the 7 year study period. In the analysis of change in Hcy level over 7 years, "RA" and "time" were found to interact with each other ($p=2.58e-7$, repeated-measures ANOVA) (figure 1). Multiple linear regression analysis in the RA population revealed a relationship between the level of Hcy and MTX dose at baseline ($p=0.048$), but no relationship between the level of Hcy and MCV ($p=0.165$).

Abstract THU0139 – Figure 1. Homocysteine level during the study period in the patients with rheumatoid arthritis (RA) and healthy volunteer (Vo)

Conclusions: MTX intake leads to folate deficiency, which is thought to cause elevation of the Hcy level. Ageing is another significant factor related to Hcy increase.

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THU0140 EFFECTS OF STATIN-TREATMENT ON CORONARY PLAQUES IN PATIENTS WITH INFLAMMATORY JOINT DISEASES

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Background: Statins have an established preventive effect on coronary artery disease in the general population. The effect of statins on coronary plaque progression and characteristics in patients with inflammatory joint diseases (IJD) is unknown.

Objectives: Our aim was to evaluate the change in coronary atherosclerosis in long-term statin-treated patients with IJD.

Methods: Sixty-eight patients with IJD and carotid artery plaque, underwent coronary computed tomography angiography before and after 4.7 (range 4.0–6.0) years of statin treatment. The treatment target for low density lipoprotein cholesterol (LDL-c) was ≤ 1.8 mmol/L. Changes in coronary artery calcification (CAC) and coronary artery plaque volume (calcified, mixed/soft and total) from baseline to follow-up were assessed using the 17-segment model of the American Heart Association. Linear regression analysis was used to identify predictors of atherosclerotic progression.

Results: Coronary plaques were present in 42% of the patients at baseline and in 51% at follow up. Mean CAC score increased with 173 ± 284 , calcified plaque volume with 39.4 ± 78.3 mm³ and total plaque volume with 22.8 ± 54.6 mm³ ($p\leq 0.01$, for all) (figure 1). Mean mixed/soft plaque volume decreased with -10.4 ± 27.5 mm³ ($p\leq 0.01$). At follow-up, 51% of the patients had obtained LDL-c treatment target. Compared to patients above LDL-c target, patients with an LDL-c ≤ 1.8 mmol/L experienced reduced median progression of both CAC (21^{2-143} vs. 69 [16–423],

$p<0.01$) and total plaque volume (0.08 [–1.0–13.9] vs. 13.0 [0.0–60.8], $p=0.02$) (table 1).

	LDL ≤ 1.8 mmol/L n=34	LDL > 1.8 mmol/L n=34	p-value*
LDL-c level baseline, mean $\pm$ SD	3.7 $\pm$ 0.9	4.4 $\pm$ 1.0	<0.01
LDL-c level follow-up, mean $\pm$ SD	1.5 $\pm$ 0.2	2.4 $\pm$ 0.7	<0.01
Change LDL-c level, mean $\pm$ SD	–2.2 $\pm$ 0.9	–1.9 $\pm$ 1.3	0.38
Change in CAC, median (IQR)	21 (2–143)	69 (16–423)	<0.01
Change in soft/mixed plaque volume, median (IQR)	0 (–3.5–0.0)	0 (–15.7–0.0)	0.71
Change in calcified plaque volume, median (IQR)	1.7 (0.0–17.3)	13.4 (1.5–107.6)	0.02
Change in total plaque volume, median (IQR)	0.08 (–1.0–13.9)	13.0 (0.0–60.8)	0.02

*independent samples t-test

Abstract THU0140 – Figure 1. Mean change in plaque volume (mm³)

Conclusions: We revealed a progression of atherosclerotic plaque volume in statin-treated patients with IJD, mainly due to calcifications. However, soft, unstable plaques were reduced, probably as a result of an alteration in plaque composition from mixed/soft plaques into calcified plaques. Patients with recommended LDL-c levels at follow-up experienced a reduced atherosclerotic progression compared to patients with LDL-c levels above the treatment target. Our results support the importance of treatment to guideline recommended lipid targets in IJD patients.

Disclosure of Interest: None declared

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THU0141 LONG-TERM EFFECTS ON BONE MINERAL DENSITY AFTER FOUR YEARS OF TREATMENT WITH TWO INTENSIVE COMBINATION STRATEGIES, INCLUDING INITIALLY HIGH DOSE PREDNISOLONE, IN EARLY RHEUMATOID ARTHRITIS PATIENTS: THE COBRA-LIGHT TRIAL

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Background: COmbinatie therapie Bij Reumatoide Artritis (COBRA)-light therapy (methotrexate and initially 30 mg/day prednisolone) has proven to be non-inferior to COBRA therapy (methotrexate, sulfasalazine and initially 60 mg/day prednisolone) in the first year of treatment of early rheumatoid arthritis (RA) patients.

Objectives: This study assessed changes in bone mineral density (BMD) after four years in early RA patients initially randomised to one year of COBRA or COBRA-light therapy.

Methods: In the open-label, randomised, non-inferiority trial patients were assigned to COBRA or COBRA-light therapy. After one year, treatment was at the discretion of the treating rheumatologists. BMD in g/cm² was measured at baseline, after one, two and four years at total hip, femoral neck, and lumbar spine with dual-energy X-ray absorptiometry (DXA).

Results: Of the 164 original patients, 154 could be assessed after a follow-up of four years (range 34 to 74 months); 68% were female; mean (SD) age at follow-up 52¹³ years. In the COBRA-light group, 11% of the patients used bisphosphonates after four years; the mean cumulative prednisolone dosage was 2.6 g (inter quartiles: 1.9; 5.9) and 49% of the patients had minimal disease activity (DAS44 <1.6).

Abstract THU0141 – Table 1. BMD values and changes in BMD during four years of treatment

	Baseline	COBRA-Light Bone Loss		Baseline	COBRA Bone Loss		Mean difference	GEE analyses	P
		Absolute	% of baseline		Absolute	% of baseline		CI	
Lumbar Spine	1.10 (0.15)	−0.01 (0.08)	−0.5	1.12 (0.18)	−0.01 (0.08)	−1.0	−0.01	−0.03– 0.02	0.56
							−0.01*	−0.03– 0.02	0.56
							−0.01**	−0.03– 0.02	0.59
Total Hip	0.96 (0.13)	−0.03 (0.05)	−3.3	0.95 (0.15)	−0.02 (0.05)	−1.7	−0.01	−0.02– 0.00	0.05+
							−0.01*	−0.02– 0.00	0.05+
							−0.01**	−0.03– 0.00	0.02+
Femoral Neck	0.88 (0.12)	−0.03 (0.05)	−3.7	0.90 (0.16)	−0.03 (0.06)	−3.0	−0.01	−0.04– 0.02	0.38
							−0.01*	−0.04– 0.01	0.37
							−0.01**	−0.04– 0.01	0.29

In the COBRA group, these numbers were 10%, 3.2 g (2.5; 6.2) and 49%, respectively. At the lumbar spine, both groups showed no significant decline in BMD over four years and no difference between treatment groups in BMD change (table 1). At the hips, 1.7% to 3.7% BMD loss over four years was found with slightly but significantly more loss in the COBRA-light group (table 1). Outcomes are mean (SD) unless stated otherwise. BMD in g/cm². *Significant difference between COBRA-light and COBRA on average over time. *Adjusted for bisphosphonate usage (yes vs no). **Adjusted for bisphosphonate usage (yes vs no), cumulative prednisolone usage, age, gender and disease activity based on DAS44 (DAS44 <1.6 in remission vs DAS44 ≥1.6 not). CI, confidence interval; GEE, Generalised Estimating Equations; T4, measurement after four years; SD, Standard Deviation.

Conclusions: In modern treat-to-target management of RA, including bone surveillance, a high starting dose of prednisolone, either 30 or 60 mg/day, was not associated with a dramatically increased bone loss at the lumbar spine, and minor losses at the hip over four years.

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THU0142 THE PROGNOSIS OF HEART FAILURE IN PATIENTS WITH RHEUMATOID ARTHRITIS

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Background: Heart failure (HF) is a condition with high rates of hospital admission and mortality. The impact of rheumatoid arthritis (RA) and its treatment on the prognosis of prevalent HF has been insufficiently studied.

Objectives: To evaluate deterioration of HF and mortality in patients with RA and concomitant HF.

Methods: The prospectively followed cohort of the German register RABBIT continuously includes RA patients with a new start of a DMARD after at least one csDMARD failure. Among all patients enrolled between 05/2001 and 10/2017 (n=15,037) patients with prevalent HF were selected (n=393). HF patients were followed until their end of observation or death. Deterioration of HF requiring hospital admission, and death were analysed as composite outcome. Incidence rates (IR) were calculated for current treatment at time of event (9 months risk window after last infusion of rituximab). Generalised estimation equations (GEE) were used to investigate risk factors for the composite outcome. To avoid uncertainties when allocating therapies, only treatment episodes >6 months were included in the GEE analysis. Missing values (DAS28, CRP, physical function) were addressed by multiple imputations.

Results: Of the 393 patients with prevalent HF and 1490 patient years (PY) of follow-up, a total of 131 patients had at least one outcome (19 HF deteriorations, 123 deaths). Infections (30%) and cardiovascular events (25%) were most frequently

reported as causes of death. The mean time until deterioration/death was 30/35 months. At baseline, patients with an event were older (69 vs. 67 years), more often male (43 vs. 32%), rheumatoid factor positive (80 vs. 74%), had higher CRP-values (39 vs. 23 mg/L) and a worse FFbH (% of physical function: 43 vs. 50) than patients without event. All HF patients had high numbers of comorbidities (mean of 7/6 in patients with/without event).

Crude IR were highest in patients under csDMARD only exposure (figure 1). IR were similar during the first 3 or 6 months after start of treatment and thereafter (data not shown). Biologic treatment was not associated with the outcome (table 1). Male gender, higher age, a higher glucocorticoid dose, worse physical function and elevated CRP under treatment were significantly associated with hospitalisation due to HF or a fatal outcome.

Abstract THU0142 – Table 1. Adjusted relative risk of HF deterioration or death in RA patients with underlying HF. CRP, FFbH and glucocorticoid doses were averaged for the time of a therapy episode.

	Relative risk	95% Confidence interval	
csDMARD	Ref.		
TNF inhibitors	0.7	0.4	1.3
Abatacept	0.8	0.3	2.3
Rituximab	0.5	0.2	1.1
Tocilizumab	0.9	0.3	2.6
Baseline age per 5 years	1.3	1.1	1.5
Male vs. female	2.4	1.4	3.9
CRP per 5 mg/L	1.03	1.004	1.1
% of physical function per 10 points	0.9	0.8	0.999
Sum of baseline comorbidities	1.1	0.96	1.3
Glucocorticoids per 5 mg/d	1.4	1.03	1.8
Smoker vs non-smoker	1.7	1.02	3.0

Abstract THU0142 – Figure 1. Incidence rates of composite outcome per 100 patient years