to compare incident HF events by decade, adjusting for age, sex and cardiovascular risk factors: smoking, obesity, diabetes mellitus, hypertension, dyslipidemia. Cumulative incidence of HF adjusted for death was also computed.

Results: The study included 905 patients with RA (mean age 55.9 years; 69% female; median follow-up 13.4 years). The 10-year cumulative incidence of HF in RA cohort in the 1980s was 8.5% (95%CI 5.3-13.6%), 1990s was 10.8% (95%CI 7.7-15.1%), and 2000s was 7.1% (95%CI 4.9-10.3%). There was no difference in incidence of HF in 1990s (hazard ratio [HR] 0.91, 95% Confidence Interval [CI] 0.62-1.35) and 2000s (HR 0.73; 95%CI 0.46-1.18) compared to 1980s. Patients with incident RA were then compared to 903 individuals without RA (mean age 56.0 years; 69% female; median follow-up 13.8 years). The 10-year cumulative incidence of HF in these individuals in the 1980s was 7.4% (95%CI 4.5-12.3%), 1990s was 7.5% (95%CI 4.9-11.3%), and 2000s was 7.3% (95%CI 5.0-10.7%). Similar to RA, there was no statistically significant difference in incidence of HF in 1990s (HR 0.96, 95%CI 0.60-1.51) and 2000s (HR 0.75, 95%CI 0.44-1.30) compared to the 1980s. When comparing the risk of HF in RA and non-RA subjects, patients with RA in 2000s had no excess in HF risk as compared to the general population (HR 1.14, 95%CI 0.73-1.78, Figure 1). This is in contrast to the 2-fold excess risk of HF in patients with RA in 1980s (HR 2.20, 95%CI 1.44-3.34) and ~1.5-fold increase in risk of HF in 1990s (HR 1.54, 95%CI 1.04-2.29).

Figure 1. Cumulative incidence of any HF event in RA and non-RA patients by decade of RA incidence/index

Conclusion: We found a reduction in excess HF risk in patients with RA compared to individuals without RA in 2000s compared to 1980s. There were no statistically significant changes in incidence of HF in patients with RA and in individuals without RA over time. More studies are needed to understand the reasons and implications of these trends.

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OP0103

5-YEAR CARDIOVASCULAR EVENT RISK IN EARLY RHEUMATOID ARTHRITIS PATIENTS WHO RECEIVED TREAT-TO-TARGET MANAGEMENT: A POPULATION-BASED COHORT STUDY

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Background: Rheumatoid arthritis (RA) is associated with higher cardiovascular disease (CVD) risk due to the underlying inflammation⁽¹⁾. It is uncertain if the excess CV risk could be reduced by effective suppression of inflammation using the treat-to-target (T2T) approach in patients with early RA (ERA).

Objectives: This study compared the 5-year cardiovascular event (CVE) rate among ERA patients managed by a T2T strategy with a CV risk factor-matched non-RA population.

Methods: This was an observational study using the Hong Kong Hospital Authority population-based hospital database known as the Clinical Data Analysis and Reporting System (CDARS) and the Clinical Rheumatology Systematic Treat-to-target in Asia Leadership (CRYSTAL) registry. ERA subjects from the registry with baseline disease duration less than 2 years were recruited between 2012-2016. All patients received a tight control, T2T treatment strategy aiming at remission and had been followed for at least 5 years. Each ERA subject was matched to 3 non-RA controls according to age, gender, smoking status, and the presence of diabetes mellitus (DM), hypertension (HT) and hyperlipidaemia (HL). All subjects on antiplatelet agents, with pre-existing CVD or chronic kidney disease were excluded. All subjects were analysed up to 5 years from baseline. The primary end point was the first occurrence of a CVE, namely ischaemic cerebrovascular disease, ischaemic heart disease and heart failure.

Results: The study identified 261 ERA subjects and 783 matched controls. Their characteristics were shown on Table 1. The mean age was 60+/-12 years, 78% were female, 18% were smokers, 8%, 25% and 22% had DM, HT and HL respectively.

Table 1. Characteristics of ERA subjects and the matched controls

	ERA (n=261)	Controls (n=783)	p-value
Age (mean +/- SD)	60.2 +/- 12	60.3 +/- 12	0.99
Total cholesterol (mean +/- SD)	4.8 +/- 0.94	4.9 +/- 1.00	0.34
High density lipoprotein-cholesterol (median, IQR)	1.4 (1.2-1.6)	1.4 (1.2-1.7)	0.612
Low density lipoprotein-cholesterol (mean +/- SD)	2.8 +/- 0.8	2.6 +/- 0.8	0.27
Triglyceride (median, IQR)	0.96 (0.7-1.3)	1.2 (0.8-1.7)	0.00
Atherogenic index# (mean +/- SD)	-0.07 +/- 0.29	-0.15 +/- 0.28	0.48
RA disease characteristics	Baseline	last visit	
Seropositivity	89.3%		
Disease duration (median days, IQR)	177 (96-299)	2026 (1818-2126)	
Erythrocyte sedimentation rate (mean +/- SD)	55.7 +/- 34	32.6 +/- 21.3	
C-reactive protein (median, IQR)	7.9 (3.5-22.2)	2.1 (1.0-5.0)	
Swollen joint count (median, IQR)	3 (2-6)	0 (0-1)	
Tender joint count (median, IQR)	6 (3-10)	0 (0-1)	
Patient's global assessment (mean mm +/- SD)	50.7 +/- 26.8	16.0 +/- 18.3	
DAS28-ESR (mean +/- SD)	5.2 +/- 1.3	2.4 +/- 1.8	
DAS28-CRP (mean +/- SD)	4.4 +/- 1.3	1.8 +/- 1.5	
Health assessment questionnaire (mean +/- SD)	0.77 +/- 0.74	0.38 +/- 0.51	

Atherogenic index = $\log_{10}$ (triglyceride/HDL-cholesterol)SD: standard deviation, IQR: inter-quartile range, DAS28: disease activity score-28

CVEs occurred in 2.3% ERA subjects and in 3.3% controls. The difference was statistically insignificant. The CVE-free survival was shown in Figure 1.

Figure 1. CVE-free survival curves of ERA subjects & matched controls

In the ERA cohort, after adjusting for baseline age and atherogenic index, cox-regression analysis showed that i) a longer DAS28 remission duration was protective for CVE with an adjusted hazard ratio (AHR) of 0.46 (95% CI 0.23-0.93),

while ii) poor baseline function as reflected by a higher health assessment questionnaire (HAQ) score was predictive of CVE with an AHR of 5.2 (95% CI 1.2-23). **Conclusion:** ERA patients treated by a T2T strategy did not develop excess CVE compared to CV risk factor-matched controls over 5 years. A longer disease remission duration was protective while a higher baseline HAQ was associated with a higher CVE risk.

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OP0104

DIFFERENCES IN LOW-DENSITY LIPOPROTEIN (LDL) PARTICLE COMPOSITION AND OXIDATION MAY UNDERLIE THE PARADOXICAL ASSOCIATION OF LOW LDL WITH HIGHER CORONARY ATHEROSCLEROSIS BURDEN IN RHEUMATOID ARTHRITIS

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Background: The association between cholesterol and cardiovascular disease (CVD) risk is attenuated in Rheumatoid arthritis (RA). In fact, RA patients in the lowest low-density lipoprotein (LDL) group (<70mg/dl) may experience unexpectedly high CVD risk. **Objectives:** We here explored whether patients with LDL<70mg/dl (Group 1) had higher coronary atherosclerosis burden compared to other LDL groups (Group 2: 70≤LDL≤130 and Group 3: LDL>130), as a reason for this risk. We further evaluated whether low LDL in group 1 associated with differences in inflammation, LDL particle composition or oxidation.

Methods: One hundred fifty RA patients without symptoms or history of CVD underwent coronary atherosclerosis evaluation with computed tomography angiography. Coronary artery calcium (CAC), number of segments with plaque (segment involvement score), stenotic severity (segment stenosis score), and extensive (>4 segments with plaque) or obstructive disease (>50% stenosis) were assessed. Lipoprotein classes and subclasses were directly measured. Oxidized LDL (oxLDL) was measured with monoclonal antibody E06. Chemiluminescence Elisa quantified IgG and IgM antibodies to oxLDL (anti-oxLDL) and apoB100 immune complexes (IC). Proinflammatory cytokines were measured with Erenna Immunoassay. Robust linear and logistic regression models adjusted for Framingham D'Agostino score, obesity, disease activity, bDMARD and statin treatment- evaluated associations between LDL groups and plaque outcomes. Similar models evaluated adjusted differences in LDL subclasses, oxLDL, anti-oxLDL, anti-ApoB100 IC, and cytokines across LDL groups.

Results: Group 1 patients had higher coronary plaque burden (Figure 1A) and 2.8 times greater risk of extensive or obstructive disease (adjusted OR 2.82 [95% CI 1.12-7.17], $P = 0.031$) compared to LDL>70 groups. Among statin naïve patients,

those with LDL<70 also had higher oxLDL (log-transformed adjusted mean 2.55 [95% CI 2.34-2.77] versus 2.27 [95% CI 2.19-2.36], $P = 0.018$ for LDL>70). Notably, Group 1 patients also had higher anti-oxLDL IgG and anti-ApoB100 IgG IC levels compared to other groups (Figure 1B). LDL subclass relative content in the LDL particle differed across groups (Figure 1C). Lp(a) was higher in LDL particles in Group 1 (adjusted mean 16.04% [95% CI 11.75-20.33], versus 10.48% [95% CI 8.20-12.75] in Group 2, $P = 0.026$ and 7.41% [95% CI 0.77-14.04] in Group 3, $P = 0.033$). Notably, Lp(a) content strongly associated with oxLDL overall ($r = 0.83$, $P < 0.0001$). This association was stronger for Group 1 compared to others ($P < 0.005$, Figure 1D). No differences in RA activity, CRP, TNF- α , IL-17A, or IL-17F were seen across groups. However, Group 1 had higher IL-6 (log-transformed adjusted mean 1.98 [95% CI 1.64-2.32] versus 1.57 [95% CI 1.45-1.70], $P = 0.028$ in Group 2 and 1.32 [95% CI 0.84-1.80], $P = 0.031$ in Group 3). IL-6 associated with both IgG anti-oxLDL ($P = 0.015$) and anti-ApoB100 IC ($P = 0.016$). Log-transformed IL-6 further associated with higher log-transformed CAC (adjusted B 0.41 [95% CI 0.01-0.81], $P = 0.049$).

Conclusion: RA patients with LDL<70mg/dl had higher coronary atherosclerosis burden. Low circulating LDL in that group may reflect higher oxidation; this was mostly linked to the larger Lp(a) relative content of LDL and its significantly higher oxidation potential in that group. OxLDL immune recognition was linked to higher IgG anti-oxLDL Ab and anti-ApoB100 IC levels in the LDL<70 group, which further associated with higher IL-6 elaboration and atherosclerosis burden.

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OP0105

FEASIBILITY AND USEFULNESS OF MAPPING BIOLOGIC REGISTRIES TO A COMMON DATA MODEL: ILLUSTRATION USING COMORBIDITIES

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Background: The Observational and Medical Outcomes Partnerships (OMOP) common data model (CDM) provides a framework for standardising health data with a view towards federated analyses, thus maximising the use and power of combining disparate datasets.

Objectives: To assess feasibility and usefulness of mapping biologic registry data from different European countries to the OMOP CDM and present initial descriptive data regarding comorbidities.

Methods: Five biologic registries, as part of a funded FOREUM project, have been mapped to the OMOP CDM: 1) the Czech biologics register (ATTRA), 2) Registro Español de Acontecimientos Adversos de Terapias Biológicas en Enfermedades Reumáticas (BIOBADASER), 3) British Society for Rheumatology Biologics Register for Rheumatoid Arthritis (BSRBR-RA), 4) German biologics register 'Rheumatoid arthritis observation of biologic therapy' (RABBIT), and 5) Swiss register 'Swiss Clinical Quality Management in Rheumatic Diseases' (SCQM). The mapping includes socio-demographic, observation period within the studies, baseline comorbidities, and baseline medications. Only patients with RA were included. Using R, registers received identical scripts to run on their mapped databases to produce an initial description of patient characteristics without the need to share patient-level data.

Results: A total of 54,458 individuals are included in the five registries being mapped to the OMOP CDM, see table. Age and gender distribution was similar across registries. All registers reported on cardiovascular system comorbidities, diabetes mellitus, mental disorders, and respiratory system comorbidities. However, it was noted that results of comorbidity mapping relies on what each register collect on each patient at the point of registration.

Whilst the Charlson comorbidity index could be calculated within each registry, due to lack of the specific coding needed, such as "uncomplicated diabetes mellitus" / "end-organ damage diabetes mellitus", it was felt to be an inaccurate

Figure 1 (A) Mean segment involvement score (SIS), segment stenosis score (SSS), and log-transformed coronary artery calcium (CAC-In) across LDL groups, adjusted for Framingham D'Agostino score, DAS28-CRP, waist-to-height ratio, bDMARD use, and statin use. (B) Mean serum levels of IgG and IgM anti-oxLDL and apoB100 immune complexes (IC) across LDL groups, adjusted for Framingham D'Agostino score and statin use. (C) LDL particle composition across LDL groups, adjusted for Framingham D'Agostino risk score, statin use, and total HDL-C. (D) Association between Lp(a) content and LDL oxidation across LDL groups, adjusted for Framingham D'Agostino score and statin use. * $p < 0.050$, ** $p < 0.010$, † $p = 0.051$, ‡ $p = 0.056$