OBJECTIVES: We here explored whether abdominal obesity (waist-to-height ratio >0.58 in females and >0.63 in males) might moderate the effect of low LDL (<70 mg/dl) on coronary atherosclerosis burden, progression and long-term CVD risk in RA.

Methods: One hundred fifty patients without symptoms or diagnosis of CVD underwent coronary computed tomography angiography. Plaque progression was evaluated 6.9±0.3 years later in 101 patients. Coronary artery calcium, number of segments with plaque (segment involvement score), and extensive (>4 segments with plaque) or obstructive disease (>50% stenosis) were assessed. CVD events were prospectively recorded, including cardiac death, myocardial infarction, unstable angina, revascularization, stroke, claudication, and heart failure hospitalization over 6.0±2.4 years of follow-up. Lipoprotein classes were directly measured. Oxidized LDL (oxLDL) was assessed with monoclonal antibody E06. Adjusted robust linear regression evaluated interactions between abdominal obesity and LDL groups on plaque outcomes. Per segment, adjusted robust logistic regression models explored obesity x LDL group interactions on new plaque formation and stenotic plaque progression of prevalent plaques. Robust Cox regression models stratified by abdominal obesity evaluated the effect of LDL group (<70 mg/dl) on CVD events.

Results: Non-obese patients with low LDL had the highest plaque burden (Figure 1A, all p < 0.02). Obesity moderated the effect of LDL on likelihood of extensive/obstructive disease (P for interaction = 0.061); specifically, LDL<70 associated with higher baseline coronary atherosclerosis burden, progression, and long-term CVD risk in RA.

Conclusion: Compared to LDL>70 mg/dl hearts, non-obese patients (adjusted OR 1.55 [95% CI 0.39-6.08], P = 0.27, Figure 1D). Obesity further moderated the effect of LDL on likelihood of future development of plaque (P for interaction = 0.011) but not obese patients (adjusted OR 0.50 [95% CI 0.11-2.21], P = 0.36, Figure 1B). Obesity further moderated the effect of LDL on likelihood of future development of plaque (P for interaction = 0.002) and increased stenotic severity of existing plaques (P for interaction = 0.040); in non-obese patients, low LDL associated with a greater likelihood of new plaque forming in segments without baseline plaque (adjusted OR 4.68 [95% CI 2.26-9.66] and worsening stenotic severity in segments with prevalent plaque (OR 5.35 [95% CI 1.62-17.67]). This was not observed in obese patients (Figure 1C). Notably, in non-obese low patients, low LDL associated with higher CVD event risk compared to those with LDL<70 mg/dl (HR 7.94 [95% CI 1.52-41.36], P = 0.015). This was not the case in obese patients (HR 0.32 [95% CI 0.04-2.40], P = 0.27, Figure 1D).

Disclosure of Interests: None declared.