We forwarded the measurements for the control group to the general practitioner for assessment. We collected information on adherence to statin treatment at the yearly interview and by review of medical records. Each reason for treatment discontinuation were recorded and classified as temporary or permanent.
Results: Out of 963 patients, 89.3% (n=434) in the intervention and 82.0% (n=391) in the control group were persistent to statin treatment after a median of 3.9 years of follow-up (p=0.001). The most prevalent reason for permanent discontinuation in the intervention group was advanced disease (27.5%, n=14) while in the control group it was side effects without a compelling relation to treatment (32.8%, n=22). A total of 27.8% (n=135) of the patients in the intervention group and 20.5% (n=98) in the control group discontinued treatment at some point during the period (p=0.009). The most common reasons for a first discontinuation were in both arms side effects without a compelling relation to treatment and lack of treatment motivation.
Conclusion: A nurse-based, long-term follow-up by telephone after an ACS with individualised medical adjustments results in a higher persistence to statin treatment than usual care.
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P5379
Predictive value of omega-3 polyunsaturated fatty acids (PUFAs) for cardiovascular- and all-cause mortality in chronic haemodialysis patients

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Background: Decreased omega-3 (n-3) polyunsaturated fatty acids (PUFAs) levels are closely associated with incidence of cardiovascular disease (CVD). Although haemodialysis (HD) patients are widely recognized to be at highest risk of CVD, the association of the PUFAs with CVD prognosis remains unclear. We investigated the predictive value of n-3 PUFAs for CVD- and all-cause mortality in chronic HD patients.
Methods: A total of 360 outpatients stably undergoing maintenance HD therapy (male 68%, age 66±11years, diabetes 41%) were enrolled. They underwent measurement of eicosapentaenoic acid (EPA), docosahexaenoic acid (DHA) and arachidonic acid (AA), and were followed-up for 3 years.
Results: During follow-up period (32 months), 84 patients died (23.3%) including 48 CVD cause (13.3%). On Cox multivariate analysis, DHA [hazard ratio (HR) 0.89, 95% confidence interval (CI) 0.81–0.97, p=0.013], EPA (HR 0.86, 95% CI 0.78–0.95, p=0.0014) and EPA/AA ratio (HR 0.77, 95% CI 0.65–0.89, p=0.0002) were identified as independent predictors of CVD mortality. Similar results were also obtained for all-cause mortality. However, the addition of DHA to a prediction model based on traditional risk factors had no effect on model discrimination as measured by C-index. On the other hand, only the addition of EPA/AA ratio could improve C-index for both CVD- and all-cause mortality, moreover, even if compared to the prediction model with DHA (Table). Kaplan-Meier survival rate at 3-year was lower in patients with EPA/AA<0.56 as a value of third quartile, compared to EPA/AA≥0.56 for CVD mortality (82.6% vs. 95.3%, p=0.0035), and all-cause mortality (72.9% vs. 87.7%, p=0.0042), respectively.

Table 1

	Cardiovascular death		All-cause death	
	C-index (95% CI)	P value	C-index (95% CI)	P value
Basic model	0.722 (0.643–0.800)	Reference	0.799 (0.742–0.856)	Reference
+ DHA	0.739 (0.669–0.809)	0.38	0.806 (0.751–0.861)	0.29
+ EPA	0.752 (0.682–0.822)	0.13	0.822 (0.769–0.875)	0.025
+ EPA/AA	0.773 (0.704–0.842)	0.0091	0.823 (0.769–0.876)	0.028
+ EPA/AA vs. + DHA	0.034 (0.005–0.072)*	0.048	0.016 (0.002–0.035)*	0.026
+ EPA/AA vs. + EPA	0.021 (-0.001–0.041)*	0.089	0.001 (-0.012–0.011)*	0.93

*Estimated differences between two groups.

Conclusion: Although DHA, EPA and EPA/AA ratio levels were associated with CVD- and all-cause mortality in HD patients, the measurement of EPA/AA ratio was only clinically meaningful for screening from the view point of the predictability for CVD mortality with increased C-index.

P5380
The association between apolipoprotein A1 and HDL-cholesterol with acute myocardial infarction is modified by plasma choline. A cohort study of patients with suspected stable angina pectoris

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Background: Choline is related to 1-carbon metabolism and essential for lipoprotein assembly. Higher plasma choline has been associated with an increased risk of cardiovascular events, whereas serum high density lipoprotein (HDL)-cholesterol (HDL-C) and apolipoprotein (apo) A1, the main apolipoprotein of HDL, is inversely related to cardiovascular risk. There is evidence suggesting that the choline metabolism and HDL metabolism are interconnected; however, their potential interactions according to future cardiovascular events are not known.
Purpose: To investigate the potential effect modification by plasma choline on the relationship between apo A1 and HDL-C with incident acute myocardial infarction (AMI).
Methods: We studied patients evaluated for suspected stable angina pectoris, and who were followed up for long-term cardiovascular events as identified according to regional health registries. By Cox regression, we investigated the associations between serum apo A1 and HDL-C with incident AMI according to median plasma choline concentration.
Results: Median (5th-95th percentile) age of the 4153 patients (2988 (71.9%) men) was 62 (44–78) years. During follow-up for median (5th-95th percentile) 4.6 (1.6–6.8) years, 344 (8.3%) patients suffered from at least one AMI. As expected, we found inverse associations between serum apo A1 and HDL-C with subsequent AMI in the total population; however, the association for apo A1 was present only when plasma choline was ≥ median [age and gender adjusted HR (95% CI) 0.71 (0.61–0.83) vs. 1.06 (0.88–1.28) when plasma choline was < median; P for interaction=0.009], and a similar trend was also observed for HDL-C (P for interaction=0.09). Further adjusting for hypertension, smoking, diabetes and body mass index yielded similar results, and the 3-dimensional generalized additive model plot in Figure 1 suggests that the interaction is approximately linear.

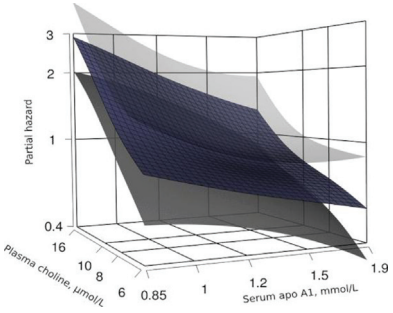

Figure 1

Conclusion: Serum apo A1 and HDL-C were inversely related to risk of future AMI, with stronger associations observed among patients with concomitant high plasma choline concentrations. Our results motivate further studies into potential ramifications between choline and lipid metabolism according to coronary heart disease.

P5381
Post-prandial remnant lipoprotein metabolism in sitosterolemia

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Background: It has been shown that sitosterolemic patients are vulnerable to diet-induced hypercholesterolemia. However, few data exist regarding post-prandial accumulation of lipoproteins in this rare condition.
Methods: OFTT cream 50 g was given per body surface area (m^2), blood sampling was performed at 2 hour intervals up to 6 hour. Plasma lipoprotein fractions and RLP fractions were determined in four sitosterolemic subjects with double mutations in ATP-binding cassette (ABC) sub-family G member 5 or member 8 (ABCG5 or ABCG8) gene (mean age=18yr, median LDL-C=154mg/dl), six heterozygous carriers (31yr, median LDL-C=105mg/dl), and five subjects with heterozygous FH (mean age=32yr, median LDL-C=221mg/dl). The area under curve (AUC) of lipids, including LDL-C, apolipoprotein B48 (ApoB48), RLP-TG, and RLP-C were evaluated.
Results: After oral fat load, the AUC of LDL-C was significantly smaller in sitosterolemia subjects than that in heterozygous FH (977 mg/dl×hour vs. 1,333 mg/dl×hour, p<0.05), whereas, the AUC of ApoB48 was significantly larger in the sitosterolemic subjects compared with that of heterozygous FH (37 mg/dl×hour vs. 9 mg/dl×hour. P<0.05). Under these conditions, the AUCs of RLP-C, and RLP-TG levels were significantly larger in the sitosterolemic subject compared with those of heterozygous FH (92 mg/dl×hour vs. 53 mg/dl×hour, p<0.05, 375

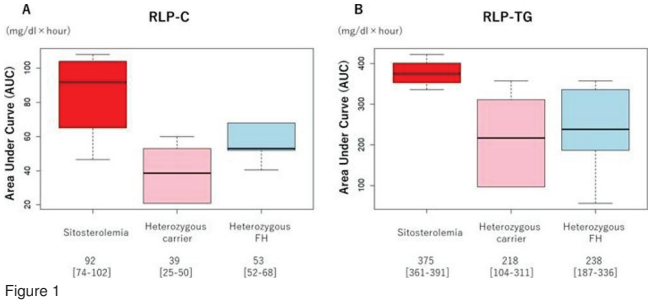

Figure 1