

SUO014 Figure 1.

**Results:**

Baseline serum fetuin levels were significantly lower in CKD patients. Spearman correlation revealed that uFCR was related to uPCR ( $r=0.582$ ,  $p<0.001$ ) and uACR ( $r=0.570$ ,  $P<0.001$ ) but had no relation to plasma Fet A levels ( $r=-0.003$ ,  $p=0.971$ ). Stratification by into high and low urinary fetuin groups (by median uFCR) was able to identify patients with adverse outcome (death or dialysis) (HR 1.8,  $p<0.001$ ) in the CKD cohort. Kaplan-Meier plot is shown. In multivariate Cox proportional modelling ln uFCR was associated with death or dialysis (HR 1.5,  $p=0.024$ ) when corrected for age, systolic blood pressure, eGFR, presence of diabetes or cardiovascular disease, albumin, cholesterol and ln uPCR.

**Conclusions:** Fetuinuria may be a useful biomarker to identify patients who are at risk of adverse outcomes in CKD.

**SUO015 EFFECTS OF N-3 FATTY ACIDS ON DECLINE OF KIDNEY FUNCTION AFTER MYOCARDIAL INFARCTION: ALPHA OMEGA TRIAL**

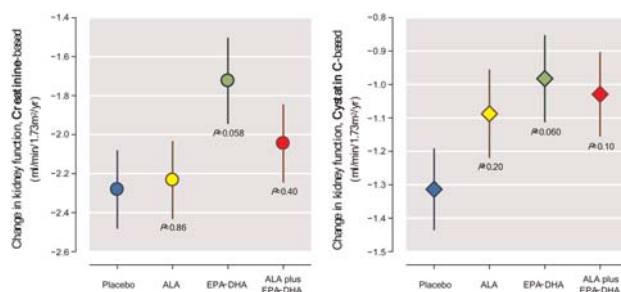
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**Introduction and Aims:** Chronic kidney disease (CKD) is a major risk factor of cardiovascular disease and mortality in both the general population and post-myocardial infarction patients. Kidney function gradually decreases with age ( $\pm 1 \text{ ml}/\text{min}/\text{y}$ ), but in patients with a myocardial infarction (MI) this deterioration is accelerated ( $\pm 2 \text{ ml}/\text{min}/\text{y}$ ). Epidemiological and experimental studies suggest that n-3 fatty acids supplementation may prevent or slow the decline of kidney function. We examined the effect of the marine n-3 fatty acids eicosapentaenoic acid (EPA) and docosahexaenoic (DHA) and of the plant-derived alpha-linolenic acid (ALA) on the decline of kidney function among patients who have had a first myocardial infarction.

**Methods:** In a secondary analysis of the randomized, double blind, placebo-controlled Alpha Omega Trial, 2426 of 4937 (49%) patients (aged 60-80y; 79% men) were included of whom kidney function was measured both at baseline and at 41 months of follow-up. Margarine spreads were used to deliver 400 mg EPA-DHA/d, 2g ALA/d, both EPA-DHA and ALA, or a placebo for 40 months. We measured at baseline and end of follow-up both serum creatinine and Cystatin-C and calculated the estimated GFR (eGFR) from the CKD Epidemiology Collaboration (CKD-EPI) equations. Baseline characteristics were compared among the four treatment groups using ANOVA. The decline of kidney function in the three treatment groups ALA, EPA-DHA and EPA-DHA plus ALA were compared to the placebo group.

**Results:** The 4 randomly assigned groups did not differ in baseline characteristics. Supplementation of ALA, EPA-DHA, EPA and DHA increased plasma cholesteryl ester concentrations of ALA, EPA, and DHA. At baseline kidney function did not differ between the four groups, mean (SD) creatinine and cystatin-C based eGFR were  $76 (20) \text{ ml}/\text{min}/1.73\text{m}^2$  and  $80 (19) \text{ ml}/\text{min}/1.73\text{m}^2$ , respectively. During 41 months of follow-up the yearly decline of creatinine and cystatin-C based eGFR (SE) were in the placebo group (background):  $-2.28 (SE 0.20)$  and  $-1.31 (SE 0.12) \text{ ml}/\text{min}/1.73\text{m}^2/\text{y}$ , respectively. In comparison to the placebo group the mean difference (SE) of the yearly decline of creatinine and cystatin-C based eGFR (SE) were in the ALA supplementation group  $0.05 (SE 0.28, P=0.86)$  and  $0.23 (SE 0.18, P=0.20)$ , in the EPA-DHA group  $0.56 (SE 0.29, P=0.058)$  and  $0.33 (SE 0.18, P=0.060)$  and in the



SUO015 Figure 1.

EPA-DHA plus ALA group 0.24 (SE 0.29,  $P=0.40$ ) and 0.28 (SE 0.18,  $P=0.10$ ; Figure 1).

Decline of kidney function (with error bars indicating SE) after 41 months, according to study group. P-value by three independent samples t-tests compared to the placebo group.

**Conclusions:** In patients who had experienced a MI, low-dose supplementation with n-3 fatty acids did not statistically significantly reduce the decline in kidney function. However, there was a trend for EPA-DHA supplementation to slow down the decline of kidney function. These results should be confirmed in a study supplementing with higher doses of n-3 fatty acids.

**SUO016 A MULTIFACTORIAL INTERVENTION WITH THE AID OF NURSE PRACTITIONERS IMPROVES RENAL OUTCOME IN PATIENTS WITH CHRONIC KIDNEY DISEASE: RESULTS OF THE MASTERPLAN STUDY**

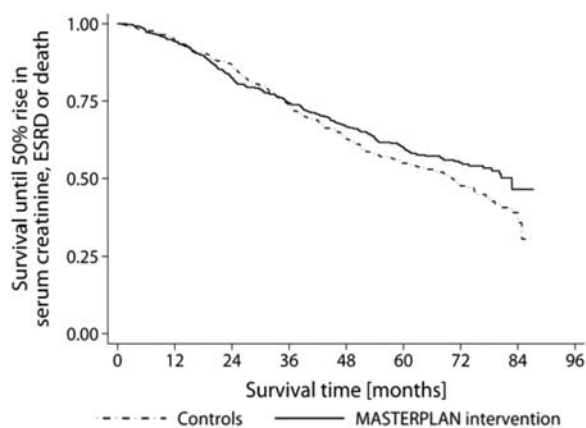
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**Introduction and Aims:** Patients with chronic kidney disease (CKD) are at risk of cardiovascular disease and of progression to end stage renal disease (ESRD). We previously reported that a multifactorial intervention with the aid of nurse practitioners did not reduce cardiovascular events in patients with prevalent CKD (EDTA 2011). In the current analysis we evaluated renal endpoints after extended follow-up.

**Methods:** In MASTERPLAN (Multifactorial Approach and Superior Treatment Efficacy in Renal Patients with the Aid of Nurse practitioners) 788 patients with mild to moderate CKD were randomized to receive nurse practitioner support added to physician care (intervention group) or physician care alone (control group). Patients were followed for a median of 5.75 years. We used a composite endpoint of death, ESRD, or 50% increase of serum creatinine. Survival was compared with adjustment for baseline serum creatinine.

**Results:** 395 patients were randomized to the intervention group, 393 to the control group. Baseline variables were balanced. During follow-up, there were significant differences between both groups in mean blood pressure ( $-3/-2 \text{ mmHg}$ ), proteinuria ( $-0.12 \text{ g}/24\text{u}$ ), LDL cholesterol ( $-0.11 \text{ mmol}/\text{L}$ ), hemoglobin ( $+0.1 \text{ mmol}/\text{L}$ ), the use of anti platelet drugs ( $+9.6\%$ ), statins ( $+4.7\%$ ), vitamin D



SUO016 Figure 1.