Introduction: Advanced glycation end-products (AGEs) are uremic toxins that result from oxidative stress and systemic inflammation and are also ingested in food cooked at high temperatures. Skin autofluorescence (SAF), a measure of tissue AGE accumulation, has been shown to be an independent predictor of mortality in dialysis patients. We have previously reported that several markers of malnutrition are important determinants of increased SAF in a haemodialysis (HD) population, suggesting that correcting malnutrition may decrease SAF. On the other hand, dietary AGE intake has been associated with higher plasma AGEs in some studies and dietary interventions that increase AGE intake may therefore increase SAF. We aimed to investigate whether improvement of nutritional status by providing individualised dietetic advice would result in a decrease in SAF in dialysis patients.

Methods: We studied 27 HD and 1 peritoneal dialysis (PD) patients with malnutrition who received individualised nutritional advice regarding food fortification and oral nutritional supplements according to estimated nutritional requirements. Participants were then followed up for 6 months. SAF was measured by using an Autofluorescence Reader at baseline, 3 and 6 months. Detailed assessment of dietary intake and nutritional status was undertaken at baseline and 6 months, including energy, protein, fat and dietary AGE intake, handgrip strength (HGS), anthropometric measurements and Subjective Global Assessment (SGA). Routine biochemical variables were also measured. We considered it unethical to randomise malnourished patients to no intervention and therefore compared the results with a control group of malnourished dialysis patients (n=41 HD and 8 PD) taken from a recent observational study, who were assessed at the same time points and using the same methodology.

Results: Table 1 shows changes in SAF, biochemical variables and nutritional markers from baseline to 6 months. In the intervention group we observed a significant increase in intake of all nutritional components, including AGEs, as well as SGA and serum albumin. SAF levels did not change significantly over 6 months in the intervention group. In contrast, in the control group there was no increase in nutritional intake, which remained below estimated nutritional requirements, except for AGE intake, which did increase. SGA increased in the control group but other markers of nutrition did not change. SAF increased significantly.

Conclusion: With individualised dietetic advice we observed improvement in dietary intake and markers of nutritional status that was associated with stable SAF levels over 6 months, despite an increase in AGE intake. In contrast, failure to improve dietary intake in a control group was associated with an increase in SAF. This suggests that individualised nutritional support may be effective in preventing the rise in SAF observed in malnourished dialysis patients over time and that the benefits of improving nutritional intake are probably outweighed by any adverse effects of increased AGE intake. Studies of nutritional interventions with larger sample sizes and longer follow-up are needed to test this hypothesis and evaluate the impact on long-term outcomes, including survival.