

carbamylation. It will be further explored whether fecal levels of SCFAs are affected in parallel and could be potential targets to restore gut dysbiosis and uremia.

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# ASSOCIATION BETWEEN CARBAMYLATED ALBUMIN, GUT MICROBIOTA AND THEIR DERIVED METABOLITES IN CHRONIC KIDNEY DISEASE

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**BACKGROUND AND AIMS:** Chronic kidney disease (CKD) is characterized by gut dysbiosis. We recently demonstrated a decrease of short-chain fatty acid (SCFA) producing bacterial species with the progression of CKD. Besides, levels of protein-bound uremic toxins (PBUTs) and post-translational modifications of protein are increased in CKD, both are risk factors for accelerated cardiovascular morbidity and mortality. The link between the gut-kidney axis and protein carbamylation is unclear. The aim of the study was to explore the relation between carbamylated albumin, estimated by the albumin symmetry factor, and plasma levels of PBUTs, fecal levels of SCFAs (ongoing), and the abundance of related gut microbiota in different stages of CKD (1-5).

**METHOD:** The study cohort includes 103 non-dialyzed CKD patients (stages 1-5). Serum proteins were detected by capillary electrophoresis and UV absorbance at 214 nm with the symmetry factor as a marker of albumin carbamylation [the lower the symmetry factor, the more carbamylated albumin]. The quantification of PBUTs and SCFAs in plasma and fecal samples, respectively, using validated UPLC methods.

**RESULTS:** The Pearson correlation coefficient (r) shows a positive correlation between the albumin symmetry factor and the estimated glomerular filtration rate (eGFR) (r=0.3025; p=0.0019).

The albumin symmetry factor correlates positively with the abundance of *Butyrivibrio* spp. (r= 0.3211; p=0.0009), *Faecalibacterium prausnitzii* (r=0.2765; p=0.0047) and *Roseburia* spp. (r=0.2527; p=0.0100) and negatively with the PBUTs, *p*-cresyl sulfate (pCS) (r=-0.2819; p=0.0039), *p*-cresyl glucuronide (pCG) (r=-0.2819; p=0.0039) and indoxyl sulfate (IxS) (r=-0.2650; p=0.0068).

**CONCLUSION:** The decreased abundance of SCFA producing gut bacteria with the progression of CKD can evoke unfavorable conditions in the gut. This can contribute to increased plasma levels of PBUTs potentially (indirectly) playing a role in albumin