**CONCLUSION:** Low SUA levels in chronic HD patients are associated with lower survival and can help identify individuals who are at risk for malnutrition. Further studies should be done to guide possible interventions.

## MO932

# SAME BUT DIFFERENT? COVID-19 METABOLOME IN HEMODIALYSIS

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BACKGROUND AND AIMS: The ongoing coronavirus disease 2019 (COVID-19) pandemic presents a major health and economical challenge. Chronic kidney disease (CKD) patients on haemodialysis (HDp) are at a particularly high risk of severe and fatal courses [1]. The COVID-19 metabolome is distinct [2] and may harbour potential prognostic markers and therapeutic targets. However, the metabolome in SARS-CoV-2 infected HDp remains unstudied.

**METHOD:** Untargeted and targeted metabolomics via high-performance liquid chromatography and tandem mass spectrometry of EDTA-plasma were performed in 39 individuals without CKD (non-HDp) and 41 HDp. Individuals were further grouped according to COVID-19 disease status and severity: negative, asymptomatic/mild (no or mild symptoms) and moderate/severe (requiring hospitalization).

**RESULTS:** Principal component analysis shows a clear separation of non-HDp and HDp alongside PC2, independent of COVID-19. Non-HDp with moderate/severe courses are shifted to the right of PC1. The influence of COVID-19 is less pronounced in HDp (Figure 1). Free fatty acids are main contributors to PC1, whereas PC2 reflects the complex metabolic disarray as seen in uraemia (Table 1).

**CONCLUSION:** Our data underlines the fundamental metabolic differences between non-HDp and HDp [3]. Furthermore, moderate/severe COVID-19 leads to a distinct metabolite signature in non-HDp. This effect is less pronounced in HDp, which could be indicative of a decreased capability to adapt to infectious challenges. In summary, these findings warrant caution when extrapolating COVID-19 metabolomics from otherwise healthy individuals, as the validity of prognostic markers and/or effectiveness of therapeutic approaches based on the metabolome may be limited in HDp.

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#### Table 1. Top 10 metabolites contributing to PC1 and PC2

PC1	PC2
FFA C16:2 (Palmitolinoleic acid)	Cytidine
FFA C20:2 (Eicosadienoic acid)	Creatinine
FFA C17:1 (Heptadecenoic acid)	Gluconic acid
FFA C16:0 (Palmitic acid)	Glucuronic acid
FFA C19:1 (Nonadecenoic acid)	Orotidine
FFA C20:1 (Eicosenoic acid)	Methylhistidine
FFA C22:4 (Adrenic acid)	ADMA
FFA C18:1 (Oleic acid)	Tryptophan
FFA C22:5 (Docosapentaenoic acid)	Trehalose
FFA C16:3 (Hiragonic acid)	Lactose

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### MO933 ARE CHRONIC INFLAMMATION AND CKD-MBD RISK FACTORS OF MORTALITY IN HEMODIALYSIS PATIENTS?

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**BACKGROUND AND AIMS:** Haemodialysis patients are known to be susceptible to a wide range of early and long-term complications such as chronic inflammation, infections, malnutrition, mineral bone disorders (CKD-MBD) and cardiovascular disease that significantly affect the incidence of mortality. The aim of this study is to assess the presence of traditional risk factors for mortality such as diabetes mellitus or cardiovascular disease on one hand and non-traditional risk factors on the other hand (chronic inflammation, CKD-MBD).

**METHOD:** We conducted a single-centre study that included 63 CKD G5D patients (haemodialysis for 1–5 years) followed up for 48 months. All patients have been assessed at baseline, regarding cardiovascular disease (medical history, echocardiography and ECG), we performed using standard methods blood biochemistry, complete blood count and markers of inflammation (CRP, IL-6) and markers of CKD-MBD (sKlotho, iPTH, serum calcium and serum phosphorus). **RESULTS:** After 24 months of follow-up, we found a mortality rate of 22.23%, while after 48 months, the mortality rate was of 50.73%.



**FIGURE 1**: Principal component analysis of non-HDp and HDp without COVID-19 and with asymptomatic/mild or moderate/severe COVID-19.