Primary endpoints were total oral/enteral caloric intake, abdominal distension and pain on POD 7. Secondary endpoints included sequential changes of total oral/enteral caloric intake after LT, numeric rating scales for abdominal distention and pain, portal venous flow and speed to the graft and so on.
Results: A total of 104 patients (DKT, n = 55; placebo, n = 49) were included and evaluated in the statistical analysis. There were no significant differences between the two groups in terms of primary endpoints. However, postoperative total oral/enteral calorie intake was significantly accelerated in the DKT group than in the placebo group ($p = 0.023$). Moreover, portal venous flow (POD 10, 14) and speed (POD 14) were significantly higher in the DKT group compared with the placebo group ($p = 0.047$, $p = 0.025$, $p = 0.014$, respectively).
Conclusion: Postoperative administration of DKT effectively enhances total oral/enteral calorie intake after LT and would contribute to performance of ERAS.

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OR54
EFFECTS OF FIBERS DERIVED FROM CEREALS IN THE MODULATION OF INFLAMMATION INDUCED BY AN OVER-CONSUMPTION OF FRUCTOSE: IMPLICATION OF THE GUT MICROBIOTA

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Rationale: The prevalence of non-alcoholic fatty liver disease (NAFLD) has increased in parallel to a rapid rise in fructose consumption. In this study we evaluated the effects of two wheat fractions characterized by different particle size on hepatic inflammation induced by fructose, in relationship with their potential effect on the gut microbiota.
Methods: Mice were divided in 4 groups (n = 6/group) receiving a control diet (CT), a CT diet plus fructose 30% w/v in the drinking water (F), a CT diet supplemented with 5% of crude wheat bran plus 30% fructose in water (normal particle size 1,690 µm, F + WB; WB with small particle size 150 µm, F + WBs) for 8 weeks.
Results: Fructose supplementation significantly increased the expression -at the mRNA level- of key inflammatory genes (MCP-1, TNFα, TLR2) and macrophage-related markers (CD11c) in the liver compared to the CT group. Neither fructose nor WB fractions significantly modified the expression of markers of inflammation, intestinal immune function or barrier function in the ileum and colon. WBs was the sole fraction to reduce hepatic (mRNA) and systemic (protein-multiplex immunoassay) markers of inflammation (IL1β, IL6, IL10, IFNγ, MCP1, TNFα). Moreover, gut microbiota analyses (qPCR of 16SrDNA) revealed that both wheat bran fractions differently affected the gut microbiota. WB increased *Akkermansia muciniphila* caecal level, whereas only WBs significantly counteracted the increase in Enterobacteriaceae induced by fructose. Enterobacteriaceae were positively correlated ($r = 0.55$ $p = 0.008$) with the hepatic expression of MCP1.
Conclusion: Our findings suggest that the different effect of WB and WBs on hepatic inflammation could be attributed to the different impact exerted on the gut microbiota. It is worth noticing that, reducing Enterobacteriaceae levels may reflect the anti-inflammatory properties of WBs.

Disclosure of Interest: None declared.

OR55
DISTURBED ENTEROHEPATIC BILE SALT SIGNALING IN INTENSIVE CARE PATIENTS WITH DIARRHEA

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Rationale: Diarrhea occurs in over 60% of critically ill patients admitted to the intensive care unit for 7 days or more and is an independent risk factor for cholestatic liver injury. We hypothesize that diarrhea disturbs the enterohepatic circulation of bile salts, potentially contributing to the development of liver injury.
Methods: We investigated plasma levels of bile salts, FGF19 (a bile salt-induced gut hormone controlling hepatic bile salt synthesis) and C4 (a marker of bile salt synthesis) in critically ill patients. Following inclusion, allocation to the diarrhea group (N = 12) or no diarrhea group (N = 18) was based on 24 hour fecal production ≥350 mg/day. Data were tested for normality and are presented as median [IQR] or mean ± SD. Mann-Whitney or t-tests were used as appropriate to test for statistical significance.
Results: Decreased FGF-19 levels were observed in the diarrhea group (0.20 ± 0.12 vs. 0.29 ± 0.10 ng/mL, $p = 0.03$) indicating disturbed enterohepatic signaling. Plasma bile salt levels were increased in patients with diarrhea (9.8 [5.0–23.9] vs. 4.5 [2.9–7.4] µmol/L, $p = 0.01$]), while C4 levels were not different between the two groups (6.8 ± 4.0 vs. 6.4 ± 3.6 ng/ml, $p = 0.77$). Bilirubin, alkaline phosphatase (ALP) and gamma-GT levels also were not different between the two groups.
Conclusion: Diarrhea in critical illness disturbs the normal enterohepatic circulation of bile salts, as evidenced by reduced plasma levels of FGF-19. There was no anticipated elevation of plasma C4 levels, suggesting that hepatic bile salt synthesis is not increased in patients with diarrhea. Furthermore, increased systemic bile salt levels suggest that hepatic uptake is affected in critically ill patients with diarrhea. The consequences of these disturbances in bile salt signaling and their relation gut function and cholestatic liver injury during critical illness requires further investigation.

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