of underweight (7/9 (78%), healthy (28/46 (61%)), overweight (14/23 (61%)) and obese (6/12 (50%)) p=0.642). Patients with >5% weight loss (13/101 (13%)) or suboptimal HGS (56/101 (55%)) were significantly more likely to be at nutrition risk using IBD-NST, MUST and SGA. Patients with suboptimal MAMC (10/66 (15%)) were not significantly more likely to be at nutrition risk using IBD-NST or MUST. Conclusion The IBD-NST identifies more patients with high nutrition risk and places less importance on BMI than SGA or MUST. Finally, we confirm that BMI is a poor indicator of HGS in IBD supporting a reduced importance in identifying nutrition risk. Further work is underway to test the repeatability the IBD-NST.

**CONCLUSIONS** Serum levels of galectins-1 and -3 were significantly higher in IBD patients compared to healthy people and this difference however could not separate active from inactive IBD.

**PHT-137** PHOSPHATE STUDIES: IMPORTANT CHANGES IN PHOSPHATE HOMEOSTASIS AND BONE METABOLISM AFTER IV IRON

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**Introductions** Iron deficiency anaemia (IDA) is the most common extraintestinal complication of inflammatory bowel disease (IBD), and IV iron is commonly used to treat IBD in IBD. Of the two commonly used IV iron preparations, ferric carboxymaltose (FCM) and iron isomaltoside (IIM), there is evidence that FCM can cause hypophosphatemia due to FGF23 mediated renal phosphate wasting, which has been associated with osteomalacia; the comparative effects of IIM are unknown.

**Methods** In two identical, open-label trials, 245 adults with IDA randomly received (1:1) IIM (1×1000 mg) or FCM (2×750 mg; 1 week apart). We compared the incidence, severity and duration of hypophosphatemia, and effects on fractional phosphate urinary excretion (FEPO4), FGF23, parathyroid hormone (PTH), vitamin D (VitD), and bone turnover biomarkers in blood and urine.

**Results** In a pooled analysis, the incidence of hypophosphatemia <2 mg/dL was markedly higher in the FCM group (74.4% vs. 8.0%, p<0.0001). Hypophosphatemia persisted at day 35 in 43.0% of FCM-treated patients compared to 0.9% of IIM-treated patients (p<0.0001). Severe hypophosphatemia ≤1 mg/dL occurred in 11.3% of FCM patients compared to 0.0% of IIM-treated patients (p<0.0001). FCM infusion significantly increased intact FGF23 compared to IIM (p<0.0001): FGF23 after FCM rose from 49.9 pg/mL at baseline to 149.5 pg/mL one day post-infusion; and to 327.9 pg/mL by day 8. The corresponding figures for IIM were 59.9 pg/mL at baseline, 58.3 pg/mL by day 1 and 66.9 pg/mL by day 8. Compared to treatment with IIM, FCM significantly: increased FEPO4; decreased serum 1,25-(OH)2 VitD; decreased ionized calcium; and increased PTH, which persisted through day 35. Post-FCM changes were accompanied by significant increases in total and bone specific alkaline phosphatase that persisted through day 35. Correction of IIA was comparable between the two treatments. Significantly fewer adverse drug reactions occurred with IIM than with FCM. Serious or severe hypersensitivity reactions occurred in 0.8% in the IIM group and 1.7% in the FCM group.

**Conclusion** Compared to IIM, FCM-induced high rates of FGF23-mediated hypophosphatemia, which was frequently...
severe and often persisted for >35 days. FCM, but not IIM, induced changes in vitD and calcium homeostasis that triggered secondary hyperparathyroidism, which likely contributed to persistence of hypophosphatemia. Alteration of bone metabolism after FCM administration may be of great clinical significance particularly for IBD patients who often have low vit D and regularly receive iv iron.

**Abstract PTH138 Figure 1** Time trend analysis of incidence rates (per 1,000 pt.-yrs.) of IBD complications in PSC liver transplantation