times, serology for Hepatitis B, C and autoimmune and Fibromax. Clinical parameters such as body mass index (BMI), type II diabetes mellitus (DMII), systemic arterial hypertension (SAH), presence of ascites, alcohol consumption (gr / week) and endoscopic findings (esophageal varices) were also analyzed.

Results: Significative difference was observed in serum ferritin levels between men and women [353.0 ng/mL (170.5–747.5 ng/mL) vs 108.3 ng/mL (55.8–253.5 ng/mL), $p < 0.0001$], as well as in serum ferritin levels between women with and without ascites, in men with different fibrosis stage (FibroTest) and necroinflammatory activity (ActiTest) (Figure 1). A poor but significant correlation was observed between serum ferritin and age, erythrocytes, MCV, MCH, uric acid, direct bilirubin, albumin and HDL cholesterol in women and alcohol consumption, uric acid, ALT and AST in men. All other evaluated clinical parameters and biomarkers showed no significant difference.

Conclusions: An association was observed between the degree of fibrosis and serum ferritin and necroinflammatory activity in men, as well as between ferritin and ascites in women. A poor correlation was observed between serum ferritin levels and the analyzed chemical biomarkers.

Conflicts of interest: The authors have no conflicts of interest to declare.

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Moringa oleífera decreases insulin resistance, novo lipogenesis and modifies the expression of mirnas in a non-alcoholic esteatohepatitis model

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Background and aim: In Mexico there is a high prevalence of non-alcoholic steatohepatitis (NASH) and liver diseases are the fourth leading cause of death. NASH is characterized by hepatocyte ballooning, inflammation, and steatosis. Moringa Oleifera (MO) extracts have been shown to have hypoglycemic, anti-inflammatory and antioxidant effects. The aim was to evaluate in a NASH model the effect of the aqueous extract of MO the gene and protein expression of molecules involved in steatosis and liver inflammation and on miRNAs involved in the development of NASH.

Material and methods: Male C57BL / 6J mice were fed a high fat diet (HF, 60% lipid, 42gr / L sugar in water) for 16 weeks. The administered dose of the MO extract was 300 and 500 mg / Kg / day from week 9 to 16. The serum levels of adipokines were measured, the HOMA-IR was calculated; In the liver miR-21a-5p, miR-103-3p, miR-34a-5p and IL1β, IL-6, TNFα, SREBP1, FASN and DAGT2 were evaluated by qRT-PCR and SREBP1 by Western Blot. The transcriptome was evaluated by microarrays. Inflammation, reactivity to αSMA and fibrosis were analyzed in histological sections. Quantitative variables were analyzed with ANOVA, Tukey for parametric data, Mann-Whitney U for non-parametric data. Approved by the CUCS Ethics, Research and Biosafety Committees: 1937.

Results: Moringa treatment reduced serum insulin, PAI-1, leptin, and resistin levels. In liver: IL1β, IL6, TNFα, SREBP1c, FAS, and DAGT2 mRNAs decreased; SREBP1 protein decreased. Expression of mir-21a, mir-103, and mir-34a were reduced. In the transcriptome, the mRNAs involved in the response to DNA damage and stress of the endoplasmic reticulum, lipid biosynthesis, and extracellular matrix synthesis were underexpressed. In liver histologies, the number of inflammatory nodules and the presence of αSMA and fibrosis decreased.

Conclusions: MO supplementation decreased serum adipokine levels; as well as the mRNAs of proinflammatory cytokines and lipogenic genes in liver. The histological quantification of MEC, collagen, inflammatory nodules and αSMA decreased; miRNAs evaluated were modified. Moringa extract showed anti-inflammatory, antifibrogenic and antilipogenic effect in a NASH model.

Conflicts of interest: The authors have no conflicts of interest to declare.

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Prolonged-release pirfenidone decreases hepatic miRNAs expression in a NAFLD/NASH experimental model

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Background and aim: Nonalcoholic steatohepatitis (NASH) is featured by lipid accumulation, inflammation, and fibrosis. miRNAs are small non-coding RNAs that participate in post-transcriptional genetic regulations and are involved in various pathologies such as NASH. The drug pirfenidone is an antifibrotic, anti-inflammatory and antioxidant agent. Aim: To evaluate the effect of prolonged-release pirfenidone on histological parameters, activation of hepatic stellar cells, expression of hepatic miRNAs and target genes in an experimental model of NAFLD/NASH.

Material and methods: Male C57BL/6J mice were fed a high fat diet (HFD, 60% lipids, 42gr/L sugar in water) for 16 weeks. Prolonged-release pirfenidone (~300 mg/kg/d, PR-PFD) was administered in food from the eighth week to the end of the protocol. α-SMA immunohistochemistry and hematoxylin-eosin, Masson's trichrome and Sirius red staining were made. Hepatic expression of miR-21a-5p, miR-103-3p, miR-34a-5p and IL-1β, TNFα, COL1A1, and SREBP1 genes was determined by qRT-PCR and the transcriptome by microarrays. Statistical significance was determined for parametric data with one-way analysis of variance and Tukey's or Bonferroni post hoc test, and Kruskal-Wallis and Mann-Whitney *U* test for nonparametric data (Graph Prism 6.0). Ethics Committee registration number: CI00518.

Results: Animals treated with PR-PFD have a decrease in inflammatory nodules, macrosteatosis, fibrosis, collagen and activation of hepatic stellar cells. PR-PFD reduced hepatic expression of miR-21a-5p, miR-34a-5p and miR-103-3p expression showed a tendency of decrease compared to HFD group. PR-PFD decreased IL-1β, TNFα, COL1A1, and SREBP1 expression. Transcriptome analysis showed that 36 genes that participate in lipid transport and antiox-