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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10

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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11

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-

idant activity were overexpressed in the treated group compared to HFD group. On the contrary, 52 genes involved in lipid and collagen biosynthesis and inflammatory response were downregulated.

Conclusions: Prolonged-release pirfenidone decreased miR-21a-5p expression, miR-34a-5p and miR-103-3p expression showed a tendency to decrease. PR-PFD exhibited an anti-steatogenic, anti-inflammatory and anti-fibrotic effect in the experimental model of NAFLD/NASH.

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

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12

Clinical and epidemiological characteristics of patients with autoimmune hepatitis in a center of Northeast Mexico

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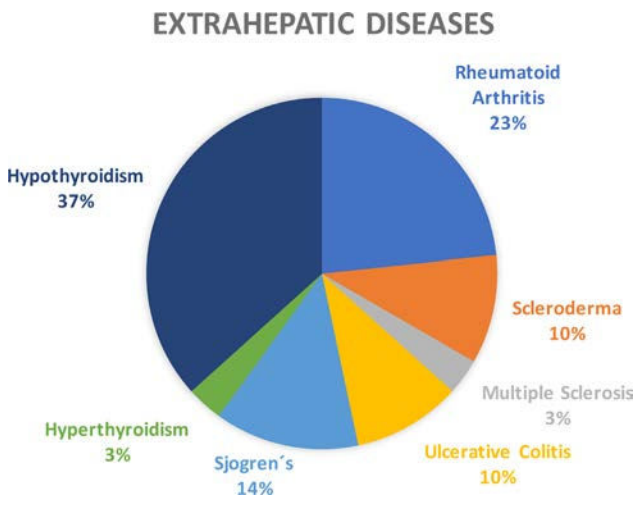
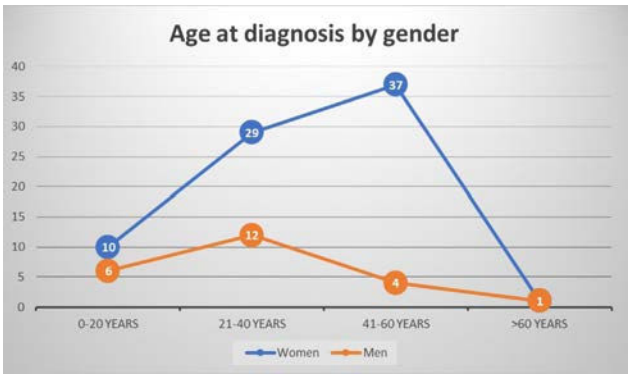
Background and aim: Autoimmune hepatitis (AIH) is a very heterogeneous disease with an impact on the morbidity and mortality of patients, which affects all ages, genders, and ethnic groups. It can present either asymptomatically, such as acute hepatitis, cirrhosis, or acute liver failure. Aim: To describe the demographic, biochemical and clinical characteristics of patients with HAI, due to the few epidemiological data that exists in Latin America.

Material and methods: A retrospective cohort study was conducted that included patients with HAI attended at the Northeast National Medical Center of the IMSS (Monterrey, Nuevo León). The information was collected from the digital file between March 01, 2019 to April 01, 2020.

Results: A total of 100 patients, 77% female, with an average age at diagnosis of 37 years were included (Graph 1). In the presentation form, 38% were cirrhotic at diagnosis, 2% debuted with acute liver failure, 16% with acute hepatitis, and 44% with asymptomatic abnormal liver tests. Regarding autoantibodies, 75% had positive antinuclear antibodies (ANAs) or smooth muscle antibodies (ASMA). 25% associated autoimmune extrahepatic diseases (Graph 2), and 46% fulfilled criteria for Overlap Syndrome, mostly primary biliary cholangitis (CBP). Regarding treatment, 40% were considered refractory to conventional therapy, meriting the use of



Graph 1. Age at diagnosis by gender ($N = 100$).



Graph 2. Associated extrahepatic diseases.

other immunosuppressors (mycophenolic acid and tacrolimus). Of the total of patients, 32% had received Orthotopic Liver Transplantation (THO).

Conclusions: Autoimmune hepatitis in our population behaves similarly to that described in the world literature, with a greater association to overlap syndrome and refractory treatment, perhaps because it is a reference center.

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

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13

Pirfenidone induces epigenetic changes modulating the activity of the ppargamma-SIRT1-DNMT1 axis in hepatic stellate cells

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Background and aim: The role of epigenetic changes in liver diseases has been described through abundant experimental evidence, highlighting the de-acetylation in residue lysine 9 of histone H3 (H3K9) regulated by SIRT1 (NAD-dependent deacetylase), and methylation of promoters of genes involved in fibrogenic response regulated by DNA-Methyl transferase 1 (DNMT1). Our research group characterized the regulation of pirfenidone (PFD) on PPARalpha/gamma-SIRT1 axis in two experimental animal models: (1) NASH and (2) hepatocarcinoma. The objective of this work was to characterize the changes in the acetylation/de-acetylation of H3K9 induced by PFD treatment in hepatic stellate cells (HSCs), in addition to analyzing the global methylation patterns

Material and methods: HSCs were treated with PFD (500 μM), SIRT172 activator (20 μM) and inhibitor EX527 (80 μM) of SIRT1 for 24 hrs. Subsequently, changes in the expression of SIRT1 and DNMT1 were analyzed by western blot. Immunofluorescence was carried out using markers that detect H3K9 acetylation and global methylation (5MeC; 5Metyl-Cytosine), images were captured with a confocal microscope in order to visualize the cellular co-location of proteins.

Results: The western blot shows that the treatments with PFD and SIRT1720 treatments increase the expression of SIRT1 and DNMT1 proteins, while EX527 reduces them. Immunofluorescence demonstrated that PFD and SIRT1720 decrease the acetylation of H3K9, but increase overall methylation; contrarily, treatment with