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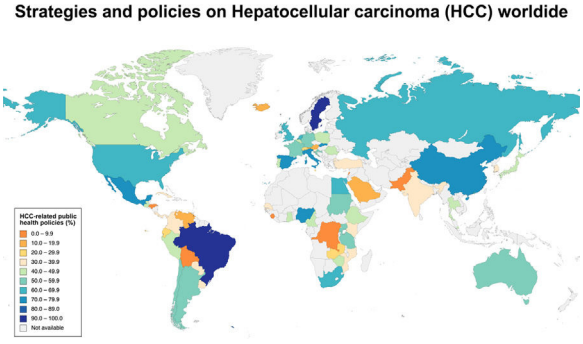
Introduction and Objectives: Hepatocellular carcinoma (HCC) is the third most common cause of cancer-related deaths worldwide. We aimed to explore HCC-related population-wide public health policies (PHP) worldwide.

Materials and Methods: We conducted a 43-item survey about HCC: policies and civil society (18 questions), clinical guidelines (5 questions), epidemiology (7 questions), and care management (13 questions). The survey was completed electronically (2022–2023). Data were collected in a spreadsheet, revised by two independent reviewers, and verified with governmental institutions, regulatory agencies, scientific societies, and scientific publications. We classified policies into eight dimensions, including criteria for low, moderate, and strong PHP establishment. We estimated an index using multiple correspondence analysis.

Results: We obtained 134 responses from 66 countries/territories (Africa N=16, the Americas N=18, Asia N=10, Europe N=21, and Oceania N=1). The median index was 43.7 [IQR: 30.9–59.3]. The lower scores were observed in Sierra Leone (0), Lebanon (5.5), and Pakistan (5.5), while Italy (79.7), Brazil (94.1), and Sweden (100) obtained the highest scores (Figure). In particular, 46 (69.7%) countries had a written national cancer strategy or action plan, but only 5 (7.6%) had a specific written national strategy or action plan on HCC. Thirty-two (48.5%) countries had national clinical practice guidelines on HCC and 54 (81.8%) countries had a national disease registry that included HCC. The most common strategies for staging HCC were Barcelona Clinic Liver Cancer (BCLC)(85%) and TNM classification (10%). The survey reflects important differences in the availability of treatments, including surgery (98.4%), tyrosine kinase inhibitors (95.1%), chemoembolization (85.2%), radiofrequency or alcohol ablation (82%), immunotherapy plus anti-VEGF (82%), liver transplant (74.2%), stereotactic body radiation therapy (42.6%), and radioembolization (36.4%).

Conclusions: The existence of PHP on HCC is insufficient worldwide. The most common strategy for staging is BCLC, but there are important differences in treatment availability across countries, especially regarding curative therapies.



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O-14 NON-ALCOHOLIC FATTY LIVER DISEASE IS INFLUENCED BY THE INTERACTION OF HELICOBACTER PYLORI INFECTION AND G-ALLELE OF PNPLA3

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Introduction and Objectives: The pathophysiology of NAFLD is only partially unrevealed; it is considered as a multifactorial disorder, attributed to multiple, parallel "hits," both genetic and environmental. It has been described that the single nucleotide polymorphism at rs738409 in the PNPLA3 gene is strongly associated with hepatic steatosis and its progression. Conversely, H. pylori infection has been related to metabolic syndrome, type-2 diabetes mellitus, and dyslipidemia, which are known risk factors for NAFLD. However, the evaluation of Infection and the rs738409 polymorphism in the PNPLA3 gene has not been explored.

Materials and Methods: this is a preliminary report of a prospective multicenter study from December 2020 to June 2021 in northeastern Argentina. 76 dyspeptic adult patients who fulfilled the ROME-IV criteria and underwent gastroscopy, of which 69 were included. The presence of H. pylori was determined by gastric histology. Biochemical and clinical parameters were recorded. NAFLD was defined by liver ultrasonography. The PNPLA3 gene was analyzed by PCR-RFLP in rs738409.

Results: The prevalence of NAFLD was 45% (31/69), with Hpyl+ 48% (17/36) and Hpyl- 42% (14/33) (p: ns). The variables significantly associated with NAFLD were BMI, dyslipidemia, Diabetes/prediabetes, presence of the G allele of PNPLA3, and the GG genotype. In the multivariate analysis, BMI (OR 1.63 95%CI 1.22-2.19) and the G-allele of PNPLA3 (OR 7.35 95%CI 1.34-40) were independently associated with NAFLD. When subjects with NAFLD were analyzed, the interaction between Hpyl and PNPLA3 allele-G was significantly associated with NAFLD (65%) and increased risk of liver fibrosis (FIB-4 > 1.3 41%).

Conclusions: the presence of NAFLD was associated with BMI and G-allele of PNPLA3. The combination of Hpyl infection and the G-allele of PNPLA3 were associated with NAFLD and risk of fibrosis (FIB-4)

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O-15 MARESIN1 REVERSES CHRONIC LIVER FIBROSIS AND IMPROVES REGENERATION

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Introduction and Objectives: Hepatic fibrosis (HF) is characterized by the progressive accumulation of extracellular matrix (ECM), which destroys the physiological architecture of the liver. Pathologically, chronic liver diseases lead to damaged hepatocytes and

infiltration of immune cells that activate collagen-producing hepatic stellate cells (HSCs), leading to excessive ECM production, and causing uncontrolled scarring. Maresin1 is a derivative of -3 docosahexaenoic acid (DHA), which has been shown to have pro-resolving and anti-inflammatory effects in various organs like those observed for DHA. This study aimed Mar1+DHA supplementation would prevent the development of fibrosis and promote regeneration in an animal model of chronic liver damage.

Materials and Methods: FH was induced in Sprague-Dawley rats by injections of diethylnitrosamine (DEN, 50 mg/kg) and treated with MaR1 (4ng/g) and/or DHA (375 mg/kg) for five weeks. Transaminases, liver histology, and proteins were analyzed by western blot.

Results: the DHA+ MaR1 group showed a greater positive response (significant) than MaR1 in terms of normalization of AST and ALT levels, and architecture of the liver. Reducing inflammation and necrosis. Furthermore, both MaR1 and DHA reduced the levels of TGF-, its receptor TGFRII, and TIMP1, increasing MMP1. Results that coincide with the quantification of type I collagen fibers in tissue. On the other hand, they would promote liver regeneration by increasing Cyclin D1.

Conclusions: Both MaR1 and MaR1/DHA improve regeneration and DEN-induced liver fibrosis parameters, promoting regeneration and acting as an antifibrotic agent. Results that open the possibility that MaR1/DHA are potential therapeutic agents in fibrosis and other liver pathologies.

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O-16 DIFFERENCES IN HEPATOCARCINOMA IN PATIENTS WITH CIRRHOSIS DUE TO NONALCOHOLIC FATTY LIVER DISEASE (NAFLD) VS. OTHER ETIOLOGIES

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Introduction and Objectives: Non-alcoholic fatty liver disease (NAFLD) is the fastest growing cause of hepatocarcinoma (HCC) in the USA and parts of Europe and is expected to increase exponentially in parallel with the global obesity epidemic. This study aimed to determine the differences in the characteristics of HCC in patients with NASH vs. other etiologies.

Materials and Methods: Observational, descriptive study of patients with a diagnosis of HCC presented to the HCC Committee and included in the local Research Registry between March and December 2022. Demographic, clinical and tumor variables at HCC diagnosis were collected. Survival was assessed based on death certificates. Chi2 was calculated considering $p<0.05$ significant.

Results: During the study period, 143 patients were presented to the HCC Committee; 109 of them fulfilled the criteria for this study, 66 with NAFLD etiology (61%) and 43 with cirrhosis due to other etiologies (39%). When comparing sociodemographic and clinical variables in relation to cirrhosis etiology, higher average age (67 vs. 63; p=0.027), lower frequency of men (51% vs. 73%, p=0.026), lower Child-Pugh (Child-Pugh A 55% vs. 40%, p=0.033) and lower average Meld-Na (10.5 vs. 12, p=0.075) were observed in the NAFLD groups vs. other etiologies. No differences were observed in laboratory analysis at HCC diagnosis. There were also no differences in tumor characteristics or recommended therapies. Survival was higher in the NAFLD group, although it was not significant (76% vs. 65%, p=0.228).

Conclusions: HCC in patients with NAFLD cirrhosis occurs more frequently in women, older patients and with better overall probably related to the severity of the chronic liver disease. No differences were observed in tumor characteristics or suggested treatment options, with loco-regional therapy being the most indicated (45% of all patients).

	Total N = 109 (%)	NAFLD N = 66 (%)	Other etiologies N = 43 (%)	P value
HCC characteristics				
N° of lesions Single Multiple	 53 (49) 56 (51)	 32 (48) 34 (52)	 21 (49) 22 (51)	 0.971
Size of the largest lesion (mm)	37 (11 – 170)	36 (11 – 140)	45 (11 – 170)	0.267
Sum of lesions size (mm)	51 (11 – 190)	51 (12 – 190)	50 (11 – 175)	0.857
Portal vein involvement	16 (15)	6 (9)	10 (23)	0.053
Extrahepatic disease	5 (5)	4 (6)	2 (5)	1
HCC treatment suggestion				
Loco-regional therapy Surgery Liver transplant Systemic chemotherapy Palliative care Image and clinical follow-up	49 (45) 16 (15) 19 (17) 14 (13) 5 (5) 6 (6)	32 (48) 9 (14) 11 (17) 7 (11) 3 (5) 4 (6)	17 (40) 7 (16) 8 (19) 7 (16) 2 (5) 2 (5)	0.432 0.784 0.801 0.397 1 1
Survival				
Mortality	31 (28)	16 (24)	15 (35)	0.228

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O-17 NUTRICIONAL AND PHYSICAL THERAPHY IMPROVES LIVER FRAILTY INDEX IN LISTED PATIENTS WITH CIRRHOSIS: RANDOMIZED CONTROLLED TRIAL. INTERIM ANALYSIS

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Introduction and Objectives: Frailty is independently associated with a lower survival in cirrhotic patients. Liver Frailty Index (LFI) determine frailty in listed patients predicting survival. This study aimed to evaluate the effect of a physical and nutritional therapy (intervention group) over LFI compared with a physical and nutritional counseling (control group).

Materials and Methods: Patients were recruited and randomized to an intervention group or a control group. Patients were followed for 12 weeks with evaluations every 4 weeks. We compared LFI and LFI at different time points between both groups during the follow-up.

Results: 46 patients were recruited, 27 of them in control group and 19 of them in the intervention group. 50% were women and the most common etiologies were metabolic associated fatty liver disease (37%) and alcoholic liver disease (19.6%), primary biliary cholangitis (6.52%), autoimmune hepatitis (6.52%) and hepatitis C virus (2.17%).