**Materials and Methods:** male C57bl/c mice were subjected to liver fibrosis by i.p diethylnitrosamine (DEN) 50 mg/kg twice a week and treated with MaR1 (4ng/g) for 9 weeks. The liver macrophages were isolated: real-time qPCR flow and cytometry were made. In addition, MaR1 was administered to healthy mice to observe the role MaR1 on hepatic macrophage populations.

**Results:** The administration of MaR1 modifies the Kupffer cells populations, generating an increase in the subpopulations of M2 F4/80+CD11b-CD206+ and F4/80intCD11b+CD206+, with a decrease in the CD86+CD11c+, both in the fibrosis as in healthy mice. This was accompanied by an increase in IL-10 cytokines and a fall in TNF-a values.

**Conclusions:** Taken together, these results indicate that Mar1 switches the Kupffer cells towards an anti-inflammatory, restorative and resolving state, acting as a hepatoprotective agent.

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## O-12 HELICOBACTER PYLORI VIRULENCE GENES ARE ASSOCIATED WITH NAFLD SEVERITY: A CROSS SECTIONAL STUDY IN DYSPEPTIC PATIENTS

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**Introduction and Objectives:** Recent studies have suggested an association between Helicobacter pylori (Hpyl) and non-alcoholic fatty liver disease (NAFLD). The current study aimed to examine the association of Hpyl virulence genes and NAFLD in dyspeptic patients.

**Materials and Methods:** prospective multicenter study from 2019 to 2022 in northeast Argentina. We evaluated 386 dyspeptic patients who fulfilled the ROME III criteria and underwent gastroscopy. NAFLD was defined by ultrasound in the absence of other known liver diseases. cagA, vacAs1/s2, vacAm1/m2 were analyzed by PCR.

**Results:** The prevalence of NAFLD was 41% (156/383), no association with Hpyl status was observed. In NAFLD subjects, Hpyl+ showed higher AST (Hpyl+: 30 (21) UI/mL vs. Hpyl-: 22 (13) UI/mL, p:0,001), ALT (Hpyl+: 32 (25) UI/mL vs. Hpyl-: 25 (17) UI/mL, p: 0,0018) and FIB-4 (Hpyl+: 1,3 (1) vs. Hpyl-: 0.99 (0.6), p: 0,009). Indeed, Hpyl+ was associated with FIB-4>1,3 (Hpyl+: 54% vs. Hpyl-: 27%, p: 0,009). cagA and vacAm1 were associated with higher ALT (cagA 40 (23) UI/mL, p:0,003, vacAm1 44 (24) UI/mL, p:0,004). Also, higher FIB-4 values were observed with cagA (1,3 (0,9), p: 0,02) and vacAm1 (1.34 (0.8), p: 0,001) with more proportion of patients with FIB-4>1,3 with cagA 54% (p: 0,008) and vacAm1 55% (p: 0.007). The allelic combination vacAs1/m1+cagA showed higher AST (34 (22) UI/mL, p: 0,001), ALT (44 (24) UI/mL, p: 0,004) Fib-4 (1,34 (0,8), p:0.001) with significantly more proportion with FIB-4>1,3 (62%, p: 0,019).

**Conclusions:** In NAFLD/dyspeptic patients, Hpyl infection was associated with markers of liver injury and fibrosis. cag-A, vacAm1 strains and the allelic combination vacAs1/m1/cagA were associated with higher ALT and FIB-4.

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## O-13 SUB-OPTIMAL GLOBAL PUBLIC HEALTH POLICIES AND STRATEGIES TO TACKLE HEPATOCELLULAR CARCINOMA

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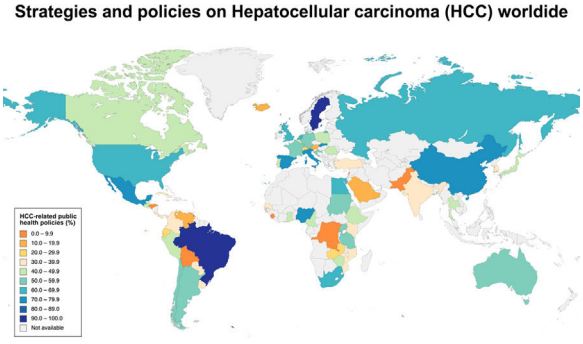
**Introduction and Objectives:** Hepatocellular carcinoma (HCC) is the third most common cause of cancer-related deaths worldwide. We aimed to explore HCC-related population-wide public health policies (PHP) worldwide.

**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