Table 1
Leukocyte profile of patients with different types of alcohol-related liver damage

	Control (n=300)	Alcoholism (n=102)	Cirrhosis (n=121)	Alcoholic Hepatitis (n=47)
Leukocytes (miles/mm^3)	6.7 (7.7, 5.8)	6.7 (7.8, 5.8)	6.1 (9, 4.4)	15 (20, 11) c*,e*,f*
Lymphocytes (miles/mm^3)	2.2 (2.7, 1.8)	1.9 (2.3, 1.7) a€	1.4 (2, 1)d*	1.7 (4.9, 1)
Monocytes (miles/mm^3)	0.4 (0.5, 0.27)	0.4 (0.5, 0.3) a€	0.5 (0.7, 0.4)	0.8 (1.2, 0.5) c+,e+,f€
Neutrophils (miles/mm^3)	3.6 (4.7, 2.9)	4.1 (5, 3.3)a+	3.3 (5.7, 2.2) d€	12 (19, 7)c*, e*,f*

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SERUM DETERMINATION OF MMP-2 AND MMP-9 ACCORDING TO THE PATTERN OF ALCOHOL CONSUMPTION AND IN ALCOHOLIC HEPATIC DISEASE

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Introduction and Objectives: The damage caused by alcohol consumption generates liver fibrosis, which is characterized by the accumulation of extracellular matrix (ECM); To limit the liver damage, MMP-2 and MMP-9 gelatinases are produced as mediators to degrade ECM products. Their importance in liver damage proposes them as possible markers and target molecules in the diagnosis of alcohol consumption, as well as in alcoholic liver disease [1]. The objective of this work is to evaluate the serum concentrations of MMP-2 and MMP-9 gelatinases in subjects with different patterns of alcohol consumption and in alcoholic liver disease patients.

Materials and methods: A cross-sectional study was carried out in which subjects with different patterns of alcohol consumption were included. The inclusion was according to the AUDIT, DSM-IV, and clinical and biochemical data of liver disease: risk (Ri), abuse (Ab), dependence (OH), cirrhosis due to alcohol (CiOH) and alcoholic hepatitis (HA). A group without alcohol consumption (TC) was also included for comparison. For the quantification of MMP-2 and MMP-9, a multiple suspension assay (Milliplex®-MERCK ©) was used. Statistical analysis was performed using SPSS V.22 software using Mann Whitney U. P <0.05 was considered statistically significant; values were expressed as mean ± standard error.

Discussion: In 2015 Prystupa, A. et al. used MMP-2 and MMP-9 as markers of progression of damage in alcohol cirrhosis; however, there are no more related studies so far. Our data shows that the synthesis of MMP-2 and MMP-9 in consumption patterns and in liver disease are decreased from risky consumption, promoting the accumulation of ECM in liver tissue.

Conclusion: Serum levels of MMP-2 and MMP-9 gelatinases are affected by alcohol consumption, even in a risk pattern. MMP-2 and MMP-9 can be used as markers of alcohol-induced damage in early stages.

Conflict of interests: The authors declare that there is no conflict of interest.

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COMPARISON OF CXCL-8 and IFN-γ PRODUCTION IN ACUTE AND CHRONIC STAGES OF LIVER DISEASE

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Introduction and objective: Hepatitis C virus and alcoholism are the main causes of Chronic Liver Disease. (1) The increase of CXCL-8 has been correlated with mortality in alcoholic hepatitis (AH), while in Chronic Hepatitis C (CHC) with the disease severity. (2) The IFN-γ has an anti-viral and anti-fibrotic function. There are few comparative studies in patients regarding the production of these mediators and their possible implication in liver damage. The objective is to evaluate the concentrations of CXCL-8 and IFN-γ in the serum of AH, alcoholic cirrhosis (CiOH) and CHC patients.

Materials and methods: A multicenter cross-sectional study was carried out. Four participant groups were included: AH, CiOH, CHC and control group (CT). For the quantification of CXCL-8 and IFN-γ a multiple suspension array assay (Milliplex®-MERCK©) was used. Statistical analysis was performed by the SPSS V.22 software using Mann-Whitney-U-test. A p-value<0.05 was considered statistically significant. Values were expressed as median (Q3, Q1).

Results: 110 individuals were included: AH (10), CiOH (25), CHC (25) and CT (50). In CXCL-8 quantification, significant differences were detected in AH vs. CT, CiOH vs. CT, CHC vs. CT, CiOH vs. AH and CHC vs. AH ($p \leq 0.001$). While in IFN-γ, the differences were detected in AH vs. CT, CiOH vs. CT, CHC vs. CT, CiOH vs. AH and CHC vs. AH ($p \leq 0.001$).

Discussion: Differences were detected in both molecules when comparing the chronic stages (CiOH and CHC) with the acute stage (HA), while no differences were found when comparing both chronic stages. The highest CXCL-8 concentration corresponds to AH, reflecting its importance in prognosis and mortality. (2) The increase of IFN-γ in CiOH and CHC may have a role in the regulation of fibrogenesis because of its anti-fibrotic function.

Conclusion: The deregulation of mediators such as IL-8 and IFN-γ promotes systemic inflammation in both acute and chronic stages, being greater in AH. This may indicate an association with the susceptibility to persistent respiratory and gastrointestinal diseases.

The authors declare that there is no conflict of interest.

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