Table: Key demographics, Time to Treatment initiation, and SVR rate of patients by region and country

	Overall LATAM	Mexico**	Brazil	Colombia
# patients	*890*	*454*	*293*	*143*
Median age, y [IQR]	54.5 [43.2; 63.5]	50.9 [40.9; 59.5]	58.6 [47.8; 65.9]	61.3 [40.3; 68.9]
Male, %	50.9	52.9	47.1	52.4
≥50 y, % (male and female)	61.9	53.7	72.2	67.4
≥50 y male, %	30.5	29.7	35.4	22.5
≥50 y female, %	31.4	24.0	36.8	45.0
Genotype 3, %	14.4	11.0	25.3	2.8
Cirrhosis stage, %	33.9	48.5	14.0	28.7
# patients	*837*	*451*	*291*	*95*
Time to Treatment ≤ 1 month, %	27.5	10.6	57.1	17.9
# patients	*788*	*440*	*237*	*111*
Overall SVR, % *	99.6	99.5	100.0	99.1
SVR in GT3 patients, %	98.2	95.7	100.0	100.0
SVR in cirrhotic patients, %	99.6	99.5	100.0	100.0
SVR in GT3 & cirrhotic patients, %	97.4	96.0	100.0	100.0

*In effectiveness population, excludes patients who did not have a valid SVR status because of non-virological reasons or unknown reasons; ** Two sites

Key outcomes for LATAM in SVR10K study

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P-12 METHYL GROUP DONOR SUPPLEMENTATION IN A MetALD MODEL: REGULATION OF GUT MICROBIOTA AND METABOLIC PARAMETERS

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Conflict of interest: No

Introduction and Objectives: Patients who meet both *MASLD (Metabolic dysfunction-associated steatotic liver disease)* and *alcohol-related fatty liver disease (ALD)* criteria are categorized as having *MetALD (Metabolic and alcohol-related liver disease*) and its damage is characterized by metabolic dysfunction, gut dysbiosis and liver inflammation, resulting in steatosis and fibrosis. Methyl group donor supplementation in MASLD demonstrated metabolic benefits; we expect to corroborate those effects in MetALD and look for microbiota changes induced by methyl availability.

Patients / Materials and Methods: Twenty-four C57BL/6J male mice (25 ± 2g) were randomly assigned to 1) Control group (ND n=8); 2) MetALD (20% ethanol in water + 45% fat diet); 3) MetALD + MetMix (MetALD + methyl donors: methionine, betaine, zinc sulfate, choline, B9, B6 and B12). Each group maintained their respective diet/supplementation for 20 weeks. Liver, epididymal fat and body weight were weighted at sacrifice and liver enzymes, adipokines and lipid profile were measured on serum. Histopathological evaluation was performed on liver, adipose and colon tissues. Gene expression of IL6 and TNF-α was analyzed, while 16S rRNA gene sequencing in fecal DNA assessed gut microbiota.

Results and Discussion: Methyl donor supplementation decreased ($p<0.05$) body and epididymal fat weight, and reduced cholesterol, HDL, and LDL serum levels. A reduction (n.s.) in AST, ALT, TG, VLDL, insulin, leptin, glucagon, and resistin, and the mRNA levels of IL-6 and TNF-α were also observed. Hepatic steatosis and adipocyte area revealed a significant decrease ($p<0.05$) in MetALD + MetMix group. Intestinal crypts tended to be restored in length, similar to the control group. Microbiota Beta diversity was comparable between groups, while alpha diversity and Firmicutes/Bacteroidetes ratio showed a trend towards increase due to Firmicutes enrichment following supplementation.

Conclusions: Methyl donor supplementation improved body weight and lipid profile and reduced liver steatosis and adipocyte area, boosting the abundance and diversity of gut microbiota.

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P-13 EVALUATION OF UNDIAGNOSED ALCOHOLIC LIVER DISEASE IN PATIENTS WITH METABOLIC DYSFUNCTION-ASSOCIATED FATTY LIVER DISEASE USING THE ANI SCORE

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Conflict of interest: No

Introduction and Objectives: The 2024 EASL-EASD-EASO guidelines on Steatotic Liver Disease emphasize the distinction between Metabolic Dysfunction-Associated Steatotic Liver Disease (MAFLD) and the subcategories with significant alcohol consumption: Alcoholic Liver Disease (ALD) and Metabolic ALD (MetALD). To this aim, a systematic record of alcohol consumption and/or validated biomarkers is recommended.

To evaluate the likelihood of associated but unrecognized alcohol-related liver disease (MetALD/ALD) in patients managed for MAFLD.

Patients / Materials and Methods: A single-center retrospective study that analyzes the probability of MetALD/ALD using the ALD/NAFLD (ANI) score in patients diagnosed with MAFLD during May/June 2024. MAFLD was defined by the presence of hepatic steatosis with alcohol intake <20/30 g/day (iwomen/men), at least one cardiovascular risk factor, and no other discernible cause. Patients with suspected advanced chronic liver disease were excluded. A probability of MetALD/ALD was considered if ANI>0 (indicative of a probability >50%). Sociodemographic variables, alcohol consumption, and hepatic fibrosis were recorded and compared between the ANI>0 and ANI<0 groups, with significance level set at $p<0.05$.

Results and Discussion: 85 patients were included, average age 61.8±9.7 years, 52.9% male. Alcohol consumption was documented in medical history for 63.3% (54/85) of patients, with 53.4% (29/54) reporting no consumption. Per ANI score, 21.2% (18/85) were identified as having a probability of MetALD/ALD (ANI>0), with half of these (9/18) showing a probability >90%. Alcohol intake (10-20/30 g/day) was significantly higher in the ANI>0 group, consisting solely of males. Hepatic fibrosis was more pronounced in this group (7.6± 3.6 vs 5.9±1.7) but did not reach statistical significance.

Conclusions: The significant prevalence of unrecognized MetALD/ALD by ANI score suggests that alcohol consumption may be underreported in the ANI>0 group. Therefore, the inclusion of the ANI score as a biomarker for accurate differentiation MAFLD from MetALD/ALD appears to be beneficial.