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Abstracts

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Conference Chairs:

- Prof. Rifaat Safadi, Israel
- Prof. Arun J. Sanjal, USA
- Prof. Quentin M. Anstee, United Kingdom
- Prof. Michael Roden, Germany

insulin resistance of NAFLD NK cells showed lower responses and decreased killing ability. On the other hand, insulin up-regulation of the NMDAR showed to depresses mTOR activity.

Conclusions: NAFLD NK cells exert insulin resistance, mainly the CD56^{Dim} cytolytic population. The NMDAR unit regulates NK activity as a result of metabolic modifications; via an mTOR dependent pathway. It stimulated F-Actin accumulations and NK granzymes complex; indicating a new cellular pathway through which NK cells contribute to the NAFLD progression.

ICFL19-0019*

Decreased Expressions of p70S6K in F4-NAFLD Patients Inhibited F-Actin Correlated with their Impaired Function in Fatty Liver Patients

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Background and Aims: Co-localization of p70S6K and stress fibers was suggested to regulate actin polymerization as rapamycin treatment could inhibit the elongation and organization of actin stress fibers via inhibition of p70S6K. We investigated effects of insulin resistance on mTOR signaling pathway of NK cells as a potential cause for their impairment in Nonalcoholic-Fatty Liver-Disease (NAFLD).

Methods: Fresh peripheral blood NK cells isolated from healthy volunteers and 72 NAFLD patients (histology documented adults lacking full criteria of metabolic-syndrome) and characterized by flow-cytometry.

Results: Histologic progression of liver injury significantly correlated with elevated pro-inflammatory serum cytokines and insulin resistance. Western blot analysis of NK cells from NAFLD patients with F4 fibrosis showed to have dramatically reduction in PI3K pathway. ERK/MAP kinase pathway showed also reductions in these patients. Notably, these results were correlated with inhibitions in mTOR, p70S6K and F-actin phosphorylation ($p = 0.001$). NK stimulations with insulin (physiologic levels) reversed these effects. Compared to normal

HOMA NK-cells by *in-vitro* co-culture with HSCs, high-HOMA CD56^{dim} cells (with F3-F4) exhibited increased apoptosis and fail to block HSCs activation. While insulin incubation stimulated NK cell activation and killing of HSCs. Rapamycin reduced CD56^{dim} expressions of insulin receptors (mimicking NAFLD insulin resistance) and prevented the insulin stimulation effect on NK cells.

Conclusion: Systemic Insulin-Resistance in NAFLD also includes the NK cells with reduced expressions of p70S6K and F-actin and therefore impairment in their function, which leads to cirrhosis and probably cancer.

ICFL19-0007

Endothelial Lipase as a New Diagnostic Marker in Patients with Nonalcoholic Fatty Liver Disease and Metabolic Syndrome

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Objective: Non-alcoholic fatty liver disease (NAFLD) is a scourge of the planet's population. NAFLD development is due to the global increase in the number of metabolic syndrome. One of the unfavourable factors contributing to the formation of cardiovascular risk in the NAFLD on the background of hypertension is the low level of high-density lipoproteins (HDL) cholesterol, in the metabolism of which activity of endothelial lipase (EL) plays a leading role. The aim was the early detection of the cardiovascular risk development with EL levels.

Methods: 42 patients with NAFLD and hypertension stage 1 and 2 with more than 3 components of the metabolic syndrome according to the IDF criteria were followed-up in group 1. Group 2 consisted of 18 hypertensive patients without NAFLD and metabolic syndrome. Control group 3 consisted of 20 healthy individuals. The concentration of EL serum was determined by ELISA using kits of reagents "Aviscera Bioscience INC" (USA). NAFLD liver fat score was used for the liver steatosis identification.

Results: See Table 1.

Table 1. (for Abstract no ICFL19-0007)

Parameters	Group 1	Group 2	Control group
BMI	29.58±4.46	2.07±3.34	-
NAFLD index liver fat score	3.69±2.95	-0.308±1.14	-
HDL, mmol/l	1.20±0.27	1.47±0.42	-
Triglyceride, mmol/l	1.87±1.05	1.17±0.39	-
Blood glucose, mmol/l	6.03±1.41	5.06±0.58	-
Blood insulin, µm/ml	29.91±17.61	17.62±7.58	-
HOMA-IR	7.93±5.04	3.64±1.92	-
EL, ng/ml	12.43±3.23	10.19±2.18	8.23±2.47

* The difference in parameters is statistically significant ($p < 0.05$).

Spearman correlation analysis showed a significant positive association between the NAFLD index liver fat score and EL level ($r = 0.278$; $p < 0.05$).

Conclusions: The concentration of EL is lowest in patients of the control group and increases progressively with steatosis and metabolic syndrome compared with those suffering from hypertension without steatosis and metabolic syndrome. In addition, an increase in EL is associated with the presence of metabolic disorders, which, in aggregate, makes it possible to attribute it to independent markers of the atherosclerotic process and cardiovascular risk.

ICFL19-0061

Annual Healthcare Costs Double for Nonalcoholic Fatty Liver Disease/Nonalcoholic Steatohepatitis (NAFLD/NASH) Patients Who Progress to Advanced Liver Disease – Multivariable Analysis of German Real-World Data

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Background and Aims: Healthcare resource utilization (HCRU) and costs significantly increase among NAFLD/NASH patients who progress to compensated cirrhosis (CC). This study evaluated comorbidities, HCRU and associated costs among NAFLD/NASH patients who progress to advanced liver disease (including CC, decompensated cirrhosis

[DCC], liver transplantation [LT], hepatocellular carcinoma [HCC]) in Germany.

Methods: Adult patients with NAFLD/NASH (ICD-10-GM) were identified retrospectively from 2011–2016 in the In-Gef database containing claims data of >4 million individuals. Following the prevalent NAFLD/NASH diagnosis, patients were identified with liver severity stages (NAFLD/NASH non-progressors (NN/NP), CC, DCC, LT, HCC) using their first diagnosis date (index date). Per patient per quarter values were annualized.

Results: Of 4,580,434 individuals in the database, the study identified 215,655 (5%) prevalent NAFLD/NASH patients. During the follow-up, 100,644 incident events of different liver severity stages were reported (NN/NP [79,245 (78.7%)], CC [411 (0.4%)], DCC [20,614 (20.5%)], LT [11 (0.01%)] and HCC [363 (0.4%)]). Comorbid burden was high, with 33–67% patients across liver severity stages having ≥ 3 conditions of hypertension, hyperlipidemia, type-2 diabetes, renal disease and cardiovascular disease. Additionally, mean annual costs among NAFLD/NASH patients with advanced liver disease were significantly higher than NN/NP [€10,291 (CC), €22,561 (DCC), €34,089 (LT), €35,910 (HCC) vs. €3,818 (NN/NP)] ($p < 0.05$ except for LT); inpatient costs were the primary driver. Multivariable analyses adjusted for demographics and comorbidities confirmed this trend.

Conclusions: Early identification and effective treatment options to halt or reverse fibrosis are needed to prevent disease progression and the associated long-term costs.

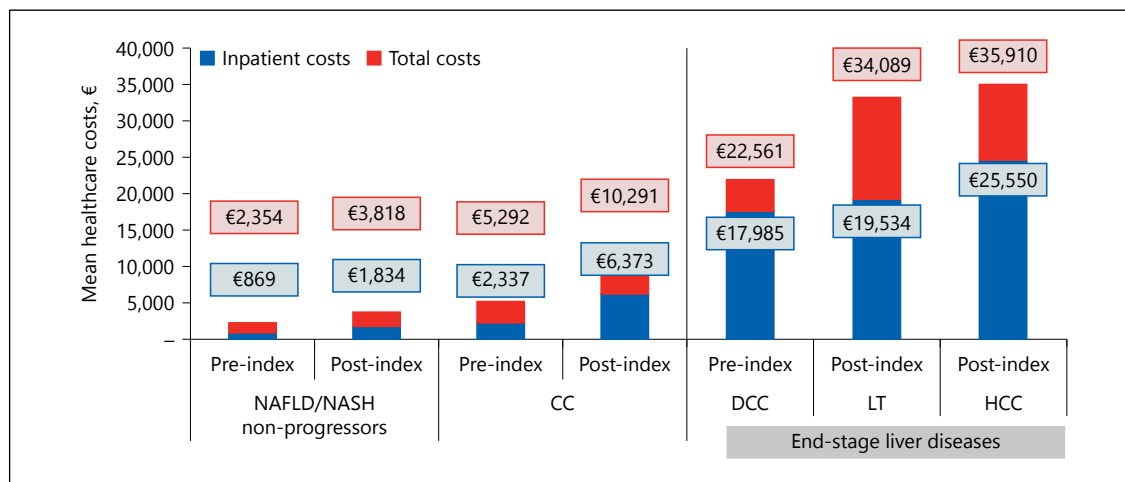


Fig. 1. Annual mean healthcare costs for NAFLD/NASH patients with advanced liver disease. NAFLD, non-alcoholic fatty liver disease; NASH, nonalcoholic steatohepatitis; CC, compensated cirrhosis; DCC, decompensated cirrhosis; LT, liver transplant; HCC, hepatocellular carcinoma; NAFLD/NASH non-progressors may include F0-F3 and possibly undiagnosed CC/DCC/LT/HCC (for Abstract no ICFL19-0061).

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