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Dear Colleagues,

Alcohol and obesity are two major causes of fatty liver disease, today the most common chronic liver disease in the Western world. The Ljubljana conference will focus on these types of nutrition-induced hepatopathies, but also parenteral nutrition-induced cholestasis and liver disease will be addressed.

On the other hand, acute and chronic liver disease as well as cholestasis affect the nutritional status by reducing appetite and intestinal absorption, by impairing digestion and by altering metabolism. Both the underlying mechanisms as well as the clinical consequences (“How to feed such patients?”) will be topics of the two-days meeting. Well-known experts in the field coming across Europe and beyond will present the State-of the Art of these most relevant and latest themes and will answer to your questions.

The conference is also a good possibility to visit Ljubljana, one of the most interesting cities of the Balkans, the capital of Slovenia, which became EU member in 2004. Ljubljana integrates German/Austrian (former name “Laibach”) and Italian (“Lubiana”) history; in 1991 the city became independent. People believe that “Ljubljana” origins from “ljubljena”, the Croatian word for “beloved” city. Because of the place, the hospitable people, and of course because of the outstanding scientific program you should come to Ljubljana, November 25-26th, 2016.

We look forward to seeing you there!

Stephan C. Bischoff  
Co-Organiser

Mathias Plauth  
Co-Organiser
WHO SHOULD ATTEND?
Hepatologists, physicians with an interest in Hepatology and nutrition, translational and clinical researchers, health professionals, and young trainees.

WHY ATTEND?
• 2 days of world-class seminars on nutrition in liver diseases.
• Gather and network with renowned specialists.

TOPICS TO BE COVERED
Patient management and nutrition in: Alcoholic liver disease, NAFLD, IFALD and PNALD, cirrhosis, ALF, biliary diseases and fibrosis.
SCIENTIFIC COMMITTEE

Stephan C. Bischoff, Stuttgart, Germany
Mathias Plauth, Dessau-Rosslau, Germany

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ACKNOWLEDGEMENTS

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EASL thanks its Premium Sponsors for their generous contributions and support of the EASL Monothematic Conference ‘Nutrition in Liver Disease’ with an unrestricted educational grant.

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GENERAL INFORMATION
GENERAL INFORMATION

CONFERENCE VENUE
Four Points by Sheraton Ljubljana Mons,
Pot za Brdom 4, 1000
Ljubljana, Slovenia

DISCOVER LJUBLJANA
City website:
http://www.ljubljana.si/en/

In Ljubljana the old meets the new; and it seems that history has spent all of the settlement’s five millennia preparing it to become the nation’s capital.

It has managed to retain traces from all periods of its rich history; from the legacy of Roman Emona; through to the Renaissance, Baroque and Art Nouveau periods characterised in the house fronts and ornate doorways of the city centre, the romantic bridges adorning the Ljubjanica river, the lopsided rooftops and a park reaching deep into the city centre. Here eastern and western cultures met; and the Italian concept of art combined with the sculptural aesthetics of central european cathedrals.

LANGUAGE
The official language of the conference is English.

CLIMATE
Ljubljana has a polar tundra climate. The month of November is characterized by rapidly falling daily high temperatures, with daily highs decreasing from 11°C to 5°C over the course of the month, exceeding 16°C or dropping below -1°C only one day in ten.

NAME BADGES
All participants are kindly requested to wear their name badges throughout the EASL Monothematic Conference in order to be admitted to the lecture halls and other scheduled activities.

REGISTRATION AND ACCOMMODATION
All participants are invited to register online in order to save time upon their arrival at the conference.

Hotel accommodation for the EASL Monothematic Conference will be offered to participants during the online registration process. Detailed information, as well as access to the online registration is available on www.easl.eu. Registered participants are entitled to reduced conference hotel rates.

REGISTRATION DESK
The onsite registration desk at the conference venue will be open at the following times:

Friday
25 November 2016 from 12:00 to 20:00

Saturday
26 November 2016 from 08:00 to 16:00
CME ACCREDITATION
An application has been made to the EACCME® for CME accreditation of this event.

All attendees will receive an email with a link to a questionnaire at the end of the conference. Upon completion of the questionnaire, attendees should receive their certificates of attendance and their CME credits by email.

No certificate of attendance will be printed onsite.

Certificates of attendance will only be sent to delegates who attended the conference.

EACCME CREDITS
Each medical specialist should claim only those hours of credit that he/she actually spent in the educational activity. The EACCME credit system is based on 1 ECMEC per hour with a maximum of 3 ECMECs for half a day and 6 ECMECs for a full-day event.

FLIGHTS TO LJUBLJANA
Direct flights are available from the European airports listed below to the Ljubljana Jože Pučnik Airport (LJU):
- Austria – Vienna
- Belgium – Brussels
- France – Paris
- Germany – Munich, Frankfurt
- Netherlands – Amsterdam
- Norway – Copenhagen
- Poland – Warsaw
- Russia – Moscow
- Switzerland – Zurich
- United Kingdom – London

TRANSPORT TO THE VENUE
The conference venue, Four Points by Sheraton Ljubljana Mons, is 20 minutes by car from Ljubljana Jože Pučnik Airport (LJU).

By public transport
Take the bus Ljubljana – Letališče Brnik from the airport to Avtobusna postaja Ljubljana (23 stops). Walk 400 metres to Slovenska cesta, then take the bus 14 B from Bavarski dvor to Podmornica (11 stops). Turn right on Tehnološki park, then walk 1300 metres on Pot za Brdom and the Four Points by Sheraton Ljubljana Mons is at the end of the road.

By car
Take route 104 and Šenčur to A2/E61 in Voglje. Follow the A2/E61 to Priključek Lj. – Brdo in Ljubljana and take exit 16 from A2/E61. Follow Priključek Lj. – Brdo, then turn right on Pot za Brdom. The Four Points by Sheraton Ljubljana Mons will be on your right-hand side.

PARTICIPANTS’ LIST
The participants’ list will be displayed and located onsite at the EASL booth.

DRESS CODE AND SMOKING POLICY
Dress code is informal for all occasions. This will be a non-smoking event.
BANKING, SAFETY AND SECURITY
The currency used in Ljubljana is the EURO. Foreign currency can be exchanged at banks, bureau de change, and automatic currency exchange machines.

Please do not leave bags or suitcases unattended at any time, whether inside or outside the session halls. Hotels strongly recommend that you use their safety deposit boxes for your valuables.

LIABILITY AND INSURANCE
The EASL Office cannot accept liability for personal accidents or loss of, or damage to, private property of participants. Participants are advised to take out their own personal travel and health insurance for their trip.
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SCIENTIFIC PROGRAMME
### DAY 1 – FRIDAY 25 NOVEMBER 2016

#### SESSION 1  BILIARY DISEASE AND CYSTIC FIBROSIS

<table>
<thead>
<tr>
<th>Time</th>
<th>Event</th>
</tr>
</thead>
</table>
| 15:30 – 17:30 | Chairs: Caroline Lemaitre, *France*  
Andre van Gossum, *Belgium* |
| 15:30 – 16:00 | **GALLSTONES AND DIET**  
Irina Blumenstein, *Germany* |
| 16:00 – 16:30 | **NUTRITIONAL MANAGEMENT OF CHOLESTATIC LIVER DISEASE**  
Nada Rotovnik Kozjek, *Slovenia* |
| 16:30 – 17:00 | **CHOLESTASIS AS A CONSEQUENCE OF PARENTERAL NUTRITION**  
Andre van Gossum, *Belgium* |
| 17:00 – 17:30 | **NUTRITIONAL MANAGEMENT OF CYSTIC FIBROSIS – THE PAEDIATRICIANS’ PERSPECTIVE**  
Reka Bodnar, *Hungary* |

#### SESSION 2  IFALD, PNALD AND OTHER NUTRITION-INDUCED LIVER COMPLICATIONS

<table>
<thead>
<tr>
<th>Time</th>
<th>Event</th>
</tr>
</thead>
</table>
| 17:30 – 18:30 | Chairs: Craig McClain, *United States*  
Manuela Merli, *Italy* |
| 17:30 – 18:00 | **PNALD – THERAPEUTIC OPTIONS (ADULT VS PEDIATRIC PATIENTS)**  
Caroline Lemaitre, *France* |
| 18:00 – 18:30 | **IFALD – LESSONS TO BE LEARNED FROM LIVER PATHOLOGY IN CELIAC DISEASE**  
Umberto Volta, *Italy* |

18:30 – 19:00  *ePoster session 1 and cocktail reception*

19:00 – 19:30  *ePoster session 2 and cocktail reception*
DAY 2 – SATURDAY 26 NOVEMBER 2016

08:30 – 09:00 **ePoster session 3**

### SESSION 3  ALCOHOLIC AND NON-ALCOHOLIC FATTY LIVER DISEASE

09:00 – 11:30 **Chairs: Stephan C. Bischoff, Germany**
**Michael Manns, Germany**

09:00 – 09:30 **NAFLD AND THE METABOLIC SYNDROME – MECHANISMS**
Hannele Yki-Järvinen, Finland

09:30 – 10:00 **NAFLD AND AFLD – WHAT IS THE DIFFERENCE?**
Craig J McClain, United States

10:00 – 10:30 **NAFLD AND AFLD – IMPACT FOR VIRAL HEPATITIS**
Michael P. Manns, Germany

10:30 – 11:00 **NAFLD AND THE METABOLIC SYNDROME – ROLE OF NUTRITION IN THERAPY AND PREVENTION**
Herbert Tilg, Austria

11:00 – 11:30 **ALCOHOLIC LIVER DISEASE – ROLE OF NUTRITION AS RISK FACTOR AND THERAPEUTIC OPTION**
Peter Ballmer, Switzerland

11:30 – 12:00 **ePoster session 4 and coffee break**

### SESSION 4  ACUTE LIVER FAILURE (ALF)

12:00 – 12:30 **Chairs: Mathias Plauth, Germany**
**Jens Kondrup, Denmark**

12:00 – 12:30 **IMPACT OF ALF ON CARBOHYDRATE, LIPID AND NITROGEN METABOLISM**
Jens Kondrup, Denmark

12:30 – 13:00 **HOW TO FEED THE ALF PATIENT**
Brian J. Hogan, United Kingdom
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<tr>
<th>Time</th>
<th>Session and Session Details</th>
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<tr>
<td>13:00 – 13:30</td>
<td>ePoster Session 5 and Lunch</td>
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<tr>
<td>13:30 – 14:00</td>
<td>ePoster Session 6 and Lunch</td>
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### SESSION 5  LIVER CIRRHOSIS

**14:00 – 15:30**  
**Chairs:** Craig J. McClain, United States  
Brian J. Hogan, United Kingdom

14:00 – 14:30  
**IMPACT OF CIRRHOSIS ON METABOLISM AND BODY COMPOSITION**  
Matthias Pirlich, Germany

14:30 – 15:00  
**MOLECULAR MECHANISMS AND MOLECULAR BIOLOGY OF SARCOPENIA IN LIVER DISEASE**  
Srinivasan Dasarathy, United States

15:00 – 15:30  
**HOW TO FEED THE PATIENT WITH CIRRHOSIS?**  
Mathias Plauth, Germany

15:30 – 16:00  
ePoster Session 7 and Coffee Break

### SESSION 6  LIVER DISEASE AND SURGERY

**16:00 – 17:00**  
**Chairs:** Mathias Plauth, Germany  
Jens Kondrup, Denmark

16:00 – 16:30  
**PERIOPERATIVE NUTRITIONAL MANAGEMENT OF PATIENTS WITH CIRRHOSIS**  
Lindsay Plank, New Zealand

16:30 – 17:00  
**SARCOPENIC OBESITY AND ASSOCIATED RISK AFTER OLT**  
Manuela Merli, Italy
POST-DOC RESEARCH

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ENTRY-LEVEL RESEARCH

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PRESENTATIONS
## DAY I – FRIDAY 25 NOVEMBER 2016

**Session I**  
ePoster presentations 18:30 – 19:00

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<tr>
<th>Screen</th>
<th>Title</th>
<th>Abstract</th>
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<tbody>
<tr>
<td>1</td>
<td>Diagnostic performance of FibroMax – FibroTest, SteatoTest and ActiTest – in patients with NAFLD using the SAF score as histological reference</td>
<td>P1.1</td>
<td>Mona Munteanu</td>
</tr>
<tr>
<td>2</td>
<td>Association between malnutrition in patients with liver cirrhosis and the presence of overt hepatic encephalopathy</td>
<td>P1.2YI</td>
<td>Ricardo Ulises Macias-Rodriguez</td>
</tr>
<tr>
<td>3</td>
<td>Serum myostatin predicts survival in patients hospitalized for decompensated advanced chronic liver disease in unexpected manner</td>
<td>P1.3</td>
<td>Lubomir Skladany</td>
</tr>
<tr>
<td>4</td>
<td>Assessment of sarcopenia in patients with chronic liver disease</td>
<td>P1.4</td>
<td>Helen Vidot</td>
</tr>
<tr>
<td>5</td>
<td>Nutritional wheat amylase trypsin inhibitors, activators of intestinal toll like receptor 4, exacerbate non-alcoholic steatohepatitis in high fat diet fed mice</td>
<td>P1.5YI</td>
<td>Muhammad Ashfaq-Khan</td>
</tr>
<tr>
<td>6</td>
<td>The impact of nutritional status on survival in cirrhotic patients with sepsis</td>
<td>P1.6YI</td>
<td>Vincenza Di Gregorio</td>
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</tbody>
</table>
### Session 2

**ePoster presentations 19:00 – 19:30**

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<tr>
<th>Screen</th>
<th>Title</th>
<th>Abstract</th>
<th>Presenter</th>
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</thead>
<tbody>
<tr>
<td>1</td>
<td>Taste sensitivity and food preferences in terminal-stage of cirrhotic patients</td>
<td>P2.1YI</td>
<td>Thomas Mouillot</td>
</tr>
<tr>
<td>2</td>
<td>Osteopontin ablation drives hematopoietic stem cell mobilization and increases hepatic iron contributing to alcoholic liver disease</td>
<td>P2.2YI</td>
<td>Fernando Verduzco</td>
</tr>
<tr>
<td>3</td>
<td>Visceral fat evaluated by ultrasound is associated with insulin resistance and liver damage in patients with non-alcoholic fatty liver disease</td>
<td>P2.3YI</td>
<td>Licia Polimeni</td>
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<tr>
<td>4</td>
<td>Potentiality of flavonoids consumption in patients with nonviral chronic hepatitis</td>
<td>P2.4YI</td>
<td>Vasyl Prysyazhnyuk</td>
</tr>
<tr>
<td>5</td>
<td>A collaborative quality improvement project to improve early nutritional support in decompensated chronic liver disease</td>
<td>P2.5YI</td>
<td>Sobia Saghir</td>
</tr>
<tr>
<td>Screen</td>
<td>Title</td>
<td>Abstract</td>
<td>Presenter</td>
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<tr>
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</tr>
<tr>
<td>1</td>
<td>Comprehensive nutritional assessment in patients with refractory ascites prior to the implantation of an alfpump system</td>
<td>P3.1</td>
<td>Guido Stirnimann</td>
</tr>
<tr>
<td>2</td>
<td>Trace elements in decompensated alcoholic liver cirrhosis</td>
<td>P3.2YI</td>
<td>Milica Stojkovic Lalosevic</td>
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<tr>
<td>3</td>
<td>Age-dependent changes in liver of adult and pubertal rats with metabolic syndrome</td>
<td>P3.3YI</td>
<td>Oleksandr Tkachenko</td>
</tr>
<tr>
<td>4</td>
<td>Dietary habits and nutritional status of patients with liver cirrhosis: results from a cross sectional survey</td>
<td>P3.4</td>
<td>Roxana Vadan</td>
</tr>
<tr>
<td>5</td>
<td>Aggressive nutrition intervention can reduce ascites in patients with cirrhosis and refractory ascites: a case series report</td>
<td>P3.5</td>
<td>Helen Vidot</td>
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### Session 4
**ePoster presentations 11:30 – 12:00**

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<th>Screen</th>
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<th>Abstract</th>
<th>Presenter</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Controlled and motivation programme of changing eating behavior reduce weight and Cytokeratin 18 levels in patients with NAFLD</td>
<td>P4.1YI</td>
<td>Nataliia Dynnyk</td>
</tr>
<tr>
<td>2</td>
<td>Systemic FGF19 and bile salt levels in adult patients with chronic intestinal failure on home parenteral nutrition</td>
<td>P4.2</td>
<td>Angelique Huibers</td>
</tr>
<tr>
<td>3</td>
<td>No consensus among nutritional assessment tools for identification of malnutrition in patients with alcoholic liver disease</td>
<td>P4.3YI</td>
<td>Chetan Ramesh Kalal</td>
</tr>
<tr>
<td>4</td>
<td>Role of malnutrition in compensated cirrhosis: a retrospective analysis answering to the proposal of the Italian consensus on portal hypertension (Baveno VI)</td>
<td>P4.4YI</td>
<td>Barbara Lattanzi</td>
</tr>
<tr>
<td>5</td>
<td>May protein malnutrition represent a risk factor for neurocognitive alterations in cirrhosis? Are protein-restricted diets in hepatic encephalopathy friend or foe?</td>
<td>P4.5YI</td>
<td>Cristina Lucidi</td>
</tr>
<tr>
<td>6</td>
<td>The impact of liver steatosis in the assessment of liver fibrosis by point share wave elastography and the validity of Hamagouchi score for quantification of steatosis compared with histology in bariatric subjects</td>
<td>P4.6YI</td>
<td>Daniele Macor</td>
</tr>
<tr>
<td>Screen</td>
<td>Title</td>
<td>Abstract</td>
<td>Presenter</td>
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<tr>
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</tr>
<tr>
<td>1</td>
<td>Erythrocyte membrane fatty acid profile in patients with cirrhosis and risk of hepatocellular carcinoma</td>
<td>P5.1YI</td>
<td>Thomas Mouillot</td>
</tr>
<tr>
<td>2</td>
<td>Sarcopenia predicts the occurrence of hepatic encephalopathy after transjugular intrahepatic portosystemic shunt</td>
<td>P5.2YI</td>
<td>Silvia Nardelli</td>
</tr>
<tr>
<td>3</td>
<td>Impact of adrenal dysfunction on psoas muscle thickness assessed by computed tomography in patients with liver cirrhosis</td>
<td>P5.3YI</td>
<td>Graziella Privitera</td>
</tr>
<tr>
<td>4</td>
<td>Phase angle as an early nutritional marker of short-term outcome in hospitalized patients with cirrhosis</td>
<td>P5.4YI</td>
<td>Astrid Ruiz-Margáin</td>
</tr>
<tr>
<td>5</td>
<td>Adherence to mediterranean diet and non-alcoholic fatty liver disease: impact on metabolic profile</td>
<td>P5.5YI</td>
<td>Francesco Baratta</td>
</tr>
<tr>
<td>6</td>
<td>Vitamin D deficiency and hepatic encephalopathy in patients with chronic liver disease</td>
<td>P5.6</td>
<td>Helen Vidot</td>
</tr>
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### Session 6
**ePoster presentations 13:30 – 14:00**

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<th>Screen</th>
<th>Title</th>
<th>Abstract</th>
<th>Presenter</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Bioelectrical impedance vector analysis (BIVA) is an objective method for nutritional assessment in patients with cirrhosis</td>
<td>P6.1YI Astrid Ruiz-Margáin</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>The role of nutritional intervention in patients with chronic liver disease awaiting transplantation</td>
<td>P6.2 Helen Vidot</td>
<td></td>
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<tr>
<td>3</td>
<td>Different responses of FGF19, FGF21, total bile acid and gut hormones in diabetics after Roux-en-Y gastric bypass</td>
<td>P6.3 Chih-Yen Chen</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>Role of Galectin-3 and IL-33/ST2 Pathway in obesogenic diet-induced nonalcoholic steatohepatitis</td>
<td>P6.4YI Ilija Jeftic</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>Effect of a probiotic on fatty liver index and liver stiffness in NAFLD patients: randomized clinical trial</td>
<td>P6.5YI Natalia Bosak</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>Parenteral nutrition dysregulates bile salt homeostasis in a rat model of parenteral nutrition-associated liver disease</td>
<td>P6.6YI Kiran VK Koelfat</td>
<td></td>
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</tbody>
</table>
### Session 7
**ePoster presentations 15:30 – 16:00**

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<th>Abstract</th>
<th>Presenter</th>
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<tbody>
<tr>
<td>1</td>
<td>Impact of bariatric surgery in the main clinical and laboratory indicators 6 and 12 months after intervention in bariatric subjects and differences between by-pass and sleeve gastrectomy</td>
<td>P7.1YI</td>
<td>Valentina Lanzilotti</td>
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<tr>
<td>2</td>
<td>Silimarvin vs. life style change: a comparison between these potential therapeutic approaches for juvenile NAFLD</td>
<td>P7.2YI</td>
<td>Veronica Marin</td>
</tr>
<tr>
<td>3</td>
<td>Effect of an oral bile acid supplement on lipid metabolism in patients with short bowel syndrome on long-term parenteral nutrition</td>
<td>P7.3YI</td>
<td>Thomas Mouillot</td>
</tr>
<tr>
<td>4</td>
<td>Food intake, anthropometry and metabolism of serum iron in patients with nonalcoholic steatohepatitis</td>
<td>P7.4</td>
<td>Joana Ribeiro Silva</td>
</tr>
<tr>
<td>5</td>
<td>Probiotics and nutraceuticals: are they one team players in NAFLD prevention</td>
<td>P7.5YI</td>
<td>Nazarii Kobyliak</td>
</tr>
<tr>
<td>6</td>
<td>The Next Generation Sequencing (NGS) platform in the assessment of the gut microbiota in bariatric surgery</td>
<td>P7.6YI</td>
<td>Giuseppina Campisciano</td>
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INVITED SPEAKERS’ ABSTRACTS
NUTRITIONAL MANAGEMENT OF CYSTIC FIBROSIS – THE PAEDIATRICIANS’ PERSPECTIVE

Reka Bodnar*1
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Abstract Body: Cystic fibrosis (CF) is an inherited disease that affects particularly the lungs, pancreas, hepatobiliar system, reproductive tract, and sweat glands (1,2). It is caused by mutations in the gene encoding the CF transmembrane conductance regulator protein. It is the most common recessive disease among Caucasian populations. The therapeutic approach to the modern treatment of chronic diseases has changed. The aim now is not only to extend life but also to improve the quality of life. To improve the quality of medical attendance in CF, pulmonologists, gastroenterologists, CF nurses, dieticians, social workers and respiratory therapists work in collaboration as members of CF team. There are some healthcare challenges regarding the management of CF. Nutrition is a critical component of the management of CF, and nutritional status is directly associated with both pulmonary status and survival. Expert dietetic care is necessary, and attention must be given to ensuring an adequate energy intake. Several factors contribute to impaired nutritional status in CF (pancreatic insufficiency, chronic malabsorption, recurrent sinopulmonary infections, chronic inflammation, increased energy expenditure, suboptimal intake). Specific nutrient deficiencies, such as fat soluble vitamins, are common in cystic fibrosis. Management of nutritional problems can be complex, and injudicious treatment can further worsen the situation, as the relationship between high daily doses of pancreatic enzymes and the development of fibrosing colonopathy illustrates. Our HRQoL study highlighted the fact that malnutrition has a significant and negative impact on patients’ HRQoL (3,4). The adequate pancreas enzyme replacement therapy is important in case of CF patients. Therefore it is urgent to introduce the measurement of the level of faecal elastase in case of Hungarian CF patients. With this technique better management of nutritional status could be obtained. Even if monitoring of nutritional status is recommended by checking the signs of malnutrition, the above mentioned objective technique could be a useful tool for CF specialists when diagnosing CF and optimizing pancreatic replacement therapy.

References:

Disclosure of Interest: None Declared
IFALD – LESSONS TO BE LEARNED FROM LIVER PATHOLOGY IN CELIAC DISEASE

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Abstract Body: Celiac disease (CD) is an immune-mediated small bowel disorder triggered by the ingestion of gluten-containing foods (1). This food intolerance, known to affect 1% of the general population, is regarded as the result of a complex interplay between gliadin and tissue transglutaminase (TG2), identified as the main CD autoantigen. Concerning the genetic background, CD is closely related to the human leukocyte antigen (HLA)-DQ2 and/or –DQ8. Although the disease primarily affects the gut, CD is a typical example of a systemic disorder. Growing evidence indicates that liver involvement is one of the extraintestinal manifestations of CD. Two main clinical phenotypes of liver damage, namely cryptogenic and autoimmune, have been observed in patients with CD. The most frequent finding is represented by a cryptogenic hypertransaminasemia, detected in about a half of untreated CD patients, as an expression of mild liver dysfunction with a histological picture of nonspecific reactive hepatitis (2). Transaminase concentrations revert to normal within 6 months of strict gluten-free diet (GFD). Patients with CD are at increased risk of nonalcoholic fatty liver disease (NAFLD) compared to the general population (3). Liver cirrhosis of unknown origin can be detected in a minority of CD patients with persistent hepatic abnormalities and it can be conceivably attributed to the progression of liver damage due to the late diagnosis of CD. Moreover, patients with CD can be affected by a wide range of autoimmune liver disorders, including autoimmune hepatitis (AIH), primary biliary cirrhosis (PBC) and primary sclerosing cholangitis (PSC) (4, 5). The pathogenic mechanisms underlying liver injury in CD are poorly understood. However, the following factors have been hypothesized as playing a role in determining the liver injury in CD: a) malabsorption and long-standing malnutrition; b) increased intestinal permeability; c) abnormalities of gut microbiota; d) humoral-mediated immune response; e) genetic factors. Malnutrition due to the severe malabsorption is rarely observed in CD nowadays, but in some cases it might concur in determining liver dysfunction. The hepatic injury seen in CD could likely depend on increased intestinal permeability resulting in the arrival of toxins and antigens in the hepatobiliary system through the portal circulation. Another important aspect concerns the role of gut microbiota in regulating the gut-liver axis. In normal conditions, commensal microbes and the host share several mutual advantages maintaining the intestinal barrier integrity. When dysbiosis occurs in CD, the changes of the normal equilibrium of gut microbiota can contribute to impair the intestinal barrier function thereby leading to bacterial translocation. Autoantibodies
directed against TG2 have been described in the liver and other extraintestinal tissues in CD, raising the possibility of a pathogenic role for the humoral-mediated immune response in the liver injury. A common genetic predisposition is present in CD, AIH and PSC. The main genetic marker of CD is HLA-DQ2, which is found in about 95% of CD patients. HLA-DQ2 is in strong linkage disequilibrium with HLA-DR3, which is the major HLA risk factor for AIH. Furthermore, in patients with PSC, associated with inflammatory bowel disease, the haplotype HLA-B8/DR3 is frequently found. Therefore, from a genetic standpoint, there is a close correlation between CD and AIH/PSC, whereas this is not present with PBC (although recent genome-wide association studies indicate some common polymorphism between CD and PBC). Due to the high frequency of liver abnormalities in CD, liver enzyme levels should be checked routinely in all patients with CD at diagnosis. Patients with raised levels of serum transaminases should be studied to exclude other causes of liver injury, including viral, autoimmune, enzyme deficiency and metabolic disorders. Once established that hypertransaminasemia is cryptogenic and likely related to CD, liver enzymes should be re-evaluated after 6–12 months of a strict GFD. The persistently elevated serum levels of transaminases must be regarded as an expression of a poor compliance with the diet or a coexistent liver disease other than celiac-type hepatitis.

References

Disclosure of Interest: None Declared
NAFLD AND METABOLIC SYNDROME – MECHANISMS

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Abstract Body: Non-alcoholic fatty liver disease (NAFLD) covers a spectrum of disorders ranging from simple steatosis (non-alcoholic fatty liver, NAFL) to non-alcoholic steatohepatitis (NASH) and cirrhosis. Even simple NAFL increases the risk of liver fibrosis. Both acquired and genetic (variants in PNPLA3, TM6SF2 and MBOAT7) factors regulate liver fat content. ‘Metabolic NAFLD’. The liver may be fatty due to causes of insulin resistance such as obesity and physical inactivity (‘Metabolic NAFLD’). The metabolic syndrome (MetS) and NAFLD share common pathophysiology although their definitions differ. The liver is the site of production of two of the key components of the MetS, fasting serum glucose and VLDL. In subjects with NAFLD, the ability of insulin to suppress production of glucose and VLDL is impaired resulting in hyperglycemia and hyperinsulinemia and hypertriglyceridemia combined with low HDL cholesterol. The liver, once fatty, also overproduces many other markers of cardiovascular risk such as C-reactive protein and coagulation factors. Although NAFLD and the MetS may be observed in non-obese subjects, obesity and intake of saturated fat and simple sugars, are major factors underlying the epidemic of NAFLD. ‘Metabolic NAFLD’ predispose to type 2 diabetes and cardiovascular disease (CVD)1,2. ‘Nutrition and NAFLD’. Study of the impact of nutritional modulation of liver fat content is of interest for understanding the mechanisms underlying NAFLD as well as its prevention and treatment3,4. Hypocaloric, especially low carbohydrate ketogenic diets rapidly decrease liver fat content and associated metabolic abnormalities. However, any type of caloric restriction seems effective long-term. Isocaloric diets containing 16-23% fat and 57-65% carbohydrate lower liver fat compared to diets with 43-55% fat and 27-38% carbohydrate. Diets rich in saturated (SFA) as compared to monounsaturated (MUFA) or polyunsaturated (PUFA) fatty acids appear particularly harmful as they increase both liver fat and insulin resistance (Figure). Overfeeding either saturated fat or carbohydrate increases liver fat content. Vitamin E supplementation decreases liver fat content as well as fibrosis but has no effect on features of insulin resistance.

Figure. Effect of dietary composition on liver fat content, expressed as relative change from baseline measured by 1H-MRS. Diets comparing isocaloric low fat/high carbohydrate (Low Fat High Carb) to high fat/low-carbohydrate (High Fat Low Carb) diets (upper panel on the left, 1=5, 2=6, 3=), isocaloric low saturated fat/high polyunsaturated fat (Low SFA High PUFA) to high saturated/low polyunsaturated fat (High SFA Low PUFA)

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Mechanisms of nutritional modulation of NAFLD. Fatty acids in intrahepatocellular triglycerides can originate from adipose tissue lipolysis, de novo lipogenesis (DNL), uptake of fatty acids from chylomicron remnants and from fatty acids released during intravascular hydrolysis of triglyceride-rich lipoproteins\textsuperscript{15}. Studies using stable isotopes to trace pathways of hepatic triglyceride synthesis have shown that both adipose tissue lipolysis and DNL are increased in NAFLD\textsuperscript{16}. We have recently compared effects of overfeeding of 3 different diets for 3 weeks on pathways contributing to intrahepatic triglyceride synthesis in 38 subjects with a BMI of 31 kg/m\textsuperscript{2}. Overfeeding a diet enriched either in mono- and polyunsaturated fat, saturated fat or carbohydrate all increased liver fat, but saturated fat was most harmful. Overfeeding carbohydrate increased liver fat via DNL while the saturated diet increased lipolysis more than the other diets. These data suggest that overeating saturated fat is even more harmful for NAFLD than that of carbohydrate or polyunsaturated fat.\textsuperscript{17}

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NAFLD AND AFLD – WHAT IS THE DIFFERENCE?

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Abstract Body: Many factors lead to the development of hepatic steatosis and steatohepatitis, including excess alcohol intake, a high-fat diet, and high-sugar beverage diets. While liver biopsies from patients consuming these diverse diets look identical histologically, the metabolic pathways involved are quite different as documented by gene profiling in experimental animals. Moreover, environmental toxins and drugs can also lead to fatty liver directly or exacerbate the above-noted pathways. This presentation will review selected differences between non-alcoholic fatty liver disease (NAFLD) and alcoholic fatty liver disease (AFLD). The type of diets consumed by these two patient groups (AFLD vs. NAFLD) are generally quite different in caloric makeup. In studies of alcoholic hepatitis/cirrhosis, patients are almost invariably consuming 12-15 standard drinks per day which equates to over 2,000 calories from alcohol. In NAFLD, many high-beverage consumers are taking in an equivalent amount of calories in high-fructose-containing drinks. High-fat consuming patients with NAFLD generally consume calories high in saturated fats and trans fats. Protein intake is almost always deficient in AFLD. We postulate that inadequate intake of leucine-rich protein (several times/day) likely plays a role in the muscle loss in ALD. This is not necessarily the case with NAFLD. The types of dietary fat intake associated with NAFLD vs. AFLD are frequently different. Consumption of a diet high in polyunsaturated fats (specifically Omega-6 fatty acids and especially linoleic acid) has been associated clinically with AFLD. Importantly, in experimental models of AFLD, the consumption of a diet rich in linoleic acid exacerbates AFLD while a diet rich in saturated fats protects against AFLD. Moreover, human intake of linoleic acid has increased dramatically over the last century. Linoleic acid in the diet is associated with increased gut-barrier dysfunction and inflammation, altered microbiome, endotoxemia, increased hepatic inflammation and TNF production, and increased liver injury. In NAFLD, diets high in saturated fats are frequently observed in humans and used in experimental animal models. Lipotoxicity is thought to be an important factor in the hepatotoxicity in NAFLD. Sarcopenia is generally more frequent in AFLD, but we are observing increasing sarcopenic obesity with NAFLD. Patients with NAFLD and sarcopenic obesity frequently have ectopic fat deposits in their muscle and have increased visceral adiposity. In experimental models, animals with NAFLD have increased visceral fat with large adipocytes, while alcohol-fed mice tend to have little or no increase in visceral fat, and the adipocytes are small. However, in both cases the adipocytes are inflamed with increased cytokine production and increased “crown-like” structures. Micronutrient deficiency can be observed in both NAFLD and AFLD, but certain micronutrients such
as zinc are more prominently affected with alcohol abuse. In cirrhosis due to either disease process, we recommend a late-night snack to prevent overnight starvation and potentially to stimulate muscle-protein synthesis. Patients with NAFLD are recommended a low-calorie, high-protein diet. Fatigue is the most common complaint we see with liver disease, and exercise should be strongly encouraged. Exercise plus adequate dietary protein consumption is necessary for maintenance of muscle mass, and inactivity/immobilization should be avoided.

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NAFLD AND THE METABOLIC SYNDROME – ROLE OF NUTRITION IN THERAPY AND PREVENTION

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Abstract Body: NAFLD is a major cause of liver disease throughout the world. It is currently considered as the hepatic manifestation of the metabolic syndrome and reflects a wide spectrum of liver diseases ranging from rather benign steatosis to steatohepatitis, cirrhosis and hepatocellular carcinoma. NAFLD is thought to develop through the interaction of various metabolic, genetic and environmental factors such as dietary and nutritional factors, and includes a complex and yet poorly understood interaction between the intestinal microbiota and the innate immune system.

Effects of food. Western diets have been shown to affect immunity, promote inflammation, and alter the microbiota via different pathways, with major health implications. The Western diet might have several features that contribute to its inflammatory effects—especially high fat content. Fatty acids promote inflammation through diverse mechanisms, including direct actions on immune cells, toll-like receptors, and cytokine signaling, as well as affecting intestinal permeability. Even in healthy subjects, a high-fat Western diet is correlated with endotoxemia and may contribute to a state of systemic low-grade inflammation including NASH. A high-fat diet (HFD) induces intestinal inflammation, increases ileal production of tumor necrosis factor and adiposity/NAFLD. The Western diet is commonly enriched in polyunsaturated fatty acids such as n−6. A diet rich in polyunsaturated fatty acids results in intestinal inflammation in mice, demonstrated by increased influx of neutrophils and macrophages. A 3-week diet of saturated milk-derived fatty acids, but not polyunsaturated fat or a low-fat diet, increased formation of taurin-conjugated bile acids in Il10−/− mice which develop spontaneous colitis. Such diets lead to the expansion of the low-abundance, sulphite-reducing pathobiont Bilophila wadsworthia. Mono-colonization of germ-free IL10-deficient mice with B wadsworthia led to colitis in association with the consumption of milk fatty acids. These findings support the association of certain dietary fats contained in Western diets with dysbiosis and development of NAFLD and colitis, and indicate the diet-induced expansion of certain pathobionts. Therefore, unhealthy lifestyles may play a major role in the development and progression of NAFLD.

Dietary approaches. Weight loss of at least 7% body weight has been demonstrated to beneficially affect liver disease. Nutritional counseling therefore should focus on such treatment targets. Increasing evidence suggests that beyond weight loss administration...
of several macronutrients, including omega-3-monounsaturated fatty acids intake and a reduction in carbohydrate consumption, particularly fructose is beneficial. Substantial weight loss as achieved by bariatric surgery has been demonstrated to affect beneficially metabolism and NAFLD, and especially improves established NASH. Weight loss eliminates expression of pro-inflammatory cytokines both in adipose tissue and liver. In conclusion, diet and nutritional aspects play a fundamental role both in pathogenesis and treatment of this prototypic food-mediated liver disease.

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IMPACT OF CIRRHOSIS ON METABOLISM AND BODY COMPOSITION

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Abstract Body: Liver cirrhosis is frequently associated with protein-energy malnutrition independently of the etiology of the liver disease. The most prominent feature of malnutrition in cirrhosis is muscle wasting ("sarcopenia", as assessed by CT-scan on L3 level, upper arm anthropometry or DEXA) or loss of body cell mass (as assessed by Bioelectrical Impedance Analysis) accompanied by expansion of the extracellular fluid compartment. The pathogenesis of muscle wasting in cirrhosis is multifactorial including low energy and protein intake, malabsorption, low glycogen storage, and chronic inflammation due to portal hypertension, increased intestinal permeability, and small intestinal bacterial overgrowth. In addition alterations of the endocrine system such as insulin resistance, low IGF-1 and low testosterone levels, and physical inactivity might also play a role.

In 30-35 % of patients the resting energy expenditure (REE) is increased when compared with predicted values according to Harris & Benedict. The overall protein turnover has found to be normal or increased and results from increased protein degradation and/or reduced protein synthesis. Increased protein degradation is particularly present after prolonged (overnight) fast due to the low glycogen storage. In this situation the body utilizes glucogenic amino acids from muscle breakdown for gluconeogenesis. The clinical relevance of the latter finding has been proven by studies using late-evening snacks showing preservation of muscle mass. The protein demand of patients with liver cirrhosis to reach nitrogen balance is increased when compared with healthy subjects.

Insulin resistance and glucose intolerance are also frequent findings in liver cirrhosis further altering muscle metabolism. In the fasting state glucose oxidation is impaired, and lipid oxidation is increased. In the postabsorptive state the plasma clearance and the lipid oxidation rate are normal indicating a normal utilization of fatty acids. Malnutrition, on the other hand, deteriorates liver function and clinical outcome of these patients even after liver transplantation. Recent data suggest that sarcopenia is an independent risk factor for poor survival in patients on the transplantation waiting list. Sarcopenia is also a risk factor for the development of liver fibrosis in NAFLD.

References


**Disclosure of Interest:** None Declared
MOLECULAR MECHANISMS AND MOLECULAR BIOLOGY OF SARCOPENIA IN LIVER DISEASE

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Abstract Body: Malnutrition in liver disease is consistently the most prevalent complication in liver disease that adversely affects clinical outcomes. However, despite nearly universal recognition of the high clinical significance, there are not effective therapies because the focus has been on deficiency replacement rather than targeting the underlying mechanisms. We have previously reported that malnutrition in liver disease includes sarcopenia and altered energy metabolism of which skeletal muscle loss or sarcopenia is the major component. Skeletal muscle mass is maintained by a balance between protein synthesis, proteolysis (proteostasis or protein homeostasis) and the regenerative potential of satellite cells. We have used a comprehensive array of models including skeletal muscle from patients with cirrhosis and controls, the portacaval anastomosis rat, hyperammonemic mice and in vitro cellular models of hyperammonemia to identify the molecular pathways and therapeutic targets to reverse sarcopenia in liver disease. We initially identified that hyperammonemia transcriptionally upregulates myostatin via a p65NFkB mediated mechanism by studies in the myostatin knockout mice and the PCA rat complemented by molecular studies in murine C2C12 myotubes. Blocking myostatin reversed the downstream molecular perturbations and increased protein synthesis and muscle mass. We also have shown that the classical ubiquitin proteasome pathway is not activated in the skeletal muscle in liver disease and during hyperammonemia. Instead, there is activation of autophagy with proteolysis. These molecular perturbations are complemented by and probably initiated by the metabolic consequences of hyperammonemia. Ammonia disposal in the skeletal muscle is mediated by cataplerosis of \( \alpha \)-ketoglutarate (\( \alpha \)-KG) a critical TCA cycle intermediate. Cataplerosis was associated with impaired mitochondrial function, increased reactive oxygen species due to leak of electrons from Complex III of the electron transport chain and consequent oxidative stress in the muscle. Supplementation with a cell permeable \( \alpha \)-KG analog replenished the intermediates and increased ATP content but worsened mTORC1 signaling and protein synthesis. We identified that the imbalance between anaplerosis and cataplerosis results in poor responses to supplementation with cell permeable TCA cycle intermediates. To overcome this limitation, we then dissected the molecular responses to branched chain amino acids, L-leucine and L-isoleucine as anaplerotic substrates and molecular activators of mTORC1. A potential reason for impaired L-leucine response is mitochondrial sequestration and consequent decreased effect on mTORC1 signaling. Deficiency of L-leucine and potentially isoleucine response
results in activation of the eIF2 kinase, general control non-derepressed 2 (GCN2).

Integrated molecular and metabolic in vivo studies in humans complemented by in vitro studies in myotubes showed that L-leucine supplementation reversed the molecular and metabolic perturbations in cirrhosis and hyperammonemia. Interestingly, unlike the classical response to phosphorylation of the global translational regulator, eIF1α, the integrated stress response mediated via the ATF4-GADD34 was not observed during hyperammonemia. We have termed this response as a hyperammonemic stress response that results in upregulation of leucine transporter, SLC7A5 with increased L-leucine uptake by the muscle and potentially, L-isoleucine. Our mitochondrial and metabolic studies also provide innovative data that glucose and fatty acid supplementation do not alter the mitochondrial function that we believe is due to impaired pyruvate dehydrogenase and fatty acid oxidation, necessitating branched chain amino acid catabolism as a source of energy generation. Potential mediators of altered oxidative metabolism include ammonia-induced activation and nuclear translocation of hypoxia inducible factor 1α. We conclude that hyperammonemia is a mediator of the liver muscle axis and targeting the molecular and metabolic perturbations and high dose administration of branched chain amino acids as a nutriceutical and signaling molecules hold potential to reverse sarcopenia and muscle dysfunction in liver disease. A number of other perturbations including reduction in testosterone, increased circulating cytokines and endotoxemia also contribute to sarcopenia and may target the myostatin, mTORC1 and eIF2 pathways.

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HOW TO FEED THE PATIENT WITH CIRRHOSIS?

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Abstract Body: Poor Spontaneous Food Intake

Many malnourished cirrhotic patients are anorexic and cannot meet their nutrient requirements by oral intake „ad lib“.

Control group data from an intervention trial showed that spontaneous food consumption was below recommended daily intake with an energy intake not even matching resting energy expenditure. In patients with chronic alcoholic liver disease, the magnitude of daily caloric intake in general has been shown positively correlated with survival.

Spontaneous protein intake in several intervention trials was well below recommended values (ESPEN guidelines recommend 1.2-1.5 g·kg⁻¹·d⁻¹) and the positive effect of nutrition therapy on survival was most obvious in those trials in which controls had the lowest spontaneous intake.

As a consequence of longstanding negative nutrient balance and the metabolic alterations of cirrhosis patients are depleted in hepatic glycogen and after an overnight fast, they are in a metabolic situation comparable to a healthy person starved for three days. Without food they start to maintain blood glucose from gluconeogenesis using amino acids from breakdown of muscle protein.

Micronutrient deficiency

Deficiency of fat-soluble vitamins is observed in LC patients with steatorrhea due to cholestasis and bile salt deficiency and in alcohol abusers. Both, vitamin A and zinc supplementation may indirectly alter nutritional state by improving gustatory function and thereby probably also volitional food intake.

A depletion in water soluble vitamins is not uncommon in cirrhosis, particularly in alcohol induced cirrhosis. Alcohol abusers are at particular risk for vitamin B1 deficiency which may be unmasked during carbohydrate refeeding and lead to lactic acidosis or Wernicke’s encephalopathy unless vitamin B1 has been given preemptively.

Zinc and selenium deficiency have been observed in both alcoholic and non-alcoholic liver disease.

Treatment Strategies

1. Patients without encephalopathy (HE)

In patients with clinically stable LC, an intake of 1.3 x REE or 30-35 kcal·kg⁻¹·d⁻¹ total energy including 1.2 g·kg⁻¹·d⁻¹ of protein is recommended for maintaining body composition and protein status. If the patient cannot eat such a quantity of food, then
standard oral nutritional supplements (ONS) offer the opportunity to provide the patient with additional energy and protein. In order to make full use of the protein consumed, patients should be counseled to eat a late evening meal or to drink ONS late in the evening and at night because nocturnal feeding has been shown to result in greater accretion of total body protein than daytime ONS. A multivitamin preparation and oral zinc should be given routinely as deficiency of these micronutrients is common.

In malnourished LC patients requiring repletion, more protein (1.5 g·kg⁻¹·d⁻¹) should be given. In such patients, enteral feeding of a high density standard formula via nasogastric tube is recommended. Tube feeding has been shown to improve liver function, mental state and survival without an undue risk of variceal bleeding. In these patients, low grade HE (I-II°) is not a contraindication to an adequate protein supply.

If patients require parenteral nutrition (PN) then this can be supplied by standard solutions preferably of a high nutrient density. As hepatic glycogen stores are depleted cirrhotics who can be fed sufficiently either by the oral or enteral route but who have to abstain from food temporarily (including nocturnal fasting!) for more than 12 hours, should be given hypocaloric PN or i.v. glucose. When this fasting period lasts longer than 72 h total PN is required.

2. Patients with encephalopathy (HE)
Adequate nutrition per se counteracts HE. In patients with HE as their main problem, other precipitating causes should be excluded before considering the patient protein-intolerant. Apart from this very rare condition, even transient protein restriction is not beneficial. In proven protein-intolerant patients, oral supplementation with branched-chain amino acids (BCAA) may be helpful in achieving an adequate nitrogen intake. PN is only indicated, when oral or enteral nutrition are not possible.

Patients in coma (HE III-IV°) can safely be given total PN regimens providing 30-35 kcal·kg⁻¹·d⁻¹ total energy including 1.0 g·kg⁻¹·d⁻¹ using BCAA-enriched amino acid solutions. The improvement of HE by BCAA is not necessarily a result of better nutrition alone. BCAA improve mental state in patients with HE provided that liver function does not further deteriorate and major clinical complications are absent. The use of BCAA-enriched solutions has no effect on survival.

3. Perioperative parenteral nutrition in chronic liver disease
Cirrhotic patients benefit from immediate postoperative nutrition and, in the absence of HE, there is no need to use BCAA-enriched rather than conventional amino acid solutions. Most likely, early enteral nutrition may be at least as effective as parenteral nutrition.
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Disclosure of Interest: None Declared
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ePOSTER ABSTRACTS
DIAGNOSTIC PERFORMANCE OF FIBROMAX – FIBROTEST, STEATOTEST AND ACTITEST – IN PATIENTS WITH NAFLD USING THE SAF SCORE AS HISTOLOGICAL REFERENCE

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Introduction: Blood tests of liver injury are less well validated in non-alcoholic fatty liver disease (NAFLD) than in patients with chronic viral hepatitis.

Aims: The aim was to improve the validation according to SAF scoring system (Bedossa et al. 2012) of three blood tests used in NAFLD patients, FibroTest for fibrosis staging, SteatoTest for steatosis grading and ActiTTest for inflammation activity grading (FibroMax panel).
Material and Methods: We pre-included new NAFLD patients with biopsy and blood tests from a single-centre cohort (FibroFrance) and from the multicentre FLIP consortium. Contemporaneous biopsies were blindly assessed using the new steatosis, activity and fibrosis (SAF) score, which provides a reliable and reproducible diagnosis and grading/staging of the three elementary features of NAFLD (steatosis, inflammatory activity) and fibrosis with reduced interobserver variability. We used nonbinary-ROC (NonBinAUROC) as the main endpoint to prevent spectrum effect and multiple testing.

Results: A total of 600 patients with reliable tests and biopsies were included. The mean NonBinAUROCs (95% CI) of tests were all significant (P < 0.0001): 0.878 (0.864–0.892) for FibroTest and fibrosis stages, 0.846 (0.830–0.862) for ActiTest and activity grades, and 0.822 (0.804–0.840) for SteatoTest and steatosis grades. FibroTest had a higher NonBinAUROC than BARD (0.836; 0.820–0.852; P = 0.0001), FIB4 (0.845; 0.829–0.861; P = 0.007) but not significantly different than the NAFLD score (0.866; 0.850–0.882; P = 0.26). FibroTest had a significant difference in median values between adjacent stage F2 and stage F1 contrarily to BARD, FIB4 and NAFLD scores (Bonferroni test P < 0.05).

Conclusions: In patients with NAFLD, SteatoTest, ActiTest and FibroTest are non-invasive tests that offer an alternative to biopsy, and they correlate with the simple grading/staging of the SAF scoring system across the three elementary features of NAFLD: steatosis, inflammatory activity and fibrosis.

ASSOCIATION BETWEEN MALNUTRITION IN PATIENTS WITH LIVER CIRRHOSIS AND THE PRESENCE OF OVERT HEPATIC ENCEPHALOPATHY

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Introduction: Hepatic encephalopathy is defined as a spectrum of neuropsychiatric abnormalities observed in patients with liver dysfunction, where malnutrition could play a role. Phase angle is a useful tool indicating the balance between cell hydration and mass, low values of phase angle reflect poor nutritional status, mainly reflecting cachexia which is the type of malnutrition seen in chronic diseases, which is a state of depletion of both muscle and fat mass as well as inflammation.

Aims: We aimed to evaluate the association between phase angle and the development of hepatic encephalopathy in the long-term follow-up of cirrhotic patients.

Material and Methods: This was a prospective cohort study with 48 months of established follow-up. Clinical (presence of esophageal varices, ascites, edema, and hepatic encephalopathy), nutritional (bioelectrical impedance derived PhA) and biochemical evaluations were performed. Overt HE was assessed with West-Haven criteria. Malnutrition was defined as PhA \(<4.9^\circ\). Student T test or X\(^2\) method were used as appropriate. Kaplan-Meier curves and Cox regression were used to evaluate the incidence of hepatic encephalopathy.

Results: 220 patients were included; the mean follow up was 34 ± 9.8 months, the most frequent etiology of cirrhosis was hepatitis C infection, 52% of the patients developed hepatic encephalopathy (18.6% covert and 33.3% overt); the main precipitating factors were infections and variceal bleeding. Kaplan-Meier curves showed a higher proportion of HE in the group with low phase angle (39%) compared to the normal phase angle group.
(13%) (p=0.012). Furthermore, creatinine and phase angle remained independently associated to hepatic encephalopathy in the Cox regression multivariate analysis [HR=1.80 (1.07-3.03)].

Conclusions: In our cohort of patients low phase angle was associated with an increased incidence of hepatic encephalopathy. Phase angle is a useful nutritional marker that evaluates cachexia and could be used as a part of the integral assessment in patients with cirrhosis.

Figure:

Disclosure of Interest: None Declared
SERUM MYOSTATIN PREDICTS SURVIVAL IN PATIENTS HOSPITALIZED FOR DECOMPENSATED ADVANCED CHRONIC LIVER DISEASE IN UNEXPECTED MANNER

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Introduction: In pts with decompensated advanced chronic liver disease ([d]ACLD), sarcopenia (S) is associated with mortality; to diagnose S in dACLD is difficult and to slow it even more so. Under physiological conditions, TFG-beta superfamily member myostatin (M) inhibits myogenesis. Findings that M is markedly increased in ACLD pts as compared to controls led to hypothesis that measuring M could be of value in diagnosing S and in refining prognostic stratification; specifically, levels of M in dACLD were expected to rise with deepening of decompensation.

Aims: To evaluate if M could be of prognostic value in dACLD

Material and Methods: Retrospective study on consecutive inpatients. Inclusion criterion: dACLD. Exclusion criteria: Malignancy, insufficient data for analysis. Recorded variables: demographics, antropometry, handgrip strenth, etiology of ACLD, MELD, Child-Pugh score, serum M levels (quantitative sandwich EIA; R&D Systems Europe, Ltd). Primary endpoints: survival at days 40 and 90. Predictors of survival were analyzed by AUROC and regression analysis.

Results: During study interval of 18 months, 253 pts with dACLD were enrolled, of median age 54,3 years (y); 111 were female; median MELD was 16,7, Child-Pugh 9,5. Median serum M was 1951 pg/ml (IQR 1006-3715). At days 40 and 90, 19 and 29 pts have not been alive, respectively (3 underwent LTx). Median M in survivors at day 40 was 2070 vs. 931 in non-survivors (p=0.001); at day 90, M in survivors was 2048 vs. 1188 in nonsurvivors (p=0.0798). Serum M below 1460 predicted mortality at day 40 with 84% sensitivity and 64% specificity, AUROC=0.726 (0.61-0.84). Stepwise logistic regression
model including M, MELD, CRP and hepatic encephalopathy (HE) identified M<1460 (OR=15.8), MELD (1.08) and HE (4.04) as independent predictors of mortality or LTx at day 40 (AUROC=0.906, p<0.0001). In subgroup of 100 pts with M<1460 pg/ml mortality was 16% as compared to 1.96% in 153 pts with M=>1460 (p<0.0001).

Conclusions: Contrary to expectation, in pts hospitalized for dACLD, lower (<1460 pg/ml) rather than higher M levels were associated with worse outcome; low level of M was found to be an independent predictor of death at day 40.

Disclosure of Interest: None Declared
ASSESSMENT OF SARCOPENIA IN PATIENTS WITH CHRONIC LIVER DISEASE

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Introduction: Malnutrition occurs in 50 – 90% of patients with chronic liver disease (CLD). The aetiology is multifactorial with the reported incidence dependent on nutritional assessment methods used. Subjective global assessment (SGA) is reported to accurately assess nutritional status in patients with CLD. Sarcopenia is characterised by muscle wasting and has been demonstrated in numerous diverse groups including CLD and is an important predictor of survival in patients with CLD.

Aims: To investigate the incidence of sarcopenia and the relationship between SGA and sarcopenia in a population assessed for liver transplantation between 2012 and 2014.

Material and Methods: Severity of liver disease was assessed using both the Child-Turcotte-Pugh (CTP) and the Model for End-stage Liver Disease (MELD) scores. Psoas muscle area and lateral abdominal wall muscle were assessed using CT imaging at the level of L3. SGA scores were determined by trained dietitians. BMI was adjusted for the presence and severity of ascites and obesity was defined by BMI > 30 kg/m\(^2\).

Results: Two hundred and five patients with CT imaging were included. Mean age was 52.8 years and 70% were male. Eighty six percent were sarcopenic and were more likely to be male (p<0.0001). There was no significant difference in MELD score and age between sarcopenic and non-sarcopenic groups. Sarcopenic patients were more likely to be encephalopathic (p = 0.033). The incidence of sarcopenia varied with SGA score (p <0.0001). Sarcopenia was identified in 90% of severely malnourished (SGA C) patients, in 67% of moderately malnourished (SGA B) patients and in 65% of well nourished (SGA A) patients. Forty-seven individuals were obese and sarcopenic with males patients over-represented in the obese sarcopenic group (83% p<0.0001) with a significant correlation (-0.29) between sarcopenia and testosterone levels (p=0.001). We have shown for the first time a significant correlation between the psoas muscle area, the anterior abdominal wall...
muscle (Pearson’s $r=0.32$, $p<0.0001$) and the lateral abdominal wall muscle (Pearson’s $r=0.46$, $p<0.0001$).

**Conclusions:** Sarcopenia is common in patients with CLD. As patients who were classified as obese or well-nourished may be sarcopenic, assessment of muscle mass is important in these individuals. The correlation of the classical psoas muscle area with anterior and lateral abdominal wall measures will enable less invasive assessment of sarcopenia using simpler modalities such as ultrasound.

**Disclosure of Interest:** None Declared
NUTRITIONAL WHEAT AMYLASE TRYPsin INHIBITORS, ACTIVATORS OF INTESTINAL TOLL LIKE RECEPTOR 4, EXACERBATE NON-ALCOHOLIC STEATOHEPATITIS IN HIGH FAT DIET FED MICE

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Introduction: Non-alcoholic steatohepatitis (NASH) is now ranked the most prevalent liver disease worldwide with an escalating demand for liver transplantation over the next decade. Apart from lifestyle changes and pharmacological strategies, specific (micro-) nutrients may play an important role in NASH pathogenesis. A possible candidate is the family of what amylase trypsin inhibitors (ATIs) that represent 3% of wheat protein. ATIs are highly resistant to digestive proteolysis and activate intestinal innate immunity via toll like receptor 4 on monocytes, macrophages and dendritic cells (Junker Y et al, J Exp Med 2012).

Aims: The effect of nutritional ATIs (equivalent to human average wheat ingestion) on the severity of diet induced NASH in mice

Material and Methods: Male C57BI/6J mice received a carbohydrate and protein (zein from corn, 22.1% of weight defined low or high fat diet (HFD, 53KJ% vs 13 KJ% of calories as saturated fats), with or without replacing 30% of the zein isocalorically by crude wheat gluten (containing approx.1g ATIs per 10g gluten) or 0.7% of the zein replaced by purified ATIs for 8 weeks. At sacrifice blood, liver and peripheral adipose tissues were collected for biochemical and histological analysis. Tissues were quantified for lipid content, inflammation, and inflammation related transcript levels were quantified by qPCR. Macrophage subsets were quantified by IHC.

Results: Compared to the HFD alone, mice fed the HFD/G/ATI or the HFD/ATI diets gained 10% and 15% more weight respectively and displayed significantly higher serum ALT, triglycerides, and hepatic, epididymal, mesenteric and inguinal fat. The
IPGTT revealed a significantly higher glucose intolerance in mice on the HFD/ATIs vs HFD alone. Compared to the HFD alone, mice on the HFD/G/ATI and HFD/ATI diets had a significantly higher histological NAS score. Transcript levels of CD68 (total macrophages) and IL-6 were increased significantly both in the liver and epididymal fats whereas alternative macrophage (and putatively anti-inflammatory) transcripts for ARG1 and CD206 were decreased in liver. This was confirmed histologically via elevated CD68+ liver macrophages in the HFD/G/ATI and HFD/ATIs fed animals. Finally, H&E stained epididymal adipose tissue revealed both significantly enlarged epididymal adipocytes and significantly increased Crown like structure (indicating inflammation) in HFD/ATI fed mice vs HFD alone.

**Conclusions:** Our study implicates dietary wheat ATIs as proinflammatory nutritional drivers of NAFLD/NASH.

**Disclosure of Interest:** None Declared
THE IMPACT OF NUTRITIONAL STATUS ON SURVIVAL IN CIRRHOTIC PATIENTS WITH SEPSIS

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Introduction: Severe infections and protein malnutrition (PM) represent two entities associated with a poor outcome in cirrhosis. In general population, a poor nutritional status has been associated to an increased susceptibility to infections due to the immune-modulatory functions attributed to leptin, and conversely, the inflammatory state related to sepsis may induce a catabolic state causing the malnourished state. No studies have analyzed a possible negative synergetic effect of sepsis and malnutrition in cirrhosis.

Aims: We aimed at analyzing the prognostic effect of PM in patients with sepsis.

Material and Methods: All consecutive cirrhotics with sepsis hospitalized admitted from 2011 to 2015 were enrolled. Demographical and clinical data were recorded, paying particular attention to the infectious history and the nutritional status. Patients were classified according to Child-Pugh class. A diagnosis of protein malnutrition (PM) was made when patients have a MAMC < 5\textsuperscript{th} percentile.

Results: Seventy-four patients with sepsis (71\% males; median age 64yars) were enrolled. PM was diagnosed in 43\% of patients. Severity of liver disease and characteristics of infection were not different in patients with and without PM. Mortality tended to be higher in PM (47\% vs 26\%, p=0.059). A multivariate analysis selected PM (p=0.0015, OR 3.2, IC: 1.4-4.8) and Child-Pugh C (p=0.001, OR 3.3, IC: 1.5-4.9) as independent predictors of in-hospital mortality. A stratified analysis according to the Child-Pugh class, showed that in patients with Child-Pugh C (29 patients) no differences were observed in in-hospital mortality according to the presence of PM or not neither any difference was found in other portal-hypertension complications; on the other hand, in Child-Pugh A-B (45 pts), an higher rate of mortality (50\% vs 16\%; p=0.01) was found in patients with PM compared with those without PM.
The mortality rate and the incidence of complications in malnourished patients classified in Child-Pugh class A/B appeared similar to those classified as Class C (63% vs 53%, p=0.56).

**Conclusions:** Our study confirms a strong influence of PM on sepsis outcome in cirrhotic patients and this is particularly evident in patients with mild-moderate cirrhosis, whereas is lost in patients with more advanced liver disease.

**Disclosure of Interest:** None Declared
TASTE SENSITIVITY AND FOOD PREFERENCES IN TERMINAL-STAGE OF CIRRHOTIC PATIENTS

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Introduction: Protein-energy malnutrition is associated to cirrhosis with a high prevalence and greatly increases morbidity and mortality of the disease. Malnutrition is the result of numerous factors including intestinal malabsorption, increased energy resting expenditure, metabolism disturbances, impaired sensory taste and changes in food preferences which might participate to a reduction in food intake.

Aims: The aim of this study was to determine the relationships that may exist between taste sensitivity and food preferences on the one hand, and eating habits, nutritional status, energy balance and biological parameters including amino acid profile on the other hand in patients with end-stage cirrhosis.

Material and Methods: Ten end-stage cirrhotic patients were included and matched with 10 healthy subjects. The following parameters were evaluated during a morning testing session: gustatory sensitivity using a triangular detection threshold method for sweet, salty and umami solutions; preferences for fat- and carbohydrate-rich foods using the PrefQuest score; liking for six foods (protein-, carbohydrate- and fat-rich foods) and wanting for 18 foods (photographs); hunger sensation; energy intake (24 h ingested food); body composition (BMI and impedancemetry); resting energy expenditure (indirect calorimetry) and physical activity (questionnaire). Several plasmatic parameters (amino-acids, leptin, ghrelin, lipid profile) were also determined.

Results: All the patients suffered from severe malnutrition. Sensitivity for sweet and salty tastes was impaired (P<0.01) and a greater preference for fatty foods (P<0.05) was noted in cirrhotic patients compared to healthy subjects, while resting energy expenditure, oral intake, body composition, physical activity or eating habits were similar. The amino-acid
profile in cirrhotic patients was different from that of heathy subjects (e.g. tryptophan and branched-chain amino acids, p<0.01), and related to sensitivity and preferences according to principal component analyses.

**Conclusions:** In this pilot study, changes in taste sensitivity thresholds and food preferences were observed in cirrhotic patients, which could be related to changes in amino-acid profile. The amino acid profile appears to be one of the most important determinant leading to eating and sensitivity disorders, at the origin of PEM.

**Disclosure of Interest:** None Declared
OSTEOPONTIN ABLATION DRIVES HEMATOPOIETIC STEM CELL MOBILIZATION AND INCREASES HEPATIC IRON CONTRIBUTING TO ALCOHOLIC LIVER DISEASE

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Introduction: Although it has been shown that hematopoietic stem cells (HSCs) restore injured parenchyma, the continuous mobilization of bone marrow-derived HSCs into the liver could contribute to alcoholic liver disease (ALD). To date, the mechanisms underlying HSCs mobilization from the bone marrow (BM) to the liver and its contribution to ALD remain poorly defined. Osteopontin (OPN) is a matricellular protein and a negative regulator of HSC proliferation that controls HSC lodgment and retention.

Aims: To investigate the role of OPN in restricting HSCs mobilization into the liver and its contribution to ALD.

Material and Methods: We analyzed young (14-16 wks.) and old (>1.5 yrs.) wild-type (WT) littermates and global Opn knockout (Opn−/−) mice for the presence of BM mobilization into the liver. In addition, 10 wks. old WT and Opn−/− mice were chronically fed with either the control or the ethanol Lieber-DeCarli diet for 7 wks. BM, blood, spleen and liver were analyzed by flow cytometry for HSC progenitors and neutrophils. Chemokines, growth factors and cytokines were measured in serum and liver. H&E staining and scoring, Prussian blue staining for ferric iron deposits and Naphthol AS-D chloroacetate esterase staining for neutrophils were performed in liver sections.

Results: Myeloid progenitor cells, CD34+ and CD127+ hematopoietic cells were lower in the BM from young compared to old Opn−/− mice, which displayed significant hepatosplenomegaly compared to their age-matched WT littermates. Opn−/− mice had significantly elevated Kitl mRNA levels albeit similar expression of Cxcr4 and Sdf-1α mRNAs. Hepatic chemokine (C-X-C motif) ligand 1 (CXCL1), macrophage inflammatory...
protein 1 alpha (MIP1\alpha) and chemokine (C-C motif) ligand 5 (CCL5) as well as granulocyte colony-stimulating factor (G-CSF) and macrophage colony-stimulating factor (M-CSF) were increased in $Opr^+$ mice suggesting potential engraftment of HSCs from the BM into the liver. Furthermore, $Opr^+$ mice had increased $Epo$ and decreased $Hamp$ mRNA expression, which are central players in iron homeostasis and iron uptake. Indeed, $Opr^+$ mice showed iron deposits in the liver and in the BM but were absent in WT littermates. Ethanol-fed $Opr^+$ mice showed significant hepatic neutrophil infiltration and hemosiderin compared to WT mice. As a result, ethanol feeding caused greater liver injury in $Opr^+$ compared to WT mice.

Conclusions: Opn ablation promotes HSC mobilization, neutrophil infiltration and iron deposits in the liver; therefore, enhancing the severity of ALD.

VISCERAL FAT EVALUATED BY ULTRASOUND IS ASSOCIATED WITH INSULIN RESISTANCE AND LIVER DAMAGE IN PATIENTS WITH NON-ALCOHOLIC FATTY LIVER DISEASE

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Introduction: Non-alcoholic fatty liver disease (NAFLD) is the most common chronic liver disease worldwide and has been regarded as the hepatic manifestation of metabolic syndrome (MetS). Insulin resistance is a hallmark of MetS and is considered to play a pivotal role in NAFLD progression. Visceral adiposity is associated with insulin resistance and visceral adiposity tissue (VAT) area evaluated by TC has been reported to be independently associated with NASH or significant fibrosis in NAFLD patients. Ultrasound examination (US) is an easy and safe technique able to detect steatosis and VAT. NAFLD fibrosis score (NFS) has been proposed as a non-invasive tool for the assessment of liver fibrosis in NAFLD. So far, there are no data regarding the relationship between VAT evaluated by US, insulin resistance and severity of liver damage in adult patients with NAFLD.

Aims: Aim of this study was to investigate the relation between VAT, insulin-resistance and liver damage in adult patients with NAFLD.

Material and Methods: The study has been performed in 176 consecutive patients referred for suspected metabolic disease and with a liver US scanning positive for NAFLD. Liver steatosis severity was defined according to Hamaguchi US criteria. All subjects underwent routine clinical and biochemical evaluation. VAT was determined during US exam. NFS was calculated to assess severity of fibrosis. A NFS was considered to be low when < -1.455 and intermediate/high when ≥ -1.455.

Results: Mean age was 55.8 (± 13.5) years and 36.9% of patients were female. Prevalence of MetS and diabetes were 61.6% and 29.9%, respectively. Median VAT was 6.1 (5.1/7.6)
cm and 32.6% of patients had severe hepatic steatosis. Median HOMA-IR was 3.4 (2.3/5.3). Median VAT was significantly higher in patients with intermediate/high NFS than in those with low NFS (6.5 (5.4/7.7) vs 5.7 (4.5/6.8) cm, p=0.013). Linear bivariate regression analysis showed a positive correlation between VAT and HOMA-IR (r=0.42; p<0.001), Hamaguchi score (r=0.47; p<0.001), ALT (r=0.38; p<0.001), AST (r=0.27; p<0.001) and NFS (r=0.19; p=0.026). In a multiple linear regression analysis, VAT (B=0.35; p=0.03) and age (B=0.16; p<0.001) were independently associated with NFS.

**Conclusions:** Our data show a positive correlation between VAT evaluated by US, insulin resistance and liver damage in a population of NAFLD patients. Our results suggest that evaluation of VAT during US exam may be useful to identify NAFLD patients with a more severe liver disease.

**Disclosure of Interest:** None Declared
POTENTIALITY OF FLAVONOIDS CONSUMPTION IN PATIENTS WITH NONVIRAL CHRONIC HEPATITIS

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Introduction: Flavonoids are compounds of plant origin, which are present in variety of fruits and vegetables: apples, grapes, nuts, onions, tomatoes, and in medicinal botanicals, including Ginkgo biloba, Hypericum perforatum, Sambucus canadensis. Flavonoids have high antioxidant activity, which is most pronounced in Quercetin, stimulate protein synthesis, regulate phospholipid metabolism.

Aims: The aim of the study was to examine the Quercetin effect on the results of treatment of nonviral chronic hepatitis (CH) patients.

Material and Methods: The study involved 41 patients with nonviral CH aged from 22 to 75 (51.3 ± 14.5) years. According to the received treatment, patients were divided into two groups. The main group consisted of 21 CH patients, which in addition to standard treatment received Quertyn (quercetyn) (Borschagivsky Chemical-Pharmaceutical Plant) at a dose of 40 mg three times a day 30 minutes before meal for 14 – 16 days. The comparison group consisted of 20 CH patients, which received standard treatment. The control group consisted of 20 healthy volunteers. Written informed consent was obtained from all the participants.

The range of examined indicators included: total bilirubin and its fractions, cholesterol, triglycerides, uric acid, total protein and albumin, urea, creatinine, plasma enzyme activity (aspartate aminotransferase (AST), alanine aminotransferase (ALT), lactate dehydrogenase (LDH), gamma-glutamyl transferase (GGT), alkaline phosphatase (AP), catalase, glutathione-S-transferase and glutathione peroxidase), reduced glutathione and reactive products of tiobarbituric acid.

Results: In CH patients, which in addition to the standart treatment received Quercetin, celebrated faster regression of clinical symptoms of hepatitis. In these patients during treatment a significant decrease in the total bilirubin concentration by 27.0% (p<0.05),
cholesterol – by 10.2% (p<0.05) and triglycerides – by 22.1% (p<0.05), decrease in AST activity – by 19.4% (p<0.05), ALT – by 28.8% (p<0.05), LDH – by 15.5% (p<0.05), AP – by 25.1% (p<0.05), GGT – by 37.3% (p<0.05) were observed. Significant increase in the concentration of reduced glutathione was observed in these patients by 26.1% (p<0.05), catalase activity – by 20.4% (p<0.05).

Conclusions: Effeciant reduction of cytolytic, cholestatic syndromes and antioxidant systems restoration with the additional to standart treatment usage of Quercetin in chronic hepatitis patients were achieved.

Disclosure of Interest: None Declared
A COLLABORATIVE QUALITY IMPROVEMENT PROJECT TO IMPROVE EARLY NUTRITIONAL SUPPORT IN DECOMPENSATED CHRONIC LIVER DISEASE

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Introduction: Malnutrition is one of the commonest complications of chronic liver disease (CLD) regardless of aetiology. Appropriate nutritional assessment and timely intervention is a critical determinant of outcome in those hospitalized with decompensated CLD.

Aims: We designed a quality improvement project to identify gaps in specialist nutritional assessment/intervention at our centre, and tested the impact of specific service developments in addressing identified areas of suboptimal practice.

Material and Methods: An audit was conducted to establish baseline performance over 1 calendar month in key areas: rates of dietetic referral, timely referral (within 24hr of admission), timely review (within 24hr of referral) and rates of monitoring and follow-up. We then convened a multi-disciplinary team (MDT) of ward junior/senior medical staff, dietitians and nurses to discuss results and suggest improvements. Agreed actions were implemented and results measured and analysed, following which necessary adjustments were made prior to a final round of assessment.

Results: In the baseline audit (n=38), just 19 (50%) of the cohort were referred, of whom only 12 (63%) were referred within 24 hr and 5(26%) were seen within 24 hr of referral. 13 (68%) were monitored and 6 (31%) followed up weekly.

Actions agreed by the MDT to enhance performance were: referral within 24hr of admission for all patients; increase expected dietitian review time to within 72hr, as no routine dietetic service at weekends; and introduction of an online review form detailing review findings, monitoring criteria and planned follow up.

Following implementation, impact analysis (n=20) showed all patients were appropriately referred, but only 9 (45%) within 24 hr. 18 (90%) were reviewed within 72 hr, 17 (85%)
monitored and 19 (95%) followed up. Online review forms were considered easy to use and efficient. To address the poor rates of timely referral, education/awareness sessions were held with ward staff.

At the final assessment (n=10), performance had again improved; 100% were referred, with 7 (70%) of these within 24hr. We achieved 100% success in review within 72hr, monitoring and follow up.

**Conclusions:** A collaborative, iterative approach between doctors, dietitians and nurses led to marked improvements in early nutritional support and intervention in patients with decompensated CLD.

To determine whether these interventions impact long-term outcomes, mortality and readmissions of the cohort will be reviewed in 1 year.

**Disclosure of Interest:** None Declared
P3.1

COMPREHENSIVE NUTRITIONAL ASSESSMENT IN PATIENTS WITH REFRACTORY ASCITES PRIOR TO THE IMPLANTATION OF AN ALFAPUMP SYSTEM

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Introduction: Malnutrition and sarcopenia are common findings in cirrhotic patients with refractory ascites and sarcopenia is an independent risk factor for poor outcome.

Aims: The aim of this study is to perform a comprehensive nutritional assessment in patients that are scheduled for the implantation of an automated low flow ascites pump (alfapump®, Sequana Medical AG, Zurich, Switzerland) to identify nutritional deficiencies and potential targets for supplementation prior and after the implantation of the alfapump system.

Material and Methods: Data have been collected from August 2013 to August 2016 in a single centre in Switzerland. Inclusion criteria were liver cirrhosis with refractory ascites, scheduled implantation of an alfapump system, age >18 years, and signed informed consent. Following parameters were collected: BMI, hand grip strength (anthropometric data) and INR, creatinine, bilirubin, albumin, prealbumin, zinc, selenium, copper, 25-OH-vitamin D3, vitamin A, and vitamin E (serum parameters).

Results: In total, 22 consecutive patients were included (64% men); mean age was 59.5 years (range 44-81). Aetiology of cirrhosis was alcohol (54.5%), hepatitis C (13.6%), hepatitis B (9.1%), NASH (9.1%), ASH/NASH (4.5%), autoimmune hepatitis (4.5%), and cryptogenic (4.5%).

Median BMI (dry body weight) was 23.9 kg/m² (range 15-37.8), median hand grip force 19.6 kg (range 10.3-34.1). Median Child and MELD scores were 9 (range 8-12) and 13 (range 7-24), respectively. Median serum creatinine was 109.5 µmol/L (range 41-334, normal level <85f, <105m), bilirubin 22.5 µmol/L (4-74, normal level <17), INR 1.2 (1.0-
1.8), albumin 28.5 g/L (20-33, normal range 35-52), and prealbumin 0.08 g/L (0.04-0.22, normal range 0.2-0.4).

Median zinc level in serum was 6.9 µmol/L (range 4-10.2, normal range 11-18), selenium 0.6 µmol/L (0.4-0.9, normal range 0.8-1.5), copper 12.5 µmol/L (8.6-29.8, normal range 13-33), 25-OH-vitamin D3 20 nmol/L (12-55, normal range 50-134), vitamin A 0.4 µmol/L (0.1-2.0 normal range 1.05-2.8), vitamin E 18.3 µmol/L (11-39.4, normal range 12-42).

Conclusions: Patients with refractory ascites are characterized by reduced muscle strength and decreased prealbumin, zinc, selenium and 25-OH-vitamin D3 levels. Vitamin A deficiency is less frequent but may be severe, copper deficiency is, if present, usually mild. Selective substitution of micronutrients und vitamins in addition to caloric and protein supplementation may help to ameliorate malnutrition in patients with refractory ascites.

TRACE ELEMENTS IN DECOMPENSATED ALCOHOLIC LIVER CIRRHOSIS

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Introduction: The relationships between chronic liver diseases and trace elements are debated. There are studies suggesting that alterations in trace elements may have a role in advancement of liver cirrhosis and in developing complications.

Aims: Aim of our study was investigate levels of trace elements in patients with decompensated alcoholic liver cirrhosis.

Material and Methods: We analyzed serum levels of iron, zinc, copper, selenium and magnesium of patients with decompensated alcoholic liver cirrhosis (n=30) and compared them with healthy control group (n=30).

Results: We observed significant differences in levels of micronutrients in group of patients with alcoholic liver cirrhosis compared to healthy controls (p<0.05). Levels of serum zinc, selenium and iron were significantly lower with a higher level of serum copper in decompensated cirrhosis group than in control group (P < 0.05). Nevertheless, there was no significant difference of serum magnesium between groups.

Conclusions: Our data indicate that trace elements might have an important role in patients with an alcoholic liver cirrhosis, and can be used as potential biomarkers of an advanced disease. Additionally, substitution of these micronutrients potentially can be useful in prevention of an advanced disease.

Disclosure of Interest: None Declared
AGE-DEPENDENT CHANGES IN LIVER OF ADULT AND PUBERTAL RATS WITH METABOLIC SYNDROME

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Introduction: Lifestyle changes over the past decade led to the rapid spread of metabolic syndrome (MS) in different age groups. Particularly the anxiety causes this pathology rapid distribution among children and teenagers.

Aims: The aim of study was the comparative estimation of MS mediated changes in liver of adult and pubertal rats.

Material and Methods: 40 Wistar male rats of two age categories (young animals of 21 days age (50-70g) and adults (160-180g)) were used in the study. They were divided into 4 groups: 1 – control 1 (intact young rats), 2 – control 2 (intact adults), 3 – young rats with MS and 4 – adult rats with MS. MS was induced by 60 days full replacing of drinking water with 20% fructose solution. Blood serum clinical chemistry parameters characterizing liver state, hepatic CYP3A, CYP2C, CYP2E1 mRNA expression and corresponding enzymes activities were measured.

Results: Serum clinical chemistry parameters show a significant difference in MS manifestation in dependence on his induction in adult or puberty. In adult rats we observed 40% increase of total cholesterol and 2 fold LDL content as compared with control. Other parameters were within the control level. In rats with MS developed in puberty we recorded 41% increase of glucose, 43% triglycerides, and 5.2 folds total bilirubin, creatinine and urea contents decrease respectively 14 and 37%. In pubertal rats liver CYP2E1 mRNA expression increased 1.8 times compared with control. Expression of CYP2C23 mRNA (ortholog of CYP2C9 and CYP2C19) in livers of both age groups was reduced. MS significantly influenced expression of CYP3A2 (ortholog of CYP3A4) only in liver of pubertal rats. p-nitrophenol-hydroxylase (CYP2E1 marker) 1.6 fold increased in liver of pubertal animals with MS. However, changes of this enzyme in adult animals were comparatively modest (+37%). Erythromycin-N-demethylase (CYP3A2 marker)
activity in liver of pubertal rats decreased 3 times. In adult animals we noted its 46% increase. In rats of different ages MS provoked the reduction of CYP2C23 marker enzyme – diclofenac-hydroxylase activity.

**Conclusions:** Thus, serum clinical chemistry parameters characterizing liver injury were greatly violated simultaneously with CYP isoforms expression rates. The greater changes of investigated parameters were observed in pubertal rats with MS. Age differences could have a serious effect on the metabolism of medications and cause unexpected toxicities or reduced effectiveness of pharmacotherapy of MS.

**Disclosure of Interest:** None Declared
P3.4

DIETARY HABITS AND NUTRITIONAL STATUS OF PATIENTS WITH LIVER CIRRHOSIS: RESULTS FROM A CROSS SECTIONAL SURVEY

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Introduction: Nutritional status has emerged as an important prognostic factor for patients with liver disease. In order to correct or prevent malnutrition, knowledge of factors influencing food intake is important, since an adequate diet is the first step of nutritional intervention.

Aims: The aim of our study was to evaluate the frequency of malnutrition and dietary habits of patients with liver cirrhosis.

Material and Methods: We performed a one day cross sectional survey (Nutrition Day in Hospitals) to evaluate the nutritional status of inpatients from a tertiary Gastroenterology and Hepatology Center. We analyzed weight, height, fluid retention (ascites, edema), BMI/BMI for ascites, weight changes, amount of weight loss, dietary habits/food intake, causes of low intake, performance status. Patients with liver diseases (LD), all with liver cirrhosis of various etiologies were compared with patients with other diagnosis – gastrointestinal or pancreatic diseases (GID).

Results: 146 patients were included, with a mean age of 58.36+/−12.92 years, 55.48% were males. 58.9% (86 patients) had LD. While as defined by BMI for ascites respectively BMI the frequency of malnutrition was low and did not differ between LD and GID patients (10.84% vs 13.33%), a significantly higher proportion of patients with LD reported weight loss (p=0.021). 32.12% of all patients reported reduced food intake, similar in both groups; the cause of low intake in LD patients was primarily reduced appetite and/or early satiety (p=0.01), while pain was the cause reported by GID patients (p=0.0001). More patients from GID group used supplemental food from outside the hospital (p=0.039). A significantly higher proportion of LD patients complained of asthenia (p <0.0001), fatigue...
(p<0.0001) and needed assistance or were confined to bed (p=0.045). We observed a tendency toward a longer hospitalization for LD patients (p=0.09).

**Conclusions:** One third of patients with liver cirrhosis have low food intake. Loss of appetite and early satiety are the principal causes. Nutritional counselling, small and more frequent meals can overcome these symptoms and should be included in the therapeutic management of these patients in order to prevent malnutrition.

**Disclosure of Interest:** None Declared
AGGRESSIVE NUTRITION INTERVENTION CAN REDUCE ASCITES IN PATIENTS WITH CIRRHOSIS AND REFRACTORY ASCITES: A CASE SERIES REPORT

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Introduction: Malnutrition occurs in up to 75% of patients with chronic liver disease (CLD) as a consequence of disturbances in metabolic and haemodynamic factors including alterations in portal circulation and nutrient metabolism, increased nutritional requirements and reduced intakes. A common sequela of hepatic decompensation is ascites, with 50% of patients with decompensated cirrhosis developing ascites and up to 22% developing refractory ascites with a reduced 12-month survival.

Aims: This case series examines the effect of supplementary 24-hour nasogastric tube feeding in a group of patients with CLD who failed to improve with standard nutritional support.

Material and Methods: Twelve patients who were moderately or severely malnourished with decompensated cirrhosis who did not respond to standard nutrition interventions received supplementary 24-hour naso-gastric feeding for a mean of 6 weeks (range 1.5 – 10 weeks).

Results: Prior to supplementary 24-hr nasogastric feeding 11 patients were severely malnourished with a subjective global assessment (SGA) score of C and 1 was moderately malnourished (SGA B). Ten patients had severe ascites and 8 required regular large volume paracenteses. The mean model for end-stage liver disease (MELD) score was 15.7 (range 7-26) and mean Child-Turcotte-Pugh (CTP) was 10. Four patients were classed as CTP B and 9 as CTP C. The majority of feeding occurred at home with no clinical deterioration during this time. One patient received a liver transplant after 1.5 weeks of feeding. Eleven patients
completed at least 6 weeks of nasogastric feeding and at completion 9 patients no longer required paracentesis. Four patients had no ascites, 5 had mild ascites, and 2 had moderate ascites requiring less frequent paracenteses. Calculated body mass index (BMI) fell from 24.7 to 23.5. All patients improved nutritionally with 6 patients assessed as SGA B and 6 as SGA C with mean grip strength increasing from 51% to 64.5% of predicted. In addition, 6 patients dropped their MELD score by 2 or more points (range 2-6) and the mean MELD score fell from 16.1 to 14.5. Mean CTP fell from 10 to 9 with 2 patients classified as CTP A.

**Conclusions:** Supplementary 24-hr nasogastric feeding can reduce ascites and improve nutritional status with an associated improvement in disease severity and should be considered as a potential treatment option in malnourished patients with decompensated cirrhosis and refractory ascites who fail to respond to oral nutrition support.

CONTROLLED AND MOTIVATION PROGRAMME OF CHANGING EATING BEHAVIOR REDUCE WEIGHT AND CYTOKERATIN 18 LEVELS IN PATIENTS WITH NAFLD

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Introduction: Lifestyle modification and weight loss is a hallmark of managing Nonalcoholic fatty liver disease according to nowadays guidelines and researches. We know that weight loss above 7-10 % improves steatosis in patients with NAFLD. But still there is a gap how to increase patient adherence for changing eating behavior and effect of weight loss. According to this we have created motivational and controlled programme for changing eating behavior and investigate its role in weight loss in patients with NAFLD. Also we discover how this programme can improve hepatic apoptosis as a main predictor of Nonalcoholic fatty liver disease progression, which can be measured by biomarker cytokeratin 18.

Aims: Investigate role of motivational and controlled programme for changing eating behavior in weight loss in patients with NAFLD. Also we discover how this programme can improve hepatic apoptosis as a main predictor of Nonalcoholic fatty liver disease progression, which can be measured by biomarker cytokeratin 18.

Material and Methods: 40 patients were randomized in a two groups by age, gender and BMI. All of them had diagnosed NAFLD. The level of cytokeratin 18 fragments was measured by Elisa method. During ultrasound investigation of the abdominal cavity we detected signs of fatty liver. Tests for Viral hepatitis B and C were negative. The history of alcohol intake was rejected. We described importance of weight loss for the improving NAFLD. We gave diet recommendations which consist of reducing portions of food, avoiding drinking soda water, reducing carbohydrates intake and increasing fiber intake. For better diet adherence every day first group was writing food diaries. Each person from first group had 5 personalised sessions with doctor during 12 weeks. On each session patients were measured of weight, waist circumference and fat percentage measured by bioelectric impedancemetry.
To those from control group were just given general recommendations of changing eating behavior without controlled sessions with doctor during 12 weeks.

**Results:** We observed significant decreasing in weight loss, cytokeratin18 fragments M30, Fatty liver index and Fat percentage measured by bioelectric impedance in group with controlled and motivational programme of changing eating behavior after 12 weeks.

**Conclusions:** Controlled and motivation programme of changing eating behavior leads to weight loss and improves steatosis and Cytokeratin 18 levels in patients with NAFLD.

**Disclosure of Interest:** None Declared
SYSTEMIC FGF19 AND BILE SALT LEVELS IN ADULT PATIENTS WITH CHRONIC INTESTINAL FAILURE ON HOME PARENTERAL NUTRITION

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Introduction: Patients with chronic intestinal failure (CIF) may have an interrupted enterohepatic circulation of bile salts. This may cause decreased or absent production of FGF19 and subsequent loss of repression of bile salt synthesis. Overstimulation of bile salt synthesis and accumulation in the liver have been implicated as one of the possible mechanisms contributing to the development of intestinal failure associated liver disease (IFALD). Currently no data are available on systemic FGF19 and bile salts levels in adult patients with CIF on home parenteral nutrition (HPN).

Aims: The aim was to assess blood levels of FGF19 and bile salts in patients with chronic intestinal failure due to different etiologies, comparing those with and without terminal ileum.

Material and Methods: Plasma FGF19 levels and bile salts were determined in stored plasma of 69 adult patients with chronic intestinal failure on HPN. FGF19 and bile salt levels were compared between patients with and without terminal ileum resection using the Mann Whitney U test. Data are expressed as median [interquartile range].

Results: In 30 patients terminal ileum was resected (76.7% female, median age 61 years), in 39 patients no ileum resection was performed (82.1% female, median age 52 years). The main reason for HPN in the terminal ileum resection group was short bowel syndrome (73.3%). Intestinal dysmotility was the main reason for home parenteral nutrition in the group without terminal ileum resection (64.1%). Plasma FGF19 levels were significantly lower in patients with terminal ileum resection (12.5 [7 to 28] pg/mL), compared with patients without terminal ileum resection (183 [107 to 296] pg/mL, P < 0.0001) Plasma
bile salt levels were comparable in both groups (with ileum: 5.9 [3.3 to 7.8] µmol/L and without ileum: 5.8 [3.5 to 17.0] µmol/L).

**Conclusions:** Adult patients with CIF on HPN have lower FGF19 levels after ileum resection compared to CIF patients with terminal ileum in situ. An impaired production of FGF19 in CIF patients may play a role in the development of IFALD.

**Figure:**

**Disclosure of Interest:** None Declared
NO CONSENSUS AMONG NUTRITIONAL ASSESSMENT TOOLS FOR IDENTIFICATION OF MALNUTRITION IN PATIENTS WITH ALCOHOLIC LIVER DISEASE

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Introduction: Malnutrition is a major concern in alcoholic cirrhosis (AC) which needs urgent attention. Prevalence of malnutrition varies with the methods used for assessment. Gamut of methods ranging from traditional like anthropometry, functional status and composite scores, to reference methods like bioelectrical impedance analysis and radiological imaging are available for defining malnutrition.

Aims: To assess nutritional status of patients with AC using various methods.

Material and Methods: 147 consecutive pts with AC, from Jun2013 to Aug2015 underwent complete nutritional workup using anthropometry [triceps skin fold(TSF) by Harpenden’s calliper;mid arm muscle circumference (MAMC)] hand grip strength (HSG) by electronic dynamometer, Royal Free Hospital-Subjective Global Assessment (RFH-SGA), Phase angle (PA) by multifrequency TANITA&skeletal muscle index (SMI) by single slice L3 CT image by Slice-Omatic software.Dietary intake using 24 hr dietary recall along with semi-quantitative food frequency method.Energy requirements (ER) were estimated using Harris Benedict equation.Protein requirements (PR) were assessed as 1.2gm/Kg IBW.Demographic, clinical, and biochemical data were also collected. The cut-offs for defining malnutrition by TSF (12.5 mm),MAMC (24.5 cm),HGS( 37.5 Kg),PA (5.4°) were taken from literature while that of SMI (36.5 cm²/m²) was from our own ethnic data of healthy controls (2SD below the mean;unpublished data)

Results: In total 147 AC[M-100%; age- 44.08±9;BMI- 18.45±6.85;Child A:B:C-14%:38%:48%, disease duration 8 (1-40 mo), alcohol intake 112±30 gm] were studied. Mean intake of proximate principles was calorie 1588.94±566.68 Kcal (69.6% of EER), protein 57.6±25.2(73% of PR)carbohydrates 263.3±20, fat 32.7±4.2; erroneous dietary restrictions were practiced by 91.5% patients. Mean values of TSF, MAMC, HGS, PA, and SMI were 1.73±5.52mm; 21.38±2.8cm;25.37±6.98Kg;4.97±1.3°;and 4.6.2±10.4cm²/m viz. Prevalence of malnutrition as n (%) was: TSF-92 (62.6%); MAMC-
137 (93.2%); HG-141 (95.9%); RFH-SGA-123 (83.6%), PA-102 (69.4%) and SMI-23 (15.6%).

**Conclusions:** Prevalence of malnutrition and dietary inadequacy is high in patients with ALD; however nutritional assessment tools do not uniformly identify patients as malnourished. Traditional as well as modern methods (TSF, MAMC, RFH-SGA, and PA) using western cut-offs may overestimate malnutrition while the reference method (SMI) using ethnic cut-offs challenge these values. Hence there is an urgent need for ethnic cut-offs for all methods to obtain uniformity.

**Disclosure of Interest:** None Declared
ROLE OF MALNUTRITION IN COMPENSATED CIRRHOSIS: A RETROSPECTIVE ANALYSIS ANSWERING TO THE PROPOSAL OF THE ITALIAN CONSENSUS ON PORTAL HYPERTENSION (BAVENO VI)

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Introduction: While is well known the high frequency and the poor prognostic effect of protein malnutrition in cirrhotics with advanced liver disease, less is written about cirrhotic patients with protein malnutrition. In this scenario, this topic has been included in the research agenda of the Italian Consensus on Portal Hypertension which was recently held in Baveno.

Aims: The aim of this study was to analyze the prognosis and the risk of complications in malnourished cirrhotic patients classified as Child Pugh A.

Material and Methods: We consecutively enrolled all cirrhotic patients admitted in our Department during the last five years and graded as score A according to Child-Pugh. Demographical, pathological and nutritional characteristics were collected for each patient. Survival and all complications due to portal hypertension occurring during the hospitalization were recorded. Each patient enrolled in the study underwent nutritional assessment by anthropometry; patients were considered malnourished when the mid arm muscle circumference (MAMC) was < 5th percentile. A cognitive assessment was also carried out for each patient and tests for minimal hepatic encephalopathy (MHE) were performed in all patients without overt HE.

Results: One-hundred-three Child Pugh A cirrhotic patients were enrolled. Twenty-two patients (22%) presented protein malnutrition. There was no difference concerning demographical, pathological and nutritional characteristics between patients with and without malnutrition. The analysis of in-hospital mortality showed a similar and low rate in both groups. The occurrence of hepatic encephalopathy was low in both group but significantly higher in the malnourished one (18 vs 1.2 %, p=0.001) and MHE was detected more frequently in patients with protein malnutrition (64 vs 25% p=0.005).
Ascites was more present in malnourished patients (27.3% vs 9.8%, \( p=0.003 \)). While the occurrence of hepatorenal syndrome was the same in the two groups, we observed an higher incidence of acute kidney injury in malnourished patients. The length of the hospital stay was longer in malnourished patients than in not-malnourished one (10 ± 9 vs 7 ± 5, \( p=0.045 \)).

**Conclusions:** Although without representing a negative prognostic factor for in-hospital mortality, protein malnutrition seems to represent a risk factor for developing complication in compensated cirrhotics leading to a longer hospital stay.

**Disclosure of Interest:** None Declared
MAY PROTEIN MALNUTRITION REPRESENT A RISK FACTOR FOR NEUROCOGNITIVE ALTERATIONS IN CIRRHOSIS? ARE PROTEIN-RESTRICTED DIETS IN HEPATIC ENCEPHALOPATHY FRIEND OR FOE?

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Introduction: Protein malnutrition frequently occurs in cirrhotic patients and could increase the risk of several complications, such as variceal bleeding, ascites, hepatic encephalopathy (HE), hepatorenal syndrome and infections. On the other hand, protein-restricted diets (PRD) are frequently prescribed to cirrhotic patients with HE leading to a deterioration of the nutritional status increasing the protein catabolism and the release of aminoacids from the muscle. The influence of protein malnutrition on minimal HE (MHE) is less clear. Our study was aimed to verify the relationship between protein malnutrition and the prevalence of HE and MHE in a large cohort of cirrhotic patients.

Aims: Our study was aimed to verify the relationship between protein malnutrition and the prevalence of HE and MHE in a large cohort of cirrhotic patients.

Material and Methods: We enrolled consecutive cirrhotic patients without neurological diseases. Protein malnutrition was diagnosed based on Mid-Arm Muscle Circumference<5th percentile. Dietary energy and macronutrient intakes was assessed in a sububgroup of consecutive patients by a dietitian A pool of standardized questions, evaluating the time and space orientation, were used to diagnose HE. Three paper and pencil tests (TMT-A, TMT-B and DST) were use to diagnose MHE in patients without HE.

Results: 370 cirrhotic patients (244 females; age 62±13 years; 56%Child B-C) were enrolled. Eighty-eight patients (24%) showed an episode of overt HE at admission or during the hospitalization. Protein malnutrition was detected in 23% of males and 47% of females. The prevalence of malnutrition was higher in patients who experienced HE vs patients without HE (56% vs 35%; p=0.003). Fasting venous blood ammonia concentration was significantly higher in malnourished vs not malnourished patients.
Two-hundred-one patients without HE were evaluated for MHE: a diagnosis was made in 122 patients (33%). Similarly, protein malnutrition was more frequent in patients with MHE vs those without MHE (49% vs 30%; \( p=0.004 \)). Similar results, but with a lower statistical significance were obtained analysing fat store depletion and decreased muscle function.

No relationship with caloric and proteic dietary intakes were observed in patients with and without neurocognitive alterations.

**Conclusions:** Our study shows that protein malnutrition is a risk factor for overt HE and MHE suggesting that PRD should be avoided to improve quality of life and decrease subclinical alteration of mental status.

**Disclosure of Interest:** None Declared
THE IMPACT OF LIVER STEATOSIS IN THE ASSESSMENT OF LIVER FIBROSIS BY POINT SHARE WAVE ELASTOGRAPHY AND THE VALIDITY OF HAMAGOUCHI SCORE FOR QUANTIFICATION OF STEATOSIS COMPARED WITH HISTOLOGY IN BARIATRIC SUBJECTS

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Introduction: Recent studies suggest that liver steatosis tend to distort the evaluation of liver fibrosis made by point share wave elastography (SWE), overestimating the real grade of liver fibrosis. Hamagoughi score (HS) is a valid tool in ultrasound evaluation of liver steatosis.

Aims: To study the impact of liver steatosis in evaluating liver fibrosis by pSWE as compared with liver histology in a population of bariatric subjects. We also compared the grade of histological liver steatosis with HS evaluation.

Material and Methods: Blood tests, pSWE and abdominal ultrasonography were performed in 85 obese subjects before bariatric surgery (58 females, 27 males). During surgery, liver biopsy was obtained; liver steatosis and fibrosis were scored by Brunt score system by the same pathologist. HS evaluation and SWE were done by the same operator on Philips IU 22 with 15 measurements. The stiffness evaluation by SWE in kPa was interpreted as a continue variable.

Results: 20 patients had grade two or more of fibrosis and 65 had grade one or less. Liver steatosis was grade 1 in 46 subjects, grade 2 in 35 subjects and grade 3 in 4 subjects. Fibrosis assessed by SWE stiffness and histology was not statistically significant by the increase of steatosis. The ratio between standard deviation (SD) and median calculated
during pSWE was higher in severe than in mild steatosis (41.20 vs 31.30 respectively, p=0.011). The comparison between median and steatosis calculated by HS showed that with the increase of steatosis there was an increase in the median value (3.96 vs 5.27, p=0.033).

**Conclusions:** The grade of liver steatosis seems to not have an impact in evaluating the fibrosis with pSWE, suggesting this technique as a good non-invasive method to evaluate liver fibrosis also in bariatric subjects. pSWE discriminates the low and high-grade fibrosis but there is an increased uncertainty in the intermediated stages of fibrosis.

**Disclosure of Interest:** None Declared
ERYTHROCYTE MEMBRANE FATTY ACID PROFILE IN PATIENTS WITH CIRRHOSIS AND RISK OF HEPATOCELLULAR CARCINOMA


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Introduction: Malnutrition and disturbances in the fatty acid (FA) metabolism have been reported in patients with cirrhosis. But their roles in the development of hepatocellular carcinoma are still unclear. The use of biomarkers of dietary fatty acid intake and fatty acid metabolism may be an interesting approach for exploring the associations between exogenous and endogenously produced FAs and the cancer risk.

Aims: The objective of the study was to investigate the associations between FA content of erythrocyte membrane and the risk of HCC in cirrhotic patients.

Material and Methods: The case-control study CiRCE ("CIrrhose et Risque de Carcinome hépatocellulaire dans le grand Est") recruited cirrhotic patients older than 35 in six hospitals of the North-East of France between 2008 and 2012. Cases were cirrhotic patients with HCC histologically confirmed (n=346); controls were cirrhotic patients without HCC at inclusion (n=533). Among 899 patients, the fatty acid composition of the erythrocyte membrane was determined by high performance gas chromatography. FA concentrations were divided into tertiles. Odds ratios (ORs) for HCC in relation to concentrations of FAs were estimated by multivariate logistic regression adjusted for gender, age, centre, CHILD classification and etiology of cirrhosis. Product-to-precursor ratios were used as indices of enzymatic activities.

Results: Compared to controls, HCC patients were older (median age: 58.9 versus 64.2 years, p=0.04). There was gender difference between groups (87% and 70% males in cases
and controls, respectively). In both cases and controls, saturated fatty acids represented more than 39% of all erythrocyte membrane fatty acids, monounsaturated fatty acids around 14% and polyunsaturated fatty acids (PUFA) around 46%. PUFA were mainly represented by n-6 PUFA (around 38% of all fatty acids versus 8% for n-3 PUFA). The preliminary results showed a significant positive association between HCC risk and C18:2 (n-6) (OR : 1.96, 95% CI: 1.29-2.97, p for trend=0.02) but an inverse association with C20:4 n-6 (OR:0.31, 95% CI: 0.17-0.54, p for trend<0.001). No other significant association was highlighted.

**Conclusions:** Patients with cirrhosis presented abnormalities in the FA composition of erythrocyte membrane, that may reflect both dietary patterns and an altered fatty acid metabolism. Some of these unbalances were associated with HCC risk. However, relations between erythrocyte membrane content in FAs and clinical evolution of cirrhosis remain to be prospectively investigated.

**Disclosure of Interest:** None Declared
SARCOPENIA PREDICTS THE OCCURRENCE OF HEPATIC ENCEPHALOPATHY AFTER TRANSJUGULAR INTRAHEPATIC PORTOSYSTHEMIC SHUNT

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Introduction: Hepatic Encephalopathy (HE) is a major problem in patients treated with TIPS.

Aims: Our study was aimed at investigating whether a decrease in muscle mass may independently influence the occurrence of HE after TIPS.

Material and Methods: 46 consecutive cirrhotic patients submitted to TIPS were included. All patients had a computed tomography scan at the level of the third lumbar vertebrae to determine the skeletal muscle index (SMI); sarcopenia was defined by sex-specific cut-offs. The incidence of the first episode of HE after TIPS, taking into account the competing risk nature of the data (death or liver transplantation), was estimated.

Results: 26 patients (57%) were affected by sarcopenia. 21 (46%) patients developed overt HE during 7±9 months after TIPS. All of them were sarcopenic according to SMI. The difference in the incidence of post TIPS HE between the patients with or without sarcopenia was highly significant (p<0.0001). At multivariate analysis, MELD(sHR:1.16, CI:1.01-1.34, p=0.043) and sarcopenia(sHR:31.3, CI:4.5-218.07, p<0.001) were independently associated to post TIPS HE. Both basal ammonia (43.5±18.5 vs 56.8±18.6 µg/dl) and its increment after TIPS (+28.4±11.5 vs +53±12.4 µg/dl) were significantly higher in sarcopenic patients.

Conclusions: Muscle wasting is a risk factor for the development of HE after TIPS. In sarcopenic patients the amelioration of nutritional status before TIPS may be a possible goal to decrease the incidence of HE.
Disclosure of Interest: None Declared
IMPACT OF ADRENAL DYSFUNCTION ON PSOAS MUSCLE THICKNESS ASSESSED BY COMPUTED TOMOGRAPHY IN PATIENTS WITH LIVER CIRRHOSIS

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Introduction: Sarcopenia is a feature of cirrhosis and contributes to mortality, but its pathogenesis is still poorly understood. Adrenal insufficiency (AI) is also common in cirrhosis. Nausea and weight loss are common symptoms of AI and could contribute to muscle waste.

Aims: Therefore, the aim of our study was to evaluate the role of adrenal dysfunction on psoas muscle thickness assessed by CT scan in a cohort of cirrhotic patients.

Material and Methods: A study population of 74 cirrhotic patients was examined. Laboratory parameters of malnutrition (albumin, pre-albumin, transferrin and lymphocytes count) were assessed in all patients. Axial (APMT) and Transversal psoas muscle thickness (TPMT) were measured on a computed tomography (CT) image at the level of the umbilicus. Psoas muscle thickness was normalized to stature by division by height (APMT/h and TPMT/h). Adrenal function was assessed using the Standard-Dose short synacthen test. Normal adrenal response was defined as a peak cortisol > 18 µg/dl.

Results: A significant reduction of TPMT values was observed in female compared to male (23.5±6 vs 31±8; p=0.002), while no significant correlation was found between the radiologic parameters of sarcopenia and the severity and etiology of liver disease. Indeed, when we stratified our population according to the presence of ascites, we observed a significant reduction of APMT and APMT/h values in patients with ascites (37±5 vs 43±7; p=0.02 and 23±3 vs 26±4; p=0.02 respectively). In contrast TPMT and TPMT/h were not influenced by the presence of ascites. Adrenal insufficiency was present in 23 patients. The severity of cirrhosis graded by Child and MELD score was significantly increased in patients with AI compared to normal adrenal function (NAF) patients (p=0.001). AI patients exhibited lower values of pre-albumin (6±3vs10±8mg/dl; p=0.03) and transferrin
(150±84 vs 220±76 mg/dl; p=0.003) compared to NAF independently to Child class. In contrast, cirrhotic patients with AI showed increased levels of both APMT (44±6 vs 20±5; p=0.02) and TPMT (32±8 vs 27±7; p=0.03) compared to NAF. A significant correlation was found between TPMT/h and peak cortisol (r=0.38; p=0.01).

**Conclusions:** Our data suggest that AI is associated with biochemical parameters of malnutrition, but does not contribute to sarcopenia. Cortisol is one of the most important catabolic hormone, and its deficiency seems to play a protective role on muscle waste in cirrhotic patients.

**Disclosure of Interest:** None Declared
PHASE ANGLE AS AN EARLY NUTRITIONAL MARKER OF SHORT-TERM OUTCOME IN HOSPITALIZED PATIENTS WITH CIRRHOSIS

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Introduction: Several factors influence the outcome in hospitalized patients with cirrhosis and mainly clinical and biochemical markers have been used to assess prognosis. Nutritional status has been proven to influence the outcome of cirrhotic patients in different scenarios; however its role in the acute setting has been scarcely explored. Phase angle (PhA) is an objective bedside nutritional marker and is suitable for daily assessment, thus could be a useful tool in the hospitalized population.

Aims: The aim of this study was to evaluate the performance of phase angle as an early marker of short-term outcomes in hospitalized patients with cirrhosis.

Material and Methods: Prospective cohort study conducted in a tertiary care center in a special intensive care unit (ICU). Consecutive patients with confirmed diagnosis of cirrhosis were included. Clinical, biochemical and nutritional evaluations were performed. Mid-arm muscle circumference (MAMC) and phase angle derived from bioelectrical impedance were used. Malnutrition was defined with a validated cutoff value of 4.9°. Daily follow-up was conducted in each patient until discharge or death. Kolmogorov-Smirnov, X², Fisher’s exact test or T-test were used as appropriate.

Results: 79 patients were included and followed-up for a mean of 8 ± 6 days, mean age was 52 ± 1, 52% of patients were females and the main etiology of cirrhosis was HCV (25.6%). The baseline characteristics were Child-Pugh 10 (9-11), MELD score 17 (14-26), total bilirrubin 3.6 (1.9-7.6), AST 41 (25-98), ALT 34 (18-69), albumin 2.9 (2.3-3.4), INR 1.5 (1.3-1.8), and mean arterial pressure was 78.5 ± 13.6. There was a stepwise decrease in PhA as severity progressed evidenced by ventilatory support (VS) requirement; patients
without VS showed a PhA of 4.0 ± 1.1, non-invasive mechanical ventilation (MV) 3.6 ± 0.9, invasive MV 3.0 ± 0.4.

During follow-up, 50% of patients had worsening of PhA, the mean decrease was 14.9 ± 11.2 %. The acute decrease of PhA was also evaluated, from the patients who died during follow-up 62.5% had a decrease PhA within the first 24 hours of admission. There was a higher prevalence of hepatic encephalopathy, ascites and infections in malnourished patients. There were 17 reported deaths in the population and all patients presented malnutrition.

Conclusions: In our cohort PhA was related to higher complications and changes in PhA within 24 hours of admission were related to higher mortality. PhA is an early marker of prognosis that could be a part of the integral assessment of patients.

Disclosure of Interest: None Declared
ADHERENCE TO MEDITERRANEAN DIET AND NON-ALCOHOLIC FATTY LIVER DISEASE: IMPACT ON METABOLIC PROFILE

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Introduction: Many genetic, epigenetic and environmental mechanisms are supposed to play a role in the pathogenesis of non-alcoholic fatty liver disease (NAFLD). What is known is that NAFLD is strongly associated with obesity and diabetes. So far, no effective treatment for the prevention and management of NAFLD are available. Several nutritional approaches have proposed for NAFLD patients. The Mediterranean Diet (MD) is a dietary pattern with beneficial properties in primary and secondary prevention of cardio-metabolic diseases.

Aims: Aim of the study is to investigate the relationship between MD and NAFLD in a cohort of consecutive metabolic patients and to investigate how MD adherence affects metabolic phenotype in NAFLD patients.

Material and Methods: 584 consecutive patients underwent ultrasonography to assess the presence of fatty liver, using hamagouchi’s criteria. Adherence to the MD was investigated by the administration of a short dietary questionnaire, elaborated by Martinez-Gonzalez et al (2004). For the analysis patients were divided in two groups according to the MD score: low adherence (0-5 points), and high adherence (6-9 points).

Results: Mean age was 56.1±12.8 years, 38.2% were women. According to the MD score, 227 patients (38.9%) had low, and 357 had a good adherence to MD (61.1%). Overall, the prevalence of NAFLD was 82.6%. NAFLD was significantly higher in the poor adherence group (88.5% vs 79.0%; p=0.003). Thus, MD score was significantly lower in patients with NAFLD compared to those without [6 (4/7) vs 5 (4/6); p<0.001]. Among 483 NAFLD patients, at univariate analysis, MD questionnaire score was positively correlated with age (Rs=0.135; p=0.003), while was inversely correlated with triglycerides (Rs=-0.106;
p=0.023), fatty liver index (Rs=-0.163; p<0.001), lipid accumulation product (Rs=-0.135; p=-0.002), Homa-IR (Rs=-0.127; p=0.009) and BMI (Rs=-0.101; p=0.027).

**Conclusions:** We found that adherence to MD is associated with a lower prevalence of NAFLD. Moreover, NAFLD patients with a good adherence to MD disclose a better metabolic profile. Our findings suggest that MD could be considered the optimal diet to obtain the weight loss recommended by guidelines as first-line intervention in NAFLD treatment.

**Figure:**

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<th>Not Compliant Patients</th>
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<td>BMI (kg/m²)</td>
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<td>29.6 ± 5.1</td>
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<td>Female (%)</td>
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<td>30.7</td>
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<td>Glycaemia (mg/dl)</td>
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<td>101.9 ± 20.6</td>
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<td>200.1 ± 41.2</td>
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<td>HDL-C (mg/dl)</td>
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<td>49.7 ± 13.6</td>
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<td>LDL-c (mg/dl)</td>
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<td>122.9 ± 35.6</td>
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<td>ALT (IU/l)</td>
<td>26 (18/40)</td>
<td>24 (18/39)</td>
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</tr>
<tr>
<td>GGT (IU/l)</td>
<td>27 (18/45)</td>
<td>25 (17.3/36)</td>
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<td>Lipid Accumulation Product</td>
<td>71.8 (48.9/106.9)</td>
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**Disclosure of Interest:** None Declared
VITAMIN D DEFICIENCY AND HEPATIC ENCEPHALOPATHY IN PATIENTS WITH CHRONIC LIVER DISEASE

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Introduction: Patients with chronic liver disease (CLD) develop a wide range of nutritional abnormalities associated with progression of their liver disease. Alterations in hepatic synthetic function, nutrient absorption and metabolism and worsening physical symptoms result in the nutritional compromise seen in this group. A major complication in patients with CLD is the development of hepatic encephalopathy (HE), which describes a complex array of neurological symptoms including cognitive decline, impaired motor function and fatigue. In the general population vitamin D (25-OHD) deficiency has been demonstrated to be associated with loss of cognitive function, all-cause dementia and Alzheimer disease.

Aims: To investigate the incidence of 25-OHD deficiency in a group of patients assessed for liver transplantation and its relationship to HE.

Material and Methods: HE, severity of CLD, nutritional status and 25-OHD measured using the Diasorin RIA technique were analysed in a group of patients who were assessed for liver transplantation. Severity of liver disease was assessed using the Model of Endstage Liver Disease (MELD) score. HE was assessed using West Haven criteria.

Results: The results of 165 patients were included in this analysis enrolling patients assessed for liver transplant between 2006-2010. The mean age of all patients was 53 ± 8 yrs. Moderate to severe 25-OHD deficiency was identified in 49 patients. Thirty-six of these had grade 1-2 HE compared with 13 patients who did not have HE (p= <0.0001).
There was no significant difference in the presence of HE in patients with mild 25-OHD deficiency.

There was a significant correlation between the severity of 25-OHD deficiency and the severity of liver disease ($r=0.3890$, $p=<0.0001$) and a significant correlation between liver disease severity and the presence of HE ($p=<0.0001$). Individuals are more likely to have 25-OHD deficiency if they had HE compared to patients without HE ($p<0.0001$). This significant difference was observed across the entire range of MELD scores (from 10 to 38).

**Conclusions:** Vitamin D deficiency was common in the majority of patients with CLD. Worsening vitamin D deficiency was associated with increased severity of liver disease. Importantly, patients with HE had significantly lower 25-OHD than non-encephalopathic patients with similar MELD scores. This suggests Vitamin D deficiency may have an unrecognised role in the development of HE.

BIOELECTRICAL IMPEDANCE VECTOR ANALYSIS (BIVA) IS AN OBJECTIVE METHOD FOR NUTRITIONAL ASSESSMENT IN PATIENTS WITH CIRRHOSIS

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Introduction: Malnutrition in cirrhosis is related to poor prognosis. There are few reliable methods for nutritional assessment since most of them are biased by fluid retention and synthetic liver dysfunction, and useful tools such as CT-scan are not easy accessible. Bioelectrical impedance vector analysis (BIVA) is a bedside non-expensive method to evaluate body composition and hydration status, however, data on its usefulness in cirrhosis is limited.

Aims: To evaluate the efficacy of BIVA for nutritional assessment in patients with cirrhosis.

Material and Methods: Prospective cohort study. BIVA, mid-arm muscle circumference (MAMC), triceps skinfold thickness (TSF), body mass index with ascites cutoffs (BMIa), and bioelectrical impedance (BIA) were used for nutritional assessment. Malnutrition with BIVA was defined as a vector outside the 75th percentile in the 4th quadrant of the RXc graph. Clinical (ascites and edema) and biochemical evaluations were performed at baseline. T test, Mann Whitney’s, and Xi2 were used as appropriate. Hotelling’s T test was used to compare Rxc graph groups. Kaplan-Meier curves and Cox regression models were used for survival.

Results: 308 patients were followed for 26.6 ± 9.8 months. Child-Pugh stage was A in 36%, B in 46%, and C in 18% and MELD score was 11.5 ± 4.1. Nutritional parameters showed a MAMC of 23.5 ± 3.9, TSF of 18.8 ± 8.4, and of BMI 27.12 ± 5.24. The frequency of malnutrition was 14.7% for BIA, 25% for BIVA, 32% for MAMC, 13% for TSF, and 14 for % BMIa. In the Rxc graph only patients with clinical ascites displayed
fluid retention by BIVA (55.1% vs 44.9% without ascites, p=0.005). Also, higher stages of Child-Pugh were significantly associated with a higher rate of malnutrition and fluid retention. Kaplan-Meier curves disclosed a higher mortality in the malnourished group identified by BIVA (39.7% vs. 21.4% in well-nourished, p=0.004), but malnutrition defined by BIA, MAMC, TSF, or BMIa was not associated to mortality. In univariate regression only BIVA, Child-Pugh and MELD score were associated to mortality. In multivariate analysis malnutrition by BIVA remained associated to mortality when controlled for severity scales (HR=1.68, 1.02-2.76).

**Conclusions:** BIVA is an objective method for nutritional assessment that can identify both malnutrition and fluid overload in patients with cirrhosis. Only malnutrition defined by BIVA was associated with mortality in this cohort, and it remained a significant prognostic factor when adjusted by liver disease severity scores.

**Disclosure of Interest:** None Declared
P6.2

THE ROLE OF NUTRITIONAL INTERVENTION IN PATIENTS WITH CHRONIC LIVER DISEASE AWAITING TRANSPLANTATION

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Introduction: Individuals with chronic liver disease who are malnourished have poorer outcomes compared to their well-nourished counterparts.

Aims: To investigate the efficacy of nutritional intervention for end-stage liver disease in patients awaiting liver transplantation.

Material and Methods: This retrospective study investigated end-stage liver disease patients assessed for liver transplantation between 1 January 2012 and 31 December 2013. Nutritional status was determined at the time of transplant assessment using Subjective Global Assessment (SGA). Nutritional intervention was with Resource Protein, Ensure and/or branched chain amino acids.

Results: One hundred and fifty four patients were enrolled in the study. One hundred and twenty seven of these patients received nutritional intervention. The remaining 27 patients did not receive any nutrition intervention and the majority of these were well nourished. Fifty percent of patients who received nutrition intervention were moderately malnourished (SGA B) and 18% were severely malnourished (SGA C) and 25% were well nourished (SGA A). Survival to transplantation was similar in the nutritional intervention and non-intervention group, 65% and 70% respectively. There were no significant differences between the groups in the changes in their model of end-stage liver disease (MELD) scores from assessment to transplantation, nor in their wait times. For both
groups, there was a fall in the mean body mass index (BMI) at 3 months post-transplant, followed by an increase that continued to 24 months. Importantly, the mean BMI for the malnourished intervention group remained significantly lower than that of the control group at each time point through to 24 months.

**Conclusions:** Nutritional intervention in malnourished individuals improves outcomes to be equivalent to those of well-nourished individuals and should be adopted as standard of care for all patients awaiting liver transplantation. Importantly, this is the first study showing pre-transplant BMI determines post-transplant BMI at 2 years irrespective of nutritional status or nutritional intervention.

DIFFERENT RESPONSES OF FGF19, FGF21, TOTAL BILE ACID AND GUT HORMONES IN DIABETICS AFTER ROUX-EN-Y GASTRIC BYPASS

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Introduction: Roux-en-Y gastric bypass (RYGB) is an effective way to remit human type 2 diabetes mellitus (T2DM). Decreased plasma fibroblast growth factor (FGF) 19 and increased bile acid has been observed in diabetics, while plasma FGF21 has been reported to be correlated with adiposity and fasting insulin. The changes in FGF19, FGF21 and total bile acids after RYGB remain less explored.

Aims: We investigated the roles of FGF19, FGF21 and total bile acid in patients with T2DM receiving RYGB.

Material and Methods: A total 18 diabetic patients undergone laparoscopic RYGB were enrolled into this prospective study. Remission of T2DM was defined as fasting glucose 100-125 mg/dL with HbA1c value < 7.0% (improvers) and <6.5% (remitters). Serial concentrations of FGF19, FGF21, total bile acid, acyl ghrelin, des-acyl ghrelin, and peptide YY were measured at before and after surgery.

Results: RYGB significantly lowered fasting blood sugar, HbA1c, insulin resistance, as well as decreased the body weight and waist circumference one year after surgery. Significant increased FGF19 and acyl ghrelin levels, and decreased FGF21 concentrations were also noted, while total bile acid remained unaltered after RYGB. The decrease in FGF19 negatively correlated with that in waist circumference. The changes in des-acyl ghrelin had positive correlation with those in total bile acids. However, no difference in FGF19, FGF21 and total bile acid was detected between improvers and non-improvers, as well as remitters and non-remitters.
Conclusions: RYGB effectively remits diabetes and induces significant increase of FGF19 and acyl ghrelin, and decreases of FGF21 without altering total bile acid levels one year after surgery in non-morbidly obese Asians with T2DM. However, the precise roles of FGF19, FGF21 and bile acid in human diabetic remission need further investigation.

Disclosure of Interest: None Declared
ROLE OF GALECTIN-3 AND IL-33/ST2 PATHWAY IN OBESOGENIC DIET-INDUCED NONALCOHOLIC STEATOHEPATITIS

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Introduction: Accumulating evidence suggests that immune reactivity plays an important role in obesity-associated metabolic disorders including nonalcoholic steatohepatitis (NASH). The role of Galectin-3 (Gal-3) and IL-33/ST2 axis in obesogenic diet-induced NASH is not fully elucidated.

Aims: We aimed to dissect the role of Gal-3 and IL-33/ST2 axis in obesogenic high-fat diet-induced liver inflammatory response and fibrosis in mice, key events in the pathogenesis and progression of NASH.

Material and Methods: Gal-3-deficient (LGALS3−/−) C57Bl/6 mice, ST2-deficient (ST2−/−) BALB/c mice and their wild-type (WT) littermate controls received HFD (60% kcal/fat) or standard chow diet for 1 and 6 months and histological, gene expression and immunophenotypic analyses were performed.

Results: Gal-3 deletion markedly reduced hepatic inflammation and fibrosis in mice fed HFD for 6 months, which was associated with lower proportion of hepatic mature myeloid DCs, proinflammatory monocytes/macrophages and lower CCL2 chemokine, NLRP3 inflammasome and IL-1β mRNA expression compared with diet-matched WT mice. To dissect early events in the pathogenesis of NASH mice were placed on HFD for one month and LGALS3−/− mice exhibited less numerous hepatic effector NK cells and proinflammatory macrophages, including NK1.1+IFN-γ+ and NK1.1+CD27+CD11b+ effector cells. After 6 months on HFD, profibrotic IL-33, ST2 (IL-33R) and IL-13 mRNA expression were higher in WT than in LGALS3−/− mice. Moreover, in contrast to wild-type macrophages, Gal-3 deficient macrophages failed to upregulate ST2 expression and IL-13 production in vitro and in vivo in response to rIL-33. To further explore the role of IL-33/
ST2 axis in NASH, ST2 knockout mice were placed on HFD for 6 months. Deletion of ST2 markedly reduced HFD-induced liver inflammation and collagen deposition which was associated with less numerous profibrotic CD11b^Ly6C_{low} monocytes and lower hepatic Gal-3, IL-33 and IL-13 expression. Furthermore, in comparison to WT mice, ST2 knockout mice fed HFD/high-fructose diet had markedly reduced liver fibrosis.

**Conclusions:** Gal-3 and IL-33/ST2 axis have an important role in NASH and these pathways interplay in obesogenic diet-induced liver inflammation and fibrogenesis. The obtained results suggest that blockade of Gal-3 and IL-33 may be novel therapeutic approach in obesity-associated fibrotic NASH.

**Disclosure of Interest:** None Declared
P6.5YI

EFFECT OF A PROBIOTIC ON FATTY LIVER INDEX AND LIVER STIFFNESS IN NAFLD PATIENTS: RANDOMIZED CLINICAL TRIAL

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Introduction: Probiotics have beneficial effect on nonalcoholic fatty liver disease (NAFLD) in animal models. Despite a large number of animal data, randomized placebo-controlled trials (RCT) in NAFLD are still lacking in humans.

Aims: We performed a double-blind single center RCT of alive multistrain probiotic vs. placebo in type 2 diabetes patient with NAFLD detected on ultrasonography (US).

Material and Methods: A total of 58 patients met the criteria for inclusion. They were randomly assigned to receive multiprobiotic “Symbiter” (concentrated biomass of 14 probiotic bacteria genera Bifidobacterium, Lactobacillus, Lactococcus, Propionibacterium) or placebo for 8-weeks administered as a sachet formulation in double-blind treatment. The primary main outcomes were the change in fatty liver index (FLI) and liver stiffness (LS) measured by Shear Wave Elastography (SWE). FLI a validated prediction score for hepatic steatosis severity designed Bedogni et al. Secondary outcomes were the changes in transaminases activity, serum lipids and cytokines (TNF-α, IL-1β, IL-6, IL-8, INF-γ) levels. ANCOVA was used to assess the difference between groups.

Results: In probiotic group FLI significantly decreased from 84.33±2.23 to 78.73±2.58 (p<0.001) and but remained static in the placebo group (82.57±2.45 to 81.6 ±2.36; p=0.367). In both interventional groups slight insignificant reduction of LS measured by SWE were detected. Therefore, LS from baseline in probiotic group (7.16±0.2 to 6.76 ±0.22; p=0.052) decreased more pronounced as compared to placebo group (7.28±0.22 to 7.14±0.26; p=0.396). Analysis of secondary outcomes showed that probiotic reduced level of serum aspartate aminotransferase (AST) – 14.8 %, p<0.001; gamma-glutamyl transpeptidase (GGT) – 20.4 %, p=0.001; and triglycerides (TG) on 22.5 %, p=0.001.
From markers of chronic systemic inflammatory state only TNF-α (14.5 %, p<0.001) and IL-6 (28.1 %, p=0.001) changes significantly after treatment with probiotics.

**Conclusions:** Probiotic therapies can reduce liver fat, aminotransferases activity, TG, TNF-α and IL-6 in NAFLD patients. Modulation of the gut microbiota represents a new treatment for NAFLD and should be tested in larger studies.

**Disclosure of Interest:** None Declared
Introduction: Parenteral nutrition-associated liver disease (PNALD), characterized by steatosis, cholestasis and fibrosis, is a feared complication in patients receiving parenteral nutrition (PN). To date, the pathogenesis of PNALD is poorly understood and therapeutic options are limited. Dysregulated bile salt homeostasis has been proposed to contribute to PNALD.

Aims: The aim of this study was to evaluate the effects of continuous PN on bile salt homeostasis in rats.

Material and Methods: Rats received PN via the jugular vein or normal diet for 3, 7 or 14 days. Serum biochemistry, hepatic triglycerides, circulating bile salts and C4 (marker for bile salt synthesis), IL-6 and TNF-alpha, and transcript levels of genes engaged in lipogenesis and bile salt homeostasis were assessed. The Mann-Whitney U test was used to compare differences between PN-fed rats and controls.

Results: PN increased hepatic triglycerides already after 3 days of administration ($P=0.01$), and were further elevated after 7 ($P=0.0095$) and 14 days ($P=0.0007$). PN also resulted in conjugated bilirubin elevation after 7 (2.3 vs 1.0 µmol/L, $P=0.001$) and 14 days (2.5 vs 1.3 µmol/L, $P<0.0001$). This indicates PN-induced steatosis and impaired canalicul secretion of bilirubin, the latter in line with reduced hepatic expression of Mrp2 mRNA after 3, 7 and 14 days of PN ($P<0.01$ for all time points). Interestingly, gene
expression of the lipogenic genes Fasn and Scd1 were decreased after 3, 7 and 14 days of PN \((P<0.05\) for all time points), indicating decreased \textit{de novo} lipogenesis. There was no histological evidence for liver inflammation after PN administration, and circulating levels of pro-inflammatory cytokines IL-6 and TNF-\(\alpha\) were comparable in all groups. Hepatic expression of Fxr mRNA was decreased after 7 days of PN \((-3.7\) Fold; \(P=0.0095\)), without apparent effect on expression of Fxr targets Bsep and Shp. Nonetheless, Cyp7a1 expression was reduced after 7 days of PN \((-4.9\) Fold; \(P=0.02\)), indicative for lowered bile salt synthesis as supported by reduced serum levels of C4 after 3, 7 and 14 days of PN \((P<0.05\) for all time points). Total levels of circulating bile salts were not affected by PN.

\textbf{Conclusions:} This study showed that PN in rats caused early steatosis and cholestasis, while inflammation was not present. The onset of these abnormalities was associated with reduced lipogenesis, bile salt synthesis, and bilirubin secretion. This animal model allows further investigation of the role of bile salt dyshomeostasis in the pathogenesis of PNALD.

\textbf{Disclosure of Interest:} None Declared
**IMPACT OF BARIATRIC SURGERY IN THE MAIN CLINICAL AND LABORATORY INDICATORS 6 AND 12 MONTHS AFTER INTERVENTION IN BARIATRIC SUBJECTS AND DIFFERENCES BETWEEN BY-PASS AND SLEEVE GASTRECTOMY**

Daniele Macor¹, ², ³, Silvia Palmisano¹, ⁴, Flora Masutti², ³, Valentina Lanzilotti⁵, Cristiana Abazia², ³, Nicolò De Manzini¹, ⁴, Claudio Tiribelli¹, ², ³, Lory Saveria Crocè¹, ², ³, Michela Giuricin⁴ and Bariatric Liver Group

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**Introduction:** Bariatric surgery is the first line of treatment in cases of morbid obesity not responding to dietary treatment and/or physical activity. The main surgical procedures are the sleeve gastrectomy and the gastric bypass. Both techniques have proven effective in weight loss. It is also known that liver fibrosis evaluation with point shear wave elastography (pSWE) is difficult in these patients.

**Aims:** To study the impact of bariatric surgery on main clinical and laboratory metabolic indicators and scores and SWE after 12 months from the intervention.

**Material and Methods:** We studied 68 obese subject candidate to bariatric surgery (45 female, 23 male). 28 underwent by-pass and 40 sleeve gastrectomy. Blood tests, physical examination and pSWE were performed before surgery, after 6 months (68 patients) and after 12 months (51 patients).

**Results:** In our sample, there was a statistically significant difference in the reduction in FLI (61% vs 27%, p=0.015), waist circumference (26% vs 18%, p=0.045), BMI (34% vs 28%, p=0.016), total cholesterol (23% vs 05%, p=0.001). No difference was observed in the other indicators considered. Ferritin level increased (52%) in sleeve and decreased (25%) in by-pass (p=0.02). No difference was observed for pSWE.
Conclusions: This study showed that no significant difference in clinical and laboratory terms between the two types of intervention, except for the iron balance. All steatosis scores (FLI, HSI, LAP), reduction of weight, BMI, waist circumference improved in both types of intervention, though significantly more in by-pass.

Disclosure of Interest: None Declared
SILIMARIN VS. LIFE STYLE CHANGE: A COMPARISON BETWEEN THESE POTENTIAL THERAPEUTIC APPROACHES FOR JUVENILE NAFLD

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Introduction: NAFLD is a chronic liver disease without a specific therapy.

Aims: The aim is to study the nutraceutical properties of Silimar in added to High Fat High Carbohydrate Diet (HFHCD) compared with the effects of life style changes (diet), in a juvenile NASH mice model.

Material and Methods: Initially, C57BL/6 female and male mice immediately after weaning were fed with HFHCD and chow diet (CTRL diet). After 8 weeks, HFHCD group was divided in three subgroups consisting in: 1) HFHCD 2) HFHCD added with Silimar (1mg/animal/day) 3) HFHCD switched to CTRL diet, in order to mimic a life style change. The trial went on for further 12 weeks. HFHCD and CTRL diet were used as controls. Treatment effects were assessed analyzing biochemical parameters reported in Table 1. Biomolecular analysis of some fibrotic markers were performed through Real Time PCR.

Results: Silimar decreased ALT/AST level and visceral fat in both genders, with a parallel improvement of plasmatic lipid profile. In females it was observed also a decreased hepatomegaly, in males an improvement in glycaemia without changes in insulinemia. Life style change demonstrated the best results, with the reversion of all the altered parameters under study, restoring a normal profile (Table 1). Liver histology showed that Silimar determined a slight reduction in inflammatory foci and fibrosis. This trend was confirmed also by a significant decrease (p<0.05) of the expression of α-SMA and Collagen1A1, suggesting a potential antifibrotic effect.
### Parameters

<table>
<thead>
<tr>
<th>Parameters</th>
<th>MALES</th>
<th>FEMALES</th>
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<tbody>
<tr>
<td></td>
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<td>HFHC+Sil</td>
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<tr>
<td>Body weight (g)</td>
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<tr>
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<td>TG (mg/dl)</td>
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<td>ALT (U/L)</td>
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<td>134±63a</td>
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<td>AST (U/L)</td>
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<tr>
<td>Glucose (mg/dl)</td>
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<td>226±17a</td>
</tr>
</tbody>
</table>

\(^{a}p<0.05;^{b}p<0.01;^{c}p<0.001\) vs CTRL diet. \(^{#}p<0.05;^{##}p<0.01;^{###}p<0.001\) vs HFHC diet

**Conclusions:** The life style change remains the best choice to revert the pathological features of NAFLD. However the addition of Silimarlin to the HFHC diet might exert beneficial effects mainly improving lipidemia, liver damage and decreasing fibrogenesis. This data is particularly relevant to overcome the current low patient compliance in changing dietary habits.

**Disclosure of Interest:** None Declared
EFFECT OF AN ORAL BILE ACID SUPPLEMENT ON LIPID METABOLISM IN PATIENTS WITH SHORT BOWEL SYNDROME ON LONG-TERM PARENTERAL NUTRITION

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Introduction: Short bowel syndrome patients (SBS) have lipid metabolism disorders with an increase of cholesterol synthesis and hepatic lipogenesis, often leading to chronic cholestasis or terminal liver failure. These lipid disturbances could be due to the decrease in bile acid pool inducing an inhibition of the nuclear receptor FXR and the nuclear receptor LXRα which in turn increases the expression of SREBP1c stimulating cholesterol synthesis and lipogenesis.

Aims: The aim of this study was to observe the effect of bile acids administration on lipid synthesis in SBS patients on long-term parenteral nutrition.

Material and Methods: This pilot study measured the 24 h fractional synthesis rate (FSR) of cholesterol and triglycerides by isotopic method (deuterated water) before and after 4 months of an oral intake of 20 mg/kg/day of ursodeoxycholic acid (UDCA). Five patients (2 women, 3 men) with short bowel (46 ± 39.11 cm) on long term parenteral nutrition (151 ± 74 days) with normal liver function and normal triglyceride and cholesterol levels have been studied. Plasma metabolites (glucose, total, free and esterified cholesterol, triglycerides), liver enzymes, 7-α-OH-Cholesterol level and profile bile acids were evaluated. Student t-test was performed and the results were expressed in mean (± SD).

Results: Subjects (age: 53.4 ± 19.2 years; BMI: 19.9 ± 1.9 kg.m⁻²) received 3.6 ± 1.8 bags of parenteral nutrition per week. After treatment with UDCA, absolute value of cholesterol synthesis decreased (0.31 ± 0.12 mmol.L⁻¹ to 0.24 ± 0.11 mmol.L⁻¹; p < 0.05)
while FSR of cholesterol tended to decrease (31.6 ± 4.7% to 26.4 ± 4.7%; p=0.059). FSR of triglycerides decreased (12.8 ± 5.8% to 9.2 ± 5.5%; p<0.01), but absolute value of triglycerides synthesis and triglyceride levels remained unchanged. Free cholesterol levels and alanine aminotransferase also decreased (p<0.05). There were no significant differences for the other variables assessed.

**Conclusions:** In SBS patients, an oral intake of UDCA appears to decrease the hepatic synthesis of triglycerides and cholesterol. This suggests that the increase of cholesterol synthesis and hepatic lipogenesis in SBS patients result from a reduction of the bile acid pool.

**Disclosure of Interest:** None Declared
FOOD INTAKE, ANTHROPOMETRY AND METABOLISM OF SERUM IRON IN PATIENTS WITH NONALCOHOLIC STEATOHEPATITIS

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Aims: To study a population with nonalcoholic steatohepatitis (NASH) with regard to eating habits, anthropometric data and serum iron parameters, and to compare with a control population.

Material and Methods: Fifty five patients with NASH (39 male, 20 female); average age of 44.6 years. 104 controls were selected according to gender and age. Application of semi-quantitative food frequency questionnaire. Determination of weight, height, perimeter belt (PB), body mass index (BMI), iron, total iron binding capacity (TIBC), transferrin saturation and ferritin. Statistics: analysis of variance, Kruskal-Wallis test and multiple linear regression model.

Results: Forty eight patients (81%) overweight, of which 24 (41%) with obesity criteria (BMI > 30).

<table>
<thead>
<tr>
<th></th>
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<tbody>
<tr>
<td><strong>Anthropometry</strong></td>
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<tr>
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<tr>
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<td>Iron</td>
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</tbody>
</table>

**Conclusions:** 1- NASH population is more obese and ingests significantly more carbohydrates, polyunsaturated fat, fiber and vitamin E;  
2- Unlike results from previous studies, consumption of saturated fat and proteins was significantly lower in the NASH population;  
3- Serum iron parameters differ significantly between the two populations, despite a similar iron intake.

**Disclosure of Interest:** None Declared
PROBIOTICS AND NUTRACEUTICALS: ARE THEY ONE TEAM PLAYERS IN NAFLD PREVENTION

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Introduction: Today probiotics have been suggested as a treatment for the prevention of non-alcoholic fatty liver disease (NAFLD). Omega-3 fatty acid treatment may have beneficial effects in regulating hepatic lipid metabolism, adipose tissue function, and inflammation. Smectite is a natural silicate that has the ability to bind endo- and exotoxins and restored the barrier properties of human intestinal cell monolayers.

Aims: The study aims to determine whether probiotics plus nutraceuticals such as smectite or omega-3 are superior to probiotic alone on the monosodium glutamate (MSG) induced NAFLD model in rats.

Material and Methods: Totally 75 rats divided into 5 groups were included (n=15, in each). Rats of group I were intact. Newborns rats of groups II-IV were injected with MSG. The III (Symbiter) group received 2.5 ml/kg of multiprobiotic “Symbiter” containing concentrated biomass of 14 probiotic bacteria genera. The IV (Symbiter-Omega) and V (Symbiter+Smectite) groups received combination of probiotic biomass supplemented with flax and wheat germ oil (250 mg of each, concentration of omega-3 fatty acids 1-5%) or smectite gel (250 mg) respectively. To assess morphological changes in liver we used NAS (NAFLD activity score). The content of proinflammatory cytokines (IL-1β, IL-12Bp40, INF-γ) and anti-inflammatory cytokines (IL-4, IL-10, TGF-β) were measured by ELISA.

Results: In all interventional groups reduction of total NAS score was observed. Supplementation with omega 3 fatty acids lead to 20 % higher decreasing of steatosis score (0.73±0.11 vs 0.93±0.22, p=0.848) and reduction by 16.6 % of triglycerides content in liver as compared to probiotic alone. Co-treatment with Symbiter+Smectite are associated with more pronounced reduction of lobular inflammation (0.13±0.09 vs 0.33±0.15). Both nutraceuticals combination with probiotic and probiotic alone equally
attenuated chronic systemic inflammation that was confirmed by the decrease of the pro-inflammatory cytokines level and the activation of anti-inflammatory system.

**Conclusions:** Our study demonstrated more pronounced reduction of steatosis and hepatic lipid accumulation after treatment with combination of alive probiotics and omega-3 as compared to probiotic alone. From the other hand, supplementation with smectite gel due to his absorbent activity and stabilization mucus layer properties can impact on synergistic enhancement of single effect which manifested with reduction of lobular inflammation and at list partly NASH prevention.

**Disclosure of Interest:** None Declared
THE NEXT GENERATION SEQUENCING (NGS) PLATFORM IN THE ASSESSMENT OF THE GUT MICROBIOTA IN BARIATRIC SURGERY

Giuseppina Campisciano*,1,2, Silvia Palmisano1,3, Michela Giuricin3, Carolina Cason1,2, Daniele Macor1,4,5, Claudio Tiribelli1,4,5, Lory Saveria Crocè1,4,5, Manola Comar1,2 and Bariatric Liver Group

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Introduction: Bariatric surgery is currently the most effective treatment for obesity improving a dramatic loss of fat mass and favoring metabolic improvement. Recent hypothesis include changes in gut microbiota as surrogate marker of local microenvironment. Aims: To further investigate the host–microbe relationship in patients who completed bariatric surgery, microbiota composition was evaluated before surgery and during the first follow-up visit.

Material and Methods: Sequencing of the V1-V3 region of bacterial 16S rRNA was performed by NGS technology on feces samples. The template preparation was performed using the Ion PGM Template OT2 200 kit on Ion OneTouch™ 2 System and then sequenced on the Ion PGM™ System machine (Thermo Fisher Scientific, Waltham, MA, USA). QIIME 1.8.01 was used to process the sequence data. High quality (Q>25) sequences were filtered by quality using split_libraries_fastq.py with default parameters. Differences in community composition were investigated using analysis of similarity (ANOSIM), Kruskal-Wallis test and similarity percentage (SIMPER) analysis.

Results: Microbiota analysis showed that 12 bacterial taxa distinguish intestinal microbiota at baseline and post-surgery. Bacteroides, Parabacteroides, Prevotella, Dialister were considered dominant and differences in amount distribution were observed pre and post-surgery. Specifically Bacteroides and Provetella associated to obesity, high level of HDL and a dietary rich in fat and carbohydrates decreased remarkable after surgery and microorganisms linked to inflammation including Dialister (IL-6 increase) and Sutterella (inflammatory bowel diseases) were absent or at very low amount.
**Conclusions:** NGS provide a deeper coverage of the complex gut microbial communities highlighting the importance of advanced microbiology as important marker of the bariatric treatment.

**Disclosure of Interest:** None Declared
Abstract submission deadline: 22 November 2016
End of early fee registration: 31 December 2016

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