Global Engage is pleased to announce the 2nd Global NASH Congress 2019, which will be taking place 25-26 February 2019 in London.

An increasing number of people are being diagnosed with nonalcoholic fatty liver disease (NAFLD) and nonalcoholic steatohepatitis (NASH) each year, and the primary method of treatment is weight loss. With no approved medicines on the market, the drug development race is intensifying. The pathogenesis of NASH is still not fully understood, and definitive diagnostic methods are invasive, so development has been slow.

However, promising developments in research will hopefully bolster drug development and other methods of treatment. Examples of such developments include improved in vivo liver models, non-invasive diagnostic biomarkers and better understanding of the disease's mechanisms. This year's congress will focus on these exciting advancements, as well as on the challenges of preclinical and clinical research in NASH. There will also be sessions covering regulation and business development, as well as a showcase of the most promising therapeutics in development.

Attracting experts working in all areas of nonalcoholic steatohepatitis, the conference will examine the latest research and development in pathogenesis, diagnosis and treatment of the disease. Featuring small group roundtable discussions and ample networking time, the event provides an excellent opportunity to meet and collaborate with senior representatives from industry, hospitals and universities. During the two-day conference, there will be 40 expert-led presentations, interactive roundtable discussions exploring key issues, and a dynamic exhibition room filled with technology providers showcasing their technologies.
DAY 1 - TRACK 1

Current Approaches to NASH and Preclinical Strategy
• Guidance on clinical endpoints
• Improving patient recruitment for clinical trials
• Regulatory pathways
• Target discovery and validation
• Preclinical models
• Off-label drugs and drug repurposing
• Novel therapeutic methods
• Lifestyle intervention strategies
• Liver transplants
• Combination therapy

DAY 1 - TRACK 2

Non-invasive Biomarkers and Diagnostic Tools
• Non-invasive biomarkers
• Blood-based biomarkers
• MRI/MRE-based assessments
• Non-invasive cirrhosis assessment
• Liver biopsies; the gold standard
• Developments in liver biopsy imaging analysis
• Improving experimental models

DAY 2 - TRACK 1

The Pathogenesis of NASH and Related Health Conditions
• Genetics and epigenetics
• Epidemiology
• Metabolic syndrome
• Lipotoxicity
• Mitochondrial dysfunction and apoptosis
• Insulin resistance
• The gut microbiome
• Diabetes
• Cardiovascular disease
• Hepatocellular carcinoma

DAY 2 - TRACK 2

Therapeutics in Development
• Presentations from the most exciting companies in NASH drug development
• Targeting the gut
• Targeting metabolic pathways
• Targeting oxidative stress and inflammation
• Targeting progressive fibrosis (antifibrotics)

ROUNDTABLE DISCUSSIONS
1. Liver biopsy: the gold standard
2. Regulation of Inflammation and Fibrosis in NASH
3. From triglycerides to toxic lipids in NASH
4. Invasive and non-invasive Biomarkers for NASH/NAFLD
5. NASH and CVD
6. Collaborative projects: academia, healthcare providers, industry
SPONSORSHIP AND EXHIBITION OPPORTUNITIES AVAILABLE
For more details contact Faizel Ismail at faizel@globalengage.co.uk or call +44 (0) 7415 228 053
<table>
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<th>Name</th>
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<tr>
<td>CHRISTIAN TRAUTWEIN</td>
<td>Director of the Department of Internal Medicine III, University Hospital Aachen, Germany</td>
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<tr>
<td>CYNTHIA MOYLAN</td>
<td>Associate Professor of Medicine, Duke University, USA</td>
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<tr>
<td>THOMAS JENSEN</td>
<td>Assistant Professor of Medicine, Colorado University, Denver School of Medicine in Division of Endocrinology, Diabetes, and Metabolism. Co-Director of NAFLD Multidisciplinary Clinic, USA</td>
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<td>GIULIO MARCHESINI</td>
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<td>WAJAHAT MEHAL</td>
<td>Professor of Medicine (Digestive Diseases), Yale School of Medicine, USA - Regulation of Inflammation and Fibrosis in NASH</td>
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<td>Clinical Assessor, Licensing Division 2, Federal Institute for Drugs and Medical Devices (BfArM), Germany</td>
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<td>Heisenberg Professor for Clinical Experimental Diabetology, Medical Clinic IV, University of Tübingen, Germany</td>
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<td>JEREMY W TOMLINSON</td>
<td>Professor of Metabolic Endocrinology, University of Oxford, UK</td>
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<td>YI LUO</td>
<td>Director Clinical Biomarkers in Innovative Medicine Development, Bristol-Myers Squibb, USA</td>
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<td>NIKOLAI NAQUMOV</td>
<td>Executive Director, Hepatology Science and Innovation, Novartis, Switzerland</td>
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<td>KARINE CLÉMENT</td>
<td>MD, PhD, Director of INSERM/ Sorbonne University, NutriOMics team, France</td>
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<td>LUCA VALENTI</td>
<td>Associate Professor of Internal Medicine, University of Milan, Italy</td>
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<td>KARIN CONDE-KNAPE</td>
<td>CVP Cardiovascular and Liver Disease Research, Novo Nordisk, UK</td>
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<td>OREN Tirosh</td>
<td>Institute of Biochemistry, Food Science and Nutrition, Faculty of Agriculture, Food and Environment, The Hebrew University of Jerusalem, Israel</td>
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<td>MICHAEL FEIGH (Chair)</td>
<td>Principal Scientist, Gubra</td>
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<td>GUIDO BASELLI</td>
<td>PhD Student, University of Milan, ItalyPoster Competition Winner Presentation</td>
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<td>JOOST BOECKMANS</td>
<td>PhD-Researcher, Department of In Vitro Toxicology &amp; Dermato-Cosmetology (IVTD), Faculty of Medicine and Pharmacy, Vrije Universiteit Brussel, Belgium</td>
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2nd GLOBAL NASH CONGRESS 2019
Dissecting the urinary steroid metabolome

Professor of Metabolic Endocrinology, University of Oxford, UK

Jeremy W Tomlinson

Circulating biomarkers for fibrosis in NASH: collagen biomarkers and beyond

Director Clinical Biomarkers in Innovative Medicine Development, Bristol-Myers Squibb, USA

Yi Luo

Fibrosis is a key readout of disease progression in NASH and reflects mortality risk. Non-invasive biomarkers capable of diagnosing fibrosis stages and monitoring fibrosis changes in NASH patients are needed. Fibrosis results from the imbalance of fibrogenesis and fibolysis due to sustained tissue injury. We have evaluated serum collagen biomarkers in an observational cohort of patients with biopsy proven NASH and demonstrated that elevated PRO-C3 levels are associated with advanced fibrosis stages in NASH. We further explored novel biomarkers that are correlated with fibrosis stages in this cohort using metabolomics and proteomics analysis.

Improving patient recruitment for clinical trials

Professor of Internal Medicine and Hepatology, Head Division of Hepatology, University of Würzburg, Germany

Andreas Geier

Patient recruitment for NASH trials is often difficult despite the fact that NAFLD is the most frequent chronic liver disease in Western countries affecting 25-30% of the population.

• Awareness of the disease and its natural course is limited in both the general public and medical doctors.
• Screening algorithms to identify NAFLD subjects at risk (NASH or present fibrosis) are recommended by international guidelines but infrequently followed in primary care.
• Specialized secondary diagnostic platforms for elastography and or advanced “direct” fibrosis testing are not widely available in some countries.
• Programs to foster the disease awareness, utilization of screening measures and second line diagnostics are key to improve patient recruitment for clinical trials.

Improving patient recruitment for clinical trials

Professor of Medicine (Digestive Diseases), Yale, USA

Cynthia Moylan

Factors influencing initiation and progression of NASH

• Alpha1-anti-tryspin
• Genetic factors
• Animal models

SOLUTION PROVIDER PRESENTATION:

Samuel Beckett

Commercialisation Coordinator, Helena Biosciences Europe

Glycomics, a powerful new method in the detection, diagnosis and monitoring of chronic liver disease

Glycomics is the study of glycans – polysaccharide structures found on secreted and membrane bound glycoproteins, with roles including cell structure maintenance, signalling, protein folding and cellular recognition. The Glyco Liver Profile is a simple, non-invasive, serum test and has been shown to provide a highly sensitive and specific method for the detection of liver diseases such as NASH as well as an essential tool for the diagnosis and monitoring of chronic liver disease, and predicting the development of HCC. This presentation provides an overview of liver glycomics and the diagnostic capabilities of glycans, discussing how the test is conducted, its role in the detection of NASH, fibrosis and cirrhosis and its potential in providing risk analysis for the development of HCC.

Current Approaches to NASH and Preclinical Strategy

Track Chair: Wajahat Mehal, Professor of Medicine (Digestive Diseases), Yale, USA

Non-invasive biomarkers and diagnostic tools

Track Chair: Julia Brosnan, Senior Director, External Alliances, Internal Medicine Research Unit, Pfizer

The European Medicines Agency is expecting to publish a “Reflection paper on chronic liver disease (PBC, PSC, and NASH)” in autumn 2018, and will conduct a stakeholder meeting at the beginning of December 2018. A detailed display of the contents of this first regulatory guidance with regard to NASH, as well as a preliminary evaluation of the initial feedback and contents of this first regulatory guidance with regard to NASH, as well as a preliminary evaluation of the initial feedback and

Elmer Schabel

Clinical Assessor, Licensing Division 2, Federal Institute for Drugs and Medical Devices (BfArM), Germany

The EMA reflection paper on chronic liver disease and its implications for drug development in NASH

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Christian Trautwein

Director, Medizinische Klinik III, University Hospital, RWTH Aachen, Germany

Keynote Address:

Factors influencing initiation and progression of NASH

• Alpha1-anti-tryspin
• Genetic factors
• Animal models

Cynthia Moylan

Epigenetics and development and progression of nonalcoholic fatty liver disease

• Summarize data on key epigenetic mechanisms of NAFLD development and progression.
• Update current research on DNA methylation, NAFLD and liver fibrosis.
• Discuss the role and future application of DNA methylation as an emerging non-invasive indicator of NAFLD and its progression.
input received by stakeholders is planned to be given in this talk. Both, the reflection paper as well as the feedback received are expected to foster further discussions, including problems of patient selection and endpoints, and on reflections on potential future approval pathways, in a situation when the current unmet medical has already been met.

Adopting machine learning-based analysis using generalised matrix learning vector quantisation, we have achieved excellent separation of controls and NASH groups with area under curve analysis of learning vector quantisation, we have achieved excellent separation of controls and NASH groups with area under curve analysis of

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**POSTER COMPETITION WINNER PRESENTATION**

**GUIDO BASELLI**

PhD Student, University of Milan, Italy

**Transcriptomics highlights interleukin-32 a novel NAFLD biomarker showing higher accuracy in carriers of the pnppl3 i148m variant**

Efforts to manage NAFLD are limited by the absence of accurate noninvasive biomarkers. Thus, we looked for novel candidate NAFLD biomarkers by examining the hepatic transcriptome variability in obese individuals at-risk for progressive NAFLD. We identified the PNPLA3 I148M variant as the major modifier of the transcriptome variability, which was linked to overexpression of inflammatory pathways, and downregulation of oxidative metabolism. Presence of severe NAFLD was associated to overexpression of both metabolic and inflammatory pathways. Notably, IL32 was the most robustly upregulated gene in severe NAFLD, more markedly in carriers of the I148M variant. Plasma IL32 levels were associated with both NAFLD and severe NAFLD independently of serum aminotransferases, and were able to improve aminotransferases accuracy in predicting NAFLD diagnosis.

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**POSTER COMPETITION WINNER PRESENTATION**

**JOOST BOECKMANS**

PhD-Researcher, Department of In Vitro Toxicology & Dermato-Cosmetology (IVTD), Faculty of Medicine and Pharmacy, Vrije Universiteit Brussel, Belgium

**In vitro investigation of the anti-NASH properties of elafibranor and lanifibranor using a human stem cell-derived disease model**

- Human skin precursors (hSKP) are adult stem cells that exhibit the ability to differentiate towards cells with hepatic characteristics (hSKP-HPC) that also hold a well-pronounced lipid metabolism.
- hSKP-HPC exposed for 24h to factors involved in the onset of NASH (insulin, glucose, fatty acids and an inflammatory cytokine cocktail) significantly increase their lipid load and express/secrete inflammatory interleukins (IL6, IL1a and IL11) and chemokines (CCL2, CCL7, CCL8, CXCL5 and CXCL8).
- The observed steatotic and inflammatory responses were restricted in the presence of elafibranor, whereas lanifibranor only reduced the inflammatory response. As such, this novel in vitro system represents a valuable model to investigate potential anti-NASH drugs.

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**REBECCA TAUB**

Chief Medical Officer and Executive Vice President, Research & Development, Madrigal Pharmaceuticals, USA

**MGL-3196 (resmetirom), a liver-selected thyroid hormone receptor-beta agonist, improves NASH and dyslipidemia in Phase 2 studies**

In Phase 2 studies primary and key secondary endpoints were achieved including reduction of liver fat on a sensitive non-invasive imaging test, lowering of multiple atherogenic lipids including LDL-cholesterol and triglycerides, and resolution of NASH on liver biopsy. Based on evidence of broad activity and downregulation of oxidative metabolism. Presence of severe NAFLD was associated to overexpression of both metabolic and inflammatory pathways. Notably, IL32 was the most robustly upregulated gene in severe NAFLD, more markedly in carriers of the I148M variant. Plasma IL32 levels were associated with both NAFLD and severe NAFLD independently of serum aminotransferases, and were able to improve aminotransferases accuracy in predicting NAFLD diagnosis.

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**RUI CASTRO**

Assistant Professor, Department of Biochemistry and Human Biology, Faculty of Pharmacy, University of Lisbon, Portugal

**NASH preclinical models for the study of microRNAs as biomarkers and therapeutic targets**

- At the moment, no single animal model recapitulates all features of human NASH. As such, investigators should carefully choose the dietary or genetic model that best suits their research goals and expected outputs.
- Dietary models have proven particularly useful to test...
hypotheses on NASH molecular triggers and drivers of progression, as well as to identify therapeutic targets or test putative pharmacological agents. In this regard, microRNAs are being increasingly recognized as potential biomarkers and therapeutic targets in NASH.

- In particular, circulating miRNAs embody novel means of disease diagnosis and monitoring, while modulation of metabolism-related miRNAs delays disease triggering and halts NASH progression.

GIULIO MARCHESINI
Professor of Diabetes, "Alma Mater" University, Department of Medical and Surgical Sciences, Italy
A web-based intervention to support lifestyle changes in NAFLD
- Lifestyle changes are mandatory in NAFD, but are scarcely implemented by busy liver units. Lifestyle programs may be jeopardized by job- and time-constraints of patients, unable to regularly attend programmed meetings.
- A web-based program was set-up to facilitate patients education to healthy diet and habitual physical activity, thus promoting and weight loss. The contact with the center was maintained through mails, food diaries exchange, physical activity monitoring.
- The web program compares favorably with face-to-face education, and is better suited for young, busy patients, and for cases living far from Liver units who cannot regularly attend educational programs.

2) Regulation of Inflammation and Fibrosis
WAJAHAT MEHAL
Professor of Medicine (Digestive Diseases), Yale, USA
- Inflammation and fibrosis provides many points of regulation.
- Pattern recognition receptor and inflammasome pathways are attractive for regulation of inflammation.
- Hepatic stellate cell activation and matrix degradation are attractive site for regulation of fibrosis.

3) Collaborative projects: academia, healthcare providers, industry
JULIA BROSnan
Senior Director, External Alliances, Internal Medicine Research Unit, Pfizer, USA
I will highlight the major PPP that are going on in the NAFLD space and why it is advantageous to work together in a precompetitive environment.

4) NASH in diabetes. Is it a priority?
WILLIAM ALAZAWI
Reader in Hepatology, Bart’s Liver Centre, Queen Mary University of London, UK
- Does NASH alter the management of people living with diabetes?
- Does NASH lead to worse cardiovascular outcomes in people living with diabetes?
- Should we screen patients with diabetes for fatty liver?
Multiple compounds are currently in clinical development targeting different pathways involved in NASH pathogenesis. Combination regimens involving compounds directed at different pathophysiological processes would allow tailoring therapy for different disease stages and are expected to be more successful with a larger proportion of treatment responders, as well as greater efficacy.

**Karine Clément**  
Director of INSERM/Sorbonne University, NutriOomics team, France  
**Gut Microbiome signature of NAFLD/NASH; can we disentangle from metabolic signals**

This talk will address the current knowledge on microbiota composition/signatures in NAFLD/NASH and liver fibrosis taking advantage of current research activities of European consortia (EU-ePos and Litmus) and existing literature background. Some published literature has addressed the potential mechanistic relationships between gut microbiota and human NAFLD phenotypes. However, there is still a need to address the question whether it exists peculiar signatures of liver diseases being independent of metabolic disorders such as obesity and Type 2 diabetes, common diseases increasing the risk of liver injury. There is indeed a need to disentangle these conditions in the context of gut microbiota studies and in the future the challenge will be to identify gut microbiota and microbiota-derived metabolites that could predict the disease stage of progression.

**Luca Valenti**  
Associate Professor of Internal Medicine, University of Milan, Italy  
**Genetics of progressive nonalcoholic fatty liver disease**

- Hepatic fat accumulation and nonalcoholic fatty liver disease (NAFLD), especially the progressive form of the disease, have a strong heritable component.
- The I148M variant of PNPLA3 is the main common genetic determinant of NAFLD, and is associated with the whole spectrum of liver damage, ranging from simple steatosis to cirrhosis and hepatocellular carcinoma development. However, variation in TM6SF2, MBOAT7, GCKR, PPP1R3B and HSD17B13 also contribute to disease risk, and rare mutations in genes involved in lipid metabolism, liver disease and cancer predisposition contribute as well.
- We are now starting to translate these new discoveries into the clinics, to improve stratification of the risk of progressive NAFLD and identify new therapeutic approaches.
CONGRESS SCHEDULE

3) NASH and CVD
FADY NTANIOS
Senior Director, Global Medical Affairs, Pfizer, USA

*Full talk details can be found at the end of Day 2

Lunch
12:50-13:50

ROBERT WALCZAK
EVP, Head of Research, GENFIT SA, France
Elafibranor as a potential first-line therapeutic and a scaffold for multiple combination therapies in NASH

Elafibranor is a novel PPAR α/δ agonist and the first NASH drug candidate to demonstrate NASH resolution without the worsening of fibrosis while concurrently improving cardiometabolic risk factors. Furthermore, NASH resolution was correlated with fibrosis improvement in patients of the GOLDEN505 study. Elafibranor is safe, with good tolerability and is now being investigated in a large phase 3 trial, RESOLVE-IT. Given the favorable profile of Elafibranor, GENFIT has explored therapeutic combinations to identify synergistic mechanisms of action. Here, we provide an overview of Elafibranor including disease model data from our combination program.

SUSANNE KASER
Associate Professor, Department of Internal Medicine I, Medical University Innsbruck, Austria
Specific effects of anti-diabetic therapies on fatty liver disease

• Significant weight reduction has been proven to improve steatosis, inflammation and fibrosis in patients with NASH.
• Some anti-diabetic therapies that are commonly associated with weight reduction are currently under evaluation as treatment strategies for NAFLD.
• Common treatment targets include GLP-1 signaling and PPAR gamma in diabetes and NASH.

DAVID FRASER
CSO, NorthSea Therapeutics, Norway
Targeting inflammatory and fibrotic pathways in NASH via a structurally engineered fatty acid, icosabutate

• Fatty-acid responsive pathways play a pivotal role in regulating hepatic inflammation and fibrogenesis/fibrolysis
• A liver-targeted structurally engineered fatty acid, icosabutate, effectively targets these pathways and exhibits potent anti-inflammatory and anti-fibrotic effects in multiple, differentiated, rodent NASH models
• Normalisation of elevated liver enzymes in dyslipidemic humans supports rodent data and, in addition to insulin sensitising and hypolipidemic effects, suggests that icosabutate could offer a potent oral treatment for NASH and its associated comorbidities.DAVID FRASER – NorthSea Therapeutics (SEFAs – Structurally Engineered Fatty Acid)

ALEXANDRE GRASSIN
VP Finance and Administration, Genkyotex, Switzerland
GKT831: A novel PII anti-fibrotic small molecule

• GKT831, a NOX1 and NOX4 inhibitor, is evaluated in a phase 2 clinical trial in primary biliary cholangitis (PBC, a fibrotic orphan disease). Positive interim efficacy results were released November 5th, final results expected in Spring 2019 – over 75% of patients have completed the full 24 week treatment.
• GKT831 is also evaluated in an investigator-initiated Phase 2 clinical trial in Type 1 Diabetes and Kidney Disease (DKD).
• A grant from the US NIH of $8.9 million was awarded to fund a multi-year research program evaluating the role of NOX enzymes in idiopathic pulmonary fibrosis (IPF), the core component of the program will be to conduct a Phase 2 trial with the GKT831 in patients with IPF.

PIERRE BROQUA
CSO/COO, Inventiva, France
Lanifibranor: a moderate and well-balanced panPPAR agonist for the treatment of NASH

• Evidence that PPARsa, δ and γ regulate multiple pathways involved in the physiopathology of NASH
• Lanifibranor panPPAR profile
• Data supporting lanifibranor therapeutic potential in NASH

Room: Lindbergh Lobby
2:00-2:25

Track Chair: Ronit Shiri-Sverdlov, Professor of Hepatic Inflammation and Metabolic Health, Maastricht University, The Netherlands

RONIT SHIRI-SVERDLOV
Professor of Hepatic Inflammation and Metabolic Health, Maastricht University, The Netherlands
Role of lysosomes in NASH

• Disturbed lipid metabolism during NASH contributes to lysosomal dysfunction
• Circulating (plasma) lysosomal enzymes can be used as biomarkers for NASH
• Reducing the activity of circulating lysosomal enzymes is a novel approach to treat NASH

Track Chair: Richard Marshall, Chief Medical Officer, Galecto Biotech, UK

AXEL GRASSIN
VP Finance and Administration, Genkyotex, Switzerland
GKT831: A novel PII anti-fibrotic small molecule

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2:00-2:25

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• Evidence that PPARsa, δ and γ regulate multiple pathways involved in the physiopathology of NASH
• Lanifibranor panPPAR profile
• Data supporting lanifibranor therapeutic potential in NASH

Room: Lindbergh Lobby
2:00-2:25
ADIL MARDINOGLU
Professor of Systems Biology, King's College London, UK
Employment of systems biology in treatment of liver diseases
To develop novel strategies for prevention and treatment as well as to gain detailed insights about the underlying molecular mechanisms of liver diseases, it is vital to study the biological functions of liver and its interactions with other tissues and gut microbiota. Biological networks can provide a scaffold for studying biological pathways operating in the liver in connection with disease development in a systematic manner. In my presentation, I will present our recent work where biological networks have been employed to identify the reprogramming in liver physiology in response to NASH/NAFLD. I will further discuss how this mechanistic modelling approach can contribute to the discovery of biomarkers and identification of drug targets which may lead to design of targeted and effective treatment strategies.

1) From triglycerides to toxic lipids in NASH
OREN TIROSH
Institute of Biochemistry, Food Science and Nutrition, Faculty of Agriculture, Food and Environment, The Hebrew University of Jerusalem, Israel
Treatment of NASH will require the development of novel anti-lipotoxic treatments. The progression of NASH and development of fibrosis is a multifactorial process that comprises metabolic, inflammatory and cell death related events. Some of the lipid involved in disease progression are pro-inflammatory and some are toxic. The session will discuss the following topics in NASH and lipotoxicity: 1) Insulin resistance, metabolism and lipotoxicity: are they connected and what are the clinical evidences in lean and obese NAFLD patients? 2) toxic lipids leading to liver toxicity compared to pro-inflammatory lipids and mediators, 3) Drugs and dietary treatments to ameliorate lipotoxicity and to protect the liver: are we there?

2) Invasive and non-invasive Biomarkers for NASH/NAFLD
VINOOD PATEL
Reader in Clinical Biochemistry, University of Westminster, UK
• Whilst liver biopsy is the gold standard for diagnosing NASH and determining the stage the procedure, lacks the ability to be high throughput, is invasive and has associated risks, and can result from sampling variation.
• Biomarkers both invasive and non-invasive provide a real solution for both diagnosing, staging and prognosis of NASH/NAFLD. A number of blood-based markers or when combined with clinical variables have shown good clinical diagnostic outcomes. In addition, several non-invasive algorithms have shown excellent diagnostic accuracy for determining advanced fibrosis.
• The challenge is to obtain clinical validation of current and new markers and assessing best practice in terms of their application in diagnosing, staging or prognosis of NASH/NAFLD.

3) NASH and CVD
FADY NTANIOS
Senior Director, Global Medical Affairs, Pfizer, USA
• Non-alcoholic fatty liver disease (NAFLD) / non-alcoholic steatohepatitis (NASH) and the associated cardiovascular (CV) disease risks.
• Critical first steps towards successfully developing NASH treatments will be improving our understanding of the disease, the CV risk factors associated with NASH and the patients at higher risk of having NASH and CVD.
• Key questions for discussion at the Roundtable:
  1. Is NASH a driver of cardiovascular disease?
  2. Can we reverse or decrease CV risk factors by treating NASH?
  3. What is the relationship between various stages of NASH and CVD risks?
  4. How will new NASH therapies be regarded by CV specialists such as cardiologists?
  5. How do we increase the awareness of NASH among CV professional and patients associations?

VENUE INFORMATION
London Heathrow Marriott Hotel
Bath Road, Heathrow Airport Hayes, UB3 5AN, United Kingdom
Located less than half a mile away from the Heathrow Airport, this four-star deluxe hotel offers comfortable, noise-free accommodations and is near attractions such as Legoland and Windsor Castle. Modern and vibrant, discover the culinary delights and more in the London Heathrow Marriott.
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<th>POSTER PRESENTATION TITLE</th>
<th>PRINCIPAL AUTHOR(S)</th>
<th>AFFILIATION</th>
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<tr>
<td>An obese mouse model of diet-induced NASH with biopsy-confirmed advanced fibrosis and progressive tumor development</td>
<td>Denise Oro, Magnus Sæholt Larsen, Niels Vrang, Michael Feigh</td>
<td>Gubra, Harsholm, Denmark</td>
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<tr>
<td>Mobile health, physical activity and obesity: subanalysis of a randomized controlled trial</td>
<td>Chang Hee Lee, PhD, Booyoon Cheung, MD, Ga-Hye Yi, MD, Bumjo Oh, MD, MPH, Yun Hwan Oh, MD, MS</td>
<td>SMG-SNU Boramae Medical Center, Seoul, Korea</td>
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<td>Targeting hepatic Glutaminase 1 ameliorates Non-Alcoholic Steatohepatitis by restoring disrupted hepatic Very-Low-Density Lipoproteins triglyceride assembly</td>
<td>Jorge Simon1, Maitane Nuñez-Garcia1, Pablo Fernandez-Tussy1, Lucia Barbier-Torres1, David Fernandez-Ramos1, Beatriz Gomez-Santos1, Maitane Nuñez-Garcia1, Mikel Azkarate1, Virginia Gutierrez-de Juan1, Marta Varela-Rey1, Naraa Goolkotseg Usandizaga1, Pablo Fernandez-Tussy1, Patricia Aspichueta1, Diego Saenz de Urturi1, Xabier Bueque1, Paula Inzulbietza1, Javier Crespo1, Shelly C. Lu1, Jose M. Mato1, Felix Ertortza1, Maria Luz Martinez-Chantar1</td>
<td>SMG-SNU Boramae Medical Center, Seoul, Korea</td>
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<td>1RenaSci, Nottingham, United Kingdom; 2DeuteRx, Andover, MA, United States</td>
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<td>1Liver disease laboratory, Metabolomics Unit, CIC bioGUNE, Basque Country; 2Technical Park of Biscay, 48160; 3Proteomic Platform, CIC bioGUNE, CIBERehd, Technologic Park of Biscay, 48160; 4University of Basque Country &amp; Biocruces Health Research Institute, Physiopathology, Faculty of Medicine and Nursing, Leioa &amp; Barakaldo, Spain; 5Division of Digestive and Liver Diseases, Cedars-Sinai Medical Center, 4Marques de Valdecilla University Hospital, Santander, Cantabria; 6Centre of investigation Biomedica in Red de Enfermedades Hepáticas y Digestivas (CIBERehd), Instituto de salud Carlos III, Madrid</td>
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<td>Michigan State University</td>
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<td>Department of In Vitro Toxicology &amp; Dermato-Cosmetology (VITD), Faculty of Medicine and Pharmacy, Vrije Universiteit Brussel</td>
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