A joint lipid conference 2026
21st GERLI international lipid meeting
65th International Community on the Bioscience of Lipids

LIPIDS

IN ALL STATES

**FROM MEDICINE AND PHARMACY
TO PHYSICAL CHEMISTRY AND PLANT SCIENCES**

Bordeaux, FRANCE
September 21–24, 2026

BOOK OF ABSTRACTS

https://www.gerli.com/congres/icbl-gerli-2026-lipid-conference/

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GENERAL INFORMATION

PROGRAM

ABSTRACTS

Welcome to Bordeaux for the ICBL-GERLI Conference

Dear Attendees,

The ICBL (International Conference on Lipid Biosciences) and the GERLI (Lipid Study and Research Group, France) are joining forces to organize a symposium on lipid science entitled "Lipids In All States." This symposium will be held in Bordeaux, a magnificent city known worldwide for its renowned wines, cultural heritage, and scientific activities.

The conference venue is located on the University of Bordeaux campus, in a former church that has been converted into a magnificent conference hall, baptized Agora. The poster sessions will take place in the cloister. We hope that these surroundings will inspire the mind and foster elevated intellectual exchanges in the field of lipid science, among others.

As we know, lipids are ubiquitous. A wide variety of topics will therefore be covered, including Membrane Lipids, Lipids in Algae and Plants, Lipids in Nutrition and Health, Lipids in the Brain, Biopolymers and Lipids, Lipids Biodiversity, Lipids in Medicine and Pharmacy, as well as Lipids, Analysis and Technology.

The conference will begin on Monday, September 21, with a workshop on lipid and membrane imaging techniques, a rapidly expanding field. It will be followed by two opening lectures: the GERLI lecture and the ICBL lecture.

We look forward to your active participation through invited lectures, oral presentations, one-minute flash-talk poster presentations, poster discussions during the eight scientific sessions, the two poster sessions as well as a session dedicated to technological and industrial challenges and the role of artificial intelligence in science.

Participation in pseudoscientific activities, such as the public lecture on tannins and lipids in wine, accompanied by hands-on wine and cheese tasting workshops, will also be encouraged on Wednesday afternoon during the wine tour and congress diner.

We look forward to welcoming you in Bordeaux!

On behalf of the Scientific and Organizing Committees

Erick Dufourc, Chair and Hubert Schaller, co-chair and GERLI president

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- Nicolas VITALE

Invited Speakers

Clotilde Théry, Institut Curie, Paris (France)

Clotilde Théry is a professor and INSERM director of research at Institut Curie in Paris, France. She is president of the International Society for Extracellular Vesicles (ISEV), where she previously served as founding secretary general and as editor-in-chief of the Journal of Extracellular Vesicles. She is the team leader of the group « Extracellular Vesicles, Immune Responses and Cancer » within the INSERM Unit 932 focusing on « Immunity and Cancer. » Clotilde Théry researches extracellular vesicles that are released by immune and tumor cells, including exosomes that originate in the multivesicular body. After a PhD in Paris (France), and a post-doc in Oxford (UK) and Columbia Universities (USA), where she studied the development of the nervous system, she turned to the cell biology aspects of immune responses in 1996 when joining the lab of S. Amigorena, at Institut Curie in Paris.

She is the author of more than 300 papers that are cited more than 80 000 times.

George Carman, Rutgers, The State University of New Jersey (USA)

Dr. Carman is a Rutgers University Board of Governors Professor, Distinguished Professor, and Founding Director of the Rutgers Center for Lipid Research.

His laboratory has made significant contributions to the understanding of lipid synthesis and its regulation through the isolation and characterization of key genes and their encoded enzymes. He is an Inaugural Fellow of the American Society for Biochemistry and Molecular Biology and Fellow of the American Academy of Microbiology.

Awards/honors include the Herbert Tabor Research Award, Avanti Award in Lipids, Supelco/Nicholas Pelick Research Award, Faculty Mentor of the Year Award-Compact for Faculty Diversity, Journal of Lipid Research Lectureship Award, Selman A. Waksman Honorary Lectureship Award, and a Highly Ranked Scholar by ScholarGPS. Editorial service includes Associate Editor of *the Journal of Biological Chemistry*, Associated Editor of *the Journal of Lipid Research*, Executive Editor of *Biochimica et Biophysica Acta-Molecular and Cellular Biology of Lipids*, and Executive Editor of Analytical Biochemistry.

Raphaël Rodriguez, Institut Curie, Paris (France)

Raphaël Rodriguez, is a Director of Research at the CNRS, and a researcher at the Institut Curie, where he holds the Skłodowska-Curie Chair in Chemical Biology.

He trained as a doctoral student and then postdoctoral researcher under the mentorship of Jack Edward Baldwin and Shankar Balasubramanian, with whom he acquired his knowledge of synthetic chemistry and supramolecular chemistry. He then studied cell biology with Stephen Jackson with whom he also co-founded Adrestia Therapeutics. He then joined the Institut Curie in Paris, 2015, where he studied the molecular basis underlying cancer biology.

There, he discovered the key role of metals in regulating the plasticity of cancer and immune cells. He is a member of the Royal Society of Chemistry (FRSC) and was named “Chevalier de l’Ordre National du Mérite”. He has received several distinctions and awards for his scientific contributions, including the Tetrahedron Young Investigator Award, the Klaus Grohe Award, the Lacassagne Award (Collège de France) and the Charles Defforey Grand Prize (Institut de France), the Liliane Bettencourt Prize for Life Sciences in 2023, and the CNRS Silver Medal in 2024.

He published more than 150. Papers which were cited about 10,000 times

Eric Maréchal, Laboratoire Physiologie Cellulaire & Végétale, Grenoble (France)

Eric Maréchal is a CNRS scientist and deputy Director of the Laboratoire de Physiologie Cellulaire & Végétale (LPCV) in Grenoble, France.

His research explores the evolution of algal and plant cell membrane architecture in plastid-containing eukaryotes. He investigates membrane and storage lipid metabolic pathways in the context of evolutionary processes - particularly primary and secondary endosymbiosis - as well as environmental constraints such as nutrient limitation and temperature variation. His experimental models include green algae and plants, diatoms and other stramenopiles, and apicomplexans. More recently, he has developed a strong research focus on the adaptive mechanisms of algae thriving in cryophilic environments, including snow, glaciers and sea ice.

He currently coordinates the AlpAlga project.

Parveen Yaqoob, University of Reading (UK)

Professor Parveen Yaqoob is Deputy Vice-Chancellor and Pro-Vice-Chancellor Research and Innovation at the University of Reading, where she has worked for 27 years.
Her research has focused on nutritional modulation of immune function, inflammation and vascular function, and she has a particular interest in the influence of n-3 polyunsaturated fatty acids on the generation and function of extracellular vesicles.
She has contributed significantly to both national and international research funding panels for food, nutrition and health and was appointed OBE for services to higher education in 2022.

Richard Bazinet, Department of Nutritional Sciences, Temerty Faculty of Medicine, University of Toronto (Canada)

Pr Richard Bazinet is a Professor and Canada Research Chair in Brain Lipid Metabolism at the University of Toronto, internationally recognized for his pioneering research into how fatty acids impact the brain.

His major scientific contributions center on understanding the regulation of brain lipid metabolism, especially the roles of dietary polyunsaturated fatty acids (PUFA) like arachidonic acid (AA), eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) in neurological health. His discoveries have shed light on how these fats are metabolized in brain phospholipids and their critical involvement in neurodegenerative and neuropsychiatric disorders.

Recently, Bazinet's research demonstrated that the liver can generate palmitic acid to support brain health, and his lab's kinetic and biochemical approaches have revealed novel mechanisms regulating fatty acid turnover and inflammation in the brain.

Since 2006, he has published over 250 scientific papers cited more than 7500 times (H43) and contributed to international guidelines on dietary fats for brain health. His work influences policy and public health recommendations worldwide. He is the past-president of the International Society for the Study of Fatty Acids and Lipids, editor-in-chief of Prostaglandins, Leukotrienes and Essential Fatty Acids, and recipient of numerous prestigious awards including the Ralph Holman Lifetime Achievement Award and the Supelco AOCS Research Award for Outstanding, Original Research in Fats, Oils, Lipid chemistry or Biochemistry.

Rochus Benni Franke, Institute of Cellular and Molecular Botany, Bonn (Germany)

Dr. Rochus Benni Franke graduated with a Diploma in Biology from the University of Kaiserslautern in Germany.

In his PhD research in Dr. Heinrich Kauss' lab in Kaiserslautern and partly in Dr. Steven Fry's lab at University of Edinburgh he studied cell wall modifications during plant defense reactions. As a post-doc in Dr. Clint Chapple's lab Benni chemically phenotyped new lignin-engineered plants. In a second project he identified and biochemically characterized an unaccounted enzyme in the essential phenylpropanoid pathway. As research associate at the University of Heidelberg he investigated and optimized the biosynthesis of tumor inhibiting alkaloids from cultures of medicinal plants.

In 2001 he joined the Institute for Cellular and Molecular Botany at University of Bonn as assistant professor where he initiated molecular genetic research approaches in the field of plant-environment-interfaces. These pioneering analyses resulted in the identification of multiple genes in the formation of physiologically important biopolymers such as cutin, suberin and Casparian strips. Benni continued in Bonn as adjunct professor and research activities focusing on the biosynthesis of apoplastic diffusion barriers and how their chemistry affects the plants water and nutrient homeostasis and biotic interactions.

Gwendolyn Barceló-Coblijn,

Fundació Institut d'Investigació Sanitària Illes Balears (IdISBa)
— Hospital Universitari Son Espases (Balearic Islands, Spain)

Dr. Gwendolyn Barceló-Coblijn established her research group in 2012 at the Health Research Institute of the Balearic Islands (IdISBa). She is a full researcher at this institution since 2019. Dr. Gwendolyn Barceló-Coblijn's research focuses on unraveling the role of membrane lipid remodeling in cell physiology and pathology, focusing on how spatial lipid distribution dictates cell identity and disease progression. Her group uses matrix-assisted laser desorption ionization-mass spectrometry imaging (MALDI-MSI) to profile the lipidomes of complex microenvironments at near-single-cell resolution.

In cancer research, her research has established that lipid remodeling acts as a highly structured, spatially organized dimension of tumor heterogeneity. Conversely, by integrating transcriptomic and lipidomic spatial profiles, her team uncovered how the depletion of total plasma fatty acids serves as an intrinsic metabolic alteration in colon cancer, occurring even in the early stages of the disease.

Her recent work profiling ulcerative colitis (UC) demonstrated that chronic intestinal inflammation severely alters epithelial membrane lipid gradients, reverting cells back to a less-differentiated, stem-like state. Additionally, her work extends to identifying spatial lipid patterns in other inflammatory conditions. Dr. Gwendolyn Barceló-Coblijn is member of the ICBL steering committee since 2023.

Currently, she is the scientific coordinator of a IdISBa's newly stablished facility on Molecular Imaging and Mass Spectrometry.

Per Larsson, Department of Pharmacy, Uppsala Biomedical Center, Uppsala University (Sweden)

Per Larsson is an Associate Professor in the Department of Pharmacy at Uppsala University, Sweden, where he leads a research group in computational pharmaceutics within the national drug delivery competence center SweDeliver.

His work uses multiscale simulation, from coarse-grained molecular dynamics to computational fluid dynamics, together with scattering and imaging methods, to understand how the lipids of the gastrointestinal tract shape oral drug performance.

A central theme is the self-assembly of bile salts, phospholipids and digestion-derived fatty acids into the colloidal structures that solubilize poorly water-soluble drugs and modulate the activity of fatty-acid permeation enhancers for oral peptide delivery. Spanning these molecular-to-luminal scales, his simulations have been central to the development of a “virtual intestine”, a multiscale model of drug behavior in the gut.

He holds a PhD from Stockholm University and carried out postdoctoral research at the University of Virginia, USA. He serves on the editorial advisory board of the journal Molecular Pharmaceutics, and has authored more than 50 publications, cited approximately 13,000 times.

Claire Berton-Carabin, Laboratoire Biopolymères Interactions Assemblages, Nantes (France)

Claire Berton-Carabin is currently Research Director at INRAE (the French National Research Institute for Agriculture, Food and the Environment), in the research unit BIA (Biopolymers, Interactions, Assemblies) in Nantes, France, where she leads a research team of about 30 members, titled Interfaces and Dispersed Systems.

She also holds a Visiting Associate Professor position at Wageningen University and Research (Netherlands) in the Laboratory of Food Process Engineering (FPE), where she was fully appointed between 2013 and 2020.

Before that, she performed her PhD research at INRAE (formerly INRA) and received her PhD degree in 2011. She then conducted two postdoctoral projects (Penn State University, Department of Food Science, USA, 2011-2013; and Danone-Nutricia Research, the Netherlands, 2013).

Claire's research is focussed on understanding the interplay between the structure of food emulsions (in particular, at the level of the oil-water interface) and their functionality and chemical reactivity.

Her research interests therefore range from the characterization of the early formation and stabilization of emulsion droplets, all the way to their subsequent physicochemical stability and final fate during digestion.

To date, Claire is the author of 120 scientific publications with more than 7200 citations, 1 patent and 9 book chapters.

She is a member of the executive board of the French Society for Lipid Research (SFEL). She received the Euro Fed Lipid 'Young Lipid Scientist Award' in 2017, and the 'Promising Researcher Award' of INRAE in 2022.

Katelyn Camille Cook, Max Planck Institute for Molecular Cell Biology and Genetics, Dresden (Germany)

A cell biologist at heart and a mass spectrometrist by training, Katelyn develops interdisciplinary workflows to uncover the organizing principles of organelle membranes.

She is a postdoctoral scientist at the Max Planck Institute for Molecular Cell Biology and Genetics in Dresden, which she joined in 2023 after earning her PhD in Molecular Biology from Princeton University in 2022 and her BA in Molecular, Cellular, and Developmental Biology from the University of Colorado Boulder in 2016.

Working with André Nadler, Katelyn recently established a discovery pipeline for mechanistic lipid cell biology that uses bifunctional phospholipid probes, lipid imaging, and proteomic affinity capture to map the protein interactomes of individual lipid species and generate testable hypotheses about lipid function and the formation of specialized membrane domains.

GERLI and ICBL thank all institutional and private sponsors for their kind support

International Lipidomics Society (iLS)

ADVANCING LIPIDOMICS • CONNECTING COMMUNITIES • TRANSFORMING HEALTH

Our Mission:
Advance lipidomics through global collaboration, innovation, education, and standardization, and promote the translation of lipidomics into biological and clinical. applications.

1. SCIENTIFIC LEADERSHIP

- Develop best-practice guidelines and reporting standards (e.g., Lipidomics Standards Initiative)
- Coordinate inter-laboratory studies and reference material development
- Support methodological and technological advances in lipidomics

2. COMMUNITY BUILDING

- Foster international collaborations across academia, healthcare, and industry
- Coordinate thematic Interest Groups covering diverse areas of lipidomics
- Create a welcoming and inclusive global community

iLS INTERNATIONAL LIPIDOMICS SOCIETY

3. EDUCATION & TRAINING

- Organize international conferences, workshops, webinars, and training courses
- Provide educational resources on lipidomics
- Support early-career scientists through travel awards and networking opportunities

4. INNOVATION & INFRASTRUCTURE

- Promote open data, digital tools, and cloud-based resources (e.g., LipidCascade)
- Support bioinformatics and data standards
- Facilitate communication between researchers, funding agencies, and industry partners

5. ADVOCACY & IMPACT

- Raise awareness of the value of lipidomics
- Engage with policymakers and funding bodies
- Promote the translation of lipidomics into biomedical and clinical practice

INFRASTRUCTURE FOR QUANTITATIVE LIPIDOMICS

i) LipidCascade
for Lipidome evaluation

LipidCascade

ii) LipidCompass
for Lipidome publication and sharing

LipidCompass

REPRESENTATIVES FROM **23** COUNTRIES

GENDER BALANCE: ♀ 44% ♂ 56%

STUDENTS 9%

POSTDOCS 8%

REGULAR 76%

OVERALL IMPACT

Building a global, collaborative ecosystem that accelerates lipidomics research and its translation into biomedical and clinical practice.

- Stronger Global Community
- Better Science & Standards
- Open Data & Shared Resources
- Trained Next Generation
- Health Impact for Society


CARDIOVASCULAR RISK • L5 ASSAY
HEART
Health Resource Technology, LLC
Go Beyond LDL-C to Identify Cardiovascular Risk
L5 is critical!
01 WHY L5 MATTERS
LDL can be divided into five subclasses (L1–L5). L5, the most electronegative subclass, is associated with atherogenesis and thrombosis.
Healthy
Non-L5: 99.55%
L5: 0.45%
02 L5 is increased in...
Hyperlipidemia
Autoimmune Diseases
Chronic Kidney Disease
Diabetes Mellitus
Coronary Syndrome
Chronic Smoker
ASCVD
Non-L5: 92.87%
L5: 7.13%
03 OUR SERVICE
Rapid
Multistep UC/SEC/FPLC/HPLC in 6 hrs
2 wks
6 hrs
Reliable
Consistent assay performance
Risk Stratification
Precision risk profiling beyond standard LDL
L5 AND CARDIOVASCULAR RISK
High LDL-C does not always predict atherosclerotic cardiovascular disease (ASCVD) events.
Low LDL-C does not guarantee freedom from ASCVD.
Higher L5% in LDL is associated with higher ASCVD.
Learn more about L5 testing
visit us at www.hearthrtllc.com/en/
HEART, Health Resource Technology, LLC


ABITEC
Larodan Research Grade Lipids
Custom Research Grade Lipids
Fatty Acids
Food Oils
Glycerides
MCPD
Phospholipids
LNP Lipids


JBC
JOURNAL OF
BIOLOGICAL
CHEMISTRY


JLR
JOURNAL
OF LIPID
RESEARCH

SCIENTIFIC PROGRAM

Venue: Agora du Haut-Carré, Université de Bordeaux,
43 rue Pierre Noailles – 33400 TALENCE
Tram B, get off at the FORUM stop, then walk for 5 minutes

Monday, September 21, 2026

Workshop on Imaging techniques for lipids and membranes (Agora)

Chairpersons **Emmanuelle Bayer & Nicolas Desbenoit**

14:00 – Registration to the workshop

14:15 – 14:30 Welcome to the workshop

Invited lecture

14:30 – 15:00 **Katelyn Camille Cook,** MPI for Cell Biology & Genetics, Dresden, Germany.
Identifying the lipid-protein interactions that build organelle membranes.

Oral Communications

15:00 – 15:15 **Emmanuelle Bayer,** Laboratory of Membrane Biogenesis, INRAE Bordeaux Aquitaine, France.
Talking through walls: Lipid-protein interactions controlling plasmodesmal communication.

15:15 – 15:30 **Nicolas Desbenoit,** Institute of Chemistry & Biology of Membranes & Nanoobjects, CNRS, University of Bordeaux**,** Bordeaux INP, France.
Multimodal Mass Spectrometry Imaging for the Spatial Characterization of Lipids and Cellular Microenvironments.

15:30 – 15:45 **Anthony Don,** Charles Perkins Centre and School of Medical Sciences, The University of Sydney, Camperdown, Australia.
Quantification and Localisation of New Brain Lipid Synthesis Using Deuterium Oxide and High-Resolution Mass Spectrometry.

15:45 – 16:00 **Jamie Bride,** PhyMedExp, Université de Montpellier, Inserm, CNRS, Montpellier, France.
Characterization of non-enzymatic oxidized polyunsaturated fatty acids (NEO-PUFAs) interactomes and the associated signalling pathways by the use of click-NEO-PUFAs.

16:00 – 16:15 **Teresa Ximelis,** Institut d'Investigació Sanitària Illes Balears (IdISBa), Palma, Balearic Islands, Spain.
A coordinated peroxisome–DHA–plasmalogen programme marks secretory and metastatic epithelial states in human colon cancer

16:15 – 16:30 **Raphaël Vatin,** Structural Virology Unit, UMR3569, Institut Pasteur, 75015 Paris, France.
Impact of the Rift Valley fever virus NSm protein on lipid droplet metabolism

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21st GERLI and 65th ICBL Conference

16:45 – 17:30 **Registration and Coffee** (Badiane room)

17:30-18:00 **Opening and Welcome Addresses** (Agora)
Erick Dufourc

Opening Lectures

Chairpersons **Hubert Schaller & Markus A. Wenk**

GERLI Polonovski lecture

18:00 – 19:00 **Clotilde Thery**, INSERM U932/Institut Curie, Paris, France
Extracellular vesicles: complex lipid-enclosed structures with roles in tumor-immune system cross-talk.

ICBL Van Deenen lecture

19:00 – 20:00 **George Carman**, Rutgers Center for Lipid Research, Rutgers University, New Brunswick, New Jersey 08550, USA
Mechanistic Insights into Phosphatidic Acid Phosphatase in Lipid Synthesis Regulation.

20:00 – 22:00 **Welcome reception** (Badiane room)

Tuesday, September 22, 2026

Session 1 Membrane Lipids (Agora)

Chairpersons **Erick Dufourc & Markus Keller**

Invited lecture

9:00 – 9:30 **Raphael Rodriguez,** Institut Curie, CNRS, INSERM, PSL Research University, Paris, France.
Chemical control of cell adaptation.

Oral Communications

9:30 – 9:45 **Katrin Watschinger,** Institute for Molecular Biochemistry, Biocenter, Medical University of Innsbruck, Innsbruck, Austria.
Ether lipid deficiency alters bone architecture in mice and impairs osteoclast differentiation in vitro.

9:45 – 10:00 **Leja Perne,** Department of Molecular and Biomedical Sciences, Jožef Stefan Institute, Jamova cesta 39, Ljubljana, Slovenia.
Lipid droplet composition and oxidative state determine their role in ferroptosis.

10:00 – 10:15 **Marina Placci,** Institute for Bioengineering of Catalonia (IBEC), Barcelona Institute of Science and Technology (BIST), Barcelona, Spain.
Membrane Remodeling in Lysosomal Storage Disorders: The Role of GlcCer and Gb3.

10:15– 10:30 **Louis Bunel,** École Normale Supérieure (ENS), Université Paris Sciences et Lettres (PSL), CNRS, Sorbonne Université, Université Paris-Cité, Paris, France.
Phospholipid-cation interactions alter membrane proteins activity: The Case of SNARE-Catalysed Membrane Fusion.

10:30 – 11:00 **Coffee break** (hanging posters in Cloister) & **Sponsors Booth** (Badiane room)

Session GERLI *PhD* Awards (Agora)

Chairpersons **Cécile Gladine & Hubert Schaller**

11:00 – 11:15 **Clara Piersson,** Univ. Bordeaux, CNRS, Bordeaux INP, CBMN, UMR 5248, IECB, Pessac, France.
Local Organization of Biological Membranes Modulates Tau−Lipid Interactions and Fibril formation.

11:15 – 11:30 **Louise Fougère,** Laboratoire de Biogenèse Membranaire, Université de Bordeaux, CNRS UMR5200, Villenave-d'Ornon, France.

Revisiting the early secretory pathway in plant cells: identification of a dynamic tubulo-vesiculated ER-Golgi intermediate compartment (ERGIC) that matures in Golgi.

11:30 – 11:45 **Aline Abou Assi (GERLI-SFN Prize),** Institute for Global Health (ISGlobal), Barcelona, Spain.
Biomarkers of early exposure to polyunsaturated fatty acids and their links with child health and development.

Session Poster flash-talks (Agora)

Chairperson **Gérard Lambeau**

11:45 – 12:30 Students Competing for Poster Prize: 1 slide, 1minute

12:30 – 14:00

➔ Lunch Buffet (Badiane room, along with the **sponsor booths**), & **Poster viewing, Even numbers** (Cloister)

13:00-14:00, **Presentation of Posters to the Jury**

Session 2 Algae and Plant Lipids (Agora)

Chairpersons **Frédéric Domergue & Hubert Schaller**

Invited lecture

14:00 – 14:30 **Eric Maréchal,** Laboratoire de Physiologie Cellulaire et Végétale, CNRS, CEA, INRAE, UGA, CEA-Grenoble, Grenoble, France.
Evolutionary repurposing of MGDG synthases from photosynthetic membrane biogenesis to new eukaryotic functions

Oral Communications

14:30 – 14:45 **Aleksandra Weremczuk,** Institute of Biochemistry and Biophysics Polish Academy of Sciences, Warsaw, Poland.
Very long-chain polyisoprenoids accumulate in rose leaves represent an overlooked class of plant lipids.

14:45 – 15:00 **Birgit Habenstein,** Univ. Bordeaux, CNRS, Bordeaux INP, CBMN, UMR 5248, IECB, Pessac, France.
Remorin-mediated protein-lipid networking during nanodomain formation in plant cells.

15:00– 15:15 **Ana Paula Alonso,** BioDiscovery Institute and Department of Biological Sciences, University of North Texas, Denton, USA

^{13}C-Metabolic Flux Analysis Elucidates Carbon Partitioning during Unusual Fatty Acid Synthesis

15:15 – 15:30 **Claire Bréhélin,** Laboratoire de Biogenèse Membranaire, UMR5200 CNRS, Université de Bordeaux, Villenave-d'Ornon, France.
Dynamics and role of 3-hydroxylated lipids in the regulation of dormancy in cherry floral buds.

15:30 – 16:00

➔ Coffee break (Badiane room, along with the sponsor booths), & Poster viewing, Even numbers (Cloister)

Session 3 - Lipid Nutrition & Health (co-organized with SFEL) (Agora)

Chairpersons **Cécile Gladine & Anne Le Guillou**

Invited lecture

16:00 – 16:30 **Parveen Yaqoob,** University of Reading, Reading, United Kingdom.
Extracellular vesicles as mediators of the anti-thrombotic effects of n-3 polyunsaturated fatty acids

Oral Communications

16:30 – 16:45 **Catherine Mounier,** Département des sciences biologiques, Université du Québec à Montréal, Montréal, Québec, Canada.
Apolipoprotein D modulates inflammation in a sex-dependent manner.

16:45 – 17:00 **Eva Larrieu**, CarMeN Laboratory, INSERM U1060, INRAE U1397, University of Lyon, Pierre-Bénite, France
Plant-based omega-3 and lipid metabolism in obesity: comparison of plant glycolipids and linseed oil as naturel carriers of ALA

17:00 – 17:15 **Chika Mochizuki-Ono**, Laboratory of Microenvironmental and Metabolic Health Science, Center for Disease Biology and Integrative Medicine, The University of Tokyo, Tokyo, Japan
sPLA2-XIIA drives extracellular vesicle phospholipid metabolism to promote pathogenic Th17 responses.

17:15 – 17:30 **Magdalena Osowiecka,** Institute of Physiological Chemistry, Faculty of Chemistry, University of Vienna, Vienna, Austria.
Dietary Lipid Epoxide as Novel Inducer of Dose-Dependent Intestinal Inflammation

18:00 – 19:00

GERLI General Assembly & ICBL Steering Committee (Agora and Councils room)

20:00

➔ GERLI & ICBL Steering committees' dinner (Le café français, 5 Place Pey-Berland, 33000 Bordeaux, Tram B, get off at the HOTEL DE VILLE stop)

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Wednesday, September 23, 2026

Session 4 Lipids and Brain (Agora)

Chairpersons **Sophie Layé & Nicolas Vitale**

Invited lecture

9:00 – 9:30 **Richard Bazinet,** Department of Nutritional Sciences, University of Toronto, Canada.
New methods lead to new insights on the role of diet and the liver in maintaining a healthy fat brain.

Oral Communications

9:30 – 9:45 **Yushan Cai,** School of Medical Sciences, Faculty of Medicine and Health, The University of Sydney, Camperdown, Australia.
Aging and APOE Genotype Significantly Impact Myelin Lipid Synthesis and Turnover in Mice.

9:45 – 10:00 **Marta Turri,** Department of Medicine, Faculty of Medicine and Health Sciences, Université de Sherbrooke, Sherbrooke, Canada.
Correction of hippocampal fatty acids rescues synapse and energy programs of 5xFAD glutamatergic neurons.

10:00 – 10:15 **Luís Freiría-Martínez,** Chemistry of Marine Products Group, Marine Research Institute - Spanish National Research Council (IIM-CSIC), Vigo, Spain.
The Chemistry of Inflammation Relief: Lipid Mediators Profiling Reveals How Gene Therapy Restores Brain Homeostasis in 5xFAD Alzheimer Mouse Model.

10:15 – 10:30 **David Perrais,** IINS, Université de Bordeaux, CNRS UMR 5297, Bordeaux, France.
Membrane lipid poly-unsaturation selectively affects dopamine D2 receptor endocytosis

10:30 – 11:00

➔ Coffee break (Badiane room, along with the **sponsor booths**), & **Poster viewing, Odd numbers** (Cloister)

Session 5 Biopolymers and Lipids (Agora)

Chairpersons **Frédéric Domergue & Benjamin Buaud**

Invited lecture

11:00 – 11:30 **Rochus B. Franke,** Institute of Cellular and Molecular Botany, University of Bonn, Bonn, Germany.
Long chain and very long chain lipids in the plant's extracellular space: from physiological importance to biopolymers and biotechnological potential.

Oral Communications

11:30 – 11:45 **Antoine Ide,** Louvain Institute of Biomolecular Science and Technology, UCLouvain, Louvain-la-Neuve, Belgium.
Accelerated ageing of vegetable oils rich in conjugated linolenic acids reveals polymerisation processes and the formation of highly toxic aldehydes.

11:45 – 12:00 **Sébastien Baud,** Université Paris-Saclay, INRAE, AgroParisTech, CNRS, Institute Jean-Pierre Bourgin for Plant Sciences, Versailles, France.
Direct transcriptional activation of suberin biosynthesis by MYB9 and MYB107 protects Arabidopsis seeds against abiotic stress from late maturation through germination.

12:00 – 12:15 **Vanessa Esquivel Bertran,** ICGM, University of Montpellier, CNRS, ENSCM, Montpellier, France.
Synthesis of biobased rheology modifiers for cosmetic applications.

12:15 – 12:30 **Boris Bizet,** ITERG, Canéjan, France
Bio-based Estolides from metropolitan Erucic Acid: synthesis and performances

12:30 – 14:00

➔ Lunch Buffet (Badiane room, along with the **sponsor booths**), & **Poster viewing, Odd numbers** (Cloister)

13:00-14:00 **Presentation of Posters to the Jury**

Session 6 Advanced topics and industrial challenges (Agora)

Chairpersons **Erick Dufourc & Hubert Schaller**

Oral Communications

14:00 – 14:15 **Elsevier**
Generative AI in Scientific Writing and Scholarly Publishing.
Sponsored by Elsevier

14:15 – 14:30 **Gerhard Liebisch,** Institute of Clinical Chemistry and Laboratory Medicine, University Hospital Regensburg, Germany
The ILS approach to lipidomics data quality — from the Lipidomics Reporting Checklist to lipidome concentration databases
Sponsored by ILS

14:30 – 14:45 **(Mendel) Chen Chu-Huang,** Vascular and Medicinal Research, The Texas Heart Institute at Baylor College of Medicine, Houston, Texas, USA.
L5 and Lp(a): Emerging Biomarkers for ASCVD Risk and Prevention
Sponsored by HEART

14:45 – 15:00 **Magdalene Reinkensmeier,** Bruker Daltonics GmbH & Co. KG, Fahrenheitstrasse 4, 28359 Bremen, Germany
Profiling cell-type specific mitochondrial sub-populations in Arabidopsis thaliana with multi-omics mass spectrometry
Sponsored by Bruker

15:00 – 15:15 **Barbara Perrone,** Bruker Switzerland, Fällanden, Switzerland.
A Multi-Phase NMR View of Lipids in Their Native Context
Sponsored by Bruker

15:15 – 15:30 **Anna Grygier,** Faculty of Food Science and Technology, University of Life Sciences, Poznań, Poland.
Mustard oil and press cake in the production of vegan mayonnaises
Sponsored by Chairmen

➔ Social Events: Visit of chateau Luchey-Halde cellar, wine-tasting, gala dinner, etc.

16:00 –16:30 Departure by Bus N°1 to Château Luchey Halde “Pessac Léognan”
16:30 –17:00 Departure by Bus N°2 to Château Luchey Halde “Pessac Léognan”
17:00 –17:30 Departure by Bus N°3 to Château Luchey Halde “Pessac Léognan”

16:30 – 18:00	30-40 minutes visit of the vineyards and cellars, explanation of wine making, visit of the “wine boutique”. 3 successive groups of 50 people.
17:00 – 19:00	3 x Public conferences by **Erick Dufourc**: *Wine tannins and lipids: a love story, including a wine and cheese tasting* 3 successive groups of 50 people.
19:00 – 19:30	Aperitif in the vineyard
19:30 – 22:00	Conference Dinner, **Poster Awards and PhD Prizes Ceremony**
22:00	Bus departures to Bordeaux downtown.

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Thursday, September 24, 2026

Session 7 – Biodiversity of lipids

Chairpersons **Hubert Schaller & Jérôme Nigou**

Invited lecture

9:00 – 9:30 **Gwendolyn Barceló-Coblijn,** Health Research Institute of the Balearic Islands (IdISBa), Palma, Balearic Islands, Spain.
Defining Ulcerative Colitis through lipids: multicompartment lipid signatures discriminate microbiome-linked phenotypes.

Oral Communications

9:30 – 9:45 **Shao-Chi Hung**
Department of Medical Laboratory Science and Biotechnology, College of Health Sciences, Kaohsiung Medical University, Taiwan.
ASAH2 is a dual-function lipid hydrolase with ceramidase and lysophospholipase A1 activities.

9:45 – 10:00 **Sarah Brunner,** Institut of Clinical Chemistry and Laboratory Medicine, Functional Lipidomics and Metabolism Research, University Hospital Regensburg, Regensburg, Germany.
Gut microbiota restricts intestinal lipid uptake via modulation of bile phosphatidylcholine metabolism in mice.

10:00 – 10:15 **Sarah Vellemans,** Louvain Institute of Biomolecular Science and Technology (LIBST), UCLouvain, Louvain-la-Neuve, Belgium.
Bioavailability of punicic acid from pomegranate seed oil: in vitro and in vivo evidence.

10:15 – 10:30 **Kyndall Nicholas**, Faculté de médecine et des sciences de la santé, Université de Sherbrooke, Sherbrooke, QC, Canada.
Fish Oil and Krill Oil do not Increase the Omega 3 Index Equally.

10:30 – 11:00

➔ Coffee break (Badiane room, along with the sponsor booths)

Session 8 Lipids for Medicine & Pharmacy (Agora)

Chairpersons **Frédéric Carrière & Ofelia Maniti**

Invited lecture

11:00 – 11:30 **Per Larsson,** Department of Pharmacy, Uppsala University, Uppsala, Sweden.
Self-assembly of medium-chain fatty acids and mixed lipid colloids in the gut: solubilization and membrane permeation enhancement.

Oral Communications

11:30 – 11:45 **Angelina Angelova,** Institut Galien Paris-Saclay CNRS UMR8612, Université Paris-Saclay, 91400 Orsay, France.
Lipid Self-Assembly as a Platform for Next Generation Nanomedicine in Neurodegenerative Disorders.

11:45 – 12:00 **Paola A. Corsetto,** Department of Pharmacological and Biomolecular Sciences "Rodolfo Paoletti", Università di Milano, Milan, Italy.
Circulating lipid and metabolic biomarkers for Prediction of Immunotherapy Response in advanced NSCLC patients.

12:00 – 12:15 **Michel Record,** INOV Team "Cholesterol Metabolism and Therapeutic Innovations" Cancer Research Center of Toulouse, CRCT, UMR 1037, Toulouse, France.
Dendrogenin A, a Cholesterol-Derived Metabolite, Reprograms Endolysosomal BMP Metabolism to Generate Immunogenic Antitumour Extracellular Vesicles.

12:15 – 12:30 **Gábor J. Tigyi,** Department of Physiology, University of Tennessee Health Science Center, Memphis, Tennessee, USA.
Loss of the lysophosphatidic acid-induced immediate early gene IEX-1 exacerbates acute radiation syndrome.

12:30 – 13:30

➔ Lunch Buffet (Badiane room, along with the sponsor booths),

Session 9 Lipids, Analysis and Technology (Agora)

Chairpersons **Fernando Leal-Calderon & Makoto Arita**

Invited lecture

13:30 – 14:00 **Claire Berton-Carabin,** INRAE, UR BIA, 44000 Nantes, France.
Lipids in disguise: Hidden co-passengers in plant protein ingredients with potential impacts on plant-based foods.

Oral Communications

14:00 – 14:15 **Marisa Pinho,** CESAM-Centre for Environmental and Marine Studies, Department of Chemistry, University of Aveiro, Campus Universitário de Santiago, Aveiro, Portugal;
Drying-induced lipidome remodeling in the two edible halophytes Salicornia ramosissima and Atriplex portulacoides.

14:15 – 14:30 **Arash Zarrine-Afsar,** University Health Network and the University of Toronto, Toronto, Canada.
A Decision-support Scheme for Clinical Implementation of Lipidomic Classifiers to Guide Cancer Care.

14:30 – 14:45 **Gerhard Hagn,** Institute of Analytical Chemistry, Faculty of Chemistry, University of Vienna, Vienna, Austria.
Integrating tissue proteomics and bioactive lipid analysis of ovine tissue samples identifies functional relations of bioactive lipids.

14:45 – 15:00 **Delphine Vergoz,** Univ Rouen Normandie, INSA Rouen Normandie, CNRS, Normandie Univ, CARMeN, UMR 6064, INC3M 76000 Rouen, France.
Multidimensional Lipidomics Profiling of Acinetobacter baumannii Persister Cells using LC-IMS-MS.

15:00 – 15:30

➔ Coffee break (Badiane room)

Oral communications Awards (Agora)

Chairpersons **Erick Dufourc & Hubert Schaller**

15:30 – 15:45

Next Meetings

15:45 – 15:55 **Anthony Don**, Australia, Presentation of **ICBL 2027**

15:55 – 16:05	**Marie-Caroline Michalski, Cécile Gladine,** Presentation of **GERLI 2027**
16:05	**Closing**

ABSTRACTS

Invited Lectures and Oral Communications

Workshop on Imaging techniques for lipids and membranes (Agora)

Invited Lecture

Identifying the lipid-protein interactions that build organelle membranes

Katelyn Camille Cook[1], Palacio-Rodriguez, Karen[2], Böhlig, Kristin[1], Lennartz, H. Mathilda[1], Lenz, Swantje[1], Honigmann, Alf[3], Shevchenko, Andrej[1], Hummer, Gerhard[2], von Appen, Alexander[1], and Nadler, André[1]

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Organelle membrane identity is encoded by protein and lipid composition. Human cells produce thousands of distinct lipid species for this purpose. Yet, the precise molecular functions of most lipids remain unknown, owing to limited methods for *in situ* investigations of individual lipid species. Here, we integrate lipid imaging with quantitative proteomics to create a framework for mechanistic lipid cell biology. We map time-resolved protein interactomes of individual lipids representing major membrane lipid classes as they are transported through the organelle system of human cells. We identify hundreds of unique lipid-protein interaction candidates, measure lipid-induced protein abundance changes, and distinguish between directly and indirectly interacting subunits of associated protein complexes. Informed by this multimodal dataset, we interrogate the lipid regulation of membrane organizing complexes, specifically the selective co-association of phosphatidylethanolamine with the ancestral transmembrane subunits of the nuclear pore (NDC1) and MICOS (MIC60) complexes. Using super-resolution microscopy and molecular dynamics simulations, we demonstrate that PE stabilizes both NDC1 and MIC60 in highly curved membrane nanodomains, indicating that major membrane organizing complexes are positioned by selective lipid-protein interactions. Altogether, our experimental strategy provides a blueprint for discovering and characterizing cellular functions of individual lipids.

Oral Communication

Talking Through Walls: Lipid-Protein Interactions Controlling Plasmodesmal Communication

Jessica Pérez-Sancho[1], Marija Smokvarska[1], Gwennogan Dubois[2] Yvon Jaillais[2] **Emmanuelle Bayer**[1],

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Inside a cell, organelles often form close contacts with one another. At these membrane contact sites (MCSs), membrane compartments exchange lipids and signals and coordinate their activity. We asked whether similar contacts could also help neighbouring cells communicate with each other. In plants, cells are connected by tiny channels called plasmodesmata, which let molecules pass from one cell to the next. Hundreds of these structures connect plant cells together, throughout the entire plant body. We show that these channels are built around an unusual membrane contact site: a direct, tube-shaped connection between the endoplasmic reticulum (ER) and the plasma membrane (PM). As in other MCSs, this connection is held in place by a protein-lipid tethering complex but in this case the complex serves communication between cells rather than between organelles. Using high-resolution microscopy, we directly visualised these nanometre-scale membrane interfaces to see how they are organised. Combining this imaging with molecular modelling and pharmacological and genetic approaches, we found that intercellular trafficking is controlled by an interplay between membrane-tethering proteins and anionic lipids at the ER-PM interface. The amount of these lipids present determines how well the tethering proteins bind the PM, whether they build up at plasmodesmata, and ultimately how open or closed the channel becomes.

Oral Communication

Multimodal Mass Spectrometry Imaging for the Spatial Characterization of Lipids and Cellular Microenvironments.

Desbenoit, Nicolas[1]

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Matrix-assisted laser desorption/ionization mass spectrometry imaging (MALDI-MSI) has emerged as one of the most attractive ex vivo techniques for the spatial characterization of molecules. MALDI-MSI combines high chemical specificity with label-free molecular analysis and has become a powerful tool for molecular histology. In particular, it provides unique opportunities to investigate the spatial distribution and heterogeneity of lipids within tissues and complex biological microenvironments. More broadly, MALDI-MSI has been applied to a wide range of research fields and molecular classes, from pharmacokinetics and tumor detection to the spatial analysis of lipids, proteins, metabolites, and drugs, including antibiotics. With the increasing spatial resolution enabled by recent advances in MALDI ion sources, cellular-scale imaging is now becoming possible, opening new opportunities to investigate molecular heterogeneity and cellular interactions within complex microenvironments. However, MSI alone does not yet allow the reliable identification of the different cell types present within a tissue.

To overcome these limitations, we have developed a multimodal approach combining MALDI-MSI with immunofluorescence (IF) imaging. This strategy enables the integration of molecular information, including lipid distributions, with cellular phenotyping and tissue organization. The analytical strategies developed will be illustrated through several practical examples.

Beyond the analytical challenges, it is essential to develop robust computational strategies to extract, integrate, and correlate imaging data acquired using different modalities. Such multimodal approaches may ultimately provide a deeper understanding of the complex spatial organization and molecular interactions occurring within biological microenvironments.

Oral Communication

Quantification and Localisation of New Brain Lipid Synthesis Using Deuterium Oxide and High Resolution Mass Spectrometry

Don, Anthony[1]; Zhang, Catherine[1]; Michael, Jesse[2]; Teo, Jonathan[1]; Song, Huitong[1]; Westerhausen, Mika[2]; Mutuku, Shadrack[2]; Ellis, Shane[2]

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Myelin is the lipid-rich membrane that surrounds neuronal axons and is essential for neurological function in vertebrates. The development of therapeutics that stimulate myelin repair to treat demyelinating disorders such as multiple sclerosis is hampered by the inability to distinguish newly-synthesised from pre-existing myelin. We therefore developed a method to quantify and localise new myelin lipid synthesis in the mouse brain. Deuterium oxide was administered for two weeks in the drinking water of mice fed normal chow, chow containing the demyelinating toxin cuprizone, or during spontaneous remyelination following cuprizone withdrawal. Liquid chromatography-tandem mass spectrometry and matrix-assisted laser desorption/ionisation mass spectrometry imaging (MSI) were used to quantify and localise the newly synthesised, deuterated lipids in the brain. While most glycerophospholipids were constitutively deuterated, deuteration of myelin-enriched sulfatides, hexosylceramides, and phosphatidylethanolamine plasmalogens was only apparent during remyelination. Most deuterium atoms were found in the fatty acyl chains, indicative of de novo lipid synthesis. MSI showed selective localisation of deuterated hexosylceramides and phosphatidylethanolamine plasmalogen species in the corpus callosum, the white matter tract that is most heavily demyelinated in response to cuprizone. Additionally, MSI captured rapid myelin lipid synthesis in subregions of the corpus callosum where remyelination was not apparent by histological staining. In summary, the method described herein provides the means to quantify and spatially profile dynamic lipid synthesis across diverse biological contexts, including understanding myelin homeostasis and preclinical evaluation of remyelinating therapeutics.

Oral Communication

Characterization of non-enzymatic oxidized polyunsaturated fatty acids (NEO-PUFAs) interactomes and the associated signaling pathways by the use of click-NEO-PUFAs

Jamie Bride[1], Anna Abramova[2], Marie Saurat[1], Valérie Bultel[2], Thierry Durand[2], Jean-Louis Baneres[2], Jean-Yves Le Guennec[1], Jean-Marie Galano[2], Marie Demion[1]

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Introduction: 4(RS)-4-F_{4t}-NeuroProstane (4-F_{4t}-NeuroP), a DHA non-enzymatic oxidation product, has emerged for its beneficial biological effects. Our interdisciplinary team has shown its anti-arrhythmic effect (*Roy et al.,2015)*, along with potential anti-apoptotic activity (*Roy et al.,2017*). Additionally, 4-F_{4t}-NeuroP has demonstrated anti-inflammatory properties in macrophages (*Bosviel et al.,2017*). Despite these effects, the exact molecular mechanisms remain unclear. The aim of the study is to identify the potential targets of 4-F_{4t}-NeuroP using click chemistry.
Methods: 4-F_{4t}-NeuroP was functionalized with a bio-orthogonal chemical group (alkyne) to generate a clickable probe named 4-F_{4t}-NeuroP-alkyne (NP0). To evaluate the impact of this modification on biological activities, we assessed the number of arrhythmias on isolated mice cardiomyocytes in presence or not of NP0. Then, using FACS analysis, we measured the pro-inflammatory markers (CD86, NOS2) and anti-inflammatory marker (CD206) expression in LPS-stimulated RAW 264.7 macrophages (LPS 100ng/mL) pretreated with 30µM of NP0. Finally, using confocal microscopy, we visualized the cellular localization of NP0 after performing the click reaction with a complementary clickable fluorophore, the AZDye647A-Picolyl-Azide (10µM) in both cell types.
Results: The anti-arrhythmic properties of NP0 were validated on isolated ventricular mice cardiomyocytes treated with 30µM NP0. These results are consistent with the confocal microscopy images which revealed that NP0 accumulates in nucleus, intercalated disc and T-tubules. The anti-inflammatory properties were not validated on RAW 264.7 macrophages in response to LPS. Despite this, confocal microscopy images revealed that NP0 accumulates in macrophages without a precise localization.
Conclusion: The preliminary data showed that the NP0 displays partial biological properties of the native 4-F_{4t}-NeuroP and accumulates in a different way depending on the cell type.

Oral Communication

A coordinated peroxisome–DHA–plasmalogen programme marks secretory and metastatic epithelial states in human colon cancer

Ximelis,Teresa[1,2], Rodriguez, Ramon M.1[,2,*], Pérez-Romero, Karim[1,2], Maimó-Barceló, Albert[1,2], Huergo-Baños, Cristina[3], A. Martínez, Marcos[4], Aransay, Ana M.[5,6],Fernández, José A.[3], Barceló-Coblijn, Gwendolyn[1,2,7,*]

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Spatially organised intratumoral heterogeneity characterises tumour progression, but the molecular basis of this organisation is still poorly understood. Although spatial transcriptomics has provided valuable insights into cellular states of cancer, the spatial organisation of tumour lipid metabolism is little known.

We combined high-resolution mass spectrometry imaging with spatial transcriptomics on consecutive sections of primary and intraperitoneal metastatic human colon cancer and validated our findings in an independent cohort of 15 primary tumours.

Using this strategy, we identified four distinct epithelial lipid phenotypes (EpLP1–EpLP4), corresponding to absorptive-, transient-amplifying-, stem- and secretory-like states), each characterised by a specific phospholipid membrane composition. We then focused on the secretory-like phenotype (EpLP4), enriched in metastatic lesions and set apart by its lipid programme. EpLP4 accumulated docosahexaenoic acid (DHA)-containing phospholipids, particularly plasmalogens such as 38:6 and 40:6. Accordingly, EpLP4 showed the strongest upregulation of peroxisomal enzymes spanning ether-lipid synthesis and β-oxidation (AMACR, GNPAT, ACAA1, ACSL1), peroxisome biogenesis (PEX16, PEX19), and ROS detoxification (CAT), pointing to a coordinated peroxisome–DHA–plasmalogen programme.

Thus, we conclude that membrane phospholipid composition correlates with distinct epithelial states within the human colon epithelium, and that a peroxisome-driven DHA–plasmalogen programme marks secretory and metastatic states. These results provide the basis for functional organoid studies addressing the role of peroxisome-derived lipids in tumour progression.

Oral Communication

Impact of the Rift Valley fever virus NSm protein on lipid droplet metabolism

Vatin Raphaël[1], Tamietti Carole[1], Imler Noé[1], Romanet Charlotte[1], Nassim Mathal[2] Rey Félix[1] and Flamand Marie[1]

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Lipid droplets (LD) are used by multiple viruses such as SARS-CoV-2, Hepatitis C, Dengue, Zika or Chikungunya as platforms to promote viral replication or to inhibit the cellular anti-viral responses. Rift Valley fever virus (RVFV) is a negative strand RNA virus that expresses two virulence factors, NSs and NSm. The RVFV NSm protein is a 12 kDa membrane protein that colonizes the mitochondrial surface. Proteomic and phosphoproteomic analyses of cells infected with wild type (wt) RVFV or a mutant defective in NSm expression (def-NSm) revealed that NSm has a strong impact on LD metabolism.

To study the cellular processes controlled by NSm, we used a murine hepatocyte cell line (AML12) that displays high LD number and size diversity, and is naturally susceptible to RVFV infection. We quantified the number of contact points at 8 hours post-infection (hpi) between mitochondria and LDs in AML12 cells infected with wt or def-NSm viruses using a proximity ligation assay and high-throughput fluorescence imaging acquisition (Zeiss CellDiscoverer 7). We recorded the evolution of LD populations within these cells over time, integrating their size and number by diverse image processing methods (Cellpose, Fiji). We observed that NSm triggers the formation of contact sites between mitochondria and LD, leading to a decrease in LD populations, likely due to fatty acid beta-oxidation, at 12 hpi onwards. We are currently investigating multiple cell lines, including bovine, human and simian cell lines, to analyse the versatile role of NSm during RVFV infections.

Our future research aims at identifying the cellular proteins interacting with NSm to further understand the spatial and temporal reprogramming of LD metabolic networks by the viral protein. Inhibiting NSm function in infected cells is expected to decrease viral virulence, paving the way for a novel therapeutic approach against RVFV.

Opening lectures

GERLI Polonovski Opening Lecture

Extracellular vesicles: complex lipid-enclosed structures with roles in tumor-immune system cross-talk

Clotilde Thery, INSERM U932/Institut Curie, Paris, France

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Extracellular Vesicles (EVs) are small lipid bilayer-enclosed particles released by all cells in their environment. EVs contain multiple components from the producing cells, especially proteins, lipids, and nucleic acids, and expose additional proteins bound to their surface. EVs are now recognized as central players in intercellular communication processes, since they carry multiple signals at once from one producing cell to its local neighbours, or at further distance. Structurally, EVs display the same membrane orientation as cells, thus exposing at their surface the extracellular domains of membrane-inserted proteins, and protecting various cytosolic components in their internal lumen. EVs can signal to surrounding cells via their surface proteins, when they are recognized by a specific ligand on a target cell, or by delivering their content inside the target cell. EVs are extensively explored as potential tools for therapeutic approaches, in various diseases. I will present examples of our current projects aiming at analysing EV lipid composition, and their functions and immunotherapeutic potential in cancer.

ICBL Van Deenen Opening Lecture

Mechanistic Insights into Phosphatidic Acid Phosphatase in Lipid Synthesis Regulation

George M. Carman, Rutgers Center for Lipid Research, Rutgers University, New Brunswick, New Jersey 08550, USA

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Phosphatidic acid phosphatase (PAP) is a conserved eukaryotic enzyme that plays a key role in lipid homeostasis and cell physiology by catalyzing the Mg2+-dependent dephosphorylation of phosphatidic acid to produce diacylglycerol. Mutational, biochemical, and cellular studies have revealed structural features essential to its function, as well as mechanisms regulating its activity through phosphorylation and dephosphorylation. Because PAP is critical for the growth and virulence of pathogenic fungi, it has emerged as a promising therapeutic target for combating fungal infections.

Session 1

Membrane Lipids

Invited Lecture

Chemical control of cell adaptation

Raphaël Rodriguez, Institut Curie, CNRS, INSERM, PSL Research University, Paris, France.

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Cells can adopt distinct states independently of genetic alterations, a biological process commonly referred to as 'cell-state transition'. Acquisition of distinct cell states is characterized by the upregulation of the plasma membrane glycoprotein CD44 in development, immunity and cancer. Although often described as a cell-surface marker, the molecular function of CD44 has remained elusive for half-a-century. We found that CD44 mediates the uptake of specific metals, including copper and iron using hyaluronate carriers in various tissue types. This glycan-mediated metal endocytosis mechanism enables immune cell activation and acquisition of a drug-tolerant state of cancer cells (cell adaptation). Increase of copper(II) in mitochondria sustains NAD(H) redox cycling, promoting the production of metabolites that co-regulate the epigenetic programming of cell identity. In contrast, increase of iron in the cell nucleus fuels the activity of specific iron- and ketoglutarate-dependent demethylases, enabling specific transcriptional programs. We developed new classes of small molecules that selectively interfere with these metal-catalyzed chemical processes in cells. Inactivating mitochondrial copper(II) prevents acute inflammation in vivo demonstrating that control of cell-state transition confers therapeutic benefits. Pharmacological activation of lysosomal iron, on the other hand, induces ferroptosis in drug-tolerant persister cancer cells, impacting tumor progression. These findings illuminate a universal metal ion uptake mechanism and the critical role of metal ions as regulators of cell adaptation, paving the way towards the development of next generation therapeutics.

Selected publications

- Activation of lysosomal iron triggers ferroptosis in cancer. T. Cañeque,…, R. Rodriguez*. ***Nature*** 642, 492-500 (2025).
- Ageing of stem cells reduces their capacity to form tumours. T. Cañeque, R. Rodriguez* ***Nature*** 637, 36-37 (2025)
- Multiple estradiol functions inhibit ferroptosis and acute kidney injury. W. Tonnus,…, R. Rodriguez,..., A. Linkermann*. ***Nature*** (10.1038/s41586-025-09389-x)
- Lymph node environment drives FSP1 targetability in metastasizing melanoma. M. Palma,…, R. Rodriguez,..., J. M. Ubellacker*. ***Nature*** (Accepted for publications)
- A druggable copper-signalling pathway that drives inflammation. S. Solier,…, R. Rodriguez*. ***Nature*** 617, 386-394 (2023).
- Copper catalyses changes in cell state. S. Müller, R. Rodriguez*. ***Nature*** (10.1038/d41586-023-01330-4)
- Chemical inhibition of NAT10 corrects defects of laminopathic cells. D. Larrieu,…, R. Rodriguez*, S. P. Jackson*. ***Science*** 344, 527-532 (2014)

Oral Communication

Ether lipid deficiency alters bone architecture in mice and impairs osteoclast differentiation *in vitro*

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Ether lipids are an emerging class of lipids critical for tissue integrity in brain, eye, and bone. They exist as plasmanyl and plasmenyl (plasmalogen) species, the latter requiring plasmanylethanolamine desaturase (PEDS1) for vinyl ether bond formation. Human defects in early ether lipid synthesis cause rhizomelic chondrodysplasia punctata (RCDP) with severe skeletal abnormalities, including shortening of the proximal long bones and calcifications in cartilage. The recently described human *PEDS1* deficiency presents with a milder skeletal phenotype. How plasmanyl and plasmalogen species differentially support bone homeostasis remains unclear.

Using mouse lines on a common genetic background, we compared *Gnpat* knockout mice, which lack all ether lipids, with *Peds1* knockout mice, which lack plasmalogens but retain plasmanyl lipids. We analysed bone density and length, as well as serum bone markers; profiled bone marrow proteomes and differentiated bone marrow-derived mesenchymal and hematopoietic precursors into osteoblasts and osteoclasts, respectively, and performed RNA-seq.

Ether lipid loss produced graded skeletal phenotypes. *Gnpat* knockout mice showed altered bone architecture and shortening of proximal bones with elevated serum markers indicating remodeling imbalance. *Peds1* knockout mice were largely normal, with only mild changes in bone density parameters. Bone marrow proteomics revealed genotype-specific remodeling of the hematopoietic niche. *In vitro*, osteoblast differentiation was preserved in both knockouts, whereas osteoclast maturation was markedly reduced, most prominently in *Gnpat* knockout. Lipid supplementation experiments significantly improved osteoclast multinucleation and size across genotypes, suggesting that membrane-active lipid availability supports maturation. Transcriptomics indicated that core osteoclast programs remain largely intact, with transcriptional defects concentrated in late maturation genes.

Total ether lipid deficiency disrupts bone architecture and osteoclast maturation, whereas selective plasmalogen loss is largely compensated by retained plasmanyl lipids. These findings align with the milder human *PEDS1* deficiency and highlight subclass-specific requirements of ether lipids for bone homeostasis.

Oral Communication

Lipid droplet composition and oxidative state determine their role in ferroptosis

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Lipid droplets (LDs) are increasingly recognized as regulators of ferroptosis, an iron-dependent cell death driven by lipid peroxidation. Although LDs are often viewed as protective organelles that sequester oxidation-prone fatty acids (FAs) away from membranes, accumulating evidence, including ours, indicates that they can also promote ferroptotic vulnerability. The basis for this functional duality remains unclear. Here, using cancer cell models with distinct lipid metabolic profiles, we show that the role of LDs is determined by two properties of the LD compartment: the availability of oxidizable polyunsaturated fatty acids (PUFAs) and the oxidative state of stored lipids. In cells with a PUFA-rich lipid environment and low oxidation, LDs function as protective reservoirs by buffering peroxidation-prone FAs through diacylglycerol acyltransferase (DGAT)-dependent neutral lipid storage. This protective LD function is reduced in cells with limited PUFA availability, demonstrating that LD abundance alone is insufficient to confer protection. Conversely, in cells with elevated basal LD-associated lipid oxidation, we find that balance between the LD redox state and PUFA load can shift the functional outcome of LD accumulation, revealing that oxidized LDs can contribute to, rather than suppress, ferroptotic damage. By combining genetic and pharmacological perturbation of LD metabolism with untargeted lipidomics, we find that the ferroptosis-modulating capacity of LDs depends on dynamic FA partitioning between neutral lipid stores and oxidation-prone membranes. Together, our findings establish that LDs are not intrinsically protective or detrimental organelles but context-dependent regulators of ferroptosis whose function is dictated by lipid availability, oxidative status and the metabolic organization of the cell. This explains the context-dependent roles of LDs and highlights PUFA availability and LD oxidative state as key determinants of ferroptosis susceptibility.

Oral Communication

Membrane Remodeling in Lysosomal Storage Disorders: The Role of GlcCer and Gb3.

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Beyond being metabolic hallmarks of Gaucher and Fabry diseases, glucosylceramide (GlcCer) and globotriaosylceramide (Gb3) are key structural players in cell membranes. While their genetic and biological origins are well-documented, the biophysical mechanisms of how they alter membrane organization and cellular nanomechanics remain poorly understood.
Here, we assess how GlcCer and Gb3 act as physical modulators of membrane architecture. Lipidomic profiling of patient-derived fibroblasts revealed distinct pathological lipidome remodelling under metabolic stimulation. To isolate the physical consequences of these glycolipids on bilayer organization, we reconstructed their environments in biomimetic supported lipid bilayers (SLBs) using physiologically relevant mixtures of DOPC, sphingomyelin, and cholesterol.
Using atomic force microscopy (AFM) and AFM-based force spectroscopy (AFM-FS), we probed bilayer structural and mechanical alterations. By measuring breakthrough force (F_b) and Young's modulus—both indicators of lipid packing—we discovered that these species dictate the membrane's physical fate. GlcCer drastically reinforces domain rigidity. Gb3 also enhances membrane stiffness, and may trigger phase inversion in membrane topology, acting as a major structural disruptor. To translate these nanoscale cues, we are extending these findings to living patient-derived fibroblasts, exploring whether these bilayer alterations correlate with pathology-specific signatures in elasticity and membrane tension.
Ultimately, this study demonstrates that pathological glycolipid accumulation directly rewrites the membrane's physical state. By defining these distinct nanomechanical fingerprints, our work offers a compelling biophysical perspective on how specific lipids govern cell mechanics, shedding light on the transition from molecular accumulation to disease pathogenesis.

Oral Communication

Phospholipid-cation interactions alter membrane proteins activity: The Case of SNARE-Catalyzed Membrane Fusion

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Biological membranes are complex assemblies of lipids and proteins whose composition and dynamics govern essential cellular processes. It is now well established that membrane protein activity depends strongly on the surrounding lipid environment. This coupling arises from both specific lipid–protein interactions and global physico-chemical properties such as elasticity, curvature, fluidity, and surface charge. In the cell, soluble chemical species can interact strongly with lipid headgroups. Among these, monoatomic cations exhibit a diverse range of interactions, engaging lipids through coordination, electrostatics, or hydrogen bonding mediated by their hydration shells. Here, we investigate how s-block cations interacting with lipids at ångström to nanometer scales indirectly regulate membrane protein activity by altering membrane properties. To isolate lipid–cation effects from direct protein–cation interactions, we employ an in vitro microfluidic platform that enables the formation of suspended asymmetric bilayers with leaflet-specific protein functionalization. Each leaflet is exposed to a distinct solution of controlled composition, while protein orientation remains fixed. This configuration maintains a constant chemical environment for the protein while selectively tuning lipid–cation interactions in the opposite leaflet. Using SNARE proteins as a model system, we monitor single fusion events to quantify how lipid–cation interactions influence membrane fusion. In membranes mimicking plasma membrane composition, coordinating cations such as Li^+ and Ca^{2+} significantly reduce fusion efficiency, whereas weakly coordinating ions as K^+ and Mg^{2+} show minimal effects. Inhibition of Ca^{2+} coordination with cryptands restores fusion activity, underscoring the importance of coordination chemistry. We also observe Li^+ reduces SNARE-mediated fusion by approximately 50%, suggesting a physico-chemical basis for its biological effects as a bipolar disorder therapeutic agent. Complementary studies reveal that lipid–cation interactions increase membrane rigidity and raise the activation energy barrier for fusion. This work thus demonstrates lipid-cation interactions to be a new control layer of membrane processes.

Session

PhD Awards

GERLI PhD Award

Local Organization of Biological Membranes Modulates Tau−Lipid Interactions and Fibril formation

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Tau is a protein found in the brain where it regulates microtubule activity. It is an intrinsically disordered protein, which means that it does not have a unique three-dimensional structure. Tau can form highly ordered aggregates that are directly involved in several neurodegenerative diseases, called tauopathies, including Alzheimer's disease. Recent research has shown that Tau protein can interact directly with the neuronal membrane, mainly composed of lipids. Observations of Alzheimer's brain tissues show the association of Tau fibers with membrane lipids such as phosphatidylcholine, cholesterol, and sphingolipid. Yet, how membranes modulate tau aggregation pathways remains poorly understood. We present a biochemical and biophysical characterization of tau amyloid filament formed in presence of different membrane models. We used Electron Paramagnetic Resonance (EPR) to directly quantify the population of tau bound to the membrane and to quantify the affinity between Tau and lipids. After showing the importance of the electrostatic interaction between Tau and anionic lipids, we investigate how the amount and density of charges influence Tau aggregation. This work allows us to draw a general model where membrane-induced Tau aggregation is a two-step process. First, the binding to the membrane through electrostatic interactions is a necessary but not a sufficient step. Second, the nucleation of Tau amyloids at the membrane surface occurs only when specific conditions are fulfilled, namely, high surface density altering Tau conformation and spatial proximity between aggregation-prone conformers. A direct implication of this model is that local membrane heterogeneities, such as phase separation or lipid rafting, are strong modulators of Tau aggregation. This work provides the molecular basis to predict how different membrane states regulate Tau aggregation.

GERLI PhD Award

Revisiting the early secretory pathway in plant cells: identification of a dynamic tubulo-vesiculated ER-Golgi intermediate compartment (ERGIC) that matures in Golgi.

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In the conventional secretion model, cargoes synthesized in the Endoplasmic Reticulum (ER) are transported to the Golgi apparatus, which in animal cells forms a single ribbon-like structure supplied by the ER-Golgi Intermediate Compartment (ERGIC). In contrast, plant cells contain hundreds of mobile mini-Golgi stacks closely associated with the ER, and the existence of an ERGIC has long been questioned.
Our work identifies a dynamic tubulo-vesicular network at the ER-Golgi interface that behaves as a plant-ERGIC. This compartment transiently interacts with the ER, stabilizes near pre-existing Golgi stacks, and contributes to Golgi biogenesis by forming a pre-cisterna that later integrates into the Golgi.
Importantly, we demonstrate that sphingolipid ceramides are essential for both ERGIC-network integrity and Golgi formation. A delicate balance of ceramide-dependent remodeling appears to control the stabilization of the ERGIC network from dynamic independent to a stable pre-Golgi cisterna.
These findings completely change our vision of the Golgi organization in plant cells and reveal the existence of ERGIC in this kingdom of life, a question that has been hotly debated until now. Furthermore, these results also redefine the early-secretory pathway in plant cells and uncover a previously unrecognized lipid-dependent mechanism controlling Golgi biogenesis.

GERLI-SFN PhD Award

Biomarkers of early exposure to polyunsaturated fatty acids and their links with child health and development

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Polyunsaturated fatty acids (PUFAs), nutrients derived from diet or endogenously synthesized, play a major role in brain development and cardiometabolic health. Epidemiological studies assessed PUFA exposure during the perinatal period using biomarkers measured in maternal blood, cord blood, and breast milk, yet findings regarding their associations with child neurodevelopment and later cardiometabolic health remain inconsistent. Several reasons could contribute to this heterogeneity. Firstly, these biofluids, which reflect distinct but interrelated exposure windows, have been examined separately, thereby limiting the ability to disentangle their respective contributions. Secondly, the residual confounding from environmental or social factors, which is inherent to observational studies design. In the French mother–child cohort EDEN, we applied an integrative approach combining PUFA levels from the three biofluids to identify perinatal exposure patterns. We examined the associations between these perinatal patterns with child neurodevelopment and cardiometabolic health at age 5–6 years, using both conventional statistical methods and Mendelian randomization. A pre- and postnatal pattern, characterized by higher levels of omega-3 long-chain (LC-PUFAs) and lower levels of omega-6 LC-PUFAs across the three biofluids, was associated with higher verbal and total intelligence quotient (IQ). Conversely, a pre- and postnatal pattern with higher levels of linoleic acid (LA) and its direct metabolite dihomo-gamma-linolenic acid (DGLA) across the three biofluids was associated with lower performance IQ (visuospatial skills and logical reasoning). A pattern specifically related to the lactation period showed an independent positive effect of both omega-3 and omega-6 LC-PUFAs in the breast milk on performance IQ. By contrast, few associations were observed with cardiometabolic health: only the LA- and DGLA-rich pattern was linked to a modest reduction in insulin sensitivity in girls. These findings suggest a long-term impact of perinatal PUFA exposure on child cognitive development and, to a much lesser extent, on cardiometabolic health, and support the importance of adhering to nutritional recommendations during this critical developmental period.

Session 2

Algae and Plant Lipids

Invited Lecture

Evolutionary repurposing of MGDG synthases from photosynthetic membrane biogenesis to new eukaryotic functions

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This presentation aims to present, in a clear and accessible way, a complex evolutionary history for a non-specialist audience.
All living cells rely on membranes whose lipid composition determines their function. In photosynthetic membranes, one lipid carrying a galactose polar head group—monogalactosyldiacylglycerol (MGDG)—is particularly abundant and essential. Because this lipid is already present in cyanobacteria, the ancestors of chloroplasts, one might expect its biosynthetic machinery to have been vertically inherited. Yet its evolutionary history is far less straightforward.
A first major transition occurred when PRIMARY ENDOSYMBIOSIS gave rise to the chloroplast, following the engulfment of an ancestral cyanobacterium by a eukaryotic host. In this new cellular context, the enzymes producing chloroplastic MGDG (MGDG synthases, or MGDs) were not inherited from the cyanobacterial machinery. The lipid was conserved, but the enzymes were evolutionary innovations recruited from a distinct origin. A similar replacement occurred for the synthesis of digalactosyldiacylglycerol (DGDG). Among photosynthetic eukaryotes derived from this event, further diversification followed. In angiosperms, MGD genes duplicated and specialized, with one major isoform maintaining a central role in chloroplast membrane biogenesis, while others contribute to adaptive responses.
A second layer of complexity emerged with SECONDARY ENDOSYMBIOSIS, when a photosynthetic eukaryote was engulfed by another host. Diatoms provide a key example: although distant from plants, they independently diversified their MGD repertoire, with isoforms linked to distinct localizations and functions. Such diversification occurring independently in these distants groups of photosynthetic eukaryotes, may have contributed to the remarkable ecological expansion of angiosperms in aeroterrestrial ecosystems and of diatoms in marine and freshwater environments.
Finally, in some non-photosynthetic parasites derived from lineages that lost their secondary plastids, MGDG synthesis persists through unusual enzymatic systems, including MGD-like fusion proteins, suggesting repurposing beyond photosynthesis.
Together, these transitions illustrate how lipid biosynthesis pathways can be repeatedly reinvented during eukaryotic evolution.

Oral Communication

Very long-chain polyisoprenoids accumulate in rose leaves represent an overlooked class of plant lipids

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Polyisoprenoids are a group of linear isoprenoid-derived lipids. These compounds are synthesized through the sequential polymerization of isoprene units and range in size from short molecules to extremely long polymers, including natural rubber. Polyisoprenoids occur in all living organisms, either as single molecular species or as complex mixtures. Despite their widespread occurrence, the biological functions of polyisoprenoids, particularly very long-chain forms, remain poorly understood.
Roses are widely recognized as a model for studying volatile terpenoids involved in floral scent biosynthesis. Our analyses revealed that rose plants also possess an exceptional capacity to synthesize and accumulate very long-chain polyisoprenoid lipids. We characterized polyisoprenoid composition in different rose organs and identified several polyisoprenoid families spanning a broad range of chain lengths, including exceptionally long compounds containing up to 50 isoprene units. These lipids accumulated predominantly in leaves and young shoots.
The abundance and composition of polyisoprenoid families varied considerably among leaves from individual plants but showed little response to short-term temperature and light treatments. In contrast, leaf age strongly affected accumulation. Mature leaves contained substantially higher amounts of polyisoprenoids than young leaves, particularly compounds longer than 26 isoprene units. While shorter-chain species increased several-fold during development, very long-chain polyisoprenoids increased more than forty-fold.
The physiological role of these compounds remains unknown. Their accumulation in photosynthetic tissues and strong increase during leaf maturation suggest involvement in fundamental cellular processes. By revealing the abundance and diversity of these lipids, this work provides a basis for future studies of their biological function in plants.

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Oral Communication

Remorin-mediated protein-lipid networking during nanodomain formation in plant cells

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Biological membranes are highly dynamic platforms that can regulate essential cellular processes through the recruitment and organization of peripheral membrane proteins into functional supramolecular assemblies. In plants, remorins are multifunctional proteins, regulating immunity, development and symbiosis (1). Their biological functions depend on membrane association, where they sequester specific lipids into functional membrane nanodomains. By integrating solution and solid-state NMR spectroscopy, advanced molecular imaging, and molecular dynamics simulations, we uncover the structural principles that govern remorin membrane recognition, self-assembly, and biological function (2, 3, 4). We show that remorins form coiled-coil oligomeric pre-assemblies decorated with a dynamic, fuzzy region and a barcode-like distribution of positively charged residues prior to membrane binding. Membrane association then promotes lipid sequestration, modulates membrane biophysical properties, and reorganizes the electrostatic landscape at the protein–membrane interface (5). Together, our findings provide a molecular framework for understanding how remorins couple protein self-assembly with lipid organization to generate functional membrane nanodomains.

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Oral Communication

^{13}C-Metabolic Flux Analysis Elucidates Carbon Partitioning during Unusual Fatty Acid Synthesis

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Physaria fendleri is a Brassicaceae native to the southwestern U.S. and northern Mexico that produces high levels of hydroxy fatty acids (HFAs) in its developing embryos. Free of toxins and rich in HFAs, *Physaria* is an attractive domestic alternative to castor bean, the only current commercial source of HFAs used in lubricants, polymers, cosmetics, etc. *Physaria* has a short life cycle, can be grown on marginal lands, and does not compete with food crops, making it a promising industrial oilseed. However, its oil yield and oil composition must be improved to enhance its economic viability.

The predominant HFA in *Physaria* seed oil is lesquerolic acid (C20:1OH), produced by elongation of ricinoleic acid (C18:1OH). From an industrial perspective, increasing total oil content while shifting HFA composition toward ricinoleic acid is desirable, as this shorter HFA is the form currently used in industry. Because genetic engineering of *Physaria* remains challenging, we developed embryo culture conditions that mimic development *in planta*, enabling chemical inhibition of defined metabolic steps and assessment of carbon partitioning. Using this system, we tested a chemical inhibitor of cytosolic acetyl CoA carboxylase (ACCase), a key enzyme required for fatty acid elongation. ACCase inhibition increased ricinoleic acid accumulation but reduced total oil yield. We hypothesized that inhibiting fatty acid elongation perturbs carbon partitioning during seed filling and tested this hypothesis using a comparative multi omic analysis of developing embryos grown in the presence or absence of the inhibitor.

This study provides the first flux map of carbon partitioning in developing *Physaria* embryos. ACCase inhibition reduced carbon conversion efficiency, largely due to decreased Rubisco activity, and lowered carbon allocation to oil by increasing fatty acid turnover and altering precursor availability. Together, these results provide a framework to guide strategies for improving oil yield and composition in *Physaria*.

Oral Communication

Dynamics and role of 3-hydroxylated lipids in the regulation of dormancy in cherry floral buds.

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Since winter and spring temperatures greatly influence bud dormancy progression, with acute impacts on flowering and fruit production, understanding the mechanisms that take place in buds during dormancy onset, maintenance and release, appears crucial in the current context of global warming. However, the mechanisms that govern lipid metabolism during bud dormancy remain poorly understood. We have followed the evolution of lipid content during bud formation and dormancy breakage in sweet cherry (*Prunus avium* L.) flower buds. Surprisingly, we detected significant amounts of 3-hydroxylated fatty acids, accounting for approcimately 50% of the total fatty acids present in dormant flower buds. These 3-hydroxylated fatty acids seem to be specific to bud dormancy, as they are absent from leaves, and their concentration increases during bud formation, though it drops sharply during flowering. Their presence is limited to the *cerasus* sub-section of *Prunus* plants, suggesting an evolutionary origin linked to cold climates. Their metabolic origin and functional significance in dormant buds are currently being investigated.

Session 3

Lipid, Nutrition and Health

Invited Lecture

Extracellular vesicles as mediators of the anti-thrombotic effects of n-3 polyunsaturated fatty acids

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Extracellular vesicles (EVs) are lipid bilayer-enclosed structures released by nearly all cell types under physiological and pathological conditions. They have been implicated in endothelial dysfunction, inflammation, and thrombosis, with elevated circulating EV numbers linked to cardiovascular risk factors such as hypercholesterolaemia, dyslipidaemia, and hypertension. Platelet-derived EVs represent the predominant EV population in the circulation and strongly promote platelet activation, thrombin generation, and thrombus formation. Remarkably, they are reported to be 50–100 times more procoagulant than activated platelets, owing to increased expression of phosphatidylserine, P-selectin, and factor X.

While dietary and lifestyle interventions are known to modify cardiovascular disease (CVD) risk, little was previously understood about how diet influences EV generation or function. EV membranes are active biochemical platforms whose phospholipid composition determines coagulation and inflammatory signalling, so the impact of n-3 polyunsaturated fatty acid (PUFA) supplementation on platelet EV production activity may be important.

Through a series of studies, we demonstrated that circulating EV numbers are strongly associated with 10-year CVD risk, and that n-3 PUFA supplementation in individuals at moderate CVD risk significantly reduced circulating EV levels. Platelet-derived EVs from supplemented subjects were enriched in eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), effectively doubling their n-3 PUFA content, and displayed a diminished capacity to support thrombin generation.

In conclusion, EV membranes are procoagulant lipid platforms, and these findings suggest that dietary n-3 PUFA alter EV number and remodel EV membranes, thereby altering EV biology and thrombotic potential.

Apolipoprotein D modulates inflammation in a sex-dependent manner

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Background: The lipocalin Apolipoprotein D (ApoD) is a multi-functional protein mainly express in the central nervous system (CNS), where it has been associated with a reduction of inflammation and oxidative stress. We recently demonstrated that ApoD can exit the central nervous system and accumulate in peripheral organs. Transgenic mice overexpressing the human ApoD in the CNS (Thy1) develop a hepatic steatosis without inflammation. Using Thy1 mice, we evaluate the effect of a high fat high sugar (HFHS) diet on the development of systemic inflammation.

Methods: Male and female Thy1 and Wild-Type (WT) mice were fed with a HFHS diet for 12 weeks. Insulin, and glucose tolerance were assessed at week 1/2, 5/6, and 11/12. Mouse were also subjected to Open field test (OFT) and Forced swim test (FST) at week 11 and were put in metabolic cages. Finally, Inflammation markers (IL-6, TNF-a) and oxidative stress markers (MDA, Nrf2, Gpx4) were quantified by RT-qPCR, Western Blot, ELISA, and TBARS in liver and brain. Lipidomic analysis were also performed in the liver.

Results: Our results show a differential response to ApoD overexpression between male and female mice on the HFHS diet. First, males Thy1 mice have better insulin sensitivity in response to the diet while females show increase insulin tolerance. Inflammation was increase in the liver in Thy1 females but decreased in males. Regardless of sex, overexpression of ApoD also decrease oxidative stress in the liver and decreased anxiety and depression.

Conclusions: Our results suggest that ApoD acts differently between males and females, while retaining its capacity to neutralize reactive lipid species.

Oral Communication

Plant-based omega-3 and lipid metabolism in obesity: comparison of plant glycolipids and linseed oil as naturel carriers of ALA

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It has become important to better understand the metabolic effects of plant-derived nutrients. While plant proteins are deeply studied, plant glycolipids (GLs), carriers of omega-3 alpha-linolenic acid (ALA) in green vegetables, remain poorly explored. This study investigated the effects of ALA-rich GLs on lipid metabolism in obese mice and compared them with a conventional plant source of ALA.

Swiss male mice were fed with four diets for 12 weeks (n=12 each): a standard balanced diet, a high-fat high-sucrose control diet (HFHS), a HFHS diet supplemented with ALA delivered by spinach GLs (HFHS+GL), and a HFHS supplemented with ALA equivalent amount delivered by flaxseed oil triglycerides (TGs) (HFHS+flax). An oral lipid tolerance test was performed at week 11. Fecal and plasma lipidomics (GC-MS and LC-MS/MS) were performed at week 12 after animal euthanasia.

After nutritional intervention, high-fat groups displayed greater body weight gain and lower food intake than standard group (p <0,0001), while HFHS+GL and HFHS+flax did not differ from HFHS. Fasting plasma TGs levels were similarly higher in all high-fat groups compared to the standard group (p <0,0001). However, HFHS induced a marked postprandial hyperlipemic response at 2h attenuated by ALA supplementation (p <0.05), with comparable effects in HFHS+GL and HFHS+flax groups. Fasting plasma lipidomics showed no changes in total fatty acid (FA) concentration between groups but increased n-3 FAs and reduced n-6 FAs proportions in HFHS+GL and HFHS+flax groups *vs* HFHS (p <0,0001). Total fecal FAs were reduced in HFHS+GL and HFHS+flax *vs* HFHS (p <0.001) without changes in free fecal FAs. Among esterified FAs, GLs were specifically increased in HFHS+GL feces (p <0.001).

Plant GLs beneficially modulate key parameters of lipid metabolism with similar effects to flaxseed TGs. These findings support the potential of GLs as an alternative or complementary plant-based source of n-3 fatty acids for metabolic health.

Oral Communication

$sPLA_2$-XIIA drives extracellular vesicle phospholipid metabolism to promote pathogenic Th17 responses

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Introduction: Extracellular vesicles (EVs) have recently emerged as important regulators of intercellular communication and lipid signaling. Here, we investigated whether EVs function as a hydrolytic platform for extracellular phospholipid metabolism and whether secreted phospholipase A_2 ($sPLA_2$) regulates this pathway during pathogenic Th17 responses.
Methods: We investigated the function of group XIIA $sPLA_2$ ($sPLA_2$-XIIA), an atypical member of the $sPLA_2$ family, using genetic mouse models, *ex vivo* Th17 cell differentiation, lipidomics, proteomics, RNA sequencing, EV analyses, pharmacological approaches, and murine models of Th17-related diseases such as psoriasis and arthritis.
Results: Among all PLA_2 isoenzymes expressed in Th17 cells, $sPLA_2$-XIIA was the most highly expressed. We also found that its expression was transiently upregulated by TCR activation. $sPLA_2$-XIIA deficiency suppressed pathogenic Th17 differentiation and ameliorated Th17-related diseases. $sPLA_2$-XIIA hydrolyzed phosphatidylethanolamine-rich EV membranes, generating lysophosphatidylethanolamine, which was converted by autotaxin into lysophosphatidic acid. This pathway promoted pathogenic Th17 differentiation through LPA_2 signaling. EV secretion increased during Th17 differentiation in WT cells but not in KO cells. In addition, WT-derived EVs were taken up by Th17 cells, whereas KO-derived EV uptake was markedly reduced. Multi-omics analyses revealed that $sPLA_2$-XIIA deficiency altered EV lipid composition, reduced the abundance of EV-associated proteins, and changed the expression of a small subset of EV-associated miRNAs. Furthermore, treatment with a neutralizing anti-$sPLA_2$-XIIA antibody suppressed Th17 responses and ameliorated Th17-related diseases.
Conclusions: Our findings identify $sPLA_2$-XIIA as a key regulator of EV phospholipid metabolism that drives intercellular lipid signaling and pathogenic Th17 responses, establish EVs as a hydrolytic platform for extracellular phospholipid metabolism, and support $sPLA_2$-XIIA as a promising therapeutic target for Th17-related diseases.

Oral Communication

Dietary Lipid Epoxide as Novel Inducer of Dose-Dependent Intestinal Inflammation

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Lipid epoxides are abundant oxidation products in edible oils and processed foods, yet their biological impact on the intestine remains largely unknown. Here we show that dietary supplementation with synthesized 1-(9,10-epoxyoctadecenoyl)-2-stearoyl-3-oleoyl-sn-glycerol (EpTAG), at dietary-relevant doses of 4-100 µg/day, induced dose-dependent, region-specific intestinal inflammation in C57BL/6 mice fed a high-fat diet. Using targeted multivariate analysis of gene expression, oxylipin profiles, fatty acids, and gut microbiota across six intestinal segments, we demonstrated that EpTAG weakened pro-resolving lipid mediators while enhancing pro-inflammatory signaling along the gastrointestinal tract, with the strongest response in the proximal jejunum, where soluble epoxide hydrolase (sEH) was induced up to 26-fold. Additionally, the multivariate integration analysis identified PGJ_2 as a candidate biomarker of dietary epoxide exposure. EpTAG supplementation further disrupted colonic microbiota composition, shifting butyrate-producing communities. These results established that diet-derived epoxidized triacylglycerols act as bioactive intestinal inflammatory mediators at levels found in food. Together, these results challenged the paradigm that peroxide value alone is sufficient to characterize dietary oxidized lipids and highlighted the need for a reassessment of lipid oxidation monitoring practices and exposure thresholds in food safety guidelines.

Session 4

Lipids and Brain

Invited lecture

New methods lead to new insights on the role of diet and the liver in maintaining a healthy fat brain

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Docosahexaenoic acid (DHA; 22:6n-3) is prevalent in the brain, playing a crucial role in regulating cell survival, neurogenesis, and neuroinflammation. While DHA can be obtained through dietary sources, particularly fish, it can also be synthesized from alpha-linolenic acid (ALA; 18:3n-3), a plant-based precursor, via a series of desaturation and elongation reactions in the liver. The efficiency of DHA synthesis from ALA remains a topic of considerable debate, particularly important in light of ecological concerns regarding fish stocks and an increase in non-fish-eating populations. Compounding this issue is a limited understanding of human DHA requirements. In this presentation, I will discuss new isotopic methods that have revealed several unexpected insights into DHA metabolism, including a feedback inhibition mechanism whereby DHA suppresses its own synthesis in the liver. These and other findings will be contextualized within the framework of DHA homeostasis and requirements.

Oral Communication

Aging and APOE Genotype Significantly Impact Myelin Lipid Synthesis and Turnover in Mice

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Myelin is comprised 70-80% of lipids and undergoes continuous turnover to sustain its structural integrity. Genetic studies strongly implicate altered brain lipid trafficking and catabolism as key contributors to the etiology of Alzheimer's disease. We hypothesise that risk genes including APOE4 synergise with ageing to promote neurodegeneration through impairing myelin lipid turnover.
This study introduces a new method to track brain lipid turnover by administering deuterium oxide in the drinking water of mice for 8 weeks, commencing at 3 and 12 months of age, resulting in deuteration of newly synthesised brain lipids. High-resolution liquid chromatography-tandem mass spectrometry was employed to quantify deuterium incorporation into lipids (~200 lipids from 17 structural classes) from the corpus callosum, hippocampus, and biochemically-isolated myelin.
This approach revealed tremendous heterogeneity in turnover rates for different myelin lipid components. Myelin-specific glycosphingolipids (hexosylceramides and sulfatides) demonstrated very long half-lives, with only 10% of the lipid pool replaced in 8 weeks. In contrast, over 90% of common cellular glycerophospholipids (phosphatidylcholine, phosphatidylethanolomine, e.t.c.) were replaced within 8 weeks. The rate of myelin sphingolipid and cholesterol replacement declined significantly between 3 and 12 months of age, while deletion of ApoE selectively impaired cholesterol turnover. Accumulation of cholesterol and myelin sphingolipids, particularly sulfatide, was seen in corpus callosum and hippocampus of ApoE knockout and APOE4 knock-in compared to WT and APOE3 knock-in mice. This result indicates that APOE4 is likely to be a loss of function allele for myelin lipid turnover.
Aging and ApoE genotype are by far the greatest risk factors of Alzheimer’s disease. We therefore propose a model in which ageing synergises with APOE4 genotype to impair myelin cholesterol and sphingolipid turnover, leading to myelin degeneration. This improved understanding is important in the new therapeutic frontier of myelin repair for neurodegenerative diseases.

Oral Communication

Correction of hippocampal fatty acids rescues synapse and energy programs of 5xFAD glutamatergic neurons

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Alterations in brain lipid metabolism are a central feature of Alzheimer's disease (AD), yet effective therapeutic strategies targeting these disturbances remain lacking. Prior work showed that inhibition of the fatty acid desaturase stearoyl-CoA desaturase (SCD) improves cognition in the slow-progressing 3xTg-AD mouse model. Here, we combined lipidomic, morphological, and single-nucleus RNA sequencing (snRNA-seq) approaches to investigate how AD mutations and SCD inhibition remodel hippocampal lipid metabolism and glutamatergic neuron subpopulations in the rapidly progressing 5xFAD model.

Inflammatory and amyloid neuropathologies were assessed by immunohistochemistry and ELISA. Fatty acid profiling of hippocampus, cortex, and plasma was performed by gas chromatography (GC-FID), while cell-specific transcriptomic changes were characterized by snRNA-seq following one month of intracerebroventricular infusion of an SCD inhibitor or vehicle.

5xFAD mice exhibited early hippocampal lipid alterations, including increased C16:1/C16:0 desaturation index and increased C16 desaturation within the major phospholipid fraction. Multi-omic analyses identified a conserved excitatory neuron signature of lipid pathway dysregulation, characterized by increased cholesterol biosynthesis and reduced phospholipid, glycerophospholipid, triglyceride, and arachidonic acid pathways, together with subpopulation-specific disturbances in synaptic and energy metabolism programs affecting hippocampal dentate gyrus and subiculum neurons. SCD inhibition remodeled hippocampal lipid metabolism, correcting the conserved lipid dysregulation signature and increasing DHA, and total polyunsaturated fatty acids. At single-cell resolution, SCD inhibition restored energy metabolism pathways in dentate gyrus and subiculum neurons and enhanced synapse-associated pathways across multiple hippocampal excitatory neuron populations, including reversal of AD-associated reductions in activity-dependent immediate-early genes and vGlut/PSD95 protein signal. These transcriptomic changes correlated with increased dendritic spine density in dentate gyrus and CA1, without altering amyloid-β_{42} levels.

Together, these findings demonstrate that targeting SCD remodels brain lipid metabolism and restores neuronal energetic and synaptic programs in the aggressive 5xFAD model, without changing amyloid deposit, supporting fatty acid metabolism as a promising therapeutic avenue for AD.

Oral Communication

The Chemistry of Inflammation Relief: Lipid Mediators Profiling Reveals How Gene Therapy Restores Brain Homeostasis in 5xFAD Alzheimer Mouse Model

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Alzheimer's disease (AD) is characterized by a multifactorial pathophysiology with mutual interaction and influence between nervous and immune systems. Deposition of amyloid-β (Aβ) outside neurons activates microglia and astrocytes, inducting cytokine production and causing chronic inflammation. Interventions for targeting neuroinflammation have demonstrated to protect against the progress of AD. A growing body of evidence has demonstrated the therapeutic potential of gene therapies based on transcription factors that regulate cellular quiescence, tissue homeostasis, and gene networks disrupted in AD. Lipid dysregulation has emerged as a critical therapeutic target. Together with oxidative stress and neuroinflammation, alterations in lipid metabolism are increasingly recognized as key contributors to AD pathogenesis and progression.

Research on lipid mediators (LMs) that play a role in neuroinflammation resolution has recently addressed. In healthy organisms, the resolution of inflammation can be considered as an active programmed response, returning the organism to homeostasis. It is a 'turned on' and highly coordinated process in which recent research has demonstrated the key role of the specialized pro-resolving mediators (SPMs) including lipoxin, resolvin, protectin and maresin families.

This study investigates the effects of a central nervous system–targeted gene therapy on the brain cortex LMs profile of the 5xFAD mouse model of AD. The therapy is based on transcription factors capable of interacting with key cytoplasmic proteins and is designed to regulate the cell cycle while promoting tissue homeostasis and regeneration. The neuronal expression of the transcription factor induced a shift on the LOX, COX and CyP450 activities in cortical tissue, leading the formation of SPMs and oxygenated lipid mediators following systemic administration of the Gene Therapy. Understanding the lipid profiling of metabolite formation helps to elucidate the molecular mechanism underlying the contribution of E2F4-Based Gene Therapy to target AD neuroinflammation.

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Oral Communication

Membrane lipid poly-unsaturation selectively affects dopamine D2 receptor endocytosis

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The brain is highly enriched in poly-unsaturated fatty acids (PUFAs) and their deficiency has been associated with several neuropsychiatric disorders. We have tested the effect of varying cellular PUFA content on the endocytosis of G protein coupled receptors. Overnight incubation of cultured HEK-283 cells or rat cortical neurons with the n-3 PUFA docosahexaneoic acid (DHA) or the n-6 PUFA docosapentanoic acid (DPA) is sufficient to affect their PUFA membrane composition specifically. We found that membrane enrichment with either DHA or DPA significantly impairs agonist-induced endocytosis of the dopamine D2 receptor (D2R), a main target of antipsychotics, without affecting clathrin-mediated endocytosis or the internalization of several other GPCRs, including D1R. Moreover, we show that PUFA treatments do not affect D2R clustering at endocytic pits, but strongly impair β-arrestin2 recruitment and slow down endocytic vesicle formation. Finally, mutation of key residues in intracellular loop 2 abolishes the sensitivity of D2R endocytosis to PUFA enrichment. We conclude that D2R trafficking is specifically dependent on membrane PUFAs, which could influence its role in the control of brain function and behavior.

Session 5

Biopolymers and Lipids

Invited Lecture

Long chain and very long chain lipids in the plant's extracellular space: from physiological importance to biopolymers and biotechnological potential

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The plant's extracellular space is not merely an insignificant, lifeless compartment outside a living cell, but performs vital functions. Structurally important, carbohydrate dominated polymers are common to all cell walls in a plant. However, specialization of tissues is often characterized by particular secondary cell wall modifications involving spatially controlled lipid depositions. The latter include partially oxygenated long-chain and very-long-chain fatty acid derivatives that can cross-link to extended polyester. One example is the biopolymer suberin, best known as the predominant compound in the bark of the cork oak tree. Other occurrences include root dermal tissues and the periderms of starch-rich tubers (e.g. potato) and storage roots (e.g. cassava). There, suberin acts as a protective barrier, enabling the plant to regulate the transport of water and nutrients and prevent the entry of pathogens.

We identified key metabolites (ω-hydroxyacids), structural genes (fatty acid hydroxylases) and transcriptional regulators (NACs) of suberin biosynthesis. Corresponding mutants exhibit a delayed and reduced suberin deposition resulting in higher permeability for water, solutes and organic compounds. On the other hand, genotypes with higher suberin content are more tolerant to drought and salt stress conditions. Thus, developing our understanding of the chemistry, biosynthesis and deposition of suberin may enable the targeted modification of diffusion barrier in plants and the engineering of stress resilient crops. Furthermore, adapted industrial extraction processes can solubilize the suberin polymer in an almost native stage indicating a potential for producing long lasting, bio-inspired materials from agricultural and food waste including suberized peels and barks.

Oral Communication

Accelerated ageing of vegetable oils rich in conjugated linolenic acids reveals polymerisation processes and the formation of highly toxic aldehydes

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Lipid oxidation is a major determinant of the quality and safety of vegetable oils. While the oxidative pathways of conventional polyunsaturated fatty acids (PUFAs) have been extensively characterised, those of conjugated fatty acids, and particularly conjugated linolenic acids (CLnAs), remain poorly understood despite their exceptional oxidative reactivity. This knowledge gap is especially relevant as CLnAs are attracting increasing attention for their anticancer properties through ferroptosis, a mechanism intrinsically linked to lipid peroxidation. Here, we investigated the oxidative behaviour of two CLnA-rich vegetable oils, pomegranate seed oil and tung seed oil, during 80 days of accelerated ageing at 30°C. Linseed oil, rich in the non-conjugated α-linolenic acid, served as a reference. Oxidation was monitored using complementary approaches, including GC-FID, HPLC, HPSEC, LC-MS/MS, peroxide value and *p*-anisidine value. The cytotoxicity of the major oxidation-derived aldehydes was further assessed in healthy and cancer cells. In contrast to linseed oil, CLnA-rich oils exhibited limited hydroperoxide accumulation. Instead, primary oxidation was characterised by extensive polymerisation. Secondary oxidation also followed a distinct pathway, leading to the formation of conjugated dienals, particularly 2,4-*tt*-nonadienal, rather than the aldehyde profile typically associated with conventional PUFAs. Toxicity assays further revealed that these conjugated aldehydes were the most cytotoxic oxidation products identified. These findings demonstrate that conventional oxidation markers alone substantially underestimate primary oxidation in CLnA-rich oils and highlight the need for analytical approaches specifically adapted to conjugated lipid systems. More broadly, this work provides new mechanistic insights into the oxidative chemistry of CLnAs and raises important questions regarding the technological stability and toxicological implications of conjugated lipids.

Oral Communication

Direct transcriptional activation of suberin biosynthesis by MYB9 and MYB107 protects Arabidopsis seeds against abiotic stress from late maturation through germination

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In angiosperms, seed development involves tissues of distinct origins. The zygotes (embryo, endosperm) result from the double fertilization of the embryo sac. They are surrounded by maternal integuments (*i.e*. the seed coat) that contribute to their nutrition and protection. The various cellular layers that make up the seed coat generally display specialized metabolisms that allow the seed coat to fulfil its protective role, even in the dry seed after these cellular layers have died. In the model species *Arabidopsis thaliana*, the integument consists of five cell layers, three of which synthesize protective surface lipid polymers (two are cuticles in nature, and the third is suberin). In this research project, we are evaluating the importance of suberin produced by the outer integument 1 (oi1) layer in seed physiology in response to various abiotic stresses. To do this, we are using mutants with defects in MYB9 and MYB107, two direct transcriptional activators of the suberin biosynthesis pathway in the seed. In the double *myb9 myb107* mutant, the seed produces almost no suberin, but under optimal growing conditions in the laboratory, these mutant seeds do not exhibit any particular phenotype compared to wild-type seeds. Under abiotic stress conditions, however, we observed various physiological alterations leading to impaired dry matter accumulation, accelerated seed aging, and increased susceptibility to toxic compounds in the environment at the time of germination - all characteristics that could negatively impact yield in crop species.

Oral Communication

Synthesis of biobased rheology modifiers for cosmetic applications

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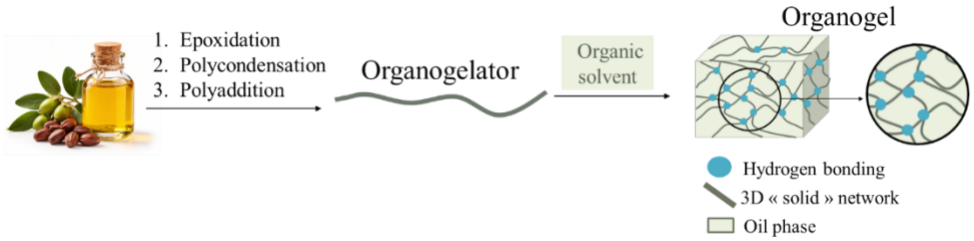
Environmental concerns and the rising demand for sustainable materials are driving the shift from petroleum-based ingredients to biobased alternatives. Key examples are lipids, including fatty acids, fatty alcohols, triglycerides, sterols, and waxes. Jojoba oil, a wax ester, was selected as the starting lipid due to its wide availability, oxidative stability, and non-competition with food uses to create a biosourced organogelator for gelled or thickened cosmetic formulations[1]. The unsaturated structure of Jojoba oil offers numerous chemical functionalizations, enabling the modulation of viscosity through the introduction of groups capables of weak interactions (hydrogen bonds, π-π interactions, Van der Waals forces, etc.), thus contributing to the formation of networks that structure the oily phases, creating an organogel[2]. The initial chemical modification involved epoxidizing the unsaturations with hydrogen peroxide to replace environmentally harmful organic peroxides like mCPBA. The subsequent step entailed ring-opening of the epoxy with bulk-synthesized polyamide-type polymers without solvent or catalyst. Several polyamides were designed by adjusting the spacing between amide groups, the length of the aliphatic chain, and the degree of polymerization, aiming to study how these variations affect the physical properties of the resulting organogelators, including softening temperature and solubility[3]. This strategy enables the introduction of polar functions (such as hydroxyl, ester, and amide groups) that can enhance intermolecular interactions and, consequently, the viscosity of the final product. Finally, formulations containing different weight percentages of organogelator in oils were prepared and characterized by oscillatory and rotational rheological measurements to determine key parameters such as dynamic viscosity and gel point for cosmetic applications.



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Oral Communication

Bio-based Estolides from metropolitan Erucic Acid : synthesis and performances

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Climate change and environmental concerns foster the search for more eco-friendly products and solutions. In this context, the design of bio-based chemical products, with reduced dependance to petroleum, has been established as a relevant alternative. Specializing in the derivatizing and functionalizing vegetable oil-based materials, ITERG has been working for the last ten years on the development of estolides, a specific range of polyester-based oligomers. Estolight-1E and Estolight-1AH are specific bio-based estolides designed to address the needs of the cosmetic industry. Being now produced at the ton scale, they have paved their way towards industrial reality and are products of choice, having demonstrated interesting performances with respect to improved dispersion of fillers and filters, improved textures, and more generally improved sensoriality. They are however based on castor oil, an exotic oil from India or South America.

The search for a local source of product – made in France – with a higher degree of traceability and, at least, similar performances is thus of very high interest. Non-edible Erucic Rapeseed Oil, especially its derived erucic acid, is thus thought to be a promising candidate. This 22-containing carbons fatty acid, containing one unsaturation, can be utilized as a platform molecule and derivatized to obtain a mono-hydroxylated saturated C22-compounds, analogue to 12-hydroxysteartic acid derivatives.

The obtained free fatty acids and methyl esters have then been engaged in polycondensation reactions to yield erucic acid-based estolides, with various structures including amphiphiles. Their performance have been further investigated with respect to physico-chemical properties and use as stabilizers of water/oil phase interfaces.

Session 6

Advanced Topics and Industrial challenges

Oral Communication Sponsored by Elsevier

Generative AI in Scientific Writing and Scholarly Publishing

Elsevier

Generative artificial intelligence is rapidly becoming embedded in research and scholarly communication. Researchers are using AI tools to discover and summarise literature, support data analysis, develop research ideas, draft funding proposals, improve language, and prepare manuscripts. These applications offer substantial benefits, including greater efficiency, improved accessibility for authors who use English as an additional language, and new ways to communicate and promote research.

However, generative AI also introduces significant risks. Outputs may be inaccurate, incomplete, biased, outdated, or based on fabricated sources, while extensive reliance on AI may weaken critical thinking and subject expertise. Additional concerns include confidentiality, data privacy, intellectual property, research integrity, and environmental impact.

This presentation examines how the scholarly publishing community is responding through evolving policies and guidance. It outlines Elsevier's principles for the responsible use of generative AI by authors and reviewers, emphasising human oversight, accountability, transparency, disclosure, and protection of confidential material. It also considers the appropriate use of AI-generated visual content and introduces LeapSpace, Elsevier's AI-assisted research workspace, which grounds responses in traceable scholarly evidence. The session will help the audience understand both the opportunities and limitations of AI while maintaining trust and integrity in research.

Oral Communication Sponsored by ILS

The ILS approach to lipidomics data quality — from the Lipidomics Reporting Checklist to lipidome concentration databases

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Mass spectrometry-based lipidomics has transformed lipid research and driven a rapid expansion of lipidomic data over the past two decades. Yet this growth has been accompanied by persistent concerns about data quality: numerous studies still report lipid misidentification and inaccurate quantification, undermining both the comparability of results between laboratories and their biological interpretation. Reliable quantification is not merely a reporting formality — accurate absolute concentrations are a prerequisite for relating lipidomes to membrane properties and function.

The International Lipidomics Society (ILS; https://lipidomicssociety.org) was founded to address these challenges by driving standardization and harmonization across the field. A central instrument of these efforts is the Lipidomics Minimal Reporting Checklist (https://lipidomicstandards.org/reporting_checklist/), which describes all essential steps of a lipidomic experiment in a standardized way, serving both as guidance for researchers and as a practical tool for reviewers assessing data quality.

Building on this foundation, we introduce two complementary resources: LipidCascade, a database for hosting and evaluating ILS inter-laboratory ring trials, and LipidCompass, a FAIR community database for qualitative and quantitative lipidomes that supports the review of submitted datasets. Together, these resources extend the checklist from documentation toward active data curation: ring-trial benchmarking supports method harmonization and reduces misidentification, while curated absolute concentrations enable cross-laboratory comparison and the interpretation of lipidomes in terms of membrane biology.

These interconnected efforts advance lipidomics from a rapidly growing field into a robust, quantitative analytical discipline spanning basic research and clinical application.

Oral Communication Sponsored by HEART

L5 and Lp(a): Emerging Biomarkers for ASCVD Risk and Prevention

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Although low-density lipoprotein cholesterol (LDL-C) remains the cornerstone of atherosclerotic cardiovascular disease (ASCVD) prevention, substantial residual risk persists despite achieving guideline-recommended LDL-C targets. Increasing evidence suggests that lipoprotein quality, rather than cholesterol content alone, contributes importantly to atherogenesis.

Electronegative LDL (L5) is a naturally occurring, highly atherogenic LDL subfraction enriched in apolipoprotein(a) [Apo(a)] and other modified proteins. Unlike native LDL, L5 activates lectin-like oxidized LDL receptor-1 (LOX-1), triggering endothelial apoptosis, oxidative stress, inflammation, senescence, and increased vascular permeability independent of plasma LDL-C. Elevated L5 has been associated with myocardial infarction, ischemic stroke, metabolic syndrome, diabetes, and accelerated coronary artery disease, making it a promising biomarker of residual ASCVD risk.

Lipoprotein(a) [Lp(a)] is an independent causal risk factor for ASCVD and calcific aortic valve disease. Through its Apo(a) moiety, Lp(a) promotes vascular inflammation, thrombosis, plaque progression, and impaired fibrinolysis. Emerging evidence suggests that L5 and Lp(a) act synergistically to amplify endothelial dysfunction and vascular injury, while dysfunctional electronegative HDL (H5) further impairs cholesterol efflux and promotes vascular inflammation.

Advances in precision lipidomics, including rapid assays for electronegative lipoproteins, are facilitating clinical translation. Assessment of L5, Lp(a), and complementary biomarkers such as H5 provides a more comprehensive evaluation of residual cardiovascular risk than LDL-C alone and may enable earlier intervention and more personalized prevention. Together, these findings support a shift from quantitative lipid management toward qualitative lipoprotein characterization for precision prevention of ASCVD.

Oral Communication Sponsored by Bruker

Profiling cell-type specific mitochondrial sub-populations in *Arabidopsis thaliana* with multi-omics mass spectrometry

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Introduction

Intracellular, mitochondrial diversity and division of labor are emerging concepts with potentially far-reaching consequences in biology. To elucidate their molecular basis in the model organism *Arabidopsis thaliana*, we developed a novel approach for cell-type specific and protein-based isolation of mitochondria, mRACE [1], and applied trapped ion mobility spectrometry (TIMS) and high-resolution mass spectrometry (MS) to characterize mitochondrial lipidomes and proteomes. TIMS-MS-based profiling of intercellular mitochondrial diversity can uncover molecular signatures associated with, and potentially mediating, plant heat stress responses in functionally different cell types and guide further insight into intracellular mitochondrial diversity.

Main results

Proteomics and lipidomics experiments revealed mitochondrial diversity in mesophyll and guard cells under heat stress. Specifically, we observed significant remodeling of the mitochondrial proteome and lipidome particularly after prolonged heat stress, with distinct protein and lipid networks unique to each cell type. Hundreds of proteins were differentially expressed. Furthermore, over 200 lipid species were annotated and several lipid classes, such as distinct cardiolipin species, discovered to be upregulated in response to heat. Integration of proteomic and lipidomic data uncovered interaction networks and cell-type-specific molecular markers, highlighting the power of multi-omics mass spectrometry for discovering biomarkers of heat resilience and facilitating the study of mitochondrial diversity across systems.

Methods

Mitochondria were isolated from rosette leaves of 17-day-old *Arabidopsis* grown under control or heat stress using mRACE1. We analyzed the lipidomes and proteomes of four biological replicates per condition of separate extracts obtained from split sample aliquots. Lipids were analyzed by C8 reversed-phase separation and 4D-TIMS-PASEF acquisition on a timsMetabo. Protein digestion was performed with iST-Kit followed by dia-PASEF protein analysis on a timsTOF Ultra 2 using 22 min nanoLC gradients. Data were processed in MetaboScape (Bruker Daltonics) and Spectronaut-19 (Biognosys), respectively. Subcellular protein localization was assessed via SUBA5 and interaction networks via STRING data base [2].

[1] Hater et al., Mitochondrial cell-type-specific profiling: Differential function of mesophyll and guard cells is reflected by their mitochondrial proteome. Physiologia Plantarum. 2025.

[2] Szklarczyk et al., The STRING database in 2023: protein–protein association networks and functional enrichment analyses for any sequenced genome of interest. Nucleic Acids Res. 2023

Oral Communication Sponsored by Bruker

A Multi-Phase NMR View of Lipids in Their Native Context

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Lipids rarely exist as uniform solutions. In foods, tissues, membranes and formulations, they coexist with water, proteins, carbohydrates and other components, forming domains that range from highly mobile to rigid. Composition alone therefore provides only part of the picture: lipid function is also shaped by mobility, phase behaviour, molecular organisation and interactions with the surrounding matrix. Nuclear magnetic resonance (NMR) is well suited to this complexity because different experiments can selectively observe molecules according to how freely they move, while preserving much of the original sample context. This presentation will illustrate a complementary strategy that links molecular fingerprints to lipid organisation across several levels of complexity. Comprehensive multi-phase NMR and high-resolution magic-angle spinning provide detailed spectra of mobile constituents in heterogeneous samples and can be combined with solid-state measurements of less-mobile matrix components. In intact rat liver tissue, this approach gives direct access to mobile metabolites and phosphorus-containing constituents without prior extraction. In pistachios, an extraction-free workflow combines mobile metabolite and lipid-related fingerprints with information from the more rigid kernel matrix, supporting exploratory differentiation by geographical origin. At the rigid end of the mobility range, natural-abundance solid-state NMR of native stratum corneum distinguishes solid and fluid fractions within the skin barrier. Sensitivity-enhanced multidimensional measurements reveal minor signals from cholesterol, ceramides and triacylglycerols and provide information on the spatial organisation of protein and lipid phases. Together, these examples illustrate how complementary NMR measurements can connect lipid composition, mobility and molecular organisation while preserving the complexity of the sample.

Oral Communication Sponsored by Chairmen

Mustard oil and press cake in the production of vegan mayonnaises

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Mayonnaise is a popular culinary product used in salads, sauces, and meat dishes. From a physical point of view, it is an oil-in-water emulsion. High-fat mayonnaises contain more than 70% oil, so it is important that the oil used for mayonnaise production is of good quality. Recently, the market has seen an increase in vegan products. Therefore, there is a growing need for solutions that replace egg yolk, which is one of the three main ingredients of mayonnaise. In this study, rapeseed oil was replaced with oil from white mustard Warta variety (low erucic acid, low glucosinolate). At the same time, a mayonnaise formulation with mustard press cake (2.5%; 5%; 10%) instead of egg yolk was developed. Emulsion stability (microscopy), viscosity (rheometry), oil quality (acid value, peroxide value), and bioactive compounds during storage (GC, HPLC, spectrophotometry) were examined. The addition of mustard seed press cake made it possible to obtain a mayonnaise emulsion without the addition of egg yolk. A stable product was obtained. In addition, both the phytosterol content and the n-3 to n-6 fatty acid ratio were higher than in the mayonnaise based on rapeseed oil. The antioxidant activity of the mustard oil-based mayonnaises was also greater than that of the rapeseed oil-based mayonnaise. However, the addition of mustard seed press cake reduced the overall sensory score of mayonnaise. An increase in the proportion of press cake caused a decrease in the overall sensory score of the mayonnaises.

Social Events

Wine making and tasting experience

Public conference

Alchemistry and physics of wine tasting: a love and hate story

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The “conference-wine tasting” will be divided into two parts: one “theoretical” and the other practical.
The 8,000-year history of wine, as well as the gentle chemical processes involved in winemaking, will be discussed: grape composition, crushing, destemming, maceration, alcoholic and malolactic fermentation, blending, clarification, aging, bottling, etc. The physical processes related to wine tasting will then be addressed. The five primary taste sensations (salty, sweet, bitter, sour, umami) as well as astringency will be presented. The interaction of tannins (particularly in red wines) with proteins in human saliva will be described. The distinction between wines with soft tannins (velvety, silky) and those with rough tannins (astringent) will be discussed. The concept of affinity (attraction-repulsion) between tannins and salivary proteins will be introduced to explain astringency. The presence of lipids in food and on the oral mucosa will also be described. Finally, an overview of the phenomena occurring in the mouth when wine is consumed with fatty foods will be presented (role of taste receptors, affinity scale).

The second part will be devoted to tasting wines described as “tannic”—that is, astringent—and wines with silky, velvety tannins. A comparison will be made between the physical aspects of the interactions between tannins and saliva and the participants’ perception of astringency. During a second tasting of an astringent wine, after consuming cheeses and/or cold cuts containing fat, the “protective effect” produced by the fat—which is explained by the interactions between the fat and the tannins—will be demonstrated through the following experiment: participants will taste a full-fat cheese followed by an astringent wine.

Reference: Erick J. Dufourc, Wine tannins, saliva proteins and membrane lipids, Biochimica Et Biophysica Acta-Biomembranes, 1863 (2021). DOI.10.1016/j.bbamem.2021.183670.

Session 7

Biodiversity of Lipids

Invited Lecture

Defining Ulcerative Colitis through lipids: multicompartment lipid signatures discriminate microbiome-linked phenotypes.

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Inflammatory bowel diseases, primarily Crohn's disease and ulcerative colitis (UC), are chronic inflammatory disorders of unknown etiology affecting the gastrointestinal tract. Their incidence has increased markedly in recent decades, with prevalence projected to approach or exceed 1% in industrialized countries. This trend underscores the need for robust molecular biomarkers to improve disease stratification. Focusing on UC, characterized by continuous inflammation of the colonic and rectal mucosa, we previously demonstrated profound remodeling of the epithelial lipidome using spatially resolved lipidomics. Building on these findings, we extended our approach to systemic and luminal compartments to identify translational lipid-based biomarkers. Eighteen newly diagnosed UC patients were included. Platelet-free plasma and fecal samples were collected at diagnosis (V1) and after 16 weeks of mesalazine treatment (V2). Plasma lipidomes were profiled using Bligh and Dyer extraction with non-endogenous internal standards, followed by high-resolution direct flow injection analysis on a hybrid quadrupole-Orbitrap platform, enabling quantitative assessment of major lipid classes and fatty acyl composition. In parallel, fecal sterol and stanol profiles were quantified by GC-MS, and microbiome composition was assessed by 16S rRNA sequencing. At diagnosis, lipidomic analysis revealed a consistent depletion in the mass of linoleic acid-containing species in plasma, including cholesteryl esters and phospholipids, consistent with our previous results in a systemic inflammatory context. Concomitantly, the fecal coprostanol-to-cholesterol ratio was reduced. Both plasma and fecal molecular profiles showed partial normalization after treatment. Unexpectedly, stratification based on fecal sterol profiles identified two patient subgroups with distinct metabolic signatures. Patients with low coprostanol levels exhibited the most pronounced depletion of linoleic acid-containing plasma lipids and a marked reduction in microbial taxa involved in cholesterol-to-coprostanol conversion. These integrative lipidomic and metabolite data indicate that disrupted host–microbiome lipid metabolism defines a distinct UC phenotype, potentially reflecting increased mucosal damage and offering a basis for biomarker development.

Oral Communication

ASAH2 is a dual-function lipid hydrolase with ceramidase and lysophospholipase A_1 activities

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Atherogenic lipids are key drivers of atherosclerotic cardiovascular disease (ASCVD), with ceramide (Cer) and lysophosphatidylcholine (LPC) implicated in vascular dysfunction and inflammation. To date, no enzyme has been clearly established to convert circulating Cer or LPC directly into biologically less bioactive metabolites. N-acylsphingosine amidohydrolase 2 (neutral ceramidase, ASAH2) has been primarily characterized as an intestinal ceramidase with two reported isoforms involved in dietary Cer homeostasis. Here, we identified a previously unrecognized lysophospholipase A1 (lysoPLA$_1$) activity in human ASAH2 and investigated the structural basis underlying its dual lipid-hydrolase function.
Quantitative PCR analysis demonstrated that isoform 2 is the predominant variant in the human intestinal tract. Structural modeling and molecular docking revealed that Cer and LPC occupy overlapping positions within a shared catalytic site. Notably, isoform 2 exhibits more stable ligand interactions, likely due to an expanded binding pocket. Guided by these computational insights, we expressed recombinant ASAH2 isoform 2, which robustly hydrolyzed Cer and LPC in human ASCVD LDL–C and in purified lipid standards. Product analysis by mass spectrometry identified sphingosine and L-α-glycerylphosphorylcholine (L-α-GPC), supporting the ceramidase and lysoPLA$_1$ activities of ASAH2. Mutation of shared pocket residues abolished hydrolysis of both substrates, confirming that Cer and LPC are processed through a common functional pocket.
Collectively, our findings define a previously unrecognized lysoPLA$_1$ activity in ASAH2 and establish it as a dual-function lipid hydrolase that integrates Cer and LPC metabolism. This newly identified function positions ASAH2 as a potential therapeutic agent for modulating lipid metabolism in ASCVD.

Oral Communication

Gut microbiota restricts intestinal lipid uptake via modulation of bile phosphatidylcholine metabolism in mice

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The gut microbiota profoundly influences host metabolism, yet its role in dietary lipid uptake and the underlying mechanisms remain poorly understood. Here, we combine stable isotope–labeled tracers with gnotobiotic mouse models to demonstrate that systemic uptake of dietary lipids critically depends on microbial colonization. Physiology-based kinetic modelling revealed that the gut microbiota restricts intestinal lipid absorption from the gut lumen. Within six hours after tracer administration, intestinal contents of microbiota-colonized mice contained up to 12-fold higher lipid levels than those of germ-free animals, attributable to a two-fold decrease in intestinal lipid absorption. Integrative lipidomics and proteomics analyses showed that gut microbes trigger myeloid differentiation primary response 88 (Myd88) signalling, leading to a downregulation of hepatic cytochrome P450 family 7 subfamily B member 1 (Cyp7b1) activity and increased taurocholate production. Taurocholate activates a previously unrecognized phospholipase A1 activity in bile, driving phosphatidylcholine degradation and thereby impairing micelle formation and intestinal lipid uptake. Notably, both a diverse microbiome and Akkermansia muciniphila lower the biliary phosphatidylcholine content essential for luminal lipid solubilization and epithelial uptake. These findings identify a bile-mediated enzymatic pathway linking microbial colonization to host lipid absorption and metabolic homeostasis. Targeting microbiome composition or biliary glycerophospholipid metabolism may therefore offer novel strategies to modulate dietary lipid uptake in metabolic disease.

Oral Communication

Bioavailability of punicic acid from pomegranate seed oil: *in vitro* and *in vivo* evidence

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Pomegranate seed oil (PSO) has attracted increasing interest due to its potential anti-diabetic, anti-obesity, and anti-carcinogenic properties, largely attributed to its high content of conjugated linolenic acids (CLnAs), particularly punicic acid, the predominant fatty acid in PSO. Despite growing evidence supporting its health benefits, the bioavailability of punicic acid and other CLnAs remains insufficiently characterized, limiting a better understanding of their physiological effects.

The present study investigated the bioaccessibility of fatty acids present in PSO using the standardized INFOGEST *in vitro* digestion protocol, with olive oil as reference. Bioaccessibility, a key aspect of bioavailability, was defined as the fraction available for intestinal absorption and was determined by measuring the ratio of fatty acids present in the absorbable fraction (monoglycerides and free fatty acids) relative to total fatty acids, quantified by gas chromatography following solid-phase extraction. Results showed that punicic acid (in PSO) and oleic acid (in olive oil) - both 18-carbon fatty acids present in similar amounts - exhibited high and equivalent bioaccessibility (around 70%). The presence of proteins (whey and soy protein isolates) did not affect the bioaccessibility of major fatty acids. A high recovery index indicated minimal fatty acid degradation during *in vitro* digestion, regardless of oxygen exposure.

To complement the *in vitro* results, the bioavailability of punicic acid was evaluated *in vivo* following oral administration of PSO to mice. Blood lipid analysis over a 4-hour postprandial period revealed that punicic acid accounted for more than 10% of circulating fatty acids, confirming its effective digestion and absorption and supporting the *in vitro* bioaccessibility results. These findings demonstrate that punicic acid in pomegranate seed oil is highly bioaccessible and bioavailable, supporting the nutritional and health-promoting potential of this oil.

Oral Communication

Fish Oil and Krill Oil do not Increase the Omega 3 Index Equally

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The Omega-3 Index (O3I), defined as the percentage of EPA and DHA in red blood cell membranes, reflects long-term omega-3 intake and is inversely associated with cardiovascular risk. We previously showed that, at equivalent doses, krill oil increases plasma EPA and DHA by ~50% compared to fish oil, likely due to differences in lipid form (phospholipids vs triacylglycerols). We therefore examined whether this translates into differences in the O3I in a randomized controlled trial (n = 72, 26 females and 10 males in the fish oil group; 27 females and 9 males in the krill oil group). Baseline O3I did not differ between groups (P = 0.052; fish oil: 4.9 ± 1.0; krill oil: 4.5 ± 0.8). A significant time-by-supplement interaction indicated a greater increase in O3I with krill oil compared to fish oil (P_interaction = 0.0169). Females had higher O3I than males at baseline and after 12 weeks (P_sex = 0.0008). Overall, krill oil increased O3I more than fish oil. These findings suggest that, at equivalent doses, krill oil may be more effective than fish oil for increasing the O3I, a clinically relevant biomarker of cardiovascular risk, and could therefore represent a more efficient strategy for improving cardiovascular risk profiles.

Session 8

Lipids for Medicine & Pharmacy

Invited Lecture

Self-assembly of medium-chain fatty acids and mixed lipid colloids in the gut: solubilization and membrane permeation enhancement

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In the small intestine, bile salts, phospholipids and the fatty acids released during digestion of dietary and pharmaceutical lipids self-assemble into a dynamic population of colloids that governs two functions central to oral drug performance: the solubilization of poorly water-soluble drugs and the membrane activity of fatty-acid permeation enhancers. We combine small-angle neutron scattering with contrast variation, cryo-TEM and coarse-grained molecular dynamics to follow these lipid assemblies from colloidal structure through to function.
Medium-chain fatty acids such as caprate (C10), used to enhance peptide absorption, act largely as free monomers, yet the surrounding colloids continually partition them between free and aggregated states. C10 self-assembly is strongly pH dependent, forming large ellipsoidal aggregates, vesicles and bilayer discs near pH 6.5 and small spherical micelles near pH 8.5, and bile salt and phospholipid media co-assemble with C10 into mixed structures whose morphology tracks fasted and fed composition. A co-formulated peptide is not a passive passenger: octreotide behaves as an amphiphilic cosurfactant, reshaping the assemblies and becoming sequestered within them, lowering its own free fraction. At the membrane, caprate inserts deeply and enhances permeability in a concentration-dependent manner, whereas the derivative SNAC resides near the headgroups and shows threshold behavior. The same colloids also solubilize hydrophobic drugs: in simulations, solutes such as probucol partition into micelle cores and more polar drugs sit nearer the surface, with the drug-micelle contact ratio tracking measured solubility and fed-state effects.

Together the results recast intestinal lipid colloids as active participants in both solubilization and permeation enhancement, set by bile salt, phospholipid, fatty-acid and peptide composition, offering structural principles for the rational design of biorelevant media and fatty-acid-based enhancers.

Oral Communication

Lipid Self-Assembly as a Platform for Next Generation Nanomedicine in Neurodegenerative Disorders

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Lipid self-assembly provides a versatile and highly tunable platform for engineering next-generation nanomedicines aimed at treating neurodegenerative disorders. Advances in understanding lipid polymorphism and supramolecular organization have enabled the rational design of biomimetic nanostructures, including liquid crystalline nanoparticles such as cubosomes, hexosomes, and spongosomes [1-5]. These nanoarchitectures offer unique internal geometries, high loading capacities, and controlled-release properties that are particularly suited for the delivery of fragile therapeutic agents, including neurotrophic proteins, peptides, nucleic acids, and antioxidant lipids like PUFA-plasmalogens. Our recent works demonstrated how plasmalogen-enriched systems and nanoantioxidants can counteract oxidative stress, while neurotrophin-loaded lipid carriers support neuronal survival and regeneration [3-5]. Synchrotron small-angle X-ray scattering (SAXS) studies have further elucidated the structural transitions and protein–lipid interactions that govern nanocarrier stability and performance, guiding the optimization of formulations for central nervous system delivery [2,3]. I shall present recent results on the structural engineering of liquid crystalline lipid nanoparticles and their application in neurotrophin and plasmalogen-based therapies for brain repair. Together, these advances highlight the potential of self-assembled lipid nanostructures to overcome biological barriers, enhance therapeutic precision, and open new avenues for regenerative strategies in Alzheimer's, Parkinson's, and related neurodegenerative diseases.

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Oral Communication

Circulating lipid and metabolic biomarkers for Prediction of Immunotherapy Response in advanced NSCLC patients

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Immunotherapy has been the most clinical innovative strategy for lung cancer treatment. However, most NSCLC patients do not respond to Immune checkpoints inhibitors (ICIs) or will eventually experience disease progression. Lipid metabolism seems to be primarily involved in many cancer and immune processes and could therefore be a good candidate to understand ICI resistance. The aim of this study is to correlate the circulating lipid profile with NSCLC evolution and ICI response. Blood was collected at baseline and after ICI treatment in advanced NSCLC patients. Plasma and erythrocytes were separated by centrifugation and circulating fatty acids, phospholipids, neutral lipids, oxysterols and TCA intermediates were assessed by gas and liquid chromatography approaches. Cox regression analysis showed that higher baseline levels of omega-6 polyunsaturated fatty acids (PUFAs) in whole blood were associated with shorter progression-free survival (PFS). After treatment, higher percentage of linoleic acid was also linked to shorter PFS, whereas increased plasma arachidonic acid was associated with improved overall survival (OS) both at baseline and after treatment. Higher baseline plasma percentages of palmitic acid were associated with shorter OS.

The assessment of neutral lipids composition suggests a decrease in the omega-3 PUFA induced by immunotherapy, related to docosahexaenoic (DHA) and docosapentaenoic acids, but not to eicosapentaenoic acid. After immunotherapy the evaluation of plasma phosphatidylcholines (PCs) highlights an increase in oleic, linoleic and of gamma linolenic acids. Conversely, in plasma PCs immunotherapy determines a significant reduction of omega-3 PUFA, in particular of DHA. Further studies will clarify the molecular mechanisms involved in ICI response to develop targeted strategies.

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Oral Communication

Dendrogenin A, a Cholesterol-Derived Metabolite, Reprograms Endolysosomal BMP Metabolism to Generate Immunogenic Antitumour Extracellular Vesicles

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Dendrogenin A (DDA) is an antitumour epoxycholestanoid that promotes the secretion of immunogenic small extracellular vesicles (DDA-sEV) enriched in bis(monoacylglycero)phosphate (BMP). BMP is a phospholipid specific to late endosomes and lysosomes, where it plays a central role in lipid degradation, regulates endosomal cholesterol trafficking, and contributes to intraluminal vesicle biogenesis. Dysregulation of BMP biosynthesis has been associated with several human diseases, but its role in cancer remains poorly understood.

The aim of this study was to investigate the role of BMP in tumour immunity. We found that DDA, through activation of liver X receptor β (LXRβ), induces the expression and activity of phospholipase D (PLD) and CLN5, two enzymes recently identified as key mediators of BMP biosynthesis. Pharmacological inhibition of PLD in DDA-treated tumour cells decreases BMP levels in DDA-sEV, reduces their secretion, and impairs their antitumour immune activity. Blocking BMP on DDA-sEV with a specific anti-BMP antibody abolishes their antitumour efficacy, prevents the recruitment of activated dendritic cells (DC) and T cells into tumours, and reduces mouse survival. This blockade also markedly impairs DDA-sEV uptake by immature DC (iDC), thereby preventing DC maturation and subsequent Th1 T-cell activation. In addition, neutralisation of the BMP-presenting receptor expressed on iDC inhibits DDA-sEV uptake and DC maturation. Treatment of iDC with free BMP was sufficient to induce their functional maturation. Furthermore, BMP-enriched DDA-sEV enhance the therapeutic efficacy of anti-PD-1 treatment in melanoma.

Collectively, these findings identify BMP as a critical phospholipid regulating the immunogenic activity of tumour-derived sEV and indicate that pharmacological activation of the DDA/LXRβ/BMP axis represents a promising lipid-based strategy to enhance anticancer immunity and improve responses to immune checkpoint blockade.

Oral Communication

Loss of the lysophosphatidic acid-induced immediate early gene IEX-1 exacerbates acute radiation syndrome

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Long-lasting activation of ERK and Akt mediated pro-survival signal transduction pathway is a key element of protection and mitigation of acute radiation syndrome (AES). To dissect the initial steps downstream of the lysophosphtaidic acid receptor subtype 2 (LPAR2) is not clearly understood. The immediate early response gene-3 (IER3/IEX-1) encodes a radiation-inducible adaptor protein that integrates stress-responsive and pro-survival signaling pathways, including ERK-dependent survival and apoptosis regulation; however, its role in ARS remains undefined. Here, we demonstrate for the first time that IEX-1 is a critical determinant of survival in murine models of both the hematopoietic (H-ARS) and gastrointestinal (GI-ARS) radiation injury. Following 9 Gy total body irradiation, IEX-1 knockout (KO) mice exhibited significantly reduced 30-day survival compared with wild-type (WT) controls (5% vs. 32%). Similarly, after 16 Gy partial body irradiation with 5% bone marrow shielding, survival was markedly decreased in IEX-1 KO mice (18% vs. 46%). IEX-1 expression was enriched in the intestinal mucosa relative to blood and moreover was robustly induced by lysophosphatidic acid receptor (LPAR) stimulation *in vivo* and in intestinal enteroids cultures. Although *in vivo* crypt numbers were unchanged, IEX-1 KO-derived enteroids displayed delayed growth and impaired differentiation. Mechanistically, IEX-1 deficiency altered ERK signaling kinetics, characterized by elevated basal phosphorylation but reduced early post-irradiation activation. These findings establish IEX-1 as a regulator of tissue radiosensitivity, identify LPAR-mediated induction of IEX-1 as a novel regulatory mechanism, and implicate dysregulated pro-survival signaling as a mechanism underlying radiation susceptibility. Supported by NIAID grant U19AI150574 and the Harriet Van Vleet endowment.

Session 9

Lipids, Analysis and Technology

Invited Lecture

Lipids in disguise: Hidden co-passengers in plant protein ingredients with potential impacts on plant-based foods

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The increasing use of plant protein ingredients is a key lever for the transition towards more sustainable diets. Protein isolates and concentrates derived from legume seeds such as pea and faba bean are widely used in plant-based foods, yet their functional and sensory properties are often not optimal. While these ingredients are commonly considered as essentially proteinaceous, their non-protein fractions remain poorly characterized, limiting our understanding of their behaviour in food matrices.
We highlighted that protein isolates and concentrates from pea and faba bean seeds contain substantial amounts of endogenous lipids (up to 12 g per 100 g dry matter), thereby largely exceeding the lipid content of the original seeds (Keuleyan et al., 2023; Koomen et al., 2025). This enrichment reveals a co-accumulation of lipids with proteins during both dry and wet fractionation processes. Remarkably, phospholipids account for more than half of the lipid fraction, suggesting the presence of structured lipid assemblies potentially analogous to oleosome-derived or lipid-protein association structures. In addition, minor lipid constituents such as tocopherols and carotenoids were detected in variable amounts, depending on the plant cultivar, growing conditions and processing route.
Despite this marked antioxidant occurrence, lipid fractions exhibited significant and heterogeneous levels of hydroperoxides, indicating substantial primary oxidation of unsaturated lipids, possibly involving enzymatic pathways (Koomen et al., 2025). In contrast, secondary oxidation products, including volatile compounds typically associated with off-flavours, remained low in dry protein powders. However, the accumulation of lipid hydroperoxides suggests a high risk of rapid secondary oxidation upon hydration and incorporation of these ingredients into food matrices.
Altogether, these findings highlight that endogenous lipids act as “hidden co-passengers” in plant protein ingredients, possibly originating from disrupted lipid droplets and/or persisting as submicron lipid-protein assemblies in protein-rich powders. This has potential implications for both the functional properties (dispersibility, solubility, interfacial behaviour) and the sensory quality of plant-based foods. A better understanding and control of lipid co-accumulation, structural organization and oxidative status, through tailored fractionation processes or by leveraging native antioxidants, appears essential for improving the quality, stability and acceptability of next-generation legume-based protein ingredients.

Keuleyan, E., et al. Pea and lupin protein ingredients: new insights into endogenous lipids and the key effect of high-pressure homogenization on their aqueous suspensions. **2023**, *Food Hydrocoll.*, 141, 108671.
Koomen, J., et al. Faba bean and pea protein ingredients: Endogenous lipid accumulation and lipid oxidative state. **2025**, *Food Res. Int.*, 217, 116884.

Oral Communication

A Decision-support Scheme for Clinical Implementation of Lipidomic Classifiers to Guide Cancer Care

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Several ‘ambient’ mass spectrometry methods leverage alterations in tissue lipidome – correlated to cancer states – to rapidly diagnose cancer types. However, as the complexity of lipidomic classifiers and the number of cancer classes in differential diagnosis grows, novel approaches in decision science are required to reduce the clinically burdensome misclassifications.

Multiclass predictions on large, often imbalanced datasets are facilitated by hierarchical "decision-trees" that reduce the number of classes that must be simultaneously compared, further allow customization of machine learning approaches at each ‘tree’ node. A ‘data-driven’ decision-tree - based on unsupervised spectral similarities- logically regress the decision confidence across tree branches and enables the users to scale back the depth of diagnosis to ‘higher’ nodes to match a desired level of confidence in prediction threshold. This offers several advantages. First, the simultaneous display of classification results and associated probabilities allows clinicians to implicitly incorporate a ‘cost function’ based on expected clinical burden of misclassification. Second, assessing the “flatness” of classifier model’s ‘learning curve’ at each node provides yet another confidence metric to be used in decision-support. This ‘adaptive’ decision-making approach addresses limitations of low confidence in weak predictions and/or classifier models, providing a better assessment of the pathology classification depth warranted for robust patient care.

Using a dataset of 10,225 mass spectra from 1,052 central nervous system (CNS) cancer patients collected with ambient picosecond infrared laser mass spectrometry, we propose lipidomic classifier models (across fatty acid and phospholipid signals) layered onto an adaptive decision-tree alongside a software for the display of confidence-based decision paths for differential diagnosis. Integrating this platform with machine learning approaches has resulted in > 90% sensitivity and specificity for the detection of 24 prognostically important CNS cancer types. This enables integrated molecular and morphometric diagnosis using a single MS instrumental based on analyzing tissue lipids in seconds.

Oral Communication

Integrating tissue proteomics and bioactive lipid analysis of ovine tissue samples identifies functional relations of bioactive lipids

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Bioactive lipids are potent effector molecules that may help to reveal specific information about biological processes. However, analytical challenges and incomplete functional annotations remain as obstacles. We hypothesized that the functional annotation of these lipids may be improved by projecting enriched Gene Ontology (GO) terms derived from proteomics to correlating bioactive lipids. To that aim, thirteen organs and tissues from sheep (Ovis aries) were analyzed using mass spectrometry-based untargeted proteomics and bioactive lipid analysis. In total, 4717 proteins and 168 bioactive lipids, including fatty acids and their conjugates, oxylipins (octadecanoids, eicosanoids, and docosanoids), lyso-phosphatidylcholines (LPCs) and lyso-phosphatidylethanolamines (LPEs), endocannabinoids and related fatty amides, and bile acids and related sterol lipids were determined. Thereof, the correct identification of 114 lipids was confirmed using commercially available analytical standards, whereas the identity of the previously uncharacterized oxylipin 4-hydroxy-eicosatetraenoic acid (4-HETE) was verified through in-house chemical synthesis. Co-expression and clustering analyses validated our hypothesis via independently confirming known bioactive lipid functions and suggested novel functional relations. Amongst other observations, 18-HETE and 19-HETE were related to mitochondrial fatty acid beta-oxidation, 11,12-dihydroxy-eicosatetraenoic acid (DiHETE), 15-deoxy-prostaglandin J2 (15-deoxy-PGJ2) and 5(6)-epoxy-eicosatrienoic acid (EET) were related to chemical synaptic transmission, arachidonoyl ethanolamide phosphate was related to muscle contraction and numerous LPCs and LPEs were related to exosome formation. Tissue levels of polyunsaturated fatty acids (PUFAs), pro-resolving mediators and the newly characterized 4-HETE were related to the cellular protein synthesis and folding machinery. The ladder finding suggested that PUFAs and their derivatives support protein synthesis fidelity in addition to efferocytosis, both essential contributors to the effective resolution of inflammation.

Oral Communication

Drying-induced lipidome remodeling in the two edible halophytes *Salicornia ramosissima* and *Atriplex portulacoides*

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Halophytes are increasingly valued as nutritious and functional foods because of their essential fatty acids (FA) and bioactive compounds. Drying is widely used to improve the shelf-life stability and preserve the quality. However, the effects of post-harvest processing on their lipid composition remain poorly understood. This study evaluated the influence of freeze-drying (−55°C), oven-drying (60°C), and sun-drying (17–27°C) on the lipid profiles of edible halophytes *Salicornia ramosissima* and *Atriplex portulacoides*. An integrated analysis combining FA profiling by GC–MS with untargeted lipidomics by LC–MS/MS was used to assess changes in lipid content, FA and lipidome composition. Total lipid content was not affected by the drying treatments. However, oven- and sun-drying caused significant reductions in esterified FA, particularly FA 18:3 *n-3*, FA 18:2 *n-6* and FA 16:0, compared with freeze-drying. These decreases were more pronounced in sun-dried samples. Since total FA remained comparatively stable, the observed changes suggest lipid hydrolysis rather than extensive oxidative degradation. By LC-MS analysis, 270 lipid species were identified in *S. ramosissima* and 275 in *A. portulacoides*, including glycerophospholipids, glycolipids, acylglycerols, sphingolipids, sterol lipids, and prenol lipids. Drying promoted a decrease in the relative abundance of major membrane phospholipids, especially phosphatidylcholine and phosphatidylethanolamine, together with an increase in their corresponding lysophospholipids, further supporting phospholipid hydrolysis. Relative increases in ceramides, diacylglycerols, and triacylglycerols were also observed, although the magnitude of the response varied between species and drying treatments. Sun-drying produced the most extensive lipidomic remodeling, followed by oven-drying. Overall, post-harvest drying substantially reshapes the halophyte lipidome without altering total lipid content. These findings demonstrate that the selection of drying conditions is critical for preserving lipid quality and controlling the nutritional and functional characteristics of halophyte-derived foods.

Oral Communication

Multidimensional Lipidomics Profiling of *Acinetobacter baumannii* Persister Cells using LC-IMS-MS

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Persister cells form a bacterial subpopulation able of surviving high concentrations of antibiotics that are involved in the recurrence of infections and development of new resistance [1]. *Acinetobacter baumannii* is currently a major public health problem due to its ability to develop resistance to antibiotics [2]. In previous study, we have shown that changes in the lipidome are involved in this ability to better tolerate high concentrations of antibiotics [3]. Lipids of persister cells are uncommon : modification of acyl chain with di-unsaturation or hydroxylation in phospholipids, (Lyso)1-phosphatidyl-2-acyl-glycerol-3-phosphoethanolamine (L)PAGPE, wax ester (WE) with two chain acyl and one diol,. In order to characterize this lipidome in detail, we have developed a multidimensional analytical approach combining liquid chromatography, ion mobility spectrometry, and mass spectrometry (LC-IMS-MS). This strategy is based on the integration of three molecular descriptors: *m/z*, retention time, and collision cross section (CCS). During this work, yielding 115 new CCS values were determined for the persister lipids. Some of these CCS values, to our knowledge, were determined for the first time for uncommon lipid classes, such as MLCLs, (L)PAGPEs, and uncommon WEs. Additionally, differences in CCS values between lipid families with identical acyl chains to be systematic and characteristics, providing additional information. These results show that CCS descriptors can facilitate or confirm annotation. Finally, an in-depth study of uncommon WEs reveals how the experimental conditions or instrumentation influence the preferential formation of adducts, highlighting the complexity of annotating certain classes of bacterial lipids.

[1] Balaban *et al., Nat Rev Microbiol.* 2019 17(7):441-448 https://doi.org/10.1038/s41579-019-0196-3
[2] WHO publishes report: https://www.who.int/publications/i/item/9789240093461
[3] Vergoz *et al., Biochim Biophys Acta Mol Cell Biol Lipids.* 2023 1869(7):15 10.1016/j.bbalip.2024.159539

POSTERS

P1

Resolving the intracellular mechanisms underlying the formation of the suberin

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Plant have evolved several strategies to protect their vasculature from external stress. One of the most effective is the deposition of a suberin layer in the endodermis, the final cellular barrier before the vasculature tissues. Suberin is a complex biopolymer composed of both acyl-chain derivatives and phenolic compounds. Although many enzymes involved in its biosynthesis as well as key transcriptional regulators have been already identified, the intracellular mechanisms underlying suberin assembly remain largely unknow.

Beyond a few existing hypotheses, the processes governing monomer trafficking, the involvement of specific endomembrane compartments, and the export of suberin precursors to the apoplast are still unclear. By combining cutting-edge imaging, genetic, and biochemical approaches, this project aims to establish a mechanistic framework for how plant cells build extracellular hydrophobic barriers. Next to previous publication from our lab, we strongly hypothesize the role of the Extra Vesicular Body in exporting suberin pre-polymerized structure to the apoplast, but also the involvement of the endomembrane system during this process, via the secretory trafficking pathway. We seek to map the successive steps of suberin synthesis, identify the molecular actors involved, and characterize the endomembrane structures and their spatial organization during this process.

P2

Extracellular vesicle lipid remodelling in response to environmental stress: the PFAS-exposed hepatocytes model

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Extracellular vesicles (EVs) are membrane-delimited extracellular particles that mediate intercellular communication and contribute to cellular homeostasis by removing unnecessary or harmful cellular cargo. Their molecular composition reflects both the physio-pathological status of the originating cell and the microenvironmental conditions. Among their biochemical cargo, increasing evidence indicates that EV lipids are not only structural components of the vesicle membrane, but play key roles in EV biogenesis, stability, uptake by recipient cells and downstream signaling.

Over the past years, our group contributed to define the lipid composition of EVs isolated from both in vitro and in vivo disease models (1-3). These studies demonstrated that EVs display a conserved lipid signature, characterized by enrichment in cholesterol, sphingolipids, ether and saturated phospholipids compared with parental cells, indicating selective lipid sorting. Furthermore, this composition is modulated by intracellular/extracellular stimuli and is affected by cellular responses to stress.

In the present study, we investigated EV release as cellular response to an environmental toxicant, with particular focus on EV lipid composition. To this aim, we used human hepatocarcinoma Huh-7 cells exposed to per- and polyfluoroalkyl substances (PFAS) as an in vitro model. PFAS induced ER stress, altered cellular bioenergetics, and changes in the lipid composition of both cells and EVs. Moreover, EVs released from PFAS-exposed cells altered energetic metabolism in recipient hepatocytes, indicating that EVs may transfer functional signals associated with toxicant exposure. Overall, these findings suggest that EV lipid composition may represent a potential biomarker of PFAS exposure and also suggest a potential role for EVs in the systemic interorgan dissemination of the biological effects associated with PFAS exposure.

1. Buratta et al. (2021) Sci Rep 11: 4613
2. Chiaradia et al. (2023) Eur J Cell Biol 102: 151285
3. Forte et al. (2024) Nat Commun 15: 10878

P3 (Competition for the best poster award)

A *pcyt-1* allelic series reveals in vivo consequences of reduced phosphatidylcholine synthesis in *C. elegans*

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Phosphatidylcholine (PC) is the most abundant phospholipid in eukaryotic membranes, and maintaining its synthesis is critical for membrane homeostasis. The rate-limiting enzyme PCYT1A catalyzes a key step in PC synthesis, and hypomorphic variants in PCYT1A cause a range of disorders in humans. To explore how reductions in PC synthesis affect organismal physiology, we generated and characterized a series of mutant alleles, including variants corresponding to disease-causing human mutations and an auxin-inducible degradation (AID) allele, in the C. elegans homolog pcyt-1. We identified a clear allelic hierarchy, ranging from an embryonic lethal variant to a largely benign variant. Along this spectrum, we identified the P154A variant as temperature sensitive while the C211Y variant results in delayed growth and sterility. Supplementation with choline, CDP-choline, or PCs rescued the C211A mutant phenotypes supporting reduced PC synthesis as the underlying cause. Lipidomic analysis revealed that decreased PC synthesis increases long-chain polyunsaturated fatty acids while reducing short-chain saturated fatty acids, and although the PC/PE is unaltered, at elevated temperatures the P154A variant exhibits a decreased PC/PE ratio.

Depletion of PCYT-1 via AID resulted in a lipid profile similar to those of disease-associated variants and caused developmental arrest in larvae and disrupted oogenesis in adults, demonstrating a continuous requirement for PC synthesis throughout the life cycle. This work establishes a functional pcyt-1 allelic series and demonstrates that reduced PC synthesis triggers compensatory membrane remodeling while revealing a particular sensitivity of the germline to PC shortage.

P4

Functional organisation of the mitochondrial tafazzin transacylation network

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Cardiolipin is the signature phospholipid of the mitochondrial inner membrane and undergoes continuous acyl chain exchange catalysed by the transacylase tafazzin. Defective tafazzin function causes Barth syndrome, highlighting the importance of cardiolipin remodelling for mitochondrial function. Traditionally, tafazzin has been viewed as a constitutive remodelling enzyme that establishes the characteristic molecular composition of cardiolipin. The same enzymatic transacylation network also supports a second distinct physiological function by facilitating the replacement of oxidatively damaged phospholipids during oxidative stress. How these apparently separate functions arise from a common enzymatic mechanism remains poorly understood.

Here, we combine mathematical Markov chain modelling with experimental analyses to investigate mitochondrial phospholipid remodelling from a systems perspective. Rather than considering individual transacylation reactions in isolation, our model describes cardiolipin remodelling as a highly interconnected network of phospholipid interconversions that links cardiolipins, monolyso-cardiolipins and other mitochondrial phospholipids. This framework is coupled with functional measurements of oxidative stress responses.

Our results support a model in which remodelling and oxidative repair represent two physiological modes of the same mitochondrial phospholipid network, with different physiological conditions engaging distinct regions of the transacylation network. Tafazzin loss reshapes network organisation and dynamics while leaving the phospholipid composition largely unchanged. Without repair capacity, tafazzin-deficient cells experience chronically elevated oxidative stress and adapt accordingly. Unexpectedly, this adaptation renders them more resistant to ferroptosis: rather than reflecting a change in membrane repair, the protection arises through lipid peroxidation-independent mechanisms that counteract oxidative stress.

P5 (Competition for the best poster award)

Dissecting the roles of ether lipid enzymes AGMO and PEDS1 in macrophage immune function

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Ether phospholipids, characterized by an ether bond at the *sn*-1 position, include plasmanyl and plasmalogen species. Beyond their role as membrane components regulating organization, signaling, and protection against oxidative stress, they are closely linked to immune regulation, exemplified by lipid mediators such as platelet-activating factor. In the intestine, lipid metabolism is critical for epithelial barrier integrity and immune homeostasis, while its dysregulation contributes to inflammatory bowel disease (IBD). However, the role of ether lipid remodeling enzymes in intestinal immune regulation remains poorly understood.

Alkylglycerol monooxygenase (AGMO) and plasmanylethanolamine desaturase (PEDS1) are key enzymes in ether lipid metabolism with distinct intestinal activity patterns in mice, suggesting specialized roles in dietary ether lipid processing and immune-related pathways. To investigate their function in immune cells, we generated stable *Agmo* knockout (KO), *Peds1* KO, and *Agmo*/*Peds1* double KO models in the murine RAW264.7 cell line using CRISPR/Cas9 technology. Clones were validated by sequencing and confirmed functionally by HPLC-based activity measurements. Loss of AGMO and/or PEDS1 altered macrophage polarization, assessed by flow cytometry and RT-qPCR, indicating that ether lipid remodeling can influence macrophage states.

To evaluate human relevance, we analyzed AGMO and PEDS1 expression and activity in intestinal biopsies from healthy donors and IBD patients. AGMO showed highest expression and activity in the ileum, whereas PEDS1 was predominantly expressed in distal colonic segments. Notably, colonic AGMO and PEDS1 activity was modulated in ulcerative colitis patients, independent of transcriptional regulation.

P6

Identification of bacterial cell envelope factors that influence tuberculosis disease and treatment outcome

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M. tuberculosis (Mtb) factors, including cell envelope lipids and lipoglycans, play a central role in host-pathogen interactions in both cellular and animal models. They are therefore assumed to be key determinants of clinical outcomes, influencing tuberculosis (TB) disease progression and response to treatment in humans. However, this has remained insufficiently explored. Within the SMA-TB EU-funded project (GA847762), a Phase 2b, double-blind, placebo-controlled clinical trial (NCT04575519) evaluating ibuprofen and acetylsalicylic acid as anti-inflammatory adjunctive therapies to standard-of-care treatment for pulmonary TB, we collected 155 baseline Mtb clinical isolates in Georgia and South Africa. The strains were cultured on solid medium, and lipoglycans and lipids were quantified using a combination of dot-blot assays and lipidomic analyses to investigate the relationship between bacterial component variability and patient clinical data. The inflammatory potential of strains was also measured by their ability to induce NF-kB activation in the human macrophage THP-1 cell line. Strikingly, we observed that the production of several lipids, including phthiocerol dimycocerosates (DIM), triacylglycerols (TAG), sulfoglycolipids (SGL), and phosphatidylinositol mannosides (PIM), correlated with baseline disease severity scores and with specific blood parameters. Moreover, among patients in the placebo arm, those showing poor TBscore evolution at week 24 were infected with strains producing higher levels of lipoarabinomannan (LAM). Similarly, patients in the placebo arm who exhibited unfavourable chest X-ray evolution at week 8 were infected with strains displaying a higher inflammatory potential. In both cases, these associations were abolished in the ibuprofen and acetylsalicylic acid arms, highlighting the impact of adjunctive anti-inflammatory therapy. Altogether, our findings strongly suggest that bacterial factors, along with the intrinsic inflammatory potential of Mtb strains, influence human TB disease progression and treatment outcomes. Such pathogen-derived biomarkers could support the development of urgently needed patient-stratification strategies to improve TB clinical management and therapeutic success.

P7

PNPLA6 establishes an intercellular phosphatidylcholine recycling circuit across the retinal pigment epithelium–photoreceptor unit

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Phosphatidylcholine (PC) turnover is essential for retinal membrane homeostasis, but how endogenous choline is recycled within retinal pigment epithelial (RPE) cells and supplied to neighboring photoreceptors remains unclear. We investigated how PNPLA6-mediated phospholipid catabolism is coupled to PC resynthesis and intercellular choline transfer.

Lipidomics of the RPE layer isolated from ocular-selective Pnpla6-deficient mice showed accumulation of PC and lysophosphatidylcholine (LPC) with reduced choline, demonstrating that PNPLA6 deficiency impedes the catabolism of PC/LPC to choline in vivo. In cultured RPE cells, glycerophosphocholine generated downstream of PNPLA6 was converted to choline mainly by glycerophosphodiester phosphodiesterase 2, with additional contributions of glycerophosphodiesterase 1 and glycerophosphodiesterase 5. Silencing of choline kinase α, PCYT1A, or choline phosphotransferase 1 suppressed cell proliferation despite preserved or increased intracellular choline, indicating that the metabolic flux of PNPLA6-derived choline into PC resynthesis through the Kennedy pathway, rather than choline abundance alone, is required for RPE homeostasis. These metabolic and functional defects were reproduced in primary human RPE cells.

Experiments using conditioned medium from RPE cells further showed that PNPLA6-dependent release of choline from RPE cells supports the survival and growth of 661W photoreceptor cells. Silencing of the choline transporter SLC44A1 impaired this response, and d9-choline tracing demonstrated rapid incorporation of extracellular choline into photoreceptor PC. Inhibition of choline kinase prevented the choline-mediated rescue, confirming that imported choline must enter the Kennedy pathway to support the survival and growth of photoreceptor cells.

Together, we identify a PNPLA6–GDE–Kennedy pathway that couples phospholipid degradation to PC regeneration in RPE cells and extends this metabolic flux to photoreceptors through SLC44A1-dependent choline transport. Our data demonstrate that PNPLA6 links phospholipid turnover in RPE cells to choline-dependent PC synthesis in photoreceptors.

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P8

Deuteration-induced changes in acyl chain compositions and their impact on membrane organization - A neutron reflectivity study

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Deuterated lipids are powerful tools for investigating the structure of biological membranes using neutron scattering techniques. While deuteration provides exceptional contrast for the study of membrane organization, the physiological effects of deuterium incorporation into cellular lipids remain poorly understood. Previous studies conducted in our laboratory demonstrated that adaptation of the yeast '*Pichia pastoris*' to growth media containing D_2O resulted in substantial changes in lipid compositions. In particular, pronounced changes in acyl chain distribution were observed across multiple phospholipid classes, suggesting that deuteration influences lipid biosynthesis and their remodeling pathways. Neutron scattering analyses further revealed that such alterations in the fatty acid abundances had a significant impact on the structural properties of biomimetic membrane bilayers reconstructed from the extracted lipids. Collectively, these findings suggest that deuterium incorporation may affect metabolic pathways and gene regulatory networks involved in lipid homeostasis.

Building upon these observations, the objective of the present project is to investigate the impact of deuteration on lipid metabolism and membrane properties in *Saccharomyces cerevisiae*. Specifically, the project aims to determine whether the lipidomic changes previously observed in *P. pastoris* are conserved in *S. cerevisiae* and to examine how these changes vary under different environmental and physiological conditions. Given that temperature is a key regulator of membrane lipid composition and cellular adaptation, the effects of deuteration will be evaluated across a range of growth temperatures and at distinct stages of the yeast growth cycle. This approach will enable us to identify potential links between isotopic adaptation, cellular metabolism, and membrane homeostasis. In conclusion, all these findings will improve our understanding of how microbial organisms adapt to growth under deuterated environments and thereby provide critical insights for the interpretation of neutron scattering experiments that rely on biologically produced deuterated lipids.

P9

Long-chain acyl-CoA synthetase 4-mediated lipid remodeling promotes the progression of pulmonary and hepatic fibrosis

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Organ fibrosis is a highly lethal condition with limited therapeutic options, partly because the molecular mechanisms are incompletely understood. Although dysregulated wound repair and oxidative stress are known to contribute to fibrotic progression, the upstream regulators controlling this process remain unclear. Long-chain acyl-CoA synthetase (ACSL) 4 is an acyl-CoA synthetase that prefers highly unsaturated fatty acids (HUFAs) as a good substrate and plays an important role in maintaining HUFA-containing phospholipids, thereby regulating sensitivity to oxidative stress-induced ferroptosis. In this study, we investigated the role of ACSL4 in pulmonary and hepatic fibrosis.

First, single-cell RNA sequencing analysis revealed that expression levels of ACSL4 were elevated in patients with pulmonary fibrosis. Consistent with these findings, in bleomycin-induced pulmonary fibrosis mouse model, wild-type (WT) mice exhibited increased ACSL4 expression, marked lipid peroxidation, and progressive fibrosis compared to ACSL4 knockout (KO) mice. ACSL4 KO mice were protected from fibrotic remodeling and maintained higher oxygen saturation. Lipidomics demonstrated reduced accumulation of lipid peroxides in ACSL4 KO mice lungs. Furthermore, pulmonary fibrosis was significantly attenuated by pharmacological inhibition of ferroptosis during transitional and early progressive stages of fibrosis.

Next, we examined the role of ACSL4 in hepatic fibrosis using a choline-deficient, L-amino acid-defined, high-fat diet (CDAHFD) mouse model. Similar to our findings in pulmonary fibrosis model, WT mice exhibited increased collagen accumulation, whereas hepatic fibrosis was significantly attenuated in ACSL4 KO mice.

Collectively, these results demonstrated that ACSL4-mediated lipid remodeling promotes fibrotic progression by maintaining HUFA-containing phospholipids that increase ferroptosis susceptibility across multiple organs. Our findings suggest that targeting ACSL4 during tissue repair phase may represent a promising therapeutic strategy for organ fibrosis.

P10 (Competition for the best poster award)

Quantitative contribution of oxidative damage to cardiolipin homeostasis

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The transacylase tafazzin is the key enzyme for mitochondrial cardiolipin (CL) side chain exchange, so-called remodeling, to generate mature CL. The importance of CL remodeling is shown in the Barth syndrome (BTHS), caused by impaired tafazzin function. BTHS is characterized by a dysregulated CL / monolyso-cardiolipin (MLCL) ratio and an accumulation of reactive oxygen species (ROS) and lipid peroxidation. This suggests that *de novo* biosynthesis without remodeling is insufficient to maintain healthy CL homeostasis but also raises the question if tafazzin contributes to a repair mechanism by actively replacing oxidized acyl side chains after lipid peroxidation.

To unriddle a potential repair mechanism by tafazzin, the contributions of biosynthesis, remodeling and repair to CL homeostasis have to be quantified. Therefore, we established a mass spectrometric ^{13}C-labeling assay using labeled metabolic precursors of cardiolipins, namely glucose and fatty acids, in healthy and tafazzin deficient HEK cells. Tracing *de novo* CL biosynthesis by ^{13}C-glucose incorporation showed us cardiolipin species-specific exchange rates indicating a faster turnover of saturated cardiolipins, when compared to more unsaturated species. The use of different ^{13}C-labeled fatty acids as flux tracers provided information about side chain incorporation and exchange rates in different lipid environments. Results indicated a faster incorporation of palmitic acid into cardiolipins, when compared to linoleic acid. The labeling assay was further applied to decreased and increased oxidative stress environments. We observed reduced MLCL formation accompanied by reduced ROS in tafazzin deficient cells. Whereas additional mitochondrial ROS showed distinct consequences for CLs and MLCLs with and without intact tafazzin function. Our results strongly emphasize a tafazzin-driven shift of CL transacylation activity triggered by mitochondrial ROS, suggesting targeted repair of oxidized CLs by tafazzin.

P11

Inducible phosphatidylserine decarboxylase knockout reveals its essential role in *Plasmodium falciparum* survival and the absence of serine decarboxylase activity in *the parasite.*

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Plasmodium falciparum, the malaria parasite, grows and multiplies within human erythrocytes. Phospholipids (PL) are critical for parasite development and are synthesized *de novo* through multiple coexisting and interconnected metabolic pathways. However, the regulation of these pathways and their respective contributions during the parasite's intraerythrocytic stages remain poorly understood. Our goal is therefore to identify bottlenecks and compensatory mechanisms that could reveal new therapeutic targets. In this study, we focus on phosphatidylethanolamine (PE) synthesis, the second most abundant PL. We used deuterated or radiolabeled precursors to monitor and quantify their incorporation into metabolic intermediates and final PL products by metabolomics. In *Plasmodium falciparum*, PE can be synthesized either from ethanolamine via the Kennedy pathway or from phosphatidylserine (PS) through decarboxylation catalyzed by phosphatidylserine decarboxylase (PSD). Previous studies reported that, in *P. falciparum*, ethanolamine feeding the Kennedy pathway can be produced through the serine decarboxylation by serine decarboxylase (SD), as in plants (Rontein *et al.*, JBC, 2001). However, no SD-encoding gene encoding has been identified in the *P. falciparum* genome to date, raising questions about the existence of SD activity in the malaria parasite. Finally, ethanolamine can also be provided indirectly through PSD-mediated decarboxylation of PS to PE, followed by a base-exchange reaction that regenerates PS and releases ethanolamine. Since ethanolamine potentially generated by either SD or the combined PSD/base-exchange steps cannot be distinguished by metabolic labeling, we generated conditional knockout of the *Pf*PSD. Indeed, in absence of this latter enzyme, the only way to produce ethanolamine and PE from serine will be through the action of SD. We demonstrate that deletion of *Pf*PSD is lethal to the parasite, completely abolishes PE synthesis from serine, and results in an increase of PS levels. These findings revealed that *P. falciparum* lacks SD activity and identify PSD as an essential enzyme for parasite survival.

P12 (Competition for the best poster award)

Advancing Membrane-Mimetic Models: Development of Physiologically Relevant Bicelles for Studying Bacterial Membrane Proteins

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Membrane proteins, despite being major drug targets, remain understudied due to difficulties in purifying them from their native lipid environments. These proteins often require specific protein-lipid interactions to maintain their native conformation and activity. Standard isolation approaches often use detergents that form homogeneous micelles, failing to accurately mimic the structural and compositional complexity of the protein's native membrane environment. Bicelles, defined as disc-shaped nanoparticles with a lipid bilayer core and a detergent rim, may serve as a better membrane mimic than micelles due to their high compositional tunability. Yet, most bicelles developed and characterized to date still do not reflect the compositional diversity of many native bacterial membranes.

To bridge this gap, we investigated the formation of bicelles composed of detergent CHAPS or DH6PC, and lipids native to the inner membrane of *Escherichia coli*: phosphatidylethanolamine and phosphatidylglycerol with varying degrees of acyl chain saturation. We evaluated two preparation methods: solubilizing preformed liposomes versus simultaneous mixing of lipids and detergents. Dynamic light scattering (DLS) and laurdan General Polarization (GP) were used to assess the size of formed assemblies and membrane lipid packing within them, respectively. Cryo-TEM imaging revealed that bicelles with biologically relevant lipid compositions can be successfully formed; however, their formation is highly dependent on specific lipid-detergent ratios and the detergent used, as DH6PC seems to form bicelles from liposomes more effectively than CHAPS. Moreover, the preparation method impacts the bicelle quality, as liposome solubilization yields significantly more homogeneous and reproducible samples than simultaneous mixing. These findings expand the range of available physiologically relevant membrane mimetics for studying membrane proteins. This will particularly benefit research focusing on proteins that preferentially associate with specific local lipid environments.

P13 (Competition for the best poster award)

Exogenous cardiolipin species differentially modulate mitochondrial function and morphology in cardiolipin-deficient human cells

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Cardiolipin (CL) is a mitochondria-enriched phospholipid essential for inner membrane organisation and oxidative phosphorylation. Disruption of its synthesis or remodelling is associated with inherited mitochondrial disorders, including disease caused by pathogenic variants in CRLS1, highlighting cardiolipin homeostasis as a potential therapeutic target. However, whether exogenous cardiolipin can modify disease-associated phenotypes remains poorly understood. We therefore investigated whether supplementation with defined molecular species could improve mitochondrial function and morphology in human cells with impaired cardiolipin synthesis. Wild-type CAL51 and CRLS1 knockout cells were treated with 10 µM tetrapalmitoyl cardiolipin ($CL(16:0)_4$), tetraoleoyl cardiolipin ($CL(18:1)_4$) or tetralinoleoyl cardiolipin ($CL(18:2)_4$). Cellular viability, ATP abundance and mitochondrial membrane potential were assessed using complementary functional assays, while network morphology was examined by fluorescence microscopy and quantitative image analysis. Treatment produced species-dependent effects on mitochondrial physiology. Cell viability improved in both wild-type and CRLS1 knockout cells. In the knockout, $CL(16:0)_4$ increased ATP levels, while membrane potential improved following supplementation, with $CL(18:2)_4$ producing the strongest response. Quantitative imaging further identified changes in mitochondrial network morphology that varied by molecular species and cellular background. Together, these findings demonstrate that exogenous cardiolipin can modulate mitochondrial function and organisation when its synthesis is impaired. The distinct responses to individual molecular species highlight the importance of acyl-chain composition and support further investigation of cardiolipin supplementation as a potential therapeutic strategy

P14 (Competition for the best poster award)

Combinatorial modeling deciphers fatty acyl patterns underlying lipid profiles

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Glycerolipids exhibit extensive structural diversity that is largely driven by the combinatorial incorporation of different fatty acyl side chains. This diversity is particularly pronounced in lipids containing multiple acyl chains, such as triacylglycerols (TAGs) and cardiolipins (CLs), with numerous isomeric and isobaric molecular species. Resolving these side-chain compositions is essential for understanding lipid metabolism and enzymatic specificity, but remains challenging because conventional lipidomics approaches often provide poor fragment spectra coverage and convoluted MS/MS spectra, limiting the structural resolution to sum compositions (i.e. CL 72:8). Here, we developed Fatty Acylizer, a combinatorial modeling framework that uses constrained optimization to infer fatty acyl compositions from measured lipid profiles. We validated the approach across three CL datasets covering different cell culture models and tissues from model organisms. The resulting acyl-chain profiles revealed substantial differences in the relative contributions of distinct fatty acyl chains within the same sum-composition species across biological systems. Importantly, these differences could be explained by the underlying fatty acyl availability without requiring additional tissue- or organism-specific enzymatic specificity. Together, indicating that tafazzin, the central CL remodeling enzyme, primarily redistributes available fatty acyl chains rather than determining the CL acyl-chain specificity. To facilitate the analysis of lipidomics datasets, we provide Fatty Acylizer both as a user-friendly web server (http://fatty-acylizer.lipid.at) and an open-source Python package implementing the underlying mathematical model and fitting procedures. Because Fatty Acylizer uses no spectral information, legacy datasets and high-throughput experiments without available MS/MS can be enhanced with biologically informative fatty acyl profiles. Moreover, this MS/MS independence positions Fatty Acylizer as an optimal orthogonal quality-control layer for automated lipid annotations.

P15

Characterization of Membrane Fluidity of Cells in Contact with Implant Materials in the Absence or Presence of Mechanical Stimulation

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The success rate of dental implants is overwhelmingly high, yet in 10 % of cases, implant failure occurs. Successful osseointegration to prevent such failure is dependent on many aspects: the biomaterial itself, surface topography and roughness, the quality and quantity of the host bone, and absence of external complications after integration such as contamination, occlusal overload, or peri-implantitis. Therefore, new innovative alloys together with new testing protocols for dental implants are important to improve the rate of success of this type of surgery with is costly and rather invasive.
Cellular membranes are the first contact frontier between the implant substrate and oral sphere cells. Therefore, the contact surface as well as the wear nanoparticles or the corrosion ions released by the substrate in the medium may exert a direct influence on the membranes and on their physico-chemical characteristic. We propose thus measurement of membrane fluidity as an early sensor of cellular reaction in response to material or to mechanical stress. In this work we describe a first dataset of membrane testing protocols to reveal the effect of Ti or Ti-alloy substrates on primary murine or canine chondrocyte membranes. To mimic the mechanical stress exerted on cells upon mastication, cells were grown under static and dynamic conditions in homemade bioreactors [Hannoun, 2020]. After 15 days of growth, cells were labelled with a fluorophore sensitive to membrane dynamics, Dioll, and harvested. Fluorescence was analyzed using fluorescence spectroscopy and confocal microscopy, in parallel with cell viability and rheological characterization of the formed tissue. Overall, data collected from the bioreactors showed similar GP trends for both confocal and spectroscopic measurements and suggest that mechanical stimulation increases cell membrane rigidity.

P16

Metabolic remodeling mediated by phytosterols and brassinosteroids and biotic interactions in barley

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Phytosterols are essential components in membrane structure and dynamics, and play several functions in plant developmental processes. In addition, phytosterols are precursors of brassinosteroids. These polyhydroxylated sterol derivatives act in cell division and elongation, and in *plant signaling networks involved in biotic and abiotic stress responses.* Here, a series of semi-dwarf barley lines displaying mutations in enzymes or signalling components of the sterol/brassinosteroid pathway are studied for their biochemical and biological features, and particularly their lipid profiles. Of note, the *dim1* (*diminuto/dwarf1*) barley lines revealed a severely altered sterol profile due to the a loss-of-function mutation in the gene encoding a sterol-24-reductase. Furthermore, we found modifications in the lipidome of these plants compared to the wild-type counterpart. Surprisingly, these modifications were closely similar to those of other barley steroid mutants considered here, eventhough their genetic lesions causing altered steroid/sterol profiles were in distinct genes. We also found a similar transcriptome reprogrammation in these mutants, including an enhanced expression of some defense-related genes and genes involved in lipid homeostasis. We finally explored the tolerance of semi-dwarf barley lines impaired in sterol/steroid pathways, to fungal pathogens such as *Pyrenophora teres f. teres*, a major threat to barley considered as a crop. Ultimately, this project will reveal components of a signaling pathway implied in defense against pathogens in the particular mutant backgrounds.

P17

9-PAHPA Redirects Hepatic Fatty Acid Metabolism towards Triacylglycerol Synthesis in Lean Mice

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Fatty acid esters of hydroxy fatty acids (FAHFAs) are a group of bioactive endogenous lipids with diverse metabolic functions. Some FAHFAs have been shown to exert antidiabetic and anti-inflammatory effects. The present study investigates the short-term metabolic effects of 9-palmitic acid hydroxy palmitic acid (9-PAHPA) in lean mice, with particular emphasis on lipid metabolism and fatty acid synthesis.

Adult male C57BL/6J mice received 9-PAHPA or vehicle orally for three consecutive days. Lipid synthesis and turnover were assessed using D_2O labelling. Plasma and liver samples were collected for lipidomic and isotopic analyses.

Short-term administration of 9-PAHPA markedly altered hepatic lipid metabolism. Treatment was associated with enhanced de novo synthesis of several fatty acids, particularly myristic acid (14:0), myristoleic acid (14:1), and palmitic acid (16:0), in the liver. Increased incorporation of newly synthesized fatty acids was also detected in circulating lipids. Moreover, the hepatic accumulation of triacylglycerol species containing these fatty acyl chains indicated that 9-PAHPA promoted triacylglycerol synthesis and/or remodelling. In contrast, 9-PAHPA treatment was accompanied by lower concentrations of medium- and long-chain acylcarnitines, particularly species ranging from C10 to C18, in both liver and plasma.

In conclusion, short-term exposure to a high dose of 9-PAHPA substantially modified fatty acid and complex lipid metabolism in lean mice. The increased synthesis of selected fatty acids together with their incorporation into triacylglycerols and the concomitant reduction in medium- and long-chain acylcarnitines suggests that 9-PAHPA redirects hepatic fatty acid flux towards lipid synthesis and storage while potentially altering mitochondrial fatty acid utilisation. These findings provide further evidence that 9-PAHPA acts as a metabolically active lipid capable of modulating lipid handling in vivo.

P18

In vitro digestion and bioaccumulation of food-born processing contaminant 3-MCPD diesters in rats

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3-Monochloropropane-1,2-diol (3-MCPD) is a process-born contaminant formed during edible oil refining. While toxicity of free 3-MCPD is well established and strictly regulated, the digestive fate, absorption and tissue distribution of its fatty acid diesters remains insufficiently characterised. This study examined the hydrolysis and bioaccumulation of 3-MCPD diesters through in vitro gastro-intestinal digestion, their absorption/transport in lymph and plasma as weel as their bioaccumulation in rat tissues. We showed that both, gastric and pancreatic lipases can hydrolyze 3-MCPD diesters, reaching 4.34 ± 0.24% and 15.38 ± 0.98%, respectively. This resulted in a cumulative absorption in lymph of 17.4 ± 1.5% over 24 hours, with approximately 65% occurring within the first 6 hours postprandially (11.3 ± 2.1%). Moreover, after four weeks of dietary exposure, 3-MCPD diesters predominantly accumulated in the liver, gonads, kidneys, brain and heart, with higher tissue concentrations observed in females, particularly in liver and ovaries. Although the digestive hydrolysis of 3-MCPD esters remains limited, their absorption nevertheless leads to a selective storage in tissues, contributing to sex-related differences in toxicity.

P19

APOB48 Knock-Down in Caco2 cells to mimic familial hypobetalipoproteinemia does not impact lipid secretion

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Familial Hypobetalipoproteinemia (FHBL) is a rare genetic disorder characterized by lipid malabsorption and decreased plasma levels of fat-soluble vitamins. Impaired lipoprotein assembly constitutes the hallmark defect in FHBL, which is caused by loss-of-function mutations in the *APOB* gene. Numerous attempts have been made to model APOB dysfunction in cellular and murine models. Nevertheless, establishing a reliable model has proven more challenging than expected due to the complexity of the APOB-lipoprotein assembly process. In this work, we modeled FHBL by deleting a fragment of the *APOB* gene in an intestinal cell model, in order to assess the impact of APOB disruption on enterocyte lipid metabolism. To this aim, we used the CRISPR-CAS9 method in the intestinal Caco-2/TC7 cell line. The selected APOB knockdown (APOBKD) clone exhibited markedly reduced *APOB* mRNA (1.01±0.03 in wild-type cells vs 0.19±0.01 in APOBKD clone; $p<0.0001$) and protein expressions (-45%) compared to the control cell line. After a lipid load, the APOBKD cells also exhibited an intracellular lipid accumulation compared to control cells, as expected. Surprisingly, unlike FHBL patients, the APOBKD clone did not show a reduction in vitamin E and carotenoids secretion, and even showed enhanced triglycerides (TG) secretion compared to the control cell line. Subsequent, qPCR analyses revealed increased APOA-IV (1,01±0,08 in Caco-2/TC7 wild-type cells vs 15,32± 1,2 in APOBKD, $p<0,0001$) and APOE (0,84±0,09 vs 1,91±0,15, $p<0,001$) expressions in the APOBKD clone compared to control cell line. Our findings suggest that impaired APOB expression in APOBKD cultured intestinal cells triggers upregulation of *APOA-IV and APOE* genes, thus activating a compensatory mechanism to maintain lipid absorption. These insights may pave the way for novel pharmacological approached to treat FHBL disorders by activating compensatory pathways.

P20

Improving nutritional quality and stability of crude palm oil produced by African smallholders : Breeding for palms with enhanced traits regarding unsaturated fatty acids, carotenes and tocochromanols contents

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The oil palm (*Elaeis guineensis* Jacq.) is the predominant oil crop in the world. Crude Palm Oil (CPO) is extracted from the mesocarp of the ripe fruit. Unlike industrial CPO (70% of total production) whose ecological footprint is often highlighted and is typically destined to refining, smallholder-produced CPO (30%) is generated by subsistence farming on small surfaces. It is a key component of food systems across West and Central Africa, and an essential ingredient of many local recipes. As CPO is the richest natural source of carotenoids and is also rich in tocochromanols, it could play a critical role in combating vitamin A deficiency in sub-Saharan Africa. However, it is often of poor quality and stability, due to high acidity and peroxides contents inherent to extraction methods used by smallholders, hence causing rancidity of the oil.

The present work aimed at identifying palm genotypes producing CPO with improved nutritional quality, increased stability and that are more suitable for cultivation by African smallholder. For this purpose, numerous genotypes of oil palm planted in research stations in Ghana and Cameroon were phenotyped with regards to carotenes (pro-vitamin A), tocochromanol (vitamin E) and fatty acids compositions.

Our results showed an important intraspecific variability for these traits. Unlike similar studies on the same topic, screening of included wild palm accessions allowed us to identify specific outliers with altered carotene and tocochromanol contents (Gutbrod *et al*., 2024). The introduction of genes from identified interesting traits into elite breeding lines represents a potent strategy to enhance oil stability, hence allowing the development of a value-added CPO. Ongoing GWAS studies to identify molecular markers (SNPs) are quite promising and will enable marker-assisted selection for future improvement programs.

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P21

Oral Intake of Linseed Oil That Suppresses Obesity-Related Skin Barrier Dysfunction Is Associated with Suppression of Brown Adipose Tissue Whitening

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Objectives: We previously reported that oral intake of linseed oil, rich in omega-3 fatty acids, suppresses skin barrier dysfunction in obese mice (Horie et al., 2024). Linseed oil also reduced brown adipose tissue (BAT) weight. Obesity promotes BAT whitening, leading to impaired thermogenesis and reduced energy expenditure. However, little is known about the relationship among obesity, skin, and BAT. This study aimed to examine whether linseed oil, which suppresses skin barrier dysfunction in obese mice, is associated with alterations in BAT morphology and fatty acid composition.

Methods: Obese mice (TSOD) were divided into Control and Omega groups. The Control group received water, and the Omega group received linseed oil orally for 8 weeks. Body weight was measured weekly. After intervention, transepidermal water loss (TEWL) was measured, and skin and BAT were collected. BAT weight, histological analyses using H&E staining, and fatty acid composition in skin and BAT were evaluated. This study was approved by the Yokohama City University Animal Experiment Committee (F-A-23-051).

Results: Body weight did not differ between groups. Compared with Controls, the Omega group showed significantly lower BAT weight and reduced lipid droplets in BAT. Fatty acid analysis revealed increased alpha-linolenic acid (ALA) and decreased palmitic acid levels in both skin and BAT in the Omega group. ALA levels in BAT were positively correlated with those in skin. For TEWL, ALA levels in skin and BAT were negatively correlated, whereas palmitic acid levels were positively correlated.

Conclusions: These findings suggest that omega-3-rich linseed oil alters fatty acid composition in skin and BAT, with increased ALA and decreased palmitic acid levels, and is associated with suppression of obesity-induced skin barrier dysfunction and BAT whitening. These changes may contribute to improved systemic metabolism, warranting further investigation of energy metabolism-related factors.

P22

Lysophosphatidylcholine induces human chorionic gonadotropin production and impairs insulin sensitivity in gestational diabetes mellitus

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Gestational diabetes mellitus (GDM) is one of the most common metabolic disorders during pregnancy and is characterized by insulin resistance and dysregulated lipid metabolism. Increasing evidence suggests that abnormal lipid profiles contribute to placental dysfunction and metabolic imbalance in GDM. Among these lipids, lysophosphatidylcholine (LPC) has been reported to be elevated in patients with GDM and is associated with mitochondrial impairment and trophoblast dysfunction. Trophoblasts are the major source of human chorionic gonadotropin (hCG), a placental hormone involved in maternal metabolic adaptation during pregnancy and potentially associated with pregnancy-related insulin resistance. However, whether elevated LPC contributes to insulin resistance by modulating trophoblast-derived hCG under GDM conditions remains unclear.

To mimic the GDM environment, JEG-3 trophoblast cells were cultured under high-glucose conditions (4.5 g/L glucose) and treated with LPC. hCG secretion was measured by ELISA, and protein expression was analyzed by western blotting. To further investigate the metabolic effects of LPC-induced trophoblast signaling, conditioned culture media from LPC-treated JEG-3 cells were applied to HepG2 cells, followed by the evaluation of insulin signaling-related proteins.

LPC treatment significantly increased hCG expression and secretion in JEG-3 trophoblast cells in a dose-dependent manner. Furthermore, conditioned media derived from LPC-treated trophoblast cells altered insulin signaling in HepG2 cells, including changes in p-IRS1, p-PI3K, and p-Akt expression, indicating reduced insulin sensitivity.

Overall, these findings suggest that LPC-mediated trophoblast dysfunction may contribute to impaired glucose homeostasis during pregnancy through dysregulated hCG production and support a potential lipid–hormone axis involved in GDM pathogenesis.

P23

Analysis of phytosterols in PEGylated and resveratrol-loaded liposomes.

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Phytosterols are bioactive lipids highly susceptible to oxidation, particularly under thermal stress. Encapsulation in PEGylated liposomes may improve their stability, while resveratrol (RES), a natural antioxidant, could further reduce oxidative degradation. Stigmasterol esters containing oxidation-prone fatty acid residues also require characterization to better understand their thermal behavior. Dipalmitoylphosphatidylcholine (DPPC) lipids were encapsulated in PEGylated liposomes containing free stigmasterol (ST), stigmasteryl myristate (ME), or stigmasteryl oleate (OE), with or without RES. Liposomal systems were characterized using transmission electron microscopy (TEM), zeta potential, and hydrodynamic diameter analyses. Samples were heated at 60 °C and 180 °C for 8 h to evaluate structural stability, degradation of stigmasterol and fatty acid residues, and oxyphytosterol (SOP) formation. The liposomes fulfilled their carrier function and remained structurally stable at 60 °C, whereas major structural alterations were observed at 180 °C. ST liposomes exhibited the smallest particle size, while ME and OE systems showed larger hydrodynamic diameters. Thermal degradation depended on sterol structure and temperature. After heating at 60 °C, stigmasterol degradation ranged from 3.5% in ST liposomes to 4.3% in ME and 6.5% in OE systems. At 180 °C, the lowest degradation was observed for OE (7.3%), compared with 13.4% for ST and 10.1% for ME. RES supplementation improved thermal and oxidative stability, reducing stigmasterol degradation and limiting SOP formation. SOP content after heating at 60 °C reached 23.2 mg/g in ST liposomes, while lower values were observed for ME (6.3 mg/g) and OE (6.4 mg/g). Heating at 180 °C markedly increased SOP formation, particularly in OE liposomes (88.7 mg/g). PEGylated liposomes effectively stabilized stigmasterol and its esters under moderate thermal conditions, while RES further enhanced oxidative stability. These findings highlight the potential of RES-enriched phytosterol liposomes as thermally stable delivery systems and emphasize the importance of controlling high-temperature processing conditions.

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P24

Searching for FAHFA transporters in blood

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Fatty acid esters of hydroxyl fatty acids (FAHFAs) are a class of bioactive lipids with anti-diabetic and anti-inflammatory effects. They elicit their effects in many sites within the organism, such as adipocytes, macrophages, intestine and pancreatic islets. FAHFAs can be obtained through diet or synthesised in adipose tissue, kidney and liver. However, it is unclear how synthesised or dietary FAHFAs are transported through the circulatory system. We have investigated candidate vehicles capable of carrying hydrophobic lipids in blood to see if they can transport FAHFAs. Microscale thermophoresis was used to characterise the interaction between serum albumin protein and 9-PAHSA – the dissociation constant of the interaction was too high to be physiologically relevant. We turned to serum particles as the next transporter candidate. Serum lipoproteins and exosomes were each purified using different density ultracentrifugation-based techniques and their protein and lipid content was characterised using proteomics and lipidomics. Proteomics confirmed the identity of the lipoprotein particles. Lipidomics further enforced the characteristic identities of the lipoprotein particles and exosomes, and revealed that chylomicrons and very low-density lipoprotein particles contain FAHFAs, as well as a FAHFA storage molecules triacylglycerol estolides (TG-EST). We established that FAHFAs can be liberated from TG-EST by lipoprotein lipase, an enzyme which normally hydrolizes triacylglycerol present in chylomicrons and very low-density lipoproteins, indicating that FAHFAs and TG-EST are a part of classic lipoprotein transport and delivery function. The data suggests that chylomicrons and very low-density lipoprotein particles are FAHFA and triacylglycerol estolide transporters and distributors in blood serum.

P25

Rice bran-derived 6-acyl-glucosylceramides are potent Mincle agonists that activate dendritic cells and enhance antigen-specific adaptive immunity

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The macrophage-inducible C-type lectin (Mincle, CLEC4E) is an FcRγ-coupled pattern-recognition receptor that drives pro-inflammatory responses to glycolipids. Cell-derived β-glucosylceramide (β-GlcCer) is an endogenous Mincle ligand, but whether structurally related dietary glucosylceramides engage Mincle is unknown. Rice bran contains diverse GlcCer species and, more recently identified, 6-acyl-GlcCers bearing a fatty acid ester-linked to the C6 of the glucose headgroup. We purified 13 GlcCer species and three 6-acyl-GlcCers (6-palmitoyl-, 6-oleoyl-, and 6-linoleoyl-GlcCer) from rice bran and tested their activity toward Mincle. In a Mincle-FcRγ NFAT-GFP reporter assay, GlcCer activation required a cis double bond at the Δ8 position of the sphingoid base, whereas all three 6-acyl-GlcCers strongly activated Mincle with dissociation constants comparable to the mycobacterial cord-factor analog trehalose-6,6′-dibehenate (TDB). Molecular docking indicated that the additional 6-acyl chain occupies a second hydrophobic groove on Mincle, rationalizing the enhanced affinity. In mouse bone marrow-derived dendritic cells (BMDCs), 6-acyl-GlcCers induced NF-κB activation and up-regulated pro-inflammatory cytokines and chemokines (IL-6, TNF-α, IL-1β, MIP-2, IL-12p40, OX40L) and increased IL-6 and TNF-α secretion. They further up-regulated the co-stimulatory molecules CD80/CD86 and MHC class II and, in an allogeneic mixed-leukocyte reaction, promoted the proliferation of CD8+ but not CD4+ T cells. Intranasal co-administration of 6-palmitoyl-GlcCer with ovalbumin enhanced antigen-specific serum IgG. These findings identify dietary 6-acyl-GlcCers as potent, food-derived Mincle agonists with adjuvant activity, and suggest a molecular link between rice bran intake and modulation of innate and adaptive immunity.

P26

Ether Phospholipid Responses to Dietary Patterns Incorporating Pasture-Raised Red Meat or Plant-Based Meat Alternatives

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Background: The effects of incorporating moderate amounts of pasture-raised red meat within a healthy dietary pattern on circulating lipidomic profiles remain poorly understood. This secondary analysis examined plasma lipidomic responses in a controlled dietary intervention comparing healthy dietary patterns incorporating either pasture-raised red meat (RM) or plant-based meat alternatives (PBMAs).

Methods: Plasma samples collected at baseline (T0), week 5 (T5), and week 10 (T10) were analysed using targeted lipidomics, quantifying 259 lipid species. Lipid abundances were $\log_2$-transformed and analysed using linear mixed-effects models with Benjamini-Hochberg false discovery rate (FDR) correction. Exploratory within-diet analyses were subsequently undertaken to investigate longitudinal lipidomic responses.

Results: Principal component and lipid subclass analyses demonstrated substantial overlap between dietary interventions, indicating limited global lipidomic restructuring. Among 259 lipid species, PE-O 39:6 was the only species remaining significant following FDR correction for the intervention × time interaction (FDR = 0.020). Concentrations of PE-O 39:6 declined in the vegetarian PBMA group but remained stable in the RM group. Exploratory within-diet analyses identified 34 FDR-significant lipid species in the RM group and 11 in the PBMA group. Responsive lipids in the RM group were enriched for ether phosphatidylcholine (PC-O) species (16/36 species; OR 8.98, $p = 2.9 \times 10^{-7}$) and plasmalogen phosphatidylethanolamine (PE-P) species (5/5 species; $p = 3.0 \times 10^{-5}$). Multiple DHA-containing phospholipids were represented among responsive species. Coordinated reductions in several PC-O and PE-P species were evident by week 5 and persisted through week 10.

Conclusions: Inclusion of moderate amounts of pasture-raised red meat within a healthy dietary pattern resulted in limited overall lipidomic differences compared with a vegetarian dietary pattern incorporating PBMAs. However, PE-O 39:6 emerged as a candidate intervention-responsive lipid, while exploratory analyses identified coordinated remodelling of ether phospholipid species, particularly within the red meat intervention.

P27

Comparative Lipidomic Analysis of Contrasting Oil Content Accessions in *Physaria fendleri*

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Hydroxy fatty acids are widely utilized in chemical, food, and cosmetic industries. Currently, Castor oil is the primary commercial source of hydroxy fatty acids, containing approximatively 90% ricinoleic acid (18:1-OH). However, the production and processing of castor seeds generate waste byproducts including ricin, a highly toxic protein, which presents significant safety and handling challenges. Lesquerella (*Physaria fendleri*), a plant native to the southwestern United States, has emerged as a promising industrial oilseed crop for hydroxy fatty acid production. Its seeds contain approximately 25% oil (w/w), of which nearly 60% is lesquerolic acid (20:1-OH), making it an attractive and safer alternative to castor oil. The overarching goal of this research is to enhance the accumulation of unusual fatty acids in Lesquerella to support industrial applications and biofuel production. To characterize the lipid composition of Lesquerella, a targeted lipidomics panel was developed and optimized using LC-MS/MS. The lipidomics platform includes: i) a polar lipid method capable of detecting 12 glycerophospholipids classes (PC, PE, PG, PI, PS, LPC, LPE, LPG, LPI, LPS, MGDG, DGDG), separated according to their headgroup, and i) a non-polar lipid method for the analysis of three glycerolipid classes (MG, DG, TG) resolved based on their fatty acyl composition. Using state-of-the art LC-MS/MS, lipid identification and relative quantification were performed on two Lesquerella accessions with contrasting oil content: PI 596432 (low oil, LO) and W6 20859 (high oil, HO).

P28

Maternal alkylglycerol supplementation during lactation increases tissue bioavailability of docosahexaenoic acid in offspring in rats

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Alkylglycerols possess an ether bond between the sn-1 fatty alcohol and the glycerol backbone, whereas conventional lipids have an ester bond. They are naturally present in breast milk but are absent from infant formulae. However, they exert significant biological activity in breastfed infants, by strengthening immune protection and preventing brown adipose tissue from differentiating too early into white adipose tissue. The aim of the present study was to explore further biological effects of alkylglycerols in breast milk. Rat mothers were therefore supplemented with alkylglycerols during the three weeks of lactation. The metabolic impact was then measured in the offspring, to determine the effect of the mother supplementation on the tissue accumulation of docosahexaenoic acid (DHA), an omega-3 fatty acid essential for cognitive development. The hypothesis put forward was based on the enrichment of cell membranes, particularly those in the brain, with plasmalogens, which are ether phospholipids derived from alkylglycerols and act as important reservoirs of DHA. The results showed that the milk of supplemented mothers was enriched in alkylglycerols, as well as in DHA, from as early as 7 days into lactation. This effect subsequently diminished gradually until weaning, at which stage DHA enrichment in the offspring's tissues, specifically the liver, plasma, and heart, was observed, particularly in males. In the brain, particularly in females, maternal supplementation with alkylglycerols had promoted a slight accumulation of DHA in phosphatidylethanolamine, and had also increased the proportion of plasmalogens to the level measured in males. Thus, the maternal dietary intake of alkylglycerols had increased the tissue bioavailability of DHA in suckling rats during the growth phase. The metabolic effects observed reinforce the case for adding alkylglycerols to infant formulae, although the longer-term physiological effects and mechanisms of action need to be clarified.

P29 (Competition for the best poster award)

Inhibition of Stearoyl-CoA Desaturase 1 restrains Hepatic Stellate Cell activation and induces oxidative stress

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Hepatic stellate cells (HSCs) are key mediators of fibrotic processes in the liver and undergo activation in response to liver injury or chronic inflammation. Thereby, quiescent HSCs transdifferentiate into proliferative and migratory myofibroblast-like cells that secrete large quantities of extracellular matrix proteins. This phenotypic shift is highly energy demanding, provoking metabolic reprogramming of activated HSCs. Recent advances in therapy of metabolic-dysfunction associated steatotic liver disease (MASLD) and related conditions focused on inhibiting HSC activation, including approaches that target stearoyl-CoA desaturase 1 (SCD1), the rate-limiting enzyme in monounsaturated fatty acid biosynthesis. Pharmacological inhibition and genetic knockdown of SCD1 have been associated with reduced activation of HSCs *in vitro*, yet the underlying mechanisms that link diminished SCD1 activity to antifibrotic effects in HSCs remain only partially defined.

To address this, we used an *in vitro* model to characterize HSC activation on the proteome level using the widely employed immortalized human HSC line LX-2. To delineate the mechanisms underlying reduced activation upon loss of SCD1 activity, we specifically inhibited SCD1 with the small-molecular inhibitor A939572 and assessed its effect in both pseudo-quiescent and activated states.

We found activated LX-2 cells to express elevated levels of SCD1 which underlines its importance in the activation process. Inhibition of SCD1 markedly reduced key fibrogenic markers, including α-SMA and COL1A1, demonstrating a strong suppression of HSC activation. Impaired SCD1 activity induced pronounced alterations in redox-regulatory pathways, most prominently within the glutathione synthesis. SCD1 inhibition further increased oxidative stress which underscores its direct impact on cellular redox homeostasis within HSCs. These findings provide a step towards elucidating the anti-fibrotic effect of SCD1 inhibition in HSCs and corroborate its therapeutical potential for fibrosis therapy.

P30

DHA retroconversion to EPA exists in cellular and animal models. Why wouldn't it occur in humans?

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Dietary DHA (C22:6 n-3) supplementation has been observed to increase EPA (C20:5 n-3) levels in rodents and humans. This metabolic response has been primarily suspected to result from a poorly characterized peroxisomal b-oxidation (retroconversion) pathway. However, recent original studies based on carbon-13 isotopic abundance (d^{13}C specific signature of □3 PUFAs) have challenged this assertion, suggesting that increases in EPA in response to DHA supplementation are not the result of increased retroconversion, but that dietary DHA may downregulate its own liver synthesis by inhibiting EPA elongation into n-3 DPA (C22:5 n-3). Focusing on the DHA retroconversion pathway, the purpose of this work was to better characterize the molecular mechanisms explaining why and how DHA increases EPA concentration, in cellular liver models and rats *in vivo*. Results first showed that cultured rat hepatocytes incubated with ^{2}D-DHA for 24h were able to synthesize detectable amounts of ^{2}D-EPA, according to a 6-7% retroconversion rate. HepaRG human hepatoma cells were also able to perform this pathway, with similar levels. In HepaRG cells, inhibiting peroxisomal β-oxidation by using thioridazine significantly reduced ^{2}D-EPA synthesis, whereas no effect was observed when mitochondrial β-oxidation was blocked (etomoxir treatment). In HepaRG cells incubated with DHA, EPA increased continuously over 48 hours, and mRNA levels of genes encoding enzymes involved in peroxisomal b-oxidation (Acox1/Decr2/Hsd17b4) were transiently significantly decreased, whereas elongases (elovl2/elovl5) mRNA levels were transiently significantly increased. In male rats fed for 8 weeks a diet containing a-linolenic acid (ALA, C18:3 n-3) compared to a diet supplemented with both ALA+DHA, EPA was significantly increased in liver phospholipids. mRNA expression level of genes encoding enzymes involved in peroxisomal b-oxidation (Acox1/Decr2/Acaa1) tended to decrease, whereas the elovl2 mRNA level was significantly increased.
These results may help to optimize the tissue status in DHA, which is an important current goal in human nutrition.

P31 (Competition for the best poster award)

Phospholipid-remodeling is key for differentiation and function of thermogenic adipocytes

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Mammals contain three major adipose tissue depots: white (WAT), beige and brown adipose tissue (BAT). While white adipocytes primarily store energy in the form of triglycerides in lipid droplets, brown and beige adipocytes dissipate chemical energy as heat through uncoupling protein 1 (UCP1), a process called non-shivering thermogenesis. An enhanced thermogenic adipocyte activity is associated with reduced obesity and cardiometabolic disease risk. Hence, understanding the mechanisms underlying beige and brown adipocyte development could open new therapeutic avenues for disease treatment. In this context, we explored the regulation and functional relevance of the metabolism of phosphatidylcholine (PC) and phosphatidylethanolamine (PE) as the major glycerophospholipids in mammalian cell membranes. To assess PC and PE synthesis via Kennedy pathway and their remodeling through Lands cycle in thermogenic adipocytes, we applied stable isotope labelling using deuterated choline and ethanolamine in combination with lipidomics. We found that brown and beige adipocytes exhibit an exceptional high capacity for PC and PE metabolism. At later stages of adipocyte differentiation, *de novo* PC and PE synthesis declined, whereas their remodeling increased, indicating a particular importance of Lands cycle in brown adipose tissue. In line with these results, adipose tissue browning induced by cold or β-adrenergic stimulation substantially altered acyl chain composition of PC and PE. A proteomic analysis revealed that specifically phospholipase enzymes of the Patatin-like phospholipase domain-containing protein (PNPL) family relevant for de-acylating PC in lands cycle are highly abundant in thermogenic adipocytes. In line with this finding genetic deletion of PNPL enzymes impaired UCP1 activity, while overexpression increased it. We conclude that acyl-chain remodeling of glycerophospholipids is key for the differentiation and function of brown adipocytes. It will be fascinating to explore in future studies whether targeted manipulation of PC and PE metabolism can be leveraged to promote brown adipose tissue thermogenesis and impede metabolic disease progression.

P32 (Competition for the best poster award)

Differential associations between dietary fatty acids and gut microbial metabolites in monozygotic twins discordant for autism

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Background: The gut microbiota may contribute to differences in host metabolism and health through bioactive metabolites. Dietary fatty acids (FA) modulate microbial metabolism, yet their relationship with microbiota-derived metabolites in autism spectrum disorder (ASD) remains poorly understood. Monozygotic twins discordant for ASD provide a unique model to explore these associations while minimizing genetic variability.

Methods: In 2023, fecal samples were collected from 16-year-old monozygotic male twins, one diagnosed with ASD and one neurotypical. Fecal short-chain fatty acids (SCFAs), branched-chain fatty acids (BCFAs), and selected aromatic microbial-derived metabolites, including indole, skatole, phenylacetic acid and p-cresol, were quantified by gas chromatography–mass spectrometry (GC-MS). Dietary intake was assessed using a 5-day food diary to estimate dietary lipid intake. Correlations between dietary lipid variables and the fecal microbial metabolite panel were assessed separately in each twin.

Results: Despite comparable dietary nutrient and FA intakes, distinct associations between dietary FA and microbial metabolites were observed between the twins. Dietary eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) showed significant positive correlations with multiple SCFAs and BCFAs in the ASD twin ($r=0.919–0.941$, $p=0.017–0.027$), whereas these associations were not observed in the neurotypical twin. EPA and DHA were also significantly associated with indole and phenylacetic acid ($r=0.931–0.957$, $p=0.0106–0.0217$). In contrast, the neurotypical twin showed significant associations between saturated fatty acids and butyrate and propionate ($r=0.9095–0.9266$, $p=0.0236–0.0322$), as well as between monounsaturated fatty acids and valerate ($r=0.911$, $p=0.0316$). Dietary cholesterol was also positively correlated with aromatic microbial metabolites, whereas n-6 fatty acids showed negative correlations ($r=−0.876–0.939$, $p=0.018–0.0499$).

Conclusion: Despite highly similar genetic backgrounds and dietary intakes, the twins displayed distinct nutrition–microbiota–metabolite associations. These findings suggest that dietary FA–microbiota metabolic relationships may differ in ASD and warrant validation in larger cohorts integrating dietary, metagenomic, and metabolomic data.

P33 (Competition for the best poster award)

Molecular Mechanisms of Ceramide Homeostasis in Dry Eye Disease

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Dry eye disease (DED) is the most prevalent ocular surface disorder worldwide. The major subtype, evaporative dry eye (EDE), is primarily associated with meibomian gland dysfunction (MGD), leading to tear hyperosmolarity and chronic inflammation. Recent studies have suggested that sphingolipid dysregulation, particularly altered ceramide (Cer) homeostasis, is involved in DED pathogenesis. However, the molecular mechanisms regulating ceramide metabolism under dry eye conditions remain poorly understood.

N-acylsphingosine amidohydrolase 2 (ASAH2) is a neutral ceramidase that hydrolyzes ceramide into sphingosine and a free fatty acid under neutral pH conditions. Although ASAH2 has been primarily characterized in intestinal epithelial cells, its biological function in corneal epithelial cells has not been established. Human corneal epithelial cells (HCECs) were exposed to hyperosmotic stress to mimic dry eye conditions. Quantitative PCR analysis revealed that ASAH2 expression was transiently upregulated during the early stage of hyperosmotic stress, suggesting an adaptive response to maintain ceramide homeostasis. In parallel, SMPD3, which encodes neutral sphingomyelinase 2 (nSMase2), an enzyme responsible for generating ceramide through sphingomyelin pathway, was continuously upregulated under hyperosmotic stress. However, prolonged hyperosmotic stimulation significantly reduced ASAH2 expression at both the mRNA and protein levels, shifting sphingolipid metabolism toward ceramide accumulation. The concurrent increase in SMPD3 and decrease in ASAH2 were associated with increased intracellular ceramide and reactive oxygen species (ROS) levels. To further investigate the functional role of ASAH2, HCECs were transfected with an ASAH2-overexpression plasmid prior to hyperosmotic stimulation. ASAH2 overexpression significantly improved cell viability under hyperosmotic stress.

These findings suggest that ASAH2 may play a protective role in maintaining ceramide homeostasis during dry eye-related stress. Further studies are warranted to clarify the mechanistic role in sphingolipid metabolism and evaluate its potential as a therapeutic target for dry eye disease.

P34

Beyond Cholesterol Lowering: Brazilian Green Propolis and artepillin C Modulate Lipid Metabolism and Hepatic Redox Homeostasis

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Brazilian green propolis, produced by *Apis mellifera* bees and derived primarily from *Baccharis dracunculifolia*, contains bioactive compounds with antioxidant and cardiometabolic potential. This study investigated the effects of its hydroalcoholic extract and artepillin C-rich fraction on lipid metabolism, cholesterol homeostasis, and hepatic oxidative *status* in hypercholesterolemic guinea pigs. Antioxidant capacity and HMG-CoA reductase inhibitory activity were evaluated *in vitro*. *In vivo*, male *Cavia porcellus* were fed control or hypercholesterolemic diets for 10 weeks and treated by oral gavage with hydroalcoholic green propolis extract (75, 150, or 300 mg/kg), an artepillin C-rich fraction (20, 40, or 80 mg/kg), or simvastatin (1.5 mg/kg). Serum and liver samples were analyzed for biochemical, oxidative, and mRNA expression parameters. All procedures were approved by the Ethics Committee on Animal Use of the University of São Paulo (CEUA/USP; protocol No. 18.1.999.60.0). The artepillin C-rich fraction showed marked antioxidant activity and inhibited HMG-CoA reductase *in vitro*. The hypercholesterolemic diet increased total and non-HDL cholesterol and reduced HDL cholesterol. Both propolis preparations favorably modulated the lipid profile, reducing total and non-HDL cholesterol and increasing HDL cholesterol. The hydroalcoholic extract also reduced triacylglycerol levels at higher doses. Propolis treatments increased *SREBP-2* and *HMG-CoA reductase* expression at specific doses, while *SR-BI* expression remained at control levels in most treatment groups. Green propolis also reduced hepatic TBARS levels under several experimental conditions. Collectively, these findings indicate that Brazilian green propolis and its artepillin C-rich fraction exert multifaceted effects on lipid metabolism and hepatic redox homeostasis, highlighting their potential as bioactive modulators of dyslipidemia and cholesterol metabolism.

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P35 (Competition for the best poster award)

Extrinsic lipids are absorbed and accumulate in colorectal cancer

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Lipid accessibility is key for cancer metabolism. Fatty acids (FAs) are β-oxidized in mitochondria to generate energy or used for membrane lipid synthesis in proliferating cancer cells. The current understanding is that those originate from de novo lipogenesis, which is drastically induced in colorectal cancer (CRC). Whether CRC cells absorb dietary lipids from the intestinal lumen to support their endogenous synthesis is unclear. Therefore, we here investigated the uptake and functional relevance of extrinsic FAs in CRC. Total FAs were quantified by gas chromatography-mass spectrometry (GC-MS) in tumor tissue and non-diseased mucosa from a discovery (n = 152) and an independent validation cohort (n = 28). Apc^{1638N} mice, an established mouse model for CRC, were used to track the flux of stable isotope-labelled lipids from the intestinal lumen into tumors. The relevance of lipid uptake for cancer cell proliferation and tumor progression was investigated in 2D and 3D cell models. Germ-free Apc^{1638N} mice were employed to investigate the role of the microbiome for CRC development. We demonstrate that diet-derived polyunsaturated fatty acids (PUFAs), including arachidonic acid (FA20:4 n-6) and docosahexaenoic acid (FA22:6 n-3) accumulate in tumor tissue of Apc^{1638} mice and CRC patients. Stable isotope tracing revealed a time-dependent absorption of FAs from the intestinal lumen into CRC tumors, supporting hepatic and intra-tumor de novo lipogenesis. Inhibition of FA import or β-oxidation in cancer cells hinders their proliferation. In the absence of the microbiome, fewer tumors were developed and survival was increased. In summary, our findings extend the understanding of cancer lipid metabolism by revealing that extrinsic FAs are taken up and accumulate in CRC, promoting cancer cell development and tumor growth. It will be intriguing to explore to what extent targeting this flux pathway together with the interrelated microbiome opens new therapeutic avenues for CRC in humans.

P36

Application of mCPBA Derivatization for Structural Analysis of Unsaturated Lipids

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Localization of double bonds in unsaturated lipids remains an important challenge in lipidomics. Derivatization with meta-chloroperoxybenzoic acid (mCPBA) enables selective epoxidation of carbon–carbon double bonds and produces characteristic MS/MS fragmentation useful for structural identification. In this work, phosphatidylcholine PC 36:2 is presented as a model system to demonstrate how mCPBA derivatization combined with mass spectrometry analysis can be used to determine fatty acid composition and double bond positions. This approach improves the structural characterization of lipid isomers in complex samples.

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P37

Effect of Combining Alpha-Linolenic Acid with EPA and DHA on Omega-3 Bioavailability and Metabolism in Rats

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Introduction. Omega-3 polyunsaturated fatty acids (n-3 PUFA) are essential nutrients involved in cardiovascular, inflammatory, and neurological functions. Alpha-linolenic acid (ALA, 18:3n-3) is the plant-derived precursor of the long-chain n-3 PUFA eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), but its endogenous conversion remains limited. The impact of combining ALA with preformed EPA and DHA on omega-3 bioavailability and metabolism is still poorly understood. This study evaluated the effects of different dietary combinations of ALA, EPA, and DHA in rats.

Methods. Forty male Wistar rats were randomly assigned to five dietary groups (n = 8/group) and fed experimental diets for 8 weeks: ALA alone, EPA+DHA without ALA, ALA+EPA/DHA (1:1 ratio), ALA+EPA/DHA with EPA predominance (1.7:1), or ALA+DHA without EPA. Fatty acid composition was measured in liver, plasma, erythrocytes, and brain using gas chromatography with flame ionization detection (GC-FID). Hepatic lipid fractions were also characterized.

Results. Diets combining ALA with long-chain n-3 PUFA produced the highest hepatic omega-3 levels (6.8-7.9% of total fatty acids), compared with ALA alone (5.3%) or EPA+DHA alone (3.4%). Hepatic EPA and DHA concentrations increased markedly when ALA was associated with long-chain n-3 PUFA. The ALA+DHA and balanced ALA+EPA/DHA diets resulted in the greatest DHA accumulation in liver and plasma. In erythrocytes, combined diets increased total omega-3 content by approximately 30% and improved the Omega-3 Index compared with single-source diets. The ALA+DHA diet produced the highest erythrocyte DHA levels and Omega-3 Index, whereas EPA-rich diets reduced DHA accumulation. No significant differences were observed in brain DHA levels among groups.

Conclusion Combining ALA with preformed EPA and DHA improves omega-3 bioavailability and tissue incorporation more effectively than supplying either precursor or long-chain n-3 PUFA alone. Diets containing DHA, alone or in balance with EPA, appear most effective for optimizing omega-3 status.

P38 (Competition for the best poster award)

High-Fat Diet Promotes Phenotypic Remodeling of Perivascular Adipose Tissue: Molecular and Spectroscopic Insights

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Perivascular adipose tissue (PVAT) surrounding the aorta displays regional heterogeneity, reflected in distinct PVAT phenotypes along the thoracic (TA) and abdominal aorta (AA). As a paracrine organ, PVAT secretes a range of bioactive mediators that regulate vessel function and participate in bidirectional communication with the vascular wall. High-fat diet (HFD) is known to impair PVAT function and impact the PVAT-vascular wall cross-talk [1]. However, the molecular mechanisms underlying HFD-induced PVAT dysfunction and the biological basis of lipid profile alterations have not been fully characterized. Here, we integrated mRNA transcriptome sequencing (RNA-seq) and gene expression analysis coupled with Raman spectroscopy to characterize phenotype and the lipid composition of murine TA and AA PVAT upon 8 weeks of HFD. RNA-seq analysis identified up- and down-regulated genes resulting from HFD influence on TA and AA PVAT. HFD feeding induced differential expression of genes involved in, *inter alia*, lipogenesis (*Aacs, Slc5a6, Scd1*), mitochondrial function (*Mtarc1, Mb*), ECM remodeling and inflammatory response (*Arsb, Orm3*). Distinct gene expression profiles related to the lipid composition, were accompanied by differences in the Raman marker, lipid unsaturation degree (I_{1660}/I_{1444}) [2]. We observed a decrease in lipid unsaturation after HFD, due to affected *de novo* lipogenesis. Molecular and spectroscopic characterization of heterogeneous PVAT facilitates the distinction between TA and AA PVAT, unveiling the influence of HFD on PVAT phenotype and lipid profile. These effects are linked to alterations in lipid metabolic pathways and inflammatory response. Together, these findings improve our understanding of the mechanisms underlying PVAT dysfunction in obesity.

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P39 (Competition for the best poster award)

Bioactive Lipid Alterations in Long COVID and their Modulation by Apheresis

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Post COVID-19 condition (long COVID) is a post-infectious condition characterised by persistent immune activation, inflammatory dysregulation, and metabolic perturbation. Current treatment options remain limited and largely focus on symptom management and rehabilitation, underscoring the need for effective treatment approaches. Apheresis has been investigated as a potential therapeutic strategy through extracorporeal removal of a broad range of molecules in blood, most likely including bioactive lipid mediators. Oxygenated polyunsaturated fatty acids (oxylipins) together with other bioactive lipids mediate and modulate inflammation and may provide insights into pathophysiological processes underlying long COVID. To investigate alterations in bioactive lipid profiles associated with apheresis treatment, plasma samples of 23 long COVID patients undergoing apheresis and 25 healthy controls were analysed using untargeted liquid chromatography coupled to high-resolution mass spectrometry (LC-HRMS). Prior to apheresis, patients with long COVID exhibited a distinct bioactive lipid profile compared to healthy controls, including differences in arachidonic acid (AA)- and linoleic acid (LA)-derived metabolites, significantly increased levels of conjugated bile acids, and significantly reduced sphingosine-1-phosphate (S1P). Upon apheresis, significant changes in the bioactive lipid profile were observed, including perturbations in S1P, AA- and LA-derived oxylipins, and conjugated bile acids. Several bioactive lipid alterations shifted towards levels observed in healthy controls, indicating attenuation of multiple perturbations. Overall, long COVID was associated with alterations in the bioactive lipid metabolism, notably involving S1P, AA- and LA-derived oxylipins, and conjugated bile acids. Following apheresis, multiple dysregulated bioactive lipid metabolites shifted towards levels observed in healthy controls, suggesting that apheresis may alleviate the metabolic burden associated with long COVID.

P40

Lipid composition in mouse brain and enriched myelin fractions by mass spectrometry. Comparison with demyelinated damaged tissues.

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Myelin is affected in various central nervous system disorders and in degenerative diseases, particularly in multiple sclerosis. This phenomenon suggests that variations of lipid composition of myelin can disrupt its insulating function and may contribute to myelin degradation: e.g., metabolic defects inducing changes in lipid composition would produce loosening of opposing bilayers of myelin membrane thus leading to detachment of consecutive bilayers, decompaction and disruption of its structure. To better understand the biological processes occurring in multiple sclerosis and to help in the diagnosis/prognosis of patients, it is essential to determine the lipid composition and their quantitation in myelin. To this aim, we analysed by mass spectrometry the whole brain and an enriched-myelin fraction contained in white matter, in healthy and demyelination/remyelination model mice.

For this study, lipids were extracted from the tissues using the well-known Folch extraction method. Analyses were performed with a high-resolution Orbitrap mass spectrometer (Exploris 120, Thermo Fisher Scientific) with an EASY-IC ion source operating in positive and negative ion modes and coupled to a liquid chromatographic system (Vanquish Flex, Thermo Electron) with a reverse phase column. Lipid identification was based on retention time, accurate mass, natural isotopic distribution and MS^2 fragment patterns. The preliminary results revealed differences in lipid composition among whole-brain and myelin-enriched fraction samples from healthy mice. Notably, the most important variations were observed in the levels of cholesterol, phosphatidylcholine, phosphatidylethanolamine, and glycosphingolipids. Healthy mice were then compared to a reversible demyelination mice model (cuprizone model). The global lipid composition showed variation of cholesterol, phosphatidylcholine, phosphatidylethanolamine and phosphatidylserine. Interestingly, for the phospholipid species, the unsaturation number of fatty acid chains seemed to be affected by the demyelination/remyelination process.

P41 (Competition for the best poster award)

Ceramide carried by electronegative LDL impairs BBB integrity in Alzheimer's Disease

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Alzheimer's disease (AD) is a progressive brain disorder in which systemic metabolic dysregulation may precede neurodegeneration. Lipidomic studies have consistently shown that ceramide levels are elevated in plasma, brain tissue, and cerebrospinal fluid (CSF) in AD. However, the mechanisms by which peripheral ceramide influences the central nervous system (CNS) remain largely unknown. As circulating ceramide requires lipoprotein for transport, electronegative low-density lipoprotein (LDL(−)), a highly toxic low-density lipoprotein (LDL) subfraction, may serve as a potential mediator of this process. Furthermore, dysregulation of sphingolipid signaling has been implicated in blood-brain barrier (BBB) dysfunction, an early and independent pathological event in AD. We therefore hypothesize that ceramide carried by LDL(−) may impair BBB integrity and contribute to neurovascular dysfunction in AD. To investigate the effects of ceramide carried by LDL(−) on barrier function, LDL(−) was isolated from human plasma and applied to a human brain microvascular endothelial cell (BMEC)-based *in vitro* BBB model. Immunofluorescence analysis revealed that LDL(−) treatment reduced Claudin-5 and VE-Cadherin expression, as well as disrupted its junctional localization, indicating compromised tight junction organization. Mechanistically, LDL(−) activated lectin-like oxidized low-density lipoprotein receptor-1 (LOX-1) signaling, accompanied by increased reactive oxygen species (ROS) production and elevated ceramide levels, suggests that ceramide-associated oxidative stress may contribute to BBB dysfunction. Taken together, our findings identify that ceramides carried by LDL(−) cause BBB dysfunction by activating LOX-1 signaling, increasing oxidative stress, and disrupting tight junction integrity. These findings suggest that LDL(−)-associated ceramide may connect peripheral lipid dysregulation and early BBB dysfunction in AD.

P42 (Competition for the best poster award)

Circulating and red blood cell omega-3 polyunsaturated fatty acids in relation to plasma p-tau biomarkers of Alzheimer's Disease pathology

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Consortium for the Early Identification of Alzheimer's Disease – Quebec (CIMA-Q)

Introduction: Higher circulating concentrations of omega-3 polyunsaturated fatty acids (n-3 PUFA) have been associated with reduced dementia risk. However, their relationship with fluid biomarkers reflecting Alzheimer's disease (AD) neuropathology remains unclear. We investigated associations between circulating fatty acids (FA) and plasma biomarkers of AD-related tau pathology in individuals at preclinical or early stages of cognitive impairment and explored relationships between red blood cell (RBC) FA and plasma p-tau217 status.

Methods: Plasma samples were collected from 272 CIMA-Q participants (mean age 74.3 years; 60.3% female), including cognitively healthy individuals, subjective cognitive decline, mild cognitive impairment, and AD. Plasma p-tau181 and p-tau231 were quantified using Simoa HD-X, and p-tau217 using S-PLEX MesoScale. Plasma FA profiles were analyzed by gas chromatography and expressed as percentages of total fatty acids. RBC fatty acid profiles were explored in relation to p-tau217 status using an exploratory threshold (MSD assay >4 pg/mL vs ≤4 pg/mL). Correlation analyses assessed relationships between fatty acids, tau biomarkers, and clinical characteristics.

Results: Plasma fatty acid profiles were largely comparable across diagnostic groups, except for higher total saturated FA in AD, driven by palmitic acid ($p<0.001$), and lower very-long-chain SFA ($p=0.02$). Females had higher n-6 and n-3 PUFA, whereas males had higher SFA. DHA and linoleic acid were inversely associated with waist circumference and BMI. Plasma n-3 PUFA, particularly DHA, were inversely associated with p-tau181 ($p<0.01$), p-tau217 and p-tau231 (both $p<0.05$). Exploratory RBC analyses suggested differences in selected omega-3 fatty acids, including ALA and EPA, between participants with higher versus lower p-tau217 concentrations (both $p<0.05$).

Discussion: Plasma n-3 PUFA, particularly DHA, were associated with lower circulating tau biomarkers, supporting further investigation of omega-3 status in relation to AD-related neurodegeneration. Preliminary RBC findings suggest that longer-term FA measures may also capture differences related to tau pathology and need further validation.

P43

Metabolic crosstalk between astrocytes and neurons in the protective fatty acid synthesis under hypoxia.

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Brain hypoxia is associated with many pathological conditions. Because accumulation of lactic acid and reduced cofactors under hypoxia result in significant brain damage, alternative O2 hydrogen acceptors might aid brain anaerobic metabolism and survival. Considering that fatty acid synthase (FASN) uses reduced cofactors in de novo fatty acid synthesis (FAS), we hypothesized that FAS might maintain a reductive potential in the brain during hypoxia, thereby protecting tissue from hypoxic damage. Previously, consistent with this hypothesis, we demonstrated a significant increase in FAS in primary neurons but not in astrocytes or other cell types in response to hypoxia, and showed the protective role of FAS under hypoxia in vitro. However, our recent in vivo data using an astrocyte-specific inducible FASN KO model demonstrated a crucial role of astrocytic FAS in brain protection under hypoxic insult. Because astrocytes do not increase FAS under hypoxia from direct precursors tested, such as aspartate, asparagine, glucose, glutamate (Glu), glutamine (Gln), these data indicate a possible metabolic crosstalk between neurons and astrocytes, which involves the synthesis of an alternative FAS substrate in neurons, which can be further used by astrocytes for FAS under hypoxia. To test this hypothesis, we performed fluxomics analysis of neuronal cell metabolism under hypoxia. Using stable isotope-labeled substrates, we demonstrated, for the first time, that neuronal cells significantly increase N-Acetyl-Aspartate (N-Ac-Asp) synthesis from Glu/Gln under hypoxia. Interestingly, the flux analysis revealed a key role for reductive carboxylation and the presence of a self-contained pathway in which no free NH3 is released during N-Ac-Asp synthesis from Glu. Further, we demonstrated that the N-Ac-Asp synthesized in neurons is a preferred substrate for FAS activated in astrocytes under hypoxia, indicating novel metabolic crosstalk between neurons and astrocytes in the protective FAS under hypoxia.

P44

The Contribution of Ultra-High-Field Nuclear Magnetic Resonance Spectroscopy to the Characterization of Brain Lipid Extracts

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In the central nervous system, myelin, composed of lipids and proteins[1], forms the white matter, whose fundamental role is to conduct signals to the gray matter, where information processing takes place. Numerous studies have revealed a close link between abnormalities in brain lipid metabolism and neurodegenerative diseases such as Alzheimer's[2]. It is therefore essential to have efficient tools for characterizing and quantifying these lipids. In this study, we used proton, carbon, and phosphorus nuclear magnetic resonance (NMR) spectroscopy to analyze and quantify lipid mixtures, including mouse brain extracts and reference lipid mixtures[3,4]. For the first time, we compared, using model lipid mixtures, the gains in sensitivity and resolution obtained at very high magnetic field strength (1.2 GHz, Infranalytics platform in Lille) relative to a lower field strength (400 MHz). Using 1D and 2D NMR experiments, we were able to identify several species of phospholipids, plasmalogens, and sterols.

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P45 (Competition for the best poster award)

Investigate the underlying mechanism of sphingolipid dysregulation in an animal model of Alzheimer's disease

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Alzheimer's disease (AD) is the most prevalent form of dementia, contributing to approximately 60–70% of cases globally. AD is characterized by amyloid-β (Aβ) deposition and tau hyperphosphorylation; however, the molecular mechanisms linking these two pathological hallmarks remain unclear. Emerging evidence supports that sphingolipid dysregulation is implicated in Aβ accumulation, tau pathology, synaptic dysfunction, and memory impairment, suggesting that altered sphingolipid metabolism may represent a potential link between Aβ-driven pathology and tau hyperphosphorylation. The 5XFAD mouse model, which harbors five human familial AD mutations and develops aggressive Aβ-driven pathology, was employed in this study. One- and three-month-old male and female 5XFAD mice, along with non-carrier (NCAR) littermates, were included. After brain tissues were collected, histological staining and western blotting were performed to characterize pathological progression. At 3 months of age, 5XFAD mice exhibited increased vacuolation in the brain. Phosphorylated tau (p-tau) levels were also significantly elevated in the cornu ammonis 1 (CA1) region and cortex, with more pronounced changes in female mice, indicating region-specific and sex-dependent pathological alterations. Transcriptomic analysis revealed progressive remodeling across multiple branches of sphingolipid metabolism, which was positively associated with tau phosphorylation-related transcriptional changes. Among the genes involved in tau phosphorylation, *Ppp2r2a*, *Gsk3b*, and *Mapk8* were identified as candidate genes potentially linking sphingolipid dysregulation to tau pathology. Notably, subtle changes in the sphingolipid pathway were already shown at 1 month of age, with *Sgpp1* significantly increased despite limited overall transcriptomic changes and the absence of overt tau pathology at this stage. Collectively, these findings suggest that disruption of sphingolipid metabolism may emerge early and progressively develop into broader dysregulation associated with tau pathology. This study provides a potential molecular framework linking Aβ accumulation to subsequent tau pathology and highlights sphingolipid metabolism as a potential target for early intervention in AD.

P46

Quantitative lipidomics of healthy and demyelinated mouse brain by multinuclear NMR and Mass spectrometry

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Myelin is a highly specialized multilamellar membrane that wraps around axons in the central nervous system and is essential for rapid nerve conduction and neuronal survival. Owing to its exceptionally high lipid content, composed mainly of phospholipids, glycolipids and cholesterol, alterations in myelin composition are associated with numerous demyelinating and neurodegenerative disorders, including multiple sclerosis. Despite its biological importance, the quantitative lipid composition of myelin and its changes during demyelination and remyelination remain incompletely characterized. To address this question, we developed a quantitative analytical workflow combining multinuclear NMR spectroscopy (^{1}H, ^{31}P and ^{2}H) with LC-MS/MS lipidomics to characterize lipid composition in whole brain, corpus callosum and myelin-enriched fractions from mouse brain. The complementary nature of these techniques enables both absolute and relative quantification of major lipid classes together with detailed molecular lipid profiling. The approach was applied to healthy mice, to the cuprizone model, which reproduces reversible demyelination followed by remyelination, and the shiverer mouse model, a genetic model of congenital hypomyelination caused by the absence of myelin basic protein (MBP). Quantitative comparison of these models reveals distinct alterations in phospholipid and cholesterol composition associated with myelin loss and recovery. In parallel, solid-state ^{2}H-NMR performed on reconstituted lipid membranes provides complementary information on membrane organization and lipid dynamics. Overall, this integrated strategy provides a comprehensive framework for investigating myelin lipid composition and membrane organization under physiological and pathological conditions and establishes a quantitative platform for studying myelin-related diseases.

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P47

A subpopulation of GPR120+ lipid-sensitive dopamine neurons modulates excitatory tone and the tradeoff between exploration and feeding in mice

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Mesolimbic dopamine (DA) neurons are key contributors to food motivation, locomotion and mood states. Dietary lipids influence DA neuronal activity in the ventral tegmental area (VTA), yet much remains to be elucidated about lipid-sensing mechanisms in these neurons. We studied the contribution of G-protein coupled receptor 120 (GPR120, also known as FFAR4), a receptor with high affinity for polyunsaturated fatty acids, to VTA DA neuronal activity and motivated behaviors.

Subregional RNAscope analysis revealed that GPR120 is expressed in less than 10% of DA neurons in the VTA. To investigate function, we generated adult DA-specific GPR120 knockout mice (VTA^{DA} GPR120 KO) using an adenoviral Cre-loxP approach. In behavioral tests, VTA^{DA} GPR120 KO mice exhibited reduced exploration in novel environments, alongside increased motivation for sucrose reward in an operant task and increased snacking (frequency of short feeding bouts) in metabolic cages.

Primary culture experiments reveal that pharmacological activation of GPR120 increases intracellular calcium via G*q* signaling in DA neurons. Electrophysiological recordings showed that theta burst stimulation-induced excitatory postsynaptic potentials in the dorsal VTA were significantly reduced in VTA^{DA} GPR120 KO mice, indicating impaired excitatory synaptic transmission. Notably, RNAscope analysis also revealed enrichment of GABAergic neurons in the dorsal VTA, suggesting a stronger inhibitory microenvironment in this region. Consistent with this, primary cultured DA neurons derived from GPR120 KO mice exhibited increased surface expression of $GABA_A$ receptor co-localized with synapsin I, indicating enhanced inhibitory synaptic input.

Collectively, these findings indicate that GPR120-expressing DA neurons represent a specialized subpopulation that modulates the exploration-feeding balance by regulating excitatory and inhibitory synaptic integration within the VTA. *Supported by an NSERC grant to SF.*

P48 (Competition for the best poster award)

Brain lipidome signatures of chronic stress and their modulation by antidepressants in zebrafish

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Major depressive disorder (MDD) is a highly prevalent disease in which alterations in brain lipid metabolism have emerged as important contributors to its pathophysiology. A previous lipidomic study in zebrafish exposed to unpredictable chronic stress (UCS) demonstrated extensive brain lipidome remodeling, suggesting that dysregulated lipid homeostasis may underlie stress-induced neuronal dysfunction. In this study, we aimed to evaluate whether the antidepressants fluoxetine (FLU) and nortriptyline (NOR), two widely prescribed drugs, could counteract these stress-induced lipid alterations. For this, adult male zebrafish were exposed to UCS for 7 days in the presence or absence of FLU or NOR, followed by behavioral assessment, cortisol quantification, and untargeted brain lipidomic analysis by liquid chromatography-mass spectrometry (LC-MS). Both antidepressants attenuated UCS-induced behavioral impairments and reduced cortisol levels, indicating mitigation of stress-axis activation. Lipidomic analysis in the brain revealed that treatment appears to reverse stress-induced alterations by restoring plasmalogen lipid levels, endogenous antioxidant molecules whose increase under UCS may reflect enhanced oxidative stress, as well as phosphatidylinositol (PI) and phosphatidylserine (PS) species involved in neuronal signaling and synaptic plasticity. Also, FLU and NOR attenuated the accumulation of arachidonic acid-containing phospholipids, which can be correlated with reduced phospholipase-mediated membrane remodeling and inflammatory signaling. These findings support a role for lipid metabolism in the mechanisms underlying antidepressant action beyond classical monoaminergic pathways and highlight brain lipid remodeling as a promising target for therapeutic intervention in MDD.

P49

Genetic polymorphism of *TOMM40*, adjacent to *APOE*, is associated with opioid-induced delirium in patients with cancer pain

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Background: Opioid analgesics are recommended for treating cancer pain. Genetic predisposition can affect opioid analgesic responses and complications. Opioid-induced delirium (daily somnolence) is one of barriers to interfere with opioid use, which is a likely factor contributing to inadequate pain management. Recently, some genetic polymorphisms in the apolipoprotein E (*APOE*) and adjacent genes have been reportedly associated with postoperative delirium. We conducted this study to examine whether these predispose an individual to opioid-induced delirium. Methods: 71 cancer patients rated their pain on an 11-point numerical rating scale twice before and after increasing opioid analgesics. Opioid-induced delirium was assessed on a 5-point Likert scale as daily somnolence. Based on genotypes of 41 single nucleotide polymorphisms (SNPs) in the *APOE*, *TOMM40* (Translocase of outer mitochondrial membrane 40) and *APOC1* and *APOC1P1* (Apolipoprotein C) genes. We conducted the rate-of-change analysis of opioid-induced delirium after increasing opioid doses among genotypes of the SNPs by non-parametric comparison tests. As an exploratory nature, we did not consider the multiplicity of statistical analyses. Results: Only one SNP of the *TOMM40* gene reached significance in changes of opioid-induced delirium (K-W test, p=0.033). Its minor homozygosity was associated with the aggravation, but the major homozygosity and heterozygosity improved by increasing opioid. This *TOMM40* SNP did not demonstrate a linkage disequilibrium with any *APOE* SNPs. Conclusion: One *TOMM40* gene polymorphism was associated with opioid-induced delirium, independent of *APOE* genotype. Reduced *TOMM40* expression in the brain reportedly suppressed traits associated with Alzheimer's disease pathogenesis via effects on cholesterol metabolism and mitochondrial function. Our results should be validated in a large-scale study with a larger sample size.

P50 (Competition for the best poster award)

Obese adipose tissue secretome induced remodeling of the cellular lipidome of breast cancer cells.

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Obesity is associated with a poorer prognosis in breast cancer, yet the metabolic mechanisms driving this relationship remain incompletely understood. Peroxisome proliferator-activated receptors (PPARs) are ligand-activated nuclear receptors that function as cellular lipid sensors, with free fatty acids (FFAs) serving as endogenous ligands. To investigate obesity-driven metabolic crosstalk, adipose tissue-conditioned medium (ACM) was generated from obese human adipose tissue explants and used to treat two triple-negative (MDA-MB-231, SUM-159-PT) and two hormone receptor-positive (MCF7, T47D) breast cancer cell lines. ACM lipid composition was characterized by Flow Injection Analysis Fourier Transform Mass Spectrometry (FIA-FTMS) and gas chromatography-mass spectrometry (GC-MS), while cellular lipid remodeling was assessed by FIA-FTMS. Lipidomic data were integrated with matched RNA sequencing (RNA-seq) datasets to identify the transcriptional changes underlying the observed obesity-induced metabolic adaptations.

Our findings indicate that obese adipose tissue releases FFAs that are readily taken up by breast cancer cells. These FFAs act as endogenous PPAR ligands, driving transcriptional reprogramming of lipid metabolism. ACM-treated cells exhibit coordinated suppression of de novo lipogenesis alongside increased expression of genes involved in lipid uptake, neutral lipid storage, and mitochondrial fatty acid β-oxidation (FAO). Consistent with these transcriptional changes, lipidomic analyses reveal extensive intracellular lipid remodeling, supporting a metabolic shift toward exogenous lipid utilization to meet the energetic and biosynthetic demands of tumor growth. Together, these findings identify PPAR-mediated metabolic reprogramming as a mechanism by which obesity-derived adipose tissue drives metabolic adaptations in breast cancer cells and suggest that this pathway may represent a therapeutic vulnerability.

P51 (Competition for the best poster award)

Leishmania amazonensis subverts macrophage lipid metabolism to drive endogenous lipogenesis and PUFA synthesis

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Successful *Leishmania* infection relies on the metabolic subversion of macrophages. Lacking sufficient *de novo* lipid biosynthesis, these protozoa must exploit host lipids to secure their intracellular niche. This study characterizes the profound lipidomic remodeling induced by *Leishmania amazonensis* in THP-1 macrophages, defining a critical host-pathogen metabolic vulnerability. Using HPTLC, GC-MS, BODIPY confocal microscopy, and isotopic tracing ([³H]LDL, [³H]HDL, [³H]palmitate), we mapped intracellular lipid dynamics over 48h. Strikingly, early infection (24h) triggers a severe downregulation of key structural and signaling phospholipids (PC, PA, PS, PI), alongside an immediate surge in cholesteryl esters and diacylglycerols. This early remodeling precedes a massive cellular accumulation of neutral lipids at 48h, including triacylglycerols, sterols, and free fatty acids, which corroborates the expansion of lipid droplets. Radiotracing proved this lipid overload is not driven by extracellular scavenging; while [³H]palmitate uptake remained unaltered, the internalization of exogenous lipoproteins ([³H]LDL, [³H]HDL) was actively suppressed. Corroborating a shift toward endogenous synthesis, GC-MS exposed a pronounced accumulation of saturated fatty acids and an active elongation and desaturation cascade, prominently enriching dihomo-gamma-linolenic, arachidonic, and osbond acids. The detection of exclusive infection biomarkers, such as 10-heptadecenoic acid, confirms a distinct metabolic signature. Crucially, this targeted omega-6 PUFA enrichment provides the essential lipid pool for generating immunomodulatory eicosanoids, facilitating macrophage immune evasion. Ultimately, *L. amazonensis* establishes an endogenous lipogenic shunt. By dismantling exogenous uptake and aggressively reprogramming host biosynthesis, the parasite fabricates a rich lipid niche essential for its survival and proliferation, unveiling targets for host-directed antileishmanial therapies.

P52

Plant vs Animal – Food lipids in drug delivery

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Rationale The distinction between food and pharma is becoming more blurred – in part due to the increased importance of understanding lipid digestion and interactions of digestion products with poorly water-soluble drugs to enable dissolution and absorption. In this context we have been building a framework to understand the differences in efficacy of different lipid sources in enabling drug dissolution and absorption which can enable both improved formulation approaches for lipid-based drug delivery systems but also potential recommendations on the interactions of plant drinks compared to milk-based products for consumption with a tablet to achieve the same effect.

Methods We utilise two main in situ methods to study digestion of lipid formulations and drug solubilisation, synchrotron X-ray scattering and low frequency Raman spectroscopy to track both fatty acid production and drug dissolution. When coupled with HPLC to quantify drug independently the rate and extent of drug solubilisation can be rationalised against in vivo absorption data.

Results and Conclusion The results emerging from a matrix of drugs and lipid compositions indicates that while the drug ionisation state is important (weakly-basic drugs such as clofazimine have a clear affinity for ion pairing with liberated fatty acids), the opposite is not necessarily true for neutral and acidic drugs such as tolfenamic acid where lipid digestion can still provide significant dissolution. The generation of colloidal structures in which the drug has an increased solubility compared to the undigested triglyceride matrix can still provide a sink to drive dissolution, providing a more nuanced selection of lipid composition to suit specific compounds. While mammal triglyceride sources are well suited to the solubilisation of some drugs, vegetable sources and be better suited for others.

P53

Aging impairs lipid droplet turnover in the murine aortic endothelium

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Lipid droplets (LDs) regulate endothelial lipid metabolism, but their turnover during aging remains poorly understood. This study investigated age-associated changes in LD turnover in the murine aortic endothelium and mechanisms of LD degradation. Aortic lipid overload was studied in vivo following oral olive oil gavage (10 mL/kg body weight) and ex vivo using isolated aortic rings exposed to oleic acid (OA, 500 μM) or palmitic acid (PA, 200 μM) for 24 h. LDs were quantified by fluorescence microscopy, while Western blotting, biochemical assays, and adipose triglyceride lipase (ATGL) and lysosomal acid lipase (LAL) inhibition were used to assess LD turnover. Aged aortas showed higher basal LD accumulation despite similar circulating free fatty acids and triglycerides, together with reduced CGI-58 expression and increased inflammation. Postprandial LD clearance was slower in aged mice. Both OA and PA increased LD accumulation ex vivo however, lipolysis was reduced after OA treatment in aged aortas, whereas PA responses were unchanged. ATGL inhibition caused greater LD accumulation after OA than PA exposure, while combined ATGL and LAL inhibition produced the strongest effect following OA treatment. These findings indicate impaired endothelial LD turnover with aging and suggest that altered coordination between lipolysis and lipophagy may contribute to lipid accumulation and vascular dysfunction.

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P54 (Competition for the best poster award)

Renal and metabolic effects of empagliflozin in a prediabetic rat model assessed by urinary proteomics and metabolomics

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Background SGLT2 inhibitors have pleiotropic effects beyond glucose-lowering, including reducing heart failure and the risk of kidney diseases. The mechanisms remain unclear. Beneficial effect of empagliflozin is observed regardless of the presence of diabetes. This study investigated the metabolic effects of empagliflozin on renal function in prediabetic hereditary hypertriglyceridemic (HHTg) rats. We used liquid chromatography with tandem mass spectrometry to determine the proteomic and metabolomic profiles of empagliflozin in urine.

Methods: Wistar strain as controls, and HHTg strain as a model of metabolic syndrome without obesity and fasting hyperglycemia were used. The rats were fed a standard diet, either with or without empagliflozin (10 mg/kg/day for 8 weeks).

Results: Compared to controls, the HHTg rats exhibited increased lipid accumulation in the kidneys ($p<0.001$), elevated NGAL ($p<0.001$) or altered urinary protein excretion ($p<0.05$). Empagliflozin treatment has been shown to partially improve renal parameters in HHTg rats, by reducing serum triglyceride levels ($p<0.001$) and renal ectopic triglyceride ($p<0.05$) and cholesterol ($p<0.01$) deposition. There was also a decrease in albuminuria and NGAL in urine ($p<0.05$). Targeted urinary proteomic analyses showed reduction in inflammatory markers MCP-1, IL-6, IL-8 and leukotrienes ($p<0.01$). In the renal cortex, mRNA expression of MCP-1 was reduced ($p<0.05$). Increased expression of β-hydroxybutyrate dehydrogenase 1 ($p<0.01$) in the renal cortex may improve utilisation of ketone bodies and enhance kidney energy efficiency. In addition, empagliflozin increased glutathione levels ($p<0.01$), activity of glutathione peroxidase and superoxide dismutase ($p<0.05$) in the renal cortex.

Conclusion: Empagliflozin's renoprotective effects may be due to its anti-inflammatory and antioxidant properties, and its improvement of renal lipid metabolism. Urinary proteomics can indicate renal disorders in prediabetic condition.

P55 (Competition for the best poster award)

Vectorization of antibodies for the treatment of cutaneous lymphomas

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Primary cutaneous lymphomas are a heterogeneous group of rare non-Hodgkin lymphomas, arising from either T cells or B cells, that primarily affect the skin [1]. These two types are referred to as “cutaneous T-cell lymphoma” (CTCL) and “cutaneous B-cell lymphoma” (CBCL), CTCL accounting for the majority of cases (approximately 75%). In advanced stages, systemic treatments such as chemotherapy or immunotherapies, are prescribed [2]. Immunotherapy is a revolutionary treatment that has proven beneficial for many cancers. However, the use of antibodies makes it expensive, and its systemic administration via intravenous injection has been shown to cause numerous adverse effects [3,4]. A topical route of administration via nanovectorization would prevent these adverse effects, increase efficacy, and reduce treatment costs, as the amount of antibody required would be reduced. The objective of this study was to develop a new alternative to the intravenous route of administration by directly targeting the tumor within the skin layers using a lipid-based vector. Multilamellar liposomes (MLLs) were chosen as a delivery system for the antibody as they offer several advantages, such as good stability and a more prolonged release compared to unilamellar liposomes [5,6]. Previous studies have also demonstrated that it is possible to deliver a molecule to a specific layer of the skin by encapsulating an active ingredient in MLLs with controlled physicochemical parameters [7,8]. To this end, empty MLLs, composed of pharmaceutical-grade ingredients—which distinguishes this study from previous ones—were prepared with different physicochemical parameters to analyze their impact on the depth of skin penetration prior to antibody encapsulation. Observation of the penetration depth of MLLs into artificial skin using Raman imaging confirmed that, by modifying the physicochemical parameters, it was possible to target different layers of the skin. Finally, the encapsulation of antibodies in MLLs proved feasible with an encapsulation efficiency exceeding 50%.

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P56 (Competition for the best poster award)

Headgroup identity shapes the immunomodulatory effects of arachidonoyl-phospholipids in macrophages

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Membrane phospholipids are increasingly recognized as active regulators of immune cell function. Among them, aminophospholipids (APL), including phosphatidylserine (PS) and phosphatidylethanolamine (PE), contribute to membrane organization, intracellular signaling, and inflammatory regulation. However, the roles of individual molecular species in macrophage activation remain poorly defined. In this work, we investigated how supplementation with two arachidonoyl-phospholipids that share an identical fatty-acyl composition but differ in their polar headgroups (PS16:0/20:4 and PE16:0/20:4), modulates macrophage responses under basal and inflammatory conditions. RAW 264.7 macrophages were supplemented with each phospholipid at rest and prior to lipopolysaccharide (LPS) stimulation, then profiled by untargeted LC-MS/MS proteomics with qPCR validation of selected inflammatory and antioxidant genes. Both phospholipids drove extensive proteome remodeling under basal and inflammatory conditions, showing that exogenous phospholipids reshape macrophage metabolism. A shared response was the enrichment of antioxidant and oxidative stress-control proteins, notably Hmox1 and Prdx1, which APL supplementation further increased under LPS stimulation. Transcriptional data corroborated this observation, with elevated *Hmox1* and *Prdx1* mRNA levels following supplementation with either APL species. Nevertheless, PS16:0/20:4 and PE16:0/20:4 also induced distinct changes in protein expression, highlighting headgroup-dependent effects. PS16:0/20:4 induced broader proteome remodeling, characterized by enrichment of proteins associated with PS recognition, Nrf2-associated antioxidant pathways, and endoplasmic reticulum stress response. In contrast, PE16:0/20:4 preferentially regulated proteins involved in glutathione metabolism, detoxification pathways, and oxidative stress control, while attenuating LPS-induced expression of Il1a, Il1rn, Nos2, and Nfkb2. Overall, these findings establish PS16:0/20:4 and PE16:0/20:4 as bioactive aminophospholipids that promote redox homeostasis and constrain excessive inflammation, with headgroup identity as a key determinant of macrophage functional remodeling.

P57

Pre-clinical Evaluation of Targeting Lipoprotein Receptors for Solid Tumors Therapeutics: Epithelial Ovarian Cancer

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Lipid nanoparticles (LNPs) modulate drug PK/PD via controlled release. Cancer resistance to receptor-targeted LNPs persists due to compensatory signaling. We identified lipoprotein receptors (LRs) as key biomarkers driving tumor progression via lipidomic reprogramming. A paclitaxel-loaded LR-targeted LNP for epithelial ovarian cancer (EOC) was developed, with preclinical evaluation conducted across a five-stage R&D pipeline.1st, we demonstrated both LDLR (gate for cholesterol; promotes chemosensitivity) and VLDLR (gate for neutral lipid; suppresses development) are prognostic manipulators in EOC patients, which can be used as diagnostic markers. 2nd, the LNP mimicking LRs (using ApoE peptide) were designed, and small-scale production (named LMND) with defined loading and C.M.C. LMND particle size is 116 nm, PDI 0.063, and a Zeta potential of -33 mV. Cryo-TEM imaging demonstrates that particles are spherical in nature and electron-dense, indicating the drug is in the polymeric core. 3rd, we've established and validated a method using liquid chromatography coupled with electrospray ionization tandem mass spectrometry (LC-ESI-MS/MS) for quantification of PTX in LMND and biological distribution. The detection was performed using electrospray ionization in positive ion mode and quantified by multiple reaction monitoring. The inter- and intra-day precision of LQC to HQC for PTX ranged from 2.0% to 18.1%, indicating a reliable method for defining PTX (0.1810 mg ± 0.27/mL). 4th, intravenous administration of PTX and LMND (2mg/kg, 12 hrs) in B6 mice to evaluate biodistribution. It's particularly abundant in the lung, spleen, and liver, which contain higher concentrations: 396.1, 67.4, and 48.2 ng/g, respectively. 5th, the PTX and LMND were injected into the orthotopic EOC model to compare therapeutic efficacy. Quantitative LC-ESI-MS/MS analysis revealed significantly higher intratumoral PTX concentrations in LMND-treated EOC mice (5.0 ng) compared with PTX group (1.62 ng). Conclusion: the LMND exhibited superior tumor reduction caliber compared to others.

P58

Docosahexaenoic acid supplementation restores lipid composition and relieves bortezomib-induced peripheral neuropathy in rats

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Background and aims: Bortezomib (BTZ), a proteasome inhibitor, widely used in cancer treatment, is frequently limited by chemotherapy-induced peripheral (CIPN). CIPN significantly affects patient quality of life and compromises patient survival due to dose reduction or interruption of treatment as no preventive or curative treatment is currently available. In this context, identifying biological mechanisms driving CIPN is essential for the development of new therapeutic targets. Our previous studies showed that BTZ induces lipid dysregulation *in vivo* and further investigation of BTZ mechanism of action *in vitro* highlighted that omega-3 polyunsaturated fatty acid (DHA) supplementation seems to be a potential solution to relieve CIPN induced by this anticancer drug. The aim of this study is to investigated membrane remodeling in a rat model of CIPN induced by BTZ and the impact of omega-3 supplementation on CIPN.

Methods: Male rats were injected i.p. with 2 mg/kg BTZ cumulative dose, or its vehicle, on days 1, 2, 4, 5 and 8. Daily lipid supplementation with 60 mg/kg DHA i.v. or its vehicle, between day 1 and 8 were organized 4 hours before anticancer injection. CIPN was assessed using behavioral tests, including nociceptive test (von Frey, cold immersion) and functional conduction velocity test. Lipid and protein composition was investigated in sciatic nerve, dorsal root ganglion, spinal cord and brain using HPTLC and Western blot respectively.

Results: Our preliminary work shown a relief of Bortezomib-induced peripheral neuropathy by DHA supplementation. This supplementation appears to restore the specific lipid content that is altered by BTZ in the sciatic nerve.

Conclusions: These preliminary findings suggest that CIPN is linked to a membrane remodeling in vivo. DHA, an omega 3 seems to be a potential solution to relieve bortezomib-induced neurotoxicity. Further experiments are needed to confirm these initial results.

P59 (Competition for the best poster award)

Protective effect of DHA supplementation against neurotoxic mechanism of action of Bortezomib

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Background and aims: Emerging evidence suggest that omega-3 polyunsaturated fatty acid supplementation may reduce the prevalence and severity of chemotherapy-induced peripheral neuropathy side effect (CIPN). CIPN negatively impacts quality of life and can even threaten patient survival due to dose reduction or interruption of treatment. Bortezomib (BTZ), the first approved proteasome inhibitor anticancer drug, seems to cause lipid dysregulation as shown in our previous work *in vivo*. BTZ effect is counteracted by Omega-3 polyunsaturated fatty acid (DHA) supplementation. This protective result could be explained by recent studies associating BTZ toxicity to ferroptosis, a cell death pathway linked to other neuropathies. The aim of this study is (1) to establish the mechanisms of BTZ neurotoxicity by the study of oxidative stress and ferroptosis *in vitro* and (2) to investigate the protective effect of DHA supplementation on the newly characterized mechanism.

Methods: BTZ toxicity was investigated in ND7/23 cells after 24h using spectrometry (MTT, ATP and LDH assays), transmission electron microscopy and flow cytometry (Annexin V-FITC/IP staining). Lipid peroxidation and cell death was examined using C11-bodipy and lysotracker labeling. To visualize lipid rafts in cells, ganglioside GM1 was tagging with fluorescently labeled cholera toxin B.

Results: BTZ induces neurotoxicity *in vitro* reflected by ferroptosis mechanism and linked to lysosomal accumulation, mitochondrial alterations and lipid peroxidation. DHA supplementation blocks BTZ toxic effects and exerts a protection against lipid rafts aggregation due to BTZ.

Conclusions: These preliminary findings suggest that ferroptosis could be connected to BTZ neurotoxic mechanism in line with existing literature. Omega-3 polyunsaturated fatty acid supplementation seems to be a potential solution to relieve anticancer BTZ-induced neurotoxicity. Further experiments are needed to confirm these initial results.

P60 (Competition for the best poster award)

Beyond peritumoral microenvironment: stage-dependent impact of colon cancer on the mesenteric adipose molecular signature

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Background. Peritumoral adipose tissue is increasingly recognized as an active component of the tumour microenvironment (TME), yet the temporal sequence and molecular mechanisms underlying its remodelling during colon cancer (CC) progression remain poorly understood. We investigated whether mesenteric visceral adipose tissue (VAT) undergoes stage-dependent molecular changes before direct tumour invasion. **Methods.** Matched peritumoral (pVAT) and distant mesenteric VAT (mVAT) samples were collected from patients with TNM T stage T2-T4 colon cancer. Transcriptomic profiling, adipokine/cytokine quantification, quantitative lipidomics of isolated adipocytes, and histomorphometric analyses were integrated to characterize adipose remodelling across tumour stages. **Results.** Transcriptomic analysis identified 1,607 differentially expressed genes in pVAT relative to mVAT. Tumour progression was associated with sequential activation of extracellular matrix remodelling and fibrosis, followed by proliferative programmes and widespread repression of adipocyte lipid metabolism. Importantly, these alterations were already evident at T2, before direct tumour-adipose contact, and intensified through T3 and T4. Molecular changes were accompanied by a broad reduction in adipocyte-derived mediators, including FABP4, resistin, IL-6, and TNFα. Lipidomics revealed selective remodelling of membrane phospholipids rather than neutral lipid stores: phosphatidylcholines, ether phosphatidylcholines and sphingomyelins increased significantly in pVAT, together with enrichment of arachidonic acid-containing phosphatidylcholine species and depletion of linoleic acid-containing species, whereas triacylglycerol content remained largely unchanged. Histological analyses confirmed significantly smaller adipocytes in pVAT, consistent with increased membrane phospholipid abundance. Notably, distant mVAT also exhibited progressive molecular and lipid alterations with advancing stage, suggesting that adipose remodelling extends beyond the immediate TME. **Conclusions.** Mesenteric VAT undergoes coordinated transcriptomic, inflammatory, lipidomic and morphological remodelling that begins before direct tumour invasion and intensifies throughout CC progression. These findings identify mesenteric adipose tissue as a dynamic component of the TME and reveal membrane phospholipid remodelling as an early hallmark of adipose reprogramming with potential diagnostic and therapeutic relevance.

P61 (Competition for the best poster award)

Unraveling the lipidome in mental health: Identifying biomarkers of depression during the perinatal period

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Mental health disorders are among the leading causes of global disease burden, with particular incidence during endocrine-sensitive life stages, such as pregnancy. Perinatal depression (PND) affects an estimated 10-13% of women, with significant consequences for both maternal well-being and offspring mental health development. In recent years, biomarker research on neuropsychiatric disorders has accelerated, driven by major advances in high-throughput omics technologies. Given the crucial roles of lipids in neurobiological processes, lipidomics has emerged as a promising tool to explore lipids as potential biomarkers of PND. Under the scope of the Re-MEND project, this study aimed to evaluate the plasma lipidome plasticity throughout pregnancy and postpartum in healthy and depressed women with different depression trajectories. Untargeted lipidomic profiling using liquid chromatography-mass spectrometry (LC-MS) was performed on plasma samples from the BASIC cohort, a Swedish population-based, prospective cohort (n=660). Sampling occurred at pregnancy weeks 17 and 38 and postpartum week 8. Depression status and depression trajectories were defined at each timepoint according to predefined study criteria. Raw data processing was conducted using Compound Discoverer, and statistical analysis was performed in R software. Multivariate analysis showed that sampling timepoint, i.e. stage of pregnancy, is the most influential factor at the lipidome level, with clear clustering of samples by timepoint, independently of the depression status and associated trajectories. Within this global structure, week 38 of pregnancy exhibited one of the strongest group separations, where patients not yet depressed at sampling but with an associated trajectory of depression at postpartum already showed distinct lipidomic profiles from healthy controls, suggesting a remodeling at the lipidome level preceding a future clinical onset of depression. Among 39 significantly altered lipid species (adj.P.Val < 0.05 and |logFC| > log2(1.2)), triacylglycerides were most represented (n=30), followed by acylcarnitines (n=5), suggesting a lipid metabolism shift with potential predictive power for PND.

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P62 (Competition for the best poster award)

Effect of 4-F4t-Neuroprostane Molecule X on the evolution of Duchenne muscular dystrophy (DMD) in rat

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Rational: Duchenne muscular dystrophy (DMD) is an X-linked genetic disorder, due to the absence of functional dystrophin, responsible for progressive muscle degeneration lost and dilated cardiomyopathy (Bellinger et al,200.9). No compensatory treatment is currently available. This deficiency is accompanied byAmong molecular alterations, an early dysfunction of ryanodine receptors (RyR) is reported, causing diastolic calcium leakage of calcium from the sarcoplasmic reticulum in skeletal (Bellinger et al,2009) muscle and in the heart (Faucounnier et al, 2010) which participate to the progression of the disease. We previously described a molecule (molecule X, an oxidized metabolite od ixidizedof DHA, an omega-3 fatty acid) able to stabilize RyR by preventing its pathological posttranslational modifications. We, we thus hypothesized that itAmong the therapeutic avenues, omega-3 polyunsaturated fatty acids, particularly "Molecule X", an oxidized derivative of DHA, stabilize these calcium channels and could slow the progression of DMD.

Materiels and Methods: Therapeutic efficacy will be evaluated in vivo in a DMD rat model (Taglietti et al 2022), whose phenotype is close to that of patients. Dosage will be defined by blood, urine and tissue assays. Animals will receive the compound or placebo vehicle via osmotic pump and will be longitudinally followed for during 1 or 4 months by conventional echocardiography and speckle-tracking, Holter ECG, grip test and plethysmography. Then, Ex vivo analyses (histology, biochemistry) will allow the evaluatevaluation eof the post-translational modifications of RyRs (oxidation, nitrosylation, phosphorylation, association withand calstabin1/2 depletion his partner calstabine) as well as the signaling pathways involved (oxidative stress, inflammation, fibrosis).

Expected Outcomes: Based on results obtained with other NEO-PUFADHA metabolites (4-F4t-Neuroprostanes), which prevented RyR-dependent calcium leakage and early contractile alterations in mouse models, we anticipate that "VoldemortMolecule X" will stabilize RyR and preserve muscle, ventilatory and cardiac functions in DMD rats.

Conclusion: This project aims to position "Molecule X" as a new potential therapeutic lipid molecule in DMD, acting upstream early by the stabilization of RyRs. It could pave the way for the development of a new class of oxidized lipid compounds of pharmacological interest in DMD and other related pathologies

P63

Innovations in the profiling of epidermal ceramides: use of supercritical fluid chromatography coupled with high-resolution mass spectrometry (Q-TOF) for advanced analysis of various samples

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Lipids play a crucial role in the structure and function of the skin. They are primarily composed of ceramides, fatty acids, and cholesterol. In a health context, a comprehensive characterization of the epidermal lipid composition is essential, as it varies in relation to skin diseases such as ichthyosis and psoriasis. The improvement of skin sample collection is a growing concern, as current methods are invasive (skin scraping), which has led to the development of new analytical methods, particularly in the extraction of these new matrices (strips, reconstructed epidermis, etc.). However, the diversity of epidermal ceramides, combined with the complexity of their structure and the lack of reference standards, makes their analysis particularly challenging. An innovative profiling of epidermal ceramides has been implemented through the combination of Supercritical Fluid Chromatography (UPC², Waters), which is particularly suitable for non-polar metabolites, and high-resolution Q-TOF mass spectrometry (Xevo, Waters). This analytical development will be presented, it has enabled the MS characterization and the relative quantification of 16 lipid sub-classes of skin ceramides. This method allows the fast profiling (10 minutes) of 310 molecular species, facilitating high-throughput applications in clinical research. The extraction method has also been improved to be adapted to various matrices (tissues, reconstructed human epidermis, tape stripping) while being rapid and suitable for all ceramide subfamilies, despite their differing chemical properties. Comparison of the results obtained on these three matrices will be presented. Moreover, we will demonstrate that this novel method enables the analysis of reconstructed epidermises and strips, confirming the role of the protein PNPLA1 (patatin-like phospholipase domain-containing in the synthesis of specific epidermal ceramides, which is implicated in autosomal recessive congenital ichthyosis. This approach offers a new and efficient way to analyze epidermal ceramides, providing valuable insights into skin health and disease management.

P64

Stigmasterol esters encapsulated in liposomes

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Phytosterols are bioactive compounds naturally present in plant seeds and vegetable oils. Due to the presence of double bonds in their structure, phytosterols exhibit low stability at elevated temperatures. In addition, the bioavailability of these compounds in the human body is relatively low. Therefore, new phytosterol formulations with improved stability and bioavailability are being actively investigated. The aim of this study was to evaluate stigmasterol fatty acid esters (myristic and oleic acid esters) encapsulated in liposomes. The degree of stigmasterol and fatty acid degradation, as well as the formation of oxyphytosterol derivatives, was assessed. The quality of the liposomes was evaluated using transmission electron microscopy (TEM). During the heating of liposomes at 60°C, the lowest degradation of stigmasterol was observed in liposomes containing free stigmasterol. In samples heated to 180°C, liposomes containing stigmasteryl oleate showed the highest stability. A high content of oxyphytosterols was detected in all samples. The lowest level was found after heating liposomes at 60°C in liposomes containing free stigmasterol (1.7 mg/g), whereas the highest level was observed after heating liposomes at 180°C, reaching 135.7 mg/g in liposomes containing free stigmasterol.

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P65 (Competition for the best poster award)

Membrane lipid remodeling regulates B cell function

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Membrane lipid composition varies widely across immune cell types, reflecting cell-specific functional adaptations. While lipid mediators have primarily been studied as immunological regulators, recent reports indicate that membrane lipids themselves regulate lymphocyte activity independently of ligand–receptor interactions. However, which membrane lipid species constitute immune cells and how the membrane lipid environment influences immune function remain largely elusive. Here, we applied untargeted and spatial lipidomics for murine lymphoid tissues to comprehensively characterize the lipid composition of immune cells. Untargeted lipidomics of bulk tissues and purified B and T cells identified a lymphocyte-specific lipidome signature. Spatial lipidomics further identified several membrane lipids as lipid markers of lymphocytes. Genetic deletion of the phospholipid remodeling enzyme in mice revealed a mechanism of controlling the levels of membrane lipid enriched in B cell. Furthermore, *in vivo* adoptive transfer experiments demonstrated that B cells with perturbed membrane lipid composition exhibited impaired competitive fitness compared with wild-type controls, suggesting that the membrane lipid environment is important for optimal B cell function. Collectively, our findings identify a previously unrecognized lymphocyte-specific lipidome signature and demonstrate that membrane phospholipid lipid remodeling is a key regulator of B cell homeostasis.

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Immunometabolic Phenotypes in Psoriasis Revealed by Cytokine–Lipid Profiling

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Background: Psoriasis is a chronic immune-mediated disease associated with an increased burden of metabolic abnormalities and cardiovascular risk. The interplay between inflammatory cytokines and lipid metabolism remains incompletely understood. This study investigated therapy-induced changes in cytokine profiles and explored their associations with lipid biomarkers in patients with psoriasis.

Methods: Serum concentrations of IL-17A, IL-22, TNF-α, IL-12(p40), IL-10, and VEGF-A were measured before and after therapy. Lipid parameters included triglycerides (TG), low-density lipoprotein cholesterol (LDL), and high-density lipoprotein cholesterol (HDL). Paired cytokine analyses were performed in 34 patients. Cytokine changes were assessed using Wilcoxon signed-rank tests with Benjamini-Hochberg correction. Multivariate cytokine profiles were evaluated using PERMANOVA, ANOSIM, hierarchical clustering, and correlation network analyses.

Results: Therapy significantly reduced all six cytokines, with the largest decreases observed for IL-22 and IL-12(p40), followed by VEGF-A. These findings indicate a broad anti-inflammatory effect extending beyond the Th17 axis. Multivariate analysis confirmed significant remodeling of the overall cytokine profile after treatment (R^2=0.0535, p=0.001). Among lipid parameters, only triglyceride status was significantly associated with cytokine-pattern heterogeneity (p=0.042). Although individual cytokine–lipid correlations were not statistically significant, biologically plausible trends were identified for VEGF-A–TG, IL-17A–TG, TNF-α–HDL, and IL-12(p40)–LDL relationships. Hierarchical clustering revealed three distinct inflammatory phenotypes characterized by differential cytokine expression patterns.

Conclusions: Psoriasis therapy induces consistent but moderate restructuring of systemic inflammatory networks. The association between triglyceride status and multivariate cytokine profiles suggests the presence of distinct immunometabolic phenotypes. These findings support further investigation of cytokine–lipid interactions as potential biomarkers of cardiometabolic risk and therapeutic response in psoriasis.

P67

Palm oil consumption, plasma fatty acid profile and cardiometabolic health: a comparative study across four regions of Côte d'Ivoire

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Widely consumed in Côte d'Ivoire, palm oil remains controversial in terms of its cardiometabolic effects. This study aimed to assess anthropometric parameters and lipid, lipoprotein and cardiometabolic biomarkers in palm oil consumers in Côte d'Ivoire. A 12-month cross-sectional study included 190 regular palm oil consumers spread across four regions of Côte d'Ivoire (North: area 1, West: area 2, Centre-East: area 3 and Abidjan: area 4), characterised by distinct consumption patterns. Anthropometric measurements and biological analyses, including lipid and lipoprotein profiles, plasma fatty acids by gas chromatography, the HOMA-IR index and hsCRP, were carried out. Average palm oil consumption was 31 mL per person per day, significantly higher in Abidjan. Waist circumference and BMI were higher in areas 3 and 4, with no association with abnormalities in the lipid profile, which remained normal regardless of the area. No significant differences were observed in the profile of plasma fatty acids, despite relatively high concentrations in the various regions. The HOMA-IR index was below the threshold for insulin resistance in all areas, but hsCRP was higher in areas 3 and 4. The consumption of palm oil, whether crude or refined, was not associated with an unfavourable lipid and lipoprotein profile or with insulin resistance, even at high levels of consumption. The adiposity and inflammation observed in Abidjan appeared to be linked to the nutritional transition rather than to palm oil itself, highlighting the importance of the overall dietary context in assessing cardiometabolic risk in Côte d'Ivoire.

P68

Lipolytic response to fasting reflects metabolic processes involved in obesity resistance

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The propensity to obesity varies greatly among individuals and murine strains, depending on genetic predispositions. Mechanisms defining this propensity may be targeted by future anti-obesity therapies. They may become more pronounced under conditions of metabolic stress, such as cold exposure and fasting. Previously, we characterized cold adaptations in the obesity-prone murine strain C57BL6/J and the obesity-resistant strain A/J following prolonged cold exposure. Furthermore. As these strains also differ in cold-induced turnover of triacylglycerols and free fatty acids in white adipose tissue (WAT), we now aim to assess lipolysis and fatty acid re-esterification in WAT in these mice during fasting. C57BL6/J and A/J male mice were either fed ad libitum or exposed to 24 h fasting and then sacrificed. Subsequently, hepatic triacylglycerol content and plasma lipid levels were analyzed along with the secretion of free fatty acid from pieces of gonadal and subcutaneous WAT. When fasted, WAT of A/J mice secretes higher amounts of free fatty acids and glycerol compared to C57BL6/J mice. The changes in the fatty acid-to-glycerol ratio between the fed and fasted states suggest that A/J mice can suppress re-esterification more efficiently during fasting. Changes in turnover of lipolysis and re-esterification partially reflect changes in the expression of major lipases and glyceroneogenic enzymes. These processes in WAT affect plasma fatty acid and triacylglycerol levels and hepatic triacylglycerol content, which transiently reach higher levels in C57BL6/J than in A/J mice. The obesity-resistant A/J mice thus exhibit greater metabolic flexibility, with more pronounced fasting-induced lipolysis and hepatic triacylglycerol accumulation. Whether these features are directly related to the propensity to obesity needs to be confirmed, e.g., in other obesity resistant murine strains.

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P69 (Competition for the best poster award)

The double bond dilemma: the vinyl ether bond is dispensable for retinal integrity and fertility in the mouse

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Ether lipids play essential roles in membrane organization, signaling, and stress responses in diverse tissues. Inborn errors in early ether lipid biosynthesis leading to a deficiency in all ether lipids (i.e. plasmanyl ether lipids and plasmalogens) cause Rhizomelic Chondrodysplasia Punctata (RCDP), a severe multi-organ disorder characterized by skeletal abnormalities, early-onset bilateral cataracts, microphthalmia, and infertility. Mouse models lacking all ether lipids, such as the *Gnpat* knockout (KO), recapitulate these phenotypes; however, whether the ocular and reproductive defects stem from total ether lipid depletion or whether the specific loss of plasmalogens is causative has remained unresolved. Plasmanylethanolamine desaturase (PEDS1) introduces the vinyl ether double bond to generate plasmalogens from plasmanyl precursors. The identification of the *Peds1* gene allows dissecting the impact of selective plasmalogen deficiency. Moreover, the recent description of human *PEDS1* deficiency in two young children, only one of whom presented with cataracts, underscores the need to differentiate subclass-specific functions *in vivo*.

Using matched genetic backgrounds, we directly compared *Gnpat* KO mice with *Peds1* KO mice by targeted LC-MS/MS lipidomics analysis of the ocular phospholipidome, alongside histological evaluation of eye and lens morphology and assessments of breeding performance and litter statistics.

Selective plasmalogen loss resulted in a much milder eye and fertility phenotype compared to total ether lipid deficiency. While *Gnpat* KO mice exhibited microphthalmia, cataracts, and infertility, *Peds1* KO mice developed no cataracts up to 15 months of age. Targeted lipidomics showed that the loss of plasmalogens in *Peds1* KO eyes was accompanied by compensatory accumulation of plasmanyl species with matching *sn*-1 alkyl and *sn*-2 acyl chain compositions. Furthermore, Peds1 KO mice showed no apparent breeding abnormalities and produced offspring when KO males and females were mated. This demonstrates that loss of the vinyl ether double bond is not the key determinant of all symptoms in human and murine ether lipid deficiency and can at least partly be compensated by plasmanyl lipids.

P70

Ether-Glycerophospholipid Trafficking in Cancer Prognosis and its Therapeutic Targeting via Upcycled Milk Extracellular Vesicle

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Lipid reprogramming is a hallmark of cancer that profoundly influences prognosis, yet the specific lipid species and their driving mechanisms in carcinoma remain largely unclear. This study integrates multi-omics profiling to delineate the role of ether-linked glycerophospholipids (GPL O-) in hepatocellular carcinoma (HCC) and epithelial ovarian cancer (EOC) progression. Through integrated transcriptomic and lipidomic analysis of clinical cohorts, we identified ether-linked phosphatidylcholine and phosphatidyl-ethanolamine as significant prognostic markers associated with poor clinical features. In HCC, the pathological upregulation of VLDLR facilitates the non-autonomous uptake of dietary lipids, which drives cell mobility and tumor growth. Concurrently, in EOC, we mapped an LDLR–LPC–FAM83B–FGFR regulatory axis, demonstrating how ether-linked lipids modulate lysophosphatidylcholine (LPC) levels to foster chemo-insensitivity. Mechanistically, these lipids accelerate malignant progression by inducing cytoskeletal rearrangement, driven by Transient Receptor Potential Vanilloid 2 (TRPV2) channel activity. In vivo, conditional knockout of VLDLR and the deployment of targeted lipid-based nanoparticle drugs (VMNDs) successfully mitigated tumor burden and reversed therapeutic resistance, validating these pathways as potent therapeutic targets. Looking toward sustainable clinical translation, we are expanding our nanoparticle platform by introducing a novel whey valorization project. This research leverages milk-derived extracellular vesicle (EV) as naturally evolved, bio-mimetic carriers that mirror the sophisticated lipid-trafficking principles of our synthetic VMNDs. By repurposing high-value dairy byproducts, we are developing a oral form delivery vehicle that enhances therapeutic efficacy while adhering to circular economy principles in biotechnology. This transition from synthetic nanoparticle systems to upcycled, EV-based therapeutics represents a significant advancement in sustainable, biocompatible precision oncology. In conclusion, our results define GPL O- as critical drivers of malignant progression and therapeutic resistance in both HCC and EOC. Our ongoing work demonstrates that integrating advanced lipid-based drug delivery with sustainable byproduct valorization offers a robust, translational framework to combat cancer malignancy and improve patient management.

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Lipidomic analysis of *Cordyceps sinensis*

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Cordyceps sinensis (*C. sinensis*) is an insect parasitizing fungus. The Chinese name is 'DongChongXiaCao' (冬蟲夏草) ('winter-worm summer-grass'), which is found at high altitudes on the Qinghai-Tibetan plateau. This fungus is a type of Ascomycetes which parasitizes insect larvae of one particular species of moth, *Hepialus armoricanus*, grows and gradually changes into a fruit body. it is one of the major tonics in Chinese medicine. More than 400 Cordyceps species have been described worldwide (around 90 in China). The preparation has been considered to have healing properties for the body and mind. There are reports that the bioactive ingredients such as 3′-deoxyadenosine, cordycepic acid, cordycepic polysaccharides might be beneficial for the prevention and treatment of a variety of ailments, including respiratory disease, metabolic disorders, and renal dysfunction. *C. sinensis* is also known to contain minerals, but the complete lipid content has not been reported.

In this study, we applied linear ion trap multiple stage mass spectrometry (LIT MS^n) with high resolution MS to report the lipidome of *C. sinensis*. We found that linoleic acid (n-6 18:2-FA) is the most prevalent species, followed by oleic acid, palmitic, stearic and hydroxy linoleic acids. In addition to the major FFA family, the extract also contained abundant TAG, ranging from 50:2 (16:0/16:0/18:2) to 56:4 (20:0/18:2/18:2), where 18:2-FA containing TAGs are dominant, in which18:2/18:2/18:2-TAG is the most prominent. By contrast, phospholipids including PA, PE, PC, PS, and PI are of low abundance (<1%), and are all dominated by the 1-palmitoyl 2-linoleoyl phosphatidyl lipid species. GC/MS analysis indicates the presence of low level of ergosterol, which is the typical sterol found in fungi.

The rich content of 18:2-FA (n-6) and 18:2-FA containing glycerophospholipids in *C. sinensis* is interesting. Nutritionally, linoleic and α-linolenic (9-*cis*,12-*cis*,15-*cis* 18:3) acids are essential for basal metabolism in humans, while long-chain polyunsaturated FAs have many beneficial effects on human health. Commercially, there are many reports on nutraceutical effects of *C. sinensis*, but studies on the lipid content of *C. sinensis* as biochemical makeup have not been reported. Whether if the richness of the 18:2 and 18:2-containing lipids play roles in the bioactive effect of *C. sinensis* on health remains unexplored. After all, it may be merely an edible mushroom.

P72 (Competition for the best poster award)

Trans Fat induced Metabolic Enteritis in Susceptible Mice

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Western-style diets impose a great risk to intestinal health, yet diet complexity limits our understanding of inflammatory factors and their mode of action on mucosal homeostasis. Lipid excess however is one of the known risk factors in development of inflammatory bowel disease (IBD).
We report that dietary unsaturated fatty acids in *trans* configuration (trans fats), highly prominent in ultra-processed foods, trigger Crohn-like disease in susceptible animals. Trans fat induced Crohn-like disease is specifically restricted by intestinal epithelial X-box binding protein 1 (Xbp1) and further governed by intestinal epithelial sterol regulatory binding protein 1 (SREBP1)-mediated Proprotein Convertase Subtilisin/Kexin Type 9 (PCSK9) expression.

Trans fat induced Crohn-like disease is reversed by treatment with PCSK9 monoclonal antibody Alirocumab, alleviating chronic gastro-enteritis. In a simplified small intestinal organoid model, stimulation with *trans* but not *cis* unsaturated fatty acids leads to inflammatory signature and PCSK9 induction. CD patients with ileitis further exhibited increased SREBP1 transcription factor activity along with increased PCSK9 expression, both correlating with severity of endoscopic and histologic disease activity.

Collectively, we define inflammatory actions of unsaturated fatty acids in *trans* configuration in the intestine and create novel perspectives along with potential repurposing of targeted metabolic therapies in IBD.

P73

Characterization of the repertoire of *Mycobacterim tuberculosis* lipids presented to T lymphocytes by CD1b protein

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Mycobacterium tuberculosis (Mtb), the bacterium responsible for tuberculosis, features a uniquely lipid-rich cell wall. Many of these lipids are antigenic, capable of activating T lymphocytes, and contribute to shaping the immune response during Mtb infection. As such, they represent promising targets for vaccine development against tuberculosis. However, while some mycobacterial antigenic lipids have been identified, the full spectrum of Mtb lipids presented on antigen-presenting cells (APCs) remains unknown.

This project aims to define the complete repertoire of mycobacterial lipids presented by CD1b molecules, a family of antigen-presenting proteins structurally similar to MHC class I. Most known mycobacterial lipid antigens are presented by the CD1b isoform, and their recognition by T cells triggers immune activation against the pathogen. A deeper understanding of Mtb's lipid repertoire and the resulting T cell responses could advance tuberculosis vaccine design.

To achieve this, we developed an innovative approach: engineering APCs to express recombinant CD1b molecules with a protease cleavage site between their luminal and transmembrane domains. After pulsing these APCs with Mtb lipid extracts and inducing protease cleavage, we use lipidomics to characterize lipids specifically extracted from CD1b. This method enables a comprehensive analysis of both endogenous and exogenous lipids presented by CD1b in response to Mtb stimulation.

By refining our knowledge of lipid-specific immune responses in tuberculosis, this study paves the way for identifying new vaccine candidates.

P74

The impact of different dietary marine oil sources on tissue lipidome profiles in zebrafish (*Danio rerio*) models with special reference to EPA and DHA tissue uptake and retention patterns

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Commercial marine fish oils, rich in EPA and DHA, are used as food supplements due to their purported health benefits. However, differences in oil source can result in variation in fatty acid (FA) composition, which may differentially affect EPA and DHA tissue uptake, retention and distribution in tissues. These aspects remain poorly studied.

Here, we investigated how three marine oils derived from farmed salmon (SO), herring (HO), and cod liver (CO) influence tissue lipidomes using zebrafish (Danio rerio) as a model organism. experimental feeds were standardised for EPA and DHA levels. Two-month-old zebrafish were fed the test diets for three months before tissue sampling. Brain, retina and liver were analysed for differences in FA composition, lipid classes and lipid species using chromatographic and mass spectrometric approaches. Distinct tissue specific responses were observed in the brain, retina and liver at both FA and lipid class/species level, indicating clear functional differentiation. The tissue n-3/n-6 ratio was improved in response to dietary oils in the order CO>HO>SO, with the largest improvements observed in the retina, followed by the brain. However, both the brain and retina showed limited remodelling at both phospholipid (PL) class and species levels, suggesting tight regulation of membrane composition and functions. Most diet induced separation in neural tissues occurred at triacyglyceride (TG) class and species level, indicating potentially important but still poorly understood roles for TGs in neural health. The liver lipidome was the most responsive to the different dietary oil treatments across all levels (FA, PL and TG). Overall, CO provides the most favourable lipidomic outcomes compared with HO and SO, while also revealing important insights into tissue-specific lipid compositions, their functional potential and modulation by dietary lipids.

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Thank you for joining us in Bordeaux

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