NuGOweek 2025

Book of Abstracts

Dublin, Ireland • 22–25 September 2025

PROGRAMME AT A GLANCE

Monday, 22nd September 2025

Session 1: What is a healthy and sustainable diet?

Chairs: Prof. Lorraine Brennan & Prof. Diana Ivanova

13:00	Registration
14:00	Welcome: Prof. Lorraine Brennan
14:15	*Sustainable Diets: A Side Issue or Essential* **Keynote: Prof. Tim Lang, University of London, UK**
15:00	*Nutrigenetic diet intervention is related with greater weight and fat mass loss in subjects with abdominal obesity: the INNUPREC study* **Alondra Mora-Jiménez**, University of Guadalajara, Mexico
15:20	*Nutrigenetics as a tool for prevention and control of non-communicable diseases: impact on cardiovascular risk* **Dr. Maria Vranceanu**, University of Arad, Romania
15:40	*Partially replacing animal-based protein foods with plant-based protein foods: a systematic review of randomised controlled trials in healthy adult populations* **Aoife Courtney,** University College Dublin, Ireland
16:00	*Including fruit juice as one of the 5-a-day: A randomised controlled trial exploring the impact on adherence to guidelines and metabolite responses* **Dr. Courtney Neal,** Newcastle University, UK
16:20	New NuGO members, NuGO Developments, ECN promotion
18:00	Welcome Reception & Networking

Tuesday, 23rd September 2025

Session 2: Diet and the microbiome: lessons learned

Chairs: Dr. Sergio Polakof & Dr. Suzan Wopereis

09:00	*Diet-microbiome interactions - moving beyond microbiome composition to activity* **Keynote: Prof. Henrik Munch Roager, University of Copenhagen, Denmark**
09:45	*Healthy eating as a Microbiome host* **Keynote: Prof. Jens Walter, University College Cork, Ireland**
10:30	Coffee Break
11:00	*Personalized dietary fibre mixtures based on ex vivo microbial SCFA production improve HOMA-IR, but not peripheral insulin sensitivity, in individuals with prediabetes and overweight/obesity* **Dr. Emanuel Canfora**, Maastricht University, Netherlands
11:20	*Predicting variability in human oral and gut microbiota response to starch - implications for precision nutrition* **Dr. Angela C. Poole**, Cornell University, USA
11:40	*Unveiling multi-omics insights for precision nutrition in type 1 diabetes and obesity-associated type 1 diabetes among the pediatric population in Qatar.* **Shaikha Al-Abduljabbar,** Sidra Medicine & Hamad Bin Khalifa University, Qatar
12:00	*Dietary fibre-specific effects on serum and faecal bile acids profile and associations with the gut microbiota: a randomised, controlled dietary intervention in healthy participants* **Jiemin Fan,** Newcastle University, UK
12:20	*Chewing behavior and bolus particle size of rice shape gut microbiota functionality and microbial metabolite signatures* **Dr. Zhen Liu,** Wageningen University, Netherlands
12:40	Lunch & Poster Session

Session 3: Can AI help bridge precision & public health nutrition?

Chairs: Prof. Baukje de Roos & Prof. Lynn Vanhaecke

14:00	*Big data in nutrition is here: Are we AI ready?* **Keynote: Prof. Diana Thomas, West Point, USA**
14:45	*Targeted PRECIsion NUTrition Strategy to Prevent Chronic Metabolic Diseases: A tissue-specific metabotype approach* **Prof. Ellen E. Blaak,** Maastricht University, Netherlands
15:05	*Unraveling the Power of Vitamin D Mechanistically: How Long-Term Supplementation Shapes Immune Health* **Ranjini Ghosh Dastidar, Institute of Animal Reproduction and Food Research, Polish Academy of Sciences, Poland**
15:25	Coffee Break
16:00	*Precision Nutrition: Predictive Model of Appendicular Skeletal Muscle Mass in non-institutionalised people aged 65 and over* **María Benavent**, CEU Universities, Spain
16:20	*Bridging Mechanisms Across Diseases: A Multi-Dimensional Mapping Approach with Potential for Broader Omics Integration* **Dr. Lena Möbus,** Tampere University, Finland
16:40	**Sponsor's Talk** *Metaproteomics: a new powerful omics tool. Insights from a pilot study on fermented fecal samples supplemented with prebiotics* ***Morten Danielson,*** Clinical Microbiomics AS, Denmark
19:00	ECN social event – offsite PI social event – Portmarnock Hotel

Wednesday, 24th September 2025

Session 4: Nutrition and the brain throughout the life course: insights from molecular mechanisms to clinical applications

Chairs: Prof. Carsten Carlberg & Prof. Vibeke Telle-Hansen

09:00	*Omega-3 fatty acid, cognition and dementia: It's all in the timing* **Keynote: Prof. Anne Marie Minihane, University of East Anglia, UK**
09:45	*Food for thought: Plant-rich foods and cognitive health during ageing* **Keynote: Prof. Claire McEvoy, Queen's University Belfast, UK**
10:30	Coffee Break
11:00	*Is there an interplay between fat and umami taste perception and what is the role of CD36 gene? A preliminary overview* **Francesco Piluso**, Lake Lucerne Institute, Switzerland
11:20	*A series of n-of-1 intervention studies investigating the effects of tea on mood reveal that the interventions and participant-specific factors influence overall mood and relaxation* **Edward Payne**, The Rowett Institute, University of Aberdeen, UK
11:40	*Cross-study metabolomics data integration for the identification of common metabolic syndrome phenotypes* **Dr. Estelle Pujos-Guillot,** INRAE, France
12:00	*The effect of Ramadan fasting on the methylation patterns in pediatric metabolic dysfunction-associated steatotic liver disease (MASLD)* **Salma Hayder Ahmed,** Sidra Medicine, Qatar
12:20	Lunch & Poster Session

Session 5: Nutrition and inflammation: friend or foe?

Chairs: Prof. Rosita Gabbianelli & Assistant Prof. Yiannis Mavrommatis

14:00	*Diet metabolism and innate immune training* **Keynote: Prof. Helen Roche, University College Dublin, Ireland**
14:45	*Personalised inflammaging and nutrition* **Keynote: Prof. Claudio Franceschi, University of Bologna, Italy**
15:30	Coffee Break
16:00	*Impact of dietary habits on epigenetic age acceleration: evaluating the intermediary role of inflammation* **Dr. Laura Bordoni**, University of Camerino, Italy
16:20	*Deciphering inflammatory cues from the microbiome: Bacterial membrane vesicles produced in response to prebiotic and antibiotic intake* **Jari Verbunt**, Maastricht University, Netherlands
16:40	*Beyond Inflammation: Vitamin D and LPS Co-Stimulation Uncovers Novel Gene Networks in Human Monocytes* **Mariusz Jankowski, Institute of Animal Reproduction and Food Research, Polish Academy of Sciences, Poland**
17:00	*Sex and Photoperiod Modulate Hepatic Oxidative Stress in Cafeteria Diet-Induced Obese Rats: Insights into Antioxidant Activation, Circadian Rhythms, and Melatonin Regulation* **Aina Gironès-Garreta,** Universitat Rovira i Virgili, Italy
17:20	NuGO 2026 Announcement
19:30	Conference Dinner – Portmarnock Hotel

Thursday, 25th September 2025

Session 6: Ultra-processed foods - what's the molecular evidence

Chairs: Associate Prof. Aifric O Sullivan

09:00	*Opportunities and Challenges for Processed Food and Health; Results from the RESTRUCTURE Trial* **Keynote: Prof. Ciarán Forde, Wageningen University & Research, Netherlands**

Session 7: Selected Abstracts

Chairs: Associate Prof. Aifric O Sullivan

09:45	*The Health Benefits of Fruit and Vegetable Byproducts: A Systematic Scoping Review* **Brendan Kesler,** The Rowett Institute, University of Aberdeen, UK
10:05	*Caloric restriction-induced metabolic adaptation associated with amount of body weight lost – results from the LION study* **Dr. Carmen Blanken**, Technical University of Munich, Germany
10:25	*Developing a 3D model of the bone marrow niche to investigate the influence of folate on childhood leukaemia-initiating events* **Dr. Jessica R Saville,** Northumbria University, UK
10:45	*Obesity-Satiety Phenotypes and the Gut Microbiome: A Pilot Study* **Dr. Kerstin Schorr,** TNO, Netherlands
11:05	*To model or not to model? Lessons on using an in silico-in vitro approach to identify foods that support the infant colonic microbiota* **Vitor Geniselli da Silva,** Riddet Institute, New Zealand
11:25	Awards announcement
11.45	Conference Close

Keynote Speakers

Prof. Tim Lang, University of London, UK

Tim Lang is Professor Emeritus of Food Policy at City St George's, University of London. After hill farming in Lancashire in the 1970s, he's worked on food policy across health, environment, politics and culture. Since writing *Feeding Britain* (Pelican 2021), he's focused on the state of civil food resilience, producing the *Just in Case* report for the UK National Preparedness Commission, urging more attention to public preparedness for food shocks.

Sustainable Diets: A side Issue or Essential

The presentation explores difficulties in translating the notion of a sustainable diet into policy and reality. Initially, 'sustainable' meant what the 1987 Brundtland report indicated, the sweet spot where the good economy, society and environment meet. Then gradually, 'sustainable' became synonymous with the environment only. This, the paper proposes, is a mistake. Too often it means sustainability is put into the 'too difficult to handle' box. But reality now intervenes due to food's climate impact and dependencies. The paper argues the environment is inevitably a composite of factors: not just climate / carbon, but biodiversity, water, land use, and socio-economic drivers shaping diet's impacts. These all demand attention. They interact. Whether we like it or not, 'sustainable diets' require a multi-criteria approach. Good diets, the ideal all societies nominally pursue, are a composite goal: health, environment, economy, society, morality, and more. This is no surprise to consumers; most already apply their own multi-criteria when nominally choosing what they eat – if they have that luxury. The paper then considers the blocks on pursuing the challenge of sustainable diets. It considers the lock-ins: price, commercial interests, culture, life-space. Different policy models for change exist including rational appeals, 'nudges', hidden determinants and more recently the significance of shocks. The paper suggests that while we discuss such matters, events are beginning to narrow the room for manoeuvre on sustainable diets. The food system is being drawn into sober policy terrain where food is again a matter of structural conflict. This is unlikely to end debate about sustainable diets but intensify it. Analyses of shocks affecting food systems stress that the best route to resilience (post-shock recovery) is to be more sustainable in the first place.

Assoc. Prof. Henrik Munch Roager, University of Copenhagen, Denmark

Associate Prof. Henrik Munch Roager leads the Nutrition, Microbiome and Metabolomics group at Department of Nutrition, Exercise and Sports at the University of Copenhagen. He earned his PhD in intestinal metabolomics from the Technical University of Denmark in 2016. In 2020, he was awarded a Starting Grant, enabling him to establish his research group at the University of Copenhagen. Today, he leads an international and multidisciplinary team dedicated to elucidating the role of the gut microbiota in digestion and health. Methodologies include controlled dietary intervention- and cohort studies in both adults and infants, multi-omics analyses including microbiome and metabolomics, and advanced data integration. Throughout his career, Dr. Roager has received several prestigious accolades, including the American Society of Nutrition's E.L.R. Stokstad Award in 2022 and the British Nutrition Foundation's Drummond Early Career Scientist Award in 2019. Henrik Roager has published over 40 papers including corresponding and first authorships in high-ranking journals such as Nature Microbiology, Gut, and Nature Communications. Beyond his research, Dr. Roager is committed to making a greater impact on both the research community and the public. He has co-founded the Copenhagen Gut Microbiome Hub, developed an MSc course on the Gut Microbiome in Nutrition and Health, and is committed to science communication, frequently engaging with the public.

Diet-microbiome interactions - moving beyond microbiome composition to activity

No doubt that diet shapes the composition of our resident microbes in our gut, also called the gut microbiome. Drastic dietary changes have been found to induce changes in the gut microbiome composition. However, more moderate dietary changes most often result in subtle effects on the gut microbiome composition, suggesting that a healthy adult's gut microbiome is relatively stable. To advance the field of diet-microbiome interactions further, we are studying how substrate availability and physiology in the gut determine an individual's gut microbiome composition and activity and how microbial metabolites may mediate host-microbial cross-talk. During my talk, I will give three examples of our work.

I will discuss how dietary fiber affects microbial tryptophan catabolites in the gut. Microbial tryptophan metabolism results in the formation of metabolites, which have been linked to both detrimental and beneficial health effects. Based on both human data, in vitro, and in vivo

models, we found that the availability of dietary fiber directs tryptophan metabolism towards production of more beneficial catabolites. Not by changing the abundance of certain microbes, but by changing the activity of microbes via bacterial gene regulation. The implications are that changes in gut microbial composition are not a prerequisite for changes in microbial activity in the gut.

Secondly, I will discuss how differences in gut physiology and environment may be key for understanding individuality of the human gut microbiome. Using a wireless motility capsule, we monitored segmental and whole-gut intestinal pH and transit time in 50 healthy Danish adults. We found large inter-individual differences in pH and transit time, which explained a large proportion of the variability in the gut microbiome and urine metabolome. Our work shows that the gut microbiome composition, diversity and metabolism are strongly associated with intestinal transit time and pH – in other words – our bowel habits.

Finally, I will give examples from previous and ongoing work dedicated to understanding how microbial metabolites, such as aromatic lactic acids, in the infant gut, may be important for regulating host-microbial cross-talk during infancy.

In conclusion, both substrate availability and individual factors such as intestinal pH and transit time, are important factors linked to the gut microbial composition and activity. More emphasis on understanding the influence of dietary and gut environmental factors on the production of microbial metabolites is crucial for leveraging the potential of the gut microbiota to enhance health and prevent diseases.

Prof. Jens Walter, University College Cork, Ireland

Professor Jens Walter severs as the Professor for Ecology, Food, and the Microbiome at University College Cork and the APC Microbiome Ireland. He is a leading expert in the evolutionary ecology of the gut microbiome and its relationship with human nutrition. His research explores how ecological and evolutionary processes shape host-microbe interactions, aiming to translate microbiome science into therapeutic and dietary strategies. His team has led numerous human trials examining how fibre, diet and live microbes influence the gut microbiota and health. He has authored over 180 peer-reviewed publications and is recognized as a *Highly Cited Researcher* by Clarivate. Prof. Walter is the recipient of a prestigious *Research Ireland Research Professorship* for his program on Microbiome Restoration (*Microbe Restore*) program. As part of this work, Prof. Walter developed the NiMe™ diet—based on traditional, non-industrialized eating patterns—which demonstrated rapid and significant health benefits in a controlled human trial. His long-term goal is to develop targeted, evidence-based microbiome interventions to improve health and prevent chronic disease.

Healthy eating as a microbiome host

Chronic non-communicable diseases (NCDs) have reached epidemic proportions in industrialized societies, a trend clearly linked to Western-style dietary patterns and changes in the gut microbiome. Research in several animal models suggests that these industrialization-driven shifts in diet–microbiome interactions may actually cause the development of such pathologies.

In this presentation, I will examine what constitutes healthy eating from a microbiome science perspective, focusing on mechanistic evidence that establishes host–microbe interactions as key mediators of diet's physiological effects. I will use this knowledge to address ongoing controversies in the nutrition field and to support the development of innovative dietary strategies.

I will also present findings from a recently published human trial that tested a microbiome restoration approach mimicking key characteristics of non-industrialized dietary patterns (the NiMe™ diet). This diet improved several microbiome features disrupted by industrialization and favourably shifted microbiota-derived plasma metabolites implicated in the etiology of chronic NCDs. It also led to significant cardiometabolic benefits, many of which could be accurately predicted by baseline and diet-responsive microbiome features.

Overall, the evidence supports a central role for the gut microbiome in mediating the physiological effects of diet, offering an exciting opportunity to systematically incorporate microbiome science into nutrition research to improve dietary strategies and recommendations.

Prof. Diana Thomas, West Point, USA

Diana M. Thomas received her Ph.D. from the Georgia Institute of Technology in 1996. She then completed a National Research Council funded post-doctoral fellowship at the United States Military Academy and the Army Research Laboratory. In 2000, she joined the faculty of the Montclair State University where she was a professor of mathematics and the director of the Montclair State University Center for Quantitative Obesity Research. Dr. Thomas is currently a professor of mathematical sciences at the United Military Academy at West Point. Dr. Thomas has been an active research mathematician for over 25 years with a focus on nutrition and obesity related modeling. She has worked with large complex and high dimensional datasets and co-invented the remote weight loss program, SmartLoss™, which has been clinically applied world-wide to guide and improve individual patient weight loss adherence through smartphone technology. Dr. Thomas has published over 150 peer-reviewed articles and has led the development of over 10 freely accessible health calculators. She is an associate editor for the world's top ranked journal for original research in nutrition, the American Journal of Clinical Nutrition and co-edits the series "Best (but oft-forgotten) practices", which consists of methodologic commentaries or statistical tutorials. She also serves as an editor for Nutrition and Diabetes and the European Journal of Clinical Nutrition. Dr. Thomas is currently the PI of the Artificial Intelligence, Data Engineering & Machine Learning (AIDE-ML) Center for the Nutrition for Precision Health Consortium which she served as a co-chair for the Steering Committee. She has held governance positions in the Obesity Society, the American Society of Nutrition, and the Mathematical Association of America. Dr. Thomas holds the 2012 Mathematical Association of American of NJ Distinguished Teaching Award, the 2015 Obesity Society George Bray Founder's Award, and the 2023 American Mathematical Society Mary P. Dolciani Prize for Excellence in Research.

Big data in nutrition is here: Are we AI ready?

I will present an overview of Precision Nutrition and the role of AI and Big Data in this space. One question surrounds how we combine messy multi-modal complex data within one model to make predictions of response to diets . To this end, I will show the mathematical steps on how two data sources, CGM and Questionnaire data are distilled and combined within one machine learning prediction model.

Prof. Anne Marie Minihane, University of East Anglia, UK

Anne-Marie and team's research programme investigates the impact of dietary components (omega-3- fatty acids and a Mediterranean-style dietary pattern) and hormone replacement therapy (HRT) on cognitive health and dementia risk. A particular focus is the molecular and physiological basis for the interactive impact of menopause and an *APOE4* genotype (25% of the UK population) on neuropathology and overall brain health and examining the ability of the omega-3-fatty acid DHA to mitigate the accelerated brain ageing in *APOE4* females. Recent work has focused on the menopausal transition as a window of opportunity for intervention. She is co-founder of Norwich Institute of Healthy Ageing (NIHA) which is focused on providing agency, and the capability, opportunity and motivation to individuals and communities to adopt heathier behaviours aligned with recommendations.

In addition, at UEA, Anne Marie contributes to the teaching of the Medical and Bioscience students, in the area of nutrition and disease prevention and therapeutics. She is a member of a number of external advisory and editorial boards. When away from science she enjoys the outdoors, and in particular running, good food, social and historical fiction, and travel and its planning.

Omega-3-fatty acid, cognition and dementia: it's all in the timing

The brain is enriched in the omega-3 fatty acid, docosahexaenoic acid (DHA) which constitutes about 15% of total lipids and 40% of select synaptic lipids, compared to <5% in most other major organs. In addition to its structural functions, DHA modulates inflammation, synaptic plasticity, glucose metabolism, neurogenesis and neurotransmission. Although the concentrations of EPA in the brain are several-fold lower than DHA, it efficiently crosses the blood brain barrier (BBB), is enriched in microglia, and is likely to be an important mediator of microglial and neuronal function.

Preclinical and prospective cohort data is supportive of a positive impact of increase EPA+DHA intake and status on neurocognitive function and dementia risk. However, RCT evidence is inconsistent with determinants of responsiveness poorly understood.

To date human RCTs focused on cognition and reducing dementia risk have typically fed DHA-rich supplements. However, there is emerging evidence that EPA is also important. Furthermore,

it is likely that timing is important, with bioefficacy affected by life-stage and degree of pathological progression.

The presentation will review the available evidence on the relationship between EPA and DHA intake and status and cognitive well-being and consider the impact of *APOE* genotype (main common genetic risk factor for dementia which also affects fatty acid metabolism), menopausal status and timing on the response to intervention. A better understanding of dose-response relationships in sub-groups would inform the emergence of more targeted omega-3 fatty acid recommendations.

Prof. Claire McEvoy, Queen's University Belfast, UK

Dr Claire McEvoy is Reader in Nutrition and Ageing Research at Queen's University Belfast, Centre for Public Health, School of Medicine whose research investigates the potential of plant-based diets for preventing or slowing progression of frailty and dementia. She conducts epidemiological investigations of dietary patterns and disease risk across the life-course, and community-based interventions targeting dietary behaviour change to improve nutritional status and health outcomes particularly in high-risk populations. She was project co-ordinator for the European Healthy Life Healthy Diet (HDHL) Joint Programming Initiative 'PROMED-COG' consortium to understand the balance between diet and exercise to combat undernutrition and promote healthy neurocognitive ageing (2020-2024). Dr McEvoy is the Regional Alzheimer's Research UK Co-ordinator in Northern Ireland and Co-Chair of Dementia Research Network Ireland (Health Research Board) to promote cross border collaboration and increase capacity in brain health research. She is Alumni Atlantic Fellow for Equity in Brain Health at the Global Brain Health Institute, University of California, San Francisco and TCD.

Food for thought: Plant rich foods and cognitive health during ageing

Modifying dietary behaviour could be a promising way to enhance cognition and delay or prevent dementia in later life. Several dietary factors influence dementia risk in humans, for example, vitamin E, B vitamins, omega-3 fatty acids, while high intake of saturated fat accelerates cognitive decline. Plant-based dietary patterns, particularly the Mediterranean diet, may offer neuroprotective effects, although the underlying mechanisms are not yet fully understood. Several pathways have been proposed, for example, improving vascular function and decreasing oxidative stress, inflammation and neuronal damage. Given that the pathological processes underlying dementia begin many years before clinical symptoms appear, it is critical to examine dietary influences on brain health across the life course to inform prevention strategies.

Our research has shown neuroprotective benefits of plant-diets across several populations including in older adults, in female twins accounting for shared genotype and early-life environmental exposures, and among those with higher genetic risk in the UK Biobank. We have also shown that improving nutritional status in older community dwelling adults using a personalised plant-based dietary intervention led to improvement in cognitive performance. Taken together, our data indicate that even modest adherence to a plant-based diet has benefit for cognitive performance and dementia risk during ageing and provides important understanding of the life course effect of diet on cognitive health. Further research is needed to investigate diet-associated neurological change from the earliest through to latest stages of cognitive decline and insight into mechanisms involved in diet-induced cognitive change.

Prof. Helen Roche, University College Dublin, Ireland

Helen initially trained in Human Nutrition and Dietetics, followed by Molecular Medicine. Her Nutrigenomics team focuses on Precision Nutrition – specifically the impact of diet on metabolism and inflammation, in obesity, metabolic dysfunction associated fatty liver disease (MAFLD) and obesity related cancer. Nutrigenomics uses state-of-the-art 'omics' to investigate the molecular effects of diet on health – to provide hard evidence. Whilst nutrition plays a critical role in health and disease, too often the mechanistic basis is lacking – we seek to fill that evidence gap.

In Europe, Prof Roche has led several initiatives relating to Food, Nutrition and Health. She chaired the Scientific Advisory Board of the European Healthy Life Healthy Diet Joint Programming Initiative (2015-2019). She advises several UK, Netherlands and US grant agencies, in relation to Food and Health Research Strategy. She was a board member of the Royal College of Surgeons Ireland Hospital Group.

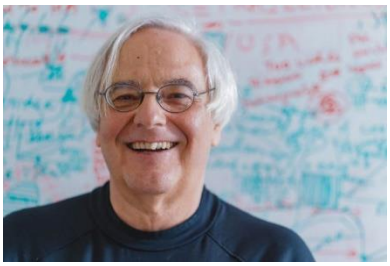
Prof Roche's research team are funded by a number of agencies. Her Research Ireland Investigator Award entitled 'Diet, Immune Training and Metabolism'. She is co-PI in Precision Oncology Ireland (POI) Roche's team are determining if/how the 'dietary environment' potentiates obesity related cancer risk. On-going research as part of the DAFM Funded PROTEIN-I Project is investigating the impact of personalised plant protein and exercise interventions innate immune function in older persons at risk of malnutrition.
Helen has supervised more than 30 PhD scientists and a similar number of post-doctoral researchers.

Diet metabolism and innate immune training

Over the last 10 years, our understanding of the inter-relationship between metabolism and inflammation in innate immune function has advanced greatly. Innate immune training is a new concept wherein monocytes and macrophage 'remember' and reflect previous insults / metabolic milieu. Metabolic dysfunction-associated steatotic liver disease (MASLD) and metabolic dysfunction-associated steatohepatitis (MASH), both involve a complex interplay between metabolic complications and innate immune dysfunction, arising in part from poor diet and obesity. Targeted reprogramming of innate immune responses, such as trained immunity, has the capacity to modulate systemic inflammation, but the impact of metabolic dysregulation on the functional plasticity of immune cells is as yet unclear.

Current work is investigating the inter-relationship between metabolism and inflammation in MASLD and MASH, as well as older people at risk of malnutrition. This review will explore how clinical, metabolic and inflammatory phenotypes influence trained immunity responses with a view to understanding mechanisms to underpin potential precision nutrition strategies that better define ‘the right diet for the right person at the right time’. The goal being to enhance understanding, underpinning more targeted nutritional approaches, which may improve efficacy of personalised nutrition interventions.

Prof. Claudio Franceschi, University of Bologna, Italy



Prof. Claudio Franceschi is Professor Emeritus at the University of Bologna (UNIBO), Italy, and Head of the Laboratory of Systems Medicine and Healthy Aging at Lobachevsky University, Russia. He previously held professorships in Immunology at the Universities of Padua, Modena, and Bologna (1980–2013). A pioneer in the biology of aging, he is internationally renowned for proposing the "inflammaging" theory, identifying key features of immunosenescence, and advancing multi-omics research on human longevity. He has authored around 900 scientific publications (117,000+ citations; h-index: 155, Google Scholar, July 2025).

Prof. Franceschi is Editor-in-Chief of *Ageing Research Reviews* (IF 2025: 12.4) and co-editor of the *Handbook of Immunosenescence* (Springer, 2019). He has delivered keynote lectures at global forums including Gordon Conferences and Cold Spring Harbor Symposia.

He coordinated major EU projects such as GEHA, NU-AGE, and PROPAG-AGEING, and led the Russian MEGAGRANT "DPM-AGEING." His contributions have earned him honorary doctorates from Argentina, France, and Russia, and prestigious awards including the 2023 Lifetime Achievement Award in Immunology and Aging Research (Canada).

Prof. Franceschi is a member of the Accademia delle Scienze dell'Istituto di Bologna and the Venetian Institute of Science, Humanities and Art (IVSLA).

Personalised inflammaging and nutrition

Inflammaging describes a chronic, systemic, low-grade inflammatory state recognized as a major risk factor for age-related diseases (ARD) and a pivotal convergence point of multiple biological mechanisms involved in aging. Inflammaging is fueled by a complex interaction of genetic, lifestyle and environmental factors. I will discuss the heterogeneity of inflammaging, proposing that it emerges as a consequence of each individual's lifelong exposures to inflammatory stimuli, shaped by a unique combination of genetics, lifestyle, socioeconomic conditions and environmental factors such as infections and pollution. Through this lens, then I will discuss how to measure inflammaging, describing the development of inflammatory clocks which quantify inflammatory age and show strong associations with ARD incidence, as well as how other aging clocks intersect with inflammaging. Research has increasingly focused on ways to mitigate/delay inflammaging with particular attention to interventions at relatively low cost and with great potential feasibility involving nutrition and lifestyle, such as Mediterranean Diet and physical activity. These interventions not only have the capability to reduce inflammation,

but they may also foster healthy aging by improving gut microbiota composition, influencing gene expression and promoting epigenetic rejuvenation. Clinical evidence from the NU-AGE study supports the link between gut microbial composition, diet, and inflammatory status in older adults. Interestingly, the NU-AGE trial revealed variations across participants from the five countries and between genders, as well as a large heterogeneity of responsiveness to the Mediterranean diet (MedDiet) at the individual level. Accordingly, MedDiet can be considered an anti-inflammatory diet, but future trials are needed to evaluate efficacy at individual level. This heterogeneity highlights the importance of personalized nutritional approaches as genetic factors, baseline inflammatory status and microbiome composition may all influence the individual's response to dietary interventions, paving the way for future personalized recommendations. To this regard, I will also consider other interventions that may counteract inflammaging, including geroprotectors. Overall, available data suggest that MedDiet is capable of fine-tuning the balance between pro- and anti-inflammaging, delaying age-related diseases, likely acting as a form of chronic hormesis. Hormesis assumes that extremely low doses of poisonous compounds such as those synthesized/present in most eatable vegetables can exert a mild stress that in turn would enhance the body cellular and molecular defense mechanisms. Indeed, MedDiet contains bioactive compounds, or "hormetins," such as resveratrol, quercetin and phenolic antioxidants, which activate pathways like Nrf2, NF-κB, mTOR, and sirtuins. Interestingly, certain hormetins are also considered geroprotectors and senolytics. Finally, I will propose that deepening our knowledge of the individual nature of inflammaging stands to enhance our understanding of personalized aging trajectories and inform precision interventions, including personalized nutrition.

Prof. Ciarán Forde, Wageningen University & Research, Netherlands

Professor Ciarán Forde is the Chair of Sensory Science and Eating Behavior at the Division of Human Nutrition and Health at Wageningen University and Research in the Netherlands. He leads research on how food sensory properties influence eating behaviors, energy intake and metabolism across the life-span. Prof. Forde coordinates undergraduate and graduate education in Sensory Science and Nutrition Behaviour at Wageningen, and has published widely on topics in sensory, nutrition and metabolism. He is an Executive Editor for the journal *Appetite* and Section Editor ('Nutrition behaviour') for the *European Journal of Nutrition*. He also holds editorial board positions at *Nutrition Bulletin*, *Journal of Future Food* and *Journal of Texture Studies*. Prof. Forde has spent the past 25 years conducting research in academic, public and private sector research roles in the UK (GSK), Australia (CSIRO) and Switzerland (Nestlé Research) and Singapore (National University of Singapore/A*STAR). He received his BSc (Hons) in Food Chemistry and a PhD in Sensory Science from the Department of Human Nutrition at University College Cork in Ireland.

Opportunities and Challenges for Processed Food and Health; Results from the RESTRUCTURE Trial

In recent years, there has been a growing research interest in the possible role of ultra-processed foods (UPFs) in a wide range of noncommunicable diseases. Almost all of this research to date has been correlational in nature. A first attempt to explore how UPFs might contribute to one of the many implicated in higher energy intakes involved a crossover randomized control trial (RCT) conducted under metabolic ward conditions and that compared *ad libitum* energy intake on diets high or low in UPFs. The diet high in UPF's led to greater energy intake and weight gain when compared to the diet low in UPFs, and the data showed that the higher UPF arm was had both higher energy density and was consumed with an increased eating rate (g/min). Food texture matters when it comes to eating rate and eating rate is an established contributory factor to weight gain. Further research is needed to clarify the mechanisms underlying observed differences in energy intake. The RESTRUCTURE (www.restructureproject.org) randomized controlled trial aimed to determine the effect of food texture-based differences in eating rate (ER) on *ad-libitum* energy intake (kcal/day) from UPF diets. Using a semi-residential design, across two 14-day diet periods, we compared average daily energy intakes on a diet based on textures that would be consumed more slowly, and a 14-day diet based on textures consumed

more quickly (Slow-ER vs. Fast-ER), with a 14-day washout period between diets. Diets were composed of >95% UPF's (Nova-4), and matched for portions served, non-beverage energy density, meal variety, volume and liking. Participants (N=41) *ad-libitum* food intake (g and Kcals) was measured across a total of 3,444 meals served, and 1,325 menu days. The diets differed by an average of 24 g/min in eating rate, with a consistent reduction in eating rate across all participants. Dietary energy intake was on average 369 kcal/day (95% CI: 221, 517) lower on the Slow-ER UPF diet compared to the Fast-ER UPF diet (main effect; $F(1,1051) = 23.98$, $p<0.001$). Texture-based differences in meal ER resulted in consistent differences in eating rate and energy intake that were sustained for the duration of each 14-day diet intervention, with a cumulative net difference of 5,232 kcals between the two diets. This resulted in a reduction of 0.43kg of fat mass when consuming the slower-UPF diet. Differences in observed daily energy intakes resulted from meal texture-based differences in ER, and were not attributable to meal liking, familiarity or macronutrient intakes (all, $p>0.05$). Secondary comparisons revealed no changes in satiety sensitivity or glucose homeostasis between the two diet arms. Our findings help to explain previously observed differences between ultra- and minimally processed diets, and highlight for the first time that texture-derived differences in ER significantly moderate sustained differences in dietary energy intake from diets comprising ultra-processed foods. results highlight that meal texture can be used to moderate consumers eating behaviours and create new opportunities to better regulate energy intake using from foods using sensory cues.

Funding: RESTRUCTURE is a public-private partnership on precompetitive research on the influence of food texture and eating rate on energy intake and is supported by the Dutch Top-Consortium for Knowledge and Innovation Agri & Food (TKI-Agri-food). For more information go to https://restructureproject.org/.

Session 1: What is a healthy and sustainable diet? Monday, 22nd September @ 2 pm

Nutrigenetic diet intervention is related with greater weight and fat mass loss in subjects with abdominal obesity: the INNUPREC study

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Background: Abdominal obesity (AO) is characterized by an excessive adipose tissue in the abdominal region that can be harmful. Factors such as diet, lifestyle, and genetics contribute to its development. Thus, the interaction between genetic variants and the response to diet can influence metabolic processes related to AO. Therefore, it has been considered that the implementation of nutrigenetic interventions could have a better metabolic impact. Objective: To compare the effect of a nutrigenetic intervention in anthropometric and cardiometabolic biomarkers vs. control intervention in subjects with AO.

Methods: In a randomized controlled clinical trial a total of 58 subjects with AO were included to follow control or nutrigenetic diet for 2 months. For subjects on the nutrigenetic diet, a genetic profile was previously performed, which included 21 genetic variants. Body composition was analyzed by electrical bioimpedance in Inbody370. Biochemical measurements were obtained by dry chemistry (Vitros350) and insulin levels using a LIASON® immunoassay kit. Statistical analysis was performed in SPSS software v29. A p value <0.05 was considered significant.

Results: At the end of the intervention, there was a greater weight reduction in the nutrigenetic group (-3.12±2.4 vs. -1.82±2.0kg), and fat mass (-2.64±2.2 vs. -1.63±1.8kg) vs. control group. In particular, women in the nutrigenetic group had a greater reduction in fat mass (-2.62±2.5 vs. -1.25±1.4kg). Regarding biochemical variables, the nutrigenetic group has a greater reduction in serum triglycerides (-12.7±40.6 vs. -9.48±53.1 mg/dL), and insulin levels (-4.55±8.6 vs. -5.88±7.8 µUI/mL) vs. control group. Besides, the group with a nutrigenetic diet decreased the percentage of subjects with high triglyceride and glucose index.

Conclusions: A nutrigenetic diet is associated with greater weight and fat mass loss, as well as a decreased prevalence of elevated triglyceride/glucose ratio vs. control diet.

Nutrigenetics as a tool for prevention and control of non-communicable diseases: impact on cardiovascular risk

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Objectives: Every two seconds, in the world, a person who has not yet reached the age of seventy dies due to a chronic non-communicable disease (NCDs). The first objective of this study was to assess the improvement of biochemical markers: total cholesterol (TC), HDLc, triglycerides (TG) and homocysteine (Hcys) plasma levels as risk factors for cardiovascular diseases, over 24 weeks of diet and 18 months follow up to understand if the nutrigenetic diet could be a valid tool for prevention of NCDs. The second aim was to assess the weight loss performance on nutrigenetic group by analyzing the interaction between APOA2 rs5082 and FTO rs9939609 genes polymorphism and diet.

Method: The investigators followed 114 overweight and obese subjects in a 2-stage process. 1) A 24-week dietary intervention. The subjects self-selected whether to follow a ketogenic diet (n = 53) or a nutrigenetic diet utilizing information from 28 SNPs (n = 61). 2) After the 24-week diet period, the subjects were followed for 18 months using standard guidelines for the Keto group (KG) vs standard guidelines modified by nutrigenetic advice for nutrigenetic group (NG). Fasting venous blood samples were taken at baseline, 24-weeks, and 104-weeks to determine TC, LDLc, HDLc, TG and Hcys.

Results: To the end of the study, TC levels were much better on the NG, 189.45 mg/dL when compared with KG, 210.33 mg/dL ($p < 0.0001$), HDLc presented higher level on the NG, LDLc for the NG was 98.82 mg/dL compared with 105.21 mg/dL in the KG. Also, Hcys and TG levels had better values in the NG. Among the subjects of NG only the carriers of risk allele for APOA2 rs5082 lost less weight compared with non-carriers.

Conclusions: Precision nutrition has been demonstrated to produce notable clinical and financial benefits. Furthermore, nutrients have a crucial role in the prevention and treatment of chronic degenerative diseases by directly and indirectly influencing and regulating gene activity.

Partially replacing animal-based protein foods with plant-based protein foods: a systematic review of randomised controlled trials in healthy adult populations

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Increasing our intake of plant-based protein may help improve both population and planetary health. The current evidence base, however, is limited. The aim of this systematic review was to identify randomised controlled trials that partially replaced animal-based protein with plant-based protein in healthy adults.

On December 5, 2024, three electronic databases were searched to identify relevant studies (Embase, PubMed, Cochrane CENTRAL). From 51 full-text articles, seven papers (four dietary interventions; mean duration 9.5 weeks; sample size range n=36–136) met the inclusion criteria. Two trials assessed nutrient status.

One 12-week trial observed a significant decrease in vitamin B-12 status in the PLANT group (70% plant protein) vs the 50/50 (50% plant protein) and ANIMAL group (30% plant protein, p< 0.001), with no significant changes in folate or iron status. Two interventions evaluated bone and mineral metabolism, the 12-week trial reported higher concentrations of biomarkers of bone formation (p=0.007), resorption (p < 0.001), and regulatory hormones (p=0.018), and lower urinary phosphate (p≤0.001) and calcium excretion (p<0.001) in PLANT vs ANIMAL. In contrast, a 6-week trial observed no significant changes in bone turnover. Participants with higher plant protein intake exhibited lower LDL cholesterol (2 trials, p=0.002–0.003), total cholesterol (1 trial, p=0.003), and body weight (2 trials, p< 0.001, p=0.037) compared to those consuming more animal protein. One 4-week study reported reduced meat intake (−63 g/day; 95% CI: 44–82; p<0.0001) and lower estimated greenhouse gas emissions (p<0.0001) in the intervention vs control group.

In conclusion, this review identified evidence that replacing animal protein with some plant-based sources may improve certain biomarkers of cardiovascular disease risk and reduce the environmental impact of dietary intake. Mixed findings for nutrient status and bone and mineral metabolism highlight the need for further research.

Including fruit juice as one of the 5-a-day: A randomised controlled trial exploring the impact on adherence to guidelines and metabolite responses

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Fruit juice overcomes many barriers linked to low fruit and vegetable intake, as it is convenient, affordable and widely available. Yet, its contribution to the UK 5-a-day fruit and vegetable targets is contested because of concerns regarding its sugar content. This randomised controlled trial explored the impact of 5-a-day advice with and without the inclusion of fruit juice on self-reported and objective biomarker measures of fruit and vegetable intake, mood, gut symptoms and metabolic responses.

Forty-two participants aged 18-65y who habitually consumed ≤2 portions of fruit and vegetables/d were assigned to one of the following four-week intervention arms: (1) Habitual diet control, (2) Whole fruit and vegetable (FV), or (3) Fruit and vegetable plus fruit juice (FV+FJ). Each participant received a weekly voucher, and the FV and FV+FJ groups received a co-designed educational booklet. Post-intervention outcome data were compared between groups using ANCOVA, controlling for pre-intervention scores, age and BMI.

Participants in the intervention groups increased their daily fruit and vegetable intake, with FV (adj. M=8.27, SE=0.54; p < 0001, d=2.96) and FV+FJ (adj. M=6.55, SE=0.54; p<.0001, d=2.10) groups consuming more portions/d than the control group (adj. M=2.37, SE=0.54) but not significantly different from one another (p=.09, d=0.86). Post-intervention depression scores (PHQ9) were significantly lower in FV+FJ (p=.02, d=1.12), but not FV (p=.05, d=0.97), versus control.

We are now objectively validating dietary changes by assessing circulating plasma ascorbic acid and serum carotenoid concentrations and exploring comprehensive metabolite responses via a targeted NMR metabolomics approach. These data will be available for presentation at NuGO Week. Clinical Trials Registration: NCT06628401.

Session 2: Diet and the microbiome: lessons learned - Tuesday 23rd September @ 9:00 am

Personalized dietary fibre mixtures based on ex vivo microbial SCFA production improve HOMA-IR, but not peripheral insulin sensitivity, in individuals with prediabetes and overweight/obesity

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Introduction: The gut microbiota ferments dietary fibres into short-chain fatty acids (SCFAs), which can improve metabolism. However, long-term supplementation with single fibres shows inconsistent effects, highlighting individual differences in microbial fermentation. This study aimed to examine the effect of a personalized fibre mixture (PFM), designed to enhance SCFA production, on insulin sensitivity.

Methods: In a randomized, double-blind, placebo-controlled study, 44 overweight/obese adults (BMI 28–40 kg/m^2, aged 35–70) with prediabetes and/or insulin resistance were assigned to receive either 24g/day of an in vitro-defined PFM (n=22) or control fibre (galacto-oligosaccharides, n=22) for 12 weeks. PFMs were tailored based on ex vivo SCFA output using the validated, dynamic TIM-2 colon model. The primary outcome was peripheral insulin sensitivity, assessed via hyperinsulinemic-euglycemic clamp.

Results: No differences in peripheral insulin sensitivity (M-value) were observed. However, PFM reduced fasting insulin (Δ -0.98 ± 2.48 vs 0.90 ± 2.96 mU/L, P=0.037), HOMA-IR (Δ -0.27 ± 0.65 vs 0.22 ± 0.76, P=0.036), body weight (Δ -0.28 ± 2.3 vs 1.37 ± 1.5 kg, P=0.014), and BMI (P=0.017), with a trend toward lower body fat mass (P=0.073) compared to placebo. No significant changes were observed in energy expenditure, substrate oxidation, blood lipids, inflammatory markers, gut hormones, or SCFA levels. Conclusion: While PFM did not improve peripheral insulin sensitivity, it improved fasting insulin, HOMA-IR, and body weight, suggesting benefits for hepatic insulin sensitivity. Personalized fibre strategies may support metabolic health in people at risk for type 2 diabetes.

Predicting variability in human oral and gut microbiota response to starch - implications for precision nutrition

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Starch consumption affects both oral and gut microbiota. Resistant starch (RS) can have beneficial effects on metabolic health, but the response, in terms of effects on the gut microbiota and host physiology, varies between individuals. Factors predicting the response to RS are not yet established and would be useful for developing precision nutrition approaches that maximize the benefits of dietary fiber intake. We sought to identify predictors of gut microbiota response to RS supplementation.

We conducted a seven-week crossover study with 59 individuals completing the study. Participants consumed RS type 2, RS type 4, and a digestible starch, for ten days each with five-day washout periods in between. We collected fecal and saliva samples and food records during each treatment period. We performed 16S rRNA gene sequencing and measured fecal short-chain fatty acids (SCFAs), salivary amylase (AMY1) gene copy number, and salivary amylase activity. Salivary amylase initiates starch breakdown, and salivary amylase gene copy number (AMY1 CN) ranges between 2-20 in humans.

Neither AMY1 CN nor salivary amylase activity were predictive of response to RS. Treatment order (the order of consumption of RS2 and RS4), alpha diversity, and a subset of ASVs were predictive of SCFA changes after RS supplementation. Surprisingly, SCFA concentrations increased the most during digestible starch supplementation. Based on our findings, prior dietary fiber intake and gut microbiome composition would be informative if assessed before recommending RS supplementation. We also collected saliva samples during this study and used these samples in an in vitro model. We grew biofilms in media with and without starch. We discovered that a gene-nutrient interaction affects the abundances of Atopobium and Veillonella, genera previously associated with cavities and periodontitis. Our findings have implications for both precision nutrition and precision dentistry.

Unveiling multi-omics insights for precision nutrition in type 1 diabetes and obesity-associated type 1 diabetes among the pediatric population in Qatar

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Type 1 Diabetes (T1D) is one of the most prevalent chronic conditions globally. Its incidence continues to increase with Qatar currently ranking 4th worldwide. Recently, obesity, referred to as "diabesity" occurring alongside T1D, has emerged with increasing prevalence among children, further complicating disease management.T1D's etiology is multifactorial with diet playing a critical role in both diseases' outcomes. This study aims to elucidate the biomarkers and dietary factors that differentiate T1D-Obese from T1D-Lean children in Qatar, utilizing a multi-omics approach.

A total of 176 children from Sidra Medicine were enrolled and categorized into T1D(n=98), Obese(n=30), T1D-Obese(n=28), and healthy-controls(n=20). Samples of blood, urine, stool and 24-hour dietary recalls were collected. Analyses included microbiome profiling via 16S rRNA sequencing, RNA sequencing for gene expression, DNA methylation analysis, and untargeted urine metabolite profiling. Integrative analysis and nutrient-omics interaction mapping were conducted using the mixOmics package in R. Significant differences in dietary patterns and gut microbiome composition were observed, with unique bacterial species identified as potential biomarkers for T1D and diabesity. Notably, ZFP57 hypermethylation was common across T1D, T1D-Obese, and Obese groups, while HLA-DBR1 and HOOK2 hypermethylation appeared specific to T1D and T1D-Obese, respectively. Differentially expressed genes (e.g. HLA-DQB, ZFP57, MMP9) were associated with T1D pathogenesis and diabesity. The methylation patterns interacted with dietary intake, gut microbiota composition, gene expression, and urine metabolites. Epigenetic modifications are reversible, making them viable therapeutic targets. As this is the first comprehensive multi-omics investigation in this population, our study presents novel biomarkers for targeted nutrition-based interventions tailored to Qatar's population, warranting further research for validation and application within clinical care pathways.

Dietary fibre-specific effects on serum and faecal bile acids profile and associations with the gut microbiota: a randomised, controlled dietary intervention in healthy participants

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Maintaining balance within the bile acid (BA) pool is crucial for cardiometabolic health and gastrointestinal function. The gut microbiota influences BA pool through transformations such as deconjugation and dehydroxylation. Dietary fibre is fermented by the gut microbiota and influences the BA pool, which changes serum and faecal BA profiles. We hypothesise that different dietary fibre sources have differential impacts on BA profiles, which may be associated with gut microbiota. We tested this hypothesis by analysing BA profiles in serum and faecal samples and gut microbiota data from the DISC Study. The latter is a double-blinded, randomised 2*2 factorial clinical trial in which 75 healthy participants were supplemented with type 2 resistant starch (RS) and polydextrose (PD) for 50 days. In participants who received RS, concentrations of conjugated BAs including TCA (p=0.006), GCDCA (p=0.018), TCDCA (p=0.018), GDCA (p=0.003), TDCA (p=0.001) and GLCA (p=0.017) in serum were higher than in those who did not receive RS. In contrast, PD supplementation had no effects on concentrations of serum BAs. Faecal BA concentrations were not significantly different after treatment with either RS or PD. Partial correlation analysis revealed RS-specific positive correlations at post-intervention between Akkermansia in faeces and TCA (Spearman's rho = 0.71, FDR p = 0.039) in serum. RS-specific positive correlations were also identified between Bifidobacterium at baseline and concentrations of GDCA (Spearman's rho = 0.71, FDR p = 0.024) in serum post-intervention. Our findings suggest RS and PD have differential effects on serum and faecal BA profile with increased serum conjugated BAs specifically induced by RS. This may be related to RS specific associations between gut microbiota. More research is needed to understand the mechanism of effects of different dietary fibre sources on the modulation of the BA pool through changes in transformation capacity of the microbiota.

Chewing behavior and bolus particle size of rice shape gut microbiota functionality and microbial metabolite signatures

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Wageningen University & Research

Differences in food oral processing can impact bolus surface area and saliva uptake, and this has been shown to influence post-prandial metabolic kinetics. Particle size and surface area may play a pivotal role in determining the accessibility of digestive enzymes and the substrate available to gut microbiomes. We sought to determine whether oral breakdown of rice influences the gut microbiota functionality, considering the oral phase, digestion, and colonic fermentation.

Using a combined in vivo and in vitro approach, we quantified bolus particle number, size, surface area and saliva uptake after expectoration and following the gastrointestinal and colonic phase of digestion for three rice varieties. Increasing the number of chews per bite produced more and smaller particles and increased total bolus surface area, regardless of rice variety. Even after colonic fermentation, the differences in bolus particles persisted, influencing microbial activity and microbial metabolite production. Higher bile salt hydrolase activity and total short-chain fatty acid (SCFA) levels were found in large particles. Focusing on SCFAs, a high concentration of acetic acid was found to correlate with larger particle sizes. Propionic acid production correlated positively with a larger surface area, while butyric acid production was highly correlated to the fiber content. Untargeted lipidomics highlighted that particle size influenced numerous metabolites beyond SCFA.

This study showed that oral chewing behavior and particle size could be innovative strategies to modulate gut microbiota functionality and microbial metabolite production.

Session 3: Can AI help bridge precision & public health nutrition: 23rd September @ 2 pm

Targeted PRECIsion NUTrition Strategy to Prevent Chronic Metabolic Diseases: A tissue-specific metabotype approach

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Obesity and type 2 diabetes (T2D) are major global health concerns, yet long-term outcomes of dietary prevention strategies remain suboptimal. One limitation is that nutritional guidelines fail to account for individual metabolic variability. We identified distinct metabolic phenotypes—metabotypes—among individuals with overweight, characterized by tissue-specific insulin resistance in either liver (LIR) or muscle (MIR). These metabotypes showed unique metabolomic, lipidomic, and transcriptomic profiles. Dietary intervention tailored to LIR or MIR led to improved cardiometabolic health (PERSON), demonstrating the potential of precision nutrition.

To further refine this approach, we employed data-driven clustering to stratify individuals based on glucose homeostasis and body composition. Using Hierarchical Clustering of Principal Components on a large cohort (The Maastricht Study), we identified six metabotypes. These were validated and classified with high accuracy (72–79%) using a Random Forest model. Optimal diets for each metabotype were derived by Reduced Rank Regression in The Maastricht Study identifying habitual dietary patterns associated with improved insulin sensitivity. Three female (F1-F3) and three male (M1-M3) metabotypes were identified, independent of age and BMI. F1 and M1 were characterized by high insulin sensitivity, low visceral adipose tissue (VAT) and liver fat (LF), and normal insulinogenic index (IGI). In contrast, F2 showed increased VAT and LF but maintained low hepatic insulin resistance (HIRI). F3 exhibited moderately elevated VAT

and LF (vs. F1), coupled with high HIRI and IGI. Among males, both M2 and M3 presented with increased VAT and LF (vs. M1); however, only M3 demonstrated elevations in HIRI and IGI. Each metabotype was associated with a distinct dietary composition to improve whole body insulin sensitivity.

This robust framework will inform the PRECINUT study, aiming to personalize dietary strategies to prevent metabolic disease.

Unraveling the Power of Vitamin D Mechanistically: How Long-Term Supplementation Shapes Immune Health

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Vitamin D3 is more than a nutrient, it functions as a pro-hormone and a critical regulator of immune function. However, its dynamic effects over time in healthy individuals remain incompletely understood. In this study, we investigated the longitudinal molecular response to vitamin D3 supplementation (a body weight adjusted vitamin D3 bolus of 1,000 IU/kg once every 28 days) in 42 disease- and medication-free adults.

Peripheral blood samples were collected at multiple time points (0, 1, 28, 29, 56, 57, and 84 days) over a 12-week period to track changes in immune-related gene expression. Using RNA sequencing (RNA-seq) and chromatin accessibility profiling (ATAC-seq), we observed a rapid and robust transcriptional response: 70 genes were significantly regulated within 24 hours of a single vitamin D3 bolus, expanding to 2,652 genes by day 84. Interestingly, the long-term response showed a convergence in gene expression profiles among individuals, suggesting a stabilizing, homeostatic effect of vitamin D. Among the differentially expressed genes, six encode for transcription factors, JUNB, ETS2, FOS, NR4A2, KLF10, and HIF1A, were consistently modulated throughout the study period. These factors are key regulators of immune signaling, inflammation, and cellular adaptation, highlighting vitamin D's role in fine-tuning immune responses.

This work establishes a reference framework for the healthy immune system's temporal response to vitamin D and sets the stage for comparison with individuals affected by immune-mediated diseases. Our ongoing efforts aim to build a comprehensive mechanistic model of vitamin D's long-term immunoregulatory effects, with the potential to inform future nutritional and therapeutic strategies. These findings emphasize that vitamin D not only supports but actively calibrates immune function, which brings us closer to unlocking its full potential in health and disease.

Precision Nutrition: Predictive Model of Appendicular Skeletal Muscle Mass in non-institutionalised people aged 65 and over

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Background and Objectives: Advances in precision nutrition directly impact public health by enabling nutritional recommendations based on the individual characteristics of each person. This study aims to develop a predictive model for Appendicular Skeletal Muscle Mass (ASM) using multiple variables in Spanish individuals aged 65 or older. A decrease in ASM is one of the main diagnostic criteria of sarcopenia, so a predictive model could facilitate its prevention and early detection. The study was part of an annual intervention project, using baseline data for the analysis.

Methods: Multiple variables were collected from 120 volunteers, including single nucleotide polymorphisms (SNPs) related to strength and sarcopenia, as well as anthropometric, nutritional, and lifestyle variables. A linear regression was performed using the full set of variables, and those that had the greatest impact on ASM were selected. A multiple linear regression model was applied to data from 80% of the individuals, reserving the remaining 20% for validation.

Results: The predictor factors in the final model were the intercept (4.43; $p < 0.01$), the rs7832552 genotype (0.33; $p = 0.11$), weight (0.35; $p < 0.01$), protein intake (-0.01; $p = 0.28$), caloric intake (-0.00007; $p = 0.89$), fibre intake (0.03; $p = 0.06$), blood folate levels (-0.09; $p = 0.08$), BMI (-0.54; $p < 0.01$), grip strength (0.11; $p < 0.01$), and hydration (0.45; $p = 0.04$). The model presented a Root Mean Squared Error (RMSE) of 1.07 and an R-squared of 0.94, indicating that the model explains 94% of the variability in the ASM. Conclusions: The developed model successfully predicts ASM in older adults using a combination of genetic, anthropometric, and lifestyle factors. The model can be used to identify individuals at risk of sarcopenia and potentially aid in the prevention of this condition through changes in nutritional and lifestyle behaviours.

Bridging Mechanisms Across Diseases: A Multi-Dimensional Mapping Approach with Potential for Broader Omics Integration

Lena Möbus, Dario Greco

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Traditional disease classification systems are largely organised by affected organs or symptom patterns. However, diseases often share underlying biological mechanisms that span across these traditional boundaries.

In our recent work, we developed a multi-dimensional framework to map mechanistic similarities among 500 diseases using public molecular, clinical, and pharmacological data. By integrating six data layers—including disease-associated genes, pathways, symptoms, drugs, and chemical interactions—we constructed a consensus disease landscape that reveals unexpected connections and challenges classical phenotypic disease groupings. We observed robust clusters driven not by anatomical proximity but by shared biological pathways. For instance, inflammatory and metabolic disorders showed mechanistic overlap with neurodegenerative diseases, including shared features between type 2 diabetes and Alzheimer's disease.

These relationships were supported by shared rare gene-pathway signatures, including insulin signalling and zinc transport, underscoring the potential for broader systemic links. Although this work does not directly involve nutrition or diet-related data, the approach is adaptable. It could be extended to include other omics data, for example from the realm of nutrigenomics, to explore gene-environment interactions or nutritional influences on disease mechanisms. By presenting this work, I aim to connect with researchers in this community and explore opportunities for methodological exchange and collaboration.

Session 4: Nutrition and the brain throughout the life course: insights from molecular mechanisms to clinical applications: 24th September @ 9 am

Is there an interplay between fat and umami taste perception and what is the role of CD36 gene? A preliminary overview

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Fat taste is the perception of free fatty acids, and Cluster of Differentiation 36 (CD36 gene) has been identified as receptor. Umami is the taste of savouriness and monosodium glutamate (MSG) is the main tastant. The combination of fat and umami is common (e.g. meats) and although an association has been hypothesized in previous literature, the two tastes have never been extensively studied together. This project aims to investigate whether there is an association between fat and umami taste perception, whether CD36 rs1761667 single nucleotide polymorphism (SNP) can play a role in both and which is the association with anthropometrics data.

The results will deepen our understanding on how tastes interact with each other, and which are the implications on food preferences. Data collection is underway. Demographics data are collected by questionnaire, saliva samples are collected for SNP genotyping of CD36 rs1761667. Body composition is analysed by bioelectrical impedance. Fat taste sensitivity (FTS) is assessed by oleic acid (OA) detection threshold (3-alternate force choice). Participants are classified as hypersensitive (= 3.8 mM), and non-tasters (>20mM). Umami and fatty taste perception, intensity and liking are assessed by rating of three broth samples (umami) with increasing concentration of MSG (29 mM, 200mM, and 400mM), and five fatty foods (cream cheese, cheese, yogurt, mozzarella, and hummus), in both regular and low-fat versions. Assessments were by Visual Analogue Scale. To date 37/69 individuals have completed data collection.

No association between FTS and umami perception has been found. CD36 rs1761667 genotype showed no difference in the distribution according to OA-detection threshold category. The rs1761667 A-allele was associated with reduced perceived intensity from regular cream cheese ($\beta = -1.07$ / $p = 0.03$) and the low-fat version ($\beta = -1.08$ / $p = 0.047$). No significant association has been found regarding anthropometry.

A series of n-of-1 intervention studies investigating the effects of tea on mood reveal that the interventions and participant-specific factors influence overall mood and relaxation

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The tea bioactive l-theanine distinguishes tea from other caffeinated drinks and has been linked with improved mood outcomes. Studies of tea and mood have focused on alertness and very few studies have reported other mood outcomes.

We recruited 13 people to individual n-of-1 studies to investigate the effects of black tea, compared to caffeine, and placebo, on overall mood (OM) and relaxation using the Caffeine Research Visual Analogue Scale, in the context of each participant's lifestyle. Each study consisted of four three-week cycles. Participants drank each intervention exclusively for one week per cycle, in random order, with a washout day at the end of each week. The caffeine and placebo interventions were made to taste and look the same as the black tea intervention.

Participants completed three measurements per day. OM and relaxation were analysed using dynamic linear, and logistic, regression, respectively. Statistical models included variables describing the interventions, time, personal factors (e.g., time, sleep), and time-lagged variables – to account for the effect of past measurements. Significant associations were found between the interventions (unblinded May 2025) and OM, and relaxation, for 3 and 2 participants, respectively ($p < 0.05$). For 9 participants OM at any given measurement was significantly associated with OM at the previous measurement ($p < 0.05$). For 3 participants, OM was significantly better at the 2nd or 3rd measurements, compared to earlier in the day ($p < 0.05$).

Sleep time, quality, or ease of getting to sleep were associated with better OM in 3 participants, and greater odds of relaxation in 4 participants ($p < 0.05$). For 1 participant, perceived effect of their menstrual cycle was associated with lower odds of relaxation ($p < 0.05$).

The participants studied revealed how the interventions and other factors (e.g., sleep, menstrual cycle) can affect their mood. This is important when people consider whether drinking tea would benefit them.

Cross-study metabolomics data integration for the identification of common metabolic syndrome phenotypes

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Due to the multifactorial character of metabolic diseases (system diseases), the understanding of the associated complex processes requires deep characterization of metabolic phenotypes in an integrated strategy. Today, metabolomics is a powerful mature tool, allowing access to a global exploration of metabolism, to stratify populations within the development of precision approaches. However, even though it generates insightful data, it often lacks a sizable population, limiting its impact.

In this context, the present work tackled the key question of cross-study metabolomics data integration, to investigate the validity of links between datasets from individuals analyzed in different health status. It addressed the major challenge of identifying early common phenotypes in the aging trajectory, with a particular focus on metabolic syndrome (MetS) for a deeper understanding of pathophysiological processes and modulators involved. In this work, a novel cooperative learning framework for vertical metabolomics data integration, designed to improve biomarker discovery by balancing integration across datasets while mitigating study-specific confounders, was applied to serum untargeted metabolomic datasets from 2 independent cohorts: one MetS case-control study (n=52 males, 22-38 y.o.) performed within the Haguenau community-based cohort, focusing on young adults; the second one conducted within the NuAGE cohort (121 males, 68-82 y.o.) in an elderly population. The proposed framework allowed an increase in MetS prediction performance.

The results showed the ability of the present approach to identify the common part of the MetS phenotype independent of age, highlighting systemic alterations related to insulin resistance, beta oxidation, inflammation and obesity. Additionally, specific aspects of the MetS phenotype were also identified, with modulated signaling metabolites in young adults, whereas specific markers of hypertriglyceridemia and dysbiosis in elderly.

The effect of Ramadan fasting on the methylation patterns in pediatric metabolic dysfunction-associated steatotic liver disease (MASLD)

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Introduction: Insulin resistance (IR) is a hallmark of metabolic dysfunction-associated steatotic liver disease (MASLD), a prevalent condition with no current pharmacological treatments. Lifestyle changes, such as diet and exercise, are the main management strategies. Fasting, especially time-restricted eating, is being studied for its role in obesity-related liver disorders. Ramadan Fasting (RF) has shown cardiometabolic benefits in adults, but its effects in children and underlying molecular mechanisms remain unclear. This study explores the impact of RF on DNA methylation and plasma lipidomics in children with MASLD.

Methods: Children (7–18 years) with NAFLD observing RF were recruited. Four visits were conducted: before RF (T1), twice during RF (T2, T3), and after RF (T4). At each visit, clinical data, body composition, and blood samples were collected. DNA methylation was assessed using the Illumina EpicArray. Dietary intake (24-hour recall) was analyzed via Nutritionist Pro. Plasma lipidomics were profiled using LC-MS.

Results: Out of 11 participants, 9 completed all visits. Kruskal-Wallis analysis found no significant changes in clinical parameters or body composition, except increased fasting glucose. Dietary analysis showed changes in calorie intake and several nutrients. DNA methylation patterns shifted over time, with both hyper- and hypo-methylated genes observed. Initially negative correlations between fasting insulin/HOMA-IR and several CpGs at T1 became strongly positive at T3/T4. Similar trends were observed for associations between CpGs and dietary components or body fat mass. Lipidomic profiling revealed persistent post-RF increases in short, saturated triglycerides—markers of de novo lipogenesis.

Discussion: RF influenced DNA methylation and lipid profiles in MASLD children, with notable shifts linked to dietary intake and clinical features. Cohort expansion and further molecular analysis are underway.

Session 5: Impact of dietary habits on epigenetic age acceleration: evaluating the intermediary role of inflammation: 24th September @ 2 pm

Laura Bordoni[1], Fatemeh Mansouri[1], Joao Agostinho de Sousa[2], Rosita Gabbianelli[1], Ferdinand von Meyenn[2].

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Low-grade inflammation (LGI) and metabolic health are key contributors to complex disease development and aging. LGI is both a cause and consequence of metabolic dysfunction and is a hallmark of inflammaging, driven by chronic immune activation. Addressing LGI and metabolic imbalance is essential for disease prevention and promoting healthy aging. Lifestyle changes, especially diet, offer practical strategies for intervention. In this study, we investigated the relationships between pro- and anti-inflammatory dietary components, LGI markers, and second- and third-generation epigenetic clocks, in the Lifelines-DEEP cohort.

We aimed to assess whether the pro- or anti-inflammatory properties of diet are associated with epigenetic age acceleration (EAA) and whether this effect is mediated by LGI. We found a significant association between overall diet quality (LLDS) and EAA, particularly using the PCGrimage clock. EAA was also associated with the intake of specific food categories: processed foods (high energy beverages (HHB) and NOVA4 food group) were linked to increased EAA, while vegetables, folate, vitamin C, and tea were associated with reduced EAA. These dietary factors also correlated with LGI levels, especially via the INFLASCORE, an integrated marker of LGI. Mediation analysis revealed that LLDS influenced EAA through both direct and indirect pathways. The effect of unhealthy food intake (HHB, NOVA4) on EAA was largely mediated by increased LGI, while beneficial foods affected EAA both directly and through reducing LGI.

Our results support the link between dietary patterns, inflammation, and epigenetic aging, corroborating the hypothesis that inflammaging and EAA are related but distinct processes, potentially contributing in parallel to aging and mortality risk.

Deciphering inflammatory cues from the microbiome: Bacterial membrane vesicles produced in response to prebiotic and antibiotic intake

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Introduction: The microbiome plays a pivotal role in human health, and gut-borne inflammatory signaling (metabolic endotoxemia) is believed involved in the etiology of metabolic syndrome. Gut-bacteria produce factors such as bacterial membrane vesicles (bMVs) that affect host physiology. These vesicles carry toll-like receptor (TLR) ligands with inflammatory properties. Dysbiosis of the microbiota yields abnormal vesicle repertoires. This study investigates the nature and characteristics of human gut-derived bMVs following microbiota disruption with the antibiotic vancomycin, and restoration through supplementation with the prebiotic 2'-fucosyllactose (2FL).

Methods: Feces-derived bMVs were isolated from 37 participants with overweight/obesity (BMI 25-40 kg/m^2) and normal glucose tolerance, randomised after a 7-day vancomycin course to 8 weeks of either 2FL supplementation or placebo (maltodextrin). Fecal-bMVs were isolated using (ultra)centrifugation and size-exclusion chromatography. Nanoparticle tracking analysis was used to assess bMV size and concentration. Human Toll-like receptor 2 and 4 (hTLR) reporters were employed to measure bMV-associated TLR-ligands such as peptidoglycan and lipopolysaccharide (LPS).

Results: Vancomycin-induced microbiota disruption yielded bMV repertoires more effective at activating both TLRs. Absolute (by volume) and relative (per vesicle) TLR4 activation was most pronounced post-vancomycin. For TLR2 activation only an absolute increase was observed. 2FL and placebo were equally efficient at restoring these parameters.

Conclusion: This study demonstrates, for the first time, that vancomycin alters the inflammatory properties of human gut bMVs. These dysbiotic vesicles are more effective at activating the host innate immune system ex vivo, and could contribute to metabolic disease. Further research into their nature and functionalities is required to unlock insight into this intricate route of communication between microbe and host.

Beyond Inflammation: Vitamin D and LPS Co-Stimulation Uncovers Novel Gene Networks in Human Monocytes

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Pathogen-associated molecular patterns like lipopolysaccharide (LPS) mimic immune responses to bacterial infections. The hormonally active form of vitamin D, 1α,25-dihydroxyvitamin D_3 [1,25$(OH)_2D_3$], supports innate immunity, yet its molecular mechanisms remain incompletely understood.

Here, we examined epigenomic and transcriptomic changes in THP-1 monocytes exposed to 1,25$(OH)_2D_3$, LPS, or both over 24–48 hours. ATAC-seq profiling revealed that co-stimulation with 1,25$(OH)_2D_3$ and LPS induced markedly more chromatin accessibility changes than either treatment alone, with up to 81% of altered regions uniquely responsive to the combination. Motif enrichment analysis identified JUN/FOS transcription factors as central mediators of this synergistic effect. RNA-seq data reflected similar patterns at the transcript level, albeit affecting fewer genes than chromatin regions. Under 1,25$(OH)_2D_3$-primed conditions, 333 genes showed synergistic expression changes upon co-treatment (responses that significantly deviated from the additive effects of individual stimuli) including 267 genes not previously recognized as vitamin D targets.

Functional annotation linked these genes primarily to monocyte and T cell differentiation, in contrast to classical vitamin D targets typically associated with inflammation. Together, our findings reveal a distinct regulatory program activated by the interplay of vitamin D and LPS, providing new insight into how vitamin D shapes immune function through coordinated chromatin and transcriptional remodeling.

Sex and Photoperiod Modulate Hepatic Oxidative Stress in Cafeteria Diet-Induced Obese Rats: Insights into Antioxidant Activation, Circadian Rhythms, and Melatonin Regulation

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Obesity is closely associated with increased oxidative stress (OS), metabolic dysfunction, and disruptions in circadian rhythms. While sex and photoperiod are known to influence OS, their combined effects under obesogenic conditions remain poorly understood.

Therefore, the aim of this study was to determine whether hepatic oxidative stress is affected by sex and photoperiod in cafeteria diet-induced obese rats (60 males and 60 females) exposed to a 6-h light (L6) or 18-h light (L18) photoperiod for 9 weeks. Females exhibited lower hepatic catalase activity and reduced glutathione (GSH) concentrations compared to males, alongside increased lipid peroxidation biomarkers such as malondialdehyde (MDA).

These changes were accompanied by elevated levels of antioxidant-related proteins, including pNrf2 and HO-1, as well as circadian regulators Bmal1 and Rev-erbα. Notably, HO-1 correlated strongly with Bmal1 ($r = 0.68$), suggesting a potential link between circadian rhythms and antioxidant responses. Plasma melatonin concentrations were significantly higher in females than males, particularly under the L6 photoperiod (0.095 ng/mL vs. 0.028 ng/mL).

Taken together, these findings highlight sex-specific differences in hepatic OS regulation, with females exhibiting greater oxidative damage but enhanced antioxidant activation potentially mediated by melatonin and circadian rhythms. The L6 photoperiod appears to amplify these effects, underscoring the complex interplay between sex, photoperiod, and hepatic oxidative stress in obesity.

Session 7: Selected Abstracts: 25th September @ 9.45 am

The Health Benefits of Fruit and Vegetable Byproducts: A Systematic Scoping Review

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Background: Fruits and vegetables (F&V) are nutrient-dense foods to the human population, yet nearly half end up being discarded. Food processing, where F&V parts such as peels, pulps, pomaces, and seeds are removed, generates approximately 16% of all global food waste. However, many of the discarded parts contain health promoting dietary components such as dietary fibre and polyphenols. The purpose of this review was to identify what is known from existing literature about the human health benefits of interventions using typically discarded F&V byproducts.

Methods: A systematic scoping review was conducted. Searches using terms relevant to F&V byproducts and human health were carried out in Medline (via PubMed), Embase, Web of Science, Cochrane (CENTRAL), and ClinicalTrials.gov. A thematic analysis of human health benefits was carried out systematically.

Results: Out of 6,252 papers identified, 16 papers (which included 19 studies) met inclusion criteria and were reviewed. The intervention studies included 13 unique byproducts and a wide range of health measures. Our review identified several positive health associations between F&V byproduct consumption and health outcomes related to glucose and insulin, cardiovascular health, gastrointestinal health, and cognitive health. However, outcomes were often heterogenous across studies and the positive association appears highly dependent on the byproduct type, quantity, and the health status of the population. No positive health outcomes were seen on anthropometric measures or in incidence of metabolic syndrome.

Conclusions: Our scoping review provides evidence that F&V byproducts, which often become waste, have several positive associations with human health outcomes and may be valorisation candidates. Finally, the heterogeneity throughout the intervention studies reinforces the need for further research using multidisciplinary approaches.

Caloric restriction-induced metabolic adaptation associated with amount of body weight lost – results from the LION study

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Background: Metabolic adaptation refers to a disproportional reduction in resting metabolic rate (RMR) during caloric restriction. Few studies have investigated longitudinal metabolic adaptation trajectories and their associations with weight loss.

Methods: For this analysis, data from the Lifestyle Intervention (LION) study (NCT04023942) were used. Indirect calorimetry-based RMR measurements were performed at baseline (V1), after an 8-week low-caloric weight loss intervention (V2), after a 12-month weight maintenance intervention (V3), and after a subsequent 12-month follow-up (V4). Using multiple linear regression on baseline data, an equation was derived to estimate RMR from age, sex, fat mass (FM) and fat-free mass (FFM). Next, metabolic adaptation was calculated at each time point by subtracting RMRestimated from RMRmeasured. Finally, multiple linear regression was performed to study the relationship between weight loss and change in metabolic adaptation from V1 to V2, adjusting for baseline age, sex, FM and FFM.

Results: In total, data from 234 participants (age 46±11 years, body mass index (BMI) 34.3±2.8 kg/m^2, 69% women) were included. Participants lost 11.7±3.5 kg from V1 to V2, gained 2.0±7.7 kg from V2 to V3, and gained 4.0±6.0 kg from V3 to V4. Mean RMRmeasured was 1598±253 kcal/day at V1, 1424±221 kcal/day at V2, 1525±258 kcal/day at V3, and 1540±247 kcal/day at V4. Metabolic adaptation was 0.0±130.0 kcal/day at V1, -70.9±109.9 kcal/day at V2, -1.2±130.0 kcal/day at V3, and 12.4±144.3 kcal/day at V4. Change in metabolic adaptation from V1 to V2 was independently associated with weight change from V1 to V2 (β=.005, p=.003).

Conclusion: A low-caloric weight loss intervention was found to induce metabolic adaptation which was in turn associated with the amount of body weight lost (-0.5 kg weight change per -100 kcal/day metabolic adaptation). The impact of the observed metabolic adaptation on long-term body weight change remains to be investigated.

Developing a 3D model of the bone marrow niche to investigate the influence of folate on childhood leukaemia-initiating events

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Acute lymphoblastic leukaemia (ALL) is the most common childhood cancer. Although aetiology is unclear, genetic aberrations, such as chromosomal translocations, are considered initiating events in ALL development and have been retrospectively detected at birth, suggesting they occur in utero. Further aberrations, such as epigenetic modifications, are required for disease progression. Epidemiological evidence suggests maternal folate status influences risk of childhood leukaemia. Folate influences DNA damage, repair and methylation status. Guidelines recommend pregnant women consume synthetic folic acid (FA) supplements during the first trimester, however, biologically active folate, 5-methytetrahydrofolate (5mTHF), is becoming a more popular supplement in pregnancy.

We aim to investigate the influence of FA and 5mTHF on leukaemia-initiators using organoid models to replicate the bone marrow niche. Mesenchymal stem cells (MSCs) were used to create 2D and 3D organoids supporting growth of GM12878 B cells in media containing physiologically relevant concentrations of FA or 5mTHF. Cell growth was evaluated through trypan blue exclusion. Intracellular and media folate levels were measured using LC-MS. Reverse-transcription PCR is used to monitor cell markers and identify leukaemia-initiating events. When grown in co-culture with MSCs, B cell growth increases compared to growing alone. A further increase in B cell growth was observed when grown in the presence of 3D spheroids compared to 2D MSC monolayers. When grown in folate deficient conditions a reduction in B cell growth compared to standard media is observed for FA and 5mTHF supplementation over 9 days. A reduction in intracellular folate was observed as early as 2 days following folate depletion. We optimised a 3D model of folate status in B cells, allowing investigation of the induction of leukaemia-initiators. Such knowledge may be useful to influence public health policy for folate guidance during pregnancy.

Obesity-Satiety Phenotypes and the Gut Microbiome: A Pilot Study

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Introduction Obesity is a chronic, multifactorial disease with increasing global prevalence, leading to a higher burden of disease. Despite various treatment efforts, obesity remains persistent, highlighting the need for personalized interventions. Eating behavior plays a significant role in the development of obesity, as it can contribute to inadequate energy intake. Recent research by Acosta et al. identified specific phenotypes describing low satiety, including "hungry brain", "emotional hunger", "hungry gut", and "slow burn", each with distinct physiological and behavioral traits. Originally developed as a treatment guide for pharmacological intervention, treatment according to satiety phenotypes has achieved greater weight loss compared to untargeted approaches. The gut microbiota may impact eating behavior and dysbiosis has been associated with inflammation in brain regions involved in satiety regulation. Since the gut microbiome is modifiable, identifying differences in microbial composition between phenotypes could result in novel personalized intervention for reducing obesity. We therefore aim to investigate the association between satiety phenotypes and gut microbiome composition.

Methods Participants (n=54, mean age=61) were provided a do-it-yourself satiety test meal and reported on eating behavior and satiety levels via questionnaires. Participants provided fecal samples which were analyzed by Metagenomic sequencing. Differences in α- and β-diversity between satiety phenotype groups are assessed using linear mixed modeling and principal component analysis.

Results In our sample we identified the phenotypes “hungry gut” (n=11), “hungry brain” (n=12), and “emotional hunger” (n=6). Preliminary results show no significant differences in α- or β-diversity across phenotypes.
Conclusion In the next step, we aim to repeat the study in a larger sample and assess taxa differences between phenotype groups.

To model or not to model? Lessons on using an in silico-in vitro approach to identify foods that support the infant colonic microbiota

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Studying diet-colonic microbiota interactions is costly, technically challenging, and ethically complex. Mathematical models can help overcome these limitations. However, their accuracy under realistic dietary conditions remains poorly established. Here, we compared short-chain fatty acid production by the colonic microbiota of weaning infants, predicted by the metagenome-scale community metabolic model MICOM, with in vitro faecal fermentation data. The data was obtained from a published study that investigated how various food ingredients, alone and in combination with other foods and/or infant formula, affect short-chain fatty acid production by the colonic microbiota of weaning infants using faecal samples as a proxy.

Overall, the model exhibited poor accuracy for all samples, and only a weak correlation was observed between measured and predicted acetate production ($r = 0.17$, $p = 0.03$). The model's accuracy improved when analyses focused on plant-based food samples, with acetate exhibiting a moderate positive correlation ($r = 0.31$, $p = 0.005$), while a trend towards weak correlation was observed for butyrate ($r = 0.21$, $p = 0.06$). In contrast, no significant correlations were observed when analysing samples primarily composed of infant formula.

These findings suggest that the model better predicts the influence of foods rich in complex carbohydrates than foods rich in protein and fat. Its further refinement is essential to advance diet-colonic microbiota research in weaning infants.

Posters

Theme 1: What is a healthy and sustainable diet?

Poster 001:
Development of an Algorithm based on Precision Nutrition to assess Nutritional Risk in the Elderly. The MyFOOD4Senior Project

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Background and aim: The elderly represent the age group at highest risk of malnutrition and nutritional deficiencies. Body mass, basal metabolic rate, and energy needs decrease with age, potentially impacting overall nutrient intake. Early diagnosis and ensuring sufficient consumption of food energy and essential nutrients is crucial for the physical, mental, and social well-being of older adults. Our purpose was to develop a scoring algorithm based in genetics, biochemical and lifestyle variables to identify potential age-related nutritional deficiencies in a group of people aged 65+.

Methods: A literature review of the most recent scientific articles, as well as a compilation of health and lifestyle recommendations from relevant national and international institutions, were conducted to identify candidate genes and nutrition related variables susceptible to intervention.

Results: A decision algorithm was developed based on four blocks: 1) Genetic variables: Single nucleotide polymorphisms in the MC4R, VDR, GC, CYP2R1, MTHFR, TMPRSS6, and CASR genes. 2) Adequacy of dietary habits, focusing on total energy intake and consumption of protein, vitamin D, iron, calcium, and folate. 3) Biochemical variables, including 25-hydroxyvitamin D, ferritin, transferrin saturation index, hemoglobin, vitamin B12, and serum folate. 4) Risk of malnutrition assessed using the short form of the Mini Nutritional Assessment (MNA-sf). Based on the values obtained for each variable, scores were assigned within each block. The algorithm enables to calculate an individual final risk score of nutritional deficiency. In addition, a specific lifestyle advice was designed for each individual case resulting from the algorithm, based on the recommendations of reference health organizations.

Conclusion: A comprehensive approach that integrates genetic, dietary, and biochemical factors enables the development of risk scores and personalized nutrition for older adults.

Poster 002:

Heterogeneity of postprandial glucose responses to standardized meals during a fully controlled dietary intervention – the RepEAT study

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Background. Existing literature indicates a large heterogeneity in postprandial glucose responses (PPGR) between individuals. However, the replicability of PPGRs within individuals and the consistency of the variation in PPGR between individuals is still unknown. We investigated whether PPGRs are sufficiently person-specific to use in personalized dietary advice.

Methods. The RepEAT study (n=63; NCT05456815) was a 9-week fully controlled dietary intervention during which PPGRs were measured using continuous glucose monitoring (CGM). All unique meals (n=105) were consumed three times at the same weekdays and time of day. PPGRs were measured as iAUC. Replicability was determined as coefficient of variation (CV). “Best” and “worst” meals for individuals were defined by within-individual lowest and highest average carbohydrate-corrected PPGR, respectively. Dietary compositions of the hypothesized best and worst meals were compared between individuals.

Results. The within-individual CV for PPGRs to identical meals ranged from 25%-105%, depending on the type of meal and subject. PPGRs to main meals, i.e. breakfast, lunch, and dinner, were more consistent (CVs ~50%) than snack meals (CVs up to 250%). Upon visual inspection of heatmaps, the main meals that induced the lowest PPGRs were person-specific. When compared to “worst” meals, “best” meals per individual were on average higher in protein (128 ± 31 vs 107 ± 32 g/1000 kcal, $P<0.001$), comparable in fat (137 ± 31 and 137 ± 24.4 g/1000 kcal, $P>0.05$), and lower in dietary fiber (26 ± 8 vs 31 ± 8 g/1000 kcal, $P<0.001$). Interestingly, the dietary composition for best meals was highly person specific. Conclusion. PPGRs are both meal- and person-specific, which emphasizes the potential of personalized dietary advice. The focus should lie on main meals, as PPGRs to snack meals are too inconsistent.

Poster 003:

Workflow for the optimisation of personalised, sustainable, plant-based, (poly)phenol-rich diets

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Sustainability in nutrition goes beyond environmental concerns, including nutritional adequacy, cultural acceptability, economic accessibility, and long-term adherence. A healthy and sustainable diet should meet individual needs, consider economic and time constraints, and be feasible to follow over time. Within this context, a structured workflow to design personalised, sustainable, plant-based, and (poly)phenol-rich diets was developed in the framework of the PRE-CARE-DIET study, to be prescribed to volunteers at cardiometabolic risk.

The study aims to demonstrate the association between aggregate phenolic metabotypes and cardiometabolic health in response to the chronic intake of (poly)phenols. The workflow combines dietary habit assessment, evaluation of energy and nutrient requirement, food preferences, national dietary guidelines and reference values, following sustainability principles and taking into account constraints mostly related to caffeine, sugar, fat, fibre, and (poly)phenol intakes.

Personal demands, including intolerances and religious restrictions, are also considered to ensure safety and acceptability. Particular attention is paid to selecting plant-based foods naturally rich in (poly)phenols, favouring seasonal and minimally processed options, when possible, while also including selected processed products to improve feasibility and reduce food waste. To align with habitual dietary patterns and improve compliance, culturally relevant foods such as pizza and dining-out options are also considered, reducing the perception of restriction and enhancing long-term adherence. Diet adherence will be evaluated and associated with health outcomes.

By integrating nutritional, social, and practical dimensions of sustainability into a personalised dietary workflow, this approach represents a novel tool for translating plant-based (poly)phenol-rich recommendations into feasible, acceptable, and long-lasting dietary changes.

Poster 004:
Moderate fish consumption manifests positive effects on sperm quality and testosterone levels

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Introduction: The incidence of male factor infertility has increased recently. Factors affecting semen quality might be genetic, hormonal, nutritional and others. An appropriate diet related to fish consumption may improve semen quality. Fish is a source of essential nutrients such as polyunsaturated fatty acids, fat-soluble vitamins, water-soluble vitamins, and essential oligo-elements like calcium, magnesium, phosphorous zinc, selenium, and others. All these nutrients have a significant contribution in semen quality and testosterone levels.

Aim: To explore a possible association between the frequency of fish consumption and sperm quality.

Methods: Seventy-four healthy men aged 25-45, were involved in this observational study. According to the filled questionnaire, volunteers were classified as low fish consumption group, ≤1/month, moderate fish consumption group, 1/week, and high fish consumption group, ≥2/week. Semen quality was evaluated by spermogram analyses. Testosterone levels were analyzed by ELISA method.

Results: The low fish consumption group has lower testosterone levels (3.6±0.9vs4.3±1.5; p=0.04), decreased non-progressive spermatozoa (11.9±6.2vs15.8±8.2; p=0.04) and lower semen volume (3.1±2.0vs3.6±1.3; p=0.06) compared to moderate fish consumption group. High fish consumption group tend to decrease sperm parameters. Moderate fish consumption group shows the better semen quality with all semen parameters in the reference range and the low fish consumption group manifested the worst semen quality parameters. Conclusions: Moderate fish consumption appears to have a positive impact on sperm quality by increasing testosterone levels, sperm volume and the non-progressive spermatozoa without affecting the sperm count. High fish consumption tend to decrease sperm quality. These results might be useful for nutritional optimization in risk groups and some cases of idiopathic infertility. European Union-NextGenerationEU: BGRRP-2.004-0009-C02

Poster 005:

Food Biodiversity in UK diets: The association of dietary species richness and consumption of fruits and vegetables, fibre, and fish

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Background and objectives: Dietary species richness (DSR), a metric of food diversity, is inversely linked to overall and specific mortality in Europe. We developed a database detailing unique species in >6000 foods from the UK National Diet and Nutrition Survey (NDNS) to estimate DSR using four-day food intake data and examine its relationship with diet quality.

Methods: The 2018- 2019 NDNS nutrient databank was expanded to include FoodEx2 food classification, ingredients, and greenhouse gas emissions data. Unique species were identified using a word-matching algorithm in R based on product ingredients. Four-day food intake and demographic data from the NDNS waves 9–11 were used to calculate dietary quality indicators and DSR. Linear regression models estimated the association between adherence to nutritional guidelines and DSR.

Results: We identified 216 unique species across UK diets. At the food level, composite dishes had the highest DSR (median 8 [Q1=4, Q3=12]), followed by seasonings, sauces, and condiments (median 7 [Q1=4, Q3=10]) and grains and grain-based products (median 5 [Q1=2, Q3=7]). At the dietary level, the median DSR over four days was 49 [Q1=43, Q3=56; range 14 - 92], with the first two days accounting for 80% of the total DSR measured over four days. DSR was significantly higher in younger age categories and among those with a higher household income or IMD (all $p<0.001$). A higher DSR was significantly associated with increased dietary fibre intake and higher consumption of fruits, vegetables, and fish (all $p<0.001$).

Conclusion: After analysing four days of food intake data, we successfully established DSR in UK diets and identified ways to enhance it by increasing fruit, vegetable, fibre, and fish consumption through composite dishes. Further research is needed to validate DSR and explore its relationships with health outcomes, personalised food biodiversity approaches, and ecological impacts.

Poster 006:

Association between MC4R Genotype and dietary intake in non-institutionalised people aged 65+. The MyFOOD4Senior Project.

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Background and aim: The melanocortin-4 receptor gene (MC4R) has been associated with an increase in body mass index (BMI) and an elevated risk of obesity, through the C allele of the single nucleotide polymorphism (SNP) rs17782313. Moreover, carrying the C allele predisposes to increased appetite. The aim was to assess the association between MCR4 genotype and dietary and anthropometric variables, in a group of individuals over 65 years of age.

Methods: A cross-sectional study was conducted with 120 non-institutionalized people (72.5% females) aged 65+ years. Data on diet were collected through a 3-day food diary. These data were then processed using the DIAL program to obtain detailed information on total caloric intake, macronutrient distribution range, and fat quality. Anthropometric measurements such as weight, height, waist and hip circumference were taken, from which the Waist-to-Hip Ratio (WHR) and Body Max Index (BMI) were calculated. Additionally, body fat percentage was assessed through bioelectrical impedance analysis. All individuals were genotyped for rs17782313.

Results: 83% of the participants were found to have the TT genotype, while the remaining 37% carried the risk allele (CC/CT). The average caloric intake was 1.989.45 ± 460.69 kcal/day. 96% of the participants met 80% of the Recommended Intake (RI) established for their sex and age (considering 1.920 kcal for men and 1.500 kcal for women). The presence of the risk allele (CC/CT) was not associated with a significant difference in energy intake, macronutrient distribution range, or fat quality. Moreover, no significant associations were found between the genotype and the following variables: body fat percentage, BMI, and WHR.

Conclusion: Although the C allele of the rs17782313 SNP of the MCR4 gene is associated with obesity, this cannot be explained through higher energy intake or differences in the energy provided by fat, protein and carbohydrates.

Theme 2: Diet and the microbiome: lessons learned

Poster 007:
Multi-Omics insights into the gut microbiome, nutrigenetics, and barrier function in obesity

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Objectives: To evaluate interactions between host genetics, gut microbiota, and intestinal barrier function in obesity management.

Methods: In a 12-month randomized trial, 133 adults (65 M, 68 F; mean age 43 ± 10 y, range 18–65; BMI ≥ 30: control group n = 62, intervention group n = 71) were assigned to a control group consuming a Western diet or to an intervention group following a low-FODMAP plan tailored by nutrigenetic profiling (PPARG rs1801282 C/C 40.6%, FTO rs9939609 A/A 33.8%, MC4R rs17782313 C/C 30.8%, LEP rs7799039 G/G 36.8%, MUC2 rs4072037 A/A 31.6%) supplemented with Lactobacillus and Bifidobacterium spp. and omega-3 agents.

Results: At 3, 6, and 12 months, the intervention group achieved greater reductions in BMI (control: 32.89, 32.00, 29.98 kg/m^2 vs. intervention: 30.56, 29.40, 27.42), waist circumference (94.32, 91.66, 83.45 vs. 101.25, 101.20, 93.52 cm), and body fat (37.55, 35.50, 31.86 vs. 39.58, 39.37, 34.63%). Metagenomic profiling revealed enrichment of Akkermansia muciniphila (2.71, 3.27, 2.18 vs. 1.55, 1.89, 3.19%), Faecalibacterium prausnitzii (11.98, 6.29, 8.53 vs. 6.83, 5.35, 5.94%), decreased LPS-producing taxa (44.47, 50.66, 30.48% vs. 28.66, 20.55, 18.34%), increased SCFA levels (1.20, 1.13, 2.34 vs. 2.23, 3.29, 2.03 mmol/L), and butyrate-producing populations (19.5, 23.70, 33.18% vs. 55.9, 35.94, 60.38%). Enterotype shifts: Ruminococcus-dominant types rose from 8.45% to 35.21%. All primary outcomes achieved statistical significance ($p < 0.01$).

Conclusion: Gene-guided dietary intervention elicited sustained improvements in anthropometric and microbial endpoints, underscoring the potential of precision nutrition in obesity therapy. Additional barrier function analysis indicated that MUC2 A/A carriers experienced significant declines in serum zonulin and LPS-binding protein, reflecting enhanced mucosal integrity. These mechanistic insights support stratified nutrition strategies. Clinical implementation requires evaluation of cost-effectiveness and scalability.

Poster 008:

Gut Microbiota as an early biomarker of Graft-versus-Host Disease in Allogeneic Hematopoietic Stem Cell Transplantation: the impact of Haemophilus and BMI

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Allogeneic Hematopoietic Stem Cell Transplantation (aHSCT) is vital for treating hematological disorders, but acute graft-versus-host disease (aGvHD) significantly impacts outcomes. This study examines the role of microbiota and pre-transplantation Body Mass Index (BMI) as potential prognostic biomarkers of aGvHD development.

375 stool samples from 72 aHSCT patients were collected and analyzed at multiple time points. Patients were categorized into normal weight (NW) and overweight/obese (OW/OB) groups based on BMI and followed longitudinally until 100 days after transplantation with serial sampling and clinical annotations.

In general, patients developing aGvHD had a lower microbial richness, with no difference between BMI groups. At day 14 prior to aGvHD development, patients showed a significant increase in Haemophilus, Faecalibacterium, Succinivibrio, and Anaeroplasma, and a loss of Sutterella and genus from the family Gemellacee. In particular, the presence of Haemophilus and the absence of Sutterella preceded the aGvHD occurrence in OW/OB subjects and were flanked by an increase of bacterial toxins pathways. In a multivariate analysis, the risk of developing aGvHD was higher in the presence of Haemophilus than well-known risk factors for aGvHD such as TBI and unrelated donor transplant. Our findings identified early microbial biomarkers and BMI associated with the development of aGvHD. Understanding the risk factors impacting gut microbiota can lead to targeted therapies and improved transplantation outcomes.

Poster 009:

In Vitro colonic fermentation of (poly)phenol-rich tablets: unravelling the inter-individual variability in (poly)phenol microbial catabolism

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(Poly)phenols are the most abundant group of phytochemicals and their consumption has been associated with cardiometabolic disease prevention. The main hindrance to exploiting their beneficial properties is the heterogeneity in the physiological response to their consumption, which mainly originates from inter-individual differences in their colonic catabolism. A key factor in determining this variability is the gut microbiota composition and activity. In this study, in vitro colonic fermentations were conducted using faecal slurries of 19 donors to investigate the inter-individual variability in the gut microbial catabolism of (poly)phenols. Three digested tablets containing 15 (poly)phenol classes in proportions reflecting the dietary intake of European populations were used as fermentation substrate. Faecal donors followed a (poly)phenol-free diet for two days before collection. The incubates derived from 2-, 6-, 24- and 48-hour fermentation were analysed with ultra-high performance liquid chromatography coupled with mass spectrometry. A total of 268 compounds were monitored, of which 130 were quantified, belonging to 22 phenolic classes. Among them, 82 were catabolites and 48 were parent compounds. The most representative flavonoid class was the flavan-3-ol group, while the richest non-flavonoid classes were cinnamic and benzoic acids. Parent compound degradation and catabolite production were assessed for pooled subjects, and several multivariate models were built to explore the inter-individual variability in (poly)phenol catabolism from a kinetic and quali-quantitative point of view. Finally, a correlation analysis between phenolic catabolites and microbial taxa was performed to assess a putative relationship between metabolic activity and composition of the gut microbiota. This study highlights the importance of exploring the inter-individual variability driven by the gut microbiota when investigating the health effects associated with (poly)phenol intake.

Poster 010:
Resolving GI Metabolome Complexity via High-Throughput Ion Mobility–Assisted Ambient MS

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Deciphering the complex metabolic interplay between the diet, gut microbiome and host physiology is essential for advancing nutritional science. Gastrointestinal (GI) biofluids, such as feces and saliva, offer a valuable window into such dynamics. Large-scale cohort studies aim to map these interactions at the population level to uncover disease mechanisms and guide clinical studies. However, as sample sizes grow, traditional chromatography coupled to mass spectrometry (MS) metabolomics/lipidomics workflows are too time-consuming and costly for routine use. Meanwhile, faster ambient MS approaches sacrifice chromatographic resolution, reducing confidence in biomarker annotation and (semi)quantification.

To address these challenges, we present an integrated platform combining cyclic ion mobility spectrometry (IMS) with laser-assisted rapid evaporative ionization MS (LA-REIMS) for the direct analysis of GI biofluids from three Flemish pediatric cohorts (n = 1500). In this setup, IMS delivers the required compound resolution at the millisecond timescale without compromising the LA-REIMS throughput set at up to 100 samples per hour. Concurrently, IMS-guided output monitoring preserves intact metabolite signals through removing contaminating and non-biological peaks (e.g., salt clusters) and background noise, improving signal specificity across complex GI samples.

We demonstrate robust annotation at both the metabolite/lipid class and individual biomarker level, including the resolution of isomers directly from biofluids. By capturing the biochemical crosstalk within the gut ecosystem at high resolution and speed, this platform provides a scalable solution for GI metabotyping as benchmarked against our 2D-LC-HRMS metabolomics/lipidomics platform. Together, these advances establish cyclic IMS–LA-REIMS as a robust platform for intact biofluid metabotyping in large cohorts.

Poster 011:

Short-term fiber mixture supplementation alters real-time intestinal gas kinetics in lean and overweight/obese individuals

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Introduction Our understanding of the diet–gut microbiota–host metabolism axis remains limited due to the lack of adequate real-time measures of microbial fermentation. To address this, we developed a novel, non-invasive system for continuous measurements of fermentation gases (H2 and CH4) and metabolic kinetics in humans (O2 and (13)CO2). Next, we investigated the effects of a two-day complex fiber mixture (inulin + resistant starch) on fermentation dynamics in lean normoglycemic individuals and in individuals with overweight/obesity and insulin resistance/prediabetes.

Method: This randomized, placebo-controlled, crossover study included 12 lean normoglycemic individuals and 13 individuals with overweight/obesity with prediabetes/insulin resistance. Participants received either a 21 gram fiber mixture (inulin + resistant starch) or a control (maltodextrin) for two days. In the evening after the start of the supplementation, participants entered the fermentation chamber for 36h. In the morning of day two a 13C-labeled fiber (inulin) was provided. Changes in fermentation gas pattern excretion rates were assessed (primary outcome) alongside other metabolic parameters.

Results: The two day fiber supplementation increased 36h H2, CH4 and 13CO2 release in both groups ($P<0.05$). Interestingly, 36h H2 release was higher in lean individuals in both the fiber and control treatment compared to the individuals with insulin resistance/prediabetes (general linear model, phenotype effect $P = 0.045$). Analyses on other metabolic parameters will be available during the symposium.

Conclusion: Our novel methodology revealed that a two-day fiber supplementation greatly impacts gut microbial H2 release as indicator of saccharolytic fermentation. Moreover, saccharolytic fermentation was higher in lean individuals compared with individuals at risk of developing diabetes.

Poster 012:
How Dietary (Poly)phenols Influence Cardiometabolic Health: A Multi-Omics Approach

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(Poly)phenols (PPs) represent a class of phytochemicals commonly found in a variety of plant-derived foods and beverages. Their absorption, distribution, metabolism, and excretion are influenced by individual factors, including genetic background and gut microbiome composition, which also shape their bioactivity. A key role is played by the gut microbiome, which converts PPs into bioactive metabolites.

Metabolomics and metagenomics analyses are used to explore these microbial-mediated transformations. However, the molecular mechanisms underlying PP effects in humans remain partially unclear, as well as the contribution of other factors to microbiota-mediated transformations and effects.

To address these ambitious questions, we integrated data from metagenomics and metabolomics investigations with genomics information, through a multi-omics strategy: we combined biological omics data obtained from the Oral (Poly)phenol Challenge Test, a study that aims at deciphering the bioavailability of dietary PPs and their impact on cardiometabolic health at the individual level. Cytoscape and publicly available interaction databases were employed to support the construction and analysis of molecular networks, offering further insights into genes involved in these pathways. Moreover, using the mixOmics R package, we performed (un)supervised integration of omics datasets, to highlight possible associations between different biological layers.

By combining multi-omics and network-based approaches, we identified interrelated molecular changes potentially linked to PP consumption and their impact on cardiometabolic health. The study also contributes to methodological advancement in the field of nutri-omics and personalized nutrition, providing a reproducible pipeline for integrating omics data in diet-health research.

Poster 013:
Effects of NatureKnit™, a Blend of Fruit and Vegetable Fibers Rich in Naturally Occurring Bound Polyphenols, on the Metabolic Activity and Community Composition of the Human Gut Microbiome Using the M-SHIME® Gastrointestinal Model

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This study evaluated the impact of a proprietary blend of fruit and vegetable fibers rich in naturally occurring bound polyphenols (commercially marketed as NatureKnitTM), compared to purified fibers (inulin and psyllium), on the human gut microbiome using the validated M-SHIME® gastrointestinal model. A short-term single-stage colonic M-SHIME® experiment (with fecal inoculum from three healthy human donors) was used to evaluate the test products compared to a negative control. Samples were assessed for pH, gas pressure, short-chain fatty acid (SCFA) production, lactate, and ammonium from 0 h to 48 h. Microbial community composition was assessed at 0 h (negative control only), 24 h, and 48 h (lumen) or 48 h (mucosal). All test products were fermented well in the colon as demonstrated by decreases in pH and increases in gas pressure over time; these changes occurred faster with the purified fibers, whereas NatureKnit™ demonstrated slow, steady changes, potentially indicating a gentler fermentation process. SCFA production significantly increased over the course of the 48 h experiment with all test products versus negative control. SCFA production was significantly greater with NatureKnit™ versus the purified fibers. Shifts in the microbial community composition were observed with all test products versus negative control. At the conclusion of the 48 h experiment, the absolute bacterial abundance and the richness of observed bacterial taxa in the lumen compartment was significantly greater with NatureKnit™ compared with inulin, psyllium, and negative control. Overall, NatureKnit™ demonstrated greater or similar prebiotic effects on study measures compared with established prebiotic fibers.

Poster 014:
Exploring Habitual Diet in Lactose Malabsorption: Preliminary Findings from the Lactobreath Study

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Lactose malabsorbers (LM) face an increased risk of being lactose-intolerant (LI) due to the genetic downregulation of lactase. Approximately 55% of the adult global population experiences gastrointestinal symptoms (GIS) after lactose ingestion. However, not all LM are considered LI, suggesting factors beyond genotype may be involved. Lactobreath aims to identify metabolomic markers of LI in relation to diet and gut microbiota.

The ongoing study assigns participants to symptomatic lactose malabsorbers (LM-S), asymptomatic lactose malabsorbers (LM-A), or asymptomatic lactose absorbers (control), based on genetic variants and GIS after lactose ingestion. Dietary intake is assessed using 4-day food diaries, and food groups, selected nutrients, and FODMAP intakes are compared between groups. Dietary patterns will also be explored through cluster analysis and various dietary and sustainability indices. In this preliminary analysis, Kruskal-Wallis and pairwise Wilcoxon tests were applied for selected outcomes.

Lactose intake was significantly lower in LM-S (n=11, mean 1.0 g/1000kcal) compared to controls (n=13, mean 3.2 g/1000kcal) (p=0.02), with LM-A showing intermediate intakes (n=10, mean 2.0 g/1000kcal). The LM-S group also tended to have lower fructose intake (4.7 g/1000kcal) compared to controls (7.1 g/1000kcal) (p=0.07). LM-S consumed significantly less insoluble fibre (4.5 g/1000kcal) than controls (7.4 g/1000kcal) (p=0.02). Other macronutrient intakes did not differ substantially, with mean carbohydrate intake contributing 39.48–41.25% of energy intake, protein 15.9–17.4%, fat 36.8–40.7% and total and soluble fibre intakes ranging from 10.9–12.8 and 2.2–3.5 g/1000kcal, respectively. Differences in FODMAP and insoluble fibre intakes indicate potential differences in dietary habits. Cluster analysis of the Lactobreath cohort (n=120) will integrate dietary patterns with metabolomic and metagenomic profiles to help reveal links to gut microbiota and host metabolism.

Poster 015:
Yogurt fermented with extra strains modulates metabolism and immune tissues in gnotobiotic mice

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Dietary diversity is increasingly recognized as a determinant of health. It is associated with better nutritional status in the elderly and protective effects against cardio-metabolic diseases. Likewise, reduced gut microbiota diversity has been linked to poorer health outcomes. Fermented dairy products are an important source of beneficial microbes and bioactive compounds, and expanding microbial diversity in fermentation may influence host metabolism and immunity.

We aimed to evaluate whether increasing microbial diversity in yogurt fermentation translates into greater metabolic diversity in the product and in the host. Specifically, we tested whether milk fermented with five lactic acid bacteria (LAB) strains (instead of the traditional two) modulates metabolic and immune-related profiles in mice. Seventeen gnotobiotic mice were fed daily for 3 days with either milk, normal yogurt, or a test yogurt fermented with two standard LAB plus three additional strains. Blood was collected on day 1 (2 h and 4 h post-gavage) and day 3 (4 h post-gavage). On day 3, mesenteric lymph nodes (MLNs) and spleens were also collected. Untargeted UHPLC-MSMS metabolomics was performed on the products, serum, spleen, and MLN samples. Bulk transcriptomic analysis was performed on ileal tissue. Multivariate analyses (PLS-DA, OPLS-DA) were applied to the metabolomics data as well as pathway analysis. Differential gene expression and GSEA was performed on the RNA sequencing data.

Metabolomics analysis of the products confirmed increased metabolic diversity in the test yogurt. Distinct metabolic profiles in the blood and organs depending on the ingested product could be revealed: (O)PLS-DA analysis clearly differentiated samples from mice treated with milk, normal yogurt, or test yogurt. Pathway enrichment using Mummichog and compound identification are ongoing and will be presented. Preliminary transcriptomic data and integrated multi-omics insights are under analysis and will be presented.

Poster 016:
Quantification and reproducibility of the response of the gut microbiome to diet

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The human gut microbiome is highly personal. Long-term dietary intake influences the structure and activity of the trillions of microorganisms residing in the human gut, but it remains unclear how reproducibly the human gut microbiome responds to diet change. The RepEAT study involved a fully controlled dietary intervention lasting nine weeks, consisting of three repetitive periods of three weeks each.

Fecal samples were collected at the end of each three-week period. Relative microbiome profiling (RMP), using amplicon sequencing, was employed to identify which microbes are present, while quantitative microbiome profiling (QMP), using droplet digital PCR, was used to quantify their absolute number.

In this study, we aim to understand how the human gut microbiome changes in response to diet over time, with a focus on reproducibility and stability.

Poster 017:
Investigating the role of human small intestine microbiota in explaining interpersonal differences in glycemic responses upon consumption of bread. The GLYSIMI proof of principle study

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Introduction: Person-specific factors, such as the fecal microbiome, are thought to influence postprandial glucose responses (PPGRs) after consumption of meals. Since the small intestine is the site of carbohydrate digestion and absorption, the small intestine microbiota may be key in explaining the interpersonal differences in glycemic responses. The main objective of our study is to investigate the role of the small intestine microbiota in regulating PPGRs towards standardized food products in humans.

Methods: 20 Males and females (age 40 to 75 years, with a BMI≥25 kg/m2) were included in a 6-day cross-over dietary intervention study. A continuous glucose monitor (CGM) was used to monitor blood glucose values for six days. Standardized meals were provided from day 3 to day 6. Participants were randomized to consume breakfast either consisting of refined wheat (RW) or whole grain wheat (WGW) bread on day 4 and the other bread on day 6. Both breakfasts contained the same amount of carbohydrates. Small intestinal content were collected at baseline and postprandially and analyzed for microbiome composition.

Results: In most of the subject's glucose values returned to baseline 180 minutes after bread consumption. For the same subject, the glucose iAUC0-120min did not significantly differ between the two bread types, but the curve of postprandial glucose was different. At the Phylum level, microbes are mainly composed of: Firmicutes, Actinobacteriota, Proteobacteria, Bacteroidota, but the composition varied over time and had inter-personal differences. Diversity analyses of small intestinal microbiota samples showed significant differences in microbial composition between subjects at baseline.

Conclusions: RW bread and WGW bread induce a differential PPGR and small intestine microbiota response. This study was registered at ClinicalTrials.gov under the identifier NCT05120661.

Poster 018:

Microbiota-Dependent Fiber Responses: A Proof-of-Concept Study on Short-chain Fatty acid production in Prevotella- and Bacteroides-Dominated Individuals

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Background/Objective: Dietary fiber supports metabolic health via microbial fermentation, producing short-chain fatty acids (SCFAs). However, metabolic responses to fiber vary between individuals, potentially due to different gut microbiota composition. The Prevotella-to-Bacteroides (P/B) ratio has emerged as a potential biomarker for fiber responsiveness. This study examined how stratified fiber supplementation affects microbial and metabolic outcomes in individuals with Prevotella- or Bacteroides-dominated microbiota.

Methods: In this single-blinded, randomized cross-over study, 23 healthy adults were classified as P-type (≥10% fecal Prevotella) or B-type (≥10% Bacteroides) via 16S rRNA sequencing. Participants consumed 15 g/day of arabinoxylan (AX), inulin (INU), or placebo (PLA) for one week each, with 2-week washouts. After each phase, fasting and postprandial plasma SCFAs, branched-chain fatty acids (BCFAs), breath hydrogen (H_2), glucose, insulin, PYY, cholesterol, appetite ratings, and fecal microbiota were assessed. Data were analyzed using repeated measures ANOVA, Friedman test, and multivariate microbiome analysis.

Results: Compared to PLA, AX increased fasting propionate in both groups ($P<0.05$) and postprandial acetate in B-types ($p=0.04$). INU reduced fasting BCFAs in B-types ($p<0.05$) but did not increase SCFAs. Breath H_2 varied widely in B-types after INU but not in P-types. Neither fiber affected glucose, insulin, or PYY. AX reduced appetite ratings in P-types ($p<0.05$). INU increased Anaerostipes and Bifidobacterium and reduced Phocaeicola in both groups (q<0.25). AX increased Fusicatenibacter in B-types ($q=0.18$) and Paraprevotella in P-types ($q=0.17$).

Conclusion: B-type individuals exhibited fiber-specific shifts in SCFA and BCFA metabolism and H_2 exhalation, while P-types remained comparably unresponsive. These findings highlight the complexity of diet-microbiota interactions and support the need for microbiota-based nutrition strategies.

Theme 3: Can AI help bridge the precision & public health nutrition?

Poster 019:
Predictive Algorithm-Based Biomarkers of Ageing in Human Nutrition Research: a focus on applications, challenges and opportunities

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In the search for healthy ageing strategies, predictive algorithm-based biomarkers of ageing (BoA), such as epigenetic clocks, are being increasingly applied within human nutrition research. Their promise lies in the ability to assess ageing in relatively short timeframes, and in case of ideal BoA that capture early ageing signals, to assess ageing in younger and preclinical populations. However, validation efforts and guidelines for implementation are lagging behind the vast and growing number of available biomarkers, introducing inconsistencies in use across nutritional studies.

As such, we critically evaluated the current applications, important considerations, challenges and limitations regarding implementation of BoA in nutrition research. Accordingly, we formulated practical insights and guidelines for future implementation, both on a general level and within different human nutrition research scenarios, to improve comparability of studies. Our four most important general guidelines comprise:

-Use multiple n ≥ 2 (complementary) biomarkers, selected according to the research aim and comparable previous studies, and substantiate choices made.

-Focus on second or next generation BoA, trained on relevant physiological or other age-related outcomes, shown predictive of prospective health (beyond CA and mortality)

-For assessment of past exposures use ageing clocks as primary endpoint; for current exposures use pace of ageing clocks.

-Establish criteria for successful anti-ageing strategy a priori e.g. 2/3 of BoA show significant association or response, and draw conclusions accordingly. Lastly, we propose several potential applications of BoA, and address directions for future nutrition research involving BoA, including promising dietary interventions and relevant study designs. Overall, despite much needed advancements, we consider these predictive BoA exciting tools for nutrition and ageing research, to ultimately refine dietary advice for healthy ageing.

Poster 020:
An integrated ML model to unravel phenolic metabotypes and assess their impact on subjects at cardiometabolic risk

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The growing availability of health-related big data has opened new opportunities in the field of precision nutrition. However, translating personalized approaches into effective strategies for improving public health remains a major challenge.

In this context, the PRE-CARE-DIET study aims at assessing how a personalized, sustainable, (poly)phenol-rich (PP) diet can affect cardiometabolic (CM) health accounting for the individual differences in these plant bioactives' metabolism. A targeted, randomized, controlled intervention study is being carried out in adults at CM risk.

A total of 500 volunteers are profiled based on their ability to metabolize phenolic compounds after an Oral (Poly)phenol Challenge Test (OPCT). Of these, 2/3 are enrolled according to their phenolic excretion phenotypes (metabotypes) and randomly assigned to either a personalized PP-rich dietary intervention or a control group receiving general dietary guidelines. Biological samples are collected and analyzed using multi-omics approaches to enable deep phenotyping.

An AI-driven feature selection process was applied to select a reduced panel of 25 key metabolites from over 210 phenolic metabolites identified in the OPCT study for participants' allocation into two phenolic metabotypes, allowing an easy-to-handle, robust, reproducible targeted recruitment. Different machine learning (ML) algorithms were tested to evaluate predictive accuracy, misclassification rates, and model robustness across several features.

Random Forest emerged as the best-performing model to cluster volunteers. Moreover, CM risk prediction scores are investigated to evaluate the effect of PP on health. The integration of metabolic, clinical, and omics data using AI/ML techniques represents a promising tool to bridge the gap between precision nutrition and public health initiatives, enhancing the development of tailored, evidence-based nutritional interventions that are either personalized or scalable at the population level.

Theme 4: Nutrition and the brain throughout the life course: insights from molecular mechanisms to clinical applications

Poster 021:
Effect of citicoline-containing supplement on the lipid profile in healthy volunteers in relation to lifestyle habits

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Stroke is one of the leading causes of mortality. Considering the fact that over 80% of strokes are due to lifestyle-related risk factors, prevention is essential. Citicoline (CDP-choline) is used as supplementary therapy in stroke recovery. It has a beneficial effect on lipid metabolism with a pronounced protective effect on the neural membrane phospholipids. There is no data on the relationship between lifestyle and the effect of citicoline on lipid metabolism. We evaluate the protective effect of a combined supplement containing citicoline on the lipid profile in healthy volunteers concerning lifestyle habits.

A six-month intervention was conducted with a dietary supplement containing citicoline in combination with rosehip, aronia and green tea extracts. Forty-two volunteers, aged 45-65 (F30/M12) were enrolled. Lipid parameters were measured before and after the intervention. Volunteers were stratified in related groups depending on their lifestyle habits. Plasma total cholesterol (TC) (5.61 mmol/L/Lvs5.33 mmol/L; p=0.02) and LDL-cholesterol (LDL-C) (3.38 mmol/L/Lvs3.15 mmol/L; p=0.03) decreased after the intervention. No differences were observed for HDL-cholesterol and triglycerides. The improved levels in TC and LDL-C seem more noticeable in the non-smokers (5.44 mmol/L/Lvs5.06 mmol/L; p=0.005 and 3.18 mmol/L/Lvs2.89 mmol/L; p=0.017, respectively). No differences were detected for the same two parameters in the smokers.

Regarding the alcohol consumption, TC and LDL-C significantly decrease equally for low and high alcohol consumption groups. Citicoline with plant extracts improve the lipid profile by modifying TC and LDL-C. Alcohol consumption does not influence this beneficial effect. In smokers, no beneficial effect on the lipid profile was found, therefore it can be assumed that tobacco neutralizes the beneficial effect of the supplement. Funding: European Union-NextGenerationEU BGRRP-2.004-0009-C02; Medical University Varna, Science Fund, Project No 21015

Poster 022:

Effect of a nutraceutical supplement on serum arachidonic acid, oxidative stress markers, and lipid profile in healthy adults

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The brain is highly susceptible to lipid peroxidation due to its high metabolic activity, energy demands, and reliance on aerobic metabolism. Arachidonic acid (AA), released from membrane phospholipids in response to inflammation and oxidative stress contributes to reactive oxygen species formation and secondary lipid mediators that can cause impaired cognitive function.

We aimed to investigate the effect of a nutraceutical supplement containing citicoline and antioxidant-rich plant extracts on markers, related to lipid metabolism and oxidative stress, such as AA, lipid profile, and malondialdehyde (MDA) in a six-months intervention study. Serum AA was quantified using a validated in-house UPLC-MS method. MDA levels were determined by a commercial ELISA immunoassay kit, and routine lipid profile parameters (TG, TC, HDL-C, LDL-C) were analyzed on automatic biochemical analyzer.

Post-intervention data revealed statistically significant reductions in serum levels of free AA compared to pre-intervention levels (median pre- 1,77, IQR: 1,18-2,47, median post- 1,30, IQR 0.95-1.89), MDA (median pre- 3,11, IQR 2,65-3,76); median post-: 2,17, IQR 2,02-2,3), TC (median pre- 5,51, IQR 4,93-6,12); median pre- 5,07, IQR 4,64-6,0), and LDL-C (median pre-: 3,10, IQR 2,90-3,66); median post-: 2,93, IQR 2,60-3,44). Specifically, a 25% reduction in serum AA concentration was observed after supplementation. A clear negative trend (Spearman r -0.27) between free AA and MDA concentration before the intervention was indicated. No correlation between AA levels and TC, LDL-C, HDL-C and TG was observed.

In conclusion, we suggest that citicoline, in combination with plant extracts, has the potential to support cognitive health by stabilising membrane phospholipids and reducing systemic oxidative stress. Acknowledgements: This research was supported by the European Union-NextGenerationEU Project No BGRRP-2.004-0009-C02 and by Medical University Varna, Science Fund, Project No 21015.

Theme 5: Nutrition and inflammation: friend or foe?

Poster 023:
Impact of chronic consumption of Alphitobius diaperinus on gut inflammation and barrier function in healthy and diet-induced obese female rats

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The search for sustainable protein sources has increased interest in edible insects for their potential health benefits. Insect proteins have been suggested to modulate intestinal inflammation and barrier function—key processes in obesity-related metabolic disturbances. However, these effects remain poorly understood, in both healthy and obesogenic conditions.

The aim of this study was to evaluate the effects of chronic consumption of A. diaperinus on the inflammatory response and intestinal barrier integrity. Forty 22-week-old female Wistar rats were assigned to five dietary groups for four weeks: casein (control), beef, insect (as sole protein source), and an obesogenic group fed a cafeteria diet (CAF) with either casein (CAF) or insect (CAF+I) Gene expression related to inflammation and barrier integrity was analysed throughout the small intestine.

Under healthy conditions, insect protein reduced inflammatory markers and modulated barrier-related genes in the duodenum compared to the control diet. In the jejunum, beef protein increased inflammatory gene expression compared to both control and insect groups, while insect protein improved barrier integrity markers. In the ileum, beef protein only increased specific inflammatory markers. In the obesogenic condition, the cafeteria diet affected the expression of inflammatory markers and and these changes persisted despite the replacement casein with insect protein (CAF+I).

Therefore, our results suggest that chronic consumption of an A. diaperinus-based diet promotes a more favourable intestinal profile than beef under healthy conditions, particularly in the duodenum and jejunum. These results support the potential of insect protein not only as a sustainable alternative but also as a modulator of gut health.

Poster 024:
Vitamin D3 Fine-Tunes Innate Immune Signaling: Transcriptomic and Pathway-Level Insights from In Vivo Human Studies

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While most prior insights into vitamin D's immunoregulatory role stem from in vitro studies using supraphysiological doses, recent in vivo intervention trials provide more physiologically relevant data. Here, we applied a novel approach combining transcriptomic profiling with in-depth manual pathway annotation, offering mechanistic insights beyond those afforded by automated methods.

Through two vitamin D3 intervention studies, VitDHiD (NCT03537027) and VitDPAS (NCT06104111), involving a total of 70 healthy individuals who each received a single bolus of 80,000 IU vitamin D3, we identified 232 consistently regulated target genes (FDR < 0.05) in peripheral blood mononuclear cells (PBMCs). Of these, 61 genes were associated with innate immune responses. Manual curation revealed that vitamin D downregulated five major inflammatory pathways, stabilized two, and upregulated one. These pathways include Toll-like receptor, NOD-like receptor, C-type lectin receptor, and IL17 signaling, which are key mediators of innate immunity. Core regulatory genes such as NFKBIA, NFKBIZ, FOSL2, JDP2, PIK3R1, DUSP6, TNFAIP3, and CLEC7A suggest a coordinated mechanism by which vitamin D limits acute inflammation and reduces cytokine production.

To expand on these findings, we are currently exploring how vitamin D modulates both shared (e.g., NFκB) and ligand-specific responses in monocyte models stimulated with pathogen- and damage-associated molecular patterns (PAMPs/DAMPs). Altogether, this gene-focused, pathway-aware strategy provides comprehensive insight into vitamin D's in vivo immunoregulatory actions, emphasizing its role in fine-tuning immune signaling and maintaining immune homeostasis in healthy individuals.

Poster 025:
Vitamin D Supplementation Modulates Inflammatory Gene Networks: A Transcriptome-Wide Analysis in Two European Cohorts

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The VitDPAS study (NCT06104111) was designed as a medical experiment to investigate the in vivo effects of vitamin D3 on immune function. A cohort of 45 healthy individuals from Olsztyn, Poland, received a single, body weight-adjusted bolus of vitamin D3 (1,000 IU/kg). Transcriptome-wide differential gene expression analysis of peripheral blood mononuclear cells (PBMCs), collected before and 24 hours after supplementation, revealed 758 significantly regulated genes ($p< 0.05$). Based on the correlation between individual gene expression changes and the increase in vitamin D status, participants were stratified into three response categories: 17 high, 19 mid, and 9 low responders.

Comparative analysis with the VitDHiD study (NCT03537027), conducted on 25 healthy participants in Finland, identified 232 overlapping target genes, allowing an integrated evaluation of vitamin D responsiveness across both cohorts ($n = 70$). Applying a stricter statistical threshold ($FDR < 0.05$) yielded 26 shared target genes, reflecting a robust and reproducible in vivo response to vitamin D3.

Functional annotation of these genes highlighted the downregulation of inflammatory signaling pathways, particularly those mediated by tumor necrosis factor (TNF) and nuclear factor κB (NFκB), as a core response. These findings reinforce the immunomodulatory role of vitamin D in healthy individuals and support the vitamin D response index as a personalized marker for supplementation efficacy. Notably, the identification of low responders within the Polish cohort emphasizes the need for individualized strategies to ensure sufficient vitamin D status during the winter months. In summary, the VitDPAS study validated key principles of vitamin D action, offering insight into the molecular basis of interindividual differences in immune responsiveness to vitamin D.

Poster 026:
Vitamin D3 Rapidly Regulates Genes Involved in Redox Homeostasis and Detoxification In Vivo

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Vitamin D3 is a vital micronutrient that can be synthesized endogenously in the skin upon UV-B exposure or obtained through diet and supplementation. One of its evolutionarily conserved roles is maintaining energetic and survival homeostasis, including detoxification. In addition, vitamin D is a key modulator of both innate and adaptive immunity. This study aimed to uncover the in vivo gene regulatory mechanisms underlying these functions.

We conducted a proof-of-concept intervention in which a healthy individual received a monthly oral bolus of 80,000 IU vitamin D3 for three months. Peripheral blood mononuclear cells (PBMCs) were collected immediately before and at 4-, 24- and 48-hours post-supplementation for transcriptome-wide differential gene expression analysis. A total of 570 genes were significantly regulated by vitamin D3 ($p < 0.05$) at one or more time points. In parallel, in vitro stimulation of PBMCs from the same participant with 1α,25-dihydroxyvitamin D3 confirmed 303 of these, as direct vitamin D receptor (VDR) target genes.

Among these, 55 primary response genes were significantly regulated as early as 4 hours post-supplementation. Notably, several of these, including SELENOS (selenoprotein S), PRDX1 (peroxiredoxin 1), TXNRD1 (thioredoxin reductase 1), and SOD2 (superoxide dismutase 2), are central to redox regulation and antioxidant defense. These results suggest that vitamin D3 initiates a rapid transcriptional program in vivo, with early activation of genes involved in detoxification and oxidative stress management. This highlights a potential primary role of vitamin D in maintaining redox homeostasis, extending beyond its classical immunomodulatory and metabolic functions in healthy individuals. This study is further extended by examining transcriptome-wide differential gene expression, stress resistance, and cellular responses at additional timepoints (2-, 4- and 6-hours post-supplementation).

Poster 027:
Evaluating Inflammatory Resilience: Insights from Energy Restriction Studies

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Assessing the health impacts of nutritional interventions in metabolically compromised individuals is challenging and requires precise tools. Composite biomarkers measured during challenge tests have shown the benefits of whole-grain wheat in overweight individuals by improving inflammatory resilience and phenotypic flexibility. Further development of these methods is essential to address low-grade inflammation.

This study tested the composite biomarker's feasibility through secondary analysis of two energy restriction (ER) trials, Bellyfat and Nutritech, analyzing fasting and postprandial inflammation using various markers. Four biomarker health space models based on postprandial responses were statistically evaluated over 12 weeks of ER. Composite biomarkers of inflammation showed significant effects of ER, thereby also correlating inflammatory resilience with BMI and body fat. This study supports using composite biomarkers to evaluate effects of ER interventions, suggesting the potential as a novel method for assessing inflammation and phenotypic flexibility.

Poster 028:
Circadian and Autophagic Dysregulation in Cafeteria Diet-Fed Rats: Protective Role of GSPE

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Obesity disrupts liver homeostasis, leading to autophagic dysfunction and altered expression of key circadian proteins such as BMAL1 and REV-ERBα. Polyphenols— natural compounds with antioxidant and anti-inflammatory properties—have been proposed to modulate these processes and contribute to the restoration of cellular homeostasis. This study aimed to assess whether supplementation with grape seed proanthocyanidin extract (GSPE) can attenuate circadian and autophagic impairments induced by a cafeteria (CAF) diet.

For this purpose, 48 rats were distributed into three groups standard diet (STD), CAF diet, and CAF diet supplemented with GSPE (25 mg/kg), administered at the onset of their active phase. The results showed that the CAF diet disrupted the circadian rhythmicity of BMAL1, and desynchronized hepatic autophagy regulation, as indicated by reduced LC3 protein expression. GSPE supplementation partially restored BMAL1 oscillation and promoted autophagy activation increasing LC3 levels at key circadian time points.

These findings suggest that GSPE can mitigate obesity-induced alterations in circadian and autophagic regulation, pointing to BMAL1 as a potential link between the circadian clock and hepatic autophagy.

Poster 029:
Vitamin D3 as a Systemic Epigenetic Modulator In Vivo: Immune-Related Gene Expression and Chromatin Accessibility in Human Immune Cells

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Vitamin D3 enhances innate immune responses and modulates adaptive immunity, with in vitro studies suggesting epigenetic mechanisms underlying these effects. However, in vivo evidence remains limited. This study integrates data from a proof-of-principle experiment, where a participant received monthly doses of 80,000 IU of vitamin D3 for three months, and the VitDPAS cohort study (n=45).

In the individual experiment, peripheral blood mononuclear cells (PBMCs) were collected before supplementation (day 0) and one and two days after (days 1 and 2). In the cohort study, PBMCs were collected on day 0 and day 1. Chromatin accessibility and gene expression were assessed via ATAC-seq and RNA-seq, respectively. RNA-seq of the proof-of-principle experiment revealed 380 vitamin D target genes with time-dependent expression changes, including immune-related genes such as DUSP6 and FOS. Functional enrichment highlighted pathways in innate immunity and interferon signaling.

Integrative analysis showed that 306 of these genes were linked to nearby vitamin D-sensitive chromatin regions, including promoters and enhancers. ATAC-seq identified 41,536 consensus peaks in the cohort, with 14,119 annotated as promoters, compared to 33,837 consensus peaks and 11,069 promoters in the individual experiment. Filtering for strongest peaks yielded 4,291 peaks in the cohort (3,872 promoters) and 3,539 in the individual case (2,535 promoters). However, chromatin accessibility patterns did not consistently align between the two approaches. Notably, substantial inter-individual variability was observed, with divergent chromatin accessibility patterns in more than 75% of detected regions post-supplementation.

These findings provide in vivo evidence that vitamin D3 serves as a systemic epigenetic modulator with immune-regulatory effects, emphasizing its potential role in personalised nutrition and immune health.

Poster 030:
Distinct postprandial metabolic and inflammatory responses in healthy individuals versus patients with metabolic dysfunction-associated steatotic liver disease

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Metabolic dysfunction-associated steatotic liver disease (MASLD) is a spectrum of conditions from simple steatosis to metabolic dysfunction-associated steatohepatitis (MASH), characterized by inflammation and fibrosis. Low-grade inflammation, possibly driven by crosstalk between immune and metabolic pathways, may play a crucial role in the progression to MASH, wherein dietary responses may contribute to this inflammatory-fibrotic state. This research aimed to explore whether postprandial metabolic and inflammatory responses to dietary triggers, e.g. saturated fat or fructose, may provide insights into the mechanisms driving MASLD-MASH progression.

34 patients with obesity and MASLD and 10 healthy controls were recruited. Patients with MASLD were stratified by liver fibrosis score (FibroScan; F0/1 MASLD, n = 18; F2/3 MASH, n = 16). MASLD, MASH, and healthy completed a high-fat mixed meal test (46g fat (33g saturated fat), 50g carbohydrates, 10g protein). MASLD and MASH also completed a high-fructose/glucose challenge (75g fructose, 25g glucose). Blood samples were drawn over 6-hours to evaluate metabolic (glucose, insulin, triglycerides (TAG), non-esterified fatty acids (NEFA)) responses. Ex vivo cytokine (IL-1β, IL-6, CCL3) responses to bacterial (TLR2/4) and viral (TLR7/8) ligands were determined in whole blood. Insulin resistance (HOMA-IR) and postprandial increases in glucose, insulin and TAG were greater in MASLD and MASH vs healthy, but were similar between MASLD and MASH. Interestingly, distinct whole blood inflammatory profiles were observed. MASLD, but not MASH, exhibited greater whole blood cytokine responses to bacterial and viral ligands compared to healthy. The IL-1β response to viral ligands was also higher in MASLD vs MASH.

The preliminary data suggests that whilst a continuum of postprandial metabolic dysfunction presents in MASLD vs MASH, there are nuances with respect to inflammatory responses which may/may not explain fibrotic disease progression.

Theme 7: Diet, Genes & Health

Poster 031:
Differential gene expression following meal consumption: A systematic review and quantitative data synthesis of postprandial transcriptomics studies

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Aim: Metabolic balance is essential for health, but it can be disrupted by short-term metabolic disturbances. Postprandial transcriptomic studies provide valuable information about how gene expression levels and metabolic processes are affected by food consumption. However, the complexities of postprandial gene expression changes across different meal types and time points remain poorly understood, partly due to variations in study designs and analytical methods.

Method and Results: This systematic review examined postprandial transcriptomic responses to various meal types by re-analysing raw data from 14 studies. We identified 36, 711, and 12 genes that were similarly changed (adj $p< 0.05$) in at least three studies at 2, 4, and 6 h after a meal, respectively. The biological processes associated with these genes included immune response at 2 h, complex cellular regulation and stress response at 4 h, and circadian rhythms at 6 h. A multivariate integrative analysis (MINT.sPLS) did not reveal significant effects of macronutrient composition on gene expression levels across the studies.

Conclusion: Despite differences in study designs and meal types, common postprandial gene expression responses were identified across studies, and these changes were linked to specific biological processes at various postprandial time points. Our findings suggest that maintaining metabolic balance involves complex biological responses in multiple pathways, particularly at 4 h after a meal. This study also emphasises that factors beyond macronutrient composition play a significant role in influencing gene expression levels, offering new insights into postprandial metabolic responses and identifying potential biomarkers for health.

Poster 032:
Changes in saliva microbiota during a follow-up study of Finnish adolescents

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The prevalence of obesity is increasing globally, which concerns children and adolescents as well. Saliva microbiota has gained increasing interest as an alternative to gut microbiota while having potential associations with various systemic diseases. The alterations in diversity or composition of saliva microbiota have been connected to various diseases including obesity, but with conflicting results. In this study we investigated the changes in microbiota diversity and composition among those with stable weight vs increased weight using a follow-up study design.

For this study we have selected 440 children from the Finnish Health in Teens (Fin-HIT) cohort study, for whom we have saliva sample available at two timepoints. Using 16S rRNA gene sequencing, we characterized the saliva microbiota of children at mean ages (SD) of 11.7 (0.3) years and 14.2 (0.4) years. We excluded subjects with recent antimicrobials use (> 3 months). Participants were divided into BMI categories using the cut-offs from the International Obesity Task Force. Here we report the preliminary findings concerning saliva microbiota composition between children with stable and increased BMI category.

Saliva microbiota composition differed between the two timepoints in all children: however, children with stable normal-weight showed no differences in saliva microbiota composition between the time points (pcorr=0.19), neither did we observe any differences in the group whose BMI increased from normal-weight to overweight (pcorr=0.7). However, in those with stable overweight/obesity the composition of microbiota differed at the two timepoints (pcorr=0.052): the most notable differences were seen in Proteobacteria at phylum level (relative abundance increased from 16.7% to 18.3%) and Haemophilus at genus level (from 7% to 9.4%).

Our preliminary findings suggest that changes in saliva microbiome with time were more pronounced in those with stable overweight/obesity, but affected my multiple factors.

Poster 033:
Genetic contributors of core oral microbiota composition – a meta-analysis of three Finnish genome-wide association studies

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Background: Oral microbiome composition presents substantial inter-individual differences, which may be partially explained by genetic variations – an underexplored area. Investigating the association of common host genetic variants with oral microbiome composition could provide insights into the biological mechanisms governing the ecological balance.

Materials and methods: We collected data on oral microbiome composition with 16S RNA gene profiling and genome-wide genetic factors across three Finnish cohorts: Panic (n=377, 7.6 (SD=0.4) yrs, 47% girls), Fin-HIT (n=629, 11.7 (0.3) yrs, 52% girls) and NFBC1966 (n=747, 46.3 (0.4) yrs, 57% female). In each cohort we conducted genome-wide association (GWA) study with 19 common bacterial genera.

Results: The core microbiome composition differed substantially between the cohorts. Five GWA-significant loci were identified in the Panic cohort (2 for Actinomyces, 1 for Fusobacterium, and 2 for Capnocytophaga), two loci in Fin-HIT (Peptostreptococcus and Veillonella), and three in NFBC1966 (TM7x, Oribacterium, and Veillonella). In NFBC1966, Oribacterium was significantly associated with genetic variation in a locus at chromosome 21 led by SNP rs141001537 (EAF=6.5%, β=-1.77 (0.31), p=6.6x10-9). The association was replicated in Fin-HIT cohort (β=-0.31 (0.14), p=0.02), but not in Panic (β=-0.16 (0.16), p=0.30). The Oribacterium genus was most abundant in Fin-HIT cohort (1.8%), followed by NFBC1966 (0.5%), but rare in Panic. Oribacterium is a strictly anaerobic bacterial genus associated with oral dysbiosis and gingivitis.

Conclusions: Common genetic variants linked to the composition of the core oral microbiota vary across Finnish cohorts with different age. Oral microbiome composition may represent a trait demonstrating genetics-by-age interactions.

Poster 034:
Diet-derived microRNA stability, cellular uptake and biological effects following gastrointestinal digestion

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Diet-derived miRNAs, of both animal and plant sources, have gained traction in recent years as novel modulators of gene expression in animals, as shown by diverse studies. In this regard, our group has recently developed and validated a protocol to purify diet-derived miRNAs (with a focus on plant origin) from gastrointestinal digesta. Using said method we performed an in vitro static digestion (INFOGEST) of frequently consumed vegetables (cauliflower, spinach, chickpea) and analyzed the stability of their miRNAs.

Our data indicates that even though there is a degradation encompassing several orders of magnitude, survival of plant-miRNAs through digestion is possible. Specifically, fibrous vegetables (i.e. cauliflower) seem to produce the highest concentrations of miRNAs in the digesta, with mir-172a, a highly conserved miRNA in the plant kingdom, being detected at a concentration of 7.4 x 104 copies/g plant tissue. Having studied the stability of miRNAs through digestion, we performed an uptake analysis with CaCo-2 cells as a model of the intestinal epithelia and a mimic of mir-172 at different concentrations. No coadjutants that facilitate the uptake were used. The data indicated that diet-derived miRNAs are absorbable in the fM range when supplemented in the media at concentrations as low as 16.5 pM. We hypothesize that both survival of miRNAs and cellular uptake are facilitated by encapsulation in exosome-like nanoparticles. Therefore, we have initiated studies to assess the role of nano-encapsulation in miRNAs for gastrointestinal stability.

Furthermore, we are comparing said stability to non-encapsulated miRNAs. In addition, the role of exosomes in mediators of cellular uptake is envisioned with plant-derived exosomes that will help us understand how these may improve absorption by the gut epithelia. In conclusion, we have conducted miRNA sequencing in gastrointestinal digesta from cauliflower to identify relevant miRNAs for human health that survive digestion

Poster 035:

Modulation of lipid metabolism in obese rats: sex and photoperiod response of a multi-ingredient based on polyphenol-enriched winter-fruits extracts.

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Fruit polyphenols have demonstrated beneficial effects on metabolic syndrome in obese rats. However, their bioactivity is influenced by several factors, including photoperiods. Seasonal rhythms, the light-dark variations during the day, modulate physiological parameters in rodents such as body weight, behavior, and lipid metabolism. Additionally, there are sex-specific differences, with females generally showing higher fat mass but a healthier lipid profile due to estrogen protection. In a previous study, we observed photoperiod-dependent effects of grape and pomegranate polyphenol-rich extracts on biochemical parameters.

This study aimed to assess a novel multi-ingredient formulation derived from these winter fruits on lipid metabolism in obese, dyslipidemic male and female rats under short (L6, 6 h light/day) or long (L18, 18 h light/day) photoperiods. Rats were fed a cafeteria diet for 11 weeks; during the last 8 weeks, they were kept under L6 or L18, and during the final 5 weeks, they received the polyphenol-rich formulation or vehicle. We evaluated plasma lipids, hepatic lipid content, and hepatic expression of genes related to lipid metabolism. Results showed that when the multi-ingredient is administrated to males exposed to L18, it reduces ALT levels and increases the hepatic phospholipids. In addition, a downregulation of Ppar-α and Fabp4 occurs without altering total hepatic lipid content. On the other hand, in females exposed to L6, multi-ingredient affects plasmatic glucose levels and upregulates Fasn and Fabp4 without altering lipid content in liver. Furthermore, the administration of the multi-ingredient during long photoperiod in females reduced total cholesterol and bile acids.

Overall, the multi-ingredient has more benefits in males under long photoperiod and in females under short photoperiod. This study could lead to novel and personalized therapeutic strategies to mitigate obesity, particularly focus to differences based on sex and photoperiod

Poster 036:
Vitamin D-Induced Epigenetic Memory: Dissecting VDR-Mediated Regulatory Programs in Human Monocytes

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Dietary compounds such as vitamin D play a central role in regulating immune cell function. Numerous studies have shown that vitamin D exerts protective effects against autoimmune diseases by modulating gene expression in immune cells. This regulation is mediated by the active vitamin D metabolite, 1,25-dihydroxyvitamin D (1,25D), through its binding to the transcription factor vitamin D receptor (VDR). However, how VDR binding contributes to long-term immune adaptation being based on epigenetic memory, remains poorly understood.

To address this gap, we investigated the time-resolved dynamics of VDR binding and chromatin remodeling in THP-1 human monocytes. Using ChIP-seq, we mapped VDR occupancy at 40 minutes, and 2, 4, 8, and 24 hours after 1,25D treatment. These data were integrated with RNA-seq-based gene expression profiles obtained at 2.5, 4, and 24 hours post-treatment. This integrative approach allowed us to classify immune-related genes based on their inducibility, basal expression, and enhancer-driven regulatory dynamics over time. We identified hundreds of genomic regions exhibiting vitamin D–responsive epigenetic signatures, suggesting a general mode of action.

The planned integration of additional spatial-temporal data, such as chromatin accessibility, histone modifications, and pioneer factor binding, will support the development of mechanistic computational models of vitamin D–dependent gene regulation. Our findings uncover a nutrigenomic mechanism through which dietary vitamin D imprints epigenetic memory in immune cells. This work links nutrient intake to long-term immune plasticity and offers insights that could inform personalized nutrition strategies to modulate immune function.

Poster 037:
Integration of Nutrigenetic Secondary Findings in Whole Exome Sequencing: A Pilot Approach Toward Personalized Medicine and Prevention

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Whole exome sequencing (WES) is primarily used for diagnosing rare genetic conditions, yet its scope allows for the detection of secondary findings with implications for preventive care. In this pilot project, we integrated nutrigenetic findings into the clinical WES workflow, enabling personalized dietary and supplementation strategies as part of a precision health approach.

The study included 100 patients with diverse clinical backgrounds, including neurodevelopmental disorders, nephrology conditions, cardiovascular disease, and reproductive failure. A hard panel of over 30 nutrigenetically relevant genes was curated based on robust clinical evidence. This included genes involved in folate and vitamin D metabolism, lipid and carbohydrate processing, micronutrient transport, and common food intolerances. Key examples included APOE (lipid metabolism), FADS1 (omega-3 processing), GC (vitamin D transport), LCT (lactose tolerance), and SLC30A8 (zinc and insulin function).

Nutrigenetic variants were systematically analyzed as secondary findings and interpreted alongside primary diagnostic results. Based on genotype, concise and actionable dietary or supplementation recommendations were generated, such as micronutrient optimization or tailored macronutrient intake. Importantly, this additional layer of interpretation had no impact on the diagnostic pipeline, but provided added clinical value for preventive care. This approach demonstrates the feasibility of integrating nutrigenetic secondary findings into clinical WES workflows. It represents a novel paradigm where genomic diagnostics can simultaneously inform diagnostic outcomes, therapeutic decisions, and long-term preventive strategies tailored to the individual's genetic profile.

Poster 038:
A scoring system for validity assessment of biomarkers of food intake

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Biomarkers of food intake (BFIs) can be a valuable tool to complement traditional dietary assessment, leading to a more precise dietary assessment and therefore, to less ambiguous associations with health-related outcomes. Although in the last years numerous new BFIs have been discovered, so far, only a very limited number has proven suitable for practical implementation in nutritional studies due to missing information about their validity. Comprehensive and consistent validation is crucial to ensure reliable and precise dietary assessment using BFIs. The degree of BFI validation varies considerably due to limited availability of validation frameworks providing clear guidance, and scattered information about validation criteria across the literature, requiring extensive effort to compile.

To propose a way forward, the FoodPhyt consortium performed a thorough systematic literature search about validation data of putative BFIs to i) identify the most important validation criteria, ii) identify missing validation information; and iii) most importantly built a scoring system based on the FoodBAll validation scheme, to standardize the scoring of BFI candidates and enable nuanced comparison of BFIs with varying validity. The bipartite score includes BFI quality (performance) and availability of supporting literature (data availability) for the following validation criteria: identification and plausibility as pass-or-fail criteria, specificity, variability in foods and biological samples, robustness, and analytical performance. In addition, the detailed validation data and validity scores of BFI candidates will be made available through a freely available online repository, BFI-Hub. The FoodPhyt scoring system, as well as the new searchable BFI-Hub database will enable the selection and application of BFIs in nutritional and health research in future.

Poster 039
Folate depletion alters DNA methylation in tissues relevant for childhood leukaemia

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Epidemiological evidence suggests maternal folate status influences childhood leukaemia risk; however, the underlying mechanisms are unclear. Genetic aberrations, are considered leukaemia-initiating events, however, alone they are not sufficient for disease, with additional 'hits' required. Epigenetic alteration, i.e. DNA methylation, is one mechanism by which maternal folate is likely to contribute to disease.

We investigated the relationship between folate depletion and DNA methylation in murine tissues relating to childhood leukaemia. Female mice were allocated low or normal folate diets for 4 weeks before mating and onwards. Offspring were sampled at 17.5 days gestation and weaning. DNA was isolated from fetal livers and bone marrow of weaned mice. Tissues correspond to specific sites of haematopoiesis throughout the lifecycle relevant to leukaemic blasts.

Epigenome-wide DNA methylation was profiled using Infinium® MouseMethylation BeadChips. R/Bioconductor packages identified differentially methylated points (DMP) and regions (DMRs). Pathway analysis was conducted by DAVID. There were 598 DMPs (382 hypermethylated/216 hypomethylated) and 363 DMRs with significantly (FDR) altered methylation in response to folate depletion in the fetal liver. In bone marrow, 1206 DMPs (334 hypermethylated/852 hypomethylated) and 565 DMRs had significantly altered methylation in response to folate depletion. Only 3 DMPs and 13 DMRs were altered in both tissue types, suggesting highly tissue specific responses.

Pathway analysis suggested 15 and 44 pathways could be altered in response to folate depletion in liver and bone marrow respectively, with 8 overlapping between tissues. Whilst loci specific altered methylation appears to be different between tissues, the impact on biological pathways is similar.

Poster 040:
Maternal α-casein deficiency extends the lifespan of offspring and programs their body composition

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Early nutrition has significant effects on physiological outcomes during adult life. We have analysed the effect of maternal α-casein (CSN1) deficiency on the physiological fate of dams and their offspring. α-casein deficiency reduces maternal milk protein concentration by more than 50% and attenuates growth of pups to 27% ($p<0.001$) of controls at the point of weaning. This is associated with a permanent reduction in adult body weight (-31% at 25 weeks).

Offspring nursed by α-casein deficient dams show a significantly increased lifespan (+22%, χ^2: 10.6; $p=0.001$). Liver transcriptome analysis of offspring nursed by α-casein deficient dams at weaning reveals gene expression patterns similar to those found in dwarf mice (reduced expression of somatotropic axis signalling genes, increased expression of xenobiotic metabolism genes). In adult mice expression of somatotropic axis genes have returned to control levels.

This demonstrates that, in contrast to dwarf mice, attenuation of the GH-IGF signalling axis in offspring nursed by α-casein deficient dams is transient, while the changes in body size and lifespan are permanent. Offspring nursed by α-casein deficient dams show permanent changes in body composition. Absolute and relative adipose tissue weights ($p<0.05$), the percentage of body fat ($p<0.001$) as well as adipocyte size in epididymal white adipose tissue are all reduced. Serum leptin levels are 25% of those found in control mice ($p<0.001$). Liver lipid content and liver lipid composition are both significantly altered in response to postnatal nutrition. This demonstrates that nutrition in early life programs adult lipid metabolism, body composition and lifespan.

Poster 041:
Nutritional Potential and Metabolomic Response Following Algae Consumption in Humans
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Algae are photosynthetic marine organism that exhibit rich nutritional profiles. Most recently, algae have emerged as promising novel protein source and functional food in Western diets due to their dense nutrient composition and sustainable production potential. Algae are rich in essential nutrients, including vitamins, minerals, essential fatty acids, antioxidants, and dietary fiber. These compounds contribute to various health benefits, such as enhancing immune function and supporting cardiovascular health. Among these nutrients, proteins and amino acids are particularly important as fundamental macronutrients. However, there is still limited evidence regarding the bioavailability of algal protein during the immediate postprandial phase, especially in comparison to conventional protein sources.

This study aims to address this gap by investigating the postprandial bioavailability of protein from the microalgae Chlorella sorokiniana in healthy participants. We will conduct a comprehensive metabolomic analyses to evaluate the metabolic effects of algae consumption. Twelve healthy adults (6 m/6 w) aged 18–40 years participated in a monocentric, randomised, placebo-controlled, crossover study. The participants consumed bread rolls with 12g microalgae as a defined test meal. Bread rolls without microalgae powder served as the control, and the order of the test meal and control consumption was randomised.

Anthropometric baseline characteristics were measured, with participants having an average age of 25.40 ± 5.21 years and an average BMI of 22.34 ± 3.22. Blood, urine and stool samples were collected at specific time points before and after meal consumption. These biospecimens will be analysed using a range of analytical techniques, such as LC-MS, GC-MS, and NMR spectroscopy, to capture a broad spectrum of metabolites. Our findings from this study may provide valuable insights into the metabolic impact of microalgae.

Poster 042:
Effects of protein replacement by alphitobius diaperinus in the enteroendocrine system in rats

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Insects have become a high-value protein source meeting essential amino acid requirements and providing unsaturated fatty acids and micronutrients. For this reason, their nutritional composition is comparable to that of protein-rich foods of animal and plant origin, such as beef, eggs, and soy. Moreover, they represent a sustainable alternative to traditional livestock farming, as their production requires less land and water, emits fewer greenhouse gases, and demonstrates high feed conversion efficiency, making them more cost-effective. Therefore, insects present a promising solution to address food supply challenges arising from population growth while providing essential nutrients.

In our study, we aim to study if the chronic consumption of such alternative protein has a metabolic impact on intestinal health analysing whether the previously observed modulation of the enteroendocrine system, induced by prior administration of Alphitobius diaperinus, also occurred when the insect was used as the sole protein source. Additionally, we sought to evaluate these effects in the context of both standard and obesogenic diets. To this end, we used female Wistar rats fed three different protein sources: casein, beef, or insect.

After assessing the enteroendocrine response to the consumption of Alphitobius diaperinus as the sole protein source, our results show that a diet where total protein is replaced by insect does not lead food intake modifications in an obesogenic induced metabolism; but seems to have different effect compared to casein or beef in a healthy one. This increase in enterohormones secretion in relation to the decrease in ingestion allows us to position insects as a good source of alternative protein with potential bioactivity that should be further explored.

Poster 043:
Gene expression in Peripheral Blood Mononuclear Cells reflects pathologies –the example of oxidative stress related Nrf2, NF-κB and HO-1 in metabolic syndrome

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Peripheral Blood Mononuclear Cells (PBMC) are suggested to respond to various endogenous and exogenous stimuli. Expression of selected genes in PBMC may reflect variety of pathologies and, such as metabolic syndrome (MetS), cardiovascular diseases, cancer etc.

The aim of the present study was to assess expression levels of oxidative stress related genes - Nrf2, NF-κB, HO-1 in PBMC in connection with plasma levels of asymmetric dimethylarginine (ADMA) and malondialdehyde (MDA) in people with MetS. Patients were as follows: MetS (n=30) and control (n=14). Gene expression analyses were performed by $2-\Delta\Delta Ct$ method. Plasma ADMA was measured with ELISA and MDA - by thiobarbituric acid method.

Results showed that mRNA of NF-kB, Nrf2 and HO-1 levels in PBMC in MetS group were by 53%, 130%, and 185% ($p<0.05$), respectively, significantly higher than in the controls. Elevated levels of MDA (by 78%, $p<0.001$) and ADMA (by 18.7%, $p<0.001$) were found in the MetS group. Expression levels of Nrf2, HO-1 and NF-kB was more than 320%, 214% and 192%, respectively, higher in MetS with ADMA>Median. Nrf2 and HO-1 were with lower expression (by 61% and 56%, respectively) in MetS MDA>Median subgroup. Our data indicate that changes in specific gene expression in PBMCs may reflect MetS pathology. Expression of NF-kB, Nrf2 and HO-1 in MetS is dependent of plasma oxidative stress parameters MDA and ADMA. Knowledge of complex cellular–molecular mechanisms would allow the use of biomarkers such as Nrf2, NF-kB, HO-1, and ADMA in clinical practice

Poster 044:
Metabolic biomarkers in colorectal cancer

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CRC development is considered to be due to genetic, lifestyle and environmental factors. Reliable biomarkers are essential at different stages of the disease management - t. The aim of this study was to check for metabolic dysregulation in CRC by measuring biochemical biomarkers in patients with CRC in comparison with adenomatous polyps (AP) and healthy Controls (C). Lactate, uric acid (UA), glucose, insulin, HOMA index, total cholesterol (TC), LDL-cholesterol (LDL), VLDL-cholesterol (VLDL), HDL-cholesterol (HDL), triglycerides (TAG), total protein (TP), Na+, Cl, osmolality and C-reactive protein (CRP) were measured in fasting plasma using validated methods.

CRP levels appeared to be significantly higher in CRC patients as compared to C (20.30 mg/L vs 1.95 mg/L, $p<0.001$) and AP group (5.75 mg/L, $p<0.01$). Although CRP levels were higher in AP than in C, the difference was with no significance. HDL concentrations were significantly lower in both CRC (1.13 mmol/L, $p<0.001$) and AP (1.23 mmol/L, $p<0.05$) in comparison with C group (1.73 mmol/L). Similarly, TC was also lower, but in CRC group only (4.96 mmol/L, $p<0.05$ vs 5.47 mmol/L in C). TAG (1.67 mmolo/L, $p<0.01$ vs 1.15 mmol/L in C) and VLDL (0.76 mmol/L, $p<0.01$ vs 0.52 mmol/L in C) concentrations were higher in AP group.

Plasma lactate was higher in AP group (3.46 mmol/L, $p<0.05$ vs. CRC) and in CRC it was lower even than the C (2.88, $p<0.01$). Similarly, UA was detected at higher concentrations in AP patients (398.94 mmol/L, $p<0.05$ vs C 295.86 mmol/L and $p<0.05$ vs. CRC 333.67 mmol/L). Na+ and osmolality were lowest in CRC (130.79 mmol/L, and 266.44 mmol/L, respectively) as compared to C (133.23, $p<0.001$ and 271.72, $p<0.001$) and to AP (133.43, $p<0.05$ and 272.34, $p<0.05$).

In conclusion, CRP appears as a strong biomarkers in CRC, making a connection between malignancy and inflammation status. Identification of higher VLDL, TAG, lactate and UA in AP, but not in CRC group may indicate metabolic dysregulation in the onset

Poster 045:
Reproducibility assessment of urinary biomarker panel for mixed strawberries and blueberries intake in a cross-sectional study

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Reproducibility assessment is a fundamental step in the validation of food intake biomarkers. However, most studies to date have not systematically evaluated the reproducibility of these biomarkers, limiting confidence in their broader application for dietary assessment and nutritional research. The objective of this study is to assess the reproducibility of the biomarkers of mixed berries A 21-urinary-biomarker panel for mixed berries intake (strawberries and blueberries) was identified in a randomized cross-over acute intervention study coupled with a dose-response study with an untargeted metabolomics approach.

Urine samples from A-Diet Confirm (ADC) study was to investigate the reproducibility of the biomarkers over 4 months. A method was optimized and validated via the urinary samples of the dose-response study to semi-quantify the 21 biomarkers of mixed berries intake in the ADC cohort. The results showed comparable intraclass correlation coefficients (ICC) observed for the biomarker panel (median ICC = 0.311) and self-reported mixed berries intake (median ICC = 0.345) and supports the potential of this panel as a reliable indicator of intake.

There were 7 biomarkers on the mixed berries biomarker panel demonstrating significant associations with reported mixed berries consumption (p<0.05) with correlations from 0.16 to 0.32 and the ICCs for reproducibility of these 7 urinary biomarkers ranged from 0.19 to 0.55 across the 2 visits. Of the 7 biomarkers, 4 are sulphated compounds 2 are glucuronidated compounds and one a non-conjugated aglycone. Overall, this study demonstrated the robustness of the biomarkers of mixed berries.

Poster 046:
Exploring Genetic Links to Fungiform Papillae, Bitter Taste Sensitivity and Liking, and the Effects of Food Modifications on Bitter Taste Liking

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Declining bitter taste perception might be caused by a decreasing number of fungiform papillae (FP) with increasing age. Number of FP has been linked to genetics. Also, a decreased bitter taste perception could be associated with increased liking. Furthermore, increasing the sweetness of bitter foods might increase liking, which could aid consumption of healthy bitter foods. Therefore, this study aims to explore whether bitter perception declines with age, and if this is linked to FP and genetics. Also, to explore whether decreasing bitter perception is associated with increased liking, and if liking of bitter food samples can be increased by increasing sweetness.

To date, 20/159 participants (F=45%, 26.3±7.22 years) have been recruited. Participant's genetics will be analysed by a custom microarray of taste genes. FP is counted by an automated software. Bitter intensity and liking are determined by detection and recognition threshold (DT / RT), and with general Labelled Magnitude and Labelled Affective Magnitude scales. The same measures will be used to explore bitter intensity and liking of food samples which are matched for bitterness but contain varying amounts of sucrose. DT / RT were not associated with age ($p=0.45$ / $p=0.24$). Reduced FP was correlated with increasing age ($p=0.04$, rho=-0.46). Increased FP was associated with decreased bitter DT and RT ($p=0.01$ / $p=0.002$). There were no differences in liking of bitter foods between bitter hyper / hypo sensitive individuals ($p=0.7$). Perceived bitterness decreased with addition of sucrose ($p<0.0001$, mean=-13.4±5.36) and sweeter samples were liked more ($p<0.0001$, mean=16.1±4.99).

Reduced FP may be linked to age and this may impact bitter taste. However, DT / RT sensitivity might not be associated with bitter food liking. Also, perceived bitterness of foods can be reduced by sucrose, and this increases liking. Further recruitment and genetic analyses are needed before final conclusions can be drawn.

Poster 047:
Exploring Genetic Links to Salt Taste Sensitivity and Liking to the Effects of Bouillon-Based Foods Modifications

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Lake Lucerne Institute

Overconsumption of salt can lead to hypertension, which is a leading risk factor for mortality. Lower salt taste sensitivity (STS), which has genetic associations, has been linked to increased salt addition to foods. Reducing salt intake remains challenging, however both chilli and citric acid have been reported to enhance salt perception. The aim of this study is to explore if STS (hypo/hypersensitivity) impacts perceived intensity and liking of salt reduction protocols, and whether genetic variation influences this.

To date, 20/159 participants (45%F, 26±7yrs) are recruited. Saliva is collected for genetic analysis by microarray panel. STS is assessed by ascending staircase to determine detection/recognition threshold (DT/RT). Intensity (general labelled magnitude scale (gLMS)) and liking (labelled affective magnitude scale (LAM)) of bouillon-based foods with varying salt (grams) were tested with increasing citric acid (CA) and chilli concentrations. Preliminary analysis indicates that taster status influences liking and intensity of reduced salt foods. In the chilli series hypersensitivity (≤0.016M DT) led to a decreased liking from 0.5g/100ml (64.6±11.6) onwards ($p<0.01$), and a decreased intensity from 0.6g/100ml (16.8±10.4) onwards ($p<0.01$). In the CA series hypersensitivity led to a decreased liking from 0.4g/100ml (50.8±10.6) onwards ($p<0.01$). No differences were found in the hyposensitive. No correlations were found between RT and liking nor intensity ($p<0.05$).

Findings suggest salt reduction using chilli and CA may be effective in the hyposensitive only, highlighting the importance of further exploring taster status in nutrition practice. Upon study completion, the impact of genetic variation on salt taste receptors will be explored, with findings aimed to influence personalised nutrition practice.

Poster 048:
Insights Relating to Heterogenous Innate Immune Training in Monocytes from Older Individuals

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Introduction: Monocytes and macrophages are key players in innate immune training (IIT), a form of immunological memory of innate immune cells. Dietary ingredients, including yeast β-glucan, can induce IIT, improving immune function. Nevertheless, IIT capacity has not been determined within the context of nutritional status in older cohorts that may benefit from immune training. Understanding this could lead to therapies enhancing IIT. Aim: Examine IIT capability in monocytes from older individuals and relate this capacity to nutritional status, body composition and health status.

Methods: Monocytes from 60 individuals over 65 were isolated and macrophage selected. IIT was tested by exposing cells to yeast β-glucan (stimulus 1) one-day post-isolation and LPS (stimulus 2) after a 4-day rest to return cells to a non-activated state. TNF-α, IL-6, and IL-10 were measured post-LPS stimulation, a ≥10% increase in cytokine response to LPS in cells pre-treated with β-glucan was classified as IIT. Ancillary dietary, body composition and health parameters were assessed.

Results: Heterogeneous training responses were observed, with 47% and 57% of the cohort displaying TNF-α and IL-6 training responses, respectively. Training responses varied among individuals, and a significant inverse relationship with BMI & training was observed. Monocytes with higher baseline cytokine responses to LPS alone demonstrated lower training responses.

Conclusion: This study is the first to demonstrate that IIT of monocytes by β-glucan can be induced in a large proportion of subjects in an elderly cohort. Increasing BMI was associated with a lower training response. On-going analysis will focus on the impact of other dietary, metabolic, immune and anthropometric factors within this cohort on training.

Poster 049:
Placental and Cord Blood DNA Methylation in Preterm Birth: Exploring the Epigenetic Role of Maternal Dietary Protein

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Recently, diet has been highlighted as a risk factor for preterm birth (PTB), a leading cause of neonatal mortality. However, the epigenetic mechanisms explaining how maternal undernutrition could be associated with PTB are still to be fully understood.
This study aims to investigate placental and cord blood DNA methylation (DNAm) alterations in a PTB compared to full-term pregnancies by investigating the link between maternal undernutrition and epigenetic changes.
Clinical data and pregnancy outcomes were collected for 18 PTB vs. 30 matched full-term subjects belonging to the Karen and Burmese populations living in Myanmar and Thailand. Nutrient intakes were computed based on 24-hour recalls and analyzed by the Nutritionist Pro™ software. DNAm was measured using the Infinium MethylationEPIC array and RnBeads analysis in 39 placental and 34 fetal cord blood samples.
The analysis revealed severe undernutrition with deficiencies of several nutrients, including proteins, in PTB compared to controls ($P<0.05$). LIPF promoter hypomethylation and SSB promoter hypermethylation were associated with PTB in the placenta and cord blood, respectively (FDR-adjusted p-value <0.05). Low protein dietary intake was associated with SSB promoter hypermethylation in cord blood samples in PTB as well as the differential methylation of IGKV1D-39 promoter in both placenta and cord blood. Gene ontology (GO) analysis revealed PTB enrichment in inflammation, developmental pathways, and metabolic functions. Remarkably, cord blood samples showed significant enrichment in the "embryo development ending in birth" process, highlighting intriguing mechanistic insights potentially unique to PTB.
Our findings uncover a novel link between maternal protein intake and DNAm alterations associated with PTB. Low protein intake was specifically linked to hypermethylation of the SSB promoter, highlighting a potential epigenetic biomarker and therapeutic target.

Poster 050:
Interaction of melatonin receptor 1B (MTNR1B) genotype and type of breakfast (protein-enriched v carbohydrate-rich) on postprandial glucose response.

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The risk allele (G) of MTNR1B rs10830963 has been associated with impaired glucose tolerance, increased fasting glucose and type 2 diabetes (T2D). Late eating, when endogenous melatonin levels are elevated, is associated with impaired glucose control in MTNR1B risk carriers. Endogenous melatonin levels remain elevated into the morning so may influence glucose response to breakfast. Aim: To investigate the interaction of MTNR1B genotype and type of breakfast on postprandial glucose response.

Methods: Healthy adults aged 18-48 years, were recruited to take part in a randomised crossover trial. Following an overnight fast, participants were assigned to consume either a carbohydrate-rich (CHO) or protein-enriched (PRO) porridge breakfast. Post-prandial glucose levels were recorded for two-hours using a continuous glucose monitor. One-week later participants repeated the protocol consuming the alternate breakfast. A two-way mixed ANOVA was used to determine the effect of breakfast and genotype on post-prandial glucose levels.

Results: Fifty-four adults (CC=19, CG=20, GG=15) completed the study (mean age 33 Â± 9 years, BMI 23 Â± 3 kg/m2). Fasting glucose was significantly higher (p=0.008) in GG (5.53 Â± 0.43 mmol/L) compared to CC or CG participants (5.02 Â± 0.42 and 5.19 Â± 0.52 mmol/L). Post prandial iAUC was significantly greater following the CHO breakfast compared to the PRO breakfast ($p<0.001$). Following the CHO breakfast iAUC was significantly greater in GG participants compared to CC (p=0.039) and CG participants (p=0.043). There was no significant difference between genotype groups in iAUC following the PRO breakfast ($p>0.05$).

Conclusion: The study findings demonstrate, in a relatively young and healthy population, MTNR1B genotype significantly affects markers associated with T2D risk. Personalised genotype-based advice to adjust timing and composition of meals eaten when endogenous melatonin levels are increased may reduce subsequent T2D risk.

PRECISION NUTRITION: The NUTRIOME Project

Poster 051

Association of Handgrip Strength with the Metabolomic Profile: Secondary Analysis of a Protein Intervention study

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Abstract: Handgrip strength is a critical indicator of functional capacity and health in older adults, and increased protein intake has been suggested to improve muscle strength. This study aims to investigate the relationship between the metabolomic profile and handgrip strength. This secondary analysis of a 12-week randomised controlled trial included 171 healthy men and women aged 50 ≥ years. Participants were randomised into three parallel groups: high plant protein (23 g/d), high dairy protein (22 g/d) and low protein (2 g/d).

The primary outcome was handgrip strength and there was no significant impact of intervention group on handgrip strength. Metabolomic analyses were performed on plasma samples using a targeted LC-MS platform. Spearman correlations analysis was used to measure the relationships between metabolites levels and handgrip strength. Baseline handgrip strength positively correlated with higher levels of certain acylcarnitines, amino acids, and lysoPCs, while weaker handgrip strength was associated with elevated levels of specific phosphatidylcholines and sphingomyelins.

Furthermore, improvements in handgrip strength were positively associated with LysoPCa20:3, LysoPCa20:4, isoleucine, and leucine. After adjusting for sex and BMI, LysoPCa20:3, LysoPCa20:4, and leucine remained significantly associated with handgrip strength improvements. Leucine levels across tertiles of positive change in handgrip strength indicated that individuals with the highest plasma leucine levels exhibited the greatest improvements in handgrip strength. Metabolite levels were strongly correlated with overall handgrip strength outcomes, suggesting the necessity to comprehensively evaluate the metabolic state of individuals prior to interventions.

Poster 052

The NUTRIOME meal study - A data-driven precision nutrition intervention study using meal responses

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Background and Aim: NUTRIOME is a Marie Curie Training Network across 9 European universities aiming to train 10 researchers in developing data-driven precision nutrition strategies to mitigate cardiometabolic risks. An integral part of this project is the 2-phase, multi-center trial that is being conducted in Sweden, Norway, and the Netherlands, which explores postprandial responses of individuals to meals varying in macronutrient composition and whether tailoring diets to these responses improves metabolic and immune health outcomes.

Design: The NUTRIOME study aims to enroll 120 adults (BMI 27-35 kg/m^2, age 40-70). During characterization (Phase 1), participants consume three isocaloric meals – animal fat-rich, marine fat-rich, and carbohydrate-rich (plant-based) (~670-683) – in a randomized cross-over design. Postprandial glucose, triglyceride (TG), and IL-6 responses are measured, along with microbiota composition, immune markers, and OMICs profiles. Using these data, an algorithm will be developed to assign personalized diets. Participants are then randomized to follow either this personalized advice or general, healthy dietary guidelines during a six-week at-home intervention (Phase 2).

Outcomes: Effects of personalized diet vs. control after six weeks will be evaluated by comparing changes in postprandial glucose, TG, and IL-6 responses between the groups via a standardized high-fat mixed meal test (PhenFlex). Additional determinants of differential metabolic and immunologic response, including lifestyle factors, glycemic variability, gastric emptying, inflammatory markers, gastrointestinal hormones and OMICs-based profiling, will be investigated throughout the study.

Expected impact: NUTRIOME will identify metabolic response types and test whether personalization improves diet efficacy. It will also provide mechanistic understanding of determinants of differential responses to animal-, marine-, or plant-based meals with different macronutrient content.

Poster 053:
Inter-Individual Variation in Circadian Vitamin D Response Reveals Targets for Personalized Supplementation

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Vitamin D3 synthesis in humans follows a circadian rhythm due to its UV-B-dependent production. As part of the VitDHiD intervention study, we identified 87 in vivo vitamin D target genes in immune cells with circadian expression patterns. These genes form a regulatory network centered on transcription factors and membrane receptors, displaying narrow basal expression ranges and showing downregulation in 80% of cases following vitamin D3 supplementation. Clustering analysis revealed six distinct gene groups, with the two most prominent regulated by the transcription factor CSRNP1 and GAS7, a known differentiation factor.

Among the 25 participants, we identified two subgroups with markedly different transcriptional responses in 14 target genes, including CSRNP1, metabolic enzymes (e.g., NAMPT, PFKFB3), and transporters (e.g., SLC2A3). All 14 genes harbor vitamin D receptor (VDR)-binding enhancers in sufficiently close vicinity to their respective transcription start sites. These results uncover a novel connection between vitamin D signaling and circadian gene regulation, with implications for personalized supplementation. However, the small cohort size limits statistical power, suggesting additional inter-individual differences may remain undetected.

To address this, we extended the analysis using data from the ongoing VitDPAS study, which follows up to 47 participants over two years. As the VDR gene itself is seasonally regulated, this expansion enables investigation of how continuous supplementation affects the expression of VDR and other seasonally responsive genes. Preliminary findings from VitDPAS reveal new interactions between vitamin D signaling and seasonal gene regulation, offering broader insights into the long-term effects of supplementation.

Poster 054:
Use of metabotyping to identify individuals with different triglycerides response curves after intake of high fat meals

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Metabotyping is a novel concept in precision nutrition that groups people based on their shared metabolic characteristics. So far, most studies use data from a single meal for metabotyping. In a randomized controlled cross-over study using four high fat meals with similar fat content from different dairy products (Hansson et al., 2019), a large variation in individual triglyceride (TG) response was observed.

The current study aims to identify different groups of TG responders and define their biological profile. 47 healthy adults (14 males) aged 25-46 y with BMI 21.0-25.8 kg/m2 were included for analysis. Metabolic parameters were measured at fasting and at 2, 4, 6h after meal consumption. The incremental AUC was calculated for metabolic parameters and lipoprotein subclasses. A latent class mixed model was applied. This method identifies subgroups in the population by modeling both the trajectory over time and the effect of different meals, while allowing for individual variability.

Four different clusters were identified. Since one cluster included only two subjects, we continued with three clusters (n = 45). Cluster 1 (n = 18) displayed low TG response, cluster 2 (n = 21) had the highest TG peak at 2h and returned to baseline at 6h, and cluster 3 (n = 6) had a continuous high TG level after 2h. Significant differences ($p < 0.05$) between the clusters were found for sex, fat mass, and visceral fat. Glucose-iAUC and GlycA-iAUC showed significant differences across the clusters, with the largest GlycA-iAUC in cluster 3. This work revealed three response groups based on dynamic postprandial TG changes. The latent class mixed model accommodated different types of data and took the meal differences into account. Since the model assigned a probability to each cluster, the repeated measures increased the reliability of clustering results. This approach holds a potential to be used to detect metabolic dysfunction and aid in delivering personalised nutritional interventions.

Poster 055

Can urine metabolites predict gut microbiota diversity, gut physiology and dietary diversity?

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The interplay between diet, gut microbiota and gut function are important for human health, however, these complex interactions are poorly understood. Within the PRIMA study (NCT04804319), we investigated the potential of using urine metabolome to predict gut physiology and microbiota, and dietary diversity. Urine and faecal samples, diet and bowel habit records were collected daily for 9 days from 61 healthy individuals. The metabolome was characterized by LC-MS based untargeted metabolomics and the gut microbiota by 16S rRNA sequencing. The gut physiology was assessed with SmartPills© measuring segmental gut transit time, pH, pressure and contractions. Machine learning was used to predict diet, gut microbiome and physiology from urine metabolite levels. The models displayed good performance at population level, but low accuracy for individual responses. Ongoing work investigates the magnitude and direction of the relationship between urine metabolites and different measurements.

Poster 056:
Functional Insights into Diet and the Microbiome using IPHOMED

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Diet is a pivotal and readily modifiable external factor significantly influencing health outcomes and disease activity. As the development of effective dietary recommendations is challenged by inter-individual variability in responses to food intake, precision nutrition seeks to identify the drivers of such variability. A major focus has been the gut microbiome, often analyzed through metagenomics, as it has been shown to be crucial in the metabolism of food components, with diet being one of its main modulators. However, studying the microbiome only through metagenomics is limiting, as compositional data and its genomic predictions may not reflect actual microbial activity. This can result in missing key contributors, or overestimating minor ones, potentially undermining observed associations.

To address this limitation, metaproteomics has emerged to reveal the true functionality of the microbial species present via its profiled proteins. However, compared to the wealth of dietary research employing metagenomic analysis of the gut microbiome, very few have considered its metaproteome. A recently developed tool in our lab, IPHOMED, addresses this concern by coupling metagenomics with a novel metaproteomics pipeline, enabling for the first time, a species-level quantification of bacterial proteins, while simultaneously allowing for an accurate identification of the dietary exposome.

In a previous study, among patients prescribed an amino acid-based nutritional therapy regimen, IPHOMED successfully identified the non-compliant individual by detecting dietary proteins. Utilizing IPHOMED in precision nutrition studies to accurately quantify dietary intake, which otherwise often relies on error-prone subjective questionnaires, as well as characterize the microbial functional landscape may be crucial for advancing findings on diet-microbiome interactions.

Poster 057:

Multi-omics integration for stratifying sex-specific phenotypes in response to changing from a diet high in saturated fat to a diet high in polyunsaturated fat

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Background: Changing from a diet high in saturated fatty acids (SFA) to a diet high in polyunsaturated fatty acid (PUFA) is associated with reduced risk of cardiovascular disease. Multi-omics integration can increase understanding of molecular health effects of changes in dietary fatty acid composition and give insights into phenotypical metabolic and lipid responses to dietary changes.

Objectives: The aim of this study is to integrate different layers of molecular and clinical data to stratify phenotypes and investigate their metabolic and lipid responses when changing from a diet high in SFA to a diet high in PUFA to increase understanding of effects of fatty acid on cardiometabolic health.

Methods: The similarity network fusion (SNF) algorithm is used to integrate untargeted transcriptomics and targeted gene expression, untargeted and targeted metabolomics, fatty acids and clinical data from 48 females and 37 males completing a double blinded, randomized controlled dietary intervention. Spectral clustering is performed after integration to identify phenotypes at screening, before and after intervention. Normalized mutual information (NMI) is used to determine the importance of features in separating the clusters at different timepoints. After stratifying phenotypes into different clusters, differences in metabolic and lipid response are investigated.

Results: Preliminary results show that both sexes separate into two clusters for screening, baseline and end of intervention. At end of intervention female cluster 1 consists of 70% intervention and cluster 2 consists of 22% intervention. Male cluster 1 at end consists of 48% intervention and cluster 2 consists of 50% intervention. Among features important for separating the two clusters in both sexes are LDL associated, apo-B, total cholesterol and saturated plasma fatty acids. At end of intervention, there is a significant difference in LDL cholesterol between intervention and control in females but not in males.

Poster 058:

Integrative analysis of lipoprotein-lipid interactions in dietary intervention studies using Nightingale metabolomics

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Dietary interventions, particularly those altering fatty acid intake, significantly impact lipid and lipoprotein metabolism. These effects are central in understanding the molecular mechanisms through which diet influences metabolic health. The Nightingale Health platform offers high-throughput ^{1}H-NMR metabolomic profiling, capturing a detailed lipid profile including lipoprotein subclasses (e.g., VLDL, LDL, HDL and their subfractions) and their associated lipid contents (e.g., cholesterol, triglycerides, phospholipids). Such profiles are increasingly used in nutritional studies to asses disease risk and monitor metabolic responses.

Despite being widely used, Nightingale data lack an analytical framework to integrate metabolomics, transcriptomics and metabolic networks for interpretation. Currently, no dedicated tools exist to investigate the lipoprotein-lipid interactions in the context of molecular mechanisms and dietary interventions. In this project, we aim to develop an integrative analysis and visualisation pipeline for Nightingale metabolic profiles. By incorporating transcriptomic data and connecting metabolite changes to pathway-based network models, we strive to create a comprehensive resource for exploring lipoprotein metabolism and its modulation by diet. This approach enables systems-level insights into nutrient-metabolism interactions, supporting more effective interpretation of dietary intervention outcomes.

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