NuGOweek 2026

BOOK OF ABSTRACTS

Healthy Aging through Diet

Olsztyn, Poland | 31 August – 2 September 2026

In Life

Institute of Animal Reproduction and Food Research Polish Academy of Sciences

NUTRIGENOMICS ERA CHAIR | WELCOME2

About NuGOweek 2026

NuGOweek 2026 brings together researchers in molecular nutrition, nutrigenomics and nutritional systems biology to discuss how nutrition can support healthy aging. The 2026 meeting in Olsztyn is organized around the theme “Healthy Aging through Diet” and combines keynote lectures, selected oral communications, poster sessions and networking.

NuGO is an association of universities and research institutes working to advance molecular and personalised nutrition, nutrigenomics, nutrigenetics and nutritional systems biology, while fostering collaboration, shared infrastructure and the exchange of data, methods and knowledge.

Structure of this abstract book

10	22	40
KEYNOTE LECTURES	SELECTED ORAL PRESENTATIONS	POSTER PRESENTATIONS

Scientific Advisory Board

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Venue

Anna Wasilewska Auditorium and Conference Hall, Ernesta Kościńskiego 11, Olsztyn, Poland

Conference website: *nugo2026.pan.olsztyn.pl*

Program at a glance

	Monday, 31.8.2026	Tuesday, 01.09.2026	Wednesday, 02.09.2026
Morning		**Session 2:** Nutrigenomics of Health and Disease	**Session 4:** Aging Clocks in Metabolic Disorders
Noon	Registration	Lunch and Poster session 1	Lunch and Poster session 2
Afternoon	**Session 1:** Emerging Science in Life Course Nutrition	**Session 3:** Microbiome Dynamics in Healthy Aging	**Session 5:** Precision Nutrition and Digital Twins in Health and Disease
Evening	City tour, then **Welcome reception** at Warmia Brewery	**ECN events, social activities** (castle visit, Ukiel lake, kayaking, music...)	**Cocktail at the beach Gala dinner** including Poster and Short talk prizes, Announcement of NuGOweek 2027

Monday, 31 August 2026

Time	Program
13.00–15.00	Registration (Aula)
15.00–19.00	**Session 1: Emerging Science in Life Course Nutrition — Chairs: Karl-Heinz Herzig / Baukje De Roos**
15.00–15.10	Welcome
15.10–15.50	**KEYNOTE: Ferdinand von Meyenn — Epigenetics: long-term cellular memory shaping health**
15.50–16.30	**KEYNOTE: Matteo Cesari — From geroscience to function: how nutrition shapes intrinsic capacity and frailty across the life course (online)**
16.30–16.50	Coffee break
16.50–17.05	Tatiana Marques — New NuGO member presentation
17.05–17.20	Claudia Favari — New NuGO member presentation
17.20–17.40	Eunike Velleuer — Fanconi Anemia: A Nutrigenomic Stress Test for Human Aging
17.40–18.00	Suzan Wopereis — Multi-study analysis reveals dynamic postprandial biomarkers for metabolic syndrome by low-level fusion of multi-platform data from nutritional challenge test studies
18.00–18.20	Thomas Agius — Benchmarking EU and US plant-based meat analogs against the nutrient needs of an aging population
18.20–18.40	Keeva Loughlin — Relations between diet and biomarkers of aging in middle-aged to older adults: an explorative cross-sectional analysis among three European cohorts
18.40–19.00	Heli Viljakainen — Average-increasing BMI trajectory in adolescence associates with multimorbidity
19.00–20.00	City Tour (ending at Warmia Brewery)
20.00–22.00	Welcome Reception at Warmia Brewery

Tuesday, 1 September 2026

Time	Program
09.00–12.00	**Session 2: Nutrigenomics of Health and Disease — Chairs: Annalisa Terranegra / Lars-Oliver Klotz**
09.00–09.40	**KEYNOTE: Jan Hoeijmakers — DNA damage at the basis of cancer and aging: the surprising impact of nutrition and gene length**
09.40–10.20	**KEYNOTE: Elina Sillanpää — Gene-lifestyle interactions in healthy aging**
10.20–10.40	Coffee break
10.40–11.00	Priman Fau — Temporal Hierarchy of Vitamin D Signaling: VDR-Centered Chromatin Dynamics Drive Immune Nutrigenomic Responses
11.00–11.20	Mirko Treccani — Genetic risk signatures in phenolic metabotypes for cardiometabolic health and disease: unravelling genomic complexity from GWAS and PGS
11.20–11.40	Lucia Ghiretti — Multi-Omics Integration to Characterize Inter-individual Variability in Dietary (Poly)phenol Metabolism
11.40–12.00	Chiara Rucci — Molecular signatures of cellular senescence and their modulation by an in vitro-derived apple callus extract
12.00–14.00	Lunch and Poster session 1
14.00–17.30	**Session 3: Microbiome Dynamics in Healthy Aging — Chairs: Agata Chmurzyńska / Lorraine Brennan**
14.00–14.40	**KEYNOTE: Patrick Veiga — The French Gut Project: Exploring Gut Microbiome, Diet, and Health Across the Lifespan**
14.40–15.20	**KEYNOTE: Katarzyna Winek — The gut-brain axis through the lens of ischemic stroke: microbiome signatures and host responses**
15.20–15.40	Coffee break
15.40–16.00	Federico Ansaloni — Hard-wired or acquired? Epigenetic and transcriptional profiling of Lgr5^{+} intestinal stem cells along the mammalian intestine
16.00–16.20	Pedro Cardenas Canto — Responders vs Non-Responders: Uncovering Individual Glucose Response Patterns to Dietary Bioactive Compounds
16.20–16.40	Axel Baumann — Bitter Taste Receptor (Tas2r) Agonists Modulate Gut Hormones and Metabolic Markers in Aged Rats
16.40–17.00	Marc Beltrán-Fernández — Bitter taste receptor agonists modulate intestinal dysfunction in aged rats under dietary stress
17.00–17.20	Jens Verbeeren — Habitual coffee consumption and longitudinal NMR metabolite trajectories in an aging Finnish cohort
17.20–17.30	Laura Bordoni — ECN presentation
From 17.30	ECN events

Wednesday, 2 September 2026

Time	Program
09.00–12.00	**Session 4: Aging Clocks in Metabolic Disorders — Chairs: Rosita Gabbianelli / Hristo Ivanov**
09.00–09.40	**KEYNOTE: Heike Bischoff-Ferrari — Learnings from DO-HEALTH and VITAL: vitamin D and omega-3 benefits on the 3 metrics of aging (online)**
09.40–10.20	**KEYNOTE: Ewelina Pośpiech — Rethinking aging: epigenetic clocks for quantifying biological age and lifestyle & nutrition effects**
10.20–10.40	Coffee break
10.40–11.00	Emilia Gospodarska — Decoding Individual Responses to Calorie Restriction: PBMC Multi-Omics Reveals Immune-Metabolic Reprogramming for Precision Nutrition
11.00–11.20	Chanachai Sae-Lee — Differential Effects of Vitamin Supplementation on DNA Methylation-Based Biological Age and Functional Outcomes in Aging Adults
11.20–11.40	Anna Kipp — Trace element supply and status in old age with a focus on zinc, selenium and copper
11.40–12.00	Alice Ricco — Association of plant-based protein intake with epigenetic age deceleration in the Netherlands Twins Register
12.00–13.45	Lunch and Poster session 2
13.45–14.00	Opening of Welcome 2 conference — Mariusz Piskula
14.00–17.00	**Session 5: Precision Nutrition and Digital Twins in Health and Disease — Chairs: Yiannis Mavrommatis / Stine Ulven**
14.00–14.40	**KEYNOTE: Elisa Dominguez-Hüttinger — The aging epidermis as a dynamic system: insights from mathematical modelling**
14.40–15.20	**KEYNOTE: Susan Coort — Connecting the dots: pathways and networks in nutrition research**
15.20–15.40	Coffee break
15.40–16.00	Tanya Tripathi — Multi-Omics Dissection of Vitamin D_3 Responses Reveals Context-Dependent Immune Regulation in Multiple Sclerosis
16.00–16.20	Catherine Graham — The Brain Derived Neurotrophic Factor Gene and Dietary Decisions
16.20–16.40	Kerstin Schorr — Adjustments in meal timing and macronutrient composition improve postprandial metabolic and inflammatory markers in night-shift workers
16.40–17.00	Ammi King — Investigation of the impact of melatonin receptor 1B (MTNR1B) genotype on continuous glucose monitor derived metrics
17.45–18.45	Cocktail at the beach of Ukiel Lake, then walk to Przystań Hotel
19.00–22.00	Gala Dinner at Przystań Hotel; poster and short-talk prizes; NuGOweek 2027 announcement; closing of NuGOweek 2026

K

Keynote Lectures

Invited lectures by leading experts

10 abstracts | K01–K10

KEYNOTE LECTURE K01

Epigenetics: long-term cellular memory shaping health

Ferdinand von Meyenn
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Monday 31 August, 15.10–15.50 | Session 1

ABSTRACT

Epigenetic information integrates genetic background with lifetime exposures, creating durable molecular states that influence physiology and disease risk – the long-term "cellular memory." DNA methylation-based epigenetic clocks are quantitative biomarkers of biological age and age acceleration. They capture interindividual differences in ageing trajectories that relate to metabolic health and lifestyle, including nutrition. We have developed new models and methods to assess epigenetic age across tissues and even at single-cell resolution. In the second part, I will present our work on obesogenic epigenetic memory in adipose tissue. Using single-nucleus transcriptomics and adipocyte epigenome profiling, we show that human and mouse visceral fat retains persistent obesity-associated transcriptional programs and stable chromatin alterations after substantial weight loss. These long-lived epigenetic changes impair adipocyte function, rewire nutrient-responsive gene regulation, and predispose to accelerated rebound weight gain upon renewed high-fat feeding. Together, these findings highlight epigenetic mechanisms that link past exposures — diet, obesity, and ageing — to future biological responses, and suggest opportunities to improve long-term health and metabolic outcomes by targeting persistent epigenetic states.

KEYNOTE LECTURE K02

From geroscience to function: how nutrition shapes intrinsic capacity and frailty across the life course

Matteo Cesari
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Monday 31 August, 15.50–16.30 | Session 1 | Online

ABSTRACT

Healthy ageing is increasingly understood as the result of dynamic interactions between biological, clinical, and environmental determinants across the life course. Among these, nutrition represents a foundational (and still under-leveraged) driver of trajectories in intrinsic capacity and functional ability. Advances in nutrition science are rapidly reshaping our understanding of ageing biology, highlighting the role of dietary patterns, macro- and micronutrient adequacy, and metabolic regulation in modulating key pathways related to frailty, sarcopenia, inflammation, and resilience. From a clinical perspective, the integration of nutrition into models of care for older adults remains fragmented, often limited to late-stage or deficit-based approaches. However, emerging evidence supports a shift towards earlier, function-oriented strategies, where nutrition is recognised not only as supportive care but as a fundamental intervention shaping trajectories of intrinsic capacity. This aligns with evolving paradigms in geroscience and life-course approaches, which emphasise the plasticity of ageing processes and the potential to delay or mitigate functional decline through targeted, multi-domain interventions. At the same time, significant gaps remain in translating nutrition science into actionable policies and care models. These include limited integration of nutrition into clinical guidelines for geriatric syndromes, insufficient alignment between research priorities and implementation needs, and ongoing challenges in operationalising personalised, context-sensitive nutritional strategies on a large scale. This keynote will explore how nutrition science can be more effectively integrated within clinical and public health frameworks for healthy ageing. It will connect biological insights with clinical practice and policy development, drawing on concepts such as intrinsic capacity, frailty, and resilience. Special focus will be given to the role of nutrition within integrated care pathways, the need for a more focused research agenda, and opportunities to better link geroscience advances with practical interventions. Ultimately, strengthening the link between nutrition science and clinical geriatrics will be essential to developing more coherent, prevention-focused, and person-centred approaches to ageing.

KEYNOTE LECTURE K03

DNA damage, cancer and aging: the surprising impact of nutrition and gene length

Jan H.J. Hoeijmakers[1,2]

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Tuesday 1 September, 09.00–09.40 | Session 2

ABSTRACT

Aging is associated with systemic functional decline and increased cancer incidence, but appears remarkably plastic: e.g., suppressing insulin/IGF somatotrophic signalling extends lifespan in numerous species and reduces cancer. On the other hand, we found defects in specific DNA repair pathways to cause accelerated aging. For instance, Ercc1Δ/- mice, carrying repair defects in multiple repair systems, including transcription-coupled and global genome nucleotide excision repair, show wide-spread progressive multi-morbidity, limiting lifespan to 4-6 month. To counteract the premature aging these mutants also mount an anti-aging 'survival response', which suppresses growth by attenuating the IGF1 somatotrophic axis and enhances maintenance and resilience mechanisms, resembling the longevity response induced by dietary restriction (DR). Interestingly, subjecting these progeroid mutants to actual (30%) DR tripled(!) lifespan, and drastically retarded accelerated aging, most profoundly neurodegeneration. We also found that DR lowered DNA damage, explaining why DR delays aging as well as cancer and why repair mutants overrespond to DR. Importantly, Ercc1Δ/- liver expression profiles showed progressive decline of transcription upon aging, preferentially affecting long genes, due to genome-wide accumulation of random, transcription-blocking lesions, which affect long genes more than small ones. Subsequently, we discovered that transcription stress also occurs in normal aging of (post-mitotic) tissues, in organisms ranging from worms to humans. This explains most -if not all- hallmarks of aging, including nutrient sensing (because of transcription stress in the long IGF1 gene) and increased protein aggregation in proteinopathies. DR alleviates transcription stress by lowering DNA damage and thereby prolongs genome function. I will present phenotypes of conditional DNA repair models targeting aging to selected organs, and translation of these concepts to rare progeroid children, which even surpasses the dramatic benefits of DR in mice. Our findings identify DNA damage as main cause of systemic aging and reveal untapped potential of nutritional interventions for reducing transcription stress in neurodegeneration (including Alzheimer's disease) and other aging-associated pathologies. Additionally, the 'survival response' triggered by short-term fasting strongly reduces ischemia reperfusion injury in surgery and organ transplantation and improves chemo- radiotherapy outcome in cancer treatment, thus impacting major areas in medicine.

KEYNOTE LECTURE K04

Gene-lifestyle interactions in healthy aging

Elina Sillanpää
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Tuesday 1 September, 09.40–10.20 | Session 2

ABSTRACT

Biological aging is characterized by progressive functional decline, increasing disease risk, and premature mortality. Individual differences in aging trajectories arise from complex interactions between genetic predisposition and lifestyle and environmental factors. Recent advances in molecular epidemiology enable more precise investigation of these mechanisms through polygenic scores (PGSs) and epigenetic clocks. PGSs summarize genome-wide genetic variation into individual-level estimates of genetic liability for traits such as age-related diseases and health behaviours, whereas epigenetic clocks capture biological aging based on DNA methylation and reflect cumulative exposure to lifestyle and environmental factors. In this presentation, I will present recent findings from longitudinal twin studies and large biobank-based cohorts examining how genetic susceptibility and modifiable behaviours jointly influence biological aging and age-related traits. Genetically informed designs and methodological triangulation help address key biases in observational research and enable the disentangling of causal effects from genetic confounding. Our recent findings suggest that physical activity, diet, and body composition are associated with biological aging, but these associations are partly explained by shared genetic factors and the clustering of health behaviours. Furthermore, emerging evidence highlights the importance of life-course exposures, particularly during adolescence, in shaping later aging trajectories. Integrating genetic, epigenetic, and lifestyle data provides a powerful framework for understanding healthy aging and for developing more targeted prevention strategies.

KEYNOTE LECTURE K05

The French Gut Project: Exploring Gut Microbiome, Diet, and Health Across the Lifespan

Patrick Veiga

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Tuesday 1 September, 14.00–14.40 | Session 3

ABSTRACT

The gut microbiome plays a key role in human health, and emerging evidence suggests that microbial alterations may precede the clinical onset of certain chronic diseases. Therefore, the microbiome has the potential to shape health trajectories throughout life and influence healthy ageing. Shaped by our diet, lifestyle, medications, environment, and previous exposures, it bears the biological imprint of our life history. Like the epigenome, it may represent a link between our past and our future health. The Le French Gut Project is a nationwide citizen-science initiative aiming to characterize the gut microbiome of 100,000 participants across the lifespan. By combining shotgun metagenomics with detailed dietary, lifestyle, environmental, and health data, the project seeks to map microbiome variation, identify its major drivers, and assess whether microbial signatures can help predict future health outcomes. This talk will explore the links between the gut microbiome and healthy ageing and present the Le French Gut Project's design and first findings, illustrating how diet, age, health status, and previous exposures contribute to shaping the gut microbial ecosystem.

KEYNOTE LECTURE K06

The gut-brain axis through the lens of ischemic stroke - microbiome signatures and host responses

Katarzyna Winek

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Tuesday 1 September, 14.40–15.20 | Session 3

ABSTRACT

Stroke, common in the elderly, is one of the most relevant causes of death and long-term disability worldwide with limited therapy options. Ischemic brain injury not only triggers a neuroinflammatory response but also affects other organs throughout the body. The crosstalk between the immune system and the central nervous system is of particular importance after stroke. Peripheral immune cells infiltrate the brain, while at the same time, systemic immune functions are suppressed during the subacute phase of the disease, increasing patients' susceptibility to infections. Our research focuses on identifying the molecular regulators of post-stroke immune responses and on characterizing the role of the "forgotten organ" – the intestinal microbiota in shaping stroke outcomes. The gut microbiota, the largest commensal collection of bacteria in the body, is crucial for many physiological processes as the commensal microbes have coevolved with the host in specific symbiosis through ages. The microbial community of the gut is perturbed after ischemic stroke in experimental models and stroke patients, and this disturbance may have functional consequences for the outcome. The gut commensals actively contribute to the regulation of the balance between anti-inflammatory and protective T regulatory cells (T regs), and pro-inflammatory IL-17 producers, linked to exacerbated neuroinflammatory response and worsening of experimental stroke outcome. Moreover, a few reports linked stroke to changes in the gut motility and permeability. To gain detailed insights into the host and gut microbiome responses after ischemic stroke, in our recent study, we profiled the transcriptome of microglia, brain-infiltrating leukocytes and peripheral blood cells on day 1, 7 and 14 in an experimental mouse stroke model (middle cerebral artery occlusion, MCAo) using single cell RNA sequencing. This profiling was coupled with investigations of intestinal proteome (5 segments from the small and large intestine and the mesenteric lymph nodes), metatranscriptomics and metagenomics of the bacterial gut community. We observed convergence of host and microbiome responses to stroke on day 7 after ischemic brain injury. Our results highlight changes in gut metabolic pathways, especially in jejunum and colon, and alterations in bacterial gene expression, delivering the first metatranscriptomic dataset in experimental stroke studies. Furthermore, specific hypothesis testing identified compartmentalized regulation of immune pathways and proteins related to gut permeability. Overall, these findings reveal critical alterations in host and microbiome responses during the subacute phase of stroke, underscoring the relevance of microbiome dynamics in the later stages following brain injury. This dimension has been largely neglected in the existing literature, yet may hold important translational implications. Elucidating the mechanisms of brain–body communication in stroke is crucial for the discovery of future therapeutic targets and strategies.

KEYNOTE LECTURE K07

Learnings from DO-HEALTH and VITAL: vitamin D and omega-3 benefits on the 3 metrics of aging

Heike A. Bischoff-Ferrari
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Wednesday 2 September, 09.00–09.40 | Session 4 | Online

ABSTRACT

Health span extension aims to slow aging across three metrics: phenotypic aging (age-related diseases and clinical events), functional aging (physical performance, frailty, and independence), and biological aging (molecular markers of the aging process). The DO-HEALTH and VITAL trials provide complementary randomized evidence that vitamin D and omega-3 supplementation may support all three. DO-HEALTH studied 2,157 generally healthy European adults aged 70+ over 3 years, testing vitamin D_3, omega-3s, and a home exercise program. VITAL studied 25,871 U.S. adults age 50+ with vitamin D3 and omega-3s over a median 5.3 years. Across phenotypic aging, omega-3s reduced infections and falls in DO-HEALTH and lowered myocardial infarction in all and several major CV outcomes among those with low nutritional intake of omega-3 in VITAL. Vitamin D improved immune and cancer-related outcomes in VITAL, including reduced autoimmune disease, advanced cancer, and cancer mortality after latency analyses. In DO-HEALTH, the combination of vitamin D, omega-3s, and exercise showed additive benefit, including a marked reduction in incident invasive cancer. For functional aging, single interventions in DO-HEALTH had limited effects in this healthy population, but the combination of vitamin D, omega-3s, and exercise reduced incident pre-frailty, suggesting that multi-component prevention may be needed to preserve resilience with age. For biological aging, DO-HEALTH showed favorable effects on second-generation epigenetic clocks, consistent with partial slowing of molecular aging, while VITAL found that vitamin D helped prevent telomere shortening. Together, these partner trials suggest that vitamin D and omega-3s—especially when combined with exercise—are affordable and scalable strategies with potential to improve health span across clinical, functional, and biological dimensions of aging.

KEYNOTE LECTURE K08

Rethinking aging: using epigenetic clocks to quantify biological age and assess lifestyle & nutritional effects

Ewelina Pośpiech

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Wednesday 2 September, 09.40–10.20 | Session 4

ABSTRACT

The emergence of epigenetic clocks has reshaped aging research by providing robust DNA methylation (DNAm)-based tools for measuring biological age, monitoring age-related changes, and offering new insights into the effects of lifestyle and environmental exposures on aging trajectories and population health. Importantly, the low-to-moderate correlations observed between different clocks suggest that they may capture distinct domains of biological aging. Therefore, exploring and comparing epigenetic clocks may facilitate the selection of appropriate tools for evaluating specific lifestyle and nutritional interventions and ultimately support the development of personalized health promotion strategies. Within the EPIGENOME project, we analyzed a broad panel of DNAm clocks in over 700 individuals from the Polish population. FitAge, GrimAge, and DunedinPACE showed the strongest and most consistent associations with lifestyle-related factors. Accelerated epigenetic aging was linked to smoking, high meat consumption, elevated BMI, stress, and short sleep duration, whereas slower aging was associated with vegetable-rich diets, higher physical activity, and yoga practice. Individuals who followed a vegetarian diet and engaged in daily exercise also exhibited reduced signs of phenotypic ageing, including fewer wrinkles, reported feeling younger, and were perceived as younger based on their appearance. These findings highlight lifestyle and nutrition as major modulators of epigenetic and phenotypic aging. Building on these results, we initiated the OMEGA project, an interventional study aimed at reducing biological aging in individuals with metabolic syndrome by targeting chronic inflammation, one of the key hallmarks of aging, through anti-inflammatory dietary interventions, physical activity, and omega-3 supplementation. The project further compares the responsiveness of different DNAm clocks to lifestyle interventions and investigates SNP × intervention interactions that may influence individual responses to dietary and lifestyle modifications, thereby providing a more personalized perspective on healthy aging. Overall, epigenetic clocks offer valuable opportunities for monitoring biological aging and identifying modifiable lifestyle factors that influence aging trajectories. Integrative approaches combining epigenetic, genomic, and phenotypic data may further improve our understanding of aging biology and support the fine-tuning of individualized strategies aimed at promoting healthy aging.

KEYNOTE LECTURE K09

The ageing epidermis as a dynamic system: insights from mathematical modelling

Elisa Domínguez-Hüttinger

Departamento de Biología Molecular y Biotecnología, Instituto de Investigaciones Biomédicas, Universidad Nacional Autónoma de México, Ciudad Universitaria, Ciudad de México, México

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Wednesday 2 September, 14.00–14.40 | Session 5

ABSTRACT

The epidermis is the outermost layer of the skin, and its correct functioning is key in in protecting us from dehydration and environmental aggressors. Epidermal homeostasis is achieved through a complex network of regulatory interactions between the physicochemical skin barrier function, innate and adaptive immune responses, the resident microbiota and epidermal proteases. Age is a key factor in shaping epidermal function. The epidermis of both the infant and the geriatric population have a decreased skin barrier recovery time, higher pH, increased epidermal proliferation and decreased differentiation of terminal differentiation markers, such as filaggrin. Elderly skin also has higher cortisol levels, which increase epidermal proliferation and further decrease keratinocyte differentiation. These differences in epidermal composition and function make the infants and the elderly more susceptible to environmental aggressors, increasing the risk of developing dermatological conditions, such as atopic dermatitis and psoriasis. It is yet to be clarified how the age-related risk factors contribute to disease onset and progression, and how to implement effective treatment strategies. The aim was to mathematically predict how of age-related changes in epidermal composition shape the vulnerability to develop dermatological conditions, and to design effective strategies for their prevention. Our in-silico approach involves the computational and mathematical analysis of published experimental and clinical data. We use MATLAB software for the numerical analysis of the mathematical model. We formalized the regulatory network controlling epidermal function into a mechanistic mathematical model and simulated the effect of age-related risk factors and preventive strategies on epidermal function. We disentangled how age-related factors shape the response of the epidermis to environmental triggers: A healthy adult epidermis is dynamically simulated as transient deviation from a homeostasis setpoint. While this phenotype is robust to parametric variations, the recovery time is proportional to age-related risk factors. Age-related risk factors that surpass a critical severity threshold lead to intermittent epidermal dysfunction, which can eventually result in persistent "inflammaging". Co-occurring age-related risk factors have synergic effects, dramatically incasing the risk for dermatological conditions. We used the model to systematically simulate the effect of different preventive strategies, such as emollients, pH buffers and topical lipid mixtures, on infant and geriatric epidermis, and identified specific regimes that optimally prevent the onset of inflammaging. Our mathematical model mechanistically links age-related risk factors to the risk of developing dermatological conditions and offers a computational framework to design effective preventive strategies that halt their progression.

KEYNOTE LECTURE K10

Connecting the Dots: Pathways and Networks in Nutrition Research

Susan Coort

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Wednesday 2 September, 14.40–15.20 | Session 5

ABSTRACT

Nutrition research is undergoing a fundamental paradigm shift from general dietary guidelines to highly customized precision nutrition. Traditional "one-size-fits-all" dietary recommendations often fail because they ignore the profound inter-individual variability in metabolic, genetic, and gut microbiome-driven responses to food. Modern multi-omics, wearables, and artificial intelligence (AI) offer unprecedented opportunities to capture this complexity, but translating high-dimensional biological data into clinical utility requires a robust network-based perspective. A 20-year scientific journey will be presented tracing the evolution of nutritional systems biology "from node to network". Beginning with influence of single proteins and how they interact. Thereafter transitioning to pathway-level biology using pathway analysis and WikiPathways, founded in 2008 to collaboratively crowd-source, curate, and annotate metabolic and gene regulatory pathways. Using these mechanistic pathway frameworks, it is demonstrated how transcriptomics and metabolomics can uncover subtle biological processes, highlighted by immunometabolic studies on vitamin D in human monocyte models. However, as precision nutrition increasingly adopts machine learning, a critical bottleneck has emerged: data quality, model reproducibility, and ecosystem fragmentation. AI models are only as robust as the datasets on which they are trained. To move beyond isolated cohorts, this talk underscores the urgent need for FAIR (Findable, Accessible, Interoperable, Reusable) data standards. Through initiatives in the ELIXIR Food & Nutrition Community and the FNS-Cloud project it is illustrate how ontologies and federated cloud infrastructures make food, wearable, and omics data truly interoperable. Ultimately, the future of nutrition lies in "computational nutrition" a hybrid field where data-driven machine learning meets mechanistically-grounded biological networks.

O

Selected Oral Presentations

Abstracts selected for oral communication

22 abstracts | O01–O22

SELECTED ORAL PRESENTATION O01

Fanconi Anemia as a Nutrigenomic Stress Test for Human Aging

Eunike Velleuer[1,2], Carsten Carlberg[3,4]

[1]Department for Cytopathology, Heinrich-Heine-University Düsseldorf, Düsseldorf, Germany; [2]Department for Pediatric Hemato-Oncology, Helios Children's Hospital, Krefeld, Germany; [3]InLife Institute of Animal Reproduction and Food Research, Polish Academy of Sciences, Olsztyn, Poland; [4]School of Medicine, Institute of Biomedicine, University of Eastern Finland, Kuopio, Finland

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Monday 31 August, 17.20–17.40 | Session 1

ABSTRACT

Fanconi anemia (FA) is classically defined as a DNA repair disorder, yet this reductionist view underestimates its broader biological relevance. FA compresses multiple hallmarks of aging, genome instability, epigenetic drift, immune dysfunction, and stem cell exhaustion, into early life, effectively creating a human "time-lapse model" of aging. Recent metabolomic and physiological evidence challenges the notion that FA pathology is driven solely by defective DNA repair. Instead, FA is characterized by profound metabolic reprogramming, including impaired glucose oxidation, increased reliance on ketogenesis, and systemic metabolic inflexibility. These alterations point to a fundamental rewiring of mitochondrial function and nutrient-sensing pathways. In turn, these metabolic shifts are likely to reshape the epigenetic landscape and immune competence, linking cellular metabolism directly to aging-associated phenotypes. Within this framework, FA emerges as an extreme model of gene-environment interaction in aging. Nutrient-responsive pathways and redox-sensitive cofactors, such as vitamin D signaling, NAD^+ metabolism, and α-ketoglutarate-dependent chromatin regulation, may critically modulate epigenetic stability and inflammatory tone. This raises the possibility that dietary inputs and metabolic state are not secondary modifiers, but active determinants of disease trajectory, influencing the progression from genomic instability to immunosenescence and early carcinogenesis. We propose that FA represents a nutrigenomic "stress test" for human aging. Its accelerated disease course provides a unique opportunity to dissect causal interactions between nutrient sensing, metabolism, and genome maintenance in vivo. Leveraging this model may enable the identification of mechanistically grounded biomarkers and dietary strategies aimed at stabilizing the epigenome, enhancing immune surveillance, and delaying cancer onset. Insights gained from FA have the potential to inform precision nutrition approaches for healthy aging, extending beyond rare disease to the general population.

SELECTED ORAL PRESENTATION O02

Multi-study analysis reveals dynamic postprandial biomarkers for metabolic syndrome by low-level fusion of multi-platform data from nutritional challenge test studies

Suzan Wopereis[1], Tim van den Broek[1], Jelle CBC de Jong[1], Milena Rundle[2], Jarlei Fiamoncini[3], Yoana Kiselova-Kaneva[4], Martien PM Caspers[1], Diana G Ivanova[4], Ben van Ommen[1], Jildau Bouwman[1]

[1The] Netherlands Organization for Applied Scientific Research (TNO), The Netherlands; [2]Imperial College London, United Kingdom; [3]University of São Paulo, São Paulo, Brazil; [4]Medical University of Varna, Varna, Bulgary

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Monday 31 August, 17.40–18.00 | Session 1

ABSTRACT

Metabolic syndrome (MetS) is a global health issue linked to increased disease risk and economic burden. Traditional biomarkers for MetS reflect disrupted homeostasis but may fail to detect earlier stages of imbalance, i.e., an increase in allostatic load. Health can be defined as the capacity to maintain or recover homeostatic control, and this capacity can be assessed via a standardized nutritional challenge test (e.g., PhenFlex), which provokes adaptive metabolic responses. We aimed to identify a metabolite signature indicative of MetS-associated allostatic load, using data from multiple nutritional challenge test (NCT) studies. A multi-study-analysis was performed using data from four human studies with nutritional challenge test (n=195). We identified 64 metabolites with significantly altered postprandial responses in individuals with MetS (n=78) compared to healthy controls (n=117). Cluster analysis revealed four clusters with a distinct temporal dynamic response to the NCT. The most prominently affected metabolites included total triacylglycerides, C-peptide, leptin, and a range of amino acids (e.g. L-glutamic acid, L-serine, and L-leucine). The response of a subset of these metabolites to the nutritional challenge test also changed after a 12-week, 20% energy-restriction intervention in an independent trial (n=72), supporting their potential as markers for monitoring intervention efficacy. Further network and ontology-based analyses revealed that the altered metabolites represented key biological processes, particularly lipid and protein metabolism, as well as higher-level physiological function domains, such as liver, muscle, adipose tissue, and pancreas function. These domains were also responsive to the energy-restriction intervention. This study identified a robust, dynamic metabolite signature for MetS that emerges in response to a NCT. This signature reflects increased allostatic load and highlights disrupted adaptability across multiple metabolic functions. A subset of the identified markers were modifiable through dietary intervention, supporting their potential as early, actionable biomarkers of metabolic health.

SELECTED ORAL PRESENTATION O03

Benchmarking EU and US plant-based meat analogs against the nutrient needs of an aging population: a cross-Atlantic compositional analysis

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Monday 31 August, 18.00–18.20 | Session 1

ABSTRACT

Plant-based meat analogs (PBMAs) are marketed as a sustainability lever and as a protein source for the general and aging populations, yet most compositional surveys are single-country and treat PBMAs as a single global food category. Whether commercial PBMAs meet the needs of older adults, who require ≥1.0–1.2 g/kg/d high-quality protein to overcome anabolic resistance, ≥25 g/d fiber to help mitigate inflammaging, and adequate B12, Fe, Zn, and Ca, has not been compared across regions. We mined the USDA FoodData Central branded-foods release (Dec 2025; n≈1,800 products) and the European retailer survey of Siegrist et al. (2024; doi:10.1016/j.foodres.2024.115213; n≈1,600 products across France, Germany, Italy, Netherlands, Spain and the UK; ~950 with full macronutrient labels). Composition was harmonized per 100 g and benchmarked against ESPEN/PROT-AGE protein, EFSA fiber AI, and adult DRI for Na, B12, Fe, Zn, Ca. Regional differences were explored by PLS-DA. EU and US PBMAs converged on protein content (14.0 vs 15.3 g/100 g) but diverged on aging-relevant nutrients. EU products contained nearly twice the fiber (4.82 vs 2.55 g, +89%; medians 5.0 vs 1.85 g), with chickpea-, mushroom-, faba-, and mycoprotein-based products in the upper quartile. US products had twice as much saturated fat (3.74 vs 1.84 g) and 22% more sodium (608 vs 501 mg). Micronutrient label coverage was strongly asymmetric: Ca/Fe/Zn/B12 were reported in 96/97/3/6% of US products vs 1/21/4/21% of EU products. Within the reported subsets, US products showed higher Zn and B12 but lower Ca and Fe, consistent with selective US fortification of B12/Zn, whereas the EU rarely reported any micronutrient levels. PLS-DA separated regions along a fiber / saturated-fat / sodium axis. At a typical serving size (~120 g), neither region reached the 25–30 g per meal protein dose required to stimulate myofibrillar protein synthesis in older adults. These data identify a transatlantic nutritional profile split in PBMAs that is invisible at the global category level: EU and US products fail the healthy-aging profile in opposite, regulator-shaped ways, and neither current product class meets the combined protein, fiber and micronutrient needs of older adults. A PBMA generation suited to healthy aging will therefore require pairing EU-style fiber and lipid quality with US-style micronutrient transparency.

SELECTED ORAL PRESENTATION O04

Relations between diet and biomarkers of aging in middle-aged to older adults: an explorative cross-sectional analysis among three European cohorts

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Monday 31 August, 18.20–18.40 | Session 1

ABSTRACT

In the past decade, advances in molecular biomarkers of aging (BoA), capturing physiological processes related to morbidity and mortality risk, have driven research on how diet may slow the rate of biological aging. This rate is malleable, variable across individuals and used as proxy for healthy aging. Prior research has mainly focused on hypothesis-driven single nutrient or food associations with BoA, potentially overlooking components and poorly resembling how these are consumed within a diet. To explore relations between GrimAge2, DunedinPACE, DamAge, AdaptAge, MetaboAge, Personal-MetaboHealth and KDM-Age, and intake of nutrients and food groups using data-driven copula graphical models (CGMs). In this study, we analysed cross-sectional data from middle-aged to older males and post-menopausal females from three European cohorts; Northern Ireland Cohort for the Longitudinal Study of Aging (n=1342, 49%♀, aged 65±9y), Rotterdam Study (n=1073, 54%♀, aged 64±8y) and the Leiden Longevity Study (n=506, 53%♀, aged 59±6y). We used CGM to infer conditional dependence relations between self-reported intake of nutrients and food groups, in separate models (all nutrients in one model, food groups in another), with several epigenetic and metabolomic BoA, while adjusting for every remaining variable. Thus, each nutrient or food group association was corrected for intake of others, reflecting whole-diet intake. Datasets were analysed independently. In the overall populations greater intake of β-cryptoxanthin, calcium, tea, yogurt, fruits and oily fish were found to partially correlate with lower aging rates in ≥1 cohort, whereas findings for alcohol were mixed. Results differed considerably per cohort, clock and underlying characteristics (sex, age and BMI). Ongoing analyses will refine these findings and quantify magnitude of the strongest relations across cohorts. Data-driven analyses revealed direct relations among certain dietary components and rate of aging, yet reproducibility was limited across three separate cohorts. This study informs on components to test in future interventions.

SELECTED ORAL PRESENTATION O05

Average-increasing BMI trajectory in adolescence associates with multimorbidity

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Monday 31 August, 18.40–19.00 | Session 1

ABSTRACT

The developmental origins of health and disease hypothesis suggests that growth patterns are influenced by genetic factors and environmental conditions, leading to diverse growth trajectories and possibly varying health outcomes. Although research has identified similarities in growth trajectories across diverse populations, the relationship of these trajectories with health outcomes later in life remains uncertain. We investigated the connection between adolescent growth trajectories and multimorbidity in young adults. Growth was modeled in participants of Fin-HIT cohort study from school-age to early adulthood using repeated BMI measurements. A Latent Class Growth Analysis was employed to identify subgroups with similar growth patterns among 8328 participants having 2-3 BMI measurements recorded from ages 11 to 19. The identified BMI trajectories were: average-stable (87%), average-increasing (4.7%), high-stable (7%), and high-increasing (1.5%). Multimorbidity was assessed through self-reported conditions, quantified by the number of conditions (NoC) and a person-centered multimorbidity weighted index (MWI) by Wei et al. (2024). This validated index reflects the burden of cumulative chronic conditions and anticipated physical function decline over time. The study sample included 3436 participants with mean (SD) age of 19.4 (0.4) years, 62% of whom were female. Of these, 575 participants (16.7%) reported at least one condition affecting their lives. The most common conditions reported were asthma (n=202, 5.88%), depression (n=50, 1.46%), atopic dermatitis/eczema (n=45, 1.31%), allergy (n=40, 1.16%), and migraine (n=37, 1.08%). Three most frequent multimorbidity combinations were asthma and allergy (n=19), depression with anxiety (n=11), and asthma and migraine (n=8). The mean MWI was 0.29 (0.85), varying from 0 to 11.9. NoC or MWI did not differ between genders. Growth data was available for 3397 participants, and a hard trajectory classification was used. In ANOVA analysis, NoC ($p<0.001$) and MWI ($p=0.032$) differed between the BMI trajectory groups, with the highest NoC and lowest MWI were observed in the average-increasing group: difference from the acerage-stable group was for NoC +0.18 (95% CI 0.06; 0.30) and for MWI -0.22 (95% CI -0.02; -0.42). In this longitudinal study, 83.3% of young adults reported no conditions affecting their lives. BMI trajectories characterized as average-increasing, indicating weight gain after puberty, were linked to a higher number of conditions and a greater decline with time in physical functioning. Though the findings are modest, they suggest the importance of monitoring growth patterns in relation to later health outcomes.

SELECTED ORAL PRESENTATION O06

Temporal Hierarchy of Vitamin D Signaling: VDR-Centered Chromatin Dynamics Drive Immune Nutrigenomic Responses

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Tuesday 1 September, 10.40–11.00 | Session 2

ABSTRACT

Vitamin D is a central regulator of immune function and a key nutritional determinant of immune resilience and healthy aging. Its effects are mediated by the vitamin D receptor (VDR), which orchestrates genome-wide transcriptional programs. However, how dynamic VDR binding translates into temporally coordinated gene regulation in immune cells remains poorly understood, limiting mechanistic insight into vitamin D-driven nutrigenomic responses. Here, we performed a time-resolved multi-omics analysis of VDR-mediated gene regulation in human THP-1 monocytes following stimulation with 1,25-dihydroxyvitamin D3. VDR ChIP-seq profiles at 40 minutes, 4, 8, and 24 hours were integrated with matched transcriptomic data to resolve the spatiotemporal architecture of vitamin D signaling. We identified >22,000 VDR binding sites, of which >5,100 exhibited ligand-inducible occupancy that expanded progressively over time. This expansion paralleled a transition from an early, focused transcriptional response to a broad late-stage regulatory program. Integrating chromatin binding with gene assignment based on genomic proximity and topological domains revealed distinct regulatory architectures. Early-responsive genes were predominantly controlled by promoter-proximal VDR binding, frequently supported by coordinated enhancer activity, whereas late-responsive genes relied more on distal enhancer elements or indirect regulatory mechanisms. Notably, promoter-proximal VDR binding displayed higher inducibility, was associated with elevated basal gene expression, and conferred stronger transcriptional responsiveness compared to enhancer-dominated regulation. These findings define a hierarchical model of vitamin D action in which rapid promoter-centric regulation initiates core immune programs, followed by progressive enhancer-driven expansion of gene regulatory networks. This temporal layering provides a mechanistic explanation for how vitamin D fine-tunes immune responses rather than inducing uniform transcriptional changes. By resolving the dynamics of VDR-driven chromatin regulation, this study establishes a conceptual framework for linking nutritional signals to immune system behavior over time. Such insights are essential for the development of precision nutrition strategies and digital twin models that account for temporal response trajectories, ultimately supporting optimized vitamin D interventions to maintain immune competence and promote healthy aging.

SELECTED ORAL PRESENTATION O07

Genetic risk signatures in phenolic metabotypes for cardiometabolic health and disease: unravelling genomic complexity from GWAS and PGS

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Tuesday 1 September, 11.00–11.20 | Session 2

ABSTRACT

(Poly)phenols (PPs) are bioactive compounds abundant in plant-based food, associated with cardiometabolic (CM) and anti-inflammatory benefits. However, responses to PP intake vary widely among individuals. Metabotyping – clustering individuals by metabolic profiles – allows to capture this variability and partially explains association with disease risk. In parallel, advances in genomics allow the identification of susceptibility loci through genome-wide association studies (GWAS) and the quantification of genetic predisposition through polygenic scores (PGS). The investigation of PP metabotypes using GWAS and their integration with PGS has not been explored yet. In this study, we investigated whether PP metabotypes reflect underlying genetic risk for CM traits and diseases. We analysed data from the Oral (Poly)phenol Challenge Test (OPCT) study, including genetic and metabolic profiles. The cohort comprised 300 individuals (18-74 years, 57% female) of Southern European origin, and in general good health. Metabotyping on urinary metabolites classified individuals into two different phenolic excretion patterns. Genotyping on blood samples followed by genotype imputation provided over 6 million variants for GWAS and PGS calculation from the PGS Catalog. GWAS highlighted significant associations ($p<1E-3$) in genes related to PP bioavailability, such as UGT1A complex, SULT, SULF, and ABC. Novel associations ($p<1E-5$) resulted in three genomic loci (chromosomes 14, 16, and 22), including TSHR gene. Functional analyses pointed at regulatory mechanisms like gene dosage and epigenetic silencing. Integration with eQTL and chromatin interaction data revealed potential cis-regulatory modulation. Metabotype-stratified analyses on 4489 PGS identified 124 significant scores ($p<0.05$), including HDL cholesterol, type 2 diabetes, and coronary artery disease. The most represented categories were anthropometric (32 PGS), CM (31 PGS) and cancer (21 PGS). PCA revealed components enriched for brain morphology (PC1) and CM traits (PC2). A computed metaPGS explained a small portion of phenotypic variability between metabotypes ($R2=0.048$; $p=0.005$). These findings suggest how PP metabotypes can partially explain genetic risk profiles for CM traits, unveiling the underlying genomic complexity and paving the way for precision nutrition strategies.

SELECTED ORAL PRESENTATION O08

Multi-Omics Integration Explains Inter-individual Variability in Dietary (Poly)phenol Metabolism

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Tuesday 1 September, 11.20–11.40 | Session 2

ABSTRACT

Diet plays a central role in human health, and part of this effect is mediated by bioactive compounds such as dietary (poly)phenols. However, their true biological impact depends on how efficiently each person can metabolise them. These compounds undergo extensive transformations, both host- and microbiome-driven, and this process varies widely among individuals, giving rise to distinct metabolic phenotypes (or metabotypes). Understanding how the factors influencing the metabolism of (poly)phenols is crucial to explain why similar diets affect people differently, and how (poly)phenols may contribute to healthier trajectories. In this study, we analysed data from the Oral (Poly)phenol Challenge Test, an intervention designed to characterise inter-individual variability in (poly)phenol metabolism under controlled dietary conditions. Metabolomic analyses revealed two distinct metabotypes, marked by differences patterns in the production of phenolic metabolites. By integrating genomics, metabolomics, and metagenomics data through a supervised multi-omics framework, we identified coordinated patterns linking (poly)phenol metabotypes to gut microbial taxa and host genetic features. Further, factors like sex, age, smoking habits, and BMI were included as supervised covariates to assess how much they contribute to variability in metabolic responses. Our extended multi-omics analysis showed that (poly)phenol metabotypes were associated with specific genetic signatures, such as rs10485285, rs9341828 and rs8003061 for metabotype 1 and rs9283799, rs9359428 and rs36093037 for metabotype 2, mainly located in intergenic or intronic regions and therefore potentially involved in transcriptional or regulatory modulation. Regarding microbial signatures instead, metabotype 1 showed increased abundance of several gut microbial taxa, including Enterobacter, Streptococcus and Eubacterium, suggesting a microbial ecosystem potentially less efficient in PP-catabolism. Overall, our findings highlight that the benefits of diets rich in (poly)phenol might depend on the individual's ability to convert dietary (poly)phenols into bioactive metabolites. Multi-omics integration offers a powerful framework to unravel the complex interplay between diet, microbial metabolism, and host factors, ultimately guiding the development of more precision nutrition strategies.

SELECTED ORAL PRESENTATION O09

Molecular signatures of cellular senescence and their modulation by an in vitro-derived apple callus extract

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Tuesday 1 September, 11.40–12.00 | Session 2

ABSTRACT

Cellular senescence plays a key role in aging and tissue homeostasis while contributing to inflammaging and age-related diseases. In this study, human umbilical vein endothelial cells (HUVECs) were used as a model of replicative senescence to investigate transcriptomic and DNA methylation changes associated with aging. Comparative transcriptomic analysis between young (yHUVECs) and senescent cells (sHUVECs) revealed alterations in genes involved in cell cycle regulation, proliferation, and inflammatory pathways. Genome-wide DNA methylation profiling, performed using PromethION (Oxford Nanopore), identified differentially methylated CpGs and promoters, highlighting epigenetic remodeling during senescence. Given the interest in diet-derived bioactive compounds as modulators of aging, the short-term effect (10µg/mL, 72 h) of an ethanolic extract from in vitro–cultured callus of Mela Rosa Marchigiana (MRME) was evaluated in sHUVECs. MRME treatment downregulated TNF-α signaling genes, suggesting an anti-inflammatory effect. No significant changes in DNA methylation were observed after short-term exposure. To explore long-term effects, HUVECs were further treated daily with MRME (2 h/day) during replicative senescence (passages 4–17), and RNA-seq analysis is ongoing. Overall, these findings show the impact of senescence on both the transcriptome and methylome of HUVECs and suggest that MRME may modulate inflammaging-related genes, supporting its potential application in functional foods targeting age-associated inflammation.

SELECTED ORAL PRESENTATION O10

Hard-wired or acquired? Epigenetic and transcriptional profiling of Lgr5^{+} intestinal stem cells along the mammalian intestine

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Tuesday 1 September, 15.40–16.00 | Session 3

ABSTRACT

Enteroendocrine cells (EEC) are intestinal sensory cells that secrete biologically active peptides, neurotransmitters and metabolites in response to environmental stimuli. Although constituting only 1% of the intestinal epithelium, EEC are required for proper gut function and represent the largest endocrine organ in the human body. By releasing hormones like GLP-1, GIP, CCK, PYY and 5-HT, EEC connect the gut with the pancreas, liver, brain and adipose tissue in response to nutrient intake. Reflecting the distinct functions of each region, EEC hormone synthesis varies along intestinal regions. Yet, EEC have a limited lifespan and are continuously renewed from intestinal stem cells (ISC). This raises the question of whether ISC already harbor different epigenetic and/or transcriptional profiles based on their intestinal spatial localization, or whether spatial-specific differences are acquired at a later differentiation stage. To address this, we isolated Lgr5^{+} ISC from the duodenum, jejunum, ileum and colon from the murine intestine and performed single-cell RNAseq. Following quality control, clustering and dimensionality reduction, colon Lgr5^{+} cells clustered separately from small intestinal Lgr5^{+} cells. In contrast, duodenum, jejunum and ileum Lgr5^{+} cells clustered together with no evidence of regional transcriptional diversity. However, epigenomic profiling of small intestinal Lgr5^{+} cells revealed substantial differences in histone mark deposition (H3K4me3, H3K4me1, H3K27ac and H3K27me3) at gene TSSs across the three regions. In particular, we observed two contrasting patterns. In duodenum and jejunum, compared to ileum, we found an increased deposition of H3K27ac and H3K4me3 and a decreased deposition of H3K27me3, at the Gata4 TSS. Gata4 is a zinc finger transcription factor whose main role is to maintain intestinal regionalization by suppressing ileal gene programs. In the ileum, compared to both duodenum and jejunum, we observed a consistent enrichment of H3K27ac and H3K4me3 at the HoxB gene cluster – which play a role in determining regional identity along the anterior-posterior axis. Altogether, these results show that, while no transcriptional differences from different intestinal regions are observed in ISC, ISC from duodenum/jejunum and from ileum are epigenetically distinct, with specific gene sets already epigenetically primed based on their spatial location along the intestine. Notably, the re-analysis of publicly available human snRNAseq and snATAC-seq validated these patterns in human LGR5^{+} cells.

SELECTED ORAL PRESENTATION O11

Responders vs Non-Responders: Uncovering Individual Glucose Response Patterns to Dietary Bioactive Compounds

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Tuesday 1 September, 16.00–16.20 | Session 3

ABSTRACT

As the global prevalence of metabolic disorders rises, understanding how dietary bioactive compounds modulate glucose regulation is increasingly important for preventive healthcare and personalised nutrition. Despite growing interest in plant bioactives, their role in postprandial glucose responses remains poorly characterised at the individual level. This study investigated the influence of dietary bioactive compounds, nutrients, and gut microbiome composition on glucose regulation using data-driven analysis and machine learning. Data were drawn from the DIME Study, a controlled crossover intervention in which 20 participants (16 with complete data) followed a high-bioactive and a low-bioactive diet for two weeks each, separated by a four-week washout. Continuous glucose monitoring, dietary records, and microbiome profiling generated ~1,024 postprandial measurements, quantified via incremental Area Under the Curve (iAUC). Six machine learning algorithms (Random Forest, Elastic Net, SVR, LASSO, k-NN, Gradient Boosting) were evaluated, with feature contributions interpreted using SHAP values. Analysis revealed a clear bifurcation in metabolic phenotypes: "responders" showed significant iAUC differences between diets ($p<0.05$), while "non-responders" maintained stable patterns regardless of intervention. Coefficient of variation analysis showed nutrient profiles were comparable between groups (2.69%), whereas bioactive intake (17.56%) and gut microbiome composition (31.24%) differed substantially. Feature importance analysis identified flavonoids—particularly anthocyanins, flavones, and flavonols—as key predictors in responders, alongside starch and iron. In non-responders, glucose regulation was predominantly driven by carbohydrate intake, with microbiome features as secondary contributors. Lunch produced significantly higher glucose excursions than snacks in both groups ($p<0.01$), identifying meal timing as a relevant modulator. These findings suggest individual metabolic responses to bioactive compounds are heterogeneous and closely linked to gut microbiome composition, supporting personalised dietary strategies for metabolic health. The machine learning framework demonstrates the potential of data-driven approaches to uncover targeted nutritional interventions, warranting validation in larger, more diverse cohorts.

SELECTED ORAL PRESENTATION O12

Bitter Taste Receptor (Tas2r) Agonists Modulate Gut Hormones and Metabolic Markers in Aged Rats

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Tuesday 1 September, 16.20–16.40 | Session 3

ABSTRACT

Ageing is associated with a progressive deterioration of metabolic homeostasis, in which alterations in intestinal endocrine function play a key role. Bitter taste receptors are a family of G protein–coupled receptors comprising approximately 26 receptor subtypes in humans and 35 in rodents. Initially characterized in the oral cavity as sensors of potentially toxic compounds, Tas2r have also been identified in extra-oral tissues, including the gastrointestinal tract. However, their functional relevance in the regulation of enteroendocrine signaling and metabolic homeostasis during ageing remains largely unexplored. In this study, we investigated whether exposure to bitter compounds acting as ligands of selected Tas2r modulates enteroendocrine hormone profiles and associated metabolic markers in aged female rats (12 months). Animals were assigned to two treatment groups: a chronic intermittent Costunolide treatment administered over three months, and a MIX treatment consisting of Costunolide + KDT501 administered during the final two weeks of the experimental period. Costunolide was used as a ligand of rTas2r105, rTas2r119 and rTas2r120, while KDT501 acts as a ligand of rTas2r108. Ageing was associated with reduced energy expenditure and respiratory quotient (RQ), lower body weight gain, and increased circulating active GLP-1 levels and total cholesterol. Chronic intermittent Costunolide administration attenuated age-associated changes in energy expenditure, RQ and active GLP-1 levels. In contrast, the MIX treatment mitigated the reduction in body weight gain and prevented the increase in total cholesterol. Together, these findings provide in vivo evidence that modulation of bitter taste receptors influences enteroendocrine signaling and metabolic outcomes in aged animals. These results highlight Tas2r as a potentially relevant and underexplored pathway linking intestinal sensing to metabolic regulation during ageing.

SELECTED ORAL PRESENTATION O13

Bitter taste receptor agonists modulate intestinal dysfunction in aged rats under dietary stress

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Tuesday 1 September, 16.40–17.00 | Session 3

ABSTRACT

Aging is associated with chronic low-grade inflammation, intestinal barrier dysfunction and accumulation of senescent cells, all of which contribute to metabolic decline and frailty. Nutrient-sensing pathways are increasingly recognized as modulators of these interconnected processes. Among them, extra-oral bitter taste receptors (TAS2Rs) have emerged as potential regulators of gut physiology and immune responses, although their role in age-related intestinal dysfunction remains poorly defined. This study aimed to evaluate whether TAS2R activation can modulate inflammation, gut barrier integrity and senescence-associated processes in the context of aging and an obesogenic challenge. A comprehensive experimental design including seven groups was implemented: young controls, aged animals under standard diet, and aged animals exposed to an obesogenic cafeteria diet, with distinct intervention strategies using Costunolide (10 mg/kg in standard diet groups and 20 mg/kg in cafeteria diet groups). This design enabled the assessment of TAS2R-mediated effects across physiological aging and diet-induced metabolic stress. Systemic inflammation was assessed using multiplex cytokine profiling and gene expression analysis in peripheral blood mononuclear cells. In the intestine, inflammatory and barrier-related markers were evaluated in the duodenum, jejunum and ascending colon. Intestinal permeability was further assessed through circulating endotoxin and antigen translocation measurements. Cellular senescence was analysed in intestinal and extra-intestinal tissues. Frailty was longitudinally assessed at multiple time points as a functional readout of aging. Overall, the data indicate that TAS2R agonists differentially regulate inflammatory status, barrier integrity and cellular senescence in aged animals, with responses depending on dietary conditions and dosing. Notably, effects were more pronounced under obesogenic conditions, supporting a role for TAS2Rs in the adaptive response to metabolic stress. These findings position TAS2R signalling as a relevant nutrigenomic interface linking dietary sensing with key mechanisms of aging. This work provides an integrative framework connecting gut barrier function, inflammation and senescence, and supports the potential of targeting TAS2Rs as a nutritional strategy to mitigate age-related metabolic and functional decline.

SELECTED ORAL PRESENTATION O14

Habitual coffee consumption and longitudinal NMR metabolite trajectories in an aging Finnish cohort

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Tuesday 1 September, 17.00–17.20 | Session 3

ABSTRACT

Habitual coffee consumption has been linked to metabolic and inflammatory markers in cross-sectional and short-term studies, but how mid-life intake relates to the trajectory of circulating metabolites across later life remains less well characterised. The Finnish elderly cohort is particularly informative: per-capita coffee intake is among the highest worldwide. Participants in the Helsinki Birth Cohort Study (HBCS; born 1934–1944; N≈2000 at 2001 baseline, mean age 62) underwent serum NMR metabolomics (Nightingale platform, ~250 biomarkers) at four waves spanning 17 years (2001, 2007, 2012, 2018). Baseline coffee intake from a food-frequency questionnaire was categorised as <1, 1–2, 2–3 and ≥3 cups/day (150 ml Finnish cup). Linear mixed-effects models with restricted cubic splines for age, coffee-category × age interactions, and random intercepts per participant were fitted to each metabolite, in the combined cohort and separately by sex. Adjusted models included BMI, diabetes, leisure-time physical activity, dietary pattern, smoking, alcohol, education, chronic disease and statin use. In the combined cohort, higher habitual coffee consumption was associated with lower longitudinal trajectories of alanine, glycoprotein acetyls (GlycA), glycerol and lactate, all robust to full covariate adjustment. A male-specific signal additionally emerged for phenylalanine, with high consumption associated with lower trajectories. The surviving metabolites converge biologically: alanine, lactate and glycerol together index gluconeogenic-substrate flux from muscle and adipose tissue, while GlycA captures low-grade systemic inflammation. In a Finnish aging cohort followed for 17 years, habitual mid-life coffee consumption is associated with systematically lower trajectories of inflammatory and gluconeogenic-substrate metabolites, independent of measured lifestyle and metabolic confounders, with an additional male-specific signal in aromatic amino acid metabolism. These findings link a widely consumed exposure to concrete longitudinal biomarker patterns of inflammageing and substrate metabolism and illustrate the utility of NMR trajectory analysis for life-course nutrition research.

SELECTED ORAL PRESENTATION O15

Decoding Individual Responses to Calorie Restriction: PBMC Multi-Omics Reveals Immune-Metabolic Reprogramming for Precision Nutrition

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Wednesday 2 September, 10.40–11.00 | Session 4

ABSTRACT

Interindividual variability remains a major barrier to the effective implementation of calorie restriction (CR) as a strategy to combat obesity, metabolic disease, and accelerated biological aging. Moving beyond "one-size-fits-all" approaches requires mechanistic insight into how individuals respond at the molecular level to dietary interventions. Here, we performed a controlled intervention study comparing two isocaloric CR strategies, a standard diet (CRS) and a ketogenic diet (CRK), in women with overweight and obesity (BMI 29.8 ± 2.17 kg/m²; CRS: n=15, CRK: n=25) over 8 weeks. To capture systemic, individual-specific responses, we applied transcriptomic (RNA-seq) and epigenomic profiling (ATAC-seq) of peripheral blood mononuclear cells (PBMCs), a minimally invasive and clinically scalable model reflecting integrated immune-metabolic regulation. Both interventions significantly improved body composition and metabolic parameters. However, multi-omics profiling revealed pronounced molecular heterogeneity between individuals, despite comparable clinical outcomes. A total of 476 genes were differentially expressed (FDR<0.05), defining a coordinated adaptive program to sustained energy restriction. Upregulated pathways (n=352 genes) highlighted mitochondrial oxidative metabolism, stress resilience, and cellular maintenance, while downregulated pathways (n=124 genes) reflected suppression of energy-intensive processes. Strikingly, immune signaling pathways exhibited bidirectional regulation, indicating a dynamic reprogramming of immune function rather than uniform anti-inflammatory effects. These findings identify CR as a driver of systemic immune-metabolic remodeling linked to mechanisms of healthy aging, including enhanced cellular efficiency and adaptive resilience. Importantly, the observed divergence in molecular responses between individuals, despite similar phenotypic outcomes, provides direct evidence that clinical readouts alone fail to capture the complexity of dietary responses. By integrating PBMC-based multi-omics signatures, this study establishes a foundation for developing predictive biomarkers of dietary responsiveness and supports the implementation of digital twin models in precision nutrition. Such approaches will enable the stratification of individuals into responders and non-responders and guide personalized dietary interventions aimed at improving metabolic health and extending healthspan.

SELECTED ORAL PRESENTATION O16

Differential Effects of Vitamin Supplementation on DNA Methylation-Based Biological Age and Functional Outcomes in Aging Adults

Pitaksin Chitta[1,2,3], Timothy M. Barrow[4], Busadee Pratumvinit[2], Atchara Dawangpa[1,2], Saliltip Khamrangsee[2], Witchayaporn Kaewboonruang[2], Viktor I. Korolchuk[5], Chanachai Sae-Lee[1,2,6*]

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Wednesday 2 September, 11.00–11.20 | Session 4

ABSTRACT

As Thailand transitions into a fully ageing society, understanding both functional and molecular determinants of healthy ageing have become increasingly important. This study investigated the effects of vitamin B9, B12, and D supplementation on physical performance, metabolic biomarkers, and epigenetic biological age in older adults. A total of 125 participants aged ≥60 years were enrolled in a 12-month intervention and assigned to four groups: Group A (placebo, n=32), Group B (vitamin B9 and B12, n=28), Group C (vitamin D, n=30), and Group D (vitamins B9, B12, and D, n=35). Between-group comparisons revealed that Group D exhibited a significant increase in GrimAgeV2_Leptin compared with Group C (P=0.003), whereas Group C showed higher DNAmGait (P=0.024) and lower DNAmFitAge and FitAgeAccel (P=0.012) compared with Group D, suggesting improved physical fitness-related biological ageing with vitamin D supplementation. Sex-stratified analysis demonstrated that males in Group C had higher DNAmGrip and lower DNAmFitAge and FitAgeAccel compared with males in Group D (P=0.015 and P=0.012, respectively), while females in Group D showed increased GrimAgeV2_Leptin compared with Group C (P=0.021). Within-group longitudinal analysis showed that the placebo group (Group A) experienced significant increases in GrimAgeV2, PhenoAge, and DNAmFitAge over 12 months, indicating accelerated biological ageing. Group B showed increases in Horvath age and AgeAccelHorvath, whereas Group C demonstrated a reduction in GrimAgeV2_Leptin (P=0.018) but a modest increase in FitAgeAccel (P=0.039). In contrast, Group D exhibited increases in GrimAgeV2_Leptin, PhenoAge, DNAmFitAge, and FitAgeAccel, alongside reductions in DNAmGait and DNAmGrip. Overall, vitamin D supplementation (Group C) was associated with favourable changes in fitness-related epigenetic ageing markers, whereas combined supplementation (Group D) showed mixed effects on biological ageing indicators. These findings highlight the complex and differential impacts of micronutrient interventions on epigenetic ageing and support the development of targeted nutritional strategies for healthy ageing in older populations.

SELECTED ORAL PRESENTATION O17

Trace element supply and status in old age with a focus on zinc, selenium and copper

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Wednesday 2 September, 11.20–11.40 | Session 4

ABSTRACT

Aging is associated with a decline in physiological functions and an increased risk of age-related diseases, highlighting the importance of developing nutritional strategies for healthy aging. Trace elements play a crucial role in maintaining optimal health. We therefore investigated how trace element status and serum concentrations change with age and whether this is associated with functional decline. Several cohort studies and one intervention study were used to analyze serum concentrations of essential trace elements such as zinc, selenium, and copper and their functional biomarkers. Analyses were also performed in aged mice fed an age-adapted, trace element-adjusted diet. Serum levels of selenium and zinc decreased with age, while copper levels increased. This was less pronounced in healthy older adults, whereas the changes were particularly strong in frail older adults. Chronic inflammation appears to be a driving factor in this latter group. Postprandial dynamics of serum selenium levels also revealed age-related differences. These age-related changes in serum trace element levels were also observed in a mouse model. However, an age-adapted diet with higher levels of zinc and selenium could not compensate for these age-related changes. This suggests that age-related changes in serum trace element levels have various causes. In addition to inflammation, insufficient trace element intake or absorption could be a contributing factor.

SELECTED ORAL PRESENTATION O18

Association of plant-based protein intake with epigenetic age deceleration in the Netherlands Twins Register

Alice Ricco[1], Stieneke Doornweerd[1], Jenny van Dongen[2], Lannie Ligthart[2], Richard IJzerman[1], Eco J.C. de Geus[2], Georges E. Janssens[1], Gajja S. Salomons[1]

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Wednesday 2 September, 11.40–12.00 | Session 4

ABSTRACT

Chronological age progresses uniformly over time, whereas biological aging varies substantially between individuals. Diet is a key modifiable factor influencing aging trajectories, yet whether dietary protein source—whether plant or animal based—specifically affects epigenetic aging remains unclear. Understanding how dietary composition relates to biological aging may help identify nutritional strategies that promote healthy aging. To address this, we analyzed dietary intake in adults from the Netherlands Twin Register, including the percentage of plant and animal protein intake as primary exposures. Participants were originally selected based on extremes of BMI relative to their cotwin or BMI polygenic risk score (PRS). After exclusion of individuals with missing epigenetic clock data, the final analytical sample included 44 subjects, comprising 13 twin pairs (n = 26) and 18 unpaired individuals. Epigenetic aging from DNA methylation arrays was assessed using five clocks: Hannum, IEAA, PhenoAge, GrimAge, and DunedinPACE. Plant protein intake (g/day) showed a consistent inverse association with epigenetic age acceleration across all epigenetic clocks, whereas animal protein intake showed positive associations across clocks. Across all five epigenetic clocks, regression coefficients were consistently negative for plant protein and positive for animal protein. Moreover, the differences between the regression coefficients for the two protein sources were statistically significant ($p < 0.001$). In multivariable GEE models adjusted for year of birth, BMI, and smoking and clustered by twin pair, the percentage of plant protein intake was significantly associated with lower epigenetic age acceleration across three clocks, whereas the percentage of animal protein intake was significantly associated with higher age acceleration. Exploratory nutrient-wide analyses of 112 nutrient components identified multiple dietary components associated with epigenetic aging. Notably, the percentage of plant protein was the strongest nutritional component significantly associated with lower biological age across all clocks, while the percentage of animal protein intake was the principal factor associated with higher biological age across clocks. Protein source is differentially associated with biological aging, with plant-derived protein showing more promising associations across multiple epigenetic clocks. Beyond protein, a broader set of dietary components contributes to aging variability, with both shared and clock-specific signals. These findings support a systems-level role of diet in biological aging and suggest that dietary composition, particularly favoring plant protein over animal protein sources, may be an important factor in strategies aimed at promoting healthy aging.

SELECTED ORAL PRESENTATION O19

Multi-Omics Dissection of Vitamin D_3 Responses Reveals Context-Dependent Immune Regulation in Multiple Sclerosis

Tanya Tripathi[1], Emilia Gospodarska[1], and Carsten Carlberg[1,2]

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Wednesday 2 September, 15.40–16.00 | Session 5

ABSTRACT

Vitamin D deficiency is a major environmental risk factor for multiple sclerosis (MS) and has been linked to impaired immune regulation and accelerated immunological aging. However, the in vivo molecular mechanisms underlying individual responses to vitamin D_3 supplementation remain poorly defined, limiting its implementation in precision nutrition strategies. Here, we applied longitudinal multi-omics profiling combining RNA-seq and ATAC-seq in peripheral blood mononuclear cells (PBMCs) from MS patients undergoing distinct vitamin D_3 supplementation regimens, alongside healthy individuals receiving monthly bolus dosing. This design enabled within-individual resolution of transcriptional and epigenetic responses over three months under real-world intervention conditions. Despite supplementation, MS patients maintained distinct transcriptomic and chromatin accessibility landscapes compared to healthy controls, indicating persistent disease-associated immune states. Rather than inducing global transcriptomic reprogramming, vitamin D_3 elicited selective, context-dependent modulation of immune regulatory networks. In MS patients receiving daily high-dose supplementation, responsive genes were enriched in innate immune and inflammatory pathways, including Toll-like receptor signaling and cytokine regulation. Integration with chromatin accessibility data revealed widespread disease-associated regulatory differences and only partial concordance between epigenomic and transcriptional responses, underscoring complex multilayered regulation. Strikingly, intersection of vitamin D-responsive genes with vitamin D receptor (VDR) binding datasets identified a focused set of candidate target genes with high relevance to MS pathophysiology, providing a mechanistic link between supplementation and immune modulation. Importantly, substantial inter-individual variability was observed at both transcriptional and epigenetic levels, highlighting personalized response patterns that are not captured by clinical parameters alone. These findings redefine vitamin D_3 supplementation as a context-dependent immune modulator rather than a uniform anti-inflammatory intervention and emphasize the importance of multi-omics approaches to resolve individual response trajectories. By integrating transcriptional and epigenetic information, this study provides a framework for developing predictive biomarkers and digital twin models of immune regulation in MS. Overall, our results support the implementation of nutrigenomics-driven precision nutrition strategies aimed at restoring immune homeostasis, improving disease management, and promoting healthy aging in chronic inflammatory conditions.

SELECTED ORAL PRESENTATION O20

The Brain Derived Neurotrophic Factor Gene and Dietary Decisions

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Wednesday 2 September, 16.00–16.20 | Session 5

ABSTRACT

The brain derived neurotrophic factor (BDNF) rs6265 G>A (Val66Met) single nucleotide polymorphism (SNP) modifies reward processing and subsequently dopaminergic-related behaviours. Psychological drivers of food choice include food adventurousness (FA), or neophilia, a high willingness to try novel foods, and loss aversion (LA), where the fear of loss exceeds any potential gain. Individual with LA may select familiar foods or opt to retain items in preloaded food choice scenarios. While rs6265 variation may modify these behaviours, the topic remains under-researched. Therefore, we aimed to assess the influence of rs6265 variation on FA and LA, considering food choices and body mass index (BMI). Two studies were completed; (1) a randomised crossover study, on 95 participants, assessing food choice via an add/remove pizza-building scenario, alongside an objective measurement of LA and rs6265 genotyping; (2) a cross-sectional analysis, on 2823 participants, using pre-existing demographic, lifestyle, FA, food liking, and rs6265 genotype data. In study 1, there was an interaction effect between LAH and LAL groups and rs6265 ($p<0.05$; Figure 1). Those that were LAL with the ValVal genotype added fewer healthy items (34 ± 24%) than Met carriers (56 ± 30%; $p<0.05$), and LAH Met carriers added more unhealthy items (47 ± 26%) than the ValVal genotype group (34 ± 22%; $p<0.05$). There was no interaction effect between LAH and LAL groups and rs6265 with removed or kept healthy and unhealthy toppings ($p>0.05$) However, in the removed setting, those that were LAH with the ValVal genotype removed more healthy items (38 ± 25%) than Met carriers (20 ± 30%; $p<0.05$). In study 2, the MetMet genotype was associated with a higher FA and BMI. Additionally, FA was associated with liking of alcohol, vegetables and fruit, and BMI was associated with liking meat and foods high in saturated fat. No association was found between rs6265 and food liking. Together, these results indicate that rs6265 variation may influence food choice through dopaminergic-related dietary behaviour. Specifically, high LA combined with genetic predispositions to reduced dopamine activity may promote greater food retention irrespective of perceived healthiness, and rs6265 variation may influence FA with potential BMI outcomes. Future longitudinal approaches are necessary to clarify the mechanisms linking BDNF variation to eating behaviour and metabolic outcomes.

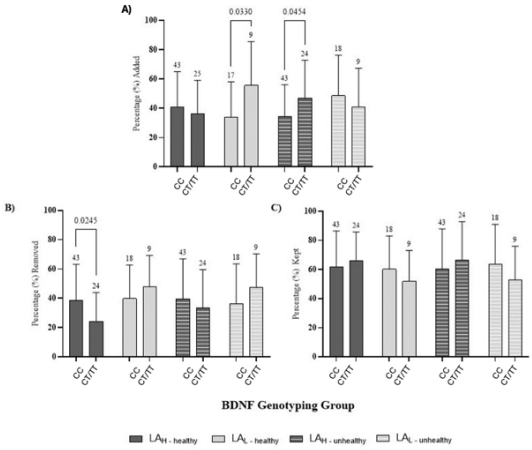

Figure 1. Total (A) added, (B) removed and (C) kept healthy and unhealthy toppings by LA and BDNF rs6252.
Exact p-values shown in figure.

SELECTED ORAL PRESENTATION O21

Adjustments in meal timing and macronutrient composition improve postprandial metabolic and inflammatory markers in night-shift workers

Kerstin Schorr[1], Suzan Wopereis[1], Maaike van der Rhee[2], Martijn E.T. Dollé[3], Inês Chaves[2], Tim J. van den Broek[1], Linda W.M. van Kerkhof[2], Alex Burdorf[2], Heidi M. Lammers-Van der Holst[2], Johanneke E. Oosterman[1,2]

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Wednesday 2 September, 16.20–16.40 | Session 5

ABSTRACT

Night work disrupts circadian rhythms and alters glucose homeostasis, increasing long term metabolic risk. Although nutritional interventions show beneficial short-term effects under controlled conditions, evidence from real life settings in night-shift workers remains limited. To evaluate whether a personalized nutrition intervention improves metabolic and inflammatory responses in night shift workers. In a non-blinded controlled intervention study, healthy male night shift workers aged 18 to 60 years participated in a three-month personalized nutrition intervention following a one-month run in period. Personalization was based on demographic characteristics combined with postprandial metabolic responses to a standardized mixed meal tolerance test. Advice focused on advancing the timing of energy intake, reducing caloric intake during night shifts, and adjusting macronutrient composition to reflect diurnal fluctuations in insulin sensitivity, with relatively higher carbohydrate earlier in the day and higher fat intake later in the day. Controls continued habitual dietary behavior. Food intake was assessed using a digital food diary, and diet quality was quantified using the Dutch Healthy Eating Index. Postprandial metabolic and inflammatory markers were measured before and after intervention. In the intervention group (n=22), the timing of total energy intake during night shifts advanced by 180 minutes at intervention start and by 120 minutes at intervention end relative to baseline, driven mainly by earlier intake of carbohydrates and fat. Total energy intake and diet quality remained unchanged, and no dietary changes were observed in controls. Postprandial total and HDL cholesterol levels improved following the intervention, while glucose, insulin and triglyceride responses did not change. Postprandial concentrations of several inflammatory markers, including IL 10, E selectin, ICAM 1 and TNF α were reduced after intervention. A three-month personalized nutrition intervention targeting meal timing and macronutrient distribution is feasible in night shift workers and associated with improved postprandial lipid and inflammatory responses. Adjusting meal timing may represent an effective strategy to mitigate metabolic disruption in real life night work settings.

SELECTED ORAL PRESENTATION O22

Investigation of the impact of melatonin receptor 1B (MTNR1B) genotype on continuous glucose monitor derived metrics

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Wednesday 2 September, 16.40–17.00 | Session 5

ABSTRACT

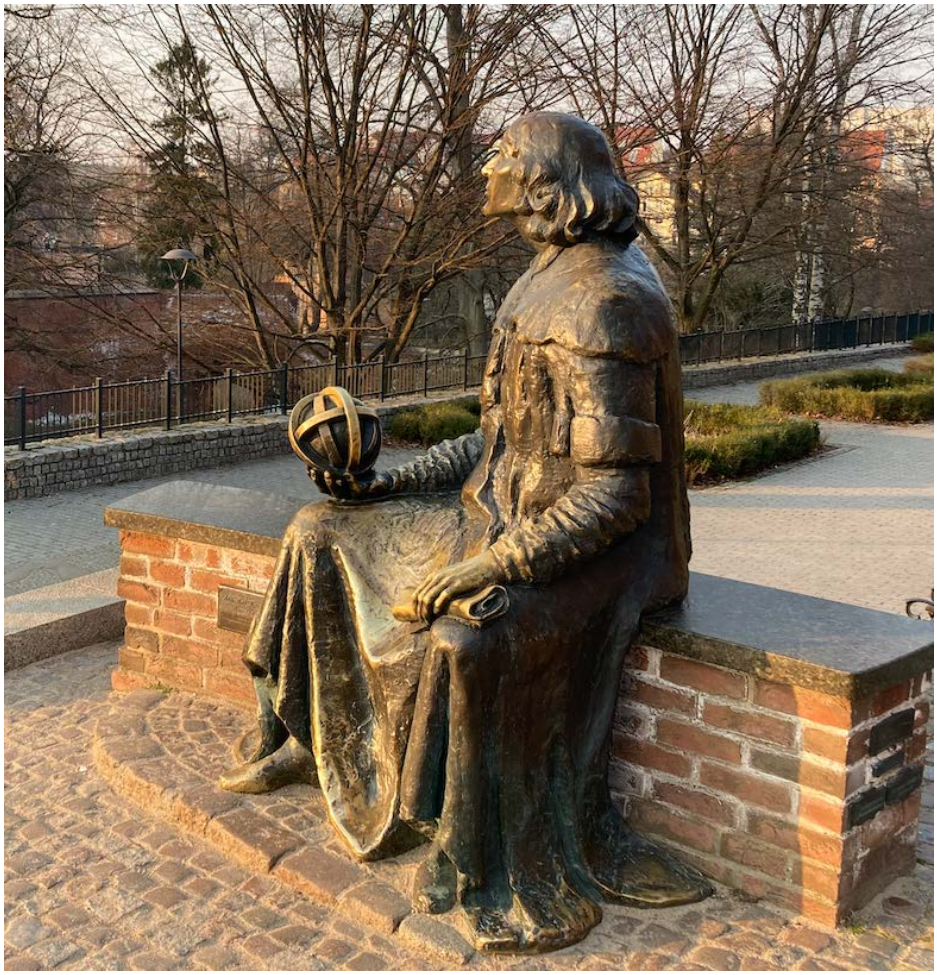
The risk allele (G) MTNR1B rs10830963 has been associated with increased fasting glucose, impaired glucose tolerance, and type 2 diabetes (T2D). Our research has demonstrated that fasting glucose and post-prandial glucose response to a high-carbohydrate breakfast were significantly greater in participants with a GG genotype than those with a CC or CG genotype. Continuous glucose monitors (CGMs) are increasingly used in individuals with euglycemia and several CGM-derived metrics have been associated with health in people without diabetes. Firstly, to determine associations between CGM-derived metrics (time in range (TIR), glycaemic variability, mean glucose concentration) and participant characteristics (age, BMI, fasting glucose, chronotype score). Secondly, to compare CGM-derived metrics between participants with a GG, GC and CC MTNR1B rs10830963 genotype. Post-prandial glucose levels were recorded for two weeks using a CGM. TIR was calculated using two different cut-offs: 3.9–7.8 mmol/L American Dietetic Association target range (TIRADA) and 3.9–5.6 mmol/L published by Berry et al. (2023) associated with need for diabetes screening (TIRBerry). The associations between CGM-derived metrics and participant characteristics were assessed using correlation analysis. CGM-derived metrics were compared between genotype groups for MTNR1B using a one-way ANOVA. Data was available for 53 participants. Age was significantly correlated with glucose variability (r_s=0.305, p=0.026). Mean glucose was significantly associated with fasting glucose (r=0.758, p<0.001). TIRBerry was significantly correlated with mean glucose levels (r=-0.947, p<0.001) and fasting glucose (r=-0.793, p<0.001). GG participants had a significantly higher mean glucose concentration over the two-week collection period than CG (p=0.004) or CC participants (p=0.009). GG participants had a significantly lower TIR using the more stringent cut-offs (TIRBerry) compared to CG (p=0.01) or CC (p=0.005) participants. The study findings demonstrate, in a relatively young and healthy population, MTNR1B genotype significantly affects CGM-metrics associated with need for T2D screening.

P

Poster Presentations

Poster abstracts grouped by the five NuGOweek scientific sessions

Poster Session 1: Tuesday 1 September, 12.00–14.00 (P01–P20) | Poster Session 2: Wednesday 2 September, 12.00–13.45 (P21–P40)



POSTER PRESENTATION P01

Maternal Fructose Exposure Reprograms Astrocytic Metabolic Resilience through a Butyrate-MCT4-Glycogen Axis

Kay L.H. Wu[1,2], Wen-Chung Liu[3-5], Chih-Wei Wu[3], Mu-Hui Fu[6,7], Hsiu-Mei Huang[7,8], You-Lin Tain[7,9], Chih-Kuang Liang[10], Chun-Ying Hung[1], I-Chun Chen[1], Pi-Lien Hung[7,9], Yu-Ju Lin[7,11], Hajime Hirase[12]

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Tuesday 1 September, 12.00–14.00 | Poster Session 1 | Session 1: Emerging Science in Life Course Nutrition

ABSTRACT

Maternal fructose overconsumption may program long-term metabolic vulnerability in offspring, but the astrocytic mechanisms linking early-life nutrition to impaired brain energy resilience remain unclear. We tested whether maternal high-fructose diet disrupts a butyrate-sensitive pathway controlling glycogen homeostasis and astrocytic polarity in the offspring hippocampus. Astrocyte-enriched cultures were prepared from female offspring exposed to maternal normal or high-fructose diet during gestation and lactation. Glycogen content, glycolytic capacity, NAD^+/NADH ratio, intracellular butyrate, glycogen-regulatory proteins, MCT4, and AQP4 were assessed. Directionality was examined using sodium butyrate and MCT4 overexpression. Maternal high-fructose exposure reduced glycogen stores, glycolytic capacity, and the NAD^+/NADH ratio. It also decreased intracellular butyrate, MCT4, glycogen synthase, and AQP4, while increasing glycogen phosphorylase B. Sodium butyrate restored intracellular butyrate, MCT4, glycogen synthase signaling, and AQP4 without normalizing glycogen phosphorylase B. MCT4 overexpression restored glycogen synthase signaling and increased AQP4-MCT4 colocalization. These findings identify a butyrate-sensitive MCT4-glycogen control node through which maternal fructose exposure programs astrocytic metabolic vulnerability. The study links early-life diet, short-chain fatty acid handling, energy buffering, and astrocytic polarity, highlighting MCT4 as a potentially modifiable metabolic target.

POSTER PRESENTATION P02

Development of a Healthful Plant-Based Diet Screening Tool

Hannah Alley[1], Alix Hughes[1,2], Sarah Brennan[3], Jayne Woodside[3], Lorraine Brennan[1,2]

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Tuesday 1 September, 12.00–14.00 | Poster Session 1 | Session 1: Emerging Science in Life Course Nutrition

ABSTRACT

Plant-based diets have become a popular option due to their positive associations with health outcomes. The healthful plant-based diet index (hPDI) measures adherence to a diet of high-quality plant-based foods. Currently, hPDI is calculated using 24h recalls, food diaries or food frequency questionnaires. There is no available screening tool to assess hPDI. To develop a hPDI screening tool for use in future studies. The hPDI encompasses 19 food groups, with high quality plant-based foods assigned a positive score, and low-quality plant-based foods and animal products assigned a reverse score. The tool was designed to capture frequency of intake of each of the food groups over the course of a week. Comparative analysis of the screening tool was completed using a previously collected data set (n=175), that scored hPDI from four 24 h recalls. The original hPDI scores from the previous data set and the screening tool scores were compared using Pearson Correlation and Bland Altman, and sex differences were analysed using an Independent Samples T-Test. Females achieved a significantly higher score than males in both the original hPDI score and screening tool hPDI score ($p<0.05$). The hPDI screening tool was significantly correlated with the hPDI score calculated from four 24 h recalls ($r=0.960$; $p<.001$). The Bland Altman showed a mean bias of -0.97 and the limits of agreement were narrow (6.009, -4.069). The new hPDI screening tool had a good level of agreement with the previously scored hPDI data. The results indicate that the hPDI is a useful screening tool to determine plant-based diet quality in future studies.

POSTER PRESENTATION P03

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

Tilde Martinsen[1-2], Janine Wirth[1-2], Lorraine Brennan[1-2]

[1]UCD School of Agriculture and Food Science, University College Dublin, Dublin 4, Ireland. [2]Conway Institute, University College Dublin, Dublin 4, Ireland

tilde.martinsen@ucdconnect.ie

Tuesday 1 September, 12.00–14.00 | Poster Session 1 | Session 1: Emerging Science in Life Course Nutrition

ABSTRACT

Higher protein intake may support muscle function in older adults, but the impact of different protein sources on the metabolomic profile is unclear. This study aimed to assess the effects of plant versus dairy protein on the plasma metabolomic profile and explore associations between metabolites and handgrip strength. This secondary analysis used data from a 12-week double-blind randomised controlled trial in 171 healthy adults aged ≥50 years allocated to high plant protein (23 g/day), high dairy protein (22 g/day), or low-protein control (2 g/day). Plasma samples collected pre- and post-intervention were analysed using targeted LC-MS metabolomics. Main and interaction effects were evaluated using ANOVA-simultaneous component analysis (ASCA). Associations between metabolites and handgrip strength were tested using Spearman correlations with false discovery rate correction ($q<0.05$). ASCA showed no main effect of group ($p=0.23$) and no group×time interaction ($p=1.00$), while time significantly influenced the metabolomic profile ($p<0.05$). Twenty-six metabolites correlated with baseline handgrip strength. Creatinine showed the strongest positive correlation ($r=0.53$), followed by leucine ($r=0.38$), proline ($r=0.35$), and isoleucine ($r=0.33$). Baseline leucine ($r=0.29$) and isoleucine ($r=0.27$) correlated with improvements in handgrip strength, while PCaeC32:2 ($r=-0.28$) and SM(OH)C22:2 ($r=-0.26$) correlated inversely; leucine increased across tertiles of strength improvement ($p<0.05$). Metabolomic changes were not related to the type of protein. However, metabolite levels were correlated with handgrip strength outcomes, suggesting the need to comprehensively evaluate the metabolic state of individuals prior to interventions.

POSTER PRESENTATION P04

Pteroyouth®, a pterostilbene-silybin formulation, in combination with nicotinamide riboside, may modulate redox resilience to exercise-induced metabolic stress in healthy volunteers

Marc Alemany-Fornés , Georgina Stella, Ignaci Miquel, Joan Albertí, Fernando Barrera ,Anna Crescenti, Ma. Josefina Ruiz de Azua, Bárbara Tello-Duran, Alícia Domingo-Tàrrech, Andrea Costa, Antoni Caimari, Juan María Alcaide-Hidalgo, Anna Mur

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Tuesday 1 September, 12.00–14.00 | Poster Session 1 | Session 1: Emerging Science in Life Course Nutrition

ABSTRACT

Healthspan is increasingly linked to the capacity to maintain physiological resilience in response to repeated metabolic, inflammatory and redox stressors. Highintensity exercise provides a controlled human model to study adaptive stress responses relevant to healthy aging, since it acutely increases reactive oxygen species while contributing to hormetic signalling. This pilot study investigated whether Pteroyouth®, a combination of pterostilbene cocrystal with silibinin phytosome, plus nicotinamide riboside chloride, modulates redox and muscle-stress responses to high-intensity exercise. Methods: A registered, randomized, triple-blind, placebo-controlled crossover pilot trial in 12 healthy male amateur runners (NCT07024966). Participants consumed Pteroyouth®, providing 100 mg pterostilbene and 78.2 mg silibinin, alongside 300 mg nicotinamide riboside per day, or matched placebo for 14 days, followed by a 45-min treadmill run at 80% VO2max. After washout, participants crossed to the alternate condition. Plasma malondialdehyde (MDA), ferric reducing antioxidant power (FRAP) , high-sensitivity C-reactive protein (hsCRP) and lactate dehydrogenase (LDH) were assessed before and after exercise (15 min). Exploratory paired responder and phenotypebased analyses are ongoing. The exercise challenge induced a measurable acute stress response, with significant increases in MDA (oxidative stress/cellular damage) and LDH (muscle damage) after both treatments. MDA responses were comparable between treatment and placebo (+1.23 vs +1.26 umol/L; between-treatment p=0.546), indicating no significant suppression of exercise-induced lipid peroxidation. FRAP (total antioxidant capacity) increased significantly after supplementation (+46.5 umol/L; p=0.028), whereas placebo showed a smaller, non-significant change (+19.2 umol/L; p=0.493). LDH increased in both conditions, with a numerically smaller increase with treatment than placebo (+20.5 vs +32.8 U/L), although without a significant between-treatment difference. Highsensitivity C-reactive protein showed a trend favoring supplementation. Pteroyouth® (pterostilbene cocrystal and silibinin phytosome) + nicotinamide riboside increased post-exercise plasma antioxidant capacity without significantly suppressing acute lipid-peroxidation or muscle-stress responses. Changes in LDH and high-sensitivity C-reactive protein suggest potential modulation of musclestress and inflammatory responses. Future studies should evaluate NAD+-related metabolism, inflammatory resolution, recovery kinetics and responder phenotypes in populations with reduced stress resilience or functional reserve. The authors would like to thank the Eurecat Nutrition and Health Unit team, particularly Ma. Josefina Ruiz de Azua, PhD; Anna Crescenti Savall, PhD; and Antoni Caimari Palou, PhD, for conducting the clinical study, coordinating participant visits, and performing sample collection and laboratory analyses.

POSTER PRESENTATION P05

Genetic contribution to taste perception and food-liking in elderly: new perspectives on healthy aging

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Tuesday 1 September, 12.00–14.00 | Poster Session 1 | Session 1: Emerging Science in Life Course Nutrition

ABSTRACT

Genetic variability contributes to interindividual differences in food-liking and food choices alongside multiple factors like accessibility, familiarity, and psychological determinants. Key variants affect preferences either directly through taste-perception genes or indirectly via reward- and metabolism-related genes. Understanding how genetic contributions shape food-liking in the elderly is essential to elucidate the mechanisms underlying food choices and sustain adequate dietary quality, ultimately supporting healthier aging trajectories. A total of 140 patients aged >75 have been randomized and tested for 9 Single Nucleotide Polymorphisms (SNP) in TAS2R38, TAS1R2, GNAT3, ANKK1, DRD2 and SLC2A2 associated with interindividual variation in taste perception, food liking, and metabolic susceptibility. Taste phenotyping was performed with filter-paper kits and Labeled Magnitude Scale, and food-liking was collected through questionnaire. Dietary history, anthropometric data, and multidimensional assessment were recorded. Preliminary results from 70 patients show genotyping–phenotyping correlation trends for taste-related variants largely matching evidence from general population. TAS2R38 rs1726866(T)/rs10246939(T) were inversely associated with PROP bitterness perception (OR=0.101, 95% CI 0.024–0.408). TAS1R2 rs9701796(G) and rs35874116(C) correlated respectively with preference for sweet foods (r=0.264, p=0.027) and sweet taste liking (r=0.286, p=0,016), aversion to bitter vegetables (r=-0.254, p=0,034) and PROP bitterness (r=-0.336, p=0,004). Additionally, ANKK1 rs1800497(A) was associated to liking of cured meat/meat sauces (r=0.243, p=0,042; r=0.313, p=0.008) and to quinine bitterness aversion (OR=0.301, 95% CI 0.088–0.998). Preference for fatty foods combined with reduced liking for bitter tastes -limiting consumption of bitter vegetables- may partly contribute to obesity susceptibility described in literature. Findings in our elderly cohort suggest genetic components in taste perception may remain predominant in food-liking despite age-related physiological decline. Variants in reward- and metabolism-related genes such as ANKK1 may further modulate taste liking and food preferences contributing to dietary patterns relevant to metabolic risk with possible implications for nutritional status and healthy aging, although further confirmation in a larger sample is needed to strengthen the associations.

POSTER PRESENTATION P06

Processing-driven modulation of intestinal response to pigmented wheat: implications for nutrigenomics and healthy aging

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Tuesday 1 September, 12.00–14.00 | Poster Session 1 | Session 2: Nutrigenomics of Health and Disease

ABSTRACT

Diet–gene interactions at the intestinal level play a central role in the regulation of inflammation and oxidative stress, key processes involved in chronic diseases and aging. Whole grains, particularly pigmented wheat varieties, are rich in bioactive phytochemicals such as anthocyanins and phenolic compounds; however, their biological effects are often limited by low bioaccessibility. Food bioprocessing may represent a key strategy to enhance the release of these compounds and modulate their downstream effects. In this study, pigmented wheat was subjected to different bioprocessing strategies, including germination (0–72 h), solid-state fermentation with Aspergillus awamori and Rhizopus oligosporus (0–72 h), and enzymatic treatments with Ultraflo XL and Viscozyme (0–16 h). The optimal conditions for each treatment, identified based on the antioxidant capacity, were selected for flour production and subsequent porridge preparation. Porridges were characterized for rheological properties and subjected to in vitro digestion to assess rapidly digestible starch (RDS) and slowly digestible starch (SDS), and phenolic bioaccessibility. Polyphenol profiles (HPLC-DAD) were characterized in flours, porridges, and digested samples. The bioaccessible fraction was applied to Caco-2 cells cultured on transwell inserts to investigate intestinal responses. Epithelial barrier integrity was evaluated by TEER, while the expression of genes involved in inflammatory and antioxidant pathways was assessed by RT-PCR. The basolateral compartment was analyzed by HPLC-DAD to assess the transport and potential absorption of phenolic compounds. Processing significantly enhanced antioxidant capacity, the highest values were observed after 72 h of fermentation (7.30 ± 0.27 mg TE/g), followed by germination (5.14 ± 0.56 mg TE/g), and Viscozyme treatment (5.42 ± 0.54 mg TE/g after 16 h), corresponding to an increased antioxidant potential of up to ~3–4-fold increases compared to untreated samples. Preliminary results indicate that processing modulates starch digestibility and porridge rheology, enhances polyphenol bioaccessibility, and induces beneficial intestinal responses, including improved epithelial barrier integrity and modulation of genes related to oxidative stress and inflammation. These findings highlight food processing as a key modulator of the nutrigenomic response to pigmented wheat, with potential implications for chronic disease prevention and healthy aging.

POSTER PRESENTATION P07

Genetic Modulation of Dietary Salt Preference in a Zambian Cohort

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Tuesday 1 September, 12.00–14.00 | Poster Session 1 | Session 2: Nutrigenomics of Health and Disease

ABSTRACT

Rising hypertension rates have contributed to cardiovascular diseases becoming the leading driver of non-communicable deaths in sub-Saharan Africa (SSA). High salt intake is a primary trigger for hypertension, particularly in individuals with salt-sensitive blood pressure. Dietary salt is modulated by taste perception, which is regulated by epithelial sodium channels and TRPV1 receptors, and taste preference. It is known that genetic variations can influence taste perception and dietary behaviour. So, understanding genetic modulation is crucial. Therefore, this study investigates the association between six single nucleotide polymorphisms (SNPs) and salt taste preference and sensitivity within an SSA population, which is underrepresented in research studies. Zambian adults (n = 276) were analysed (age 45.3 ± 13.8 years). Phenotypic data regarding salt taste preference and taste sensitivity thresholds were collected. Six SNPs (rs11064145, rs239345, rs3785368, rs8065080, rs4790522, rs7956915) of genes involved in salt taste transduction (SCNN1A, SCNN1B, TRPV1) were selected based on their association with taste perception and frequency across both European and African populations. Genetic-phenotype associations were evaluated using dominant and recessive genotypic models and p-values were adjusted using Bonferroni correction. Ordinal logistic regression revealed significant associations for salt taste preference. Specifically, the TRPV1 rs8065080 alternative allele was associated with an increased preference for higher salt concentrations ($p < 0.001$; OR= 3.58; 95% CI: 1.86 - 6.90). Contrastingly, the homozygous alternative variant of SCNN1A rs11064145 was associated with a preference for lower salt concentrations ($p = 0.018$; OR= 0.29; 95% CI: 0.13 – 0.66). No significant associations were found for salt sensitivity thresholds. Overall, this study shows that genetic variations may be associated with salt preference but not with salt taste sensitivity in Zambian adults. Specifically, the rs8065080 alternative allele is associated with higher salt preference, whereas rs11064145 with lower salt preference. Additional validations should be carried out in larger cohorts to better analyse gene-environment interplay.

POSTER PRESENTATION P08

Genetic background modulates individual responses to fruit and vegetable interventions: insights from the MiBlend randomized trial

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Tuesday 1 September, 12.00–14.00 | Poster Session 1 | Session 2: Nutrigenomics of Health and Disease

ABSTRACT

Diet is a major modifier of chronic disease risk; however, increasing evidence shows that individuals respond differently to the same dietary exposures. This heterogeneity, often reflected in “responder” and “non-responder” phenotypes, contributes to inconsistent outcomes in nutrition intervention studies. Genetic variation is a key determinant underlying this variability, as gene–diet interactions can substantially modify health outcomes. The MiBlend randomized controlled crossover trial was designed to investigate whether genetic polymorphisms contribute to differential responses to fruit and vegetable (F&V) interventions. In the MiBlend study, participants completed a two-week run-in phase with standardized low F&V intake (50 g/day), followed by two randomized two-week intervention periods (450 g/day F&Vs) separated by a one-week washout. Each participant received two out of seven distinct F&V blends, specifically designed to vary in phytochemical composition and complexity. Biological samples were collected pre- and post-intervention to assess biomarkers of oxidative stress, inflammation, and transcriptomic responses. Genotyping focused on polymorphisms in genes related to antioxidant defence, phase I/II metabolism, and inflammatory regulation. The intervention resulted in overall improvements in plasma antioxidant capacity, retinal microvasculature, and DNA protection; however, substantial inter-individual variability was observed. For example, reductions in DNA damage differed significantly across SNPs including XRCC1, GSTP1, SLC23A1, ZBED3, Glu298Asp, and APOC1. XRCC1 wildtype carriers showed the strongest protection after flavonoid-rich intake, while SLC23A1 wildtype individuals consistently exhibited greater reductions compared to variants. Microvascular improvements were greatest in GSTP1 wildtypes, particularly following polyphenol-rich blends, whereas GSTM1 and CAT1 genotypes showed differences only for certain specific blends. Changes in plasma antioxidant capacity also depended on genotype, with GSTT1, HNF1A, NQO1, and TCF7L2 variants showing larger or smaller increases depending on the intervention. Additionally, SLC23A1 variants were associated with higher vitamin C excretion, and APOC1 variants influenced circulating carotenoid levels, indicating differences in absorption and retention of these nutrients. These findings demonstrate that genetic background significantly influences the efficacy of different F&V interventions. In conclusion, the MiBlend study provides mechanistic evidence supporting personalized nutrition approaches and underscores the potential of genotype-informed dietary strategies for optimizing chronic disease prevention.

POSTER PRESENTATION P09

Comparative acute effects of caffeine-supplemented and decaffeinated coffee on immune function in healthy adults: A randomized double-blind crossover trial

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Tuesday 1 September, 12.00–14.00 | Poster Session 1 | Session 2: Nutrigenomics of Health and Disease

ABSTRACT

Coffee consumption has consistently been associated with a lower risk of chronic diseases, including type 2 diabetes and cardiovascular disease. These benefits may, at least in part, be mediated through immunomodulatory mechanisms. However, the contribution of caffeine and other coffee constituents to these effects remains insufficiently understood. In a randomized, double-blind, controlled crossover study, 12 healthy adults received three interventions: water (control), decaffeinated coffee, and caffeine-supplemented coffee. Blood samples were collected before and up to 24 h after each intervention. Plasma caffeine concentrations were quantified by UPLC-MS/MS, while cytokines (IL-1β, IL-2, IL-4, IL6, IL-10, IL-12p70, IL-17A, TNF, IFN-γ) and IL-1ra were measured using ultrasensitive immunoassays. The systemic immune-inflammation index (SII) was calculated. TAS2R38 and CYP1A2 genotypes were determined, and bitter taste perception was assessed using PROP and coffee sensory testing. Caffeine-supplemented coffee significantly reduced circulating IL-6, TNF, and IL-4 concentrations four hours after consumption compared with water. A trend toward increased IL-10 concentrations resulted in a significantly lower IL-6/IL-10 ratio, indicating a shift toward a less pro-inflammatory immune profile. Both decaffeinated and caffeine-supplemented coffee reduced the baseline-adjusted AUC of the systemic immune-inflammation index. Exploratory subgroup analyses suggested that individual bitter taste perception may influence immune responses, indicating a potential role of TAS2R-mediated signaling. Acute coffee consumption induces measurable immunomodulatory effects in healthy adults. While caffeine appears to contribute to cytokine suppression, decaffeinated coffee also improved systemic inflammatory markers, suggesting that multiple coffee-derived bioactive compounds participate in the anti-inflammatory response. These findings provide mechanistic support for the beneficial health effects associated with habitual coffee consumption.

POSTER PRESENTATION P10

A longitudinal multi-omics study of vitamin D-induced epigenetic memory in human PBMCs

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Tuesday 1 September, 12.00–14.00 | Poster Session 1 | Session 2: Nutrigenomics of Health and Disease

ABSTRACT

Vitamin D is a key immunomodulatory micronutrient regulating the transcriptional programming of immune cells, including peripheral blood mononuclear cells (PBMCs). Although the molecular effects of vitamin D signaling have been extensively studied, the epigenetic mechanisms underlying long-term memory following vitamin D exposure remain poorly understood. The primary aim of this study is to determine whether different vitamin D_3 supplementation regimens, such as a monthly bolus administration versus daily supplementation, induce epigenome- and transcriptome-wide changes in human PBMCs. We investigate inter-individual variation in these responses over time. PBMCs are collected from peripheral blood of 45 participants across five defined time points (months 0, 3, 12, 15, 24) during a two-year longitudinal study. Complete longitudinal datasets were obtained from 29 participants, while the remaining 16 contributed to partial time-course datasets. During the first winter season, participants received monthly bolus vitamin D_3 supplementation (1000 IU/kg for three months) followed during the second winter by daily supplementation (40 IU/kg for three months). Isolated PBMCs are cultured in vitro for 24 hours in the presence of either 10 nM 1,25-dihydroxyvitamin D_3 (1,25D) or ethanol as vehicle control. Samples from each experimental condition are subjected to RNA-sequencing (RNA-Seq) and Assay for Transposase-Accessible Chromatin using sequencing (ATAC-Seq), followed by downstream bioinformatic analyses. The controlled in vitro stimulation approach enables the identification of both immediate and persistent vitamin D-induced changes in gene expression and chromatin organization, including alterations in regulatory elements associated with epigenetic memory. We hypothesize that wintertime vitamin D_3 supplementation induces long-term epigenetic reprogramming in immune cells and that the magnitude and nature of these responses depend on the supplementation regimen. Furthermore, this study is expected to advance our understanding of vitamin D-mediated epigenetic memory mechanisms in the context of trained immunity.

POSTER PRESENTATION P11

Vitamin D Shapes Immune Cell Epigenetic Memory under Inflammatory Challenge: Implications for Precision Nutrition in Healthy Aging

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Tuesday 1 September, 12.00–14.00 | Poster Session 1 | Session 2: Nutrigenomics of Health and Disease

ABSTRACT

Aging is characterized by progressive dysregulation of immune function, often linked to chronic low-grade inflammation and impaired adaptive responses. Nutritional factors, such as vitamin D, play a critical role in modulating immune homeostasis; however, their mechanistic contribution to immune cell programming in the context of inflammatory challenges remains insufficiently understood. In this study, we investigated how the active form of vitamin D_3 ($1,25(OH)_2D_3$) interacts with bacterial-derived lipopolysaccharide (LPS) to reshape the epigenomic and transcriptomic profile of human monocytes, a key cell type in innate immunity and age-related inflammation. Using ATAC-seq and RNA-seq in THP-1 cells, we demonstrate that co-stimulation with vitamin D and LPS induces a highly distinct and synergistic regulatory program that cannot be explained by additive effects of either stimulus alone. Epigenomic profiling revealed that up to 81% of chromatin accessibility changes were unique to the combined treatment, indicating a profound reprogramming of immune regulatory regions. Motif enrichment analysis identified JUN/FOS (AP-1) transcription factors as central mediators of this response, suggesting a convergence of inflammatory and nuclear receptor signaling pathways. At the transcriptomic level, hundreds of genes displayed synergistic regulation, including a large subset not previously recognized as vitamin D targets. Functionally, these genes are predominantly associated with monocyte differentiation and T cell interaction rather than classical inflammatory pathways. These findings support a model in which inflammatory stimuli sensitize immune cells to nutritional signals, enabling vitamin D to reprogram immune responses through epigenetic mechanisms. In the context of aging, such adaptive plasticity may contribute to maintaining immune resilience while preventing maladaptive chronic inflammation. Our results provide a mechanistic basis supporting the integration of micronutrient status into precision nutrition strategies and highlight the potential of integrating multi-omics data as a foundation for future development of digital twin models of immune aging. This approach may help enable personalized nutrition strategies aimed at promoting healthy aging and mitigating age-associated inflammatory diseases.

POSTER PRESENTATION P12

Dosing Matters: Longitudinal Immune Cell Dynamics Reveal Regimen-Specific Effects of Vitamin D_3 Supplementation

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Tuesday 1 September, 12.00–14.00 | Poster Session 1 | Session 2: Nutrigenomics of Health and Disease

ABSTRACT

Vitamin D_3 is a key modulator of immune function and has been implicated in maintaining immune homeostasis and resilience during aging. However, how different supplementation strategies shape immune cell composition over time in vivo remains poorly defined, limiting the optimization of vitamin D interventions in precision nutrition. Here, we performed high-resolution longitudinal immune profiling in 28 healthy individuals from the VitDPAS cohort undergoing two distinct vitamin D_3 supplementation regimens: daily dosing and monthly bolus administration. Peripheral blood mononuclear cells (PBMCs) were collected at nine time points, enabling detailed temporal analysis of immune cell dynamics across supplementation phases. Multiparameter flow cytometry (FACS) was applied using a standardized antibody panel (CD45, CD3, CD4, CD8, CD14, CD16, CD19, CD56) to quantify major immune cell populations, including T cells, B cells, natural killer (NK) cells, and monocytes. This approach allowed robust and reproducible assessment of immune composition over time and between supplementation strategies. Longitudinal analysis revealed that vitamin D_3 supplementation induces dynamic, time-dependent remodeling of peripheral immune cell populations rather than static shifts. Notably, distinct temporal patterns emerged between supplementation regimens, indicating that dosing strategy is a critical determinant of immune modulation. Daily supplementation was associated with more stable and gradual immune cell adjustments, whereas bolus dosing induced more pronounced fluctuations in immune cell distributions over time. These findings demonstrate that vitamin D_3 does not exert uniform immunological effects but instead drives regimen-specific immune dynamics. Such temporal variability highlights the importance of considering dosing strategies when evaluating vitamin D interventions and underscores the limitations of single time-point assessments. By providing longitudinal, high-resolution insights into immune cell remodeling, this study establishes a framework for linking supplementation regimens to immune system behavior. These results support the development of precision nutrition strategies that tailor vitamin D dosing to individual immune profiles, with the goal of enhancing immune homeostasis and promoting healthy aging.

POSTER PRESENTATION P13

Vitamin D Reprograms Primary Human Monocyte Inflammatory Networks via Chromatin Remodeling: A Nutrigenomic Mechanism for Healthy Aging

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Tuesday 1 September, 12.00–14.00 | Poster Session 1 | Session 2: Nutrigenomics of Health and Disease

ABSTRACT

Chronic low-grade inflammation ("inflammaging") is a central driver of immune dysfunction and age-related disease. Vitamin D has emerged as a key nutritional modulator of immune responses; however, the molecular mechanisms underlying its context-dependent effects and inter-individual variability remain incompletely understood, limiting its application in precision nutrition for healthy aging. Here, we applied integrated multi-omics profiling to dissect how vitamin D reshapes inflammatory signaling in primary human monocytes. Cells were stimulated for 24 hours with lipopolysaccharide (LPS) or tumor necrosis factor (TNF), in the presence or absence of 1,25-dihydroxyvitamin D (1,25D). Matched RNA-seq and ATAC-seq datasets enabled the simultaneous resolution of transcriptional and chromatin accessibility changes, with cross-validation in THP-1 cells to assess model conservation. Inflammatory stimuli induced extensive transcriptional remodeling, which was further amplified under co-stimulation with vitamin D (LPS+1,25D: 2,902 DEGs; TNF+1,25D: 2,275 DEGs). Notably, vitamin D did not act as a general anti-inflammatory agent but instead selectively reprogrammed immune responses through bidirectional modulation. Protective immune functions were enhanced, including antiviral and immune surveillance pathways such as type I interferon signaling (e.g., OAS1, IFITM2) and antigen presentation. In contrast, pathways associated with metabolic and lipid-driven inflammation, including APOE, CD36, and glycolytic programs, were attenuated. Integration with chromatin accessibility data revealed coordinated remodeling of promoters and enhancers at key regulatory loci, including ADAP1, PLXDC2, and DUSP6, demonstrating that vitamin D exerts its effects through reconfiguration of gene regulatory architecture. Cross-model analysis confirmed context-dependent conservation of these responses, particularly under inflammatory conditions, highlighting robust yet stimulus-specific regulatory programs. Together, these findings redefine vitamin D as a selective immune response modulator that fine-tunes inflammatory networks via chromatin-mediated nutrigenomic mechanisms. This targeted reprogramming of immune function provides a mechanistic basis for leveraging vitamin D in precision nutrition strategies aimed at reducing inflammaging, enhancing immune resilience, and promoting healthy aging.

POSTER PRESENTATION P14

Vitamin D_3 Induces Divergent Epigenetic State Transitions in Human Immune Cells: Implications for Precision Nutrigenomics

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Tuesday 1 September, 12.00–14.00 | Poster Session 1 | Session 2: Nutrigenomics of Health and Disease

ABSTRACT

Vitamin D_3 is a key regulator of immune function and has been implicated in promoting immune resilience and healthy aging through epigenetic mechanisms. However, the extent to which individuals exhibit distinct epigenomic responses to supplementation in vivo remains poorly understood, limiting the translation of vitamin D interventions into precision nutrition strategies. Here, we investigated inter-individual variability in chromatin accessibility dynamics using ATAC-seq in peripheral blood mononuclear cells (PBMCs) from participants of the VitDPAS cohort following high-dose vitamin D3 supplementation (1,000 IU/kg body weight monthly for three months). Samples were collected at baseline (day 0) and 24 hours after supplementation (day 1), enabling high-resolution mapping of early epigenetic responses. Chromatin accessibility profiles were integrated with ChromHMM-derived epigenetic state annotations to characterize functional regulatory changes. Unsupervised clustering of ATAC-seq signal changes revealed three distinct response phenotypes: Positive (increased chromatin accessibility), Negative (decreased accessibility), and Moderate (minimal change). These groups exhibited fundamentally different epigenetic remodeling trajectories. In the Positive group, chromatin accessibility shifted toward enhancer regions, accompanied by a relative reduction in active transcription start site (TSS) annotations, suggesting a redistribution of regulatory activity toward distal control elements. In contrast, the Negative group displayed the opposite pattern, with reduced enhancer representation and stable or increased TSS-associated accessibility. These differences were statistically robust following FDR correction and were most pronounced for enhancer and active TSS states. These findings demonstrate that vitamin D_3 supplementation induces rapid, individual-specific epigenetic state transitions rather than uniform regulatory responses. The observed divergence in enhancer versus promoter dynamics suggests that vitamin D-mediated immune regulation operates through personalized rewiring of gene regulatory networks. Importantly, this study provides in vivo evidence that inter-individual variability in nutritional epigenomics is a defining feature of immune modulation. By capturing early chromatin accessibility changes, PBMC-based ATAC-seq emerges as a powerful and minimally invasive tool to stratify individuals according to molecular responsiveness. Together, these results establish a mechanistic basis for integrating epigenomic biomarkers into precision nutrition frameworks and digital twin models, enabling targeted vitamin D interventions aimed at optimizing immune function and promoting healthy aging.

POSTER PRESENTATION P15

Multi-omics phenotyping of lactose intolerance traits in a pilot randomised placebo-controlled trial

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Tuesday 1 September, 12.00–14.00 | Poster Session 1 | Session 3: Microbiome Dynamics in Healthy Aging

ABSTRACT

Food intolerances are a common cause of gastrointestinal (GI) symptoms, typically diagnosed by symptom assessment, hydrogen breath tests and dietary exclusion. However, these approaches do not capture the interplay between host physiology, gut microbial metabolism and dietary factors underlying inter-individual variability in food responses. Breath metabolomics is a non-invasive approach that can be used to investigate host-microbial metabolic interactions, with potential to identify biomarkers of food intolerance. The Lactobreath study was designed as a randomised placebo-controlled challenge study investigating lactose intolerance (LI) by integrating breath metabolomics with complementary multi-omics and multimodal phenotyping. A total of 112 participants comprising symptomatic lactose malabsorbers (LM-S = 64), asymptomatic lactose malabsorbers (LM-A, n = 24) and lactose absorbers (controls; n = 24) received a lactose challenge (25g) or glucose placebo (13g). On-line exhaled breath analysis was performed over 6 h with concurrent urine sampling, GI symptom assessments, hydrogen breath testing, bowel sound monitoring (wearables) and intestinal gas measurement (ingestible gas-sensors). The breath metabolome was analysed by secondary electrospray ionisation high resolution mass spectrometry, while urine metabolites were characterised using targeted and untargeted GC-MS. Baseline gut microbiome composition (metagenomics), usual dietary intake (4-day food records) and selected genetic polymorphisms were assessed for integrated phenotyping. Exploratory multivariate analysis revealed distinct breath metabolome profiles distinguishing LM-S from controls. Selected urinary metabolites also showed potential to discriminate lactose malabsorbers (LM-S/LM-A) from controls. Baseline differences in dietary patterns were observed between the groups, while MaAsLin3 analysis identified microbial features associated with the LI phenotypes. These findings highlight the potential of breath metabolomics within an integrated multi-omics framework to characterise lactose responses and inform personalised dietary approaches for food intolerances.

POSTER PRESENTATION P16

Enzymatic activity of the human gut microbiota and its role in (poly)phenol bioavailability and metabolism

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Tuesday 1 September, 12.00–14.00 | Poster Session 1 | Session 3: Microbiome Dynamics in Healthy Aging

ABSTRACT

Dietary (poly)phenols have been associated with cardiovascular health benefits; however, their bioavailability and biological effects depend largely on gut microbial biotransformations. Given the substantial inter-individual differences in microbial enzymatic activity, investigating this variability may help clarify differences in (poly)phenol metabolism. This study aims to quantify gut microbial enzyme activities involved in (poly)phenol catabolism and to explore their associations with host characteristics, dietary patterns, urinary metabolites, aggregate phenolic metabotypes, and gut microbial communities. Baseline faecal samples were obtained from 77 healthy adults enrolled in a standardized oral (poly)phenol challenge test (OPCT), involving acute supplementation with multiple (poly)phenol classes. Activities of five gut microbial glycosidases (β-glucosidase, β-galactosidase, β-glucuronidase, α-glucosidase and α-galactosidase) were quantified spectrophotometrically at 405 nm using chromogenic p-nitrophenyl-conjugated substrate analogs and normalized to faecal protein content. Associations with host-related variables (BMI, age, sex), dietary patterns, urinary metabolites, phenolic metabotypes, and microbial taxa were evaluated. Gut microbial glycosidase activities showed marked inter-individual variability. Selected glycosidase activities were associated with host-related factors (age and sex), specific nutrient intakes, and dietary pattern scores. Exploratory integration with urinary phenolic metabolites and gut microbial taxa suggested potential links between microbial enzyme function. Conversely, individual glycosidase activities were not associated with the aggregate metabotype classification. Host characteristics and dietary habits are key drivers shaping gut microbial enzymatic activities involved in (poly)phenol metabolism. Understanding these factors is crucial, as they directly modulate inter-individual variability in (poly)phenol bioconversion and influence host–microbiota metabolic interplay.

POSTER PRESENTATION P17

Characterisation of circulating bioactive compounds and gut microbiota-host interactions in lung cancer patients using LC-MS metabolomics

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Tuesday 1 September, 12.00–14.00 | Poster Session 1 | Session 3: Microbiome Dynamics in Healthy Aging

ABSTRACT

Lung cancer (LC) is one of the most prevalent malignant tumours, with non-small cell lung cancer (NSCLC) accounting for approximately 85% of all cases. The interplay among diet, host, and microbiota results in diet-derived host-microbiota co-metabolites capable of influencing the tumour microenvironment, comprising cancer metabolism and growth. This can be investigated through metabolomics, which allows a comprehensive analysis of metabolites offering valuable insights into dynamic diet-induced metabolic processes. In this study, urine and plasma samples collected from LC patients enrolled in the LISTENING study will be analysed using ultra-high-performance liquid chromatography coupled with tandem mass spectrometry (UHPLC-MS/MS). Targeted metabolomic analyses will be performed to assess the circulating levels of food bioactive metabolites and to investigate inter-individual variability in the metabolism of key phenolic metabolites. This will also provide a better understanding of the impact of dietary choices on the production of metabolites related to the diet-microbiome-host interplay, like short-chain fatty acids, secondary bile acids, trimethylamine N-oxide, and indole derivatives. To date, 29 patients have been enrolled, including 2 diagnosed with SCLC and 27 with NSCLC, all at stage IV disease. Co-morbidities were reported in 79% of participants, most commonly arterial hypertension and dyslipidaemia. Among food-derived bioactive metabolites, a comprehensive mass spectrometry method was developed for the analysis of (poly)phenols. The method comprises 349 compounds, encompassing native parent compounds, gut microbiota-derived catabolites, and phase II conjugated metabolites, including methylated, sulfated, and glucuronidated derivatives. Of the 22 subclasses, those associated with considerable inter-individual variability will be further investigated, including phenyl-γ-valerolactones, urolithins, isoflavone-derived metabolites and enterolignans. By integrating diet-derived metabolites, biomarker discovery, inflammation, and microbiome–host interactions, this study supports the development of precision nutrition strategies to effectively prevent and manage LC co-morbidities.

POSTER PRESENTATION P18

Dietary Fibre, Inflammageing, and Vitamin D-Related Gene Expression in Skin (INGUTSKIN)

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Tuesday 1 September, 12.00–14.00 | Poster Session 1 | Session 3: Microbiome Dynamics in Healthy Aging

ABSTRACT

Ageing is associated with chronic low-grade inflammation ('inflammageing') that disrupts skin homeostasis and increases susceptibility to inflammatory conditions such as psoriasis. This disorder involves altered gene expression, including pathways linked to vitamin D metabolism, highlighting nutrigenomic interactions between diet, inflammation and skin function. Vitamin D contributes to keratinocyte differentiation and immune regulation. Prebiotics can modulate the gut microbiota and host inflammatory status. This trial examined whether inulin-type beta-fructans (ITFs) influence vitamin D-related gene expression in mild psoriasis via the gut–skin axis. Adults with mild psoriasis were randomised to ITFs (n = 29) or maltodextrin placebo (n = 27) at 15 g/day for 8 weeks (ClinicalTrials.gov: NCT05971992). Punch biopsies from lesional (L) and non-lesional (NL) skin were collected. RNA was extracted, reverse transcribed, and CYP2R1, CYP27B1 and VDR expression quantified by qPCR. At baseline, CYP27B1 expression was approximately 2-fold higher in L relative to NL skin ($\Delta\log_{10}$-EMM: 0.32, 95% CI [0.18, 0.46], q = 0.0004), indicating inflammation-associated dysregulation. After the intervention period, CYP2R1 and CYP27B1 expression remained elevated in L skin within the ITF group (0.10, 95% CI [0.03, 0.17], q = 0.021; 0.32, 95% CI [0.11, 0.53], q = 0.019). In the placebo group, a significant increase over time was observed for CYP2R1 and VDR expression in NL skin. No significant Group × Timepoint interaction was identified for any of the analysed genes, including after adjustment for sex, age and BMI. Expression levels across CYP2R1, CYP27B1 and VDR were positively correlated (ρ = 0.34–0.80, q ≤ 0.043), suggesting coordinated regulation. ITF supplementation did not elicit effects distinct from placebo; however, vitamin D-related genes exhibited coordinated expression over time, with consistent positive correlations between CYP2R1, CYP27B1 and VDR. These findings support interconnected nutrigenomic pathways linking diet, microbiota and vitamin D metabolism in skin biology, with relevance to inflammageing. Further work is needed to determine how targeted dietary strategies may help mitigate age-related inflammation and support skin health.

POSTER PRESENTATION P19

The impact of a personalized combined lifestyle intervention on microbiome and metabolic, hepatic and inflammatory resilience in overweight and obese adults

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Tuesday 1 September, 12.00–14.00 | Poster Session 1 | Session 3: Microbiome Dynamics in Healthy Aging

ABSTRACT

Overweight and obesity are closely associated with insulin resistance and type 2 diabetes (T2D). Since 2019, the cost-effective SLIMMER combined lifestyle intervention (CLI) has been reimbursed in the Netherlands for promoting moderate weight loss, improving quality of life, and reducing the risk of T2D. A personalized version of CLI (pCLI) has demonstrated even greater effectiveness in enhancing compliance and achieving body weight reduction. Microbiome diversity and resilience scores are important health indicators, with higher diversity and lower resilience scores reflecting better health status. This study assessed the pCLI's impact on metabolic, hepatic, and inflammatory resilience, as well as microbiome profiles. A retrospective analysis was conducted involving 33 overweight or obese adults at risk for cardiometabolic disease. Before and six months after starting the pCLI, participants underwent comprehensive body composition assessments, fecal microbiome and short-chain fatty acid (SCFA) profiling and the PhenFlex challenge test (PFT), a standardized mixed-meal tolerance test. During the PFT, multiple blood samples were collected to evaluate metabolic, hepatic, and inflammatory resilience dynamically. The pCLI intervention did not significantly change fecal microbiome diversity or SCFA composition. However, it led to significant reductions in multiple body composition parameters including body fat percentage and visceral fat, and improvements in several fasting plasma parameters. Postprandial plasma response analyses showed significant changes in NEFA, IL-6, and sICAM-1 levels. Compared to baseline, metabolic and hepatic resilience improved at follow-up and were correlated with body fat percentage The pCLI significantly reduced multiple anthropometric measurements and enhanced metabolic and hepatic resilience. This highlights the value of personalized lifestyle interventions for improving metabolic and hepatic resilience.

POSTER PRESENTATION P20

Relationships between one-carbon biomarkers and metabolic syndrome in Polish postmenopausal women: A case-control study

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Tuesday 1 September, 12.00–14.00 | Poster Session 1 | Session 4: Aging Clocks in Metabolic Disorders

ABSTRACT

Postmenopausal women are at increased risk of metabolic syndrome (MetS), characterized by abdominal obesity, dyslipidemia, hyperglycemia, and hypertension. One-carbon metabolism (OCM) may contribute to MetS in several ways. However, the relationships between OCM biomarkers—including choline, betaine, total homocysteine (tHcy), trimethylamine (TMA), and trimethylamine N-oxide (TMAO) —and MetS remain unclear. The main aim of this study was to compare concentrations of OCM biomarkers in postmenopausal women with and without MetS. A total of 121 nonsmoking postmenopausal women (48 with MetS, 73 without MetS) were enrolled. MetS was diagnosed according to the International Diabetes Federation (2005) criteria. Fasting serum triglycerides, glucose and high-density lipoprotein cholesterol (HDL-C) levels were measured using a biochemical analyzer. Waist circumference (WC) and blood pressure were measured using standard methods. Body mass index (BMI) were calculated using the standard formula. Dietary intake was assessed using 3-day food records, while physical activity with the International Physical Activity Questionnaire. Plasma OCM biomarkers were measured using isotope dilution analysis or HPLC. Student's t-test and Spearman's rank correlation analyses were performed using Statistica software, with $p < 0.05$ considered significant. Women with MetS had a higher BMI than those without MetS (31.0 ± 5.0 vs. 26.5 ± 4.1 kg/m^2, $p < 0.001$). No significant differences were observed between the groups neither in plasma concentrations of choline, betaine, TMA, TMAO, tHcy or other OCM biomarkers, nor in dietary choline and betaine intake. Choline intake was inversely correlated with plasma tHcy and positively associated with TMAO in the whole group. Plasma choline and betaine concentrations were positively correlated regardless of MetS status. Among individual MetS components, fasting glucose was positively associated with TMAO only in women without MetS. Weak associations were also found between tHcy and WC or systolic blood pressure in the group of women without MetS,. In conclusion, circulating OCM biomarkers did not differ between postmenopausal women with and without MetS. However, modest associations between dietary choline and betaine, OCM biomarkers, and individual MetS components suggest that OCM may influence specific metabolic pathways rather than MetS itself.

POSTER PRESENTATION P21

No effect of betaine supplementation on the expression of genes involved in lipid metabolism in subcutaneous adipose tissue of premenopausal women

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Wednesday 2 September, 12.00–13.45 | Poster Session 2 | Session 4: Aging Clocks in Metabolic Disorders

ABSTRACT

Betaine has been shown to affect numerous processes related to lipid metabolism in animal and in vitro studies, but evidence from human studies remains limited and inconsistent. Therefore the aim of the present study was to determine the effects of eight weeks of supplementation with 3 g/day betaine on lipid metabolism in women with increased body weight. The study was designed as a randomized, placebo-controlled, double-blind trial. Females aged 18-45 years, pre-menopausal, BMI ≥ 25kg/m2 and waist circumference ≥ 80 cm were randomly assigned to one of two groups supplemented with betaine (BET, 3g/day) or placebo (PLA) for eight weeks (Clinical Trials: NCT06344377). A Bod Pod (Cosmed, Italy) was used to analyze body mass and body composition. Plasma concentrations of total cholesterol, HDL cholesterol, LDL cholesterol, glucose, and activities of ALT, AST, and GGTP were determined using an automated analyzer Konelab 20i. Samples of subcutaneous adipose tissue for gene expression analysis were collected by needle biopsies. The selected genes included master regulators of lipid metabolism such as peroxisome proliferator activated receptor gamma (PPARG), sterol regulatory element binding transcription factor 1 and 2 (SREBF1, SREBF2), nuclear receptor subfamily 1 group H member 3 (NR1H3/LXRA), and other genes involved in lipid metabolism: a gene encoding transporter protein ATP binding cassette subfamily A member 1 (ABCA1), a gene with the biggest effect on body mass - fat mass and obesity associated (FTO). Real time reactions were performed with TaqMan Gene Expression Assays. Relative transcript quantification was performed using the 2-ΔΔCT method. Fifty three women were randomized and forty two completed the study After the intervention there were no significant group by time interaction effects on body composition or biochemical parameters. Similarly, there was no interaction effects on the transcription level of the analyzed genes. In conclusion, a mid-term supplementation with 3 g betaine/day does not appear to significantly affect lipid metabolism in premenopausal women.

POSTER PRESENTATION P22

Dietary Fiber Intake Is Associated with Transcriptional Activity of Lipid Metabolism Regulators in Adipose Tissue of Women with Overweight and Obesity

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Wednesday 2 September, 12.00–13.45 | Poster Session 2 | Session 4: Aging Clocks in Metabolic Disorders

ABSTRACT

Previous studies suggest that dietary fiber may influence adipose tissue metabolism and obesity-related pathways. This study aimed to analyze the association between dietary fiber intake and gene expression in adipose tissue of women with excessive body mass. The study included 58 premenopausal Polish women aged 18–45 years with waist circumference exceeding 80 cm. Dietary intake was assessed using a 3-day food record and analyzed with Dieta 6.0 software. Subcutaneous adipose tissue samples were collected via needle biopsy under local anesthesia. Expression levels of sterol regulatory element-binding transcription factor 1 (SREBF1) and 2 (SREBF2), nuclear receptor subfamily 1 group h member 3 (NR1H3), and peroxisome proliferator-activated receptor gamma (PPARG) were quantified using RT-qPCR with TaqMan chemistry. ACTB and LPR10 served as reference genes, and relative expression levels were calculated using the comparative Ct (ΔΔCt) method. For subgroup analysis, participants were divided according to the median dietary fiber intake. Associations between fiber intake and gene expression were assessed using multivariate linear regression adjusted for body mass index (BMI) and total energy intake. The study was approved by the Bioethics Committee of Poznan University of Medical Sciences (no. 92/24). Mean BMI of the study group was 31.3 ± 4.45 kg/m², mean total fiber intake was 18.0 ± 7.21 g/day, and median fiber intake was 17.2 g/day. In multivariate regression analyses, higher dietary fiber intake was associated with higher expression of SREBF2 ($\beta = -0.42$, $p < 0.05$), PPARG ($\beta = -0.38$, $p < 0.05$), and NR1H3 ($\beta = -0.40$, $p < 0.05$). No significant associations were observed for SREBF1. In conclusion, higher dietary fiber intake was associated with altered expression of genes involved in regulation of lipid metabolism and adipose tissue regulation among women with overweight and obesity.

POSTER PRESENTATION P23

Betaine supplementation modulates mitochondrial and redox-related adaptations in premenopausal women with overweight: A randomized controlled trial

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Wednesday 2 September, 12.00–13.45 | Poster Session 2 | Session 4: Aging Clocks in Metabolic Disorders

ABSTRACT

The aim of this study was to investigate the effects of betaine supplementation on sex hormones, as well as mitochondrial- and redox-associated parameters, in young women with excessive body weight. We hypothesized that betaine enhances mitochondrial enzyme activity and improves antioxidant defense, and may also affect sex hormone concentrations, as previously demonstrated in men. In this randomized controlled trial (https://clinicaltrials.gov/study/NCT06344377), premenopausal females with overweight or obesity were assigned to receive betaine supplementation or a control condition for eight weeks. Mitochondrial enzyme content was assessed in peripheral blood mononuclear cells (PBMCs), including citrate synthase (CS) and cytochrome c oxidase (COX) via ELISA method. Redox-related parameters were evaluated in plasma via colorimetric method; these included the activities of catalase (CAT), superoxide dismutase (SOD), glutathione peroxidase (GPx), and oxidized glutathione (GSSG). Hormones concentrations were measured via ELISA method and included plasma testosterone, cortisol, estradiol, sex hormones binding protein (SHBG), dehydroepiandrosterone sulphate (DHEA-S) and salivary testosterone. Preintervention and postintervention measurements were compared between the groups. Betaine supplementation resulted in a significant increase in CS content in PBMCs as compared with the control condition ($p = 0.043$), while no changes were observed in COX ($p = 0.326$). In plasma, CAT activity increased significantly following betaine supplementation ($p = 0.003$), whereas SOD ($p = 0.825$), GPx ($p = 0.359$), and GSSG ($p = 0.693$) levels remained unchanged. No significant changes were observed in the control group. There were no significant changes in hormone concentrations. Betaine supplementation may induce selective adaptations in mitochondrial and antioxidant systems in young women with excessive body weight, as reflected by increased CS content and CAT activity in PBMCs, but it does not appear to affect sex hormone concentrations.

POSTER PRESENTATION P24

Associations of Habitual Coffee Intake with Testosterone and Cardiometabolic Markers: the Northern Finland Birth Cohort 1966 Study

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Wednesday 2 September, 12.00–13.45 | Poster Session 2 | Session 4: Aging Clocks in Metabolic Disorders

ABSTRACT

Coffee is a widely consumed source of bioactive compounds globally, yet its associations with circulating metabolites, sex hormones, and cardiometabolic markers remain incompletely characterised. We investigated these associations in a large population-based cohort, with particular attention to sex-specific hormonal profiles and metabolomic signatures. Cross-sectional data from 2,264 participants (47% men) of the Northern Finland Birth Cohort 1966 (NFBC1966) at age 46 were analysed using sex-stratified Spearman correlations and multivariable linear regression models adjusted for BMI, educational attainment, smoking, physical activity, and alcohol intake. Higher coffee intake groups showed lower total and visceral fat and higher skeletal muscle mass, while BMI was comparable across groups. Inverse correlations were observed between coffee intake and circulating branched-chain amino acids (BCAAs) in both sexes. In men, higher coffee intake was correlated with a more favourable glucose–insulin profile and a distinct hormonal pattern: multivariable-adjusted models showed positive associations with total testosterone (β = +0.29 nmol/L per cup/day), bioavailable testosterone, and sex hormone–binding globulin (SHBG), alongside inverse associations with free testosterone and the free androgen index (FAI). In women, hormonal associations were more limited, with positive associations for SHBG and inverse associations for free testosterone and FAI. Higher habitual coffee intake groups showed a leaner body composition, correlated inversely with circulating BCAAs in both sexes, and were independently associated with a sex-specific hormonal profile that may be metabolically relevant, particularly in men. Future longitudinal and intervention studies are needed to establish causality and identify the coffee-derived bioactive compounds involved.

POSTER PRESENTATION P25

REV-ERB Signaling Modulates the Circadian Regulatory Axis for Glucagon-Like Peptide-1 Secretion in Enteroendocrine L-Cells

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Wednesday 2 September, 12.00–13.45 | Poster Session 2 | Session 4: Aging Clocks in Metabolic Disorders

ABSTRACT

Circadian rhythm regulates enteroendocrine function and plays a significant role in metabolic homeostasis. Enteroendocrine cells of the GI tract possess intrinsic clock machinery, contributing to the physiological rhythm of GLP-1 secretion. Enteroendocrine cells possess an intrinsic molecular clock, but the contribution of REV-ERB signaling to the temporal regulation of GLP-1 secretion remains poorly defined. We hypothesized whether REV-ERB activation alters clock-gene dynamics and the physiological secretory response to a nutrient stimulus in synchronized STC-1 cells. STC-1 cells were synchronized with forskolin (20 µM) and treated with vehicle or the REV-ERB agonist SR12418 (1 µM). Bmal1 and Rev-erbα gene expression was assessed at 3-h intervals from 0 to 24 h post- synchronization. Active GLP-1 secretion was subsequently evaluated during 2-h stimulation periods with α-linolenic acid (αLA) corresponding to 6–8 h and 18–20 h, under vehicle, αLA, SR12418, or SR12418+αLA conditions (n=3 independent experiments). Bmal1 expression showed a significant time effect ($p<0.0001$) and a significant time×SR12418 agonist interaction ($p=0.0032$), indicating that REV-ERB activation altered the temporal organization of Bmal1 expression. 24-hour cosinor analysis confirmed significant rhythmicity in both vehicle ($p=0.0018$) and SR12418-treated cells ($p=0.0312$), with a shift in fitted phase from approximately 7.4 h to 4.9 h. Rev-erbα expression also varied significantly with time ($p<0.0001$), but a significant 24-h cosinor rhythm was not detected in either condition, and SR12418 did not alter overall Rev-erbα expression. In the secretion experiments, αLA had a significant overall effect on active GLP-1 secretion ($p=0.0054$). The largest secretory response occurred in SR12418+αLA-treated cells at 8 h and was markedly lower at 20 h. The time×SR12418×αLA interaction showed a trend ($p=0.0719$), although it did not reach statistical significance with the current sample size. Together, these findings demonstrate that REV-ERB activation modifies circadian clock-gene dynamics in enteroendocrine cells and identify temporal regulation of nutrient-stimulated GLP-1 secretion, with the strongest response to SR12418 and αLA occurring during the early post-synchronization phase.

POSTER PRESENTATION P26

Lifestyle-behavior factors affect obesity and T2DM genetic predisposition risk

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Wednesday 2 September, 12.00–13.45 | Poster Session 2 | Session 5: Precision Nutrition and Digital Twins in Health and Disease

ABSTRACT

Obesity and type 2 diabetes mellitus (T2DM) arise from complex interactions between genetic susceptibility and modifiable lifestyle and behavioral factors. Across several sequential and complementary studies conducted in Israeli adult cohorts, several obesity-related genetic variants and their environmental interactions were identified as significant determinants of metabolic risk. FTO rs9939609 was associated with significant elevated obesity risk. inactive rs9939609 risk-allele carriers had higher obesity risk compared to their active counterparts (OR=2.54 and OR=3.7, with 3.1 and 3.5 BMI increment for heterozygotes and homozygotes, respectively; n=1720, p<0.001). Sugar-sweetened-beverages (SSB) consumption (≥1 serving/day) significantly raised obesity risk, and wine consumption (1−3 drinks/weekly) significantly lowered obesity risk for risk-allele carriers (OR=1.54, p=0.028 and OR=0.61, p<0.001, respectively). SH2B1 rs7498665 polymorphism was significantly associated with obesity, where carriers of the risk genotype showed increased odds across genetic models (OR 1.24-2; n=3,070). Physical activity (PA) and moderate wine consumption (1-3 drinks/week) reduced obesity risk (OR=0.35 and 0.71, respectively), whereas high stress levels, ≥median eating-habits-score and fasting glucose (≥90 mg/dL) increased risk up to OR=5.82 in homozygotes. TMEM18 rs939583 and TMEM18 rs1879523 were associated with increased obesity risk (O=1.35 and OR=1.66; n=3089, p<0.01), while PA and moderate wine consumption attenuated this genetic susceptibility (OR = 0.82 and OR=0.74, p<0.001). The MC4R rs17782313 variant was associated with increased obesity susceptibility (recessive OR=1.38; n=5785, p<0.005), particularly in females (OR=1.41; p=0.01). MC4R rs17782313 significantly interacted with several eating behaviors to enhance the risk of obesity. Extending these findings, FTO rs9939609 polymorphism interacted with behavioral factors affecting T2DM risk, where longer daily eating windows (OR=1.029), later last-meal timing (OR=1.066), prolonged sleeping windows (OR=1.137) and poorer sleeping quality (OR=1.185) were associated with increased T2DM susceptibility (n=12,254). BDNF rs925946 carriers had higher risk for gestational-diabetes-mellitus (recessive model, OR=2.05, p=0.03). Finally, a polygenic obesity risk score (GRSBMI, n=5,824) demonstrated that each unit increase in GRSBMI was associated with a 3.06 kg/m² increase in BMI (p≤0.0001), with unhealthy eating-habits-score and SSB intake amplifying genetic predisposition. Altogether, these findings demonstrate that obesity risk results from additive genetic effects that are substantially modified by lifestyle and behavioral exposures, highlighting the potential of community precision-nutrition approaches for prevention and management.

POSTER PRESENTATION P27

Precision nutrition interventions in cancer management: a scoping review of emerging approaches

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Wednesday 2 September, 12.00–13.45 | Poster Session 2 | Session 5: Precision Nutrition and Digital Twins in Health and Disease

ABSTRACT

Cancer is a major global health challenge, with a significant impact on mortality worldwide. Beyond its direct clinical burden, both the disease and its treatment can impair nutritional status, thereby affecting quality of life, treatment tolerance, and clinical outcomes. In this context, precision nutrition represents a promising strategy to address these issues. This scoping review aims to examine current evidence on the role of precision nutrition intervention in cancer management. A scoping review was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses Extension for Scoping Reviews (PRISMA-ScR) guidelines. The PCC framework was used to define the research question and develop the search string: population (patients with cancer of any age, sex, type and disease stage), concept (precision nutrition interventions), and context (any healthcare or care setting, with no geographical limitations). Records were identified through searches in the Scopus, Web of Science, Embase, and PubMed electronic databases. A total of 3613 records were identified, of which 5 studies were included after screening (3 articles, and 2 study protocols). Among the included articles, one was a retrospective cohort study in patients with oesophageal cancer, reporting improved postoperative outcomes following personalised parenteral nutrition based on metabolic profiling, individualized energy intake, protein tailored to catabolic state and targeted immunonutrients. The remaining studies were case reports (advanced prostate cancer and locally advanced rectal cancer), suggesting potential benefit of personalised nutrition guided by nutrigenomics within a multidisciplinary framework. The identified protocols included a randomized phase II trial in breast cancer survivors comparing a Mediterranean diet with a personalised intervention based on postprandial glycaemic response, blood parameters, and gut microbiome composition, and a multicentre real-world study involving patients with malignant tumour receiving personalised dietary advice based on their genomics and gut microbiota-derived metabolomics profiles. Due to the limited number of published studies, the search was extended to registered trials in ClinicalTrials.gov and WHO International Clinical Trials Registry Platform (ICTRP), identifying five additional ongoing or registered trials. Current evidence on precision nutrition interventions in oncology is scarce and heterogeneous. Although preliminary findings are promising, further well-designed clinical trials are needed to confirm their effectiveness.

POSTER PRESENTATION P28

Efficacy of Vitamin D Supplementation in Mitigating Aromatase Inhibitor-Induced Discontinuation in Breast Cancer Patients: A Systematic Review and Meta-Analysis Protocol

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Wednesday 2 September, 12.00–13.45 | Poster Session 2 | Session 5: Precision Nutrition and Digital Twins in Health and Disease

ABSTRACT

Aromatase inhibitors (AIs) are the cornerstone of adjuvant endocrine therapy for hormone receptor-positive breast cancer survivors. However, AI-induced musculoskeletal symptoms (AIMSS) often lead to early treatment discontinuation in up to 30% of patients, negatively impacting survival outcomes. Vitamin D is biologically vital for bone homeostasis and inflammatory modulation. Although widely used to alleviate AIMSS, its specific impact on reducing definitive clinical treatment discontinuation has not been systematically synthesized. This ongoing meta-analysis aims to evaluate whether vitamin D supplementation significantly improves AI adherence in randomized controlled trials (RCTs). A systematic search was initiated on March 26, 2026, across electronic databases including PubMed, Scopus, Web of Science, Google Scholar, Cochrane Library, Embase and ClinicalTrials.gov, from inception to the present, using related keywords such as vitamin D, breast cancer, aromatase inhibitors and AIMSS. We include RCTs comparing vitamin D supplementation (alone or with calcium) versus placebo or standard care in breast cancer patients receiving AIs. Non-randomized trials, observational studies, animal studies, case reports, and grey literature (editorials, conference abstracts) will be excluded. The primary outcome to be extracted is the rate of early AI discontinuation due to the presence of AIMSS. Pooled Relative Risks (RRs) and 95% Confidence Intervals (CIs) will be calculated using a random-effects model. Heterogeneity will be assessed via the I2 statistic. The systematic search strategy and study selection criteria have been finalized. Preliminary pilot screening confirms the availability of eligible RCTs reporting data on treatment discontinuation and safety drop-outs within their CONSORT diagrams or adverse event tables. Data extraction, risk of bias assessment (using the Cochrane RoB 2 tool), and quantitative statistical pooling are currently underway. The final quantitative results and forest plots will be fully presented at the conference. This systematic review and meta-analysis will provide a rigorous, evidence-based synthesis of the role of vitamin D in maintaining endocrine therapy adherence. By clarifying its impact on treatment persistence, this study will contribute to precision nutrition strategies aimed at optimizing long-term oncological outcomes in breast cancer survivors.

POSTER PRESENTATION P29

Reanalysis of Whole Exome Sequencing Data for Actionable Nutrigenetic Variants in Children with Autism Spectrum Disorder: A Pilot Study

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Wednesday 2 September, 12.00–13.45 | Poster Session 2 | Session 5: Precision Nutrition and Digital Twins in Health and Disease

ABSTRACT

Children with autism spectrum disorder (ASD) frequently undergo whole exome sequencing (WES) for diagnostic purposes. These data may contain actionable information beyond the primary diagnostic indication, including variants in genes relevant to micronutrient metabolism. Genotype-guided nutritional supplementation is an emerging concept in precision nutrition. To evaluate whether reanalysis of existing WES data can identify nutrigenetic variants informing personalized nutritional management in children with ASD. WES data (GRCh37) from 20 children with ASD were reanalyzed targeting 25 genes across six nutrient metabolism pathways (folate/methylation, vitamin B_{12}, vitamin D, vitamin A, antioxidant defense, neurotransmitter metabolism). Variant rsIDs were extracted from VCF files using BCFtools and annotated via GeneBe (ACMG classification, ClinVar, gnomAD frequencies). Common nutrigenetic variants (AF >1%) were interpreted based on published functional evidence rather than ACMG pathogenicity criteria. Variant frequencies were compared to gnomAD reference populations. All 20 patients carried multiple functional nutrigenetic variants (median 13, range 9–18). The folate/methylation pathway was universally affected (100% with ≥3 variants). Carrier rates for several variants exceeded gnomAD-derived expected frequencies, notably VDR rs2228570/FokI (95% vs. ~58%), BCO1 rs12934922 (95% vs. ~64%), and TCN2 rs1801198 (90% vs. ~50%). Nutrigenetic-focused reanalysis additionally revealed two rare pathogenic variants not reported in the original diagnostic setting: CBS rs121964964 (1/20, associated with classic homocystinuria, OMIM #236200) and CYP24A1 rs114368325 (1/20, infantile hypercalcemia type 1). Both were heterozygous carriers, not expected to manifest full disease phenotype, but with potential modifying effects on nutrient metabolism and direct implications for B_6 and vitamin D dose optimization. Systematic nutrigenetic reanalysis of existing WES data is feasible and identifies both common functional variants and rare pathogenic findings with potential implications for personalized nutritional management. This approach adds clinical value to already-performed genomic testing at no additional sequencing cost. These observations are descriptive and hypothesis-generating; validation in larger cohorts with matched controls and correlation with biochemical markers and supplementation outcomes is warranted.

POSTER PRESENTATION P30

Genotype-Guided Mitochondrial Nutritional Support in a Child with an ATAD3A In-Frame Deletion: A Precision Nutrition Case Report

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ABSTRACT

ATAD3A encodes a mitochondrial membrane AAA+ ATPase involved in mitochondrial dynamics, nucleoid organization, respiratory-chain assembly and cholesterol trafficking. Pathogenic variants cause Harel-Yoon syndrome, a rare neurodevelopmental mitochondrial disorder. Although mitochondrial supplements are often prescribed empirically, genotype-informed selection of nutritional support remains insufficiently explored. A 3-year-old girl, born at 35 gestational weeks from an IVF monozygotic twin pregnancy complicated by preeclampsia and perinatal asphyxia, presented with global developmental delay, intellectual disability, spasticity, ventricular septal defect and mild dysmorphism. Brain MRI showed periventricular leukomalacia and probable focal cortical dysplasia. Whole-exome sequencing identified a heterozygous in-frame ATAD3A deletion, c.63_71del, p.(Leu22_Pro24del), classified as a variant of uncertain significance. The variant affects the N-terminal region, implicated in mitochondrial membrane contacts and network regulation. We designed a stepwise workflow combining: (1) baseline profiling of lactate/pyruvate ratio, acylcarnitines, urinary organic acids, 3-methylglutaconic acid, FGF21, GDF15, leukocyte CoQ10 and lipid profile; (2) parental segregation to assess de novo occurrence; (3) fibroblast studies using Seahorse XF respirometry in glucose and galactose media, complex-specific mapping, ATAD3A protein analysis and cristae morphology; and (4) individualized mitochondrial nutritional support selected according to predicted ATAD3A-related vulnerabilities: ubiquinol and riboflavin for respiratory-chain support, thiamine and L-carnitine for energy metabolism, alpha-lipoic acid, N-acetylcysteine and vitamin E for redox balance, with folinic acid, magnesium and taurine as adjunctive support. Dosing and monitoring were age- and weight-adjusted. This case proposes a framework for moving from empiric mitochondrial supplementation toward genotype-guided precision nutrition in rare neurodevelopmental disease. Integration of variant interpretation, metabolic biomarkers and cellular respirometry may support both pathogenicity assessment and objective monitoring of response. The ATAD3A-specific rationale links mitochondrial membrane organization, respiratory-chain function, oxidative stress and cholesterol metabolism, making this gene a relevant model for nutrigenomic stratification in early-life disorders with potential lifelong health consequences. A structured ATAD3A-informed nutritional strategy may personalize mitochondrial support and generate measurable endpoints for future nutrigenomic studies.

POSTER PRESENTATION P31

Exome Sequencing Ends a 30-Year Diagnostic Odyssey in Congenital Chloride Diarrhea: Implications for Precision Nutrition

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Wednesday 2 September, 12.00–13.45 | Poster Session 2 | Session 5: Precision Nutrition and Digital Twins in Health and Disease

ABSTRACT

Congenital chloride diarrhea (CLD; OMIM #214700) is a rare autosomal recessive disorder caused by pathogenic variants in SLC26A3, encoding the intestinal Cl^-/HCO_3^- exchanger DRA. CLD presents with lifelong secretory diarrhea, metabolic alkalosis, and electrolyte disturbances. Delayed diagnosis may result in repeated invasive investigations, inappropriate empirical therapies, and lack of prognostic and reproductive counseling. A 31-year-old Bulgarian male presented with lifelong watery diarrhea, occurring 7–8 times daily, and a history of gastric and colonic polyps. Despite more than three decades of clinical follow-up, the underlying cause remained undiagnosed. Whole-exome sequencing was performed because a hereditary disorder was suspected. Whole-exome sequencing identified a homozygous pathogenic SLC26A3 variant, c.2024_2026dup, p.(Ile675dup), an in-frame duplication, establishing the diagnosis of congenital chloride diarrhea and ending a 30-year diagnostic odyssey. The molecular diagnosis enabled lifelong individualized electrolyte replacement, complication-focused renal surveillance, avoidance of further non-directed investigations into the cause of diarrhea, and reproductive counseling, including partner carrier testing and individualized offspring-risk assessment. It also provided a rationale for considering butyrate as a potential adjunctive nutritional intervention. Previous limited evidence suggests that response to butyrate may vary according to SLC26A3 genotype; however, variant-specific efficacy for p.(Ile675dup) remains uncertain. This case demonstrates that genomic diagnosis in congenital chloride diarrhea extends beyond diagnostic confirmation. It supports individualized long-term management, prevention and surveillance of complications, rational selection of potential nutritional interventions, and informed reproductive planning. CLD represents a clinically relevant model of how genomic information may guide the evaluation of nutrient-derived therapies, while emphasizing the need to distinguish established treatment recommendations from emerging genotype-associated evidence.

POSTER PRESENTATION P32

Rationale for personalised intervention diets in the NUTRIOME meal study

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Wednesday 2 September, 12.00–13.45 | Poster Session 2 | Session 5: Precision Nutrition and Digital Twins in Health and Disease

ABSTRACT

Individuals respond differently to the same foods, indicating that some may benefit from tailored dietary interventions. In this study, we (1) characterised individual postprandial responses to three test meals, (2) derived an algorithm allocating individuals to personalised diets based on these responses, and (3) evaluated the effects of a personalised vs control diet over 6 weeks on acute and long-term cardiometabolic risk factors. Participants aged 40 to 70 with a BMI of 27-35 kg/m2 (25,0-26,9 kg/m2 if waist circumference elevated) were recruited at three study centres in Europe. In Phase 1, participants consumed three isocaloric meals (~670-683 kcal) varying in macronutrient composition and quality. An algorithm was developed based on postprandial glucose and triglyceride responses to assign participants to different personalised diet groups. In Phase 2, participants were randomised to their allocated personalised diet or a control diet based on dietary guidelines. Postprandial metabolic responses and selected cardiovascular risk factors were evaluated. 113 participants were included in the study, of which 97 finished Phase 1 and 87 completed the dietary intervention. ‘Carbohydrate tolerance’ and ‘fat tolerance’ were derived in the algorithm based on minimising baseline-corrected AUCs. The postprandial response data from the test meals allowed the assignment of individuals to three metabolic response groups – one ‘carbohydrate tolerant’ and two ‘fat tolerant’ groups. These were utilised in the intervention to evaluate different carbohydrate and fat intake ratios and fat quality. The dietary intervention was overall well received by participants and results from Phase 2 are outstanding.

POSTER PRESENTATION P33

Linking Individual Urine Metabolomic Profiles to Postprandial Responses in the NUTRIOME Study

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ABSTRACT

Diet is a key factor in preventing non-communicable diseases, but individuals respond differently to the same foods, highlighting the need for personalised nutrition. The NUTRIOME meal study, a data-driven precision nutrition intervention study using meal responses, is a two-phase, multi-centre precision nutrition intervention, aimed at developing tailored dietary recommendations to reduce cardiometabolic risk factors and investigating factors driving individual differences in postprandial responses. Urine is used as a non-invasive biological sample to capture host-, microbial-, and diet-derived metabolites, enabling detailed analysis of individual metabolic profiles. The aim of this project is to investigate whether baseline urinary metabolomic profiles differ across distinct postprandial response types and to examine their associations with metabolic and immune responses. Ninety-seven overweight adult participants (74 females, 23 males) consumed, in a crossover design, three isocaloric meals differing in macronutrient composition (carbohydrate-rich, high-quality fat-rich, and low-quality fat-rich), once a week over three consecutive weeks, while consuming their habitual diet on all other days. Blood samples were collected before and after meal consumption to assess postprandial immune and metabolic responses, including glucose, triglycerides and interleukin-6. On each study day, fasting first-morning urine samples were collected before consumption of the test meal. The three urine samples of each participant were subsequently analysed using high-performance liquid chromatography–mass spectrometry (HPLC-MS)-based untargeted metabolomics. Using the postprandial responses to the three meal tests, an algorithm was developed to stratify individuals into three response types. A total of 3,094 compounds were detected across all samples (MSI level 1: n=212; level 2: n=527; level 3: n=1918; level 4: n=1247), providing broad coverage of the semi-polar urine metabolome. Inter-individual differences were observed, indicating the highly personalized nature of urine metabolomic profiles and highlighting the importance of considering individual variability in human metabolomics studies. Ongoing analyses are exploring differences in urine metabolites between response types, as well as associations between the urine metabolome and postprandial metabolic responses. These findings may provide further insight into how individual urine metabolomic profiles, including microbial-derived metabolites, relate to metabolic and immune responses following food intake.

POSTER PRESENTATION P34

Multi-omics integration for subtyping response patterns to a dietary intervention where saturated fats were replaced by polyunsaturated fats

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ABSTRACT

Integration of multi-omics data can increase understanding of interindividual differences in response to dietary interventions. The aim of this study is to use the similarity network fusion (SNF) to investigate whether we can identify subgroups of individuals showing different response patterns to replacing dietary SFA with PUFA and to characterize these patterns. Moderately hypercholesterolemic adults (n = 43) received a dietary intervention replacing food products high in SFA with products high in PUFA for 8 weeks. SNF was applied to integrate percentage change in clinical parameters, plasma fatty acids, transcriptomics, targeted gene expression, and metabolomics profiles after the intervention. Normalized mutual information (NMI) was used to determine the importance of features in separating the clusters. Cluster-specific metabolic and lipid response were investigated. Using spectral clustering, the integrated network was separated into three subgroups. Key molecular drivers of cluster separation included percentage change in LDL- and VLDL-related markers, total triglyceride, total cholesterol, and some saturated fatty acids, showing distinct metabolic and lipid response patterns. Cluster 2 showed a stronger decrease in LDL-cholesterol, while cluster 1 showed a stronger decrease in triglycerides. Compared to the two other clusters, cluster 3 showed a moderate LDL-cholesterol and triglyceride response, and a smaller decrease or even an increase in most saturated fatty acids, along with a larger increase in most polyunsaturated fatty acids. Despite receiving the same dietary intervention, subgroups of the intervention group showed different response patterns across their integrated data profile, specifically plasma fatty acids and LDL- and VLDL-related features. This calls for further investigations of interindividual differences in response to dietary interventions with potential for development of personalized dietary advice.

POSTER PRESENTATION P35

Modeling postprandial triglyceride response using a neural network framework uncovers individual-specific biological drivers

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ABSTRACT

Understanding how individual omics profiles shape dynamic physiological responses is a central challenge in precision nutrition, yet standard longitudinal models often fail when the number of covariates far exceeds the sample size and when effects are non-linear and highly interactive. We propose here a deep learning framework designed to model high-dimensional longitudinal processes in such data-scarce settings. The approach uses an over-parameterized neural network, trained with rigorous k-fold validation and ensemble averaging, to capture complex non-linearities, gene–metabolite interactions, and temporal patterns without explicit variable selection or prior dimensionality reduction. We apply the framework to postprandial triglyceride trajectories measured over six hours after four different high-fat meals in 47 subjects, integrating 625 gene transcripts, 120 metabolites, fasting triglycerides, age, and sex. Despite the limited sample size, the large network achieves stable predictive performance and avoids overfitting. Model interpretation with SHAP (Shapley Additive exPlanations) values recovers biologically meaningful features, including known regulators of lipid metabolism, and cross-conformal prediction provides valid uncertainty intervals for individual trajectories. Our results demonstrate that, when properly regularized, heavily over-parameterized neural networks can both predict and explain complex longitudinal phenotypes where classical models fail, offering a powerful alternative for integrating multi-omics data in clinical nutrition.

POSTER PRESENTATION P36

Causal structure learning for nutrition and healthy aging

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Wednesday 2 September, 12.00–13.45 | Poster Session 2 | Session 5: Precision Nutrition and Digital Twins in Health and Disease

ABSTRACT

Diet modification is a promising avenue for extending healthspan, a metric defined as the number of years lived in good health. Understanding which dietary components most impact healthy aging is inherently a causal question. However, drawing causal conclusions about the relationship between diet and aging is challenging: aging is a process that occurs over decades, and controlled, long-term dietary intervention trials are not feasible, while observational studies are susceptible to bias. Although recent decades have seen substantial advances in methods for estimating causal effects from observational data, most approaches assume a known underlying causal structure informed by expert knowledge or prior studies. For many questions related to nutrition and aging, such detailed causal knowledge is incomplete. The field of causal discovery, which concerns itself with inferring causal structure from observational data, could help address this gap. We present an overview of a PhD project involving the development of causal discovery methods to investigate questions about the links between nutrition and healthy aging. The rationale for this work, as well as some preliminary results from an analysis of the Health and Retirement Study, are presented. First, simulated data are used to evaluate different causal discovery methods and determine which perform best under different circumstances. The best-performing methods are then applied to investigate the relationship between dietary patterns and functional outcomes.

POSTER PRESENTATION P37

Machine learning regression models for predicting body mass index in adults using UK Biobank data

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ABSTRACT

The effects of DNA repair genes on obesity have been investigated in adults using UK Biobank data. Tu et al., (in preparation) reported that the number of alternative/risk alleles in the following DNA repair genes NEIL2, NEIL3, UNG, OGG1, TDG, MUTYH, NTHL1, and FEN1, together with FTO, was associated with BMI in a multivariable regression model adjusted for age, sex, MET score, and smoking status. However, the explained variance of that model was approximately 5%. In the present study, our aim was not to determine whether demographic factors (age, sex, education status), lifestyle factors (smoking, MET score and diet-related energy levels), or genetic factors (the summed risk score for DNA repair genes and FTO) were associated with BMI, but rather to evaluate how well these variables jointly predict BMI using regression-based machine learning models. To elaborate, we compared the predictive performance of decision tree, random forest, and gradient boosting regression models to investigate if any of these approaches could improve the prediction of the BMI. A total of 47,449 complete cases were included in the analysis. The data were randomly split into training (80%) and test (20%) sets. Five-fold cross-validation was then applied to the training set, with four folds used for training and one-fold used for validation in each iteration. Model performance was compared using cross-validated R^2, RMSE, and MAE. Among the evaluated models, gradient boosting showed the best predictive performance, although its explanatory ability remained poor and only slightly better than that of the other models. The selected model was then evaluated on the test set, where it also showed poor predictive performance (6%). Overall, all regression-based machine learning models performed poorly, suggesting that the selected predictors provide only limited information for predicting BMI. Although FTO is a well-established obesity-related gene and the included DNA repair genes have shown significant associations in conventional regression analyses, BMI is a highly polygenic trait influenced by many genetic variants of small effect, as well as by environmental factors.

POSTER PRESENTATION P38

Dynamic system identification from longitudinal vitamin D perturbations reveals regulatory control architecture of interindividual responsiveness

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ABSTRACT

Vitamin D signaling regulates immune and metabolic processes through vitamin D receptor-mediated transcriptional control, yet the systems-level mechanisms underlying interindividual variability in supplementation responses remain poorly defined. Here we investigated longitudinal molecular responses to repeated weight-adjusted vitamin D_3 bolus supplementation in 41 healthy adults. Transcriptome profiling of peripheral blood mononuclear cells across three supplementation cycles enabled the dissection of acute and cumulative transcriptional adaptation under physiologically relevant perturbation conditions. Repeated supplementation induced modest acute transcriptional perturbations but progressively amplified coordinated system-level gene expression reprogramming during sustained endocrine exposure. Integration of transcriptional responsiveness with serum 25-hydroxyvitamin D_3 pharmacokinetics allowed classification of individuals into reproducible response phenotypes, supporting the concept of a personal vitamin D response index. To elucidate the underlying regulatory mechanisms, we reconstructed a vitamin D-responsive transcription factor network from persistently regulated genes and reduced it to a dynamically identifiable regulatory kernel sufficient to explain temporal transcription factor dynamics. Ordinary differential equation-based modeling demonstrated that this compact regulatory system quantitatively recapitulates observed transcriptional trajectories across supplementation cycles and responder groups. Network analysis identified TP53, MYC, JUN, FOS, and HIF1A as dominant control nodes governing response heterogeneity. This links vitamin D signaling to core pathways of immune regulation, cellular stress adaptation, and metabolic control. Together, our findings establish intervention-driven dynamic system identification as a framework for quantitatively linking endocrine nutrient exposure to transcriptional regulatory dynamics in humans. Sensitivity analysis and out-of-sample prediction of an independent cohort (R^2 = 0.978) confirmed the robustness and transferability of the inferred regulatory architecture, establishing a mechanistic foundation for predictive modeling of individualized vitamin D responsiveness in precision nutrition.

POSTER PRESENTATION P39

Associations between immune gene modules and lipoproteins following PUFA intervention

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ABSTRACT

The risk of developing cardiovascular diseases (CVD) is strongly influenced by lifestyle factors, among which dietary fatty acid composition has long been recognized as a key factor. Polyunsaturated fatty acids (PUFAs) play important roles in modulating CVD indicators, including low-density lipoprotein cholesterol (LDL-C), through their regulatory effects on lipoprotein metabolism and immune function. In this study, we performed a secondary analysis of a randomized controlled dietary intervention trial by integrating transcriptomic and lipoprotein metabolomic data from the intervention group only to investigate gene expression responses to PUFA intervention and their associations with different lipoprotein subclasses and components. Multiple ApoB-containing lipoprotein particles were negatively associated with the PUFA intervention. Distinct associations with the intervention were observed among lipoprotein particles of different sizes and compositions within the same lipoprotein class. Peripheral blood mononuclear cell (PBMC) transcriptomic profiles were clustered into 22 gene co-expression modules, of which seven showed significant associations with the intervention. Four responsive modules were enriched in immune-related pathways, including macroautophagy, B-cell function, viral processes, and innate immune responses. Immune-related gene modules displayed complex association patterns with lipoprotein subclasses and their components. Notably, VLDL and HDL particles of different sizes exhibited distinct associations with immune modules, highlighting the potential of these metabolic biomarkers to reflect immune responses and disease risk. Our study revealed potential effects of dietary PUFA intake on immune function and provided a map of associations between lipoprotein metabolism and immune-related gene expression modules. These associations facilitate further validation exploring the potential roles of specific lipoprotein particles as biomarkers of inflammation and cardiometabolic disease risk.

POSTER PRESENTATION P40

Comparative analysis of human transcriptional responses to different types of dietary intake intervention

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Wednesday 2 September, 12.00–13.45 | Poster Session 2 | Session 5: Precision Nutrition and Digital Twins in Health and Disease

ABSTRACT

Diet consists of multiple components that exert diverse effects on human health. Dietary intervention studies often focus on a specific nutrient or dietary component—such as fats, proteins, or nutritional supplementation—to investigate how differences in intake affect health outcomes and metabolic mechanisms. However, the common and interconnected patterns of human responses to different types of dietary interventions remain largely unexplored. Publicly available human dietary intervention transcriptomics datasets are systematically searched and collected from GEO (Gene Expression Omnibus). Eligible studies are separated into comparable intervention groups, identifying paired pre- and post-intervention samples, and standardizing gene identifiers across different microarray and RNA-seq platforms. Data harmonizing includes expression matrix and metadata. An automated analysis pipeline is developed for data quality assessment, gene filtering, within-group differential expression analysis and functional enrichment analysis. Gene coverage diagnostics are performed to quantify coverage across groups and platforms, evaluate pairwise shared-gene proportions, and identify datasets contributing disproportionately to gene loss. Sensitivity analyses using alternative gene-coverage thresholds are subsequently used to assess the robustness of downstream results. Cross-study response patterns are evaluated using principal coordinate analysis based on shared genes and ranked differential-expression statistics, while gene set enrichment analysis was used to characterize and cluster pathway-level responses. Additional tissue-specific analyses are performed within major tissue categories to investigate whether dietary intervention responses showed tissue-dependent patterns. This project is expected to create a platform that enables nutrition researchers to identify common and distinct transcriptional response patterns across dietary interventions. It also explores how tissue type, intervention characteristics, and intervention duration contribute to cross-study variation. Tissue-specific analyses may reveal biologically meaningful patterns that are masked when all studies are analysed together, while GSEA-based clustering is expected to uncover pathway-level responses. Together, this framework will provide a reproducible strategy for integrating public dietary intervention transcriptomics datasets and may support the design and interpretation of future nutritional intervention studies.

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