

6th Paula Rantakallio Symposium on Birth Cohorts and Longitudinal Studies



10–12 June 2026
University of Oulu
Finland



**UNIVERSITY
OF OULU**

Where science meets culture – welcome to the 6th Paula Rantakallio Symposium

It is our great pleasure to gather with you for this year's event, which brings together researchers, practitioners, and partners from across disciplines who share a deep commitment to birth cohorts and longitudinal studies. In 2026, our focus turns especially to the power of transdisciplinary research in advancing our understanding of lifelong health and ageing.

This year is truly exceptional for us in Oulu, as we proudly celebrate the 60th and 40th anniversaries of the Northern Finland Birth Cohorts 1966 and 1986 – remarkable milestones that reflect decades of scientific dedication, participant engagement, and societal impact. These cohorts have profoundly shaped our knowledge of health, development, and ageing, and they continue to serve as invaluable foundations for cutting edge, transdisciplinary research.

We are equally delighted to celebrate the city of Oulu, named the European Capital of Culture 2026. The vibrant cultural landscape of Oulu provides a unique and inspiring backdrop for this symposium, inviting us to explore the rich intersections between science and culture and offering fertile ground for dialogue, collaboration, and discovery.

We are deeply grateful for the outstanding work you have contributed and will be sharing with us. We trust that you will find inspiration, new ideas, and meaningful connections throughout the symposium – and that this gathering under the midnight sun becomes truly memorable, as the nightless nights of Northern Finland offer endless daylight to explore both science and culture.

Wishing you an engaging, insightful, and truly inspiring symposium.

On behalf of the Organising Committee
Marko Kantomaa
Chair of the 6th Paula Rantakallio Symposium

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Programme

Wednesday 10th June

Time	Title	Speaker
8:00	Registration	
9:00	Session 1	Chair: Marko Kantomaa
9:00	Welcome to the 6 th Paula Rantakallio Symposium	Marko Kantomaa Chair of the 6 th Paula Rantakallio Symposium <i>Research Unit of Population Health, University of Oulu, Finland</i>
9:10	K1 – Opening lecture – The genetic architecture of obesity across the life course	Ruth Loos <i>Novo Nordisk Foundation Center for Basic Metabolic Research (CBMR), University of Copenhagen, Denmark</i>
10:00	Coffee/tea break	
10:30	Session 2	Chairs: Sylvain Sebert and Barbara Heude
10:30	O1 – The next phase of cohort studies? From observation to intervention to action	William Siero <i>Population Health, Murdoch Children’s Research Institute, Australia</i>
10:45	O2 – The causal relationships between leisure-time physical activity and body mass index in adulthood: a triangulation study	Anna Kankaanpää <i>Gerontology Research Center (GEREC), Faculty of Sport and Health Sciences, University of Jyväskylä, Finland</i>
11:00	O3 – An intersectional analysis of sex, early-life socio-economic factors, and genetic risk on adult obesity: a Northern Finland Birth Cohort 1966 study	Subhechcha Bhandari <i>Research Unit of Population Health, University of Oulu, Finland</i>
11:15	O4 – Device-measured 24-h movement behaviors and metabolic profile in young adults	Kristin Suorsa <i>Department of Public Health, University of Turku and Turku University Hospital, Finland</i>
11:30	O5 – Exploring biological pathways of heat exposure in pregnant women and infants in the climate change era: HIGH Horizons prospective birth cohort, Greece	Zacharoula Bogogiannidou <i>Laboratory of Hygiene and Epidemiology, Faculty of Medicine, University of Thessaly, Greece</i>
11:45	O6 – Municipality level changes in overweight and obesity in pregnant women in Sweden over two decades: evidence from The Swedish Medical Birth Register	Fethi Mohammed Yusuf <i>Department of Epidemiology and Global Health, Umeå University, Sweden</i>
12:00	Lunch break (Restaurant Medisiina, Aapistie 5)	
13:00	Poster walks 1–3	
14:00	Session 3	Chair: Anna Pulakka
14:00	K2 – Keynote lecture – Birth cohort studies in Malawi, a low-and middle-income setting: opportunities and challenges.	Charles Mangani <i>Malaria Alert Centre, Kamuzu University of Health Sciences, Malawi</i>
15:00	Coffee/tea break	
15:30	Session 4	Chairs: Emma Raitoharju and Sonja Rajić

15:30	O7 – FILOMENE – a new French large couple-child cohort to study the relations between the exposome and children’s health	Barbara Heude <i>Université Paris Cité et Université Sorbonne Paris Nord, Inserm, INRAE, Centre de Recherche en épidémiologie et Statistiques (CRESS), France</i>
15:45	O8 – Extensive sample and data collection on environmental exposures in a new Biofantti birth cohort	Jenni Lehtimäki <i>Built Environment Solutions Unit, Finnish Environment Institution, Finland</i>
16:00	O9 – Future Finland Birth Cohort: optimizing the use of Finnish register and welfare clinic data to advance research on family and child wellbeing	Eeva-Leena Kataja <i>Welfare Epidemiology and Monitoring Unit, Finnish Institute for Health and Welfare (THL), Finland</i>
16:15	O10 – Physical fitness in adolescents born preterm or small for gestational age in rural Malawi	Pragga Bhattacharjee <i>Clinical Medicine Research Unit, University of Oulu, Finland</i>
16:30	O11 – Supporting children’s decision-making in a large-scale birth cohort study through staff experiences in the Japan Environment and Children’s Study	Ryoko Matsuyama <i>Department of Computational Biology and Medical Sciences, Graduate School of Frontier Sciences, The University of Tokyo, Japan</i>
16:45	O12 – How do birth cohort researchers beat the survey fatigue?	Anne-Marie Nybo Andersen <i>Section for Epidemiology, University of Copenhagen, Denmark</i>
17:00	End of day 1	

Thursday 11th June

Time	Title	Speaker
8:00	Registration	
9:00	Session 5	Chair: Jouko Miettunen
9:00	K3 – Keynote lecture – Rethinking mental illness: from causal mechanisms to precision immunotherapy	Golam Khandaker <i>Bristol Medical School, University of Bristol, United Kingdom</i>
10:00	Coffee/tea break	
10:30	Session 6	Chairs: David Burgner and Toby Mansell
10:30	O13 – Prenatal stress and adverse childhood experiences as risk factors for atherosclerotic cardiovascular disease – FinnBrain Birth Cohort Study	Hasse Karlsson <i>Department of Psychiatry and Department of Clinical Medicine, University of Turku and Turku University Hospital, Finland</i>
10:45	O14 – Early determinants of parental burnout in mothers of the NINFEA birth cohort	Maja Popovic <i>Department of Medical Sciences, University of Turin, Italy</i>
11:00	O15 – Neighbourhood socioeconomic deprivation and ADHD and ASD during childhood in Finland, a country-wide register study (1994–2016)	Basho Poelman <i>Research Unit of Population Health, University of Oulu, Finland</i>
11:15	O16 – Maternal inflammation and the development of white matter microstructure: a multi-cohort study	Anni Niskanen <i>Research Unit of Clinical Medicine, Department of Psychiatry, University of Oulu, Finland</i>
11:30	O17 – Socioeconomic inequalities in childhood respiratory tract infections. Data analysis of >200,000 children in 16 birth cohorts from Europe and Australia	Demetris Avraam <i>Department of Public Health, University of Copenhagen, Denmark</i>
11:45	O18 – Socioeconomic position, inflammation, and cardiovascular health in childhood and adolescence: an international multi-cohort study	Toby Mansell <i>Department of Paediatrics, University of Melbourne, Australia</i>
12:00	Lunch break (Restaurant Medisiina, Aapistie 5)	
13:00	Poster walks 4–6	
14:00	Session 7	Chair: Johannes Kettunen
14:00	K4 – Keynote lecture – Frailty beyond old age – from late-life syndrome to lifecourse risk	Juulia Jylhävä <i>Research Unit of Population Health, University of Oulu, Finland</i>
15:00	Coffee/tea break	
15:30	Session 8	Chairs: Elina Sillanpää and Suvi Ravi
15:30	O19 – Early-life infections and life course health in the EU Child Cohort Network	Marianna Karachaliou <i>The Generation R Study Group, University Medical Center, Rotterdam, Netherlands</i>

15:45	O20 – The LIFE Child Study: a longitudinal perspective on child development and lifestyle diseases	Mandy Vogel <i>Hospital for Children and Adolescents, LIFE Child, Leipzig University, Germany</i>
16:00	O21 – Exploring the exposome and unexplained variance in biological ageing – insights from a longitudinal twin study in adolescence and early adulthood	Annika Opperbeck <i>Faculty of Sport and Health Sciences, University of Jyväskylä, Finland</i>
16:15	O22 – Impacts of social isolation on health and wellbeing: a mixed methods study	Estelle Lowry <i>School of Natural and Built Environment, Queen's University Belfast, United Kingdom</i>
16:30	O23 – More than Images: inclusion and collective deliberation in visualising ageing	Mireia Manent Blanch <i>Project Management, Communications Department, WeDo Project Intelligence Made Easy, Spain</i>
16:45	O24 – Life course drivers of frailty: a longitudinal birth cohort study	Markus Haapanen <i>Research Unit of Clinical Medicine, University of Oulu, Finland</i>
17:00	End of day 2	
19:00 – 22:00	Symposium dinner at Theatre Restaurant Aulis, Oulu Theatre Street address: Kaarlenväylä 2	

Friday 12th June

Time	Title	Speaker
8:00	Registration	
9:00	Session 9	Chair: Johanna Metsälä
9:00	K5 – Keynote lecture – The infant gut microbiome at the crossroads of environmental exposures and child health	Tanya Alderete <i>Department of Environmental Health and Engineering, Johns Hopkins University, United States</i>
10:00	Coffee/tea break	
10:30	Session 10	Chair: Ville Pimenoff
10:30	K6 – Keynote lecture – Intersectionality, health inequalities and unequal ageing across the life course	Daniel Holman <i>School of Sociological Studies, Politics and International Relations, University of Sheffield, United Kingdom</i>
11:30	Short break	
11:45	Session 11	Chairs: Leena Ala-Mursula and Ville Pimenoff
11:45	O25 – Longitudinal monitoring of biotic and abiotic exposures using wearable devices among pregnant women in Finland	Xinyue Zhang <i>Department of Genetics, Stanford University, United States</i>
12:00	O26 – Longitudinal mapping of environmental exposures with individual-level geographic coordinates and wearable devices among pregnant women in Finland	Ville Pimenoff <i>Research Unit of Population Health, University of Oulu, Finland</i>
12:15	O27 – Linking climate reanalysis data and birth registries: a scalable approach to study heat exposure effects on perinatal health	Elizaveta Fomenko <i>Study Center for Perinatale Epidemiology (SPE), Belgium</i>
12:30	O28 – Relationship between early life adversities, gestational age and their joint effect on birth of the first child	Priyanka Choudhary <i>Clinical Medicine Research Unit, University of Oulu, Finland</i>
12:45	O29 – Air pollution from livestock farms and lung function decline in neighboring residents over 7 years	Warner van Kersen <i>Institute for Risk Assessment Sciences, Utrecht University, Netherlands</i>
13:00	O30 – ExpoMapper: an interactive tool for generating environmental exposures from individual-level geographic coordinates	Justiina Ronkainen <i>Research Unit of Population Health, University of Oulu, Finland</i>
13:15	Farewell and closing remarks	
13:30	Symposium ends	
13:30	Lunch (Restaurant Medisiina, Aapistie 5)	

Poster Walks

Time	Title	Speaker
Wednesday 10th June		
13:00	Poster walk 1	Leader: Ulla Lång
	P1 – Biotic and abiotic exposure estimated from blood among pregnant women	Ville Pimenoff <i>Research Unit of Population Health, University of Oulu, Finland</i>
	P2 – The prenatal exposome and childhood asthma in Finland, does climate change play a role?	Warner van Kersen <i>Research Unit of Population Health, University of Oulu, Finland</i>
	P3 – Metabolites and lipids largely mediate the protective effect of breastfeeding on infant inflammation	Toby Mansell <i>Infection, Immunity and Global Health Theme, Murdoch Children's Research Institute, Australia</i>
	P4 – Early-life determinants and at-birth prediction of early childhood caries in a nationwide Finnish birth cohort	Pragathy Kannan <i>Faculty of Medicine, Clinical and Molecular Metabolism Research Program (CAMM), University of Helsinki, Finland</i>
	P5 – Early life small quantity lipid-based nutrient supplementation and cognitive ability at 9 years: a follow-up study in Malawi	Yvonne Muthiani <i>Center for Child, Adolescent and Maternal Health Research, Faculty of Medicine and Health Technology, Tampere University and Tampere University Hospital, Finland</i>
	P6 – Gendered impact of violence against women on children's growth up to 2-years: analysis of MINIMat longitudinal cohort data from rural Bangladesh	Jannatul Ferdous Antu <i>Maternal and Child Health Division, icddr, Bangladesh</i>
	P7 – Maternal and birth predictors of eating disorders in Northern Finland Birth Cohort 1986	Aajna Adoor <i>Research unit of Population Health, University of Oulu, Finland</i>
	P8 – Longitudinal dynamics of epigenetic entropy in DNA methylation profiles from the Northern Finland Birth Cohort 1966	Marjan Aziminezhad <i>Research Unit of Population Health, University of Oulu, Finland</i>
	P9 – Spouses of women with gestational diabetes are at increased risk of ischemic heart disease – FinnGeDi Study	Tea Taskila <i>Research Unit of Clinical Medicine, Medical Research Center, Oulu University Hospital, University of Oulu, Finland</i>
13:00	Poster walk 2	Leader: Johanna Metsälä
	P10 – The Flemish perinatal registry: from statutory registration to advances in perinatal health research for over 40 years	Elizaveta Fomenko <i>Study Centre for Perinatal Epidemiology (SPE), Belgium</i>
	P11 – Future Finland – the birth of a new nationwide birth cohort	Eero Kajantie <i>Welfare Epidemiology and Monitoring Unit, Finnish Institute for Health and Welfare, Finland</i>

	P12 – Future Finland Birth Cohort – piloting recruitment approaches in routine healthcare	Anna Parkkola <i>Welfare Epidemiology and Monitoring Unit, Finnish Institute for Health and Welfare, Finland</i>
	P13 – Future Finland Birth Cohort – Applying social and behavioural insights for supporting participant recruitment and communication	Viivi Eskelinen <i>Cultural, Behavioural, and Media Insights Centre, Finnish Institute for Health and Welfare, Finland</i>
	P14 – Arctic Biobank – Biobanking infrastructure of the Northern Finland Birth Cohorts for lifelong health and aging research	Maarit Kangas <i>Infrastructure for Population Studies, Arctic Biobank, Northern Finland Birth Cohorts, University of Oulu, Finland</i>
	P15 – Using the UK Census Longitudinal Studies to investigate long term change	Estelle Lowry <i>School of Natural and Built Environment, Queen's University Belfast, United Kingdom</i>
	P16 – Fostering lifelong resilience and deep pragmatism: A transdisciplinary proposal for integrating 'meta-morality' into longitudinal birth cohort studies	Atiqul Mazumder <i>Department of Psychiatry, Research Unit of Clinical Medicine, University of Oulu, Finland</i>
	P17 – Entanglements of science and art – rethinking the ways of distributing knowledge	Kati Leinonen <i>Independent photographic artist, Finland</i>
	P18 – Threading age: visualising ageism through participatory data physicalisation	Mireia Manent <i>Project Management, Communications Department, WeDo Project Intelligence Made Easy, Spain</i>
13:00	Poster walk 3	Leader: Yuan Wang
	P19 – Heritability estimation of BMI across early life from birth to age 15: sex-specific insights from the Northern Finland Birth Cohorts 1966 and 1986	Yuhua Fan <i>Research Unit of Population Health, University of Oulu, Finland</i>
	P20 – Body size and blood pressure of 9-year-old children born preterm or small for gestational age (SGA) in Malawi	Eeva Virtanen <i>Clinical Medicine Research Unit, University of Oulu, Finland</i>
	P21 – Adolescent weight gain trajectories and their associations with biological aging: a genetically informed study	Anni Pitkänen <i>Faculty of Sport and Health Sciences, University of Jyväskylä, Finland</i>
	P22 – Suboptimal dietary patterns are associated with accelerated biological aging in young adulthood: a study with twins	Suvi Ravi <i>Faculty of Sport and Health Sciences, Gerontology Research Center, University of Jyväskylä, Finland</i>
	P23 – Associations of habitual coffee intake with testosterone and cardiometabolic markers: The Northern Finland Birth Cohort 1966 study	Luca Verroest <i>Research Unit of Biomedicine and Internal Medicine, Faculty of Medicine, University of Oulu, Finland</i>
	P24 – Prospective bidirectional associations between leisure-time physical activity and adiposity across the transition from middle to old age	Tiina Savikangas <i>Research Unit of Population Health, University of Oulu, Finland</i>
	P25 – Association with long-term exposure to heat or cold strain measured by UTCI and blood pressure in middle age	Anna Remes <i>Research Unit of Population Health, University of Oulu, Finland</i>

	P26 – Intersectional analysis of sex and socioeconomic position on the development of obesity in later life using the Northern Finland Birth Cohort 1966	Khin Yu Yu Hlaing <i>Research Unit of Population Health, University of Oulu, Finland</i>
	P27 – Sociocultural factors of obesity by different socioeconomic positions across the life-course in high income countries: a systematic review	Fahmida Sarker <i>Research Unit of Population Health, University of Oulu, Finland</i>
Thursday 11th June		
13:00	Poster walk 4	Leader: Toby Mansell
	P28 – DNA methylation in muscle tissue of adults born preterm with very low birth weight: evidence from sibling study	Luliia Averianova <i>Clinical Medicine Research Unit, University of Oulu, Finland</i>
	P29 – Sleep duration moderates the cognitive ability in adults born with very low birth weight: a sibling-control actigraphy study	Seyedalireza Mirilavasani <i>Clinical Medicine Research Unit, University of Oulu, Finland</i>
	P30 – Body composition in mid-adolescence in relation to preterm birth: a cohort study from rural Bangladesh	Zareen Tasnim <i>Clinical Medicine Research Unit, University of Oulu, Finland</i>
	P31 – Being born preterm and having a child in young adulthood: the mediating role of mental health and educational attainment	Abate Belachew <i>Clinical Medicine Research Unit, University of Oulu, Finland</i>
	P32 – Preterm birth and relationship status and parenthood in young adulthood – the role of sex and neurosensory impairments	Johanna Metsälä <i>Clinical Medicine Research Unit, University of Oulu, Finland</i>
	P33 – Cumulative early-life exposure to climate extremes and childhood wheezing	Giovenale Moirano <i>Department of Medical Sciences, University of Turin, Italy</i>
	P34 – Adolescent stress and health in Northern Finland: an intersectional analysis of familial and adolescent perceived social positions	Eetu Haataja <i>Research Unit of Population Health, University of Oulu, Finland</i>
	P35 – Individual and joint associations of adolescent and young adult alcohol use and smoking with midlife psychological distress – a multicohort study	Noora Berg <i>Department of Healthcare and Social Welfare, Finnish Institute for Health and Welfare, Finland</i>
	P36 – Pneumonia morbidity in young and working aged adults; risk factors and immunological properties in a large birth cohort	Pia Holma <i>Research Unit of Biomedicine and Internal Medicine, University of Oulu, Finland</i>
13:00	Poster walk 5	Leader: Subhechcha Bhandari
	P37 – Psychiatric disorders in the offspring of mothers with antenatal stress and fatigue	Jesse Kankaala <i>Research Unit of Clinical Medicine, University of Oulu, Finland</i>
	P38 – Are maternal antenatal fatigue and stress associated with offspring mental health outcomes?	Tiina Riekkö <i>Research Unit of Clinical Medicine, University of Oulu, Finland</i>

	P39 – Gene–environment interactions in mental health trajectories of youth (Youth-GEMs)	Jenni Leppänen <i>Research Unit of Clinical Medicine, University of Oulu, Finland</i>
	P40 – Internalizing and externalizing symptoms from youth to adulthood in the normal population: correlates in limbic system morphology	Lassi Björnholm <i>Department of Psychiatry, University of Oulu, Finland</i>
	P41 – Parent- and teacher-rated childhood emotional and behavioral problems predicting adulthood depressive symptoms and disorders	Jouko Miettunen <i>Research Unit of Population Health, University of Oulu, Finland</i>
	P42 – Do psychotic symptoms predict future psychotic disorders in adolescent psychiatry inpatients? A 17-year cohort study	Valentina Kieseppä <i>Research Unit of Clinical Medicine, University of Oulu, Finland</i>
	P43 – Prognostic factors for adulthood psychosis in adolescent psychiatry services: a longitudinal total birth cohort study	Ulla Lång <i>Institute for Neuroscience and Cardiovascular Research, University of Edinburgh, United Kingdom</i>
	P44 – Association between severity of depressive symptoms and related costs: a Northern Finland Birth Cohort 1966 study	Fahim Tazware Himel <i>Research Unit of Population Health, University of Oulu, Finland</i>
	P45 – Social capital and depression – a 15-year bidirectional analysis	Johanna Kemppainen <i>Research Unit of Population Health, University of Oulu, Finland</i>
	P46 – Environmental inequality and mental health: a geospatial analysis in the Northern Finland Birth Cohort 1966	Zahidul Islam Khan <i>Research Unit of Population Health, University of Oulu, Finland</i>
13:00	Poster walk 6	Leader: Ville Pimenoff
	P47 – Clinical and therapeutic implications of super-enhancer–driven 3D genome regulation at the 2q3 prostate cancer risk locus	Gong-Hong Wei <i>Disease Networks Research Unit, University of Oulu, Finland</i>
	P48 – The conundrum of nc886 – methylation pattern with clear association to ancestral origins with no genetic determinants?	Sonja Rajic <i>Molecular Epidemiology, Faculty of Medicine and Health Technology, Tampere University, Finland</i>
	P49 – Effect of infant vitamin D supplementation on epigenome-wide DNA methylation	Helena Hauta-alus <i>Research Program for Clinical and Molecular Metabolism, University of Helsinki, Finland</i>
	P50 – Clinical usefulness of genetic risk scores in the prediction of gestational diabetes	Elina Keikkala <i>Research Unit of Clinical Medicine, Medical Research Center, University of Oulu, Finland</i>
	P51 – Lower blood cell mitochondrial DNA abundance associates with higher all-cause mortality and aging: a 30-year prospective epidemiological study	Attila Sebe <i>Research Unit of Biomedicine and Internal Medicine, University of Oulu, Finland</i>
	P52 – Male Youth BMI is associated with epigenetic pace of aging of their descendants in maternal line in two generations in post WWII Finland	Jo Ciantar <i>Faculty of Medicine and Health Technology, Tampere University, Finland</i>

	<p>P53 – Fragmented biodiversity in urban residential environment and childhood asthma and atopic dermatitis</p>	<p>Yuan Wang <i>Faculty of Medicine, University of Oulu, Finland</i></p>
	<p>P54 – Genetic architecture of pain heterogeneity in endometriosis</p>	<p>Samu Vanhala <i>Infrastructure for Population Studies, Arctic Biobank, Northern Finland Birth Cohorts, University of Oulu, Finland</i></p>
	<p>P55 – Implementing brain age prediction models in population-based neuroimaging cohorts</p>	<p>Varsha Jayawardena <i>Neuropsychiatric Research Group, Research Unit of Clinical Medicine, University of Oulu, Finland</i></p>



Keynote Speakers



Ruth Loos

University of
Copenhagen,
Denmark

Ruth Loos is the Vice Executive Director of the Novo Nordisk Foundation Center for Basic Metabolic Research (CBMR) at the University of Copenhagen. Ruth has dedicated her career to understanding the genetic causes of obesity, to gain insights into the biology underlying body weight regulation. She is a founding member of the GIANT (Genetic Investigation of ANthropometric Traits) consortium, where she has spearheaded numerous large-scale gene-discovery efforts for BMI and obesity. Her interests extend beyond conventional obesity outcomes, focusing on more refined adiposity phenotypes and composite phenotypes to uncover new biological insights that traditional obesity research may have missed. The emergence of new -omics data has led her team to integrate genetic and proteomic information to identify obesity subtypes, predict who is at risk of obesity, and develop tailored prevention and treatment strategies.

K1: The genetic architecture of obesity across the life course

Ruth Loos¹

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Obesity is a multifactorial disease arising from a complex interplay between genetic predisposition and environmental exposures across the life course. Despite this complexity, obesity is typically defined using a single, unidimensional metric; body mass index ($\text{BMI} \geq 30 \text{ kg/m}^2$). This definition masks substantial heterogeneity among individuals classified as having obesity, who may differ markedly in the causes of weight gain, clinical presentation, associated complications, disease trajectories, and response to treatment. These limitations have motivated growing interest in subclassifying obesity into more homogeneous subtypes.

To date, most subclassification efforts have relied on clinical and phenotypic features. However, the rapidly expanding catalogue of genetic variants associated with obesity enables genetically informed subclassification. Because genetic variation is fixed at conception, genetic subtypes can be defined early in life – well before the onset of obesity – offering unique opportunities to understand etiological heterogeneity and to enable early, targeted prevention.

Recent advances in polygenic scores (PGSs) illustrate this potential. A newly developed PGS, derived from data on over five million individuals, explains up to 18% of variation in BMI and shows strong predictive performance for severe obesity ($\text{BMI} \geq 40 \text{ kg/m}^2$). Children with higher PGSs exhibit accelerated BMI gain from early childhood through adolescence, including earlier adiposity rebound. Incorporating genetic risk into birth based predictors substantially improves prediction of BMI trajectories from childhood into adulthood.

In this talk, I will review emerging approaches that integrate genetic information into the subclassification of obesity and demonstrate how genetically defined subtypes illuminate distinct life course pathways. Ultimately, genetically informed frameworks promise more precise risk prediction, improved prognostic assessment, and more effective, tailored prevention and treatment strategies across the life span.



Charles Mangani

Kamuzu University
of Health Sciences,
Malawi

Dr Charles Mangani is a public health scientist and university teacher. He is an Associate Professor of Epidemiology and Public Health at Kamuzu University of Health Sciences (KUHeS). He also currently serves as Acting Director of Malaria Alert Centre at KUHeS. Dr Mangani's research has focused mainly on understanding the epidemiology of two key childhood conditions in sub-Saharan Africa; undernutrition in early childhood and malaria in under-five and school-aged children, and optimization of interventions to address these two major health problems in low- and middle-income countries. His nutritional research also includes studies on the life-course of vulnerable populations, by examining critical health determinants, and how to improve health outcomes in resource-limited settings. His preference is interdisciplinary community-based research studies that merge epidemiological methods with insights from the social sciences.

K2: Birth cohort studies in Malawi, a low- and middle-income setting: opportunities and challenges

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Maintaining a birth cohort is a large and expensive undertaking, placing demands on both the investigators and the participants. But they offer critical opportunities to understand long-term impact of early vulnerability in pregnancy and early childhood on growth and development over the life course including impact on human capital accumulation. In addition, globally, low- and middle-income (LMIC) countries are projected to account for most of the non-communicable disease burden and related premature mortality by 2030; birth cohorts provide opportunities to study and identify early life context-specific exposures. A collaborative research project in Malawi (Kamuzu University of Health Sciences and Tampere Universities) have maintained three birth cohorts over the past 30 years. They have been used to examine the effects of perinatal and early childhood risk factors on growth, motor and cognitive development, and cardiovascular health in later childhood, adolescence, and early adulthood. A further study is examining transgenerational cycle of health and development among women who were “small vulnerable newborns” at time of birth. However, birth cohorts are not without challenges: loss of participants, insufficient funds for multiple follow-up rounds, maintaining a continuous pool of trained investigators, and potential for selection bias. Because of their importance though, there is need to build stronger international collaborations, expertise training and sharing, data-sharing infrastructure, and sustained funding.



Golam Khandaker

University of
Bristol, United
Kingdom

Professor Golam Khandaker is a world-leading psychiatrist and translational scientist whose pioneering research sits at the intersection of immunology, epidemiology, and clinical psychiatry. His research explores how inflammation and immune processes shape the risk of neuropsychiatric conditions and cognitive outcomes across the life course, with the goal of identifying modifiable mechanisms and developing targeted, biology-informed treatments. He also has a strong interest in multimorbidity, particularly the prevention of cardiometabolic disease in people serious mental illness.

His work combines large-scale population studies, genomic and proteomic approaches, and causal inference methods such as Mendelian randomization. This is complemented by clinical trials of immunotherapy in patients, enabling bench-to-bedside translation.

Professor Khandaker has provided influential evidence for a causal role of inflammatory pathways – especially interleukin-6 – in depression and schizophrenia. These discoveries have directly informed clinical trials targeting inflammation and led to the identification of clinically relevant immunometabolic subgroups.

He is Lead Editor of the Textbook of Immunopsychiatry and serves on the editorial boards of leading psychiatry journals. He works closely with people with lived experience and charities including the McPin Foundation and MQ in the UK, and the Neuroimmune Foundation in the United States.

K3: Rethinking mental illness: from causal mechanisms to precision immunotherapy

Golam Khandaker¹

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Immune dysfunction, particularly low-grade systemic inflammation, is implicated in pathogenesis of depression, schizophrenia, and other chronic psychiatric and physical conditions. Inflammation is a clinically relevant phenotype, as it is associated with poor response to psychotropic medications. Currently, several RCTs are testing the effectiveness of anti-inflammatory drugs for patients with depression and schizophrenia. However, there are key unanswered questions, both mechanistic and clinical. Is inflammation a causal factor for depression and schizophrenia? Could anti-inflammatory drugs be used to treat these disorders? If so, which patients are likely to benefit?

Professor Khandaker will present data from population-based cohort studies (e.g., ALSPAC, UK Biobank), genetic analysis (e.g., Mendelian randomization – a genetic causal inference method) method, and clinical trials addressing the questions of causality and treatment potential. These include largescale proteogenomic investigations in the UK Biobank cohort identifying plasma biomarkers for depression, and possible novel immunological drug targets for neuropsychiatric conditions including depression, schizophrenia, and Alzheimer's disease. In addition, Professor Khandaker will present results from the recently completed Insight study – a RCT of IL-6R receptor antagonist tocilizumab in difficult-to-treat depression. This study provides useful insight into optimal patient selection strategy as well as likely treatment sensitive outcomes to inform the development of largescale definitive trials in future.



Juulia Jylhävä

University of Oulu,
Finland

Dr Jylhävä is a researcher specialising in the systems biology of ageing, with a focus on understanding the causes and consequences of biological ageing using genetic, multi-omics, AI-based, and traditional epidemiological approaches. Her work integrates large-scale longitudinal cohort data to investigate how ageing-related processes develop across the life course and how they contribute to disease and mortality risk.

A key area of her research is frailty, traditionally considered a syndrome of older age. However, her work has demonstrated that frailty is also present in younger adults and represents a significant risk factor for adverse health outcomes earlier in life. This has important implications for public health, highlighting the need for earlier identification and prevention strategies across the adult population.

Her research group is developing innovative tools, including an AI-enhanced electronic frailty index, to identify vulnerable individuals using both structured and unstructured health data. These approaches aim to enable earlier intervention and support more effective healthcare planning.

Dr Jylhävä is a member of The Lancet Commission on Frailty, contributing to global evidence synthesis and policy development on the prevention and management of frailty, positioning her at the forefront of efforts to extend healthspan and improve population health. She has authored over 140 peer-reviewed publications and serves as an Associate Editor for *npj Aging*.

K4: Frailty beyond old age – from late life syndrome to life course risk

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While frailty has traditionally been viewed as a condition of older age, emerging evidence suggests a more complex reality: vulnerability to health decline begins much earlier in life and often remains under-recognised until it is too late and opportunities for effective intervention are limited.

In this keynote, I will challenge conventional views of frailty and present new insights from large-scale longitudinal cohort studies demonstrating that frailty is not confined to older populations but is also prevalent among younger adults, where it acts as a significant predictor of adverse health outcomes. These findings highlight the need to shift from a late-life management paradigm to earlier identification and prevention across the adult lifespan.

I will discuss how advances in genetics, multi-omics, and AI are transforming our ability to study frailty and ageing-related processes and detect early signals of decline. I will also present work on developing next-generation electronic frailty indices that integrate structured and unstructured health data, enabling more comprehensive and timely identification of at-risk individuals in real-world healthcare settings.

Drawing on both epidemiological and translational research, I will explore how these approaches can bridge the gap between data and practice, informing more targeted interventions and sustainable healthcare strategies. The keynote will highlight the importance of rethinking frailty as a dynamic, life-course phenomenon and emphasise its relevance for clinicians, researchers, and policymakers alike.

Ultimately, I aim to encourage a shift in perspective, from treating frailty as an inevitable consequence of ageing to recognising it as an actionable and preventable condition across the population; we just need the right tools to identify at-risk individuals at the right time.



Tanya Alderete

Johns Hopkins University, United States

Dr. Tanya L. Alderete is an Associate Professor in the Department of Environmental Health and Engineering at the Johns Hopkins Bloomberg School of Public Health, with an adjunct appointment at the University of Southern California. Her research focuses on how environmental exposures during critical periods of development, including air pollution, PFAS, and plastics, shape metabolic, immune, and neurodevelopmental health across the life course.

With training in clinical and translational research, environmental epidemiology, and microbiome science, Dr. Alderete combines clinical and population-based approaches with multi-omics methods to understand how environmental toxicants contribute to chronic disease risk. She was among the first to show that air pollution exposure contributes to the pathophysiology of type 2 diabetes in youth and alters gut microbiome composition and function.

Dr. Alderete has authored more than 115 peer-reviewed publications and has received major honors, including an NIEHS K99/R00 Pathway to Independence Award, multiple NIEHS R01 grants, the Health Effects Institute Rosenblith New Investigator Award, a Volkswagen Foundation Night Science Grant, and the Tony McMichael Mid-Career Award from the International Society for Environmental Epidemiology.

K5: The infant gut microbiome at the crossroads of environmental exposures and child health

Tanya Alderete¹

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Environmental exposures during pregnancy and early life shape child health, yet the biological mechanisms linking exposure to developmental outcomes remain only partly understood. One missing layer is the biology in between: the processes that translate an environmental input into a physiological response. The infant gut microbiome is emerging as one of the most important of these intermediate systems—an exposure-sensitive ecosystem whose microbial composition, functional capacity, and metabolic outputs may influence growth, immune development, neurodevelopment, and later disease risk.

In this talk, I present the infant gut microbiome as a common biological interface through which diverse environmental exposures may influence early-life health. Drawing on multi-omics data from a longitudinal birth cohort integrating shotgun metagenomics, untargeted fecal metabolomics, and human milk composition, I will present two linked lines of evidence. First, I will show how prenatal air pollution mixtures are associated with shifts in infant microbial composition, functional pathways, and fecal metabolites, and what these signatures suggest about the timing and mechanisms of exposure effects. Second, I will extend this framework to per- and polyfluoroalkyl substances (PFAS) in human milk, exploring how PFAS may shape both the milk metabolome and the infant gut microbiome during a critical developmental window.

Together, these findings position the infant gut as a unifying biological interface where chemically distinct exposures converge, suggesting that tracking this system may be more informative than measuring exposures in isolation. More broadly, this work highlights the value of life-course, multi-omics approaches for understanding how early environments become biologically embedded and for identifying new opportunities for prevention.



Daniel Holman

University of
Sheffield,
United Kingdom

Dr Dan Holman is a Senior Lecturer in Sociology and Public Health at the University of Sheffield, UK, where he co-leads the Intersectionality Research Network. His research examines how social inequalities shape health and ageing across the life course, with a particular focus on intersectionality, social determinants of health, multimorbidity, and quantitative and mixed-methods approaches to understanding complex inequality.

His work brings together sociological theory, public health, and social epidemiology to investigate how gender, ethnicity, socioeconomic position, age, and other axes of inequality combine to shape health. He has led and contributed to externally funded projects on chronic disease and healthy ageing, intersectional equity in public health research, unequal ageing, later life employment, and quantitative approaches to intersectional analysis. His research has advanced an integrated intersectional life course perspective on unequal ageing, and his recent methodological work has contributed to extending and applying MAIHDA to longitudinal health research.

Dr Holman's keynote will draw on this programme of work to consider how transdisciplinary research can better understand, measure and address lifelong health inequalities.

K6: Intersectionality, health inequalities and unequal ageing across the life course

Daniel Holman¹

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Health inequalities are often described along single axes of difference, such as age, gender, ethnicity or socioeconomic position. Yet people live their lives at the intersection of multiple social locations, and these intersections shape exposure to social determinants of health, experiences of discrimination, access to resources, and opportunities for healthy ageing. As populations age and health inequalities persist across generations, understanding unequal ageing requires approaches that can account for both social complexity and change over time.

This keynote will explore how intersectionality and life course perspectives can be brought together to better understand health inequalities across adulthood and later life. Intersectionality highlights how systems of power and inequality operate together, while life course research draws attention to trajectories, transitions, cumulative exposure and historical context. Taken together, these perspectives suggest that health inequalities are not static or unidimensional, but dynamic, multidimensional and shaped by social conditions across time.

The talk will consider the conceptual, methodological and policy challenges involved in researching intersectional health inequalities. In particular, it will discuss the promise and limitations of quantitative approaches, including Multilevel Analysis of Individual Heterogeneity and Discriminatory Accuracy (MAIHDA), for studying complex patterns of inequality. Drawing on longitudinal UK data, the keynote will show how physical and mental health trajectories vary across intersectional strata defined by age, gender, ethnicity, socioeconomic position and generation. These analyses reveal that social advantage and disadvantage do not operate uniformly across outcomes or stages of the life course.

The keynote will argue that research on health and ageing needs to move beyond simple narratives of vulnerability or risk. By integrating sociological theory, public health, social epidemiology and life course research, we can develop more nuanced ways of understanding, measuring and addressing unequal ageing. The central challenge is to translate complexity into clearer thinking about prevention, policy and social justice.



Oral Presentations

O1: The next phase of cohort studies? From observation to intervention to action

William Siero¹, Susan Clifford¹, Jatender Mohal¹, Alisha Gulenc¹, Tony Frugier¹, Naomi Schwarz¹, Richard Saffery¹, Sharon Goldfeld¹, Raghu Lingam², Melissa Wake¹

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As in many prosperous countries, nearly half of all Australians live with one or more chronic health conditions, accounting for 87% of all deaths. At least a third of the chronic disease burden could be prevented by reducing key early risk factors like obesity, hypertension, poor diet and inactivity, yet population-level success remains limited. Faster, nimbler, and more efficient study designs are needed to reduce chronic disease worldwide.

Lifecourse cohort studies are rapidly transitioning from observation to action platforms. Embedding interventions and multiple nested studies within cohorts can cut costs and completion time of a trial by a third.

Generation Victoria (GenV), Australia's largest and most inclusive parent and child cohort, is a globally unique megacohort spanning >120,000 young children and adults with repeated cell-to-society measurement, intervention capability and whole-population involvement. We conceptualise four 'GenV engines' to accelerate solutions: (1) Discovery: Open Research data and biosamples to reveal causes, risks, needs and costs. (2) Intervention Hub: Real-world trials and natural and policy experiments testing what works. (3) 'What If': Simulation tools forecasting intervention impacts and costs. (4) StateLab: A learning system for innovation with many partners. This presentation will showcase how GenV's interventional capacity could address pressing health challenges through inclusive, real-time prevention and early intervention research.

O2: The causal relationships between leisure-time physical activity and body mass index in adulthood: a triangulation study

Anna Kankaanpää^{1,2}, Laura Joensuu^{1,2}, Ulf Ekelund³, Anni Pitkänen², Katja Waller², Teemu Palviainen⁴, Jaakko Kaprio⁴, Miina Ollikainen⁵, Sari Aaltonen⁴, Elina Sillanpää²

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Background: Previous studies have presented conflicting findings regarding the causal relationships between leisure-time physical activity (LTPA) and body mass index (BMI). Here, we use individual-level data and apply a triangulation framework that incorporates complementary methods to investigate the bidirectional causal effects between LTPA and BMI.

Methods: We used data from a longitudinal Finnish twin cohort with four measurement points spanning 36 years. The data included 22,696 twin individuals aged 18–50 years at baseline (52.4% women) of whom 8,527 had genetic data available. We applied three analytical approaches: Random intercept cross-lagged path model for longitudinal data, one-sample Mendelian Randomization (MR) and Direction of Causation (DoC and MR-DoC) twin models for cross-sectional data at each measurement point.

Results: All the three approaches provided evidence for causal effect from higher BMI on lower LTPA particularly at the later stages of follow-up. Only twin models suggested a negative causal effect from LTPA on BMI. The effects were similar in men and women.

Conclusions: Triangulating evidence across three methodologies provided support for a causal effect of higher BMI on lower LTPA, while evidence for the reverse effect was less convincing. The findings suggest that the role of high BMI in limiting LTPA becomes more important with advancing age and highlight the importance of accounting for timing when studying the causal effects.

O3: An intersectional analysis of sex, early-life socio-economic factors, and genetic risk on adult obesity: a Northern Finland Birth Cohort 1966 study

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Obesity inequalities arise from the interplay of social and biological factors, including sex, socioeconomic factors (SEF), and genetic risk. These factors are often examined separately, limiting understanding of how their combined effects shape obesity risk. Recent novel analytical approach allows these influences to be examined jointly.

We included 3,239 participants from the Northern Finland Birth Cohort 1966 with complete exposure and outcome data. Multilevel Analysis of Individual Heterogeneity and Discriminatory Accuracy (MAIHDA) examined how sex, early-life SEF, and genetic risk jointly relate to obesity in mid-adulthood. Individuals were nested within intersectional strata defined by combinations of these exposures.

The null model showed modest clustering of obesity across 48 intersectional strata (VPC = 12.3%). Predicted probabilities ranged from 8% to 40%, lowest among males with low genetic risk and socioeconomic advantage and highest among females with high genetic risk and socioeconomic disadvantage. Genetic risk explained nearly all between-stratum variance (PCV = 99.9%), whereas sex and early-life SEF contributed little independently. In the full model, between-stratum variance was fully attenuated (PCV = 100%), with modest discriminatory accuracy (AUC \approx 0.69).

These findings suggest obesity inequalities in mid-adulthood were largely structured by additive effects of sex, early-life SEF, and genetic risk rather than residual intersection-specific effects.

O4: Device-measured 24-h movement behaviors and metabolic profile in young adults

Kristin Suorsa^{1,2}, Joel Hynninen², Tomi Laitinen², Jarkko Heiskanen³, Tuija Leskinen¹, Jesse Pasanen¹, Juha Mykkänen², Olli Raitakari², Sari Stenholm¹, Katja Pahkala²

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Purpose: This study examined how 24-hour movement behaviors, i.e., sleep, sedentary behavior (SED), light physical activity (LPA), and moderate-to-vigorous physical activity (MVPA), associate with metabolic profile among young adults.

Methods: Overall, 335 young adults from the Special Turku Coronary Risk Factor Intervention Project (STRIP) used a wrist-worn accelerometer continuously for one week at the age of 26 years. Circulating metabolites were quantified by a nuclear magnetic resonance (NMR) metabolomics platform. Associations between 24-h movement behaviors and metabolic profile were analyzed using compositional data analysis, adjusting for sex, education, smoking, alcohol intake, and diet.

Results: Higher LPA was associated with a more favorable metabolic profile, while fewer associations were observed for other 24-h movement behaviors. Specifically, the theoretical reallocation of 30 min from SED to LPA from the mean 24-h composition (sleep 8.0 h, SED 12.8 h, LPA 1.7 h, MVPA 1.4 h) was associated with higher omega-6 fatty acids to total fatty acids (FA) and linoleic acid to FA (≈ 0.13 SD, $p < .002$ for both), as well as lower serum triglycerides, very-low-density lipoprotein (VLDL) particle size, total lipid and triglyceride concentration in VLDL particles, isoleucine and glycoprotein acetyls (from -0.10 to -0.18 SD, $p < 0.002$).

Conclusions: Our findings support a beneficial role of LPA in metabolic health and suggest underlying mechanistic pathways.

O5: Exploring biological pathways of heat exposure in pregnant women and infants in the climate change era: HIGH Horizons prospective birth cohort, Greece

Zacharoula Bogogiannidou¹, Dimitrios Papatheodorou¹, Georgia Ntouska¹, Konstantinos Tasiias², Christina I Messini³, Michalis Koureas¹, Ioanna Voulgaridi¹, Fani Kalala⁴, Varvara A Mouchtouri¹

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In the Horizon Europe–funded project HIGH Horizons, the Laboratory of Hygiene and Epidemiology, University of Thessaly, Greece, investigates the effects of heat exposure on maternal-child health to support strategies for protecting pregnancies in a warming climate. A prospective birth cohort will recruit ≥ 500 mother-child pairs, followed from the 1st trimester to the infant's 1st year. Women are enrolled at 12 weeks gestation and followed at 22 and 32 weeks, delivery, and 2, 6, and 12 months postpartum. Data collection includes questionnaires and forms capturing exposome factors like ambient heat exposure, lifestyle, diet, housing features, cooling practices, medical history, laboratory tests, fetal monitoring, clinical examinations, placental and umbilical cord morphometrics. Biological samples are collected to identify heat-related biomarkers while environmental exposures are assessed using ambient temperature and air pollution data, with a subset individually monitored by wearable devices. By March 2026, 487 women have been enrolled, and 311 pregnancies have been completed. Preliminary results ($n=221$) show a median gestational age of 38 weeks. Preterm delivery occurred in 8.4% (17/203) of pregnancies. C-sections accounted for 62% (129/207), while low birth weight was observed in 3.4% (6/176) of newborns. Further analyses will provide evidence on associations between pregnancy outcomes and exposome factors and insights into heat-related mechanisms affecting pregnancy.

O6: Municipality level changes in overweight and obesity in pregnant women in Sweden over two decades: evidence from the Swedish Medical Birth Register

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Background: Overweight and obesity among pregnant women have increased dramatically, posing significant risks for maternal and child health. While regional and sociodemographic inequalities in maternal BMI are well documented, little is known about how these patterns have evolved over time at the municipal level in Sweden. This study examined temporal and geographic changes in overweight/obesity among pregnant women in Sweden.

Method: Data from the Swedish Medical Birth Register for 1997, 2007, and 2017 were used to calculate prevalence of overweight/obesity at the first antenatal care visit. Municipal level variation in prevalence over time was visualized using maps. To examine sociodemographic associations, multi-level logistic regression analyses were performed.

Result: Overweight/obesity increased significantly in 260 of 290 municipalities. The maps showed a marked shift toward higher levels from 1997 to 2017. Several municipalities more than doubled their prevalence since 1997. Higher age (AOR 1.04–1.05), being born in Africa (AOR 1.38–1.57), and lower education (AOR 1.92–1.95) were consistently associated with overweight/obesity in 1997–2017. Municipal-level variation was small (ICC 0.01 in 1997 and 0.02 in 2007 and 2017).

Conclusion: Findings highlight a dramatic increase in overweight/obesity across municipalities. Although municipal level variation was minimal, the widespread rise underscores the need for public health strategies that address population level drivers.

O7: FILOMENE – a new French large couple-child cohort to study the relations between the exposome and children’s health

Barbara Heude¹, Valérie Benhammou¹, Romain Basmaci², the FILOMENE study group

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The environments we are exposed to, our lifestyles, and the incidence of certain pathologies, particularly rare paediatric diseases, are constantly evolving. These transformations raise new challenges in children’s environmental health and call for the establishment of a large-scale, contemporary paediatric cohort. The FILOMENE new French cohort has the ambition to include at least 100,000 children and their parents, starting from pregnancy. The overarching aim of this parent-child cohort is to characterize the exposome-related determinants of child health, development and diseases, including uncommon disorders.

The protocol currently under development includes longitudinal follow-up, combining active and passive data collection, particularly through linkage with environmental and medico-administrative databases. FILOMENE will serve as an open research platform, accessible to the international scientific community. A call for expressions of interest was organised in autumn 2025, targeting the entire French medical and scientific community. Nearly 200 expressions of interest have been submitted coming from a highly diverse range of teams representing a wide array of disciplines and geographic locations.

New cohorts dedicated to understanding the early determinants of child health and development are necessary to address new environmental and societal challenges and provide up-to-date evidence-based knowledge for public health decision-makers.

O8: Extensive sample and data collection on environmental exposures in a new Biofantti birth cohort

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Three major environmental crises, i.e., biodiversity loss, climate change and pollution, pose simultaneous threats to human health. Inclusion of individual-level data on these threats to birth cohorts provides an opportunity for addressing timely research questions. There is urgent demand for better understanding the health impacts of early-life exposure to 1) extensive (indoor) heat and humidity, 2) limited richness of microscopic and macroscopic lifeforms, and 3) various anthropogenic products.

We are establishing "Home, biodiversity and immunological health of infants and children (Biofantti)" birth cohort including 500 Finnish mother-child dyads. The main aim is to combine collections of environmental and human-origin biological samples. In the case of most infants, home is the place where the majority of the first year of life is spent. We collect detailed data on indoor conditions during infancy including measurements of indoor air quality, assessment of species across kingdoms of life, and measurements of microplastics and household chemicals. We also collect samples and data on infants' microbiomes, immunological and psychosocial development as well as growth are collected. The main outcome is the development of childhood asthma and allergic conditions.

We have not found other birth cohorts with such detailed description of infants' daily environments. We expect to produce novel understanding on health-impacts of early-life environmental exposures

O9: Future Finland Birth Cohort: optimizing the use of Finnish register and welfare clinic data to advance research on family and child wellbeing

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The Future Finland Birth Cohort (FFBC) is a recently launched nation-wide birth cohort led by the Finnish Institute for Health and Welfare (THL) and funded by the Finnish Cultural Foundation (SKR). FFBC comprises two arms: 1) National registers and other routinely collected data of all people born in Finland during 2025–2029 ($n > 200,000$), people moving to Finland and born during these years, and their parents and siblings, 2) An in-depth cohort will include questionnaire and biological data collected from the consented participants (abstracts Kajantie et al., Parkkola et al.). Key registers include e.g. perinatal data, specialty care diagnoses and certain social welfare services, medication purchases, and socioeconomic data as well as air pollution and other environmental exposures. Moreover, antenatal and child welfare clinics routinely collect data which is only partially usable largely because of technical barriers: one aim of FFBC is to harness this resource. We will prioritize data widely collected in a structured format, e.g., maternal Edinburgh Postnatal Depression Scale, and Lene screening test for child developmental disorders. We also aim to utilize unstructured data stored in electronic patient record systems. Overall, by enabling the large-scale and innovative use of data, the FFBC aims to advance pioneering research on enhancing the well-being, health, and human capital in Finland over the next century, with an emphasis on prevention and early intervention.

O10: Physical fitness in adolescents born preterm or small for gestational age in rural Malawi

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Background: Preterm and SGA infants face higher mortality and later cardiometabolic risk than term peers. Muscle strength and cardiorespiratory fitness predict long-term health and lower mortality, but evidence on effect of preterm or SGA birth on fitness mainly comes from high-resource settings.

Methods: Lungwena Antenatal Intervention Study enrolled 1,320 women to assess maternal antimalarial treatment effects during pregnancy. Gestational age was ultrasound-confirmed and children have subsequently been followed up. At 17 years follow-up, 818 adolescents were examined: 94 (11.0%) preterm appropriate for gestational age (AGA), 12 (1.4%) preterm SGA, 130 (15.2%) term SGA and 582 (67.8%) were born term AGA (reference). Muscular fitness was measured by handgrip strength and number of 30 second push-ups; cardiorespiratory fitness by heart rate after 4 min step test.

Results: Among comparison group, mean (SD) dominant-hand grip strength was 29.1 (6.3) kg and 26.1 (4.8) kg for boys and girls, respectively. In a pooled analysis adjusted for sex and age, handgrip strength was lower than in term AGA by 1.2 kg (95% CI 0.1–2.4) in preterm AGA, 3.9 kg (0.9–6.8) in preterm SGA, and 2.4 kg (1.4–3.4) in term SGA. Adjustment for height modestly attenuated the results, suggesting partial mediation through height. No group differences were observed in push-ups or heart rate.

Conclusions: Adolescents born preterm or SGA in a rural, low-resource setting had lower handgrip strength than term AGA.

O11: Supporting children’s decision-making in a large-scale birth cohort study through staff experiences in the Japan Environment and Children’s Study

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Background: Children in birth cohort studies develop decision-making capacity as they mature. Staff promote continued participation while respecting autonomy, making explanations and responses to children’s preferences important. Yet little is known about how these practices are enacted. This study examines the experiences of staff in the Japan Environment and Children’s Study (JECS), which has been following approximately 100,000 families for 15 years. It aims to clarify the practices that support continued participation and the dilemmas involved. This presentation focuses on children’s decision-making.

Methods: Between 2023 and 2025, in-depth interviews were conducted with 21 JECS staff who regularly communicate with participants. The data were analyzed using reflexive thematic analysis.

Results: Staff described supporting children’s understanding through age-appropriate explanations. They also expressed an awareness of the need to respect children’s preferences and avoid pressuring them to continue participation. Moreover, voluntary participation was found to be more complex within actual parent–child relationships.

Discussion: As participants have reached their teenage years, supporting children’s decision-making has become an important issue, and staff are exploring appropriate approaches. Given that direct benefits for the children providing data are not readily expected, careful attention is required to how children understand and interpret their research participation.

O12: How do birth cohort researchers beat the survey fatigue?

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Understanding disease etiology from a life course perspective requires data on individuals, ideally from preconception through the end of life. Information must include diverse domains, exposures, and developmental periods to ensure future scientific utility and hypotheses not yet conceivable at the time of data collection.

To provide sufficient statistical power, conception to death cohorts must be large. Loss to follow up—the Achilles heel of cohort studies—is a well known problem, which is why countries with comprehensive health register data available for research, such as Denmark, Finland, and Norway, have been considered particularly advantageous for initiating birth cohorts.

However, registries generally provide limited information on exposures, and many essential phenotypic characteristics can only be obtained through direct self-report from cohort participants. Diet, pain, body dimensions, well-being, and emotional states exemplify such data domains.

Thus, the continuous data collection needed to ensure rich, heterogeneous, and repeated measures across the life course increases the risk of participant fatigue, leading individuals to skip one or more data collection waves.

Danish National Birth Cohort (DNBC) participants are now 25 years old, and response rates are dropping. Experience shows that establishing and maintaining birth cohorts beyond childhood is challenging. So how do birth cohort researchers beat the survey fatigue? We will share our experiences from DNBC.

O13: Prenatal stress and adverse childhood experiences as risk factors for atherosclerotic cardiovascular disease – FinnBrain birth cohort study

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In-utero exposure to maternal stress reactions and reactions to adverse childhood experiences (ACEs) and other early life stress (ELS) are emerging as potentially modifiable risk factors for atherosclerotic cardiovascular disease (ASCVD). Animal studies suggest that stress induces alterations in multiple physiological systems, relevant for (AS)CVD development. Prospective research from pregnancy onwards in humans is very scarce. The main aim of this project is 1. to study how prenatal stress and ELS (incl. ACEs) are related to ASCVD risk indicators, i.e. carotid and/or abdominal aorta intima media thickness (aIMT) and 2. whether there are sex differences in these associations. 3. Potential mediators are investigated. The study population is drawn from the ongoing FinnBrain Birth Cohort Study (<https://sites.utu.fi/finnbrain/en/>). Child and parental aIMT is measured by standard ultrasonography procedures (GE Logiq S8), continuous ECG recording combined. Nine-year-old children were interviewed to assess their ACEs. Questionnaires, register data, anthropometrics, and biological samples have been used to assess prenatal stress, ELS and relevant covariates/mediators. Results (doi:<https://doi.org/10.1101/2024.06.27.24309083>) on maternal stress during pregnancy associating with infant aIMT thickness with sex differences (n=273) are presented. Preliminary findings on the 9-year assessments (n=470, analyses ongoing) will be presented in the conference. Implications are discussed.

O14: Early determinants of parental burnout in mothers of the NINFEA birth cohort

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Parental burnout (PB), characterized by emotional exhaustion in the parental role, negatively affects parents and children. This study examined whether maternal socioeconomic, health, and early caregiving factors are associated with PB when children are aged 10–16 years.

The study included 2067 mothers from the Italian NINFEA birth cohort who completed the 23-item PB Assessment 10–16 years after delivery. Exposures, collected during pregnancy and the first 2 years after delivery, covered sociodemographic factors (age, education, employment, income, family composition), maternal health and pregnancy characteristics, and early caregiving factors (daycare, breastfeeding, TV exposure, mother–child interaction, sleep, feeding, crying).

Higher PB scores were associated with maternal unemployment (Ratios of Means: 1.26, 95% Confidence Intervals: 1.05–1.52), low household income (1.37; 1.01–1.86), and maternal chronic conditions (psychiatric disorders: 1.42, 1.17–1.71). A higher frequency of infant crying was associated with higher PB scores (frequent vs. almost never: 1.35, 1.13–1.62), as was difficulty in soothing the child and more frequent feeding refusal. Higher levels of mother–child interaction were associated with lower PB scores.

PB when children are in late childhood/adolescence is linked to pre-pregnancy socioeconomic and health vulnerabilities and early caregiving factors. Early identification of populations at risk of PB is important to guide parenting support policies.

O15: Neighbourhood socioeconomic deprivation and ADHD and ASD during childhood in Finland, a country-wide register study (1994–2016)

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Background: Neighbourhood socioeconomic deprivation has been associated with the risk of Attention Deficit Hyperactivity Disorder (ADHD) and Autism Spectrum Disorder (ASD). We explored the association between high resolution socioeconomic disadvantage and childhood ADHD and ASD in Finland.

Methods: Our eligible study population were 1,140,336 singleton liveborn children born between 1994–2013 recorded in the Finnish Medical Birth Register. After excluding persons missing the outcome, exposure, or covariate data our analytical population was 999,070. We linked address at birth with information from other registers. Our exposure was a continuous neighbourhood disadvantage-score (NSES, range; -3.3–6.8) for 250m grid cells, based on mean education, income, and employment variables from Statistics Finland. Our outcome (ICD10) was ADHD (F90 codes) or ASD diagnosis (F84 codes) from specialist care during childhood (up to 10 years). We used Cox regression with follow-up until 2016, and adjusted for child sex and prematurity, maternal variables, urban-rural residence and parental ADHD or ASD.

Results: In the adjusted model, a one unit increase in the continuous NSES was associated with 34% increase in the hazard for ADHD (HR: 1.34; 95% CI 1.30–1.37), and 23% increase in the hazard for ASD (HR: 1.23, 95% CI 1.19–1.27).

Conclusions: Neighbourhood deprivation in early childhood associated with ADHD and ASD in Finland and should be considered as a modifiable risk factor in public health policies.

O16: Maternal inflammation and the development of white matter microstructure: a multi-cohort study

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The prenatal period is a critical epoch of neurodevelopment, during which inflammatory perturbations may alter the normal developmental trajectory. Extensive epidemiological literature suggests prenatal maternal immune activation (MIA) may increase the offspring's risk for neuropsychiatric disorders and neurological morbidities. However, the extent to which MIA affects neurodevelopment in the general population is unknown.

This research evaluates the effect of MIA on white matter microstructure, measured by diffusion tensor imaging (DTI) parameters, mean diffusivity (MD) and fractional anisotropy (FA). FA and MD are indicators of white matter integrity sensitive to fibre density, coherence, and myelination. Maternal C-reactive protein (CRP) is used as a marker of MIA. The research is conducted in population-based birth cohorts FinnBrain (Finland), CO-PSYCH (Denmark), PREOBE (Spain), Generation R (The Netherlands), and NFBC1986 (Finland), comprising 3,161 mother-child dyads.

Preliminary results indicate an association between prenatal maternal CRP and offspring FA at ages 1 month and 6 years, whereas no significant associations were detected in older offspring. We expect to further evaluate the results in a meta-analysis across all cohorts. We aim to provide information on white matter development in relation to prenatal inflammation in the general population to benefit research on typical brain development and neuropsychiatric conditions.

O17: Socioeconomic inequalities in childhood respiratory tract infections. Data analysis of >200,000 children in 16 birth cohorts from Europe and Australia

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Background: Respiratory tract infections (RTIs) are among the most common illnesses in childhood and a major cause of healthcare use. While socioeconomic inequalities are well documented for severe infections, less is known about social patterning in common childhood RTIs across countries and developmental stages.

Methods: We analysed harmonised individual participant data from 213,101 mother-child dyads in 16 birth cohorts participating in the EU Child Cohort Network. Maternal education during pregnancy was used as an indicator of early-life socioeconomic status. Outcomes were binary indicators of upper and lower RTIs reported at multiple ages. Inequalities were quantified using the Slope Index of Inequality and Risk Ratios using federated analyses via DataSHIELD.

Results: Upper RTIs were more common than lower RTIs. Although prevalences varied between cohorts, RTI prevalence showed a clear age pattern with peaks in early childhood. Across all cohorts combined, the highest prevalences were observed at ages 2 and 4 (22.5% and 28.8% for lower RTIs; 36.4% and 37.2% for upper RTIs). Overall, children from lower socioeconomic backgrounds tended to have higher RTI prevalence, with large variations across country settings.

Conclusion: Childhood RTI prevalence shows consistent age patterns, but both prevalence level and socioeconomic gradients vary across contexts. These findings suggest contextual factors may play a role in aetiology and prevention of childhood respiratory tract infections.

O18: Socioeconomic position, inflammation, and cardiovascular health in childhood and adolescence: an international multi-cohort study

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Background: Inflammation may partly explain socioeconomic inequalities in cardiovascular disease. As risk accrues from childhood, we investigated how cardiovascular health (CVH) in children and adolescents is patterned by socioeconomic position (SEP) and associated with inflammation.

Methods: We analysed data from: Barwon Infant Study (BIS, Australia, mean age 4.1y, n=708); Born in Bradford (BiB, UK, 9.3y, n=4576); Longitudinal Study of Australian Children's Child Health CheckPoint (LSAC-CP, 12.0y, n=1874); Northern Finland Birth Cohort 1986 (NFBC1986, 16.0y, n=9467); and Avon Longitudinal Study of Parents and Children (ALSPAC, UK, 17.8y, n=4875). SEP indicators in pregnancy/at birth were neighbourhood disadvantage, household SEP, and maternal education. Inflammatory markers were C-reactive protein and glycoprotein acetyls (GlycA). Measures of CVH were vascular structure/function and plasma lipids.

Results: From 12y, children with lower SEP had worse CVH; e.g., LSAC-CP children in the most disadvantaged neighbourhoods had higher pulse wave velocity ($\beta = 0.1$ m/s 95% CI [0.03-0.2]) than the most advantaged group. Higher inflammation was linked to worse CVH in all cohorts; e.g., 1 SD increase in GlycA was associated with higher systolic blood pressure in NFBC1986 (0.7 mmHg [0.5; 1.0]).

Conclusions: Child and adolescent CVH is patterned by SEP and linked to higher inflammation. Improving socioeconomic conditions and reducing inflammation may improve lifecourse risk trajectories.

O19: Early-life infections and life course health and disease in the EU Child Cohort Network

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Research into the developmental origins of, and life course influences on, health and disease has a significant gap: infections. Clinical and subclinical infections are nearly universal in early life and can induce structural and functional changes in developing organ systems with life-long consequences. Emerging evidence links common childhood infections—such as Epstein–Barr virus and *Helicobacter pylori*—to so-called non-communicable diseases ranging from respiratory to neurodevelopmental and cardiometabolic diseases. Identifying infectious determinants of non-communicable diseases offers substantial opportunities for prevention and early intervention. Here we describe a newly developed infectious data resource within the EU Child Cohort Network, a network of 37 pregnancy and childhood cohorts from 16 predominantly European countries. Infection data include information from questionnaires, health registries, and biomarkers across cohorts. We describe the scope, harmonization strategy, and research potential of this resource. Analyses from selected cohorts will be presented to illustrate feasibility and scientific value. Integrating infection data within the EU Child Cohort Network enables comprehensive investigation of how infections interact with genetic, environmental and social factors to shape health across the life course, strengthening the exposome framework and advancing understanding of non-communicable disease aetiology.

O20: The LIFE Child Study: a longitudinal perspective on child development and lifestyle diseases

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Understanding the complex trajectories of child development and the onset of civilization diseases requires a comprehensive, life-course approach. The LIFE Child study, established in 2011 in Leipzig, Germany, is a population-based longitudinal cohort designed to monitor healthy development from prenatal stages to early adulthood.

With over 6,000 participating children and their parents across more than 28,000 study visits, LIFE Child combines cross-sectional and longitudinal designs to capture a broad age range from the beginning. A cornerstone is its deep phenotyping approach, which integrates highly specialized medical, psychological, and sociodemographic assessments with an extensive biobank containing over 700,000 biological specimens.

Our research focuses on identifying critical time windows, early predictors, and resilience factors for modern health challenges. Key areas of investigation include the development of obesity, allergies, psychosocial adaptation, and dental health. The cohort also provides vital data on the impacts of social inequality, environmental factors, and the COVID-19 pandemic on child well-being.

By establishing robust reference populations and characterizing at-risk groups, the study facilitates interdisciplinary research aimed at improving pediatric care. With a robust infrastructure, LIFE Child continues to serve as an accessible scientific resource for translating life-course knowledge into better health outcomes.

O21: Exploring the exposome and unexplained variance in biological ageing – insights from a longitudinal twin study in adolescence and early adulthood

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Biological ageing begins before birth, with early-life exposures shaping late-life health and driving inequities. However, the composition of the ageing exposome remains undefined, partly due to a lack of agnostic investigations accounting for non-linearity and interactions. We aimed to identify childhood and adolescent exposures predictive of epigenetic ageing and explore the “missing” exposome.

In the FinnTwin12 cohort, >500 exposures were analysed using exposome-wide association studies and ML models (Knockoff Boosted Tree, sNPLS, & Boruta). Epigenetic age (blood DNA methylation at age 22) was estimated via GrimAge and DunedinPACE. Our exposure set explains ~28% of the variance in epigenetic age ($R^2_{\text{GrimAge}}=25.7\%$; $R^2_{\text{Duned-inPACE}}=30.8\%$). Predictors of increased epigenetic age included lifestyle and socioeconomic factors (smoking, alcohol, youth unemployment) and green space. Conversely, tree cover, vegetation index, neighbourhood age structure, and aerial black carbon predicted decreased epigenetic age. Twin modelling revealed that unexplained variance—the “missing exposome”—consists primarily of environmental factors unshared by twin siblings, distinct from the substantial genetic component captured by our model.

Our results underscore the need for non-linear models to reveal subtle environmental signals accumulating early in life. Because these predictors include modifiable systemic factors, they offer opportunities to alter health trajectories and mitigate inequity early on.

O22: Impacts of social isolation on health and wellbeing: a mixed methods study

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Background & Aims: Increasing numbers of older people are living alone. Living alone, as a proxy for social isolation and low social support, is important due to potential negative impacts on health and mortality. This is especially relevant in Northern Ireland, where about half the population live rurally, adding further risk. We aimed to explore the prevalence of social isolation in Northern Ireland, its association with mortality risk, and its impact on people.

Methods & Results: A mixed-methods approach was used. Data from the Northern Ireland Longitudinal Study provided a large, representative sample (~28% of the population). Census 2011 variables were linked with mortality data to 2018 (7-year follow-up). At baseline, ~13% lived alone, nearly half aged over 65. During follow-up, ~37% of older adults living alone died. Associations between living alone and mortality were examined, controlling for confounders. Four community workshops and 10 one-to-one interviews explored challenges of living alone in rural areas.

Conclusions & Further Study: Living alone was associated with increased mortality, particularly among older adults. To support this at-risk population, we are collaborating with AgeNI, an older persons charity, and Fermanagh & Omagh District Council, a predominantly rural council area in Northern Ireland.

O23: More than images: inclusion and collective deliberation in visualising ageing

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STAGE is a European Research and Innovation project exploring healthy ageing and multi-morbidity through a life-course perspective. Within this framework, we are developing a collection of 104 illustrations representing 100 years of life, portraying ageing through diverse and inclusive lenses.

To enrich this creative process, we conducted a deliberative exercise involving consortium members from different cultural backgrounds and generations. Participants engaged in discussion, critique and proposal of visual concepts using the Miro collaborative platform. This collective dialogue enabled reflection on representation, stereotypes and cultural nuance in the visual communication of ageing.

The process has strengthened inclusion within the project by incorporating multiple perspectives into the development of its visual language. It also aligns with Open Science principles, fostering transparency, shared authorship and socially responsible research practices. In this presentation, we share the lessons learned, challenges encountered and future directions of this collaborative initiative, highlighting its value as a tool for promoting inclusivity in research-based visual communication.

O24: Life course drivers of frailty: a longitudinal birth cohort study

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Individual risk factors have been linked to frailty, a state of reduced physiological reserve, but the cumulative contribution of life-course factors remains unclear. We aimed to quantify their contribution to frailty development.

We conducted a longitudinal analysis of 1,995 individuals from the Helsinki 1934–44 birth cohort. Forty-five life-course factors, from the prenatal period to midlife, were grouped into age, sex, early-life characteristics, socioeconomic position, lifestyle, and clinical measures. Frailty was assessed quarterly for two decades using an electronic frailty index derived from linked national hospital and primary care records, with follow-up to a median age of 85 years. Joint models estimated frailty trajectories and survival while accounting for differential mortality.

Life-course factors explained 41% of individual differences in frailty progression, with age accounting for most explained variance. Smoking, alcohol use, poorer diet, higher BMI, elevated systolic blood pressure, and disadvantaged childhood socioeconomic status were associated with faster frailty accumulation. Predicted frailty increased 2.5-fold between midlife and age 65 among non-smokers versus 2.8-fold among smokers; by age 85, the corresponding increases were 19.2-fold and 27.4-fold.

Frailty accumulation is largely age-dependent but is modified by lifestyle, cardiometabolic, and socioeconomic exposures across the life course, with widening divergence in trajectories at older ages.

O25: Longitudinal monitoring of biotic and abiotic exposures using wearable devices among pregnant women in Finland

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The exposome has the potential to fundamentally transform how human health is monitored, predicted, and protected across the life course. Pregnancy represents a uniquely sensitive time window, during which environmental exposures can have lasting consequences for both maternal health and fetal development. However, most studies rely on infrequent sampling or indirect exposure proxies, limiting their ability to capture individual-level exposure dynamics and lacking spatiotemporal resolution. To this end, we employed a bi-weekly personal exposure monitoring strategy using wearable exposometers to capture airborne exposures. This study included a cohort of 100 pregnant women in Finland who were monitored over a four-month period using. The wearable exposometers use filters to collect both airborne chemical and biological exposures, which were subsequently analyzed using next-generation sequencing and untargeted mass spectrometry to generate comprehensive personal exposome profiles. In this collaborative effort, we demonstrate that biotic and abiotic exposure data derived from wearables enable comprehensive, longitudinal profiling of the personal exposome. This high-resolution monitoring captures dynamic spatiotemporal exposure patterns that are not detectable through conventional ambient or episodic sampling approaches. These findings highlight the feasibility and value of using wearable exposometers for personalized environmental health exposure assessment during pregnancy.

O26: Longitudinal mapping of environmental exposures with individual-level geographic coordinates and wearable devices among pregnant women in Finland

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Individual-level biological and chemical exposure profiling will eventually change the way we monitor our health. Pregnant women represent a particularly vulnerable population as environmental exposures during pregnancy may affect both maternal and fetal health. To better understand individual-level longitudinal environmental exposures likely affecting our health we applied an innovative bi-weekly personal biotic and abiotic exposure monitoring with wearable exposometers and combined it with available high-resolution environmental exposome maps (e.g. air pollutants) to model personal exposures among 100 young women.

For the project we have recruited 100 Finnish women from which 30% were pregnant, and who all carried a filter-based exposometer sampler for four months with bi-weekly exposure sampling. Moreover, we combined the wearables exposure data with the previously developed geo-coordinated environmental exposome maps of Finland for integrated analysis of the environmental exposures.

Our results showed that with extensive aerosol sampling for biological and chemical exposure estimation combined with high-resolution environmental exposome maps allowed to infer specific environmental exposures associated to mobility, seasonality and health.

Taken together, we demonstrate that integrating individual-level wearable exposometer data with geo-coordinated environmental maps enables comprehensive biotic and abiotic exposome profiling for personalized health exposure assessment.

O27: Linking climate reanalysis data and birth registries: a scalable approach to study heat exposure effects on perinatal health

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Climate change is increasingly recognized as a determinant of health across the life course, yet impacts during pregnancy remain understudied. Population and hospital-based birth registries (BR) provide an opportunity to investigate how environmental exposures affect perinatal outcomes. When linked with climate datasets such as ERA5, it enables comparable analyses across countries.

We demonstrate the potential of linking BR with ERA5 to study short-term (7 days before delivery) and trimester-specific heat exposure during pregnancy. Using harmonized approaches, our analyses combined BR data from Africa, Europe, and Latin America (>5.4 million births). Heat exposure was characterized using indicators including temperature, the Universal Thermal Climate Index (UTCI), Wet Bulb Globe Temperature (WBGT), and heat index.

Across settings, short-term exposure to extreme heat was associated with increased odds of preterm birth (PTB). Trimester-specific analyses further showed that heat exposure during the third trimester was associated with increased risks of PTB, stillbirth, and abnormal fetal growth.

Linking BR with open climate datasets provides a scalable framework to study climate impacts on perinatal health, including temperate regions. Openly shared scripts and methods from the HIGH Horizons project will allow other countries to replicate and expand this work within their own BR systems.

O28: Relationship between early life adversities, gestational age and their joint effect on birth of the first child

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Preterm birth is associated with lower fertility, and early life adversities may modify this association. We investigated the role of gestational age (GA) at birth with having a first child, and the role of family adversity in this association. We used data from Finnish registers across 3 generations (F1 = index population, F0 = parents of F1, F2 = offspring of F1), including F1 individuals born in 1987–2008, followed until 2023. Early life adversity was defined as the number of adversities (0–3; death, disability, unemployment of a parent F0) between F1 birth and age 13 years. F1 GA was categorised as: GA1 = <34wks, GA2 = 34–36wks, GA3 = 37–38wks, GA4 = 39–41wks (reference). The primary outcome was having a first child (F2). We used Cox regression to estimate HRs and 95% CIs for independent and joint associations assessed using interaction of adverse early life exposures and GA on fertility. The models were additionally adjusted for F1 birth year and sex. Of the total F1 population (N=1,321,593), 14.1% had been exposed to any adversity and 18.2% had a first child by the end of the follow-up. Adversity was associated with higher hazard (HR: 1.16–2.73 for adversity categories 1–3), while GA was associated with lower hazard (HR: 0.74–0.98 for GA1–GA3) of having a child in adjusted models. Interaction between adversity and GA (p=0.03) was observed. Adverse family exposures and higher GA both independently influence having a first child, but the patterns of interactions were complex.

O29: Air pollution from livestock farms and lung function decline in neighboring residents over 7 years

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Longitudinal studies investigating air pollution from livestock farms and respiratory health effects in neighboring residents are lacking. The aim of this study was to assess the relationship between residential livestock exposures and lung function decline over a 7-year period in people living in livestock-dense rural areas in The Netherlands.

Spirometry was performed in 2014–15 and 2021–22 for 847 adults. We analyzed the annual rate of change in FEV1, FVC, FEV1/FVC, PEF and MMEF in relation to long-term exposure to livestock-related endotoxin and PM10 at the home address, predicted by dispersion modelling at baseline. Data analysis was performed using generalized additive models, adjusting for potential confounders.

No associations were identified between livestock-related endotoxin or PM10 and annual rate of change in lung function. Adjusted models showed that participants with a farm childhood had larger annual decreases in FEV1 (-5.63 ml/y, $p=0.018$) and MMEF (-11.15 ml/s per year, $p=0.032$), compared to those without. However, average baseline spirometry was higher in participants with farm childhood compared to those without.

We did not find evidence for a relationship between air pollution from livestock farms and lung function decline in neighboring residents. Longitudinal studies with more observations across the life course are needed to gain deeper insights into lung function trajectories and assess the impact of livestock-related air pollution in rural populations.

O30: ExpoMapper: an interactive tool for generating environmental exposures from individual-level geographic coordinates

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Environmental factors are increasingly incorporated into cohort research, but generating harmonised, high-resolution exposure measures is limited by incomplete and variable environmental maps across countries, pollutants, and time. Producing such data often requires strong geospatial modelling and advanced programming expertise. To address these challenges, we developed ExpoMapper, an R Shiny application that enables standardised, coordinate-based individual-level environmental exposure generation without the need for specialised geo-computation skills.

ExpoMapper provides an interactive interface for authenticated access to environmental maps within the EXPANSE Exposome Data Platform. Users may also upload their own environmental maps to generate the exposure estimates they require. ExpoMapper can be deployed locally in secure environments, making it suitable for studies involving sensitive address data. It streamlines exposure generation through a structured workflow allowing users to:

- i) pre-process geocoordinate data
- ii) explore environmental variables across domains and countries
- iii) upload coordinates and link them to selected maps
- iv) download harmonised, analysis-ready exposure data.

By simplifying data access, processing, and the secure use of geographical coordinates, ExpoMapper supports wider and more consistent integration of environmental information in population health research.



Poster Presentations

P1: Biotic and abiotic exposure estimated from blood among pregnant women

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Life-course profiling of biological and chemical exposures (i.e. human environmental exposome) will eventually change the way we monitor our health and environment. To estimate the effects of long-term environmental exposures to our health we used blood samples from a prospective population cohort for monitoring of biotic and abiotic exposures among pregnant women.

For the study we applied serum samples from 92 Finnish women from a population-based Finnish Maternity Cohort, a unique and extensive serum biobank of about one million Finnish women with first-trimester sera collected over 34 years. These serum samples were analysed with deep metagenome sequencing and non-targeted mass spectrometry for biotic and abiotic exposures.

Our findings revealed that using a prospective population cohort with extensive blood sampling it is possible to assess chemical and biological exposures and estimated their likely associations to our health trajectories.

P2: The prenatal exposome and childhood asthma in Finland, does climate change play a role?

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The human exposome and its link to chronic health outcomes are hypothesized to be strongly impacted by climate change. To test this, we have designed an exposome-wide association study (ExWAS) of childhood asthma focusing on prenatal exposures at the mother's residential address. This nationwide register study investigates temporal trends in the association between prenatal exposome factors and childhood asthma in Finland over three decades, the recommended period to assess climate change.

We identified three birth cohorts in Finland in 1990–91 (N=128,409), 2000–01 (N=110,010) and 2010–11 (N=119,098). Preliminary results show that childhood asthma, defined as the first hospital visit for asthma (ICD 10 J45–J46) between 5–16yrs, was diagnosed in 7,730 (6.0%), 7,314 (6.6%) and 6,169 (5.2%) children respectively. We are scaling up to an ExWAS, using sex specific Cox regression models, adjusted for confounders. The impact of climate change will be assessed by including climate anomalies, defined as the difference between climate conditions (e.g. temperature) during pregnancy and an historical climatic baseline, alongside other exposome factors such as pollen and air pollution.

Our findings will provide comprehensive evidence of how environmental and climatic changes shape childhood asthma risk. These insights could enable prevention strategies, guide adaptation policy, and might even support healthier ageing by mitigating the long-term respiratory impacts of climate change.

P3: Metabolites and lipids largely mediate the protective effect of breastfeeding on infant inflammation

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Background: Inflammation across the life course contributes to many adverse health outcomes. The extent to which breastfeeding may reduce infant inflammation burden, and the pathways involved, are poorly understood. We investigated effects of breastfeeding on infection and inflammation burden up to 12 months of age, and metabolomic and lipidomic biomarkers as potential mediators.

Methods: We used data at 6 and 12 months of age from 889 infants in the Barwon Infant Study, an Australian pre-birth, population-derived longitudinal cohort. Breastfeeding was dichotomised as any/none at 6 and 12 months of age. Plasma metabolomics was measured by nuclear magnetic resonance (NMR) (250 biomarkers) and lipidomics by mass-spectrometry (776 lipids). Infection burden was total number of parent-reported infections. Inflammation was measured by glycoprotein acetyls (GlycA), an NMR biomarker.

Results: Breastfeeding status at both 6 and 12 months of age was cross-sectionally associated with lower GlycA (mean -0.2 SD lower at both time points, $p < 0.001$ and $p = 0.02$), modestly lower infection burden, and extensive differences in metabolomic and lipidomic profiles. Several metabolites and lipids mediated the effect of breastfeeding on reducing inflammation, particularly plasmalogen class ether lipids.

Conclusions: Breastfeeding is associated with lower inflammation at 12 months. Several metabolic pathways are potential targets to optimise health for all infants, including those not breastfed.

P4: Early-life determinants and at-birth prediction of early childhood caries in a nationwide Finnish birth cohort

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Early childhood caries (ECC) is one of the most common chronic diseases in children and a preventable source of pain, infection, and healthcare burden. Most caries risk assessment approaches rely on clinical information collected after tooth eruption, limiting the use of earlier life-course information for risk identification. This study investigates whether ECC risk can be predicted at birth using nationwide Finnish registry data and examines early-life determinants of ECC in a nationwide birth cohort.

The study includes all children born in Finland between 2012 and 2017 (N≈320,000) using linked health and administrative registers from the Fin-Registry project. The outcome was a recorded diagnosis of ECC before age six. Prediction models were developed using information available at birth and evaluated using temporal validation. Regression analyses examined associations between ECC and key early-life domains including socioeconomic conditions, familial oral health indicators, perinatal characteristics, pregnancy exposures, and geographic context.

Prediction models showed moderate discrimination and identified high-risk subgroups. Determinant analyses revealed strong socioeconomic gradients and marked family clustering. Selected pregnancy exposures and perinatal growth patterns were also associated with ECC risk.

These findings suggest ECC risk stratification may be possible at birth and highlight the importance of early-life factors.

P5: Early life small quantity lipid-based nutrient supplementation and cognitive ability at 9 years: a follow-up study in Malawi

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Small quantity lipid based nutrient supplements (SQ-LNS) have been evaluated as a strategy to prevent malnutrition, but evidence on their long-term cognitive effects is limited. In a previous trial in Malawi, 1391 pregnant women were randomized to receive SQ-LNS(20g/d) during pregnancy and 6 months postpartum and SQ-LNS for children from 6 to 18 months of age or multiple micronutrients (MMN) during pregnancy and 6 months postpartum or iron and folic acid (IFA) during pregnancy only. In a follow up conducted when the children born to the trial were about 9 years, we evaluated the effect of early life SQ-LNS on cognitive ability using saccadic reaction time (SRT), manual choice reaction time (CRT), coloured progressive matrices (CPM) and early grade mathematics assessment (EGMA). We compared the SQ-LNS and non-LNS groups (MMN and IFA) and estimated the intervention effect using linear regression.

828 children participated (64% of the liveborn infants) with 53% being girls. There were no significant differences between SQ-LNS and control groups in SRT: adjusted mean difference = 2.05 milliseconds (ms), 95% CI (-1.57, 5.67); CRT: -10.45 ms (-43.84, 22.93); CPM: 0.15 (-0.34, 0.63) or EGMA: 0.03 (-0.08, 0.15).

Although we hypothesized that early SQ-LNS supplementation, which contains nutrient components that may positively influence neurodevelopment, would lead to better cognitive outcomes later in childhood, the findings at this age for the outcomes measured do not support this hypothesis.

P6: Gendered impact of violence against women on children's growth up to 2-years: analysis of MINIMat longitudinal cohort data from rural Bangladesh

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Violence against women (VAW) is associated with poor child growth, but evidence rarely considers growth trajectories or gender differences. This study examines how maternal VAW affects growth trajectories among boys and girls.

Data came from Maternal and Infant Nutrition Interventions in Matlab (MINIMat) longitudinal cohort in Bangladesh (1,636 girls; 1,704 boys). Mothers reported lifetime domestic violence at baseline, and children's height from birth to two years was used to assess growth. Trajectories were identified with latent class models, and impacts of VAW measured by multinomial logistic regression.

Stunting in the first 2 years was lower among girls than boys. Four distinct declining trajectories were identified in combined and gender disaggregated analyses: severely stunted, moderately stunted, mildly stunted, & non-stunted. In the combined sample, maternal emotional victimization increased the likelihood of being on the severely stunted trajectory (AOR-1.68; 95% CI: 1.17–2.41) compared to the non-stunted trajectory. This result held for boys (1.71; 1.06–2.76), but not for girls. Among boys, maternal emotional violence increased the likelihood of being on the moderately stunted trajectory (1.47; 1.01–2.12). Among girls, what increased the likelihood of being on the severely stunted trajectory was maternal controlling behavior (1.66; 1.08–2.54).

These results highlight to address controlling behavior and emotional violence within early childhood nutrition programs.

P7: Maternal and birth predictors of eating disorders in the Northern Finland Birth Cohort 1986

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Background: The study aims to identify the longitudinal effects of maternal and birth predictors of eating disorders (ED) in offspring.

Methods: The study uses a prospective sample comprising 9,362 pregnant women and 9,479 live-born infants from the NFBC 1986. The ICD-10 diagnoses of EDs were collected from registers and followed up until 2020. ED symptoms were defined using DSM-5 criteria based on follow-up questionnaires completed at ages 15 to 16 years. Logistic regression models were employed to identify the predictors of EDs.

Results: A total of 73 cases of EDs were identified among females, of which 31 were anorexia nervosa, and 22 were bulimia nervosa. Of the 277 cases with ED symptoms among females, 67 cases had restrictive type, 111 cases had binge eating type, and 99 cases had purging type of behavior. Factors such as small head circumference (<32 cm) (AOR = 7.45, 95% CI 1.52–36.5) and large birth length (≥54 cm) (AOR = 2.68, 95% CI 1.01–5.34) were significantly associated with any ED diagnosis. Maternal age of 27–35 years (AOR = 3.34, 95% CI 1.23–9.06) was associated with a restrictive type of ED symptoms. Any pregnancy complication (AOR = 1.87, 95% CI 1.03–3.37) and abnormal fetal growth (AOR = 2.13, 95% CI 1.02–5.05) showed a significant association with binge eating. Maternal smoking (AOR = 1.83, 95% CI 1.07–3.13) was associated with purging-type symptoms.

Conclusion: The results highlight the need for addressing modifiable risk factors to mitigate the occurrence of EDs.

P8: Longitudinal dynamics of epigenetic entropy in DNA methylation profiles from the Northern Finland Birth Cohort 1966

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Epigenetic entropy reflects the progressive loss of information content in DNA methylation (DNAm) patterns with age. Although it has been proposed as a biomarker of aging, the specific cytosine–phosphate–guanine (CpG) sites contributing to increased entropy remain unclear. We aimed to determine whether epigenetic entropy dynamics explain epigenetic aging rather than stochastic variation.

Using longitudinal DNAm data from over 1,500 individuals in the Northern Finland Birth Cohort 1966 at ages 31 and 46 years, we grouped CpGs according to their longitudinal methylation dynamics into entropic, steady, and anti-entropic categories to prioritize biologically informative loci. Entropy was then calculated for those CpG sets as well as sites used in established epigenetic clocks and previously reported differentially and variably methylated positions (DMPs and VMPs). Linear mixed models adjusting for age, sex, and cell composition identified CpGs with robust longitudinal changes.

Principal component analysis of entropy profiles showed entropic CpGs occupy a distinct dimension of methylation variability, independent of conventional clock CpGs. Moreover, only a small fraction of entropic CpGs overlapped with established epigenetic clocks indicating that entropy captures complementary aspects of epigenetic aging not represented by established DNAm signatures, providing an additional foundation for studying biological aging.

P9: Spouses of women with gestational diabetes are at increased risk of ischemic heart disease – FinnGeDi Study

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Background: We examined whether the cumulative incidence of IHD differs between the spouses of pregnant women with or without GDM.

Methods: In this longitudinal study, spouses participating in the Finnish Gestational Diabetes (FinnGeDi) study (n = 1185) completed a questionnaire at the time of recruitment in 2009–12 (spouses of pregnant women with (n=599) or without (n=586) GDM). Cumulative incidences of IHD were obtained from register data 10–13 years after the recruitment. The diagnosis of IHD was based on the Hospital Discharge Register and the Cause-of-Death Register (ICD-10 codes: I21–I25), or special reimbursement code for medication for chronic coronary artery disease. Odds ratios (ORs) with 95% confidence intervals (CIs) adjusted for age and BMI at the time of recruitment were calculated using logistic regression analyses.

Results: The spouses in the GDM group were 2.35 years older (95% CI 1.68–3.02) and had 0.78 kg/m² higher BMI (95% CI 0.34–1.21) during recruitment. Ten spouses died during the follow-up, from causes other than cardiovascular. Twenty of the 592 spouses (3.4%) in the GDM group had IHD, compared with <5/586 (0.5%) in controls (adjusted OR 4.8; 95% CI 1.4–16.6).

Conclusion: Having a spouse with GDM is associated with almost 5-fold odds of IHD within ~ 10 years, compared to having a spouse without GDM.

P10: The Flemish perinatal registry: from statutory registration to advances in perinatal health research for over 40 years

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Background: Population-based birth registries are key infrastructures for monitoring perinatal health and supporting epidemiological research. In Belgium, birth registration is legally mandated: a statistical form containing medical and socio-demographic information must be completed for every live birth and for stillbirths from 22 weeks of gestation or ≥ 500 g. In Flanders, these pseudonymized data are collected by the Study Centre for Perinatal Epidemiology (SPE), creating a registry covering all births in the region. SPE also collects data on neonatal admissions within the first 7 days after birth.

Methods: To illustrate the research potential of this registry, we will present several population-based analyses using SPE data, including studies on environmental exposures during pregnancy, induction of labour, predictors of neonatal admission, and variation in cesarean section rates between maternity units.

Results: The presented analyses illustrate the wide range of research questions that can be addressed using registry data. For example, studies have investigated environmental exposures, clinical practices, and health system factors affecting perinatal outcomes.

Conclusion: The SPE birth registry provides a powerful population-level data source for perinatal research. Future developments, including the planned eBirth 2.0 system linking hospital electronic health records, will further strengthen opportunities for longitudinal research.

P11: Future Finland – the birth of a new nationwide birth cohort

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The Future Finland Birth Cohort (FFBC) is a new national birth cohort coordinated by Finnish Institute for Health and Welfare and funded by Finnish Cultural Foundation to advance interdisciplinary scientific research and promote social sustainability over the next century.

FFBC comprises two arms. National registers and other nationally collected data: FFBC includes all individuals born in Finland during 2025–2029 ($n > 200,000$), along with parents and siblings. Families who move to Finland after the child's birth will also be included. In addition to well-established healthcare registers, FFBC will consolidate data collected in routine antenatal and child healthcare and education for use in research.

Questionnaire and sample data: A subset of pregnant women and their partners will be recruited primarily at the first-trimester ultrasound for biological samples collected in routine healthcare and questionnaires administered to both parents during pregnancy and infancy. Regional piloting is conducted in 2026–2027, followed by nationwide implementation in 2028. Later follow-up is being planned.

FFBC is a new national birth cohort transitioning from design to pilot phase. Among the spectrum of birth cohorts, FFBC will be characterised by (i) a relatively large study population, (ii) extensive use of register data, (iii) linkage of parents and siblings across generations, and (iv) integration of these data with questionnaires and biological samples obtained in routine health care.

P12: Future Finland Birth Cohort – piloting recruitment approaches in routine healthcare

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The Future Finland Birth Cohort collects data on all children born in Finland between 2025 and 2029. In addition to nationwide register data on all children (>200,000), questionnaire data and blood samples are collected from consenting participants starting in March 2026. During pregnancy and the first year of life, data collection includes four questionnaires (<29 gestation weeks, >29 gestation weeks, 4–6 months, and 9–12 months of age) and three blood samples (during pregnancy, umbilical cord blood, and newborn dried blood spot). Protocols for children beyond 12 months of age are under development. Recruitment will begin as a regional pilot in the capital area and expand nationwide in 2028.

Given the large target population, recruitment is integrated into routine prenatal care, primarily at the first trimester ultrasound visit offered free of charge to all pregnant families. These examinations are typically conducted by specialized midwives at ~30 sites nationwide, enabling efficient training for the study. Maternity clinics will also inform families, though their larger and more variable workforce increases training needs.

Participation rates will be monitored and recruitment effectiveness systematically evaluated. Interviews with recruiting professionals are ongoing to assess feasibility and acceptability. In summary, the cohort is piloting a recruitment strategy designed to maximize coverage through routine care, though limited clinician time may constrain implementation.

P13: Future Finland Birth Cohort – applying social and behavioural insights for supporting participant recruitment and communication

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Birth cohort studies such as the Future Finland Birth Cohort (FFBC) produce in-depth knowledge about the health of the (future) generation and impact of societal issues on well-being. Wide participation, engagement, and retention from diverse populations is vital in the consent-based part of the cohort. Previous research shows that trust, perceived gains, time constraints, socio-economic and cultural background affect willingness to participate in research. Furthermore, as families are recruited during routine antenatal visits, the recruitment process relies heavily on maternity healthcare professionals.

In this qualitative study, the research objectives are twofold. Firstly, we examine the factors that facilitate or hinder the participation of families in FFBC. Second, we assess how feasible and acceptable the recruitment process is for maternity healthcare professionals. Interviews with families and professionals will be conducted in spring 2026 in the regional pilot area of Uusimaa. In this symposium, we will present preliminary findings and offer evidence-based suggestions to enhance the accessibility and equity of FFBC. The findings are used to develop communication and recruitment materials and to help to establish a baseline design for the nationwide data collection, while presenting and critically assessing the feasibility of recruitment during routine antenatal care visits.

P14: Arctic Biobank – biobanking infrastructure of the Northern Finland Birth Cohorts for lifelong health and aging research

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Arctic Biobank - University of Oulu, established in 2020, provides research material from two Northern Finland Birth Cohorts (NFBC1966 and NFBC1986) and two aging cohorts (Oulu1935 and Oulu1945), which together cover approximately 16,000 biobank participants. The biobank manages longitudinal biospecimen including blood, serum, plasma, DNA, RNA, urine, fecal samples, saliva, hair, and white blood cells. Complementary phenotype data cover clinical measurements and analyses together with extensive self-reported information on lifestyle, environment, diet, sleep, mental health, medication, and disease history. Omics datasets further strengthen the resource, including genome-wide genotyping data, NMR metabolomics, DNA methylation profiles, telomere length measurements, and gut and oral microbiome data.

Arctic Biobank's resources are available for medical and life-science research, as well as for R&D activities, through the national FINGENIOUS® service (www.fingenious.fi), which provides unified access to Finnish biobanks. Biobank samples and data can also be linked to Finnish national registers via permits from Findata. Operating under the Finnish Biobank Act, GDPR, and harmonized national governance, Arctic Biobank ensures professional and ethically robust sample and data management. Its longitudinal and multigenerational design supports transdisciplinary research on lifelong health, early life determinants of disease, and mechanisms of aging in a northern population.

P15: Using the UK Census Longitudinal Studies to investigate long term change

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The Northern Ireland Longitudinal Study (NILS) is a population-based data resource enabling life-course research through linkage of successive census returns with administrative health and social data. NILS forms part of the UK Census Longitudinal Studies (UKCenLS), a coordinated infrastructure spanning Northern Ireland, England and Wales, and Scotland, providing a harmonised platform for cross-jurisdictional longitudinal analysis.

NILS supports rigorous investigation of demographic change, social stratification, migration, and health trajectories from early adulthood into later life. Its scale and linkage capacity enable examination of transitions, cumulative (dis)advantage, and the interaction between structural conditions and individual pathways. The resource is particularly well suited to trans-disciplinary research on lifelong health and ageing.

Linkage of the 2021 Census extends follow-up through a decade shaped by Brexit and the COVID-19 pandemic and introduces new domains including identity, housing, and long-term health. Updated cohort profiling highlights population ageing, increasing reporting of long-term conditions, and shifts in housing tenure and national identity.

This presentation demonstrates the analytical potential of the UKCenLS infrastructure for examining key demographic and health changes across the UK over time.

P16: Fostering lifelong resilience and deep pragmatism: a transdisciplinary proposal for integrating 'meta-morality' into longitudinal birth cohort studies

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Background: In a polarized world marked by instability and crises, small-group moral intuitions often fail to resolve “us vs. them” conflicts. Cultivating Meta-Morality—a shared moral common ground—can build resilient futures. A child-focused curriculum shifts learners from tribalism to Deep Pragmatism. The long-term effects of early cognitive reframing on lifespan development and healthy aging remain under-tested.

Aims: Integrate meta-moral cognitive assessments into longitudinal birth cohorts to test whether transcending tribalism in early life predicts adaptability, cognitive preservation, and mental-health resilience across aging.

Methods: The framework couples cognitive reframing with interactive dilemmas, moving from fast, emotional judgments to slower, deliberative reasoning that prioritizes a common currency of suffering-minimization.

Results & lifespan implications: Early workshops in Bangladesh show reduced in-group bias and higher emotional intelligence. We hypothesize that cohorts high in Deep Pragmatism will exhibit durable psychological flexibility, lower cognitive rigidity, and superior geriatric mental-health outcomes through major transitions.

Conclusion: Meta-morality equips individuals with cognitive tools to transcend local tribalism and embrace global citizenship. Integrating this framework into longitudinal and birth cohort research is a critical transdisciplinary intervention for safeguarding lifelong mental health and fostering resilient population.

P17: Entanglements of science and art – rethinking the ways of distributing knowledge

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The role of traditional research institutions, as producers and communicators of knowledge, is changing (Van Bavel et al. 2020). Although trust in science has not diminished, an increasing number of people consider themselves capable of interpreting research data. This is called the horizontalization of science and knowledge (Kristensson Uggla, 2019). The horizontalization of knowledge challenges institutions such as universities and forces them to find new ways to create knowledge and communicate research results.

One possible answer to this challenge is collaboration between science and art. Both disciplines share the goal of exploring the world and deepening our understanding of human experience. Until the eighteenth century, art and science were closely intertwined and often practiced in the same space, as exemplified by Leonardo da Vinci.

Through selected photographs we demonstrate how photographic practices can reveal temporal, material, and affective dimensions of complex phenomena. These examples suggest how interdisciplinary collaboration can broaden prevailing understandings, how scientific research can be conceptualized and represented to wider audiences through artistic practices, and how art can challenge traditional scientific perspectives.

The examples may foster dialogue between artists and researchers and provide a foundation for a collaborative initiative that integrates elements of health sciences and photographic art practices.

P18: Threading age: visualising ageism through participatory data physicalisation

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STAGE is a European Research and Innovation project investigating healthy ageing and multi-morbidity through a life-course approach. It addresses critical knowledge gaps by examining how biological, environmental, social, historical, and infrastructural factors interact across the lifespan.

During May 2025, an interactive artistic data physicalisation installation was developed to explore perceptions of ageism in everyday life. Participants were invited to complete two connected circuits using threads. In Circuit A, they reflected on how society perceives older adults through questions such as: At what age is someone considered old, how does society treat older people, and how are older adults seen in relation to technology or financial decision-making? In Circuit B, they responded to parallel questions focused on themselves, such as: When will you feel old, and how would you like to be treated in later life?

Over the course of one day, 45 complete responses were collected. The comparative analysis reveals a clear gap between perceived societal attitudes and personal aspirations for ageing. While society is seen as associating older age with decline, dependence or indifference, participants imagine their own ageing as characterised by autonomy, competence, dignity and continued contribution. These findings highlight the persistence of age-related stereotypes and emphasise the importance of reframing ageing as an evolving and valuable stage of life.

P19: Heritability estimation of BMI across early life from birth to age 15: sex-specific insights from the Northern Finland Birth Cohorts 1966 and 1986

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Body Mass Index (BMI) is a highly heritable, polygenic trait whose genetic architecture shifts dynamically across early life. From birth through adolescence, distinct transitions—the infant adiposity peak, the adiposity rebound around age 5~6, and the pubertal growth spur—represent critical developmental windows during which genetic influences on BMI intensify or recede. Accurate single nucleotide polymorphism (SNP) -based heritability estimation of early BMI can provide targeted opportunities for preventing childhood obesity. Traditional quantitative genetic analyses usually provide static estimates at isolated time points with using a simple longitudinal model that failed to model the entire growth curve and to identify genetic variants whose effects are specific to a growth phase.

In this study, we employ Random Regression Linear Mixed Models with Legendre Polynomials to repeated BMI measurements from birth to age 15 in the Northern Finland Birth Cohorts 1966 and 1986. This method estimates continuously changing SNP-heritability, decomposed into components governing baseline level, rate of change, and growth curvature. We hypothesise that SNP-heritability peaks at developmentally distinct windows that differ between boys and girls. Identifying these windows may reveal when genetic factors most powerfully shape BMI variability across early life, ultimately guiding more precisely timed and biologically informed interventions against childhood obesity and underweight.

P20: Body size and blood pressure of 9-year-old children born preterm or small for gestational age (SGA) in Malawi

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Preterm birth or being born SGA are associated with increased risk of cardiovascular diseases. Evidence of the role of birth characteristics to the burden of CVD health has emerged mainly from research conducted in high-income countries. To test the applicability of the hypothesis in low-resource setting, we used data from the follow-up of the iLINS-DYAD-M trial, measuring body size with standard anthropometrics and blood pressure with oscillometric blood pressure monitor in 9-year-old Malawian children born preterm (n=50) and term SGA (n=176) and compared the outcomes with children born term AGA (n=556). We used multiple linear or logistic regression with adjusting for socioeconomic and pregnancy-related factors.

Compared with term AGA children, height, weight, and BMI z-scores (coefficient (95% CI)) in term SGA group were -0.40 (-0.55, -0.26), -0.51 (-0.64, -0.38), -0.36 (-0.49, -0.22), and -0.31 (-0.56, -0.03), -0.30 (-0.57, -0.04), -0.15 (-0.38, 0.08) in preterms. These associations stayed significant after adjustments, with the exception of BMI z-scores among preterms. Differences in systolic (SBP) and diastolic blood pressure (DBP) were 0.52 (-0.98, 1.95), 1.68 (-0.24, 3.79) in term SGA, and 2.20 (-0.38, 4.79), 3.74 (0.66, 6.91) in preterms. In conclusion, birth characteristics were related to body size but not to blood pressure at 9 years of age among Malawian children, except for higher DBP observed among those born preterm.

P21: Adolescent weight gain trajectories and their associations with biological aging: a genetically informed study

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High body mass index (BMI) may accelerate biological aging, and genetics may partly explain this association. In this study, path analysis was used to investigate the hypothesized association between genetic predisposition to higher BMI and accelerated biological aging while considering the mediating role of adolescent BMI trajectories. Furthermore, the causal effect of adolescent BMI on later biological aging was examined using Mendelian randomization (MR). Participants were from the Young Finns Study ($n=3596$, ages 3–18 at baseline), followed from 1980 to 2018–2020. Biological aging was estimated using DNA methylation based epigenetic clock DunedinPACE at three follow-ups (ages 15–56, $n=2045$). BMI trajectories were modeled from BMI measured at ages 9, 12, 15 and 18 using latent growth curve modeling. Genetic predisposition to BMI was quantified using polygenic risk score (PRS) (941 genetic variants). Both BMI-PRS and single variants were used as instrumental variables in MR. The results indicate that higher level of adolescent BMI partly mediates the association between higher BMI-PRS and accelerated biological aging from late adolescence to middle adulthood. MR analyses supported a positive causal effect of adolescence BMI on biological aging (causal estimate=0.020, $p=0.019$). In conclusion, high BMI in adolescence can accelerate aging, especially in genetically predisposed individuals. This may have long-term implications for life expectancy.

P22: Suboptimal dietary patterns are associated with accelerated biological aging in young adulthood: a study with twins

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The role of diet quality in biological aging during early adulthood remains unclear. We examined associations between dietary patterns and biological aging in young adult twins at the individual and within-pair levels. Participants were 826 twins aged 21–25 from the FinnTwin12 cohort. Diet was assessed with a food frequency questionnaire, diet patterns were derived using latent class analysis, and biological aging was estimated by the GrimAge and DunedinPACE epigenetic clocks. Associations were analysed with linear regression. GrimAge acceleration was slower in the Plant-based, Health-conscious, and Balanced-average patterns than in the High fast food (FF), low fruits and vegetables (F&V) pattern and faster in the Western with infrequent fish pattern than in the Balanced average, independent of sex, energy and alcohol intake, and smoking (Model 2). Adjustment for BMI and sports participation (Model 3) weakened the associations, but the Balanced average versus High FF, low F&V difference remained significant. DunedinPACE was slower in the Plant-based than in the High FF, low F&V and the Western with infrequent fish patterns in Model 2. Most associations were replicated in within-pair analyses in all and in dizygotic pairs, but not in monozygotic pairs, suggesting partial genetic confounding. Diets high in FF and low in F&V are linked to accelerated biological aging in young adults. Lifestyle clustering and genetic confounding must be considered when interpreting the results.

P23: Associations of habitual coffee intake with testosterone and cardiometabolic markers: the Northern Finland Birth Cohort 1966 study

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Background: Coffee is widely consumed and rich in bioactive compounds, yet its associations with detailed metabolic and hormonal profiles remain unclear.

Objective: To examine relationships between habitual coffee intake, circulating metabolites, cardiometabolic markers, and sex hormones in midlife adults.

Methods: Cross-sectional data from 2,264 participants (47% men) of the Northern Finland Birth Cohort 1966 at age 46 were analyzed. Sex-stratified Spearman correlations and multivariable linear regression models were adjusted for BMI, education, smoking, physical activity, and alcohol intake.

Results: Higher coffee intake was associated with a leaner body composition, including lower total and visceral fat and higher skeletal muscle mass, despite similar BMI. In both sexes, coffee consumption was inversely associated with branched-chain amino acids. In men, higher intake was also linked to lower fasting and post-load insulin and a distinct androgen profile. Adjusted models showed positive associations with total testosterone and SHBG, and inverse associations with free testosterone and the free androgen index. In women, associations were mainly limited to higher SHBG and lower free androgen fractions.

Conclusions: Habitual coffee consumption was associated with a leaner phenotype and sex-specific metabolic and hormonal profiles, particularly in men. Longitudinal and intervention studies are needed to clarify causality.

P24: Prospective bidirectional associations between leisure-time physical activity and adiposity across the transition from middle to old age

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Aim: To explore bidirectional associations between leisure-time physical activity (LTPA) and adiposity indicators from middle to old age, and to assess sex differences.

Methods: Data were drawn from the Finnish Oulu45 cohort study (N=682, 56% female). Self-reported frequency of LTPA of ≥ 30 min and laboratory-measured adiposity indicators—body mass index, waist circumference, waist-to-height ratio, and waist-to-hip ratio (WHR)—were collected at approximately ages 57 and 70. Multi-group cross-lagged panel models were used to assess sex-specific associations between LTPA and adiposity indicators, adjusted for socio-demographic and lifestyle confounders.

Results: In men, weak negative cross-sectional associations were found between LTPA and all adiposity indicators at both timepoints ($r = -0.12$ to -0.28 , $p \leq 0.039$). In women, only LTPA and WHR at 70 were significantly associated ($r = -0.120$, $p = 0.031$). No cross lagged effects were observed from LTPA at 57 to adiposity at 70. In contrast, higher values in all adiposity indicators at 57 predicted lower LTPA at 70 in men ($\beta = -0.123$ to -0.160 , $p \leq 0.035$) and women ($\beta = -0.274$ to -0.321 , $p < 0.001$). Sex did not moderate the associations ($p \geq 0.2$).

Conclusions: Across the transition from middle to old age, adiposity predicts subsequent LTPA but not vice versa. Supporting physical activity engagement among middle aged adults with elevated adiposity may help reduce functional decline later in life.

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P25: Association with long-term exposure to heat or cold strain measured by UTCI and blood pressure in middle age

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Hypertension is a major global health issue associated with comorbidities and premature mortality. The effects of temperature and weather on blood pressure (BP), especially in the context of climate change, need further study. Long-term cold exposure is suggested to increase BP, whereas heat decreases it; however, most studies focus on short-term exposure and one-dimensional temperature indicators. We will investigate the association between long-term exposure to heat and cold strain, expressed as the Universal Thermal Climate Index (UTCI), and BP in middle-aged adults.

The exposure is defined as UTCI, a biometeorological index that assesses heat and cold stress on the human body based on environmental factors and thermal balance. We will link mean monthly UTCI to health data from the Northern Finland Birth Cohort (NFBC1966) using geolocated residential histories. Participants (N=3984) will be followed from age of 31 to 46.

We will first identify UTCI trajectories over a 15-year period using growth mixture modelling. Next, we will assess the association between trajectory membership and changes in BP between ages 31 and 46 using linear mixed model. Models will be adjusted for sex, education, unemployment, physical activity, unhealthy diet, alcohol use, smoking, depression, obesity, diabetes, and season of clinical examination. The results will provide insight into the link between long-term exposure to heat and cold and BP and can help adapting to the changing climate.

P26: Intersectional analysis of sex and socioeconomic position on the development of obesity in later life using the Northern Finland Birth Cohort 1966

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Sex and socioeconomic positions are determinants of obesity, but they are typically studied in isolation. Intersectional approaches examine how these positions combine to shape health outcomes across the life course. We aimed to examine their intersectional effects on midlife obesity risk in a longitudinal setting.

We included 3,195 participants from the Northern Finland Birth Cohort 1966 (38.8% male). Sex, income, education, and employment status at age 31 formed 24 intersectional strata. Longitudinal Multilevel Analysis of Individual Heterogeneity and Discriminatory Accuracy (MAIHDA) was used to estimate additive and multiplicative effects on obesity risk at age 46.

At age 46, 31.0% of participants had obesity. Females had 36.0% lower odds than males (95% CI 0.53–0.78), and high income was associated with 28.0% lower odds compared with low income (95% CI 0.57–0.92). Education and employment showed weaker associations. VPC was 2.3%, indicating low discriminatory accuracy. 90.0% of between-strata variance was explained by additive effects. Predicted probabilities ranged from approximately 20.0% to 45.0% across strata.

We observed low discriminatory accuracy across intersectional strata, with most differences due to additive effects, including higher risk among men and a clearer gradient by income than education or employment. These findings would support universal policies over targeted interventions.

P27: Sociocultural factors of obesity by different socioeconomic positions across the life-course in high income countries: a systematic review

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Obesity remains a major public health concern, and growing evidence shows sociocultural (SC) factors contribute to its development across the life-course. Socioeconomic position (SEP) is strongly linked to obesity and may modify the influence of SC factors, contributing to unequal outcomes. This systematic review followed a PROSPERO-registered protocol (CRD420251084190) and PRISMA 2020 guidelines. We reviewed evidence from high-income countries on associations between SC factors and obesity across life-course, focusing on SEP as an effect modifier. Risk of bias was assessed using the Newcastle–Ottawa Scale. We found 42 studies, mostly American, N ranging from 841 to 2.35M participants. Ethnicity was the most examined SC factor, followed by nativity, immigrant generation, acculturation, Aboriginal status, and family meal patterns. Education was the most common SEP indicator. Among ethnicity-focused studies, 24 reported higher obesity risk in minority groups. In most studies SEP modified these associations: 81% reported stronger associations between SC factors and obesity in lower SEP groups, 12% weaker associations in higher SEP groups and 7% mixed patterns. Most studies had low risk of bias. This review shows that associations between sociocultural factors and obesity vary by SEP, with stronger links more often observed in lower SEP groups. Effective obesity prevention should consider how SC and socioeconomic factors interact to support equitable and targeted interventions.

P28: DNA methylation in muscle tissue of adults born preterm with very low birth weight: evidence from sibling study

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Shorter gestational age and lower birth weight are associated with increased risks of adult cardiometabolic diseases, but the biological mechanisms underlying long-term outcomes remain unclear. Epigenetic modifications may represent one potential mechanism. This study aimed to determine whether adults born preterm with very low birth weight (VLBW, <1500 g) exhibit differences in DNA methylation patterns in skeletal muscle tissue compared with their same-sex siblings.

Epigenome-wide DNA methylation profiling was performed on vastus lateralis muscle biopsies using the Illumina EPIC 850K array in 60 participants born with VLBW (mean age 29.4) and 55 sibling controls (mean age 29.1) from the Adults Born Preterm Sibling Study. Methylation differences were analyzed using linear mixed-effects regression models adjusting for age, sex, smoking, maternal smoking, batch effects, and estimated muscle cell-type composition.

We identified 48 differentially methylated CpGs (FDR <0.05), most of which were hypermethylated in the VLBW group. Several CpG sites mapped to genes involved in metabolic regulation, insulin signaling, inflammatory pathways, and cellular signaling. We performed exploratory mediation analyses and identified 122 CpGs potentially mediating the association between VLBW and appendicular skeletal muscle mass adjusted for BMI, and 43 CpGs potentially mediating the association between VLBW and the glucose increment during the oral glucose tolerance test (120–0 min).

P29: Sleep duration moderates the cognitive ability in adults born with very low birth weight: a sibling-control actigraphy study

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Background: Very low birth weight (VLBW; <1500g) adults often score lower than term-born peers on tests assessing cognitive abilities. While sleep is associated with neurocognition, its role in moderating the relationship between VLBW and IQ remains unclear.

Methods: A sibling-control study evaluated 62 adult sibling pairs, one born at term and the other born preterm with VLBW (n=124; mean age = 28.9 ± 4.2 SD years; 50.8% female). Actigraphy was recorded over 13.2±2.8 nights. Measured metrics included total sleep time and sleep timing. Cognitive ability was assessed via the Wechsler Adult Intelligence Scale IV. Applied linear Mixed Models accounted for nested individual and family structures.

Results: VLBW status predicted lower Full-Scale IQ (Estimate = -6.7, p<0.01, 95% CI [-11.44, -2.08]). For VLBW adults, each additional hour of sleep was associated with a 3.6-point IQ increase (p=0.040, 95% CI [0.07, 7.13]), a relationship non-significant in term-born siblings. Sleep timing (bedtime; p>0.05) did not moderate outcomes.

Conclusion: These findings suggest that sleep duration is positively correlated with cognitive abilities in adults born preterm with VLBW. This highlights a disproportionate link between sleep and cognitive performance in this population, whereas term-born siblings lack this association.

P30: Body composition in mid-adolescence in relation to preterm birth: a cohort study from rural Bangladesh

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Evidence on body composition in adolescents and adults born preterm (<37 weeks) or small for gestational age (SGA) is mixed. Most evidence originates from relatively affluent countries. We aimed to assess the effect of preterm birth and SGA on body composition in adolescents in a low-income setting.

We used 15-year follow-up data from the MINIMat cohort in Bangladesh. Body composition was assessed by leg-to-leg bioelectrical impedance. The sample (n=2300) for the analysis included 162 preterm appropriate for gestational age (AGA), 45 preterm SGA, 1018 term SGA, and 868 term AGA adolescents. Outcome variables were lean body mass (LBM), LBM adjusted for height, and fat percentage (FP). We utilized sex-stratified multiple linear regression models adjusted for age.

Male adolescents born preterm AGA (mean difference: -2.45 kg; 95% CI: -3.84, -1.07), preterm SGA (-3.11; -5.63, -0.59), and term SGA (-1.93; -2.73, -1.14) had lower LBM than their term AGA peers. Among females, preterm SGA (-2.41; -3.75, -1.06) and term SGA (-1.48; -1.87, -1.08) children had lower LBM than term AGA. While adjusted for height, the effect in LBM attenuated in male preterm AGA (-1.03; -1.84, -0.22) and female term SGA (-0.53; -0.84, -0.23) children. In addition, male preterm AGA children had lower (-1.60%; -2.99%, -0.20%) FP compared to term AGA.

Findings suggest that preterm birth and SGA are associated with lower LBM and lower FP in adolescence. Moreover, some of the LBM differences might be due to shorter height.

P31: Being born preterm and having a child in young adulthood: the mediating role of mental health and educational attainment

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People born preterm (birth <37 gestational weeks, PTB) are less likely to have their own children, but the underlying mechanisms remain understudied. We investigated the extent to which mental disorders diagnosed early (age <15 yrs), and subsequent education mediate the PTB-having a child association, and whether these pathways differ by sex. Data were drawn from Finnish registers for 1,277,580 individuals born in 1987–2008, followed from age 15 yrs until 2023. Causal mediation analysis partitioned PTB's total effect into a natural direct effect and an indirect effect. We calculated the proportion mediated (PM) sequentially via mental disorders (diagnosis with ICD-9/ICD-10) and later education. The incidence of having a child was reduced across PTB categories (Hazard Ratio, 95%CI 0.57, 0.52–0.62 in women born very preterm; 0.71, 0.65–0.77 in men). In women, mental disorders and education did not mediate the association. In men, early mental disorders significantly mediated the reduced incidence. For very preterm men, mental disorders explained 7% (95% CI: 3–11) of the effect, remaining unchanged after adding education. For moderately late preterm men, mental disorders mediated 10%, increasing only marginally to 11% (PM 11%, 95% CI: 6–15) when education was added. Early mental disorders mediate the lower probability of having a child in men, with subsequent educational attainment providing no substantial additional mediation. This sequential pathway is non-mediating in women.

P32: Preterm birth and relationship status and parenthood in young adulthood – the role of sex and neurosensory impairments

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BACKGROUND: People born preterm are less likely to have a partner or become parents than those born at term. We used Finnish register data to assess the role of G1 (generation G1) gestational age (GA) in the likelihood of having a partner and/or offspring (G2) at age 25–36 years, and the roles of sex and neurosensory impairments (NSI).

METHODS: We included 751 241 G1s (5.0% preterm, 8.3% NSI) born in Finland in 1987–2000, alive on 31/12/2023. We used adjusted multinomial regression to assess the association between GA (ref: 39–41wks) and a composite variable of ever being married/ in a same-sex relationship, or having a child; had a partner (P), or a child (C) up to 12/2023:

- (1) no partner + no child(ren) ([NPNC, reference);
- (2) no P + yes C [NPYC];
- (3) yes P + no C [YPNC]; and
- (4) yes P + yes C [YPYC]).

Analyses were stratified by sex and G1 NSI status.

RESULTS: In all, 63.7% of G1s were at category NPNC and 16.0% in YPYC. Compared to term born men, men born preterm (<37 wks) had 10–50% lower odds of having a child, regardless of partnership status (YPYC or NPYC). However, this association was not evident for having a partner and no child (YPNC). All associations attenuated among men without NSI, while men born at 23–27wks with NSI had 60% lower odds for a child (YPYC or NPYC). Among women, the associations were highly similar to those of men but did not differ by NSI.

CONCLUSION: Diagnosed NSIs may be a risk factor for not having a child among preterm men, but not among women.

P33: Cumulative early-life exposure to climate extremes and childhood wheezing

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Background: While the short-term health effects of climate extreme events are documented, evidence on their long-term impacts remains limited. We investigated whether cumulative exposure to climate extremes during the first 6 years of life is associated with respiratory health at age 7.

Methods: Using data from the NINFEA Italian birth cohort, we estimated exposure to extreme heat (days with max temperature ≥ 35 °C or min temperature ≥ 20 °C), heavy precipitation (days ≥ 30 mm), wildfire-related PM₂₅ (days ≥ 5 $\mu\text{g}/\text{m}^3$), and drought (months with SPEI < -2.0). Respiratory health at age 7 was assessed based on wheezing symptoms in the previous 12 months. We applied logistic regressions adjusted for spatio-temporal factors, and maternal characteristics.

Results: Among 4,645 children included in the analysis, wheezing prevalence at age seven was 5.9%. Increased odds of wheezing were observed with higher exposure to extreme heat (maximum temperature: adjusted OR per 10-day increase = 1.12, 95% CI: 1.01–1.23; minimum temperature: adjusted OR = 1.02, 95% CI: 1.01–1.04) and heavy precipitation (adjusted OR per 10-day increase = 1.06, 95% CI: 1.01–1.11). No clear associations were observed for wildfire-related PM₂₅ or drought exposure.

Conclusions: Cumulative exposure to extreme heat and heavy precipitation during early childhood may contribute to respiratory symptoms at school age. These findings highlight the importance of considering the long-term health impacts of climate extreme events.

P34: Adolescent stress and health in Northern Finland: an intersectional analysis of familial and adolescent perceived social positions

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While the effects of different social positions on adolescents' health and wellbeing are well evidenced, the contribution of their intersections with health inequalities remain unclear. This study investigated how intersections of adolescents' familial and perceived social positions relate to self-rated stress and health.

Using data from the Northern Finland Birth Cohort 1986 (N=7,792, ages 15–16), two sets of intersectional strata were formed. The first included familial social positions: parental education, household income, family type, and sex. The second included adolescent-perceived social positions: family economic status, academic performance, number of friends, and sex. The data were analysed using a Multilevel Analysis of Individual Heterogeneity and Discriminatory Accuracy (MAIHDA).

Variance partitioning coefficients (VPC) showed that both sets of strata explained a significant proportion of adolescent self-rated stress [VPC_familial = 11.72% (6.94%–19.17%), VPC_adolescent = 13.22% (8.70%–19.85%)] and health [VPC_familial = 3.22% (1.39%–6.20%), VPC_adolescent = 16.00% (10.81%–22.86%)]. The additive effects accounted for 89.1% to 99.4% of the between-strata variance.

Differences in adolescent stress and health were mostly driven by the additive effects of social positions, rather than synergistic or buffering effects at their intersections. Adolescents' perceived social positions could be a potential target for adolescent health and wellbeing interventions.

P35: Individual and joint associations of adolescent and young adult alcohol use and smoking with midlife psychological distress – a multicohort study

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The aim of this study is to examine the separate and combined associations of heavy episodic drinking (HED) and daily cigarette smoking in adolescence and early adulthood with midlife psychological distress across four prospective cohort studies from Finland and Sweden.

Data for this prospective multicohort study were drawn from four longitudinal studies: the 'TAM' cohort (N=1334), the 'FinnTwin16' (N=4409), and the 'Northern Finland Birth Cohort 1966' (NFBC1966, N=7147) from Finland, and the 'Individual Development and Adaptation Study' (IDA, N=514) from Sweden. HED was measured as drunkenness or using 60g or more pure alcohol on one occasion, smoking as daily cigarette use, and psychological distress using the 12-item General Health Questionnaire.

No clear evidence for individual or combined associations of HED or smoking in adolescence or early adulthood with midlife distress were found. The results were overall systematic across all cohorts.

In contrast to prior research based on diagnostic criteria, this multicohort study found no evidence that heavy episodic drinking or daily cigarette smoking in adolescence or early adulthood—individually or combined—are associated with midlife psychological distress when assessed using symptom measures. These findings, overall consistent across four longitudinal cohorts, suggest that the relationship between early substance use and later mental health may be weaker than previously assumed when studied using non-diagnostic indicators.

P36: Pneumonia morbidity in young and working aged adults; risk factors and immunological properties in a large birth cohort

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Pneumonia susceptibility arises from a combination of demographic characteristics, underlying disease burden, lifestyle, and environmental exposures. Recurrent pneumonia episodes may rarely lead to suspicion of secondary or primary immunodeficiency. While pneumonia risk factors are well established among the elderly, the young and working-aged adult population is not equally well studied.

The focus of this study is to explore the role of immunological properties in relation with pneumonia risk at population level. We included Northern Finland Birth Cohort 1966 participants of the 46-year follow-up study with consents to use their data in combination with the national register data (Study arm 1) and with measured serum immunoglobulins (Study arm 2). Finally, careful characterization of the cohorts aims to identify subpopulations with a high pneumonia risk in association with evidence of immunological abnormalities for further genetic analyzing (Study arm 3).

We identified novel high-risk subpopulations for pneumonia, such as young males. Smoking shapes immunity unfavorably; it rises serum IgE, lowers serum IgG and exposes to pneumonia. Our findings indicate that both low and high serum IgG levels are surprisingly common in our unselected study population and both deviations may indicate immunological insufficiency. Our preliminary data supports the view that not only high IgE, but also low IgE concentration is associated with adverse events such as pneumonia and autoimmunity.

P37: Psychiatric disorders in the offspring of mothers with antenatal stress and fatigue

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Maternal stress during pregnancy is commonly associated with adverse obstetric outcomes, but long-term psychiatric outcomes of antenatal fatigue in the offspring remain unknown. The aim of this study is to identify potential associations between maternal antenatal fatigue and stress, and offspring psychiatric diagnoses.

This study is based on the Northern Finland Birth Cohort 1986 (N = 9432 children). Maternal fatigue was assessed with a one-item self-reported question and antenatal stress by elevated blood pressure, blood glucose and hsCRP levels. Cohort members with psychiatric diagnoses were identified using the Finnish Care Register for Healthcare. To study associations between maternal stress and fatigue and offspring psychiatric diagnoses, chi-square tests and Cox proportional hazard regression models were conducted utilizing gestational smoking, alcohol use, marital status, maternal education and parental psychiatric diagnoses as confounders.

Female offspring whose mothers had fatigue and/or stress had more borderline personality disorders, while male offspring had more mental disorders related to alcohol use and mood disorders, compared to participants without maternal antenatal fatigue or stress.

Results suggest maternal antenatal fatigue and stress may have associations with offspring psychiatric disorders, but clinical implications are uncertain. Considering the prevalence of fatigue and stress among pregnant mothers, preventive and supportive interventions could be favourable for offspring.

P38: Are maternal antenatal fatigue and stress associated with offspring mental health outcomes?

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Fatigue is a common symptom of a normal pregnancy but is also associated with mental and somatic illness. Antenatal fatigue has been found to be associated with elevated risk for obstetric complications and neonatal problems, but the research on antenatal fatigue and offspring mental health is scarce. Psychological distress affects about 20% of mothers during pregnancy and it has been associated with adverse psychiatric outcomes in the offspring. Stress can also manifest as allostatic overload resulting in organic malfunction. In this research project, we have studied maternal reports of antenatal fatigue and objectively measured stress-related factors (gestational hypertension, diabetes, and inflammation) in association to mental health in the offspring. The study is based on two birth cohorts: The Northern Finland Birth Cohort 1986 (NFBC1986, N=9362) Avon Longitudinal Study of Parents and Children (ALSPAC, N=14541) in which the mothers have reported their experiences of fatigue during pregnancy by questionnaires, and the children have been followed from pregnancy until adulthood with questionnaires and clinical examinations. Maternal sera (N=7200) and national registry data are also available for NFBC1986 participants. Offspring mental health was evaluated with clinical psychiatric questionnaires at age 7–8 and 15–16 years in both cohorts, and with register diagnoses in the NFBC1986. Antenatal fatigue was associated with offspring internalizing symptoms in both cohorts.

P39: Gene environment interactions in mental health trajectories of youth (Youth-GEMs)

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Adolescence and early adulthood are critical stages for the onset of psychiatric disorders, but how mental health develops as young people seek and receive treatment remains unclear. Improving understanding of mental health trajectories during treatment could help identify early markers of severe mental health problems as well as actionable targets and guide the development of new targeted therapeutic interventions. Therefore, this large prospective cohort study aims to examine mental health trajectories among help-seeking young people to identify transdiagnostic risk and resilience factors.

One thousand young people aged 12–24 who are seeking help for mental health problems will be recruited across six European countries to obtain a representative sample. The participants, along with their caregivers, will be asked to complete a comprehensive multi-dimensional battery, which will assess genetic, clinical, and cognitive predictors. During a two-year follow-up period, data will be collected using a mobile application and through formal assessments conducted every 3–6 months. The two follow-up approaches will provide high- and low-frequency data about participants' mental health, including wellbeing, sleep quality, habits, and physical and social environment. Utilising artificial intelligence, we will first identify mental health trajectories using both high- and low-frequency data and detect key baseline measures that predict the direction of the trajectories.

P40: Internalizing and externalizing symptoms from youth to adulthood in the normal population: correlates in limbic system morphology

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Brain limbic system morphological alterations have been observed amongst youth with internalizing and externalizing behaviors (IEB) both in typically developing and diagnosed individuals. It is, however, unknown if youth symptoms or their change associate with structural alterations in early adulthood, after completion of the critical neural adaptations of adolescence. In this study, we explore how IEB, assessed at age 16 years and symptom change until 26 years relate to morphology of key limbic structures of amygdala, hippocampus, and prefrontal cortex. The sample consisted of 420 participants of the prospective NFBC1986. IEB were measured using self-reported questionnaires and brain imaging data were collected via magnetic resonance imaging at 26 years. Linear regression analyses were controlled for the confounding effects of participant age, sex, BMI, smoking and alcohol use at 16 years, and maternal education. The results showed no associations between behavioral symptoms in youth or symptom change from adolescence to adulthood and limbic system morphology at 26 years. Association trend between internalizing behavior and lateral orbitofrontal cortex showed overlap with previous literature of brain alterations in youth. This study provides new insights into youth IEB in the general population and suggests that symptoms or their change until adulthood may not pertain to long-term macrostructural alterations in the limbic system, and the effects may be diluted by adulthood.

P41: Parent- and teacher-rated childhood emotional and behavioral problems predicting adulthood depressive symptoms and disorders

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Multiple informants are commonly used when studying children's mental health, however their predictive value is unclear. The aim of the current study is to examine whether teacher and parent reported emotional and behavioral problems in childhood predict depressive symptoms in adulthood. The Northern Finland Birth Cohort 1986 (NFBC1986) is an unselected, population-based sample recruited during mid pregnancy. At age 8, children's behavior was assessed by both teachers and parents using the Rutter Scales. At age 34, cohort members completed a survey including the 15 item depressive symptoms scale from the Hopkins Symptom Checklist. Final sample included 1053 males and 1698 females. Logistic regression analysis including several childhood and adulthood covariates was used to study associations separately for males and females. Childhood emotional problems predicted adult depressive symptoms only among females, with the highest risk seen for those who scored high on both teacher and parent ratings (adjusted Odds ratio, aOR = 2.16, 1.37–3.42). Childhood behavioral problems rated by parents associated with depressive symptoms among females (aOR = 1.37, 1.01–1.86), whereas associations attenuated after adjustments among males. Gender differences and information from multiple informants should be considered when assessing childhood predictors of later psychiatric outcomes. These findings may help guide the development of early targeted interventions.

P42: Do psychotic symptoms predict future psychotic disorders in adolescent psychiatry inpatients? A 17-year cohort study

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Individuals with a psychiatric inpatient admission in adolescence have a high risk of future schizophrenia-spectrum disorders (SSDs). The aim of this study was to examine whether psychotic symptoms predict subsequent SSDs in inpatient cohorts. The sample consisted of adolescents (aged 13–17) admitted to psychiatric inpatient care (Oulu, Finland) from April 2001 to March 2006. Specialized health care use and diagnoses were followed up in national health care registers until June 2023. Cox regression was used to predict SSDs by the presence of baseline psychotic symptoms. Of 404 adolescent inpatients admitted with non-psychotic mental disorders, 28% (n = 113) reported psychotic symptoms: 17% (n = 68) subthreshold and 11% (n = 45) full threshold. By the end of follow-up, 23% of the total cohort went on to be diagnosed with an SSD. Subthreshold psychotic symptoms did not differentiate patients who would subsequently develop SSDs (HR = 1.42, 95%CI = 0.81–2.50). Full-threshold psychotic symptoms, on the other hand, were associated with an increased risk of subsequent SSDs (HR = 2.00, 95%CI = 1.12–3.56). Most subsequent SSDs (83%), however, occurred in individuals who had not reported threshold psychotic symptoms during inpatient admission. In conclusion, despite high overall risk of subsequent SSDs in the sample, SSDs were not predicted by subthreshold psychotic symptoms. Threshold symptoms were associated with future SSDs, but with low sensitivity.

P43: Prognostic factors for adulthood psychosis in adolescent psychiatry services: a longitudinal total birth cohort study

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Introduction: As many as half of all psychotic disorders diagnosed by age 28 in the population emerge in individuals who attended child and adolescent psychiatry services, highlighting important opportunities for psychosis prediction and prevention. An important next step is to identify prognostic factors for later psychotic disorders within this clinical population. We assessed a large number of potential prognostic factors for adult-onset psychosis within adolescent psychiatry services.

Methods: Population-wide register data for all individuals born in Finland 1987–1992. Using survival analyses, we assessed a range of clinical, service use, and sociodemographic characteristics in adolescent psychiatry patients as prognostic factors for adult-onset psychotic disorders.

Results: Among adolescent psychiatry patients (N=27,626), cumulative risk of psychosis between ages 18–30 was 8.5%. Within-service significant prognostic factors for psychosis included: total number of mental disorder diagnoses received in adolescence, young maternal age, premature birth, older age at first adolescent psychiatry contact, history of child psychiatry contact, psychiatric inpatient admission in adolescence, parental history of psychosis, and parental history of inpatient psychiatric admission.

Conclusion: Our findings demonstrate that a number of clinical, service use and sociodemographic factors have prognostic significance for the development of adulthood psychosis among adolescent psychiatry.

P44: Association between severity of depressive symptoms and related costs: a Northern Finland Birth Cohort 1966 study

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Depression is a prevalent mental health condition that can lead to high healthcare costs, especially when it is more severe. Primary health services are the first point of contact in Finland; thus, it is essential to understand how severity of depressive symptoms influences costs in primary healthcare settings.

We used the Northern Finland Birth Cohort 1966 (N=5604) survey data collected when participants were aged 46 in 2012. The severity of the depressive symptoms was measured using the Beck Depression Inventory. Information on healthcare visits, including costs from mental health services, health centers, private clinics, and occupational healthcare were gathered. Two-step statistical models were used in the analysis. Logistic regression examined the likelihood of incurring costs, and linear regression assessed associations between the severity of depressive symptoms and mental and overall primary healthcare costs.

The odds of incurring mental healthcare costs and overall costs were higher (adjusted OR: 7.5; 95% CI: 3.7–15.4; and aOR: 1.7; CI: 0.6–4.8) for those with severe depressive symptoms compared to those without depressive symptoms. Those with severe depressive symptoms incurred €881 (CI: 344.5–1418.5) more mental health-related and €758 (CI: 610.0–906.8) more overall primary healthcare costs than those with no depressive symptoms.

Our results underline the importance of early interventions for depression, which may reduce societal healthcare costs for this condition.

P45: Social capital and depression – a 15 year bidirectional analysis

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Background: The relationship between depressive symptoms and social capital is complex. Prior research has raised the question of a potential bidirectional association between the variables. Yet due to limited longitudinal research, no conclusions have been able to be reached.

Methods: The main aim of this study was to examine whether social capital level at age of 31 (baseline) predicts depressive symptoms at age of 46 (follow-up), or if depressive symptoms at 31 predict social capital at 46. Data were derived from two surveys (baseline at 31 years and follow-up at 46 years) of the Northern Finland Birth Cohort 1966 (N=5106). Depressive symptoms were measured using the depression subscale of the Hopkins Symptom Checklist-25 and social capital dimensions consisting of emotional and instrumental support received from five different sources from the individual's social network.

Results: Lower emotional and instrumental support – particularly from one's spouse, colleagues, and close friends – at age 31 was associated with higher depressive symptoms at age 46. Instrumental support demonstrated a stronger predictive association with depressive symptoms than emotional support. Depressive symptoms at age 31 were only weakly associated with reduced social capital at age 46.

Conclusions: The findings suggest that it is more likely that low support predicts depression than the reverse. Consequently, our findings provide only limited evidence for a bidirectional relationship between the two.

P46: Environmental inequality and mental health: a geospatial analysis in the Northern Finland Birth Cohort 1966

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Environmental inequality (EI), defined as unequal environmental risk exposure across socioeconomic groups, may contribute to mental health disparities. This study maps EI based on neighborhood socioeconomic disadvantage (NSED) and PM_{2.5} across Finland and examines its association with depressive symptoms at age 46 in the Northern Finland Birth Cohort 1966 (NFBC1966).

NSED was derived from Statistics Finland's 1×1km Grid Database (2010) and linked to annual PM estimates (2010) from the ELAPSE project. EI was defined using binary NSED (<1=low, ≥1=high: most disadvantaged) and PM (high=3rd tertile). Among populated Finnish grids, 1.7% (n=289) showed EI (high-NSED/high-PM). After linkage to NFBC1966 residential address at age 46 (2012-2014), 7194 individuals were included: 3.0% (n=218) lived in EI areas, 54.1% (n=3894) in low-NSED/high-PM, 3.7% (n=264) in high-NSED/low-PM, and 39.2% (n=2818) in low-NSED/low-PM areas. Associations between EI (reference: low-NSED/low-PM) and depressive symptoms will be assessed by Beck Depression Inventory II using ordinal logistic regression, adjusted for sex, education, marital status, employment, income, household size, housing quality, physical activity, smoking, and alcohol use.

By integrating high-resolution spatial data with individual mental health measures, this study will provide evidence on whether PM-related EI contributes to depressive symptoms in middle age and highlight priority areas for equity-focused environmental health policy.

P47: Clinical and therapeutic implications of super-enhancer-driven 3D genome regulation at the 2q31 prostate cancer risk locus

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Although GWAS has identified numerous prostate cancer (PCa) susceptibility loci, the clinical relevance and therapeutic implications of noncoding risk variants remain poorly defined. Here we integrate GWAS signals with eQTL, epigenomic, and chromatin interactions to uncover actionable regulatory logic at the 2q31 risk locus. We identify a PCa-specific super-enhancer (SE) harboring risk variants rs12621278 and rs13414928 that drive long-range chromatin interactions with the oncogenic transcription factors DLX1/2. Functional perturbation using CRISPR-mediated enhancer deletion suppresses DLX1/2 expression and impairs tumor cell migration, supporting a direct role in disease progression. Mechanistically, TEAD1 preferentially binds the rs13414928-containing enhancer and cooperates with AR to sustain SE-driven oncogenic transcription. Disruption of TEAD1 attenuates AR signaling and reduces malignant phenotype. Importantly, a TEAD1-derived six-gene expression signature robustly predicts biochemical recurrence, metastatic progression, and overall survival across independent patient cohorts. These findings provide a direct mechanistic link between germline susceptibility variants and clinically relevant transcriptional programs in PCa. By defining a TEAD1-AR-super-enhancer axis that connects inherited risk to disease progression, our study identifies a potential biomarker for risk stratification and highlights TEAD1-dependent enhancer regulation as a therapeutic vulnerability in PCa.

P48: The conundrum of nc886 – methylation pattern with clear association to ancestral origins with no genetic determinants?

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nc886 locus displays a maternal polymorphic imprinting pattern and has been suggested to mediate the DOHaD hypothesis. Suboptimal pregnancy conditions have been associated with increased proportions of children with non-methylated nc886 locus, and this methylation pattern in turn has been linked with poorer cardiovascular health. At population level, the proportion of individuals with an imprinted nc886 locus varies, with populations of African, European and Asian ancestral origins respectively comprising of ~80%, ~75% and ~65% of individuals with imprinted nc886 locus. Our aim was to identify the genetic determinants of nc886 imprinting status using the GODMC2 consortia, with more than 60000 European study participants from more than 35 cohorts.

In the meta-analysis, 95 SNPs across 3 genomic regions were associated with the methylation status of nc886 locus ($p < 5 \times 10^{-8}$) but the effect sizes of these SNPs were not adequate for them to be causal. Our results did reveal that minor allele carriers of lead SNPs rs3746580 (PABPC1L), rs10053962 (near nc886) and rs11769787 (NOBOX) were less likely to exhibit imprinted nc886 locus than major allele homozygotes.

Despite extensive analysis, the population-level pattern of nc886 methylation remains unexplained. Our results are however intriguing, as both PABPC1L and NOBOX genes play essential roles in growth and maturation of oocytes, further linking the nc886 methylation status with oocyte development and early pregnancy conditions.

P49: Effect of infant vitamin D supplementation on epigenome-wide DNA methylation

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Early life exposures can affect individual's epigenome and lead to long-term health consequences. Data on vitamin D and epigenetic alterations are limited. We aimed to examine whether higher vitamin D supplementation dose compared with a standard dose affects epigenome-wide DNA methylation (DNAm) in 1-year-old infants.

In the Vitamin D Intervention in Infants (VIDI) study, 987 infants were randomized to receive daily vitamin D supplementation of 10µg or 30µg from age 2 weeks until age 2 years. To analyze DNAm with Illumina EPIC 850K array, we randomly selected 286 participants at age 1 year with available DNA samples isolated from whole blood within intervention groups and sexes. Epigenome-wide association analyses were conducted via R packages Meffil and rGREAT adjusted for sex, parental smoking status, maternal age and education, infant length-adjusted weight and age, cell count estimates and batch effect.

We observed no significant epigenome-wide DNAm differences between group-10 (n=143) and group-30 (n=143) ($p < 9.4 \times 10^{-8}$). We observed one differentially methylated region overlapping with gene PPM1H showing demethylation in group-30 than group-10 (FDR $p = 0.014$).

Our results indicate small effects of higher vitamin supplementation of 30 µg compared with standard dose of 10 µg on epigenome-wide DNAm in infants aged 1 year. Possibly affected genomic locations relate to metabolic processes including cholesterol metabolism as well as phosphorylation processes.

P50: Clinical usefulness of genetic risk scores in the prediction of gestational diabetes

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Background: Genetic risk scores (GRS) have been proposed to be useful in the prediction of gestational diabetes mellitus (GDM). We tested whether GRS adds predictive value to clinical risk factors for GDM.

Methods: GRS was calculated from 14 GDM-associated SNPs (Cissé et al. 2025, *BMJ Open Diabetes Res Care*) for 998 women with GDM and 973 controls, using genome-wide data from the Finnish Gestational Diabetes study. Data were randomly divided into the training and the validation sets. In the training set, multivariable logistic regression was used to create predictive models for the combination of clinical variables (maternal age, pre-pregnancy body mass index, family history of type 2 diabetes, and GDM in an earlier pregnancy) and to further combine them with GRS. The area under the receiver operating characteristic (ROC) curve (AUC) were calculated to analyze the added value of GRS.

Results: In the GDM group, standardized GRS was higher than in the controls (0.21, SD 0.98, vs 0.21, SD 0.97; OR 1.56, 95% CI, 1.42–1.71). The AUC values for GDM were 0.773 (95% CI 0.744–0.802) with clinical variables and 0.798 (95% CI 0.770–0.825) with clinical variables and GRS. No difference was observed between the AUC values ($p = 0.223$).

Conclusion: The clinical usefulness of the GRS composed of 14 genome-wide significant SNPs as a diagnostic tool for GDM is limited. In the future, genome-wide GRSs capturing more GDM-associated variants are to be explored.

P51: Lower blood cell mitochondrial DNA abundance associates with higher all-cause mortality and aging: a 30-year prospective epidemiological study

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Recent epidemiological studies with follow-up times up to two decades suggest blood cell mitochondrial DNA (mtDNA) abundance as a promising biomarker for aging and disease, though findings remain inconsistent and underlying pathomechanisms unclear. We investigated blood mtDNA abundance as a clinical biomarker and explored biological pathways driving its variation.

The OPERA cohort (Oulu Project Elucidating Risk of Atherosclerosis) comprises 1045 individuals assessed in the 1990s and followed for over 30 years, with a second visit in the 2010s. We quantified mtDNA abundance in blood samples from both time points with real-time quantitative polymerase chain reaction and compared it with clinical and epidemiological data. Transcriptomic profiling was assessed by RNA sequencing in 452 follow-up samples. At both time points, lower blood mtDNA levels were associated with increased overall morbidity and mortality. MtDNA abundance showed a small, but statistically significant decrease over two decades. Transcriptomic analysis linked lower mtDNA levels to enhanced inflammatory pathways in immune cells.

Our findings highlight blood mtDNA as a potential biomarker for aging and morbidity, with lower levels possibly linked to inflammatory processes. With three decades of follow-up, these results support its use in preventing morbidity and mortality of diverse etiology. Additionally, blood mtDNA emerges as a promising inflammatory biomarker, warranting further exploration in future studies.

P52: Male youth BMI is associated with epigenetic pace of aging of their descendants in maternal line in two generations in post WWII Finland

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Evidence supporting transgenerational inheritance in humans includes associations between pre-pubertal food availability and grandchild health and mortality. Here, we investigate the effect of early adulthood (18–22y.) male BMI on the epigenetic pace of aging, a measure of general well-being, in two generations of their descendants.

Weight and height data from military conscription of men born between 1897 and 1937 (n=1246) were obtained from the Finnish Army in World War II Database (FA2W). The conscripts were linked to their children and grandchildren who participated in the Young Finns Study (YFS), for whom DNA-methylation-based DunedinPACE is available (total n=925). Linear regression models were adjusted for the conscript's age, social class and East/West Finland origins, as well as the descendant's age, sex, BMI, and smoking-status.

The conscripts' BMI had an inverse association with the DunedinPACE of their daughters (p=0.003, B=-0.01, n=200) and daughters' daughters (p=0.02, B=-0.01, n=153) but not their sons or daughters' sons. Change of one BMI unit is associated with 1% change in epigenetic pace of ageing of the descendants.

Our results indicate that low BMI is associated with a higher pace of ageing in two generations of offspring, especially in the female line. In contrast with previous research, which used population-level harvest records, individual-level data allow us to show evidence of a transgenerational effect in a cohort with a healthy BMI range.

P53: Fragmented biodiversity in urban residential environment and childhood asthma and atopic dermatitis

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Asthma and atopic dermatitis are increasingly common chronic immunological disorders, which substantially impact children's quality of life and pose significant burden on healthcare. Recent research suggests that early-life exposure to biologically diverse natural habitats may mitigate the risk of developing these immunological disorders, potentially through the enrichment of human microbiota and enhancement immunity. However, for children live in mosaic city landscape, how fragmented biodiversity components and children's spatial connections to them may influence later health remains understudied.

We will follow all children born between 2005 and 2015 in the Finnish cities of Helsinki, Tampere, Turku, and Oulu, using national register dataset NORDCAP. Each child's residential history will be linked to environmental datasets describing remnant habitat quality, plant species richness, and soil microbial diversity around the home during ages 0–3 and from age 3 to censoring. Landscape patterns, including connectivity, aggregation and shape complexity, will also be evaluated to examine how urban structure modifies children's potential exposure to biodiverse environments. Using Cox proportional hazards models, the study will assess influence of these residential environment characteristics on the risk of asthma and atopic dermatitis by the age of 12.

This study aims to provide evidence for designing of healthier, biodiversity-rich urban environments for children.

P54: Genetic architecture of pain heterogeneity in endometriosis

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Background: Endometriosis is a chronic gynecological condition characterized by ectopic endometrium-like tissue outside the uterus, leading to pelvic inflammation, tissue damage, and chronic pain. Pain response is highly heterogeneous, suggesting a partial genetic contribution. To identify genetic factors differentiating endometriosis with abnormal pain response from non-abnormal, genome-wide association studies (GWAS) were conducted.

Methods: GWAS analyses were performed for endometriosis stratified by pain response using FinnGen data, including 20,190 endometriosis cases and 130,160 controls.

Results: We identified twenty-one genome-wide significant loci associated with endometriosis in pain status-stratified analyses. Several loci mapped to genes previously implicated in endometriosis, including TSHZ3 and RNLS. Among loci more strongly associated with abnormal pain response endometriosis, one mapped to KMT5A, encoding the primary H4K20 mono-methyltransferase. Loci more strongly associated with non-abnormal pain response included WNT4, a regulator of female sex determination and reproductive tract development. Endometriosis also showed high genetic correlations with multiple pain phenotypes, including joint, limb, and thoracic pain.

Conclusion: Our GWAS analyses identified differences in the genetic architecture between endometriosis with abnormal and non-abnormal pain response, supporting partially distinct genetic contributions to pain response heterogeneity.

P55: Implementing brain age prediction models in population-based neuroimaging cohorts

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Brain age estimation has emerged as a promising biomarker for studying brain development, aging, and psychosis risk. While the Brain Age Estimation model “brainageR” has shown robust performance across clinical cohorts, its generalizability to young adult population cohorts remains untested. This study aims to implement and evaluate the brainageR model for predicting brain age using T1-weighted MRI data from the Northern Finland Birth Cohort 1986 (NFBC 1986). The model applies a Gaussian Process Regression framework to estimate individual brain age and derive the Brain Age Gap (BAG), defined as the difference between predicted brain age and chronological age. BrainageR was applied to higher-quality T1-weighted MRI from NFBC 1986 (N=451, age 25.4–27.8 years) following automated preprocessing. The model showed modest correlation between chronological and predicted age ($r=0.070$) with mean absolute prediction error of 3.35 years (RMSE=4.27 years). Participants exhibited systematically elevated brain age relative to chronological age (mean BAG 0.96 ± 4.16 years). High BAG variance (17.32 years²) revealed substantial inter-individual neuromaturational heterogeneity despite chronological age homogeneity, among young adults from Northern Finland. These preliminary findings establish brainageR’s applicability to NFBC 1986 and motivate linking BAG to maternal, developmental, and neuropsychiatric variables available in the cohort.

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