Sydney_2025
Cardiovascular
Symposium

20 – 21 NOV 2025

PROGRAM

Innovations in Cardiovascular Science:

The Intersection of Vascular Biology, Cardiac Biology, and Organ Cross-Talk

HRI HEART RESEARCH INSTITUTE

Victor Chang Cardiac Research Institute

The George Institute for Global Health

The Westmead Institute FOR MEDICAL RESEARCH

CONTENTS

Getting to the Sydney Cardiovascular Symposium

Charles Perkins Centre Auditorium (CPC Auditorium) is located below the café.

Enter the auditorium area through the doors to the left of the café (outlined in the blue circle)

Building D17 John Hopkins Dr, Camperdown NSW 2037

From Central train station and the Four Points by Sheraton (approx. 20 minutes)

Take a bus from Central Railway Square to the Ross Street Gate (Stop: Forest Lodge 203768) located on Parramatta Road (opposite Officeworks Glebe). Get off, then turn right along Western Avenue and walk until you arrive the Charles Perkins Centre (CPC). The auditorium is below the café so enter through the door on the left according to the image above.

From Newtown train station (approx. 20 minutes)

Leave Newtown station, turn right and walk north along King Street until you reach Missenden Road. Turn left on Missenden Road, then walk until you reach the Royal Prince Alfred Hospital (RPAH) Emergency and head down John Hopkins Drive (on the right) to arrive at the Charles Perkins Centre. The café and auditorium will be on your right.

WIFI Access (connect to UniSydney-Guest)

Username: scvs2025

Password: 908415

FREE COFFEE!

Grab your free coffee/tea between 8-9am each day of the Symposium. Show your Sydney Cardiovascular Symposium registration badge at the coffee cart in the foyer outside the auditorium to claim your free drink.

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ADINSTRUMENTS

Welcome to the 2025 Sydney Cardiovascular Symposium

The Sydney Cardiovascular Symposium 2025 marks its eighth year of this flagship event, jointly hosted by the Heart Research Institute (HRI), Victor Chang Cardiac Research Institute (VCCRI), The George Institute for Global Health and the Westmead Institute for Medical Research (WIMR).

Since 2017, the Symposium has united scientists and clinicians from across New South Wales, Australia and internationally, creating a forum for collaboration across cardiology, multi-organ disease, bioengineering, bioinformatics and drug development. Together we share a commitment to advancing cardiovascular research and improving patient outcomes through multidisciplinary innovation.

This year's theme, "Innovations in Cardiovascular Science" highlights the exciting intersection of vascular biology, cardiac biology and organ cross-talk, with a focus on how integrated systems approaches can reveal new mechanisms and therapeutic opportunities.

We are honoured to welcome Professor Filip Swirski, Director of the Cardiovascular Research Institute at the Icahn School of Medicine at Mount Sinai in the United States as the Princesses' Lecturer, a lecture named in honour of Diana, Princess of Wales and Mary, Crown Princess of Denmark. Professor Swirski is internationally recognised for his pioneering work exploring immune and inflammatory processes in atherosclerosis, inter-organ communication in health and disease and the regulation of haematopoiesis in cardiovascular pathology.

The Sydney Cardiovascular Symposium Lecture will be delivered by Professor Dana Dawson, Professor of Cardiovascular Medicine and Consultant Cardiologist at the University of Aberdeen and Aberdeen Royal Infirmary in the United Kingdom. Professor Dawson is globally renowned for her research into acute takotsubo cardiomyopathy, spanning preclinical studies, cellular and organ-level mechanisms, registries, mechanistic clinical studies and clinical trials.

We are also proud to feature the outstanding work of our emerging and established researchers throughout the program. The inclusion of early and mid-career investigators, Rising Star presentations, poster sessions and a panel discussion reflect our commitment to fostering collaboration, mentorship and innovation across all career stages.

We warmly welcome you to this year's Symposium and hope that you find it inspiring, intellectually stimulating and rich with opportunities for discussion and connection. We encourage you to make the most of the sessions and social events, introduce yourself to new colleagues and take full advantage of all the opportunities to share ideas and forge new collaboration.

Thank you for joining us for the Sydney Cardiovascular Symposium 2025.

On behalf of the Organising Committee,

Prof Julie McMullen

Program: Thursday 20 November 2025

08:00 – 09:00	Coffee and Registration
09:00 – 09:10	Welcome to Country Speaker: Uncle Raymond Weatherall
09:10 – 09:15	Welcome to the Sydney Cardiovascular Symposium 2025 Prof Andrew Coats AO, *The Heart Research Institute, NSW*
09:15 – 10:00	**Sydney Cardiovascular Symposium Lecture** Sponsored talk by The Office of Health and Medical Research (OHMR) Chair: Prof Andrew Coats AO
	Prof Dana Dawson, *University of Aberdeen, UK* Takotsubo Cardiomyopathy – Organ Cross-talk and Clinical Trials
10:00 – 10:50	**Session 1: Organ Cross-talk in Health and Disease** Sponsored by AstraZeneca (Presentation: 20 min, Questions: 5 min) Chair: A/Prof Clare Arnott
10:00	Prof Andrew Coats AO, *The Heart Research Institute, NSW* Organ Cross-Talk: Clinical Perspective of Co-Morbidities in Heart Failure
10:25	Prof Luigi Fontana, *The University of Sydney, NSW* The Role of Nutrition and Exercise in Modulating Cardiometabolic Health: Strategies for Prevention and Optimisation
MORNING TEA 10:50 – 11:10	
11:10 – 12:30	**Session 2: Heart and Brain** (Presentation: 20 min, Questions: 5 min) Chairs: Prof Ben Freedman OAM and Prof Julie McMullen
11:10	Prof Perminder Sachdev, *The University of New South Wales, NSW* The Interplay of Heart and Brain Health
11:35	Dr Sonali Gnanenthiran, *The George Institute for Global Health, NSW* Blood Pressure Control After Stroke: Current Evidence and Emerging Frontiers
12:00	Prof Lin Yee Chen, *University of Minesota, USA* Atrial Myopathy – A Target for Stroke and Dementia Prevention
LUNCH 12:30 – 14:00 **POSTER JUDGING SESSION 13:00 – 14:00**	

Program: Thursday 20 November 2025

14:00 – 15:40 **Session 3: Inflammation and Organ Cross-talk**
(Presentation: 20 min, Questions: 5 min)
Chairs: Dr Ashish Misra and Dr Renping Liu

14:00 Prof Edward Fisher, *NYU Grossman School of Medicine, USA*
Metabolic and Psychological Stress and Atherosclerosis: Intersection at the Bone Marrow

14:25 Dr Siân Cartland, *The Heart Research Institute, NSW*
Reprogramming Inflammation: Can Myeloid-Derived Suppressor Cells be Therapeutic Targets in Atherosclerosis?

14:50 Prof John O'Sullivan, *The University of Sydney, NSW*
Metainflammatory Driven Strategies for HFpEF – A 21st Century Epidemic

15:15 – 15:40 **Flash Talks**
(Presentation: 5 min, Questions: 3 min)
Chair: Elaina Kelland

15:15 Lauren Watson, *The Heart Research Institute, NSW*
Exercise has Differential Cardiometabolic Benefits in Male and Female Mice on a High-Fat Diet

15:23 Niina Matthews, *University of Technology Sydney, NSW*
Human Size Bioprinted Cardiac Patches for Heart Failure Patients

15:31 Wendy (Huiwen) Zhao, *The University of Sydney, NSW*
Designing Human Platelets with Improved Reactivity for Cardiovascular Disease

AFTERNOON TEA 15:40 – 16:00

16:00 – 17:00 **Session 4: Panel Discussion on Research Career Progression/Pathways**
Chairs: Dr Emma Rath and Dr Cindy Gueguen

Panellists:
1. Prof Bronwyn Kingwell, *CSL Ltd, VIC*
2. Prof Mathias Francois, *The University of Sydney, NSW*
3. Dr Gavin Recchia, *Davies Collision Cave, NSW*

17:00 **POSTER SESSION (VIEWING) AND NETWORKING DRINKS**

18:30 **SPEAKER'S DINNER BY INVITATION**

Program: Friday 21 November 2025

08:00 – 09:00	Coffee and Registration
09:00 – 10:15	**Session 1: Cardiometabolism** (Presentation: 20 min, Questions: 5 min) Chairs: Prof Jamie Vandenberg and Dr Ivy Chiang
09:00	Prof Leonie Heilbronn, *The University of Adelaide, SA* Time Restricted Eating: Impacts on 24-Hour Blood Pressure Profiles and Cardiometabolic Outcomes
09:25	Prof Nigel Turner, *Victor Chang Cardiac Research Institute, NSW* Reduced Nicotinamide Mononucleotide Improves Metabolic Profile of High Fat Diet-Fed Mice
09:50	Prof Alex Brown, *The Australian National University, ACT* Precision Medicine in Indigenous Health
MORNING TEA 10:15 – 10:45	
10:45 – 12:40	**Session 2: Heart and Organ Cross-talk** (Presentation: 20 min, Questions: 5 min) Chair: A/Prof Jacob Qi
10:45	Prof Natasha Rogers, *Westmead Hospital, The Westmead Institute for Medical Research, The University of Sydney, NSW* Finding Missing Links in Cardiorenal Syndrome
11:10	Dr Weizhen (Eva) Li, *The Heart Research Institute, NSW* NOTCH3 at the Fat–Artery Crossroads in Atherosclerosis
11:35 – 12:15	**Flash Talks** (Presentation: 5 min, Questions: 3 min) Chair: Dr Clara Liu Chung Ming
11:35	Lee Patricia Liao, *The University of Sydney, NSW* Dietary Approaches to Stop Hypertension (DASH) Diet, Incident Heart Failure and its Associated Risk Factors in Australian Women
11:43	Dr Javad Foroughi, *The University of New South Wales, NSW* Artificial Muscle Based Soft Robotic Sleeve for End-Stage Heart Failure Treatment
11:51	Amandeep Mondal, *The Heart Research Institute, NSW* Lactate-Dependent Reprogramming of Macrophages is Associated with Fibrous Cap Formation and Atherosclerotic Plaque Stability

Program: Friday 21 November 2025

11:59 Dr Jaideep Singh, *The Heart Research Institute, NSW*
Novel Formylpeptide Receptor 1/2 Agonist Limits Hypertension-Induced Cardiovascular Damage

12:07 Hannah Parker, *The University of Sydney, NSW*
Investigating the Effects of In Utero High-Saccharide Exposure on Fetal Heart Development and Cardiovascular Health in Offspring

12:15 – 12:20 Dr Seakcheng Lim, *CVRN*
Introduction to the NSW CVRN

12:20 – 12:40 Jason Boyd, Principal Policy Officer, Evaluation and Reporting, *OHMR*
Cathy Kellick, Principal Policy Officer, Research Grants, *OHMR*
Interim Evaluation of NSW Cardiovascular Research Capacity Program

LUNCH 12:40 – 13:10

13:10 – 14:45 **Session 3: Rising Stars Award**
(Presentation: 15 min, Questions: 5 min)
Chairs: Dr Fergus Payne and Dr Ivy Guan

13:10 Alexander Lin, *The Heart Research Institute, NSW*
Vascular Smooth Muscle Cell PDGFRβ Signalling Regulates Fibrous Cap Fibroblast-Like Cells and Stability of Diabetic Atherosclerotic Lesions

13:30 Dr Lucy McGrath-Cadell, *Victor Chang Cardiac Research Institute, NSW*
High and Variable Endothelial Shear Stress Characterises Spontaneous Coronary Artery Dissection: An Imaging Case-Control Study

13:50 Dr Robert Hume, *The University of Sydney, NSW*
Do Human Hearts Have an Intrinsic Regenerative Potential? Cardiomyocyte Mitosis Post-Myocardial Infarction

14:10 – 14:42 **Flash Talks** (Presentation: 5 min, Questions: 3 min)
Chair: Dr Fergus Payne

14:10 Wei Yi Lew, *The University of Sydney, NSW*
Rapid On-Demand Monitoring of Valsartan and Sacubitril at the Point of Care with Miniature Mass Spectrometry

14:18 Lauren Cook, *Victor Chang Cardiac Research Institute, NSW*
Developing an iPSC Model of Epicardial Adipose Tissue-Mediated Atrial Fibrillation

Program: Friday 21 November 2025

14:26 Dr Tim Shiraev, *The University of Sydney, NSW*
Medium-term Outcomes of a Novel Arterial Graft in a Large Animal Model

14:34 Ramu Dumre, *University of Wollongong, NSW*
Docosahexaenoic Acid Supplementation Appears to Elicit an Omega-3 Index Response Irrespective of Dietary Patterns; Vegan/Vegetarian or Non-Restrictive

14:45 – 16:00 **Session 4: Heart Disease: Early Origins and Diabetes**
(Presentation: 20 min, Questions: 5 min)
Chairs: A/Prof Emily Wong and Dr Zijing (Albert) Zhou

14:45 Prof Sally Dunwoodie, *Victor Chang Cardiac Research Institute, NSW*
NAD Deficiency and Congenital Heart Disease

15:10 Dr Magda Montgomery, *The University of Melbourne, VIC*
Understanding the Endocrine Function of the Heart in Type 2 Diabetes

15:35 A/Prof Mary Kavurma, *The Heart Research Institute, NSW*
Sex Differences and Mitochondrial Endothelial Health Influence Vascular Recovery in Diabetes-Associated Peripheral Artery Disease

AFTERNOON TEA 16:00 – 16:15

16:15 – 17:30 **The Princesses' Lecture**
Sponsored by UNSW Cardiac, Vascular and Metabolic Medicine (CVMM)
Chair: Prof Jason Kovacic

Prof Filip K. Swirski, *Mount Sinai, USA*
Heart-Brain Communication in Health and Disease

17:30 Symposium Close
Prof Julie McMullen, *The Heart Research Institute, NSW*

NETWORKING DRINKS AND AWARDS 17:40 – 18:30

Invited Speakers

The Princesses' Lecture: Professor Filip K. Swirski

Cardiovascular Research Institute, Icahn School of Medicine at Mount Sinai, USA

"Heart-Brain Communication in Health and Disease"

The Princesses' Lecture, named in honour of Diana, Princess of Wales and Mary, Crown Princess of Denmark, will be given by Professor Filip Swirski. Filip obtained his PhD at McMaster University in Canada and completed postdoctoral studies at Brigham and Women's Hospital, Harvard. He was a Professor at Harvard Medical School and Massachusetts General Hospital before he joined Mount Sinai in 2021. Filip is currently the Director of the Cardiovascular Research Institute, Icahn School of Medicine at Mount Sinai in the USA. Filip is recognised internationally for his work on 1) Immune and inflammatory processes in atherosclerosis, 2) Systems physiology and inter-organ communication in health and disease, and 3) Haematopoiesis and its modulation in disease.

Invited Speakers

Sydney Cardiovascular Symposium Lecture: Professor Dana Dawson

School of Medicine, Medical Sciences and Nutrition, University of Aberdeen, UK

"Takotsubo Cardiomyopathy – Organ Cross-talk and Clinical Trials"

The Sydney Cardiovascular Symposium Lecture will be given by Professor Dana Dawson. Dana first qualified in medicine at University of Medicine "Grigore T. Popa", Iasi, Romania. This was followed by completion of her MRCP with the Royal College of Physicians and then read for her *D. Phil* in Cardiovascular Medicine at Merton College, University of Oxford. She further trained in Cardiovascular medicine in Edinburgh, Oxford, and London in the United Kingdom, and at the University of Virginia, Charlottesville in the USA. Dana moved to the University of Aberdeen in 2010, where she is Professor of Cardiovascular Medicine and Consultant Cardiologist at Aberdeen Royal Infirmary, in the United Kingdom. Dana is recognised internationally for her work on acute takotsubo cardiomyopathy which includes preclinical research, cell work, organ cross-talk, registries, mechanistic clinical studies, and clinical trials.

Invited Speakers

Professor Andrew Coats AO

Heart Research Institute, NSW, Australia

"Organ Cross-talk: Clinical Perspective of Co-morbidities in Heart Failure"

Professor Andrew Coats AO has been a Scientific Director and CEO at the Heart Research Institute since 2022. He is an experienced academic leader and entrepreneur with three decades of international experience in four of the world's top 50 universities. He has over 800 peer-reviewed full papers, over 200,000 career citations and an H-Index of 170. Professor Coats is a fully accredited physician and cardiologist in the United Kingdom and Australia, a qualified company director with more than 60 board years of experience. In 2017, Prof Coats was appointed an Officer of the Order of Australia for distinguished service to medical research and tertiary education in the field of cardiology, as an academic and author, and as a mentor and role model for young scientists.

Professor Luigi Fontana

Charles Perkins Centre, The University of Sydney, NSW, Australia

"The Role of Nutrition and Exercise in Modulating Cardiometabolic Health: Strategies for Prevention and Optimisation"

Professor Fontana is the Leonard P. Ullmann Chair of Translational Metabolic Health at the Charles Perkins Centre, where he directs the Charles Perkins Centre Royal Prince Alfred Clinic and the Health for Life Research, Clinical & Educational Program. He is also a Professor of Medicine and Nutrition in the Faculty of Medicine and Health at the University of Sydney and a Clinical Academic in the Department of Endocrinology at the Royal Prince Alfred Hospital. His work focuses on preventative medicine, the role of nutrition and physical exercise in retarding the aging process, and in preventing the accumulation of metabolic and molecular damage leading to multiple age-associated chronic disease.

Invited Speakers

Professor Perminder Sachdev

The University of New South Wales, NSW, Australia

"The Interplay of Heart and Brain Health"

Perminder Sachdev AM MBBS MD FRANZCP PhD FAHMS is Scientia Professor of Neuropsychiatry, Co-Director of the Centre for Healthy Brain Ageing (CHeBA), UNSW Sydney, Research Director of the Neuropsychiatric Institute (NPI) at the Prince of Wales Hospital, Sydney, and Director of the Centre of Research Excellence in Vascular Contributions to Dementia. His major areas of research are drug-induced movement disorders, brain imaging, cognitive ageing and dementia, especially vascular cognitive impairment. He has published over 900 peer-reviewed journal papers and 6 books, including one for lay readers (The Yipping Tiger and other tales from the neuropsychiatric clinic) and a book of poems (A migrant's musings). In 2011, he was appointed Member of the Order of Australia (AM) for services to medical research. He was awarded the Ryman Prize in 2022 by an international jury for the most significant contributions world-wide toward the health of older people.

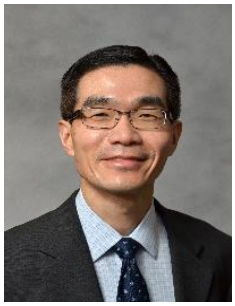
Dr Sonali Gnanenthiran

The George Institute for Global Health, NSW, Australia

"Blood Pressure Control After Stroke: Current Evidence and Emerging Frontiers"

Dr Sonali Gnanenthiran (MBBS [Hons I], PhD, FRACP, FCSANZ) is a cardiologist at The George Institute for Global Health and Concord Hospital, Australia. Her clinical and research interests include cardiovascular disease prevention and risk assessment. She completed a PhD with the University of Sydney. She was a recipient of the John Chalmers Fellowship at The George Institute, and a Heart Foundation post-doctoral fellowship. She has >50 publications and has been awarded >$18.5 million in research funding. She is the lead investigator of the national LOTUS trial that assesses new secondary prevention models for ischaemic stroke. She has been awarded the 2023 American Heart Association Karl Link Award for Thrombosis, 2022 American Heart Association Paul Dudley White International Scholar Award, 2021 Scientific Medal of the Thrombosis and Haemostasis Society of Australia and New Zealand, and 2020 American Heart Association Paul Dudley White International Scholar Award.

Invited Speakers



Professor Lin Yee Chen

Lillehei Heart Institute, University of Minnesota, USA

"Atrial Myopathy - A Target for Stroke and Dementia Prevention"

Dr Chen is a tenured Professor of Medicine, an ABIM board-certified cardiologist and cardiac electrophysiologist, and an NIH-funded physician-scientist. He is the Director of the Lillehei Heart Institute, at University of Minnesota Medical School. His patient-oriented research on atrial fibrillation and atrial myopathy is focused on three themes: (1) To identify novel risk factors for atrial fibrillation and atrial myopathy, and discover new strategies to prevent both conditions, (2) To characterise the relationship of atrial fibrillation and atrial myopathy to cardiovascular and neurocognitive outcomes such as stroke and dementia, and to elucidate the underlying mechanisms, and (3) To discover novel strategies to prevent atrial fibrillation and atrial myopathy-related stroke and dementia. Dr. Chen was elected to the American Society for Clinical Investigation in 2024 and the Association of University Cardiologists in 2025.

Professor Edward Fisher

NYU Grossman School of Medicine, USA

"Metabolic and Psychological Stress and Atherosclerosis: Intersection at the Bone Marrow"

As a preventive cardiologist at NYU Langone, Professor Ed Fisher is dedicated to making a positive impact on patients' lives by focusing on preventing and managing cardiovascular diseases. He specialises in preventive cardiology, with a particular focus on managing major risk factors such as elevated cholesterol, high blood pressure, prediabetes, and diabetes. With extensive training and years of experience, Professor Fisher is committed to providing accurate diagnoses and comprehensive preventive care, which includes medication management, heart-healthy nutrition, and regular exercise programs. Throughout his career, Professor Fisher has been part of NYU Langone's Center for the Prevention of Cardiovascular Disease, a nationally recognised program known for its excellence in patient care and research. His work includes a federally funded program aimed at reversing arterial damage caused by elevated cholesterol levels. This research has provided valuable insights into why certain clinical studies succeed or fail, ultimately enhancing patient care. He has been honoured with an award from the National Lipid Association for "extraordinary expertise and contributions to the field of clinical lipidology," reflecting his dedication to quality patient care

Invited Speakers

Dr Siân Cartland

The Heart Research Institute, NSW, Australia

"Reprogramming Inflammation: Can Myeloid-Derived Suppressor Cells be Therapeutic Targets in Atherosclerosis?"

Dr Siân Cartland is Group Leader of the Cardiovascular Immunotherapy Unit at the Heart Research Institute. Her research focuses on how immune cells, especially myeloid-derived suppressor cells and macrophages, drive vascular inflammation in atherosclerosis and peripheral artery disease, aiming to uncover mechanisms and develop targeted therapies that address residual inflammatory risk.

Professor John O'Sullivan

The University of Sydney, NSW, Australia

"New Insights and Therapeutic Strategies for HFpEF – A 21st Century Epidemic"

Professor John O'Sullivan is the inaugural Professor of Cardiometabolic Medicine at the University of Sydney and Department of Cardiology, Royal Prince Alfred Hospital. He is a Level 2 NHF Future Leader Fellow, and co-Director of the HFpEF ("Stiff Heart Failure") Clinic at RPAH. He is Director of the Heart Failure Alliance across SLHD and WSLHD, incorporating RPAH, Concord, Westmead, and Blacktown Hospitals. John's clinical practice focusses on Heart Failure and Early Prevention of Atherosclerotic Cardiovascular Disease (ASCVD). John's basic science discoveries have been translated to clinical biomarkers, Medicare Item Numbers, and new diagnostic assays.

Professor Leonie Heilbronn

The University of Adelaide, SA, Australia

"Time-Restricted Eating: Impacts on 24-Hour Blood Pressure Profiles and Cardiometabolic Outcomes"

Professor Leonie Heilbronn is a clinical research scientist based at the University of Adelaide. Her work is focused on understanding the impact of meal timing on circadian regulation of metabolism and on developing individualised strategies to optimise glycaemia and other aspects of metabolic health. She has authored over 150 research papers and currently serves as an Associate Editor for Obesity and the European Journal of Endocrinology and is the immediate past President of the Australia New Zealand Obesity Society.

Invited Speakers

Professor Nigel Turner

Victor Chang Cardiac Research Institute, NSW, Australia

"Reduced Nicotinamide Mononucleotide Improves Metabolic Profile of High Fat Diet-Fed Mice"

Professor Nigel Turner is head of the Cellular Bioenergetics Laboratory at the Victor Chang Cardiac Research Institute. He completed a PhD in comparative physiology and biochemistry at the University of Wollongong and undertook postdoctoral studies in the area of mitochondrial metabolism and insulin action at the Garvan Institute of Medical Research. From 2012-2021 he was head of the Mitochondrial Bioenergetics Laboratory in the School of Biomedical Sciences at UNSW Sydney, and in 2022 established his current research group at the Victor Chang Cardiac Research Institute. Nigel's research focuses on understanding how aberrations in cellular energy metabolism contribute to the pathogenesis of conditions including diabetes, cardiovascular disease and cancer.

Professor Alex Brown

The Kids Research Institute Australia, Australian National University

"Precision Medicine in Indigenous Health"

Professor Alex Brown is a leading Indigenous medical doctor and genomics researcher. He directs the National Centre of Indigenous Genomics and leads the Australian Alliance for Indigenous Genomics. From the Yuin nation, his work advances equity in health, empowers Indigenous researchers, and influences national policy to address chronic disease and health disparities in Indigenous communities.

Professor Natasha Rogers

Westmead Hospital, The Westmead Institute for Medical Research, The University of Sydney, NSW, Australia

"Finding Missing Links in Cardiorenal Syndrome"

Natasha Rogers is Professor in Nephrology and Transplantation Medicine at the University of Sydney. She is Head of Kidney, Pancreas, and Islet Transplantation at Westmead Hospital, and Co-Director of the Centre for Transplant and Renal Research at the Westmead Institute for Medical Research. Her main focus of research is matrix protein signalling.

Invited Speakers

Dr Weizhen (Eva) Li

The Heart Research Institute, NSW, Australia

"NOTCH3 at the Fat–Artery Crossroads in Atherosclerosis"

Dr Eva Li is an early career researcher in the Atherosclerosis and Vascular Remodelling Group at the Heart Research Institute. She was awarded her PhD in June 2025, with a research focus on targeting cell phenotypes to stabilise atherosclerotic plaques. Her work has been acknowledged through notable awards such as the 2023 Sydney Cardiovascular Symposium Rising Star Award and the 2024 Faculty of Engineering Career Advancement Award.

Professor Sally Dunwoodie

Victor Chang Cardiac Research Institute, NSW, Australia

"NAD Deficiency and Congenital Heart Disease"

Professor Sally Dunwoodie is Co-Deputy Director of the Victor Chang Cardiac Research Institute and heads the Embryology Laboratory and the Congenital Heart Disease Research Program. She is recognised for her work in identifying the genetic causes of congenital malformations. Her research explores the functional effects of human gene mutations during embryogenesis and examines how gene-environment interactions disrupt embryogenesis in mice.

Invited Speakers

Dr Magda Montgomery

The University of Melbourne, VIC, Australia

"Understanding the Endocrine Function of the Heart in Type 2 Diabetes"

Dr Magda Montgomery is the Head of the 'Metabolic Tissue Crosstalk Laboratory' in the Department of Anatomy and Physiology (University of Melbourne), previous NHMRC Early Career Fellow (2014–2018) and NHMRC Career Development Fellow (2018–2022) and leads one of four major programs in the School of Biomedical Sciences, the Metabolism Program. Dr Montgomery leads an innovative research program aimed at understanding how defects in lipid metabolism and the endocrine function of metabolic tissues drive progression of metabolic liver disease and type 2 diabetes. Dr Montgomery received her PhD at the University of Wollongong in 2011, and her postdoctoral training at the Garvan Institute in Sydney, at the University of New South Wales (Sydney), and at Monash University (Melbourne). Dr Montgomery published 60+ papers in high-impact peer-reviewed journals (Nature, Nature Communications, Science Translational Medicine) and is the Editor-in-Chief for a new metabolism journal with the Nature Portfolio (npj Metabolic Health and Disease).

Associate Professor Mary Kavurma

The Heart Research Institute, NSW, Australia

"Sex Differences and Mitochondrial Endothelial Health Influence Vascular Recovery in Diabetes-Associated Peripheral Artery Disease"

Associate Professor Mary Kavurma leads the Centre for Peripheral Artery Disease at the Heart Research Institute and heads the Vascular Complications Group. An Associate Professor at the University of Sydney, her research focuses on cellular mechanisms of atherosclerosis. She has held national leadership roles, secured major fellowships and grants, co-directs ACvA's Disease Mechanisms flagship. In 2009 she received a Young Tall Poppy Science Award.

Panellists

Professor Bronwyn Kingwell

CSL Ltd, VIC, Australia

Bronwyn was recruited to CSL as the Research Therapeutic Area Lead for Cardiovascular and Metabolic diseases (now Cardiovascular and Renal) in 2019. At CSL, Bronwyn has built a pipeline of cardiovascular, metabolic and renal drug candidates in areas of high unmet medical need. She has also introduced strategic initiatives to increase the quality and speed of research project transitions into clinical development. Throughout her career Bronwyn has made contributions to national science strategy and policy through honorary leadership roles with government and non-for-profit organisations, as well as a variety of peak bodies and learned Academies including the National Health and Medical Research Council, Australian Academy of Science, Australian Society of Medical Research, National Heart Foundation and the Australian Academy of Health and Medical Sciences where she is a current board member.

Professor Mathias Francois

The University of Sydney, NSW, Australia

Mat Francois is leading a lab that studies the control of gene regulation during development and diseases of the cardio-vascular diseases. The group research activity relies on a highly multi-disciplinary approach that combines developmental biology, molecular imaging complemented by biophysics and genomics methods to investigate the control of gene transcription in the endothelium. The aim of our research is to take advantage of a deep understanding of transcription factor mode of action during development to unlock new therapeutic avenues in vascular-related pathologies. Findings from the lab have repositioned the clinical management of a two rare orphan diseases and opened up therapeutic avenue for infantile hemangioma. A spin off company from the lab (Gertrude Biomedical Pty Ltd, Bio21 Melbourne) was founded in 2019 is currently developing the next generation of inhibitors that target an endothelial specific transcription factor.

Panellists

Dr Gavin Recchia

Davies Collision Cave, NSW, Australia

Gavin is a Principal and patent attorney with over 20 years' experience, specialising in the areas of microbiology, molecular biology, biochemistry, genetics, plant varieties and pharmaceuticals. Gavin provides strategic advice to clients in relation to their patent portfolios, including advising on aligning IP and commercial strategies, and maximising the commercial value of IP. He drafts and prosecutes patent applications in biotechnology and pharmaceutical related fields, files and prosecutes plant breeders' rights applications, advises on patent validity and infringement, provides freedom to operate advice and conducts patent oppositions.

Rising Stars

Alexander Lin, *The Heart Research Institute, NSW*

'Vascular Smooth Muscle Cell PDGFRβ Signalling Regulates Fibrous Cap Fibroblast-like Cells and Stability of Diabetic Atherosclerotic Lesions'

Background: Atherosclerotic plaque rupture is the leading cause of myocardial infarction and stroke. Diabetic patients experience a greater frequency of plaque rupture events because of a thinner fibrous cap, the collagen-rich structure which largely dictates plaque stability. However, our understanding of the cellular origins and mechanisms underlying fibrous cap depletion in diabetic lesions is limited. Addressing this knowledge gap is essential for developing strategies to strengthen the fibrous cap and prevent plaque rupture in diabetes.

Methods: We investigated the cellular origins and composition of the diabetic atherosclerotic fibrous cap throughout murine atherosclerosis using vascular smooth muscle cell (SMC) lineage tracing, clonal analysis, and single cell RNA-sequencing (scRNA-seq). Further mechanistic studies were performed using SMC-specific PDGFRβ knockout mice.

Results: As reflected in human lesions, our diabetic mouse plaques displayed features of plaque instability. Clonal analysis revealed that in contrast to the regular monoclonal expansion of SMCs in non-diabetic plaques, diabetic lesions displayed SMC polyclonality. Surprisingly, an increased proportion of SMCs retained a contractile ACTA2+ phenotype in diabetes, despite reductions in plaque-stabilising collagen. Instead scRNA-seq analysis implicated a decline in fibroblast-like cells in reduced collagen synthesis. Lineage tracing studies revealed that this reduction in fibroblast-like cells was of both SMC and non-SMC origin. SMC-specific deletion of PDGFRβ impaired SMC migration into the diabetic lesion and resulted in a loss of SMC-derived fibroblast-like cells. These plaques contained less collagen, highlighting the importance of SMC-derived fibroblast-like cells in maintaining fibrous cap stability.

Conclusions: Our findings implicate a loss of collagen-producing fibroblast-like cells in diabetic plaque instability. Our study challenges the traditional paradigm that ACTA2+ SMCs are the collagen-producing cells within the fibrous cap, instead highlighting the importance of SMC-derived fibroblast-like cells formed through PDGFRβ-dependent pathways. Targeting these cells offers a promising therapeutic approach to enhance fibrous cap stability and reduce cardiovascular events in diabetes.

Rising Stars

Dr Lucy McGrath-Cadell, *Victor Chang Cardiac Research Institute, NSW*

'High and Variable Endothelial Shear Stress Characterises Spontaneous Coronary Artery Dissection: An Imaging Case-Control Study'

Background: Spontaneous coronary artery dissection (SCAD) causes acute coronary syndrome which typically affects women. Coronary vessel wall vulnerabilities play a role, but it is not known why SCAD occurs in some segments. We aimed to identify anatomical and haemodynamic factors that lead to SCAD by comparing SCAD cases and controls at the coronary tree and territory level as well as within the SCAD segment versus the rest of an individual's coronary tree.

Methods: We studied 36 patients with angiographically-confirmed SCAD (41 lesions) and 75 sex- and ethnicity-matched healthy controls. Coronary arteries were reconstructed from the computed tomography coronary angiogram (CTCA) to quantify vessel curvature, diameter, torsion, and four haemodynamic metrics—time-averaged endothelial shear stress (TAESS), topological shear variation index (TSVI), oscillatory shear index, and relative residence time (RRT)—at the tree (left/right), territory (left anterior descending, circumflex, right coronary artery), and lesion levels. Effect sizes were determined by the ranked biserial correlation, rrb, with 95% confidence intervals (CI).

Results: Compared to controls, SCAD-affected coronary trees demonstrated higher curvature ($p<0.001$), TAESS ($p=0.007$), and TSVI ($p<0.007$). Only 2 (3%) SCAD trees lacked these adverse features compared with 107 (71%) controls, whereas 32 (45%) SCAD trees exhibited all three features versus 2 (1%) in controls ($p<0.001$). SCAD-affected territories also had smaller diameters ($p<0.001$) along with higher curvature ($p<0.001$), TAESS ($p=0.007$), and TSVI ($p=0.047$). Within individual coronary trees, segments harbouring SCAD lesions showed smaller diameters ($p<0.001$), lower torsion ($p<0.001$), higher TAESS ($p<0.001$), and higher TSVI ($p=0.001$) than non-SCAD segments. A combination of curvature, TAESS, and TSVI distinguished SCAD cases from controls with areas under the ROC curves of 0.95 (left tree) and 0.97 (right tree); adding diameter yielded AUCs >0.91 at the territory level.

Conclusions: These findings demonstrate that local vessel geometry and elevated, variable endothelial shear stress underpin SCAD pathophysiology and may explain its anatomical distribution.

Rising Stars

Dr Robert Hume, *The University of Sydney, NSW*

'Do Human Hearts Have an Intrinsic Regenerative Potential? Cardiomyocyte Mitosis Post-Myocardial Infarction'

Background: Myocardial infarction (MI) can eliminate up to a third of the cardiomyocytes (CMs) within the human heart. Although CMs proliferate during early development, most CMs cease mitosis soon after birth. In contrast, for decades rodent MI models have shown that CMs increase mitosis in response to ischemia, however this has not been shown in humans.

Methods: We performed imaging mass cytometry, 3D immunofluorescence, DNA in situ hybridisation (ISH), RNA sequencing, proteomics, metabolomics and single nucleus RNA sequencing (snRNAseq) on a unique pre-mortem post-MI human heart. We also developed a novel post-MI left ventricle biopsy method to obtain live infarct border and remote zone samples from MI patients undergoing coronary artery bypass graft (CABG) surgery. Lastly, we developed a human in vitro/ex vivo translational pipeline for future testing of novel therapeutics that amplify cardiac regeneration.

Results: Using these human samples, we demonstrated that adult human CMs exhibit increased mitosis and cytokinesis, with reduced binucleation in response to post-MI ischemia. Our findings were further confirmed through analysis of the largest published human MI snRNAseq dataset currently available and our novel CABG biopsy model. We also described the gene, protein and metabolite expression of the myocardium containing mitotic CMs, identifying pathways involved in human CM mitosis. Both novel pathways and pathways previously shown in rodent cardiac regeneration studies were identified.

Conclusions: This is the first human study (2nd round of review in Circulation Research) that proves what has previously been demonstrated in animal studies over the last 25 years. We are the first to show that, like rodents, human CMs increase mitosis post-MI and provide a novel MI CABG ex vivo model for further studies. Importantly, future development of therapies that amplify this intrinsic cardiac regenerative capacity could reverse heart failure and improve the lives of millions of MI patients worldwide.

Selected Abstracts: 5 Minute Flash Talk Speakers

Lauren Watson, *The Heart Research Institute, NSW*

'Exercise has Differential Cardiometabolic Benefits in Male and Female Mice on a High-Fat Diet'

Background: Progression, prevalence and outcomes of cardiometabolic disease are sexually dimorphic, with women typically exhibiting more beneficial cardiometabolic health than men. Regular exercise is well established to improve cardiometabolic health, through promoting muscular hypertrophy and fat loss, improving glucose homeostasis and inhibiting adipose tissue inflammation. However, whether the underlying mechanisms are different between sexes is unknown. The objective of this study was to investigate the cardiometabolic effects of exercise training in male and female mice in a disease setting.

Methods: Male and female C57Bl/6J mice were fed a high fat diet (HFD, 60% fat) for 10 weeks, with or without access to voluntary running wheels. Body composition, exercise, glucose and insulin tolerance were measured, and cardiac function was evaluated by echocardiography. Fluorescent-activated cell sorting was used to assess adipose tissue inflammation, and wheat-germ agglutination staining was used to assess cardiomyocyte dimensions.

Results: Exercise intervention resulted in attenuation of HFD-induced body and fat mass increase in females but not males, despite similar increases in exercise tolerance. Anti-inflammatory effects of exercise were differentially elicited between sexes in subcutaneous and epididymal fat pads. Glucose tolerance was unaffected by exercise in both sexes, but a slight improvement in insulin tolerance was observed in males only. Consistent with the body weight findings, cardiac diastolic function was improved with exercise in females but not males. Surprisingly, cardiomyocyte dimensions increased with exercise in females and decreased with exercise in males.

Conclusions: This study provides the first evidence that cardiometabolic effects of exercise are differentially elicited in males and females in a metabolic disease setting. Female mice were more disposed to exercise benefits related to body weight and cardiac function than males. These findings suggest that exercise may be especially beneficial for cardiometabolic health in females and provide new insight into sexually dimorphic mechanisms underlying the effect of exercise in cardiometabolic disease.

Niina Matthews, *The University of Technology Sydney, NSW*

'Human Size Bioprinted Cardiac Patches for Heart Failure Patients'

Background: Heart failure presents significant clinical challenges worldwide, as cardiovascular disease remains a leading cause of mortality globally. For patients with end-stage heart failure, conventional treatment culminates in heart transplantation; however, donor scarcity and complex surgical considerations limit this approach. This project aims to advance the clinical translation of

Selected Abstracts: 5 Minute Flash Talk Speakers

tissue engineering methodologies, including 3D bioprinting technologies, to develop patient-specific cardiac patches for targeted myocardial repair.

Methods: Patient-specific cardiac patches were designed using cardiac MRI data. A total of 2800 cardiac spheroids (CSs) were co-cultured using iPSC-derived human cardiomyocytes, human cardiac fibroblasts, and human cardiac arterial endothelial cells. CSs were mixed in hydrogels containing either alginate/gelatin alone (control) or with silk fibroin (SF) to create bioinks for biofabrication of patient-specific designs using an extrusion bioprinter. After printing, human-sized cardiac patches were characterised for their printability and durability over 28 days. Cell viability, neovascularisation, contractility, and conduction velocity were evaluated using seven CS-laden patches, with approximately 400 CSs per patch.

Results: Our findings demonstrate that patient-specific cardiac patches can be 3D bioprinted to clinically relevant dimensions, and they are durable for up to 28 days in vitro. The addition of SF improved the printability and durability of the patches. The addition of SF did not alter the viability of CSs in bioprinted patches or their contractile function, and the CSs showed levels of neovascularisation.

Conclusions: In a world-first, medical imaging was used to create personalised 3D designs for the extrusion bioprinting of cardiac patches containing viable and functional CSs. Future studies will investigate the long-term effects on safety and efficacy of the patches. If successful, personalised cardiac patches could provide a solution to overcome the risks associated with heart transplantation and address the limitations due to donor shortage of transplantable hearts.

Wendy (Huiwen) Zhao, *The University of Sydney, NSW*

‘Designing Human Platelets with Improved Reactivity for Cardiovascular Disease’

Background: Hyperreactive platelets drive cardiovascular complications such as heart attack and stroke. We recently discovered that upregulation of the platelet protein SEC61B promotes hyperreactivity by increasing cytosolic calcium. Inhibiting SEC61B reduced platelet hyperreactivity, and thrombus formation, identifying it as a promising therapeutic target (Kong et al. J Clin Invest, 2025). This project aims to establish a bone marrow-on-a-chip model populated with megakaryocytes (platelet progenitors) genetically modified to lack SEC61B, to generate platelets with reduced SEC61B expression and attenuated reactivity. This represents the first platform for engineering human platelets with favourable haemostatic properties and reduced thrombogenic potential.

Methods: To mimic the bone marrow microenvironment, a porous, sponge-like silk fibroin scaffold was fabricated following the method described by Buduo, et al. eLife 2021. Silk solution was cast into a PDMS mould with salt particles to create pores. SEC61B was deleted in immortalised human megakaryocytes (imMCKLs) using CRISPR-Cas9, followed by transfection and selection.

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Differentiating imMCKLs were introduced into the silk scaffold, cultured in the scaffold and perfused with media under shear stress mimicking physiological blood flow. Platelet production and function were assessed by flow cytometry and immunofluorescence.

Results: Cultured imMCKLs retained normal differentiation, granule formation, and platelet production. SEC61B deletion was confirmed by immunohistochemistry and flow cytometry. Platelets generated ex vivo from wild type (WT) imMKCLs expressed platelet receptors CD42a and CD41, similar to human platelets. The platform produced 3-5 x 10^3 platelets per hour. Upon thrombin stimulation, 20-30% of ex vivo-derived platelets mobilised P-selectin, approximately one-quarter the activation rate of fresh human platelets (Figure 1).

Conclusions: Our bone marrow-on-a-chip generates functional human platelets from genetically modified megakaryocytes. Targeting SEC61B reduces platelet reactivity, demonstrating the platform's utility for disease modelling, drug discovery, and future therapeutic platelet design.

Lee Patricia Liao, *The University of Sydney, NSW*

'Dietary Approaches to Stop Hypertension (DASH) diet, Incident Heart Failure and its Associated Risk Factors in Australian Women'

Background: Heart Failure (HF) affected 49 900 Australian women in 2022, leading to more deaths in women than men. Increasing evidence supports dietary modification in HF management, although large clinical trials are lacking. This study aimed to determine if high adherence to the Dietary Approaches to Stop Hypertension (DASH) diet was associated with lower incident heart failure (HF) and its associated risk factors of hypertension and diabetes mellitus (DM).

Methods: The current study was a prospective analysis of the Australian Longitudinal Study on Women's Health (ALSWH). Women aged 45 – 50 years, recruited in 1996, were followed from 2001 to 2022. DASH diet scores were calculated based on responses to a food frequency questionnaire (FFQ) and categorised into quintiles. Multivariable logistic regression was used to estimate odds ratio (OR) and corresponding 95% confidence intervals (CIs) for DASH diet association with incident HF. Adjustments were made for baseline covariates including age, socio-demographic factors, and history of risk factors. Multivariable Cox proportional hazards models were used to calculate hazard ratios (HRs) and 95% CIs for the secondary endpoints, hypertension and diabetes mellitus (DM). Dietary exposure was modelled as a time-varying covariate.

Results: 10 594 women with a mean age of 52.5 ± 1.5 years participated in this study. At 21-year follow up, there were 136 (1.3%) cases of HF, 2182 (15.0%) cases of hypertension and 994 (5.7%) cases of DM. After adjustment, no association was found between the highest (quintile 5) versus lowest (quintile 1) DASH diet adherence group and incident HF [OR 0.73, 95%CI 0.37, 1.43; p=0.20]. Those with the highest dietary adherence had lower risk of hypertension [adjusted-HR 0.73 (95%CI 0.63, 0.84); p<0.001] and DM [adjusted-HR 0.65 (95% CI 0.53, 0.81); p<0.001].

Selected Abstracts: 5 Minute Flash Talk Speakers

Conclusion: Higher adherence to the DASH diet was associated with a significantly lower risk of developing hypertension and DM in Australian women. We found no association between the DASH diet and incident HF, which may reflect the small incidence of HF in our cohort.

Dr Javad Foroughi, *The University of New South Wales, NSW*

'Artificial Muscle Based Soft Robotic Sleeve for End-Stage Heart Failure Treatment'

Background: Heart failure (HF) affects over 64 million patients globally and remains a leading cause of morbidity and mortality. For end-stage HF, ventricular assist devices (VADs) offer life-saving support but suffer from major drawbacks: thrombosis, bleeding from anticoagulation, driveline infections, and loss of physiologic pulsatility (Fig. 1). These complications reduce survival and quality of life. An alternative approach, soft robotic ventricular assist sleeves, can restore cardiac function externally without direct blood contact, thereby avoiding anticoagulation and enhancing hemocompatibility.

Methods: We developed an implantable soft robotic sleeve powered by twisted and coiled polymer (TCP) artificial muscles encapsulated in silicone elastomer (Fig. 1). Electrothermal actuation enables synchronized circumferential and longitudinal contractions, wrapping around the ventricles to augment systolic function. The modular design allows independent actuator group control for tailored left and right ventricular assistance. Performance was validated in benchtop models assessing pressure generation, frequency response, and endurance.

Results: The prototype generated peak pressures of up to 120 mmHg, sufficient to support left ventricular function. Operation at 1.4 Hz (84 bpm) closely matched physiologic heart rates, with frequency modulation enabling adaptation to rest and exercise demands. The device is powered by a safe low-voltage system (32.5 V), compatible with portable batteries, minimizing overall system footprint. Silicone encapsulation ensured electrical insulation, mechanical durability, and biocompatibility. Repeated actuation cycles confirmed robust endurance and stable contractile performance.

Conclusions: This study demonstrates the first soft robotic biventricular sleeve delivering synchronized, biomimetic contractile forces while preserving physiologic pulsatility. By eliminating blood contact, reducing infection risk, and operating with miniaturized power systems, the device addresses key limitations of conventional VADs. This work establishes a promising platform for next-generation fully implantable, physiologically compatible cardiac assist devices, with ongoing efforts directed toward in vivo validation and hardware miniaturization for clinical translation.

Selected Abstracts: 5 Minute Flash Talk Speakers

Amandeep Mondal, *The Heart Research Institute, NSW*

'Lactate-Dependent Reprogramming of Macrophages is Associated with Fibrous Cap Formation and Atherosclerotic Plaque Stability'

Background: Atherosclerosis remains a leading cause of cardiovascular mortality worldwide, with plaque rupture triggering myocardial infarction and stroke. Stable plaques feature a thick fibrous cap enriched in ACTA2^{+} myofibroblasts that maintain structural integrity, whereas rupture-prone plaques display cap thinning and loss of these reparative cells due to pro-inflammatory macrophage infiltration. Recent evidence highlights lactate as a key metabolite capable of reprogramming vascular and immune cells toward tissue repair. While lactate promotes anti-inflammatory macrophage polarization, whether it can reprogram inflammatory macrophages into ACTA2^{+} myofibroblast-like cells within the plaque remains unknown.

Methods: We analysed single-cell RNA-sequencing data from asymptomatic and symptomatic human plaques using single-cell Flux Estimation Analysis to map glycolytic and TCA cycle flux across macrophage subsets. Validation was performed by multiplex immunofluorescence imaging of human carotid endarterectomy plaques, profiling immune, smooth muscle, and lactate-responsive or matrix proteins. In vivo studies employed macrophage lineage-tracing mice (Apoe$^{-/-}$ Csf1r-Mer-iCre-Mer; ROSA26tdT/tdT), which underwent tamoxifen-induced labelling and 16-weeks of Western diet, receiving colchicine (30µg/kg/day) or vehicle. Aortic roots were examined by confocal microscopy for ACTA2, lactylated histones, Arginase1, and LDHA. In vitro, LPS-stimulated macrophages were treated with colchicine or the LDHA inhibitor FX11, and expression of Acta2 and Col1a1 measured after 48 hours.

Results: ACTA2^{+} macrophages in asymptomatic plaques exhibited enhanced glycolytic and TCA cycle flux, particularly in the pyruvate-to-lactate module. These cells co-expressed markers of lactate-associated activity and extracellular matrix remodelling. In mice, reduced inflammatory conditions induced by colchicine, increased cap thickness, and promoted ACTA2^{+}, Arginase1^{+} induction of Acta2 and Col1a1, reversed by LDHA inhibition.

Conclusions: We identify a novel subset of lactate-reprogrammed, ACTA2^{+} macrophages that stabilise the fibrous cap in atherosclerosis, uncovering a metabolic axis linking lactate signalling to macrophage-to-myofibroblast transition. Enhancing lactate-driven macrophage reprogramming may represent an innovative therapeutic strategy to prevent plaque rupture and reduce cardiovascular events.

Selected Abstracts: 5 Minute Flash Talk Speakers

Dr Jaideep Singh, *The Heart Research Institute, NSW*

'Novel Formylpeptide Receptor 1/2 Agonist Limits Hypertension-Induced Cardiovascular Damage'

Background: Chronic unresolved inflammation contributes to hypertension-induced cardiovascular damage. Although existing therapies manage blood pressure, substantial end-organ damage remains. Formylpeptide receptors (FPRs) play a critical role in regulating inflammation, an important driver of hypertension-induced end-organ damage. We have previously reported that a FPR small-molecule Compound17b (Cmpd17b) is cardioprotective against acute, severe inflammatory insults, but its impact on chronic, sustained inflammatory insult, i.e., hypertension-induced end-organ damage, has not been explored. We investigated the therapeutic potential of the Cmpd17b on blood pressure, cardiac and vascular remodelling, and function in hypertensive mice.

Methods and Results: Our approach encompassed a preclinical hypertension model (mice in vivo, human cardiac fibroblasts and aortic smooth muscle cells in vitro), quantitative cardiac and aortic proteomics, and mining of published hypertensive patient datasets. Hypertensive (Ang II-infused at 0.7 mg/kg/day, s.c.) and normotensive (saline-infused, s.c.) mice were implanted with radiotelemetry probes to record 24-hour blood pressure during 4 weeks of daily Cmpd17b (50 mg/kg/day, i.p.) or vehicle treatment. The hypertensive response to Ang II was attenuated by Cmpd17b. Impairments in cardiac and vascular function assessed via echocardiography were improved by Cmpd17b in hypertensive mice. This functional improvement was accompanied by reduced cardiac and aortic fibrosis, and vascular calcification. Cmpd17b also attenuated Ang II-induced increased cardiac mitochondrial complex-2 respiration. Proteomic profiling of cardiac and aortic tissues and cells by label-free nano-liquid chromatography-mass spectrometry quantified ~6000 proteins. Hypertension altered protein clusters associated with dysregulation of inflammatory, mitochondrial, and calcium responses, and networks associated with cardiovascular remodelling, contractility, and cytoskeletal organisation. Cmpd17b attenuated hypertension-induced dysregulation of multiple proteins in mice, and of these, ~110 proteins were identified as similarly dysregulated in humans suffering from adverse aortic remodelling and cardiac hypertrophy.

Conclusions: The FPR-agonist Cmpd17b limits hypertension-induced end-organ damage, consistent with proteome networks, supporting the development of pro-resolution FPR-targeted therapies for systemic hypertension complications.

Selected Abstracts: 5 Minute Flash Talk Speakers

Hannah Parker, *The University of Sydney, NSW*

'Investigating the Effects of In Utero High-Saccharide Exposure on Fetal Heart Development and Cardiovascular Health in Offspring'

Background: Cardiovascular diseases (CVD) are the leading cause of mortality worldwide. Growing evidence indicates that the in-utero environment is a critical factor in determining long-term cardiometabolic and cardiovascular health. This is concerning in the context of global dietary trends that prioritise low-quality, fructose-rich sugars over nutrient dense carbohydrates. Carbohydrate quality influences digestibility, absorption, and metabolism, which plays a critical role in shaping whole-body physiology. It is therefore important that maternal diets are primarily composed of high-quality carbohydrates to meet physiological and metabolic demands required to support fetal development. Suboptimal fetal development often induces compensatory changes postnatally, predisposing individuals to chronic conditions including CVD, type 2 diabetes, and obesity.

Previous work from our lab demonstrated that high-sucrose in-utero exposure can increase fetal heart weight (%body weight) in a sex-dependant manner. The isolated effects of fructose on fetal heart development and the mechanisms of maternal-fetal nutrient exchange, particularly saccharide and metabolite transport are not well understood.

Methods: C57BL6/J female mice (n=10-11/group) were allocated to one of four diets: brown chow, AIN93G, high-fructose or high-sucrose. Mice were mated at 8-weeks and sacrificed at E17.5. Fetal body and heart weight was taken during the sacrifice. Placentas were harvested for metabolomics.

Results: Fetuses from mothers consuming a high-fructose diet had reduced placental glucose transfer, which likely limited fetal nutrient and energy availability, contributing to an observed intrauterine growth restriction phenotype. This was characterised by a reduced body weight relative to the healthy control and an increased heart weight (%body weight) compared to the diet-matched control, with distinct sex-dependant effects. Placentas from these fetuses exhibited significantly elevated uric acid levels, suggesting oxidative imbalance and cardiometabolic stress.

Conclusions: These findings highlight the importance of maternal carbohydrate quality during gestation and the deleterious effects that maternal high-fructose intake can have on the in-utero environment, potentially acting as a programming agent for disease.

Selected Abstracts: 5 Minute Flash Talk Speakers

Wei Yi Lew, *The University of Sydney, NSW*

'Rapid On-Demand Monitoring of Valsartan and Sacubitril at the Point of Care With Miniature Mass Spectrometry'

Background: Heart failure pharmacotherapy largely revolves around minimising disease progression, managing symptoms, and preventing/reducing hospitalisation. Treatment guidelines emphasise the establishment of all patients on all four pillars via dose escalation to established target doses. Providing no effective 'reversal' of pathology, treatment is lifelong, and nonadherence is common. 20–50% of patients report poor adherence, of which is strongly associated with significantly worse disease progression, baseline health, and reduced symptomatic improvement. The one-size-fits-all approach to dosage, while effective, could represent an avenue for future improvement in personalised treatment. Our work establishes a rapid accessible point-of-care (POC) assay leveraging a portable miniature mass spectrometer (Mini-MS) to simultaneously quantify valsartan and sacubitril, the constituents of an angiotensin receptor–neprilysin inhibitor (ARNI) and one of four pillars, in plasma. This offers clinicians a capable tool for adherence monitoring.

Methods: The analytical performance of the assay was evaluated for sensitivity, accuracy, and reproducibility. Linearity was established using plasma spiked with valsartan in concentrations of 50, 250, 500, 2500, 5000, 10000 ng/mL, and sacubitril in 50, 250, 500, 1000, 2500, 5000 ng/mL. Reproducibility was assessed across 3 days with spiked plasma.

Results: Strong linearity was established across clinically relevant concentration ranges of 50–10,000 ng/mL for valsartan (R2 = 0.9902), and 50–5000 ng/mL for sacubitril (R2 = 0.9997). For valsartan, intra-assay coefficients of variation (CV) and inter-assay CVs ranged from 3.8–13.6% and 6.1–17.1% respectively. For sacubitril, these were 5.2–11.4% and 1.54–9.58%. Accuracies fell between 80–110% for valsartan and 93–114% for sacubitril.

Conclusions: In this early-stage proof-of-concept study, our Mini-MS–based POC assay enables rapid, accessible, and reliable quantification of valsartan and sacubitril, an ARNI representative of one of four HF pillars of treatment. This preliminary platform supports rapid low-hurdle adherence monitoring, pharmacokinetic monitoring studies, pharmacovigilance purposes, and ultimately the improvement of patient outcomes.

Lauren Cook, *Victor Chang Cardiac Research Institute, NSW*

'Developing an iPSC Model of Epicardial Adipose Tissue-Mediated Atrial Fibrillation'

BACKGROUND: Atrial fibrillation (AF) is the most common clinical arrhythmia, with obesity being a key modifiable risk factor. This is partly mediated through organ crosstalk between epicardial adipose tissue (EAT) and the adjacent atrial myocardium. However, the precise cellular mechanisms underlying this interaction remain unclear due to a lack of relevant human models.

Selected Abstracts: 5 Minute Flash Talk Speakers

Human-induced pluripotent stem cells (hiPSCs) can be used to generate adipocytes; however, current differentiation protocols lack efficiency and physiological relevance to EAT. This study aimed to develop and characterise a superior protocol for hiPSC-adipocyte generation and establish an in vitro coculture model to investigate EAT's proarrhythmic effect on atrial cardiomyocytes.

METHODS: hiPSCs were differentiated into adipocytes via a novel cardiac fibroblast progenitor pathway and compared against a standard mesenchymal stem cell (MSC)-based protocol. Adipocytes were assessed by lipid accumulation, droplet morphology, and adipokine secretome profiling. These adipocytes were cocultured with hiPSC-derived atrial cardiomyocytes to investigate proarrhythmic remodelling, with the electrophysiological properties of the cardiomyocytes assessed by optical mapping using calcium- and voltage-sensitive fluorescent dyes.

RESULTS: Adipocytes derived from the fibroblast pathway showed a 12-fold increase in lipid droplet number and a 1.8-fold increase in average lipid droplet area, demonstrating improved differentiation efficiency compared to the MSC method. They also exhibited a more physiologically relevant adipogenic profile, characterised by differential secretion of pro-inflammatory chemokines (e.g., MCP-1) and immune-recruitment molecules (e.g., ICAM-1). When cocultured with atrial cardiomyocytes, these adipocytes induced a proarrhythmic phenotype, characterised by slowed conduction velocity and increased electrical variability.

CONCLUSION: We have developed an improved protocol for generating hiPSC-derived adipocytes through a fibroblast progenitor pathway, producing cells that recapitulate key inflammatory features of EAT. Our coculture system provides a human in vitro platform to investigate the mechanisms of EAT-mediated atrial remodelling and offers a valuable tool for studying pathophysiology and screening novel therapeutic strategies to prevent AF.

Dr Tim Shiraev, *The University of Sydney, NSW*

'Medium-Term Outcomes of a Novel Tropoelastin Arterial Graft in a Large Animal Model'

Background: Cardiovascular diseases such as ischaemic heart disease, peripheral arterial disease and stroke contribute significant morbidity and mortality internationally. The gold standard for revascularisation of these stenosed or occluded vessels is bypass surgery in many cases, where a graft (either autologous or synthetic) is used to bypass the stenosis or occlusion. Unfortunately, long term patency rates of these bypass grafts are poor (especially polytetrafluoroethylene or PTFE, a commonly used synthetic graft), and obtaining these grafts also contributes morbidity and extra surgical time. An 'off the shelf' graft that addresses the above limitations would revolutionise both cardiac and vascular surgery, but despite significant research interest over decades, such a graft has not been developed.

Methods: The current study assessed the outcome of a novel tropoelastin graft in a large animal model. Ten sheep underwent implantation of bilateral carotid interposition grafts (randomised to

either sham surgery, PTFE, or a novel tropoelastin-polycaprolactone graft), and imaged with angiography and ultrasound for eight months.

Results: At 240 days, PTFE demonstrated a patency rate of 12.5%, versus 66.7% for the tropoelastin graft, and 100% for sham surgery. Aneurysmal change was noted in the tropoelastin grafts, with diameters increasing from 3.7mm to 6.1mm. Graft volumes were not significantly different between sham and tropoelastin grafts. Histology of the explanted grafts demonstrated significant remodelling, with formation of an elastic lamina in the tropoelastin grafts.

Conclusions: This study demonstrates excellent patency of a novel tropoelastin graft in a large animal model, and is the first to demonstrate formation of an internal elastic lamina in a large animal model. The aneurysmal change seen in the tropoelastin group indicates need for further modifications to graft structure to ensure positive remodelling.

Ramu Dumre, *The University of Wollongong, NSW*

'Docosahexaenoic Acid Supplementation Appears to Elicit an Omega-3 Index Response Irrespective of Dietary Patterns; Vegan/Vegetarian or Non-Restrictive'

Background: The Omega-3 Index (O3I) is the percent of eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) in total erythrocyte membrane fatty acids and is a biomarker of cardiovascular disease risk. Vegan and vegetarian (VGN/VTN) dietary patterns are associated with lower O3I status than non-restrictive (NR), however, both groups are at risk of sub-optimal status. Most studies have relied on fixed-dose and duration interventions but fail to reach, or not all participants reach, ≥8% O3I. There is a need for tailored study designs to ensure participants achieve 8-11% cardioprotective O3I. This study aimed to: (1) determine the duration required for daily 1g DHA supplementation to achieve 8% O3I in individuals consuming VGN/VTN or NR diets and (2) identify the factors that explain the variability of response.

Methods: Healthy study participants (mean age 36.4±15.1 years and BMI 24.9±3.9 kg/m2) consuming NR (n=38) and VGN/VTN (n=20) volunteered for this open-label pilot clinical trial (#ACTRN12625000253404). Blood pressure, height, weight, and waist circumference were measured at baseline (week 0). O3I was monitored at weeks 0, 4, 8 and 12 using finger-prick blood analysis (Fatty acid labs, Victoria).

Results: Baseline, mean ± standard deviation O3I in NR was 6.1%±1.1% compared to 5.2%±1.6% in VGN/VTN (p=0.005). At week 4, O3I increased to 8.1%±1.1% in NR (47% reached 8% O3I) and 7.4%±1.4% in VGN/VTN (20% reached 8% O3I) (p=0.036). O3I and DHA plateaued in weeks 4-12. 71% in NR and 40% in VGN/VTN reached 8% O3I by week-12. Baseline O3I explained 50% variability in O3I response, irrespective of dietary pattern.

Conclusions: The O3I increased substantially in the first 4 weeks of DHA supplementation, then appeared to plateau and not all participants reached ≥8% O3I. O3I response was mainly explained by baseline O3I. A higher DHA dose may be warranted, especially for VGN/VTN to achieve target ≥8% O3I.

Posters

1. Saksham Bhatia, *The University of New South Wales, NSW*
   'The Histological Impact of Sodium-Glucose Cotransporter-2 (SGLT2) Inhibitors in Patients with Non-Ischaemic Cardiomyopathy'

2. Zaid Bahi, *The University of Auckland, NZ*
   'The Impact of Pulsatility in Mechanical Circulatory Support on Renal Function'

3. Dr Dhanya Ravindran, *The Westmead Institute of Medical Research, NSW*
   'Novel Approaches for Recombinant AAV-mediated Gene Therapy in A Pre-clinical Model of Cardiac Arrhythmia'

4. Dr Nicholas Murray, *Victor Chang Cardiac Research Institute, NSW*
   'Exploring the Nkx2-5 locus architecture and its role in heart development'

5. Dr Jordan Thorpe, *Victor Chang Cardiac Research Institute, NSW*
   'Identifying New Therapeutic Approaches for Inflammatory Driven Atrial Fibrillation Utilizing Engineered Atrial Tissues'

6. Sutapa Saha, *Victor Chang Cardiac Research Institute, NSW*
   'Modelling the Influence of the Autonomic Nervous System in Atrial Fibrillation Using iPSC Models.'

7. Angela Rofail, *Victor Chang Cardiac Research Institute, NSW*
   'Exploring the Role of "Modifier Alleles" in KCNH2-related Long QT Syndrome'

8. Dr Chai Ng, *Victor Chang Cardiac Research Institute, NSW*
   'Patch Clamp Data Improve Risk Stratification in KCNH2-Related Long QT Syndrome'

9. Dr Joanne Ma, *Victor Chang Cardiac Research Institute, NSW*
   'A calibrated SCN5A functional assay enables the reclassification of 'Variants of Uncertain Significance' in patients with Brugada Syndrome'

10. Dr Matthew Graus, *The University of Sydney, NSW*
    'Propranolol Rescues an NR2F2-driven Orphan Syndrome via SOX18 Inhibition'

11. Eleni Dimos, *Victor Chang Cardiac Research Institute, NSW*
    'Molecular Insights into Congenital NAD Deficiency Disorder using Murine Models'

Posters

12. Dr Emma Rath, *Victor Chang Cardiac Research Institute, NSW*
    'Bile Acid Transporter Variants with Cross-Organ Influence May Be Contributing to Bad Outcomes After Paediatric Surgery'

13. Linda Dei-Awuku, *The University of Sydney, NSW*
    'High-Resolution 3D Bioprinting for Biomimetic Modelling of Neurovascular Thrombosis'

14. Muskaan Gupta, *The University of New South Wales, NSW*
    'Impact of Statin Therapy on the Risk of Stroke Recurrence, Mortality, and Dementia After Ischemic Stroke (ISMARDD Study): A Comprehensive Meta-Analysis'

15. Renata Sawyer, *The University of Sydney, NSW*
    'Developing Cyclic Peptides Targeting Protein Disulfide Isomerase A6, a Key Modulator in Ischemic Stroke-Associated Thrombosis''

16. Jianfang (Jenny) Ren, *The Heart Research Institute, NSW*
    'Unravelling Vorticity-Enhanced Platelet Aggregation in a Microfluidic Model of Stenotic Arterial Thrombosis'

17. Tiana Pelaia, *The University of Sydney, NSW*
    'Validation of a platelet storage device for research assays and point-of-care applications'

18. Ruby Soueid, *The University of Sydney, NSW*
    'Prescribing Patterns of Antiplatelet Therapy Following Percutaneous Coronary Intervention: Is CYP2C19 Testing of Clinical Value?'

19. Ashley Abe, *The University of Sydney, NSW*
    'Can Chimpanzees Provide the Answers to Coronary Artery Disease'

20. Amulya Regulagedda, *Victor Chang Cardiac Research Institute, NSW*
    'Establishing a Drug-Induced Aortic Aneurysm-Dissection Model to Study Spontaneous Coronary Artery Dissection (SCAD)'

21. Keziah Ambatt, *Victor Chang Cardiac Research Institute, NSW*
    'The Developmental Role of a Novel Low-Density Lipoprotein Receptor-Related Protein 1 Variant Implicated in Spontaneous Coronary Artery Dissection'

22. Dr Fergus Payne, *The Heart Research Institute, NSW*
    'cis-WOOH, a Tryptophan-Derived Hydroperoxide, Mediates Arterial Relaxation via the PKG-1α/BKCa Axis'

Posters

23. Millard Phiphatphol, *The Heart Research Institute, NSW*
'Mitochondrial H2O2 Contributes to Arterial Signalling via Formation of a Novel Hydroperoxide'

24. James Gallagher, *The Heart Research Institute, NSW*
'Detection of Mitochondrial Targeted Probes in Cardiovascular Disease'

25. Elaina Kelland, *The Heart Research Institute, NSW*
'Endothelial and Fibro-adipo-progenitor Cell Interactions as Drivers of PAD Pathogenesis in Diabetes'

26. Natalie Le, *The Heart Research Institute, NSW*
'Does Lower-Limb Ischemia Impact Cardiac Health? A Pilot Study'

27. Joshua Han, *The University of Sydney, NSW*
'Influence of Dietary Interventions or Fat/Oil Supplementation on LDL Oxidation in People with Diabetes Mellitus: A Scoping Review'

28. Laura Smith, *The University of Wollongong, NSW*
'Early Postprandial Exercise Improves Endothelial Function in Healthy Adults: A Systematic Review and Meta-analysis.'

29. Tewodros Wonde, *The University of Technology Sydney, NSW*
'Cardiovascular Risk Prediction in Women with Gestational Diabetes Using Risk Assessment Tools: A Scoping Review'

30. Fatemeh Heidari, *The University of Technology Sydney, NSW*
'Evaluating Potential Treatments Targeting Angiogenesis in Preeclampsia'

31. Samuel Hansen, *The University of Technology Sydney, NSW*
'In Vitro Biocompatibility of Zinc-Copper-Lithium Bioresorbable Alloys for Vascular Application'

32. Dr Qianyi Zhang, *The University of Sydney, NSW*
'Plasma adhered micropatterned DNA and protein microfluidic devices for DNA nanostructure and cell assembly'

33. Prof Zhen (Jeff) Luo, *The University of Technology Sydney, NSW*
'Auxetic Metamaterial-Inspired Coronary Artery Stents'

Posters

34. Kaan Ozguven, *The Heart Research Institute, NSW*
    'The Effects of L-Tryptophan on Blood Pressure in L-NAME Treated Mice'

35. Eustacia Karunalia, *The Heart Research Institute, NSW*
    'MitoQ: From Decreasing Superoxide to Blocking Calcium Channels'

36. Maryam Dashti, *Deakin University, VIC*
    'Exploring Relaxin Family Peptide 3 Receptor Expression In The Mouse Heart'

37. Dr Amy Nicks, *Victor Chang Cardiac Research Institute, NSW*
    'Layers of Silencing: DNA Methylation at Metabolic Gene Promoters Coordinates Cardiomyocyte Maturation'

38. Prof MD Golam Hossain, *Khulna University, Bangladesh*
    'Unlocking the Secrets of MyomiR Signatures: Novel Theranostic Strategies for Cardiovascular Disease Management'

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