



South Australian
Cardiovascular
Research Network

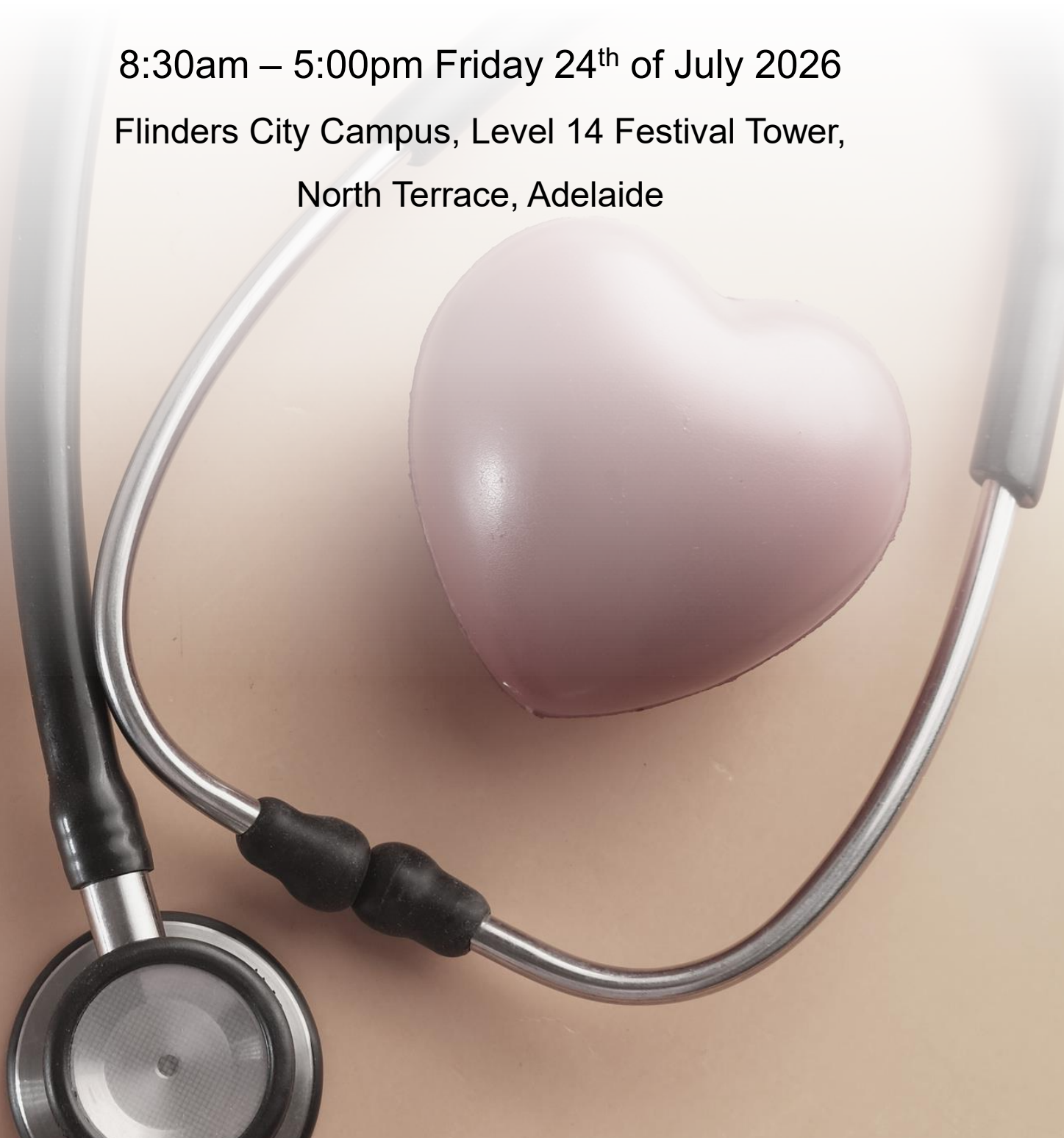


South Australian Cardiovascular Research Network Emerging Leaders Symposium

8:30am – 5:00pm Friday 24th of July 2026

Flinders City Campus, Level 14 Festival Tower,

North Terrace, Adelaide



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SA CVRN Membership



**South Australian
Cardiovascular
Research Network**

The SA CVRN, supported by the Heart Foundation, is a collaborative network connecting researchers, clinicians and consumers across South Australia. Through grants, fellowships, professional development opportunities and networking events, SA CVRN supports cardiovascular research excellence at all career stages.

How SA CVRN supports members

- Collaboration – Connecting researchers, clinicians, consumers and industry partners
- Capacity Building – Delivering workshops, webinars, mentoring and professional development opportunities
- Leadership Opportunities – Supporting member involvement through committees and working groups
- Funding and Fellowships – Promoting and supporting grants, travel awards and fellowship opportunities
- Advocacy – Championing the importance of cardiovascular research in South Australia

Why join?

- Connect with cardiovascular researchers across South Australia
- Access exclusive events, grants and professional development opportunities
- Stay informed about funding opportunities and research initiatives
- Help shape the future of cardiovascular research in South Australia

Membership is free and open to researchers, clinicians, health professionals, consumers and industry representatives interested in cardiovascular health.

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Join [SACVRN](#) today and help shape the future of cardiovascular research in South Australia.

Welcome

The Rising Stars Subcommittee of the South Australian Cardiovascular Research Network is delighted to welcome you to the **2026 Emerging Leaders Symposium** on **Friday, 24 July**.

This symposium celebrates the quality, diversity, and impact of South Australia's emerging leaders in cardiovascular research. Our goal is to create opportunities to share ideas, build meaningful connections, foster collaboration, and support professional development across all stages of the research career pathway.

Today's program showcases an outstanding breadth of cardiovascular research, featuring presentations from PhD candidates through to early- and mid-career researchers. The sessions span the full spectrum of cardiovascular research, from basic discovery science and translational research to clinical, public health, and population research, highlighting the strength and diversity of expertise within our network.

We are pleased to host two exceptional interstate plenary speakers, Dr Niamh Chapman and A/Prof Simon Foster, who will share their current research programs and provide advice and guidance to inspire the next generation of research leaders.

Today's program also incorporates a panel discussion featuring leaders in cardiovascular research, who will share their perspectives on building a successful and strategic research career with guidance on how to successfully navigate fellowship applications. New to this year's symposium is a debate session exploring contrasting views on the value of cross-disciplinary experience versus early career specialisation, as well as traditional approaches to peer review.

We are also excited to include a 'micro' mentoring session this year. We encourage you to use this opportunity to gain advice and insight from senior researchers in attendance. An exciting prize awaits those who engage in the most conversations during this session.

We encourage you to make the most of the day by reconnecting with existing collaborators, meeting new colleagues from different disciplines and institutions, and engaging with researchers whose work inspires you. By working together across disciplines and institutions, we can accelerate innovation and translate discoveries from the laboratory to the clinic, the community, and ultimately into better cardiovascular health outcomes for people across South Australia.

We sincerely thank our sponsors, the Heart Foundation, Flinders University, and Adelaide University, for their generous support and their commitment to investing in the future of cardiovascular research. Your partnership has made this symposium possible. We also extend a heartfelt thank you to all our invited speakers for their expertise, generosity and continued dedication to fostering and advancing the next generation of cardiovascular researcher leaders.

As you enjoy today's program, we encourage you to reflect on how our network can continue to foster collaboration and support career development of our emerging leaders.

Thank you for joining us, and we hope you find the symposium engaging, inspiring, and rewarding.

Yours sincerely,

Dr Emma Solly and Dr Sanuri Liyanage

Co-Chairs SA CVRN Emerging Leaders Symposium

Planning Committee



Dr Emma Solly



Dr Sanuri Liyanage



Dr Joyce Ramos



Dr Kacie Dickinson



Mr Jackson Howie



Ms Sania Aijaz

Flinders Festival Tower

Location:

- Flinders University Festival Tower (look out for the Flinders Uni banners), Level 14 via lifts.

Access Instructions:

- Level 14 requires swipe card access.
- Security personnel will be present at ground level to guide you if needed.



Program

Opening

8:30 – 9:00am	Registration
9:00 – 9:05am	Welcome/Acknowledgment Dr Anna Calkin

Plenary

Chairs: Dr Emma Solly and Dr Sanuri Liyanage

9:05 – 9:30am	Team-based and patient-centered approaches to transform hypertension management Dr Niamh Chapman
9:30 – 9:55am	Dissecting Cardiac Fibrosis: From Mechanisms to Drug Target Discovery A/Prof Simon Foster

PhD Award Presentations

Chair: Dr Clementine Labrosciano

9:55 – 10:05am	Raised lipoprotein(a) associates with rapidly progressive coronary atherosclerosis after acute coronary syndrome Rajesh Pathy Adelaide University and SAHMRI
10:05 – 10:15am	Deletion of the asialoglycoprotein receptor 1 (ASGR1) has atheroprotective effects with improved macrophage lipid handling and immune cell regulation Demeke Debebe Adelaide University and SAHMRI
10:15 – 10:25am	Impact of age on autonomic symptom burden and quality-of-life in individuals with Orthostatic Intolerance- Postural Orthostatic Tachycardia Syndrome (POTS) Vs. Postural Symptoms Without Tachycardia (PSWT) Gemma Wilson The Australian Dysautonomia and Arrhythmia Research Collaborative
10:25 – 10:35am	Coronary Angiography and Reperfusion Therapy Following Out-Of-Hospital Cardiac Arrest in Women Charlotte Miedel Capio St Göran's Hospital, Stockholm, Sweden
10:40 – 11:15am	Morning Tea and Poster Session

Program

Early Career Researcher Award Presentations

Chair: Dr Katharine Brown

11:15 – 11:25am	Cardiovascular risk classification changes in Australian primary care: comparing the old and new CVD Risk Calculators Kedir Ahmed Adelaide University
11:25 – 11:35am	Basilar artery versus anterior circulation large vessel occlusion code stroke process delays: a single centre cohort study. Pavandeep Singh Dhaliwal Royal Adelaide Hospital Neurology and Stroke Service
11:35 – 11:45am	Digital Home Monitoring of Obstructive Sleep Apnoea and Arterial Stiffness: a Longitudinal Study of Over 29,000 Adults Lucia Pinilla Flinders University
11:45 – 11:55am	Proteomic and Lipidomic Profiling of In-Stent Neoatherosclerosis Using a Novel Murine Model Jiandi Liu SAHMRI
11:55 – 1:00pm	Lunch and 'Micro' Mentoring

Panel Discussion

Chair: Dr Kacie Dickinson

1:00 – 2:00pm	Setting Yourself up for a Successful Research Career Dr Niamh Chapman A/Prof Simon Foster Dr Courtney Ryder Dr Sarena La
2:00 – 2:30pm	Afternoon Tea and Poster Session

Program

Debate Session

Chairs: Dr Emma Solly and Dr Sanuri Liyanage

2:30 – 3:45pm	1. Should early-career cardiovascular researchers prioritise broad cross-disciplinary experience over early specialisation?
	2. Is traditional peer review still the best mechanism for ensuring scientific reliability, or should it be replaced by more open and rapid evaluation models?
	Dr Niamh Chapman Prof Robyn Clark A/Prof Adrian Elliott Dr Sophie Wiszniak
3:45 – 4:00pm	Conclusions and Awards
4:00 – 5:00pm	Networking

Plenary Speakers

Dr Niamh Chapman



Dr Niamh Chapman is a Senior Research Fellow and leads the People and Systems Hypertension Research team at the University of Sydney. Dr Chapman's research focuses on adapting the way we deliver health services for the management of high blood pressure to better meet the needs of patients and healthcare providers.

Dr Chapman is an emerging leader in hypertension and cardiovascular research, as demonstrated by her roles as Chair of the Australian Cardiovascular Alliance Emerging Leaders committee and Chair of the International Society of Hypertension Women in Hypertension Research committee.

A/Prof Simon Foster



A/Prof Simon Foster is a Team Head at QIMR Berghofer, where he leads the Cardiac Drug Discovery Laboratory. He completed BA/BSc (Hons) degrees at the University of Melbourne and a PhD in biomedical sciences at the University of Queensland in 2014, focusing on cell signalling and G protein-coupled receptor (GPCR) biology, including the discovery of a novel receptor signalling system in the heart.

He subsequently worked in Denmark, leading a major orphan GPCR project supported by fellowships from the Lundbeck Foundation and the Danish Council for Independent Research, where he identified novel pairings between predicted human peptides and receptors. In 2018, he returned to Australia and developed phosphoproteomics workflows to analyse global signalling networks, before joining the Cardiac Bioengineering Group at QIMR Berghofer in 2020 to study inflammation-induced cardiac dysfunction in human cardiac organoids.

He is currently supported by a Bellberry-Viertel Senior Medical Research Fellowship and a National Heart Foundation Future Leader Fellowship, and his work focuses on identifying new therapeutic targets for cardiac fibrosis and heart failure.

Invited Speakers

A/Prof Courtney Ryder



Ryder is a nationally recognised Aboriginal injury epidemiologist, Co-Director of the FHMRI Health Equity Impact Program, Discipline Lead for Trauma and Injury at Flinders University, and a Matthew Flinders Fellow. With a background in biomedical engineering and a PhD examining health inequities in Aboriginal children with acute burn injuries, she brings expertise spanning quantitative, qualitative, and Indigenous research methodologies, including Indigenous Data Governance and Sovereignty. Her work is grounded in community-led approaches and relational ways of knowing, being, and doing.

Ryder has secured over \$31.3 million (\$14.4m CIA) in competitive funding. Her research includes the NHMRC-funded HEAL Cohort Study and the Nra:gi Ya:yun diabetes remission program, recognised as an NHMRC and Consumer Health Forum case study for excellence in community engagement. She is a recipient of multiple awards, including the 2025 SA Young Tall Poppy Science Award.

Dr Sarena La



Dr Sarena La recently completed her PhD with the Translational Vascular Function Research Collaborative at the University of Adelaide, the Basil Hetzel Institute and The Queen Elizabeth Hospital, where she was awarded the Dean's Commendation for Doctoral Thesis Excellence. She is currently a Postdoctoral Research Fellow at Adelaide University, supported by the South Australian Cardiovascular Network's inaugural Kick Start Fellowship. Her research focuses on patients with Non-Obstructive Coronary Artery (NOCA) syndromes, with particular interests in health outcomes, coronary vasomotor disorders, and integrating patient advocacy into cardiovascular research.

Invited Speakers

Distinguished Emeritus Professor Robyn Clark



Distinguished Emeritus Professor Robyn Clark previously held a joint appointment as Professor of Acute Care and Cardiovascular Research at Flinders University's Caring Futures Institute and Clinical Chair & Director of Nursing and Midwifery Research at the Southern Adelaide Local Health Network. She was the inaugural recipient of the NHMRC National Institute of Clinical Studies PhD Scholarship (supported by the National Heart Foundation), completed an NHMRC Training Fellowship (CIA) at QUT in 2013, and was a Heart Foundation Future Leader Fellow (2016–2020). Prof Clark holds fellowships with the FAAN, Royal College of Nursing, FAHA, FCSANZ, and FESC, and maintains adjunct appointments with multiple universities and institutes in Australia and internationally. She is a Life Member of the Australian College of Critical Care Nurses and the Australasian Cardiovascular Nurses Association.

She is internationally recognised for research improving access to evidence-based cardiovascular care for underserved populations, including women, older adults, rural and remote communities, low literacy and socioeconomic groups, and Aboriginal and Torres Strait Islander peoples. She has received over \$30 million in competitive research funding, led 28 Category 1 projects, and published more than 300 peer-reviewed papers (H-index 43).

A/Prof Adrian Elliott



A/Prof Elliott is a senior investigator within the Centre for Heart Rhythm Disorders at Adelaide University and leads a multidisciplinary team of cardiologists, exercise physiologists, and health scientists. His primary research interests focus on the care of patients with atrial fibrillation and heart failure through lifestyle intervention and new treatment pathways. He also leads a program of research focussed on the mechanisms and treatment of arrhythmias in endurance athletes.

Invited Speakers

Dr Sophie Wiszniak



Dr Sophie Wiszniak leads the Neural Crest and Cardiac Development Group at the Centre for Cancer Biology, Adelaide University. She is particularly interested in neural crest cell interactions with the heart and vasculature, and how defective neural crest development can underpin congenital heart defects and other developmental disorders. Her team uses genetic mouse models of embryo development, combined with imaging and molecular approaches to understand how these complex cell-cell interactions drive tissue morphogenesis, and how this can go wrong to underpin congenital disease.

Oral Abstracts

OP01: Raised lipoprotein(a) associates with rapidly progressive coronary atherosclerosis after acute coronary syndrome

Rajesh Pathy^{1,5}, Naomi Wattchow^{1,5}, Momina Allahwala^{1,5}, Thalia Salagaras¹, Lauren Sandeman¹, Natalie Cutri¹, Sanuri Liyanage¹, Julie Bradley², Adam J Nelson^{2,5}, Damini Dey³, Dennis Wong⁴, Jessica A Marathe^{1,2,5}, Christina Bursill^{1,5}, Peter J Psaltis^{1,2,5}.

¹South Australian Health and Medical Research Institute, Adelaide, Australia. ²Central Adelaide Local Health Network (Cardiology), Adelaide, Australia. ³Cedars Sinai Medical Group Inc, Los Angeles, United States of America. ⁴Victoria Heart, Melbourne, Australia. ⁵Adelaide University.

Background:

Lipoprotein(a) [Lp(a)] is a genetically mediated lipid molecule that independently associates with cardiovascular events, including recurrent myocardial infarction. Here, we examined for relationships between Lp(a) levels and progression of coronary atherosclerosis after acute coronary syndrome (ACS), as a potential mechanism for this increased risk.

Methods:

This multicentre, prospective study recruited 82 patients who had recent ACS with residual coronary stenoses after invasive coronary angiography. Participants underwent photon-counting coronary computed tomography angiography within 30 days and 12 months later to track the response of their residual atheroma to treatment directed by their usual clinicians. Paired scans were analysed by semi-automated software to quantify plaque burden, volume and composition across the entire coronary tree. Lp(a) concentration was measured at 12 months using an immunoturbidimetric assay, with levels >125 nmol/L considered elevated.

Results:

Mean age of participants was 61.8 years, 22% were female and 50% presented with STEMI. 96% were on statins at follow-up, with median LDL-C reducing from 2.9 to 1.4 mmol/L. 18% had elevated Lp(a). No significant differences in baseline characteristics existed for strata of Lp(a) levels for other risk factors, medication use, and risk factor control. Similar baseline measurements were observed for plaque volumes, burden and composition. High Lp(a), however, associated with greater progression of total plaque (TP) volume (+34.5 vs +5.0 mm³, P=0.020) and burden (+5.9 vs +0.1%, P=0.005), and noncalcified plaque (NCP) volume (+28.9 vs -3.8 mm³, P=0.030) and burden (+4.9 vs -1.2%, P=0.007) over 12 months. Using a pre-specified threshold of a >2% increase in total plaque burden to define plaque progression, 73% with high Lp(a) were plaque progressors compared to 34% with low Lp(a) (P=0.01).

Conclusion:

In this post-ACS cohort, where almost 20% had Lp(a) >125 nmol/L, high Lp(a) levels associated with a near five-fold increase in total and noncalcified plaque over the first year despite clinician-directed secondary prevention.

Oral Abstracts

OP02: Deletion of the asialoglycoprotein receptor 1 (ASGR1) has atheroprotective effects with improved macrophage lipid handling and immune cell regulation

Demeke Debebe Geremew^{1,2,3}, Liam Stretton^{1,2}, Lauren Sandeman¹, Benjamin Pullen^{1,2}, Victoria A. Nankivell^{1,2}, Emma Solly^{1,2}, Stephen J. Nicholls⁴, Nicole H. Packer⁵, Stephen Blake^{6,7}, Yung-Chih Chen⁴, Joanne T.M. Tan^{1,2}, Karlheinz Peter^{8,9}, Peter J Psaltis^{1,2,10}, Christina A. Bursill^{1,2}.

¹Vascular Research Centre, Lifelong Health Theme, South Australian Health and Medical Research Institute, Adelaide, South Australia, Australia 5000. ²College of Health, Adelaide Medical School, Adelaide University, Adelaide, South Australia, Australia 5000. ³Department of Medical Laboratory Sciences, Immunology and Molecular Biology Unit, College of Medicine and Health Sciences, Bahir Dar University, Bahir Dar, 6000, Ethiopia. ⁴Victorian Heart Institute, Monash University, Clayton, Australia 3168. ⁵Department of Molecular Science, Macquarie University, NSW 2109. ⁶Lynn Systems Immunology Laboratory, Precision Cancer Medicine Theme, South Australian Health and Medical Research Institute (SAHMRI), Adelaide, South Australia 5000, Australia. ⁷College of Medicine and Public Health, Flinders University, Adelaide, South Australia 5042, Australia. ⁸Atherothrombosis and Vascular Biology, Baker Heart and Diabetes Institute, Melbourne, Victoria, Australia 3004. ⁹Department of Cardiometabolic Health, Medical School, University of Melbourne, Parkville, Victoria, Australia 3010. ¹⁰Department of Cardiology, Central Adelaide Local Health Network, Adelaide, Australia 5000.

Background:

Loss-of-function mutation in asialoglycoprotein receptor 1 (ASGR1) associates with reduced coronary artery disease (CAD) risk. We aimed to determine if ASGR1 deletion causes atheroprotection and elucidate changes in macrophage function and immune cells.

Methods:

Flow cytometry assessed ASGR1 expression in bone marrow-derived macrophages (BMDMs) and immune cells. Apolipoprotein (Apo)e^{-/-}, Apoe^{-/-}Asgr1^{+/-} and Apoe^{-/-}Asgr1^{-/-} mice were fed high-cholesterol diet (HCD) for 16-weeks. Immune cells were assessed by flow cytometry, and lipids were measured enzymatically. Wildtype, Asgr1^{+/-} and Asgr1^{-/-} BMDMs were subjected to cholesterol efflux and oxidised low-density lipoprotein (oxLDL) uptake assays.

Results:

ASGR1 protein is expressed in cultured BMDMs, and in a host of immune cells in blood, aorta, liver and lymph nodes (LNs). Atherosclerotic Apoe^{-/-}Asgr1^{-/-} mice developed smaller (-22%) plaques in aortic sinuses than Apoe^{-/-} mice, P<0.05. Plasma and liver lipids were unchanged between groups. Apoe^{-/-}Asgr1^{-/-} mice had fewer aortic monocytes (-38%), inflammatory M1-like (-40%), fatty TREM2⁺ (-77%) and anti-efferocytotic CD47⁺ (-54%) macrophages than Apoe^{-/-} mice, P<0.01. Apoe^{-/-}Asgr1^{-/-} livers and iliac LNs also had fewer M1-like (Livers: -60%; LNs: -31%) and TREM2⁺ (Livers: -77%; LNs: -36%) macrophages, P<0.01. Circulating and aortic cytotoxic CD8⁺ T-cells (aorta: -45%; blood: -70%) and NKT cells (aorta: -69%; blood: -49%) were lower in Apoe^{-/-}Asgr1^{-/-} mice. In vitro, Asgr1^{-/-} BMDMs elicited more cholesterol efflux (+39%) and less oxLDL uptake (-15%) than wildtype BMDMs, P<0.05.

Conclusions:

ASGR1 is expressed in macrophages and other immune cells. ASGR1 deletion reduces plaque size, mediated by lowered monocyte, macrophage, CD8⁺ T and NKT cells, and enhanced macrophage lipid handling functions.

Oral Abstracts

OP03: Impact of age on autonomic symptom burden and quality-of-life in individuals with Orthostatic Intolerance- Postural Orthostatic Tachycardia Syndrome (POTS) Vs. Postural Symptoms Without Tachycardia (PSWT)

Gemma Wilson¹, Bora Nadlacki¹, Marie-Claire Seeley^{1,2}, Kristina Comacchio^{1,2}, Jonathan Chieng¹, Danielle Bailey¹, Eric Ong¹, Jonathan Foote^{1,2}, Nikki McCaffrey³, Celine Gallagher^{1,2}, Dennis H Lau^{1,2,4}.

¹The Australian Dysautonomia and Arrhythmia Research Collaborative (ADARC), College of Health, Adelaide University. ²South Australian Health and Medical Research Institute (SAHMRI). ³School of Health and Social Development, Institute for Health Transformation, Deakin University. ⁴Department of Cardiology, The Royal Adelaide Hospital.

Background:

Individuals with Postural Orthostatic Tachycardia Syndrome (POTS) frequently present with severe autonomic symptom burden and poor health-related quality-of-life (HrQoL). Our understanding of symptoms and HrQoL in those with orthostatic intolerance (OI) who do not meet the strict heart-rate criteria for POTS, is poorly understood. While research has demonstrated regression of orthostatic tachycardia with aging, there are no age-based adjustments within the diagnostic criteria for POTS beyond ≤ 19 years. We compared autonomic symptoms and HrQoL in POTS compared to Postural Symptoms Without Tachycardia (PSWT); examining age-related effects on Delta heart-rate (DHR) and symptoms.

Methods:

Consecutive patients undergoing OI assessment completed patient-reported outcome measures (PROMS) capturing autonomic symptoms and HrQoL. Between group comparisons utilised Chi-square for categorical variables and an independent t-test or Mann-whitney u-test for continuous variables as appropriate. Correlations between age, DHR, and PROMS were explored using Spearman's test. A secondary analysis was conducted utilizing age and sex-matching. Analyses used SPSS v29.0; significance set at $P < 0.05$.

Results:

A total of 414 POTS and 151 PSWT participants were included (88.6% female in POTS vs. 89.4% female in PSWT; $P = 0.800$). Median age was lower in POTS (28 IQR 21–36) than PSWT (38 IQR 26–51; $P < 0.001$). Overall autonomic symptom burden (COMPASS-31) was similar between groups ($P = 0.349$), while those with POTS demonstrated worse OI (COMPASS-31 OI subdomain; $P = 0.005$) and HrQoL (EQ-Utility; $P = 0.019$) than PSWT. After age and sex-matching, these differences were no longer significant ($P > 0.05$). Weak-to-moderate negative correlations were seen between age and DHR ($r = -0.34$, $P < 0.001$) and age and total COMPASS-31 ($r = 0.11$, $P = 0.01$).

Conclusion:

Comparable symptom burden and HrQoL in POTS and PSWT highlight limitations of DHR based criteria. As orthostatic tachycardia declines with age, current diagnostic thresholds may require re-evaluation.

Oral Abstracts

OP05: Cardiovascular risk classification changes in Australian primary care: comparing the old and new CVD Risk Calculators

Fernanda Nobre¹, Kedir Y Ahmed^{1*}, David Gonzalez-Chica^{1, 2}, Carla Bernardo¹, Abira Chandrakumar¹, Nigel Stocks¹.

¹*Discipline of General Practice, School of Medicine, Adelaide University, Adelaide, Australia.*

²*Adelaide University Rural Health, Adelaide University, Adelaide, Australia.*

**Presenting author*

Background:

In 2023, a modified Australian cardiovascular disease (CVD) risk calculator was introduced to improve the accuracy of risk prediction compared with the 2012 version. This study compared CVD risk estimates from the 2012 and 2023 calculators among adults attending Australian general practices and identified sociodemographic and clinical factors associated with reclassification to high risk using the updated calculator.

Methods:

We conducted a cross-sectional analysis using the MedicineInsight database, encompassing data from 427 general practices in Australia. Participants were regular adult patients (aged ≥18 years; 3+ consultations across two consecutive years) who attended a general practice in 2021 and were eligible for CVD risk assessment. CVD risk was estimated using information from multiple sources in the database, including reasons for encounters, prescriptions, diagnoses, pathology results, clinical observations, and medication records.

Results:

Of 438,865 adults without a history of CVD and eligible for risk calculation using the 2012 tool, 70.0% were classified as low risk. With the new 2023 calculator, 18.0% of these were reclassified as intermediate risk and 2.2% as high risk. Of that previously categorised as moderate risk, 18.0% were upgraded to high risk while 8.8% were downgraded to low risk. The new calculator also classified additional 63,781 adults considered ineligible by the 2012 tool, including First Nations aged 30-34 years (n=1,147; 97.9% intermediate risk), diabetic patients aged 35-44 years (n=11,379; 58.7% low risk) and those aged 75-79 years (n=51,255; 53.5% high risk). Older age, socioeconomic disadvantage, inner regional residence, and smoking were associated with higher odds of reclassification to high risk with the new calculator, while females and individuals from Western Australia had lower odds of reclassification to high risk.

Conclusion:

The 2023 CVD risk calculator reclassified approximately one in five adults from low to moderate or high risk, with more common in regional and socioeconomically disadvantaged populations. This reclassification should result in CVD risk factor management changes, including increased use of pharmacotherapy.

Oral Abstracts

OP06: Basilar artery versus anterior circulation large vessel occlusion code stroke process delays: a single centre cohort study

Pavandeep S. Dhaliwal¹, Michael Waters¹, Jackson Harvey¹, Matthew Wright¹, Laura Grimshaw¹, Allan Sabado¹, Timothy Kleinig¹.

¹Royal Adelaide Hospital.

Background:

Acute basilar artery occlusion (BAO) is a time-critical high-morbidity, high-mortality stroke subtype. Non-focal signs and coma in BAO may delay stroke recognition and treatment. We compared stroke recognition and imaging metrics for anterior large vessel occlusion (aLVO) (intracranial internal carotid artery and M1 middle cerebral artery) and BAO stroke patients in a contemporary large single centre cohort.

Methods:

This retrospective cohort study used prospectively collected data on all admitted non-transferred Royal Adelaide Hospital adult LVO stroke patients (2018-2025). We compared Code Stroke activation frequency, reperfusion rates and timing metrics.

Results:

Of 13,477 total stroke admissions, 1,538 LVO patients were identified (119 BAO and 1419 aLVO, median NIHSS 11(IQR5-25)vs 16(IQR8-22) and age 74 (IQR65-82)vs 76(IQR65-85) years. Onset-arrival time in patients with BAO in comparison to aLVO was similar (101 minutes in BAO vs 97 minutes aLVO, $p=0.316$). Code Stroke activation frequency was also similar (85% BAO vs 90% aLVO, $p=0.06$). Arrival door-imaging times for BAO patients were slower (32 vs 22 minutes, $p<0.001$), with prolonged door-needle (56 vs 45 minutes, $p=0.04$) and door-arterial puncture times (98 minutes vs 81 minutes, $p=0.02$). Thrombolysis (23% vs 20%, $p=0.47$) and thrombectomy rates (65% vs 60%, $p=0.452$) were similar.

Conclusion:

Patients with BAO had significantly longer door-to-imaging, door-to-needle and door-to-puncture times than with aLVO. This is potentially due to both delayed recognition and delays due to decreased conscious state. This study highlights a quality improvement opportunity.

Oral Abstracts

OP07: Digital Home Monitoring of Obstructive Sleep Apnoea and Arterial Stiffness: a Longitudinal Study of Over 29,000 Adults

Lucía Pinilla¹, Kelly Sansom^{1,2}, Philomène Letzelter³, Andrew Vakulin¹, Ashley Montero¹, Anna Hudson¹, Pierre Escourrou⁴, Jean-Louis Pepin⁵, Robert Adams¹, Peter Catchside¹, Bastien Lechat¹, Danny J Eckert¹.

¹Flinders Health and Medical Research Institute (FHMRI): Sleep Health, Flinders University Adelaide, Australia. ²Centre Interdisciplinaire du Sommeil, Paris, France. ³University Grenoble Alpes, HP2 Laboratory, Grenoble, France.

Background:

Pulse wave velocity (PWV), a proxy of arterial stiffness, is an independent predictor of cardiovascular events and mortality. Obstructive sleep apnoea (OSA) may accelerate vascular ageing through intermittent hypoxia and sleep fragmentation; however, previous studies relying on single-night sleep assessments have reported inconsistent associations with PWV. Given the substantial night-to-night variability in OSA severity, we investigated the independent associations of multi-night OSA severity and OSA variability with arterial stiffness using novel home-monitoring technologies.

Methods:

Adults were monitored using two FDA-cleared home devices over ~4 years (January 2021 to November 2024): an under-mattress sleep sensor (validated against gold-standard polysomnography) and a smart weight scale. Nightly OSA severity was quantified using the apnoea-hypopnoea index (AHI), and OSA variability was calculated as the root mean square of successive nightly AHI differences. Aortic-leg PWV was measured using the smart scale (validated against gold-standard applanation tonometry). Multivariable mixed-effects regression models assessed independent associations of OSA severity and OSA variability with PWV, adjusting for age, sex, and time-varying body mass index (BMI).

Results:

The study included 29,653 participants from 20 countries (age 52 ± 12 years; 84% male; BMI 27.3 ± 4.9 kg/m²), contributing a median [IQR] of 252 [103] sleep recordings and 38 [64] PWV measurements per year. OSA prevalence was 35% mild, 17% moderate, and 8% severe, and 40% no OSA. Mean PWV was 7.56 ± 1.02 m/s. Compared with participants without OSA, increasing OSA severity was associated with progressively higher PWV: +0.08 (0.06–0.10) m/s for mild, +0.14 (0.12–0.16) m/s for moderate, and +0.16 (0.13–0.19) m/s for severe OSA. Independently of severity, greater night-to-night OSA variability (13.7 vs. 2.4 events/h) was associated with a further increase in PWV of 0.24 (0.22–0.27) m/s. Participants with mild OSA but high variability had PWV levels comparable to those with severe OSA.

Conclusion:

Multi-night OSA severity and night-to-night variability were independently associated with greater arterial stiffness. These findings suggest that multi-night sleep monitoring captures clinically meaningful dimensions of OSA that are not reflected by single-night assessments and may enhance cardiovascular risk stratification and care.

Oral Abstracts

OP08: Proteomic and Lipidomic Profiling of In-Stent Neoatherosclerosis Using a Novel Murine Model

Jiandi Liu^{1,2}, Lauren Y. Sandeman¹, Lei Xiang³, Mingyue Liu³, Victoria A. Nankivell^{1,2,4}, Liam G. Stretton^{1,2}, Emma L. Solly^{1,2}, Robert A. McLaughlin^{2,4}, Joanne T. M. Tan^{1,2}, Peter J. Psaltis^{1,2}, Claudine S. Bonder^{2,5}, Jiawen Li^{3,4}, Christina A. Bursill^{1,2,4}.

¹Vascular Research Centre, Lifelong Health Theme, South Australian Health and Medical Research Institute (SAHMRI), Adelaide, South Australia, Australia. ²Faculty of Health and Medical Sciences, Adelaide University, Adelaide, South Australia, Australia. ³School of Electrical and Mechanical Engineering, Adelaide University, Adelaide, South Australia, Australia. ⁴Australian Research Council (ARC) Centre of Excellence for Nanoscale BioPhotonics (CNBP), Adelaide University, Adelaide, South Australia, Australia. ⁵Centre for Cancer Biology, Adelaide University and SA Pathology, Adelaide, South Australia, Australia.

Background:

In-stent neoatherosclerosis is increasingly recognized as a major cause of late stent failure and myocardial infarction, yet its molecular mechanisms remain poorly understood due to the lack of suitable preclinical models.

Methods:

We developed a murine model of in-stent neoatherosclerosis using stent implantation combined with carotid interposition grafting in apolipoprotein (Apo)e^{-/-} mice. Following surgery, mice were fed a high-cholesterol diet (HCD) or chow for 28 days. Plaque size and composition were assessed on vessel sections spanning stented regions. For omics analyses, mice were prepared for assessment of vessels with: (1) no plaque (healthy controls), (2) de novo atherosclerosis, (3) in-stent neointimal hyperplasia and (4) in-stent neoatherosclerosis. Proteomic and lipidomics were performed on vessel groups using mass spectrometry.

Results:

HCD induced lipid-rich, macrophage-infiltrated neointimal lesions within stented vessels, resembling human neoatherosclerosis. By contrast, chow-fed mice developed smooth muscle cell-rich neointimal hyperplasia. Proteomic analyses identified 27 neoatherosclerosis-specific proteins ($P < 0.05$) that distinguished neoatherosclerosis from neointimal hyperplasia, de novo atherosclerosis, and healthy vessels. Pathway enrichment highlighted alterations in cholesterol metabolism, inflammatory signaling, extracellular matrix remodeling, and lipid-handling pathways. Lipidomic analyses showed neoatherosclerosis shared a highly overlapping lipid profile with de novo atherosclerosis, supporting its identity as accelerated atherosclerosis. Class-level lipid analyses revealed higher levels of phosphatidylcholines, phosphatidylinositols and phosphatidylserines in neoatherosclerotic vessels, compared to neointimal hyperplasia and healthy vessels ($P < 0.001$ for all).

Conclusion:

This study establishes a translational murine platform for mechanistic investigation of in-stent neoatherosclerosis. Multiomics show neoatherosclerosis has a unique biological signature, with implications for the development of novel targeted therapies.

Poster Abstracts

PP01: Implementation of Evidence-Based, Person-Centred Alternative Delivery Models for Cardiac Rehabilitation in a Rural and Remote Population: The Country Heart Attack Prevention (CHAP) Project

Alline Beleigoli¹, Maria Alejandra Piñero de Plaza^{1*}

Lemlem G. Gebremichael¹, Norma Bulamu², Aarti Guyliani¹, Derek P. Chew², Stephen J. Nicholls³, Joyce S. Ramos¹, Carol Maher⁴, John Beltrame⁵, Billingsley Kaambwa², Vincent L. Versace⁶, Jeroen M. Hendriks^{1,5}, Rosanna Tavella⁵, Jonathon Foote¹, Katie Nesbitt¹, Sarah Powell¹, Wendy Keech⁷, Marie Ludlow⁸, Anthony Maeder⁹, Ken Wanguhu¹⁰, Hendrika Meyer¹¹, Ivanka Prichard¹, Orathai Suebkinorn¹, Christopher Zeitz⁵, Alex Brown¹², Robyn A. Clark¹

¹Caring Futures Institute, College of Nursing and Health Sciences, Flinders University; ²Flinders Health and Medical Research Institute, College of Medicine and Public Health; ³Victorian Heart Institute, Monash University; ⁴Alliance for Research in Exercise, Nutrition and Activity, University of South Australia; ⁵Adelaide Medical School, University of Adelaide; ⁶Deakin Rural Health, Deakin University; ⁷Health Translation SA; ⁸National Heart Foundation of Australia; ⁹Flinders Digital Health Research Centre; ¹⁰Royal Australian College of General Practitioners; ¹¹Yorke and Northern LHN, SA Health; ¹²South Australian Health and Medical Research Institute

**Presenting author*

Background:

Cardiac rehabilitation (CR) reduces cardiovascular mortality and readmissions, yet only 20–50% of eligible patients participate globally. Rural and remote populations face disproportionately high cardiovascular morbidity compounded by inadequate access to CR services. Before COVID-19, alternative delivery methods (telehealth, telephone, web-based) were rarely implemented, and absence of patient co-design hindered engagement. The CHAP Project aimed to develop, implement, and evaluate a co-designed, evidence-based, person-centred model of care to address barriers to CR participation in rural Australia.

Methods:

The CHAP model was introduced across fifteen rural and remote CR programs in South Australia (14 centre-based, 1 telephone-based), compared with eight metropolitan face-to-face programs (usual care). Using the Model for Large-Scale Knowledge Translation, we conducted systematic reviews, identified barriers through clinician collaboration, audited CR utilisation, and engaged rural services via the '4Es' approach (Engage, Educate, Execute, Evaluate). The model expanded delivery options (face-to-face, telephone with/without GP support, web-based) and included a 21-session continuing professional development program for clinicians. A matched prospective cohort (1:1 on age, sex, DRG complexity; n=1,913 per group) compared CR attendance, completion (primary outcomes), 12-month cardiovascular readmissions, mortality, emergency department visits, patient-reported outcomes, and cost-effectiveness. The RE-AIM framework evaluated implementation reach, adoption, fidelity, and maintenance.

Poster Abstracts

PP01 continued

Results:

CR attendance was comparable between groups (24.2% vs 23.8%; OR 1.15, 95% CI 0.89–1.47; $p=0.16$), but completion rates were significantly higher in CHAP (77.1% vs 57.5%; OR 1.69, 95% CI 1.30–2.18; $p<0.001$). Patient satisfaction was greater (85.9% vs 77.1%; $p<0.001$). No statistically significant differences were observed between groups in 12-month cardiovascular readmissions (HR 1.19, 95% CI 0.96–1.49; $p=0.17$), cardiovascular mortality (HR 1.70, 95% CI 0.92–3.16; $p=0.09$), or emergency department visits (HR 1.06, 95% CI 0.94–1.21; $p=0.33$). Cost per completion was lower in CHAP (\$6,542 vs \$8,689). Statewide implementation was estimated to generate healthcare savings of approximately \$2 million per year at 20% uptake, or \$10 million if all patients referred to CR in South Australia completed the program. Eighty percent of rural services adopted multiple delivery modes. The CHAP model directly informed the new SA Health Cardiac Rehabilitation Services Model of Care, now implemented statewide.

Conclusions:

The CHAP Project demonstrates that flexible, person-centred CR delivery significantly improves program completion and satisfaction without compromising clinical outcomes, while reducing cost per CR completion and showing potential healthcare savings at scale. Co-design with patients and clinicians, combined with quality improvement and primary care integration, enabled rural services to meet evidence-based standards more effectively. Broader implementation across all geographic areas is recommended to enhance the uptake and impact of CR in underserved populations. This work exemplifies successful translation from research to statewide policy and practice.

Poster Abstracts

PP02: Beyond Understanding: How Enjoyment and Preference Drive Emergency Clinicians' Adoption of RAPIDx AI

Maria Alejandra Pinero de Plaza, PhD¹

¹*Caring Futures Institute, College of Nursing and Health Sciences, Flinders University, Adelaide, SA, Australia*

Background:

The RAPIDx AI randomised controlled trial tested an AI-assisted clinical decision-support pathway for suspected cardiac chest pain assessment in emergency departments. While AI decision support can improve aspects of evidence-based care, sustained clinical adoption depends on how clinicians experience and integrate the tool in practice. The PROLIFERATE_AI framework was created to address this gap by quantifying the behavioural and contextual factors, including enjoyment, preference, trust and comprehension, that may shape whether clinicians adopt new technology in practice. This study asks: beyond understanding the interface, what makes emergency clinicians actually want to use RAPIDx AI?

Aim:

To examine whether intended use of the RAPIDx AI clinical interface is associated with behavioural constructs, including enjoyment, preference and trust, as well as comprehension, and to compare the relative strength of these associations.

Methods:

This study used a secondary analysis of de-identified clinician end-user data from the RAPIDx AI RCT (NHMRC GNT1191914; ANZCTR ACTRN12620001319965; SACHREC OfR no. 272.20). The analytic sample was restricted to clinicians with complete responses across the four threshold items and the trust measure. We analysed completed responses from a respondent-level Qualtrics export (n=18 emergency clinicians). Each clinician answered four threshold items that asked them to estimate, on a 0–100 scale, how many out of 100 trained users would: understand the interface (Q17—Comprehension); use it in their daily practice (Q18—Use); choose it preferentially over existing alternatives (Q19—Preference, interpreted as a proxy indicator of perceived workflow fit); and enjoy the experience of using it (Q20—Enjoyment). To convert these five-category threshold responses into continuous construct scores, we fitted a Beta(α, β) distribution for each respondent and each construct, enforcing monotonicity so that the survival probabilities matched the observed thresholds. Construct scores were taken as the fitted Beta medians. A separate 0–10 scale item measured Trust in AI decision support. We then estimated associations between all constructs using Spearman rank correlation coefficients, a non-parametric approach suited to small samples and bounded distributions.

Results:

Intended Use correlated strongly with Enjoyment ($p=0.78$, $p<0.001$) and Preference ($p=0.66$, $p=0.003$), and also with Comprehension ($p=0.69$, $p=0.001$). Trust in AI decision support (0–10) correlated with Enjoyment ($p=0.49$, $p=0.040$). The behavioural constructs (Enjoyment, Preference) showed comparable or stronger associations with Use than did Comprehension alone. Notably, the coupling between Enjoyment and Use ($p=0.78$) was the strongest observed relationship, exceeding even the Comprehension–Use association. These findings suggest that clinicians' enjoyment of the tool, and their preference for it over existing alternatives, may be at least as important for intended use as technical comprehension.

Poster Abstracts

PP02 continued

Conclusions:

These findings suggest that going beyond understanding may be important for successful AI adoption in emergency care. Behavioural factors captured by PROLIFERATE_AI—particularly enjoyment and preference—are tightly coupled to intended interface use. Adoption strategies should prioritise engagement design, trust-building, and seamless workflow integration alongside traditional training and information provision. These findings support a shift from conventional 'train-and-hope' implementation models toward behaviourally informed, human-centred AI deployment in high-stakes clinical environments. PROLIFERATE_AI offers a structured, quantifiable approach to evaluate adoption readiness and identify optimisation priorities before scale-up, helping bridge the gap between technical performance and real-world implementation in South Australian emergency departments.

Figure: Quadrant summary of respondent-level PROLIFERATE_AI constructs (n=18): A) Spearman correlation matrix; B–D) key bivariate associations showing Enjoyment–Use, Preference–Use, and Trust–Enjoyment relationships. Abbreviations: AI, artificial intelligence; RCT, randomised controlled trial.

Poster Abstracts

PP03: The Double-Edged Role of Glucocorticoids in Heart Development: Insights From a Sheep Model

Reza Amanollahi¹, Stacey L. Holman¹, Ashley S. Meakin¹, Kathy L. Gatford¹, Michael D. Wiese¹, Mitchell C. Lock¹, Janna L. Morrison¹

¹Early Origins of Adult Health Research Group, School of Pharmacy and Biomedical Sciences, Robinson Research Institute, College of Health, Adelaide University

Background:

Glucocorticoids (GCs), particularly cortisol, are essential for cardiac maturation, but premature or excessive exposure due to stress, disease, or antenatal GC therapy may disrupt this process. We hypothesised that GCs exert a dual effect on cardiac development, supporting maturation under physiological conditions but becoming detrimental when dysregulated.

Methods:

Fetal sheep left ventricle (LV) tissue was accessed from our Biobank including two studies: 1) preterm elevated cortisol (CORT, 109-116 days gestation (dG), term=150dG, n=8) vs near-term elevated cortisol (AGE, 140dG, n=7); 2) Antenatal betamethasone (BMZ, 138-140dG, n=12). Protein expression and hormone concentrations were analysed using western blot and LC-MS/MS, respectively. Data were analysed using one-way ANOVA (CORT-AGE) or a linear mixed model (BMZ) compared to its respective control group. $P < 0.05$ was considered significant.

Results:

Cardiac cortisol:cortisone was increased in CORT ($P < 0.0001$) and AGE ($P = 0.0462$), whereas it was reduced in BMZ ($P < 0.001$). Protein expression of $GR\alpha$ -A (main GC-responsive GR) was reduced in all cohorts, while the ratio of the antagonistic GR ($GR\beta$) to $GR\alpha$ -A was increased in AGE ($P = 0.0359$) and BMZ ($P < 0.001$), but not in CORT ($P = 0.4008$). IGF-1R was reduced as a result of GC elevation in all cohorts. In contrast, PCNA was increased in both CORT ($P = 0.0404$) and AGE ($P = 0.0213$) but reduced in BMZ ($P = 0.003$). A positive correlation between $GR\beta$: $GR\alpha$ -A and GLUT4 was observed in BMZ, but not in CORT or AGE.

Conclusions:

These findings highlight that the late-gestation rise in cortisol supports normal cardiac maturation, whereas exposure to synthetic antenatal GCs elicits distinct molecular responses. Such differences may have implications for cardiac health later in life.

Poster Abstracts

PP04: Fetal growth restriction alters mechanisms regulating cardiac oxygen sensing, oxidative stress, vascular remodelling and cell survival across gestation in the fetal sheep heart

Melanie R. Bertossa^{1\$}, Mitchell C. Lock^{1\$}, Stacey L Holman¹, I Caroline McMillen¹, Kimberley J Botting¹, Song Zhang^{1*} and Janna L Morrison^{1*\$} and *denote equal contribution

¹Early Origins of Adult Health Research Group, Health and Biomedical Innovation, School of Pharmacy and Biomedical Sciences, Robinson Research Institute, College of Health, Adelaide University, Adelaide, SA, 5000, Australia

Background:

Babies born with fetal growth restriction (FGR) are at risk of cardiovascular disease, which may have its early origins in fetal life. Using a sheep model of human pregnancy, we previously showed that FGR fetuses exhibit altered blood pressure regulation, fewer but larger cardiomyocytes, and a high-risk cardiometabolic phenotype. Herein, we investigated how FGR impacts the normal developmental maturation of oxygen sensing, oxidative stress, vascular remodelling, and cell survival mechanisms across early, mid, and late gestation in the fetal sheep heart.

Methods:

Non-pregnant ewes underwent carunclectomy surgery to induce FGR. Pregnant ewes were humanely killed at 55 (Control, n=19; FGR, n=17), 90 (Control: n=13; FGR, n=15) or 140 (Control, n=17; FGR, n=12) days gestation (dGA (term 150d)). RNA was extracted from fetal ventricular myocardium for targeted gene analysis (qRT-PCR). Data were analysed using two-way ANOVA (factors: FGR, age), with Bonferroni post hoc (*P<0.05).

Results:

FGR decreased fetal body and heart weight compared to Control fetuses at all ages (PFGR<0.0001). FGR increased cardiac PHD3 expression in late gestation compared with Controls (Pintx<0.0018). Other oxygen-sensing markers, either decreased with gestational age (HIF1 α ; Page<0.0001) or increased (PHD1 and PHD2; Page<0.0001), regardless of treatment. FGR increased expression of SOD1 and HO1 at all ages compared to Controls (PFGR=0.0040, <0.0001), suggesting an early and sustained antioxidant response. FGR increased SIRT1 expression in late gestation compared to Controls (Pintx<0.0065). FGR increased expression of S1PR11, but decreased ADM expression at all ages (PFGR<0.0001), indicative of early and sustained vascular remodelling phenotype. In contrast, vascular growth markers (VEGF, TIE2, and ANGPT1) decreased only in mid gestation, regardless of treatment (Page<0.0001), suggesting a low angiogenic developmental window. FGR increased expression of cell death markers, p27 and BNIP3, at all ages compared with Controls (PFGR=0.0112 and <0.0001).

Conclusions:

This study demonstrates that the fetal heart's normal developmental trajectory of oxygen sensing, oxidative stress response, vascular remodelling/growth, and cell survival mechanisms is altered when impacted by FGR, leading to a chronic-stress adaptive phenotype. Specifically, FGR induces phenotypes consistent with chronic oxidative stress with evidence for activated antioxidant defence, vascular remodelling without angiogenesis and cardiac cell death.

Poster Abstracts

PP05: Multifractal Fluctuations in Electrogram Dynamics Distinguish Atrial Fibrillation Phenotype, Drug Response, and Imminent Termination: Implications for Mechanism and Treatment

Darius Chapman¹, Campbell Strong¹, Ivaylo Tonchev^{1,2}, Scott Lorensini^{1,2}, Sobhan Salari Shahrababaki¹, and Anand Ganesan^{1,2}

¹College of Medicine and Public Health, Flinders University, Bedford Park, SA, Australia;

²Department of Cardiology, Flinders Medical Centre, Bedford Park, SA, Australia.

Background:

Atrial fibrillation (AF) is maintained by complex dynamics, characterised by periods of organisation and disorganised electrograms. We hypothesised that AF behaves as a critical complex system, and that multifractal analysis of AF electrograms could quantify non-stationary fluctuations to distinguish paroxysmal (PAF) from non-paroxysmal AF (NPAF), predict pharmacologic response, and identify imminent spontaneous termination, providing a marker of proximity to transition.

Methods:

>1.4 million seconds of high-density bipolar electrograms from 106 patients (52 paroxysmal, 54 non-paroxysmal) were mapped at standardised sites. Multifractal analysis using the Wavelet Transform Modulus Maxima (WTMM) method was applied to derive multifractal parameters c_0 (support dimension), c_1 (spectrum location), and c_2 (fluctuations). Mixed-effects models accounted for nesting within locations/patients. A flecainide sub-study ($n=15$) provided paired pre-/post-infusion recordings, and 27 spontaneous termination events in 15 patients were analysed.

Results:

AF electrograms exhibited robust multifractality, confirming multifractal fluctuations as an intrinsic property of AF. Non-paroxysmal AF showed significantly reduced fluctuations versus paroxysmal AF (c_2 : $\beta \approx -0.01$, $p=0.001$), indicating a paradoxical loss of fluctuations with disease progression. Flecainide selectively increased fluctuations in paroxysmal AF ($\Delta c_2 \approx +0.04$, $p<0.01$; $\Delta c_0 \approx +0.06$, $p<0.01$) but had no significant effect in non-paroxysmal AF, revealing phenotype-dependent drug response. Immediately prior to spontaneous AF termination, fluctuations increased significantly compared with sustained AF (c_2 : 0.198 vs 0.181, $p=0.024$). Spatial variogram analysis revealed heterogeneous patterns in paroxysmal AF, whereas non-paroxysmal AF displayed a homogenised, flattened fluctuations landscape.

Conclusions:

AF exhibits robust multifractal dynamics rather than random electrical activity. Reduced fluctuations characterise non-paroxysmal AF, whereas higher fluctuations are observed in paroxysmal AF, during flecainide modulation, and immediately prior to spontaneous termination. Multifractal fluctuations (c_2) may serve as a quantitative biomarker of disease progression, pharmacologic responsiveness, and proximity to termination, with implications for intra-procedural assessment of AF phenotype, drug selection, and recognition of imminent reorganisation.

Poster Abstracts

PP06: Exploring the Role of Neural Crest Derived Notch Signalling in Thymus Development

Briarn Dixon^{1,2}, Sophie Wiszniak^{1,2}

¹Adelaide University, Adelaide, Australia. ²The Centre for Cancer Biology, Adelaide, Australia

Background:

Neural crest cells are a critical source of the Notch ligands, which are imperative for thymus development, however the mechanisms of this relationship are unknown. A greater understanding of the importance of neural crest cell derived Notch signalling and its involvement with thymus growth and maturation is key to providing insight into developmental mechanisms and in turn may be used to treat thymus atrophy and involution, which can be seen in congenital diseases such as SCID and DiGeorge syndrome, allowing for the regeneration of T-lymphocyte production and increase in immune function.

We have previously demonstrated the importance of Notch signalling in neural crest cell development and migration using a novel mouse model where the Notch ubiquitin ligase Mindbomb1 (Mib1) is removed from all neural crest cells (Wnt1-Cre; Mib1^{fl/fl}). Following this, we hypothesised that defective activation of Notch ligands in neural crest cells leads to inadequate Notch signalling in other interacting cell types within the developing thymus, including the vasculature, underpinning thymus defects.

Methods & Results:

Through histological (H&E) and immunofluorescent (GFP-NCC, NG2, CD31) staining, we have shown that Wnt1-Cre; Mib1^{fl/fl} embryos have altered thymus vasculature, with a decrease seen in the vessel density within the thymus lobes, along with changes in the neural-crest derived pericytes that are imperative for vessel formation. Changes can also be seen in the surrounding carotid arteries, with the arteries often displaced, likely due to a decrease in Notch signalling and its downstream effects. Furthermore, we see changes in development and differentiation of thymic epithelial cell populations, suggesting neural crest-derived Notch ligands are involved in instructing this process.

Conclusions:

Current work is focussed on understanding the molecular mechanisms by which neural crest-derived Notch ligands regulate development of the vasculature, pericytes, thymic epithelia and T-cells, to provide new insight into the developmental mechanisms underpinning thymus defects.

Poster Abstracts

PP07: Roles for Neural Crest Cell-Derived Notch Ligands in Development of Pulmonary Stenosis

Dee Domingo PhD^{1,2}, Briarn Dixon^{1,2}, Lily Bright^{1,2}, Jasmine Hartmann^{1,2}, Ceilidh Marchant^{1,2}, Quenten Schwarz^{1,2}, Sophie Wiszniak^{1,2}

¹Adelaide University, Adelaide, Australia. ²The Centre for Cancer Biology, Adelaide, Australia

Background:

Congenital Heart Defects (CHDs) are the most common birth defect, affecting 1% of live births and accounting for 20% of miscarriages and 10% of still births. 20% of CHDs cases are caused by genetic or environmental factors however, in the remaining 80% the underlying cause is unknown, highlighting an important knowledge gap in CHD aetiology.

The pharyngeal arch arteries (PAAs) are a series of paired embryonic vessels with remarkably different fates. During development PAAs remodel into major arteries, including the aorta and pulmonary artery which facilitate blood flow to and from the heart. Neural crest cells (NCCs) are key architects of this process, forming the smooth muscle surrounding the PAAs and directing molecular signalling within the PAA environment. Aberrant PAA remodelling during development can lead to CHDs, including aortic arch malformations and pulmonary stenosis.

Methods & Results:

Using Immunofluorescence and Immunohistochemistry, We discovered that the PAAs, particularly the 6th PAA which contributes to the future pulmonary artery, are surrounded by unique expression patterns of Notch signalling target genes. To investigate differential PAA molecular environments driven by Notch signalling, we created a novel mouse model in which NCCs surrounding the PAAs can no longer initiate Notch signalling (Wnt1-Cre;Mib fl/fl). We found that NCCs are essential drivers of Notch signalling within the PAA environment and loss of this signalling disproportionately affects 6th PAA development, phenotypically presenting as pulmonary stenosis at full penetrance.

Using light-sheet microscopy, micro CT, and immunofluorescence sequencing we show that 1) Wnt1-Cre;Mib1fl/fl mice have pulmonary artery and valvular stenosis akin to 'Alagille Syndrome', 2) The 6th PAA is surrounded by a unique molecular environment with distinguishable expression of Notch, mTORC and mechanosensory components/downstream effectors and 3) loss of NCC driven Notch signalling disproportionately affects this unique molecular environment.

Conclusions:

Uncovering spatially restricted signalling pathways driving vascular development provides new mechanistic insight into CHDs affecting specific cardiovascular compartments.

Poster Abstracts

PP08: Dynamic Right Ventricular–Pulmonary Artery Coupling Responses Following Atrial Fibrillation Ablation

Kylie Hollitt^{1,2,3}, Jenelle, Dziano^{1,2,3}, Alexis Puvrez^{1,2,3}, Jackson Howie^{1,2,3}, Genevieve Kenny^{1,2,3}, Nicola MacDonald^{1,2,3}, Jonothan Ariyaratnam^{1,2,3}, Prashanthan Sanders^{1,2,3}, Adrian Elliott^{1,2,3}

¹Adelaide University, ²Centre for Heart Rhythm Disorders, ³Royal Adelaide Hospital

Background:

Right ventricular–pulmonary artery (RV–PA) coupling is an emerging marker of RV performance and load adaptation. Its response to atrial fibrillation (AF) ablation remains poorly characterised, despite the known relationship between AF, atrial remodelling, and RV afterload.

Methods:

TAPSE/PASP ratio identifies RVD undetected by conventional TAPSE alone. Although macro prevalence metrics remained static, continuous tracking reveals that individual responses to AF ablation are highly dynamic with over half of baseline RVD patients achieving meaningful functional recovery, regardless of rhythm status.

Results:

The cohort included 232 patients (age 69±10years; BMI 28±5 kg/m², 63% male and 57% paroxysmal AF). Prevalence of hypertension was 69.7% and obstructive sleep apnoea was 32.8%. Baseline uncoupling was present in 12.9% of patients (95% CI: 9.3%–18.4%). The TAPSE/PASP ratio identified a significantly higher proportion of right ventricular dysfunction (RVD) than TAPSE alone (McNemar's $p = 0.002$), with the two modalities showing only moderate diagnostic agreement (Cohen's kappa = 0.405). At 12 months ($n=108$) macro prevalence remained unchanged (McNemar's $p = 1.000$). However continuous TAPSE/PASP ratios significantly improved across the cohort (Wilcoxon $p=0.019$) driven by a strong trend towards reduced PASP ($p=0.07$). These changes were not significantly associated with atrial remodelling assessed by LAVi ($r_s = 0.375$ $p = 0.55$). These changes were independent of baseline rhythm status.

Conclusions:

TAPSE/PASP ratio identifies RVD undetected by conventional TAPSE alone. Although macro prevalence metrics remained static, continuous tracking reveals that individual responses to AF ablation are highly dynamic with over half of baseline RVD patients achieving meaningful functional recovery, regardless of rhythm status.

Poster Abstracts

PP09: Rapid Angiography-Based Determination of Coronary Shear Biomarkers

Yaofeng Ju¹, Nelson Tansu¹, Peter J. Psaltis^{2,3,4}, Mergen H. Ghayesh¹

¹School of Electrical and Mechanical Engineering, Adelaide University, Adelaide, South Australia, 5005, Australia; ²Vascular Research Centre, Lifelong Health Theme, South Australian Health and Medical Research Institute, Adelaide, South Australia, 5000, Australia; ³Adelaide Medical School, Adelaide University, Adelaide, South Australia, 5000, Australia; ⁴Department of Cardiology, Central Adelaide Local Health Network, Adelaide, South Australia, 5000, Australia.

Background:

Wall shear stress is a useful biomechanical marker for assessing local coronary flow conditions, but its clinical use is limited by the need for complex vessel reconstruction and computational modelling. This study presents a rapid angiography-based approach for estimating coronary flow and shear-related biomarkers from routine clinical images.

Methods:

Two sequential coronary angiographic frames acquired during contrast-agent washout were analysed. Vessel centreline and wall boundaries were extracted using image-processing techniques. Local changes in pixel intensity were then used to estimate contrast transport, from which near-wall velocity and wall shear stress were calculated.

Results:

The method was demonstrated in a representative patient with a stenotic mid-left anterior descending coronary artery. The image-derived wall shear stress distribution showed good consistency with the computational reference result, supporting the feasibility of rapid shear biomarker estimation from standard angiographic data.

Conclusions:

This approach provides a fast and practical way to obtain coronary flow and wall shear stress information without full three-dimensional CFD simulation. A wider range of patient cases has recently been published in *Frontiers in Bioengineering and Biotechnology* (DOI: 10.3389/fbioe.2026.1805161).

Poster Abstracts

PP10: The effect of catheter ablation-induced changes in heart rate on symptoms, functional capacity and cardiac remodelling

Nicola Macdonald^{1,2,3}, Jackson Howie^{1,2,3}, Jenelle Dziano^{1,2,3}, Genevieve Kenny^{1,2,3}, Alexis Puvrez^{1,2,3}, Shaun Evans^{1,2,3}, Melissa E. Middeldorp^{1,2,3}, Jonathan P. Ariyaratnam^{1,2,3}, Prashanthan Sanders^{1,2,3}, Adrian D. Elliott^{1,2,3}

¹Centre for Heart Rhythm Disorders, Adelaide University, ²South Australian Health and Medical Research Institute, ³Central Adelaide Local Health Network, Royal Adelaide Hospital

Background:

An increase in heart rate is frequently experienced after atrial fibrillation (AF) ablation. It has been postulated that this is due to modulation of the intrinsic autonomic nervous system during ablation and may in fact augment ablation success. However, the clinical implications on patient outcomes are unclear.

Methods:

Consecutive patients with symptomatic paroxysmal or persistent AF undergoing de novo catheter ablation were prospectively studied prior to, and 12-months post-ablation. The following were determined at each time point: AF symptoms using the atrial fibrillation severity score (AFSS) questionnaire; heart failure (HF) symptoms using Minnesota living with heart failure questionnaire (MLHFQ); functional capacity (VO₂peak) using cardiopulmonary exercise testing; left ventricular (LV) and left atrial (LA) structure and function by transthoracic echocardiography; NT-ProBNP was measured; and resting HR from 4-day holter monitors in patients in sinus rhythm at both timepoints. An increase in HR was determined if daily HR increased by >10%.

Results:

Of 93 patients, 53 had <10% HR change, while 40 had >10% HR change. Both groups had comparable age, BMI, risk factor profile and majority were male (76%). Patients showed no differences in type of energy used for ablation with majority having radiofrequency ablation (68%). At 12-months, there were no statistically significant between-group differences in AF(-2.7, 95% CI:-6.1 to 0.6 p=0.11) or HF(-6.1, 95% CI:-15.2 to 3.0 p=0.18) symptom scores; VO₂peak (0.2mL/kg/min, 95% CI:-2.0 to 2.4 p=0.31); NT-ProBNP(-30, 95% CI:-113 to 53 p=0.46); ventricular size and function; or LA volume indexed(-1.6, 95% CI:-6.1 to 3.0 p=0.48).

Conclusions:

Amongst patients who have undergone AF ablation, HR changes were not associated with differences in patient symptoms, functional capacity or cardiac structure and function at 12-months post ablation, suggesting autonomic changes do not influence patient outcomes.

Poster Abstracts

PP12: Patient-Specific Digital Twin Modelling of Carotid Fibromuscular Dysplasia for Biomechanical Risk Assessment

Kaveh Moghadasi¹, Eric Hu¹, Robert Fitridge^{2,3,4}, Jiawen Li^{1,5}, Mergen H. Ghayesh¹.

¹School of Electrical and Mechanical Engineering, Adelaide University, South Australia, ²Vascular and Endovascular Service, Royal Adelaide Hospital, Adelaide, Australia, ³Discipline of Surgery, Adelaide University, Adelaide, Australia, ⁴Basil Hetzel Institute for Translational Health Research, The Queen Elizabeth Hospital, Adelaide, Australia, ⁵Institute for Photonics and Advanced Sensing, Adelaide University, Adelaide, SA, Australia.

Background:

Fibromuscular dysplasia (FMD) of the carotid artery is a non-atherosclerotic and non-inflammatory vascular disorder associated with abnormal structural remodelling of the arterial wall. Although carotid FMD may alter local hemodynamics and vessel-wall mechanics, its biomechanical implications remain insufficiently understood. This study investigates the hemodynamic and structural responses of carotid arteries with different FMD morphologies to identify clinically relevant indicators of disease severity.

Methods:

A patient-specific carotid digital twin was developed based on a three-dimensional, two-way coupled fluid-structure interaction (FSI) framework, that simultaneously resolved pulsatile blood-flow dynamics, nonlinear arterial wall deformation, and downstream vascular impedance through physiologically based outlet constraints. Healthy, focal, non-focal, and severe non-focal carotid geometries were analysed to evaluate changes in hemodynamic indices and arterial wall responses.

Results:

The FMD models were associated with a markedly altered hemodynamic response compared with the healthy artery, including accelerated flow, increased wall shear stress, and reduced distal pressure. These findings indicate greater local flow disturbance and residence effects within the FMD-affected regions. Structurally, arterial wall deformation and von Mises stress were most pronounced in the non-focal phenotypes and focal model, respectively.

Conclusions:

Carotid FMD demonstrated more unfavourable hemodynamic behaviour and altered mechanical loading, providing insight into disease-related vascular behaviour across different FMD phenotypes. The proposed digital twin approach may support the identification of clinically meaningful biomechanical biomarkers for future patient-specific assessment. A more broader version of this study will be published in the journal of "Biomechanics and Modeling in Mechanobiology" for interested readers.

Poster Abstracts

PP13: Diroximel fumarate has atheroprotective and plaque-stabilizing effects in the tandem stenosis mouse model of plaque instability

Victoria Nankivell^{1,2}, Lauren Sandeman¹, Afreen^{1,2}, Dion Turner³, Joanne Tan^{1,2}, Liam Stretton^{1,2}, Demeke Geremew^{1,2}, Jiandi Liu^{1,2}, Peter J. Psaltis^{1,2,4}, Karlheinz Peter⁵, Yung Chih Chen⁶, Andrew Abell³ and Christina A. Bursill^{1,2}

¹Vascular Research Centre, South Australian Health and Medical Research Institute. ²Adelaide Medical School, Adelaide University. ³School of Physics, Chemistry and Earth Sciences, Adelaide University. ⁴Department of Cardiology, Central Adelaide Local Health Network. ⁵Atherothrombosis and Vascular Biology, Baker Heart and Diabetes Institute. ⁶Victorian Heart Institute, Monash University.

Background:

Oxidation and inflammation drive atherosclerotic plaque growth and instability. Diroximel fumarate (DRF) exhibits antioxidant and anti-inflammatory properties through activation of nuclear factor erythroid 2-related factor 2 (Nrf2) and suppression of NF- κ B respectively. We determined whether DRF exerts atheroprotective and plaque stabilizing effects.

Methods:

Using the tandem stenosis model of plaque instability, high-cholesterol diet fed apolipoprotein (Apo)e^{-/-} mice had two partial stenoses placed around the right carotid artery. Mice received vehicle or DRF (DRF50 or DRF100 mg/kg) daily via gavage. Unstable plaques were assessed histologically and circulating cells by flow cytometry. Nuclear protein immunoblotting and cholesterol efflux were assessed in immortalised bone marrow-derived macrophages (iBMDMs). Antioxidant and inflammatory mediators were measured by qPCR.

Results:

DRF100 reduced unstable plaque area (-68%, $P < 0.05$), Oil Red O+ lipid (-41%, $P < 0.001$) and CD68+ macrophage content (-42%, $P < 0.05$). There were fewer Ter119+ erythrocytes (DRF100: -93%, $P < 0.05$) and increased α SMA+ smooth muscle cells (DRF50: 3.1-fold, $P < 0.05$), indicating improved stability. DRF reduced circulating neutrophils (DRF50: -82%; DRF100: -54%, $P < 0.0001$), activated monocytes (DRF50: -24%; DRF100: -29%, $P < 0.01$) and CD8+ cytotoxic T-cells (DRF50: -47%; DRF100: -38%, $P < 0.01$). DRF increased antioxidants Gsta3 (DRF100: +46%) and Hmox1 (DRF100: +29%) in aortic arches ($P < 0.05$). DRF increased nuclear Nrf2 (+70%) and decreased NF- κ B (-58%) protein, upregulated Gsta3 (7.1-fold), Nqo1 (1.5-fold), Gpx1 (2.5-fold), downregulated Il6 (-82%), Il1b (-63%), Ccl5 (-88%), and increased cholesterol efflux (+29%) in iBMDMs (all $P < 0.05$).

Conclusions:

DRF reduces unstable plaque size and instability, via anti-inflammatory and antioxidant effects. Our findings have implications for DRF to stabilize high-risk plaque.

Poster Abstracts

PP14: A digital twin for coronary atherosclerosis: a case from the COCOMO study

Oscar Zi Shao Ong¹, Farajollah Zare Jouneghani¹, Kaveh Moghadas¹, Peter J. Psaltis², Mergen H. Ghayesh¹.

¹*School of Electrical and Mechanical Engineering, Adelaide University, Adelaide, SA, Australia,*

²*Vascular Research Center, Lifelong Health Theme, South Australian Health and Medical Research Institute (SAHMRI), Adelaide, SA, Australia.*

Background:

Although well-established clinical and biological factors, such as high lipids and uncontrolled inflammation, are known to contribute to ischemic risk in patients with coronary artery disease, they do not fully explain why certain plaques continue to progress and destabilise despite current standard-of-care. This unresolved gap highlights the need for mechanistic approaches that can capture plaque-specific vulnerability beyond conventional risk profiling. Accordingly, biomechanical modelling has emerged as a powerful framework for characterising plaque stress, hemodynamic loading, and arterial wall structural instability.

Methods:

Digital twin biomechanical modelling provides a patient-specific framework for simulating coronary hemodynamics and arterial wall mechanics. This modelling approach is known as fluid-structure interaction (FSI). A three-dimensional digital twin of the artery is first reconstructed through medical image analysis and cross section reconstruction of a patient from the COCOMO study. Within the fluid-structure interaction framework, displacement of the solid components, including the artery and plaque, is transferred to the fluid domain, while hemodynamic pressure and shear loading from the blood flow are transferred back to the artery-plaque interface.

Results:

The results show that, based on a comparison between baseline and follow-up optical coherence tomography (OCT) images, nearly 80% of plaque progression occurred in regions with high oscillatory shear index (OSI), indicating that OSI may be a reliable variable. Additionally, these high-OSI regions were co-localised with contraction and expansion areas identified by wall shear stress (WSS) divergence. Based on von Mises stress distribution through the arterial wall, the highest values occurred at minimum fibrous caps where is vulnerable to rupture. These areas are indicated and compared with corresponding real OCT images.

Conclusions:

Patient-specific digital twin FSI modelling identified biomechanical markers associated with plaque progression and vulnerability. These findings will support the use of biomechanical modelling as a new tool for improving heart attack prediction.

Poster Abstracts

PP15: A Digital Twin for Coronary Stent Deployment and Associated Stress Biomarkers in an Atherosclerotic Coronary Artery

Amir Sarviha¹, Rey Chin¹, Mergen H. Ghayesh¹

¹School of Electrical and Mechanical Engineering, University of Adelaide, South Australia, 5005, Australia

Background:

Coronary stenting is widely used to restore blood flow in narrowed coronary arteries; however, complications such as in-stent restenosis (ISR) and thrombosis may still occur following treatment. These adverse outcomes are influenced by post-implantation haemodynamics and arterial mechanics. This study investigates the stent deployment and post-implantation haemodynamics of a coronary artery.

Methods:

An idealised atherosclerotic coronary artery consisting of a multilayer arterial wall, plaque, and an Onyx Frontier-inspired coronary stent is developed. Stent deployment is first simulated using finite element analysis to obtain the deformed geometry. Subsequently, a two-way FSI model is employed to capture the interaction between pulsatile blood flow and the deformable artery under physiological conditions. Haemodynamic and mechanical biomarkers, including wall shear stress and von Mises stress, are evaluated following stent implantation.

Results:

The simulations demonstrate that stent deployment significantly alters the local flow field and arterial mechanical environment. Regions of disturbed flow and reduced wall shear stress are observed near the stent struts, while elevated von Mises stresses are concentrated within the plaque and adjacent arterial tissue. The maximum plaque von Mises stress reaches approximately 0.2 MPa, indicating substantial local mechanical loading following stent deployment. Haemodynamic analysis shows that 11.5% of the arterial surface is exposed to low time-averaged wall shear stress (TAWSS), while 7% exhibits high relative residence time (RRT), indicating flow stagnation. The two-way FSI approach captures arterial deformation throughout the cardiac cycle and identifies recirculation zones associated with neointimal hyperplasia and ISR.

Conclusions:

The proposed two-way FSI framework successfully quantifies both mechanical and haemodynamic biomarkers following coronary stent implantation. The model identifies flow recirculation zones, low-TAWSS regions, and increased RRT associated with neointimal hyperplasia and ISR. These findings highlight the potential of FSI modelling to improve understanding of post-stenting vascular responses and support future optimisation of coronary stent treatments.

Poster Abstracts

PP16: Asgr1-silencing lipid-based nanoparticles reduce inflammatory mediators and improve macrophage efflux

Liam G. Stretton^{1,2}, Afreen^{1,2}, Joanne T. M. Tan^{1,2}, Lauren Sandeman^{1,2}, Demeke Geremew^{1,2}, Juan Chen^{3,4}, Peter Psaltis^{1,2,5}, Gang Zheng^{3,4}, Victoria A. Nankivell^{1,2}, Christina A. Bursill^{1,2}

¹College of Health, School of Medicine, Adelaide University, Adelaide, South Australia, Australia 5000, ²Vascular Research Centre, Lifelong Health Theme, South Australian Health and Medical Research Institute, Adelaide, South Australia, Australia 5000, ³Princess Margaret Cancer Centre, University Health Network, Toronto, Ontario, M5G 1L7 Canada, ⁴Department of Medical Biophysics, University of Toronto, Ontario M5G 1L7, Canada. ⁵Department of Cardiology, Central Adelaide Local Health Network, Adelaide, Australia 5000.

Background:

A loss-of-function mutation in the asialoglycoprotein receptor 1 (ASGR1), was found to associate with a 34% reduction in coronary artery disease risk, highlighting a potential role for ASGR1 in atherosclerosis. Our lab has demonstrated that genetic deletion of Asgr1 reduces stable and unstable atherosclerotic plaque size and instability in preclinical models.

Aims:

To evaluate if Asgr1-siRNA targeted lipid-based nanoparticles, with fluorescent porphyrin lipid for detection, are internalised by cultured macrophages and elicit changes in cholesterol efflux and inflammatory mediators.

Methods:

Immortalised bone marrow-derived macrophages (iBMDMs) were incubated for 24 h with either a non-targeting scramble control (siScr-LNP) or an ASGR1 targeting (siAsgr1-LNP) lipid-based nanoparticle (50nM and 100nM). Nanoparticle uptake was assessed via fluorescence confocal microscopy (653nm). Inflammatory mediators and Asgr1 mRNA levels were measured using qPCR. Cholesterol efflux from 3[H]-cholesterol-loaded macrophages was assessed with a scintillation counter.

Results:

siScr-LNP and siAsgr1-LNPs were robustly taken up into macrophages and readily detectable at 653nm. siAsgr1-LNP treated iBMDMs had significantly lower Asgr1 mRNA levels at 50nM (↓78%, $P<0.05$) and 100nM (↓63%, $P<0.05$), compared with siScr-LNP controls. siAsgr1-LNP treated macrophages had lower mRNA levels of inflammatory chemokines Ccl2 (↓23%, $P<0.05$) and Ccl5 (↓16%, $P<0.05$), cytokine Il1b (↓23%, $P<0.05$) and transcription factor Rela (↓18%, $P<0.05$). siAsgr1-LNP incubated macrophages exhibited improved cholesterol efflux capacity (↑9%, $P<0.05$) compared to siScr-LNP controls.

Conclusions:

siAsgr1-LNPs are internalized by cultured macrophages, inhibit Asgr1 and elicit atheroprotective anti-inflammatory and cholesterol efflux enhancing effects. Our findings have implications for a novel plaque-targeting nanoparticle that inhibits ASGR1 efficiently and reduces atherosclerosis.

Poster Abstracts

PP17: Trends in the use of permanent pacemaker implantations in Australia: A 15-year nationwide study from 2008 to 2023

Neshi Weerasooriya¹, Anthony Salib², Brandon Truong¹, Samuel J. Tui^{1,3}, Shaun Evans^{1,3}, Alessandra Pina^{1,3}, Jonathan Ariyaratnam^{1,3}, Mehrdad Emami^{1,3}, Kurt C. Roberts-Thomson^{1,3}, Dennis Lau^{1,3}, Glenn D. Young^{1,3}, Prashanthan Sanders^{1,3}, Christopher X. Wong^{1,3}

¹Adelaide University, Adelaide, SA, ²Austin Health, Melbourne, VIC, ³Royal Adelaide Hospital, Adelaide, SA

Background

Permanent pacemakers are widely used to manage bradyarrhythmias; however, contemporary national trends are incompletely described.

Methods

Prospectively collected 15-year data on pacemaker insertions nationwide in Australia between 2008 and 2023 from the Australian Institute of Health and Welfare were analysed.

Results

Absolute pacemaker implantation counts increased by 79% from 12,153 in 2008 to 21,745 in 2023. Population-adjusted implantation rates increased by 43% from 57.2 to 81.6 per 100,000 persons. The rate of pacemaker implantations increased by 2.2% per year (IRR 1.022, 95% CI 1.021–1.022, $p<0.001$). Males had a 47% higher pacemaker implantation rate than females (IRR 1.47, 95% CI 1.46–1.48, $p<0.001$). Annual increases were observed in both males and females, at 2.5% in males (IRR 1.025, 95% CI 1.024–1.026, $p<0.001$) and 1.6% in females (IRR 1.016, 95% CI 1.015–1.017, $p<0.001$). While significant age-specific increases were observed in the 70–79 and >80 year age groups, trends were inconsistent or non-significant in younger age groups.

Conclusions

Population-adjusted pacemaker implantation rates rose significantly nationwide in Australia between 2008 and 2023, with greater increases amongst males and older age groups. The rising burden of devices has significant implications for healthcare resource planning and long-term clinical outcomes.

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