The STM Graduate Research Student Committee presents

The 17th Annual

Student Symposium & Careers Expo

The Innovation & Education Hub

@ The Alfred

3-4 October 2024

Contents

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We would like to acknowledge the Wurundjeri people of the Kulin nation, the Traditional Custodians of the land on which we gather today, and pay our respects to their Elders past, present, and emerging. We extend that respect to Aboriginal and Torres Strait Islander peoples here today.

Introduction

School of Translational Medicine (STM)

ACBD • APOM • Diabetes • Gastroenterology • HER Centre • Immunology
Infectious Diseases • Medicine & Surgery • Melbourne Sexual Health Centre
NCHA • Neuroscience • NTRI • Psychiatry • Respiratory • Surgery

STM Graduate Research Student Committee

The STM Graduate Research Student Committee (STM-GRSC) is a student-run committee that represents the voices and research of all STM graduate research students. The main objective of STM-GRSC is to serve as the primary liaison and advocate to the graduate research executive committee of the school and FGRC regarding STM graduate students' concerns and activities.

The goals of the committee are to:

- Represent the STM graduate research student body
- Provide useful resources to STM graduate research students that assist with transition and ongoing navigation through respective courses, including the creation of the STM Graduate Research Student Handbook
- Provide an accessible platform for communication among STM graduate research students
- Facilitate connections and networking between STM graduate research students and the wider community by organising social and professional events
- Organise the annual STM Graduate Research Symposium

Student Symposium & Careers Expo

The GR Student Symposium aims to showcase our students innovative research projects to their peers, colleagues, and industry partners. The Careers Expo provides an opportunity for students to network with the attending exhibitors and learn about the wide range of career opportunities available following their graduate research degrees. Previously two independent events, this year the committee decided to bring them together as a ***Research Extravaganza*** across two days!

Meet the Committee

Donggyu Rim
Co-Chair
Neuroscience

Jesse Mulder
Co-Chair
Immunology

Tamara Baker
Administration Manager
Neuroscience

Siqi Li
Treasurer
ACBD

Crystal Li
Faculty Graduate Student Committee Representative
Neuroscience

Lauren Evans
Media & Communications Manager
Neuroscience

Meet the Committee

Elyssa Osianlis
Media & Communications Team Member
Psychiatry

Thanh Xuan Le
Events Team Manager
Neuroscience

Mon Wittayacharoenpong
Events Team Member
Neuroscience

Julien Tran
Events Team Member
MSHC

Irja Isaksen
Events Team Member
Diabetes

Kristina Vanguardia
Events Team Member
Medicine

Careers Expo

Day One
Thursday 3rd October

TIME	SESSION
13:45 – 14:00	**Check-in**
14:00 – 15:15	**Panel Discussion –** *Alternate Post-Graduate Career Pathways*
15:15 – 15:25	**Exhibitor Presentation –** *EY*
	Exhibitor Presentation – *Mind Medicine Australia*
15:25 – 15:45	**Break – Afternoon tea**
15:45 – 16:45	**Networking Session** **Professional Headshots ***
16:45 – 17:00	**Closing session**

*Pre-registered spots only. Unfortunately there will be no opportunity for walk-ups.

Student Symposium

Day Two
Friday 4th October

TIME	SESSION	
9:30 – 10:00	Check-in	Hot drinks poured by  COFFEE RIDER & CO
10:00 – 11:00	Introduction & Keynote Speaker	
11:00 – 11:30	Break – Morning tea	
11:30 – 12:30	Oral presentations (5)	
12:30 – 13:30	Break – Lunch Networking	
13:30 – 14:30	Oral presentations (5)	
14:30 – 15:45	Poster Session* Break – Afternoon tea	
15:45 - 16:30	The Great Debate	
16:30 - 17:00	Award Ceremony & Closing Remarks	

*If you are presenting a poster, please stand by your poster during this time slot as this is when the judging will occur.

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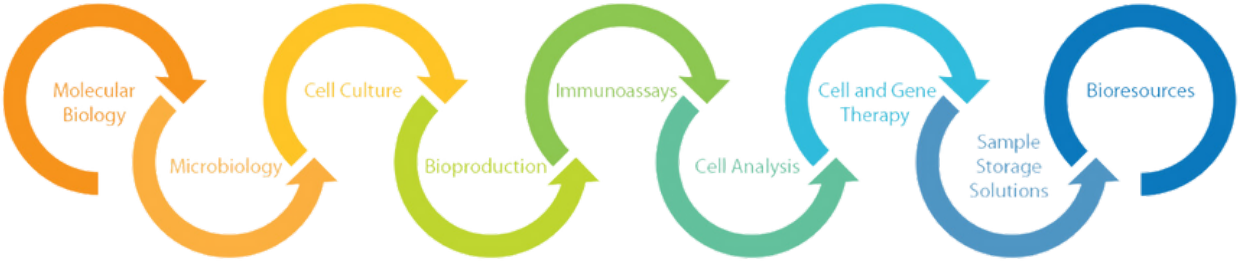

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Come and say hi to **Chris** during the Networking session or breaks in programme!

Day 1

October 3rd 2024

Panel Discussion

Alternative post-graduate career pathways

Okay, so we're all here because we are in research – but what are the other options apart from continuing on as a post-doctorate researcher? Do you fancy heading into private industry? Or the university space? What about starting your own business? How do you make these choices? We hear from our discussants on post-PhD career moves.

Chaired by

Crystal Li @LiCrystal_

PhD Candidate – *Department of Neuroscience*

Crystal is a third year PhD candidate under the supervision of Professor Richelle Mychasiuk in the Department of Neuroscience. Using female rodent models, her research explores how the cerebellum - an often-neglected brain region - is associated with processing pain relief, and the modulation of neuroanatomical pathways for pain inhibition.

A/Prof Vilija Joukabaitis @vjokubaitis

Deputy Head of Department (Laboratory Head) – *Department of Neuroscience*

A/Prof Joukabaitis leads the Neuroimmunology Genomics and Prognostics Group. She is a clinical and translational neuroscientist with skills in molecular medicine and biostatistics. Her research interest lies specifically, in the intersection between biology and clinical outcomes research. The Jokubaitis group is dedicated to improving the lives of those living with Multiple Sclerosis (MS) and related disorders. Their multidisciplinary team uses epidemiological, biostatistical, bioinformatic, and lab-based research methods to uncover important insights into what drives disability outcomes, and therefore leads to better care for people with MS and related disorders.

Dr Nick Parsons @nick_parsons_

Post-Doctoral Research Fellow (Imaging Neuroscience) – *Department of Neuroscience*

Dr. Parsons is a neuroscientist with a PhD in Traumatic Brain Injury. He is a research fellow in the Department of Neuroscience, focused on understanding neuroplasticity following stroke and other vascular diseases. He served as CEO of BrainCast Neurotechnologies, where he led the development of advanced quantitative MRI analysis software designed for sports physicians to quantitatively map, track, and predict subtle brain injuries in professional athletes, and for radiologists to automatically quantify neuropathology in patients.

Dr Claretta Dsouza @claryd85

Commerical Project Manager (Vaccine Development) – *Burnet Institute*

Dr Dsouza is a neuroimmunologist with six year's experience in the research commercialisation and industry partnerships space, coupled with over eight year's experience as a medical researcher in neurodegenerative diseases. Claretta investigated the role of platelets as precursors of neuroinflammation in diseases such as Multiple Sclerosis with an aim to determine the molecular basis of the disease and consequently, targets for therapy. As the Commercial Project Manager, Vaccine Development, at Burnet, her overall responsibility is to ensure the smooth execution of the Burnet Vaccine Initiative.

Dr Michelle Zajac

School Manager – *School of Translational Medicine*

Dr Michelle Zajac is currently the Manager of the School of Translational Medicine (STM). In this role, she has overall responsibility for planning and managing the School's business, (~$180M/year including $90M/year research funding) and professional service functions. This includes budgeting and financial planning; fostering and maintaining external relationships including with Alfred Health and the A+ precinct partners; recruitment; communications; space, infrastructure and facilities; planning and resources. She works closely with the Head of School and SubFaculty Dean to shape and action local strategic, business and operational plans for STM and the Sub-Faculty of Translational Medicine and Public Health. Michelle has a background in neuroscience, with a specific interest in gene x environment interactions, and 15 years experience in general management; strategic planning; research administration and operations; project and program management; and collaborations and partnerships. Prior to joining Monash, she had roles at the Cancer Therapeutics CRC and Biomedical Research Victoria.

Chris Mintoff

Territory Manager – *In Vitro Technologies*

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Chris has completed an MPhil in Medicine (Cancer Research) from the University of Melbourne, where his research focused on investigating the biological heritability of melanoma metastasis patterns using patient-derived xenografting. Following his academic pursuits, he gained further practical experience in breast cancer research before transitioning to lab management. His career in life sciences, research & development and management has been diverse, reflecting a commitment to advancing medical science. Recently, he joined In Vitro as a Life Science Territory Manager, where he continues to leverage his expertise to drive innovation and support scientific progress.

Professional Headshots

Hynesite Photography will be available for complimentary LinkedIn-style headshots for students!

Students who have **registered** will be assigned to one of two sessions to receive their headshots and will be informed of their allocation in advance.

Students **MUST** attend their assigned sessions and headshots will be taken on a first come, first serve basis.

Pre-registered spots only. Unfortunately, there will be no opportunity for walk-ups.

Open Networking

In the afternoon, students are all invited to an open networking session with **over 20 professionals** who will be representing organisations from across academia, education, government, and industry.

Networking exhibitors will be available to provide students with information about their respective organisations and **share their experiences and insights** into the career pathways that are available to graduate research students upon completion of their studies.

The full list of exhibitors has been provided in the coming pages. Exhibitors have provided several suggested **conversation starters** and topics to ask them about, however students are encouraged to also ask other questions based on their own professional interests and experiences.

What challenges did you experience as a post-doc?

How did you find the transition from academia to industry?

Did you find mentors useful in early career transition?

Do you find science skills to be well transferred in your industry?

Exhibitors

Chris Mintoff
Territory Manager

Bronze sponsor

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Rebecca Craythorn
Field Application Specialist

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Daniel Gilbertson
Director

Dr Nadia Khan
Senior Consultant

Dr Stephi Sievers
Senior Consultant

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Talk to us about: Working environment, projects, recruitment pathway

Exhibitors

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Peng Fan
Strategic Account Manager

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Talk to me about: Abcam products and services

Michael Valentine
Business Development Manager

APR.INTERN

Australian Postgraduate Research Intern (APR.Intern) is Australia's only national **industry internship program** for PhD, other Doctoral and Masters by Research students that spans all sectors and disciplines. A not-for-profit, the program works with all universities, nationally, to streamline pathways for postgraduate research students to be **industry-literate and career ready**; to expand university research collaborations; and to connect businesses with the country's brightest emerging research talent.

Find out more: www.aprintern.org.au

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Quinn Nguyen
Consultant
(Risk Consulting)

Lucillia Colman
Senior Manager
(Consulting)

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Talk to us about: Career pathways, opportunities at EY

Exhibitors

Emily Papakyriakopoulos
Student Experience Coordinator

Jake Chua
Postdoctoral Researcher

Pin Sun
Research Officer

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Talk to ua about: Career pathways, interviews, CVs, networking, opportunities at WEHI

Kevin Ponce
Field Account Manager

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Talk to me about: Working environment, career pathway, scientific sales

Ilan Hayman
Head of Operations

MIND MEDICINE AUSTRALIA

Mind Medicine Australia exists to help alleviate the suffering caused by mental illness in Australia through **expanding the treatment options** available to medical practitioners and their patients. We will establish safe and effective psychedelic-assisted treatments, focused specifically on the **clinical application of medicinal psilocybin and medicinal MDMA.**

Find out more: www.mindmedicineaustralia.org.au

Talk to me about: Career pathways, psychedelic therapies.

Exhibitors

Michele Sallaberger
Head of Clinical Operations

Adam Pirie
Business Development Manager

Alexia Smileski
Clinical Research Associate

neuroscience trials australia

NTA is a highly respected CRO playing a key role in transitioning research and development into **clinical practice**, and creating improved clinical outcomes for patients. NTA, a not-for-profit, continues to invest in the **neuroscience clinical ecosystem** by funding neuroscience research undertaken at **The Florey.**

Find out more: www.neurosciencetrialsaustralia.com

Talk to us about: NTA's services and capabilities, clinical research career opportunities

EMERITUS RESEARCH

Blair Stevens
Training Coordinator

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Talk to me about: Clinical trials, career pathways

Notes

Day 2

October 4th 2024

Keynote Speaker

Dr Shane Huntington OAM

Science Communication 101

Shane is an accomplished speaker, executive coach, workshop facilitator, MC, and strategist with a passion for science and communication.

In 1993 while doing his **Honours degree in Astrophysics/Optics,** Shane had his first serious taste of science communication **broadcasting on 3RRR** in Melbourne. Although already an accomplished presenter, successfully leading the RRR science team enabled him to finetune his communication and production skills. Shane has interviewed thousands of researchers, including Jane Goodall and Apollo astronaut Captain Gene Cernan.

Outside of the studio, Shane has given hundreds of **workshops and seminars** on communication, feedback, time management, and strategy. Shane has also provided career coaching - ranging from early career researchers to senior executives.

Since 1999 Shane has successfully run his own company, **The Innovation Group Pty Ltd.** Initially focused on commercialisation of research, the company is now the leading supplier of Atomic Force Microscopes in Australia and New Zealand - an accolade it has held for **almost two decades**.

Between 1998 and 2008 Shane worked as a Physicist at the University of Melbourne, successfully acquiring some $10m in grants over a 10-year period and **publishing some 80 papers**. Between 2005 and 2008 Shane established and led Quantum Communications Victoria (QCV), a $9m program to commercialise Australia's first export based on Quantum Physics.

Shane is currently the CEO of **Little Big Steps**, a cancer charity focussed on support for children undergoing treatment.

Presentation Judges

Oral Presentations

Ali Dvorscek
Immunology

Ross Dickins
ACBD

Zikou Lui
ACBD

Marcus Robinson
Immunology

Maria Lambouras
ACBD

Neha Kaul
Neuroscience

Rong Xu
ACBD

Angela Wan
Respiratory

Presentation Judges

Poster Presentations

Ren Giesler
Neuroscience

Be'eri Niego
ACBD

Madeleine Smith
Neuroscience

Will O'Brien
Neuroscience

Notes

The Great Debate

The much-loved (and often chaotic) Great Debate provides students and staff the chance to battle it out about the BIG issues (like whether it is better to be too hot or too cold, or that Gromit is the ideal sidekick).

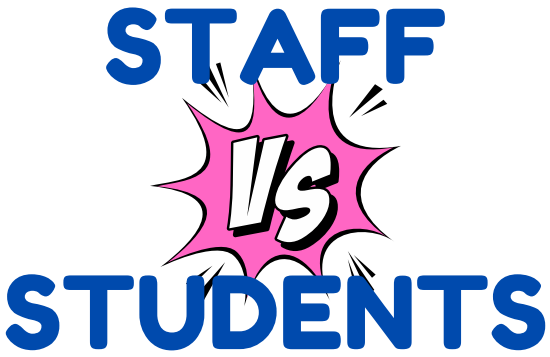
The Staff are the reigning champions...who will take the crown this year?

Three topics, two sides, one winner!

Glenn Yamakawa
Neuroscience

Bernadette Jones-Freeman
Immunology

Mastura Monif
Neuroscience



Sydney Harris
Neuroscience

Irja Isaksen
Diabetes

Joshua Nicholls
Neuroscience

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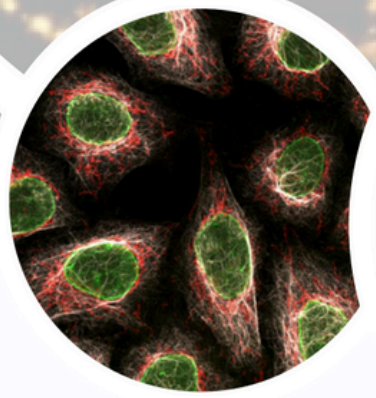

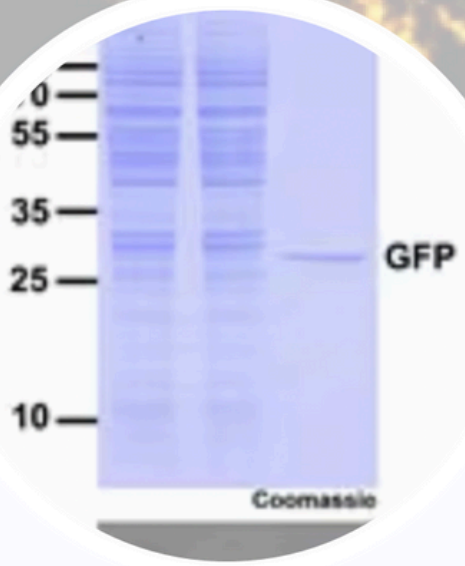

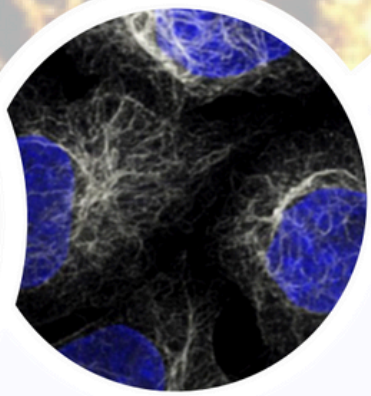

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Come and say hi to **Rebecca** during the Networking session or breaks in programme!

Student Abstracts

Alexandra Bosak Karaviotis

Department of Immunology

Poster Oral

Identification of a unique plasma cell population that resists survival factor depletion

INTRODUCTION: Plasma cells (PC) are terminally differentiated B cells that confer protective humoural immunity through the production of antibodies. It is evident that PC rely on a supportive tissue microenvironment and the provision of trophic signals to survive in the body. However, the relative contribution of external stimuli within the microenvironment to downstream PC survival programs and persistence is unclear. We aimed to examine how the in vivo depletion of two external survival factors, APRIL and BAFF, influences PC persistence and determine whether PC subsets are differentially sensitive to the loss of these factors.

METHODS: A genetic time-stamping approach was used to indelibly label PC with human CD4 at the time of tamoxifen gavage. At the point of maximal PC labelling, APRIL, BAFF, or both were depleted using neutralising monoclonal antibodies. The proportion, phenotype and isotype of persisting hCD4+ PC of the spleen and bone marrow was assessed using flow cytometry.

RESULTS: Splenic and bone marrow PC populations were substantially reduced upon neutralisation of both APRIL and BAFF but were unperturbed upon single blockade. In the small marrow population that was resistant to the loss of APRIL and BAFF, PC that were IgG+ and had acquired an aged phenotype (MHC-II-SLAMF6-) were enriched for.

CONCLUSION: This work identified unique phenotypic and molecular traits of the PC that resist death during extrinsic factor, specifically APRIL and BAFF, dearth. Future work will look to dissect the downstream PC survival programs affected by APRIL and BAFF signalling.

Amalia Khayyira

Department of Diabetes

Poster

Exploring the gut-kidney-axis in a mouse model of diabetic kidney disease

INTRODUCTION: Emerging research has unveiled a potential link between intestinal barrier integrity and kidney injury, known as the gut-kidney-axis. We have shown evidence of an impaired intestinal epithelial barrier in a mouse model of DKD (Leprdb/db mice). Larazotide is intended to improve intestinal permeability, but it has not been investigated in DKD. This research aims to investigate whether larazotide can improve diabetic kidney disease (DKD) in a mouse model of type 2 diabetes.

METHODS: Eight-week-old Leprdb/db mice were treated with 20mg/kg/day larazotide in drinking water for 10 weeks. FITC-dextran was used in vivo to assess intestinal tight junction permeability. Kidney injury was evaluated by urinary albumin-creatinine ratio, and structural damage was assessed by glomerulosclerotic index scoring.

RESULTS: Diabetic Leprdb/db mice displayed greater intestinal permeability when compared to non-diabetic controls, as shown by the FITC-dextran assay (4.322±1.081µg/ml vs 1.597±1.126µg/ml, $p<0.05$) which was not improved by larazotide treatment. Unexpectedly, larazotide exacerbated albuminuria in diabetic mice (81.81±28.38µg/µmol vs 125.6±34.93µg/µmol, $p<0.05$). This finding corresponds to the further increase in kidney weight in the larazotide-treated diabetic mice (0.014±0.002 vs 0.013±0.001, $p<0.05$). No changes in glomerulosclerosis were observed with larazotide treatment.

CONCLUSIONS: Leprdb/db mice exhibited increased albuminuria, kidney hypertrophy and intestinal permeability compared to non-diabetic controls. However, larazotide treatment did not improve intestinal permeability. Notably, kidney injury was exacerbated in Leprdb/db mice treated with larazotide. This finding has raised an intriguing question of whether larazotide is nephrotoxic in the setting of diabetes. Further investigations are required to unravel the underlying mechanisms of this observation.

Cassandra Marotta

Department of Neuroscience

Poster Oral

Brain-derived tau as a biomarker of treatment effect in Alzheimer's disease and behavioural variant frontotemporal dementia

INTRODUCTION: Novel biomarkers are needed to measure treatment response in neurodegenerative diseases. Brain-derived tau (BD-tau), a cerebrospinal fluid (CSF) protein biomarker specifically from brain-derived sources, has previously been shown to differentiate Alzheimer's disease (AD) from other neurodegenerative diseases. This study aimed to determine if BD-tau may also be a potential biomarker of treatment effect in clinical trials of a potential disease modifying drug, sodium selenate, which acts by reducing pathological hyperphosphorylated tau, in patients with AD and behavioural variant frontotemporal dementia (bvFTD).

METHODS: We investigated BD-tau and p-tau217 levels after treatment with sodium selenate in two clinical trials in patients with AD and bvFTD. We also examined the association of these measures with levels of t-tau, p-tau181 and A42.

RESULTS: CSF BD-tau levels decreased after treatment with sodium selenate in patients with AD, however they did not change in patients with bvFTD. No change in p-tau217 was seen after treatment in either AD or bvFTD cohorts. CSF t-tau and p-tau181 correlated with CSF BD-tau in both the AD ($r = 0.9113$ and 0.7746, $p < 0.0001$) and bvFTD ($r=1.0$, $p=0.004$ and $r=0.79$, $p<0.05$) cohorts. In the bvFTD cohort CSF BD-tau did not correlate with serum or plasma BD-tau ($r<0.32$, $p>0.5$).

CONCLUSIONS: CSF BD-tau shows potential as a biomarker of treatment effect in patients with AD, however not in patients with bvFTD. Further research is needed to investigate the feasibility of BD-tau as a measure of treatment effect in blood-based samples and its use in other neurodegenerative diseases.

Charlotte Copas

Department of Neuroscience

Poster

Aerobic exercise as a potential therapeutic intervention for post-traumatic stress disorder and persistent post-concussion symptoms in women with a history of intimate partner violence

INTRODUCTION: Intimate partner violence (IPV) is a pervasive medical concern affecting millions of people worldwide, with the majority being women. The nature of IPV often results in severe neurological consequences, with the majority of victim-survivors being at risk for a lifelong prevalence of post-traumatic stress disorder (PTSD) and persistent post-concussion symptoms (PPCS) due to repeated physical assaults to the head and neck.

Despite the global prevalence of IPV and high comorbidity rates of PTSD and PPCS in victim-survivors, there is a profound lack of evidence-based treatment options for this population. The primary aims of this study are to explore the potential therapeutic effects of a four-week aerobic exercise program versus a passive stretching paradigm on PTSD and PPCS symptomology in women with a history of IPV. Second, we will be investigating whether aerobic exercise leads to changes in blood biomarkers or cognitive measures compared to passive stretching, and finally assessing the feasibility and adherence of a 4-week exercise program in this population.

METHODS: Using participant-directed surveys, blood biomarkers, and a battery of cognitive tests, we aim to compare physiological and psychological changes in victim-survivors who complete a four-week aerobic exercise program versus a passive stretching paradigm. To measure feasibility and adherence, we equip each participant with a Fitbit smartwatch to monitor their heart rate in order to ensure they follow their assigned routine.

CONCLUSIONS: Results from this research will aid in the facilitation of improved therapeutic practices and individualized treatment options for victim-survivors experiencing PTSD and PPCS related to IPV.

Christina Kazzi

Department of Neuroscience

Poster

Clinical utility of a computerised cognitive test in identifying immune effector cell associated neurotoxicity syndrome following chimeric antigen receptor t-cell therapy

INTRODUCTION: Change in cognition is characteristic of immune effector cell-associated neurotoxicity syndrome (ICANS), which is a potentially life-threatening complication of chimeric antigen receptor T-cell therapy (CAR-T). This study investigated the clinical utility of a computerised test of processing speed, visual attention, and working memory (CARTcog) in identifying ICANS.

METHODS: Thirty-one patients underwent CAR-T at the Alfred Hospital in Melbourne between 28 August 2023 and 23 April 2024. 28 patients consented to the study. Participants completed serial CARTcog assessments and immune effector cell-associated encephalopathy (ICE) scores at baseline, daily during inpatient admission, and 1-, 3-, and 6-month time points. Trajectory plots of ICE scores and CARTcog scores across time were fitted with loess smoothing functions. Linear mixed-effect modelling was constructed to investigate the association between ICANS status (predictor) and CARTcog response measures (outcome). Receiver operator characteristic curves were constructed to investigate the discriminative ability of CARTcog measures in predicting ICE scores.

RESULTS: Twenty-eight participants (78% male, 64.07±12.49 years old at infusion) completed 345 CARTcog assessments. Eight (29%) patients developed ICANS. Trajectory plots illustrated a temporal relationship between ICE scores and CARTcog scores; in general, CARTcog scores worsen with decreases in ICE scores and recover with the improvement of ICE scores. Linear mixed-effects models revealed a significant interaction between time and ICANS groups ($p<0.05$), such that the patients who developed ICANS performed worse over time compared to those who did not develop ICANS. CARTcog scores were found to distinguish between patients with ICANS (i.e., ICE score<10) and those without ICANS (i.e., ICE score=10) (AUC>0.95 across different scores).

CONCLUSION: CARTcog has the potential to be used in conjunction with ICE scores to improve the detection of ICANS in patients who receive CAR-T. Earlier detection of ICANS, and hence, earlier treatment and management, can lead to improved patient outcomes.

Ebony Blight

Department of Immunology

Poster Oral

Functional assessment of the NOD2 signalling pathway in patients with inborn errors of immunity

INTRODUCTION: Majority of patients with an inborn error of immunity have no pathogenic variant identified, limiting access to targeted therapeutics and leaving patients susceptible to severe infection, immune dysregulation and major organ damage. To overcome this, we developed functional screening assays in 5 commonly affected, major immune signalling pathways. Here, we aimed to functionally evaluate rare, novel variants of unknown significance (VUS) affecting the NOD2 signalling pathway.

METHODS: Peripheral monocytes were evaluated by flowcytometry for L18-MDP-induced (NOD2-dependent) TNF-α production, and phosphorylation of p38 (p-p38) and p65 (p-p65). NOD2-independent controls and unstimulated controls were run concurrently.

RESULTS: In healthy donors (n=11), L18-MDP induced TNF-α production in 57.23% (range 22.0-90.02%) of monocytes, and a fold-change of 4.71 (range 2.97-7.62) and 3.18 (range 2.67-10.29) for median fluorescent intensity (MFI) of p-p38 and p-p65. A patient with a hemizygous XIAP variant had complete absence of NOD2-dependent TNF-α, p-p38 and p-p65, whereas a patient with a heterozygous XIAP variant had low TNF-α production (7.32%), but p-p38 and p-p65 increase within, or near, range (4.27 and 2.5, respectively).

CONCLUSION: Here we show assessment of NOD2-dependent TNF-α, p-p38 and p-p65 can identify patients with complete loss-of-function phenotypes, and potential to identify other pathway defects. In future, we will assess more patients with a pathway VUS, and screen patients without a candidate causal variant. This ex vivo functional evaluation of immune pathways, such as NOD2, could provide rapid insights into impact of VUS and into mechanisms of disease, thereby expediting genetic diagnosis and treatment in PAD patients.

Eliza Moore

Department of Neuroscience

Poster

Addressing Drug Resistant Epilepsy: A1R targeting Positive Allosteric Modulators

INTRODUCTION: Anti-seizure medications (ASMs) are the mainstay for epilepsy treatment and act as anti-convulsants by targeting neuronal ion channels or excitatory neurotransmitter receptors to reduce seizure frequency. Despite the availability of numerous ASMs, approximately 30% of patients remain resistant to these treatments. This persistent challenge highlights the urgent need for novel therapeutic strategies that explore alternative anticonvulsive mechanisms and broaden the scope of epilepsy management.

Adenosine 1 Receptor Positive Allosteric Modulators (A1R-PAMs) have emerged as promising novel ASM candidates. This study will systematically screen A1R-PAM candidates in combination with clinically approved ASMs to guide future clinical trials, where patients would continue their existing treatments while introducing these new therapeutic agents.

METHODS: The screening of A1R-PAMs will be conducted in vitro using induced pluripotent stem cell (iPSC)-derived neuronal cultures, wherein 'seizure-like events' will be induced through the application of 4-aminopyridine (4-AP). Functional outcomes will be measured using multi-electrode array (MEA) analysis, allowing for the assessment of neuronal activity patterns. Additionally, isobolographic analysis will be employed to elucidate drug interactions and determine the efficacy of the combinations tested.

CONCLUSION: This research endeavours to provide critical preclinical insights into the potential of A1R-PAMs for effective seizure control. By advancing our understanding of these novel agents, the study aims to inform and refine future treatment strategies for patients with drug-resistant epilepsy, ultimately contributing to improved clinical outcomes and quality of life.

Grace Jin

Department of Neuroscience

Poster

Hidden influences on response latencies within the TUNL task of working memory

INTRODUCTION: The Trial-Unique Nonmatch to Location (TUNL) task is a touchscreen-based working memory (WM) assessment for rodents that can investigate the mechanisms underpinning WM processing when combined with other investigative techniques. However, behavioural nuances observed during the task can impact result interpretation. Here we characterised TUNL response latency variation across different conditions.

METHODS: We trained male C57BL/J mice (n=18) on a 5-window TUNL protocol until training accuracy reached >80% success. Mice were either connected to an electrophysiological tether or left untethered in the testing chamber. Response latency distributions for task Sample and Choice phases were calculated, and compared for correct and incorrect trials across tethering conditions. We included 3662 trials in this analysis.

RESULTS: Both Sample and Choice response latencies were significantly faster for correct trials than incorrect trials (Mann-Whitney U: $p<0.0001$). Latency variances exhibited exponential curves, with curves shifting towards increased latencies for incorrect responses. All conditions also exhibited >10 second responses which did not fit the exponential curve. In tethered sessions the proportion of these responses was strikingly lower for correct responses compared to incorrect responses (Sample: 3.2% vs 13.2%; Choice: 16.6% vs 39.1%), while these differences were modest in untethered sessions (Sample: 1.7% vs 6.9%; Choice: 1.9 vs 7.4%).

CONCLUSION: Response latency is important to consider when interpreting TUNL data, especially while performing tethered measurements. These discrepancies are particularly important when introducing cognition-affecting procedures such as pharmacological treatments.

Holly Fryer

Department of Immunology

Poster Oral

Discovering targets of long-lived humoral immunity for Group A Streptococcus vaccine design

INTRODUCTION: There is an urgent need for a vaccine against Group A Streptococcus (Streptococcus pyogenes, GAS), a bacteria that causes over 600 million pharyngitis cases and 160,000 deaths from invasive infectious disease every year. The current lack of knowledge of immunity in humans is a barrier to vaccine development. We aimed to bridge these knowledge gaps and improve vaccine design and evaluation, we aim to comprehensively map the memory B cell responses specific for 7 GAS candidate vaccine antigens in human tonsils.

METHODS: Using spectral flow cytometry, we have quantified and sorted memory B cells specific for GAS protein and carbohydrate antigens from current vaccine candidates, in tonsils from paediatric donors with and without GAS colonisation. Subsequently, single-cell RNA sequencing is performed to phenotype the antigen-specific memory B cell response in detail. Results: We have quantified tonsil memory B cells specific for all studied GAS antigens. Flow cytometry data revealed different isotypes and activation phenotypes of antigen-specific B cells, and a variation in response magnitude based on GAS colonisation status. We have performed single cell sequencing on GAS antigen-specific memory B cells sorted from these donors, and this dataset enables detailed analysis of the subsets of vaccine antigen-specific B cells as well as their B cell receptor repertoire.

RESULTS: Memory B cells specific for multiple GAS vaccine antigens are present in human secondary lymphoid tissue. Furthermore, we have observed that most antigens can elicit germinal centre responses, which generate high-affinity and long-lasting antibody responses.

CONCLUSIONS: Our study provides critical knowledge about immunity induced by natural infection with GAS, to inform development of vaccines that can reduce the devastating global burden of GAS diseases.

Jack Edwards

Department of Immunology

Poster Oral

Response and resistance to combination immune checkpoint blockade associate with distinct baseline and on-treatment blood T-cell profiles in melanoma patients

INTRODUCTION: Despite the success of immune checkpoint blockade (ICB), most melanoma patients fail to respond or experience severe toxicity. Currently, there are no biomarkers available to predict these events and guide treatment. We evaluated peripheral T cells to identify immune features associated with ICB outcome.

METHODS: Blood samples were collected from 41 advanced melanoma patients at baseline and after one cycle of PD-1 + CTLA-4 ICB. Patients were classified as responders or non-responders based on best overall response to treatment. Absolute immune cell counts were obtained prior to spectral flow-cytometric T-cell immunophenotyping.

RESULTS: 19 patients (46%) failed to respond to treatment. At baseline, non-responders had fewer T cells than healthy controls (median 780 vs. 1297 cells/μL, p=0.00012), mostly due to reduced naive CD4+ (p=0.00203) and CD8+ (p=0.00149) T cells, and showed increased highly immunosuppressive T regulatory cells and expression of proliferation marker Ki67 compared to responders. One ICB cycle expanded memory and regulatory subsets, and responders showed greater Ki67 upregulation in CD4+ central memory (Tcm) (p=0.0086) and regulatory (p=0.0257) T cells compared to non-responders. Compared to Ki67- cells, Ki67+ cells expressed more PD-1 at baseline, expanded to a greater degree on-treatment, and co-expressed higher TIGIT, TIM-3, CD39, and ICOS. The on-treatment fold change of Ki67 expression in CD4+ Tcm cells differentiated responders and non-responders (AUC=0.7545, p=0.0094).

CONCLUSIONS: ICB response was associated with distinct T-cell profiles before and after one cycle of treatment and differentiated responders and non-responders. Further work using combinations of immune features promises to improve this predictive capacity.

Jessica Canning

Department of Immunology

Poster

Dissecting germinal centre B cells in malaria

INTRODUCTION: Malaria remains a global health priority, causing over 600,000 deaths predominantly in children, in 2022. Infection-induced antibodies are slow to develop in previously exposed individuals. Responses to vaccination are poor, with booster doses failing to provide adequate protection. To date, no studies have characterised malaria specific B cells within secondary lymphoid tissues (SLOs) nor following vaccination in children.

METHODS: We used tonsil cells from Ugandan children with prior malaria exposure and/or current asymptomatic infection to investigate the phenotype of malaria specific B cells within the germinal centre (GC) SLOs compared to the periphery. Methods: Samples comprise of tonsils and matched PBMCs from Ugandan children aged 2-11 (n=50, 25% infected at collection) and control malaria-naïve tonsils. Expression of malaria antigens was conducted in Expi293F-BirA cells. Plasmid constructs include a leader sequence, 6x His tag for purification and an AviTag for biotinylation. In vivo biotinylation allowed for site-specific tagging with fluorescent streptavidins to form tetramers. Tetramers are used to identify B cell populations using 25-colour high-dimensional spectral flow cytometry.

RESULTS: We have successfully generated site-specific biotinylated C-terminus CSP, NANP10 repeat region, MSP1-19, AMA-1 proteins for tetramerisation. B cells of particular interest are various malaria-specific MBCs, Ig isotypes and subclasses, atypical B cells, GC plasmablasts and marginal zone B cells.

CONCLUSION: Data will inform our understanding of B cell development and diversity in the GC compared to periphery in human malaria. This knowledge may inform the impact of prior infections on vaccination responses and provide insights into antigen inclusion in future malaria vaccines.

Joshua Law

Clinical Genomics Laboratory

Poster Oral

Detecting Genetic Copy Number Changes in Targeted Sequencing: A Step Forward for Population Health Screening.

INTRODUCTION: DNA Screen is an Australian pilot study of population genomic screening for Hereditary Breast and Ovarian Cancer (HBOC), Lynch Syndrome (LS) and Familial Hypercholesterolemia (FH) involving >10,000 participants, aged 18-40 years. It uses a small panel targeting the coding regions of 9 genes associated with high risk of HBOC (BRCA1, BRCA2, PALB2), LS (MLH1, MSH2, MSH6) and FH (APOB, PCSK9, LDLR). To date, medically actionable variants detected in DNA Screen have consisted of small variants (up to 20bp). Yet, it is estimated that at least 10% of all pathogenic variations across HBOC, LS and FH are copy number variations (CNVs). The analytical validity of the DNA Screen panel (88 kbp total) for the detection of CNVs is still unknown. We aim to explore the possibility of detecting CNVs in the DNA Screen participants and develop a customised framework for CNV interrogation.

METHODS: CNVs were simulated in a series of DNA Screen samples, representing various CN states in different genomic locations. VS-CNV (Golden Helix) was used to detect these simulated CNVs, and the output was used to build 95% confidence intervals to distinguish CNV events from technical noise in the data.

RESULTS: Through the application of our custom framework to the entire DNA Screen dataset, we were able to identify a number of potential CNVs. Assays to experimentally validate and further refine this framework are currently being developed.

CONCLUSION: These preliminary results show that there is potential for CNVs to be identified from DNA Screen data. This is likely to lead to the identification of more at-risk individuals in the population, allowing them to access life-saving early interventions, rather than late-stage treatment.

Judy Choi

Baker Institute

Poster Oral

Selectively targeting the Gasdermin-D pore formation attenuates cardiac inflammation and fibrosis after ischemia reperfusion injury

INTRODUCTION: Inflammation plays a critical role in the clearance of cellular debris to promote tissue repair in cardiac injury pathophysiology. However, inadequately controlled inflammation contributes to adverse cardiac remodelling after acute myocardial infarction (AMI). This is driven by persistent activation of the NLRP3 inflammasome-Gasdermin-D (GSDMD) pathway with subsequent secretion of the inflammatory cytokine IL-1β. Aims: We investigated whether the FDA-approved therapeutic, Disulfiram, used to treat chronic alcoholism but recently shown to inhibit GSDMD, could reduce inflammation and thus improve ischemia/reperfusion (I/R)-mediated cardiac injury.

METHODS: Left coronary artery ligation was performed for 1h in 12-week-old C57BL6 mice, followed by reperfusion with or without 25/50mg of Disulfiram administered at reperfusion and daily until termination. At termination, (day7 and 28) cardiac function was measured by echocardiography, whilst fibrosis and inflammation were assessed by histology and RT-PCR. Flow cytometry assessed leukocyte populations in blood, spleen, bone marrow and heart. Control and Disulfiram-treated mouse BMDMs and human THP-1 cells were investigated for secreted inflammatory cytokines.

RESULTS: Echocardiography showed significant improvements in ejection fraction after 50mg/kg Disulfiram, 7-days post-I/R injury($p<0.01$). Cardiac fibrosis and cardiac inflammatory and fibrosis gene expression was attenuated by Disulfiram D7 and D28 post-AMI (~$p<0.001$). This was associated with reduced inflammatory cell abundance in blood, spleen, bone marrow and heart. In LPS and ATP/Nigericin treated BMDMs and THP-1 cells, Disulfiram attenuated IL-1ß and IL-6 secretion ($p<0.001$).

CONCLUSION: This study demonstrates that Disulfiram reduces inflammation by inhibiting IL-1ß secretion. Therefore, targeting the GSDMD pore may represent a novel way to provide cardio-protection post-AMI.

Meltem Karadeniz

Department of Neuroscience

Poster

Circulatory immune cell counts and clinical outcomes in multiple sclerosis relapse versus remission

INTRODUCTION: Multiple sclerosis (MS) relapse diagnosis is impeded by 'pseudo-relapses' whilst relapse treatment lacks specificity. Before we can improve clinical care, we must first improve our understanding of relapse pathogenesis. Currently, the circulatory immune mechanisms underpinning relapse are poorly understood. We aimed to determine changes in circulatory cell counts from people with MS during relapse versus remission and their association with clinical outcomes.

METHODS: Data was collected retrospectively by screening 2316 patient files through which we identified 97 episodes of MS relapse and remission. From these participants' full blood examination data, we calculated immune cell counts and ratios.

RESULTS: In participants with contrast enhancing lesions on MRI (n=50), total monocyte (p=0.03) and lymphocyte (*p=0.05) counts were significantly lower, and neutrophils/total white cell count (N%) was higher (p=0.02) in relapse versus remission. Monocyte counts were also lower in patients not on any disease-modifying therapies (n=53) during relapse (p=0.02) compared to remission. In participants with a new lesion on MRI (n=65), total neutrophil counts (p=0.02) and N% (p=0.02) were significantly higher during relapse versus remission. Univariable and multivariable regression analyses demonstrated that all three cell types were associated with changes in clinical outcomes.

CONCLUSIONS: This study determined that lymphocyte and monocyte counts are lower whilst neutrophil counts are higher in the circulation during MS relapse compared to remission. All three of these cellular alterations were also associated with clinical outcomes of relapse. These findings support the notion that alterations in circulating immune cells may play a role in MS relapse pathogenesis.

Muhammed Kiyar

Baker Institute

Poster

Dapagliflozin treatment improves cardiac remodelling in a mouse model of heart failure with preserved ejection fraction (HFpEF)

INTRODUCTION: Despite global efforts, HFpEF remains a major issue of modern cardiology and is plagued by a paucity of effective evidence-based therapies. Recently, SGLT2 inhibitors (SGLT2i) have demonstrated robust clinical benefits in HFpEF patients. However, the mechanisms underlying their cardioprotective effects are not well understood and thought to be distinct from their kidney-mediated pathways. This study aims to assess the effects of SGLT2i in a clinically relevant mouse model of HFpEF induced by high-fat diet (HFD) and angiotensin-II (AngII) infusion in aged female mice.

METHODS: 18-month-old Female C57BL/6J mice were randomly assigned to three treatment groups: (i) Normal chow diet (NCD), (ii) HFD+AngII, and (iii) HFD+AngII+Dapagliflozin. AngII infusion began at week 6. DAPA treatment started at week 6 in the HFD+AngII+DAPA group and continued for 6 weeks.

RESULTS: HFD+AngII challenge induced a HFpEF phenotype in aged mice, demonstrating diastolic dysfunction as evidenced by the elevated IVRT and reduced E/A ratio. Concomitantly, the perturbations induced cardiometabolic remodeling including obesity, hypertension, exercise intolerance, inflammation and vascular dysfunction. DAPA treatment did not ameliorate any parameter of the cardiac function. Notably, DAPA treatment attenuated cardiac hypertrophy and overall had mild effects on the cardiometabolic HFpEF phenotype.

CONCLUSION: This study highlighted that DAPA treatment only improved aspects related to cardiac remodeling without a significant impact on cardiac function. The minimal benefits in the cardiometabolic HFpEF phenotype may explain, in part, why SGLT2i treatment did not reduce mortality in HFpEF patients. Further investigation is needed to explore the mechanisms underlying the cardioprotective effects of SGLT2i in HFpEF.

Rhiannon Grant

Department of Immunology

Poster Oral

P-cresol sulfate acts on epithelial cells to reduce allergic airway inflammation

INTRODUCTION: P-cresol sulfate (PCS) is a microbial metabolite derived from L-tyrosine and was recently discovered to have immunoregulatory influences on allergic airway inflammation. Administering PCS to mice reduced house dust mite-induced CCL20 production, a chemokine that recruits lymphocytes and dendritic cells (Wypych et al. Nature Immunology 2021). We are using PCS as a molecular template to develop novel therapeutics against allergic asthma. Our aim is to determine the molecular mechanism of action of PCS and its molecular derivatives in alleviating allergic airway inflammation.

METHODS: We isolated and stimulated primary mouse lung cells with lipopolysaccharide, a strong inducer of CCL20, identifying airway epithelial cells as the main cell type affected by PCS**.**

RESULTS: RNA sequencing of ex vivo mouse lung epithelial cells revealed that PCS influenced heat shock protein 90 (HSP90) gene expression, and indeed, blockade of HSP90 reduced CCL20 production, suggesting it may be involved in the mechanism of action. In silico molecular modelling indicated a shared putative binding site for PCS and two molecular derivatives in the epidermal growth factor receptor (EGFR). Additionally, RNA-sequencing of the A549 human alveolar epithelial cell line identified an increase in genes regulated by the aryl hydrocarbon receptor (AHR) in PCS and molecular derivative treated cells.

CONCLUSIONS: These results implicate EGFR and AHR in mediating the effects of PCS and its molecular derivatives. Overall, PCS acts on lung epithelial cells to reduce CCL20 production and consequently allergic airway inflammation. Elucidation of the molecule's mechanism of action could lead to development of novel therapeutics against allergic asthma and other atopic diseases.

Samin Iranfar

Australian Centre for Blood Diseases (ACBD)

Poster

Lineage-restricted differentiation therapy of acute myeloid leukaemia

INTRODUCTION: Acute myeloid leukaemia (AML) is a haematological malignancy caused by oncogenic mutations that drive aberrant proliferation and block maturation of myeloid progenitors. Differentiation therapies specifically target molecular differentiation blocks in immature AML blasts to induce their maturation and promote subsequent clearance. The rate of relapse in AML patients remains a consistent clinical challenge, highlighting the importance of understanding sources of relapse. Most differentiation therapies rely on irreversible maturation of blasts, however recent studies have demonstrated maturation state plasticity of AML cells whereby persistent mature AML-derived cells can regain leukemogenicity through de-differentiation, seeding relapse. AML cells can undergo multi-lineage differentiation and whilst short-lived lineages such as neutrophils are rapidly cleared, specific persistent sub-lineages de-differentiate and cause relapse

METHODS: To investigate strategies to prevent relapse following differentiation therapy, we utilized a MLL-AF9-driven mouse model of AML. In this model, in vitro or in vivo differentiation therapy triggers lineage bifurcation, producing a short-lived population of AML-derived neutrophils and a persistent population of AML-derived macrophages. Given the known role of haematopoietic cytokine granulocyte-colony stimulating factor (G-CSF) in instructing neutrophil differentiation of normal myeloid progenitors, we aimed to interrogate its instructive potential in restricting AML blast differentiation into short-lived neutrophils.

RESULTS: We have observed that G-CSF administration in vitro can accelerate clearance of mature AML-derived cells. Impeding macrophage differentiation through near-complete restriction into the neutrophil lineage requires prolonged G-CSF administration prior to differentiation therapy use.

CONCLUSIONS: These findings support the use of specific cytokines alongside differentiation therapies to alter multi-lineage differentiation and eradicate mature AML-derived cells to ultimately mitigate relapse.

Sydney Harris

Department of Neuroscience

Poster Oral

The development and characterisation of a mouse model of abusive head trauma

INTRODUCTION: Abusive head trauma (AHT), formerly known as "shaken baby syndrome", is the leading cause of child abuse mortality and morbidity. One-third of children under 3 years who present to the emergency department with a head injury are believed to be victims of abuse. AHTs can lead to enduring behavioural, social, and cognitive deficits that often do not become apparent until years later.
Lack of diagnostic and intervention strategies increases the risk for repetitive AHTs, with reports indicating a single child may experience up to 30 AHTs. Due to a paucity of preclinical models for AHT, particularly repeat AHTs, we aimed to develop and characterise a mouse model of AHT to aid in early diagnosis and prevent repetitive abuse.

METHODS: On postnatal days (P)8-12 mice were subjected to a shaking injury of varied frequencies (1, 3, or 5) or were assigned to a sham group. Mice were euthanised on P14 or P21 for brain tissue analysis; gene expression and immunohistochemistry. Anxiety-like behaviours, nociception, and social interaction were measured in a separate group of mice.

RESULTS: AHTs produced brain swelling 48 hours post-injury and gene expression alterations in the hippocampus and prefrontal cortex at 48 hours and one-week post-AHT. Behavioural deficits, including increased anxiety, as well as impaired thermal nociception and social interactions, were identified in a sex-dependent manner.

CONCLUSIONS: This novel model of AHT demonstrates that gene expression changes in the brain can be detected before behavioural problems arise, suggesting early brain pathology might enable timely diagnosis and intervention.

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