33rd Combined Health Science Conference

New Horizons 2016
Innovative science with impact:
Strengthening alliances between research

Dr Chau Chak Wing Building, University of Technology Sydney
21st – 22nd November 2016

FINAL PROGRAM & ABSTRACT BOOK

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www.newhorizons2016.org
ACKNOWLEDGEMENTS

The New Horizons 2016 Program Committee would like to thank the following who, at the time of printing, had given their support to this conference:

INSTITUTIONS

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EXHIBITORS & OTHER SPONSORS (in alphabetical order)

UTS:HEALTH  VWR  We Enable Science
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On behalf of the Organising Committee and all co-hosting organisations I would like to extend a very warm welcome to delegates, plenary speakers, presenters and invited guests of New Horizons 2016. This is the 33rd Combined Health Science Conference co-hosted by the University of Technology Sydney, the Kolling Institute, The University of Sydney and Northern Sydney Local Health District.

These annual conferences promote advancements in healthcare through research and education, and by building bridges between basic science, the clinic and delivery of care. There is a rich history of collaboration between the institutes involved which, over the past 33 years, has resulted in significant progress in all of these areas.

This year our conference will focus on research with impact - research that increases our understanding of disease and leads to the development of new therapies and better outcomes for patients. It will include presentations on the latest scientific research, new developments in technology, clinical excellence and the translation of research from bench to bedside.

New Horizons 2016 will be held in the Frank Gehry designed Dr Chau Chak Wing Building at the University of Technology, Sydney which was also the venue for the 2015 meeting. The building is a symbol of creativity and innovation, providing an excellent space for interaction and discussion. We are very grateful to our plenary speakers, Professor Nikolai Petrovsky, Professor Tania Sorrel, Professor Phil Hansbro, Professor Johanna Westbrook and Professor Iain McGregor, for agreeing to share their latest research with us. We also look forward to presentations and posters from health care providers, research assistants, honours students, PhD students, masters students, post-doctoral researchers as well as research leaders.

We acknowledge and sincerely thank all of those who have committed time and effort into the organising of this conference, as well as our academic and health sponsors who have made the meeting possible.

We hope that you find this meeting productive and informative, and take the opportunity to form new cross-institutional and inter-disciplinary collaborative and personal relationships. We have tried to create a relaxed and friendly atmosphere that allows students and younger researchers to feel comfortable presenting their research and interacting with more experienced researchers.

Please join us for drinks and canapés in Bar 80 on level 2 after the meeting - take the opportunity to interact with the speakers and other delegates and enjoy being part of a vibrant research community.

A/Professor Ken Rodgers, Organising Committee Chairman, 2016
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<td>0900 – 1030</td>
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<td>Collaborative theatre</td>
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<td>Morning tea, posters, exhibitions</td>
<td>Morning tea, posters, exhibitions</td>
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<tr>
<td>1100</td>
<td>Plenary 2: Fungi in the mix – improving health outcomes in an era of increasing drug resistance</td>
<td>Plenary 4: Microbiomes and respiratory diseases</td>
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<td>1130</td>
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<td>1100 – 1200</td>
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<td>1200</td>
<td>Abstract 1: Rapid Fire Prize Session</td>
<td>Merck/Sigma Sponsored presentation: CRISPR/Cas9 technology</td>
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<td>1230</td>
<td>Lunch, poster, exhibitions</td>
<td>Lunch, poster, exhibitions</td>
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<td>1245 – 1400</td>
<td>1230 – 1330</td>
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<td>1400</td>
<td>Plenary 3: Innovative approaches to measuring large-scale health system interventions: The impact of digital health on hospital work and patient outcomes</td>
<td>Plenary 4: The therapeutic potential of medicinal cannabis and plant-derived cannabinoids</td>
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<td>Afternoon tea, posters, exhibitions</td>
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<td>Wrap up and Prizes</td>
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Bar 80, Level 2
The Organising Committee of the New Horizons 2016 Conference includes (in alphabetical order):

- A/Prof Beata Bajorek, Graduate School of Health, UTS and Academic Pharmacist, Royal North Shore Hospital, NSLHD

- Dr Hui Chen, Senior Lecturer, School of Life Sciences, UTS

- Dr Emily Colvin, Post-doctoral Research Fellow, Bill Walsh Cancer Research, Kolling Institute

- Dr Deborah Debono, Lecturer, Faculty of Health, UTS

- A/Prof Sheila Donnelly, School of Life Sciences, UTS

- Dr Amanda Hudson, Post-doctoral Fellow, Sydney Neuro-Oncology Group, Kolling Institute

- Dr Kristine McGrath, Lecturer, School of Life Sciences, UTS

- Dr Leigh Monahan, Post-doctoral Fellow, The iThree Institute, UTS

- A/Prof Ken Rodgers, School of Life Sciences, UTS

- Dr Sonia Saad, Senior Research Fellow, Kolling Institute and School of Life Sciences, UTS

- A/Prof Stella Valenzuela, Centre for Health Technologies, School of Life Sciences, UTS
Plenary Speakers of the New Horizons 2016 Conference (in alphabetical order):

Prof Phil Hansbro, *Hunter Medical Research Institute, University of Newcastle*
Phil Hansbro is chair in immunology and microbiology, and an NHMRC Principal Research Fellow at the Hunter Medical Research Institute and University of Newcastle, Australia. He is Associate Director of the Priority Research Centre for Lung Health there. He has established research programs in COPD, asthma and infection. His group has developed several novel mouse models of the important diseases (COPD, severe, steroid-insensitive asthma, early life infection & lung cancer). He has interrogated them (immune, histological, pathological, lung function & molecular analysis) to further our understanding of pathogenesis and develop novel therapies. He performs complimentary collaborative clinical and multi-disciplinary studies and collaborates widely. He publishes extensively (~150 pubs) in influential journals and is regularly invited to present internationally including as plenary speaker and to chair sessions. He has a substantial funding record of obtaining nationally competitive grant that support his group. He undertakes considerable mentoring and supervision activities of junior researchers, regularly sits on grant review panels and is on the editorial board of three journals. He is an active advocate for respiratory research in lobby groups and is regularly in the press promoting research and funding.

Prof Iain McGregor, *School of Psychology, University of Sydney*
Iain McGregor is Professor of Psychopharmacology, NHMRC Principal Research Fellow and Director of the Psychopharmacology Laboratory at the University of Sydney. Iain's research focuses on the effects of recreational drugs and prescription drugs on brain and behaviour and on the development of new medications for the treatment of various diseases. His research spans medicinal chemistry, the use of cellular assays and preclinical animal models of disease, and also clinical trials in humans. Major recent areas of research interest include the beneficial effects of oxytocin on social behaviour, the development of novel treatments for addiction-related and mental health problems, and analysis of the increasing use of prescription psychotropic drugs in Australia and other countries. Iain has a strong interest in the area of medicinal cannabis and cannabinoids and is Director of Preclinical Research at the Lambert Initiative for Cannabinoid Therapeutics at the University of Sydney. The Lambert Initiative was established in 2015 with a $33.7 million philanthropic gift and aims to fast track research into the therapeutic benefits of cannabinoids in various disease states.

Prof Nikolai Petrovsky, *Clinical Endocrinologist, Flinders University*
Nikolai is a Clinical Endocrinologist at Flinders Medical Centre, Professor of Medicine at Flinders University and the founder of Vaxine Pty Ltd, a company engaged in vaccine research and development. He has been principal investigator on multiple large research grants from the U.S. National Institutes of Health and conducts diverse research covering immuno-informatics, adjuvants, vaccines, diabetes and autoimmunity. He holds multiple patents for discoveries across these fields and his work has been honoured with awards including the 2010 Ernst & Young Entrepreneur of the Year Award, 2011 Vaccine Industry Excellence Award for Most Innovative Asian Biotech and 2013 Biopharma Asia Asian Executive of the Year Award. He has authored over 140 research papers and has taken multiple novel vaccines including for influenza, hepatitis B and allergy, all the way from conception at the laboratory bench to human clinical trials.
Prof Tania Sorell, Director of the Marie Bashir Institute for Infectious Diseases and Biosecurity, Deputy Dean University of Sydney

Tania Sorell is Professor of Clinical Infectious Diseases, Director of the Marie Bashir Institute for Infectious Diseases and Biosecurity and Deputy Dean, Sydney Medical School, The University of Sydney, Australia; Director of the Centre for Infectious Diseases and Microbiology, Westmead Institute for Medical Research, Westmead, NSW; and Service Director, Infectious Diseases and Sexual Health, Western Sydney Local Health District. She has had a longstanding clinical interest in mycology and infections in the immunocompromised host and a more recent interest in emerging infectious diseases. Her research has focused on the pathogenesis of fungal infections, emerging fungal diseases, new antifungal drug development, new diagnostics and clinical trials of antifungal diagnostic and treatment strategies. She has served on state and national advisory committees in Infectious Diseases and therapeutics, and on the Research and Human Ethics Committees of NHMRC.

Prof Johanna Westbrook, Director of the Centre for Health Systems and Safety Research, Australian Institute of Health Innovation, Macquarie University

Professor Johanna Westbrook is internationally recognised for her research evaluating the effects of information and communication technology (ICT) in health care. She has over 390 publications and been awarded > $40M in research grants. Johanna has led important research in the development and application of approaches to evaluate ICT, including new tools and methods which have been adopted internationally. She has contributed to theoretical models regarding the design of complex multi-method ICT evaluations. Her research has led to significant advances in our understanding of how clinical information systems deliver (or fail to deliver) expected benefits and has supported translation of this evidence into policy, practice, and IT system changes. Johanna is currently leading research investigating the role and impact of ICT in the community and aged care sector. Johanna was elected as a Fellow of the American College of Medical Informatics (ACMI) in 2005, and is one of only three Australians to receive this honour. In 2014 Johanna was named Australian ICT professional of the year by the Australian Information Industry Association for her research contributions. In 2015 she was appointed Associate Editor of the Journal of the American Medical Informatics Association (JAMIA). In 2016 she was appointed to the Board of the Australian Digital Health Agency.
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### FULL PROGRAM – Day 1 – Monday 21st November 2016

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<td>0800 – 1700</td>
<td>Registration Desk Open</td>
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<tr>
<td>0900 – 1030</td>
<td><strong>WELCOME AND OPENING PLENARY</strong></td>
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<td>Session Chair: Associate Professor Ken Rodgers</td>
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<td>Location: Collaborative Theatre</td>
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<tr>
<td>0900 - 0915</td>
<td>Welcome to Country</td>
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<td>Aunty Joan Tranter, UTS Indigenous Leader</td>
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<tr>
<td>0915 - 0930</td>
<td>Welcome and Introduction to the Conference</td>
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<td><strong>Professor Judith Smith, Dean of Science, UTS</strong></td>
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<td>0930 – 1030</td>
<td><strong>OPENING PLENARY: Professor Nikolai Petrovsky</strong></td>
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<td></td>
<td><strong>Title:</strong> Alzheimer's disease vaccine and other unorthodox vaccines</td>
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<td>Session Chair: Prof. Rob Baxter</td>
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<td>Location: Collaborative Theatre</td>
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<tr>
<td>1030 – 1100</td>
<td>Morning tea, poster presentations and sponsored table displays</td>
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<tr>
<td>1100 – 1200</td>
<td><strong>PLENARY SESSION 2: Professor Tania Sorrell</strong></td>
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<td><strong>Title:</strong> Fungi in the mix – improving health outcomes in an era of increasing drug resistance</td>
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<td>Session Chair: Dr Valery Combes</td>
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<td>1200 – 1245</td>
<td><strong>ABSTRACT SESSION 1: RAPID FIRE PRIZE SESSION</strong></td>
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<td>Session Co-chairs: Associate Professor Sheila Donnelly and Dr Deborah Debono</td>
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<td>Location: Collaborative Theatre</td>
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<td>1200 – 1203</td>
<td>Self-medication with anti-hypertensive medicines in Indonesia: patients’ perspectives</td>
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<td>Riana Rahmawati, Graduate School of Health (Pharmacy), University of Technology Sydney, NSW 2007, Australia</td>
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<td>1204 – 1207</td>
<td>Identification of patients with diabetes mellitus that benefit most from a chronic disease management health coaching program</td>
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<td>Rachel T. McGrath, Department of Diabetes, Endocrinology &amp; Metabolism, Royal North Shore Hospital, St Leonards, Sydney, NSW 2065, Australia</td>
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<td>1208 – 1211</td>
<td>Retrospective analysis of a canine study assessing the safety of a novel tumour vaccine process (2010 – 2016)</td>
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<td>Chris Weir, Bill Walsh Translational Cancer Research Laboratory, Kolling Institute of Medical Research, Royal North Shore Hospital and the Sydney Medical School, University of Sydney, Sydney Australia</td>
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<tr>
<td>1212 – 1215</td>
<td>Understanding mechanisms of IDH-mutant glioma progression and chemoresistance through use of latest-generation mass spectrometry</td>
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<td></td>
<td>Angela Cho, Bill Walsh Translational Cancer Research Laboratory, Hormones and Cancer, Kolling Institute of Medical Research, Royal North Shore Hospital, St Leonards, Sydney, Australia</td>
</tr>
<tr>
<td>1216 – 1219</td>
<td>N-of-1 trials for assessing the effects of deprescribing medications on short-term clinical outcomes in older adults: a systematic review</td>
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<td>Alexander J. Clough, Faculty of Pharmacy, University of Sydney, Camperdown, Sydney, NSW</td>
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<tr>
<td>1220 – 1223</td>
<td>RET-related microRNAs, miR-1277-5p and miR-153-3p, as novel therapeutic agents in medullary thyroid carcinoma</td>
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<td></td>
<td>Lauren Joob, Cancer Genetics Laboratory, Kolling Institute of Medical Research, Royal North Shore Hospital, St Leonards, NSW, Australia</td>
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<td>1224 – 1227</td>
<td>The roles of SIRT1 in maternal obesity induced metabolic disorders in the offspring</td>
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<td>Long Nguyen, Renal Research Group, Kolling Institute, Royal North Shore Hospital, University of Sydney, NSW 2065, Australia</td>
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<td>1228 – 1231</td>
<td>Differentiating bacteria associated with cystic fibrosis lung infections</td>
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<td>1232 – 1235</td>
<td>FXYD1 is an endogenous protector against oxidative stress induced vascular dysfunction by Thomas Hansen.</td>
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<td>1236 – 1239</td>
<td>Insulin-like growth factor receptor and sphingosine kinase co-expression has therapeutic potential in breast cancer by Aleksandra M. Ochniak.</td>
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<td>1240 – 1243</td>
<td>Heterogeneity of thawed and cored frozen blood aliquots by Kathleen M. Phillips.</td>
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**1245 – 1400**
Lunch, poster presentations and sponsored table displays

**1400 – 1500 hours**
**PLENARY SESSION 3: Professor Johanna Westbrook**
*Title: Innovative approaches to measuring large-scale health system interventions: The impact of digital health on hospital work and patient outcomes*
*Session Chair: Professor Michael Wallach*
*Location: Collaborative Theatre*

**1500 – 1530**
Afternoon tea, poster presentations and sponsored table displays

**1530 – 1700 hours**
**ABSTRACT SESSION 2: POST-DOCTORAL SESSION**
*Session Chair: Associate Professor Beata Bajorek*
*Location: Collaborative Theatre*
*Short listed for Early Career Researcher Award*

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<tr>
<td>1530 – 1545</td>
<td>Functional prediction of long non-coding RNAs in ovarian cancer-associated fibroblasts indicate a role in metastasis by Emily K. Colvin.*</td>
</tr>
<tr>
<td>1545 – 1600</td>
<td>Utilising metabolomics to identify predictive biomarkers for pregnancy complications by Katie L. Powell.*</td>
</tr>
<tr>
<td>1600 – 1615</td>
<td>Assessment of variables affecting lung protein in exhaled breath condensate by Sarah A. Hayes.*</td>
</tr>
<tr>
<td>1615 – 1630</td>
<td>Insulin pump therapy in pregnant type 1 diabetic women is not associated with improved perinatal outcomes by Rachel T. McGrath.</td>
</tr>
<tr>
<td>1630 – 1645</td>
<td>Awake versus sleep prediction methods for selecting Obstructive Sleep Apnoea patients for oral appliance therapy by Kate Sutherland.*</td>
</tr>
<tr>
<td>1645 – 1700</td>
<td>Development of an international geriatric pharmacology curriculum for medical schools: systematic review and curriculum mapping by Mandavi Kashyap.</td>
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**FULL PROGRAM – Day 2 – Tuesday 22\(^{nd}\) November 2016**

0800 – 1700  Registration Desk Open

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<td>0900 – 1030 hours</td>
<td><strong>ABSTRACT SESSION 3: YOUNG INVESTIGATORS PRIZE SESSION</strong></td>
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<td>Session Chair: Dr Leigh Monahan</td>
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<td>Location: Collaborative Theatre</td>
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<tr>
<td>0900 – 0915</td>
<td>Vascular TRAIL expression is increased in endothelial cells by Angiotensin II</td>
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<td><strong>Pradeep CHOLAN</strong></td>
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<td>Heart Research Institute, Sydney</td>
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<td>0915 – 0930</td>
<td>Nurses are underutilised in Antimicrobial Stewardship - results of a multisite survey in paediatric and adult centres</td>
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<td>Graduate School of Health, University of Technology Sydney, Sydney, Australia</td>
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<td>0930 – 0945</td>
<td>Intra-operative amylase in peri-pancreatic fluid predicts pancreatic fistula after distal pancreatectomy</td>
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<td><strong>Christopher B. NAHM</strong></td>
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<td></td>
<td>Department of Gastrointestinal Surgery, Royal North Shore Hospital, St. Leonards NSW, Australia</td>
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<tr>
<td>0945 – 1000</td>
<td>Uncovering the missing genetic component of familial amyotrophic lateral sclerosis using bioinformatic approaches</td>
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<td><strong>Emily McCANN</strong></td>
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<td>Department of Biomedical Sciences, Faculty of Medicine and Health Sciences, Macquarie University, Sydney, New South Wales, Australia</td>
</tr>
<tr>
<td>1000 – 1015</td>
<td>MicroRNAs: Novel diagnostic biomarkers for diabetic nephropathy</td>
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<td><strong>Qinghua CAO</strong></td>
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<td>Kolling Institute of Medical Research, Royal North Shore Hospital, Sydney Medical School, University of Sydney, Sydney, New South Wales, Australia</td>
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<tr>
<td>1015 – 1030</td>
<td>Multiple myeloma causes T cell immunosenescence in tumour induced clones: Implications for immunotherapy</td>
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<td><strong>Hayley SUEN</strong></td>
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<td>Institute of Haematology, Royal Prince Alfred Hospital, Camperdown NSW Australia</td>
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<tr>
<td>1030 – 1100</td>
<td>Morning tea, poster presentations and sponsored table displays</td>
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<tr>
<td>1100 – 1200 hours</td>
<td><strong>PLENARY SESSION 4: Professor Phil Hansbro</strong></td>
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<td>Title: Microbiomes and respiratory diseases</td>
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<td>Session Chair: Professor Elizabeth Harry</td>
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<td>Location: Collaborative Theatre</td>
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<td>1200 – 1215 hours</td>
<td><strong>MERCK SPONSORED PRESENTATION: CRISPR/Cas9 technology</strong></td>
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<td>Dr Fabien Delerue; CRISPR/Cas9 genome editing in mice: streamlining the process for fast generation of disease models</td>
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<td>Mr Oliver Distler; Technologies to model and correct disease in cells using CRISPR: Towards gene therapy approaches</td>
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<td>Session Chair: Dr Emily Colvin</td>
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<tr>
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<td>Location: Collaborative Theatre</td>
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<tr>
<td>1215 – 1330</td>
<td>Lunch, poster presentations and sponsored table displays</td>
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<td>1330 – 1430 hours</td>
<td><strong>PLENARY SESSION 5: Professor Iain McGregor</strong></td>
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<td>Title: The therapeutic potential of medicinal cannabis and plant-derived cannabinoids</td>
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<td>Session Chair: Professor Andrew Hayen</td>
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<td>1430 – 1500</td>
<td>Afternoon tea, poster presentations and sponsored table displays</td>
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<tr>
<td>1500 – 1700 hours</td>
<td>**New Horizons 2016</td>
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# ABSTRACT SESSION 4

Session Chair: Dr Kristine McGrath  
Location: Collaborative Theatre

*Short listed for award honours award*  
*Short listed for Josephine Anderson oral award*  

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<th>Time</th>
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<tr>
<td>1500 – 1510</td>
<td>Biomarker discovery by MALDI-MS imaging in triple-negative breast cancer</td>
<td>Robert C. BAXTER, Kolling Institute, University of Sydney, Royal North Shore Hospital, St Leonards NSW 2065</td>
</tr>
<tr>
<td>1510 – 1520</td>
<td>Bacterial Communities Vary between Sinuses in Chronic Rhinosinusitis Patients</td>
<td>Catherine M. BURKE, Faculty of Science, The iThree Institute, University of Technology, Sydney, NSW, Australia</td>
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<tr>
<td>1520 – 1530</td>
<td>Impact of Novel Drugs in Rats with Mild Traumatic Brain Injury (mTBI)</td>
<td>Arjun SAPKOTA*, School of Life Sciences, Faculty of Science, University of Technology, Sydney</td>
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<td>1530 – 1540</td>
<td>Public perceptions and attitudes towards antibiotic resistance in Sydney, Australia</td>
<td>Annie ZHUO*, Asia – Pacific Natural Hazards and Disaster Risk Research Group, School of Geosciences, The University of Sydney, Sydney, NSW 2006</td>
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<tr>
<td>1540 – 1550</td>
<td>Transcriptional Profiling of Macrophages Predicts a Therapeutic Application for a Parasite-Derived Peptide</td>
<td>Akane TANAKA, The School of Life Sciences, University of Technology Sydney, New South Wales, Australia</td>
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<td>1550 – 1600</td>
<td>Measuring frailty in CHF: validation of a question only approach</td>
<td>Lily MARTIN*, Centre for Cardiovascular and Chronic Care, Faculty Health, University of Technology Sydney</td>
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<td>1600 – 1610</td>
<td>The protective role of trail in NAFLD</td>
<td>Scott GENNER*, Heart Research Institute, Sydney</td>
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<td>1610 – 1620</td>
<td>The tumour suppressive roles of microRNA-497 in Adrenocortical Carcinoma</td>
<td>Nunki HASSAN, Cancer Genetics Laboratory, Kolling Institute, Northern Sydney Local Health District, St Leonards, NSW, Australia</td>
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<td>1620 – 1630</td>
<td>The potential of nanomedicine: non-toxic, stable nanoparticles for enhanced cancer therapy</td>
<td>Samuel YUEN*, Bill Walsh Translational Cancer Research Laboratory, Kolling Institute of Medical Research, NSW 2065, Australia</td>
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<tr>
<td>1630 – 1640</td>
<td>RIPK1: the next novel target for renal fibrosis</td>
<td>Ying SHI, Kolling Institute, University of Sydney, Royal North Shore Hospital</td>
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<td>1640 – 1650</td>
<td>Sentinel surveillance of Plasmodium falciparum Kelch propeller for the detection of artemisinin resistance</td>
<td>Christiane PROSSER*, School of Medical and Molecular Biosciences, University of Technology Sydney, NSW, Australia</td>
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<tr>
<td>1650 – 1700</td>
<td>Modulating the innate immune system in rats following spinal cord injury using a novel molecule derived from a helminth parasite</td>
<td>Alison RICAFRENTE*, The School of Life Sciences, University of Technology Sydney, New South Wales, Australia</td>
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**1700 – 1715 hours**  
CONFEERENCE WRAP UP AND PRESENTATION OF AWARDS  
Session Chair: Associate Professor Ken Rodgers  
Location: Collaborative Theatre

**1715 – 1800 hours**  
CLOSING DRINKS  
Location: Bar 80, Level 2
Speaker abstracts are provided in presentation order (presenting author underlined):

**MONDAY 21ST NOVEMBER 2016**

**ABSTRACT SESSION 1: RAPID FIRE PRIZE SESSION**

**Self-medication with anti-hypertensive medicines in Indonesia: patients’ perspectives**
RAHMAWATI R1,2, Bajorek BV1,2

1Graduate School of Health (Pharmacy), University of Technology Sydney, NSW 2007, Australia 2Department of Pharmacy, Royal North Shore Hospital, Sydney, NSW, Australia

**Background:** Despite their status as prescription-only medicine, anti-hypertensive medicines can be purchased without prescription in Indonesia.

**Aims:** This study reports the period prevalence, type of medicines purchased, patients’ reasons for self-medicating and patients’ self-reporting regarding information gathered by community pharmacist.

**Methods:** This study was conducted from August to November 2015 in eight rural villages in the Bantul district, Yogyakarta province, Indonesia. It included villagers diagnosed with hypertension aged ≥45 years. A researcher-administered questionnaire was used to collect information from the participants.

**Results:** In total, 384 participants were included in this study. Of these, 64 (18%) self-reportedly purchased anti-hypertensive medicines in community pharmacies (CP) without prescription within 30 days prior to conducting the study. Thirty-nine participants (61%) did not personally attend a CP, but instead a family member purchased the medicines on their behalf. Captopril was the most commonly purchased (73%). Obtaining an additional supply (prescription refill) of anti-hypertensive medicines was the most frequently reported reason for purchasing without prescription. Five participants claimed to have disclosed this practice to their clinicians and obtained their approval. The participants preferred to obtain medicines from a CP because of their accessibility, flexible opening hours, short waiting time and perceived better quality of medicines compared to those from public healthcare services. Avoidance of doctors’ consultation fees was mentioned as another reason for bypassing appointments with medical practitioners and purchasing anti-hypertensives over the counter. Twenty-six participants stated that CP staff asked them who they were purchasing the medicine for, while 10 reported that anti-hypertensive medications were dispensed without any questions.

**Conclusion:** Patients who lived in rural villages acknowledged the benefits of self-medicating with anti-hypertensives. Nevertheless, improving the role of CP staff in assisting patients to obtain adequate medication and information is mandatory for improving the appropriate use of medicines.

**Identification Of Patients With Diabetes Mellitus That Benefit Most From A Chronic Disease Management Health Coaching Program**
Rachel T. MCGRAITH1,2,3, Grace Delaney1, Neroli Newlyn1, Elline Pamplona1, Samantha L. Hocking1,2,4, Sarah J. Glastras1,3 and Gregory R. Fulcher1,2

1Department of Diabetes, Endocrinology & Metabolism, Royal North Shore Hospital, St Leonards, Sydney, NSW 2065, Australia 2University of Sydney, Northern Clinical School, Royal North Shore Hospital, St Leonards, NSW 2065, Australia 3Kolling Institute of Medical Research, University of Sydney, Royal North Shore Hospital, St Leonards, NSW 2065, Australia 4Charles Perkins Centre, University of Sydney, Australia

**Background:** Diabetes mellitus is a chronic disease associated with significant morbidity, premature mortality and psychological distress. Chronic disease management programs (CDMP) that include health coaching are used to facilitate and coordinate diabetes management. Further, health coaching programs tailored towards diabetes self-management have been identified as effective ways to engage patients in management decisions and to improve health outcomes.

**Aims:** The aim of this study was to assess the change in general knowledge of diabetes (DK), self-reported health status (HS), diabetes distress (DD), BMI and glycaemic control following enrolment in a face-to-face CDMP health coaching session (with telephone follow-up) or participation in telephone CDMP health coaching alone over a 12-month period.

**Methods:** Patients with diabetes were enrolled into a health coaching program at Royal North Shore Hospital (RNSH), Sydney, Australia. Questionnaires were administered at baseline, 3, 6 and 12 months and the results were compared to baseline. Glycaemic control (HbA1c) and BMI were measured at baseline and 12 months.

**Results:** Overall, 283 patients attended a face-to-face CDMP health coaching session followed by telephone health coaching (n = 178) or participated in telephone health coaching alone (n = 60). There was no change in BMI over 12 months; however patients with HbA1c levels above target at baseline demonstrated a significant improvement. Patients with the lowest self-reported HS at baseline showed an improvement. DK improved in all patients and DD was reduced for those with the highest levels of distress at baseline.

**Conclusion:** We propose that diabetes health coaching programs could be best utilised by targeting specific patient groups based on their baseline characteristics, specifically patients with the highest level of DD and/or poor glycaemic control.
Retrospective analysis of a canine study assessing the safety of a novel tumour vaccine process (2010 – 2016)

Chris WEIR1, Angus Ross2, Miles Alexander2, Veronika Langova2, Peter Bennett3, Peter Britton4, Mathew McClennan5, Kathryn Fernside5, Richard Mullins6, Rachel Allavena3, Ross Davey1 and Viive M. Howell1

1Bill Walsh Translational Cancer Research Laboratory, Kolling Institute of Medical Research, Royal North Shore Hospital and the Sydney Medical School, University of Sydney, Sydney Australia; 2Ku-ring-gai Veterinary Hospital, Sydney, Australia; 2Small Animal Specialist Hospital, Sydney, Australia; 4Sydney University Veterinary School, Sydney, Australia; 3St George Veterinary Hospital, Sydney, Australia; 5Normanbury Vet Clinic, Sydney, Australia; 7Lane Cove Veterinary Hospital Sydney, Australia; 8School of Veterinary Sciences, University of Queensland, Gatton Queensland

Canine malignancy rates are similar to those of humans; however canine cancer patients have limited therapeutic options. Inducing a patient’s own immune system to elicit an anti-tumour response is an attractive approach to cancer therapy. In this safety study, autologous, allogeneic and xenogeneic tumour vaccines produced by a unique method developed in our laboratory were used to treat canine cancer patients.

Dogs enrolled in this study required a diagnosis of cancer, the ability to obtain fresh tumour and informed consent from the owner. Tumour tissue was frozen, transported to the laboratory and stored until required for vaccine production. Dogs received a minimum of 2 subcutaneous doses of vaccine. The first 2 doses were 3 weeks apart and often given concurrently with chemotherapy, prednisone and other medications.

Ninety two dogs representing 30 different breeds, presented with 17 different tumour pathologies. In total, 79 (86%) dogs received the vaccine course. The remainder were excluded generally due to very late stage presentation and death prior to receiving the vaccine. Two minor adverse events were observed in the 290 doses delivered. Approximately 45% of dogs treated with vaccine alone had longer than predicted survival. When the vaccine was combined with chemotherapy or steroid treatment a benefit over that predicted by standard of care alone was observed in 75% of dogs. Eighteen dogs (22.7%) derived no benefit from being vaccinated. The highest response rate (91%) was seen in dogs that had their tumours resected, compared with debulking (68%) and biopsy (53%). However, no significant difference in survival time was seen in responders in these groups.

All versions of the vaccine were safe, well tolerated and demonstrated greatest efficacy as an adjunct therapy warranting further controlled studies.

Understanding mechanisms of IDH-mutant glioma progression and chemoresistance through use of latest-generation mass spectrometry

CHO, Angela1,2, Hayes, Sarah A.1,2, Hudson, Amanda L.1,2, Colvin, Emily K.1,2, Molloy, Mark P.3, Wheeler, Helen R.1,2, Howell, Viive M.1,2

1Bill Walsh Translational Cancer Research Laboratory, Kolling Institute of Medical Research, Royal North Shore Hospital, St Leonards, Sydney, Australia
2Sydney Medical School, Northern Clinical School, University of Sydney, Sydney, Australia
3Australian Proteome Analysis Facility, Macquarie University, North Ryde, Sydney, Australia

Gliomas are the most common primary brain tumours in adults. Mutations in Isocitrate Dehydrogenase 1 and 2 (IDH1/2) define a glioma subtype that includes both low and high grade gliomas. Although IDH- mutated low grade gliomas have a relatively indolent clinical course, recurrence is inevitable and progression to a higher grade after treatment is common. The standard treatment for glioma patients following surgery includes radiation and chemotherapy. This regimen was introduced over a decade ago and is not curative. At recurrence, the molecular profile of the tumour may have changed with significant therapeutic consequences including the acquisition of chemoresistance.

To determine the proteomic profiles and differences between primary and recurrent IDH-mutated tumours, protein was extracted from six matched primary and recurrent IDH-mutated tumours and analysed by SWATH-MS (on a Sciex TripleToF 6600). Overall, 1781 proteins were identified, with 53 proteins being significantly differentially expressed (FC ≥1.5-fold; p ≤ 0.05) between primary and recurrent specimens from patients who had not received therapy. In patients who had received radiotherapy, 39 proteins were significantly differentially expressed between primary and recurrent tumour. In both datasets, these key proteins were associated with activation of the Acute Phase Response, Cell Death and Survival and Cellular Compromise, Development and Movement. Proteins significantly differentially expressed included HNRPL and MT2A, which are involved in tumour angiogenesis, development and chemoresistance resistance.

The identification of differentially expressed proteins will improve our understanding of the mechanisms driving recurrence and progression of gliomas and may also reveal potential novel targets for additional therapies for this deadly cancer.

N-of-1 trials for assessing the effects of deprescribing medications on short-term clinical outcomes in older adults: a systematic review

Alexander J CLOUGH1,2, Sarah N Hilmer2,3, Sharon L Naismith4,5, Luke Kardell1,2, Danijela Gnijdic1,2,5

Faculty of Pharmacy, University of Sydney1, Camperdown, Sydney, NSW
Kolling Institute of Medical Research and Sydney Medical School, University of Sydney, St Leonards, Sydney, NSW2
Department of Clinical Pharmacology and Aged Care, Royal North Shore Hospital, St Leonards, Sydney, NSW3
Brain & Mind Centre, University of Sydney, Camperdown, Sydney, NSW4
Charles Perkins Centre, University of Sydney, Camperdown, Sydney, NSW5

Introduction: Deprescribing research, the investigation of the effects of supervised discontinuation of treatments, is a growing field. Most studies have been randomised controlled trials (RCTs), however methods more applicable to clinical practice providing rigorous data on causation and reversibility have been recommended. The N-of-1 methodology may allow this and provide evidence on individual responses to medications – and inform patient-centred care.
Aims: To determine the feasibility of using the N-of-1 method for deprescribing trials in older adults.

Methods: A search was conducted from inception through to the 26th of September 2016 in Embase, PubMed, Informit, Scopus, International Pharmaceutical Abstracts, PsychINFO, Cochrane Central Register of Controlled Trials (CCCTR) and CINAHL for studies conducted in older adults (≥ 50 years), deprescribing any long-term treatment conducted over less than a year using the N-of-1 method. Two authors independently reviewed all articles for eligibility and extracted data. The review was conducted according to PRISMA guidelines. Quality assessment of trials was carried out using the PEDro scale.

Results: Six studies of deprescribing treatments using the N-of-1 method in older adults were found. These trials all investigated the efficacy of treatments for treating diseases including cardiovascular disease, asthma, chronic airflow limitation and skeletal muscle cramps. Four trials resulted in a significant number of patients (44-64%) discontinuing their medication due to non-significant treatment benefit. Two studies determined that the respective treatment was effective, and the majority of patients continued their treatment.

Discussion: The ability of the N-of-1 method to effectively determine the individual efficacy of treatments was powerful, resulting in strong patient-specific outcomes impacting on care of adults. However, use of the N-of-1 method has rarely been reported in deprescribing trials, although it has been used in other fields.

RET-related microRNAs, miR-1277-5p and miR-153-3p, as novel therapeutic agents in medullary thyroid carcinoma

Lauren Jin Suk JOO1, Justin S. Gundara1, Anthony R. Glover1, Anthony J. Gill2, Matti L. Gild1, Bruce G. Robinson1, Stan B. Sidhu1,4 and Jing Ting Zhao1

1Cancer Genetics Laboratory, Kolling Institute of Medical Research, Royal North Shore Hospital, St Leonards, NSW, Australia
2Department of Anatomical pathology, Royal North Shore Hospital, St Leonards, NSW, Australia
3Department of Endocrinology, Royal North Shore Hospital, St Leonards, NSW, Australia
4University of Sydney Endocrine Surgery Unit, Royal North Shore Hospital, St Leonards, NSW, Australia

Background: Medullary thyroid carcinoma (MTC) originates from a small population of neuroendocrine C-cells of thyroid gland. Surgery remains the only curative treatment. The human REarranged during Transfection (RET) proto-oncogene is recognized as the key driver of MTC tumigensesis. This has been targeted by tyrosine kinase inhibitors (TKIs) but with modest efficacy. Growing evidence has shown that cancer biology can be modified by targeting miRNA expression. Progress of miRNA studies in MTC has been hampered due to the lack of normal C-cell tissue as a differential expression comparator.

Objective: We aimed to identify miRNAs whose expression is altered by RET proto-oncogene in MTC. We propose that specific RET-associated miRNAs play fundamental roles in MTC tumorigenesis and modulation of TKI responses, with the ultimate goal of establishing systemic miRNA-based treatment as a novel therapeutic regimen.

Methods: RET of human MTC cells were silenced with TKI, Cabozantinib, or siRNA. Enriched miRNA samples were prepared for small RNA sequencing performed by Australian Genome Research Facility. MTC cells were transfected with miRNA mimics and functional assays were performed to assess the miRNA effects.

Results: A list of differentially expressed miRNAs was first identified with small RNA sequencing then validated in a large cohort of MTC tissues using RT-qPCR. Restoration of under-expressed miR-1277-5p and miR-153-3p significantly reduced cell proliferation in MTC cells. Combined treatment of miR-1277-5p and Cabozantinib significantly increased cell responses to individual treatment, leading to enhanced cell proliferation inhibition. Furthermore, mRNA and protein expression of ribosomal protein S6 kinase B1 (RPS6KB1) was significantly reduced with increased expression of miR-1277-5p and miR-153-3p.

Conclusions: To our knowledge, this is the first study identifying RET-associated miRNAs in MTC. Functional characterisation of under-expressed miRNAs as tumour suppressors in MTC will offer potential to establish novel miRNA therapy.

The Roles of SIRT1 in Maternal Obesity-Induced Metabolic Disorders in the Offspring

Long T NGUYEN1, Hui Chen2, Carol Pollock1, Sonia Saad1

1Renal medicine, Kolling Institute, Royal North Shore Hospital, The University of Sydney, Sydney, NSW, Australia
2School of Life Sciences, Faculty of Science, University of Technology Sydney, Sydney, NSW, Australia

Background: Obesity is a well-known risk factor of multiple metabolic disorders. Importantly, due to the effects of fetal programming during pregnancy, not only obese mothers but also their offspring are affected. Sirtuin (SIRT) 1 is a metabolism-regulating deacetylase whose activation has been shown to mimic the effects of caloric restriction and ameliorate metabolic disorders associated with obesity. However, in the setting of maternal obesity, the role of fetal/neonatal SIRT1 is yet to be elucidated.

Hypothesis: In this study, we tested the hypothesis that overexpression of SIRT1 in the fetus is protective to the abnormal fetal metabolic programming induced by maternal high-fat diet (HFD) consumption during pregnancy.

Methods: Female C57BL/6 wild-type mice (WT, 6 weeks old) were fed either chow or HFD for 6 weeks, then bred with hemizygous transgenic males (SIRT1-tg, Col1a1tm1(CAG-Sirt1)Dsin) to produce WT and SIRT1-tg pups (1:1 ratio).

Results: In the genotyped WT offspring, maternal HFD induced a significant reduction of liver and hypothalamic SIRT1 mRNA expression in association with increased body weight, fat mass, levels of plasma triglycerides (TG), free fatty acids, as well as glucose intolerance. Maternal HFD also increased liver TG concentration and the mRNA expression of Sterol regulatory element-binding proteins (SREBP)-1c and Fatty acid synthase (FAS), while reducing that of PPARY, PGC-1α and GLUT1. By contrast, the genotyped SIRT1-tg male offspring either from Chow- or HFD-fed mothers showed higher liver and hypothalamic SIRT1 expression in association with lower BW, fat deposition and glucose intolerance compared to the WT controls. Additionally, liver mRNA levels of PPARY and FAS were respectively up- and down-regulated in correlation with SIRT1 overexpression.

Conclusion: these results suggest potential protective effects of SIRT1 on maternal obesity-induced metabolic diseases, including type 2 diabetes and hepatic steatosis in the offspring.
Differentiating Bacteria Associated With Cystic Fibrosis Lung Infections
Katie D. NIZIÖ1, Katelynn A. Perrault1, Amanda N. Troobnikoff1, Maiken Ueland1, Shereen Mohsin2, Jonathan R. Iredell2, Peter G. Middleton3,4 and Shari L Forbes1
1Centre for Forensic Science, University of Technology Sydney, Broadway, NSW, Australia
2Centre for Infectious Diseases and Microbiology, Westmead Millennium Institute for Medical Research and Marie Bashir Institute, Westmead Hospital and The University of Sydney, Westmead, NSW, Australia
3Ludwig Engel Centre for Respiratory Research, Westmead Millennium Institute, Westmead, NSW, Australia
4CF Service, Department of Respiratory & Sleep Medicine, Westmead Hospital, Westmead, NSW, Australia

Background: Chronic pulmonary infections are the principal cause of morbidity and mortality in individuals with cystic fibrosis (CF). Due to the polymicrobial nature of these infections, the identification of the particular bacterial species responsible is an essential step in diagnosis and treatment. Current diagnostic procedures are time-consuming, and can also be expensive, invasive and unpleasant in the absence of spontaneously expectorated sputum. The development of a rapid, non-invasive methodology capable of diagnosing and monitoring early bacterial infection is desired. Future visions of real-time, in-situ diagnosis via exhaled breath testing rely on the differentiation of bacteria based on their volatile metabolites.

Aim: The aim of this proof-of-concept study was to investigate whether a range of CF-associated bacterial species could be differentiated based on their in-vitro volatile metabolomic profiles. Future studies will address the long term goal of developing a portable instrument to rapidly and non-invasively diagnose common lung infections based on the volatile by-products produced via exhaled breath.

Methods: Headspace samples were collected using solid phase microextraction (SPME), analysed using comprehensive two-dimensional gas chromatography – time-of-flight mass spectrometry (GC×GC-TOFMS) and evaluated using principal component analysis (PCA).

Results: The particular pattern of volatile organic compounds (VOCs) detected for each bacterial species was found to be dependent upon the bacterial growth phase (e.g. logarithmic vs. stationary) and sample storage conditions (e.g. short-term vs. long-term storage at -18 °C).

Conclusions: Although it was not possible to effectively differentiate all six bacteria investigated using this method, the results revealed that the presence of a particular pattern of VOCs (rather than a single VOC biomarker) is necessary for bacterial species identification. Future studies will benefit from the approaches presented in this study and further facilitate the production of diagnostic tools for the early detection of bacterial lung infections.

FXYD1 Is An Endogenous Protector Against Oxidative Stress Induced Vascular Dysfunction
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1North Shore Heart Research, Kolling Institute, University of Sydney
2Department of Neurosurgery, Massachusetts General Hospital & Harvard Medical School, Boston, Massachusetts
3Department of Cardiology, Royal North Shore Hospital

Background: The FXYD protein family are type I membrane proteins have been demonstrated to be protective against redox modification of key membrane proteins, including the Na+/K+ pump and endothelial nitric oxide synthase (eNOS). We have previously shown acute beneficial effects of FXYD1 on Na+-pump and eNOS activity. Here, we explore the effects of FXYD1 on vascular pathophysiology in the systemic and pulmonary circulations in vivo.

Aims: To explore the effects of FXYD1 on vascular pathophysiology in the systemic and pulmonary circulations in vivo.

Methods: Six-month old male mice were treated with angiotensin II infusion (1 week). Implantable blood pressure catheters were inserted into the aortic arch, and relaxation in response to administration of bradykinin measured. Post-sacrifice, heart/aorta were collected, paraffin-embedded, and stained (van Gieson’s/Masson’s Trichrome) to detect changes in aortic wall thickness and cardiac perivascular fibrosis. Echocardiography was performed to estimate pulmonary artery pressures using a high frequency transducer in 3-month old mice anaesthetised with isoflurane.

Results: Knockout (KO) of FXYD1 in a six-month old mouse model was associated with both significant increases in aortic medial thickness and cardiac perivascular fibrosis. There were no significant differences in baseline systemic blood pressures in FXYD1 KO mice. However, FXYD1 KO mice had a 50% reduction in endothelial-dependent vasodilation compared to WT. Further, pulmonary artery pressures were significantly elevated: (pulmonary acceleration time [PAT]: WT:23.29 ± 0.9932, n=7, KO:18.57 ± 0.7190, n=7, KO:18.57 ± 0.7190, n=7, p=0.002, PAT/ejection time [ET]: WT:0.4086 ± 0.01353, n=7 KO:0.3257 ± 0.02125, n=7, p=0.006).

Conclusion: FXYD1 has a protective effect on medial hypertrophy, perivascular fibrosis, and endothelial dysfunction in the systemic vasculature, and has a newly defined role in pulmonary arterial circulation, consistent with our previous findings of its ability to prevent redox-mediated impairment of key membrane signalling proteins.

Insulin-Like Growth Factor Receptor And Sphinicosine Kinase Co-Expression Has Therapeutic Potential In Breast Cancer
Aleksandra M. OCHNIK1 and Robert C. Baxter1
1Kolling Institute of Medical Research, University of Sydney, NSW 2065, Australia

Insulin-like growth factor receptor (IGF1R) signaling is oncogenic, yet has not led to high clinical success as a monotherapy. There is now re-ignited hope that co-targeting the IGF1R in conjunction with other breast cancer therapies will be more clinically effective. The sphingosine-kinase (SpK1) signaling pathway is activated by IGF1R, and since both genes are associated with therapy-resistance and worse prognostic outcomes, we hypothesized that co-targeting IGF1R and SpK1 may be an effective...
novel breast cancer therapy. In vitro studies using MTT-assay and clonogenic assay were performed using the luminal, estrogen receptor (ER)-positive MCF7 and T47D and the basal-like, ER-negative HCC-1806 and HCC70 breast cancer cell-lines to test the effectiveness of the dual IGF1R-inhibitor (OSI-906; 0.1 – 10(µM) and the SphK1-inhibitor (SKI-11; 1 - 20(µM) as single-agents and combined. Statistical analysis was completed using a one-way ANOVA (p<0.05), repeated measures (p<0.05) and calculation of drug synergism by a combination index (CI) <1 (CommSyn). IGF1R, SphK1 and Ki67 expression were measured by immunohistochemistry in 238 formalin-fixed breast cancer tissues and sub-divided into seven subtypes based on ER, progesterone receptor (PR) and human epidermal growth factor receptor (HER2) expression. 4 µM SKI-11 acted synergistically with OSI over the range 0.1 - 6.4 µM (CI<1), and significantly increased sensitivity towards OSI (p<0.05). IGF1R expression was highest in the ER+, PR+ and HER2- (37%), ER+, PR+, HER2+ (17%) and ER-, PR-, HER2- (17%) subtypes compared to SphK1 and Ki67 which was highest in the ER-, PR+, HER2+ (87% SphK1 and Ki67) and ER-, PR-, HER2- (81%; SphK1 and 72%; Ki67) subtypes. The highest IGF1R and SphK1 co-expression was observed in the ER+, PR+ and HER2- (24%), ER+, PR+, HER2- (13%) and ER-, PR+, HER2+ (13%) subtypes. Collectively, these studies have identified that co-targeting IGF1R and SphK1 has potential therapeutic benefit in specific molecular breast cancer subtypes.

Heterogeneity Of Thawed And Cored Frozen Blood Aliquots
Kathleen M PHILLIPS1, Anna L Campbell1, Leo Phillips3, Danson Wooll1, Ussha Pillai1, Ross C Smith1,2, Viive Howell1,2, Alexander Engel1, Deborah J Marsh1,2, Robert C Baxter1,2
1Hormones and Cancer Group, Kolling Institute of Medical Research, RNSH, St Leonards, NSW, 2065
2Sydney Vital Translational Research Centre, Kolling Institute of Medical Research, RNSH, St Leonards, NSW, 2065
3Mass Spectrometry Core Facility, DVC Research, University of Sydney, The Hub, Camperdown, NSW 2006

Background: Freeze-thawing of stored blood samples for distribution to researchers has raised concerns over losses in protein integrity, leading to inaccurate quantification of biomarkers. The CXT350 frozen sample aliquotter can extract multiple frozen homogenous aliquots (cores), preserving sample quality. The potential for an uneven distribution of analytes within a single cryotube upon sample freezing to influence the measurement of biomarkers has not been tested. By doing so, the Kolling Tumour Banks can provide researchers with higher quality samples, leading to faster developments in precision therapies for our patients.

Aim: To determine if heterogeneity exists in serum and plasma protein concentrations between aliquots cored at different depths using the CXT350, and thawed aliquots of the same volume, using matrix-assisted laser desorption/ionisation time-of-flight mass spectrometry (MALDI-TOF MS).

Methods: Blood was collected from five volunteers and processed using local protocols. Samples were aliquotted into cryovials and stored at -80°C and -175°C. One set of samples from each temperature group was thawed, aliquotted and re-frozen, while the other set was cored multiple times at different depths using the CXT350. Samples were then thawed, plated in duplicate with matrix, and intact proteins analysed by MALDI-TOF MS. Protein ions were quantitated by peak intensities. Data was analysed by ANOVA using SPSS v22.0.

Results: Plasma samples showed a significantly greater concentration of proteins in cored aliquots than thawed aliquots (*p<0.05), and top cored aliquots had higher protein concentrations than middle cored aliquots across 56% of protein peaks (*p<0.05). In serum samples, the effect of aliquotting method and coring depth was not statistically significant.

Conclusion: This preliminary study suggests that frozen whole core aliquoting of plasma and serum is equal or superior to freeze-thaw aliquotting in the preservation of protein content and distribution.

ABSTRACT SESSION 2: POST-DOCTORAL SESSION *SHORT LISTED FOR PRIZES

Functional Prediction Of Long Non-Coding RNAs In Ovarian Cancer-Associated Fibroblasts Indicate A Role In Metastasis*
Emily K COLVIN1,2, Fatemeh Vafaei3,4, Samuel C. Mok3, Michael J. Birrer6, Viive M. Howell1,2, Goli Samimi7
1Bill Walsh Translational Cancer Research Laboratory, Kolling Institute, Northern Sydney Local Health District, St Leonards NSW 2065, Australia
2Sydney Medical School Northern, University of Sydney, NSW 2006, Australia
3Charles Perkins Centre, University of Sydney, Sydney, Australia
4School of Mathematics and Statistics, University of Sydney, Sydney, Australia
5Department of Gynecologic Oncology and Reproductive Medicine Research, Division of Surgery, The University of Texas MD Anderson Cancer Center, Houston, TX
6Harvard Medical School, Massachusetts General Hospital Cancer Centre, Boston, MA
7Garvan Institute of Medical Research, The Kinghorn Cancer Centre, Darlinghurst, Australia

Background/Aims: Ovarian cancer is the most lethal gynaecological malignancy in women. Cancer- associated fibroblasts (CAFs) contribute to this poor prognosis by enhancing tumour cell survival and metastasis, making them an attractive therapeutic target. DNA mutations are rare in CAFs, raising the likelihood of other mechanisms to regulate gene expression such as long non-coding RNAs (lncRNAs). We sought to identify lncRNAs specific to CAFs and by highly novel computational approaches predict those involved in metastasis.

Methods: RNA expression from 67 ovarian CAF samples and 10 normal ovarian fibroblast samples was analysed using Affymetrix U133 Plus 2.0 Arrays. Differentially expressed probes corresponding to lncRNAs were calculated using linear models for microarray data (limma) package from Bioconductor and a moderated t-statistic used to assess significance. A functional network was constructed by integrating our data with publically available datasets (CHIPBase) to predict those CAF-specific lncRNAs involved in metastasis.
Results: Based on a significance cutoff of fold-change $>2$ and a p-value $<0.05$, 62 lncRNA probes were identified including 41 unique lncRNAs, as differentially expressed in CAFs versus normal fibroblasts. Logistic regression modelling and ROC curve analysis demonstrated that these lncRNAs were able to confidently distinguish CAFs from normal fibroblasts. Interrogation of known transcription factor- lncRNA interactions, transcription factor-gene interactions and construction of a context-specific interaction network identified multiple lncRNAs involved in metastasis. These included known lncRNAs GAS5 and MALAT1 as well as novel lncRNAs.

Conclusion: Until recently, lncRNAs were thought to be “transcriptional noise” but are now recognised to play important roles in several diseases, including cancer. We have identified for the first time in any cancer type that lncRNAs can be used to distinguish CAFs from normal fibroblasts and contribute to the metastasis-promoting phenotype of CAFs.

Utilising Metabolomics To Identify Predictive Biomarkers For Pregnancy Complications*
Katie L POWELL1,3,4, Anthony Carrozzi2,3, Vitomir Tasevski1,4, Anthony W Ashton1,3, Anthony C Dona2,3
1Division of Perinatal Research, Kolling Institute, Northern Sydney Local Health District, St Leonards, NSW, 2065, Australia
2Department of Cardiology, Kolling Institute, Northern Sydney Local Health District, St Leonards, NSW, 2065, Australia
3Sydney Medical School Northern, University of Sydney, NSW, 2006, Australia
4Pathology North, NSW Health Pathology, Royal North Shore Hospital, St Leonards, NSW, 2065, Australia

Background: Pregnancy complications, including pre-eclampsia (PE) and intrauterine growth restriction (IUGR) can severely impact the short and long-term health of the mother and/or baby. Currently, there are no diagnostic tests available to identify women at high risk of developing either condition at some stage during pregnancy.

Aim: This study aims to identify novel serum biomarkers using metabolomics, which can identify women at risk of developing PE and/or IUGR during pregnancy. Metabolomic profiling is an emerging technology that provides a snapshot in time of all upstream biological processes, which ultimately represent a patient’s phenotype.

Methods: Third trimester serum samples were collected from women with known pregnancy outcomes of PE, IUGR, PE/IUGR or healthy pre-term or term pregnancies (< or >38 weeks gestation, respectively). Samples were analysed by 1H Nuclear Magnetic Resonance (NMR) spectroscopy using a Bruker Avance III 400 MHz spectrometer. The resulting serum spectra were analysed used multivariable statistical techniques to identify trends in the metabolite profiles.

Results: Differences in metabolite profiles could identify PE, IUGR and PE/IUGR pregnancies separately to healthy pregnancies consisting of samples collected pre-term and at term, based on a principle component analysis. A panel of 20 metabolites were identified and used to develop predictive models. A model comparing PE and healthy pregnancies correctly identified 98% of patient outcomes.

Conclusions: Metabolic profiling of symptomatic pregnancies has shown strong predictive power in correctly classifying patients according to their clinical outcome. The novel metabolites identified herein demonstrate the potential for developing an early, pre-symptomatic, diagnostic test.

Assessment Of Variables Affecting Lung Protein In Exhaled Breath Condensate*
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Background/Aims: Lung cancer is a leading cause of cancer-related mortality. Survival is improved by diagnosis at an early stage before the disease has spread and therapeutic options are limited. Analysis of exhaled breath condensate (EBC) has recently been proposed as a non-invasive method to diagnose early-stage lung cancer. Since this sample type has not been comprehensively profiled before, here, we investigated variables that may affect protein yield in EBC, prior to constructing a protein profile of human breath.

Methods: Using EBC from healthy volunteers, we assessed the effect on total EBC protein of storage tube, method of concentrating the sample, cooling temperature, addition of protease inhibitors, the use of filters/nose-clips and the effect of delaying processing for up to 24hrs. Total protein was quantitated using the Protein Assay Kit on a Qubit 3.0 Fluorometer. Protein mapping of EBC from a healthy volunteer was performed by Information Dependent Acquisition MS on a Sciex 6600 TripleTOF.

Results: We determined that EBC should be collected at lower cooling temperatures (-80°C) for greatest EBC volume and protein yield. EBC should be stored at -80°C in plastic with the addition of protease inhibitors to ensure long-term stability of the sample. Protease inhibitors also assist in stability of EBC protein when the sample was left at room temperature for up to 24hr. Use of nose-clips/filters do not affect total EBC protein. In a preliminary protein MS profile, 27 proteins were identified in EBC from a healthy volunteer.

Conclusion: Understanding the factors that affect EBC yield and quality is crucial for reproducible high yield samples and the generation of robust EBC proteomic profiles. Improving our knowledge of protein pathways and their changes during carcinogenesis is crucial in the evaluation of EBC to provide an “exhaled biomarker fingerprint” of early-stage lung cancer.
Insulin Pump Therapy In Pregnant Type 1 Diabetic Women Is Not Associated With Improved Perinatal Outcomes
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Background: Continuous subcutaneous insulin infusion (CSII; insulin pump therapy) is widely regarded as the optimal treatment modality for individuals with type 1 diabetes (T1D); however, retrospective studies have shown that insulin pump therapy in T1D pregnancy does not improve perinatal outcomes, in comparison to multiple daily injections (MDI) of insulin.

Aims: To compare the perinatal outcomes of women with T1D using MDI versus CSII therapy in pregnancy.

Methods: A prospective, observational study was conducted at Royal North Shore Hospital, Sydney, to investigate the relationship between glycaemic control and perinatal outcomes in T1D pregnancy. 21 women were recruited over 2 years and ~50% of women (n = 11) used CSII.

Results: Women with T1D using MDI therapy were more likely to be older and to weigh less before pregnancy than women using CSII. Both groups required increasing doses of basal insulin as pregnancy progressed, however women in the CSII group required significantly more in the second (31.2 ± 4.7 vs. 19.4 ± 3.8; p = 0.049) and third (38.7 ± 5.7 vs. 22.3 ± 5.2; p = 0.034) trimesters. Women using CSII were more likely to have LGA or macrosomic neonates (p = 0.015 and p = 0.011, respectively). Following adjustment for maternal age and pre-pregnancy body weight, the significant association between CSII use and LGA was retained (p = 0.027 and p = 0.044, respectively). There was no difference in rates of caesarean section, neonatal hypoglycaemia, jaundice, respiratory distress or NICU admission.

Conclusions: In the present study, use of CSII in T1D pregnancy was associated with a higher incidence of LGA and macrosomic neonates, with no improvement in other pregnancy outcomes. This finding is unexpected and further research on possible mechanisms is required.

Awake Versus Sleep Prediction Methods For Selecting Obstructive Sleep Apnoea Patients For Oral Appliance Therapy*
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Background: Obstructive Sleep Apnoea (OSA) is a common sleep disorder requiring lifelong treatment to reduce symptoms and circumvent downstream health consequences. Oral appliance (OA) therapy treats OSA by protruding the mandible and enlarging the pharyngeal airway but is not efficacious in all patients. Current inability to pre-select OA responders is a major clinical barrier. Simple awake prediction tests are the ideal goal; however a single test is not prospectively validated. Sleep tests have good preliminary results but are resource-intensive involving prototype devices.

Aims: To assess optimal 1) awake and 2) sleep prediction tests for OA treatment response in a clinical sleep centre.

Methods: OSA patients underwent prediction testing before OA therapy. Awake testing involved a novel multimodal phenotyping approach applying a range of simple office-based tests. Sleep testing used a newly commercially-available device following a published protocol, yet to be validated in other centres. OA treatment response was determined by polysomnography. Accuracy of awake and sleep prediction methods was assessed.

Results: Awake assessments were completed in 142 OSA patients. OSA severity and BMI were the strongest predictors and combined awake tests did not improve prediction. Predictive accuracy (ROC AUC 0.71) is inadequate for clinical use. Sleep testing was performed in N=42 with a test fail rate of 21.5%. However in N=33 successful tests, there was good sensitivity (81.8%) and specificity (92.6%) for OA response.

Conclusion: We have demonstrated in a generalizable clinical sample that awake tests and patient characteristics do not have sufficient predictive accuracy to select OSA patients for OA therapy. Overnight sleep testing shows greater accuracy, although there is a high failure rate of the current method. Our work suggests that sleep tests are likely necessary for accurate prediction of OA treatment response.

Development of an international geriatric pharmacology curriculum for medical schools: systematic review and curriculum mapping
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Introduction: Understanding and application of pharmacology and therapeutics in old age and multi-morbidity is critical to ensure quality use of medicines in our ageing population. Education in geriatric pharmacology in medical schools is starting to emerge internationally.

Aims: To systematically review the published literature for evidence of existing geriatric pharmacology education in medical schools and use this to inform development of an international geriatric pharmacology curriculum.
Methods: A systematic literature review using subject terms “geriatric, pharmacology, clinical pharmacology, curriculum, medical students, medical education, skills, knowledge, and attitude in combination” was performed in MEDLINE, EMBASE, PsyCINFO and Pubmed databases for studies published 2000-2016. Overall, 457 articles were screened by title and abstract. Based on inclusion criteria (available in English, focus on content or learning outcomes for pharmacology, clinical pharmacology or geriatric pharmacology, medical school education), 43 full text articles were screened further and any relevant curriculum and learning outcomes were mapped. Once literature had been identified, a thematic analysis was undertaken in order to extract themes, which were learning topics related to geriatric pharmacology course.

Results: Seventeen relevant peer-reviewed articles were identified. Of these, 10 articles described geriatric pharmacology related learning topics. Geriatric pharmacology learning topics included changes in pharmacokinetics and pharmacodynamics, polypharmacy, dose adjustment to account for ageing physiology, medications that should be avoided or used with caution in older adults, consideration of the patient's goals of care, and collaboration with other health professionals to ensure safe prescribing for the elderly. No detailed, systematic or complete geriatric pharmacology curricula or learning outcomes were identified.

Discussion: This systematic review of existing literature highlighted that there is a need for a comprehensive international curriculum in geriatric pharmacology to teach medical students safe and effective prescribing for older patients.

TUESDAY 22ND NOVEMBER 2016

ABSTRACT SESSION 3: YOUNG INVESTIGATORS PRIZE SESSION

Vascular TRAIL expression is increased in endothelial cells by Angiotensin II
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Background: Angiotensin II (AngII) is a hormone that controls cell proliferation and apoptosis and is an important regulator of endothelial cell (EC) function, necessary for vascular homeostasis. Recently, we showed that TNF-related apoptosis-inducing ligand (TRAIL) promoted EC proliferation, migration, and blood vessel growth. Whether AngII regulates EC function via TRAIL is unknown.

Aim: Determine whether AngII regulates TRAIL transcription/expression, and how this influences EC function.

Method: Serum-arrested human microvascular EC (HMEC-1) were transfected with -1523TRAIL-Luc, a human TRAIL promoter reporter, followed by AngII (50 ng/ml) for 24 h. Luciferase activity was assessed. RNA was extracted from HMEC-1 treated with AngII; TRAIL and TRAIL-receptor expression was measured by qPCR. AngII-induced TRAIL mRNA was also examined in response to AT1R (Losartan) and AT2R (PD123177) antagonists. EC proliferation, migration and apoptosis were examined in response to AngII. Cells were transfected with siRNA targeting TRAIL to determine the involvement of TRAIL in AngII-induced migration.

Results: AngII increased TRAIL transcriptional activity. mRNA expression of TRAIL and its receptor DR5 was increased following AngII treatment. AngII-inducible TRAIL mRNA was inhibited with losartan, but not PD123177. While AngII did not alter HMEC-1 proliferation or apoptosis, it increased the ability of HMEC-1 cells to migrate. Importantly, this was impaired with siTRAIL, but not with control siRNA.

Conclusion: This is the first demonstration showing that AngII positively regulates TRAIL transcriptional activity and expression via AT1R, and that AngII-induced migration of EC involves TRAIL. Understanding how EC function is regulated under normal and pathological conditions, may offer new direction in the development of therapies associated with cardiovascular diseases.

Nurses are underutilised in Antimicrobial Stewardship - results of a multisite survey in paediatric and adult centres
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**Background:** Nurses represent a large, stable workforce that play a pivotal role in the delivery of patient care. Antimicrobial Stewardship (AMS) activities are embedded within the daily workflow of nurses. To date, however, the role of nurses in AMS has not been clearly defined.

**Method:** An electronic survey was administered to nursing staff across three Australian tertiary metropolitan institutions with AMS facilitated by a shared electronic approval and decision support system. The survey explored perceptions and attitudes of nurses in regard to AMS, their roles as nurses, and sought to identify differences in perceptions and attitudes across paediatric and adult settings.

**Results:** One hundred and forty two completed responses were received. Paediatric nurses comprised 40% of responders. Sixty five percent of nurses were familiar with the term AMS and 75% recognised that nurses were expected to have a role. Over 80% of nurses selected hand hygiene and infection control, patient advocacy and knowledge of antimicrobials as AMS roles for nurses, but 57% reported their knowledge of antimicrobials was limited. Only 49% identified ensuring approval for restricted antimicrobials as part of their role. Most nurses identified AMS or infectious diseases (ID) teams and pharmacists as sources of support for AMS, indicating an interest in receiving education on appropriate antimicrobial selection (73%). Few statistically significant differences were found between the two settings.

**Conclusion:** Nurses consider AMS activities within their roles, but are being underutilised in AMS programs. Further engagement, education, support and acknowledgement is required to improve nursing participation.

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**Intra-Operative Amylase In Peri-Pancreatic Fluid Predicts Pancreatic Fistula After Distal Pancreatectomy**

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**Introduction:** Distal pancreatectomy (DP) is performed for the resection of tumours involving the body and/or tail of the pancreas. Post-operative pancreatic fistula (POPF) is a complication involving the leakage of pancreatic enzymes occurring in approximately 30% of DPs that may lead to several disastrous sequelae including post-operative haemorrhage and intra-abdominal sepsis. Intraoperative prediction of POPF may help guide management and post-operative monitoring of DP patients. The aim of this study was to assess the predictive value of intra-operative amylase concentration (IOAC) in peri-pancreatic fluid after distal pancreatectomy for the diagnosis of POPF.

**Methods:** Consecutive patients who underwent a distal pancreatectomy between November 2014 to September 2016 were included in the analysis. IOAC was measured intraoperatively followed by drain fluid analysis for amylase on post-operative days (POD) 1, 3 and 5. Receiver operator characteristic (ROC) analysis was performed to evaluate the discriminative capacity of IOAC as a predictor of POPF.

**Results:** IOAC was measured after distal pancreatectomy in 26 patients. The IOAC correlated significantly with (i) POD 1,3 and 5 drain amylase (p<0.01), (ii) the development of POPF (p <0.01), (iii) the grade of POPF, and (iv) the Clavien-Dindo grade of surgical complications. ROC curve analysis confirmed the predictive relationship of IOAC and POPF as a good test with an area under the curve of 0.92, (95%CI 0.81-0.99, p<0.01).

**Conclusion:** Measurement of IOAC allows early and accurate categorisation of patients at risk for POPF in distal pancreatectomy.

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**Uncovering The Missing Genetic Component Of Familial Amyotrophic Lateral Sclerosis Using Bioinformatic Approaches**

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**Background:** Amyotrophic lateral sclerosis (ALS, also known as motor neuron disease) is a debilitating and fatal neurodegenerative disease. Approximately 10% of cases are hereditary (familial ALS), while the remainder occur sporadically. The only known causes of ALS are gene mutations, which have driven research into understanding disease pathogenesis underlying ALS. However, known mutations account for just two thirds of familial ALS. ALS families without a causal gene mutation are generally small and often exhibit incomplete penetrance of disease, and are not amenable to classical gene discovery strategies. Thus new approaches are required to uncover the missing genetic component of ALS.

**Aims:** We propose novel bioinformatics and biostatisticians approaches to uncover the remaining genetic component of familial ALS.

**Methods:** Custom R code has been developed to parse patient exome sequencing data for 1) shared variants in families or 2) candidate genes, and to apply Fisher’s Exact testing. Identified novel variants were prioritised using a combination of: gene function, predicted functional consequence, amino acid conservation, genic tolerance and presence in online ALS database cohorts.

**Results:** Shared variant analysis and bioinformatics filtering was performed on five ALS families and found 18, 27, 31, 26 and 22 variants segregating with disease in each respective family. Subsequent prioritisation classified remaining variants as high, medium or low priority for further analysis. Candidate genes from proteomic (n=14), genome wide association (n=4), transgenic mouse (n=1) or other genetic (n= 6) studies were investigated. Our statistical approach provided evidence
implicating two novel variants in ALS, and suggesting four and eight known SNPs may respectively confer predisposition and protective effects for ALS.

Conclusion: Identification of causal ALS mutations or ALS associated variants inspires follow up functional studies into ALS pathogenesis. New causal genes can provide patients and families the opportunity for diagnostic testing or potentially pre-implantation embryonic screening.

**MicroRNAs: Novel Diagnostic Biomarkers For Diabetic Nephropathy**

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**Background:** Diabetic nephropathy (DN), is a worldwide public health problem, with adverse outcomes of kidney failure and premature death. Currently, clinical treatments of DN have only slowed the progression to end stage kidney disease, and eGFR and albuminuria are used as key markers to define DN, however both markers have their limitation as a predictor of renal outcome. Hence novel diagnostic biomarkers are urgently needed to predict disease prognosis and enable personalized therapy (precision medicine). This project aimed to identify urinary miRNAs as non-invasive diagnostic biomarkers in patients to predict the development of DN.

Methods: Fresh urine samples were collected from 10 normal volunteers, 14 diabetic patients without nephropathy, and 12 diabetic patients with nephropathy. miRNAs were extracted from 1ml urine using Exiqon kit, cDNAs were synthesized using miScript II RT Kit (Qiagen) and miRNAs were detected using Fibrosis Pathway-Focused miScript® miRNA PCR Array (Qiagen).

**Results:** miRNA PCR Array results have shown five significantly downregulated miRNAs (miR-744, miR-204, miR-21, miR-26a and miR-328) and one upregulated (miR-451a) in the urine of diabetic patients with nephropathy compared to both diabetic patients without impairment of renal function and normal volunteers (P<0.001).

**Conclusion:** Either single or a combination of miRNAs (miR-744, miR-204, miR-21, miR-26a, miR-328 and miR-451a) may potentially serve as novel non-invasive diagnostic biomarkers for DN and validating these miRNA in a large scale of clinical study warrants.

**Multiple myeloma causes T cell immunosenescence in tumour induced clones: Implications for immunotherapy**

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**Background:** Tumour induced T cell clones have been detected in multiple myeloma (MM) patients and are related to improved survival, despite being dysfunctional, suggesting a role in anti-tumour immunity.

**Aim:** To characterise the mechanism(s) of T cell dysfunction in order to determine target(s) for restoring the function of these protective cells, with implications for development of an autologous T cell therapy.

**Method:** T cell clones (CD3+CD8+TCRβ+CD57+) were identified using the Betamark kit and proliferation was measured using CFSE dilution. Phenotypic markers were measured using flow cytometry and signaling protein expression by phosphoflow cytometry. Telomere length was determined using qPCR and Flow-FISH.

**Results:** T cell clones were identified in 75% of MM patients (n=103) and were associated with an improved survival ($\chi^2=21.01; \ p<0.0001$). The cells were hypo-responsive as they were non-proliferative in response to TCR ligation and immune modulators. Multiple dysfunctional pathways were identified including suppression of the ERK proliferative pathway and activation of the TGF-β pathway which is involved in suppressing T cell activation. Low PD-1, CTLA-4, Tim-3 and LAG-3 levels were detected, suggesting that these cells were not anergic or exhausted. Expression of CD57, CD160, KLRG-1 and lack of CD28 expression, indicated cellular senescence. Telomere length was normal, indicating telomere-independent senescence in T cell clones.

**Conclusion:** Tumour-induced T cell clones in MM are senescent. As telomere length is not shortened, the dysfunction is potentially reversible and two potential targets for restoring T cell function have been identified. Reversing the dysfunction of these protective cells would constitute an immune cell therapy based on the restoration of the host immune response. Low levels of PD-1 and CTLA-4 expression suggest that immune checkpoint blockade would be unsuccessful in reversing the T cell dysfunction, as reflected in recent clinical studies.
Biomarker Discovery By MALDI--MS Imaging In Triple--Negative Breast Cancer
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Background: About 15% of breast cancers, lacking estrogen and progesterone receptors, and HER2 overexpression, are designated as triple--negative (TNBC). Patient outcomes are often poor for women with TNBC, with few prognostic biomarkers.

Aim: This study used MALDI--mass spectrometry imaging (MSI) of FFPE tissue to discover proteins that distinguish TNBC from adjacent unaffected tissue, with the aim of evaluating these proteins for prognostic utility.

Methods: 10 µm sections from ten FFPE TNBC tumours were mounted onto ITO--treated glass slides, and marked for regions of tumour and non--tumour tissue. Sections were deparaffinised, antigen--retrieved, and sprayed with trypsin to generate peptides, then CHCA matrix, using the ImagePrep workstation (Bruker). Imaging data was acquired using a Bruker UltraFlexT++ TOF--TOF MS in positive ion reflectron mode. Mass spectra were collected in the m/z range 700--3500 at 50 µm raster resolution. In parallel, tissue samples were extracted, trypsin--digested, and peptides identified by LC--MALDI-MS/MS to generate a reference library. Data were processed and analysed using SciLS Lab software, and distinguishing peptides were characterised by their ROC--AUC values.

Results: A shortlist of 13 proteins was generated, identified on the basis of m/z values of imaged peptides, having high ROC--AUC values in at least 3 tissues, when comparing cancer and non--cancer tissue regions. COL1A2 peptides were identified with ROC--AUC values >0.8 in most sections. SOX11, identified in 3 tissues, has been reported as a poor prognostic indicator in TNBC (Shepherd, Oncotarget 2016). Other identified proteins are being evaluated in TNBC TMA's for prognostic significance.

Conclusion: MALDI MSI is a powerful tool for cancer biomarker discovery, having the potential to identify novel prognostic markers with utility in the management of women with TNBC.

Bacterial Communities Vary between Sinuses in Chronic Rhinosinusitis Patients
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Background: Chronic rhinosinusitis (CRS) is a common and potentially debilitating disease characterized by inflammation of the sinus mucosa for longer than 12 weeks. Bacterial colonization of the sinuses and its role in the pathogenesis of this disease is an ongoing area of research. Recent advances in culture-independent molecular techniques for bacterial identification have the potential to provide a more accurate and complete assessment of the sinus microbiome, however there is little concordance in results between studies, possibly due to differences in the sampling location and techniques.

Aims: This study aimed to determine whether the microbial communities from one sinus could be considered representative of all sinuses, and examine differences between two commonly used methods for sample collection, swabs, and tissue biopsies.

Methods: High-throughput DNA sequencing of the bacterial 16S rRNA gene was applied to both swab and tissue samples from multiple sinuses of 19 patients undergoing surgery for treatment of CRS. Sequencing results were compared to hospital culturing results of swabs from the same sinuses.

Results: Swabs and tissue biopsies showed a high degree of similarity, indicating that swabbing is sufficient to recover the microbial community from the sinuses. Microbial communities from different sinuses within individual patients differed to varying degrees, demonstrating that it is possible for distinct microbiomes to exist simultaneously in different sinuses of the same patient. The sequencing results correlated well with culture-based pathogen identification conducted in parallel, although the culture missed many species detected by sequencing.

Conclusions: This finding has implications for future research into the sinus microbiome, which should take this heterogeneity into account by sampling patients from more than one sinus.

Impact of Novel Drugs in Rats with Mild Traumatic Brain Injury (mTBI)*
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Oxidative stress and mitochondrial dysfunction have been considered a major factor in the pathogenesis of central nervous system (CNS) injury such as TBI and stroke. Following mild TBI, overproduction of reactive oxygen species (ROS), and malfunctioning of antioxidant defence system such as manganese superoxide dismutase (MnSOD) have been recognised as a common mechanism in the pathogenesis of the TBI. Due to the overproduction of ROS, mitochondrial dynamics such as fission, fusion and autophagy is deregulated. This project aims to investigate the effects of two novel antioxidants, CTM10 and DTME10 (structure confidential) in the cellular outcomes following the treatment. A weight drop injury model was used to create a mild contusion at 3.0 mm lateral and 2.5 mm posterior to the bregma. After the treatment with two antioxidants, rats were sacrificed 24 hours and six weeks post injury. Using western blotting, level of MnSOD, dynamin-related protein 1 (Drp1), Optic atrophy 1 (Opal1) and light chain 3 (LC3) were measured. Using immunohistochemistry, glial fibrillary acid protein
Public perceptions and attitudes towards antibiotic resistance in Sydney, Australia*

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**Background:** The overuse of antibiotic resistance globally has contributed to the development and spread of antibiotic resistance. Current antibiotic effectiveness needs to be maintained and the overuse and misuse of antibiotics in the community needs to be addressed.

**Aim:** The study explored knowledge, attitudes and perceptions towards antibiotic use and antibiotic resistance among members of the general public in Sydney, Australia. Methods: A self-administered questionnaire was conducted online, in pharmacies and through a letterbox drop in 2015.

**Results:** A total of 583 individuals completed the survey and were included in the study. The study found that while most people in the sample had heard of antibiotic resistance, many had optimistic bias beliefs, held misconceptions about the causes of antibiotic resistance and impacts to themselves and the community. Inappropriate use of antibiotics for the common cold and flu and storage of unfinished antibiotics from previous prescriptions were reported by nearly a third of the sample. These self-reported behaviours and levels of trust in different sources of information were found to be significantly associated with demographic and cultural factors. Knowledge of antibiotic effectiveness and level and type of education attained, were identified as significant in influencing individuals’ levels of risk perception towards antibiotic resistance.

**Conclusion:** The findings point to the importance of local scale strategies and education interventions and the important role of doctors and pharmacists in educating the public on antibiotic use and antibiotic resistance.

Transcriptional Profiling of Macrophages Predicts a Therapeutic Application for a Parasite-Derived Peptide

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To minimise tissue damage and to prevent their expulsion from a mammalian host, parasitic worms (helminths) release excretory/secretory (ES) products, which interact with cells of the immune system to prevent excessive pro-inflammatory responses, and induce mechanisms of wound repair. The surprising impact of this immune modulation is the demonstration that live worm infection inhibits the pro-inflammatory responses mediating auto-immune diseases, such as Type 1 diabetes (T1D) and multiple sclerosis (MS). To understand the mechanisms involved, we have identified and characterised the biological activity of the proteins within the ES of the human parasite, Fasciola hepatica. Of these, a single peptide, FhHDM, displayed an ability to preferentially bind to macrophages (human and mouse) and localise to the endolysosomes. Addition of FhHDM to macrophages did not induce any changes in gene expression, cytokine or chemokine production, or the expression of surface markers. However, macrophages treated with FhHDM were inhibited in their ability to secrete pro-inflammatory cytokines in response to ligands of toll-like receptor (TLR) -3 (polyinosinic:polycytidylic acid [Poly I:C]) and -4 (lipopolysaccharide [LPS]); but not in response to ligands for TLR-2 and TLR-9. This observation combined with the inhibition of IFN-β production, indicated that FhHDM was selectively modulating the TIR-domain-containing adapter-inducing interferon-β (TRIF)-dependent pathway. To further elucidate FhHDM’s mechanism of action, a global transcriptomic analysis was conducted on macrophages treated in vitro with FhHDM and/or LPS. Pathway mapping of the microarray data confirmed the association of FhHDM with the TRIF pathway. In addition, the differential gene expression of macrophages treated with LPS compared to those treated with FhHDM and LPS, predicted a therapeutic effect for FhHDM-1 in the treatment of T1D and asthma.

Measuring frailty in CHF; validation of a question only approach*

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**Background:** Frailty is an independent predictor of poorer outcomes in people with chronic heart failure (CHF). The aim of this study was to compare a questionnaire only (TRILOGY ACS) measure versus a questionnaire plus handgrip strength (SHARE-FI) as measured by a dynamometer to determine which measure correctly identifies frail patients and better predicts worse outcomes.

**Methods:** Patients with a diagnosis of CHF were recruited to this study. Both frailty assessments consist of 5 domains: weakness, exhaustion, slowness, physical inactivity, and weight loss. A score
≥3 was classed as frail, 1-2 as pre-frail, and 0 as non-frail for TRILOGY. SHARE-FI uses a DFactor Score (DFS) based on an algorithm to assess frailty. Both inpatients and outpatients were recruited from a tertiary hospital in metropolitan Sydney. Frailty was assessed during a routine outpatient visit clinic close to discharge for inpatients.

**Results:** Twenty-seven patients (19M: 8F; age 53±14 years, range 23-77; LVEF 32±19%) underwent assessment with both tools. Using the result from the SHARE-FI, 13 (48%) were frail, 9 (33%) were pre-frail, and 5 (19%) were non-frail. The results from the TRILOGY measure showed that 21 (78%) participants were frail, 5 (19%) were pre-frail, and 1 (4%) was non-frail. In both measures, frailty was independent of age, sex, and LVEF. The SHARE measure showed that frailty was associated with a lower functional status score.

**Discussion:** Frailty is highly prevalent in people with CHF. A question-only measure reports higher rates of frailty than one that uses an objective measure to assess weakness. This is an ongoing study to determine if a questionnaire only measure correctly identifies frailty and predicts those patients most likely to die or be readmitted to hospital.

**The Protective Role of TRAIL in NAFLD**

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**Background:** Non-alcoholic fatty liver disease (NAFLD) is characterised by steatosis, inflammation and fibrosis in the liver, and is associated with type-2 diabetes and cardiovascular disease. Recent work in our laboratory has shown that high fat diet (HFD)-fed TRAIL/-/- mice display more severe symptoms of diabetes and atherosclerosis than Apoe/-/- mice. The role of TRAIL in NAFLD is unclear.

**Aims:** To determine whether HFD-fed Trail/-/-/Apoe/-/- mice display features of NAFLD and investigate if TRAIL treatment protects against NAFLD in vitro.

**Methods:** The in vivo study involved 6 week old Apoe/-/- and Trail/-/-/Apoe/-/- mice fed a HFD for 12 weeks. Livers were dissected and snap-frozen for gene expression analysis by qPCR, or processed for histology. Plasma alanine transaminase (ALT) was measured by ELISA. In vitro, human hepatocytes (HepG2) were treated with the free-fatty acid palmitate for 24 h, ± TRAIL treatment (1 ng/mL); lipid uptake and expression of inflammatory/fibrotic markers were assessed.

**Results:** Liver from Trail/-/-/Apoe/-/- mice had increased expression of the inflammatory marker MCP-1, and fibrotic markers collagen IV and MMP2 compared to Apoe/-/- mice. Masson’s Trichrome staining revealed increased steatosis and fibrosis in these mice. Plasma ALT was also elevated. In vitro, oil-red O staining revealed ~270% increase in steatosis, associating with elevated inflammatory and fibrotic markers. Importantly, lipid uptake in hepatocytes was significantly reduced, ~60% with TRAIL treatment.

**Conclusion:** In mice with TRAIL-gene deletion we observed increased hepatic steatosis, associating with increased expression of inflammatory and fibrotic markers. In vitro, TRAIL blocked palmitate-induced lipid accumulation. These results suggest that TRAIL protects against NAFLD, and may be useful as a potential therapeutic in the treatment of liver disorders.

**The Tumour Suppressive Roles of MicroRNA-497 in Adrenocortical Carcinoma**

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**Background:** Adrenocortical carcinoma (ACC) has high recurrence rates and poor prognosis with limited response to conventional cancer therapy. Recent contributions of high-throughput transcriptomic profiling studies have identified microRNAs (miRNAs) to have biological roles in ACC. miRNA-497 (miR-497) was identified significantly underexpressed in two major miRNA clusters in the study of Assié et al (2014). Furthermore, miR-497 has emerged as a tumour suppressor in several other cancers, including pancreatic, ovarian and lung cancers.

**Aim:** To investigate functional roles of miR-497 in ACC.

**Methods:** miR-497 mimics were transfected to restore expression level of miR-497 in ACC cell line NCI-H295R. Cellular proliferation was assessed by Cell Titer 96® Aqueous One Solution Cell Proliferation Assay (Promega). Cell cycle and apoptosis were evaluated by fluorescence-activated cell sorting analysis. Cell migration and invasion were tested using Cytoselect™ cell migration and invasion transwells (Cell Biolabs). Gene and protein expression were evaluated by RT-qPCR and western blot, respectively. Luciferase assay was performed to confirm the direct interaction of miRNA and its target genes.

**Results:** miR-497 was confirmed to be significantly underexpressed in our collected cohort of ACC tissues compared to normal adrenal cortex. Overexpression of miR-497 in ACC cells significantly suppressed cellular proliferation, induced G1 cell cycle arrest, and promoted early-stage apoptosis in vitro. Migration and invasion was also significantly inhibited by miR-497. Gain-of-function studies identified that miR-497 significantly reduced mRNA expression of eukaryotic translation initiation factor 4E (eIF4E), ribosomal protein S6 kinase B1 (RPS6KB1) and vascular endothelial growth factor A (VEGF-A). Protein expression of RPS6KB1 was also reduced. Moreover, the luciferase activity of the reporter construct containing 3' UTR of eIF4E was downregulated following co-transfection with miR-497.

**Conclusion:** Our functional studies establish miR-497 as a tumour suppressor, indicating its potential as a therapeutic target in ACC.
The Potential Of Nanomedicine: Non-Toxic, Stable Nanoparticles For Enhanced Cancer Therapy*

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Background/Aims: Iron oxide nanoparticles show great promise in cancer diagnostics and therapy, however, to date their use has been limited due to prolonged retention in the body causing organ toxicity. We determined the biodistribution and toxicity of our customised sterically stabilised super-para-magnetic iron oxide nanoparticles (SPIONs) of 10 nm and 25 nm average core diameters in healthy nude mice.

Methods: Mice underwent intra-peritoneal (IP) injection of SPIONs up to 90 mg Fe/kg body weight. SPION accumulation in tissues was determined at 5 time points between 1 hour and 7 days by Prussian Blue staining and graphite furnace atomic absorption spectroscopy (g-f-AAS). Toxicity was assessed by histology.

Results: SPIONs were detected in all tissues examined except the brain and kidney. The level of 10nm and 25nm SPIONs peaked by 4hrs and was cleared from all tissues by 1 week. Iron staining and g-f-AAS showed that the omentum retained the highest accumulation of SPIONs (10nm and 25nm). High levels of SPIONs were also seen in the spleen and liver. Iron staining patterns suggested macrophage uptake of SPIONs in selected tissues. This was confirmed through immunohistochemical labelling for the macrophage marker F4/80. Clearance of SPIONs was also detected in spot faecal samples in both 10nm and 25nm SPIONS.

Conclusion: Our results demonstrate single administration of SPIONs is non-toxic and biodistribution is broad but transient. The main clearances mechanisms of SPIONs appear to be via macrophages and faecal excretion. High accumulation in the omentum, a common metastatic site in ovarian cancer, may prove beneficial for SPION-mediated cancer therapy.

RIPK1: The Next Novel Target for Renal Fibrosis

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Background: Current therapies for renal fibrosis are largely ineffective. Therefore identification of novel therapeutic targets is essential. Recent studies have demonstrated RIPK1 is a crucial regulator of necrosis, apoptosis and inflammation, which have been well recognised to be involved in renal fibrogenesis. To date, the role of RIPK1 in renal fibrosis has not been reported.

Aims: To define the role of RIPK1 inhibition in alleviating renal fibrogenesis.

Methods: To examine the role of RIPK1 inhibition in fibrotic responses in renal tubular cells, RIPK1 expression levels were determined in normal and diabetic mice kidney tissue by immunohistochemistry. For in vitro studies, HK2 cells (human proximal tubular cells) were incubated with/without TGF-β1 (2 ng/ml) in the absence or presence of 80μM nec-1s (a selective pharmacological inhibitor of RIPK1) for 48 hours. Supernatant, cell lysate and total RNA were collected for Western blotting and quantitative RT-PCR analysis. The mRNA and protein expression of ECM proteins collagen I, IV, and fibronectin were measured.

Results: Renal tubular cells from diabetic mice had significantly higher expression level of RIPK1 compared to normal mice. TGF-β induced the protein expression of collagen IV (3.0 fold change; P<0.001), and fibronectin (1.5 fold change; P<0.05) compared to control, which were attenuated by the RIPK1 inhibitor nec-1s (n=4; both P<0.05). Quantitative RT-PCR showed TGF-β increased the mRNA level of collagen I (4.6 fold change; P<0.001) and IV (1.6 fold change; P<0.05), and fibronectin (2.5 fold change; P<0.001) compared to control, which were partially reversed by RIPK1 inhibitor nec-1s in HK2 cells (n=4; all P<0.05).

Conclusion: These results suggest that RIPK1 may be a potential novel target in renal fibrosis. Further studies will examine the role of RIPK1 inhibition in inflammatory cytokines and TGF-β signalling molecules in HK2 cells and in mouse models of CKD.

Sentinel Surveillance Of Plasmodium Falciparum Kelch Propeller For The Detection Of Artemisinin Resistance*

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Background: Malaria parasites, particularly Plasmodium falciparum, still cause fatalities in over 400,000 people annually. The World Health Organisation recommends artemisinin (and derivatives), in combination with a longer half-life partner, as the frontline treatment for clinical P. falciparum malaria. Resistance to artemisinins emerged in 2006 on the Thai-Cambodian border, and is now present in growing proportions throughout the Greater Mekong Subregion. Artemisinin resistant phenotypes (ARTR) are defined by delayed parasite clearance and the presence of defined ARTR molecular markers (Kelch propeller mutations, and background alleles pfCRT, pfFD, pfARPS10 and pfMDR2).
Aims: To develop a protocol for molecular surveillance of resistance-associated genes in P. falciparum samples from travellers returning to Australia. Additionally, to analyse archived P. falciparum samples from the NSW parasitology reference laboratory determining pfK13 and pfCRT mutation status.

Methods: Case information was recorded and DNA extracted from samples (n=153) using whole blood DNA extraction kit. Gene regions of interest (propeller region of Kelch, and short region around pfCRT codon 76) were amplified using nested PCR protocols. Kelch fragments were sequenced, and aligned to the P. falciparum 3D7 reference sequence for variant analysis. The mutation status of pfCRT at codon 76 (denoting chloroquine resistance) was determined by allele specific restriction digestion with Apo1, and gel electrophoresis visualisation.

Results: The project found seven propeller mutations, including the C580Y mutation most strongly associated with artemisinin resistance. The C580Y specimen originated in Papua New Guinea, where resistance-associated kelch mutations are unreported. The pfCRT protocol determined the codon 76 mutations status for all samples, generating regional proportions.

Conclusions: This protocol identifies ARTR genotypes which will assist to pre-empt treatment failure of traveller’s malaria, and will allow Australian laboratories to participate in the global surveillance of emergent ARTR.

Modulating the innate immune system in rats following spinal cord injury using a novel molecule derived from a helminth parasite*

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Current understanding of the neuroinflammatory responses in spinal cord injury (SCI) has revealed a disproportionate presence of pro-inflammatory M1 macrophages compared to anti-inflammatory M2 macrophages in the injured spinal cord. This phenomenon is driven by an excessive innate immune response and closely mirrors the cellular profile of chronic non-healing wounds, promoting the permanent nature of SCI. Following the recent characterisation of secreted proteins from helminth parasites as potent anti-inflammatory agents, their potential as a therapeutic for SCI was tested. In particular, the ability of the helminth defense molecule FhHDM-1, to influence the innate immune response after SCI was investigated. Adult female Sprague-Dawley rats were subjected to a mild SCI and compared to sham surgery. All rats received a daily intraperitoneal injection (ip) of either FhHDM-1 or saline for seven days. Flow cytometric analysis of their innate immune responses at one week and three weeks after surgery revealed that FhHDM-1 did not affect the uninjured spinal cord, but significantly affected the early innate immune response in the injured spinal cord. This translated to an increased early infiltration of total immune cells, including M1 macrophages, despite positive physiological patterns during the post-operative recovery period. However, there was also a significant increase of M2 macrophages following treatment with FhHDM-1 that was not observed in the saline treated SCI, suggesting beneficial effects during recovery, and is supported by a decrease of pro-inflammatory cytokines within the spinal cord tissue. Supplementary analysis of other biological parameters such as behavioural and histological studies may be extremely useful in determining the neurological and functional outcome of the SCI after treatment with FhHDM-1.
**POSTER PRESENTATION ABSTRACTS**

Posters are listed alphabetically by the last name of the presenting author (underlined).

**MONDAY 21ST NOVEMBER 2016**

**DELEGATE POSTERS *SHORT LISTED FOR PRIZES***

**Improving Implantable Technologies by Optimizing Specificity and Efficiency Between Electrode-Cell Interfaces**

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Mimicking normal function of neuron cells in vitro is a crucial step towards developing technologies that can compensate for missing brain function via bio-implants. Making the necessary intimate contact between the electrode and cell plasma membranes has always been a challenge to existing procedures for stimulating and reading ionic currents in neurones and other electrically active cells. An appropriate interface should reduce the leakage pathways between adjoining cells to avoid electrophoretic ion currents being lost into the interstitial medium before arriving at the reading electrode. Our aim is to prepare biocompatible electrode coatings that permit localized electrode-cell intimate contact and communication. This improves the selectivity and sensitivity of the electrodes that are employed to stimulate and monitor cell activity in vitro and in vivo. One possible solution is to functionalize the gold surface of the electrode to provide more efficient cell-surface crosstalk points. This is now feasible via the use of novel tethered bilayer membrane technologies. Tethered bilayer membranes are stable and tough. Moreover, they provide a hydrophilic ionic reservoir of 2nm. This spacing readily enables alternating current electrical impedance measurement to mimic the structural, sensing and transport properties of biological membranes. Cell spreading and proliferation surface interactions can be analysed by fluorescence microscopy, the efficiency of electrode-cell contacts is assessed using electrical impedance spectroscopy. This study will further investigate the influence of a range of chemicals and chemo-attractants on different cells. The ultimate significance of the project is to improve the quality of life for visually and hearing impaired patients by developing a more effective interface by which to directly communicate with biological neuronal tissue, for superior biomedical devices such as cochlear and retinal implants.

**Telephone-based management of pressure ulcers in people with spinal cord injury in low- income and middle-income countries: a randomised controlled trial**

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**Background and Aims:** Pressure ulcers are very common in low-income countries and people are commonly left to manage their pressure ulcers at home. The aim of this study was to determine whether a low-cost model of care involving weekly telephone contact with patients was effective for helping these individuals manage their pressure ulcers.

**Methods:** A 12-week multicenter, prospective, assessor-blinded, parallel randomised controlled trial was conducted. Participants were included if they sustained a spinal cord injury more than 3 months prior and had at least one pressure ulcer. Participants were randomly allocated (1:1) to usual care (Control group) or to usual care with weekly telephone contact (Intervention group). The weekly telephone contact was with a trained healthcare professional for 15 to 30 minutes a week. The healthcare professional provided ongoing advice and support. Specifically, this included reinforcing self-help strategies for managing pressure ulcers, minimising psychological stress and enhancing engagement with life important. The primary outcome was the size of the pressure ulcer at 12 weeks. Some of the secondary outcomes included Pressure Ulcer Scale of Healing, Depth, Braden Scale, Undermining Distance and satisfaction with healthcare service delivery.

**Results:** One hundred and twenty participants were randomised from 3 centers. The mean between-group difference for size of pressure ulcer at 12 weeks was 1.1 cm² (95% CI -0.8 to 4.3).

**Conclusion:** Pressure ulcers remain a major problem in low-income countries. Weekly telephone contact does not help heal pressure ulcers although people value the regular contact and support with a health professional.

**Potential Health Effects of Electronic Cigarettes in Chronic Obstructive Pulmonary Disease**
Objective: Electronic cigarettes or Electronic Nicotine Delivery Systems (ENDS) have become the consumer-preferred alternative to smoking tobacco. ENDS have been proposed and are used as a smoking cessation device, but there is a lack of information on their potential health risks or benefits. Chronic Obstructive Respiratory Disease (COPD) is a disease caused by smoking, in which quitting smoking is the most efficacious method of slowing disease progression. Therefore it is likely that patients with COPD will use ENDS as a quit smoking device. A number of studies have shown that ENDS vapour is pro-inflammatory and toxic in vitro, which could possibly have negative implications for COPD patients using ENDS. It has also been shown that there is an increase in toxic by-product formation when the devices are used at high temperature. Hence we hypothesised that E- vapour would increase pro-inflammatory mediator production and have cytotoxic effects that could be increased through increasing vaporisation temperature of ENDS.

Methods: In an in-vitro model, Primary human airway smooth muscle (ASM) cells were stimulated with ENDS Vapour Extract (EVE) from 4 different E-liquids. Assessment of toxicological effects were obtained using an MTT assay, and pro-inflammatory mediators IL-6 and IL-8 were measured using ELISA.

Results: All 4 EVE’s up-regulated the production of IL-8, while only 3 of 4 EVE’s up-regulated IL-6 production. Cell viability was decreased after stimulation with all 4 EVE’s, demonstrating cytotoxic effects. Increasing the ENDS power settings with 18mg nicotine menthol flavoured EVE further enhanced cytotoxic effects.

Conclusions: The use of ENDS is likely cause an inflammation in users, through the up-regulation of IL-8, and IL-6 when using some E-liquids. Cell death from EVE exposure in vitro might translate to structural changes in vivo such as airway remodelling and emphysema. Increasing the vapourisation temperature in vitro increased toxicity and should be avoided by users.

Impact of maternal smoking on mitophagy markers in mice offspring with hypoxic-ischemic brain injury

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We have previously shown that maternal smoking during pregnancy can change mitochondrial functional markers in the offspring brain in adulthood. Maternal smoking is also a significant risk factor for hypoxic ischemic (HI) encephalopathy in newborns. Mitochondrial wellbeing is critical for cell survival. However, the effect of maternal smoking on mitochondrial damage following HI injury in offspring is not yet known. Female Balb/c mice were exposed to air (SHAM) or cigarette smoke (SE) for 6 weeks prior to mating, during pregnancy and lactation. At postnatal day (P) 10, half of the male pups in each litter underwent left carotid artery occlusion followed by hypoxia (SHAM-HI and SE-HI) and were sacrificed on P45. Proteins were measured using immunoblotting. Mitochondrial oxidative phosphorylation (OXPHOS) complexes III, IV and V were reduced in SE compared to SHAM offspring. OXPHOS complexes I, II and III were increased in SE-HI compared to SE offspring. Mitochondrial fission marker Parkin-8 was reduced but light chain (LC)3A/B-I was increased by maternal SE. HI injury reduced mitophagy markers phosphatase and tensin homolog-induced putative kinase-1, mitochondrial fission marker Optic atrophy-1, and LC3A/B-I and II protein in the SE offspring. Mitochondrial density in the cerebral cortex was reduced in SHAM-HI and SE-HI compared to SHAM and SE offspring, respectively. HI injury led to an increase in the mitochondrial fission marker while maternal smoking elicited an even higher level in the injured offspring. Our results suggest that maternal SE can worsen mitochondrial damage in offspring following HI injury.

Interaction of IGFBP-3 with NONO/SFPQ in the breast cancer response to DNA damaging chemotherapy*

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Background: Triple-negative breast cancers, lacking estrogen receptor and HER2, are typically treated with chemotherapy that induces DNA double-strand breaks (DSB). DSB damage repair allows cancer cells to become chemoresistant. Inhibitors of poly(ADP-ribose) polymerase-1 (PARP1) have been trialled to block DNA repair. We previously reported that insulin-like growth factor binding protein-3 (IGFBP-3) interacts in the nucleus of basal-like triple-negative breast cancer cells with EGFR and DNA-dependent protein kinase (DNA-PKcs) to modulate DSB repair by non-homologous end-joining (NHEJ) (Lin, Oncogene 2014). We found by proteomic discovery that Non-POU domain-containing octamer-binding protein (NONO) and its binding partner Splicing factor, proline/glutamine-rich (SFPQ) interact with IGFBP-3 in response to etoposide treatment and might be part of the IGFBP-3-EGFR-DNA-PKcs complex.

Aim: To demonstrate the significance of IGFBP-3 interactions with NONO and SFPQ in mediating DNA repair by NHEJ, and delineate the role of PARP in this process.

Methods: TNBC cell lines HCC1806 and MDA-MB-468 were treated with 20 μM etoposide ± 20 μM PARP inhibitor ABT-888 (Veliparib), and protein interactions measured by communoprecipitation or proximity ligation assay. NHEJ activity was assessed by in vitro end-joining assays.

Results: IGFBP-3-NONO and IGFBP-3-SFPQ interactions were enhanced by etoposide treatment, peaking 2 h following treatment. Both PARP inhibition and EGFR kinase inhibition blocked the formation of these complexes. DNA ending activity was reduced when IGFBP-3 was downregulated, and NONO downregulation inhibited DNA-PKcs autophosphorylation, essential for NHEJ.

Conclusions: Interaction of IGFBP-3 with NONO and SFPQ in response to chemotherapy is prevented by...
PARP inhibition, suggesting a PARP-dependent role for these proteins in IGFBP-3-dependent DSB repair. We propose that targeting the DNA repair function of IGFBP-3 may enhance chemosensitivity in basal-like triple-negative breast cancers, thus improving patient outcomes. Supported by ARC and Cancer Institute NSW.

**Instruments assessing attitudes toward or capability regarding self-management of osteoarthritis: a systematic review of measurement properties**

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**Background:** Although osteoarthritis (OA) self-management programmes may not dramatically improve pain and functional outcomes, this does not necessarily reflect a failed strategy if participants improve their self-management capabilities and live with an acceptable quality-of-life despite their disease.

**Objective:** To make a recommendation on the “best” instrument to assess attitudes toward and/or capabilities regarding self-management of OA based on available measurement property evidence.

**Methods:** Electronic searches were performed in MEDLINE (PubMed), EMBASE (OvidSP), CINAHL (Ebsco) and PsychINFO (OvidSP) (inception to 5 January 2016). Two reviewers independently rated measurement properties using the Consensus-based Standards for the selection of Health Measurement Instruments (COSMIN) 4-point scale. Best evidence synthesis was determined by considering COSMIN ratings for measurement property results and the level of evidence available for each measurement property of each instrument.

**Results:** Seven studies out of 5103 publications were included, with seven instruments identified for evaluation: Multidimensional Health Locus of Control, Perceived Behavioural Control, Patient Activation Measure, Educational Needs Assessment, Perceived Efficacy in Patient–Physician Interactions scale (PEPPI-5), The Stages of Change Questionnaire in Osteoarthritis and Effective Consumer Scale. Measurement properties assessed for these instruments included internal consistency (n=7), structural validity (n=7), test-retest reliability (n=2), hypothesis testing (n=2) and cross-cultural validity (n=2). No information was available for measurement error, content validity, responsiveness or minimal important change/difference. The Dutch PEPPI-5 demonstrated the best measurement property evidence; strong evidence for internal consistency and structural validity but limited evidence for reliability and construct validity.

**Conclusion:** Although PEPPI-5 was identified as having the best measurement properties, overall there is a poor level of evidence currently available concerning measurement properties of instruments to assess attitudes toward and/or capabilities regarding OA self-management. Further well-designed studies investigating measurement properties of existing instruments are required.

**Investigation of the association between blood glucose levels, anxiety and depression**


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**Background & Objectives:** Mental illness leads all non-communicable diseases in loss of productivity and occupational function. In Australia, one in five people will be affected by a mental disorder, with depression and anxiety being the most common. Furthermore, glucose metabolic dysfunction is associated with a variety of physiological complications, including diabetes mellitus (DM), however knowledge regarding its influence in psychiatric illnesses remains unknown. This research sought to investigate the relationship between blood glucose levels (BGLs) and anxiety and depression. It was hypothesised that an increase in BGL would be linked to increased anxiety and depression symptomology.

**Methods:** Healthy participants (n=31) and participants with DM (n=15) were recruited from the general population (n=46 in total). Blood glucose concentration was determined in the fasting and postprandial states using a glucometer and lancet device. A dietary diary was administrated to calculate the nutritional intake of each subject. The Lifestyle Appraisal Questionnaire (LAQ) assessed both lifestyle risk factors and stress perception, whilst the Beck Depression Inventory (BDI), Beck Anxiety Inventory and Depression-Anxiety-Stress-Scale (DASS) were administered to assess anxiety and depression. Data was analysed to identify any correlations between BGLs, anxiety and depression.

**Results & Conclusion:** A negative relationship was found between fasting BGL, DASS-anxiety (p=0.07) and BDI (p<0.05) within the DM sample population. Although these findings did not support the hypotheses of this study, they do suggest the need for further research into the potential relationship between metabolic dysfunction and mental illness prevalence in Australia.

**Microvesicle Cargo: Biomarkers for the Future**


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Cell-derived microvesicles (MVs) play a key role in intercellular communication and regulation of biological processes. Once regarded as cellular ‘trash’ by multiple research groups, the utilisation of these particles and their bioactive cargo in clinical studies has generated worldwide

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popularin both as a biomarker and as a mediator of infection, cancer metastasis and other biological processes. Proteomic studies of the MVs cargo have identified over 100 proteins including ion channel proteins which may provide a clue for MV function and the origin of their healthy or diseased cell parent.

The heightened interest surrounding MVs and their subtypes Exosomes (EXs)(50-100nm) and Microparticles (MPs) (0.1-1.0um) has also encouraged the development of scientific communication and visualisation as a form of reinforcement, education and increasing public awareness towards research. By aiming to condense quantitative and qualitative data, scientific visualisation and data communication can excel in translating key discoveries for the public eye.

The present study amalgamates elements of research and design to develop a highly accurate account of the structure, function and interactions of MVs and their bioactive cargo with cell membranes. The outcome of this work will be the production of a short biomedical animation detailing the processes and importance of cell-derived MVs and their cellular interactions.

**Associations between heart rate variability and blood glucose levels**

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**Background:** Diabetes mellitus (DM) is the fastest growing chronic condition in Australia. The most effective means of managing the adverse complications of DM is stringent glycaemic control. Current glucose-monitoring devices and procedures are invasive; as such, the development of non-invasive, continuous measures of blood glucose levels (BGLs) is the focus of many studies. Non-invasive heart rate variability (HRV) measures have the potential to assess autonomic function in response to glucose fluctuations. However, there is limited evidence on associations between HRV measures and glucose variability.

**Aim:** To investigate the relationship between HRV and BGLs.

**Methods:** Twenty-five healthy participants (44% male; 27.56 ± 9.26 years of age) uninhibited by chronic illness or daily medications were recruited for this exploratory study. BGLs were assessed by glucometer and lancet device in a fasting state (no caloric intake for eight hours), as well as three-hours post-fasting and six-hours post-fasting. Following each glucose assessment, a ten-minute electrocardiogram (ECG) was recorded for the determination of HRV parameters. Participants recorded all food or drinks consumed over this period, as such, kilojoule intake was derived and applied as a covariate in the analysis.

**Conclusion:** Fasting low frequency (LF) HRV was correlated with the variability between the first (fasting) and second (three-hour post-fasting) BGL readings, the second BGL reading, and the variability between second and third (six-hour post-fasting) BGL readings. LF power and total HRV power (TP) in the second session were correlated positively with the variability between the second and third BGL readings. LF power and TP in the second session were negatively associated with the second BGL reading. These findings suggest that HRV may be useful in the prediction of the magnitude and direction of BGL changes.

**Mechanisms of erlotinib resistance in vitro by latest-generation mass spectrometry**

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Recent developments in molecular-targeted therapies have transformed the way lung cancers are now treated. One such therapy, erlotinib, a tyrosine kinase inhibitor has shown dramatic and prolonged response in a specific subset of non-small-cell lung cancer (NSCLC) patients with activating EGFR mutations. However, the development of treatment resistance is inevitable. The aim of this study was to identify proteins associated with the development of erlotinib resistance in NSCLC in vitro.

Erlotinib-sensitive HCC827 cells were treated with repeated cycles of erlotinib to generate an erlotinib-resistant subline, HCC827er. To identify proteins associated with erlotinib resistance, each cell line was profiled using Sequential Windowed Acquisition of All Theoretical Fragment Ion Mass Spectra (SWATH-MS, Sciex 6600 TripleTOF mass spectrometer). A total of 3,954 proteins were included in the spectral library, with information extracted for 3,416 proteins in SWATH-MS analysis (at peptide confidence >99%). Of these, 77 proteins were differentially expressed between HCC827 and HCC827er (Fold Change >2, p<0.05). Several of these proteins are already known to be associated with drug resistance (e.g. MET), while others may be involved in novel mechanisms of resistance.

Pathway Analysis identified “Cellular Movement” as the leading Molecular and Cellular Function in the HCC827er cells. This was validated in vitro showing an increased number of HCC827er cells migrating through transwells compared to the sensitive counterpart. Such migratory capacity may provide insight into the metastatic propensity of erlotinib resistant- NSCLC.

In an era of personalised treatment, the development of resistance presents as the next challenge in targeted therapy. Here, we have produced the first high-resolution protein profile for NSCLC cell lines to aid identification and characterisation of mechanisms of resistance and ultimately improve patient outcomes.

**Targeting an IGF1R-3 signalling pathway for the treatment of triple-negative breast cancers**

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**Background:** Triple-negative breast cancers (TNBCs) are aggressive with few treatment options. TNBCs typically express the epidermal growth factor receptor (EGFR) and insulin-like growth factor binding-protein-3
(IGFBP-3), which potentiates growth-stimulatory signalling through the EGFR by activating sphingosine kinase-1 (SphK1). We hypothesised that, because IGFBP-3 and EGFR are co-expressed in TNBC, combined inhibition of their signalling pathways may have potential as a novel treatment for TNBC.

**Aims:** To investigate in vitro and in vivo the potential therapeutic efficacy of combined EGFR and IGFBP-3/SphK1 inhibition in TNBC.

**Methods:** The effects of gefitinib (EGFR inhibitor) and FTY720 (SphK1 inhibitor) on cell proliferation were assessed using real-time imaging. For in vivo studies, nu/nu mice bearing orthotopic TNBC tumour xenografts were treated with gefitinib (25 mg/kg), FTY720 (5 mg/kg) or both agents, and sacrificed when tumours reached ~1,000 mm³.

**Results:** Among cell lines representing the six subtypes of TNBC, EGFR and IGFBP-3 were highly expressed in basal-like TNBC cells, the most common subtype. The combination of gefitinib and FTY720 abolished cell proliferation when the drugs were used at concentrations that had minimal effect when used alone, and markedly sensitised TNBC cells to the growth inhibitory/apoptotic effects of chemotherapeutic agents doxorubicin and etoposide. In vivo, gefitinib plus FTY720 significantly improved survival of mice bearing basal-like MDA-MB-468 or HCC1806 cell xenograft tumours, compared with those receiving single agents.

**Conclusions:** This pre-clinical evaluation suggests that combined inhibition of EGFR and IGFBP-3 signalling through SphK1 using agents already approved for clinical use may be of value as a novel treatment for TNBC. Supported by Cancer Council NSW.

**The role of TRAIL in regulating insulin expression and function**

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**Background:** Type 2 diabetes (T2D) is characterised by insulin resistance, β-cell dysfunction, and inadequate insulin production. Recently, TNF-related apoptosis-inducing ligand (TRAIL) was implicated in protecting against T2D. Whether TRAIL regulates glucose/insulin homeostasis is unknown.

**Aim:** To investigate the effect of TRAIL on insulin expression and β-cell function, and insulin signalling pathways.

**Method:** Min6 cells were transfected with an insulin promoter-reporter followed by addition of human TRAIL (hTRAIL) for 24 h. Luciferase activity was assessed. mRNA expression of the murine insulin genes, Ins1 and Ins2 in response to hTRAIL were measured. Proliferation and apoptosis assays were performed. C57Bl/6J mice were administered hTRAIL (2 μg/mouse) by i.p. and mice were euthanased 30 min and 24 h later. Pancreata were collected for histology and assessment of insulin expression islet size. Skeletal muscle was collected for gene expression studies.

**Results:** Min6 cell proliferation was increased in response to hTRAIL, associating with augmented insulin transcription and mRNA expression. An increase in islet of Langerhans area, is considered a marker of β-cell proliferation in vivo. hTRAIL significantly increased islet area 30 min and 24 h after TRAIL administration in mice. Consistent with our in vitro data, hTRAIL also stimulated insulin protein expression in pancreatic islets, without affecting plasma insulin. Importantly, ERK was phosphorylated in response to hTRAIL in skeletal muscle.

**Conclusion:** This is the first demonstration implicating TRAIL in regulating insulin expression and β-cell function, and TRAIL stimulating insulin signalling pathways in peripheral tissues. As β-cell function and insulin signalling is impaired in T2D, these results highlight a potential role for TRAIL as a therapeutic in this metabolic disease.

**Confirming T-cell Responses in Intrauterine Growth Restriction**

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**Background:** Intrauterine growth restriction (IUGR) is the second leading cause of perinatal mortality in humans and is associated with many long term health consequences. Thus it is important to use a robust animal model that replicates an IUGR phenotype to develop new therapies.

**Aims:** To quantify the maternal peripheral blood T-cell profile and fetal genomic sex in the CBA/CaH x DBA/2J model of IUGR and fetal mortality.

**Methods:** Twelve-week old female CBA/CaH mice were mated with male CBA/CaH (intra-strain control) Balb/c (inter-strain control) and DBA/2J mice (IUGR prone model) or unmated (non-pregnant control). Peripheral blood mononuclear cells (PBMCs) were isolated at term and analysed for Th1, Th2, Th17 and Treg by flow cytometry. Fetal sex was determined by PCR for X-chromosome Xir and Y-chromosome Sly.

**Results:** CBA/CaH x DBA/2J pregnancies showed a 3-fold greater prevalence of IUGR (p<0.01) and increased fetal mortality (p<0.05), when compared to the CBA/CaH x Balb/c pregnancies. Maternal T-cell production of IFNγ, IL-4, IL-17 and TGFβ was higher in pregnant vs non-pregnant mice (p<0.05). Unexpectedly, maternal Th1 cells were decreased in CBA/CaH x DBA/2J pregnancies when compared to CBA/CaH x Balb/c (p<0.05). Significantly more non-viable fetuses were female at mid-gestation and term however, no gender bias was observed for IUGR fetuses.

**Conclusion:** The CBA/CaH x DBA/2J model is a robust model of IUGR and fetal mortality however, further studies will be required to determine the maternal T-cell contribution and gender bias of IUGR and mortality to endeavour new therapies.

**Knowledge gaps on hypertension among patients from a rural Indonesian community**

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**Background:** Lack of knowledge is an important barrier to hypertension awareness and treatment.
Aims: This study aimed to report patients’ basic knowledge on hypertension and the predicting factors.

Methods: The study was conducted in eight rural villages in the Bantul district of Yogyakarta province, Indonesia, from August to November 2015. Hypertension knowledge was assessed using a 10-item questionnaire (one point for each correct answer). Participants with a total score ≥8 were considered to have good knowledge. Logistic regression was used to identify the predictors of hypertension knowledge.

Results: Of the 384 participants, 75% were female and 36% had completed elementary school. Overall, 59 (15%) participants had good hypertension knowledge. Items where less than 50% of participants responded correctly were: ‘High blood pressure usually lasts for the rest of life’ (16%), ‘High blood pressure is associated with kidney problems’ (19%), ‘People with high blood pressure should take their medicines every day’ (29%), ‘High blood pressure can cause heart attacks’ (38%) and ‘Losing weight usually makes blood pressure go down’ (38%). Most participants (62%) responded that anti-hypertensive medicines should be taken only when they feel sick. Participants with good hypertension knowledge had a higher formal education (OR = 2.25, CI = 1.24–4.09) and lived closer to a community health centre (OR = 1.82, CI = 1.01–3.29) compared to those with poor knowledge.

Conclusion: The knowledge gaps pertinent to hypertension in the rural population of Indonesia require customised educational programs aimed at addressing the information needs, especially for patients with low levels of education and those who live far away from public health care services.

The role of LncRNA in Diabetic Kidney Disease

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Background: Diabetic kidney disease (DKD) is one of the major complications of diabetes. Current therapies are insufficient to halt the progression to kidney failure. Recent studies have shown that long non-coding RNA (LncRNA) play a role in DKD and are involved in epigenetic regulation.

Aim: To identify/validate LncRNAs in the diabetic animal kidney and to assess the functional relevance in human kidney proximal tubular cells (HK2) exposed to high glucose (HG) and transforming growth factor beta (TGFb1).

Method: RNA sequencing and bioinformatics (AGRF) were employed to identify differentially expressed LncRNA from 3 groups of C57Bl/6 mice (n=3): control, Type 1 (streptozotocin induced) and Type 2 DKD (high fat fed and obese). Only LncRNA that were common in both diabetic groups were analysed. This approach was to narrow down the LncRNA relevant to kidney disease rather than other metabolic factors. HK2 cells exposed to HG and TGFb1 were used to assess regulation to diabetic stimuli in vitro.

Results: RNA sequencing identified four novel LncRNAs that were upregulated (Log FC >1, P value< 0.05) in both models of diabetic mice. Real-time PCR confirmed only one of those (LncRNA #1) to be consistently expressed across human and mice kidney hence this was analysed further. In HK2 cells, LncRNA#1 was upregulated by TGFb1 at (P=0.05) but to a lesser extent with high glucose (p=0.06) at 48h.

Conclusion: LncRNA#1 is upregulated in both types of DKD. In HK2 cells, LncRNA#1 was upregulated by TGFb1 and less significantly by HG. We are currently using a sequence specific LNA GapmerR (Exiqon) in HK2 cells to assess the functional relevance of this target.

Emotion Regulation and Stress: physiological assessment

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Background: The ability of an individual to employ emotion regulation strategies to cope with a stressful situation can impact on the nature of physiological stress response. Limited research exists in the area of autonomic physiological stress and emotion regulation even though there are clear associations of emotion dysregulation in psychopathology. The aim of this study was to determine associations between emotional regulation, perceived stress and the autonomic nervous system. It was hypothesised that increases in maladaptive emotion regulation and perceived stress would be linked to increases in autonomic physiology.

Methods: A total of 40 participants between the ages of 19 and 53 were recruited. Skin conductance measure was acquired to derive autonomic activity measure to identify links with demographic and psychometric variables, measured using self-report questionnaires.

Results and Conclusions: This study offers evidence that use of reappraisal, as an adaptive technique, may lead to decreases in skin conductance (r=-0.332, p=0.037). Although maladaptive techniques demonstrated no associations, adaptive strategies are thought to reduce maladaptive strategies. Maladaptive emotion regulation techniques were associated with psychopathology and increases in perceived stress. A significant correlation between increases in perceived stress and an increase in the change in skin conductance was recognised, demonstrating a physiological stress response (r=0.327, p=0.040). Only the group of subjects who had experienced a traumatic event, demonstrated associations between skin conductance and psychometric measures suggesting that autonomic arousal is implicated in trauma and post-traumatic stress disorder. The results of this study suggest emotion regulation may impact on the development of a physiological stress response and psychopathology. To further this study, emotion regulation and physiology should be explored in clinical and non-clinical populations.

Quantitative analysis of the effects of microparticles and parasitised red blood cells on the endothelium using an in vitro model of human cerebral malaria

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Malaria is one of the world’s most prevalent infectious diseases infecting millions and killing hundreds of thousands people each year. Cerebral malaria (CM) is one of the most severe complications of malaria. Recently, microparticles (MPs) and other subcellular
vesicles have been proposed as key player in CM pathogenesis. We hypothesised that *P. falciparum* parasitised red blood cells (PRBCs) would adversely affect the integrity of the endothelial cell monolayer leading to disruptions and alterations in the cell junction proteins and adhesion receptors and that MP interactions with human endothelial brain cells would modulate the effects of PRBCs. We used an *in vitro* model of co-culture between brain endothelial cells, RBC, PRBC and MPs. The effects that PRBCs, PRBC-MPs and PRBCs + PRBC-MPs (PRBC-Mix) have on human brain endothelial cell (HBEC) junction molecules (ZO-1 and VE-cadherin), adhesion receptors (ICAM-1 and VCAM-1) and monolayer integrity were assessed by combining immunofluorescence, flow cytometry and permeability assays. Using quantitative image analysis, we observed significant decreases in VE-cadherin expression after HBEC co- incubation with PRBCs and PRBC-Mix. Furthermore, the image analysis, also showed that PRBC-MPs were internalised by HBEC at twice the rate of nRBC-MPs. Moreover, the expression of eCAMs, ICAM-1 and VCAM-1 in HBEC was significantly increased in the presence of PRBCs and during the co-incubation with PRBC-Mix. Finally, the permeability assays showed significant increases in the permeability of the endothelial cell monolayer when PRBC were present but only a trend towards an increase when co- incubated with the PRBC-Mix.

Our results show that PRBCs do affect the physiology and overall function of human brain endothelial cells. These results support our hypothesis that MPs are able to modulate cellular responses triggered by PRBCs.

**Improving care standards for patients with traumatic spinal cord injury - a modified e-Delphi survey.**

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**Introduction:** Defining ‘agreed practice standards’ is one of the first and most crucial steps in the translation of knowledge into policy and practice, enabling identification of current evidence-practice gaps and facilitating implementation of an improved model of care. This study aimed to develop consensus and agreed standards of care for patients with acute TSCI and identify barriers to achieving this.

**Methods:** A rapid literature review identified 'best practice' evidence across specific clinical practice areas in early TSCI care (2005 – 2015). A modified e-Delphi survey process was used to build consensus across pre-hospital care, spinal immobilisation, imaging, haemodynamic management, time to surgery and referral pathways and processes.

**Results:** Survey respondents were experienced practitioners from Emergency Medicine (33%), Trauma Medical (16%), Surgical (15%), Nursing (11%) and Paramedic (12%); 66% using locally written protocols for acute TSCI care. There was >80% consensus regarding: pre-hospital diagnosis of isolated TSCI and transfer decisions, the transfer of patients with TSCI to a specialist service within 24 hours, specific preparation for patient transfer and imaging requirements for diagnosis/clearance. Induced hypertension with related invasive monitoring was not agreed by the majority, neither the location of necessary decompressive surgery or closed reduction in the first 12-24 hours.

**Conclusions:** These findings will be explored within individual stakeholder interviews to further understand specific drivers (barrier/facilitators) of achieving agreed standards. Clinical variation audit will measure evidence-practice gaps, finding target areas amenable to change. These findings will inform national discussion toward consistent approaches to triage, treatment, transport and definitive care for TSCI.

**Using proteomic analysis to uncover the mechanisms of toxicity of non-protein amino acids implicated in neurological diseases**

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**Background:** Neurodegenerative diseases such as Parkinson’s disease and motor neurone disease are characterised by protein misfolding and deposition of protein aggregates in the nervous system. In 90% of cases these diseases are sporadic with no known cause. It is essential therefore that we identify the environmental factors involved. Non-protein amino acids (NPAs) present in the environment have been implicated as causative factors in neurological disorders since they can be misincorporated into proteins in place of protein amino acids modifying native protein structure and generating misfolded aggregate-prone proteins.

**Methods:** We use quantitative proteomic approaches to examine the expression of proteins in human neuroblastoma cells (SH-SY5Y) after exposure to NPAs to determine the mechanisms underlying their toxicity.

**Results:** Quantitative TMT labeling of proteins from human neuroblastoma cells treated with BMAA, a NPA produced by cyanobacteria (blue-green algae), generated a comprehensive data set of differentially expressed proteins. Analysis revealed a proteotoxic stress response consistent with protein misfolding as well as changes to many pathways known to be involved in neurodegenerative diseases. Incorporation of certain NPAs into proteins also resulted in a loss of solubility of specific proteins such as histone H4. In order to detect NPAs in proteins with a greater sensitivity and specificity we developed a new method that allows NPAs to be detected in peptides using mass spectrometry. This novel approach overcomes many limitations associated with protein hydrolysis which is currently the only method available.

**Conclusions:** The data presented supports the hypothesis that NPAs can be misincorporated into proteins, cause protein misfolding and can impact on pathways implicated in neurodegenerative diseases.

**Effect of maternal cigarette smoke exposure on the lung**
expression of RAGE-mediated signaling pathway in male mice offspring

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Maternal smoking during pregnancy contributes to long-term health problems in adulthood, especially respiratory disorders. Receptors for advanced glycation end-products (RAGE) are multi-ligand receptors abundantly localized in the lung. Several studies have suggested a role of RAGE in cigarette smoking-related disease that RAGE signalling is a key regulator of inflammation in pulmonary diseases. Cigarette smoke induces the formation of advanced glycation end-products (AGE)s resulting in the development of diseases through AGES-RAGE axis. The ligation of RAGE mediates the generation of reactive oxygen species and consequent activation of the pro-inflammatory signaling. This study aims to investigate the effect of maternal cigarette smoke exposure on RAGE expression, as well as RAGE-mediated signaling pathway in the lung of the offspring. Female Balb/c mice (8 wk) were divided into sham group (exposed to air) and SE group (exposed to cigarette smoke before mating and throughout gestation and lactation). Protein expression in lung from male offspring at 13 weeks was measured by Western blotting. RAGE and its downstream signaling including NF-κB and MAPK family consists of ERK1, ERK2, and JNK were significantly increased in the lung from the SE offspring, however, not their phosphorylated forms. In the SE offspring, Nrf-2, a transcription factor which is high sensitivity to oxidative stress was significantly increased. Furthermore, mitochondrial antioxidant manganese superoxide dismutase (MnSOD) was reduced in the SE offspring. Our findings reveal that maternal cigarette smoke exposure enhanced RAGE expression and promoted the RAGE-mediated mechanisms associated with oxidative stress and inflammation in offspring lung.

Characterisation of a Diet induced intra-hepatic immune foci representing a model of Nonalcoholic Steatohepatitis

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Introduction: Non-alcoholic fatty liver disease (NAFLD) is the commonest liver disease and affects one third of the adult population in affluent societies. In about 20% of cases, NAFLD progresses to chronic liver inflammation defined as non-alcoholic steatohepatitis (NASH). High dietary intake of cholesterol is a risk factor for NASH.

Objectives: The underlying role of immune response in diet promoted NASH is poorly understood to date. The aim of this study was to investigate whether and how an atherogenic (Ath) diet alters the hepatic immune signature responsible for inflammation and induces the pathology of NASH.

Methods: Male C57BL6 mice fed an atherogenic diet containing 2% cholesterol, 0.5% cholate and 33% sucrose to induce liver inflammation or received normal chow for 12 weeks. The phenotype of immune response within the liver and spleen was examined using immunoflowcytometry, nCounter nanostring gene expression and immunostaining.

Results: The Ath diet induced histological features of NASH including higher liver/total body weight ratio and an increase in the serum ALT levels (both p<0.05). H&E staining showed the infiltration of immune cells in the liver, and inflammatory foci formation. Nanostring mRNA expression analysis demonstrated Ath diet significantly upregulated the hepatic expression levels of F4/80, CD11b, CD11c, CD3 and B220 (all P value<0.001). Immunophenotyping using flowcytometry revealed a trend towards an increased level of inflammatory macrophages, monocytes and dendritic cells in Ath group.

Conclusion: The hepatic inflammation in an Ath diet model of NASH includes both innate and adaptive immune response. This model suggests the critical role of cholesterol in altering the hepatic immune signature leading to NASH.

Risk Factors for Giardiasis in South Western Sydney: A Case-Control Study

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Background: Giardiasis is a common cause of gastrointestinal illness notifiable in New South Wales. However, risk factors for giardiasis in Australia remain unclear and there is limited information about local sources of exposure to inform prevention strategies. Giardiasis cases are not routinely investigated in NSW.

Aims: This study seeks to describe the epidemiology of giardiasis in south western Sydney; and identify modifiable risk factors for giardiasis in south western Sydney.

Methods: A retrospective analysis of giardiasis notifications for January 2011 to December 2016 will be conducted. A case-control study of giardiasis cases notified from January to December 2016 will investigate the risk factors for giardiasis.

Results: From 2011 to 2015, 779 cases (mean 156 cases per annum) giardiasis were notified to south western Sydney Public Health Unit. At 30 June 2016, notifications have exceeded the annual average with 173 cases notified. A significant increase in notifications was observed in February 2015 particularly from Camden LGA, suggestive of an outbreak; however, giardiasis cases were not investigated. Preliminary geo-spatial analysis of surveillance data for January 2011 to December 2015 has identified significant clustering of giardiasis cases outside of urban areas in the LHD. Geospatial analysis may be useful to further explore environmental risk factors driving giardiasis cases in SWSLHD. Hypothesis around potential risk factors for giardiasis such as water supply, onsite septic and exposure to wildlife should be investigated using geo-spatial tools.

Conclusion: Further investigations are needed to identify potential risk factors for giardiasis to inform enhanced surveillance and control strategies.
Members of the Chloride Intracellular Ion Channel Protein Family Demonstrate Cell Protective Effect Against Oxidative Stress

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The chloride intracellular ion channel (CLICs) proteins are atypical anion selective channel proteins, with some members also known to have enzymatic activity. Structural studies demonstrate that the CLIC proteins share strong structural homology with members of the glutathione-S-transferase superfamily of enzymes, in particular the omega glutathione-S-transferase (GST-Ω) members. Human CLIC proteins are found in most tissues and cells, where they localise onto intracellular membranes of various intracellular organelles, as well as being found as soluble proteins within the cell cytosol. Six CLIC members exist in humans: CLIC1-6, with all members containing a common 240 amino acid residue cassette.

Our group has recently shown via an in vitro HEDS assay system, that members of the CLIC family have intrinsic enzymatic activity in addition to their known ion channel function. The current study aimed to investigate whether expression of CLIC proteins by bacterial cells could provide increased tolerance to oxidative stress. Recombinant CLIC proteins were expressed in bacterial E. coli cells followed by their exposure to the oxidising agent H2O2. Expression of the proteins CLIC1, 3 or 4 by the E. coli cells was found to provide increased tolerance of up to 5mM H2O2. This was assessed using spectrophotometric absorbance measurements of cell growth density at 600nm over time. This work for the first time assigns that the soluble form of the CLIC proteins 1, 3 and 4 can function as oxidoreductase enzymes within a living cell and can serve to protect cells against oxidative damage and circumvent oxidative stress.

Placental Exosomes: A New Mechanism to Improve Intrauterine Growth Restriction

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Introduction: IUGR is a leading cause of perinatal death with no preventative or therapeutic strategies. The mechanisms underlying the pathology of IUGR remains unknown. Recent evidence shows that IUGR is strongly associated with a pro-inflammatory Th1/Th17 T-cell phenotype. Our group has shown that NF-xB p65 is a critical regulator in controlling Th1/Th17 immune responses, and is degraded during normal pregnancy. We hypothesise that the mechanisms underlying the degradation of p65 during pregnancy are mediated by placentally derived exosomes through Fas/Fasl signalling.

Aims: To determine whether p65 suppression during normal pregnancy, is mediated via Fas signalling and that p65 expression and Fas/Fasl signalling is aberrant during UIGR.

Methods and Results: Fas activation of Jurkat T-cells resulted in suppressed p65 expression determined by western analysis. In addition, p65 expression was reduced in T-cells from pregnant women, compared to UIGR women, as determined by flow cytometry. Moreover, exosomes isolated by ultracentrifugation from normal maternal plasma induced p65 suppression in T-cells of non-pregnant women, whereas exosomes from UIGR plasma, was less capable of reducing p65. Finally, Fasl expression was reduced in UIGR placenta compared to normal placenta detected by immunohistochemistry and western blot.

Discussion: Our data suggests that exosomes derived from the placenta have the ability to regulate p65 expression via Fas signalling in T-cells, and this is necessary for pregnancy success. The reduced FasL expression and inability of exosomes to reduce p65 in T-cells during UIGR, may explain the increased Th1/Th17 inflammatory environment associated with the pathology of this pregnancy complication.

A Telephone-Based Version Of The Spinal Cord Injury–Secondary Conditions Scale: A Reliability And Validity Study

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Background and Aim: The Spinal Cord Injury Secondary Conditions Scale (SCI-SCS) was first published in 2007 and is an adaptation of the generic Seekins Secondary Conditions Questionnaire for people with injury related disabilities. The scale to date has received little attention although it may provide a useful way of screening patients over the telephone for complications. The purpose of this study was to determine the reliability and validity of the telephone-based version of the SCI-SCS.

Methods: Forty people with SCI were recruited from the Royal North Shore Hospital, Australia. Inter-rater reliability was tested by comparing the telephone-based version of the SCI–SCS administered on two different days by two different telephone assessors. Validity was tested by comparing the telephone-based version of the SCI–SCS with the paper-based version of the SCI–SCS.

Results: The intraclass correlation coefficient (95% CI) reflecting the agreement between the telephone-based and paper-based versions of the SCI–SCS was found to be 0.85 (0.76–0.91), indicating excellent reliability. The validity of the telephone-based version was also found to be good, with a Pearson correlation coefficient of 0.79 (0.64–0.89) between the telephone-based and paper-based versions. This suggests that the telephone-based version of the SCI–SCS is a reliable and valid measure of secondary conditions.
version of the SCI–SCS administered on two different days by two different assessors was 0.96 (0.93–0.98). The corresponding value reflecting agreement between the telephone-based assessment and the paper-based assessment was 0.90 (0.83–0.95).

**Conclusion:** The telephone-based version of the SCI–SCS is a simple and a quick questionnaire to administer that has both inter-rater reliability and validity. It may be a useful way to screen patients for complications especially in low and middle-income countries where patients cannot always be regularly followed up at face-to-face clinics.

### The Reliability Of Measuring Wound Undermining In People With Spinal Cord Injury

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**Background and Aim:** An important feature of a pressure ulcer is the extent to which it is undermined. However, little attention has been directed at the measurement of undermining. The purpose of this study was to determine the reliability of measuring wound undermining in people with SCI.

**Methods:** Thirty people with SCI and a pressure ulcer with wound undermining were recruited from Indian Spinal Injuries Centre, India. Wound undermining was measured using the four cardinal points from a clock face namely 12 O’clock, 3 O’clock, 6 O’clock and 9 O’clock. Inter-rater reliability was tested by comparing the wound undermining scores from two different assessors. Intra-rater reliability was tested by comparing the wound undermining scores from the same assessor on two different days.

**Results:** The median (IQR) extent of wound undermining was 3.2 cm (1.0 to 7.1). The intraclass correlation coefficients (95% CI) for inter-rater and intra-rater reliability were 0.996 (0.992 to 0.999) and 0.998 (0.996 to 0.999), respectively. Repeat measurements by the same and different assessor were within 0.3 cm of each other, 80% and 83% of the time, respectively.

**Conclusion:** Wound undermining can be reliably measured and could be more widely incorporated into clinical practice to reflect the real size of a pressure ulcer.

### Health Literacy and its Impact on Social and Health Outcomes in a Motor Vehicle Crash Injury Population

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**Background:** Motor Vehicle Crash (MVC) injury is a significant problem in Australia. Health Literacy (HL) may influence recovery and rehabilitation following crash events. HL is complex and requires knowledge, motivation and capacity to access, understand, appraise and apply information to make decisions about health and healthcare. It’s a rapidly expanding area of research and has been shown to mediate recovery and lead to improved outcomes across various health domains.

**Aims:** To determine if HL is a predictor of recovery after MVC and to assess whether HL changes over time after injury.

**Methods & Results:** This longitudinal cohort study, observes adult outcomes resulting from MVC injury across NSW. Recruitment is being conducted via major NSW hospital emergency departments capturing diversity across the region. Emergency attendees with non-catastrophic injuries are eligible for the study. Those with severe traumatic brain injury and spinal cord injury are excluded. Participants are contacted within 28 days of a crash, and subsequently followed up at 6, 12 and 24 months respectively. The Health Literacy Questionnaire tool is being administered via telephone to participants at these time points. Cross-sectional reporting will describe the differences in HL between demographic sub-groups. The study is prospective, therefore it’s expected that data on longer-term outcomes will become available for analysis in future studies.

**Conclusion:** HL may aid recovery from MVC injury and could be a mechanism in achieving equitable outcomes across different population groups. Establishing HL as a recovery predictor may reveal differences in two key areas, cognition and engagement. This should provide deeper insight into the relationship between HL and the social and environmental positions of participants. This may influence improved health solutions and interventions for participants using health services following an MVC event.

### Does Modern Acupuncture Address The Clinical Features Of The Original Acupuncture Theory?

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**Background:** The seminal acupuncture text The Systematic Classic of Acupuncture and Moxibustion 282AD (SCoAaM) details the scope of clinical acupuncture. These signs and symptoms appear as differential diagnostic patterns grouped into 14 channels that encompass different body regions. Acupoints are particularly sensitive channel areas which influences physiological change. However, epidemiological changes over two millennia may have changed the focus of clinical acupuncture due to changes in disease prevalence, technology and treatment modalities.

**Aims:** To compare the original clinical diagnostic features presented in SCoAaM with those reported in modern clinical and experimental acupuncture research studies.

**Methods:** An extensive search of all human clinical and experimental acupuncture studies published between 1995 - 2016 was conducted. For each acupoint, the main clinical focus of relevant studies was compared with those for the original SCoAaM diagnosis. Results in this presentation are restricted to the six channels on the arm.

**Results:** For all channels, there have been both clinical and experimental studies. However, the number of studies was limited, with only a minority judged as methodologically sound. Clinical focus varied between channels but had
minimal relationships to those from SCoAaM. Many of the signs and symptoms from SCoAaM dealt with serious infection – febrile conditions, pneumonia, tuberculosis and tetanus which are all treated effectively with antibiotics or vaccinations. One important difference concerns cardiac indications, theses were additions that could not have occurred prior to technological advances from the last century. Also, the studies showed an increase focus on fMRI studies with growing awareness of acupuncture effects involving the nervous, endocrine and lymphatic systems.

**Conclusion:** There were identified significant changes in the clinical focus of acupuncture reflected in changes in control of disease prevalence and severity and related treatment modalities and technological advances.

**Determinants of the geospatial distribution of giardiasis in New South Wales, Australia**

Stephanie FLETCHER-LARTEY, Soumya Mazumdar, Patricia Zajaczkowski, John Ellis

**Background:** Giardiasis is a common cause of gastrointestinal illness notifiable in New South Wales. However, little is known about the distribution of risk factors for giardiasis in Australia. Although giardiasis is routinely notified by laboratories to the disease surveillance system, cases are not routinely investigated in NSW.

**Aims:** This study seeks to identify the determinants of geospatial distribution of giardiasis in New South Wales, Australia.

**Methods:** Confirmed (de-identified) giardiasis cases notified to the Notifiable Conditions Information Management System in NSW for January 2011 to July 2016 will be analysed. Spatial cluster analyses will be implemented to delineate areas with high rates of giardiasis. Hypothesis around potentially modifiable risk factors for giardiasis such as use of tank water, well water supply, onsite septic and exposure to wildlife will be explored using geo-spatial tools.

**Results:** The results will show the distribution of giardiasis cases based on Local Health District; the association between giardiasis notifications and certain environmental risk factors including but not limited to municipal water supply, use of rain harvested/ tank water, bore wells, and onsite septic systems in addition to individual variables such as age, gender and socio-economic status of cases.

**Conclusion:** The application of advanced geospatial analysis to the investigation of confirmed cases of giardiasis in New South Wales will improve understanding of the epidemiology and geographical distribution of this parasitic disease. This is important to assist with identifying modifiable risk factors not detected through routine surveillance measures in order to inform disease prevention and control strategies at the State and Health District levels.

**Acquired Chemotherapy Resistance In Vitro: MiRNA Profiles Of Chemotherapy Resistant Squamous Lung Cancer Cell Lines**

Simon A. HAEFLIGER, Amanda L. Hudson, Sarah A. Hayes, Nick Pavlikis, Viive M. Howell

**Background:** Lung cancer is the leading cause of cancer death worldwide. 25% of lung cancers are histologically squamous cell carcinomas (SCC). Cytotoxic chemotherapies are currently the mainstay of treatment. However, patients with lung SCC inevitably acquire chemotherapy resistance. This results in poor overall survival of advanced stage lung SCC of only 9 to 11 months. Repetitive exposure of lung cancer cell lines to chemotherapeutic drugs enables investigation of molecular mechanisms of acquired chemotherapy resistance in vitro. We are studying the role of miRNAs in this process.

**Methods:** We induced chemotherapy resistance in lung SCC cell lines LUDLU-1, Calu-1, SK-MES-1 in vitro by repetitive drug treatment. Agents used to develop resistance included Cisplatin, Gemcitabine, Paclitaxel and Vinorelbine. Cell viability after 3 or 5 days of chemotherapy treatment was measured by MTT assay and drug dose causing a 50% growth inhibition (IC50) was calculated. Expression of 754 miRNA was measured by TaqMan OpenArray MicroRNA array.

**Results:** After 15-25 cycles of chemotherapy lung SCC resistant cells showed a statistically significant increase in IC50 values (n-fold): Cisplatin 4.9 - 12.4; Gemcitabine 12.6 - absolute resistance; Paclitaxel 30.9 - 110.1; Vinorelbine 4.8 - 19.3. MiRNA expression of resistant cells was compared to parental, drug sensitive cells and is illustrated by heatmaps. Analysis of expression patterns revealed upregulation and downregulation of specific miRNAs in drug resistant cells. We are currently investigating the function of these dysregulated miRNAs in promoting chemotherapy resistance. Further, we are testing if certain miRNA are suitable targets to improve chemotherapy response.

**Conclusion:** We identified changes of miRNA expression patterns after induction of chemotherapy resistance with various drugs used for lung SCC treatment. These findings may lead to development of new predictive biomarkers and to new miRNA-based drugs.

**Matched Pre- And Post- Treatment Analysis Of Glioblastoma Tumours**

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**Background:** Glioblastoma, the most common and aggressive brain cancer in adults, has a very grim prognosis and a 5 year survival rate of less than 10%. While treatment with surgery, radiotherapy and/or chemotherapy may prolong life, progression is inevitable. What is still unknown however, is how much these treatments affect the molecular profiles of these tumours and how these tumours adapt to withstand these treatment pressures. Understanding such changes will
uncover pathways used by the tumour to evade destruction and will elucidate new targets for treatment development. We hypothesise that molecular changes will be evident following treatment and progression of disease and these will correlate with resistance and the ability of the tumour to escape from the host’s immune system. Nineteen matched pre-treatment and post-treatment glioblastoma tumours were subjected to gene expression profiling (Fluidigm, TaqMan assays), MGMT promoter methylation analysis (pyrosequencing) and protein expression analysis of the DNA repair pathways, known to be involved in treatment resistance (immunohistochemistry). Gene expression profiling used to molecularly subtype tumours revealed that 26% of recurrent post-treatment specimens did not match their primary diagnostic specimen. Post-treatment specimens had molecular changes which correlated with known resistance mechanisms including increased expression of APEX1 (p<0.05) and altered MGMT methylation status. Increased expression of GPNMB, CCL5 and KLRCl (associated with immune suppression, invasion and aggression) and polarisation of macrophages to an M2 (anti-inflammatory and immune suppressive) phenotype in post-treatment tumours demonstrated an overall change in the tumour microenvironment favouring aggressive tumour growth and disease progression. These findings highlight the ability of glioblastomas to evade not only the toxic onslaught of therapy but also to evade the immune system suggesting that immune-altering therapies may be of value in treating this terrible disease.

Investigating indirect photobiomodulation-induced neuroprotection in a mouse model of Alzheimer’s disease

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Background: Photobiomodulation – the treatment of tissue with low-—intensity light (λ=600–1100nm) – is neuroprotective in various animal models of neurodegeneration when applied transcranially. In humans, clinical utility is limited by skull thickness; however evidence suggests that irradiating peripheral tissues may also confer neuroprotection. This ‘indirect’ effect shows promise in the translation of neuroprotective photobiomodulation for clinical use.

Aims: To determine whether indirect photobiomodulation mitigates amyloid plaque pathology in an APP/PS1 transgenic mouse model of Alzheimer’s disease.

Methods: Male APP/PS1 mice were treated with 670nm light (50mW/cm2) from a WARP 10 LED device for 3min/day, 3days/wk (8J/cm2 dosage/3min treatment). Light was targeted at the dorsum only, with the head shielded by foil. Following 8wks of treatment (at age 41wks), mice were perfused with formalin, and brains harvested and sectioned at 20µm thickness. Sections were stained with Thioflavin S for amyloid plaques, scanned using a Zeiss Axioscan, and images analysed for measures of plaque pathology using ImageJ 2. Statistical analysis was performed in Prism 6, using an unpaired two-tailed t-test with Welch’s correction. Values are given as means ± SEM.

Results: Treated (n=8) and sham---treated (n=5) animals did not differ significantly in either cortical plaque load (0.30±0.02 vs. 0.30±0.03 %tissue area; P=0.88) or density (14.16±0.71 vs. 13.22±1.39 plaques/mm2; P=0.53).

Conclusions & future directions: In APP/PS1 mice with established pathology, indirect photobiomodulation did not affect cortical fibrillary amyloid plaque load or density. This may be due to the animals’ advanced age at commencement of treatment; future studies may investigate whether earlier treatment mitigates disease development. Future analyses will examine other signs of earlier-stage pathology, including non-fibrillar β-amyloid deposition and markers of mitochondrial function and oxidative damage, as well as comparison with baseline pathology (i.e. at age 33wks).

Low-Dose Specific Inhibitors Of The Sodium-Potassium Pump Increase Cell Viability In Breast Cancer Cells

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Background: The Na/K/ATPase membrane pump is critical in regulating ion homeostasis, and thus cell survival. Due to its importance in maintaining life, the pump has gained interest as a potential target for the treatment of many types of cancer – such as prostate, breast, and lung cancer. In preclinical studies, cardiotonic steroids - specific inhibitors of the Na/K/ATPase – used at high doses have shown promising results as cancer treatment.

Aim: To investigate the effect of low-dose cardiotonic steroids on cell viability in two breast cancer cell lines: MCF7 and MDA468.

Method: Two breast cancer cell lines – MCF7 and MDA468 cells – were incubated with either vehicle or nanomolar to subnanomolar doses of the cardiotonic steroids ouabain, digoxin, or ouabagenin. Following the 24h treatment, PrestoBlue cell viability reagent was added to the cell media, and the absorbance was measured after 1h.

Results: Following treatment with ouabain, MCF7 cell viability increased up to 116±10% (P<0.05) from a baseline of 100%, whilst MDA468 cell viability showed a more modest increase of up to 110±8% (P<0.05). Incubation with either digoxin or ouabagenin did not significantly decrease viability in either cell line.

Conclusion: Taken together, these results suggest that Na/K/ATPase inhibitors may exacerbate breast cancer at low doses by stimulating cancer cell proliferation rather than effectively treating it. Cardiotonic steroids administered at higher doses that have previously been shown to be effective will eventually be metabolized and drop to lower concentrations, which will remove the beneficial effect. This suggests that the dose and rate of administration must be carefully studied. Future studies will test the effects of other cardiotonic steroids on cell viability, and on breast cancer cells implanted in mouse models.
MicroRNA Modulation Of Chemosensitivity In Adrenocortical Carcinoma
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Introduction: Adrenocortical carcinoma (ACC) is a rare cancer with a poor prognosis. Five-year survival over all stages approaches 35%. Current therapies include surgery with chemotherapy and mitotane. However, the first international RCT of chemotherapy and mitotane regimens for advanced ACC showed no increase in overall survival. MicroRNAs (miRNAs) are small non coding RNAs functioning in RNA silencing and post- transcriptional gene expression regulation. They are implicated in tumourigenesis and cancers’ susceptibility and response to chemotherapy and radiation.

Methodology: Frozen stage 4 ACC tissue obtained from the Kolling Institute of Medical Research Tumour Bank were stratified into “responsive” and “non- responsive” groups based on clinical survival and treatment response. Tumour miRNA profiling was performed with Taqman Low Density Array Cards (ThermoFisher Scientific). A list of differentially expressed miRNAs between the comparing groups were selected and further confirmed with individual RT-qPCR. The candidate miRNA mimic was transfected onto the ACC H295R cell line using Lipofectamine RNAiMax (ThermoFisher Scientific). Transfected H295R cells were treated with chemotherapy drugs/mitotane at various concentrations. Cell proliferation/viability was assessed with MTS assays (Promega).

Results: Several miRNAs, including miR-431 were significantly underepressed in the “non-response” group. Various effects of miRNAs interacting with chemo/mitotane treatment were identified.

Conclusion: In vitro data shows miRNA therapeutic potential of in increasing chemotherapy/mitotane responses in ACC treatment. Further work to ascertain its underlying molecular mechanisms is required.

The effects of Green Tea Polyphenols on ovarian steroidogenesis in ovarian cells
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Background: Green Tea Polyphenols have been shown to have wide variety of health benefits, especially for metabolic syndrome. However, there has been contradictory data for the effects of GTP on steroidogenesis and may differ between males and females. Our previous studies have shown that GTP inhibits adipocyte differentiation while increasing osteoblast differentiation in human adipose derived stem cells. Given the role of reproductive hormones such as estrogen on adipogenesis and bone formation, clarification of the role of GTP in ovarian steroidogenesis is warranted.

Aims: To investigate the effects of different concentrations of Green Tea Polyphenols (GTP) on estrogen, progesterone and testosterone secretion in murine ovarian cells and explore the molecular mechanisms related to the steroidogenic pathway.

Methods: A co-culture of ovarian theca and granulosa cells were treated with concentrations of GTP (1, 10 and 25µg/mL). Media were collected for immunoassay for estrogen, progesterone and testosterone. Immunofluorescence staining techniques were also used to determine expression of cholesterol side-chain cleavage (CYP11A1) and 17α-hydroxylase (CYP17A1) involved in ovarian steroidogenesis.

Results: The results show that GTP significantly increases estrogen, progesterone and testosterone in ovarian cells (P<0.01). Immunofluorescence staining and western blot are still ongoing.

Conclusions: These results suggest that GTP increases steroidogenesis in ovarian cells. It is anticipated that this is primarily through upregulation CYP11A1, the rate-limiting step in the steroidogenic cascade. Increased estrogen production by GTP may be useful in the treatment of conditions such as osteoporosis related to menopause. However, the effects of GTP on the hormones may also negatively impact some other disease states and therefore further experiments are needed to clarify the role of GTP on steroidogenesis.

Explosive cell lysis is involved in the biogenesis of staphylococcal “public goods” and membrane vesicles
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The Gram-positive Staphylococcus aureus is a major cause of nosocomial and community-acquired infections. Many of those develop into chronic infections and are difficult to eradicate due to the formation of biofilm- complex multi-cellular structures that enhance antibiotic resistance and evasion of human defences. Biofilms are characterized by cell attachment to a surface and encapsulation in self-produced extracellular polymeric substances, comprised of extracellular DNA (eDNA), membrane vesicles (MVs) and bacterial cytosolic proteins referred as “public goods”. These extracellular products enhance the structural integrity of biofilm matrix, protect the community from antimicrobial agents and contribute to virulence and chronic infection. However, the mechanism of “public goods” and MV production by S. aureus has not been fully elucidated. Staphylococcal interstitial biofilms were monitored by time-lapse microscopy and release of eDNA detected using a cell impermeant fluorescent dye specific for DNA. We found that occasionally individual cells within a cluster lysed and at the same time eDNA was produced. Two type of cell lysis were observed: slow lysis, in which DNA remained partially trapped inside dead cells and after several minutes it was released into the extracellular space; and explosive cell lysis, a rapid event occurring within seconds characterized by efficient eDNA released in bursts. Super-resolution microscopy (OMX 3D-SIM) also revealed the presence of MVs at the sites of eDNA smears. The main enzyme involved in cell lysis in S. aureus is the peptidoglycan hydrolase Atl. We found that mutants lacking atl showed significantly impaired explosive cell lysis, and were abrogated in the production of eDNA and MVs in interstitial biofilms. Finally, we also found that staphylococcal cell lysis accounts for MV production in response to antibiotic stress.
Optimising a preclinical model to identify adverse geriatric outcomes from polypharmacy and Drug Burden Index

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Background: Polypharmacy (using ≥5 drugs) and exposure to medicines with anticholinergic and sedative effects (measured with Drug Burden Index, DBI) are common in older people and are associated with adverse outcomes including impaired physical function, frailty, falls, hospitalization and mortality. Preclinical models of polypharmacy and DBI are required to rigorously assess causation of these associations.

Aim: Establish models to determine the effect of polypharmacy and increasing DBI on adverse outcomes.

Methods: Young (3 months) and middle aged (12 months) male C57BL/6 mice were treated for 1 or 3 months, respectively, with control or treated feed/water containing different drug regimens at therapeutic doses. Treatment regimens were Polypharmacy Zero DBI (simvastatin, metoprolol, omeprazole, paracetamol, irbesartan), Polypharmacy Low DBI (simvastatin, metoprolol, omeprazole, paracetamol, cilostazol), Polypharmacy High DBI (simvastatin, metoprolol, oxbytynin, oxyocodone, cilostazol) and single drugs: simvastatin, metoprolol, oxbytynin, oxycodone and cilostazol. Toxicity and functional tests were conducted at baseline and after treatment.

Results: Drug regimens were well tolerated. Compared to control, locomotor activity (open field distance, time mobile and centre exploration entries) was significantly decreased, with the Polypharmacy Low and High DBI regimens in young and middle aged mice and with the single drug cilostazol in middle aged mice only. No other exposures affected locomotor activity and none of the exposures affected grip strength (wire hang) or muscle endurance (rotor rod) in either age group. Conclusion: We have established preclinical models to determine the effects of polypharmacy and DBI. Polypharmacy with increasing DBI and chronic cilostazol significantly impair locomotor activity in young and/or middle aged mice. The observed differences between shorter exposures in young adult mice and longer exposures in middle aged mice highlight the importance of pre-clinical evaluation relevant to the intended clinical exposure period and population.

Biochemical Characterization of CLIC members and the Influence of the Six Histidine Tag on Enzymatic Activity

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Chloride intracellular ion channel (CLIC) proteins are novel redox enzymes in humans that catalyze glutathione-dependent oxidoreductase reactions. In the current study, we investigate the biochemical properties and enzyme kinetics of two members of the CLIC family, CLIC1 and CLIC3. Interestingly, in the course of this investigation our results revealed distinct activity in the HEDS oxidoreductase enzyme assay between the His- tagged recombinant CLIC proteins versus their non-His-tagged counterparts.

Our results demonstrate that the His-tagged form of CLIC1, CLIC3 and HcTrx5 (a worm oxidoreductase) is more active than the non-His-tagged form of each protein, with distinct enzyme kinetics. Most significant and unexpected is the enzymatic activity by the His-tagged version of the inactive mutant, C24A-CLIC1. Furthermore, the drug IAA94 that was shown to abolish CLIC1 enzyme activity had no significant effect on the His-tagged CLIC1 enzymatic activity. In addition, the His tag appears to confer heat resistance to the proteins, with His-tagged CLIC 1 and CLIC3 becoming heat stable up to 60oC for 10mins, in contrast to their non-His-tagged form. This work conclusively demonstrates that the commonly used six Histidine tag confers additional enzymatic activity upon His-tagged proteins and therefore, its removal from recombinant proteins prior to their use in such assays is essential.

Deletion of Hypoxia-inducible factor 1α in brown and beige adipocytes increases energy expenditure and reduces body weight in mice

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There are two major types of adipose tissue (fat) in mammals. White adipose tissue (WAT) is highly adapted to store excess energy; while brown adipose tissue (BAT) produces heat as a defence against hypothermia and obesity. The appearance of brown-like adipocytes within white adipose tissue (beige adipose tissue) is related to improved metabolic phenotypes. Brown and beige fat cells express uncoupling protein-1 (UCP1), a unique protein that facilitates heat generation by allowing proton leaks across the inner mitochondrial membrane. Oxygen availability is a key element of energy homeostasis which influences both the rate and the mechanism of substrate utilization for energy production. Hypoxia-inducible factor 1α (HIF1α) is a bHLH-PAS transcription factor that regulates energy metabolism pathways that become dysregulated in obesity.

We have created mice that lack HIF-1α specifically in brown and beige fat cells by breeding UCP1-Cre mice with mice possessing a floxed HIF-1α allele. These mice tend to weigh less (5%) and have (4.8%) increased energy expenditure and reduced adiposity (4%) compared to Ucp1-Cre mice. These results demonstrate that HIF may have effects on brown/beige adipose tissue metabolism. Inhibitors of HIF1α can provide a potential therapeutic target for the treatment of metabolic diseases.
Gender-Specific Regulation Of Renal Fat Metabolism And Autophagy By Maternal High-Fat Consumption*

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Background: Maternal obesity has been associated with kidney damage and dysfunction in male offspring.

Aim: However, the underlying mechanisms as well as gender differences of this transgenerational effect remain unclear and need to be investigated. Methods: In this study, female rats were fed a high-fat diet (HFD) for 6 weeks prior to mating, throughout gestation and lactation, the kidneys from both male and female offspring at weaning were examined for lipid metabolic and stress response markers. 

Results: Our results indicate that renal lipid deposition was increased in male offspring only. In addition, the expression of Sirtuin (SIRT1) and Peroxisome proliferator-activated receptor gamma (PPARγ) coactivator 1-alpha (PGC-1α), two regulators of fatty acid oxidation, was significantly reduced. Phosphorylated 5'-AMP-activated protein kinase (pAMPKα) and Forkhead box (FOX) O3a, two molecules in the SIRT1 signalling network, were also downregulated. By contrast, in female offspring, renal fatty acid content was unchanged, and PPARγ2 and PGC-1α expression was reduced instead. Renal autophagy was also selectively regulated. In male offspring, there was reduction in most autophagy markers including Beclin-1, Atg12-Atg5 complex, LC3-I/II and p62; in female offspring, only Atg7 and LC3-I/II proteins were significantly decreased. On the other hand, G6Pase-1, a marker of antioxidant defence, was similarly reduced in both genders.

Conclusion: Together, these findings suggest that under the effects of maternal HFD, there was a gender difference in the regulation of renal lipid metabolic and autophagy markers, but not the antioxidant defence marker. Understanding these differences is essential to develop gender-specific therapies to reduce the risk of kidney disorders at young ages.

The effects of Radix Albus Paeoniae Lactiflorae on androgen production in ovarian theca cells*

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Background: Polycystic Ovary Syndrome (PCOS) is a complex endocrine and reproductive disorder affecting approximately 10% of women of reproductive age. Hallmarks of PCOS include hyperandrogenism and ovulatory dysfunction which are closely linked with insulin resistance and subfertility. Chinese herbal medicine (CHM) has been traditionally used in the treatment of reproductive disorders such as PCOS, however the molecular mechanisms of these herbs have not yet been fully elucidated.

Aim: To examine the effects of Radix Albus Paeoniae Lactiflorae (Bai Shao), a herb commonly used in CHM on testosterone and progesterone secretion in ovarian theca cells and to define the molecular mechanisms involved.

Methods: Murine theca cells were treated with concentrations of Bai Shao extract (1-100µg/mL) in the presence of dexamethasone (10 µM). Media were collected for immunoassay of testosterone and progesterone. The effects of Bai Shao on cell proliferation were also assessed using SYBR green assay. Protein expression of cholesterol side-chain cleavage (CYP11A1) and 17α-hydroxylase (CYP17A1) involved in ovarian androgen production was also investigated using immunofluorescence staining and later, Western blotting.

Results: Bai Shao extract (100µg/mL) significantly decreased testosterone in dexamethasone treated theca cells (P<0.05). Dexamethasone suppressed progesterone levels which was also reversed by Bai Shao (P<0.05). There were no significant changes to cell proliferation with Bai Shao concentrations of 1-1000µg/mL compared to control. Immunofluorescence staining also revealed that Bai Shao inhibits the expression of CYP11A1. Preliminary Western blot analysis of CYP11A1 also confirms these findings.

Conclusion: Bai Shao reduces testosterone secretion in theca cells which appears to be through suppression of CYP11A1 that regulates the rate-limiting step in the ovarian steroidogenic pathway. These findings suggest that the herb may be useful in targeting hyperandrogenism in the treatment of PCOS with further experiments warranted.

Self-medication among people living with hypertension: a review*

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Background: Self-medication is commonly practised by patients, underpinned by health beliefs that affect their adherence to medication regimens and impacting on treatment outcomes.

Aims: This review explores the scope of self-medication practices among people with hypertension in terms of the scale of use, types of medication and the influencing factors.

Method: A comprehensive search of English language, peer-reviewed literature published between 2000 and 2014 was performed. Twenty-seven studies met the inclusion criteria; 22 of these focused on complementary and alternative medicines (CAMs).

Results: Anti-hypertensive medications are listed among 11% of products that patients reportedly obtain over-the-counter (OTC) for self-medication. On average, 25% of patients use CAMs, mostly herbs, to lower their blood pressure. Recommendations by family members, friends and neighbours are the most influential factors for self-medication. Most (70%) patients with hypertension take OTC medicines to treat minor illnesses. The concurrent use of anti-hypertensive medications with analgesics and herbal medicines is commonly practised. The socio-demographic profile of patients engaging in self-medication differs markedly in the articles reviewed; self-medication practices cannot be attributed to a particular profile. Low disclosure of self-medication is consistently reported.

Conclusion: This review highlights that a high proportion of people with hypertension practise self-medication. Health professionals involved in hypertension management should be mindful of any types of self-medication practices. Further
Inadequate supply of hypertension medication for patients in rural Indonesia

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Background: Adequate supply of medicines is essential to facilitate medication adherence.

Aims: This study investigated the duration of medication intake within a 30-day time frame and the place of medication procurement.

Methods: Data pertaining to patients with hypertension (aged > 45 years) were collected from eight rural villages in the Bantul district, Yogyakarta province, Indonesia. A researcher-administered questionnaire was used to collect socio-demographic data, hypertension history and the use of hypertension medication.

Results: Of 384 participants, 203 (52.9%) had taken antihypertensive medicines in the previous 30 days. More than half of them (n = 109, 54%) had taken captopril as a monotherapy, while 18 (9%) participants had taken a combination therapy. The total duration of medicine use over the 30 days was mostly less than 7 days (n = 88, 43%). As reported by participants, the supply of medicines was typically only enough for 3–5 days, particularly for the ones who obtained their medicines from the Integrated Health Service Post for Elderly (43 of 47 participants), village doctor (7 of 8 participants), village midwife (10 of 13 participants) and village nurse (10 of 15 participants). There were only 40 (10%) participants who took the medicines every day within the 30-day timeframe; they obtained their medicines from the community health centre (n = 18), public hospital (n = 4), community pharmacy (n = 5), private hospital (n = 2), and multiple sources (i.e. community pharmacy plus other sources of supply) (n = 11).

Conclusion: Based on patients’ self-report this study indicates inadequate provision of medicines across health care providers. Acknowledging the inventory management challenges prevailing in the current Indonesian rural primary health care system, future research should explore ways to address and overcome these issues.

Beta Blockers Are Associated With Impaired Spontaneous Development Of Endothelial Progenitor Cells In Humans Suspected Of Having Coronary Artery Disease

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Background: Production of endothelial progenitor cells (EPCs) is an essential part of vascular regeneration after a myocardial infarction (MI). They are thought to improve vessel formation and aid in the formation of new capillaries. Research that shows that certain clinical variables, including both degree of arterial disease, smoking, diabetes, and the use of statins and exercise can modify EPCs levels and characteristics.

Aims: To identify clinical co-variates that are associated with altered spontaneous growth of EPCs in patients susceptible or resistant to atherosclerotic disease.

Methods: Patients were recruited to the BIOHEART study run at North Shore Radiology, and data was collected on age, sex, medication and cardiovascular risk factors. Blood samples were collected in heparin coated vacuettes prior to patients then undergoing Computed Tomography Coronary Angiogram (CTCA) for investigation of cardiac atherosclerosis. Peripheral blood mononuclear cells were extracted via Ficoll gradient centrifugation and these cells were cultured in 0.1% gelatin coated 24 well plates in endothelial cell growth medium.

Results: Time to spontaneous growth of EPCs was recorded over a 30 day period. Cell colonies were expanded and EPC cell characteristics were confirmed by morphology and functional characteristics. EPCs were capable of tubule formation on matrigel, similar to endothelial cells from other sources. Samples from 17.5% of patients spontaneously developed EPCs. Spontaneous development of EPCs in samples was not affected by age, smoking or family history of cardiovascular disease. Interestingly, of the patients taking ß-blockers, 0/31 demonstrated spontaneous development, versus 28/129 of those not taking ß-blockers (p<0.01).

Conclusion: The use of beta-blockers was associated with complete absence of spontaneous EPC development. This may be relevant to patients with coronary artery disease, impairing adaption and collateral formation.
Awards and prizes include:

- Best Oral Presentation by an honours student ($500)
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GENERAL INFORMATION

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Dr Chau Chak Wing Building (Building 8), Ultimo NSW 2007.

CAR PARKING
Limited car parking is available at UTS for mobility-impaired visitors. Local car parks include:

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TRAIN
T1, T2, T3, and T4 trains stop at Central station. For directions from Central Station to UTS see: [http://www.uts.edu.au/sites/default/files/uts-tag.pdf](http://www.uts.edu.au/sites/default/files/uts-tag.pdf). From the South Concourse or Railway Square follow the Goods Line or TAFE signage for direct access to the Dr Chau Chak Wing Building.

LIGHT RAIL
Dulwich Hill to Central line (Closest stops are Paddy’s Market or Capital Square)

SMOKING
Smoking is banned on the UTS campus, except for within designated smoking areas. Smoking is specifically prohibited within 4 metres of all pedestrian access points to a building.
The conference will be held on level 3 of the Dr Chau Chak Wing Building (one level street level). Enter via the Goods Line Pedestrian Walkway (labeled on map) OR from the street level and go up the stairs/lifts.

LEVEL 3

Goods Line Pedestrian walkway from Central Station and Railway Square
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