The Australian Society for Medical Research

The Queensland branch of the ASMR are proud to present the

2015 HEALTH & MEDICAL RESEARCH AWARDS

Proudly Supported by

Queensland Government

The Australian Society for Medical Research

"Medical Research - Bringing Health to Life"

ASMR is the peak professional organisation representing health and medical researchers in Australia. Direct members, seventy-five affiliated professional societies, institutions and medical colleges, together with disease related foundation memberships bring more than 100,000 Australians into the ASMR network

ASMR planning the future

Investing in health and medical research drives the 3I's of invention, innovation and implementation, creating jobs and underpinning future manufacturing.

Most importantly, investment into health and medical research underpins a healthier, more productive Australia

- Establishd 1961 - Not for Profit
- Fostering excellence in Health & Medical Research
- Non politically aligned
- Annual National Scientific Conference
- Proud initiator and trademark owner of "The Australian Health and Medical Research Congress"
- Career and Professional Development
- Research Awards - Student Travel Grants
- Expanding the interface between basic science, clinical and transdisciplinary research
- Newsletter - Dissemination of information
- Member Directory
- Public Outreach and Political Advocacy
- Online Mentor Program

ASMR Medical Research Week(R)

- ASMR Medallist tour
- Career Development Seminars
- State Scientific Meetings and Awards
- Public Lectures/Debates
- Rural and Metropolitan Schools Events
- National Schools Quiz
- Cinema/Pub and Trivia Events
- Dinner with a Scientist

Submissions 2009/10/11/12/13/14/15

- Strengthening iMRI's Discussion Paper
- Harmful use of alcohol in Aboriginal and Torres Strait Islander communities
- Response to the McKeon Review
- Submission to the Chubb Review
- Submission to the McKeon Review
- Pre-Budget Submissions
- ToR HMR Review
- DIISR Research Workforce
- House SC on Economics
- NHMRC Strategic Plan Consultation
- NHMRC Partnership Centres
- NEAF Evaluation
- NHMRC Rsh. Fellowship Scheme

See all submissions at

http://www.asmr.org.au/Submissions.html

2014 Deloitte Access Economics Report
"Extrapolated returns on investment in medical research future fund (MRFF)"
2012 Deloitte Access Economics Report
"Extrapolated returns on investment in NHMRC medical research"
2011 Deloitte Access Economics Report
" Returns on NHMRC funded Research And Development"
2009 HMR Workforce Planning 2009-2019

Queensland Health is proud to again support the Australian Society for Medical Research (ASMR) Queensland Health and Medical Research Awards. I would like to congratulate all applicants and recipients for 2015. These awards provide an important opportunity to recognise the exceptional scientific contributions of Queensland's health and medical researchers.

Research is vital to improving Australia's health service delivery and it is clear that Queensland is at the forefront of medical research and innovation to achieve this improvement in delivery. From the world's first cancer vaccine, Gardasil, to a needle free vaccination method, the Nanopatch, Queensland continues to achieve medical breakthroughs.

With our world-class facilities and outstanding researchers, I believe Queensland is succeeding at fostering innovation and research that contributes to quality patient care and outcomes, and health system improvement.

By rewarding early to mid-career researchers we are not only highlighting the skill and talent of researchers in this State but creating an environment where research excellence is valued and supported. Given the high quality of research undertaken by the outstanding awards finalists, we can see that Queensland research will continue to thrive in the future.

It is exciting that this inspiring group of talented scientists, clinicians and researchers has the potential to build a health system designed to meet the challenges of the future.

Congratulations and thank you to all applicants and finalists. I look forward to seeing the positive impact of your research for patients in Queensland, across Australia and ultimately the world.

Dr Michael Cleary
Acting Director-General, Department of Health

Queensland Government

The Australian Society for Medical Research

The Queensland branch of the ASMR are proud to announce the finalists for the

ASMR QUEENSLAND HEALTH & MEDICAL RESEARCH AWARDS, 2015

Proudly supported by the Queensland Government

The finalists for these prestigious awards are as follows:

Postdoctoral Researcher Award Finalists (1:00-1:45pm)

Andrea Schuessler (QIMR)
Amirali Popat (UQ)
Shin Foong Ngiow (QIMR)

Senior Researcher Award Finalists (1:45-2:30pm)

Shyuan Ngo (UQ)
Sarah Medland (QIMR)
Kyle Upton (Mater Medical Institute)

Clinical Researcher Award Finalists (2:30-3:15pm)

Yogavijayan Kandasamy (TTH & UON)
Christopher Carty (QCGL & Griffith University)
Alwyn Todd (Griffith University)

Venue: QIMR Berghofer Medical Research Institute (Level 3 Auditorium)
300 Herston Rd, Herston
Date: Monday 25th May 2014
Time: 1:00pm-3:15pm

Postgraduate Student Award Finalists

Jonathon Fanning (Prince Charles Hospital)
Shani Stuart (QUT)
Mitchell Stark (QIMR)
Gillian Fisher (Griffith University)
Laure Martine (QUT)
Arabella Young(QIMR)

Venue: QIMR Berghofer Medical Research Institute (Level 3 Auditorium)
300 Herston Rd, Herston
Date: Wednesday 27th May 2014
Time: Registration from 8:00-8:45am and closing at 5:10pm

All are welcome to hear this year's finalists present their research

The Australian Society for Medical Research ACN 000 599 235 - ABN 18 000 599 235
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Email: asmr@alwaysonline.net.au, Website: www.asmr.org.au Snr Executive Officer: Catherine West

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JONATHON FANNING

The University of Queensland

Transcatheter aortic valve implantation (TAVI)

Aortic stenosis (AS) is the most common heart valve disease in the western world. One-half of patients with severe AS are rejected for valve surgery because of excessive risk. Transcatheter aortic valve implantation (TAVI) is an innovative life-saving treatment for these patients. However, the possibility of brain injury from TAVI has limited its use. This study performed the most comprehensive neurological/cognitive assessment to date to characterize the brain injury associated with TAVI and identify strategies to minimize its occurrence. While no patient had symptoms of stroke or cognitive dysfunction, 60% had silent strokes detected by brain imaging.

Scientific Abstract:

Background: Transcatheter aortic valve implantation (TAVI) has expanded the therapeutic options for high-risk and inoperable patients with severe aortic stenosis (AS). However, the poorly-defined risk of neurological injury post-TAVI has raised concerns that both impact informed decision-making and limit its widespread application.

Methods: In 2014, 30 high-risk patients undergoing transfemoral TAVI at The Prince Charles Hospital, Brisbane, Australia, were prospectively enrolled. Participants underwent the most rigorous assessment for neurological injury to date: serial neurological and cognitive assessments; pre-procedural carotid Doppler ultrasonography; pre-procedural chest computed tomography; intra-procedural INVOSTM 5100 (Covidien, CO, USA) cerebral oximetry and haemodynamic monitoring; and brain magnetic resonance imaging, including diffusion weighted imaging (DWI) sequences, pre-procedure and 3 days post-procedure.

Results: The mean participant age was 82.7±18 years; 66.7% were female. There were no clinically-apparent cerebrovascular events and only one delirium episode. Compared to baseline, a trend towards improved cognition was noted at 6-week follow-up (Montreal Cognitive Assessment scores: 25.6 [95% CI, 24.4–26.9] vs 23.2 [95% CI, 21.7–24.6]). Fifty new post-procedural (day 3) DWI lesions were identified in 12 of 20 participants (60%), with 4.2 lesions per affected patient and mean volume of 250 mm3/lesion. Among these, 54% were in the posterior cerebral artery territory. The incidence of DWI lesions did not correlate with presumed risk factors, including cerebral desaturation or aortic calcification score.

Conclusions: Subclinical neurological injury is common post-TAVI. The predominance of posterior-circulation DWI lesions poses unique considerations for neuroprotective strategies. The previously assumed aetiological importance of calcium dislodged from the aorta/aortic valve or hypoperfusion was not supported. In addition to describing improved perioperative monitoring for neurological injury in clinical practice, this study has formed the template for an international investigation of post-TAVI neurological injury. It is the pilot for the multi-centre Australian Cerebrovascular Hazard/Injury chaLLEnging aortic Stenosis management (ACHILLES) study.

SHANI STUART

Queensland University of Technology

Mitochondria, the energy producing organelles of the body are linked with migraine susceptibility

Sufficient energy production to enable our neural system to function correctly could be related to migraine, a common neurological disease affecting approximately 12% of the general adult population. Mitochondria are found in all mammalian cells to supply the tissues of our body with energy, especially muscle and neural cells which have the highest energy requirements. This study investigated the link between mitochondrial dysfunction and migraine susceptibility by studying the underlying genetic variation in migraine sufferers compared to healthy controls. The Norfolk Island genetic isolate population was used as a discovery cohort and follow up studies were undertaken in large Australian Caucasian migraine case control populations. The latest genomic next generation sequencing technology was used to perform whole mitochondrial sequencing and subsequent statistical modelling analysis was used to identify significant findings. This analysis showed a clear link between mitochondrial function and migraine pathogenesis, which may lead to the identification of new therapeutic targets.

Scientific Abstract:

Migraine is a common neurological disorder affecting approximately 12% of the Australian Caucasian population with three times more females affected than males. It represents a significant personal and economic burden and remains sub-optimally treated, presenting a need for the identification of novel therapeutic targets. While some progress had been made, a large proportion of the genetic variance underlying migraine pathogenesis remains to be identified. We aimed to pursue a new avenue of research and hypothesized that the pathophysiology of migraine is influenced by mitochondrial dysfunction. The aim of this study was to investigate genetic variation within the mitochondrial genome and nuclear encoded genes affecting mitochondrial function in relation to migraine susceptibility. In total n=315 individuals from the genetically isolated Norfolk Island population underwent full mitochondrial genome sequencing. We identified 3 homoplasmic and 11 heteroplasmic variants to be significantly associated with migraine susceptibility. Logistic regression analysis showed that 930G>A located within the 12S rRNA subunit is significantly associated with migraine, an interesting finding given that five causative mutations for sensineuronal hearing loss have been identified in this region. A Fisher's exact test identified two rare variants associated with migraine susceptibility namely 6480 G>A and 11930 A>G situated in the COX 1 and NADH dehydrogenase genes respectively. Of further interest was the discovery that haplogroup K significantly increases an individual's risk of developing migraine. Investigation of nuclear encoded genes involved in mitochondrial function revealed that the PCDHG gene cluster plays a pivotal role in migraine susceptibility in both the Norfolk Island population and an outbred Australian Caucasian population. This is the first molecular genetic study of this scale to show a conclusive link between genetic variation and mitochondrial dysfunction. Future studies will expand on these findings and strengthen the theory of mitochondrial dysfunction influence in migraine pathophysiology.

MITCHELL STARK

QIMR Berghofer Medical Research Institute

MicroRNA Expression Regulates the Sensitivity of Targeted Therapy in Melanoma

Melanoma is the 4th most common cancer in Australia. Targeted therapy in patients with stage IV melanoma (distal spread of disease) is one of the first-line treatments for advanced disease. However, most patients do not have a durable response to the treatment due to either inherent or acquired resistance to therapy. One of the many mechanisms for this resistance is due to loss of a 'tumour-suppressor' gene called NF1. We found that loss of NF1 function can be explained in part via regulation by a microRNA called miR-514a. MicroRNAs are small ribonucleic acid sequences that control protein levels in the cell. We found that when miR-514a was turned on, melanoma cells were resistant to the main targeted therapy. On the other hand, when miR-514 was 'turned off', melanoma cells responded well to the therapy. Results from this study will help to devise better treatments for late stage melanoma patients.

For the past 15 years and currently as a PhD student Mitchell has been working towards understanding the aetiology of melanoma studying gene dysregulation during tumour progression. More recently during his PhD candidature, his research focus is identifying 'melanoma-specific' microRNAs that may be useful for clinical management of disease.

Scientific Abstract:

To identify 'melanoma-specific' microRNAs we used an unbiased microRNA profiling approach to comprehensively study cutaneous melanoma in relation to other solid malignancies, which revealed 233 differentially expressed (≥2 fold, $p<0.05$) microRNAs. Among the top 20 most significantly different miRNAs was hsa-miR-514a-3p. miR-514a is a member of a cluster of microRNAs (miR-506-514) involved in initiating melanocyte transformation and promotion of melanoma growth. We found miR-514a was expressed in 38/55 (69%) melanoma cell lines but in only 1/34 (3%) other solid cancers. To identify miR-514a regulated targets we conducted a miR-514a-mRNA 'pull-down' experiment, which revealed hundreds of genes, including: *CTNNB1*, *CDK2*, *MC1R*, and *NF1*, previously associated with melanoma. NF1 was selected for functional validation because of its recent implication in acquired resistance to BRAFV600E-targeted therapy. Luciferase-reporter assays confirmed *NF1* as a direct target of miR-514a and over-expression of miR-514a in melanoma cell lines inhibited NF1 expression, which correlated with increased survival of BRAFV600E cells treated with PLX4032. These data provide another mechanism for the dysregulation of the MAPK pathway which may contribute to the profound resistance associated with current RAF-targeted therapies. In combination with currently used therapies of the MAPK, PI3K, and mTOR pathways, pharmacological intervention of miRNAs may allow for more durable outcomes in late stage melanoma patients.

GILLIAN FISHER

Griffith University

Investigating the Antimalarial Potential of Primary Sulfonamide Compounds

Malaria is a significant infectious disease, causing ~600,000 deaths annually, mainly in African children under the age of five. Unfortunately all current antimalarial drugs are failing due to the widespread emergence of drug resistant parasites. Thus, new antimalarial drugs are urgently needed, particularly those that act differently to current drugs as this may help prevent treatment failure due to resistance. Primary sulfonamides are a type of chemical compound not yet exploited for malaria, but with proven efficacy and safety as drugs for other diseases. In this study novel primary sulfonamides with antimalarial activity were identified and one of these compounds was used to generate laboratory resistant malaria parasites. These resistant parasites are an important new tool that will allow us to determine whether these compounds kill malaria parasites differently to current antimalarials, potentially identifying a novel target in the parasite that can be used to develop new antimalarial drugs.

Scientific Abstract:

Malaria is a major public health threat, causing hundreds of millions of clinical cases and ~0.6 million deaths annually. Unfortunately malaria parasite resistance has emerged to all currently-used antimalarial drugs. Added to this, the majority of agents under investigation in the antimalarial drug discovery and development portfolio contain known antimalarial pharmacophores. This may compromise their widespread use due to potential issues of cross resistance with existing drugs. This alarming situation is driving efforts to discover and develop new chemical types (chemotypes) as antimalarial drugs. In this study primary sulfonamides were investigated as a potential new antimalarial chemotype. Primary sulfonamides have a proven track record of efficacy and safety in many clinical applications including glaucoma, renal disorders and epilepsy. To investigate their antimalarial potential, both clinically used and novel primary sulfonamides were screened in vitro against Plasmodium falciparum drug sensitive (3D7) and resistant (Dd2) malaria parasites. One of these compounds (PS-3) was found to have a sub-micromolar 50% inhibitory concentration for both parasite lines and good selectivity for P. falciparum versus mammalian cells (Selectivity Index >50). Target identification studies were carried out, including generation of a recombinant P. falciparum carbonic anhydrase (rPfCA) protein (a known target of primary sulfonamides in other organisms) and in vitro selection of resistant parasites. Enzyme inhibition studies identified rPfCA as a putative target of PS-3. Analysis of the phenotype of parasites resistant to PS-3 showed no cross-resistance with clinically used antimalarials or amplification of the multi-drug resistance transporter (Pfmdr1) gene which is associated with antimalarial resistance. Whole genome sequencing studies are currently underway to detect possible genetic alterations associated with P. falciparum resistance to PS-3. This information may identify a novel malaria parasite drug target which may aid in developing the primary sulfonamide chemotype as an alternative antimalarial drug lead with less potential for cross resistance.

LAURE MARTINE

Queensland University of Technology

Tissue-engineered humanised xenograft models to dissect mechanisms of breast cancer-induced bone metastasis

Bone metastasis occurs in 80% of women suffering from advanced breast cancer (BC) and remains incurable to date. The development of new therapies is hampered by the lack of appropriate models to mimic this disease. Current studies use human BC cells grown in the mouse skeleton, but due to differences between both species the results are often not transferable to the clinical situation. We have developed a model that reproduces the interactions of human BC cells and human bone in vivo. Human bone-forming cells obtained from adult patients are transplanted under the skin of immune-deficient mice with a biomaterial carrier and bioactive factors promoting bone formation. This tissue engineering approach leads to the formation of a human-derived bone tissue in mice. We have applied this “humanised” bone model to reproduce the development of metastases from injected human BC cells and investigate biological mechanisms involved in human BC metastasis to bone.

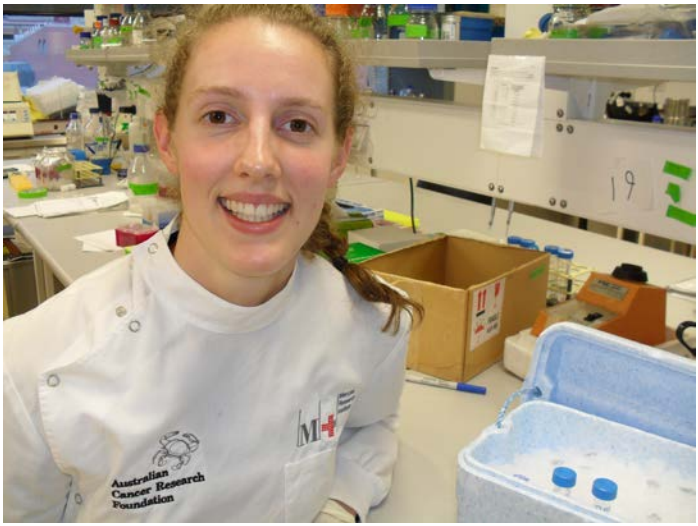
Scientific Abstract:

The skeleton is a preferred site for breast cancer (BC) metastasis, but the mechanisms involved in this disease are still poorly understood due to a lack of suitable animal models to interrogate human-specific tumour-bone interactions. We have established a tissue engineering method to reconstitute a human bone microenvironment in mice. Human tissue engineered bone constructs (hTEBCs) based on scaffolds combined with human osteoblasts and bone morphogenetic protein 7 are implanted ectopically in immunodeficient mice to engineer humanized bone. The model reproduces closely the physiology and structure of native bone, and comprises human bone cells and extracellular matrix. We have shown that systemic inoculation of BC cells via the intracardiac route leads to the development of osteolytic metastases in the hTEBCs that reproduce closely clinical bone lesions in BC patients. In this work we further investigated the use of the hTEBC as a platform to study the role of integrins during the establishment of BC cells in the bone microenvironment. In fact, β1 integrins have been identified as a prognostic marker of invasive BC but their role during bone colonisation by BC cells remains unclear. Using preclinical fluorescence imaging and histological analysis we have shown that suppression of β1 integrins in BC cells significantly inhibited tumour growth within hTEBCs. Immunohistochemical analysis revealed a decrease in BC cell proliferation in bone in the absence of β1 integrins. Despite their role in modulating tumour development in bone, β1 integrins did not influence osteoclast activation and osteolytic bone resorption. Analysis of β1 integrin expression in tissue micro-arrays assembled from a cohort of 22 BC patients showed that these adhesion molecules are detected in clinical bone metastases across different BC subtypes. Taken together, the hTEBC provides a platform to investigate potential therapeutic targets and improve our understanding of bone metastatic disease.

ARABELLA YOUNG

QIMR Berghofer Medical Research Institute

Targeting immunosuppressive adenosine to promote an immune response against cancer



Within the clinic, recent advances in immunotherapies that aim to reinvigorate the immune system's fight against cancer have shown great success. Nevertheless, variation in treatment efficacy is evident and while some patients respond well, others show resistance. By combining therapies that inhibit a diverse range of immunosuppressive pathways it is predicted that patient's will receive durable benefits. However, identifying synergistic combination approaches remains a significant challenge. Notably, production of the immunosuppressive metabolite adenosine is increased within the tumour microenvironment. This suppresses tumour cell killing performed by immune cells allowing for disease progression. We identified that targeting adenosine improves survival and reduces tumour burden in preclinical models. Importantly, by combining clinically utilised immunotherapies with targeted therapies against adenosine we further improve disease control. Our studies advocate that targeting adenosine in immunotherapeutic combinations, has potential to reduce tumour burden and progression, increasing patient life expectancy and quality of life.

Scientific Abstract:

CD73 generation of immunosuppressive adenosine in the tumour microenvironment causes dysregulation of immune cell infiltrate. This results in tumour progression, metastases and poor disease outcomes for patients. The emergence of targeted therapies towards the adenosinergic pathway, which enhance anti-tumour immune defences, appear promising. However, their ability to synergize in combination with alternate immunotherapeutics remains unknown. We have utilized clinically approved immunomodulating therapies in combination with A2A adenosine receptor antagonists in order to identify their synergistic potential in preclinical tumour models. Firstly, we aimed to identify whether antagonism of the A2A adenosine receptor in combination with blockade of immune checkpoint molecules could enhance anti-metastatic activity. Importantly, the immune checkpoint molecules targeted include FDA approved therapies, anti-PD-1 and anti-CTLA-4. We found a significant improvement in metastatic control and prolongation of survival following treatment with these combinations. In particular, the mechanism by which A2A adenosine receptor antagonism and anti-PD-1 elicited anti-metastatic activity was immune-mediated, reliant on NK, T cells and IFN-γ, and to a lesser extent perforin. Following, using a de novo melanoma model, in which tamoxifen-induced aberrant expression of BRAF and PTEN initiates tumourigenesis, we identified that immune cell deletion of the A2A adenosine receptor inhibits tumour initiation and severely impacts on tumour growth. This was confirmed using a transplantable BRAF-mutated melanoma model, in which genetic deletion of the A2A adenosine receptor delayed tumor growth. Currently, we are investigating whether the efficacy of the clinically approved targeted BRAF inhibitor Vemurafenib may be enhanced alongside A2A adenosine receptor antagonism. Of note, use of A2A adenosine receptor antagonists in Parkinson's disease have progressed to late stage clinical trials with minimal adverse events. Therefore, it is feasible that the combination therapies tested within these studies could progress to clinical utility in the treatment of cancer.

asmr Australian Society for Medical Research

54th National Scientific Conference

15-18th November 2015, Stamford Plaza, Adelaide

Bugs, Bowels & Beyond

Innovations in Digestive Health and Disease Research

Our exciting program will bring together leading international and Australian-based scientists, clinicians and health professionals with a wide-ranging focus on conditions affecting the digestive tract.

Themes

Microbiome
Obesity and nutrition
Inflammatory bowel conditions
Gastrointestinal cancers
Pancreatic and liver diseases
Gut, brain and microbiota

Firkin Oration

Professor Eran Elinav

Weizmann Institute of Science, Israel.

Professor Elinav is a clinician and scientist leading a team that investigates interactions between the innate immune system, the intestinal microbiota and their effects on health and disease.

Edwards Oration

Professor Nicholas Talley

Pro Vice Chancellor, Faculty of Health, University of Newcastle

Professor Talley is an icon of Australian health and medical research with over 1,000 publications and recipient of more than $10 million in research funding. His team investigates the molecular basis and treatment of Irritable Bowel Syndrome (IBS), as well as the link between bacteria and dyspepsia, gastroesophageal reflux disease (GORD) and gastritis.

Invited Speakers

Professor Mark Morrison, Microbiome

Dr Vicki Whitehall, Colon Cancer

Dr Ilse Rooman, Pancreatic Cancer

Associate Professor Phil Sutton, Mucosal Immunity

DR ANDREA SCHUESSLER

QIMR Berghofer Medical Research Institute

A new immunotherapy for brain cancer

Glioblastoma multiforme (GBM) is the most aggressive human brain cancer and affects almost 1000 Australians each year. Most patients survive only little more than one year after diagnosis and this grim prognosis has hardly changed despite decades of research into better medications. We have developed a new immunotherapy that aims to help a patient's own immune system fight the cancer more effectively. We tested this new therapy in 10 patients with recurrent GBM and could show that the treatment is absolutely safe and has no major side effects. Our patients survived longer than would be expected after tumour relapse, for example one patient was still alive more than 4 years after therapy. Most importantly, 4 out of 10 patients showed no signs of the disease recurring. This study was an essential first step in the development of new treatments to improve the outlook for patients with brain cancer.

Scientific Abstract:

Glioblastoma multiforme (GBM) is one of the most severe human brain malignancies and invariably fatal due to tumour relapse. Even with optimal treatment, median survival is less than 6 months for patients with recurrent GBM. Improvements to this poor prognosis have remained elusive despite extensive research into new chemotherapies. Immune-based therapies have the potential to improve patient outcome by supplementing standard treatment. Expression of human cytomegalovirus (CMV) antigens in GBM tissues provides the unique opportunity to target viral antigens for GBM therapy. We have completed a formal clinical assessment of the safety and potential clinical efficacy of autologous CMV-specific T-cell therapy as a consolidative treatment for recurrent GBM. From a total of 19 patients with recurrent GBM, CMV-specific T cells were successfully expanded from 13 patients (68.4%), 11 of whom received up to four T-cell infusions. Combination therapy based on T-cell infusion and chemotherapy was well tolerated, and we detected only minor adverse events. The overall survival of these patients since first recurrence ranged from 133 to 2,498 days, with a median overall survival of 403 days. Most importantly, 4 of 10 patients who completed the treatment remained progression free during the study period. Furthermore, molecular profiling of CMV-specific T-cell therapy from these patients revealed distinct gene expression signatures, which correlated with their clinical response. Our study suggested that a combination therapy with autologous CMV-specific T cells and chemotherapy is a safe novel treatment option and may offer clinical benefit for patients with recurrent GBM. Building on these initial findings, I am currently developing novel immunoprofiling strategies to identify markers in the tumour microenvironment with prognostic significance. The identification of such biomarkers will be essential in advancing innovative immunotherapies for brain cancer and bring us closer to the ultimate goal of improving the life expectancy of patients.

DR AMIRALI POPAT

University of Queensland

Programmable Nanoparticles for the treatment of Inflammatory Bowel Disease

Inflammatory Bowel Disease (IBD) is affecting over 2.2 million people in Europe, 1.4 million people in USA and around 70,000 Australians with an annual net disease burden of $2.7 billion on the Australian economy. A key challenge in current treatment for IBD is achieving localised delivery of drugs, reducing effective systemic dose and side effects associated with non-targeted therapy. The overarching aim of this research is to target intestinal inflammation using programmable nanoparticles and to understand the mechanism of the same. Our results suggest that these smart nanoparticles can release anti-inflammatory drugs in the colon avoiding systemic side effects and improving bioavailability of these drugs. Outcomes of this research can lead to improved therapeutic efficacy and has the potential to have a large impact on the quality of life of millions of patients suffering from IBD and other colonic disease such as colon cancer around the world.

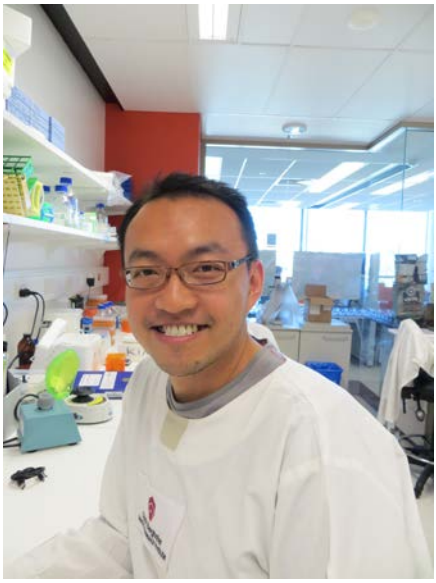
Scientific Abstract:

Smart nanomaterials delivering cargo at the targeted site with zero premature release is an enchanting area of translational pharmaceutical research. Traditional nanomaterials made from polymers and lipids suffer from many disadvantages such as premature drug release, stability issues in complex biological environment and low encapsulation capacity. In the past decade, mesoporous silica nanoparticles (MSNs) have been in the limelight as one class of the most promising nanocarriers owing to their biocompatibility, enormous loading capacity, and adaptable surface chemistry, just to name a few. In this study, we propose the first example of programmable drug delivery system based on MSNs using a programmable grate using structurally modified soy protein isolate (SPI) which is commonly available dietary supplement. First we have adsorbed large amount of drug on to the 3D cubic pores of highly ordered MSNs with small particle size (~150 nm) and high surface area (~1300 m2/g). Later this drug loaded MSNs are coated with multi stimulie protein called soy protein isolate (SPI) to achieve stepwise pH and Enzyme responsive release in simulated gastrointestinal fluid. The particles show an extremely high pro-drug loading with programmable drug release in simulated gastrointestinal fluid in different pH and enzymatic conditions. Additionally, we have found that nanoparticles with specific size and functional group can be selectively taken up by macrophages (representing inflamed cells in-vivo) in in-vitro assays. This unique and precisely programmed oral drug delivery system will be useful for targeting the small intestine or the colon, which is the absorption site of most orally delivered drugs. We are currently testing its in-vivo distribution and targeting in various IBD animal models. The research project aims address important problems in the area of targeted medicine and the knowledge generated could potentially be extrapolated to a variety of disease associated with gastro-intestinal tract.

DR SHIN FOONG NGIOW

QIMR Berghofer Medical Research Institute

A threshold level of intratumor CD8+ T cell PD1 expression dictates therapeutic response to anti-PD1



Tumors hijack immune regulatory mechanisms, known as immune checkpoint pathways to suppress anti-tumor immunity, facilitating tumor progression. Excitingly, these immune checkpoint pathways can be blocked using specific monoclonal antibodies (mAbs), reactivating the host anti- tumor immunity to combat the disease. The recently approved anti-PD1 mAbs (Pembrolizumab and Nivolumab) are the revolutionising immunotherapy agents of this class, currently used to treat melanoma and non-small cell lung cancer patients. Unfortunately, while some patients are presented as exceptional responders, a proportion of the anti-PD1-treated patients with various malignancies do not respond. This new survival profile now raises questions about how to predict and increase the number of patients who receive long-term clinical benefit from PD1-targeted therapy. However, it remains hard to study the differences between responder and non-responder in patients. Using mouse tumor models, we have now determined which tumor types are responsive to anti-PD1 therapy.

Scientific Abstract:

Immune checkpoint molecules refer to a group of immune receptors, that when engaged by their ligands, transmit an inhibitory signal to suppress immune effector function. We now appreciate cancer can use these pathways to evade tumor immunity. Notably, by inhibiting immune checkpoint pathways like programmed death 1 (PD1) using specific monoclonal antibodies (mAbs), we now observed variable, but impressive, levels of clinical responses in a broad spectrum of cancer patients. Anti-PD1 mAbs are currently FDA-approved for the treatment of advanced melanoma and non-small cell lung cancer, fast tracking its application to treat other malignancies. Unfortunately, a proportion of the anti-PD1-treated patients of various malignancies were presented with insignificant clinical response. This new survival profile now raises questions about how to predict and increase the number of patients who receive long-term clinical benefit from anti-PD1 therapy. Recent studies in patients reported that the presence of CD8 T cells, PD1 and PDL1 expressions are positive predictive biomarkers for anti-PD1 therapy. Here, we have refined the proposed biomarker profile with even robust immune signatures to predict the treatment outcome of anti-PD1 mAb. We showed an inverse correlation of PD1 and PDL1 expressions in the tumor microenvironment by comparing anti-PD1-sensitive to resistant tumors. We demonstrated that the expression levels of T cell PD1 (^{hi} and ^{lo}) and immune cell PDL1 (^{hi} and ^{lo}) in the tumor microenvironment dictated the efficacy of anti-PD1 mAb. We revealed that certain threshold for the PD1 downregulation is achieved for the release of adaptive immune resistance. Mechanistically, an induction of intratumoral Treg is partly responsive to the development of anti-PD1 resistant tumor. In light of this, our study also provided translational approaches to treat anti-PD1-resistant tumors.

Health and Medical Research investment strategy for reducing future Australian health expenditure[1]

The Australian Society for Medical Research

- The Australian Society for Medical Research (ASMR) is the peak professional body representing Australia's 24,000 health and medical research (HMR) workforce.
- Established in 1961, the Society promotes HMR in all its aspects through public, political and scientific advocacy.
- ASMR is committed to clearly articulated, evidenced-based political advocacy; in the past decade (2001 and 2006) playing a key role in two doublings of investment in National Health and Medical Research Council (NHMRC).

Australia faces unprecedented health and economic challenges

- Health and Ageing expenditure: currently >¼ of total Government spend increasing to almost ½ of total spend by 2049–50.[1]
- Total health and residential aged care expenditure escalating from 9.3% GDP in 2003 to 12.4% GDP in 2033.[2]
- Health system expenditure to grow from $113 billion in 2012 to $3.3 trillion by 2062.[3]

Economic benefit of health and medical research

- HMR between 1993 and 2005 is estimated to have returned a net benefit of $29.5 billion.[4]
- Every dollar invested in HMR returns on average $2.17 in health benefits.[4]
- Australian HMR returns 117%, exceeded only by mining (159%) and wholesale/retail (438%) sectors.[4]

ASMR's request to government

- **Lift investment into the NHMRC to 3% of the total health expenditure in Australia by 2023.[5]**

Exceptional returns on investment into NHMRC

- Investment in NHMRC between 2000–10 is projected to save $966 million in direct/indirect costs to the health system.[6]
- Gains of $6 billion linked to increased well-being from NHMRC investment between 2000–10.[6]
- Increasing investment in NHMRC to 3% of total health spend has a conservative saving of $25.9 billion to the Australian economy.[3]

Support ASMR's case for Australian Health and Medical Research.

This investment will feed directly into more cost effective and efficient treatment of patients.
It will build knowledge for prevention, intervention and innovation.
It will reduce the predicted unsustainable escalation of health spending, safeguarding both the community and government.

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DR SHYUAN NGO (UQ)

University of Queensland

Fuelling the fight against neurodegenerative disease: translational studies in motor neuron disease

Nerve cells (neurons) in the brain are normally lost as we age. The unnatural loss of these cells however, is a hallmark of diseases like Motor Neuron Disease (MND). In MND, the loss of neurons in the brain and spinal cord leads to muscle paralysis and death. Research from my laboratory suggests that glucose is not properly used to generate energy in the brains and muscles of mouse models of MND. This change in how the body generates energy might starve the neurons and muscle cells, leading to their death. Using a chemical compound to improve glucose use, we have delayed the disease in our mice. We are now studying how changes in the way the body generates energy can affect how quickly the disease progresses in MND patients. Thus, our studies have the potential to treat diseases that occur due to the loss of neurons, including MND.

Scientific Abstract:

Motor Neuron Disease (MND) is a fatal neurodegenerative disease for which there is no cure. For most patients, death occurs within 2–5 years of diagnosis. While the primary pathology in MND is the death of motor neurons, there is evidence that defective energy metabolism affects disease pathogenesis. Moreover, increased body mass index and high calorie supplementation is associated with improved prognosis in MND patients. Thus, we hypothesise that in MND, the state of metabolic flux modifies disease course, and modulation of metabolic homeostasis will aid in sustaining survival. We have studied mouse models and human subjects to investigate how altered metabolic homeostasis impacts disease outcome.

We have found that defects in glucose metabolism occur in the brains and muscle of mouse models of MND. In muscle, this drives a switch towards the use of fat as an energy substrate. Because metabolic flux underpins the generation of energy to maintain cell survival, we have developed targeted strategies to ameliorate defective glucose metabolism, with the aim to prevent the catastrophic degeneration of neurons and progressive muscle pathology in MND. By orally administering a chemical compound to recover glycolytic capacity, we have successfully prevented metabolic defects and have improved the motor deficit that is associated with neuronal death in MND mice.

To translate observations in mice to humans, we have studied subjects with MND. Analysis of muscle biopsies reveals defects in glucose metabolism in MND patients. Moreover, MND patients have altered expression of circulating proteins that demonstrate metabolic responses that match altered demand on fat as an energy substrate. Our studies not only establish the disease-promoting role of defective energy metabolism in MND, but also pave the way for future clinical testing of novel therapeutics aimed at improving metabolic capacity to prevent the death of neurons and muscle in neurodegenerative diseases.

DR SARAH MEDLAND

QIMR Berghofer Medical Research Institute

Common genetic variants influence human subcortical brain structures

At the individual level, genetic variations exert lasting influences on brain structures and functions associated with behaviour and predisposition to disease. Within the context of the Enhancing Neuro Imaging Genetics through Meta-Analysis (ENIGMA) Consortium, we conduct collaborative large-scale genetic analysis of magnetic resonance imaging (MRI) scans to identify genetic variants that influence brain structure. Our recent findings will be discussed focusing on genetic variants influencing volumetric measures derived from a measure of head size (intracranial volume, ICV) and seven subcortical brain structures corrected for ICV (nucleus accumbens, caudate, putamen, pallidum, amygdala, hippocampus, and thalamus). In addition, we will present the first findings from joint meta-analyses of ICV and hippocampal volume from 26,577 participants from the ENIGMA and CHARGE cohorts. These new analyses have identified novel variants influencing brain structure and have revealed substantial overlap between genetic variants influencing hippocampal volume and those influencing risk for Alzheimer's disease.

Scientific Abstract:

At the individual level, genetic variations exert lasting influences on brain structures and functions associated with behaviour and predisposition to disease. Subcortical brain regions form circuits with cortical areas to coordinate movement, learning, memory, and motivation, and altered circuits can lead to abnormal behaviour and disease. Identification of these genetic variants provides insight into the causes of variability in human brain development, and help elucidate mechanisms of neurological and psychiatric dysfunction.

Within the context of the Enhancing Neuro Imaging Genetics through Meta-Analysis (ENIGMA) Consortium, we conduct collaborative large-scale genetic analysis of magnetic resonance imaging (MRI) scans to identify genetic variants that influence brain structure. In 2014 we conducted genome-wide association studies (GWAS) of the volumes of seven subcortical regions and intracranial volume derived from MRI scans of individuals from 50 cohorts (N discovery =13,688; N replication =17,029). We identified five novel genetic variants influencing the volumes of the putamen and caudate nucleus. We also found stronger evidence for three loci with previously established influence on hippocampal volume and intracranial volume. These variants showed specific volumetric effects on brain structures rather than global effects across structures. The strongest effects were found for putamen, where a novel intergenic locus with replicable influence on volume (rs945270; P=1.08x10-33; 0.52% variance explained) showed evidence of altering the expression of the KTN1 gene in both brain and blood tissue. Variants influencing putamen volume clustered near developmental genes that regulate apoptosis, axon guidance, and vesicle transport.

In addition, we will present the first findings from joint meta-analyses of ICV and hippocampal volume from participants from the ENIGMA and CHARGE cohorts, yielding a discovery sample of 26,577 individuals. These new analyses have identified novel variants influencing brain structure and have revealed substantial overlap between genetic variants influencing hippocampal volume and those influencing risk for Alzheimer's disease.

DR KYLE UPTON

Mater Medical Institute

Single cell genomics demonstrates somatic mosaicism in development and disease

Mobile Genetic Elements (MGEs) are DNA sequences which use a copy and paste mechanism to replicate within our DNA, generating mutations which can cause genetic disease. Understanding the impacts of this process has been limited by technical difficulties in identifying mutations. To overcome this obstacle, I have played a lead role in the development and application of a sensitive and accurate technique to map MGE mutations. Using this technique, I have previously demonstrated that MGE mutations occur extensively as part of normal brain development, and contribute to tumour evolution. To better understand the prevalence and impact of MGE mutations, I have developed single cell DNA sequencing techniques. I have now demonstrated that MGEs generate a large number of mutations in all neurons in normal development, and are likely to impact neuronal function. I am currently investigating the role of MGE mutations in driving tumour evolution and chemoresistance in ovarian cancer.

Scientific Abstract:

Retrotransposons are mobile genetic elements which use a copy and paste mechanism to replicate, and comprise more than 40% of the human genome. Of the millions of retrotransposon elements in the human genome, fewer than 0.1% remain active, with as few as six believed to drive the majority of new mobilisation events. Nonetheless they remain a powerful and underappreciated source of insertional mutagenesis. Over 100 instances of genetic disease have been linked to retrotransposon insertions, however new insertions are difficult to identify and their prevalence is greatly underestimated. To this end, I have played a lead role in the development of a novel high throughput sequencing technique to identify retrotransposon insertions in the human genome (RC-seq). Using RC-seq I am defining the effects of retrotransposon mutagenesis in disease and development. I have demonstrated retrotransposon mobilisation is very rare in most healthy somatic tissues, with the stark exception of the brain. In 2011 I published a landmark first-author paper in Nature demonstrating prolific retrotransposon mobilisation in the human brain. I have recently extended this work by developing single cell genomics techniques which can be used in combination with RC-seq. In a recent first author publication in Cell I have demonstrated that MGEs mobilise in all human neurons of the cortex and hippocampus. Further, these insertions are likely to have functional implications affecting neuronal phenotype. While somatic mosaicism is an unexpected feature of normal brain development, it is also a well-known feature of many cancers. I am currently applying single cell sequencing to understand the mosaicism present in ovarian cancers, and how this genomic diversity contributes to prolific chemoresistance in this disease.

The Queensland branch of

The Australian Society for Medical Research

is proud to announce

THE ASMR MEDICAL RESEARCH WEEK® GALA DINNER 2015

Share and enjoy the fellowship of the Queensland medical research community.

Keynote speaker and ASMR 2015 Medallist

Professor Ashok Saluja

The ASMR medal will recognise Professor Saluja's outstanding work on pancreatic diseases and the huge impact his findings have had on science and medical research. Professor Saluja is committed to finding a cure for pancreatic cancer as it is one of the most lethal cancers. One of many highlights of Professor Saluja's career was his discovery that a stress-induced protein protects pancreatic cancer cells from death. His group developed a small molecule to inhibit this protein, Minnelide™, which kills tumors in several models of pancreatic cancer in mice and is currently being tested in Phase 1 clinical trials in human patients with pancreatic cancer.

Presentation of the finalists and winners of the ASMR QLD Health and Medical Research Awards

FRIDAY 29TH MAY 2015

BRISBANE CONVENTION & EXHIBITION CENTRE (SKY ROOM)

SOUTH BANK, BRISBANE

DOORS OPEN AT 7PM FOR 7.30PM START

$135 PER ATTENDEE ($90 FOR STUDENTS)

Dinner registrations are now open, please visit:
http://www.asmr.org.au/MRWQld.html

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Enquiries about this event are to be directed to the ASMR QLD Gala Dinner Sub-Committee
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DR YOGAVIJAYAN KANDASAMY

The Townsville Hospital & The University of Newcastle

The effects of preterm birth on renal development

The World Health Organization estimates that every year 15 million babies are born premature and this number is rising. Three-quarters of these babies could be saved with current, cost effective interventions. In Australia, approximately 25,000 babies (8%) are born premature each year. Survival of these babies has dramatically improved and majority of them now survive to adulthood but often with long-term health complications. Recent research indicates that these babies may also have an increased risk of developing kidney diseases as adults. We therefore carried out a study to determine the effects of prematurity on kidney growth in a group of premature babies admitted to a specialist baby unit. From this study, we found that prematurity decreases kidney growth and kidney function compared to full term babies. We concluded that these babies need to be followed regularly to monitor the kidney growth and function.

Scientific Abstract:

Background: Nephrogenesis in a human fetus is completed in utero by 36 weeks of gestation. Approximately 8% of the babies born in Australia are born premature (before 37 weeks). Animal and human studies indicate that prematurity, independent of birth weight, results in abnormal renal development and predispose adults to the development of chronic kidney disease. The objective of this study was to investigate the effects of preterm birth on extra uterine renal growth and function.

Methods: Data was collected as part of an ongoing observational study currently being conducted in a tertiary perinatal center. Preterm babies less than 32 weeks of gestation were recruited and followed until discharge. Term infants were recruited for comparison. The babies underwent renal sonography and measurements of renal function at 38 weeks corrected age. Total kidney volume was measured as a surrogate for nephron numbers and estimated glomerular filtration rate (eGFR) was calculated by replacing creatinine with Cystatin C.

Results: A total of 88 babies were part of this study (52 preterm and 36 term). The mean birth weight was 1067± 324 g and the mean gestational age was 27.5 ± 2.3 weeks. The median corrected gestational age for the preterm babies when comparison was carried out with term babies (control) was 38 [37.3- 38.3] weeks. At term corrected age, preterm babies had smaller total kidney volume (21.2 ± 5.5 vs.24.6 ± 6.2 cm3; P = 0.009) and lower eGFR (49.4 [44.5 - 56.4] vs. 53.4 [47.6 - 67.4] mL/min/1.73 m2; P = 0.027) respectively. Preterm babies were also smaller compared to their term counterparts (2532 ± 388 vs.3380 ± 450 g; P = 0.001)

Conclusion: Premature birth causes oligonephronia. Premature infants have smaller total kidney volume and likely decreased nephron number and lower eGFR relative to infants born at term. We propose that current clinical practice for follow-up of premature babies should include regular assessment of renal function.

DR CHRISTOPHER CARTY

QLD Children's Gait Laboratory & Griffith University

Informing medical intervention for movement disorders using innovative musculoskeletal modelling technology

Throughout my career I have contributed to medical research by applying engineering principles in the clinical management of patients with movement disorders. These patients include young adults with bone cancer, older adults with an increased falls risk and children with cerebral palsy. Currently I am appointed in a consultant clinical role in the Queensland Children's Gait Laboratory and my vision is to use computer modelling techniques to identify optimal surgical interventions for the paediatric population. My current projects are focused on incorporating medical images from MRI with 3D motion of children during walking. The ultimate purpose of these projects is to predict outcomes from surgery using 'virtual surgery' simulations. This will allow the surgical team to have access to better information for surgical decision making for children who require surgery and also will assist in determining that those children who do not need surgery will not be subjected to surgery.

Scientific Abstract:

The human neuromusculoskeletal system is an impeccably designed machine. Unfortunately, due to various factors including congenital abnormalities, disease and/or injury the integrity of this machine can become compromised. Throughout my career I have contributed to medical research by applying engineering principles to objectively assess movement disorders, which have enabled me to develop informed and appropriate clinical management recommendations for patients across the lifespan. To provide two examples: (1) throughout my PhD at the University of Queensland I assessed the outcome of surgical interventions for children with bone cancer of the lower limb. The application of 3D rigid body modelling identified important walking impairments that would not have been identified using current clinical management. The results of this project allowed better recommendations for post-operative rehabilitation and joint replacement design. (2) During my post-doctoral research fellowship at Griffith University I used inverse kinematic modelling techniques to determine the major biomechanical factors that contribute to falls in older adults allowing development of targeted preventative recommendations for reducing the incidence of falls. Currently, I am appointed in a consultant clinical role in the Queensland Children's Gait Laboratory (50%) and a research role in the Centre for Musculoskeletal research at Griffith University (50%). The research vision of my team is to use advanced modelling techniques to best identify surgical interventions for the paediatric population. Current projects are focused on merging medical images from MRI with 3D motion capture in ambulatory patients with patellofemoral joint pain and cerebral palsy. My intention would be to use this award to progress an exciting field of research focused on 'virtual orthopaedic surgery'. The significance of this project will be that the surgical team will have access to better information for surgical decision making, which will also assist in determining the children with movement disorders who do not need surgery.

DR ALWYN TODD

Griffith University

The effect of dietary sodium modification on blood pressure in studies of subjects with systolic blood pressure less than 140mmHg: A systematic review of quantitative evidence

In healthy individuals with normal blood pressure excess dietary salt is excreted by the kidney. Through this process normal blood pressure can be maintained despite changes in dietary salt intake. However, in the presence of hypertension (high blood pressure), blood pressure may rise in response to salt intake as the kidney is not able to effectively get rid of excess salt. The Australian Dietary Guidelines currently state that sodium reduction can change blood pressure in healthy individuals with normal blood pressure. This statement is based on analyses of data including both healthy subjects with normal blood pressure and some subjects with hypertension. This paper has analysed data from studies that only include healthy individuals with normal blood pressure (not individuals with hypertension), and found that blood pressure does not change by more than 1% when dietary sodium intake is varied.

Scientific Abstract:

Background: The pressure–natriuresis relationship described by Guyton links dietary sodium intake and renal sodium handling. In a normotensive individual, consumption of dietary sodium will elicit a transient rise in blood pressure (BP) that stimulates the kidney to excrete excess sodium, leading to restoration of normal BP. Intervention trials in normotensive subjects should therefore observe minimal variation in BP with changes to dietary sodium. Previous trials have, however, reported a wide range of response (systolic BP [SBP] change -1mmHg to 8.2mmHg), due to changing definitions of normotension, and inclusion of subjects with SBP≥140mmHg. Existing meta-analyses have not accounted for this, and estimates of normotensive BP response from these analyses are inaccurate.

Objectives: To identify the effect of sodium modification on BP and arterial function in normotensive subjects with SBP<140mmHg.

Search methods: We searched MEDLINE, CINAHL, PROQUEST, Scopus, EMBASE, Cochrane Library, Wiley InterScience, the reference lists of relevant articles, grey literature and clinical trial registrars.

Selection criteria: We included randomised controlled trials and non-randomised controlled trials with a minimum duration of 4-weeks in subjects with SBP<140mmHg.

Data collection and analysis: Methodological validity was assessed using the Joanna Briggs Institute Meta-Analysis of Statistics Assessment and Review Instrument. Quantitative data were extracted independently by two reviewers. Meta-analysis was conducted using a random-effect model.

Main results: Five trials (n=1214) were included, the mean change in urinary sodium was - 74.5mmol/24hrs (-4.4grams/24hrs). Meta-analysis found that SBP was maintained within 1% of subjects baseline blood pressure readings after dietary sodium modification (p>0.05). Two trials studied arterial function by pulse wave velocity, the pooled effect was insignificant (+0.57m/s, 95%CI: -0.86, 2.00, p>0.05, I^2=22%).

Conclusion: Normotensive subjects effectively respond to changes in sodium intake by regulating BP. Reducing dietary sodium may be cardio-protective independent of the BP pathway, however further studies on arterial function and cardiovascular risk are required.

ASMR Medical Research Week®

ASMR Medallist 2015 – Professor Ashok K. Saluja

A Champion for Translation from Bench to Bedside

Dr. Ashok Saluja obtained his B.Sc. and M.Sc. in Punjab, India before moving to the US where he obtained his doctoral degree in Biochemistry from Washington State University and completed his post doc at Cornell. He then spent twenty years at Harvard, after which he joined University of Massachusetts Medical School. In 2006, Dr. Saluja joined the faculty of the Department of Surgery at the University of Minnesota Medical School as Professor and Vice Chair. He also holds the Sit Family Chair in Pancreatic and GI Cancer Research as well as a University of Minnesota McKnight Presidential Endowed Chair.

The American Cancer Society's has estimated that around 49,000 people in the US alone will be diagnosed with pancreatic cancer in 2015. Dr Saluja is internationally renowned for his work on the pathogenesis of pancreatitis. His group has shown that pancreatic tumors overexpress Heat Shock Protein 70 and its inhibition causes death of these cells not only in in vitro settings, but also in mouse models of pancreatic cancer. Dr Saluja's group has been successful in developing a small molecule which they have named Minnelide, a drug extracted from the lei gong ten or 'thunder god vine' (Tripterygium wilfordii). The compound is effective in several models of pancreatic cancer and entered Phase I clinical trials in 2013.

Dr Saluja said about his work on this most aggressive of cancers, *"I start imagining that one day, and the one day in my lifetime is very soon," he said. "Maybe this curse of pancreatic cancer will be over."*

Dr. Saluja has published more than 120 original research papers along with review articles and book chapters. He is an inventor on two patents and is also Chief Scientific Officer and co-founder of a start-up biotechnology company.

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ASMR QUEENSLAND HEALTH & MEDICAL RESEARCH AWARDS 2015

Proudly supported by the Queensland Government

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These prestigious Awards recognise excellence in all areas of health-related research performed in Queensland.

1. Post-graduate Student Award.

Eligibility - enrolled students and within one year of completing a higher degree.
Prize - winner $1500, each finalist runner-up $200

2. Post-doctoral Researcher Award.

Eligibility - between one and five years (inclusive) of completing a higher degree.
Prize - winner $2000, each finalist runner-up $500

3. Senior Researcher Award.

Eligibility - between six and ten years (inclusive) of completing a higher degree.
Prize - winner $3000, each finalist runner-up $500

4. Clinical Researcher Award.

Eligibility – clinicians within ten years (inclusive) of completing a higher degree/specialist training. Prize - winner $3000, each finalist runner-up $500

Application Deadline: Friday 17th April 2015 (5pm)

Please see the ASMR website for complete information on eligibility and application instructions: http://www.asmr.org.au/MRWQld.html

Finalists in the Post-doctoral (3), Senior (3) and Clinical Researcher (3) Award categories will be selected to give oral presentations at the QIMR Berghofer Medical Research Institute on **Monday 25th May 2015**.

Finalists for the Post-graduate Student Award (6) will be selected to give oral presentations at the Post-Graduate Student event at the QIMR Berghofer Medical Research Institute on **Wednesday 27th May 2015**

Presentations to the finalists and winners will be made during the ASMR Medical Research Week® Dinner on **Friday 29th May 2015**.

Please direct enquiries about these awards to the Convenors of the ASMR Queensland Awards for Health & Medical Research Sub-Committee (**Antonia Pritchard** (chair): antonia.pritchard@qimrberghofer.edu.au, **Michael Weible**: m.weible@griffith.edu.au or **Olga Panagiotopoulou**: o.panagiotopoulou@uq.edu.au).

The Queensland Government is the Principal Sponsor of the Queensland Health and Medical Research Awards

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Health and Medical Research, within the Preventive Health Unit, Health Service and Clinical Innovation Division, Department of Health, provides leadership for Queensland Health in the advancement of Queensland Health's research expertise and translation of research outcomes to better healthcare for Queenslanders.

Health and Medical Research inputs into national initiatives to advance research (e.g. National Health and Medical Research Council projects to increase clinical trials) and coordinates Queensland Health's input into State Government initiatives (e.g. Queensland's Science and Innovation Action Plan and projects led by the Department of Science, Information Technology and Innovation).

Health and Medical Research oversees state-wide policy for research ethics review and governance, supports researchers through a fellowship program, and provides assistance with knowledge transfer and realising the health, commercial and social benefits of research outcomes. It is also responsible for services such as the provision and monitoring of approvals for using confidential health information for research under the *Public Health Act 2005 (Qld).*

For more information visit
www.health.qld.gov.au/ohmr/default.asp.

About the 2015 ASMR Queensland Postgraduate Student Conference

As part of Australian Society for Medical Research Medical Research Week®, ASMR Queensland holds a student conference to showcase the high quality of research undertaken by students in universities and other institutions. The work presented at this conference is that of postgraduate students and the conference is well attended by a broad range of researchers and academics. The 2015 ASMR Queensland Postgraduate Student Conference will take place at the QIMR Berghofer Medical Research Institute on May 27th 2015.

As well as having many students present their work as oral and poster presentations, the conference is held in conjunction with the Postgraduate Student category of the ASMR Health and Medical Research Awards. This year the six finalists for this award are: Jonathon Fanning (Prince Charles Hospital), Shani Stuart (QUT), Mitchell Stark (QIMR Berghofer), Gillian Fisher (Griffith University), Laure Martine (QUT), Arabella Young (QIMR Berghofer).

The organising committee would like to take this opportunity to thank our keynote speakers, Dr Steven Lane (QIMR Berghofer), Professor Maree Smith (UQ School of Pharmacy), and Professor Lyn Griffiths (QUT, Institute of Health Biomedical Innovation) for sharing their science and experience with the young researchers of Queensland.

We would also like to show our appreciation to all of our judges, sponsors, presenters, supervisors and registrants; without you, this conference would not be possible.

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