The Australian Society for Medical Research

2019 QUEENSLAND HEALTH & MEDICAL RESEARCH AWARDS

2019 ASMR Queensland Health and Medical Research Awards is proudly supported by the Queensland Government

asmr Australian Society for Medical Research

Public, Political, Scientific Advocacy

Empowering research for health and well-being

A vision for a healthy and equitable Australia

The current state of the health and medical research workforce

Between 2012 and 2017 the NHMRC full-time workforce declined by

20%

At the end of 2016, 1 in 4 Australian researchers did not have job security for 2017

Investing in health and medical research, through the National Health and Medical Research Council (NHMRC), will drive economic growth, reduce healthcare costs and enable Australians to prosper...

ASMR's vision for the future

Incrementally increasing investment into NHMRC to reach 3% of total health expenditure by 2025/26 will generate $58 billion in health and economic benefits

Investing in the health and medical research workforce...

Every $1 invested into the NHMRC supported workforce returns $3.20 in health and economic benefits

Increasing the NHMRC supported workforce by 40% could lead to net gains of $35 billion

$35 billion

Ongoing expansion to the science and innovation economy

In an ageing population, investment into health and medical research will underpin a healthier, more productive Australia, reducing the projected spend on health and aged care

WWW.ASMR.ORG.AU

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Foreword

Queensland Health is once again proud to support the ASMR Queensland Health and Medical Research Awards in 2019.

Health and medical research and innovation plays a crucial role in improving health outcomes by finding new and better ways to deliver care and prevent, diagnose and treat disease. As such, it is a critical part of a world class healthcare system. The importance of research is recognised by our *Queensland Advancing Health Research 2026* Strategy, which is currently being implemented and supported by an additional $10 million investment in research.

A key action toward achieving the Strategy's vision of healthier Queenslanders through research-informed healthcare was the development and launch of the new $6 million *Queensland Advancing Clinical Research Fellowships* program earlier this year.

The program supports eligible Queensland Health clinician researchers to undertake research linked to their practice and includes multiple categories, catering for different levels of research experience, so there is a pipeline of opportunities. The Fellowships will support research that will lead to better health outcomes for Queenslanders, while building competitiveness for national funding and strengthening collaborative linkages between Queensland Health and the wider Queensland health and medical research community.

The ASMR Queensland Health and Medical Research Awards showcase and celebrate the achievements of some of Queensland's leading and emerging health and medical researchers. Congratulations to all the award finalists and a sincere thank you to the broader health and medical research community for the valuable contribution you make to improving Queensland's healthcare.

Michael Walsh
Director-General, Queensland Health

Table of Contents

Queensland Health and Medical Research Awards Seminar Schedule

8:30 am	**Opening Remarks** Dr Gautam Rishi
8:45 – 9:30 am	**Postgraduate Researcher Presentations** **Chair:** Dr Ran Wang Ms Marietta Landgraf – Page 09 Ms Jessica Sexton – Page 11 Dr Shuichi Suetani – Page 13
9:30 – 10:15 am	**Early-Career Researcher Presentations** **Chair:** Dr Ran Wang Dr Jamie Kutasovic – Page 19 Dr Tianqing Liu – Page **Error! Bookmark not defined.** Dr Abu Sina – Page 23
10:15 – 10:45 am	*Morning tea*
10:45 – 11:30 am	**Mid-Career Researcher Presentations** **Chair:** Dr Jay Gunawardana Dr Emanuele Pelosi – Page 27 Dr Ronan Kapetanovic – Page 29 Dr Denuja Karunakaran – Page 31
11:30 – 12:15 pm	**Clinical Researcher Presentations** **Chair:** Dr Jay Gunawardana Mr Joshua Tobin – Page **Error! Bookmark not defined.** Ms Lisa Gillinder – Page 37 Dan Siskind – Page 39
12:15 pm	**Closing Remarks** Dr Jay Gunawardana
12:20 pm	*Judges Meeting*

ASMR Medallist 2019 – Dr Elizabeth Finkel

Dr Elizabeth Finkel is an award winning Australian science journalist with a background in laboratory research. Dr Finkel is not a stranger to the ASMR. In 1982 she was the Campion Ma Playoust Award winner, an award for the best presentation of original research at the National Conference by an early career scientist. Now, in 2019, ASMR recognises Dr Finkel's impressive contributions to science communication as the ASMR Medallist. After being awarded her PhD in Biochemistry from the University of Melbourne, Dr Finkel subsequently pursued a research career at the University of California, San Francisco. During this time, her investigation of the genes that sculpt a fruit fly egg into an embryo were published in *Nature*.

Upon returning to Melbourne she turned to freelance journalism, and since then has written for *Science, Lancet, Nature Medicine, New Scientist, The Age* and *The Monthly* among others, and has also broadcast for ABC Radio National. In 2005 Dr Finkel co-founded the popular science magazine, *Cosmos*, and from 2013 to 2018, served as Editor in Chief.

Dr Finkel also edited the 2012 edition of the Best Australian Science Writing. And she has written two books: 'Stem Cells: Controversy at the Frontiers of Science' which not only provides a clear lay explanation of just what stem cells are, but why they are important for medical research and how Australia found itself in the forefront of stem cell research. Her last book 'The Genome Generation' which covers genetic developments in diverse areas such as medicine, agriculture, and evolution, clearly contextualises their relevant applications to our society.

Elizabeth has received numerous awards for her journalism, including a Michael Daley Award for Best Radio Feature Broadcast, the Queensland Premier's Literary Award, four Publishers Australia Excellence Awards and the National Press Club's Higher Education Journalist of the Year Award. Her story "Fields of Plenty" for Cosmos Magazine won the Crawford Prize for agricultural journalism and more recently, she won the Department of Industry and Science Eureka Prize for Science Journalism for her Cosmos article "A Statin a day" – the first print article to win the award in 11 years. Notably she also received a Member of the Order (AM) for her work in science communication and support of a range of not-for-profit organisations.

Dr Finkel is not afraid to tackle complex and controversial issues. She has interrogated the promise of the stem cell revolution, the human genome project and gene therapy, delved into the conflict around GM crops and organic agriculture, and not shied from fierce debates around the use of statins to prevent heart disease or cannabis as a modern-day panacea.

Speaking of the team at *Cosmos*, Dr Finkel has said "We are a troupe of like-minded souls, passionate about journalism and sharing the wonders of science with the world." And in her final Editor's note for Cosmos, "Rational, evidence-based discourse is under threat everywhere. Our mission as science journalists has never been more poignant."

Public, Political, Scientific Advocacy

ASMR Research Fund

You can help build strength in Australia's Health and Medical Research Sector

The Australian Society for Medical Research is the peak body representing and supporting Australian Health and Medical Researchers. Supporting and encouraging early career researchers is an essential element in building and maintaining the Health and Medical Research sector in Australia.

In order to facilitate and promote learning and collaboration in early career researchers, the **ASMR offers two Research Awards each year**, enabling early career researchers to travel to another laboratory either within Australia (Domestic Research Award) or overseas (International Research Award) to learn and diversify their skills and networks.

The ASMR Research Awards are made possible by the generosity of ASMR members who make donations to the ASMR Research Fund.

The ASMR Research Fund was established by ASMR with the sole purpose of providing research awards to ASMR members, in particular early career members.

Without continued generosity, ASMR would not be able to continue to build strength and expertise in Australian Health and Medical Researchers via its support of early career researchers with the ASMR Research Awards.

If you would like to make a contribution to the ASMR Research Fund, and support the development of Australian Health and Medical Researchers, please follow link below.

https://asmr.org.au/research-awards/

Note: all donations to the ASMR Research Fund are tax deductible

Postgraduate Researcher Award Finalists

MS ALEENA WOJCIESZEK

Mater Research Institute
University of Queensland

Care in Pregnancies after Stillbirth: Generating Evidence to Inform Clinical Practice

Aleena is a researcher at the National Health and Medical Research Council (NHMRC) Centre of Research Excellence in Stillbirth (Stillbirth CRE). Her research background is in health psychology and clinical perinatal epidemiology specific to reproductive health and decision-making. Her current research is focused on implementation science in the context of perinatal health, particularly improving mothers' and babies' health and preventing adverse pregnancy outcomes such as stillbirth. Aleena recently submitted her Ph.D. thesis on informing clinical practice for care in pregnancies after stillbirth, and she co-leads the Stillbirth CRE's pregnancy after stillbirth research stream.

Scientific Abstract:

Background: Stillbirth is a devastating outcome of pregnancy with lasting impacts on families. Parents who embark on a subsequent pregnancy have an increased risk of stillbirth and of various pregnancy complications, and their pregnancies are frequently laden with anxiety. While it is clear that expectant parents who have had a stillborn baby comprise a distinct group with unique medical and psychosocial needs, there is currently scant evidence to support optimal care.

Methods: A range of methodologies were employed as part of this broad-ranging foundational work. These included an international survey of parents to explore current patterns and variations in pregnancy-after-stillbirth care, and Cochrane Systematic Reviews of the available evidence to underpin this care. Additionally, stillbirth researchers and advocates were surveyed to identify future research priorities and explore potential methodologies in this important area**.**

Results: Additional medical care, such as increased antepartum surveillance, appeared common in pregnancies after stillbirth, particularly in pregnancies following late-gestation stillbirths. Psychosocial support, on the other hand, was seldom provided to parents, revealing a major deficiency in practice across international settings. No eligible randomised controlled trials (RCTs) were identified to assess the utility of diagnostic investigation protocols in identifying the causes of stillbirth. Likewise, extremely limited evidence could be drawn from RCTs to inform specific intervention strategies to improve medical and psychosocial outcomes of families, highlighting another extraordinary gap in research. The most pressing avenue for future research surrounded clinical trials to evaluate the effects of low-dose aspirin.

Conclusions: This work has identified critical deficiencies in practice and research regarding care in pregnancies after stillbirth, and enabled a well-informed pathway for future scientific enquiry. Ultimately, this work serves as vital preliminary work towards the development of clinical practice guidelines, which are an essential component to improving the care and experiences of, and outcomes for, affected families.

MS JESSICA SEXTON

University of Queensland

Spatial Dependence of Risk Factors among Stillbirths at the Mater Mothers' Hospital (1997-2012)

Jessica Sexton is currently a Faculty of Medicine epidemiology PhD student at the NHMRC Centre of Research Excellence in Stillbirth, Mater Research Institute – University of Queensland, studying prognostic modelling and risk prediction for stillbirth (Primary Advisor: Prof. Vicki Flenady). Prior to joining the Stillbirth CRE, Ms. Sexton was an Allan Rosenfield Global Epidemiology Fellow at the U.S. Centers for Disease Control and Prevention in Lilongwe, Malawi. There, she was responsible for the design and implementation of surveillance programs designed to address HIV drug resistance and improve laboratory networks. Her background includes a Bachelor of Science in Biomedical Science from the University of New Hampshire (2012), a Master of Public Health in Epidemiology from Georgia Southern University (2015), and recently, a Master of Science in Spatial Analysis from Johns Hopkins University (2019).

Scientific Abstract:

Background: Australia's Health 2018 (AIHW) identified a gap in our epidemiological understanding of stillbirth due to an inability to identify and respond to vulnerable populations through existing health data. This study provides a proof of concept for larger-scale studies that explore and address this specific gap for stillbirths in Australia.

Methods: Clinical data for all births at the Mater Mothers' Hospital (MMH) in Brisbane, QLD (1997-2012) were collected retrospectively. Stillbirths with major/fatal congenital abnormalities as cause of death and multiple pregnancies were excluded. We quantify key risk factors for stillbirth using logistic regression and describe non-constant intensity patterns while accounting for clustering (spatial dependence) using semivariograms and Ripley's K function. A geodatabase was developed using publicly available spatial data and spatially linked to MMH clinical data using postcode.

Results: There were 282 eligible singleton stillbirths (rate 2.3 per 1000) recorded in a total population of 121,200 births. Logistic regression of key risk factors found that overweight/obesity (OR 1.65, 95% CI 1.28-2.11), public insurance (OR 1.82, 95% CI 1.42-2.34), smoking (OR 1.61, 95% CI 1.17-2.21), pre-eclampsia (OR 2.5, 95% CI 1.20-5.41), Type I/Type II diabetes (OR 3.01, 95% CI 1.08-8.42), nulliparity (OR 1.26, 95% CI 1.00-1.59), previous stillbirth (OR 3.89, 95% CI 1.92-7.89), small for gestational age (OR 4.08, 95% CI 3.10-5.37), fetal growth restriction (OR 4.87, 95% CI 1.89-12.54), and pre-term birth (OR 18.6, 95% CI 14.5-23.8) were associated with increased risk of stillbirth. Our semivariograms indicate that these risk factors demonstrate distinct, spatially dependent geographic patterns.

Conclusions: For the first time, this study identified unique, statistically significant spatial patterns for risk factors among stillbirths. Improvement in access to clinical data with postcode-level locality at larger population levels is needed to fully assess rurality in the context of stillbirth and to describe community-level ecological factors that may also contribute to risk of stillbirth.

DR SHUICHI SUETANI

Queensland Centre for Mental Health Research

What is the relationship between physical activity and mental health when you are growing up? : Findings from in the Mater-University of Queensland Study of Pregnancy

Dr Shuichi Suetani is a community psychiatrist in Logan, Queensland. He completed his medical degree with distinction in psychological medicine from University of Otago in New Zealand, before moving to Adelaide to start his medical career. He has numerous first author publications in high impact journals including *JAMA Psychiatry*, and currently sits on the editorial board for *Australasian Psychiatry.* Dr Suetani is also completing a PhD exploring the epidemiological relationships between physical activity and mental disorders using large Australian datasets.

Scientific Abstract:

The beneficial effects of physical activity (PA) for both physical and mental wellbeing are well established. Given that adolescence presents a critical developmental period for development both physically and psychologically, the exploration of the bidirectional longitudinal relationship between mental health and PA status in this period is of clinical interest.

We aimed to examine the bidirectional longitudinal association between mental health and PA status at ages 14 and 21 using a large birth cohort study from Brisbane, Queensland.

We analysed prospective data from over 3,000 individuals in the Mater-University of Queensland Study of Pregnancy. At age 14, psychopathology was measured using the Youth Self-Report (YSR) and PA status was categorized into; (1) frequent, (2) infrequent, or (3) no PA engagement group. At age 21, mental health outcomes consisted of; (1) common mental disorders, (2) psychosis-related outcomes, and, (3) emotional and behavioural problems, and PA engagement was dichotomized into either none or any. Using logistic regression, we examined the association between the (1) YSR score and subsequent PA engagement, and (2) PA engagement in adolescence and later mental health outcomes in young adulthood.

Although we found no longitudinal association between psychopathology at age 14 and PA engagement at age 21, no PA engagement at age 14 was associated with the increased likelihood of lifetime diagnosis of affective disorders, elevated delusional ideation, and endorsement of visual perceptual disturbance at age 21. Conversely, infrequent PA engagement at age 14 was associated with the decreased likelihood of subsequent substance use disorder.

Our findings suggest that while there is no longitudinal association between adolescent psychopathology and PA in young adulthood, lack of PAE in adolescence influences some later mental health outcomes. Thus, interventions to increase PA engagement in adolescence may represent an opportunity to prevent future mental health problems.

MR NAVID TOOSI SAIDY

Institute of Health and Biomedical Innovation

BIOINSPIRED 3D PRINTING FOR PERSONALIZED HEART VALVE TISSUE ENGINEERING

After completing a bachelors in mechanical engineering with first class honours from Taylors University, Malaysia (2017), Navid Toosi Saidy started his PhD in regenerative medicine group at the institute of health and biomedical innovation, QUT led by D/Prof Dietmar Hutmacher. His research project involves the convergence of advanced biomaterials and additive manufacturing techniques to fabricate scaffolds for cardiovascular tissue engineering applications. He is the recipient of world biomechanics congress travel award and have presented his work at a number of national and international conferences. In addition, Navid spent 8 months as a visiting scientist at the Center for Biohybrid Medical systems, Aachen, Germany where he has been involved with characterising heart valves in costume-made cardiovascular bioreactor systems.

Scientific Abstract:

Valvular heart disease is the third leading contributor to cardiovascular disease resulting in more than 5 million deaths annually. Heart Valve Tissue Engineering (HVTE) aims to provide mechanically stable valves that support tissue growth and remodelling to replace synthetic and non-bioactive prosthetics which are incapable of these features. Heart valves exhibit a complicated deformation profile instigated by the unique properties and microarchitecture of the underlying tissue constituents. Fabrication of biomimetic scaffolds that recapitulate the native properties have been a major challenge in HVTE. In an effort to address this challenge, we combined a novel fabrication technique called Melt Electrowritting (MEW) with a biomimetic design approach that mimics the wavy architecture of collagen fibres found in native leaflets for the manufacture of heart valve leaflets with outstanding anisotropic and soft tissue-like mechanical properties.
Scaffolds with precisely defined serpentine architectures reproduced the J-shaped strain stiffening, the anisotropic and the viscoelastic behavior of the native heart valve leaflets, as demonstrated by quasi static and dynamic mechanical characterization. Human Umbilical vein smooth muscle cells (HUVSMCs) seeded directly on the scaffold and encapsulated in fibrin showed high cell viability, infiltration and deposition of collagen I, III after 14 days of culture at physiological conditions. The tissue-engineered showed excellent functionality with transvalvular pressure drop and effective orifice area satisfying the ISO requirements, illustrating its great potential for long-term application of these scaffolds in HVTE. The convergence of a biomimetic design methodology and MEW was illustrated as a promising approach for fabricating highly tenable and personalized scaffolds for HVTE applications.

Queensland Health
Health and Medical Research

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.

Queensland
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Early-Career Researcher Award Finalists

DR JAMIE KUTASOVIC

University of Queensland

Breast cancer metastasis to gynaecological organs: a clinico-pathological and molecular profiling study

Jamie was awarded her PhD in 2016 from UQ. Her work focuses on understanding the molecular underpinnings of breast cancer progression with particular interest in invasive lobular carcinoma and oestrogen receptor positive breast cancer.

Scientific Abstract:

Breast cancer metastasis to gynaecological organs is an understudied pattern of tumour spread. We explored clinico-pathological and molecular features of these metastases to better understand whether this pattern of dissemination is organotropic or a consequence of wider metastatic dissemination. Primary and metastatic tumours from 54 breast cancer patients with gynaecological metastases were analysed using immunohistochemistry, DNA copy-number profiling, and targeted sequencing of 386 cancer-related genes. The median age of primary tumour diagnosis amongst patients with gynaecological metastases was significantly younger compared to a general breast cancer population (46.5 versus 60 years; $p < 0.0001$). Median age at metastatic diagnosis was 54.4, time to progression was 4.8 years (range 0-20 years), and survival following a diagnosis of metastasis was 1.95 years (range 0-18 years). Patients had an average of five involved sites (most frequently ovary, fallopian tube, omentum/peritoneum), with fewer instances of spread to the lungs, liver, or brain. Invasive lobular histology and luminal A-like phenotype were over-represented in this group (42.8 and 87.5%, respectively) and most patients had involved axillary lymph nodes ($p < 0.001$). Primary tumours frequently co-expressed oestrogen receptor cofactors (GATA3, FOXA1) and harboured amplifications at 8p12, 8q24, and 11q13. In terms of phenotype conversion, oestrogen receptor status was generally maintained in metastases, FOXA1 increased, and expression of progesterone receptor, androgen receptor, and GATA3 decreased. *ESR1* and novel *AR* mutations were identified. Metastasis to gynaecological organs is a complication frequently affecting young women with invasive lobular carcinoma and luminal A-like breast cancer, and hence may be driven by sustained hormonal signalling. Molecular analyses reveal a spectrum of factors that could contribute to *de novo* or acquired resistance to therapy and disease progression.

DR TIANQING LIU

QIMR Berghofer Medical Research Institute

Can nanotechnology give an old iron removal drug a new life? — Development of a novel nanomedicine to treat iron overload diseases

Dr. Tianqing Liu received her PhD in June 2014. She is an early career research fellow working at QIMR Berghofer Medical Research Institute. She was awarded NHMRC Peter Doherty - Australian Biomedical Fellowship in 2016, Women in Technology Rising Star Award in 2015, and is shortlisted for L'Oréal-UNESCO for Women in Science Australian & New Zealand Fellowship in 2019. Her current research focus is on the use of bio-inspired materials and nanotechnology to generate multifunctional nanostructures to overcome the current medical barriers for neurological disorder treatment. Her research is highly productive, leading to 20 original research publications, including 8 first author articles and 2 last/corresponding author articles in leading journals such as ACS NANO (Impact Factor [IF] 13.9), Biomaterials (IF 8.6), Nanoscale (IF 7.2), ACS Applied Materials & Interfaces (IF 8.1) etc.

Scientific Abstract:

Iron loading haemoglobinopathies, such as β-thalassaemia, are treated with iron chelators to remove excess iron. Deferoxamine (DFO) is an effective iron chelator with a favorable safety profile, but an onerous parenteral administration regimen limits its routine use. In order to develop more effective methods for delivering iron chelators, we have examined whether amphiphilic copolymer nanoparticles (NPs), can be used to deliver DFO more efficiently. We found DFO-NPs were much more effective at depleting iron than free DFO using cellular models and three different iron overload animal models. Pharmacokinetic analysis showed that NP-encapsulated DFO had a much longer half-life than free DFO, and that DFO-NPs could be readily taken up by tissues, and, in particular, by hepatic Kupffer cells.

Iron accumulation has been reported in neurological disorders as well. Iron chelation therapy using deferoxamine (DFO) may inhibit this nigrostriatal degeneration and prevent the progress of PD. We used a polymeric nanoparticle system modified with brain targeting peptide rabies virus glycoprotein (RVG) that enables intracerebral delivery of DFO. Administration of these nanoparticles significantly decreased iron content and oxidative stress levels in the brain of PD mice and effectively reduced their dopaminergic neuron damage as well as reversed their neurobehavioral deficits, without causing any overt adverse effects in the brain or other organs. This novel DFO-based nanoformulation holds great promise for safe and effective delivery of DFO into brain and for realizing iron chelation therapy in PD treatment.

DR ABU SINA

University of Queensland

Highly Porous Superparamagnetic Nanoparticles for Cancer Diagnostics

Dr Abu Sina is a research fellow at the Australian Institute for Bioengineering & Nanotechnology (AIBN), The University of Queensland. His research is focused on developing clinically actionable technologies for detecting disease biomarkers. Most of his works are published in high impact scientific journals like Nature Communications, Biotechnology Advances, Nanoscale, Biosensors and Bioelectronics, and Lab on a Chip, etc. He is one of the main discoverers of 10-minute universal cancer test which has been highlighted in >400 international media outlets including CNN, The Guardian, Forbes, etc. He has had several media appearances which include interviews in national (Channel 9, Channel 7, ABC News, Fox News) and international Television (CBC News, CTV News Canada) and Radio (4EB, 4BC, ABC Perth, ABC Sunshine coast, SBS) outlets. His vision is to develop point of care diagnostic technologies for early disease detection which could deliver a healthier and better life to the human.

Scientific Abstract:

DNA methylation is one of the major epigenetic modifications which involve the addition of a methyl group to the 5 position of cytosine nucleotides. Normal cell DNA carries a distinct methylation pattern across the genome to regulate the key molecular machinery of the cells. In cancer, this methylation pattern experiences a significant reprogramming with a net loss of global methylation at the intergenic regions of the genome together with a concomitant increase in methylcytosine levels at clustered CpG sites involved in regulatory roles (e.g., selective hyper-methylation at promoter regions). Herein, we discover that cancerous DNA with this altered methylation pattern forms a unique nanostructure in solution which is completely different from the structure formed by normal DNA. We find that the purified genomic DNA from normal cells has a greater tendency towards aggregation in aqueous solutions than genomic DNA from cancer cells. We also find that the solution properties of cancer and normal epigenomes influenced their affinity towards bare gold surfaces. In addition to the solvation properties, gold-DNA interaction is also modulated by the higher affinity of methylcytosines towards gold in comparison to the regular cytosines, and as a function of their clustered or dispersed patterning across the genome**.** Based on these gold-DNA affinity properties, we develop a one-step pan-cancer detection technology using interfacial biosensing which utilizes bare gold-biomolecule affinity to identify disease-associated biomolecules. The assay is simple and can be carried out in ≤ 10 minutes with small DNA input. More importantly, it can detect cancer without extensive sample preparation (e.g., bisulfite or enzyme treatment and PCR), sensor surface modification and sequencing. We believe this test will potentially be integrated into the clinic as a universal cancer screening method which will have enormous significance in the field of biotechnology, diagnostics, and medicine.

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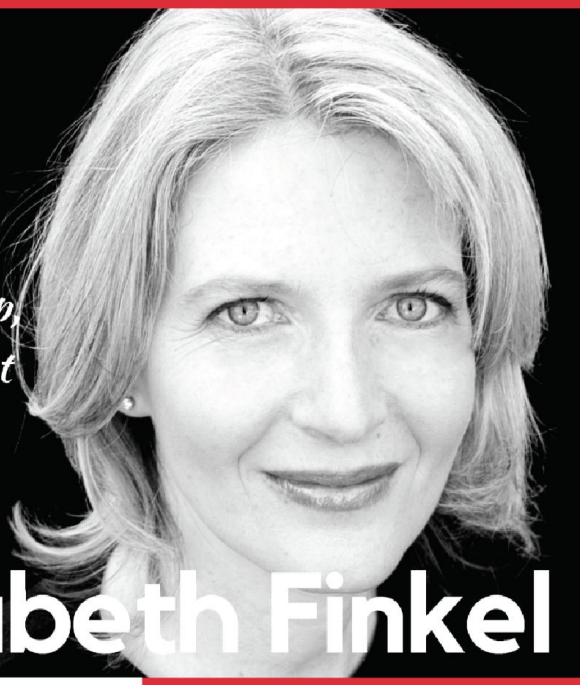

"Science excited and inspired me growing up, and that's what I want to give back"
– Elizabeth Finkel

Dr Elizabeth Finkel

Dr Finkel is a renowned Australian science journalist and regarded by many as a master storyteller. Using characters and experiences, Dr Finkel connects science to the people.

Dr Finkel is a co-founder of Cosmos and has received several awards for her work, including the National Press Club's Higher Education Journalist of the Year and a Eureka Prize for Science Journalism

Dr Finkel will excite and inspire at the ASMR Gala Dinner as she describes her journey from being a scientist to a renowned journalist and author

Mid-Career Researcher Award Finalists

DR EMANUELE PELOSI

Institute for Molecular Biosciences
University of Queensland

Genetics of Mayer-Rokitansky-Kuster-Hauser syndrome: role of Hnf1b and Wnt5a in Mullerian duct development

Dr. Pelosi received his PhD in Medical Biotechnology from the University of Bologna, Italy. He joined the Laboratory of Genetics and Genomics (LGG) of the National Institute of Health (NIH), USA, as a postdoctoral Visiting Fellow, and later accepted a position as Research Fellow in LGG. In 2016, Dr. Pelosi moved to Australia, and he took up a position as Senior Research Officer at the Institute of Molecular Biology of the University of Queensland.

His research efforts have focused on the genetic bases of sex-determination, ovarian development, and primary ovarian insufficiency, and menopause. He also studies the development of the female reproductive tract, and the genetics of Mullerian conditions including Mayer–Rokitansky–Küster–Hauser (MRKH) syndrome. Dr. Pelosi also serves as a Special Volunteer for the NIH, and he is actively involved in aging studies to characterize the molecular mechanisms of factors affecting age-related diseases and lifespan.

Scientific Abstract:

Mayer-Rokitansky-Kuster-Hauser (MRKH) syndrome results from the incomplete development of the embryonic Müllerian ducts, which give rise to the female reproductive tract including oviducts, uterus, and upper third of the vagina. MRKH syndrome affects 1 in 4500 women and it is found in 10% of primary amenorrhea cases. Type I MRKH is characterized by the absence of a fully developed uterus and vagina without any associated malformation. Type II MRKH is syndromic, and features additional malformations, mostly in the renal and skeletal systems. To date, the aetiology of MRKH syndrome remains unknown due to the lack of genome-wide approaches on large patient cohorts, and functional analyses using specific mouse models. We have identified novel candidate genes in a population of women with MRKH using an integrated approach of microarray and whole exome sequencing. We focused on two genes with unknown functions in the uterus: *Hnf1b*, expressed in the Müllerian epithelium; and *Wnt5a*, expressed in the Müllerian mesenchyme. We ablated *Hnf1b* specifically in the epithelial cells of the Müllerian ducts by crossing a mouse line harbouring a floxed *Hnf1b* gene ($Hnf1b^{fl/fl}$) with a $Wnt7a^{Cre}$ mouse strain. This is the first mouse model of MRKH type II. $Hnf1b^{fl/fl};Wnt7a^{Cre/+}$ mice showed Müllerian hypoplasia ~40% reduction in uterus length. Additionally, around 15% of $Hnf1b^{fl/fl};Wnt7a^{Cre/+}$ embryos developed additional malformations, including unilateral kidney agenesis and skeletal abnormalities. Similarly, uterus length was reduced by ~50% in *Wnt5a-/-* compared to wild-type mice due to a block in Müllerian duct elongation. Interestingly, we found *Hnf1b* and *Wnt5a* having similar roles and acting along the same pathway. These studies identify *Hnf1b* and *Wnt5a* as critical in Müllerian duct development and demonstrate the requirement of epithelial–mesenchymal interactions for proper uterine development. These findings will have direct implications in the diagnosis of MRKH and will improve clinical care for affected women.

DR RONAN KAPETANOVIC

Institute for Molecular Biosciences
University of Queensland

Uropathogenic Escherichia coli employs both evasion and resistance to subvert innate immune-mediated zinc toxicity for dissemination

I am working in the field of Immunology, in particular on the interactions between innate immune cells (macrophages) and bacterial pathogens, with a background in cell biology, biochemistry and molecular biology.
I have completed my PhD at the Pasteur Institute (Paris, France) in 2008, studying the detection of bacteria by the innate immune system. I have then moved to Edinburgh (Scotland, UK) and had the opportunity to work in the group of Prof. David Hume at the Roslin Institute for 4 years. There, I investigated the differences in innate immunity between species, with a particular interest in pigs. In 2013, I received an ARC DECRA to work in Australia at the Institute for Molecular Bioscience in the group of Prof. Matthew Sweet. At the moment, my main projects are understanding how macrophages use metal ions as antimicrobial weapons and characterising the role of mitochondria in the immune response.

Scientific Abstract:

Urinary tract infections are one of the most common bacterial infections of humans, with ~150 million cases globally per year, accounting for >1 million hospitalizations and $3.5 billion in medical expenses each year in the USA. Urinary tract infections usually begin as a bladder infection that can ascend to the kidney to cause pyelonephritis and potentially lead to sepsis. Recurrence rates are high, and infections often become chronic with many episodes. There is therefore an urgent need to understand mechanisms of uropathogenic *Escherichia coli* (UPEC) pathogenesis, host evasion, and new antimicrobial pathways in macrophages. We recently found that toll-like receptor-mediated activation of human macrophages triggers a zinc toxicity response against intracellular bacteria such as *Salmonella* and *E. coli*. Thus, we investigated whether EC958, a globally-disseminated UPEC, is also targeted by this zinc stress response. We first used transposon-directed insertion site sequencing to identify the complete set of UPEC genes conferring protection against zinc toxicity. We found that, compared to non-pathogenic *E. coli* K-12 strain MG1655, EC958 displayed an enhanced resistance to zinc toxicity. We also identified a full suite of genes involved in zinc resistance, including zinc exporters and genes involved in envelope integrity. Using this knowledge, we then developed innovative tools to track innate immune-mediated zinc toxicity intracellularly by microscopy. We generated highly specific bacterial reporter systems demonstrating that the majority of intramacrophage EC958 evades the zinc toxicity response, enabling survival within these cells. Finally, an intraperitoneal challenge model in mice revealed that EC958 employs both evasion and resistance against zinc toxicity, enabling its dissemination to the liver and spleen. Our work demonstrates that a pathogen of global significance uses multiple mechanisms to effectively subvert innate immune-mediated zinc poisoning, and provides us with a list of candidate genes that could potentially be targeted to enhance macrophages zinc-dependent antimicrobial responses.

DR DENUJA KARUNAKARAN

University of Queensland

RIPK1 directs immunometabolism in humans and can be therapeutically silenced to improve metabolic dysfunction in diet-induced obesity

Dr. Denuja Karunakaran is an IMB Fellow at University of Queensland and an emerging leader in the field of atherosclerosis. She completed her B. Biomed. Sci. (hons) and PhD in platelet biology at Monash University, Australia. She then pursued the prestigious Heart Foundation Postdoctoral Fellowship at the University of New South Wales, Australia, followed by an Endowed Cardiovascular Genetics Postdoctoral Fellowship at the Ottawa Heart Institute, Canada, where she trained in macrophages, microRNAs and cardiometabolic mouse models. Her high profile research publications include Science Advances, Circulation Research, ATVB, Nature Immunology, and Blood. In recognition of her research contributions, she is a recipient of various awards, including the 2019 American Society for Investigative Pathology (ASIP) Young Scientist Leadership Award and 2018 Australian Atherosclerosis Society (AAS) Early Career Research Award finalist. She's also an early career member of the ATVB journal editorial board. Her current research interests include RIP kinases and microRNAs in inflammation, cell death and efferocytosis in cardiometabolic diseases such as atherosclerosis, obesity and diabetes.

Scientific Abstract:

Obesity is a major public health burden worldwide, greatly increasing the risk of diabetes, cardiovascular diseases and cancer. Obesity and associated insulin resistance are characterized by chronic low-grade inflammation driven by the cooperation of the innate immune system and dysregulated metabolism in adipose tissue and other metabolic organs. RIPK1 (Receptor-Interacting serine/threonine Protein Kinase 1) is a master regulator of inflammatory cell function that coordinates inflammation, apoptosis and necroptosis in response to inflammatory stimuli. We found that therapeutic silencing of RIPK1 *in vivo* in a mouse model of diet-induced obesity dramatically reduces fat mass, total body weight and improves insulin sensitivity by reducing pro-inflammatory macrophages and promoting IL-10 producing invariant natural killer T cells (iNKTs) within the adipose tissue. We observed no significant difference in physical activity, metabolic respiration, food consumption and excretion. In humans, we identified 8 novel genetic variants (SNPs) in strong linkage disequilibrium in or nearby RIPK1 gene exon 5. Notably, in a cohort of >1,800 people, individuals carrying the minor allele of these RIPK1 SNPs have a 75-89% increase in the risk of developing obesity (adjusted odds ratios: 1.75-1.89, p<10-4) and a significant increase in RIPK1 mRNA expression in adipose tissue (eQTL association in METSIM cohort, p=10-23). Minor allele variation in one of these SNPs disrupts E4BP4, a repressor of RIPK1 transcription, promoting increased RIPK1 expression in adipose tissue. These findings demonstrate RIPK1 is a genetic driver of inflammation in human obesity, and that reducing RIPK1 expression is a potential novel therapeutic approach to target obesity and related diseases. Together, these studies show that RIPK1 genetic variants can be utilised as a diagnostic tool to predict risk of becoming obese, and RIPK1 can be therapeutically targeted to treat obesity.

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Clinician Researcher Award Finalists

DR JOSHUA TOBIN

Mater Research Institute

The Tumour Microenvironment as an Independent Prognostic Model in Follicular Lymphoma

Dr Joshua Tobin completed his medical degree at the Australian National University in 2013 before returning to his home-town of Brisbane. He has since undertaken a 2-year translational haematology fellowship at the Princess Alexandra Hospital where he maintains an honorary research position as an academic fellow. He is currently completing his training with the Royal Australian College of Physicians and is undertaking his PhD at the University of Queensland. His research interests are focused on the intratumoural immune response to lymphoid malignancy with the goal of developing clinically-applicable biomarkers to provide personalised therapeutics to lymphoma patients. For his work in this field Dr Tobin has been awarded the 2017 Achievement Award from the Haematology Society of Australia and New Zealand as well as the American Society of Hematology Achievement award in 2017 and 2018.

Scientific Abstract:

Follicular Lymphoma (FL) is the most common indolent Non-Hodgkin Lymphoma. Despite generally favorable survival outcomes, 20% of FL patients experience 'Progression of Disease within 24 months' (POD24) and subsequently have dismal long-term overall survival (OS). Accurate pre-therapy prognosticators are vital for clinical trial design and are increasingly mandated by funding agencies for stratification of patients to emerging front-line treatments. Recently, a clinicogenetic prognostic index (POD24-PI) has been developed which supplements clinical parameters with genetic mutational status however this remains to be validated in independent studies. The established biological importance of the tumor microenvironment (TME) in FL suggests that prognostic models would be enhanced by incorporating information on host immunity.

Forty-five pre-treatment FL biopsies were categorized into 'hot' or 'cold' immune nodes by multiplex immunofluorescent imaging and respectively characterized by concordant high or low expression of immune effector and checkpoint proteins. We applied these findings to an independent population-based cohort of 175 cases of FL treated with immunochemotherapy. The aims were to: a) identify novel, targetable immune markers of prognostic importance in the rituximab-era; and b) compare and contrast these with published clinicogenetic tools.

Low gene expression of multiple immune markers including PD-L2, TIM3, LAG3, CD137, TNF and CD4 predicted poor PFS. PD-L2 demonstrated the strongest association with PFS ($p=3.4 \times 10^{-7}$, HR=3.32). This finding was then validated in two independent, uniformly-treated cohorts derived from clinical trial cohorts (BCCA Cohort HR 2.272, p=0.002, GLSG Cohort HR 5.989, $p=9.2 \times 10^{-4}$). PD-L2 expression was independent and additive to previously described prognosticators. A combined 'clinico-immuno-genetic' model significantly improved the specificity of the POD24-PI to predict POD24 events (68% vs 88%)

Low expression of PD-L2 appears to be a surrogate of a broadly co-ordinated downregulation of the immune response (i.e. cold TME) which may point to the pathobiology underpinning the poor outcomes in these patients.

DR LISA GILLINDER

Mater Centre for Neurosciences

Defining the clinical phenotype of neuronal autoantibody associated epilepsy

Dr Gillinder has a joint clinical appointment as an Epileptologist at the Mater Advanced Epilepsy Unit, and Neurologist / Epileptologist at the Princess Alexandra Hospital in Brisbane. Dr Gillinder is the first person to undertake a surgical epilepsy fellowship in Queensland and the first person to be trained in Stereo-electroencephalograph (SEEG) within Australia. She is now one of only 4 epileptologists in Australia qualified in SEEG. She completed her Physicians examinations while concurrently completing a research higher degree at the University of Queensland, studying the quantification of myocardial steatosis using Magnetic resonance spectroscopy. Dr Gillinder's current research focus is to define the clinical features associated with chronic autoimmune epilepsy. She is the first to describe an association between neuronal autoantibodies and perisylvian epilepsy. This work offers potential new treatment options for this unique epilepsy subtype, which is often refractory to conventional therapies.

Scientific Abstract:

Epilepsy affects more than 250,000 Australians, with many of these being refractory to conventional therapies. This is a very costly condition to treat and it causes significant psychosocial burden. Our current research into a unique epilepsy subgroup is addressing this important health issue. This study will define the clinical phenotype associated with neuronal autoantibodies in chronic refractory epilepsy. This involves a multimodality approach to diagnosis that will test the hypothesis that these antibodies are associated with a definable clinical phenotype, which can be used to generate diagnostic criteria similar to what exists for autoimmune encephalitis. Patients will undergo thorough clinical evaluation guided by the stereo-electroencephalography methodology aimed at localisation of the epileptogenic zone. This involves evaluation of seizure semiology, epilepsy risk factors, medical background, neuropsychiatric and neuropsychological assessments. Prolonged electroencephalographic (EEG) recordings will be used to define the electroclinical features of these epilepsies and advanced neuroimaging techniques will be used to evaluate for inflammatory abnormalities in these cases. Our pilot study found that these antibodies are associated with perisylvian network epilepsies, as defined by seizure semiology and unique EEG changes. We also found an association with certain epilepsy risk factors, and a very high prevalence of mental health disorders in this population. Most importantly, we demonstrated a clear response to immunotherapy, with many of these refractory patients becoming seizure free. If our hypothesis is correct and we can confirm our preliminary findings in a large cohort, this will change the clinical approach to chronic epilepsy. Once these patients are clinically recognisable as a distinct subgroup they can be more easily identified and will therefore receive more appropriate and effective treatment. This will also lead directly to randomised trials to determine which immunotherapies are most effective, with the potential for curing this epilepsy subgroup.

A/PROF DAN SISKIND

Metro South Addiction and Mental Health Service

Reducing the Burden of Cardiometabolic Disease among People with Schizophrenia

A/Prof Siskind trained as a psychiatrist in Australia and the United States. He works clinically as a psychiatrist at Metro South Addiction and Mental Health Service with people with treatment refractory schizophrenia. His research interests include clozapine, treatment refractory schizophrenia and the cardiometabolic comorbidities of schizophrenia. He was awarded an NHMRC Early Career Fellowship (2016-2019) looking at the cardio-metabolic health of people with severe and persistent mental illness. He has over 115 peer reviewed publications, including first author in the highly ranked BJPsych, ANZJP, & EPS and has over $8 million in research grants in the past 5 years.

Scientific Abstract:

Treatment refractory schizophrenia (TRS) is a debilitating disorder that affects one in three adults with schizophrenia. Clozapine is the only antipsychotic licensed for TRS and can be transformative for the lives of these people. However, clozapine leads to significant weight gain and cardio-metabolic adverse effects. Weight gain is the most concerning side event reported by people on clozapine and can lead to non-adherence and relapse. Given the lack of other effective medications for TRS becoming available, reducing the cardio-metabolic adverse drug reactions of clozapine is essential. My COMET randomized controlled trial (RCT) found that exenatide can reduce clozapine-associated obesity people with schizophrenia, with a 4.2kg greater weight loss than placebo (Siskind et al 2018, Diabetes Obesity Metabolism). A similar result was found in my just published individual patient data meta-analysis of all studies of GLP-1RAs (including exenatide and liraglutide) for antipsychotic associated obesity among people with schizophrenia. Our recent meta-analysis reported that metformin holds promise for weight loss in clozapine-associated obesity (Siskind et al 2016 PLoSOne). We are currently recruiting for our CoMET RCT which randomizes people being newly commenced on clozapine to either metformin or placebo We aim to test the hypothesis that metformin compared to placebo will lead to significantly lower 24-week endpoint weight. We will also examine the effects of metformin on other markers of metabolic syndrome, visceral and hepatic adiposity,and participant preference, safety and tolerability regarding metformin and exenatide. This trial has the potential to establish acceptable, cost-effective treatment to prevent abdominal obesity and metabolic syndrome in people on clozapine.

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Medical Research: Bringing Health to Life

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