Day 1, Monday the 7th of November

It was a typically overcast Melbourne afternoon, the hesitant low pressure system eventually decided on some patchy rain to the inconvenience of those caught with no umbrella. But to more down-to-earth science, an enthusiastic crowd of medical researchers gathered at the Melbourne Convention Centre. Old friends were greeted and hands were shaken with the invited guests. The inaugural Joint Australasian Wound & Tissue Repair Society (AWTRS) and Molecular & Experimental Pathology Society of Australasia (MEPSA) conference, opened by the respective society presidents Dr Rachael Murray and Dr Scott Byrne, began with an exciting line-up of speakers and promises of a great three days away from lab, sponsored by the wonderful Phoenix Eagle, Smith & Nephew, The Company of Biologists, QUT IHBI, Wound Management Innovation and Visualsonics Fujifilm.

Plenary 1: Wound & tissue repair highlighted the fascinating work of Prof Boris Hinz, who has impressively managed to be the president of the European Tissue Repair Society while holding a professorial appointment at the University of Toronto – extending, as Prof Ian Darby puts it, the definition of Europe. The expert on fibrospasts (thankfully the original name never caught on) demonstrated that a feedback exists between dermal compliance and fibroblast activity, which may have implications on the amplification of scarring in healing wounds under high mechanical stress. His videos on the lives of fibroblasts showed how mechano-sensing of tissue stiffness causes differentiation into myofibroblasts that could be prevented by soft rubber-based culture substrates. Interestingly, their new properties could not be reversed after the cells were plated on new substrates, as epigenetic factors preserved their long-term mechanical...
memory. Perhaps more than one of us walked away disheartened to learn that our precious fibroblasts cultured in plastic flasks and wells are all myofibroblasts. Our immersion into each other’s research continued with the first poster display during afternoon tea.

With the skies not any clearer, **Plenary 2: The good, the bad and the ugly side of sunlight** began under bright fluorescent lighting. What better setting than this for a talk from the charming Dr Richard Weller from the University of Edinburgh on the benefits of sunlight. After analysing terabytes of data from dialysis centres across America, he presented some shocking yet convincing epidemiological evidence that sunlight is correlated to lower blood pressure, reduced atherosclerosis and reduced cardiovascular disease — independent of the beloved vitamin D. Dr Weller went further to reveal a mechanism to account for this surprising association with help of medical student “volunteers”/experimental subjects. His lab identified that UVA, augmented by skin thiols, photoreduces the large stores of nitrogen oxides contained in human skin to nitric oxide, which subsequently enters the systemic circulation where it acts as a vasodilator. The link between increased sun exposure and decreased all-cause mortality suggests we should go outdoors a bit more after all. To the dismay of everyone who has been taught to “slip slop slap”, Dr Weller also challenged our obsession with sunscreen, showing data that sunscreen while reducing skin aging and non-melanocytic cancer rates, has no effect on mortality.

On the other hand, Dr Katie Dixon of the Bosch Institute at the University of Sydney presented on a health benefit of sunlight that was indeed vitamin D related. She described a novel relationship by which vitamin D’s active metabolite (1,25D) increases a tumour suppressor called phosphatase and tensin homolog (PTEN) following UV exposure both in vitro and in vivo, which was interesting as UV exposure can down-regulate PTEN in skin cells. Her work with human melanoma cell lines suggested that through this vitamin D receptor- and PTEN- dependent manner, 1,25D acts to reduce melanoma cell viability. Taken together, this body of work may lead to therapeutic agents for both the prevention and inhibition of melanoma cell growth.

Continuing on the sunlight story, Prof Rex Tyrrell from the University of Bath questioned the need for antioxidant additives in sunscreens. His work since the 70s revealed that UVA leads to dramatic oxidative stress on skin through generation of ROS, as well as and increasing labile iron pools through ferritin degradation and release of free heme. More recently his team observed that UVA activates superoxide generating NADPH oxidases 1 and 4. To counter this disruption of heme and redox homeostasis, the body has evolved endogenous antioxidants and stress pathways involving proteins such as heme oxygenase 1 and MMPs. Pharmaceutical companies often add phytochemicals and antioxidant vitamins to sunscreen on the assumption that they will enhance this natural protection. As antioxidants (like green tea) are not regulated to the extent of approved drugs and there is limited evidence that they can even reach through the skin barrier, Prof Tyrrell argued that without a thorough understanding of their role in skin biology and importantly UV protection, the addition of exogenous antioxidants may upset a delicate signalling network already under stress. We look forward to what develops in this area of research and hope that Rex can relieve us with a definitive answer next time.

The day was closed with a welcome reception and more poster viewing, before the first social event commenced on Monday evening. It was dedicated for the gathering of ECRs at Munich Brauhaus, a few minutes shy from Melbourne Convention Centre. A good turnout was anticipated and surely we
were not disappointed. We were welcomed by friendly staff and the venue itself housed many exotic and rustic décor. We were quick to be accustomed by each other’s presence as the social dynamic swiftly turned from the initial what-do-you-do and where-are-you-from situation to various dialogues touching on all sorts of worldly matters. Most importantly, the amicable ambience filled up the room and perhaps the German food eased everyone into conversation. The night had to end a tad earlier for some as we headed back to prepare for our own deliveries of talks the following day as the agenda for Day 2 was bounded by another round of exciting talks from invited guests and speakers.

Day 2, Tuesday the 8th of November

We began the morning with the Plenary 3: Epithelial Biology chaired by Assoc Prof Terrence Piva, whom kindly introduced both speakers Prof Wolfgang Weninger from University of Sydney and Prof Birgitte Lane from the Institute of Medical Biology, Singapore. Prof Wolfgang began the session by describing a novel subset of specialized macrophage subpopulation, which he termed perivascular macrophage (PVM) due to their localization at the perivascular niche. He proceed to show that the high number of PVMs has a strong correlation on the incidence of breast cancer progression. It was believed that the high expression of matrix metalloproteinases by PVM could facilitate the metastatic spread of breast cancer. Next, Prof Birgitte Lane started by introducing the endeavours of her institute on the understanding of skin biology and their diseases. She then proceeded to talk about a rare skin fragility disorder called epidermolysis bullosa simplex, which could be caused by a mutation on the cytokeratin 14 (K14), an intermediate filament protein. Her lab showed that this mutation causes the inability of K14 to form fine filaments, which leads to the formation of aggregates. Currently, Prof Birgitte Lane’s lab is in the progress researching the possibility of using of certain inhibitors that may reverse the formation of K14 aggregates.

Symposium 1: Tissue Stress, Inflammation and Immunity began in-step with a recurring theme; that sun exposure has unexpected health benefits after accounting for induced vitamin-D, with Dr Prue Hart from the University of Western Australia speaking first. She introduced us to her research on autoimmune disorders by revealing that populations with greater sun exposure see a lower frequency of autoimmune disease. Interestingly, this effect seems to be embedded at a young age. Dr Hart sought the mechanism behind this phenomenon by comparing the metabolic activity of dendritic cells that differentiated from bone marrow progenitors of either UVB-irradiated or non-irradiated (control) mice. Dr Hart revealed that UVB-irradiation hindered the trafficking and metabolism of dendritic cells in the skin and that this in turn reduced their ability to elicit T-cell responses. Convincingly, data from further experiments illustrated that the bone marrow of UVB-irradiated mice, transplanted into naive mice, produced dendritic cells that remained in this suppressed state. Her research is now focussed at applying this newfound mechanism to the prevention and treatment of the autoimmune disease, multiple sclerosis.

Retaining a focus on the immune system, Dr Susan McLennan from the Charles Perkins Centre, University of Sydney, improved our understanding of the bacterial mechanisms that impede healing in diabetic ulcers, her talk focussing on the bacterial effect on MMP-9 in the infected wound. Firstly Dr McLennan reminded us that MMP-9 produced by the immune cells that infiltrate a wound
diminishes the levels of growth factors and extracellular matrix when not tightly controlled, ultimately impairing wound closure. Building on this knowledge she described her research findings which indicate that bacterial colonisation impairs wound closure via two means. Firstly, Dr McLennan explained that MMP-9 expression in the diabetic wound is already aberrantly high and that this titre is further increased by bacterial colonisation. Yet, MMP-9 must be activated from its inactive pro-enzyme form to become functional. Dr McLennan then revealed data indicating that predominant wound colonisation by gram-negative bacteria in particular, has the added effect of enhancing MMP-9 activation. So far, Dr McLennan’s research has provided strong evidence of both the importance of MMPs and the extensive influence of wound microbiota to wound healing.

After lunch, we saw the first of the dedicated sessions for AWTRS members **Stem Cell and Regeneration**, chaired by Prof Brigitte Lane and Prof Chris Jackson. The session was opened by Dr Pritinder Kaur from Curtin University, Perth, Australia – an expert in pericyte biology. Pritinder reminded us that pericytes are contractile cells that wrap around the endothelial cells of blood vessels and perform various functions including regulation of vascular diameter and capillary blood flow. Pritinder’s group isolated a mesenchymal stem cell-like pericyte population from human dermis and co-cultured these cells with skin fibroblasts. Skin regeneration studies suggested that pericytes are critical contributors to epithelial regeneration via enhancement of extracellular matrix components, basement membrane and hemidesmosome assembly. Pritinder shared images that demonstrate a new function, namely the ability of pericytes to influence symmetric division of keratinocyte stem cells – a determining factor in skin regeneration.

Another important factor in keratinocyte and epidermal stem cell proliferation is Endothelial Protein C Receptor (EPCR). EPCR is an anticoagulant protein C receptor which is highly expressed in the basal layer of human epidermis. The fact that EPCR overexpressing keratinocytes showed increased expression of stem cell markers and growth potential compared to siRNA treated keratinocytes with reduced expression of EPCR. Dr Meilang Xue from the University of Sydney concluded that EPCR is associated with increased keratinocyte proliferation. In addition, Meilang shared immunofluorescent images of adult skin biopsies co-stained with EPCR and stem cell markers p63 and integrin β1 which suggest that EPCR could serve as a potential human epidermal stem cell marker. Xanthe Strudwick and Gink Yang both from Future Industries Institute, University of South Australia presented previously unknown functions of a protein Flightless I, which was previously identified as a negative regulator of wound healing, but a positive regulator of hair follicle regeneration. Using a murine model of digit amputation, Xanthe investigated whether Flightless protein overexpression in genetically altered mice would have an influence on distal and proximal digit amputation. Although both WT and Flightless overexpressing mice grew claws following a distal amputation, only Flightless overexpressing mice showed regeneration of the missing digit, in part, due to activated Wnt signalling pathway. Gink, on the other hand, showed that Flightless protein may also play an important role in proliferation and number of cutaneous epithelial stem cells. Using mice with high
and low expression of Flightless, Gink assessed the influence of this protein on proliferative capacity of epithelial stem cells. Results suggest that although the number of epithelial stem cells is not affected, low Flightless levels were associated with lower expression of quiescence markers.

The final two presentations pointed out the importance of stem cells in organs other than the skin. Dr Nadira Ruzehaji, from the Institut National de la Sante et de la Recherche Medical in France indicated the importance of mesenchymal stromal cells in driving profibrotic responses in the bone marrow, such as that observed in primary myelofibrosis, a type of chronic leukemia. Her work demonstrated that downregulation of important activators of profibrotic TGF-β1 pathway, thrombospondin 1 and αvβ6, either with the chemical compound LSKL which inhibits the ability of thrombospondin 1 to activate profibrotic TGF-β1 or neutralizing antibodies αvβ6 against have anti-fibrotic affects in an in vivo model of primary myelofibrosis. Huan Ting Ong of the Ear Science Institute Australia, Murdoch University spoke about the benefits of adipose-derived mesenchymal stem cells in healing of perforated eardrum. Huan Ting pointed out the paracrine potential of mesenchymal stem cells positively influencing proliferation and migration of human tympanic membrane keratinocytes.

After an interesting poster display session in the afternoon, the second AWTRS session focused on Tissue Engineering and Repair. Chair Dr. Zlatko Kopecki first introduced us with A/Prof. Rodney Dilley from the University of Western Australia – an expert in ear sciences related tissue engineering. A/Prof. Dilley first illustrated the current shortcomings in treating ear tympanic membrane (TM) perforations after infections or trauma, and introduced his idea of using silk fibroin to address the issues involved during surgical interventions. A/Prof. Dilley’s group isolated TM epithelial and fibroblast cell populations from umbo explants and evaluated the growth of these cells on silk fibroin under the influence of growth factors and cytokines in vitro. A/Prof. Dilley shared the data of how silk fibroin grafts promoted cell proliferation, migration and collagen remodelling. Their group has also developed a new chronic TM perforation mouse model, upon which A/Prof. Dilley’s group is going to test their silk fibroin based devices in the near future.

The second speaker was Dr. Simon R. Corrie, an industry-engaged researcher specialised in nanosensor engineering with tissues. Dr. Corrie spoke about his work in using microneedle arrays to capture and detect proteins via the skin for medical diagnosis. We learnt from his introduction that the current chemistry based diagnostic methods lack applicability in remote locations and new technologies such as microneedle arrays (MPAs) could address this issue. Dr. Corrie and his team modified the surfaces on the MPAs with various antibody concentration to capture biomarkers, and they found the capture efficiency was greatly improved when antibody concentration was elevated. Their studies were further tested in mice dermis to detect dengue and malaria proteins and were proved to be great success.

The second chair Dr. Brooke Farrugia of this session first spoke about her own work on using chitosan sulphate to deliver growth factors. Dr. Farrugia’s study aimed at engineering materials to mimic cell surface molecules in this case heparan sulphate (HS) to bind growth factors. She showed
us the water soluble chitosan-arginine was able to bind growth factors FGF-2 and -7 during sulphation process, and illustrated how the incorporated molecule improved epithelial migration.

Dr. Brooke Farrugia then introduced Mr. Chee-Wai Wong from Curtin University to the stage. Mr. Wong has been working on his Ph.D thesis for the past three years aiming to develop an interesting solution for human keratinocyte expansion upon clinical application. He showed us a xenogenic-free method to expand cells using human dermal fibroblast ECM. Mr. Wong first illustrated how the human dermal ECM was created with pictures and graphs and then evaluated the effect of his modified ECM on primary keratinocyte proliferation and differentiation. He found his modified ECM improved proliferation marker Ki-67 expression and under-differentiation marker p63 in the cells as compared to being cultured on the conventional way of using collagen and tissue plastic. He also showed the cells growing on his modified ECM have better re-epithelialization potential as compared to the conventional method.

Another Ph.D candidate from Curtin University Ms. Elizabeth Grenlik continued on with the story of heparan sulphate which was first introduced by Chair Dr. Farrugia. Ms. Grenlik showed her work on using HS covalently immobilised to a 3D hydrogel in tissue engineering applications. She illustrated how the hydrogel-HS improved fibroblast and myoblast cell adhesion with statistics and graphs as compared to using hydrogel-RGD alone. And she found the co-presentation of HS and RGD on hydrogel enhanced cell differentiation and increased myotube formation.

The last talk of this session was presented by Ms. Rachael L. Moses from the University of Cardiff. Ms Moses introduced us with a novel pharmaceutical compound called Epoxy-Tigliane (ET) with the potential to battle cancer. We learnt from her that this naturally found compound could stimulate keratinocyte proliferation and migration and she aimed to investigate its underlying mechanisms. Using microarrays, Ms Moses discovered key genes/proteins upregulated upon ET treatment in keratinocytes, the most profound upregulated candidates include Keratin 13 and 15, proliferation regulator CCNB2, CDKN3, CDCA7, GINS2 and proteinases MMP-1, MMP7, MMP-10, and the most down-regulated candidates include Keratin 6B, 16, 17 and cytokine IL-6, IL-8, CCL5, CXCL10.

Ludlow Bar and Dining Room, situated conveniently along the Yarra Promenade was the booked social venue for Tuesday night. It was a chilly evening but it didn’t deter us from having a splendid time with the great company that we had. Not to mention, the superb hospitality and the mouth-watering three-course meal consisted of an eclectic selection from cured kingfish ceviche to a whopping size of steak to the soft and delicate vanilla panacotta or the decadent chocolate and walnut torte. The meals, one after the other, made their way to our tables, interspersed with voices mingling, outbursts of laughter and the clinking wine glasses. Before the night concluded, Dr Rachael Murray, the President of AWTRS announced the names of the eight travel award recipients: Xanthe Strudwick, Ruilong Zhao, Chee Wai Wong, Lucas Wager, Nadira Ruzechaji, Leila Cuttle, Gink Yang and Duy Anh Tao. With their widest and brightest of photographs taken with Dr Murray as they graciously accepted their awards. Congratulations went to Dr Zlatko Kopecki for receiving the Young Investigator Award. The most prestigious award of the night was the announcement of The Lifetime
Member Award which recognised and congratulated the efforts of Professor Allison Cowin whom have initiated the inauguration of AWTRS ten years ago. Thanks to Professor Cowin’s dedication and continuous efforts, the society has become a distinguished society like it is today. As midnight fast approached soon after the award session, so too was the closing of the second day of the conference. Nevertheless, the final day of the conference promised to offer another run of great talks awaiting to be delivered.

Day 3, Wednesday the 9th of November.

The final day of the conference started with a session on Wound Healing chaired by Dr Rachael Murray. Dr Matthew Hardman from the University of Hull kicked off the 4th and final plenary session with a sample of his research on the influence of microbiota on wound healing. Matt uses an extensive range of wound healing models in his research, but focussed on a NOD2 deficient mouse model in this session. NOD2 is a pattern recognition receptor (PRR) involved in recognising potentially harmful pathogens and was first appreciated for its role in wound healing when it was linked to gut inflammation and Crohn’s disease. Dr Hardman explained how he has expanded on this work by investigating NOD2 in cutaneous wounding. He presented interesting experiments in which commensal cutaneous microbes were discovered to vary between NOD2-/- and wild type control mice. Furthermore he demonstrated that the types of microbes inhabiting NOD2-/- mice impaired their ability to heal cutaneous wounds and that transfer of these bacteria to wild type controls also transferred this healing deficiency. Applying this knowledge to human patients, when NOD2 expression was investigated in clinically uninfected human wounds, Dr Hardman showed that lower levels of NOD2 expression correlated with non-healing wound status. The inaugural AWTRS Lifetime Member, Prof Allison Cowin covered extensively her work on Flightless I (Flii), showing that it is a negative regulator of wound healing with a significant impact upon scarring. She proceeded to show how Flii could be a potential target for the development of wound healing and anti-scarring treatment, which has been the long standing aim of her research. Currently, she is looking on the use of porous silicon nanoparticles to deliver Flii neutralizing antibodies to wounds as an approach to treat diabetic ulcers.

After morning tea, we had the second combined AWTRS-MEPSA symposium on Skin Cancer which was chaired by Dr Graeme Walker. First to speak was Prof Peter Soyer, an academic dermatologist based at UQ and Princess Alexandra hospital. Prof Soyer spoke about his naevus (mole) research and particularly the NHMRC Centre for Research Excellence (CRE) he is leading on the Study of Naevi to investigate why some naevi transform into cancers while others do not. The CRE has 3 program arms; 1) genetics and epidemiology of naevi, 2) consumer facilitated naevi monitoring and 3) molecular and genetic properties of changing naevi. The core study in the CRE is a population-based
cohort study of naevi in Brisbane adults over 3 years. Participants will have 3D total body photography and dermoscopy to catalogue the number, size, type and changes in naevi over that time and any excised naevi will be studied in terms of their pathology, and the cells will be cultured and genetically profiled. Naevogenesis susceptibility markers including the Melanocortin-1 receptor (MC1R) genotype and the MITF E318K allele were mentioned specifically as of interest. In terms of Program 2 he described a project where mobile teledermoscopy (people using a teledermoscope on their phone to take photos of their naevi and sending them) will be utilised to examine the effectiveness of self-monitoring and identification of melanoma in people of different skin types. This would be compared to conducting skin self-examination without the teledermoscope and will demonstrate if technologies of this type will enable faster presentation to a doctor and more accurate diagnosis.

The second speaker was Dr Elke Hacker, a post-doc at QUT who presented her work examining the effect of sunscreen in preventing melanoma. From biopsies of participant’s skin which was exposed to UV with and without sunscreen, she saw that people with wild type MC1R gene had a significant increase in melanocyte density after UV exposure compared to those with MC1R variant genes. Her work has also shown that sunscreen reduced p63 levels and DNA damage. She has been most recently working on a public health project where participants are using a sunsmart app in combination with a UV monitoring device to help them measure and track their UV exposure levels and alerts to inform them when to apply and re-apply sunscreen to prevent melanoma.

After lunch, we moved into the third AWTRS session on The Molecular and Cellular Basis of Tissue Repair and Fibrosis chaired by Prof Boris Hinz and Assoc Prof Ian Darby. Our current society president Dr Rachael Murray from the Institute of Health and Biomedical Innovation began the session with a neat description of MMP dependent migration of macrophages into injured tissue and the pathways which lead to surface presentation of the protein which facilitate this process. The research presented the application of inhibitors of the intracellular transport pathway that regulate surface delivery of MT1-MMP, also known as MMP-17, as a possible target for therapies aimed at the reduction of inflammation during wound healing. Mr Ruilong Zhao from the Kolling Institute then described a novel engineered activated protein C (APC) which may be used to enhance wound healing whilst avoiding the problems associated with the anticoagulant properties of native APC. Dr Lyn Wise from the University of Otago in New Zealand discussed the use of IL-10 and VEGF-E produced by the Orf Virus found in sheep to treat limb wounds in horses which have characteristically delayed healing. While the application of the proteins was seen to dampen the inflammatory response in both normal body and the limbs wounds, further optimization of the treatment schedule may be needed to improve the rate of healing. Mrs Betoul Baz from University of Queensland Centre for Clinical Research then gave an update on her research utilising the Collaborative Cross panel of mouse strains which has identified Aldose Reductases as key regulators of wound healing speed. Mr Krupesh Patel from Monash University spoke on pharmacological targeting of epithelial repair and fibrosis associated with asthma, where it was found that the administration of relaxin reduced epithelial damage and in combination with corticosteroid treatment may offer optimal treatment for chronic airway disease. Mr Lucas Wager from the Institute of Health and Biomedical Innovation presented his research into the regulation of epithelial-mesenchymal transition by microRNAs, identifying altered expression of miR-501-3p, miR-4972 and miR-181a-5p in the leading edge keratinocytes in an in vitro model of migration into a wound. The final speaker in the session, was Dr Leila Cuttle also from the Institute of Health and Biomedical Innovation described research by her group assessing the cytotoxicity of silver wound dressing in vitro, in in vivo burns and using a novel ex vivo burn model which showed that while they were toxic to skin cells in vitro they do not appear to delay burn wound healing.
After a short afternoon tea break, the final AWTRS session on *Chronic Wounds and Inflammation*, chaired by Dr Matthew Hardman and Dr Rachael Murray was opened with an engaging talk by Prof Greg Schultz from the University of Florida describing the battle with biofilms in chronic skin wounds and the need for effective debridement, accurate detection of biofilm presence and the correct identification of bacterial species to direct treatment and win the war against chronic wounds. Prof David Margolis continued the discussion on chronic wounds identifying lower extremity amputation and diabetic foot ulcers as major risk factors for death in diabetic patients. Interestingly the hazard ratios cannot be fully attenuated by adjusting for comorbidities such as renal failure and cardiovascular disease, highlighting the need for close medical follow-up for patients with a history of these chronic wounds. Dr Danqing Min from the University of Sydney presented a small study performed on diabetic patients with foot ulcers at the Royal Prince Alfred Hospital which revealed altered monocyte profiles, both in circulating numbers and in the expression of CCR2 and CCR5 during healing. It is hoped that further investigation may allow for identification of altered inflammation and be markers for healing versus non-healing wounds. Ms Anh Tao, also from the University of Sydney, then discussed the potential utility of NGAL, a regulator of MMP9, as a marker of healing in diabetic foot ulcers. It was found that higher MMP9/NGAL complex was associated with longer ulcer duration and that the changes did not appear related to bacterial load. Ms Sumeet Sandhu from the Institute of Health and Biomedical Innovation then spoke on the use of qPCR as a better method for the determination of total bacterial load in diabetic foot ulcers, and the finding that an increased total bacterial load may be used as a prognostic for non-healing foot ulcers. The final talk for the meeting was from Dr Mariena van der Plas from Lund University who discussed the action of pseudomonas aeruginosa which unlike many bacteria found in chronic wounds is not associated with increased inflammation. She demonstrated that these bacteria evade the immune response by cleaving thrombin to produce a peptide which mimics host defence peptides to dampen inflammation.

Dr Rachael Murray concluded the day by announcing the recipients of the prizes for the best ECR podium presentations and posters. Congratulations went to Mariena van der Plas (Lund University) and Xanthe Strudwick (University of South Australia), who were awarded first and second places for their podium presentations, and to Krupesh Patel (Monash University) and Lucas Wager (Institute of Health and Biomedical Innovation, QUT) who were awarded first and second places for their posters. The joint AWTRS-MEPSA meeting was then closed with thanks for society presidents Dr Rachael Murray and Dr Scott Byrne, and the Conference Organising committee for a well organised and informative conference bringing together research from a broader community which demonstrated a common scientific interest and is sure to benefit all researchers who attended.