

ASMR is pleased to announce finalists for the 2008  
**QUEENSLAND PREMIER'S AWARDS FOR HEALTH AND MEDICAL RESEARCH**  
**Senior Researcher and Postdoctoral Researcher**  
Awards to be presented at the ASMR Medical Research Week® Dinner on  
Friday May 30, 2008 at the Marriott Hotel, Brisbane

**Senior Researcher Award Finalists**

**Patricia C Valery - QIMR**

**High prevalence of metabolic syndrome in youth of the Torres Strait Islands of Australia**

Findings have important public health implications for the Torres Strait region. One in four Indigenous Australian adults are obese and 26% of Indigenous adults in the Torres Strait (TS) region have type 2 diabetes mellitus (T2DM). Obesity plays a central role in the development of the metabolic syndrome, a group of risk factors for cardiovascular disease and T2DM. Risk factors for cardiovascular disease tend to coexist in the same individual from childhood and persist over time. A study of 158 school-age Indigenous youths residing in the TS region revealed 46% were overweight or obese, 38% had a large waist circumference, 43% had acanthosis nigricans and 27% had high blood pressure. The metabolic syndrome was present in 17% of the total sample and one-third of the overweight or obese subgroup. Girls in particular seem at risk for obesity associated metabolic complications. T2DM was diagnosed in two youths.

**Ben Goss - Institute of Health and Biomedical Innovation, Queensland University of Technology**

**Reduction of Secondary Degeneration after Spinal Cord Injury by Acute Delivery of Vascular Growth Factors**

Spinal cord injury has a devastating personal and social impact. There is currently no cure for spinal cord injury and the majority of people who survive this injury are destined to a life of disability, often with a reduced life expectancy. There are many animal studies on the potential of neuroregenerative strategies including stem cell therapy, gene therapy, tissue engineering and many others. Most of these studies concentrate on understanding and assisting neuronal regeneration but to date there has been no successful clinical translation. Part of the problem is that after the initial trauma, the compensatory mechanisms in the spinal cord exacerbate and greatly extend the injury. The International Spinal Research Trust has stated that "minimising deleterious effects of early trauma inflammation and scar tissue" must be the first fundamental step in any strategy for repair of the injured spinal cord. Our approach is to promote early inflammation and angiogenesis in an attempt to reduce chronic inflammation and subsequent secondary degeneration. We have shown that a combination of VEGF and PDGF applied topically, at the time of injury, to a spinal cord hemisection in the rat reduced the size of the lesion and reduced the size of the glial scar. It also increased blood vessel and nerve cell density at the injury site when examined 90 days after injury. We have achieved the prevention of spinal scar formation which is the critical first step in spinal cord regeneration. This a starting point for future neuroregenerative strategies to potentially restore function. The concepts developed here have broader implications in the treatment of other diseases including stroke and brain injury.

**Dr Kathy Andrews – QIMR**

**A "piggy back" platform for antimalarial drug discovery**

In Queensland, Australia and globally there is a significant amount of research being undertaken towards the development of new drugs for treatment of diseases such as cancer and HIV/AIDS. This existing expertise is not currently being exploited effectively against the global threat posed by major tropical infectious diseases. The aim of my research is to use a "piggyback" platform to develop new drugs for malaria, the worlds most significant tropical infectious disease, by exploiting drugs currently used for (or under development for) diseases such as cancer and HIV/AIDS. Using this approach we have shown that some HIV drugs (protease inhibitors) can kill malaria parasites at clinically relevant concentrations. This may have major public health implications due to the risk HIV/malaria co-infection in many regions of the world. We are also investigating different classes of anti-cancer drugs for potential antimalarial use. One of these compound classes includes anticancer agents that target an enzyme involved in gene regulation and cell death. A major strength of this "piggyback" platform is a reduction in the time it takes to develop products for potential clinical use against malaria because extensive safety and tolerability testing have already been carried out or the drugs are already approved for clinical use for other diseases. This is an important consideration as many of the drugs currently used to prevent and treat malaria are now failing due to malaria parasite resistance. Time is running out – to save lives we urgently need to identify and develop the next generation of antimalarial drugs.

## Postdoctoral Researcher Awards Finalists

**Trent Woodruff, School of Biomedical Sciences, University of Queensland**

### Treatment of Neurodegenerative Diseases with a Novel C5a Receptor Antagonist

Inflammation is increasingly recognised as a significant disease process in degenerative brain diseases. Diseases such as Alzheimer's, Parkinson's, Huntington's and Motor Neuron disease, all display inflammation as a major component of their disease progression. The complement system is a large component of inflammation, and plays a role in several inflammatory diseases. Over recent years, our laboratory has developed a new type of anti-inflammatory drug. This drug has been successfully tested in humans for safety. In the current study, we have used rat models of Huntington's disease and Motor Neuron disease to test the ability of our novel anti-inflammatory agent to reduce the signs of disease in these animals. Animals were administered drug in the drinking water before the onset of disease, or once disease symptoms were already present. We found that the drug was able to reduce signs of disease in both animal disease models. In the model of Huntington's disease, drug-treated rats displayed reduced behavioural and postural problems. Furthermore, when the brains were examined post-mortem, we found a significantly reduced degree of brain damage in the region of the brain associated with Huntington's disease. In the model of Motor Neuron disease, drug-treated rats had reduced movement problems and muscle paralysis. In addition, we found a significantly reduced degree of inflammation in the spinal cord of these rats. This research suggests that our novel, orally active anti-inflammatory drug may be a viable new drug treatment for diseases such as Huntington's disease and Motor Neuron disease.

**Jenny Ekberg, Eskitis Institute for Cell and Molecular Therapies, Griffith University**

### Sea snail toxins and nerve cell enzymes may relieve pain and neuronal dysfunction

Over-activity of nerve cells causes a number of severe disorders affecting a large proportion of the population in Australia and worldwide, including pain, epilepsy and stroke. Chronic pain results from injury to nerves and is estimated to affect one in 6 Australians at some point during their life-time, often with devastating consequences severely hampering the quality of life of the person affected. Current drugs used to manage severe pain, such as morphine, are often inadequate and associated with serious side-effects.

In this study, a natural product from a Great Barrier Reef snail, *Conus marmoreus*, was found to selectively block the activity of specific nerve cells involved in pain, instead of affecting all types of nerve cells like conventional pain medications. The results are exceptionally promising and the compound is now being synthetically improved at the University of Queensland to further increase the therapeutic benefits before it can be tested on humans and developed into a proper drug. Furthermore, this study describes the characterisation of a new enzyme, *Nedd4*, involved in regulating the activity of nerve cells. This enzyme, which is expressed in the human brain, therefore constitutes a novel potential therapeutic target not only for pain, but also for epilepsy, stroke and other diseases involving hyperactivity of nerve cells. If we can learn to modulate the behaviour of *Nedd4* in the brains of patients with these disorders, we may be able to control the activity of the affected nerve cells and thereby alleviate the symptoms. My research has so far been focused on blocking over-activity of nerve cells associated with various disease states. I have recently moved to the Eskitis Institute for Cell and Molecular Therapies, Griffith University, to continue my work related to neurological disorders, now pursuing an interesting new approach using a specialised cell found in the nasal mucosa to regenerate damaged nerve cells after spinal cord injury.

**Michael Piper The Queensland Brain Institute, The University of Queensland,**

Understanding how the brain develops is central to comprehending nervous system function in both normal and disease states. There are many complex developmental programmes that occur synchronously during embryonic development and which form the basis for subsequent brain maturation and functional connectivity. One family of molecules, the Nuclear Factor One (*Nfi*) transcription factors, may be an integral component of many of these programmes. *Nfis* have been implicated in development of specific cells known as glia, which regulate how neurons form functional connections during development. *Nfis* may also contribute to how the complex, layered structure of neurons in the brain is initiated and subsequently formed, and how these neurons extend processes to other regions in the nervous system, such as the spinal cord. My research has identified the developmental and molecular mechanisms by which the *Nfi* genes regulate these complex processes. This research will have a significant impact on our understanding of human disorders of cortical development, and may form the basis from which potential therapeutic drug targets for clinical disorders can be developed.

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