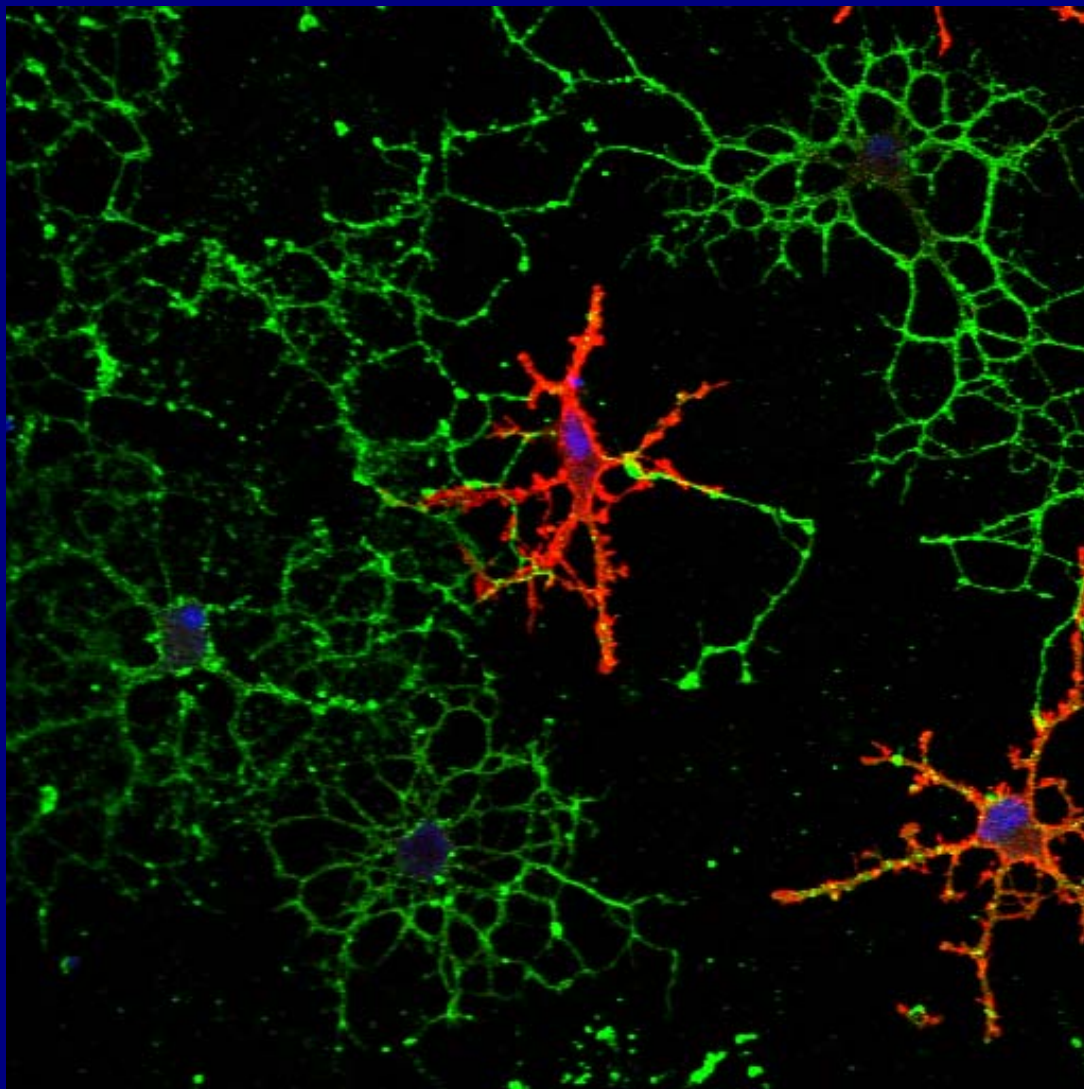


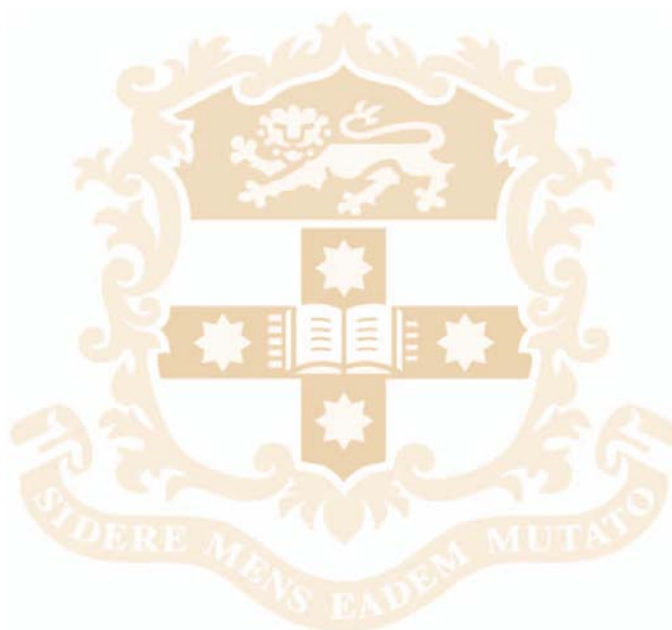
Annual Report 2008

The Nerve Research Foundation
The University of Sydney



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Highlights of 2008

Below are Highlights from 2008 for the Nerve Research Foundation

- Prof John Prineas was awarded the Charcot Prize. This is the most prestigious international award for work in the field of Multiple Sclerosis.
- Prof John Prineas was invited to deliver the keynote lecture on MS Immunopathology at the major international MS Conference of the European Committee for the Treatment and Research into Multiple Sclerosis (ECTRIMS) in September in Prague.
- Prof Pollard invited to deliver the E G Robertson Memorial Lecturer to the Australian & New Zealand Association of Neurology in May.
- Dr Roberta Chow, Assoc Prof Armati, Dr Ariel Arthur and Laita Bokhari awarded the prize for the best basic science presentation at the International Conference of the World Association of Laser Therapy, focused on laser therapy for chronic pain in South Africa in October.
- NRF researchers were awarded \$1.5 million in competitive research grants (NH&MRC, ARC, MSRA).
- Ms Isa Guha, an Honours Graduate from University College London and the world's best women's fast bowler, joined the Neuroinflammation Group as a volunteer researcher. Isa was in Australia bowling for England in the Women's World Cup Cricket Competition (http://iccwomensworldcup.yahoo.net/gallery/videos/pre_event_videos.html)



John Prineas



President's Report

Nerve Research Foundation



Benjamin Chow

The last twelve months have seen some new initiatives taking shape in the form of fund raising activities in partnership with other established organizations. Our Foundation managed to secure the position of being the sole beneficiary of the annual Chinese New Year Fund Raising Dinner of Australian Chinese Charity Foundation Inc. ACCF was founded by The Hon Dr Peter Wong AM some sixteen years ago. It now has a foundation capital of more than \$1.2M. It is a most respected Chinese community charitable organization in Sydney.

This fund raising activity will allow our usual supporters to support our Foundation and at the same time enjoy a ten course Chinese banquet in a leading restaurant in Chinatown with much colour and sound typical of the Chinese New Year celebration in Sydney and in other parts of the world.

We are also in the process of securing a similar position with the newly formed Sydney Business Executive Lions Club at its annual fund raising dinner to be held in August 2009. This new Lions Club is a world first and is designed to suit the business sector members in large cities. Lions International head office is keeping a close watch on this club as it intends to replicate this club in other cities around the world if the Sydney Business Executive Lions Club proves to be successful.

Managing the Foundation's supporter data base has always been a challenge with our limited resources. Associate Professor Armati has been working very hard to complete the update and it looks like we are getting there for which we are most grateful.

As the global financial crisis deepens we will most likely see a continued economic deterioration for much of 2009. The next twelve months will be challenging for most of us but with the support of Council members and other supporters we will continue to provide much needed resource to assist with the important research work in Multiple Sclerosis, Motor Neuron Disease, Peripheral Neuropathy, Pain and Dementia.

Benjamin Chow AO

Director's Report

Nerve Research Foundation



John Pollard

In 2008 the Nerve Research Foundation made important contributions to research at the University of Sydney by supporting several research projects, providing travel funds to allow young researchers to attend important overseas international conferences and providing salaries for young scientists. The research projects included the fields of multiple sclerosis, peripheral neuropathy, chronic pain, Motor Neuron Disease, and Alzheimer's disease, and progress made in 2008 is highlighted in the following pages.

Nerve Research Foundation researchers were very successful in winning competitive grants from national funding bodies such as the National Health & Medical Research Council (NH&MRC) and the Australian Research Council (ARC). However, these grants are insufficient to purchase new and expensive technologies which are a necessary part of cutting edge research methodologies; funds raised by the Nerve Research Foundation thus complement and strengthen these grants and scholarships.



Patricia Armati

We wish to thank our many benefactors who have given generously to support the work of the foundation. In particular, Mr & Mrs James Graham, Mr John Armati, Dr Ruth Kerr, Mr & Mrs Alston, Mr & Mrs John Anderson, and Mr & Mrs Athol Yeomans have continued their generous help despite the difficult financial situation. We thank Stephen Ainsworth for support for the gene study of MS which has not been published in an international journal.

In 2008, the Michael Bannigan Postgraduate Prize, which was established to recognise the best Ph D thesis supervised by a member of the Nerve Research Foundation, was jointly awarded to Dr Kim Alexander Kauffman and Dr Michael Barnett.

We were saddened to learn of the death in November of our second President, Mr Ed Barnum. The Barnum family requested donations to the NRF rather than flowers on that sad occasion and we used the very generous donations to establish The Ed Barnum Postdoctoral Research Fellowship to provide a continuing salary for a gifted young research worker in the field of MS and inflammatory neuropathy.

Professor J D Pollard, Co-Director
Associate Professor P J Armati, Co-Director

Members of the Nerve Research Foundation

Council

- Professor Marie Bashir AC, Chancellor, The University of Sydney
- Dr Michael Spence, Vice Chancellor, The University of Sydney
- Professor Andrew Coats, Deputy Vice-Chancellor, The University of Sydney
- Professor Bruce Robinson, Dean, Faculty of Medicine, The University of Sydney
- Mr Benjamin Chow AO, President
- Associate Professor Patricia Armati, Vice President and Co-Director
- Professor John Pollard AO, Co-Director
- Professor Robert Ouvrier AOM
- Dr Ruth Kerr
- Mr John Armati AOM
- Mr Ross Low
- Mr David Jacobs
- Mr John Baker
- Mr Stanislav Carroll AO
- Mr Stephen Parry
- Mr Roy Melick

Scientific Committee

- Professor John Pollard AO
- Associate Professor Patricia Armati
- Professor Robert Ouvrier AOM
- Professor Bruce Robinson

Honorary Governors

- Mr John Armati AOM
- Ms Rikki O'Neill

Honorary Life Members

- Mr David Jacobs
- Ms Rikki O'Neill
- Dr Ruth Kerr
- Mr John Armati AOM
- Mr Stanislav Carroll AO





NEUROINFLAMMATORY DISEASES

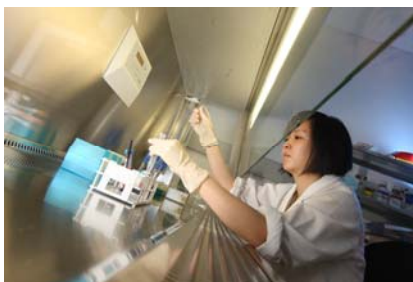
Inflammatory Demyelinating Neuropathies

M Wang, E Mathey, M David, P J Armati, J D Pollard, J Spies

These disorders are autoimmune diseases of peripheral nerves and represent the commonest treatable neuropathies in the western world. Acute and chronic varieties occur. The acute variety is known as the Guillain-Barre Syndrome and characteristic subtypes have recently been described. The chronic disorder, chronic inflammatory demyelinating neuropathy, is now known as C.I.D.P. and various subtypes are also now recognised.

Guillain-Barre Syndrome

This is an intriguing condition, often resulting in severe paralysis of all limbs, and even the respiratory muscles, yet many patients eventually recover if managed appropriately. Most cases follow a mild infective illness of the gastrointestinal or upper respiratory tracts. Recent research has shown that antibodies produced by the immune system to counteract the infective organisms, cross react with target molecules on patient's nerve thus causing nerve damage with resulting paralysis. The molecules which are targeted are unique lipids called gangliosides which are particularly concentrated in nerve membranes. In 2008 our group studied how antiganglioside antibodies cause nerve damage.



Antibodies, similar to those found in patients, were obtained from our collaborator in Julius Hopkins University, Baltimore, USA, and were injected into rat nerves. We found that these antibodies bound to nerves in the region where the insulating layer of myelin is deficient – the node of Ranvier. In this region the sodium channels are anchored in a dense cluster by specialised connections between Schwann cell and the axons. The antibodies blocked the conduction of nerve

impulses, presumably by interfering with ion channel function. In high concentration, these antibodies caused structural damage to axons. These interesting findings explain why some patients may recover quickly and completely from severe paralysis whilst others sustain significant nerve cell impairment. In continuing research we are examining methods of treatment to prevent the axonal damage and hence prevent the disability.

Funding: NH&MRC grants

C.I.D.P.

This disorder is regarded as the peripheral nerve analogue to Multiple Sclerosis and like that disease may present with a relapsing remitting or progressive course. However, most patients are successfully treated by plasma exchange or intravenous immunoglobulin suggesting that antibodies also play an important pathogenic role. Dr Emily Mathey has been studying the role of antibodies to Neurofascin 155, an important adhesion molecule which attaches the Schwann cell paranodal loops to the axon. Emily identified such antibodies in M.S. patients with chronic progressive disease, and in preliminary studies has shown them in a significant proportion of C.I.D.P. patients. Current studies are exploring the pathogenicity of these antibodies and how they may cause clinical symptoms and signs. Since, in both M.S. and C.I.D.P., long-term disability equates with axonal damage and loss, we are also currently studying in C.I.D.P. the role of antiganglioside and other antibodies which may damage axons. If these are present, early treatment of patients may prevent later disability.

Funding: NH&MRC grant and Rebecca Cooper Medical Research Foundation

Pain relieving effects of laser therapy – investigations into mechanisms

P J Armati, R Chow, L Bokhari

Chronic pain is a major medical problem, costing the Australian Government approximately \$10 billion a year. Pharmacological remedies are widely used but their efficacy is limited by serious side effects.

Low- level laser therapy, the use of laser applied to skin without any heating or burning of the skin, has been used empirically for the treatment of painful conditions such as arthritis and neck pain, for more than thirty years. Mechanisms for these analgesic effects have not been well understood. The Nerve Research Foundation has been engaged in recent years in examining how laser interacts with nerves to reduce pain.



We propose that laser therapy reduces pain by directly acting on pain fibres and hence, altering pain signals transmitted to the brain. Our previous study using infrared laser has shown that this wavelength temporarily interrupts the transport system in the nerves. This transport system is responsible in maintaining the vitality of the nerves and providing sufficient energy for nerve functions. We propose that transient interruption of this system by laser disrupts the conduction of pain signals along the nerve, resulting in the relief of pain in patients.

We were awarded an NHMRC grant for 2009-2011 to extend this research to examine how laser acts with nerves in the skin of living animals and human subjects, as well as in nerve cultures. We now have a PhD Student, Laita Bokhari working on this project. In October, 2008, our preliminary work presented at the World Association of Laser Therapy in South Africa, hosted by the University of Cape Town, was presented the Best Basic Science award.

As the level of pain in our aging community is projected to reach epidemic proportions in the next 20 years, low-level laser therapy offers a non-invasive, non-drug based technique for treatment of both acute and chronic pain. This has important implications for reducing costs and side-effects of drugs and improving quality of life. Understanding neural mechanisms of laser therapy will increase understanding of this modality and facilitate the development of optimal parameters and clinical guidelines.

Funding: NRF, BMRI & NH&MRC, Wenkart Foundation Scholarship

Leprosy

J D Pollard, W Britton

Leprosy is an inflammatory/infective neuropathy which has been recognised for thousands of years and remains one of the commonest neuropathies world wide. The well-known disfigurement which may occur in Leprosy effected individuals results from damage to nerves. We are collaborating with Professor Warwick Britton from the Centenary Institute and a group of veterinarians from Texas, in a study of Leprosy infection in the Armadillo. These interesting animals are infected endemically in Central America and Southern USA and hence provide a useful means to examine the mechanism of nerve damage in the disease. Work to date has shown that the Leprosy bacillus first infects the Schwann cell (the myelin producing cell) and that the earliest sign of nerve damage is demyelination and axonal damage and loss follows later. This finding has important implications for the initiation of treatment.

Funding: NH&MRC grant

The mechanism of Action of Intravenous Immunoglobulin

Intravenous Immunoglobulin (IVIg) is the most effective treatment for inflammatory neuropathies. Despite an annual cost to the health budget of at least 60 million dollars, how

IVIg works to remedy these conditions remains unknown. Current theories are based largely on the premise that its efficacy depends on the enormous diversity of individual antibodies contained within each unit (hundreds of thousands) which in turn depends on pooling blood from thousands of donors. Our research, however, suggests that the constant region of the immunoglobulin molecule activates receptors on cells in the patients' own immune system which down regulate the inflammatory reaction. If we can prove this, new means of treating autoimmune diseases may be possible. We have been awarded an NHMRC Grant to investigate this possibility.

Funding: NH&MRC grant

Multiple Sclerosis

MS is the leading cause of neurological disability in young adults, but its cause remains unknown and current therapies are only partially effective.

MS research projects in 2008 have included:

Immunopathology of Multiple Sclerosis

J W Prineas, M H Barnett, J D E Parratt, A P D Henderson

In 2004, the MS Research Unit proposed a novel paradigm of multiple sclerosis pathogenesis, namely that the myelin-forming oligodendrocyte is the target of the initial insult in newly forming lesions and the recruitment of a systemic immune response in large part a subsequent phenomenon. This landmark publication (MH Barnett and JW Prineas) was validated in 2008 by new findings published by our group in 2008 (The distribution of immunoglobulins and complement in MS, *Annals of Neurology*; MH Barnett et al), which also described the first pathological 'biomarker of the disease'. A third paper which describes the nature of, and quantitates, the inflammatory infiltrate in MS lesions of varying age has been submitted for publication (APD Henderson et al) and will complete this 'trilogy'. A review of these findings has been accepted for publication (MH Barnett et al) in *The International Journal of MS* (Figure 1).

Funding: MSRA Project Grant, Nerve Research Foundation, NSW Spinal Cord and Other Neurological Diseases Grant

Multiple Sclerosis Brain Bank

S Hawke, K Ting

With funding from MSRA and the NSW Government's Office of Science and Medical Research Australia, we have established an Australia-wide donor program enabling the collection of high quality post-mortem material from people with MS. Since the opening of the Multiple Sclerosis Research Australia Brain Bank (MSRABB) in February 2008 by Minister Hon Verity Firth, over 600 people with MS have signed up to register as potential donors. The aim of the MSRABB is to provide scientists with high quality post mortem material suitable for use in a broad range of modern scientific techniques. To date, 13 MS brains have been retrieved and stored to our specification by the Australian Brain Bank Network. As well as facilitating a wide array of research on MS in Australia, the MSRABB Tissue Characterisation Laboratory will service a number of groups working on human MS Tissue. In addition to optimising immunohistochemical analyses of the tissue, a goal will be to undertake gene and protein expression analyses of the MS post mortem material in a systematic way, and make this library of information widely available to scientists in the field.

Funding: MSRA Project Grant, BMRI

Defining the role of the blood brain barrier in MS in molecular terms

S Hawke, A Arthur, K Ting

The cerebral blood vessels may have a key role in the pathogenesis of MS. Firstly, inflammation is often centred around blood vessels in both the cerebral white matter and in the cerebral cortex. Secondly, treatment blocking interactions between activated lymphocytes and mononuclear cells and inflamed cerebral endothelium (e.g. natalizumab) is amongst the most effective current therapy for acute inflammatory episodes and MS lesion formation, and thirdly, an antibody to a water channel aquaporin-4, expressed in astrocytic foot processes that encapsulate the blood brain barrier, has been found in a sub-type of MS, Devic's disease or neuromyelitis optica. Experiments with knockout mice suggest that this water channel may have a key role in the maintenance of the blood brain barrier.



We have been purifying brain microvessels from post mortem MS brain tissue and determining which genes are dysregulated compared to control microvessels using highly sensitive DNA Array technology. In addition to surveying acute MS lesions, we have been focusing on the so-called normal appearing white matter (NAWM) in order to gain insight into the pathology of progressive MS. To date we have found that the NAWM is very abnormal in terms of gene expression and we hypothesize, given the extreme dependence of the brain on its blood supply, that defective endothelial function in MS may have a global impact on brain function and play a role in disease progression.

Funding: MSRA Project Grant, University of Sydney Medical Foundation (Jessie and Isabel Alberti Program Grant)

Proteomics of Multiple Sclerosis

M H Barnett, J W Prineas, B Crossett, L Ly

In a collaborative project with Dr Ben Crossett of the Biomedical Node of the Australian Proteomics Facility at the University of Sydney, the MS Research Unit is conducting an exciting new study which aims to define the protein profile (or proteomics) of MS lesions. Tissue is derived from the newly established University of Sydney MS Brain Bank, the NSW Tissue Resource Centre and the paraffin MS archive at the University of Sydney. In particular, this MSRA and NRF funded-project will examine newly forming MS lesions and the tissue immediately bordering expanding MS lesions. The study, which is driven by Dr Linda Ly, Postdoctoral Research Fellow, will help define molecules important in the pathogenesis of MS and ultimately facilitate the development of novel therapeutic agents.

Funding: MSRA Project Grant, Nerve Research Foundation

MS Chronobiology

M H Barnett, B Boot, S Naismith, N Rogers

Dr Brendon Boot was appointed as the inaugural MS Fellow at BMRI and Royal Prince Alfred Hospital, a position funded by the NRF and generous support from Merck-Serono. Dr Boot's research project is focused on the alteration of circadian rhythms in MS, and will make use of state of art sleep research facilities at the BMRI. Sleep disturbance is common in MS and may contribute to both fatigue and cognitive dysfunction, both prominent symptoms in MS. This pilot study will help to define the nature of sleep disturbance in MS to guide therapy which may help alleviate these debilitating symptoms.

Funding: Nerve Research Foundation, BMRI, Merck-Serono (B Boot MS Fellowship)

Alzheimer's Disease Cell Biology

C Goldsbury

The risk of Alzheimer's disease (AD), the most common form of dementia, increases exponentially at ages >65 years. Due to our rapidly ageing population, between 2000 and

2050, the number of people with dementia in Australia is expected to increase by 327%, while the total population increases by less than 40% (Alzheimer's Australia). The total financial cost of dementia in 2002 was \$6.6 billion; by 2051 the impact will increase threefold to a total of 3.3% of GDP (Access Economics). Research at all levels – cause, cure, prevention, delay and management are required if we are to tackle this major health challenge and impending epidemic. Our research aims to determine mechanisms of nerve cell dysfunction in AD with the overall goal of achieving a positive impact into understanding the underlying molecular cell biological causes of this disease.

In the AD brain, rod-like cytoskeletal inclusions containing hyperphosphorylated tau protein (tau rods) form striations in neurites called "neuropil threads". These structures often form near plaques of mis-folded amyloid- β ($A\beta$) peptides, are a strong correlate to cognitive decline and a likely underlying molecular cause of the disease. The rods span the width of neurites and there is evidence that they impair trafficking of vesicles and organelles to synaptic terminals. However little is known about their mechanism of assembly or how they impede neuronal function. Our research addresses this important issue, by focusing on the cause, mechanism and consequences of tau rod assembly in neurons. Current work builds on our new discovery that impairment of mitochondria, the cell's energy producing structures, causes the formation of tau rods and that their assembly invokes novel interactions of tau with elements of the actin cytoskeleton¹. The outcomes of our work will reveal new targets for vital future interventions to prevent the assembly of tau rods and their adverse effects on neuronal function.

Funding: NH&MRC Grant, Novartis Pharmaceuticals Australia Educational Grant, Rebecca L Cooper Medical Research Foundation, Mason Foundation, Sir Zelman Cowen Universities Fund

Motor Neuron Disease

R Pamphlett

Associate Professor Pamphlett continues to look for possible causes of motor neuron disease (MND), also known as amyotrophic lateral sclerosis (ALS). He has set up an Australia-wide DNA Bank to look for genetic differences that could make people susceptible to MND. The DNA Bank now contains samples from more than 2,400 people, and is used by local and international researchers. He also contributes to a MND tissue bank via the "Using our Brains" donor program.

His interest is in looking for novel genetic mechanisms that could underlie sporadic ALS. These include mutations that occur only in the brain, gene-environment interactions, and epigenetic gene silencing. If such a cause were found, gene therapy could be of use in treating MND.

Funding: NH&MRC grant

Refereed Publications

1. Arthur AT, Armati PJ, Bye C, Consortium SM, Heard RN, Stewart GJ, Pollard JD, Booth DR. (2008). Genes implicated in multiple sclerosis pathogenesis from consilience of genotyping and expression profiles in relapse and remission. *BMC Med Genet*, 9(1): 17.
2. Barnett MH, Parratt JDE, Pollard JD, Prineas JW. MS – one disease? Invited Review. *Int Journal MS* (accepted for publication in 2009).
3. Barnett MH, Parratt JDE, Cho E, Prineas JW. Immunoglobulins and Complement in Multiple Sclerosis: implications for pathogenesis. *Ann Neurol* (accepted for publication in 2009).
4. Goldsbury C, Whiteman IT, Jeong E, Lim YA (2008). Oxidative stress increases levels of endogenous amyloid-beta peptides secreted from primary chick brain neurons. *Aging Cell*, 7(5): 771-5
5. Granot RL, Barnett MH, Theocharous C, Pamphlett R, Hersch M. What lies beneath the tent? JC virus granule cell neuronopathy complicating sarcoidosis. *J Clin Neurosci* (accepted for publication in 2009).
6. Jufas NE, Roediger B, Armati PJ. (2008). A microwave technique for double indirect immunostaining of human brain tissue cultures with mouse monoclonal antibodies. *Appl Immunohistochem Mol Morphol*, 16(1): 83-6.
7. Lincoln JA, Cadavid D, Pollard JD, McLeod JG, Prineas JW, Dowling P, Cook SD..We should use MRI to classify and monitor the course of Multiple Sclerosis. *Archives of Neurol* (accepted for publication in 2009)
8. Luquin N, Yu B, Trent RJ, Morahan JM, Pamphlett R. An analysis of the entire SOD1 gene in sporadic ALS. *Neuromuscul Disord* 2008;18:545-52.
9. Luquin N, Yu B, Trent RJ, Pamphlett R. DHPLC can be used to detect low-level mutations in amyotrophic lateral sclerosis. *Amyotroph Lateral Scler* 2008:1-7.
10. Ng K, Howells J, Pollard JD, Burke D. (2008). Up-regulation of slow K(+) channels in peripheral motor axons: a transcriptional channelopathy in multiple sclerosis. *Brain*, 131(Pt 11): 3062-71.
11. Owe-Young R, Webster NL, Mukhtar M, Pomerantz RJ, Smythe G, Walker D, Armati PJ, Crowe SM, Brew BJ. (2008). Kynurenine pathway metabolism in human blood-brain-barrier cells: implications for immune tolerance and neurotoxicity. *J Neurochem*, 105(4): 1346-57.
12. Pamphlett R, Kum Jew S. TDP-43 inclusions do not protect motor neurons from sporadic ALS. *Acta Neuropathol* 2008;116:221-2.
13. Ruff ME, Pamphlett R. A simple method for comparing microarray genotype data between brain and other tissues. *J Neurosci Methods* 2008;173:315-7.
14. Saunderson RB, Yu B, Trent RJ, Pamphlett R. A comparison of the lengths of androgen receptor triplet repeats in brain and blood in motor neuron diseases. *J Neurol Sci* 2008;267:125-8.
15. Sutton I, Lahoria R, Tan IL, Barnett MH. CNS Demyelination and Quadrivalent HPV Vaccination. *Multiple Sclerosis* (accepted for publication in 2009)
16. Vallat J-M, Ouvrier RA, Pollard JD, Magdelaine C, Zhu D, Nicholson GA, Grew S, Ryan MM, Funalot B. (2008). Histopathological findings in hereditary motor and sensory neuropathy of axonal type with onset in early childhood associated with mitofusin 2 mutations. *J Neuropath & Exp Neurol*, 67(11): 1097-102.

Invited Lectures, Seminars and Conference Presentations

M Barnett – Multiple Sclerosis: current research directions. BMRI Scientific Retreat, Sydney, April

M Barnett – Multiple Sclerosis. Sydney GPCE Conference, Sydney, May

M Barnett – Multiple Sclerosis: a Role for Proteomics, RPAH Scientific Meeting, Sydney, July

M Barnett – CNS Demyelination and HPV Vaccination. Sydney Neuroimmunology Meeting, Sydney, August

M Barnett – ECTRIMS Update, Sydney, October

M Barnett – Multiple Sclerosis Pathogenesis. 35 year anniversary WA MS Society, Perth, November

M Barnett – CIS: Is it already MS? Ms Global Experts Board, Berlin, December

R Chow, P Armati, A Arthur, L Bokhari - The effects of 830nm and 650nm continuous wave laser irradiation on rat dorsal root ganglion neurons: relevance to neural mechanisms of clinical pain relief in acute and chronic pain. World Association of Laser Therapy, Sun City, South Africa, October

R Chow, L Laakso, P Armati, J M Bjordal, G D Baxter. A systematic review of the literature of the effects of laser irradiation on peripheral mammalian nerves: relevance to the pain relieving effects of low-level laser therapy. World Association of Laser Therapy 2008 Conference, Sun City, South Africa, 19 - 22 October

C Goldsbury, I T Whiteman. Roles of oxidative stress and energy deprivation in modulating Alzheimer's Disease-associated proteins. *Australian Neuroscience Society Meeting*, Hobart, January

E Mathey, Axonal loss in demyelinating diseases. Neurological Meeting, Royal Prince Alfred Hospital, Sydney, October 2

J D Pollard, Mechanism and Management of Inflammatory Neuropathies. The E G Robertson Memorial Lecture. Annual Scientific Meeting of Australian and New Zealand Association of Neurologists. Brisbane May

J D Pollard, Demyelinating Neuropathies. National Lecture Series for Neurology Trainees. Royal Australasian College of Physicians. Sydney March

J D Pollard, Chronic Inflammatory Demyelinating Polyneuropathy. Asian and Oceanic Congress of Myology. Melbourne November

J D Pollard, The role of IVIg in GBS and CIDP – INDAPS. Melbourne November

J W Prineas, Research objectives of the MS research group. BMR Scientific Retreat, Brain and Mind Research Institute, University of Sydney, 4-5 April

J W Prineas, Multiple sclerosis – a neuropathologist's perspective. Festschrift for Dr Gytis Danta, Canberra Hospital. 16 February

J W Prineas, Immune-mediated inflammatory demyelinating diseases of the peripheral nervous system. Festschrift for Professor John Pollard, Centenary Institute, Royal Prince Alfred Hospital, 2 June

J W Prineas, The pathology of multiple sclerosis. Festschrift for Dr Cedric Raine. Albert Einstein College of Medicine, Bronx, New York, 30 June

J W Prineas, Changing concepts in the immunopathology of multiple sclerosis. Postgraduate Course in Neurology. Westin Hotel, Sydney 4-5 April

I T Whiteman, K M Cullen, J R Bamburg, C Goldsbury. Mitochondrial dysfunction induces the accumulation of microtubule-associated protein in Alzheimer-like striated neurites. *Society of Neuroscience Meeting (USA)*, Washington DC, November



Staff of The Nerve Research Foundation 2008

Academic Staff

- Prof John Pollard AO BSC (Med) MB BS PhD, FRACP, FRCP
- A/Prof Patricia Armati, BSc, MSc, PhD
- Professor John Prineas, MB Bs, FRCP, FRCP (Edin)
- Dr Judy Spies, MB BS PhD, FRACP
- Dr Min-Xia Wang MB MD
- Dr John Parratt MB BS MD – Honorary Associate
- Dr Roberta Chow MBBS PhD, FRACGP, FAMAC, MAppSci (Med Acu)
- Dr Michael Barnett MB BS (Hons 1) FRACP PhD
- A/Prof Roger Pamphlett, MD, FRACP, FRCPATH

Post Doctoral Fellows

- Dr A Arthur BSc(Hons) PhD
- Dr E Mathey BSc(Hons) PhD

Senior Research Fellow

- Dr Claire Goldsbury PhD

Clinical Trials Staff

- Mrs Linda Pallott RN, Trials Co-Ordinator

Technical Staff

- Mr Toan Nguyen
- Ms Hung Jiew Lee
- Ms Jennifer Lee

Research Assistants

- Ms Laita Bokhari
- Ms Jennifer Lee

Higher Degrees Awarded & Students 2008

Degrees Awarded

- Ms Monique Davis PhD

Enrolled PhD Students

- Ms Laita Bokhari
- Ms Hung Jiew Lee
- Ms Ineka Whiteman

Honours Students

- Ms Xian-Jun Mah MB BS (Hons)
- Ms Erica Jeong





Research Grants

<i>Title</i>	<i>Granting Body</i>	<i>\$2008</i>
Project grant: Mechanisms of autoantibody mediated axonal injury in inflammatory demyelinating neuropathies (J Pollard, E Mathey, D Burke)	NH & MRC	\$557,250 2008-2010
Project Grant: Neural plasticity following lesions of the central nervous system in multiple sclerosis. (D Burke, K Ng, J Pollard)	NH & MRC	\$502,750 2008-2010
Project grant - Investigation of neural mechanisms of 670 & 830nm laser acupuncture in pain relief, using rat peripheral nervous tissue models. (P Armati, R Chow)	NH & MRC (alternative and complementary medicine)	\$326,207 2008-2010
Equipment Grant – Transgenic Micro-injection Setup (J Pollard, J Goetz, I Campbell, S Hawke, I Hickie)	NH & MRC	\$73,000 2008
Direct Project Grant – The MSRA Brain Bank Director (S Hawke)	MSRA	\$180,000 2008-2012
Direct Project Grant – The MSRA Brain Bank (S Hawke, M Barnett, J Pollard, J Prineas)	MSRA	\$1,000,000 2008-2012
Rehabilitating at-risk youth & young offenders through responsive service learning programs (C Lennings, D Kenny, P Armati, C Riley)	ARC Linkage Grant	\$190,000 2008 - 2011
Molecular Studies of the Blood Brain Barrier (S Hawke, J Pollard)	J and I Alberti Program Grant	\$1,500,000 2005-2010
Neuroinflammatory Mechanisms in Neurodegeneration and regeneration (J Pollard, M Bennett, R Banati, J Prineas)	NSW Health (spinal chord and other neurological diseases)	\$1,000,000 2005-2010
A DNA Bank for Motor Neuron Disease (R Pamphlett, N Laing, R Trent, B Yu,)	NH&MRC Enabling Grant	\$125,000 2006-2011
Educational Grant (C Goldsbury, I Whiteman)	Novartis Pharmaceuticals Australia	\$2,750 2008
Pathogenic mechanisms of neuronal dysfunction and drug discovery for neurodegenerative diseases (C Goldsbury, M Lovelace, M Kassiou, E Mathey, P Strappe)	R L Cooper Medical Research Foundation	\$20,000 2008
Mitochondrial dysfunction as an initiator of cytoskeletal abnormalities in Alzheimer's disease (C Goldsbury)	Mason Foundation	\$50,000 2008
Alzheimer's Disease Research Grant (C Goldsbury, K Cullen)	Sir Z Cowen Universities Fund	\$100,000 2008-2009
Neuronal energy crisis in Alzheimer's disease: Evidence from human neuropathology and cell culture models (K Cullen, C Goldsbury)	R L Cooper Medical Research Foundation	\$20,000 2008
Pain relieving effects of laser therapy – investigations into mechanisms (PhD scholarship)	Wenkart Foundation Scholarship	\$44,000 2007-2009

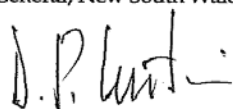
Financial Statement

	31 December 2008 \$	31 December 2007 \$
Income		
Grants	218,591	-
Scholarships, Donations and Bequests	32,639	66,990
Business & Investment Income	95,389	101,737
Internal and Other Income	403,799	250,672
Total Income	750,418	419,399
Expenditure		
Salaries	269,515	343,930
Consumables	99,802	17,640
Equipment & Repairs/Maintenance	103,107	180,722
Services and Utilities	6,300	33
Travel and Conferences	14,487	15,502
Other Expenses	85,498	126,191
Total Expenditure	578,709	684,018
Surplus/(Deficit)	171,709	(264,619)
Total Accumulated Funds as at 1 January	1,651,011	1,915,630
Total Accumulated Funds	1,822,720	1,651,011

Balance Sheet

	Notes 31 December 2008 \$	31 December 2007 \$
Assets		
Current Assets		
Funds Participating in University Pool Interest	1,620,137	1,451,011
Total Current Assets	1,620,137	1,451,011
Non-Current		
Growth Fund Investment Pool	2 202,583	200,000
Total Non-Current Assets	202,583	200,000
Total Assets	1,822,720	1,651,011
Net Assets	1,822,720	1,651,011
Equity		
Accumulated Funds	1,822,720	1,651,011
Total Equity	1,822,720	1,651,011

I certify that the Income Statement and Balance Sheet of the Foundation have been prepared in accordance with the University's accounting practices and procedures. These Foundation accounts form part of The University of Sydney's financial reports which have been audited by the Auditor-General, New South Wales.



Dominic Curtin, CA
Finance Director
Faculties of Health
March 18, 2009

With Special Thanks for our benefactors and supporters in 2008

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Nerve Research Foundation



The University of Sydney

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Front Cover: Rat oligodendrocyte progenitor cells and mature oligodendrocytes in vitro. Differentiated cultures stained for NG2 (red) and O4 (green)

We would like to thank the following for contributing to the costs of producing this Annual Report:



The Nerve Research Foundation

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