The Australian and New Zealand Society for Neuropathology
Annual Scientific Meeting, April 29th, 2006
John Dwyer Lecture Theatre, Prince of Wales Medical Hospital,
Sydney

Program

8:00-9:00am Registration
9:00-11:00am Slide Seminar
11:00-11:20am Morning Tea
11:20am-12:10pm Invited Speaker – Dennis Dickson (USA). Introduction by Glenda Halliday.
“Recent Advances in the Neuropathology and Genetics of Parkinsonism and Dementia with Lewy Bodies”
12:10-1:00pm Lunch break

Oral presentations.
Ten minutes presentation & five minutes questions.
1:00-2:30pm Oral Presentations (* Presentations eligible for the Bill Evans Memorial Young Investigator Award)
1:00-1:15 * Y Huang, W Gai, H McCann, G Halliday: LRRK2 pathology in sporadic and α-synuclein A53T mutant Parkinson’s disease.
1:15-1:30 * Z Ahmed, D Dickson: Pathological progression in progressive supranuclear palsy: the ups and downs.
1:30-1:45 * E Schofield, J Kril, G Halliday: How destructive is neuronal tau deposition in PSP?
1:45-2:00 * YJC Song, Y Huang, WP Gai, PH Jensen, GM Halliday: Correlations between striatal oligodendrogial abnormalities and neuron loss in the multiple system atrophy.
2:00-2:15 * V Hansen, R. Pamphlett: Somatic differences in gene polymorphisms or chromosome copy number – possible causes of sporadic motor neuron disease?
2:15-2:30 * JM Morahan, B Yu, RJ Trent, R Pamphlett: Does genetic susceptibility to metal toxins underlie sporadic ALS?
2:30-3:00pm Afternoon tea
3:00-4:00pm Oral Presentations (continued)
3:00-3:15 * D Pountney, MJ Raftery, F Chegeni, PC Blumbergs, WP Gai: Role of the small Ubiquitin-like Modifier, SUMO-1, in Neurodegeneration.
3:15-3:30 * V Young, GM Halliday, JJ Kril: The pathological correlate of white matter hyperintensities.
3:30-3:45 * T Garrick, L Azizi, C Harper: Brain Donation for Research: How Do Next-of-Kin Respond?
3:45-4:00 * A Green, T Garrick, D Sheedy, H Blake, C Harper: The Repeatable Battery for the Assessment of Neuropsychological status (RBANS): Normative Data for Australian Adults.

4:05-4:55pm Brain Bank Case Discussion Forum

CLOSE OF MEETING
5:00-5:30pm ANZSNP Business Meeting
5:30-6:00pm Australian Brain Bank Network Meeting

7:00 for 7:30pm ANZSNP Dinner
Thai Pothong,
294 King Street, Newtown. (ph 9550 6277).
Banquet menu: Cost ~$40 per head plus drinks and tips (BYO available).
Booking under the name of: Neuropath Society.
**Title:** Recent Advances in the Neuropathology and Genetics of Parkinsonism and Dementia with Lewy Bodies.

**Dennis W. Dickson, M.D., Department of Pathology (Neuropathology), Mayo Clinic College of Medicine, Jacksonville, FL 32224 USA**

The most common parkinsonian disorders can be assigned to one of two categories based on biochemical abnormalities in tau protein or α-synuclein. As a group these disorders have been termed tauopathies and synucleinopathies. The recent discovery of mutations in the tau gene (*MAPT*), located on chromosome 17, and their relationship to frontotemporal dementia with Parkinsonism (FTDP-17) has lead to renewed interest in tau and lead to animal models of tau pathology. Synuclein was discovered as a non-amyloid component of senile plaques in AD. Interest in synuclein was fueled by the discovery that its gene (*SNCA*), which is located on chromosome 4, was mutated in rare familial forms of Parkinson’s disease (PD). Subsequently, synuclein was shown to be present in Lewy bodies (LBs) in PD and glial inclusions in multiple system atrophy. Both tau and synuclein have a tendency to form pathologic fibrils, and mutations in each molecule favor fibril formation. The best characterized posttranslational modification of tau and synuclein is phosphorylation. Many protein kinases have been shown to phosphorylate tau, but only a few kinases phosphorylate synuclein. It is of interest that one of the most common genetic causes of PD, LRRK2, is a protein kinase. Given evidence that parkin, another molecule involved in familial juvenile-onset parkinsonism, is a ubiquitin ligase and involved in proteasome-mediated proteolysis, additional studies are required to understand the role of ubiquitination of tau and synuclein and in parkinsonian disorders.
**Abstracts:**

*Presentations marked with an * are eligible for “The Bill Evans Memorial Award” for Young Investigators.*

**Title:** LRRK2 Pathology in Sporadic and α-Synuclein A53T Mutant Parkinson’s Disease.
**Authors:** Yue Huang\(^1\), W Gai\(^2\), H McCann\(^1\) and G Halliday\(^1\)
\(^1\) Prince of Wales Medical Research Institute and UNSW, \(^2\) Human Physiology, Flinders Medical Centre, Adelaide.

**Background:** Mutations in α-synuclein and leucine-rich repeat kinase 2 (LRRK2) genes cause autosomal dominant Parkinson’s disease (PD) and Lewy body pathology. Whether LRRK2 protein directly contributes to Lewy bodies remains unknown. **Objective:** To determine LRRK2 associated pathology in PD patients with or without α-synuclein gene mutations. **Methods:** Rabbit antisera were raised against different recombinant peptides of human LRRK2 and affinity purified. Antibody specificity was ascertained by antigenic peptide absorption and for this study the antibody raised against residues 946-962 used at 1:500 dilution, due to its robust staining of human brain sections. Formalin-fixed, paraffin-embedded 10µm thick tissue sections from the substantia nigra and anterior cingulate cortex in two PD patients with the α-synuclein A53T mutation and four with sporadic cases were visualised with peroxidase and adjacent sections used to co-localise LRRK2 with α-synuclein (1:200, Transduction Laboratories, Lexington, USA) using immunofluorescence. **Results:** A similar density of LRRK2-positive Lewy neurites (LN) were found in both sporadic and A53T cases. LRRK2-positive Lewy bodies (LB) were only observed in sporadic PD, although few LB were found in the two A53T cases. Double labelling revealed that LRRK2 concentrated more centrally in a proportion of LN and LB and that there was an increased density of α-synuclein-positive LN in A53T cases (but note less LB). While the majority of LN and LB (70-80%) in sporadic PD also contained LRRK2 protein, only 25-35% of the more abundant LN were LRRK2-positive in the A53T cases. LN were fine and thin in sporadic PD and enlarged in the A53T cases. Some substantia nigra neurons in the A53T cases had LRRK2-positive cytoplasm. **Conclusion:** LRRK2 participates in LN and LB pathology in sporadic PD. In A53T cases, significantly more α-synuclein deposition occurs in expanded LN, supporting a toxic gain of function of this protein. Mutant α-synuclein appears to contribute to abnormalities in the normal cellular processing of LRRK2 in midbrain neurons.

**Title:** Pathological Progression in Progressive Supranuclear Palsy: The Ups and Downs.
**Authors:** Zehsan Ahmed and D Dickson.
Mayo Clinic College of Medicine, Jacksonville, Florida, 32224, USA

**Background:** Progressive supranuclear palsy (PSP) is a multi-system neurodegenerative tauopathy characterized clinically by an atypical parkinsonian syndrome and pathologically by the presence of tau-associated lesions, gliosis and neuronal loss in specific cortical and subcortical regions. Although diagnostic pathological criteria have been established and validated for PSP, they give no indication of the pathological severity or disease progression. **Aims:** To investigate the pathological progression of PSP in a collection of cases with varying disease durations. **Methods:** PSP cases with sufficient clinical information to determine disease duration were selected from Society for PSP brain bank (Mayo Clinic, Jacksonville, Florida). Neuronal density, tau-related degeneration and gliosis were measured in diagnostic regions and correlated with disease duration. **Results:** In the subthalamic nucleus (STN) and substantia nigra (SN) neuronal densities declined with disease duration (significant for STN). The density of STN neurofibrillary tangles (NFTs) followed a similar pattern. In contrast, the density of tufted-astrocytes (TAs) in the caudate nucleus (CN) increased with disease duration. Pathological variables in the STN, SN and CN were interrelated. Pathology in motor cortex or dentate nucleus of the cerebellum did not correlate with disease duration. **Conclusions:** This study demonstrates the pathological progression of PSP in regions with high diagnostic value. An assessment of these regions may be helpful in differentiating patients at different disease stages. Identifying early and late disease stages in PSP will assist with further understanding changes in the cellular mechanisms underlying these degenerative changes.
Progressive supranuclear palsy (PSP) is diagnosed by abnormal deposition of tau in basal ganglia and cerebellar pathways, although many cortical regions also have abnormal tau deposition of similar severity to other tauopathies. The relationship between cortical tau deposition and neuron loss in PSP has not been assessed and may be informative in determining the degree of tau neurotoxicity in PSP. Three studies have been performed on autopsy-confirmed PSP cases collected via regional brain donor programs. 1) A comparison between the gross severity of cortical atrophy using staging techniques in different tauopathies (24 pathologically-proven PSP versus 11 corticobasal degeneration versus 24 frontotemporal dementia). Compared with other endstage tauopathies, endstage PSP has limited gross cortical atrophy despite cortical tau deposition. 2) A direct comparison between the density of cortical neurons (cresyl violet stained) and those containing abnormal tau (AT8-immunoreactive) in multiple cortical regions of 11 PSP cases and 10 age- and sex-matched controls. Severe neuronal loss (average 43% reduction) was observed in focal parietal (supermarginal gyrus) and frontal (iniept) cortices, with a mild loss of neurons (average 22% reduction) observed in other frontal and anterior temporal regions (orbitofrontal, superior frontal and perirhinal cortices). In contrast, tau-immunoreactive neurons concentrated in the caudate and amygdala nuclei, hippocampus and motor cortex, with some deposition also in the anterior cingulate, superior frontal and inferior temporal cortices. Overall the distribution of tau-immunoreactive neurons did not correlate with the severity of neuron loss. 3) A direct comparison between tissue loss using volumetric morphometry and the density of cortical neurons containing abnormal tau (AT8-immunoreactive) in multiple cortical regions of 24 PSP cases and 22 age- and sex-matched controls. As expected, gross atrophy occurred subcortically (50% loss of internal globus pallidus). Focal volume loss was observed in parietal (35% reduction in supramarginal gyrus) and frontal (33% reduction in inferior frontal and 21% in motor cingulate) cortices. The distribution of AT8-immunoreactive neurons was the same as that described above, and did not correlate with volume loss. These studies show that cortical foci of neuron and tissue loss occur in PSP independent of neuronal tau deposition. In regions with significant tau deposition (eg caudate, motor cortex, superior frontal cortex) there was no measurable neuron or tissue loss. There appears to be limited neurotoxicity and tissue destruction associated with abnormal tau deposition in cortical regions in PSP.

References: Lindersson et al. Mov Disord 2005;20:34-9

---

**Title:** How Destructive is Neuronal Tau Deposition in PSP?

**Authors:** Emma Schofield, J Kri and G Halliday

1Prince of Wales Medical Research Institute & UNSW, 2Depts of Pathology and Medicine, University of Sydney

**Abstracts:**

* Presentations marked with an * are eligible for “The Bill Evans Memorial Award” for Young Investigators.

**Title:** How Destructive is Neuronal Tau Deposition in PSP?

**Authors:** Emma Schofield, J Kri and G Halliday

1Prince of Wales Medical Research Institute & UNSW, 2Depts of Pathology and Medicine, University of Sydney

**Background:** Multiple system atrophy (MSA) is characterised by severe neuron loss in basal ganglia and cerebellar pathways in association with widespread a-synuclein-positive oligodendroglial cytoplasm inclusions (GCI) but without gross demyelination. Current grading systems for MSA suggest that high GCI load occurs early in the disease decreasing with increasing severity of neuron loss. The mechanism of neuron loss remains unknown and the relationship between GCI and neuron loss is poorly understood. **Objectives:** To determine 1) any correlations between GCI load and neuron loss in the putamen and 2) the relationship between neuronal loss and the glial-concentrating a-synuclein-interacting proteins p25a and aß-crystallin. **Methods:** Ten mm thick formalin-fixed paraffin-embedded sections of the putamen from thirteen postmortem-confirmed MSA cases and eight neuropathologically normal controls were immunostained with antibodies to a-synuclein (1:200, Transduction Laboratories, USA), p25a (1:1000 from Professor Poul Henning Jensen, Lindersson et al. 2005) and aß-crystallin (1:1000, Novo Castra Laboratories, USA) respectively using peroxidase visualization counterstained with cresyl violet (0.5%). Neuron loss and GCI load (morphologically similar a-synuclein-, p25a, or aß-crystallin-immunopositive inclusions) were assessed in the putamen using the neuropathological grading system of Jellinger et al. (2005). Spearman rank correlations were used to identify significant associations. **Results:** One MSA case had stage I, nine had stage II and three had stage III striatal disease. P25a immunopositive GCI load significantly decreased in association with neuron loss. The ratio of P25a to a-synuclein-immunopositive GCI significantly reduced with increasing disease stage. aß-Crystallin-immunopositive GCI load remained consistently high across all MSA stages. **Conclusions:** Our data show that p25a accumulates early in GCI, and that with increasing neuron loss and a-synuclein aggregation into GCI, there is a reduction in p25a in MSA oligodendroglia. Further, the early and consistent presence of aß-crystallin-immunopositive GCI suggests an early and prolonged cell-stress activation in response to GCI formation.


---

**Title:** Correlations between Striatal Oligodendroglial Abnormalities and Neuron Loss in Multiple System Atrophy.

**Authors:** Yun Ju (Christine) Song, Y Huang, WP Gai, PH Jensen and GM Halliday

1Prince of Wales Medical Research Institute, and UNSW, 2Human Physiology, Flinders Medical Centre, Adelaide, South Australia, 3Institute of Medical Biochemistry, University of Aarhus, Denmark.

**Background:** Multiple system atrophy (MSA) is characterised by severe neuron loss in basal ganglia and cerebellar pathways in association with widespread a-synuclein-positive oligodendroglial cytoplasm inclusions (GCI) but without gross demyelination. Current grading systems for MSA suggest that high GCI load occurs early in the disease decreasing with increasing severity of neuron loss. The mechanism of neuron loss remains unknown and the relationship between GCI and neuron loss is poorly understood. **Objectives:** To determine 1) any correlations between GCI load and neuron loss in the putamen and 2) the relationship between neuronal loss and the glial-concentrating a-synuclein-interacting proteins p25a and aß-crystallin. **Methods:** Ten mm thick formalin-fixed paraffin-embedded sections of the putamen from thirteen postmortem-confirmed MSA cases and eight neuropathologically normal controls were immunostained with antibodies to a-synuclein (1:200, Transduction Laboratories, USA), p25a (1:1000 from Professor Poul Henning Jensen, Lindersson et al. 2005) and aß-crystallin (1:1000, Novo Castra Laboratories, USA) respectively using peroxidase visualization counterstained with cresyl violet (0.5%). Neuron loss and GCI load (morphologically similar a-synuclein-, p25a, or aß-crystallin-immunopositive inclusions) were assessed in the putamen using the neuropathological grading system of Jellinger et al. (2005). Spearman rank correlations were used to identify significant associations. **Results:** One MSA case had stage I, nine had stage II and three had stage III striatal disease. P25a immunopositive GCI load significantly decreased in association with neuron loss. The ratio of P25a to a-synuclein-immunopositive GCI significantly reduced with increasing disease stage. aß-Crystallin-immunopositive GCI load remained consistently high across all MSA stages. **Conclusions:** Our data show that p25a accumulates early in GCI, and that with increasing neuron loss and a-synuclein aggregation into GCI, there is a reduction in p25a in MSA oligodendroglia. Further, the early and consistent presence of aß-crystallin-immunopositive GCI suggests an early and prolonged cell-stress activation in response to GCI formation.

Abstracts:

* Presentations marked with an * are eligible for “The Bill Evans Memorial Award” for Young Investigators.

**Title:** * Somatic Differences in Gene Polymorphisms or Chromosome Copy Number - Possible Causes of Sporadic Motor Neuron Disease?

**Authors:** Valerie Hansen and R Pamphlett

Depts of Pathology and Molecular and Clinical Genetics, University of Sydney

**Introduction:** Motor neuron disease (MND) is a progressive neurodegenerative disorder with death 3-5 years after onset. In 90% of cases the disorder is sporadic, with no other family members affected. Although mutations in the superoxide dismutase 1 gene cause 2% of cases, no common blood DNA gene mutation for sporadic MND has been found. One hypothesis for the cause of sporadic MND is somatic mutations occurring in embryogenesis. A somatic mutation in neuronal progenitor cells would be detectable only in neurons and not in peripheral blood cells. Studies have begun to reveal increasing numbers of DNA sequence and chromosomal structural variations within the human genome. We are attempting to find if any of these changes in brain tissue underlies sporadic MND. **Methods:** We collected blood samples during life from patients with sporadic MND via the Australian MND DNA Bank, and brain tissue from the Using our Brains donor program. DNA was extracted using standard techniques from peripheral white cells and from the frontal cortex of brains. We are using Affymetrix GeneChip Human Mapping 500K Array Sets to interrogate (at the Ramaciotti Centre at UNSW) over 500,000 single nucleotide polymorphisms per DNA sample. With these we are looking for differences in single nucleotide polymorphisms, chromosome copy number and loss of heterozygosity in brain, as compared to blood, DNA in cases of sporadic MND. **Results:** The computer analysis of the GeneChip results is in progress at present. Preliminary results will be presented.


**Title:** * Does Genetic Susceptibility to Metal Toxins Underlie Sporadic ALS?

**Authors:** Julia M Morahan, B Yu, RJ Trent and R Pamphlett

Depts of Pathology and Molecular and Clinical Genetics, University of Sydney

**Introduction:** Exposure to environmental toxins such as heavy metals has been linked to an increased risk of sporadic amyotrophic lateral sclerosis (SALS). Genes that protect against heavy metals include the metallothionein (MT) family (MT-1a, MT-1e, MT-1f, MT-1g, MT-1h, MT-2a), the transcription factor for metal induced upregulation of MTs (metal transcription factor-1, MTF-1), and glutathione synthetase (GSS) which is involved in glutathione synthesis. We were interested to see if differences in these genes could lead to susceptibility to SALS. **Methods:** A case-control association study of differences in the above genes in SALS was undertaken. We examined DNA polymorphisms from 186 SALS and 186 control subjects using the Taqman® genotyping assay, a high-throughput single nucleotide polymorphism (SNP) analysis with probe-based real-time PCR. Alleles, genotypes and haplotypes were compared between SALS and controls. **Results:** A SNP associated with MT-1e (rs7403881) was different at the allele (p = 0.023) and genotype level (p = 0.025) in SALS. Haplotypes formed in the region of MT-1a and MT-1e were also significantly different. No MTF-1 SNPs in isolation differed between cases and controls, but MTF-1 haplotypes overall were different in SALS (p = 0.03), as were two specific MTF-1 haplotypes (p = 0.001 and p = 0.004). For GSS, no allele, genotype or haplotype distribution differed between groups. **Discussion:** We found an association of certain polymorphisms in MTF-1, MT-1e and MT-1a and SALS. These genes are involved in heavy metal detoxification and their dysfunction may predispose metal toxins to poison motor neurons. In addition, oxidative stress is a potential mechanism in SALS and MTs have antioxidant functions. MTF-1 upregulates several genes involved in antioxidant responses. Therefore, these genes may also be involved in SALS because of a reduced capacity to combat oxidative stress.
**Abstracts:**

* Presentations marked with an * are eligible for “The Bill Evans Memorial Award” for Young Investigators.

**Title:** Role of the Small Ubiquitin-like Modifier, SUMO-1, in Neurodegeneration

**Authors:** Dean L Pountney¹, MJ Raftery², F Chegini³, PC Blumbergs⁴, WP Gai³

¹Griffith University, Gold Coast, ²University of New South Wales, ³Flinders University, Adelaide, ⁴Institute of Medical and Veterinary Science, Adelaide.

Many neurodegenerative diseases, including Parkinson’s disease and Huntington’s disease, are characterized pathologically by the occurrence within brain cells of microscopically visible intracytoplasmic or intranuclear inclusion bodies composed of protein aggregates. We have found that proteins conjugated to the ubiquitin homologue, SUMO-1, occur in intracytoplasmic protein aggregates in dementia with Lewy bodies and multiple system atrophy, as well as in large (4-6 micron) intranuclear inclusion bodies (NIs) in the rare familial ataxia, neuronal intranuclear inclusion disease (NIID) and in smaller intranuclear aggregates in Huntington’s disease and spinocerebellar ataxia type 3 (SCA3). SUMO-1 modification plays an important role in a growing list of cellular systems, including nucleocytoplasmic trafficking, cell cycle progression, the ubiquitin proteasome system, mitochondrial fission and apoptosis. To further understand the nature of the SUMO-1 modified proteins, we isolated NIID NIs from fresh, frozen cortical tissue. Purified NIs were then dissolved in SDS and the SUMO-1-modified proteins immunoprecipitated using an anti-SUMO-1 antibody. The proteins, NSF, dynamin I and Hunc-18, involved in membrane trafficking of proteins, and the chaperone, HSP90, were identified by tandem mass spectrometry and database searching. The presence of NSF, dynamin I, Hunc-18 and HSP90 in NIs was confirmed by immunostaining of NIID tissue sections and smears of tissue homogenates. Further studies aimed at identifying the SUMO-1 modified proteins present in intracytoplasmic neuroaggregates are underway.

---

**Title:** The Pathological Correlates of White Matter Hyperintensities.

**Authors:** Vanessa Young¹, GM Halliday² and JJ Kril¹.

¹Dept of Pathology, University of Sydney, ²Prince of Wales Medical Research Institute.

Magnetic resonance imaging (MRI) often reveals areas of high signal intensity in the cerebral white matter of elderly people. These lesions are known as white matter hyperintensities (WMHs) and have been associated with numerous disorders including cerebrovascular disease, dementia, and gait disturbances. Pathological investigations into this phenomenon have so far been limited and hence the pathological correlates of WMHs remain unclear. We undertook a more comprehensive investigation by analysing each of the major components of white matter in 23 aged, formalin-fixed brains. Myelin was examined using both the Weil stain and degraded myelin basic protein antibody; vessels were examined using the Periodic acid-Schiff stain and PECAM-1 (CD31) antibody for endothelial cell-cell adhesion; glia were examined using GFAP antibody for upregulated astrocytes and human GLUT-5 antibody for microglia. We correlated measures of the various tissue components with the severity of WMHs on premortem MRI scans as rated by a semiquantitative rating scale. No correlation was found between the severity of WMHs and glia or between the severity of WMHs and myelin. However, a significant negative association was found between WMH severity and PECAM-1 score (Kruskal-Wallis statistic, p < 0.0001). A similar negative relationship was found between WMH severity and periventricular PAS score (Welch statistic, p < 0.007). These results therefore indicate that the underlying pathogenesis of WMHs is associated with a decrease in vessel number as well as a loss of vascular integrity.

Abstracts:

* Presentations marked with an * are eligible for “The Bill Evans Memorial Award” for Young Investigators.

**Title:** Brain Donation for Research: How Do Next-of-Kin Respond?
**Authors:** Therese Garrick, L Azizi and C Harper.
Dept of Pathology, University of Sydney, NSW.

**Purpose:** To investigate responses of the next-of-kin (NOK) of deceased persons to the question of brain donation for research, and to examine response pattern over a three-year period. Further, to elucidate what factors influence NOK decision-making under the necessary time pressure. **Background:** Post-mortem brain tissue is becoming increasingly valuable to psychiatric research, enabling progress in understanding and treatment. Australian rates of organ and tissue donation are low, and in an attempt to identify the reasons for this, studies have examined prospective motivation to donate. The post-mortem situation is different. NOK must make a decision under emotive circumstances and within a restricted time-frame. There is considerable confusion about the distinction between donation for research and donation for transplantation. A previous study at our centre examined NOK responses to brain donation for research over one year. The present study extends this to encompass three-year data. **Methods:** On the day of autopsy a phone call was made to the deceased's family. The senior available NOK was asked to consider donating the deceased's brain tissue to research. Responses were tabulated over three years for analysis. **Results and Conclusions:** Donation ratios were high (60%) and remained constant across the three-year data. The presence or absence of a psychiatric illness in the deceased did not affect the response. In fact, results showed that most families who decided to donate did so because the donation allowed an 'altruistic' outcome to the death. The organ donation literature indicates that people who discuss the donation question with family members are much more comfortable with their ultimate decision than those who decide alone. It is encouraging that in our study, consultation with family members more often led to a “yes” response than did a lone decision. This suggests that the positive response was a considered one, and likely to be a comfortable one.

---

**Title:** * The Repeatable Battery For the Assessment of Neuropsychological status (RBANS): Normative Data for Australian Adults.
**Authors:** Alisa Green, T Garrick, D Sheedy, H Blake and C Harper.
Dept of Pathology, University of Sydney

**Aim:** To provide preliminary normative data for the RBANS in an Australian context. **Background:** Studies of individuals with neurological and psychiatric illness indicate that cognitive impairment is often a hallmark feature of the illness. Unfortunately, traditional cognitive batteries suffer from a number of limitations including excessive test length, difficulty and cost. Also brief screening tools are often insensitive to milder forms of impairment, suffer from a lack of accompanying normative data and provide only a general indicator of cognitive functioning limiting their use with differential diagnosis. The RBANS is a relatively new screening tool that attempts to overcome these limitations by assessing diverse cognitive domains in short format using extensive normative data. Early studies suggest that the RBANS demonstrates good psychometric properties across diverse patient groups. As yet there has been no examination of differences between healthy Australian participants and the original RBANS North American normative group despite a number of studies revealing that Australian participants perform differently to other English speaking populations across a number of traditional neuropsychological measures. **Method:** Healthy control subjects (N=179) completed the RBANS (Part B) as part of joining the “Using our Brains” (UoB) tissue donor programme. This programme invites members of the general community to donate their brain to medical research when they die. Means and standard deviations were calculated across six age bands and subsequently across two educational groups. **Results:** Analysis of the results revealed higher performance in favour of Australian subjects relative to the RBANS standardisation sample on all RBANS summary scores with the largest differences found on language based tasks. Age and education were correlated significantly with a number of RBANS subtests, with education the primary predictor of RBANS performance for our sample. **Conclusion:** This study highlights the need to employ culturally specific normative data even amongst other English speaking populations to help reduce the risk of erroneous decision-making.