

Project title	Case type	Tissue supplied	Brain regions requested	Project summary
1. Localisation of 5-HT1A receptors in human brain. 2.Variability in isoform expression patterns of tyrosine hydroxylase in Parkinson's disease.	Control	Fixed	Basal Ganglia, Brainstem	Project one localised a receptor in the motor regions of the healthy human brain as this receptor is thought to participate in the beneficial effects of a new drug to treat Parkinson's disease. In project two we described differences in expression of an enzyme, which may make cells more vulnerable to death in Parkinson's disease.
Clinicopathological relationships in patients with progressive supranuclear palsy.	Control	Fixed	Various Cortical and Sub-Cortical Areas	Most existing literature on clinicopathological correlations in progressive supranuclear palsy (PSP) only address the relationship between clinical features and subcortical structures, as PSP is considered to be primarily a subcortical disease of the basal ganglia. In this study we plan to analyse the relationship between cortical (particularly frontal lobe) damage and neuropsychological features in PSP, particularly concentrating on language and visuospatial functions which have not been previously investigated.
Dopaminergic neurons in the olfactory bulb in neurodegenerative diseases.	Parkinson, Alzheimer, Frontotemporal dementia, Control	Fixed	Olfactory Bulb	There have been limited neuropathological studies of olfactory bulb in the aged and in neurodegenerative diseases. It is well established that olfactory ability declines with age. Hyposmia and anosmia (complete loss of olfaction) occur very early in Parkinson's disease and late in Alzheimer's disease. There have been no studies regarding the integrity of the olfactory system in frontotemporal dementia.
The staging and severity of multiple system atrophy.	Control	Fixed	Basal Ganglia, Brainstem, Cerebellum	The parkinsonian disorders, multiple system atrophy (MSA) and progressive supranuclear palsy (PSP), differ from Parkinson's disease (PD) pathologically in their pattern of cell loss, type of neuronal inclusions, but particularly by their glial inclusions. There have been limited quantitative studies demonstrating neuronal loss in only a few selected regions in both PSP and MSA, but few of these studies have assessed any association with clinical features. In MSA clinical correlates have been made between akinesia and cell loss in the putamen and substantia nigra, between rigidity and nigral loss, and between cerebellar ataxia and neuronal loss in the inferior olives and the cerebellar cortex. If true these relationships should also occur in PSP. Oligodendroglia in MSA and PSP accumulate intracellular inclusions, although the association with neuronal degeneration has yet to be determined.
Molecular basis of dependence in the mesocorticolimbic system.	Alcoholic, Control	Frozen	Prefrontal, Nucl. Accumbens Amygdala, Ventral Tegmental Area.	Our research aims to elucidate the molecular mechanisms underlying addiction to alcoholism and tobacco smoking. We are investigating gene and protein expression in the dopaminergic mesocorticolimbic system of alcoholics, smokers and control subjects.
Somatic mutations in Motor Neuron Disease.	Motor Neuron Disease	Frozen	Spinal Cord, Motor Cortex, Brainstem, Cerebellum.	In this group of experiments we are looking for evidence that DNA in the brain in motor neuron disease is different from DNA in other tissues, i.e. that there are somatic mutations present. These differences could be either at the genetic, chromosomal, or epigenetic level.
Analysis of gene expression and epigenetics underlying schizophrenia.	Schizophrenia, Substance Abuse	Frozen	Prefrontal	We examine the brain mechanism underlying schizophrenia with diversity of genetics and epigenetics. The quantitative difference of gene expression, splicing variations, methylation on genome and RNA modification will be examined.
Analysis of gene expression and epigenetics underlying addiction	Alcoholic, Control	Frozen	Prefrontal	The aims of the studies are to find the brain mechanism underlying vulnerability for addiction. We compare the quantitative and qualitative difference in genetics and in epigenetics between cases and controls
Analysis of the proteome of the BA46 prefrontal cortex region from post-mortem samples of subjects affected with schizophrenia compared to controls.	Schizophrenia, Control	Frozen	Prefrontal	The BA46 region has been implicated in working memory deficit and abnormal activation of this region has been observed in schizophrenia patients. We are using proteomics approach to identify proteins differentially expressed in the grey and white matter that may underlie abnormal function of the BA46 region.
Identification of disease-specific molecules expressed on cerebral endothelium - preliminary studies on cerebral endothelium from brain tissue not-suitable-for-research.	Tissue not suitable for research	Frozen	Frontal and Temporal	The aim of this project is to identify disease-specific molecules expressed on cerebral endothelium, which can assist in the transport of therapeutic molecules into the CNS, in Alzheimer's disease, Parkinson's disease, Creutzfeldt-Jakob disease and multiple sclerosis.
Protein Profiling the Human Alcoholic Brain.	Alcoholic, Wernicke-Korsakoff	Frozen	Cerebellum, BA9	Using proteomics technology, protein expression profiles of various regions of the normal post-mortem human brain will be created and compared to alcoholic brain tissue. By identifying proteins altered in the alcoholic brain, an array of potential markers will be developed for the characterisation of alcohol-related brain damage.
Analysis of the proteome of the hippocampal formation in schizophrenia.	Schizophrenia, Control	Frozen	Hippocampus, Parahippocampus	Hippocampal formation, a region critical for learning, memory consolidation and sensory gating, has been implicated in schizophrenia (SZ) pathogenesis. We are using proteome methodology to identify differentially expressed proteins in the anterior, posterior and parahippocampal gyrus that may underlie abnormal functioning of this region in SZ.
The role of Schwann cells in diabetic peripheral neuropathy.	Diabetes	Fixed	Peripheral Nerve	The role of Schwann cells in diabetic peripheral neuropathy: a pilot study using paraffin-preserved archival human material.

Proteomics in the Hippocampus of Alcoholic Human Brain.	Alcoholic, Substance Abuse, Cirrhosis, Control	Frozen	Hippocampus	Analysis of protein expression profile in the hippocampus of alcoholic human brains using proteomics-based approach.
Gene Expression Profiling in Schizophrenia.	Schizophrenia, Control	Frozen	BA9, Temporal	We are investigating the changes to gene expression in post-mortem brains in schizophrenia and controls to better understand what causes the disorder.
Investigating changes in myelin associated proteins in multiple system atrophy (MSA).	Control	Frozen	Motor Cortex White Matter	Multiple system atrophy (MSA) is pathologically characterised by the abnormal deposition of alpha-synuclein in both neurons (also occurs in other neurodegenerative diseases) and oligodendroglia (unique to MSA). Alpha-Synuclein is not normally found in oligodendroglia (Gai et al., 1999), and the intracellular changes to critical oligodendroglial proteins that must occur due to this abnormal process have not been explored
The roles of adrenoceptors and the intracellular signal transduction proteins in the aetiology of schizophrenic symptoms.	Schizophrenia, Bipolar Disorder, Control, Depression	Frozen	BA9, Visual	This study will investigate the expression of adrenoceptors and their respective intracellular signal transduction proteins in schizophrenia.
Investigation of PARK and associated gene mutations and polymorphisms in autopsy confirmed Lewy body diseases.	Parkinson	Frozen and Fixed	Occipital Pole	LRRK2 mutations account for 5% of familial and 2% of sporadic Parkinson's disease (PD). We aim to screen pathological cases of PD for LRRK2 gene mutations to understand any differences in pathological phenotypes.
The neuropathology of white matter hyper-intensities.	Specified cases	Fixed	Periventricular & Deep White Matter	The pathological substrate of white matter hyper-intensities (WMH), a common finding on MRI scans of elderly individuals, is unknown. In this study WMH will be investigated histopathologically using a number of markers of vascular integrity.
International collaboration to perform a genome wide association study in autopsy confirmed frontotemporal lobar degeneration with ubiquitin inclusions.	Frontotemporal Dementia, Control	Frozen	Cerebellum, Cortex	Research has shown that one of the most common pathologies underlying the clinical features of frontotemporal dementia is frontotemporal atrophy with ubiquitin-positive inclusions (FTD-U). The protein/s at the heart of the ubiquitinated inclusions in this disease remains a mystery, even though there is a current world-wide effort to determine their identity. For other neurodegenerative disorders significant clues concerning the proteins involved and their critical processing pathways in the brain have been determined by focusing on genetic determinants. Although FTD-U is a rare disorder, Dr Pickering-Brown's request to international colleagues currently publishing in the field for the numbers of available autopsy confirmed cases with DNA has determined that 240 FTD-U cases are available world-wide for genetic analyses. As only 20-30% of these cases have a family history of disease, the approach considered most appropriate for genetic analysis of FTD-U is a genome-wide association study. We will participate in this study by contributing sporadic and unique familial cases
Mechanisms of neurodegeneration and cognitive impairment induced by alcohol: development of neuroprotective pharmacotherapy.	Alcoholic, Control	Frozen	Hippocampus, Prefrontal	Our project is focused on transcriptional and cellular mechanisms of neurodegeneration in the human brain in association with alcohol abuse. We found that the NF-kappa B system critical for neuroplasticity and neurodegeneration is dysregulated whereas alterations in the cell suicide machinery contribute to presynaptic injury in the PFC in chronic alcoholics. These adaptations apparently contribute to cognitive impairment associated with alcoholism, and to alcohol dependence.
Changes in proteomics of brain in schizophrenia.	Schizophrenia, Control	Frozen	BA17, BA9, Caudate.	We investigate changes in brain protein expression profiles due to schizophrenia and antipsychotic drugs. The identified proteins will point out on molecular mechanisms for the disease pathogenesis and on potential targets for novel drugs development.
Impaired insulin signalling in brains of chronic alcoholics.	Alcoholic, Control	Frozen and Fixed	Cerebellum, Cingulate.	The TRC makes available, human alcoholic brain tissue that is in excellent condition and suitable for performing molecular and biochemical studies. Through analysis of the human tissue samples, we were able to determine the degree to which the impairments in insulin signalling observed in experimental rat models of chronic alcohol feeding also occur in human subjects. The research data provided us with new information about how alcohol mediates its adverse effects on brain function.
Alzheimer risk and polymorphisms of the folate/ methionine pathway.	Control	Frozen	Hippocampus	To investigate DNA damage markers and genotyping of polymorphisms within the folate /methionine pathway in Alzheimer patients and in gender and age matched controls.
A novel physiological role for cytochrome P450 enzymes in the brain?	Tissue not suitable for research	Frozen	Frontal, Hippocampus, Amygdala, Basal Ganglia	This project involves the characterisation of cytochrome P450 distribution in various regions of the brain. This work is directed towards defining the involvement of these enzymes in metabolism of indole in the human brain.
Membrane phospholipid composition in superior temporal gyrus (STG) of schizophrenia brain.	Schizophrenia, Control	Frozen	Temporal Lobe	We examined membrane phospholipid composition and neurotransmitter receptor density in the superior temporal gyrus of schizophrenia and control subjects. It was found that there is increased GABAa and reduced muscarinic M1 and M2/4 receptor binding in the STG in schizophrenia.
Schizophrenia and Neuropathology of Amygdala.	Schizophrenia, Control	Frozen	Amygdala	The aim of this project was to investigate the neuronal alterations in the amygdala that are correlated with schizophrenia. This project examined size, shape, and density of brain cells in the amygdala of control and schizophrenia cases.
Membrane phospholipid composition	Schizophrenia, Control	Frozen	Anterior Cingulate	This study examined membrane phospholipid composition and serotonin binding in the anterior cingulate cortex in

and 5HT binding efficiency in schizophrenia.				schizophrenia. Altered membrane phospholipid composition and serotonin binding was found.
Autoimmunity in Schizophrenia.	Schizophrenia, Control	Fixed	Hippocampus, BA9	My project involves looking for antibodies against the brain in the serum of people with schizophrenia. Using human brain tissue samples and commercial antibodies to selected neurotransmitter receptors we have established that the receptors are the same in people with schizophrenia and healthy controls, but that the levels of receptors are different. We are now screening serum samples for antibodies.
Proteomics of human post-mortem brain.	Alcoholic, Control	Frozen	Hippocampus	We have compared the protein expression pattern between two different regions of the normal human brain white matters (BA9 vs CC). It will show the heterogeneity among the white matters.
Analysis of the proteome of the corpus callosum from post mortem samples of subjects affected with schizophrenia compared with controls.	Schizophrenia, Control	Frozen	Corpus Callosum	Abnormalities within the corpus callosum are thought to affect inter-hemispheric communication and this in turn may underlie some of the schizophrenia symptoms. We are applying proteomics approach to identify differentially expressed proteins that may underlie these abnormalities.
3D Golgi analysis of the prefrontal cortex and hippocampus in schizophrenia/ Immunohistochemical analysis for chromogranin A and B expression in schizophrenia brains.	Schizophrenia, Control	Fixed	Prefrontal, Hippocampus	Significant decrease of chromogranin A expression was observed in the brain of schizophrenia. 3D Golgi staining was not stable enough to be used to analyse this type of brain tissue.
Immunohistochemical analysis for chromogranin A and B expression in schizophrenia brains.	Schizophrenia, Control	Fixed	Prefrontal, Hippocampus	Significant decrease of chromogranin A expression was observed in the brain of schizophrenia.
The expression of hNP22 in the frontal cortex, the hippocampus and anterior cingulate cortex of schizophrenia.	Schizophrenia, Control	Fixed	Frontal, Hippocampus, Anterior Cingulate	Significant decrease of hNP22 expression was observed in the brain of schizophrenia.
Investigation of the role of the posterior cingulate cortex as an anatomical locus of disturbed neural circuitry in schizophrenia.	Schizophrenia, Control	Fixed	Posterior Cingulate	This project examines neurotransmitter receptors in the posterior cingulate cortex in schizophrenia. To date, we have found specific alterations in glutamatergic, cholinergic, gaba-ergic, serotonergic and cannabinoid systems in this brain region in schizophrenia subjects compared to controls
Glutamate transporters in human hippocampus: alterations in schizophrenia.	Schizophrenia, Control	Fixed	Hippocampus, Visual	We have investigated whether there were changes in glutamate transporter expression in the human hippocampus in schizophrenia. We have used a panel of antibodies to examine if there are anomalies in astrocytes in this region. Our findings suggest that there is no clear evidence for an overall anomaly in glutamate regulating proteins but here may be some subtle idiosyncracies.
Understanding the molecular basis of bipolar affective disorder.	Schizophrenia, Alcoholic, Control	Frozen	Cerebellum	Using tissue from the Australian Brain Donor Programs we have been able to examine the nature of the expression patterns, mRNA splicing, etc of a recently identified bipolar disorder susceptibility gene, the protocadherin FAT. The study is also examining other genes that have altered expression in response to the FAT susceptibility alleles.
Tachynin receptors in human brain.	Schizophrenia, Control	Fixed and Frozen	Amygdala	Investigation of the localisation of tachykinin receptors in the human brain and comparison with schizophrenia.
Investigate neurological basics of schizophrenia.	Schizophrenia, Control	Frozen	Posterior Cingulate	Compare neuronal and glial cell densities within the posterior cingulate, between people who suffered with schizophrenia and controls
Molecular cell biology of hNP22: role in alcohol dependence-Amygdala, VTA, NA, PFC.	Alcoholic, Control	Frozen	Amygdala, Ventral Tegmental Area, Nucl. Accumbens, Prefrontal	Our research aims to elucidate the molecular mechanisms underlying addiction to alcoholism and tobacco smoking. We are investigating the RNA and protein expression of a novel alcohol-responsive gene in various tissue.
Molecular mechanisms of abnormal acetylcholine neurotransmission in schizophrenia	Schizophrenia, Control	Frozen	Caudate-Putamen	The role of the caudate-putamen in the pathophysiology of schizophrenia is not well understood. In this project, we aim to reveal the molecular mechanisms of abnormal acetylcholine and GABA neurotransmissions in the caudate-putamen in schizophrenia.
Entorhinal Transcriptome of Schizophrenia	Schizophrenia, Control	Frozen	Entorhinal	This project will compare glutamate receptors and related mRNAs and proteins in the entorhinal cortex of individuals diagnosed with schizophrenia with matched controls. Similar assessments will be made of the entorhinal cortex of rhesus monkeys treated chronically with the antipsychotic medications clozapine and haloperidol.
Proteomics of the nucleus accumbens in schizophrenia and substance use in schizophrenia.	Schizophrenia, Control	Frozen	Nucleus Accumbens	Using proteomic analysis, we will identify the differentially expressed proteins in the nucleus accumbens in the brain of subjects with schizophrenia and animal models of developmental vitamin D deficiency (DVD) and chronic antipsychotic treatment. Co-operating the data from both animal models with findings in humans will allow us to identify specific changes in protein expression in nucleus accumbens in schizophrenia.
Follow-on project to look at p25 $\alpha$	Controls	Frozen	Pons	In a preceding study on multiple system atrophy (MSA) we found that p25 $\alpha$ relocalizes in oligodendroglia prior to

phosphorylation				disease-associated intracellular protein accumulation. This study will use proteomic techniques to identify posttranslation modifications (eg. phosphorylation) on p25a in normal and diseased brain tissue, and analyse whether disease associated modifications can reveal pathogenic signalling pathways.
Synchrotron microprobe analysis and speciation of trace metals in neuromelanin in the healthy and parkinsonian brain.	Parkinson, Control, Incidental Lewy Body, Alzheimer	Fixed	Caudate, Putamen, Ventral Tegmental Area, Midbrain, Pons	This project using a highly sophisticated technology, the synchrotron microprobe, to investigate differences in chemical elements in the brains of persons who died with Parkinson's disease and healthy controls. This information is hoped to increase our understanding of why a small brain region, called the substantia nigra, is particularly vulnerable to brain cell death in Parkinson's disease.
Preservation of neurochemical substances in postmortem brain tissue	Controls	Frozen	Cingulate, Frontal, Striatum, Cerebellum	Postmortem brain tissue used for research often has poor clinical characterisation, particularly regarding agonal state. We propose to standardise the measurement of levels of neurochemicals such as tryptophan, phosphofructokinase, GAD and caspase-3 along with tissue pH in control cases with and without terminal hypoxic/ischaemic insult in order to find correlations that may improve quality control and case selection for the tissue banking community.
Differential regulation of human tyrosine hydroxylase isoforms and the development of Parkinson's disease	Parkinson, Control	Fixed	Ventral Tegmental Area, Midbrain, Pons	This project investigates the distribution and regulation of tyrosine hydroxylase, the rate limiting enzyme for the production of dopamine and possible changes in these factors in Parkinson's disease. Identified changes may explain the relative selective vulnerability of the dopamine-producing cells in the midbrain in this disorder.
Distribution of brain pigments in the healthy brain and in Parkinson's disease	Control	Frozen	Frontal, Midbrain	This project will characterise the apparent differential distribution of different brain pigments in the healthy brain and identify changes to these pigments in different brain regions in Parkinson's disease. This information will increase our understanding of the physiological roles of these pigments in health and disease.
New therapeutic approaches for ALS based on ER stress and induction of the UPR.	Control	Frozen	Cervical Spinal Cord	We aim to assess human ALS patient spinal cords for the presence of ER stress markers proteins in comparison to normal controls. Evidence of ER stress and induction of the unfolded protein response provides new therapeutic targets for ALS.
Gene expression in the human alcoholic brain	Alcoholic, Control	Frozen	Amygdala, Frontal	Chronic alcohol abuse produces persistent changes in brain function that are manifested as tolerance, physical dependence, craving, and other behavioral changes. We are testing the hypothesis that patterns of brain gene expression can identify novel functional pathways that are altered in alcoholics
Protein expression of the nicotinic receptors in the human adult and infant brainstem and hippocampus using immunohistochemistry.	Control	Fixed	Midbrain, Pons, Hippocampus	Comparative protein expressions of the nicotinic receptors with age in the human brainstem and hippocampus using immunohistochemistry.
Molecular mechanisms of abnormal acetylcholine neurotransmission in schizophrenia	Schizophrenia, Control	Frozen	Basal Forebrain	
Analysis of Neuroinflammatory markers in human alcoholic brain	Alcoholic, Control	Frozen	Hippocampus, Midbrain, Ventral Tegmental Area, Amygdala	
Alcohol dependence: integrating genetic and fMRI methods	Alcoholic, Control	Frozen	Ventral Tegmental Area, Prefrontal, Nucleus Accumbens	
Brain glucocorticoids and alcohol: mechanisms	Alcoholic, Control	Frozen	Frontal, Hippocampus, Cerebellum	
Functional studies of variants in the muscarinic acetylcholine receptor M2(CHRM2) that demonstrate association with alcohol dependence, depression and related phenotypes	Alcoholic, Control	Frozen	Prefrontal	
The roles of $\alpha$ - and $\beta$ -adrenoceptors, and the intracellular signal transduction proteins in the aetiology of schizophrenic symptoms.	Schizophrenia, Control, Depression	Frozen	Anterior Cingulate	
Gene Methylation and Schizophrenia	Schizophrenia, Control	Frozen and Fixed	Temporal	
Relation between dopamine, GABA and glutamate receptors in the anterior	Schizophrenia, Control	Fixed	Anterior Cingulate, Temporal	

cingulate cortex and superior temporal gyrus of schizophrenia				
Involvement of the kynurenine pathway in Alzheimer's disease	Alzheimer, Control	Fixed	Hippocampus, Temporal	
Localisation of galanin peptides and galanin receptors in human brain	Control	Fixed	Midbrain, Pons, Cerebellum	
Neuropathological changes in SID's infants and associations with known clinical risk factors.	Alzheimer	Fixed	Hippocampus	
Molecular genetic analysis of functional psychosis using postmortem brains	Schizophrenia, Control	Frozen	Hippocampus, BA9, BA17.	
An investigation of the role of alterations in soluble tau in the neurodegeneration of frontotemporal dementia subtypes.	Frontotemporal dementia	Frozen	Temporal	Frontotemporal dementia (FTD) is a spectrum of disorders characterized by a heterogeneous range of clinical phenotypes and pathologies the cause of which is unknown. The aim of this project is to determine whether alterations to soluble tau form a common mechanism of neurodegeneration across the range of FTD subtypes.
DNA microarray analysis of hypothalamus in patients with bipolar disorder.	Schizophrenia, Control, Bipolar	Fixed and Frozen	Anterior Hypothalamus	In this study, DNA microarray analysis will be performed in the hypothalamus of patients with bipolar disorder to identify the neuronal systems responsible for bipolar disorder.
Amino Acid Neurotransmission In Human Chronic Alcoholism.	Alcoholic cirrhosis	Frozen	Frontal, Motor	To determine the effect of chronic alcoholism on the levels of glutamate and GABA neurotransmitter receptors in the human brain with particular consideration of an individuals genotype. It is hoped that this will further our knowledge of the molecular mechanisms behind alcoholic brain damage and the genetic factors that could increase our susceptibility to, not only becoming alcoholic but also, the damage alcoholism can cause.
Gene Expression in Human Brain/Collaborative Study on the Genetics of Alcoholism	Alcoholic, Control	Frozen	Prefrontal, Visual, Cingulate, Thalamus, Caudate, Hippocampus, Amygdala, Putamen, Cerebellum	To characterize differences in expression of genes associated with alcohol dependence and related phenotypes as a function of genotypes and haplotypes, in specific brain regions.
Somatic mutations in ALS	Motor Neuron Disease, Control	Frozen	Prefrontal	We are collecting brain DNA from patients with sporadic MND and from controls to see if any differences in genotype or gene copy number can be detected using whole-genome analyses.
Somatic mutations in ALS	Motor Neuron Disease, Control	Pre-freezing and Frozen	Prefrontal Cortex and White Matter, Putamen, Motor, Occipital, Spinal Cord	We are analysing brain DNA from patients with sporadic MND and from controls to see if any differences in genotype, epigenetic or gene copy number can be detected using whole-genome analyses.
Assessment of cortical atrophy in MND	Motor Neuron Disease	Photographs	Whole Brain	Clinical signs of MND are often observed in cases of FTD, and vice versa. This project aims to contribute to the debated view that these be regarded as a spectrum of diseases, by determining if there are any similarities in the patterns of gross atrophy between FTD and MND
Differential regulation of human tyrosine hydroxylase isoforms and the development of Parkinson's disease.	Control	Frozen	Prefrontal, Substantia Nigra, Ventral Tegmental Area, Caudate, Putamen	This project investigates the distribution and regulation of tyrosine hydroxylase, the rate-limiting enzyme for the production of dopamine and possible changes in these factors in Parkinson's disease. Identified changes may explain the relative selective vulnerability of the dopamine-producing cells in the midbrain in this disorder.
Characterisation of Schizophrenia associated genes using a model of antipsychotic drug action.	Schizophrenia, Control	Frozen	Prefrontal	
Somatic mutations in ALS	Parkinson's Disease	Frozen	Prefrontal	We plan to analyse brain DNA from patients with sporadic ALS and compare these to controls (people with Parkinson disease and people with no neurodegenerative disease) to see if genetic differences can be detected that could be responsible for ALS.
Characterization of NSW TRC Schizophrenia cohort	Schizophrenia, Control	Frozen	Prefrontal	We will characterize the University of Sydney TRC cohort of about 32 patients with schizophrenia compared to 32 matched controls for gene expression studies in postmortem human brain.
Characterising the phenotypes of a novel causative dementia gene.	Frontotemporal dementia, Alzheimer, Control	Paraffin	Frontal, Hippocampus	A novel causative gene for dementia and motor neuron disease has recently been identified. This study will investigate the role of the gene protein product in the characteristic pathological protein deposits found in frontotemporal dementia and Alzheimer's disease, to aid in identification of potential intracellular partners interacting with this protein.

Gene Expression Profiling in Schizophrenia	Schizophrenia, Control	Frozen	Prefrontal, Superior Temporal	We are investigating gene expression profiles in a number of brain regions in subjects with schizophrenia in an effort to further the understanding of what causes this disorder.
A comprehensive methylation analysis of 1p and 19q in oligodendrogliomas.	Control	Frozen	DNA	This project will perform, for the first time, a comprehensive analysis of the methylation status of all gene promoters in the chromosomal locations 1p and 19q in oligodendrogliomas. One copy of these genes is already lost in most oligodendrogliomas and our hypothesis is that the second copy of genes involved in oligodendroglioma formation are silenced by methylation of their promoters. By studying the methylation status of genes in these locations in a large group of oligodendrogliomas we hope to identify tumour suppressor genes whose existence in these locations has long been suspected but not yet proven.
The contribution of cerebrovascular disease to Parkinson's disease.	Lewy body disease, Control	Paraffin	Frontal, Motor, Basal Ganglia	Small vessel cerebrovascular disease (SVD) is a common finding in the elderly where it is often associated with parkinsonism, however whether SVD also contributes to the movement disorder in patients with Lewy body disease is unknown. This project will examine the distribution and severity of SVD in patients with Lewy body disease and establish any correlation with severity of parkinsonism.
Neuropathological assessment of human hypothalamic tissue from patients with Huntington disease and controls.	Huntington's, Control	Fixed	Hypothalamus	Changes indicative of disturbed hypothalamic function occur early in Huntington's disease, including increased energy metabolism, weight loss, disruption of circadian rhythm and sleep changes. This project will determine whether there is substantial hypothalamic pathology in Huntington's disease using stereological methods.
The kynurenine pathway and inflammation in ALS.	Motor Neuron Disease, Control	Paraffin	Motor, Spinal cord.	The focus is on inflammation profile and so the AD tissue is compared with control and cases with other neurodegenerative diseases with and without neuroinflammation. The kynurenine pathway is a significant pathway in microglial and macrophage immune response".
MCP-1 in Alzheimer's disease	Control	Paraffin	Temporal	This project investigates inflammatory cytokine and chemokine expression (MCP-1, IL-6 and IL-8) in Alzheimer's disease brain tissue compared to age-matched controls
Differential Composition of Methionine Synthase in Brain.	Alcoholic, Control	Fixed and Frozen	Cingulate, Cerebellum	Previous studies indicate alternative splicing of methionine synthase mRNA in brains of elderly (>70 yrs) but not younger (<30 yrs) individuals, and ethanol inhibits methionine synthase activity. This project will evaluate the splicing status of methionine synthase mRNA in brain samples from alcoholic subjects vs. normal, age-matched controls.
Aquaporin 1 and 4 in paediatric brain tumours: role in oedema and infiltration.	Control	Frozen	Cerebral	Our research aim is to quantify and map the expression of AQP1 and 4 in samples of paediatric brain tumours. We also will investigate the relationship of AQPs expression with tumour-related brain oedema and tumour cell migration and dissemination.
The overlap between FTD and other neurodegenerative disorders.	Frontotemporal, Lewy body disease, Alzheimer, Control	Paraffin	Hippocampus, Frontal	The prevalence of FTD in the elderly is not known and until recently it was not possible to detect tau-negative FTD when it overlaps with other pathologies. This study will use TDP-43 staining to detect FTD in patients whose dementia onset is over 65 years.
Somatic mutations in ALS	Motor Neuron Disease	Frozen	Frontal, Spinal Cord	This group will analyse DNA from laser-dissected cells in sporadic ALS spinal cord to see if we can detect mutations in ALS candidate genes.
Neuregulin-ErbB4 pathway in schizophrenia.	Schizophrenia, Control	Frozen	Prefrontal	The Schizophrenia Research Institute collaborative project aims to understand the relationships among causative and consequential factors of the NRG1-ErbB4 pathway in the brains of patients with schizophrenia.
Characterisation of a new gene that causes FTD and MND.	Control	Frozen	Frontal	We have identified a new gene that causes familial frontotemporal dementia (FTD) and motor neuron disease (MND) in pedigrees affected with dementia and/or MND. This project will study the expression and function of this new FTD/MND gene to determine its role in the aetiology and pathology of this complex of neurodegenerative disorders.
Molecular Actions of Alcohol and Smoking on the Drug Reward Pathway of the Brain	Alcoholic, Control (smoker and non smoker)	Frozen	Prefrontal, Accumbens, Ventral Tegmental Area	This project aims to characterise alcohol- and smoking-sensitive molecular pathways, which result in structural remodelling in core regions of the mesolimbic system of the alcoholic and smoker. This pathway has a high potential to identify novel pharmaceutical targets for alcohol and nicotine addiction.
Inflammation in the pathogenesis of FTD.	Frontotemporal dementia, Pick's Control, Corticobasal degeneration,	Paraffin	Temporal	This study investigates the contribution of inflammatory processes in FTD. Information about the mechanisms involved in FTD will contribute to our understanding of the pathogenesis and progression of this debilitating disease. Collectively, studies such as this facilitate future development of therapeutic strategies aimed at slowing or preventing disease progression.
Amyloid deposition in stroke in the human brain.	Haemorrhagic stroke, Alzheimer	Fixed	Region of Stroke, Temporal, Frontal, Parietal	We will test whether the deposition of beta-amyloid occurs at the site strokes of different sizes in the aging brain. The value of these observations arises from recent evidence that amyloid-rich plaques considered Alzheimer-like dementias form at the site of bleeding. We are testing the hypothesis that bleeding precipitates the deposition of insoluble forms.