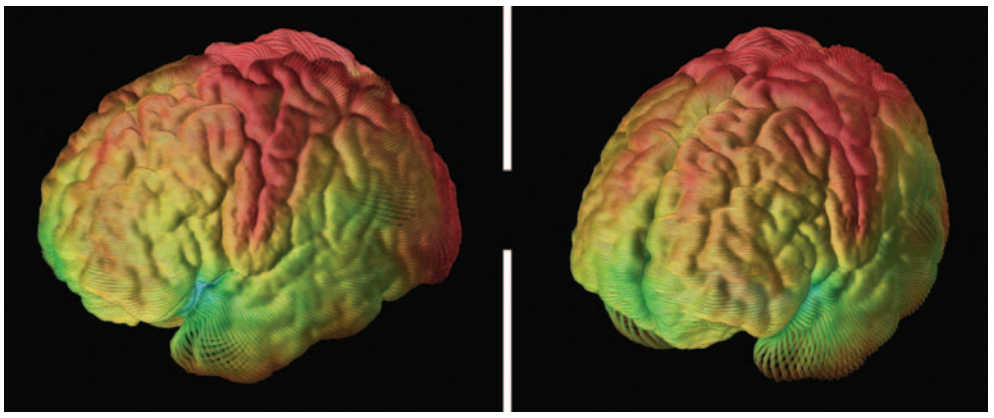
HEADLINES
Neuroscience Institute of Schizophrenia and Allied Disorders. NOVEMBER 2005

A WORLDWIDE WEB OF SCHIZOPHRENIA RESEARCH



NISAD's Paul Rasser pioneered the introduction into Australia of the 'Brain Atlasing' imaging technique which allows differences in anatomy between brains to be accurately compared. The images above show one of the brains on the 'Virtual Brain Bank' at two stages of its full 3D rotation.

The NISAD Virtual Brain Bank has been awarded a $95,000 grant from the Australian Research Council (ARC) to help the initiative expand into the world's biggest online collaborative mental health research facility.

The Virtual Brain Bank is a growing database of three-dimensional digital images of brains, currently of 250 schizophrenia patients and normal control subjects. These images are held at four separate locations: the Centre for Mental Health Studies, and the School of Behavioural Sciences at University of Newcastle; the Schizophrenia Research Unit at Liverpool Hospital, and the Laboratory of Neuro Imaging at University of California Los Angeles.

It was always intended that the Virtual Brain Bank would link to the Schizophrenia Research Register and to the DNA Bank to offer an unrivalled source of data to worldwide researchers. The ARC funding will fast track this plan with massive mobilisations of hardware and software, creating an online global centre of integrated mental health research collaborations.

This NISAD innovation has arisen in response to the new understanding within the neuroscientific community that the complexities of mental illness will only be unravelled by allowing cross-pollination of data from such diverse research fields as neuroimaging, neurobiology, cognitive neuroscience, clinical studies and genetics. Each field is now highly complex and specialised, so the need is for large-scale collaborative programs spanning multiple laboratories and mobilising many types of expertise.

It is hoped that the expanded Virtual Brain Bank will eventually provide the information and the venue for such collaborations to take place.

Cocktails & Consciousness

NISAD Gold Sponsor Margarete Ainsworth and husband Len (front row) enjoy the wit and wisdom of the Celebrity Panel with 160 other guests at 'Cocktails & Consciousness'.

Cocktails & Consciousness, NISAD's annual event for NISAD Society members, sponsors and scientists, filled the Garvan Institute's Galleria on 20 October. With the help of a celebrity panel including Greg Pickhaver ('H.G. Nelson'), Kerry Chikarovski, Andrew O'Keefe, Sally Loane and Bruce Hawker - not forgetting the wines donated by Hungerford Hill - the event was a memorable success.

For more details of the great night, see Debbie Willcox's report overleaf.

Investigating the Origins of Emotional Response

NISAD's Newcastle team combines two research techniques to investigate how schizophrenia affects recognition of emotion.

For family members one of the most upsetting characteristics of schizophrenia is how it sometimes changes a normally responsive and empathetic person into what seems to be a detached stranger. No matter how hard the family tries to convey its concern, the affected member seems not to care, or to be incapable of recognising the anxiety and disruption his/her behaviour is causing.

Earlier studies of facial emotion recognition in schizophrenia have indicated that patients scored very poorly at recognising negative facial expressions such as fear, disgust and anger, but scored close to normal at recognising happy expressions. Opinion among scientists about how this happens is divided: some say it may be due to a conscious decision to avoid acknowledging negative responses, others suggest it may be due to abnormal processing in brain regions specifically responsible for recognising negative emotions.

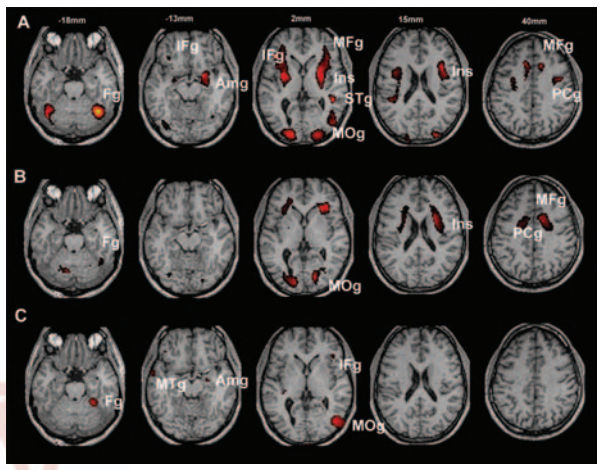
Investigating the latter hypothesis, some studies have linked this negative emotion recognition abnormality to dysfunction of the brain's limbic structures, which emotionally 'colour' all received impressions. This limbic system includes the amygdala, and is what accounts for the fact that we do not choose what to feel before we feel it.

The limbic system assigns emotional content to all incoming impressions independently and informs the cognitive frontal lobes. During emotional confrontations, for example, we all observe what is happening immediately in terms of cold information, and feel the emotional impact of it shortly after, as interpreted by the limbic system. The process takes from a fifth to a half second.

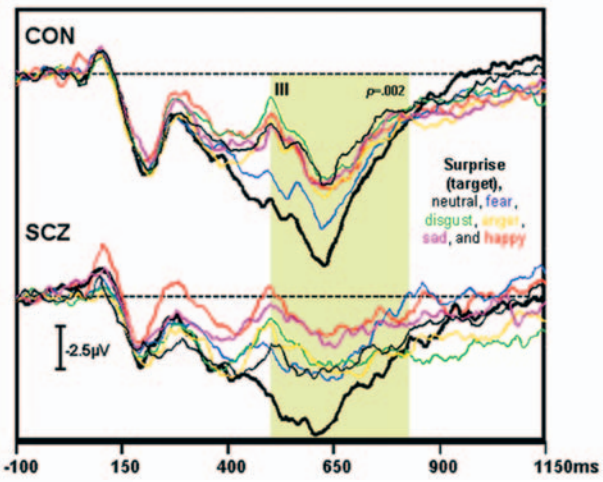
In a new study*, NISAD scientist Pat Johnston and colleagues at the University of Newcastle have combined the latest fMRI brain scanning methods with Event-Related-Potentials (ERPs - measurements of the brain's electrical activity collected via electrodes placed on the scalp) to explore how schizophrenia affects the brain's processing of emotions. The combination of the two techniques allowed the team to investigate *when* (with ERPs) and *where* (with fMRI) deficits in facial expression recognition and emotion processing occur.

ERPs were collected from 11 schizophrenia patients and 15 controls. fMRI scans were obtained from 10 patients and 10 controls. All research subjects were asked to perform the same two tasks. First, each was shown a series of 56 photos of male and female faces displaying different emotions (happiness, fear, anger, etc.) and asked to simply count the numbers of male and female faces. The ERP and fMRI records of each subject's brain activity during this 'attention to gender' performance were used to highlight any differences recorded in the second task, which was about emotion recognition. In this 'attention to expression' task, all subjects were shown the same series of faces and asked to count the number of faces showing surprise. In order to do this, they had to assess the emotional content of all 56 face images while the ERPs or fMRI recorded their brain activity.

Compared to the control subjects, schizophrenia subjects showed reduced fMRI activity in the fusiform and superior temporal gyri, a brain area known to be involved in the earliest stage of the facial recognition process. The ERP results supported this finding by showing a distortion at the beginning of the vertex positive potential, the electrical waveform associated with facial recognition. Importantly, these abnormalities were evident whether the schizophrenia subjects were recognising gender differences or expression differences.



***A.** Composite fMRI scans of all normal control subjects' brains (at five different levels) while they completed a facial emotion recognition task. The red colour shows areas of special activation. **B.** Composite Images of all schizophrenia subjects performing the same task. **C.** Significant areas of different activation are revealed to be in the fusiform gyrus (Fg), middle temporal gyrus (MTg), amygdala (Amg), inferior frontal gyrus (IFg) and middle occipital gyrus (MOg).*



Composite ERP brainwave activity recordings of normal controls (CON) and schizophrenia subjects (SCZ) while performing a facial emotion recognition task. Of particular significance is the schizophrenia subjects' uniform 2.5 microvolt difference at the start of each trace.

These results indicate that schizophrenia disrupts the brain's facial perception processes at the very earliest 'encoding' stage, *before* the more elaborate emotion recognition mechanisms of the limbic system are activated.

Further research is needed to investigate whether this disruption is indeed the root source of the emotional alienation observed in schizophrenia.

* Johnston P, Stojanov W, Devir H, Schall U. Functional MRI of facial emotion recognition deficits in schizophrenia and their electrophysiological correlates. *European Journal of Neuroscience* 2005.

INSIDE ■ Whose Brain Is It Anyway?... ■ The Sensory Deprivation Suspect .... ■ Prevention is Better than Cure....

An entertaining cocktail of consciousnesses: (L-R) panel members Andrew O'Keefe, Kerry Chikarovski, Sally Loane, Greg Pickhaver, Bruce Hawker, Dr Maryanne O'Donnell and Prof. Vaughan Carr.

On the evening of October 20, 'Cocktails & Consciousness' brought the Garvan galleria to life with good humour, great intellect and inspiration. Andrew O'Keefe hosted a most entertaining debate 'Smart drugs, is that an oxymoron?' NISAD was delighted that Greg Pickhaver, Kerry Chikarovski, Sally Loane, Bruce Hawker and Dr Maryanne O'Donnell could join NISAD Scientific Director Professor Vaughan Carr to discuss this very topical issue. More than 160 guests were entertained, and I sincerely thank the panel for making 'Cocktails & Consciousness 2005' such a successful event. I also want to acknowledge the generosity of our sponsors, Exhibition Hire, Sydney Props - and a special thank you to the team at Hungerford Hill for providing such high quality wines.

The NISAD Society is one way individuals can show their support for NISAD. For more information please visit the NISAD web site at www.nisad.org.au.

Book Your Table for 'Spark of Genius' 06

Readers will recall that NISAD took the difficult but strategic decision not to host a major fundraising event in 2005 due to the impact of the tsunami and the enormous need that it rightly demanded. I am very pleased to report that on March 24, 2006 'Spark of Genius' will return, and this time at the Sydney Town Hall. The evening will see a magically dressed Town Hall, with each table having a mystery guest. That mystery guest will be a real-life 'Spark of Genius' from the breadth of Australian life, such as the arts, sports, politics, science and media. To whet your appetite, already a number of notable Australians have agreed to be 'Sparks': Adam Elliot, Kerry O'Brien, Andrew Denton, David Williamson, Bryce Courtenay and Justice Michael Kirby are but a few. It is shaping up to be a big event on the Sydney calendar - and 100 percent of funds raised on the night will go directly to support mental health research.

However, what we now need is your support in getting friends and colleagues together to purchase tables for 8 at $5,000 per table. For further information contact Lee Drury on 02 9295 8407, or email l.drury@nisad.org.au

Update on 'The Chair'

The next phase of establishing Australia's first University Chair of Schizophrenia Research is underway. With our partners the University of NSW and the Prince of Wales Medical Research Institute, NISAD has appointed a search consultant to identify a suitable candidate. More than 40 applications have been received from eminent scientists in Australia and from overseas.

Applications closed in late October

and we hope to have identified a successful candidate before Christmas. So I look forward to introducing the Chair of Schizophrenia Research to you early in 2006, and to providing more details on the dynamic new research centre which will be established under his/her direction.

Sponsors Lunch and DVD Launch

The Hon. John Hatzistergos MLC. The NSW Minister for Health's first public engagement was at the NISAD Sponsors and Supporters Annual Lunch.

Once again the Annual Sponsors and Supporters Lunch was generously hosted by NISAD Board member and CEO of AMP, Andrew Mohl, at the AMP Board room in August.

More than 80 guests expressed their appreciation to John Hatzistergos for making the occasion his first public engagement as NSW Minister for Health.

The event also served as the launch platform for the new DVD 'Protecting Future Generations From Schizophrenia', a documentary presentation of facts about the illness, and NISAD's work to discover the means of prevention and cure.

I thank all the scientists, sponsors and supporters who took part in the filming, and especially Doug Hawkins of TVU who produced and directed it so effectively.

I would also like to express warm thanks to Angela Greensil and to Peter Foggitt who shared their personal stories about schizophrenia.

Whose brain is it, anyway?

The NSW Tissue Resource Centre investigates the barriers to brain donation - and breaks through them.

In April last year the Australian laws applying to transplant organ donation were changed to allow a registered donor's organs to be collected without first obtaining permission from relatives. This important amendment assigns first priority to the donor's wishes, and is expected to significantly reduce the number of people who die while waiting for transplants - currently around 150 deaths per year.

Under current regulations, however, a distinction is made between donation of organs for transplantation and donation for research. Unlike other organs the brain is not included in the 'list' that is available via the drivers license system or with the Australian Organ Donor Register. These regulations severely handicap mental illness researchers - and at a time when new technology is available to gain maximum research value from the donated brain tissue.

In 1995 the transplant donor rate in Australia was 10.3 donors per million of population, half the rate of the United States. This poor performance is at odds with current opinion polls, which report that around 90 per cent of Australians approve of organ donation. The discrepancy between such public opinion and actual numbers of organs donated can only be due to the bureaucratic complexity which must be unravelled before collection is permitted. And in the case of the brain, Tissue Resource Centre staff have only 48 hours after death to complete collection before the organ deteriorates and may not be useful for research purposes.

In a recent NISAD-supported study[1] Therese Garrick and colleagues have investigated the views of 180 registrants on the 'Using Our Brains' donor program, which shares the NSW Tissue Resource Centre with NISAD's 'Gift of Hope' brain donor program. Around 90 percent of the total group believed that their brains would be collected after death because they had signed the donor section of their licences or had completed donor cards. This is not the case, but is a belief held by most of the population. Most participants reported that they had decided to become a brain donor because they knew someone with a brain disease or disorder and wanted to help advance medical research. Importantly, although all donors had discussed the desire to be a brain donor with their next-of-kin, the donors believed that they shouldn't have to gain 'permission' from their next-of-kin.

Also significantly, 78 percent of registrants were members of transplant organ donor programs, as well as members of the 'Using Our Brains' research-based program. This indicates that the current legal distinction between the two is redundant, and that most donors are not concerned whether their organs are used for research or transplantation.

These findings suggest that the complex logistic and bureaucratic processes required for organ donation to research are contrary to donors' opinions or wishes, and severely handicap Australian mental health research. It is hoped that the publication of the study will help to redress the current inequity.

1. Garrick TM, Howell S, Terwee P, Redenbach J, Blake H, Harper CG. Brain donation for research - who donates and why? *Journal of Clinical Neuroscience* (in press).

The Power of Human Contact

In a second study[2], NISAD's Lisa Azizi and colleagues investigated the responses of families to the request for brain tissue donation for research. Previous research on organ donation for transplantation indicated that in NSW, refusal by families for transplant donation occurred in 56 per cent of cases in 1995, and had risen to 82 percent in 1999. Despite these figures, public opinion polls show that 90 percent of Australians are in favour of organ donation in principle. The only way to account for this discrepancy is by examining the methods whereby next of kin are provided with the opportunity to donate organs and tissues.

Earlier studies had shown that when organ coordinators were able to meet with relatives to discuss the transplant donation issue, consent was granted in 71 percent of cases, and that permissions were also readily obtained when relatives were contacted by phone. The key element seemed to be that next-of-kin needed to discuss the matter sympathetically and to have their questions answered, which was more successfully achieved by telephone or face-to-face interviews than by less personal methods such as written letters.

These findings all related to transplant donors, so Lisa Azizi and Therese Garrick set out to discover if similar results could be obtained for brain donations for research. Hitherto, telephone approaches were considered to be intolerable intrusions on grieving families. It was also believed that requests for brain donation in particular would cause offence and receive negative responses since the brain, like the heart and eyes, is considered one of the defining organs for an individual. These perceptions proved inaccurate.

Of the 48 families contacted by phone, 58 percent gave permission for the brain tissue to be collected for research. Most of the families that preferred not to donate explained that they did so only because they knew the deceased's wishes on the matter. No families were upset or offended by being contacted on the day of the autopsy, and all were interested in the details and potential of the research proposed. It was unanimously agreed upon by families that the option to donate brain tissue for research should be spread more widely.

The research team was overwhelmed by this response, which ran against all public perception. The majority of permitting families commented that the phone call and their decision had a positive effect on their emotional condition. The thought that the deceased would make such a valuable contribution was felt as a comfort in a time of sadness.

To register as a brain donor please call NISAD's 'Gift of Hope' on (02) 9295 8398 or 'Using Our Brains' on (02) 9351 2410.

2. Azizi L, Garrick TM, Merrick J, Harper CG. An Australian response to brain donation for research. *Journal of Clinical Neuroscience* (in press).

A New Model of Schizophrenia

Is sensory deprivation during brain development the single cause?

Schizophrenia is now considered to be a neurodevelopmental disorder with origins in the prenatal or neonatal period. The variety of symptoms which appear in later life are considered to be the end results of a cascade of effects possibly dating back to a single original abnormality.

The brains of people with schizophrenia have enlarged ventricals, reduced cortical thickness, increased neuronal density in the prefrontal cortex, and differences in other brain areas. Establishing the fact of these differences has occupied much of neuroscience research for the last decade, and the results of these earlier investigations have equipped researchers to now seek the cause or causes of them.

Along with brain structural and functional abnormalities, schizophrenia is also characterised by reduced pain sensitivity and reduced niacin (vitamin B3) skin flare reaction. Following this 'clue', a NISAD/University of Newcastle team proposed that the cascade of schizophrenia effects may originate with a chronic neurodevelopmental deficit in the sensory neurons. These 'afferent' neurons send information from our sensory receptors (e.g., skin, eyes, nose, tongue, ears) toward the central nervous system and the brain. They are some of the largest neurons in the body, some extending to 40 cms in length, and are responsible for triggering our senses including touch, temperature and pain, and for our sense of posture, movement and facial expression. The Newcastle team's hypothesis was that original deficits in the afferent neuronal system could starve the infant brain of the continual flow of sensory information needed for normal development.

To test this, the team employed a number of male and female newborn rats, and used injections of capsaicin under anaesthetic to produce significant permanent loss of afferent neurons in half the group, thus subjecting their developing brains to a degree of continual sensory deprivation. Then the behavioural differences of both groups were observed during 40 days of development, before both the treated and untreated groups were euthanised and differences in brain structure examined.

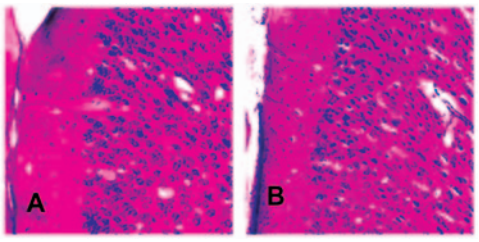
Remarkable similarities

The biological effects of the induced sensory deprivation on the treated rat brains, and their parallels with abnormalities found in human schizophrenia brains were remarkable.

Compared to the untreated rats, the brain weights of the treated male rats were significantly lower, but the female treated rat brains were the same weight as that of the untreated females.

The cortices of both male and female treated rats were thinner than normal - as found in schizophrenia.

The ventricles of the treated rats were significantly larger than those of untreated rats, and their hippocampus areas and corpus callosums were smaller. Similar abnormalities are found in schizophrenia.

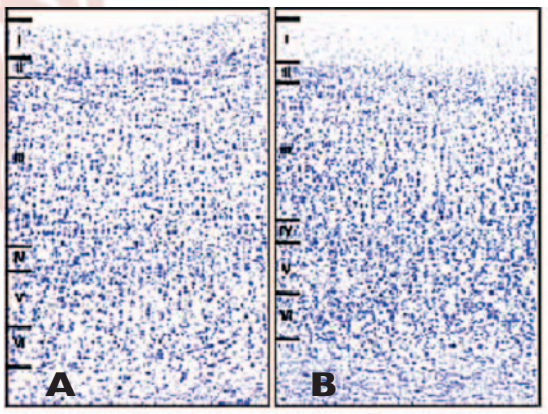


Images from the Newcastle study showing the increased neuronal density of the capsaicin-treated rat brains (A) compared to untreated rats (B).

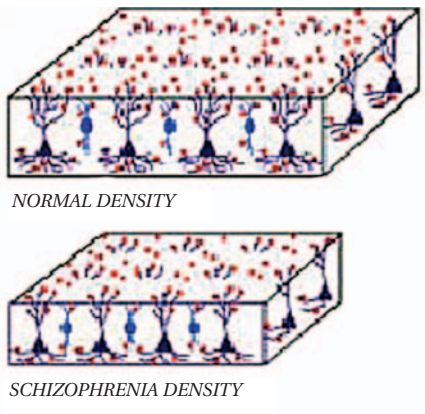
Increased neuronal density in many brain areas was observed in male and female treated rats. This is reflected in similar densities in schizophrenia.

Overall, the male treated rats were affected more severely than the females, and this aligns with the preponderance of males affected by schizophrenia.

The development of the visual cortex was unaffected in the treated rats. This brain area is unaffected by schizophrenia.



Above: Images from a 2004 study showing neuronal densities in the prefrontal cortex of normal controls (A) and schizophrenia subjects (B). Ref: LD Selemon. Am J Psychiatry 2004.



A new direction for research

Recent studies have added to the evidence that schizophrenia causes a reduction in cortex volume, and a 10 - 17 percent increase in density of brain cells - probably due to reduction of the 'neuropil' material (synaptic elements and neuronal connections) between cells. This loss of cellular connectivity could underlie abnormalities of information processing and produce the cognitive dysfunctions of the illness.

Building on this, the Newcastle study results support the adoption of a promising new model for the cause of schizophrenia, and suggest that the illness results from an intrinsic somatosensory deprivation that causes subnormal proliferation of brain cell connectivity during the brain's development.

This deficit remains undetected until the normal process of synaptic 'pruning' in adolescence further reduces connectivity to a level below the threshold required for normal processing - resulting in the gradual onset of schizophrenia symptoms.

As well as demonstrating for the first time the importance of the somatosensory nervous system to brain development, the study has signposted a valuable new direction for schizophrenia research.

Newson P, Lynch-Frame A, Roach R, Bennett S, Carr V, Chahl LA. Intrinsic sensory deprivation induced by neonatal capsaicin treatment induces changes in rat brain and behaviour of possible relevance to schizophrenia. *British Journal of Pharmacology;* 2005

The Missing Matter

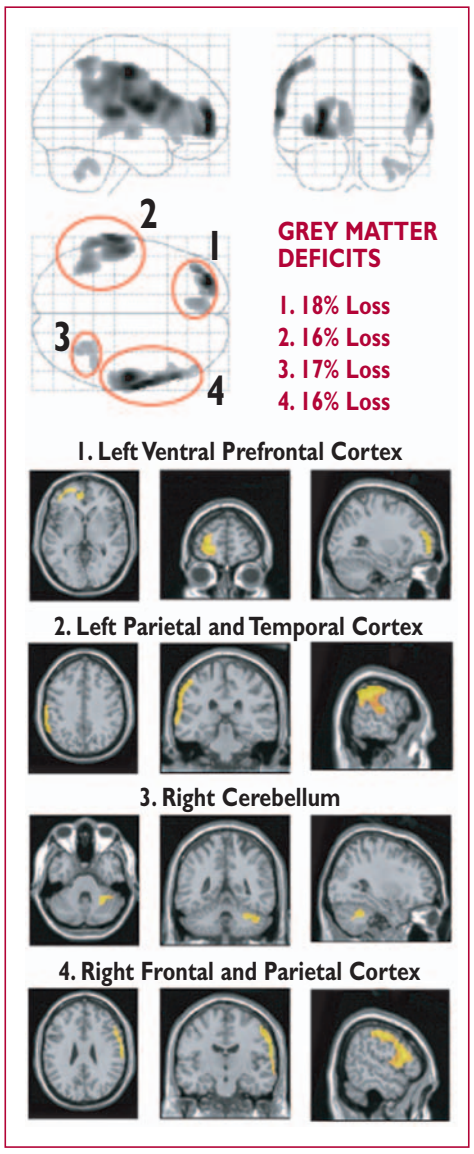
Many studies have confirmed that schizophrenia causes reductions in the grey matter of the brain, but few have investigated which brain areas are most affected and whether these areas relate to the symptoms of the illness.

A NISAD-supported study* led by Tom Whitford at the Brain Dynamics Centre, Westmead Hospital, has used MRI to compare the brain structures of 31 male and female patients with 30 healthy control subjects. All participants were around the age of 19 years, and all patients were recruited to the study within 3 months of being diagnosed with first episode schizophrenia - therefore medication effects were minimal. To further rule out any brain abnormalities not due to schizophrenia, subjects with any history of brain injury, drug abuse, depression/anxiety, or mental retardation were excluded.

The aim of the study was to identify regions of grey matter reduction, and to test whether the severity of such reductions corresponded with the type and severity of patients' symptoms.

Each patient was assessed using established tests to measure the three basic syndromes of schizophrenia: Psychomotor Poverty (inertia of thought), Disorganisation (disordered thinking), and Reality Distortion (delusions, hallucinations). The research team predicted that Psychomotor Poverty and Disorganisation would be associated with grey matter reductions in the prefrontal cortex, while Reality Distortion would be associated with such reductions in the temporal regions.

The results confirmed the findings of other studies that there was an overall deficit of grey matter in the brains of young people experiencing first episode schizophrenia, and also identified four distinct regions of such reduction (see illustrations). However, the research team found significant correspondance between degrees of grey matter reduction and severity of symptom only in the case of Reality Distortion. It was found that patients assessed with more severe Reality Distortion symptoms had correspondingly *less* grey matter deficit than other patients in three of the four brain areas identified.



The study found significant reductions of grey matter in 4 brain areas of schizophrenia subjects.

*Whitford TJ, Farrow TF, Lavier Gomes, Brennan J, Harris AW, Williams LM. Grey matter deficits and symptom profile in first episode schizophrenia. *Psychiatry Research: Neuroimaging* 2005.

Delusions as Self-Defence

NISAD-supported graduate Ryan McKay's thesis attracts high-level attention.

Dr Ryan McKay

After completing his NISAD-supported PhD thesis 'Sleights of mind: delusions and self-deception' at Macquarie University last May, Ryan McKay was offered a position at the National Hospital for Neurology and Neurosurgery in London. In September, however, Dr McKay took up a Research Associate position at Boston's Tufts University Centre for Cognitive Studies, which is headed by one of the world's most influential contemporary philosophers of mind, Prof. Daniel Dennett.

Completed at the Macquarie Centre for Cognitive Science, Dr McKay's study* investigated the motivation underlying persecutary delusions using a new instrument of measurement, the 'Paranoid, Persecutory and Delusion-Proneness Questionnaire'. His results to date have brought to light the self-defensive motivation of some delusory beliefs.

Medical opinion remains divided as to the cause of paranoid and delusury beliefs in schizophrenia. To what degree are they due to a self-defensive need to 'blame' undesirable events on delusory causes or, alternatively, to biologically malfunctioning brain cells?

Individuals with persecutory delusions may be protecting their self-esteem by projecting their negative self-representations onto others. The cost of maintaining self-esteem in this way, however, is that such people must live in a world populated by hostile, malevolent beings. A key motivation for such beliefs may also be the need to explain why the life events and social disrruptions caused by the mental illness are happening. If the individual continues to deny the reality of their mental illness, such delusions are the means which enable them to maintain such denial.

As every family member coping with a delusional relative knows, it is often impossible to convince individuals that their distorted interpretations of events are due to their illness. The human mind usually prefers to believe any other explanation, rather than the explanation that it is mentally ill.

Dr McKay's research is valuable for the ongoing development of a balanced therapy for psychosis, particularly as a component of the Cognitive Behaviour Therapy (see overleaf) which is applied with medication as standard treatment.

*McKay R, Langdon R, Coltheart M. Paranoia, persecutory delusions and attributional biases. *Psychiatry Research;* 2005.

.....Profile of a NISAD Scientist.....

Matthew Hughes

PhD student at the School of Behavioural Sciences, University of Newcastle

I am investigating the brain networks underpinning the capacity to inhibit actions which are in progress; actions that have been launched cortically but have not yet resulted in overt action. In particular, I am interested in why patients with schizophrenia have an impaired ability to trigger activity in these networks which enable us to control our behaviour. To achieve this I use functional magnetic resonance imaging (fMRI) and event-related potentials (ERPs) which permit analyses of the spatial and temporal properties of the networks.

What does NISAD mean to you? NISAD is a committed group of scientists working to achieve a common goal. Hopefully the on-going efforts of NISAD will enable us to understand the origins of schizophrenia and ultimately provide a cure.

What got you interested in researching schizophrenia? It has mostly been the motivation of my supervisor Professor Pat Michie. She has an intense passion for science, particularly schizophrenia research. Subsequently I have become fascinated by the neurological changes which result as a consequence of the disorder and how these manifest cognitively and behaviourally; it is a great intellectual challenge.

What is the most difficult thing about research? Technical problems are far and away the biggest menace to my research. They pop up out of the blue and can take months to solve. Needle-in-a-haystack stuff.

If you were not a scientist, what would you be doing? Right now I could not imagine doing anything else except perhaps traveling more extensively.

What do you do when not researching? Well there is not much time to do anything much outside of attempting to complete a PhD! However, I am quite into practicing Ashtanga yoga and spending time socialising with friends and family.

Neuroscience Institute of Schizophrenia and Allied Disorders

Patron: Her Excellency, Professor Marie Bashir AC, Governor of NSW

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NISAD also thanks 'Friends of NISAD' members...and... 'NISAD Society' members

The National Health & Medical Research Council (NHMRC) has provided $606,000 to NISAD affiliated scientists for the study 'The Effectiveness of Cognitive Behaviour Therapy for Young People at Risk of Serious Mental Disorders'.

It has long been known that schizophrenia onset is usually preceded by an often lengthy 'prodrome' period of steadily worsening symptoms, and several studies have investigated the efficacy of 'early intervention' strategies to prevent onset during this period.

Using the current 'At Risk Mental States' (ARMS) criteria to assess individual risk has successfully predicted onset in 40-50 percent of assessed cases. That is, around half of all young people tested and considered to be at risk did indeed develop psychosis within 6-12 months after assessment. It is this demonstrated ability to identify those at risk which has made preventative treatment a realistic proposition.

Early intervention treatment with Cognitive Behaviour Therapy (CBT) and low doses of antipsychotic medication has been relatively successful in interrupting the decline into psychosis and improving overall outcomes, but it remains unclear what proportion of such successes are due to CBT 'counselling' or to medication. The new study will treat a cohort of young people at risk solely with a course of CBT formulated especially for psychosis prevention. If the effectiveness of treating with CBT alone is comparable to that of CBT + low dose medication, the treatment could negate the ethical difficulties of prescribing antipsychotics to young people

who prove not to be at risk, and become viable for wide application as an early intervention therapy.

Mental Health in Rural Areas

The NHMRC has also provided $718,000 for the study 'Mental Health and Well Being in Rural and Remote NSW'. The largest investigation of its kind yet undertaken, This multicentre study will be administered from four locations in rural NSW (Orange, Broken Hill, Moree and Lismore) representing the bases of the four collaborating rural research units, and will be coordinated from the Centre for Rural and Remote Mental Health, in Orange.

This unique rural Australian study will investigate individual, family and community factors associated with the mental health and wellbeing of rural communities.

The 'VineFire' Glows Again for NISAD

Hungerford Hill once again dedicated their annual 'VineFire' event to NISAD. On 12 November, 100 guests attended the sumptuous black-tie dinner and dance, held among the giant vats in the picturesque Hunter Valley winery. Many thanks again to James Kirby and the Hungerford Hill team for the $10,500 raised!

Some of the 100 VineFire guests making merry in Hungerford Hill's 'cellar'.

New Boardmember Janet McDonald, AO

Janet McDonald has been very actively involved in women's health issues for more than 20 years. She has been a member of the National Breast Cancer Centre since 1996 and was appointed Chair in 2003. Mrs McDonald is also a member of the Drug Utilisation Sub-Committee for the Commonwealth Government. Former board memberships include; National Breast Cancer Foundation; Trustee of the Powerhouse Museum and Chair of Royal Hospital for Women.

Yen Lim

New 'Gift of Hope' Coordinator Yen Lim

Appointed as Brain Donor Program Coordinator, Yen is based in the NSW Tissue Resource Centre at the University of Sydney to promote the growth of the 'Gift of Hope' program and to assist in conducting enrolments and assessments with donors. Yen has a keen interest in schizophrenia research and was a telephone volunteer for the NSW Schizophrenia Fellowship whilst completing a degree in psychology.

YOUNG SCIENTIST SCHOLARSHIP

This year's End of Financial Year Appeal to HeadLines readers aimed at raising $30,000 to fund the first year of a 3-year schizophrenia research PhD scholarship. **The response has been the best ever recorded for any NISAD appeal.** Readers sent in donations amounting to $34,277, and the scholarship has now been advertised. The successful applicant will be announced in the next issue, and all contributors will be kept informed of the young scientist's progress over the three years of the Scholarship.

THANK YOU!

To include yourself on the mailing list to receive free HeadLines, call (02) 9295 8407.
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HeadLines is written, designed and produced by NISAD Communications Director Alan Tunbridge.
The opinions expressed in HeadLines do not necessarily represent the views of all NISAD's participating scientists.

NISAD Schizophrenia Research