bipolar disorder are because of a breakdown in communication between neurons and glia, which occurs most potently at the tripartite synapse. Our published data and recent data from our microarray study have now shown that there is a decrease in the expression of specific apolipoprotein E receptors in the CNS of subjects with schizophrenia, further supporting our hypotheses of altered neuronal glia communication in psychiatric disease and will be summarized in this presentation.

Time course of illness prior to a diagnosis of bipolar disorder or schizoaffective disorder

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Background: Evidence suggests that diagnostic delay from first episode of illness may be many years. Treating illness early in its time course is associated with a better prognosis.
Methods: Participants (n = 240) were enrolled in the Bipolar Comprehensive Outcomes Study (BCOS). A questionnaire was devised to collect information about participants from their first onset of symptoms of mental illness to when they received a diagnosis of bipolar disorder or schizoaffective disorder. The questionnaire was administered at interview by BCOS researchers when the participant was euthymic.
Results: Symptoms of mental illness were first experienced at 20.17 ± 10.26 years (mean ± SD; n = 207) and mood swings at 21.19 ± 11.76 years (mean ± SD; n = 191). Symptoms of depression were first experienced at 21.11 ± 9.98 years (mean ± SD; n = 195), a full episode of depression at 23.64 ± 9.76 years (mean ± SD; n = 191), symptoms of mania at 24.24 ± 11.48 years (mean ± SD; n = 202) and a full episode of mania at 26.43 ± 10.41 years (mean ± SD; n = 196). Medical treatment was first sought at 26.26 ± 10.18 years (mean ± SD; n = 207). Participants first received a diagnosis of bipolar disorder or schizoaffective disorder at 31.43 ± 11.34 years (mean ± SD; n = 206). Having had a previous diagnosis other than bipolar disorder or schizoaffective disorder was reported by 116 of 206 participants who answered this question.
Conclusions: Prior to being diagnosed and treated for bipolar disorder or schizoaffective disorder, participants typically experience a long time course of symptoms, episode and treatments.

A central dilemma in the mental health sector: structural imbalance

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Background: The provision of mental health services is subject to persistent criticism, often with the implication that allocated resources are inadequate. However, the mental health sector is also subject to another dilemma, which we define as a 'structural imbalance' problem.
Methods: The study shows the dimensions of structural imbalance in Australia's mental health sector by recourse to two data sets: the Mental Health and Wellbeing: Profile of Adults, Australia 1997, published by the ABS; and the National Minimum Data Set – Institutional Mental Health Services published by the AIHW. This study also examines the concept by reference to the Australian Government's COAG mental health initiatives announced in April and state government responses in July 2006.
Results: The two dimensions of the structural imbalance are 1) that some people, with no clinical manifestations of mental illness, consume mental health services and 2) another group of people have clinical manifestations of mental illness but (for various reasons) do not consume mental health services. We show how the situation coexists with various patterns of resource distribution in the public and private health sectors, acute vs. chronic conditions, institutional vs. noninstitutional service provision and private vs. public medical practice.
Conclusions: 'Throwing more money' at the preexisting structures will do nothing to address the structural imbalance problem. Remedies are discussed by reference to the content and processes of reform undertaken in the British National Health Service in recent years.

Anxiety, depression and the HPA axis in human pregnancy: links to postpartum mood

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Background: Most studies investigating maternal mood across the transition from pregnancy to the postnatal period have focused on depression. In contrast,

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little is known about patterns of anxiety across this period. This study aimed to 1) assess patterns of anxiety and depression across pregnancy and the postpartum, 2) investigate associations between antenatal mood and HPA axis hormones and 3) determine the extent to which antenatal anxiety, depression and HPA axis activity predict postnatal mood disorders.
Methods: Participants were recruited antenatally as part of a prospective study undertaken at the Royal Hospital for Women, Sydney. Ninety-four women completed self-report measures of anxiety and depression at 30–32 and 36-38 weeks gestation, and at 6 months postpartum. They were also administered a structured diagnostic interview (MINI-Plus) at 36–38 weeks gestation and at 6 months postpartum to determine the presence of DSM-IV anxiety and depression. Blood samples were collected at 30–32 weeks gestation for bioassays of HPA axis hormones (CRH, ACTH and cortisol).
Results: The data indicate significant stability in maternal mood across pregnancy and the postpartum and associations between anxiety and depression were moderate-high at each assessment. Despite the stability of depression, an anxiety disorder in pregnancy appears to be a greater risk factor for a postnatal anxiety [odds ratio (OR) = 10.20, $P < 0.005$] or depressive disorder (OR = 7.90, $P < 0.005$) than antenatal depression. Antenatal neuroendocrine parameters were unrelated to either antenatal or postnatal anxiety or depression.
Conclusion: These results clearly highlight the importance of anxiety in both the pre- and postnatal periods.

Phenotypic correlates of the serotonin transporter gene

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Background: A genetic variation within the promoter region of the serotonin transporter (5-HTT) gene has been found to moderate the effect of stressful life events on onset of major depression (Caspi et al. 2003; Wilhelm et al. 2006). This paper examines for observable characteristics underlying the genetic liability to depression following stressful events associated with differing 5-HTT genotypes within two study samples.
Method: Study 1 – 'diabetes study'. Commencing in July 2006, patients presenting to a hospital-based diabetes clinic were recruited. Participants provided data on psychiatric diagnosis, personality traits (NEO) and coping styles (COPE), as well as provided saliva samples for genetic analysis. Study 2 – 'teachers cohort study'. Between 1978 and 1998, episodes of major depression, life events, coping behaviours and trait anxiety measures (EPQ, TCI) were recorded at 5 yearly intervals. In 2003, blood or saliva samples were collected for genetic analysis.
Results: Associations between the 5-HTT gene and candidate phenotypes (trait anxiety and coping styles) were examined using preliminary data from the diabetes sample (anticipated $n > 100$). For the teachers cohort study, no associations between the 5-HTT genotype and trait anxiety were found for those who provided genetic material ($n = 128$). There were, however, significant differences on the coping behaviours used by differing genotype groups when under stress.
Conclusions: These findings raise the possibility of a genetic disposition to emotional reactivity to stressors that may predispose individuals to use different coping strategies. Replication of these findings will be examined within the diabetes sample.

Clozapine and cardiotoxicity: echocardiography findings from Barwon Health

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Background: Clozapine continues to have a unique efficacy profile that to date has not been matched or enhanced by other second-generation antipsychotics. Although agranulocytosis is a well-documented vulnerability for these patients, other serious risks, such as myocarditis and cardiomyopathy, are less well recognized and there remains a dearth of examination in this area. The current study aims to investigate changes in cardiac functioning in a group of patients treated for the first time with clozapine.
Methods: Transthoracic echocardiograms were conducted on 77 clozapine-naïve patients, prior to commencing clozapine treatment (time 1) and were repeated after 6–12 months (time 2). Patient psychiatric and medication history were documented, as were full white blood count, troponin 1 and creatinine kinase results. The rate of clozapine titration was also recorded.
Results: Preliminary analyses of the data set indicate a decrease in left ventricular shortening, a measure of ventricular contractility, from time 1 (pre clozapine) to time 2. Further analyses will be presented.

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