

IN THIS ISSUE

This issue contains one theoretical paper on the implications for neurobiological research of cognitive models of psychosis. Other sets of papers examine various aspects of psychosis, post-traumatic stress disorder (PTSD), and cognitive behavioural therapy (CBT) for panic disorder.

Cognitive models of psychosis and neurobiological research

Garety *et al.* (pp. 1377–1391) review recent evidence relevant to cognitive models of psychosis and, from this, discuss the implications for neurobiological research. The authors argue that the evidence increasingly supports a number of hypotheses proposed by cognitive models, including psychosis as a continuum, the importance of emotional processes in the occurrence and persistence of psychotic symptoms, and the importance of social adversity and trauma. A number of implications are identified, including the importance of incorporating emotional processes in neurobiological investigations, the need to study more specific clinical phenotypes (i.e. single psychotic symptoms), and the potential value in studying gene × environment × cognitive/emotion interactions.

Psychosis

This issue contains four papers on aspects of psychosis, particularly schizophrenia. In the first, Valencia *et al.* (pp. 1393–1402) report findings from a randomized controlled trial of psychosocial skills training (PSST) for schizophrenia, conducted in Mexico. Eighty-two out-patients with long-standing schizophrenia were randomly assigned to PSST ($n=43$) or treatment as usual (i.e. antipsychotic medication, $n=39$). The authors found that the PSST group showed greater improvements at 1-year follow-up across a range of domains, including symptoms and psychosocial functioning. The authors conclude that PSST, already shown to be effective in the USA and Europe, can be effective in a Latin American context.

Lorente-Rovira *et al.* (pp. 1403–1412) examined confabulation and its clinical and neuropsychological correlates in a study of 34 cases with long-standing schizophrenia (chosen to include those with and without thought disorder) and 17 healthy matched controls. Subjects were given a false recall test to elicit confabulation. The authors found that confabulation was more common in cases than controls, and more common in those cases with thought disorder than those without. The authors further report associations between confabulation and semantic memory impairment.

Kirkbride *et al.* (pp. 1413–1425) investigated individual and neighbourhood socio-economic risk factors (SERFs) for schizophrenia in 218 subjects drawn from the London arm of the AESOP study. They found that 23% of the observed variation in the incidence of schizophrenia across small geographical areas in the AESOP London site could be attributed to neighbourhood-level SERFs [e.g. ethnic segregation, voter turnout (a proxy for social capital)], independent of individual-level SERFs. The authors conclude that individual risk factors for schizophrenia may be mediated by area-level risk factors.

Bergemann *et al.* (pp. 1427–1436) examined associations between oestrogen levels during the follicular, peri-ovulatory and luteal phases of the menstrual cycle and psychotic symptoms in a sample of 125 pre-menopausal women with schizophrenia. Hormonal levels were assessed at two or more time-points in all subjects. The authors found significant improvements in psychotic, but not depressive, symptoms during the luteal phase when compared with other phases. The authors conclude that this is consistent with a possible anti-psychotic effect for oestrogen in women with schizophrenia.

Post-traumatic stress disorder

This issue contains five papers on aspects of PTSD. In the first, Breslau & Alvarado (pp. 1437–1444) investigated the impact of applying DSM-IV clinical significance criteria for PTSD on the lifetime prevalence of PTSD in two community-based samples – the 1996 Detroit Area Survey of Trauma

($n=2181$) and the Mid-Atlantic Urban Youth study ($n=1698$). In both samples, the addition of clinical significance criteria resulted in a reduction of approximately 30% in the prevalence of PTSD. Those meeting criteria of clinical significance had more persistent and pervasive disturbance, and greater functional impairment. The authors conclude that these findings support the construct validity of the clinical significance criteria.

Vythilingam *et al.* (pp. 1445–1455), in a sample of 22 patients with PTSD, 21 trauma controls, and 20 non-traumatized controls, examined two conceptualizations of the basis of emotional attention biases in PTSD: (1) emotional hyper-responsiveness, and (2) reduced priming of task-relevant representations due to ‘top-down’ regulatory systems. Subjects completed two tasks – the affective Stroop task (aST) and the emotional lexical decision task (eLDT). The authors found that, compared with the two control groups, patients showed increased interference for negative, but not positive, stimuli on the aST and increased emotional facilitation for negative words on the eLDT. These findings, the authors conclude, are consistent with emotional hyper-responsiveness being the basis of emotional attention biases in PTSD.

Kleim *et al.* (pp. 1457–1467) investigated early predictors of subsequent chronic PTSD following assault in a sample of 205 subjects recruited from a UK emergency room. Predictors were assessed 2 weeks following the assault and PTSD at 6 months. The authors found that, at 6 months, 24% met criteria for PTSD and that most of the measured predictors were associated with PTSD, including acute stress disorder and a number of cognitive and biological variables. In multivariable analyses, the predictors independently associated with PTSD were mental defeat, rumination and pre-trauma psychological problems.

Zatzick *et al.* (pp. 1469–1480) also investigated the prevalence and correlates of PTSD, this time at 12 months follow-up, in a sample of 2931 subjects hospitalized with a physical injury drawn from a multi-centre study of trauma. The authors found that, at 12 months, approximately 23% of the sample met criteria for PTSD, with a range from 0% to 44% across the study sites. Variables associated with PTSD included: early post-injury emotional distress and physical pain and pre-injury depression. Rates of PTSD were higher in Whites who were uninsured than in Whites who were insured. No similar interaction was found for Hispanic subjects.

Peres *et al.* (pp. 1481–1491) report findings from an experimental study of the neural substrates of psychotherapy-related symptom changes in 27 patients with sub-threshold PTSD. Subjects were allocated to psychotherapy ($n=16$) or waiting-list control ($n=11$) and scanned during traumatic memory retrieval using single positron emission tomography pre- and post-intervention. The authors found significantly higher activity in the parietal lobes, left hippocampus, thalamus and left prefrontal cortex during memory retrieval after psychotherapy. The authors suggest that psychotherapy may influence the development of a narrative pattern overlaying the declarative memory neural substrates.

Cognitive behavioural therapy for panic disorder

This issue concludes with two papers by Dow *et al.* (pp. 1493–1509), which report findings from analyses of the predictors and moderators of outcome following CBT for panic disorder. Data were used from a randomised controlled trial of three forms of CBT (two brief, one standard) for panic disorder, which was conducted across two sites and included 186 patients. The first paper reports on analyses of predictors of outcome at 6 months follow-up. The authors found that a number of factors were predictive of outcome, including age of initial onset of panic symptoms, co-morbid social anxieties and degree of agoraphobic avoidance. The second paper reports on analyses of moderators of outcome for two of the three treatment groups, brief (6-week) and standard (12-week) CBT. A number of factors were found to predict poor outcome for those receiving brief CBT, including higher baseline symptom severity, disability and co-morbidity and duration of current episode of panic disorder. In other words, while there were no differences in brief and standard CBT for those with less severe panic disorder at baseline, brief CBT was less effective than standard CBT for those with more severe panic disorder.

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