Psychological Medicine, 2007, **37**, 607–608.
doi:10.1017/S0033291707000451 Printed in the United Kingdom

IN THIS ISSUE

This issue contains two reviews, one on the question of 'what is a mood stabilizer?', and one on genetic influences on measures of the environment. Other sets of papers examine various aspects of genetics, bipolar disorder, and five individual papers examine a variety of topics.

What is a mood stabilizer?

In the first review, Goodwin & Malhi (pp. 609–614) examine the concept of mood stability and assess which drugs currently meet reasonable criteria as mood stabilizers. If the ideal mood stabilizer should show efficacy in the treatment of acute mania and depression, and be effective in preventing recurrence, the authors conclude from their review that, at present, only lithium comes close to being a mood stabilizer. Other drugs, the authors suggest, may have achieved the status of mood stabilizer prematurely.

Kendler & Baker (pp. 615–626) present findings from a systematic review of 55 studies that have examined the heritability of measures of environments relevant to psychiatry. Weighted heritability estimates for environments, including stressful life events and social support, ranged from 7% to 39%. The authors conclude that genetic influences on measures of the environment are pervasive in extent and modest to moderate in impact.

Genetics

This issue contains four papers examining the impact of genetics on various psychiatric disorders. In the first, Klump *et al*. (pp. 627–634) using data on 510 female adolescent twins from the Minnesota Twin study, tested the hypothesis that genetic influences on disordered eating are greater in pubertal than prepubertal girls. They found that the model of best fit in their data was consistent with their hypothesis. The authors conclude that puberty may influence the expression of genes for disordered eating.

Wade & Bulik (pp. 635–644), in a sample of 1002 female twins, aged 28–39 years, from the Australian Twin Registry, investigated the degree to which environmental and genetic factors influence two phenotypes previously associated with anorexia and bulimia: (1) undue influence of body shape and weight on self-evaluation and (2) perfectionism. They found that both genetic and environmental factors influenced both phenotypes (genetic estimates ranging from 25% to 39% of the variance). However, they could reject a model which assumed that these phenotypes were entirely caused by the same factors.

Reichborn-Kjennerud *et al*. (pp. 645–653) investigated genetic and environmental influences on cluster C personality disorders (PD), using data from 1386 young adult twin pairs from the Norwegian Institute of Public Health Twin Panel (NIPHTP). They found cluster C PDs to be moderately heritable (estimates ranging from 27% to 35%). There was no evidence of shared environmental or sex effects. Common genetic and environmental factors were found to account for a substantial proportion of avoidant and dependent PDs, but not obsessive–compulsive PD. The authors conclude that the absence of common aetiological factors across the three cluster C PDs does not support the validity of this construct.

Kendler *et al*. (pp. 655–665) using data on 3334 young adult twin pairs from the NIPHTP, examined the effect of using questionnaires or personal interviews to assess cluster A PDs on heritability estimates. First, they found that latent liabilities to paranoid PD, schizoid PD and schizotypal PD were highly heritable, but that they were assessed by current methods with only moderate reliabilty. Second, they found that questionnaires had lower specificity for all three cluster A PDs than did personal interviews.

Bipolar disorder

Four further papers examine aspects of bipolar disorder. The first continues the genetics theme. Hall *et al*. (pp. 667–678) in a sample of 94 twin pairs, examined associations between event-related potentials (ERP) and bipolar disorder (BPD) with psychosis, and whether these associations were genetic or environmental. They found that smaller P300 amplitude and decreased P50 suppression were associated with BPD. Genetic factors were the main reason for the association. The authors conclude that P300 amplitude and the P50 suppression ratio may be endophenotypes for BPD with psychosis.

Antila *et al*. (pp. 679–687) investigated cognitive function in a population-based sample of patients with familial BPD ($n=32$), unaffected relatives ($n=40$) and healthy controls ($n=50$). They found that both patients and relatives were impaired in psychomotor performance speed, compared with controls. There was weaker evidence of impairment in executive function. There were no differences between subjects in simple attention and working-memory tasks. The authors conclude that impaired psychomotor performance speed and, to a lesser degree, executive function may be endophenotypes for BPD.

Tzemou & Birchwood (pp. 689–698) examined whether dysfunctional thinking and dysregulation of traumatic memories are trait vulnerability factors for BPD, in a sample of patients with BPD ($n=29$), unipolar depression (UPD) ($n=21$), and controls ($n=20$), followed prospectively over 12 weeks. They found that the BPD (but not UPD) group was indistinguishable from controls in dysfunctional thinking. However, levels of intrusive memories were high in both BPD (45%) and UPD (48%) groups. Having fewer intrusions was associated with over-general memory retrieval. The authors conclude that affect regulation may be important in BPD and UPD and that this may be useful in informing future therapeutic interventions.

Atmaca *et al*. (pp. 699–704) investigated the morphology of the corpus callosum (CC) in 12 first-episode patients with bipolar I disorder and 12 healthy controls, using magnetic resonance imaging. They found that patients had significantly smaller areas of total CC, anterior body, posterior body, and isthmus, compared with controls. The authors conclude that CC morphology may be associated with the pathophysiology of bipolar disorder.

Other topics

This issue concludes with five papers examining a variety of topics. Korhonen *et al*. (pp. 705–715) examined the association between smoking and depression in a prospective cohort study of 10 977 adult twins in Finland. They found that persistent smoking (defined as smoking at two time-points separated by 6 years) was associated with an increased risk of depression in men, but not women, after a number of confounders were adjusted for. Further, genetic modelling among men suggested a modest association between genetic risk factors for smoking and depression.

Ayuso-Mateos *et al*. (pp. 717–725) evaluated predictors of compliance with two forms of psychological treatment for depression in 236 subjects who had taken part in the multi-centre Outcomes of Depression International Network (ODIN) study. The primary predictors of engaging with the start of treatment were: presence of a confidant, previous use of services, and desire for change score. The predictors of treatment completion were: presence of a confidant, use of antidepressants in the previous 6 months, and use of any social or health services.

Michalak *et al*. (pp. 727–736) report on quality-of-life findings from the Canadian Seasonal Affective Disorder (Can-SAD) study, a randomized-controlled trial of antidepressants *versus* light therapy for SAD in 96 patients. They found that, over an 8-week follow-up period, quality of life for both treatment groups improved markedly; there were, however, no differences in amount of improvement between the two groups. The authors conclude that treatments for SAD should routinely include quality-of-life outcomes.

Smith *et al*. (pp. 737–746) report on the development of a health-related quality-of-life measure for people with dementia, with self-report and proxy report versions. The development included item reduction and psychometric evaluation field tests. The self-report version had high reliability and moderate validity for those with mild/moderate dementia. For all dementia groups, the proxy measure had good acceptability and internal consistency; there was moderate evidence of validity.

In the final paper, Dierckx *et al*. (pp. 747–755) investigated the ability of cued recall tests (the Visual Association Test and the Memory Impairment Screen-plus Test) to differentiate between Alzheimer's disease (AD), mild cognitive impairment (MIC), and depression in a total sample of 173 subjects, including 52 controls. They found that AD patients had lower cued recall scores than those with MIC, who in turn had lower scores than those with depression. The depressed subjects and controls did not differ. When a cut-off score of 8 on the cued tests was used, the tests had high specificity (85%) and sensitivity (83%) for differentiating AD and depression; for MIC the respective figures were 85% and 58%.

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