

IN THIS ISSUE

This issue contains two reviews, one on the concept of recovery in major depression and one on internet-based cognitive behavioural therapy (CBT) for depression and anxiety. Four papers report findings from randomized controlled trials (RCT) of interventions for depression and other sets of papers examine various aspects of depression, suicide, and co-morbid generalized anxiety disorder (GAD) and major depression (MD).

Recovery in major depression

Fava *et al.* (pp. 307–317) review the literature on residual symptoms in MD, and consequent implications for current conceptualizations of depression and its treatment. The authors found that a majority of patients report residual symptoms, despite apparently successful treatment, and that such symptoms are strongly predictive of poor outcomes. The authors conclude that subclinical symptoms have important clinical implications and that treatment of residual symptoms may produce better long-term outcomes.

Internet-based CBT for depression and anxiety

Spek *et al.* (pp. 319–328) conducted a meta-analysis of 12 RCTs, with a total of 2334 participants, of internet-based CBT for depression and anxiety. They found a large mean effect size (0.96) for treatment of anxiety. When an outlier study was removed, the authors found a more modest effect size for depression (0.22). Those interventions which included therapist support showed the largest effect sizes.

Trials of treatments for depression

This issue contains four papers reporting on RCTs of treatments for depression. In the first, den Boer *et al.* (pp. 329–339) report findings from a RCT of cognitive self-therapy (CST) involving 151 subjects. They found that both the CST and a treatment as usual (TAU) comparison group improved, with no significant differences between them. Adherence to CST was 50%. Medical care utilization was lower for CST than TAU.

Loo *et al.* (pp. 341–349) report findings from a sham-controlled trial of the efficacy and safety of repetitive transcranial magnetic stimulation (rTMS) in MD over a 2-week period. In a group of 38 subjects, the authors found that active rTMS, delivered twice daily, was more effective than sham rTMS in reducing symptoms of depression. Side-effects reported from those receiving active rTMS included scalp pain and headaches, but none were long lasting.

Schene *et al.* (pp. 351–362) conducted a RCT of TAU plus occupational therapy (OT) for MD, focusing on the impact of OT on recovery, work resumption, work stress and costs, in a sample of 62 adults followed for 42 months. They found that the addition of OT to TAU did not have an extra effect on reducing symptoms, but that it did lead to a reduction in the number of work-loss days during the first 18 months, without any increase in work stress, and with consequent substantial cost savings.

Merritt *et al.* (pp. 363–372) used a cluster RCT to examine the effectiveness of an educational campaign to increase knowledge of depression and its treatment among undergraduates. Twenty-eight Oxford University colleges were randomized. The authors found no differences between intervention and control clusters in those reporting that depression could be treated effectively. However, subjects in the intervention groups were more likely to recognize symptoms of depression and to report that antidepressants were not addictive.

Other aspects of depression

Four further papers continue the focus on depression. Kivimäki *et al.* (pp. 373–382) investigated associations between socio-economic status (SES), antidepressant treatment and mortality in a cohort of over 69 000 subjects, followed over a 7-year period, as part of the Finnish 10-Town study. Although psychiatric disorders are known to be more common among those of lower SES, the authors found that men of lower SES were prescribed fewer antidepressants; there were no differences for women from different social groups. Low SES was associated with deaths from suicide and alcohol-related causes.

Naarding *et al.* (pp. 383–392) used data from two large population-based cohorts in The Netherlands, comprising over 8000 subjects, to investigate whether a distinct group of vascular depression could be identified. They found that those who were depressed and had vascular risk factors showed more loss of energy and physical disability than those without vascular risk factors. However, they did not find evidence that those with vascular risk factors had more of the symptoms presumed characteristic of vascular depression, such as psychomotor retardation and anhedonia.

Leyman *et al.* (pp. 393–402) investigated attentional biases for angry faces in 20 cases with depression and 20 controls, in order to test the proposition that depression is characterized by attentional biases for negative information. The authors found that cases maintained attention on angry faces more than on neutral faces. They further found that cases, compared with controls, showed stronger attentional engagement for angry faces. Controls more rapidly disengaged from angry faces than neutral faces.

Le Masurier *et al.* (pp. 403–410) examined emotional processing and salivary cortisol in 25 female relatives of depressed patients and 21 controls, with the aim of assessing whether similar emotional biases and raised cortisol levels are observed in relatives as in those with depression. The authors found that there were no differences in waking salivary cortisol between relatives and controls. However, compared with controls, relatives showed significantly faster recognition of fear and reduced positive bias in the emotion categorization task.

Suicide

In the first of three papers focusing on aspects of suicide, Raust *et al.* (pp. 411–419) investigated prefrontal cortex (PFC) neuropsychological function in 30 patients with suicidal behaviour and 39 healthy controls. They found significant executive function deficits in patients compared with controls, specifically in visuospatial conceptualization, spatial working memory, inhibition and visual attention. All of these are related to dorsolateral PFC rather than orbitofrontal cortex. The authors conclude that the observed deficits are consistent with prefrontal dysfunction in suicidal behaviour.

Pritchard & Amanullah (pp. 421–430) used WHO mortality data to investigate rates of suicide and other violent deaths (OVD) in 17 Islamic countries. Focusing on male rates, they found considerable variations between countries, with suicide rates ranging from 0 per million people (Qatar) to 506 per million (Kazakhstan) and OVD rates ranging from 1 per million (Syria) to 420 per million (Qatar). Only three countries had higher suicide rates than the UK, and 10 had higher OVD rates. The authors conclude that high OVD rates in Islamic countries may reflect the use of this category to record suicides.

Boden *et al.* (pp. 431–440) investigated the relationship between anxiety disorders and suicidal behaviours in adolescents and young adults using data on over 1000 subjects drawn from the Christchurch Health and Development Study. Using fixed-effects regression models, the authors were able to take account of non-observed fixed confounding factors. They found that all anxiety disorders were associated with substantial increased risk of suicidal ideation (OR 7.96) and suicide attempts (IRR 5.85). When observed and non-observed confounders were controlled, these risks reduced. The authors further found that risk of suicidal behaviours increased as the number of co-morbid anxiety disorders increased.

Co-morbid depression and anxiety

This issue concludes with two papers examining co-morbid MD and GAD. Moffitt *et al.* (pp. 441–452) used data from the Dunedin Longitudinal Study to examine whether the risk factors for GAD, MD and co-morbid GAD/MD were similar or different. Thirteen risk factors across a number of domains were considered. The authors found that co-morbid GAD/MD was associated with risk factors across all domains. GAD had risk factors similar to GAD/MD, whereas MD did not.

Kendler *et al.* (pp. 453–462) using data on over 23 000 members of same-sex twin pairs drawn from the Swedish National Twin Registry, examined the genetic correlation between GAD and MD, and the degree to which co-morbidity of GAD/MD could be explained by genetic and environmental risk factors shared with neuroticism. They found high genetic correlations between GAD and MD, although this was slightly stronger for women (+1.00) than men (+0.74). They further found that genetic risk factors indicated by neuroticism contributed only around 25% to the genetic correlation between GAD and MD in men and women.

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