

Highlights of this issue

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VISITS FOR PSYCHOSIS – TOO MUCH OF A GOOD THING?

The surprising result from the UK700 study published last year was that intensive case management produced no benefits in outcome over standard case management for severe psychotic illnesses. One reason put forward for the negative finding was that subjects had not actually received intensive care as planned. However, Burns *et al* (pp. 427–433) put those doubts to rest by reporting that contact frequency was more than doubled in the intensive case management group. An accompanying editorial by Tyrer (pp. 386–387) poses the obvious question – why was this greater frequency of contact not translated into any clinical improvement? Since standard care has already absorbed many of the key elements of assertive treatment, the differences between the groups were quantitative rather than qualitative. He suggests that additional home visits are not therapeutic in themselves and some patients may even find them intrusive. Case management is not a treatment but a structure for delivering treatments, and more evidence-based research is needed to determine which specific interventions are most effective. Bellack *et al* (pp. 434–439) also find out the hard way that more of a good thing is not necessarily better. In this instance the intervention is family therapy in schizophrenia. The intensive family therapy group received frequent (weekly or fortnightly) home visits with structured training in communication skills, while the standard therapy group attended monthly family meetings. At the end of the 2-year study period both patient groups had improved, but there were no differences between the intensive and standard groups. In fact, most

families in the intensive group did not learn or apply the communication skills taught. The authors conclude that communication training and home visits are not a cost-efficient addition to family therapy.

NEW AND NOT-SO-NEW TREATMENTS FOR DEPRESSION

Cognitive therapy is well-established as a treatment for acute depression but its effect in residual depression is not clear. Scott *et al* (pp. 440–446) report that the addition of cognitive therapy to clinical management for residual depression produced significant improvements in specific psychological symptoms: guilt, self-esteem, pessimism and hopelessness. A thought-provoking review by McQuade & Young (pp. 390–395) presents evidence for dysfunction in glucocorticoid-receptor-mediated auto-regulation of the hypothalamic–pituitary–adrenal axis in depression. Novel pharmacological strategies for treating mood disorders could involve reinstatement of normal feedback mechanisms. Glucocorticoid receptor antagonists and inhibitors of steroid synthesis are promising therapeutic targets.

TAKING A CHANCE WITH TRYPTOPHAN

Rapid tryptophan depletion reduces brain serotonin and leads to decreased mood in patients recovered from unipolar depression. A brave study by Hughes *et al* (pp. 447–451) set out to investigate the effects of acute tryptophan depletion on patients with bipolar disorder stabilised on lithium. Luckily, the authors found no alteration of mood or suicidality in their subjects, and

they conclude that the beneficial effects of lithium are not reversed by perturbation of brain serotonin levels.

NEUROSIS, PSYCHOSIS AND OCD

Neuroticism is known to be a risk factor for depression. However, this risk-increasing effect appears to extend to other disorders also. Lewis *et al* (pp. 416–420) find an increased risk of later schizophrenia among those with a diagnosis of neurosis or personality disorder at age 18 years. Samuels *et al* (pp. 457–462) find that that neuroticism and obsessive–compulsive personality disorder are common among relatives of patients with obsessive–compulsive disorder, and speculate that these conditions may be part of a familial spectrum of OCD.

HOW DO ATYPICAL ANTIPSYCHOTICS WORK?

Two papers in this month's *Journal* investigate this question. A SPET study by Stephenson *et al* (pp. 408–415) finds that quetiapine shows marked limbic-selective D₂/D₃ receptor blockade. A PET study by Liddle *et al* (pp. 402–407) demonstrates that a single dose of risperidone causes significant reduction of glucose metabolism in cortico–striato–thalamic circuits and the left hippocampus. The magnitude of decrease in hippocampal activity predicts subsequent reduction in positive symptoms. We do not yet know whether these disparate findings will fit together into a coherent mechanism of action for atypical antipsychotics.

THE DIANA EFFECT

The publication of Goethe's *The Sorrows of Young Werther* was followed by a series of suicides of young people seemingly related to the novel – better known as the 'Werther effect'. The UK now has its own 'Diana effect'. Hawton *et al* (pp. 463–466) report an increase in suicides in Oxford in the month following the death of the Princess of Wales. This increase occurred particularly in young women and emphasises the symbolic importance of the Princess among this age group.