Highlights of this issue

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NUTRITIONAL TREATMENT FOR PSYCHOSIS (POSSIBLY)

Amino acid mixtures lacking tyrosine decrease the release of dopamine produced by the psychostimulant drug amphetamine. McTavish *et al* (pp. 356–360), in a double-blind crossover trial, administered a tyrosine-free amino acid mixture and a control mixture to 16 healthy volunteers and 20 with mania 4 hours before methamphetamine. The tyrosine-free mixture lowered both subjective and objective measures of the psychostimulant effects of methamphetamine. Ratings of mania were lower in patients who received the tyrosine-free mixture. The intriguing clinical implication of this finding is that certain nutritional strategies may be useful adjuncts in the treatment of psychiatric disorders such as mania, schizophrenia and substance misuse.

COGNITIVE FUNCTION AND DURATION OF UNTREATED PSYCHOSIS

It has been suggested that the toxic effect on the brain from untreated psychosis may adversely affect cognitive functioning. Norman *et al* (pp. 340–345) administered a battery of cognitive tests to 113 first-episode patients and examined possible predictors of poor cognitive performance, including duration of untreated psychosis (DUP). Although poor performance was associated with female gender, being left-handed and having had fewer years of education, there was no evidence that DUP adversely affected cognitive performance early in the course of treatment.

DEPRESSION ACROSS EUROPE

The Outcome of Depression International Network (ODIN) aims to provide data on the prevalence of and risk factors for depressive disorders in Europe using uniform methodology across sites. Ayuso-Mateos *et al* (pp. 308–316), in a cross-sectional two-phase community study using the Beck Depression Inventory followed by the Schedules for Clinical Assessment in Neuropsychiatry, report the prevalence of depression across five European countries (at nine urban and rural sites) to average 8.6%, but with considerable variation between sites. Study centres fell into three categories – high, medium and low depression prevalence – with the urban UK site having the highest prevalence of all.

INCREASED RISK OF SCHIZOPHRENIA IN CITIES

People with schizophrenia are more likely to live in socially deprived areas and occupy lower socio-economic positions. It is unclear whether these factors contribute to the aetiology of the illness or result from social 'drift' after birth. Methods used to estimate rural/urban incidence vary across studies and as such it is unclear whether reported differences are real or artefactual. Allardyce *et al* (pp. 335–339), compare service-based incidence rates for schizophrenia in a rural part of Scotland with those in an inner-city area of London using identical methods in both areas. The incidence in the urban area was found to be 61% higher than in the rural area. This excess could be largely explained by the high incidence of schizophrenia in the non-White urban population. Harrison *et al* (pp. 346–350), using a matched case–control design, investigate the relationship between social inequality at birth and later development of schizophrenia. Risk of schizophrenia increased with increasing deprivation at birth, adding to the accumulating evidence for the role of environmental factors in the aetiology of schizophrenia.

PATIENTS PREFER DEPOTS

Two review papers in this issue provide an update on depot antipsychotic drugs. Adams *et al* (pp. 290–299) provide a synthesis and quantitative summary of the findings of the Cochrane depot reviews. The overall evidence is that depots are safe and effective and confer a small benefit over oral drugs on global outcome. No statistical difference in relapse rates has been demonstrated. Walburn *et al* (pp. 300–307), examine patient and nurse attitudes to depots. From the limited evidence available, patients appear to prefer depots.

AGGRESSION AND COMT

Aggression in schizophrenia has previously been associated with polymorphisms of the catechol-*O*-methyltransferase gene (COMT). The COMT gene inactivates catecholamines and a common polymorphism results in high and low enzyme variants. Jones *et al* (pp. 351–355) investigate this association using the largest sample to date. Findings suggest an association between the high-activity COMT homozygote and aggression – in contrast to previous links with the low-activity homozygote.

OCD – PREVALENCE AND STRUCTURAL ABNORMALITIES

Heyman *et al* (pp. 324–329) report the prevalence of OCD to be 0.25% in a UK nationwide epidemiological study of young people aged 5–15 years. Results show exponential increases in each increasing age band and equal rates in boys and girls. Compared with normal controls, children with OCD were more likely to be from lower socio-economic class and of lower intelligence, in contrast to the beliefs stated in much of the early anecdotal literature. Contradictory findings with regard to structural brain abnormalities have been reported in OCD. Kim *et al* (pp. 330–334) investigate these using a novel voxel-based analysis of magnetic resonance images. Results are consistent with functional studies suggesting orbitofrontal and subcortical hyperfunction. The finding of a marked reduction of cerebellar grey matter requires further study to clarify its possible involvement in the pathophysiology of OCD.