Highlights of this issue

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Autism: possible prenatal risks and the impact of parent–child interaction

Following the emergence of evidence implicating selective serotonin reuptake inhibitor (SSRI) exposure during pregnancy in the development of a range of adverse early life outcomes for offspring, El Marroun *et al* (pp. 95–102) report an association between such exposure and development of autistic traits in a sample of children followed prospectively up to age 6 years. Of note, the authors also found that maternal depressive symptoms without SSRI use were associated with both pervasive developmental and affective problems in the children, although these effects were weaker. Two commentaries on the paper are also published in the *Journal* this month. Jones & McDonald (pp. 103–104) highlight the fact that, until relatively recently, the limited evidence base regarding the safety of SSRI prescription during pregnancy indicated little of concern, but that a number of studies have recently raised the possibility of adverse consequences for offspring exposed to SSRIs prenatally. The proportion of pregnant women taking antidepressants has also been noted to have increased in recent years. The authors call for caution to be exercised when decisions about such prescribing in pregnancy are made and comment that, while women should be reassured that absolute risks do not appear to be large and that causation is not yet clearly established, there is emerging reason for caution and that other non-pharmacological treatments should be seriously considered if treatment for depression in pregnancy is required. In contrast, Petersen *et al* (pp. 105–106) have concerns about the validity of the study findings and argue that the association reported may well be explained by an underlying association between maternal depressive symptoms and childhood features of autism, a possibility not fully discounted by the results presented in the paper. The authors of this commentary caution against accepting the potential implications of such observational findings.

Another paper focused on autism in the *Journal* this month concerns an investigation of the oxytocin functioning of young children with autism spectrum disorder (ASD) and the impact of parent–child contact on such functioning. Comparing 40 preschoolers with high-functioning ASD with 40 matched controls, Feldman *et al* (pp. 107–112) found lower baseline oxytocin levels in the ASD group, levels which normalised during parent–child interactions. The authors comment on the implication that the oxytocin dysfunction identified in children with ASD appeared to be malleable, giving support for the notion that environmental inputs including caregiver interaction can have a positive impact on a biological system potentially important in the development of autism.

Predicting and intervening to improve outcomes in psychosis

In a systematic review and meta-analysis of papers analysing data from 33 independent early psychosis samples, Penttilä *et al* (pp. 88–94) found that duration of untreated psychosis (DUP) correlated with the following: poor general symptomatic outcome, the presence of more severe positive and negative symptoms, reduced likelihood of remission, poor social functioning and poor global outcome. Long DUP was not associated with a number of other outcomes considered (employment, hospital admissions and quality of life). The authors comment that early intervention may have positive effects on long-term outcome but that, in the absence of a study able to randomise individuals to DUP length, conclusions about causation remain difficult to make.

Although individual placement and support (IPS) for those with psychosis has a strong evidence base, translation into clinical practice has proved difficult, in part because of the ambivalence of staff to the likelihood of positive occupational outcomes for this group and the persistence of largely unfounded concerns about the impact of employment-related stress. Intending to address such barriers, Craig *et al* (pp. 145–150) describe a cluster randomised trial of motivational interviewing directed at clinical staff working in early intervention teams offering IPS in London and the Midlands. At 12-month follow-up, employment was higher among patients of the teams that had received the motivational intervention than among those of the control teams. In another psychosis intervention trial, Fleischhacker *et al* (pp. 135–144) report no efficacy disadvantage for once-monthly long-acting injectable aripiprazole for maintenance treatment of schizophrenia when compared with oral aripiprazole.

In addition to the psychosis outcome-focused studies already described, another paper examines psychosis incidence. Lasalvia *et al* (pp. 127–134) report on the results of a first-contact incidence study of psychosis undertaken in a large area of northern Italy. The annual incidence rate per 100 000 was 18.1 for all psychoses, and 14.3 for non-affective psychoses, findings lower than reported in other Western studies. Rates for all psychoses were higher among young people, immigrants and those living in deprived areas but similar incidence rates were found in men and women.

Polygenic gene × environment interaction in depression

Rather than consider the interaction between one candidate gene and an environmental factor, Peyrot *et al* (pp. 113–119) examined the interaction between polygenic risk scores based on genome-wide meta-analysis results and childhood trauma in relation to the presence of major depressive disorder. Evidence of interaction as a departure from both multiplicative and additive effects was found. The authors comment on the need for their findings to be replicated given the history of inconclusive findings in relation to interaction effects in depression to date.