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Highlights of this issue

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Risk factors for psychosis: interaction and evolution

Schizophrenia is considered to have a multifactorial aetiology. Initial interest was in the relative contribution of these individual factors to developing schizophrenia, but more recently there has been a much greater focus on the interactions between these factors, driven by an interest in specific gene–environment relationships. Zammit *et al* (pp. 207–211) report the analysis of a large epidemiological study of schizophrenia, looking at the additional value of examining interactions between established risk factors. They conclude that some people will only develop schizophrenia if more than one risk factor is present; this significant additive interaction was found for most of the observed risk factors. However, despite these statistically significant interactions, interestingly, they do not consider that this offers any significant additional understanding of aetiology, or guidance towards potential targeting of therapies. A related editorial by Kendler & Gardner (pp. 170–171) is invaluable in setting out a 'guide for the perplexed' – the core of the theoretical and practical issues required to understand this complex area. They are also very cautious in their assessment of this approach, and recommend a simple analysis strategy predicated on maximising our ability to predict and explain risk factors, and actually avoiding interactions as a major research focus *per se*. There has been a more modest renewed interest in the dimensional view of psychotic symptoms, and the observation that these symptoms are reported commonly in the population. An editorial by Kelleher and colleagues (pp. 167–169) considers an evolutionary perspective in explaining the relative frequency of symptoms: while there are clear disadvantages in developing a psychotic illness, there may be evolutionary benefits associated with carrying some of the risk-related genes and subclinical symptoms. They advocate this area, of symptoms in the non-clinical population, as deserving more research.

Culture, depression and self-harm

There has been considerable variation in the rates of suicide reported in different ethnic groups. Cooper *et al* (pp. 212–218) conducted a three-city study and found higher rates of self-harm in young Black females in all three sites, while rates for south Asian groups varied between the different sites. They also report that minority ethnic patients were less likely to receive specialist psychiatric assessment and follow-up. They concluded that services need to be better tailored to respond more sensitively to differential local cultural variations in people presenting with self-harm. Cultural competency training is suggested as one option to help in changing skills and attitudes of clinical staff. The raised incidence of depression and self-harm in south Asian women, along with lower levels of antidepressant use, was the rationale for a social intervention trial among south Asian women with depression in the UK. Gater and colleagues (pp. 227–233) report that the social group intervention was acceptable to the women in the study, and was associated with greater improvement in social functioning, although not significantly greater improvement in depression. An accompanying editorial by Bhui (pp. 172–173) emphasises the value of contemporary research focused on understanding cultural influences in the aetiology of mental illness, but suggests that insufficient attention has been paid to cultural adaptations of clinical interventions and service delivery. He argues that such adaptation is not necessarily expensive, and the increased attention to the core elements of the intervention can also benefit the wider population.

Negative symptoms, personality and cortisol

In the absence of any novel therapeutic interventions for the negative symptoms of schizophrenia, Singh and colleagues (pp. 174–179) report that adjunct antidepressant treatment was effective in improving negative symptoms. They performed a meta-analysis of 22 publications and report a significant result for treatment with the antidepressants fluoxetine, trazodone and ritanserin. Combination treatments are likely to cause additional side-effects and there are insufficient data on the levels of this side-effect burden, or on the benefits for specific negative symptoms. Personality disorder is relatively common in the population, but there is no standard measure of severity. Yang *et al* (pp. 193–199) overcame this difficulty by using the extent of comorbidity between different personality disorder types, and clusters, as an index of severity. They report increased levels of dysfunctional behaviour and impairments in social functioning with increased levels of severity. Intriguingly, the suggestion is made that it is difficult to implement targeted service provision for a disorder that has a prevalence of 13%; but by indexing severity, it should be possible to target provision at the much smaller proportion of the people assessed as having a severe disorder. Cortisol measures have been widely used as to demonstrate change associated with depressive illness within the hypothalamic–pituitary–adrenal axis. Vreeburg and colleagues (pp. 180–185) show that the same pattern of elevated awakening cortisol levels is present in non-depressed individuals with a parental history of depression as in patients with depression or anxiety disorder. They suggest that genetic factors could account for their results, and represent a trait factor for vulnerability to developing depression or anxiety disorders.