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Highlights of this issue

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Mental illness: public understanding and creativity

Two contentious beliefs are examined in this issue of the *Journal*: the link between mental illness and increased creativity, and the utility of an improved public understanding of biological mechanisms in reducing the stigma associated with mental illness. The link between creativity and mental illness is often suggested as a rare beneficial aspect of mental disorder, although robust data to support this association have been difficult to find. Kyaga and colleagues (pp. 373–379) use data from a Swedish registry to demonstrate that patients with bipolar disorder were over-represented in creative professions, patients with schizophrenia were more frequently found in artistic occupations and healthy siblings of patients with psychotic illness were overrepresented in creative professions. The authors suggest that the results are relatively robust, on the basis of a much larger sample than in earlier studies. The absence of any observed link between creativity and depressive illness indicates some specificity for psychotic disorders. They raise the question of whether this is a secondary evolutionary advantage, allowing these disorders to persist in society. An accompanying editorial by Jamison (pp. 351–352) clarifies that most people who are creative do not have mental illness and most people with mental illness do not have unusual levels of creativity. She also points out the unintended societal consequences of potential treatments for bipolar disorder. If genetic testing were made available for bipolar disorder, then reducing the risk of bipolar disorder will also carry a risk of losing greater flexibility of cognitive styles, risk-taking and temperament. There is a widespread view that improving public knowledge of the biological basis of mental illness results in decreased stigma and increased tolerance. However, in their review article, Angermeyer *et al* (pp. 367–372) show that biological or genetic causal attributions were not associated with increased tolerance towards mental illness. The authors suggest that in some cases offering an improved biological understanding may actually serve to increase public stigma, and that alternative methods of reducing stigma will require investigation.

Psychosis: prodrome, cannabis and *COMT*

There is an increasing interest in what can be learnt from the prodromal phase of psychotic illness. Chuma & Mahadun (pp. 361–366) review the predictive validity of several studies examining prodromal criteria and find that ultra-high-risk criteria predicted schizophrenia with 81% sensitivity and 67% specificity. They conclude that these criteria are useful in predicting the risk of developing schizophrenia, but that longer follow-up times would be useful in reducing possible false positives, should there be a long prodromal period. Cannabis use has been associated with increased risk of developing psychosis, with at least one study suggesting that variation in the catechol-methyl-transferase gene on chromosome region 22q11 may contribute to increasing the risks of psychosis associated with cannabis use. Zammit and colleagues (pp. 380–385) fail to demonstrate this specific gene–cannabis association, finding an increased association between psychotic experiences and cannabis use – regardless of the genetic variation. They suggest that the link between cannabis and psychosis is important, and caution against viewing any genotype as conferring a lower risk of psychosis. Welch *et al* (pp. 386–390) use structural neuroimaging to demonstrate that cannabis use is associated with decreases in bilateral thalami of people at familial high risk of schizophrenia. The authors draw together other data showing reduced thalamic volume in high-risk populations and during the transition to frank psychosis – suggesting that cannabis use may enhance this vulnerability further and lead to worsening of any initial subtle symptoms.

Serotonin, dopamine and addiction

Lower levels of brain 5-hydroxytryptamine (5-HT) have been associated with impulsive and aggressive traits, which in turn are related to increased levels of stimulant misuse. Cox *et al* (pp. 391–397) used neurochemical imaging to demonstrate that lowering 5-HT levels leads to an increase in cocaine-induced craving and striatal dopaminergic response, a possible mechanism for increased drug-seeking behaviour. In an accompanying editorial, Nutt (pp. 353–354) questions the clinical implications of this finding, commenting on the lack of effect of selective serotonin reuptake inhibitors as anti-addiction agents. He also raises the questions of whether 5-HT may also modulate other neurotransmitters and influence the use of other drugs, and whether one should also examine the role of well-established genetic variation in 5-HT reuptake sites.

Youth suicide prevention, CNVs and ADHD

A multi-level National Youth Suicide Prevention Strategy was put in place following a rise in youth suicide in Australia between 1970 and 1990. Page *et al* (pp. 423–429) show little discernable impact of these prevention activities on suicide rates. However, there was a general decrease in suicide rates and they discuss wider reasons, including an increase in economic prosperity, as well as changes in primary care provision and increased consumption of anti-depressants. Chromosomal copy number variants (CNVs) are associated with several neurodevelopmental disorders, including schizophrenia, autism and, more recently, attention-deficit hyper-activity disorder (ADHD). Langley and colleagues (pp. 398–403) demonstrate that ADHD CNV carriers did not differ from non-carriers in the frequency of any clinical characteristics, except for having higher rates of intellectual disability. The authors conclude that CNVs are not associated with an atypical subgroup of people with ADHD.