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Depression among educated youths regarding personality and social factors

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Abstract
Depression, as a heterogeneous collection of disorders, is likely to include subgroups that are more genetic in origin. In common with other neuropsychiatric disorders such as schizophrenia, Alzheimer's disease and Huntington's disease, earlier age at onset in depression is associated with higher genetic loading and poorer long-term outcome. Adolescents and young adults with depression are also at high risk of developing a bipolar illness. This article reviews depressive illnesses that occur for the first time in adolescence and young adulthood. Case studies are used to discuss atypical presentations and the evolving concept of bipolar-spectrum disorders.

Keywords: Depression, educated youths, social factors

Introduction
Affective illnesses may present at any age, but it is becoming clear that patients who suffer from recurrent and severe forms of mood disorder often experience their first episode of illness early in life. The clinical presentation of depression at this stage of life can be atypical and is often complicated by personality difficulties and substance misuse. A significant proportion of young people presenting with recurrent depression will go on to develop a bipolar disorder, with important implications for future pharmacological treatment choices.

Epidemiology
In the UK, suicide is now the most common cause of death in young men between the ages of 25 and 34 Epidemiological studies suggest that although factors such as poor schooling, poverty and un-employment are important, the strongest risk factors for suicide in this group are a history of mental illness, and a family history of suicide or mental illness (Agerbo *et al*, 2002) [3]. In a psycho-logical autopsy study of completed suicide in young people aged between 15 and 24, found that 19 out of 27 individuals (70%) had suffered from a mental illness and that depression was the most common diagnosis, affecting 15 (56%) of those studied. Eight indivi-duals (30%) had had a personality disorder and nine (33%) had had a comorbid psychiatric disorder. It is notable that very few of these young people were receiving psychiatric care when they died. The Government's recently published National Suicide Prevention Strategy (Department of Health, 2002) acknowledges the importance of improved recognition and treatment of mood disorders in young adults, particularly within the young male population.
Although most epidemiological studies estimate that around 5% of the adult population suffer from depression, there are relatively few studies focusing on populations of adolescents and young adults. Although depressive symptoms appear to be common – a recent Finnish study of young adults identified a 1-month prevalence for major depression of 10% – only a small proportion of this group are likely to present to mental health services (Aalto-Setala *et al*, 2001) [1].
Adolescents with sub-diagnostic levels of depressive symptoms show higher rates of early-adulthood depression, substance misuse and adverse psychological and social functioning (Aalto-Setala *et al*, 2002) [2]. When symptom severity reaches the threshold for diagnosis, there is a likelihood that depression will continue into early adult life.

The Maudsley long-term follow-up study of child and adolescent depression, which followed 149 participants over 20 years, found that 62% experienced a recurrence of major depression. Similarly, rates of suicidal behaviour were high, with 44% attempting suicide at least once. Levels of social dysfunction and service utilisation were much higher than in the general adult population.

The emergence of gender differences during adolescence

That women are twice as likely as men to have depression is a consistent finding in psychiatric epidemiology and is not simply a consequence of females being more likely to report, recall or seek help for depressive symptoms. Before puberty, boys are slightly more likely than girls to be depressed, but between the ages of 11 and 13 this trend is reversed, with girls outnumbering boys by two to one. This predominance of females over males persists for the next 35 to 40 years. Changes in gonadal steroids are only part of the explanation for this gender gap. Hormonal changes in ado-lescence, combined with dramatic changes in social environment and relationships, stimulate the development of greater affiliative needs in females such as a preference for intimacy and emotional responsiveness. One result of this is that adolescent girls can be left more vulnerable to the effects of negative life events, especially ones that have interpersonal consequences (Cyranowski *et al*, 2000) [15].

Progression from early-onset unipolar depression to bipolar disorder

Age at onset of depression and severity of depressive episodes are important factors in determining rates of ultimate progression to bipolar disorder. Pre-pubertal onset of depression is a strong marker for bipolar disorder, with some studies finding that at least one-third of depressed children will develop bipolar disorder in adult life. In 7-year prospective study of 28 out-patient adolescents with depression, detected a rate of bipolar outcome of almost 20%.

Rates of switching polarity are higher for those with more severe episodes of depression. A 15-year follow-up of 74 young adults hospitalised for unipolar depression found that 27% subsequently developed hypomania, with an additional 19% experiencing at least one episode of mania. The presence of psychotic symptoms during the index depressive episode was strongly predictive of bipolar disorder, with psychotic depression eventually becoming bipolar in eight out of ten patients. Unsurprisingly, patients with a positive family history of mania were also at higher risk of an outcome of bipolar disorder.

Aetiology

Depression in young adults occurs as a result of the dynamic interaction of a wide range of risk factors.

Depression in young adults

The role of genetics

Adoption and family studies have established that depression runs in families and that most of this familiality occurs as a result of genetic rather than environmental influences. Unipolar depression, as a heterogeneous disorder, is likely to include subgroups that represent more genetic forms of depressive illness. Recurrent, early-onset depression, defined as two or more episodes before the age of 25, is associated with a strong family history of affective disorder and appears to follow a particularly malignant course, with frequent recurrence, poor response to treatment and high psychiatric and physical comorbidity. Although the heritability estimate of major depression across the life span is between 31% and 42%, recurrent early-onset depression carries an estimated heritability of 70%, a figure which is close to estimates for bipolar disorder.

A recent family study of recurrent, early-onset depression found that over one-third of first-degree relatives and one-fifth of extended relatives had a history of depression. Segregation analysis of these families was consis-ent with a single major locus being responsible for the expression of the disorder (Maher *et al*, 2002). Findings such as these have given added impetus to genetic studies of affective disorders.

Neuroticism

Neuroticism, defined as a general vulnerability to neurotic breakdown under stress, is a heritable personality trait and has been positively associated with depression. In a longitudinal study of 897 young adults followed between the ages of 18 and 21 years, found a strong association between high premorbid neuroticism and the subsequent development of a depressive illness.

However, the relationship between neuroticism and depression is complicated. Genes that pre-dispose to mood disorders overlap with those implicated in neuroticism and individuals with high levels of neuroticism are more likely to experience depression after stressful life events than those with low levels. Furthermore, evidence is emerging for a significant person–environment interaction whereby indivi-duals with high neuroticism scores select themselves into high-risk environments and as a result become more likely to experience stressful life events.

For young adults with high levels of neuroticism, a vicious circle can be hypothesised in which they are more likely to place themselves in high-risk situations and, as a result of a high genetic loading for depression, are less able to withstand the adverse effects of stressful life events when they occur.

Early adversity

Childhood physical, emotional and sexual abuse are established as important risk factors for the development of a range of psychiatric disorders in adult life and are increasingly recognised as important in early-adulthood psychopathology. Traumatic experiences can interfere with normal emotional and psychological development, with the result that abused or neglected individuals often struggle to negotiate the maturational tasks of adolescence and early adulthood (Brown *et al*, 1999) [14].

The observation that not all abused individuals develop significant psychopathological disorders in later life suggests that our susceptibility to stress is heavily dependent on our genetic make-up. This notion of genetic resilience in some individuals is supported by recent work on depression in adolescent girls, which confirms that genetic factors play an important role in determining their level of susceptibility to environmental stress.

Life events

Although it is established that negative life events can precipitate depression, the association is a complex one and probably operates in both direc-tions. People with depression are more likely to generate stressful events, and individuals

with a higher genetic loading for affective disorder are more likely to experience depression after a stressful event than those with low genetic loading.

In recurrent depressive disorder, the association between life events and depression is strongest for early episodes and becomes weaker as the number of episodes increases. Recurrent depressive episodes tend to become more autonomous and are progressively less linked to environmental adversity, a phenomenon which has been called 'kindling'. Kindling tends to be most marked in individuals at low genetic risk of depression; those at high genetic risk tend to exhibit 'prekindling'. Prekindled individuals appear to become depressed after only minimal environmental provocation. One important implication of this is the possibility that young people with a strong family history of affective disorder are constitutionally vulnerable to the effects of even minor psychosocial stressors.

Substance misuse

Drug and alcohol use in adolescence are important risk factors for the development of affective disorders in early adulthood and are likely to complicate the long-term course of depression. In a 5-year longi-tudinal study of 155 adolescent females, found that 19% developed a substance use disorder and that substance use was a marker for the eventual occurrence of depression. Conversely, when followed 274 formerly depressed adolescents to age 24, two-thirds had experienced another depressive episode and, from the remaining third who had not, 77% were found to have a substance misuse disorder. This suggests that an episode of depression in adolescence, or a diagnosis of substance misuse, represents an opportunity for early intervention to prevent recurrence of both disorders in later life. Alcohol As in older adults, there is significant comorbidity between alcohol misuse and depression in young adults. That alcohol use at a young age leads to a higher risk of depression in young adulthood is supported by the findings of the Children in the Community Study mentioned above. Earlier alcohol use significantly predicted not only depression but also any substance use disorder and alcohol dependence by age 27.

Pathophysiology: the neurogenic theory of depression

In recent years the monoamine theory of depression has given way to a molecular and cellular theory that suggests that antidepressants work by pro-ducing sustained activation of second messenger systems such as cyclic adenosine monophosphate (cAMP). This in turn leads to increases in brain levels of neurotrophic factors such as brain-derived neurotrophic factor (BDNF) that can reverse the detrimental effects of stress in brain areas such as the cerebral cortex and hippocampus (Reid & Stewart, 2001). The neurogenic theory of depression posits a central role for dysregulation of the hypothalamic–pituitary–adrenal (HPA) axis and adrenal steroid-induced changes in hippocampal function in the pathophysiology of depression. It is supported not only by new insights into how antidepressants work, but also by a growing body of evidence from basic science and clinical studies (for reviews see.

High ratios of cortisol to dehydroepiandrosterone (DHEA) are consistent findings in affective disorders and contribute to hippocampal atrophy. Excess cortisol impairs neurogenesis in the hippocampus, whereas DHEA may offer some protection (Young *et al*, 2002) [3]. Prospective studies of adolescents with no past history of depression have found that those who subsequently become depressed have higher ratios of cortisol to DHEA than controls. It is suggested that this can occur by two separate routes: genetic predisposition (a high familial loading for affective disorder); and early adverse experiences such as childhood sexual abuse.

The finding of neurocognitive deficits in young adults even during the early stages of their illness has important implications for the way in which psychoeducational and cognitive– behavioural interventions are delivered. Information and therapy should be provided in a way that takes account of cognitive impairments in areas such as recollection memory and verbal learning.

Classification

The unipolar/bipolar dichotomy

Kraepelin, writing in the 1920s, had a broad view of manic–depressive illness that encompassed not only less-severe, attenuated forms but also much of the domain of major depressive disorders. Several important studies in the second half of the 20th century divided this unitary model of affective disorders into unipolar and bipolar disorders. In the 1950s, Leonard observed from his cohort of patients with recurrent depression that those who also had a history of mania tended to report more.

The overlap with personality disorder

There is a great deal of debate about whether the primary diagnosis in a substantial proportion of cases of borderline personality disorder is more usefully one of a primary mood disorder. In young adults, there is considerable overlap between the symptoms of mood disorder and cluster B person-ality disorder, particularly with regard to mood lability, impulsivity and self-harm. An 11-year follow-up of early-onset depression demonstrated that those who went on to develop bipolar disorder had many of the features of borderline personality disorder when first assessed (Akiskal *et al*, 1995) [4]. Given the damaging long-term implications of a diagnosis of personality disorder early in life, it seems good practice to exclude a primary mood disorder in young adults presenting with behaviours that might initially be suggestive of personality pathology. This kind of approach is supported by a recent international consensus that when patients satisfy the diagnostic criteria for both bipolar disorder and borderline personality disorder, a bipolar diagnosis is preferred (Akiskal *et al*, 2000) [6].

Conclusions

Depression that has its onset in adolescence or young adulthood represents a severe form of affective disorder associated with a range of poor long-term out-comes. It often arises in families where multiple first-and second-degree relatives have a mood disorder and is frequently complicated by substance misuse.

Although we have highlighted the high rate of progression to bipolar disorder in this group, and ways in which subtle indicators of bipolarity can be used to guide treatment choices, further studies of the validity of the proposed diagnostic criteria for bipolar-spectrum disorder are clearly required.

Genetic, neuroendocrine and brain-imaging studies are unravelling the complex interaction of constitutional and environmental risk factors in early-onset depression, but there remains a need for long-term prospective studies of young people in the early stages of illness. These studies will

have to allow the integration of findings from a diverse range of disciplines, from basic and clinical neurosciences to social science and epidemiology.

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