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Depression among adolescents and young adults – A Review

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ABSTRACT

Depression among adolescents and young adults is a significant public health concern with far-reaching implications for individuals, families, and societies. The frequency and demographic trends of depression in adolescents and young adults, highlighting the complex interactions between biological, psychological, and social elements that influence the illness's development. Since depression is a diverse range of illnesses, it is likely to have subgroups with a stronger hereditary component. Similar to other neuropsychiatric conditions including Huntington's disease, schizophrenia, and Alzheimer's disease, depression is linked to a worse prognosis in the long run and a greater genetic loading at the early age of start. It highlights the importance of addressing comorbidities such as substance abuse, self-harm, and suicidality in treatment planning. Depressed adolescents and young adults are also more likely to get bipolar disorder. This article discusses depressions that initially manifest in youth and early adulthood. The paper stresses the need of tackling depression in adolescents and young adults through a holistic and multidisciplinary strategy. In order to create efficient preventative and intervention plans that support mental health and wellbeing in this susceptible group, it is necessary for researchers, and other community members to collaborate more closely.

***Keywords:** Depression, Young adults, Adolescence, Bipolar disorder,*

INTRODUCTION

As a diverse set of illnesses, depression is likely to have subgroups with a stronger hereditary component (Cai et al., 2020). A multitude of risk variables combine dynamically to cause depression in young adulthood. At this point in life, personality issues and drug abuse frequently exacerbate the clinical presentation of depression, which can be unusual. A substantial percentage of young individuals who initially appear with recurrent depression will go on to develop bipolar illness, which has substantial consequences for the selection of future pharmaceutical treatments (Goodwin et al., 2007). Due to young adult melancholy, suicide is currently the most prevalent cause of death for young men in India and other nations between the ages of 25 and 34 (Bachmann, 2018). The greatest risk factors for suicide in this demographic, according to epidemiological research, are a history of mental illness and a familial history of suicide or mental illness, even if variables like low education, poverty, and unemployment are also significant (Agerbo et al, 2002). Houston et al. (2001) discovered that depression was the most prevalent diagnosis, affecting 15 (56%) of the 27 people who had been examined, and that 19 out of 27 people (70%) had experienced a mental disorder. Nine people (33%) had a co-occurring mental condition and eight people (30%) had a personality problem. Remarkably, when these young people passed away, relatively few of them were receiving mental health treatment. Relatively few epidemiological research has focused on populations of adolescents and young adults, despite the fact that the majority of studies estimate that 5% of people suffer from depression (Angold et al., 2001; Saluja, et al., 2004). Just a tiny percentage of this population are likely to come to mental health services, despite the fact that depressive symptoms appear to be frequent (a recent Finnish research of young people found a 1-month prevalence for serious depression of 10% (AaltoSetala et al., 2001).

The appearance of gender disparities in teenage years

A persistent finding in psychiatric epidemiology is that women are twice as likely as men to experience depression. This result goes beyond the simple fact that women are more likely to report, recollect, or seek treatment for depressed symptoms (Kessler, 2003; Sloan et al., 2006). Adolescent hormonal changes together with significant changes in relationships and the social environment encourage the development of higher affiliative demands in females, including a need for closeness and emotional reactivity (Spear, 2000; Walke et al., 2013; Guyer et al., 2016). Adolescent females may be more susceptible as a result to the negative impacts of life experiences,

particularly those that have interpersonal repercussions (Cyranowski et al, 2000; Green et al., 2005; Cash, 2012).

Early-onset unipolar depression developing into bipolar disorder

The age at which depression first appears and the intensity of depressive episodes have a significant role in predicting the rates at which bipolar illness eventually progresses (Kessler, et al., 2003; Goodwin et al., 2007; Treuer et al., 2010). According to certain research, at least one-third of youngsters who experience depression will go on to acquire bipolar illness as adults (Kessler, et al., 2003; Goodwin et al., 2007). Pre-pubertal start of depression is a significant indicator of bipolar disease (Geller et al, 2001). Individuals experiencing more severe periods of depression have increased rates of polarity flipping (Riemann et al., 2020). In a 15-year follow-up, 27% of the 74 young people hospitalized for unipolar depression went on to develop hypomania, and 19% had at least one manic episode (Goldberg et al., 2001). Eight out of ten patients had psychotic depression that subsequently turned into bipolar disorder, with the prevalence of psychotic symptoms during the index depressive episode serving as a significant predictor of bipolar disorder (Aminoff et al., 2022; Uher et al., 2024). It should come as no surprise that individuals with a positive family history of mania were also more likely to experience bipolar disorder symptoms (Akiskal et al., 2003; Johnson, 2005; Alloy et al., 2012; Zimmerman, et al., 2014).

The role of genetics

Depression runs in families, and adoption and family studies have shown that most of this familiarity is due to genetics rather than external factors (Sullivan et al., 2000; Smoller et al., 2003; Kendler et al., 2007). A strong family history of affective disorder is linked to recurrent, early-onset depression, which is defined as two or more episodes before the age of 25 (Zisook et al., 2004; Holmans et al., 2007). It also appears to have a particularly malignant course, with frequent recurrence, a poor response to treatment, and high psychiatric and physical comorbidity (Zubenko et al., 2001). While severe depression has an estimated heredity of 31% to 42% throughout a person's life, recurring early-onset depression has an estimated heritability of 70%, which is comparable to estimates for bipolar illness (Goodwin et al., 2007).

Early adversity

Physical, emotional, and sexual abuse throughout childhood have been shown to be significant risk factors for the emergence of a variety of mental illnesses in adulthood, and they are also becoming more well acknowledged for their role in early-adult psychopathology (Fergusson et al., 2008; Carr et al., 2013) . According to Brown et al. (1999), abuse victims and those who have been neglected or mistreated may find it difficult to deal with the challenges of adolescence and the early stages of life as a result of traumatic events interfering with normal emotional and psychological development. Our genetic makeup has a major role in determining our vulnerability to stress, as evidenced by the finding that not all mistreated persons go on to acquire substantial psychopathological illnesses in later life (McCrory et al., 2012; Doom et al., 2013). According to current research on depression in adolescent girls, genetic variables are essential in determining an individual's vulnerability to environmental stress (Silberg et al., 2001). This suggests that some people may be genetically resilient.

Life events

Although the link between unfavorable life circumstances and depression is well-established, it is complicated and likely works both ways (Lacovides et al., 2003). Individuals with a greater genetic loading for affective disease are more likely to develop depression following a stressful event compared to those with a lower genetic loading. People with depression are also more inclined to create stressful circumstances (Kendler & Karkowski-Shuman, 1997; Silberg et al, 2001; Lacovides et al., 2003).

Alcohol consumption

Young persons' depression and alcohol abuse are significantly comorbid, just like it is in older adults (Fergusson et al, 2002; Deas et al., 2006; Wu et al., 2014). The results of the previously described Children in the Community Study support the idea that early alcohol usage increases the likelihood of depression in early adulthood (Magee et al., 2021). Previous alcohol consumption was a strong predictor of alcohol dependence by age 27, any drug use disorder, and depression (Brook et al, 2002).

PATHOPHYSIOLOGY: THE DEPRESSION-RELATED NEUROGENIC HYPOTHESIS

According to the neurogenic hypothesis of depression, alterations in hippocampal function brought on by adrenal steroids and dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis play a

major role in the pathophysiology of depression (Duman et al, 1997; Drevets, 2000; Juruena et al., 2004; DeMorrow, 2018). It is corroborated not just by recent discoveries on the mechanisms of action of antidepressants, but also by an increasing amount of data from clinical trials and fundamental science (reviews may be found in D'Sa & Duman, 2002; Kempermann, 2002). Affective illnesses are frequently associated with high cortisol to dehydroepiandrosterone (DHEA) ratios, which also lead to hippocampus shrinkage (Jin et al., 2016). Young et al. (2002) found that while DHEA may provide some protection, excessive cortisol inhibits neurogenesis in the hippocampal regions. Cortisol to DHEA ratios are greater in depressed individuals than in control groups, according to prospective studies of teenagers without a history of depression (Goodyer et al., 2000; Jin et al., 2016). Genetic predisposition (a high familial loading for affective disease) and early unpleasant experiences, such as childhood sexual abuse, are proposed as the two distinct pathways by which this might happen (Charney et al., 2004; Zimmermann et al., 2007). In order to solve this issue, one research is really interesting since it looks at the role and hippocampal volume in a case-control study of depressed young people MacQueen et al. (2003) where 20 patients in their first episode of depression who had never received treatment were compared to 17 patients who had a history of many bouts of depression and matched controls. Hippocampal volume was quantified by magnetic resonance imaging, and hippocampal function was evaluated using neuropsychological tests of verbal memory and recall memory (Den Heijer et al., 2010; Bonner-Jackson et al., 2015). Importantly, neuropsychological testing revealed decreased hippocampus function in both the first- and multiple-episode depressive groups (Frey et al., 2007; Gałecki et al., 2015). Only the group with several prior incidents, meanwhile, showed signs of a diminished hippocampal volume. This shows that decreased hippocampal volume results from recurrent episodes of disease rather than being a cause of the illness itself (Henje Blom et al., 2015).

The discovery that young adults have neurocognitive impairments even in the early phases of their disease has significant ramifications for the delivery of cognitive-behavioral and psychoeducational therapies. Therapy and information should be given with consideration for cognitive deficits in verbal learning and remembrance memory.

Depression-related bipolar illnesses in young adulthood

There is a strong case for carefully considering bipolar illness in young adults presenting with recurrent or chronic depressive episodes because bipolar disorders typically present initially in a depressive phase, most people with bipolar illness experience their first episode in adolescence, and the rate of progression from adolescent depression to bipolar disorder is about 20% (Rao et al., 1995).

The connection to personality disorders

There is much discussion on whether a main mood disorder would be a more appropriate diagnosis in a significant number of patients of borderline personality disorder (Silk et al., 2010; Asherson et al., 2014). In young adults, there is a significant overlap between cluster B personality disorder and mood disorder characteristics, especially in relation to impulsivity, self-harm, and mood instability (Chanen et al., 2016). An 11-year follow-up study on early-onset depression revealed that many of the characteristics of borderline personality disorder were present in individuals who later developed bipolar disorder (Akiskal et al, 1995). When young adults arrive with actions that may first reflect personality pathology, it is best to rule out a main mood illness due to the detrimental long-term effects of a diagnosis of personality disorder at an early age. This sort of approach is reinforced by a recent global agreement that a bipolar diagnosis is preferred when individuals meet the diagnostic criteria for both borderline personality disorder and bipolar disorder (Akiskal et al, 2000).

CONCLUSION

Adolescent or young adult depression is a severe kind of affective illness linked to a number of unfavorable long-term consequences. It commonly develops in families with several first- and second-degree relatives who suffer from mood disorders, and drug abuse usually makes it worse. Long-term prospective studies of young individuals in the early stages of the disease are still necessary, even as genetic, neuroendocrine, and brain-imaging research help to clarify the intricate interplay between constitutional and environmental risk factors in early-onset of depression. These investigations must enable the integration of results from a wide variety of fields, including social science, epidemiology, and fundamental and clinical neurosciences.

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