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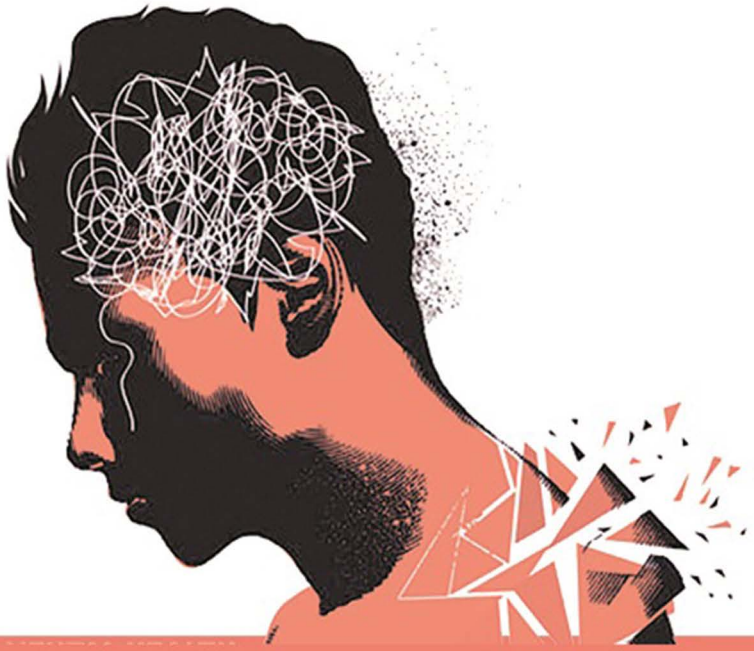
Handbook of Clinical Depression



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CHAPTER

Etiology of Depression

Nikhil Gautam

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INTRODUCTION

The etiology of depression is considered to be multifactorial and is not completely understood. It is not known exactly what causes depression, however, what is understood is that it results from a complex interaction of social, psychological, and biological factors. The purpose of this chapter is to review what is known or suspected about the causes of depression. Depressive symptoms such as sad mood, pessimism, and lethargy, are considered normal reactions to the struggles of everyday life. However, for some individuals, the intensity and persistence of depressive symptoms are not typical and can lead to illness. This chapter discusses some of the characteristics of individuals that may make them vulnerable, as well as the features of environments that are likely to provoke depression. The chapter also emphasizes the interplay between persons and environments—the ways in which, for example, stressful life events may provoke depression.

GENETIC FACTORS

Mood Disorders tend to run in families, and this has been proven by twin Studies. The twin studies data

provide compelling evidence that genes explain only 50–70% of the etiology of mood disorders. The environment or other nonheritable factors must explain the remainder. Family data indicates that if one parent has a mood disorder, a child will have a risk of between 10 and 25 percent for mood disorder. If both parents are affected, then this risk roughly doubles. It is to be noted that it is the susceptibility to disease that is actually inherited and the not the disease itself. Gene-mapping studies of unipolar depression have also found very strong evidence of linkage to the locus for cyclic AMP response element-binding protein (CREB1) on chromosome 2.

BIOLOGICAL FACTORS

Biogenic Amines

There are three major amines that have played a role in causation of depression. The lack of monoamines namely serotonin, dopamine and noradrenaline availability at receptor sites in the brain can lead to depression. The fact that certain drugs and diseases that deplete monoamines (e.g., reserpine and Parkinson disease) cause depressive symptoms and antidepressants that cure said symptoms act on the monoamines (particularly catecholamines as noted above) led to the theory that depression is the result of deficiency or dysregulation in brain monoamines. Patients with suicidal impulses have shown low cerebrospinal fluid (CSF) concentrations of serotonin metabolites. Although this theory has become prevalent, several lines of evidence suggested it as an inadequate explanation for the etiology of depression.

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Most notably, antidepressants take weeks to work, even though their presumed action of increasing monoamine levels happens quickly, often within hours after administration.

A number of studies have investigated the role of genetics in the serotonin-related genes in the etiology of depression. Research in individuals with changes in alleles of serotonin transporter (5-HTTLPR) gene experienced more depressive symptoms and higher rates of depressive disorder in response to stressful life events.

Neuroendocrine Regulation

Recent studies in depressed humans indicate that a history of early trauma is associated with increased hypothalamic pituitary adrenal (HPA) axis activity accompanied by structural changes (i.e., atrophy or decreased volume) in the cerebral cortex. Elevated HPA activity is a hallmark of the mammalian stress response and shows the role of chronic stress in biology of depression. There is also a correlation between the hypersecretion of cortisol and depression. Hypercortisolemia in depression suggests one or more of the following central disturbances: decreased inhibitory serotonin tone; increased drive from norepinephrine, acetylcholine, or corticotropin-releasing hormone; or decreased feedback inhibition from the hippocampus. Hypothyroidism is one of the most common disorders found in depression and should always be checked for. Approximately, 5–10% of patients evaluated for depression have previously undetected thyroid dysfunction, which is reflected by an elevated basal thyroid-stimulating hormone (TSH) level. Such

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abnormalities are often associated with elevated anti-thyroid antibody levels and, unless corrected with hormone replacement therapy, can lead to poor response to anti-depressive treatment.

Immune System Processes

Depressive disorders are associated with immunological abnormalities such as decreased lymphocyte proliferation in response to mitogens and other forms of impaired cellular immunity. There appears to be a link with clinical severity, hypercortisolism, and immune dysfunction. A possibility exists that chronic stressors might prime the immune system to make a heightened response to stress. Alternatively, chronic stress may interfere with the capacity of the immune system to return to baseline even after termination of a stressor, perhaps due to dysregulation of the HPA response and the production of glucocorticoids in response to stress. The inflammatory response may also contribute to symptoms of depression like disruptions in appetite, sleep, and social activity.

PSYCHOSOCIAL FACTORS

Adverse life events can trigger depression and often precede the development of symptoms. Life events that lead to feelings of entrapment and humiliation may be particularly relevant. One theory proposed to explain this observation is that the exposure to stress initially results in long-lasting changes in the brain's biology. These long-lasting changes may alter the functioning of various neurotransmitter and intraneuronal systems, changes that may include the loss of neurons and an

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excessive reduction in synapses. As a result, a person has a high risk of undergoing depression, even without an external stressor. The environmental stressor that is most often associated with the onset of depression is the loss of a spouse. Another risk factor is unemployment; persons out of work are three times more likely to report symptoms of an episode of major depression than those who are employed. Guilt may also play a role.

PSYCHOLOGICAL FACTORS

Cognitive Theory of Depression

According to cognitive theory people with depression tend to form have characteristic ways of interpreting events and circumstances that are excessively pessimistic, with perceptions of helplessness and hopelessness about changing or improving their situations. This way of thinking includes developing negative views about self, environment and future i.e., a cognitive triad of depression that consists of (1) views about the self —a negative perception of oneself, (2) about the environment—a tendency to experience the world as hostile and demanding, and (3) about the future—the expectation of failure and suffering.

Learned Helplessness

The learned helplessness theory of depression connects depressive phenomena to the experience of uncontrollable events in life. For example, when dogs in a laboratory were exposed to electrical shocks in a cage from which they could not escape, they showed behaviors that differentiated them from dogs that had not been exposed to such uncontrollable events. The

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dogs that were exposed to the shocks when placed in a new environment without the cage would not escape to stop the flow of electric shock even when put in a new situation without the cage. They remained passive and did not move. According to the learned helplessness theory, the shocked dogs learned that outcomes were independent of responses, so they had both decreased motivation (i.e., they would not attempt to escape the shock) and decreased emotional response (indicating decreased reactivity to the shock). This theory, as applied to human depression, notes that this phenomenon of helplessness in a depressed individual is thought to produce a loss of self-esteem after adverse external events.

ENDO-REACTIVE

Depression is diagnosed, respectively, when sadness is overly intense and continues beyond the expected impact of a stressful life event. Depressive disorders are conceptualized as endo-reactive i.e., once released due to external factors, they tend to persist autonomously in response to these factors. The term reactive depression which is known as adjustment disorder in current classification terminology signifies that the symptoms can be reversed with the removal from stressful situation which might be the cause.

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Handbook of Clinical Depression

Salient Features

- Easy-to-understand text
- Provides a simple and adequate understanding of clinical and other aspects of Depression
- Treatment strategies are explained in a simple manner
- Helps bridge gap in learning for all medical practitioners
- Helps all medical practitioners in understanding and approaching a person presenting with clinical depression
- Suitable for reading for all medical practitioners

Nikhil Gurjar MBBS MD (Psychiatry) is a Consulting Psychiatrist from Mumbai. He is currently practicing at Dr DY Patil Medical College, Navi Mumbai, Maharashtra, India in the department of Psychiatry. He is passionate about teaching and is currently involved in teaching medical undergraduate students and postgraduate students of psychiatry as well as allied healthcare branches including students of Physiotherapy, Occupational Therapy and Nursing. His areas of interest include mood disorders, anxiety disorders, and substance use disorders. He is also interested in the field of biological psychiatry and is currently doing research in the field. He has also won an award for best original research paper presentation in the field of biological psychiatry. He actively attempts to spread awareness about psychiatric disorders and combating stigma associated with mental health difficulties in the medical fraternity.

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