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Depression: New Horizons

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Aetiology of depression is not yet well understood. Depression and co-morbid anxiety are often triggered by stressful life events. A complex interaction between innumerable variables that include genetics, cognition, personality, environment and gender, eventually correlate with predisposition and surfacing of clinical depression. Despite elaborate studies and deliberations on the genetic and mono-amine hypothesis of depression, several issues remain unanswered.

A news item in the Times of India, Mumbai edition dated 8th September 2007, cited Dr. Somnath Chatterjee from an article in The *Lancet* of September 07 – 14, 2007, where the WHO team noted depression to be more harmful than chronic physical diseases like angina, arthritis, asthma and diabetes.[1] Medical practitioners appreciate that depression may cause, contribute, contaminate, complicate and at the very best, co-exist with a physical illness, Prince et al[2] state that 'about 14% of the global burden of disease has been attributed to neuropsychiatric disorders, 'mostly due to the chronically disabling nature of depression and other common mental disorders, alcohol-use and substance-use disorders and psychoses'. The burden of mental disorders is likely to have been underestimated because of inadequate appreciation of the connectedness between depression and other health conditions. [2]

Ever since the serendipitous discovery of tricyclic antidepressants, followed by selective serotonin reuptake inhibitors, medical fraternity and those suffering from the illness expected significant, almost magical symptom relief with the use of antidepressant drugs. Unfortunately, several adverse events that include treatment failures and treatment emergent adverse events necessitate new research to better understand and manage the disorder.

The emerging data now emphasises that not only the intensity but also subjective interpretation, learning and effectiveness of strategies to cope with stress, play causal roles in depression.[3]

Trend setting research by Sheline in 2003[4] has triggered extensive and elaborate studies on the neurodegenerative effects of stress induced elevated levels of cortisol in depression. Symptoms of cognitive deficits in depressive illness like indecisiveness, impairments of executive functions[5] and slow mentation are now re-interpreted to facilitate reversal of this degenerative process with drugs that promote neuroplasticity. White matter changes in the brains of depressed patients have been identified.[6] This research highlights the neurotoxic effects of high levels of cortisol in a person suffering from clinical depression.[7] New research highlights the neurotoxic effects of high levels of cortisol in a person suffering from clinical depression.[8]

Significance of negative life events[9] and stress induced rise in cortisol levels and drug treatments to heal, prevent and hopefully, reverse the neurotoxic effects of high levels of cortisol on the amygdala and hippocampus[10,11] and strategies to promote brain reorganization,[12] have been the aim of some of the recent research on understanding depression.
Measures to treat depression with medicines that promote neuroplasticity is the emerging trend of this decade.

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