THE CONCEPT OF SYNAPTIC PLASTICITY IN MAJOR DEPRESSION

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Psikiyatride Yeni Ufuklar

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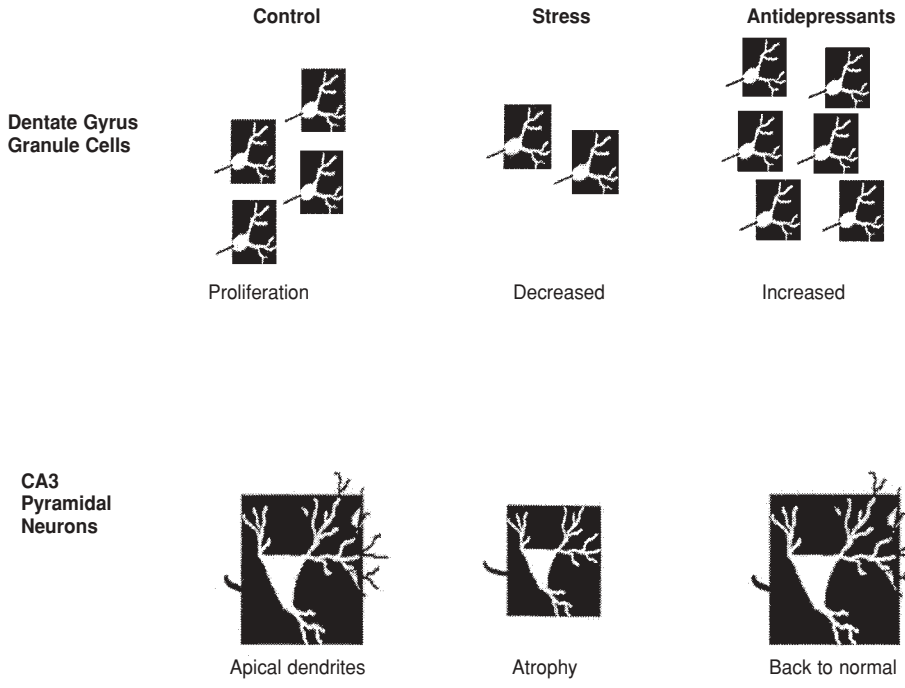
The word neuroplasticity denotes the remarkable capacity of brain to adapt continually to the demands placed on it by experience. Neuroplastic changes are not restricted to a certain stage of development but occur throughout the life. One major neurotrophic factor, brain-derived neurotrophic factor (BDNF) has been demonstrated to play a critical role in long-term potentiation, a cellular model of learning and memory, demonstrating that this neurotrophic factor can influence plasticity. BDNF is also not only needed for the survival and guidance of neurons during development but also required for the survival and function of neurons during adulthood (Duman et al. 2000, McAllister et al. 1999, Thonen et al. 1995).

Duman and colleagues (2000) propose a hypothesis that stress-related affective illness results in part from a loss of neuronal plasticity. Support for this hypothesis is provided by the studies demonstrates that structural alterations also occur in response to stress and in patients with mood disorder (Gönül 2001). It is possible that the structural alteration might be result of atrophy and death of vulnerable neurons and glial cells in some brain areas, i.e. hippocampus and subgenuel prefrontal cortex (Ongur et al. 1998). The atrophy and loss of neurons or glial cells in the hippocampus and cerebral cortex could be resulted from stress-induced loss of neurtrophic factors or other factors that compromise neuronal function and activity (e.g., hyperactivation of hypothalamic-pituitary-adrenal (HPA) axis, glutamatergic excitoxicity, hypoglycemia, hypoxia-ischemia and other insults as a result of genetic background).

In fact, it was showed that stress decreases the neurogenesis of dentate granule neurons and BDNF in hippocampus of adult animals (McAlister et al. 1999, Thoenen 1995, Gould et al. 1997). Pre-clinical studies demonstrate that antidepressant treatments, including selective norepinephrine and serotonin reupta-

Figure 1: Adapted from Duman et al 2000



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ke inhibitors and electroconvulsive seizure, increase the expression of BDNF (See figure) (Nibuye et al. 1995, 1996). When BDNF is administrated to stressed animals, it produces an antidepressant like effect by antagonizing learned-helplessness (Siuciak et al. 1997).

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