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## Melanocyte growth factor deprivation is intertwined with oxidative stress and autoimmune theories in the etiology of vitiligo: A basis for treating vitiligo

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Vitiligo is an acquired depigmentary disorder characterized by the patchy depigmentation of skin that may become progressive over time. The etiology of the disease is far from clear. Recent experimental and clinical related evidence suggests that the vitiligo melanocytes are more prone to oxidative stress than the normal melanocytes. The sensitivity of melanocytes to oxidative stress is interlinked with melanocyte growth factor deprivation theory as proposed by us and of autoimmunity. In the present paper, I will discuss how melanocyte growth factor deprivation theory as cause of vitiligo is intertwined with the other prevailing theories of oxidative stress and auto immunity. Based on the growth factor deprivation theory as one of possible causes for loss of melanocytes in vitiligo, experiments were done to develop a therapeutic agent to treat vitligo. The active basic fibroblast growth factor related peptides (bFGFRP) act as mitogen on melanocytes in culture or in mixed cultures of melanocytes and keratinocytes obtained from untreated vitiligo patients. bFGFRP increases repigmentation of depigmented patches in experimental animal models compared to corresponding controls. bFGFRP was considered as potential repigmenting agent for vitiligo macules. Various phases of clinical trials were conducted in India to study the safety and efficacy of the bFGFRP in patients with vitiligo. Based on the results of these trials, the deca peptide (bFGFRP) was listed as a drug to treat vitiligo in 2001 and sold in India, through online by trade names Melgain and Melbild. The protocols on using bFGFRP to treat vitiligo will be described in detail not only as a single agent for non segmental and segmental vitiligo, but also in combination with PUVA/PUVASOL, vitiligo resistant to further treatment with PUVA, NBUVB or with steroids or with surgical procedures.

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