

Clinical Images

Melanocyte Growth Factor Deprivation is Intertwined with Oxidative Stress and Autoimmune Theories in The Etiology of Vitiligo. A Basis for Treating Vitiligo.

Ramaiah Abburi¹

¹ Samastha Laboratories Hyderabad, India.

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Double Blind Randomized Phase IV Clinical Trial on bFGFRP in Vitiligo

All the above described clinical trials on bFGFRP were open ended. In view of the fact that western countries require only double blind clinical trials on any prospective drug for it to be approved as a drug, a multi-center double blind clinical trial was done on bFGFRP in combination with NBUVB or without. Two macules in non-sun exposed areas were selected on each patient with stable non-segmental vitiligo. The duration of treatment of volunteers was for 3 months starting with or without the topical application of bFGFRP followed by NBUVB on 30 patients. The results demonstrated that bFGFRP acted synergistically with NBUVB in repigmentation of the macules. More repigmentation occurred with NBUVB + bFGFRP at all-time points of evaluation (1, 2, and 3 months) than with NBUVB alone. NBUVB+ bFGFRP was superior to NBUVB alone in repigmenting vitiligo macules (23). Thus, it is clearly demonstrated that bFGFRP can be successfully used by itself or with synergistic effect in combination with many other modalities of treatment to treat vitiligo.

Himalayan Institute of Medical Sciences Jolly Grant with deca peptide lotion alone, PUVASOL with and without deca peptide lotion and with oral mini pulse therapy with and without deca peptide lotion and Prof AS Kumar at Owaisi hospital Hyderabad who supervised the clinical trial with PUV-A+deca peptide lotion on vitiligo patches resistant to PUV-A alone and above all to all the volunteers who participated in the various clinical trials described herein.

Table-1: Growth Characteristics of Normal and Vitiligo Melanocytes.

	Normal	Uninvolved from Vitiligo	Perilesional from Vitiligo	Uninvolved from Repigmenting	Perilesional from Repigmenting
Initial seeding capacity	124#12.38	53.8#10.1	27.6#3.92	168.9#12.4	134#1.6
Lag in growth (d)	0	6-11 (d)	no growth	0	0
Doubling time	8.1#1.2	8.5#0.89	No growth	4.6#0.47	4.25#0.25
Number of passages	Four	No passageability	No growth	>9	>9

Major Conclusions

Growth factor deprivation theory as a possible cause of vitiligo (3) is in the light of the present literature mentioned above intimately intertwined with other prevailing theories of oxidative stress and autoimmune theories (5). These theories are not mutually exclusive but interrelated and all together contribute to the loss of melanocytes resulting in vitiligo.

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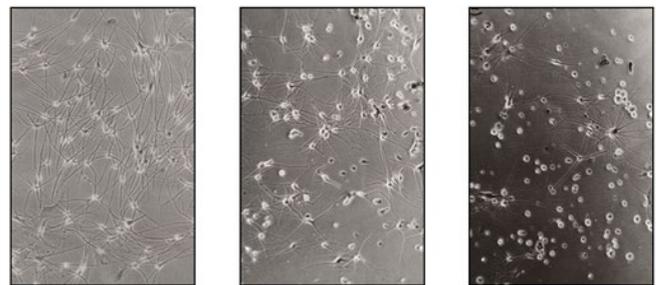


Figure 1: The Melanocytes from Normal, the perilesional & far away from the vitiligo macule.

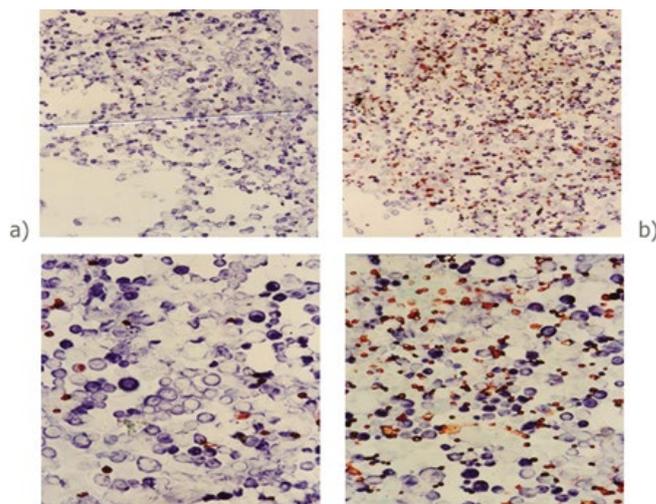


Figure 2: bFGF increased the proliferation of melanocytes in mixed cultures of keratinocytes and melanocytes from vitiligo patients.

*Corresponding author: Ramaiah Abburi, Samastha Laboratories Hyderabad, India, Tel: +91-9012952001; Fax: +91-9012952001; E-mail: ramaiah_abburi@yahoo.com

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a. Animal 5 before treatment and

Figure 3: Guinea pig ears before and after depigmentation.

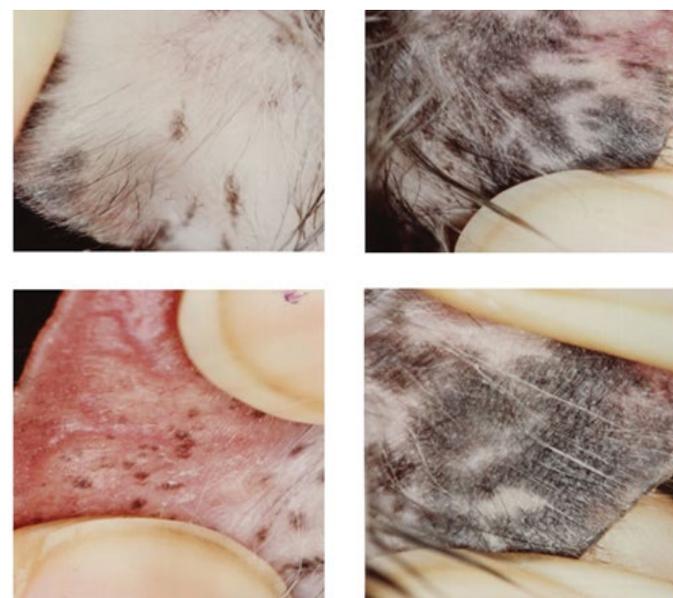
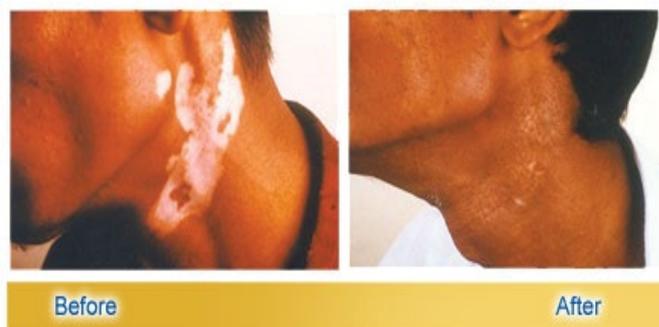
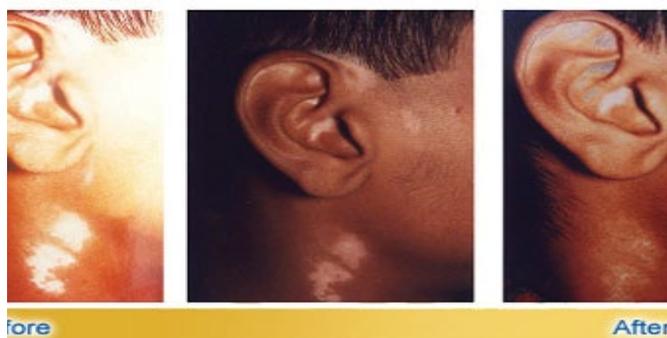


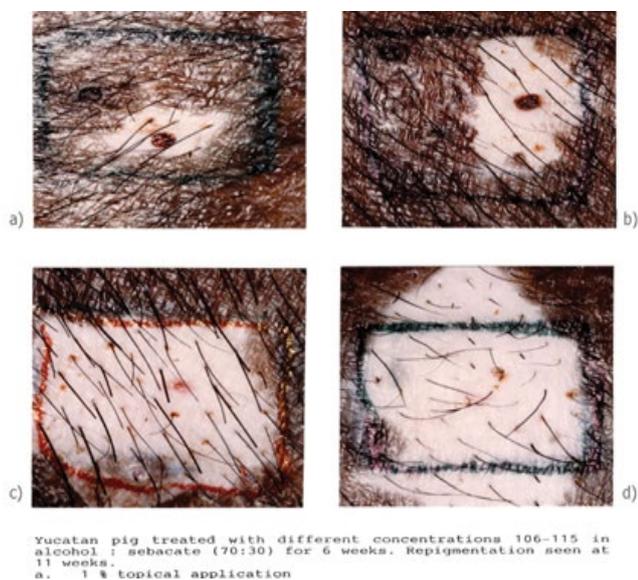
Figure 4: Effect of bFGFRP on the repigmentation of depigmented patches of guinea pig ear lobes compared to controls.



Figure 6: Representative Photographs before & after treatment with bFGFRP.



Figure 7: Combinatorial therapy of Vitiligo with bFGFRP + PUV-A SOL.



Yucatan pig treated with different concentrations 106-115 in alcohol : sebacate (70:30) for 6 weeks. Repigmentation seen at 11 weeks.
a. 1% topical application

Figure 5: Topical application of (bFGFRP) at varying concentrations on depigmented patches of skin of Yucatan swine.



Figure 8: Combinatorial therapy of Vitiligo Resistant to PUV-A with bFGFRP +PUV-A.



Figure 10: Combinatorial therapy of fast spreading Vitiligo with bFGFRP+ OMP.



Figure 9: That this combinatorial therapy was successful even in repigmenting most recalcitrant macules in the palms was shown below.



Figure 11: Effect of bFGFRP application after punch Graft.