J Cosmo Trichol 2016, 2:3(Suppl)
http://dx.doi.org/10.4172/2471-9323.C1.003

Expression of caspases-8, -9 and -3 in vitiligo

Maryham S Ibrahim[1], **Michel R Hanna**[1, 2], **Mohamed A El-Khayyat**[1], **Sherif S Awad**[1] and **Fatma Y Saleh**[1]
[1]Minia University, Egypt
[2]Burnham-Prebys Medical Discovery Institute, USA

Vitiligo is a multifactorial polygenic disorder with a complex pathogenesis. The precise cause behind melanocyte destruction remains unknown. The epidermal melanocytes form a functional and structural unit with neighboring keratinocytes. The keratinocytes produce certain growth factors required for melanocyte growth, and damage to keratinocytes may result in passive melanocyte death with the development of vitiligo. We performed this study to confirm the role of apoptosis in the pathogenesis of vitiligo through studying the expression of caspases-8,-9 and -3 and to determine the relation between the disease activity and the expression of these apoptotic markers. Twenty skin biopsies were obtained from the edge of vitiligo lesion. Immunohistochemical staining for caspases-8, -9 and -3 was carried out. We demonstrated the expression of these apoptotic markers within both, the epidermis and the dermal lymphocytes. We found that the expression of caspases-8,-9 and -3 was higher in depigmented epidermis when compared to normally pigmented epidermis either from vitiligo patients or from the normal control. The majority of apoptotic keratinocytes was found in the basal and suprabasal layers of epidermis. Regarding disease activity, the expression of these apoptotic markers was significantly higher in cases with active disease when compared to those with stable disease. Also, these apoptotic markers were expressed in the dermal lymphocytes. In conclusion vitiligo is not a disease limited to melanocyte death. Apoptosis of keratinocytes also clearly occurs and may play an important role in the development of the disease.

mishmish13@hotmail.com

Hair follicle regeneration from human pluripotent stem cells

Michel R Hanna[1, 2], **Yang Liu**[2], **Hasan El-Fakahany**[1], **Walid Medhat**[1], **Hamza Abdel-Raouf**[1] and **Evan Y Snyder**[2]
[1]Minia University, Egypt
[2]Sanford-Burnham-Prebys Medical Discovery Institute, USA

Hair follicle (HF) morphogenesis and regeneration depend on intensive and reciprocal interactions between epithelial and mesenchymal components. Currently, attempts to regenerate HFs depend on combining receptive epithelial and trichogenic dermal mesenchymal components and grafting them into an in vivo environment. Unfortunately, human HF bulge stem cells (BSCs) are not suitable for this purpose because the isolation and propagation of human HF BSCs for tissue engineering purposes remains a challenge. Here, we developed a strategy to differentiate human embryonic stem cells (hESCs) and induced pluripotent stem cells (hiPSCs) into $CD200^+/ITGA6^+$ BSCs that can reconstitute the epithelial components of the HF. Importantly, co-transplantation of hESCs or hiPSC-derived $CD200^+/ITGA6^+$ cells with trichogenic mice dermal cells into immunodeficient Nude mice resulted in HF formation. Histological analysis revealed that the obtained HFs possess all HF lineages including the hair shaft, and the inner and outer root sheaths. Human HF stem cell markers such as keratin-15 were detected in reconstituted HFs. The human origin of the epithelial cells in the new HFs was confirmed by positive reactivity for human-specific nuclear antigen. In this context derivation of functional HF BSCs capable of inducing a new hair formation suggest a major step toward developing cell-based treatments for alopecia.

mibrahim@sbpdiscovery.org