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Prevention of Hair Loss by Scalp Cooling During Chemotherapy

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Description

Hair graying is an early and obvious phenotypic and physiological trait with age in humans. Several recent advances in molecular biology and genetics have increased our understanding of the mechanisms of hair graying, which elucidate genes related to the synthesis, transport, and distribution of melanin in hair follicles, as well as genes regulating these processes above. Therefore, we review these advances and examine the trends in the genetic aspects of hair graying from enrichment theory, Genome-Wide association studies, whole exome sequencing, gene expression studies, and animal models for hair graying with age, aiming to overview the changes in hair graying at the genetic level and establish the foundation for future research. Meanwhile, by summarizing the genetics, it's of great value to explore the possible mechanism, treatment, or even prevention of hair graying with age. A major hair care concern for women is thinning hair, which occurs during aging. The causes of age-related hair thinning in women are complex and multifactorial, including influences such as hormonal changes, genetic susceptibility, and environmental, along with other changes that occur during chronological aging. There is growing evidence that cellular senescence is a major contributor to age-related decline in cellular activity. Given the role of senescence in the aging phenotype, it is of great interest to evaluate its potential role in aging hair. Previous studies have shown that Dermal Papilla Cells (DPC) from balding hair follicles were more susceptible to go into senescence after exposure to oxidative stress and growth inhibition by androgens than DPC from non-balding hair follicles. One source of oxidative stress, is known to naturally accumulate in the aging hair follicle, and this increase in has been linked to a decrease in naturally occurring detoxifying enzymes, such as catalase, which is a major mechanism for detoxification.

DNA Damage

It is well established that can induce sufficient DNA damage to lead to cellular senescence, *in vitro*. Herein we report the development of a high-throughput screening assay, using low, repeat dosing of to induce senescence. In this assay, over 40 actives were screened for their ability to stimulate the proliferation of hair follicle dermal papilla cells and protect against induced cellular senescence. Of the promising actives, three types of activities were observed actives that stimulated cellular proliferation without an observable protection against induced senescence. Actives that stimulated cellular proliferation and protected against induced senescence. Actives that protected against induced senescence with no cellular proliferative activity. Hair follicles (HFs) enjoy a relative Immune Privilege (IP) that is characterized by downregulation of Major Histocompatibility Complex (MHC) class I and local expression of potent immunosuppressants. Normally, Natural Killer (NK) cells attack cells with absent/low MHC class I expression. However, because few perifollicular NK cells are found around healthy human anagen HFs, we asked how HFs escape from NK cell attack. This study suggests that this happens via an active NK cell suppression. Alopecia Aerate (AA), an organ-specific autoimmune disease thought to result from a collapse of HF-IP, in contrast, and shows striking defects in NK cell inhibition/containment. We show that the NK cell inhibitor macrophage migration inhibitory factor is strongly expressed by the HF epithelium, and very few NK cells are observed in and around normal anagen HFs compared to AA with prominent aggregations of NK around. By flow cytometry, many fewer NK function-activating receptors and significantly more killer cell Ig-like were found to be expressed on peripheral blood $CD56_+$ NK cells of healthy controls than on those of AA patients. In addition, only weak immunoreactivity for MHC class I chain-related A gene was observed in normal anagen HFs compared to AA. To our knowledge, this defect is previously unreported and must be taken into account in AA pathogenesis and its management. Hair graying in mice is caused by various injuries such as X-ray radiation and repeated plucking that ultimately damage melanocytes and their stem cells (melanocyte stem cells). In X-ray–induced hair graying, injuries first manifest as a loss-of-niche function of hair follicular keratinocyte stem cells to maintain melanocyte stem cells. Thus, we hypothesized that hair follicular keratinocyte stem cells could be a practical target to prevent hair graying.

Preventing Hair

We found that hydroxygenkwanin exerted a remarkable effect in preventing hair graying; however, when receptor Y kinase Kit-mutant mice were used, no prevention effect was observed. Therefore, we propose that Kit signaling might be involved in the hydroxygenkwanin-induced protective effect against hair graying. Finasteride, a selective Type II 5α-reductase inhibitor,

has been approved as an oral drug for the treatment and prevention of Androgenetic Alopecia (AGA). The daily administration of finasteride by oral route may lead to various undesirable systemic side effects. In the present study, Finasteride Niosomes (FIN-NIS) as a dermal topical delivery system were developed evaluated in an attempt to overcome the limitations of the oral administration of finasteride, enhance the retention of finasteride into hair follicles, and cause fewer adverse effects. FIN-NIS were prepared by ethanol injection method and characterized for particle size, Entrapment Efficiency (EE), *in vitro* release and *in vitro* skin retention. Testosterone-induced AGA mice model was used to evaluate the therapeutic effect of FIN-NIS. The results show that the particle size of FIN-NIS was about 260 nm with a good loading capacity, and the EE was greater than 90%. FIN-NIS had the sustained-release effect to some extent. Compared with the hydroethanolic solution of finasteride (FIN-hydroethanol) and finasteride suspension (FIN-suspension), FIN-NIS showed higher drug concentration in the excised rat skin, especially in the hair follicle area. The drug accumulation of FIN-NIS in the hair follicles was 20 times higher than that of FIN-hydroalcohol. In addition, FIN-NIS was more effective to promote hair regeneration in AGA mice than FIN-suspension, and the effectiveness of FIN-NIS even exceeded that of minoxidil. In conclusion, FIN-NIS could become a promising dermal topical delivery system for enhancing the performance of finasteride for the management of AGA and avoiding the possible risk of systemic side effects associated with oral administration. In this study, we investigated the in vivo effect of the flavonoid hydroxygenkwanin, which has been shown to exert the best protection on human epidermal keratinocytes against in vitro X-ray–induced cytological effects, using X-ray–induced and repeated hair plucking–induced hair graying mice models.