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Graying: gerontobiology of the hair follicle pigmentary unit

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Abstract

The visual appearance of humans derives predominantly from their skin and hair color. The phylogenetically ancient biochemical [corrected] pathway underling this phenomenon is called melanogenesis and results in the production of melanin pigments in neural crest-derived melanocytes, followed by its transfer to epithelial cells. While melanin from epidermal melanocytes clearly protects human skin by screening harmful ultraviolet radiation, the biologic value of hair pigmentation is less clear. In addition to important roles in social/sexual communication, one potential benefit of pigmented scalp hair in humans may be the rapid excretion of heavy metals, chemicals, toxins from the body by their selective binding to melanin. The hair follicle and epidermal melanogenic systems are broadly distinct, though open. The primary distinguishing feature of follicular melanogenesis, compared to the continuous melanogenesis in the epidermis, is the tight coupling of hair follicle melanogenesis to the hair growth cycle. This cycle appears to involve periods of melanocyte proliferation (during early anagen), maturation (mid to late anagen) and melanocyte death via apoptosis (during early catagen). Thus, each hair cycle is associated with the reconstruction of an intact hair follicle pigmentary unit... at least for the first 10 cycles or so. Thereafter, gray and white hairs appear, suggesting an age-related, genetically regulated exhaustion of the pigmentary potential of each individual hair follicle. Melanocyte aging may be associated with reactive oxygen species-mediated damage to nuclear and mitochondrial DNA with resultant accumulation of mutations with age, in addition to dysregulation of anti-oxidant mechanisms or pro/anti-apoptotic factors within the cells. While the perception of "gray hair" derives in large part from the admixture of pigmented and white hair, it is important to note that individual hair follicles can indeed exhibit pigment dilution or true grayness. This dilution is due to a reduction in tyrosinase activity of hair bulbar melanocytes, sub-optimal melanocyte-cortical keratinocyte interactions, and defective migration of melanocytes from a reservoir in the upper outer root sheath to the pigment-permitting microenvironment close to the dermal papilla of the hair bulb. Animal models with mutations in apoptotic survival factors (e.g. bcl-2) and in melanogenic enzymes (TRP-1) are providing valuable insights into the aging hair pigmentary unit. It is from these and other advances, including our ability to grow hair follicle melanocytes in vitro, that the possibility of reversing canities has been raised. Indeed, it is not too uncommon to see spontaneous repigmentation along the same individual hair

shaft in early canities. Moreover, melanocytes taken from gray and white hair follicles can be induced to pigment in vitro. One of the surprising results of pigment loss in canities is the alteration in keratinocyte proliferation and differentiation, providing the tantalizing suggestion that melanocytes in the hair follicle contribute far more that packages of melanin alone. Furthermore, there have been some unconfirmed reports in the literature suggesting that canities may link (although not causally) with more systemic alterations in homeostasis e.g. osteoporosis. Here, we review the current state of knowledge of the development, regulation and control of the human hair follicle pigmentary unit during life.

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