



Oxidatív stressz és a hajhullás összefüggése

(Egyelőre nincs jelentős mennyiségű tapasztalat e tekintetben.

A probléma, tehát az oxidatív stressz szerepe mindenesetre felvetődött. További vizsgálatok szükségesek.

Az oxidatív stressz szűrése és korrekciója mindenképpen hasznos)

1. Cell Biochem Funct. 2000 Sep;18(3):169-73.

Antioxidants and lipid peroxidation status in the blood of patients with alopecia.

Naziroglu M, Kokcam I.

Department of Physiology, Veterinary Faculty of Firat University, 23119 Elazig, Turkiye.

The aim of this research was to determine levels in blood of vitamin E, beta-carotene, lipid peroxidation as thiobarbituric-acid reactive substances (TBARS) and reduced glutathione (GSH) and activity of glutathione peroxidase (GSH-Px) in patients with alopecia. Studies were carried out on 37 patients with alopecia and 34 healthy age-matched controls. Red blood cell (RBC) and plasma samples from healthy and patient subjects were taken. Beta-carotene levels ($P < 0.001$) in plasma and levels of GSH ($P > 0.05$) and the activity of GSH-Px ($P < 0.05$, $P < 0.01$) in both plasma and RBC samples were significantly lower in patients with alopecia than in controls, whereas TBARS levels in plasma ($P < 0.05$) and RBC ($P < 0.001$) samples were significantly higher in patients with alopecia than in controls. However, vitamin E levels in plasma did not differ statistically. Although being far from conclusive, these results provide some evidence for a potential role of increased lipid peroxidation and decreased antioxidants in alopecia. Copyright 2000 John Wiley & Sons, Ltd.

PMID: 10965354 [PubMed - indexed for MEDLINE]

(Távolról sem egységesek a tapasztalatok, de számos bizonyíték van az oxidatív stressz és csökkent antioxidáns védelem és hajhullás összefüggéséről)

2. Clin Interv Aging. 2006;1(2):121-9.

Pharmacologic interventions in aging hair.

Trüeb RM.

Department of Dermatology, University Hospital of Zurich, Zurich, Switzerland.
ralph.trueeb@usz.ch

The appearance of hair plays an important role in people's overall physical appearance and self-perception. With today's increasing life-expectations, the desire to look youthful plays a bigger role than ever. The hair care industry has become aware of this and is delivering active products directed towards meeting this consumer demand. The discovery of pharmacological targets and the development of safe and effective drugs also indicate strategies of the drug industry for maintenance of healthy and beautiful hair. Hair aging comprises weathering of the hair shaft, decrease of melanocyte function, and decrease in hair production. The scalp is subject to intrinsic and extrinsic aging. Intrinsic factors are related to individual genetic and epigenetic mechanisms with interindividual variation: prototypes are familial premature graying, and androgenetic alopecia. Currently available pharmacologic treatment modalities with proven efficacy for treatment of androgenetic alopecia are topical minoxidil and oral finasteride. Extrinsic factors include ultraviolet radiation and air pollution. Experimental evidence supports the hypothesis that oxidative stress



also plays a role in hair aging. Topical anti-aging compounds include photoprotectors and antioxidants. In the absence of another way to reverse hair graying, hair colorants remain the mainstay of recovering lost hair color. Topical liposome targeting for melanins, genes, and proteins selectively to hair follicles are currently under investigation.

Publication Types:
Review

PMID: 18044109 [PubMed - indexed for MEDLINE]

(Kísérletes bizonyítékok támasztják alá a feltételezést, mi szerint az oxidatív stressz a haj öregedésében is szerepet játszik. Gyakran sikerrel alkalmaznak helyi antioxidáns hatású készítményeket)

3. FASEB J. 2009 Jul;23(7):2065-75. Epub 2009 Feb 23.

Senile hair graying: H₂O₂-mediated oxidative stress affects human hair color by blunting methionine sulfoxide repair.

Wood JM, Decker H, Hartmann H, Chavan B, Rokos H, Spencer JD, Hasse S, Thornton MJ, Shalhaf M, Paus R, Schallreuter KU.

Clinical and Experimental Dermatology/Department of Biomedical Sciences, University of Bradford, Bradford, BD7 1DP, West Yorkshire, UK.

Senile graying of human hair has been the subject of intense research since ancient times. Reactive oxygen species have been implicated in hair follicle melanocyte apoptosis and DNA damage. Here we show for the first time by FT-Raman spectroscopy in vivo that human gray/white scalp hair shafts accumulate hydrogen peroxide (H₂O₂) in millimolar concentrations. Moreover, we demonstrate almost absent catalase and methionine sulfoxide reductase A and B protein expression via immunofluorescence and Western blot in association with a functional loss of methionine sulfoxide (Met-S=O) repair in the entire gray hair follicle. Accordingly, Met-S=O formation of Met residues, including Met 374 in the active site of tyrosinase, the key enzyme in melanogenesis, limits enzyme functionality, as evidenced by FT-Raman spectroscopy, computer simulation, and enzyme kinetics, which leads to gradual loss of hair color. Notably, under in vitro conditions, Met oxidation can be prevented by L-methionine. In summary, our data feed the long-voiced, but insufficiently proven, concept of H₂O₂-induced oxidative damage in the entire human hair follicle, inclusive of the hair shaft, as a key element in senile hair graying, which does not exclusively affect follicle melanocytes. This new insight could open new strategies for intervention and reversal of the hair graying process.

PMID: 19237503 [PubMed - indexed for MEDLINE]

(Kísérletes bizonyítékok szerint a hidrogéperoxid keltette oxidatív stressz az öregedéssel járó őszülés kulcsfontosságú eleme.)

4. Biol Trace Elem Res. 2010 Jan 5. [Epub ahead of print]

Fluorine-Induced Apoptosis and Lipid Peroxidation in Human Hair Follicles In Vitro.

Wang ZH, Li XL, Yang ZQ, Xu M.

Department of Otolaryngology-Head and Neck Surgery, the Second Hospital, Xi'an Jiao Tong University, Xi'an, 710004, China, ehui4298@163.com.

Fluoride is an essential trace element for human body; however, exposure to high amounts of fluoride has been documented to be correlated with an increasing risk of hair loss. To date, little is



known about the mechanism(s) of how fluoride affects hair follicles. Here, we demonstrated that middle (1.0 mmol/L) and high (10.0 mmol/L) concentrations of sodium fluoride (NaF) significantly inhibited hair follicle elongation in vitro, but low NaF (0.1 mmol/L) showed little influence. Moreover, treatment with high levels of NaF resulted in a marked increase in terminal dUTP nick end labeling-positive cells in the outer layer of the outer root sheath, the dermal sheath, and the lower bulb matrix surrounding dermal papilla. Furthermore, the enhanced apoptosis was coupled with an increased oxidative stress manifested as higher malondialdehyde content. Additionally, the presence of selenium considerably antagonized the effects of middle NaF on hair follicles, with regard to either the suppression of hair growth or the induction of oxidative stress and apoptosis. In conclusion, exposure to high levels of fluoride compromises hair follicle growth and accelerates cell apoptosis in vitro. The toxicity of fluoride can be reduced by selenium, at least partially via the suppression of intracellular oxidative stress.

PMID: 20049553 [PubMed - as supplied by publisher]

(A fluorid többszörösen igazolt hatása: a hajvesztés sebességének fokozása... A fluorid fenti hatása kivédhető szeléniummal. Ennek oka a sejten belüli oxidatív stressz csökkentése)

5. Micron. 2004;35(3):193-200.

Hair cycle and hair pigmentation: dynamic interactions and changes associated with aging.

Van Neste D, Tobin DJ.

Skinterface, 9, rue du Sondart, B-7500 Tournai, Belgium. info@skinterface.be

The tight coupling of hair follicle melanogenesis to the hair growth cycle dramatically distinguishes follicular melanogenesis from the continuous melanogenesis of the epidermis. Cyclic reconstruction of an intact hair follicle pigmentary unit occurs optimally in all scalp hair follicles during only the first 10 hair cycles, i.e. by approximately 40 years of age. Thereafter there appears to be a genetically regulated exhaustion of the pigmentary potential of each individual hair follicle leading to the formation of true gray and white hair. Pigment dilution results primarily from a reduction in tyrosinase activity within hair bulbar melanocytes. Thereafter, 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 follicular papilla of the hair bulb, will all disrupt normal function of the pigmentary unit. Evidence from studies on epidermal melanocyte aging suggest that reactive oxygen species-mediated damage to nuclear and mitochondrial DNA may lead to mutation accumulation in bulbar melanocytes. Parallel dysregulation of anti-oxidant mechanisms or pro/anti-apoptotic factors is also likely to occur within the cells. Pigment loss in canities may also affect keratinocyte proliferation and differentiation, providing the tantalizing suggestion that melanocytes in the hair follicle contribute far more than packages of pigment alone. Here, we review the current state of knowledge of the development, regulation and control of the aging human hair follicle pigmentary system in relation with hair cycling. The exploitation of recently available methodologies to manipulate hair follicle melanocytes in vitro, and the observations that melanocytes remain in senile white hair follicles that can be induced to pigment in culture, raises the possibility of someday reversing canities. The perspective of rejuvenation of the whole hair follicle apparatus are still part of the dream but optimizing its functional properties is clinically relevant and is close to reality. Finally as hair color influences its visibility when optical methods such as scalp photography are used to count hair fibers, the attention is drawn to possible interpretations of statistically significant changes in visible hair. Such changes may not exclusively be related to improved hair growth itself but also to changes in natural hair color that makes the hair more visible with the method used to count hairs.

PMID: 15036274 [PubMed - indexed for MEDLINE]



(Tudományos bizonyítékok szerint a bőr melanocitáinak öregedése oxidatív stressz által kiváltott, a sejtmagban és a mitokondriumokban bekövetkező DNS károsodás következménye)

6. J Dermatol Sci. 2002 Aug;29(2):85-90.

Antioxidant enzymes and lipid peroxidation in the scalp of patients with alopecia areata.

Akar A, Arca E, Erbil H, Akay C, Sayal A, Gür AR.

Department of Dermatology, GATA School of Medicine, 06018, Ankara, Turkey.
aakar@gata.edu.tr

Alopecia areata (AA) is an autoimmune inflammatory disease. However, little is known about the alterations in lipid peroxidation and antioxidant enzymes in the scalp of patients with AA. Therefore, the aim of this study was to investigate the status of oxidative stress in the scalp of patients with AA. We measured the levels of thiobarbituric acid reactive substances (TBARS) as lipid peroxidation status, superoxide dismutase (SOD) and glutathione peroxidase (GSH-Px) as antioxidant enzymes in the scalp of ten patients with AA and ten control subjects. The levels of TBARS in scalp of patients with AA (3654.1±621.2 nmol/g tissue) were significantly higher than those of controls (1210.2±188.8 nmol/g tissue) (P=0.002). The levels of SOD (134.8±23.8 U/g tissue) and GSH-Px (332.7±66.2 U/g tissue) in scalp of patients with AA were also significantly higher than those of controls (63.2±8.8 U/g tissue, 112.0±18.4 U/g tissue, respectively) (P=0.019, P=0.002, respectively). The mean levels of TBARS, SOD and GSH-Px in early phase of disease were increased 2-fold as compared with late phase of the disease. These results indicate that oxidative status is affected in AA. Lipid peroxidation and antioxidant enzymes may be involved in the pathogenesis of AA. Furthermore, we found high SOD and GSH-Px activities in the scalp of patient with AA. These high levels could not protect the patients against the reactive oxygen species, because lipid peroxidation could not be lowered in AA patients.

PMID: 12088608 [PubMed - indexed for MEDLINE]

(A TBAR, SOD és GSH-Px anyagok szintje kétszeresére emelkedett; ez bizonyítja az oxidatív stressz szerepét foltos kopaszságban)